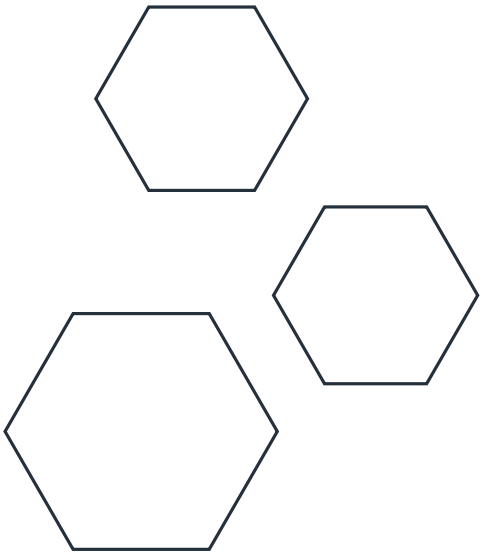
EC MTB 2026

ECMTB 2026

Book of Abstracts

14th European Conference on Mathematical and Theoretical Biology

Joint event by SMB and ESMTB · Graz, Austria



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We thank our partners and sponsors for their generous support of ECMTB'26.

Welcome

Dear Participants,

A warm welcome to the University of Graz, the host of the 14th European Conference on Mathematical and Theoretical Biology 2026 (ECMTB), a joint event by the Society for Mathematical Biology (SMB) and the European Society for Mathematical and Theoretical Biology (ESMTB).

It is a true pleasure to welcome each and every one of almost 1300 participants from around the world. The local organisers have the honour in wishing you a most productive and inspiring conference.

We would like to express a most appreciative Thank You to the University of Graz headed by Rector Peter Riedler, in providing the venue and its facilities. In particular, we are very grateful to the extraordinary support received from the event management, facility management, human resources, UniIT, cleaning services and all the many people contributing in organising such an event. Without their dedicated support ECMTB 2026 would not have been possible.

As local organisers, we are most grateful to ESMTB and SMB for the constructive and trustworthy dialogue to bring ECMTB 2026 to life. We also warmly acknowledge our sponsors, the Styrian government, the Field of Excellence COLIBRI, the National Institute for Theory and Mathematics in Biology, BioTechMed and Numericor. A very special thank you also to the colleagues of Graz Tourism with its great support for hotel bookings and social events.

Finally, I would like to personally express my deepest gratitude to all my colleagues of the local organisers, who have put in such an enormous workload.

Klemens Fellner and the local organisers

Talks

Code	Title	Speaker(s)	Date / time
PLT-01	Mathematical modeling of spatial evolution with applications to biomedical systems	Natalia L. Komarova	mon, 09:20
MS124-02	Empirical and mechanistic modeling of SARS-CoV-2 transmission in the United States to design or evaluate and possibly improve mitigation measures	John Glasser	mon, 10:40
MS127-01	Mechanosensitive Feedback Organizes Cell Shape and Motion During Hindbrain Neuropore Morphogenesis	Fernanda Pérez Verdugo	mon, 10:40
MS145-01	From Cell Alignment to Axonal Pathfinding: Mechanics as a Guidance Cue	Chiara Giverso	mon, 10:40
MS146-01	Microbial community metabolic models of the human gut microbiome: From objectives to validation	Christian Diener	mon, 10:40
MS163-01	The whole baby PK/PD model of anti-VEGF therapy that big pharma doesn't want you to see	Jessica Crawshaw	mon, 10:40
MS164-01	Mosquito biting heterogeneity and the community impact of targeted malaria interventions	Emma Fairbanks	mon, 10:40
MS18-01	Qualitative properties for certain PDEs derived from neuroscience	Delphine Salort	mon, 10:40
MS34-03	Dynamical modeling of phenotypic plasticity in melanoma reveals strategies of drug-induced adaptation	Sarthak Sahoo	mon, 10:40
MS36-01	Developing a handbook of mathematical modelling of infectious diseases for decision-making	Philip Gerlee	mon, 10:40
MS38-01	Field theories and quantum methods for multiscale complex systems	Mauricio del Razo Sarmina	mon, 10:40
MS40-01	A Stage-Structured Seasonal Model for the Spread of Flavescence Dorée in Vineyards	Attila Denes	mon, 10:40
MS55-05	An Invitation to Reaction Networks	Gheorghe Craciun	mon, 10:40
MS61-01	Deciphering Alzheimer's Disease Dynamics: Modelling Protein Pathology, Inflammation, and Therapeutic Strategies	Georgia Brennan	mon, 10:40
MS62-12	Biofilm Rheology: Properties, Applications, and Parameter Estimation	Nicholas Cogan	mon, 10:40
MS69-01	Spatial immune-tumor ecology in the multiple myeloma bone niche: an agent-based modeling approach	David Basanta	mon, 10:40
MS70-01	A cell–cell adhesion model: a multiscale derivation	Anna Zhigun	mon, 10:40
MS86-01	Data-Based Calibration and Fast–Slow Decomposition of a Patient-Specific Hypothalamus–Pituitary–Thyroid Axis Model	Clara Horvath	mon, 10:40
MS87-01	Data-Driven Modelling of Cell Cycle Regulation	Luke De Bretton-Gordon	mon, 10:40
MS88-01	Predicting outcomes of combination treatment of Tuberculosis using a multi-scale quantitative systems pharmacology model	Christian Michael	mon, 10:40
MS127-02	Linking vertex models with experiments in tissue morphogenesis	Gonca Erdemci-Tandogan	mon, 11:00
MS146-02	Thermodynamic Feasibility in Genome-Scale Microbial Consortia	Jürgen Zanghellini	mon, 11:00
MS163-03	How Mechanical Forces Shape Stratification and Wound Healing Dynamics in the Corneal Epithelium	Neda Khodabakhsh Joniani	mon, 11:00
MS164-02	Simulating Mosquito Flight Paths with a Random Walk Model	Luzia Nora Felber	mon, 11:00
MS18-02	Disentangling pulse-coupled oscillators in the mean-field regime through the pseudo-inverse in a dilated timescale	Pierre Roux	mon, 11:00
MS34-04	Critical assessment of Waddington's Epigenetic Landscape and its modern interpretations	Eric Fanchon	mon, 11:00
MS36-02	Interactive education and interdisciplinary communication via VisualPDE	Benjamin Walker	mon, 11:00
MS38-02	Multi-resolution simulations of intracellular processes	Radek Erban	mon, 11:00
MS40-02	On Multiscale Modeling of Vector-Borne Diseases	Jorge Velasco	mon, 11:00
MS61-02	Spreading of pathological proteins through brain networks	Samira Breitling	mon, 11:00
MS62-09	Modeling of biofouling dynamics in membrane filtration systems	Vincenzo Luongo	mon, 11:00
MS69-02	Integrating longitudinal MRI and clinical data into a biomechanistic tumor growth model for spatial forecasting of prostate cancer aggressiveness	Guillermo Lorenzo	mon, 11:00
MS70-02	Scaling limits for a population model with growth, division and cross-diffusion	Diane Peurichard	mon, 11:00
MS86-02	Mathematical modelling of thyroid response to incidental radiotherapy exposure	Isabel Gonzalez Crespo	mon, 11:00
MS87-02	Using inference to obtain models for biological systems	Marta Sales Pardo	mon, 11:00
MS88-02	A mathematical model of TCR-T cell therapy	Doron Levy	mon, 11:00
MS124-01	Long-range forecasting of seasonal influenza vaccine uptake using web search data	Edward Thommes	mon, 11:20
MS127-03	Cell-cell heterogeneities in vertex models for tissue mechanics	Oliver Drozdowski	mon, 11:20
MS145-03	Bayesian parameter inference of stochastic two-population cell movement models with spatial statistics	Duncan Martinson	mon, 11:20
MS146-03	Elementary vectors reveal minimal interactions in microbial communities	Stefan Müller	mon, 11:20
MS163-04	Estimating effective diffusion in the vitreous: A profile likelihood approach	Patricia Lamirande	mon, 11:20
MS164-03	Immunity-driven biases in malaria incidence: a study for Senegal and The Gambia	Miguel Garrido Zornoza	mon, 11:20
MS18-03	Navigational-enabling settings of a PDE modelling noisy grid cell activity	Suzanne Solem	mon, 11:20

Code	Title	Speaker(s)	Date / time
MS34-02	Analyzing the impact of proliferation and treatment parameters on low-grade glioma growth using mathematical models	Maria Vela Pérez	mon, 11:20
MS36-03	Using bibliometrics to analyse trends in mathematical biology	Kira Pugh	mon, 11:20
MS38-03	Adding Drift to Stochastic Reaction-Diffusion Processes	Samuel Isaacson	mon, 11:20
MS40-03	Underlying Complexity in Dengue Reinfection Dynamics: Backward Bifurcation and Serotype Invasion in a Multi-Serotype Model	Mostafa Adimy	mon, 11:20
MS55-04	From Continuous-Time Markov Chains to Spatial Stochastic Simulation of Biochemical Reaction Networks	Hye-Won Kang	mon, 11:20
MS61-03	A mathematical model for tau aggregation and diffusion: An approach via two-scale homogenization of the Smoluchowski equation with transmission boundary conditions	Silvia Lorenzani	mon, 11:20
MS62-11	Mixing and transport in the Drosophila melanogaster oocyte	David Stein	mon, 11:20
MS69-03	How evolvability dynamics affect tumor growth and treatment response	Jill Gallaher	mon, 11:20
MS70-03	Spatial phase transition in collective dynamics	Léo Meyer	mon, 11:20
MS86-03	Hormone level estimation in the pituitary-thyroid axis with infrequent sampling	Seth Siriya	mon, 11:20
MS87-03	Making PINNs ready for parameter inference from noisy data	Roman Vetter	mon, 11:20
MS88-03	Illuminating antigen presentation dynamics in the pancreatic cancer microenvironment with omics-informed agent-based modeling	Daniel Bergman	mon, 11:20
MS124-04	Modelling disease transmission with game theoretic determinants: a look at MPOX modelling	Monica Cojocaru	mon, 11:40
MS127-04	A quantitative vertex model of neural tube closure informed by imaging data	Rubén Perez-Carrasco	mon, 11:40
MS145-04	A Physiologically Based Pharmacokinetic (PBPK) Model of Whole-Body CAR T-Cell Trafficking in Humans	Elio Campanile	mon, 11:40
MS146-04	Automated Discovery of Mechanistic Models for Microbial Communities	Artai Rodríguez Moimenta	mon, 11:40
MS163-02	Heterogeneity, Bias, and Translation Bottleneck in Age-Related Macular Degeneration	Moussa Zouache	mon, 11:40
MS164-04	Identifying robust triggers for controlling epidemic waves in real-time	Nathan Doyle	mon, 11:40
MS18-04	Resolving the Blow-Up: A Time-Dilated Numerical Framework for Multiple Firing Events in Mean-Field Neuronal Networks	Zhennan Zhou	mon, 11:40
MS34-01	Uncovering underlying physical principles and driving forces of cancer from single-cell transcriptomics	Jin Wang	mon, 11:40
MS36-04	A Sensational Approach to Inclusive STEM outreach	Parmvir Kaur Bahia	mon, 11:40
MS38-04	Coarse-Grained Modeling of Particle Clustering Dynamics	Stefanie Winkelmann	mon, 11:40
MS40-04	Modelling West Nile spread in a habitat heterogeneous in space and time	Andrea Pugliese	mon, 11:40
MS55-03	Stochastic Reaction Networks: Definition, Classical Scaling and Long-Term Behavior	Daniele Cappelletti	mon, 11:40
MS61-04	Linking Astrocyte Morphology to Metabolic Dysfunction in Alzheimer's Disease: A Multiscale Modelling Approach	Sofia Farina	mon, 11:40
MS62-10	Modeling and Optimization of Microfluidic Spheroid-on-Chip Systems Using Computational Fluid Dynamics and Neural Network–Based Bayesian Optimization	Mohsen Rezaeian	mon, 11:40
MS69-04	Quantifying Tissue Architecture in the Tumour Microenvironment with Multi-Parameter Persistent Homology	Kylie Savoye	mon, 11:40
MS70-04	Emergent modes of collective cell invasion: a multiscale perspective	Martina Conte	mon, 11:40
MS86-04	Human-Readable Tabular Reinforcement Learning for Dosing Decisions in Graves' Disease	Thomas Benninger	mon, 11:40
MS87-04	Towards Inferring Biological Mechanisms with Physics-Informed Neural Networks	William Lavery	mon, 11:40
MS88-04	Towards Optimization of Monoclonal Antibody Therapeutics Using a 3D Model of Cancer-Immune Interaction	Jordana O'Brien	mon, 11:40
PLT-02	Self-organisation in mouse development	Takashi Hiiragi	mon, 14:00
MS11-01	Computational Approaches to Multiple Sclerosis: Immune Dynamics and Data-Driven Lesion Modeling	Barbara Quintela	mon, 15:00
MS121-07	A Research Program on Modularity in Biological Systems	Reinhard Laubenbacher	mon, 15:00
MS124-05	Integrating Machine Learning Techniques in Mathematical Models of Dengue Transmission Dynamics	Naveen Vaidya	mon, 15:00
MS13-01	A harmful cycle: understanding the roles of inflammation, serotonin and the blood brain barrier in neurological development through mathematical models.	Ami Radunskaya	mon, 15:00
MS145-02	Multiscale modelling of cancer invasion and metastasis: the effects of tumour heterogeneity	Dimitrios Katsaounis	mon, 15:00
MS148-01	Multiscale mathematical modeling of pulsed field cardiac ablation	Annabelle Collin	mon, 15:00
MS173-01	The role of human behavior in shaping infectious disease dynamics	Chris Soria	mon, 15:00
MS22-01	Universal design for learning applied to the development of a new a second-year undergraduate statistics course	Adriana Zanca	mon, 15:00
MS29-01	The impact of sojourn distribution assumptions on malaria parasite transmission dynamics: a flexible within-host modeling framework to accommodate empirically driven distributions	Jordan Pellett	mon, 15:00

Code	Title	Speaker(s)	Date / time
MS31-01	Revealing the influence of cell motility on immune processes within complex tissue environments	Frederik Graw	mon, 15:00
MS39-01	Manifold robust perfect adaptation and structural limits on steady-state reachability	Ankit Gupta	mon, 15:00
MS41-01	Bifurcation Structures in Low-Dimensional Models of Ant–Coffee Berry Borer Dynamic	Gerard Olivar-Tost	mon, 15:00
MS42-01	Spatial organization of adhesion-deficient cells governs multiscale rigidity transitions in epithelial tissues	Pilar Guerrero	mon, 15:00
MS50-06	Biochemical reaction networks: systematic design, limit cycles and properties of Hilbert-like numbers	Radek Erban	mon, 15:00
MS52-06	PEtab-SciML: The missing layer for accessible SciML modelling	Sebastian Persson	mon, 15:00
MS53-01	The role of transient crosslinks in the chromatin search response to DNA damage	Caitlin Hult	mon, 15:00
MS62-14	From Langevin Functions to Macro Closures: Multiscale Modeling of Viscoelastic Biofluids	Paula Vasquez	mon, 15:00
MS74-07	Feeding Less, Modeling More: Can Mathematics Optimize Intermittent Fasting in Cancer?	Víctor M. Pérez-García	mon, 15:00
MS90-01	Phase i trials in cancer: From board to bench to bedside and back again	Alexander Anderson	mon, 15:00
MS11-03	Mass-conserving boundary motion in a model of invasion and recession	Georgia Weatherley	mon, 15:20
MS124-06	A dimension-reduction technique for modelling evolution in structured populations	Troy Day	mon, 15:20
MS13-02	Mathematical Modeling of the Gut-Brain Axis	Cornelia Mihaila	mon, 15:20
MS145-08	The biomechanics of the single cell-ECM interaction during migration	Nicolas Louviaux	mon, 15:20
MS173-02	Social Dilemma of Disease Control	Feng Fu	mon, 15:20
MS22-02	Mentoring First-Year STEM Students Through Collaborative Research in the Haynes Scholars Program	Alex Capaldi	mon, 15:20
MS29-02	Forecasting Malaria using test positivity and reported case rates	Katie Gurski	mon, 15:20
MS31-02	Beyond MIC: Defining a Minimum Inhibitory Dose (“MID”) for Periodic Antibiotic Treatments	Jessica Conway	mon, 15:20
MS39-02	Biological functions and functional modules originated in structure of chemical reaction network.	Atsushi Mochizuki	mon, 15:20
MS41-02	SIT-based control of Aedes aegypti mosquitoes featuring spatial heterogeneity	Olga Vasilieva	mon, 15:20
MS42-02	Modelling single and collective cell migration through confined non-isotropic environments using geometric surface PDEs	Anotida Madzamuse	mon, 15:20
MS50-03	A bimolecular reaction network with chaotic dynamics	Josef Hofbauer	mon, 15:20
MS52-03	Universal Differential Equations Trained on Remotely Sensed Datasets Can Identify Tipping Points in Large-Scale Ecosystems	Jack Buckner	mon, 15:20
MS53-02	Harnessing Discrete, Multiscale Modeling and Topological Data Analysis to Forecast Stem Cell Behavior	Daniel Cruz	mon, 15:20
MS62-15	Dynamic deformation and positioning of nucleus	Yuan-Nan Young	mon, 15:20
MS90-02	Tumours, risk, and waves: learning from multi-modal data to predict cancer outcomes	Chay Paterson	mon, 15:20
MS11-05	Exploring immune pathways and remyelination in Multiple Sclerosis	Maria Carmela Lombardo	mon, 15:40
MS121-06	Design principles of complex cellular decision-making networks in cancer	Mohit Kumar Jolly	mon, 15:40
MS124-07	Modeling the invasion of novel SARS-CoV-2 variants and their co-existence with or replacement of ancestral variants in the United States	Zhilan Feng	mon, 15:40
MS13-03	The Four Ds of Ovulation: Dynamics, Dysfunction, Disparities, and Dosing	Erica Graham	mon, 15:40
MS145-06	Heterogeneous elastic wires: a toy model for biological membranes	Gaspard Jankowiak	mon, 15:40
MS148-05	Computational assessment of wireless powering of a pulmonary artery intravascular sensor via volume conduction	Mar Gadea Saez	mon, 15:40
MS173-03	Behavioral Adaptation to Novel Pandemics: Learning to Move from Mobility Restrictions to Less Costly Measures	Binod Pant	mon, 15:40
MS22-03	Structured Onboarding of Undergraduate Researchers in Mathematical Biology	Ashley Ford Versypt	mon, 15:40
MS29-03	Modeling Insecticide Resistance and Management of Malaria Mosquitoes	Lihong Zhao	mon, 15:40
MS31-03	Hemogenic endothelium-derived multipotent progenitors shape prenatal hematopoiesis	Connor Shrader	mon, 15:40
MS39-06	The structural concept(s) of cores in reaction networks	Nicola Vassena	mon, 15:40
MS41-03	About the Combination of Control Tools Against the Oriental Fruit Fly Using a Metapopulation Approach	Yves Dumont	mon, 15:40
MS42-03	Mathematical Models for the Mechanics of Soft Tissues: From Linear Elasticity to Morpho-Visco-Poroelasticity	Sabia Asghar	mon, 15:40
MS50-04	Stability and Robustness in Biochemical Networks with Bifunctional Enzymes	Matthew Johnston	mon, 15:40
MS52-05	Guiding the optimal use of hybrid mechanistic and data-driven models for individual prediction of platelet dynamics	Marie Steinacker	mon, 15:40
MS53-03	Learning collective multicellular dynamics with an interacting mean field neural SDE model	Lin Wan	mon, 15:40
MS62-16	Monte Carlo simulations of membrane microfiltration for water purification	Francesca Bernardi	mon, 15:40
MS74-01	From patient vascular data to radiotherapy outcome prediction: a multiscale mechanistic framework	Cristina Macaluso	mon, 15:40
MS90-03	A Multiscale Physiologically Based Pharmacokinetic Model for mRNA-Encoded Therapeutics: Preclinical Predictions and Translational Perspectives	Elio Campanile	mon, 15:40

Code	Title	Speaker(s)	Date / time
MS121-01	Biconnected components and structural robustness in directed networks	Byungjoon Min	mon, 16:00
MS124-08	Waning immunity and the critical vaccination threshold	Jane Heffernan	mon, 16:00
MS13-04	Bridging Structured PDEs and ODE Models in Ovarian Follicle Dynamics	Claudio Iuliano	mon, 16:00
MS145-07	Towards optimal scaffolds for cell migration	Luigi Preziosi	mon, 16:00
MS148-03	Pulsed Field Ablation: electroporation based cardiac ablation	Damijan Miklavčič	mon, 16:00
MS173-04	SIR model with the distribution of preventive behaviors in a community	Zhiqiong Fu	mon, 16:00
MS22-04	Putting Math Bio in its Place	Kyle Dahlin	mon, 16:00
MS29-04	Likelihood-Free Confidence Regions for Stochastic Epidemic Models via CTMC Simulation and Neural Networks	Madison Pratt	mon, 16:00
MS31-04	Uncovering the T cell differentiation pathway	Mason Lacy	mon, 16:00
MS39-04	Cycling Reaction Network Robustness Analysis	Franco Blanchini	mon, 16:00
MS41-04	Modelling Movement and Stage-Specific Habitat Preferences of a Polyphagous Insect Pest	claudia ferreira	mon, 16:00
MS42-04	Contact guidance and ECM architecture jointly regulate cancer invasion	Fabian Spill	mon, 16:00
MS52-04	Theme and Variations: Conditional Universal Differential Equations for Biological Heterogeneity	Max de Rooij	mon, 16:00
MS53-04	Multi-scale regulation of organ growth through a gene-regulatory network that drives a transient proliferative signal	Marcos Nahmad	mon, 16:00
MS62-13	A spheroidal squirmer model for euglenids	Henry Shum	mon, 16:00
MS74-05	Multimodality Stacking with Blockwise missing values and application to the PIONeeR biomarkers study for prediction of resistance to immunotherapy	Mohamed Boussena	mon, 16:00
MS90-04	Identifying population interactions in multiple myeloma immunotherapy with SINDy	Ryan Woodall	mon, 16:00
MS11-06	Derivation from kinetic theory of chemotaxis models for Multiple Sclerosis	Marzia Bisi	mon, 17:00
MS117-01	Spatial dynamic modelling to understand how dendritic cell clustering affects T cell activation	Domenic Germano	mon, 17:00
MS121-02	Attractors are less stable than their basins: Canalization creates a coherence gap in gene regulatory networks	Claus Kadelka	mon, 17:00
MS124-09	Spatial Pattern Formation and the Evolution of Cooperative Behavior	Daniel Cooney	mon, 17:00
MS13-05	Models of Regulation and Disease Dynamics in Ovarian Cancer Invasion	Leili Shahriyari	mon, 17:00
MS148-02	A multiscale cardiac pulsed field ablation model: from cellular electroporation effects to lesion formation prediction	Argyrios Petras	mon, 17:00
MS173-05	Extending an SEIR behavioural model with structures for supply chain efficiency and vaccine allocation	Jim Duggan	mon, 17:00
MS22-05	Advances in Accessibility of Undergraduate Numerical Methods	Laura Ellwein Fix	mon, 17:00
MS27-02	Reaction-transmission delays in flocking models	Elisa Continelli	mon, 17:00
MS29-05	New approaches to old problems: data-driven models of dengue in emergent and endemic environments	Michael Robert	mon, 17:00
MS31-05	Modelling the HBV immune response: insights from virtual humanised mouse cohorts	Melanie Prague	mon, 17:00
MS39-05	Structural properties and biological oscillators: ideas from strongly 2-cooperative systems	Giulia Giordano	mon, 17:00
MS42-05	Emergence of Cellular Stress and Metabolic Dynamics within GBM progression	Dumitru Trucu	mon, 17:00
MS50-05	Alternative equations for studying bifurcations in mass action networks	Murad Banaji	mon, 17:00
MS52-08	Functional Identifiability for Universal Differential Equations	Torkel Loman	mon, 17:00
MS53-05	Geometric control and mechanics in growing biological tissues	Pascal Buenzli	mon, 17:00
MS62-19	Seeing new depths: Three-dimensional flow of a free-swimming alga	Sookkyung Lim	mon, 17:00
MS71-09	Scientific Machine Learning Methods for Extracting ODE Models of EBV-Driven B-Cell Fate Trajectories from scRNA-seq Data	Melanie Sadecki	mon, 17:00
MS74-02	NHOC: A PET imaging biomarker for survival forecasting in oncology	Jesús J. Bosque	mon, 17:00
MS11-07	A New Paradigm for the Mathematical Modelling of Multiple Sclerosis	Thomas Hillen	mon, 17:20
MS117-02	TRAIL sensitivity: Decision boundaries in a continuous cell-state landscape with implications for NK-mediated cytotoxicity	Giada Fiandaca	mon, 17:20
MS121-03	Principles of dynamical modularity in biological regulatory networks	Erzsebet Regan	mon, 17:20
MS124-10	Rotating waves driven by asymmetric cognitive map in nonlocal aggregation-diffusion model driven by spatial cognitive map	Junping Shi	mon, 17:20
MS13-06	Modeling PSA Dynamics to Characterize Prostate Cancer Treatment Response	Renee Brady-Nicholls	mon, 17:20
MS148-06	Multiscale modelling, analysis and simulation of cancer invasion processes	Mariya Ptashnyk	mon, 17:20
MS173-06	Not Everyone Panics Alike: When Heterogeneous Behavioral Responses Change Epidemic Outcomes	Navid Ghaffarzadegan	mon, 17:20
MS22-06	Learning Differential Equation Modeling through Student-Driven Projects	Rebecca Everett	mon, 17:20

Code	Title	Speaker(s)	Date / time
MS27-03	On the local stability of the elapsed-time model in terms of the transmission delay and interconnection strength	Nicolás Torres Escorza	mon, 17:20
MS29-06	Can We End the HIV Epidemic in the U.S.? A Multiscale Model Linking Clinical and Surveillance Data	Necibe Tuncer	mon, 17:20
MS31-06	Mechanistic mathematical modeling of PD-1/IL-10 co-blockade predicts post-ART SIV control		mon, 17:20
MS39-03	Monomial parametrizations and absolute concentration robustness in reaction networks	Elisenda Feliu	mon, 17:20
MS42-08	Modelling the dynamics of focal adhesions in cell migration using geometric surface PDEs	Sofie Verhees	mon, 17:20
MS50-01	Structural classification of chemical reaction networks via visual and algebraic decompositions and its implications to steady state analysis	Bryan Hernandez	mon, 17:20
MS52-02	Elucidating Regional and Global Epidemiological Trends Using Neural Network Model-form Error Corrections	Erin Acquesta	mon, 17:20
MS53-06	Uncovering noisy dynamics during cell growth using biologically-informed neural networks	Rebecca Crossley	mon, 17:20
MS62-17	Swimming-limited aggregation of Escherichia coli bacteria in liquid crystals	Maria Tătulea-Codrean	mon, 17:20
MS71-07	Generation of Model-Based Virtual Patient Cohorts Using Neural Networks	Kathleen Wilkie	mon, 17:20
MS74-04	Forecasting Patient Specific Treatment Response by Leveraging a Mathematical Model and Patient-Reported Outcomes	Diya Upadhyaya	mon, 17:20
MS11-04	Mathematical Model of Multiple Sclerosis	Mo'tassem Al-arydah	mon, 17:40
MS117-03	The co-evolution of a tumour's genomic landscape and TCR repertoire	Isabella Sodi	mon, 17:40
MS121-04	Network dynamical stability analysis of age-related health	Glen Pridham	mon, 17:40
MS124-11	Are the mathematical biological models governed by non-linear first order Caputo fractional differential equations solvable?	Kunquan Lan	mon, 17:40
MS13-07	Longitudinal Biomarkers and Alzheimer's Disease in Down Syndrome	Samuel Handelman	mon, 17:40
MS148-07	Homogenization of a phenomenological electropermeabilization model	Tobias Gebäck	mon, 17:40
MS173-07	Optimizing infectious disease mitigation under dynamic conditions	Laura Mueller	mon, 17:40
MS22-07	Building Modeling Workshops at Biology Conferences	Angela Peace	mon, 17:40
MS27-04	Models with Memory and Delay in Molecular, Cellular, and Population Biology	Radek Erban	mon, 17:40
MS29-07	From Bird Viremia to Bird Surveillance: Identifiability in a Multiscale Vector-Borne Model of Usutu Virus Infection	Quiyana Murphy	mon, 17:40
MS31-07	Uncovering the mechanisms of SHIV dynamics in rhesus macaques undergoing immune therapy	Stanca Ciupe	mon, 17:40
MS39-07	Non-monotonic dose-response curve in biochemical systems	Polly Yu	mon, 17:40
MS42-07	Multiphase cross-diffusion models for tissue structures	Cordula Reisch	mon, 17:40
MS50-07	Exploring the effect of parametric model reduction on the structural identifiability of linear models	Sabina Haque	mon, 17:40
MS52-01	Sensitivity analysis for biological differential equation models with many parameters	Dilan Pathirana	mon, 17:40
MS53-07	First-passage times and queueing behavior of stochastic search with dynamic redundancy and mortality	Samantha Linn	mon, 17:40
MS62-20	Parallel computing methods for large-scale simulations of flagellar dynamics	Weifan Liu	mon, 17:40
MS71-11	Predicting the impact of gene therapy in preventing HIV transmission	Steffen Docken	mon, 17:40
MS74-03	From measurement to decision: a tissue-aware digital-twin platform for CAR T cell dosimetry	Luciana Luque	mon, 17:40
MS117-04	Optimizing combined oncolytic virotherapy and chemotherapy for neuroblastoma: a mathematical approach	Matteo Italia	mon, 18:00
MS121-05	From self-sustained intracellular oscillations to whole-cell network organization in Physarum polycephalum	Linnéa Gyllingberg	mon, 18:00
MS124-12	Evolutionary Escape in Antibiotic Cycling Therapies	Esteban Hernandez Vargas	mon, 18:00
MS13-08	Analyzing (Sub-phenotyping) the Longitudinal Progression to Type 2 Diabetes using Machine Learning and Physiological Simulations	Vijaya Subramanian	mon, 18:00
MS148-04	Multiscale Modeling of Electroporation Capturing Long-Term Membrane Permeability	Emil Timlin	mon, 18:00
MS173-08	Measles and mandates: A mathematical assessment of Florida's proposed school-entry vaccination mandate removal	Alice Oveson	mon, 18:00
MS22-08	Does classroom-flipping improve analytical skills? Results from a cross-sectional comparison between undergraduate calculus exams	Stacey Smith?	mon, 18:00
MS27-05	An Exact Solution and Amplitude Enhancement in a Non-Autonomous Delay Differential Equation	Toru Ohira	mon, 18:00
MS29-08	An immuno-epidemiological model with consideration for symptom score	Summer Atkins	mon, 18:00
MS31-08	Multiscale modelling identifies a predictive relationship between circulating biomarkers and antiviral efficacy of new treatments for chronic hepatitis B virus infection	Tyler Cassidy	mon, 18:00
MS39-08	A Structural Approach to Identifying Indicator Species in Chemical Reaction Networks	Yong-Jin Huang	mon, 18:00
MS42-06	Upscaling between an agent-based model (smoothed particle approach) and a continuum-based model for cellular forces	Qiyao (Alice) Peng	mon, 18:00

Code	Title	Speaker(s)	Date / time
MS50-08	Simple chemical systems with chaos	Tomislav Plesa	mon, 18:00
MS52-07	Symbolic regression enables coarse-grained model discovery for cancer-relevant signalling dynamics	Theodore de Pomereu	mon, 18:00
MS53-08	Plant Cortical Microtubules: Curvature Sensing by Elastic Curves on Smooth Surfaces	Tim Tian	mon, 18:00
MS62-07	Multi-timescale dynamics of microswimmers	Sara Drummond-Curtis	mon, 18:00
MS71-12	3D reaction-diffusion model-based biopsy simulation for dynamic tumor growth parameter estimation	Veronika Hofmann	mon, 18:00
MS74-08	Data-efficient early prediction of treatment response using mechanistic tumor growth models	Yi Jiang	mon, 18:00
PLT-03	Impact of horizontal gene transfer on population evolutionary dynamics	Sepideh Mirrahimi	tue, 08:30
PLT-04	An intro to shape spaces - from differential geometry to the drosophila testis	Benedikt Wirth	tue, 09:20
MS106-02	Introductions + Junior member mini-presentation	Lorenzo Veschini	tue, 10:40
MS108-01	Sub-diffusion Compartmental Model for Diffusion MRI to Probe Brain Microstructural Information: Insights from a Simulation Study of White Matter	S M Erfanul Kabir Chowdhury	tue, 10:40
MS114-01	The role of DNA methylation and DNA sequence in epigenetic plasticity	Elizabeth Read	tue, 10:40
MS138-01	Quantifying the effect of initial cell density on hypoxia-induced radioresistance: experimental analysis and compartmental modeling of cell state dynamics	Botao Dai	tue, 10:40
MS144-02	Involving communities in epidemic modelling: an introduction to co-production principles and tensions	Lucy Rycroft-Smith; Sarah Barnes	tue, 10:40
MS150-01	Inferring RNA processing dynamics across cell states and fates from single-cell snapshot data	Tara Chari	tue, 10:40
MS158-01	Informing Macrophage phenotypic change over the endometrial cycle using omics data	Domenic Germano	tue, 10:40
MS171-01	Information delays, network structure and adaptive human behavior jointly shape epidemic dynamics	Claus Kadelka	tue, 10:40
MS174-08	A Data-Driven Model of Polarity Reversal in Migrating Cells	Jupiter Algorta	tue, 10:40
MS33-01	Kinetic equations and mathematical epidemiology	Angela Stevens	tue, 10:40
MS47-01	Modelling monoclonal antibody transfer across the human placenta	Eleanor Doman	tue, 10:40
MS62-21	Swimming Strategies of Bipolar Flagellated Bacteria	Jeungeun Park	tue, 10:40
MS66-01	Mechanical regulation of the G1/S checkpoint in collective cell migration: a two-stage age-structured model	Stéphanie Abo	tue, 10:40
MS80-01	Stability of reaction networks with randomly switching parameters	Aidan Howels	tue, 10:40
MS83-02	Sparse identification of renewal equations with application to disease transmission dynamics	Dimitri Breda	tue, 10:40
MS89-01	How perception and neural weighting can drive the geometry of decision-making in target pursuit	Christopher Strickland	tue, 10:40
MS92-01	Mathematical Models for Understanding and Managing Biological Invasions	Ousmane Ousmane Seydi	tue, 10:40
MS95-01	From CRN to CNN: Deep Learning with Stochastic Chemical Reaction Networks	Bernie Daigle	tue, 10:40
MS106-03	Multiscale modeling of the innate immune response to respiratory pathogens	Reinhard Laubenbacher	tue, 11:00
MS108-02	Energy Constraints and Neural Strategy Transitions in Alzheimer's: A Game-Theoretic Model	Irina Kareva	tue, 11:00
MS114-02	Joint trajectory and gene regulatory network inference using a mechanistic optimal transport-based framework	Elias Ventre	tue, 11:00
MS138-02	Exploring how metabolism comes into play with radiations: a mechanistic approach	Damian Bimbenet	tue, 11:00
MS144-03	Neat Models, Messy Lives: Social Science as a Bridge to Lived Experience	Shema Tariq	tue, 11:00
MS150-02	Inferring Cellular Dynamics through Ordered Diffusion Kernels	Jack Soulsby	tue, 11:00
MS158-02	Transcriptome-informed flux predictions revealed temporal metabolic reprogramming and branch points mediating cold stress-growth trade-offs in rice	Fatemeh Soltani	tue, 11:00
MS171-02	Coupled Dynamics: How Genes Shape Epidemics (and Epidemics Shape Genes)	Joan Ponce	tue, 11:00
MS174-04	Chaste: a computational framework for multicellular modelling	James Osborne	tue, 11:00
MS33-02	Bacterial movement by run and tumble: the flux-limited Keller-Segel equation	Benoit Perthame	tue, 11:00
MS47-02	Modelling Hematopoietic Dynamics in H3K27M-Driven Preleukemia and Implications for AML Therapy	Mia Brunetti	tue, 11:00
MS62-22	Modeling the Transposition of Deformable Bacteria Through Membrane Pores	Abigail Drumm	tue, 11:00
MS66-02	From discrete models of cell mechanics to continuum descriptions of tissue growth	Shahak Kuba	tue, 11:00
MS80-02	Weakly Reversible Chemical Reaction Networks Are Recurrent in 2d	Andrea Agazzi	tue, 11:00
MS83-01	Discovering Dynamical Digital Twins from Time Series through Bayesian Sparse Regression	Clémence Métayer	tue, 11:00
MS89-02	Small Organism Collective Behavior in Fluid Environments	Margaret Knight	tue, 11:00
MS92-02	A Hybrid Modeling Framework for Forecasting the 2025 Texas Measles Outbreak and Evaluating Vaccination Strategies	Chidozie Williams Chukwu	tue, 11:00

Code	Title	Speaker(s)	Date / time
MS95-02	Buffered Chemical Reaction Networks for Reusable Computation	Dominic Scalise	tue, 11:00
MS106-04	A Large-Scale Logic-Based Model of the Human Immune System as a Foundation for Mechanistically Interpretable Clinical Prediction and Immune Digital Twins	Tomas Helikar	tue, 11:20
MS108-03	Biomechanical Forces and Tensions that Contribute to Cortical Folding in the Brain	Julianna Capece	tue, 11:20
MS114-03	A domain decomposition approach to the construction of Markov state models for stochastic gene regulatory networks	Maryam Yousefian	tue, 11:20
MS138-03	Geometric Adversarial Learning for Reconstruction of Right-Censored Tumor Dynamics	Nahum Puebla-Osorio	tue, 11:20
MS144-04	How Close is Too Close? Modelling Sexual and Non-sexual Transmission Pathways of Mpox in the UK	Zviiteyi Chazuka	tue, 11:20
MS150-03	Inferring causal gene regulatory relationships from time-resolved single-cell transcriptomics	Dimitris Volteras	tue, 11:20
MS158-03	From genome sequencing to metabolic modelling and dynamic flux balance analysis extensions	Thomas Munn	tue, 11:20
MS171-03	Models that ignore behavior underestimate the basic reproduction number	Marko Lalovic	tue, 11:20
MS174-01	Vivarium: a compositional framework for multiscale modeling	Eran Agmon	tue, 11:20
MS33-03	Oncogenic transformation of tubular epithelial ducts: how mechanics affects morphology	Davide Ambrosi	tue, 11:20
MS47-03	How much biological realism is enough? An application to ovarian cancer treatment response	Patricia Lamirande	tue, 11:20
MS62-24	Morphology and dynamics of chemotacting droplets	Giulia Celora	tue, 11:20
MS66-03	Toward a unified theory of curvature-guided epithelial migration: ER remodeling, cytoskeletal polarity, and cell mechanics	Fabian Spill	tue, 11:20
MS80-03	Sensitivity of dynamics in Autocatalytic Reaction Networks of Togashi-Kaneko type	Lea Popovic	tue, 11:20
MS83-03	Bayesian discovery of biochemical reaction networks from time-course data with projection predictive inference	Julien Martinelli	tue, 11:20
MS89-03	Pattern formation through collective behavior in plant pigment systems	Patrick Shipman	tue, 11:20
MS92-03	Coupled Dynamics of COVID-19 and Drug Overdose Deaths	Folashade Agusto	tue, 11:20
MS95-03	Event-based reservoir computing via chemical reaction networks	Jinsu Kim	tue, 11:20
MS106-01	Using an Agentic AI Critical Illness Digital Twin to rescue from immune exhaustion in sepsis	Gary An	tue, 11:40
MS108-04	A Biomchemical Model of Cortical Folding via Turing Patterns	Monica K. Hurdal	tue, 11:40
MS114-04	From Simulation to Macroscopic Dynamics: Learning Reaction Coordinates and Sampling Rare Transitions in Stochastic Gene Regulatory Networks	Alexander Sikorski	tue, 11:40
MS138-04	A new metric for functional drug screening: combing radiation with systemic therapies	Sarah C. Brüningk	tue, 11:40
MS144-01	The MEMVIE public involvement framework: Developing a framework for public involvement in epidemiological and health economic modelling to bring dynamism to vaccination policy recommendations	Edward Hill	tue, 11:40
MS150-04	Decoding gene regulatory networks and cellular dynamics	Weixu Wang	tue, 11:40
MS158-04	Inferring mass isotopomer distribution dynamics from partial isotopic labeling data: Implications for flux estimation	Anika Küken	tue, 11:40
MS171-04	Highlights of projects funded through the Mathematical Biology program and other initiatives at the US NSF	Zhilan Feng	tue, 11:40
MS174-02	Bringing biology to the browser with Artistoo	Inge Wortel	tue, 11:40
MS33-04	Minimal Principles for Dynamics in Reaction Networks	Nicola Vassena	tue, 11:40
MS47-04	Modeling immune-mediated effects of RAGE inhibition and radiotherapy in malignant gliomas	Miguel Perales-Patón	tue, 11:40
MS62-23	Efficient Immersed Boundary method for flows with rigid objects	Brittany Leathers	tue, 11:40
MS66-04	Modelling bulk mechanical effects in a planar cellular monolayer	Natasha Cowley	tue, 11:40
MS80-04	Noise induced stabilization in stochastic chemical reaction network.	Lucie Laurence	tue, 11:40
MS83-04	Global modelling of algae blooms from time series: comparison of chaotic attractors	Martin Rosalie	tue, 11:40
MS89-04	Data Assimilation and Model Selection for Collective Motion in Locust Swarms	Rebecca Everett	tue, 11:40
MS92-04	Associations of spatial dynamics of COVID-19 transmissions with social distancing measures on household visits in the Netherlands	Ward Spee	tue, 11:40
MS95-04	Heterochiral DNA for intracellular computing	Matthew Lakin	tue, 11:40
MS102-04	Thermodynamics of Chemical Reaction Networks: From Circuits to Metabolism	Massimiliano Esposito	tue, 15:00
MS139-01	Mechanistic modeling of joint size-dependent cell-free DNA concentrations and tumor kinetics for immunotherapy resistance prediction	Linh Nguyen Phuong	tue, 15:00
MS160-03	Integrating Single-Cell Experiments and Stochastic Models to Infer a Predictive, Mechanistic Understanding for Glucocorticoid Receptor Transport and DUSP1 mRNA Expression Dynamics	Brian Munsky	tue, 15:00

Code	Title	Speaker(s)	Date / time
MS162-05	Could malaria mosquitoes be controlled by periodic releases of transgenic mosquitocidal Metarhizium pingshaense fungus? A mathematical modeling approach.	Salman Safdar	tue, 15:00
MS1644-03	Thirty Years of Cell Cycling with Bela	John Tyson	tue, 15:00
MS174-03	Compucell3D: Python-scripted open-source multiscale modeling framework	James Glazier	tue, 15:00
MS28-02	Toward Sustainable Corrosion Control: Identifying tuning parameters associated with Biofilm-Induced corrosion Inhibition in a mathematical framework	Blessing Emerenini	tue, 15:00
MS30-01	Introduction to Catalyst.jl and Applications in Chemical Reaction Network Modeling	Samuel Isaacson; Torkel Loman	tue, 15:00
MS31-09	Accurate estimation of the frequency of asymptomatic SARS-CoV-2 infections from serosurveys	Narendra M Dixit	tue, 15:00
MS48-02	Spatial Mechanistic Modelling of T-Cell Engagers: Insights into Trimer Formation and its effect on Cancer Killing	Gibin Powathil	tue, 15:00
MS62-01	The Immersed Boundary Single-Layer (IBSL) method for reaction-advection-diffusion systems	Owen Lewis	tue, 15:00
MS67-02	Integrating human behaviour and epidemiological modelling: unlocking the remaining challenges	Edward Hill; Matthew Ryan	tue, 15:00
MS71-01	Hybrid Mechanistic-Machine Learning Frameworks for Personalized Oncology	Alvaro Köhn-Luque	tue, 15:00
MS73-01	Effects of molecular noise on bacterial size control	Andrew Mugler	tue, 15:00
MS76-01	Development of agent-based modelling of STI's: Can we use AI methods for improving implementation of partnership dynamics?	Mirjam Kretzschmar	tue, 15:00
MS78-02	Evolutionary game theory and the emergence of noisy learning	Christian Hilbe	tue, 15:00
MS81-01	Mathematical models of brain networks: propagation of diseases and of brain activity	Ashish Raj	tue, 15:00
MS84-07	Drug Release Dynamics from a Three-Layer Composite Contact Lens	Rayanne A. Luke	tue, 15:00
MS94-01	Computing with reaction networks at input-independent speed	Badal Joshi	tue, 15:00
MS1644-01	Unlike networks, molecules are dreams - the early years of systems biology with Bela Novak.	Andrea Ciliberto	tue, 15:15
MS102-05	Hypergraph-based models of random chemical reaction networks: Conservation laws, connectivity, and percolation	Nahuel Freitas	tue, 15:20
MS139-02	ctDNA precedes imaging: A predictive model for real-time treatment adaptation in HPV-associated anal squamous cell carcinoma	Phebe Havor	tue, 15:20
MS162-06	Modeling the Geospatial Dynamics of Lyme Disease in Maryland Under Current and Projected Climate Change Scenarios	Salihu Musa	tue, 15:20
MS174-05	Morpheus: User-friendly multiscale modeling	Jörn Starruß	tue, 15:20
MS28-01	A Predictive Model of Subaerial Biofilms: Insights into Daily Dynamics and Environmental Changes	Alberto Tenore	tue, 15:20
MS31-10	Modeling the Interferon Responses to Influenza A Virus Infection	Cailan Jeynes-Smith	tue, 15:20
MS48-01	From Image to Simulations: Integrating Imaging Biopsy Data into Agent-Based Models to Understand the Impact of Tumour-Microenvironment Spatial Heterogeneity on Cancer Treatment	Blanche Mongeon	tue, 15:20
MS62-02	Suppression of upstream swimming in shear flows by memory-mediated chemotaxis	Smitha Maretvadakethope	tue, 15:20
MS67-01	Epidemics Don't Just Spread - People React: Modelling Behaviour in Real Time	David Haw	tue, 15:20
MS71-02	Deciphering antibody repertoire evolution using protein language models and B cell lineage inference	Daphne van Ginneken	tue, 15:20
MS73-02	Cell growth under high antibiotic concentrations drastically reshapes the bacterial transcriptome	Daniel Schultz	tue, 15:20
MS76-02	Early-warning signals for infectious diseases with a social-media compartment	Stacey Smith?	tue, 15:20
MS81-02	Non-Linear Dynamics and Catalytic Exchange Govern Prion Assembly and Spreading	Human Rezaei	tue, 15:20
MS84-01	A mathematical model of fluid and solute transport across the corneal endothelium	Federica Vanone	tue, 15:20
MS1644-04	Modeling the Cell Cycle: Reflections Inspired by Béla Novák	Laurence Calzone	tue, 15:30
MS102-06	Positive algebraic geometry for mathematical biology	Stefan Müller	tue, 15:40
MS139-03	Coupled SDE-ODE Modeling of Tumor-Immune Dynamics to Infer Biomarker Release	Pujan Shrestha	tue, 15:40
MS160-05	When Trajectory Inference Fails: The Challenges of Inferring Dynamical Models from Single-Cell RNA-seq Data	Meichen Fang	tue, 15:40
MS162-07	Investigating the Impact of Novel Transmission-Blocking Anti-malarial Drugs: A Mathematical Modeling Approach	Woldegebriel Assefa Woldegerima	tue, 15:40
MS174-06	PhysiCell: multiscale modeling with a grammar	Randy Heiland	tue, 15:40
MS28-05	Mathematical Modeling of Microbial-Induced Corrosion of Bioplastic in Marine Environments	Luigi Frunzo	tue, 15:40
MS31-11	Towards a generalized framework for validating biological fidelity in immunobiological virtual patients	Chapin Korosec	tue, 15:40
MS48-03	Mechanistic Modeling of Heterogeneous Tumor Response to Anti-NUPR1 Therapy in Docetaxel-Resistant Prostate and Pancreatic Cancers	Harsh Jain	tue, 15:40
MS62-03	Mechanical Interactions between a Contact Lens and the Eye	Lucia Carichino	tue, 15:40
MS67-03	Modelling social influence through discussion-based contacts in coupled behaviour-epidemic models	Elisabetta Colosi	tue, 15:40

Code	Title	Speaker(s)	Date / time
MS71-03	Establishing a Taxonomy of Computational Modeling as the Foundation Towards Certifying Trustworthy Artificial Intelligence	Erin Acquesta	tue, 15:40
MS73-03	Cell Size Regulation from Bacteria to Mammalian Cells	Kuheli Biswas	tue, 15:40
MS76-03	Mathematical Modeling of Post-Infection Mortality and Transmission Structure: Implications for Persistent Infections and Public Health Dynamics	Zhisheng Shuai	tue, 15:40
MS78-01	Direct reciprocity under constraints: information and evolutionary dynamics	Nikoleta Glynatsi	tue, 15:40
MS81-03	Modelling Optimal Anti-Inflammatory Regimens and Outcomes in Alzheimer's Disease	Wissam El Hajj	tue, 15:40
MS84-02	V-Cornea: A Multi-Scale In Silico Framework for Ocular Toxicology and IVIVE	Joel Vanin	tue, 15:40
MS94-02	Molecular Machines and the EM algorithm	Carsten Wiuf	tue, 15:40
MS1644-02	Positive feedback + negative feedback = "bloody complicated"	Attila Czikasz-Nagy	tue, 15:45
MS102-07	Widespread biochemical reaction networks enable Turing patterns without imposed feedback	Tian Hong	tue, 16:00
MS139-04	Linking cfDNA clearance to fragment length with mechanistic models	Thomas Rachman	tue, 16:00
MS160-06	Deep learning for the Analysis and Reconstruction of Transcriptional dynamics from live-cell imaging data	Muhan Ma	tue, 16:00
MS162-08	Mathematics of Wolbachia-based biocontrol of mosquito-borne diseases	Abba Gumel	tue, 16:00
MS1644-05	A systems biology study of autophagy induction playing a key role in survival upon cellular stress	Orsolya Kapuy	tue, 16:00
MS174-07	PolyHoop & SimuCell3D: Open-source tools for high-resolution cell dynamics	Roman Vetter	tue, 16:00
MS28-04	Towards a mechanistic model of the mercury methylation process	Grace D'Agostino	tue, 16:00
MS31-12	Novel regulators found by network perturbation of sex-specific influenza A infection in clinical samples	Jason E. Shoemaker	tue, 16:00
MS48-04	Model-supported patient stratification using multi-objective synergy optimization in combination therapy	Irina Kareva	tue, 16:00
MS62-04	An immersed boundary model for fluid membrane interaction in the cochlea	John Stockie	tue, 16:00
MS67-05	Feedback loops in multi-season influenza vaccinations strategies	Joel Winterton	tue, 16:00
MS71-04	Exploring the Spatial Dynamics of the Immune Response to Viral Infections	Fatemeh (Azade) Beigmohammadi	tue, 16:00
MS73-04	Essential Role of Extrinsic Noise in Models of E. Coli Division Control	Mattia Corigliano	tue, 16:00
MS76-05	Modelling COVID-19 vaccine breakthrough infections in highly vaccinated Israel—The effects of waning immunity and third vaccination dose	Daihai He	tue, 16:00
MS78-03	The role of structure and mobility in the evolution of cooperation	Diogo Pires	tue, 16:00
MS81-04	ATM-ApoE crown formation in Alzheimer's disease	Felipe Olivares	tue, 16:00
MS84-05	A coupled fluid-dynamics-heat transfer model for 3D simulations of the aqueous humor flow in the human eye	Marcela Szopos	tue, 16:00
MS94-04	Analog computation with transcriptional networks	David Soloveichik	tue, 16:00
CT1229	Decision-making in heterogeneous self-propelled systems	Víctor Villegas-Morral	tue, 17:00
CT1237	Predicting clogging in complex networks through a minimal continuum model	Cara Neal	tue, 17:00
CT1259	Sparsest steady-state relations from biochemical network structure	Ahmad Mokhtar	tue, 17:00
CT1311	Evaluating the impact of school measures on early SARS-CoV-2 transmission in Portugal: a retrospective study	Ganna Rozhnova	tue, 17:00
CT1374	Revealing Hidden Organization in High-Dimensional Cancer Proteomic Data: A Geometric and Topological Perspective	SeyedSamie Alizadeh Darbandi	tue, 17:00
CT1440	Estimating directed connectivity from fMRI and structural priors via network diffusion model	Ashish Raj	tue, 17:00
CT1565	Decomposing noise in RNA sequencing experiments of adaptive immune repertoires	Matthew Cowley	tue, 17:00
CT1586	A mathematical model of ultrasensitive sequencing protocol	Eszter Lakatos	tue, 17:00
CT216	Conflict-Gated Gradient Scaling: Resolving gradient pathology in physics-informed epidemiological models	Woldegebriel Assefa Woldegerima	tue, 17:00
CT232	Markov Process based Mathematical Model of Vascular Stenosis Formation and Treatment	Taiga Kadowaki	tue, 17:00
CT273	3D-Analysis of tumor spheroids – a synergistic combination of mechanistic modeling and data-driven computational methods	Anja Voss-Böhme	tue, 17:00
CT383	Quality of information impacts growers' adoption of crop protection methods	Frédéric Grognard	tue, 17:00
MS102-02	Genetic recombination, deterministic reaction systems, stochastic partitioning, and Markov embedding	Ellen Baake	tue, 17:00
MS116-01	Simplicity in complexity: A probabilistic view microbial ecology	Mario Castro	tue, 17:00
MS151-01	Radiotherapy as an evolutionary force: modeling phenotypic heterogeneity and treatment response in tumors	Lara Schmalenstroer	tue, 17:00
MS155-04	Eye2Heart: A reduced mathematical model bridging cardiovascular and ocular hemodynamics	Marcela Szopos	tue, 17:00
MS157-01	LLM-Guided Mechanistic Model Discovery in Population Pharmacometrics	Sébastien Benzekry	tue, 17:00
MS160-04	Statistical inference of chromatin- and transcription dynamics in living cells	Christoph Zechner	tue, 17:00
MS177-03	Numerical Modelling for Glucose Metabolization in the Retina	Kohl James	tue, 17:00

Code	Title	Speaker(s)	Date / time
MS28-06	Bacterial Biofilm Growth Modelling in a Counter-Diffusion System, Coupled with Biozone Formation in the Aqueous Phase by Planktonic Bacteria, driven by Directed Movement.	Rachana Mandal	tue, 17:00
MS30-02	Parameter Identifiability and Inference with Catalyst.jl and PEtab.jl	Sebastian Persson; Torkel Loman	tue, 17:00
MS31-13	Inside the wavering mind of an NK cell: Mathematical modeling of NK cell activation in health and disease	Montana Ferita	tue, 17:00
MS48-05	Assessing the Role of Model Complexity in Virtual Clinical Trial Outcomes	Jana Gevertz	tue, 17:00
MS62-05	Drag on Microtubule Asters	Sarah Olson	tue, 17:00
MS67-04	Modelling the adoption of Integrated Pest Management (IPM) by UK crop growers	Elliot Vincent	tue, 17:00
MS68-03	Integrating multiscale multimodal imaging-based and PK/PD-informed models to predict triple negative breast cancer response to neoadjuvant therapy	Guillermo Lorenzo	tue, 17:00
MS71-05	Curriculum Learning for Patient-Specific Parameters in Glioblastoma Multiforme	Erica Rutter	tue, 17:00
MS76-06	An agent-based platform for simulating the impact of chlamydia vaccine in the US population	Qi Deng; Edward Thommes	tue, 17:00
MS78-04	Exploiting evolutionary trade-offs in Anaplastic Large Cell Lymphoma though optimal targeted therapy	Franco Pradelli	tue, 17:00
MS81-05	Modeling the Prion Aggregation Process During Polymerization Experiments Using Delay Differential Equations	Theo Loureaux	tue, 17:00
MS94-05	Limits of Equilibrium Computation in DNA Seesaw and Dimerization Networks	Ho-Lin Chen	tue, 17:00
CT1234	Multi-stage volume exclusion models for cell proliferation	John Carlo Dimaculangan	tue, 17:20
CT1236	Optimizing the resources allocation between biochemical defence and counter-counter defence in pathogenic and trophic interactions.	Lukas Korn	tue, 17:20
CT1305	Optimal epidemic mitigation using focused and unfocused interventions	Martin Bootsma	tue, 17:20
CT1438	Construction and simulation of a path-valued model of dendrite growth	Andrew Nugent	tue, 17:20
CT1466	An Information-Based Dynamical Network Model of Multicellularity Across Dimensions	Sabahat Defne Plattürk	tue, 17:20
CT1499	Quantitative Modelling of Microbial Colony Growth Dynamics	Attila Csikász-Nagy	tue, 17:20
CT1510	Mechanistic modelling of adipocyte lipid droplet dynamics	Thomas Reed	tue, 17:20
CT1625	Bistability between acute and chronic states in a Model of Hepatitis B Virus Dynamics	Hayriye Gulbudak	tue, 17:20
CT258	Saddle-Node Bifurcation in Macrophage Proliferation Determines Atherosclerotic Plaque Stability	Emine Atıcı Endes	tue, 17:20
CT274	Modelling interclonal cooperation in epithelial carcinogenesis using spatial models	Muhammad Asim Farooq	tue, 17:20
CT334	Extension of an established dynamic thyroid model for capturing Graves' disease including antibody production	Thomas Benninger	tue, 17:20
CT384	Transmission Dynamics of Eastern Equine Encephalitis: Global Sensitivity Analysis and SHAP Parameter Importance in an Age-Structured Vector-Host Model	Christopher Mitchell	tue, 17:20
MS116-02	An evolutionary approach to ecological transitions in population dynamics	Luciano Stucchi	tue, 17:20
MS151-02	Multi-phase field approach towards modelling mechanical heterogeneity in tissues	Aleksandra Ardaševa	tue, 17:20
MS155-01	An agent-based model of atherosclerotic plaque angiogenesis grounded in human spatial and single-cell data	Katrin Schröder	tue, 17:20
MS157-02	Deep Reinforcement Learning for Sequential Decision-Making in a Polytherapeutic Cancer Landscape	Alvaro Köhn-Luque	tue, 17:20
MS160-07	Quantifying the impact of cell division and size control on stochastic auto-regulatory gene expression	Abigail Kushnir	tue, 17:20
MS177-05	Mathematical Models of Retinal Drug Delivery	Paul Roberts	tue, 17:20
MS28-03	Aspects of Nonlinear Dynamics, Spatial Effects and Cellular Memory in Bacterial Quorum Sensing	Christina Kuttler	tue, 17:20
MS31-14	From binding to neutralization: A mathematical model for flavivirus antibody responses	Nathanael Hoze	tue, 17:20
MS48-06	Cellular kinetics of drug response: are they conserved when altering experiment and biological context?	Matt Faria	tue, 17:20
MS62-06	How do glioma cells infiltrate the narrow gap between astrocytes endfeet and blood vessel through ion-exchange and physical deformation for the critical cellular infiltration?	Yangjin Kim	tue, 17:20
MS67-06	Memory mechanisms in Bayesian behavioural change epidemic models	Rob Deardon	tue, 17:20
MS68-05	Linking Primary and Metastatic Growth: Testable Predictions from a Shared-Carrying-Capacity Model	Pirmin Schlicke	tue, 17:20
MS71-06	Experimental and theoretical investigations of rotating algae biofilm reactors (RABRs): Areal productivity, nutrient recovery, and energy efficiency	Jia Zhao	tue, 17:20
MS78-05	Stackelberg evolutionary game theory: how to manage evolving systems	Alexander Stein	tue, 17:20
MS81-06	An optimal control problem for anti-inflammatory treatments of Alzheimer's disease	Nicolás Torres Escorza	tue, 17:20
MS94-06	Designing optimal computational networks: a case study from maximum likelihood estimation	Oskar Henriksson	tue, 17:20
CT1233	Emergent mechanics from material replacement in biological systems	Matthew Butler	tue, 17:40

Code	Title	Speaker(s)	Date / time
CT1235	Role of phenotypic plasticity in community assembly and coexistence: from a model organism to theory and back	Ata Kalirad	tue, 17:40
CT1261	Autocorrelation-Based Inference of Transcription Dynamics via Infinite-Server Queueing Models	Jeremy Worsfold	tue, 17:40
CT1303	Mapping bovine trypanosomosis under diagnostic uncertainty: spatial prevalence estimates in Nigeria and Ethiopia	Alex Kaye	tue, 17:40
CT1362	New topologies in the unfolding of a high codimension singularity organizing neuronal dynamics	Marisa Saggio	tue, 17:40
CT1428	The exponential distance rule-based network model predicts topology and reveals functionally relevant properties of the Drosophila projectome	Balazs Pentek	tue, 17:40
CT1451	Generating Vascular Network Structures from Real Data	Rui Travasso	tue, 17:40
CT1576	Membrane Lipid Dynamics in Macrophage Chemotaxis	Joshua Forrest	tue, 17:40
CT243	Topological Morphological Descriptor Enables Artery–Vein Discrimination in Whole-Mouse Vasculature	Sofia Farina	tue, 17:40
CT277	Mathematical modeling of radiotherapy response in murine glioma using MRI-defined tumor habitats	Ayesha Das	tue, 17:40
CT293	Interpretable measures for sensitivity and identifiability in models of infectious disease	Tarek Alrefae	tue, 17:40
MS102-01	A Graph grammar view on chemical reaction networks	Christoph Flamm	tue, 17:40
MS116-03	Species interactions do matter in microbial dynamics	Aniello Lampo	tue, 17:40
MS151-03	Phenotype structuring in collective cell migration: a tutorial of mathematical models and methods	Chiara Villa	tue, 17:40
MS155-02	Modelling Cerebral Autoregulation at the Microvascular Scale: From Health to Post-Stroke Dysfunction	Chryso Lambride	tue, 17:40
MS157-03	Towards personalized glioblastoma radiotherapy using growth modeling and recurrence prediction	Lucas Zimmer	tue, 17:40
MS160-02	Predicting Single-Cell RNA Expression Variability from DNA Sequence and Epigenetics	Andrew Nicoll	tue, 17:40
MS177-06	Mathematical analysis of photoreceptor changes in conditions of separation from the underlying retinal pigment epithelium	Stephen Wirkus	tue, 17:40
MS28-07	Modelling accumulation of toxic substances in bacterial cells: a route to suppressing antimicrobial resistance	Sandeep Shirgill	tue, 17:40
MS31-15	Mathematical and Mouse Models Identify T Regulatory Cell Influx as A Key Determinant of Acquired Resistance to PD-1 Immunotherapy	Rachel Sousa	tue, 17:40
MS48-08	An Agent Based Modelling Framework for the Role of Tumour-Stroma Dynamics in Metastatic Colorectal Cancer	Sam Oliver	tue, 17:40
MS62-08	The Method of Regularized Stokeslets as an Introduction to Fluid-Structure Interactions	Amy Buchmann	tue, 17:40
MS67-07	Oscillatory epidemic dynamics driven by non-linear behavioural feedback	Ryosuke Omori	tue, 17:40
MS68-06	Data-driven reconstruction of phenotypic transition networks in colorectal cancer	Rodrigo Garcia-Tejera	tue, 17:40
MS71-10	Multivariate drivers of the heterogeneous within-host immune response during influenza	Nicole Bruce	tue, 17:40
MS76-07	Decision-making, epidemiological dynamics, individuals, and societies	Chadi Saad-Roy	tue, 17:40
MS78-06	When policy shapes selection: anticipating evolutionary feedbacks in conservation	Maria Kleshnina	tue, 17:40
MS81-07	Machine learning accelerated modeling of tau protein spread with the Network Transport Model	Brandon Imstepf	tue, 17:40
MS94-07	Recurrent neural chemical reaction networks: a versatile way of generating complex dynamics in chemical systems	Tom Ouldridge	tue, 17:40
CT1247	AI-enabled discovery of mechanistic mediators in Alzheimer's disease progression	Ashish Raj	tue, 18:00
CT1254	Cellular resource allocation in budding yeast beyond steady state	Weida Liao	tue, 18:00
CT1294	Bayesian Inference for Outbreak Final Size Datasets with Underlying Household Structure	Joseph Brooks	tue, 18:00
CT1366	Multi-scale Modelling of Astrocyte Metabolism in Pathological Morphologies	Ingrid A. Lizcano-Prada	tue, 18:00
CT1518	The Race Between Entropy and Maximum Entropy	Jiří Nedomlel	tue, 18:00
CT1600	Deciphering Spatiotemporal Dynamics of Oocyte Selection during Ovarian Development: A 3D Multiscale Agent-Based Model	Pawan Kumar	tue, 18:00
MS102-03	From Mean Control to Variability Control in Biological Systems	JaeKyoung Kim	tue, 18:00
MS116-04	Robust time-independent metrics for evaluating drug efficacy	Marta Sánchez-Díez	tue, 18:00
MS151-04	A multiscale discrete-to-continuum framework for structured population models	Eleonora Agostinelli	tue, 18:00
MS155-03	Mechanistic modeling of closed-loop cerebrovascular reactivity	Laura Ellwein Fix	tue, 18:00
MS157-04	Image-based prediction of skin cancer evolution for clinical decision support through machine learning	Daniel Camacho-Gomez	tue, 18:00
MS177-07	Predicting the efficacy of optogenetic strategies for vision restoration	Chiara Boscarino	tue, 18:00
MS28-08	Ecological interactions and spatial dynamics in microbial aggregates: A novel modelling framework	Viktoria Freingruber	tue, 18:00
MS31-16	Decoding Influenza Transmission from Ferret Contact and Viral Dynamics	Veronika Zarnitsyna	tue, 18:00
MS48-07	Mechanistic Learning of Engineered Immune Cell-Tumor Cell Interactions and Heterogeneity	Philipp Altrock	tue, 18:00
MS62-18	Thin-film modelling and parameter optimisation for biofilms	Alex Tam	tue, 18:00
MS67-08	Predicting contact rates in response to control measures: a validated protocol for generating contact matrices relevant to transmission modelling of respiratory diseases	Willem Frederiks	tue, 18:00

Code	Title	Speaker(s)	Date / time
MS68-07	A data-integrative approach to building ABMs for TNBC metastasis initiation and growth in the lungs	Tatiana Miti	tue, 18:00
MS71-08	Equation Learning and Distributional Inference for Modeling Mitochondrial Inheritance in Yeast	Kevin Flores	tue, 18:00
MS76-04	Modular arithmetic as an alternative to model seasonal time-varying transmission rates: Influenza as a case study	Woldegebriel Assefa Woldegerima	tue, 18:00
MS78-07	Evolutionary Therapy in Non-Small Cell Lung Cancer	Kailas Honasoge	tue, 18:00
MS81-08	Study of a bi-monomeric Becker-Döring-type model	Mathieu Mezache	tue, 18:00
CT10	Mathematical modeling of cell proliferation in a scaffold with elastic branching channels	Pejman Sanaei	wed, 08:30
CT123	Modeling the impact of host diversity on the evolution of vector feeding preferences and implications for disease control	Shravani Shetgaonkar	wed, 08:30
CT1239	Path-Independent Transformation of Antagonistic Interactions under Minimal Evolutionary Conditions	Hanna Isaksson	wed, 08:30
CT1255	Coarse-grained PDEs for polar auxin transport in plants.	Alexis Farman	wed, 08:30
CT1297	Model-informed optimisation of dosing strategies using mechanistic tumour growth modelling	Esha Joshi	wed, 08:30
CT1301	Mechanistic Modeling of Cell–Cell Contact Networks in Collective Cancer Invasion	Edwin Weinholtz	wed, 08:30
CT1365	Enhancing Patient Privacy and Model Interpretability with Tensor Trains	Juliette Sinnott	wed, 08:30
CT1390	Biophysical Modeling of Molecular Glues: Mechanistic Insights for Design	Seokjoo Chae	wed, 08:30
CT1396	Simulating how turnover influences actomyosin contraction and bundle formation	Alex Tam	wed, 08:30
CT1404	Prey-predator-parasite models with infection-dependent attack rates	Frank Hilker	wed, 08:30
CT1418	Modeling water-related dynamics in subaerial biofilms interacting with porous stone substrates	Fabiana Russo	wed, 08:30
CT1422	Mechanistic modelling of cluster formation in metastatic melanoma	Nathan Schofield	wed, 08:30
CT1492	Mathematical insights into the dynamics of prostate cancer under phytocannabinoid therapy	Marianna Cerasuolo	wed, 08:30
CT1540	Joint Transmission-Mutation Modelling Shapes the Long-Term Dynamics of Evolving Pathogens	Marvin Schulte	wed, 08:30
CT1572	How does cross-resistance for disease strains influence coexistence outcomes?	Daniel Maes	wed, 08:30
CT1584	Inferring rooted phylogenies from distances	Klaus Schliep	wed, 08:30
CT17	Blocking of Bistable Front Propagation: Application to Wolbachia Population Replacement	Saoussen Latrach	wed, 08:30
CT195	Understanding microbiome assembly and stability through a simplified model	Andreas Eilersen	wed, 08:30
CT256	Resource Limitations Induce Phase Transitions in Optimal Estimation Strategies	Takehiro Tottori	wed, 08:30
CT266	Accelerating Parameter Estimation in Large Systems Biology Models	Andrea Polo Rodríguez	wed, 08:30
CT337	Extending Time to First Subsequent Therapy in the Adjuvant Treatment of High-Grade Serous Ovarian Cancer	Cristina Koprinski	wed, 08:30
CT368	How much should we trust non-identifiable models? Reliable model selection in face of parameter non-identifiability	Elijah Foo	wed, 08:30
CT376	The evolutionary rarity of bacterial endosymbiosis	Eric Libby	wed, 08:30
CT382	From intracellular oscillations to complex behaviour in the unicellular slime mould Physarum polycephalum	Linnéa Gyllingberg	wed, 08:30
CT54	Targeting metabolic fluxes to prevent drug tolerance in Mycobacterium Tuberculosis under host-derived stress	Tanishk Patodi	wed, 08:30
CT9	Qualitative Analysis of a Caputo Fractional-Order System Arising from Within-Host Virus Dynamics	ShyanShiou chen	wed, 08:30
CT109	Balancing Stability and Complexity in Boolean Models of Biological Systems	Venkata Sai Narayana Bavisetty	wed, 08:50
CT110	Metabolic games in an autotroph-heterotroph consortium: eco-evolutionary analysis of cooperative, private and cheater exoenzyme strategies	Tanvir Hassan	wed, 08:50
CT1257	On the Emergence of Traveling Wave Patterns in Resource-Mediated Tissue Competition	Natalia Briñas-Pascual	wed, 08:50
CT1266	Allocation dynamics of stage-specific interventions in structured populations	Daniel Strömbom	wed, 08:50
CT1380	Divergent mechanics of inner ear morphogenesis are coupled to developmental tempo over vertebrate evolution	Shuhei Horiguchi	wed, 08:50
CT1382	Memory T Cells as Predictors of Progression-Free Survival in High-Grade Serous Ovarian Cancer: A Mathematical Modelling Approach	Iman Al Buwaiqi	wed, 08:50
CT1409	The evolution of an evolvability-enhancing condition-dependent mutation rate	Jana M. Riederer	wed, 08:50
CT1427	Understanding quorum-sensing bacteria one model at a time	Silas Poloni	wed, 08:50
CT1445	Parameter Identifiability Under Limited Experimental Data in Age-Structured Models of the Cell Cycle	Ruby Nixson	wed, 08:50
CT1460	What's in a modelling formalism? On the use of graphical models in systems biology	Leo Diaz	wed, 08:50
CT1479	A multi-phase field computational framework to model endothelial cell rearrangement and blood flow-induced polarization	Marcos Gouveia	wed, 08:50
CT1505	Mathematical modeling of macrophage-precancerous epithelial cell interactions in the oral cavity	Lia Sela	wed, 08:50
CT1551	Co-modelling epidemics and specific TTI behaviours	Thomas House	wed, 08:50

Code	Title	Speaker(s)	Date / time
CT1573	A Spatiotemporal Computational Model of Ultrasound-Triggered Nanoparticle Drug Delivery in Solid Tumors	Farshad Moradi Kashkooli	wed, 08:50
CT1585	Ecological and stochastic determinants of the growth and persistence of the oral pathogen Porphyromonas gingivalis	Haralampos Hatzikirou	wed, 08:50
CT1611	A Sufficient Condition for Stable Coexistence Under a Generalist Predator	Lawrence Seminario-Romero	wed, 08:50
CT188	A mathematical model studying the interplay between malaria dynamics and economic growth	Ruijun Zhao	wed, 08:50
CT210	A Bayesian phylogenetic approach using fluctuating methylation to resolve inter- and intra-crypt stem cell dynamics	Calum Gabbutt	wed, 08:50
CT225	Catch-bond dynamics of cell-cell adhesion in the Cellular Potts model predicts dynamics in cell clustering, sorting and invasion	Lisanne Rens	wed, 08:50
CT237	Deciphering Neutrophil Complexity	Gabriel Calvo	wed, 08:50
CT271	Addressing Calibration Challenges in Generalized Lotka–Volterra Models of Microbial Ecosystems	Ana Paredes-Vázquez	wed, 08:50
CT321	Derivation and Analysis of a Low-Dimensional Hopf Oscillator Model for Macroscale Brain Dynamics	Alexander Edthofer	wed, 08:50
CT348	Mathematical Models for Non-Monotone Dose Responses	Peter Rashkov	wed, 08:50
CT370	Exploring Cell-Free Systems Through Robust Sensitivity Analysis	Emily Hunter	wed, 08:50
CT381	Plant tolerance is explained by resource-based plant-nematode interactions	Joseph Penlap	wed, 08:50
CT98	Human papillomavirus driving cervical cancer: a mathematical model with persistent infection, cancer progression, and spontaneous remission.	Rena Hayashi	wed, 08:50
CT111	Asthma-mediated control of optic glioma growth via T cell-microglia interactions: A mathematical model	Donggu Lee	wed, 09:10
CT1252	A Hybrid Model of Tumor Evolution: The Impact of Oxygen on Glioblastoma Dynamics	Alexandra Whiteside	wed, 09:10
CT1278	Metapopulation Dynamics with May–Leonard Competitive Lotka–Volterra systems	Anju Susan Anish	wed, 09:10
CT1332	Subclones, Drivers and Microenvironment in a new Genetic Model of Tumour Growth	Vivien Holmes	wed, 09:10
CT1388	Qualitative study of a tumor-immune interaction model for hepatocellular carcinoma under radiotherapy	Cristina Vilar Medina	wed, 09:10
CT1394	The role of space in the macroecological patterns of microbial abundances	Adrián Gutiérrez Arroyo	wed, 09:10
CT1417	Total biomass and dispersal: effects of local dynamics and network topology	Juan Segura	wed, 09:10
CT1436	Inference of Spatially Varying Tumor Hydraulic Conductivity and Its Impact on Drug Transport	Mohsen Rezaeian	wed, 09:10
CT1491	Venue access policies and intervention timing under vaccine acceptance heterogeneity	Maikel Bosschaert	wed, 09:10
CT1610	Invasion, replacement and coexistence in ecological models	Cameron Browne	wed, 09:10
CT180	Is (Microbial) Ecology Fair?	Teemu Kuosmanen	wed, 09:10
CT187	Modelling and inference of wildfire spread dynamics in UK moorland environment.	Axa-Maria Laaperi	wed, 09:10
CT208	Modelling multiscale cell competitions and cooperations that determine longevity of homoeostasis	Ijas Ahamed N	wed, 09:10
CT295	Identifiability-aware Neural ODEs for parsimonious learning of dynamical systems	Núria Campo-Manzanares	wed, 09:10
CT367	A Spatially and Phenotypically Structured Model of Radiotherapy under Oxygen Heterogeneity	Francesco Albanese	wed, 09:10
CT386	Physiologically Informed Modeling of Aminoglycoside Exposure During Pediatric Maturation	Felix J. Meigel	wed, 09:10
CT395	Applying Koopman operator theory to explore the dynamics of bioreactor models	Cameron Jakub	wed, 09:10
AW-2024	Impacts of genetic and phenotypic heterogeneity on tumor evolution	Simon Syga	wed, 09:40
AW-2025	Modelling and quantifying interactions in collective cell behaviour	Carles Falco	wed, 10:10
CT1204	Closed-Form Steady-State Analysis of Multi-Stage Continuous Bioprocess Cascades	Andrea Graf	wed, 11:10
CT1288	Deterministic discrete-particle modeling of ribosome stalling beyond mean-field approximations	Katrin Schröder	wed, 11:10
CT1413	Household-level patterns of vaccine uptake during the COVID-19 pandemic in the UK	Joe Hilton	wed, 11:10
CT1461	Spatial heterogeneity accelerates phase-to-trigger transitions of mitotic waves	Daniel Ruiz-Reynés	wed, 11:10
CT1502	A three process approach for insomnia-like awakenings	Joe Rowland Adams	wed, 11:10
CT1555	Competing mechanisms for anisotropic lesion formation in cardiac PFA modelling	Luca Gerardo-Giorda	wed, 11:10
CT1570	Machine Learning and Optimization Approaches for Improving Artificial Insemination Service Delivery in Philippine Water Buffalo Systems	Renier Mendoza	wed, 11:10
CT1642	A structured systems dissection of immune response dynamics in Drosophila	Manmath Panigrahy	wed, 11:10
CT270	Agent-Based Model for Superficial Spreading Melanoma Reveals the Role of Cancer Hallmarks in Shaping Intratumor Heterogeneity and Tumor Morphology	Khola Jamshad	wed, 11:10
CT285	PerTexP: scenario-based exploration of pertussis dynamics under maternal and infant vaccination	Emanuela Penitente	wed, 11:10

Code	Title	Speaker(s)	Date / time
CT331	Counterfactual evaluation of elementary and secondary school policies in the COVID-19 pandemic	Benedetta Canfora	wed, 11:10
CT49	Bonsai: Tree representations for distortion-free visualization and exploratory analysis of single-cell omics data	Daan de Groot	wed, 11:10
MS101-01	Timescale separation anxiety in capturing neuronal activity patterns in biophysically detailed models	Jonathan Rubin	wed, 11:10
MS113-01	Stochastic modeling of horizontal gene transfer via M13 bacteriophages in E. coli populations	Alexandra Loudieres	wed, 11:10
MS118-01	Feature-based prediction of preclinical pharmacokinetic profiles using machine learning and compartmental modeling	Felix Jost	wed, 11:10
MS119-01	Community-Level distribution modelling of endangered pelagic bycatch species: a joint modelling approach	Clara Lereboug	wed, 11:10
MS12-01	From Sparse Data to Smart Decisions: Surrogate-Assisted Agent-Based Modeling for Regional Outbreak Response	Carson Dudley	wed, 11:10
MS125-01	Whole-host modeling of immune-pathogen interactions during Tuberculosis infection	Christian Michael	wed, 11:10
MS134-01	A Minimal Model Framework for Robust CAR-T Cell and Oncolytic Virus Combination Therapy	Aisha Tursynkozha	wed, 11:10
MS149-01	Oscillations in the growing tip of the filamentous brown alga Ectocarpus	Nelson Serre	wed, 11:10
MS15-01	Mathematical Modeling and Analysis of Oncolytic Virus and CAR-T Cell Combination Therapy via Switched Optimal Control	Arwa Baabdulla	wed, 11:10
MS151-05	Age-structured models for cell-cycle synchrony	Benjamin Stephens	wed, 11:10
MS152-04	A Boolean Approach to Invasion and Mesenchymal–Epithelial Transition (MET)	Laurence Calzone	wed, 11:10
MS154-01	The Dummy species extension of the Chemical reaction networks; Adding additional species to derive its stationary distribution.	Hoejin Kim	wed, 11:10
MS159-01	MS159-1:Agent-based models, networks and machine-learning models as tools for pandemic preparedness	Jasmina Panovska-Griffiths	wed, 11:10
MS168-01	DNA replication and division cycles coordination in Escherichia coli	Alexandre Perrrin	wed, 11:10
MS175-01	Using reference models to compare multicellular simulation frameworks and tools	Inge Wortel; Lorenzo Veschini; Roman Vetter	wed, 11:10
MS23-01	Modeling of DNA origami electrochemical signaling for optimal biosensor design	Allison Carson	wed, 11:10
MS46-01	Branching annihilating random walk models for self-regulating populations	Alice Callegaro	wed, 11:10
MS51-01	Modelling early immune selection and evasion in tumour evolution	Eszter Lakatos	wed, 11:10

Code	Title	Speaker(s)	Date / time
MS93-01	Pattern Formation and Spike Dynamics in the Presence of Noise	Chunyi Gai	wed, 11:10
CT104	Enhancing epidemiological parameter inference using quantitative diagnostic data	Punya Alahakoon	wed, 11:30
CT1196	The Frustrating Nature of Tissues	Taylor Woodward	wed, 11:30
CT1256	Capturing complex organoid dynamics using image analysis, modeling and simulation	Franziska Matthäus	wed, 11:30
CT1416	A Hybrid Cell-Based Model of Lipid-Immune Interactions in Atherosclerosis.	Thomas Quinlan	wed, 11:30
CT1420	Seasonal Reaction-Diffusion Model of Vector-Borne Diseases: Existence of periodic dynamics	Michela Sabbatino	wed, 11:30
CT1503	Conditions on Interaction Networks for Achieving Specific Reinforcement under Global Dopamine Signals	Yonatan Savir	wed, 11:30
CT1531	Fluid Transport through the Boundary of the Human Brain	Emil Eriksson	wed, 11:30
CT1598	Breaking free from 2D: Extending the cellular Potts model to curved deforming surfaces to understand vascular remodelling	Jessica Crawshaw	wed, 11:30
CT1681	A Conservative Network Framework for Coupled Hemodynamics and Physiological Transport	Lester Caudill	wed, 11:30
CT238	Modelling Immune Dynamics in Locally Advanced MSI-H/dMMR Colorectal Cancer with Neoadjuvant Pembrolizumab Treatment: From Differential Equations to an Agent-Based Framework	Georgio Hawi	wed, 11:30
CT267	Understanding the Biology Underpinning the Interaction between Arboviruses and Aedes aegypti Mosquitoes Using Statistical and Mathematical Modelling	Junjie Chen	wed, 11:30
CT312	The effects of vertical transmission on a spatially-structured host-parasite model	Jack Woodruff	wed, 11:30
MS101-02	Unfolding neuronal excitability: dynamical paths to depolarization block during spreading depolarization events	Marisa Saggio	wed, 11:30
MS113-02	Bridging single-cell and population dynamics of plasmids with an age-structured model	Valentine Brulard	wed, 11:30
MS118-02	Mathematical modelling as a tool to investigate bell-shaped dose-response relationships in drug development	Gianluca Selvaggio	wed, 11:30
MS119-02	Restoring oyster reefs by optimizing metapopulation connectivity	Leah Shaw	wed, 11:30
MS12-02	Quantifying Uncertainty of ABM Parameters Using Explicitly Formulated Surrogate Models	Kerri-Ann Norton	wed, 11:30
MS125-02	Agent-Based Modeling of Nontuberculous Mycobacteria Infection In Vivo with Multi-Modal Patient Data Integration	Kali Konstantinopoulos	wed, 11:30
MS134-02	Using Naive Bayes to uncover probabilistic maps of SIV tissue spread	Chapin Korosec	wed, 11:30
MS149-02	Membrane flows for cell patterning and resilience in fission yeast	Dimitrios Vavylonis	wed, 11:30

Code	Title	Speaker(s)	Date / time
MS15-02	Resistance and immune response during oncolytic virotherapy	Darshak Bhatt	wed, 11:30
MS151-06	Understanding the role of space in modulating cellular heterogeneity	Paras Jain	wed, 11:30
MS152-01	Biological Battle Royale: Structural Constraints of Networks Supporting Multi-Fate Cellular Decisions Using Monotone Boolean Models	Harshavardhan BV	wed, 11:30
MS154-02	Bang–bang population control with reduced fluctuations: lessons from bacteria	Jinsu Kim	wed, 11:30
MS159-02	Starsim: A fast, flexible agent-based disease modeling framework	Fabian Sturman	wed, 11:30
MS168-04	A scenario for an evolutionary selection of ageing: from biological observations to mathematical modelling	Sylvie Méléard	wed, 11:30
MS23-02	Restoring immune regulation in cancer: a multifaceted optimization problem	Anne Talkington	wed, 11:30
MS46-02	A spectral Koopman framework for stochastic reaction networks	Ankit Gupta	wed, 11:30
MS51-02	Deciphering systemic immune cell dynamics to inform immunotherapeutic approaches	Frederik Graw	wed, 11:30
MS93-08	Recurrent disease outbreaks and their frequency	Theodore Kolokolnikov	wed, 11:30
CT1249	Building Spatial Resolution into Ebola virus VP40 Assembly Models: An ODE-to-ABM Transition	Trevor Shoaf	wed, 11:50
CT1269	Stability Analysis of a Multistage Compartmental Model of Infection	Jacobs Somnic	wed, 11:50
CT1270	A Hybrid Agent-Based Framework for Quantifying Mitochondrial Dynamics from Time-Lapse Imaging	Volkan Ozcoban	wed, 11:50
CT1403	Individual behavior can challenge the management of virulence	Clément MONAURY	wed, 11:50
CT1604	A Kuramoto Oscillator Model of Closed-Loop Auditory Stimulation of REM Sleep EEG	Daniel Knott	wed, 11:50
CT1607	Fast, Scalable, and Interpretable Persistent Homology for Biology	Joshua Bull	wed, 11:50
CT1618	The Spatial Regime Conversion Method: An Adaptive Hybrid Framework for Multiscale Reaction–Diffusion Systems	Charlie Cameron	wed, 11:50
CT1646	Biphasic responses in cell signalling systems	J Krishnan	wed, 11:50
CT1683	Integrating physics-informed machine learning and mathematical modelling for cardiac digital twinning	Federica Caforio	wed, 11:50
CT190	Efficient Parameter Estimation in Agent-Based Models of Collective Cell Invasion via Gaussian Process Surrogates and Bayesian Optimization	Ashlee Ford Versypt	wed, 11:50
CT260	Non-local models for spatio-temporal macrophage-tumour co-invasion: role of competition for resources	Johan Marguet	wed, 11:50
CT299	Linking Temporal Structure to Epidemic Dynamics in Contact Networks from Epigames	Estelle McCool	wed, 11:50
MS101-03	Multiscale Organization in a novel Cartwheel Interneuron Model: From averaging to folded node dynamics	Matteo Martin	wed, 11:50
MS113-03	DNA replication as a hidden driver of single-cell timing variability	Juan David Marmolejo Lozano	wed, 11:50
MS118-03	Joint modeling of longitudinal data and survival outcome to improve decision making in oncology drug development	Hoai-Thu Thai	wed, 11:50
MS119-04	Computational topology methods for spatial population pattern quantification	Sarah Day	wed, 11:50
MS12-03	Parameter-wise predictions and sensitivity analysis for random walk models in the life sciences	Matthew Simpson	wed, 11:50
MS125-03	Within-Host Mathematical Models of Tuberculosis: Foundations, Progress, and Future Directions	Roselyn Kaondera-Shava	wed, 11:50
MS134-04	Inferring Mobility Distributions in Heterogeneous Epidemic Models	Weiqi Chu	wed, 11:50
MS149-03	Emergence of polar cell growth in plants: A quantitative perspective.	Zhenbiao Yang	wed, 11:50
MS15-03	Cell evolution in the presence of viruses	Dominik Wodarz	wed, 11:50
MS151-07	A novel cellular automaton approach for modeling genotypic and phenotypic heterogeneity in cell systems	Simon Syga	wed, 11:50
MS152-02	Long-lived sets: The missing oscillatory phenotypes of Boolean networks	Samuel Pastva	wed, 11:50
MS154-03	On the abelian structure of noncompetitive chemical reaction networks.	Louis Faul	wed, 11:50
MS159-03	Comparative evaluation of HIV testing interventions for men who have sex with men in the Netherlands: insights for a low-incidence setting	Ganna Rozhnova	wed, 11:50
MS168-02	2-phase model of ageing : Stability analysis of coupled non-linear partial differential equations with age structure	Luce Breuil	wed, 11:50
MS175-02	Modeling Blastocyst and Organoid Morphogenesis Towards a Virtual Embryo in Morpheus	Lutz Brusch	wed, 11:50
MS23-03	Optimal and Predictive Control of Acute Myeloid Leukemia with Drug Resistance	Pauline Mazel	wed, 11:50
MS46-03	Stochastic ordering tools for reaction network models	Giulio Cuniberti	wed, 11:50
MS51-03	Mathematical modelling of cytokine dynamics in myeloproliferative neoplasm patients during treatment	Johanne Gudmand-Høyer	wed, 11:50
MS93-03	Structure of family trees in finite-population exchangeable models	Eric Foxall	wed, 11:50
CT1231	Nonlinear Stability Analysis and Pattern Formation in Reaction-Diffusion-ODE Systems	Finn Münnich	wed, 12:10

Code	Title	Speaker(s)	Date / time
CT1495	The Matrameru code of biomolecules – Enumerating unbranched fatty acids and amino acids	Stefan Schuster	wed, 12:10
CT1530	Towards digital twins for sleep-wake timing	Anne Skeldon	wed, 12:10
CT1617	Evaluation of Tissue Growth Models for Proliferating Cell Colonies	Zuzanna Szymańska	wed, 12:10
CT1654	Mathematical modeling of signal transduction and gene expression in cancer: from information processing to patient specific models	Jana Wolf	wed, 12:10
CT1655	Probing bacterial fitness in virtual chemostats	Kaan Öcal	wed, 12:10
CT250	Data-driven dynamical analysis of an age-structured model: A graph-theoretic approach	Anuraj Singh	wed, 12:10
CT265	Mathematical modelling reveals that liver architecture impacts tumour size distribution in mouse models of colorectal liver metastases.	Xiaoyuan Liu	wed, 12:10
MS101-04	Slow–fast dynamics in a neurotransmitter release model: Delayed response to a time-dependent input signal	Mattia Sensi	wed, 12:10
MS113-04	Unravelling causal interactions of coding and non-coding RNA from genome wide single-cell data	William Hilton	wed, 12:10
MS118-04	Fit-for-Purpose Modeling in Immuno-Oncology: A Practical Guide to Data and Quantitative Tools Across the Pipeline, featuring a Case Study on novel T Cell Engagers	Inmaculada Sorribes	wed, 12:10
MS119-03	Prediction of spatial population collapse with coarse grain population distribution pattern data	Laura Storch	wed, 12:10
MS12-04	Probabilistic Estimation of Agent Based Models using Hamiltonian Monte Carlo	Benjamin Olsson	wed, 12:10
MS125-04	Mathematical modeling of impact of BCG vaccination on Mycobacterium tuberculosis dynamics in mice	Vitaly Ganusov	wed, 12:10
MS149-04	Mathematical modeling of cell wall elasticity, loosening, and feedback mechanisms on tip-growing morphogenesis	Rholee Xu	wed, 12:10
MS15-04	Computational investigation of the interactions between oncolytic viruses and tumour-associated macrophages	Raluca Eftimie	wed, 12:10
MS151-08	A mathematical model of cell-state dynamics in melanoma	Charlotte Taylor Barca	wed, 12:10
MS152-03	How well do Boolean model capture continuous dynamics of regulatory networks?	Tomas Gedeon	wed, 12:10
MS154-04	On the Stability Properties of Stochastic Chemical Reaction Networks with Phosphorylation	Philippe Robert	wed, 12:10
MS159-04	Improving the use of social contact studies in infectious disease modelling	Tom Britton	wed, 12:10
MS168-03	Crossing a fitness valley in age-structured populations	Sarah Kaakai	wed, 12:10
MS175-03	Born or Made? Modeling the Origins of Leader Cells in Collective Cancer Invasion	Sheriff Akeeb	wed, 12:10
MS23-04	Probabilistic Modeling of Antibody Kinetics Post Infection and Vaccination	Rayanne Luke	wed, 12:10
MS46-04	Remodelling selection: What is it?	Gabriela Gomes	wed, 12:10
MS51-04	A Bayesian ODE model for CAR T-cell kinetics in non-Hodgkin lymphoma	Yifan Chen	wed, 12:10
MS93-04	An inverse problem for the modern Hopfield network	Linh Huynh	wed, 12:10
PLT-05	Behind the barrier: mathematical modelling of the neuroimmune system and multiple sclerosis	Adrianne Jenner	thu, 08:30
PLT-06	A theory, just a theory, or not even a theory? Perspectives on the roles of mathematical modelling	Caroline Colijn	thu, 09:20
CT1318	Clean seed use in seasonal agroecosystems: How growers behave shapes plant epidemic dynamics	Ludovic Mailleret	thu, 10:40
CT1384	Anticipating epidemic waves in non-stationary disease dynamics	Joshua Looker	thu, 10:40
CT1429	A Mathematical Model of Two-Stage Nanomedicine-Based Intraperitoneal Chemotherapy for Peritoneal Carcinomatosis	Mohsen Rezaeian	thu, 10:40
CT1475	Quantitative parameter free methods for toxicokinetics	Inzish Sajid	thu, 10:40
CT1493	Mechanistic Bayesian Mixture Modeling of Infection Establishment and Proliferation	Bruce Rogers	thu, 10:40
CT1500	Modelling mass asymptomatic testing strategies for early containment of infectious disease outbreaks in prisons	Lorenzo Pellis	thu, 10:40
CT1552	Modeling the spatio-temporal effects of broad-beam and mini-beam irradiation on a cell population	Mathilde Badoual	thu, 10:40
CT1643	Personalizing the Patient Model for Depth-of-Hypnosis Control	Gorazd Karer	thu, 10:40
CT1656	Mean first passage time as a framework for immune cell search and encounter	Fatemeh Saghafifar	thu, 10:40
CT222	Quantitative modeling of cytochrome P450 gene expression regulated by pregnane X nuclear receptor	Veronika Bernhauerová	thu, 10:40
CT230	A computational study of physicochemical interactions in DCIS microinvasions	Ana Forero Pinto	thu, 10:40
CT249	A Robust Numerical Framework for Multi-strain Epidemic	Fadilah Ilahi	thu, 10:40
MS101-05	Multimodal Synchronization of Pancreatic Islets	Richard Bertram	thu, 10:40
MS105-01	CRNT approaches in modelling carbon dioxide removal through direct ocean capture	Al Jay Lan Alamin	thu, 10:40
MS126-01	Spatial Heterogeneity in Tumours: How Tumour-Immune Spatial Relationships Impact Immunotherapy Outcomes in Glioblastoma	Blanche Mongeon	thu, 10:40
MS133-01	Optimal Bandwith Selection in Biological Field Effect Transistor Measurements	Luis Melara	thu, 10:40
MS137-01	Goshawk-Squirrel Predator-Prey Dynamics in Patchy Forests Subject to Fire and Forestry Disturbances	Christian Wiewelhove	thu, 10:40
MS14-05	Analytical and Numerical Optimization Methods for Solution of Non-smooth Dynamic Impact Problems	Victor Kovtunenko	thu, 10:40
MS142-03	Collective cell dynamics driven by active boundaries and cell turnover: a multi-scale approach.	Laurent Navoret	thu, 10:40

Code	Title	Speaker(s)	Date / time
MS161-01	Producing quantitative protein function data at scale to enable mechanistic simulation - and protein design	Cyrus Knudsen	thu, 10:40
MS26-01	Entropy-Based Information Sharing for Uncertainty Quantification in Multi-Layer Complex Systems	Grzegorz Rempala	thu, 10:40
MS45-07	Parameter identifiability for the Data-Driven Modeling	Wenrui Hao	thu, 10:40
MS46-05	Mean field limit of non exchangeable interacting diffusions on co-evolutionary networks	Julian Cabrera-Nyst	thu, 10:40
MS60-01	Identifiability of phenotypic adaptation from low-cell-count experiments and a stochastic model	Alexander Browning	thu, 10:40
MS72-03	A quantitative and phenotype-resolved model for melanoma cell populations	Keith Chambers	thu, 10:40
MS75-01	Efficacy-toxicity and toxicity-toxicity trade-offs in head and neck cancer treated with radiotherapy	Daniel Glazar	thu, 10:40
MS79-02	Estimating epidemic risk in spatially and behaviorally structured populations	Eugenio Valdano	thu, 10:40
MS82-01	Non-monotonic attack rates arising from risk-aware and imitation-driven behaviours during epidemics	Gergely Röst	thu, 10:40
MS91-01	How choices in quantifying data affect parameter identifiability in agent-based models of pattern formation	Yue Liu	thu, 10:40
MS93-02	Genealogical structures under interactive neutral reproduction: Ancestral influence graph and Frankenstein process	Ellen Baake	thu, 10:40
MS99-01	Global sensitivity-driven simplification and calibration of Cancer-on-Chip nonlocal integro-differential model	Annachiara Colombi	thu, 10:40
CT115	Mathematical Modeling of Biofilm Eradication Using Optimal Control of Antibiotic Treatment	Rehan Akber	thu, 11:00
CT1326	Spatially explicit individual-based model for schistosome transmission: A bipartite network approach	Melissa A. Iacovidou	thu, 11:00
CT1389	Forecasting SFTS risk in the Republic of Korea under population aging and climate change using machine learning	Yongin Choi	thu, 11:00
CT1494	ROSA: A Geometric Spectral Lift for Detecting Pre-Reorganisation Signals in Noisy Spatial Graphs	Ben Cardoen	thu, 11:00
CT1538	Pathogen non-planktonic phases within the urinary tract impact early infection and resistance evolution	Amanda de Azevedo-Lopes	thu, 11:00
CT1539	Modelling community and healthcare worker transmission using a two-population SEIS Framework informed by ONS survey	jingsi xu	thu, 11:00
CT1557	The Impact of Programmed Cell Death on Cell Population Dynamics: A Stochastic and Moment Dynamics Approach	Peter Boldog	thu, 11:00
CT1603	A spatiotemportal model for prostate cancer	Alethea Barbaro	thu, 11:00
CT1623	Spectral EEG differences using Eleveld and Schnider propofol PK/PD models	Duygu Aydin	thu, 11:00
CT229	Cell Trajectory Inference based on Schrödinger Problem and a Mechanistic Model of Stochastic Gene Expression	Clémence Fournié	thu, 11:00
CT231	Optimization of melanoma treatment with cocktails of CAR-T cells of different affinities	Emilie Favier	thu, 11:00
CT254	Travelling waves in a mathematical model of fireblight during bloom season	Hermann Eberl	thu, 11:00
MS101-06	Cardiomyocyte Models: Bifurcations and Fast-Slow Decomposition	Roberto Barrio	thu, 11:00
MS105-02	Spatiotemporal modeling of signaling pathways: impact of endosomal compartmentalization and application to gonadotropin receptors	Chloe Weckel	thu, 11:00
MS126-02	Granger-causality analysis reveals defective viral genomes with antiviral potential from longitudinal infection data	Tanja Laske	thu, 11:00
MS133-03	A Numerical Method for a Singular Integrodifferential Equation Model of Biosensor Dynamics	Ryan Evans	thu, 11:00
MS137-02	Starvation-driven cell patterning: integrating lab experiments and mathematical modelling	Cinzia Soresina	thu, 11:00
MS161-02	Thermodynamically Consistent Mechanochemical Modelling of Actin Dynamics	Volkan Ozcoban	thu, 11:00
MS26-02	Algebraic, Geometric, and Combinatorial Approaches to Modeling Gene Regulatory Networks	Brandilyn Stigler	thu, 11:00
MS45-01	Effects of Cellular Memory and Adaptation Cost on Optimal Survival in Fluctuating Environments	Paras Jain	thu, 11:00
MS46-06	The effects of individual versus community-influenced isolation on SIS epidemic persistence on finite random graphs	Matthew Wascher	thu, 11:00
MS60-02	From noisy cell size control to population growth: When variability can be beneficial	Arthur Genthon	thu, 11:00
MS72-05	Spin Glass Active Inference for Cancer Stochastic Switching Systems	Linh Huynh	thu, 11:00
MS75-02	Beyond Dose, What Really Drives Radiation-Induced Brain Necrosis?	Ibrahim Chamseddine	thu, 11:00
MS82-04	Behaviour-driven epidemic phenomena in heterogeneous populations and networks	Márton Karsai	thu, 11:00
MS91-02	Cancer systems immunology reveals the tumor-immune dynamics that determine responses to novel combination therapies in metastatic tumor microenvironments	Adam Maclean	thu, 11:00
MS93-05	Multi-type logistic branching processes with selection: frequency process and genealogy for large carrying capacities	Marta Dai Pra	thu, 11:00
CT1334	Estimating fitness effects of mutations that evolve in a Long-Term Evolution Experiment of HIV-1	Jenny Dunstan	thu, 11:20
CT1395	Assessing Birth Month Dependent Effectiveness of Monoclonal Antibodies against Infant RSV Hospitalization: A Modelling Study.	Beryl Musundi	thu, 11:20

Code	Title	Speaker(s)	Date / time
CT1498	Learning dynamical systems with biochemically informed neural ordinary differential equations	Lucas Böttcher	thu, 11:20
CT1542	Simulating glioblastoma microenvironment: How hypoxia and vascular occlusion shape immune dynamics	Raquel Arroyo-Vázquez	thu, 11:20
CT1581	Quantifying biological heterogeneity in nano-engineered particle–cell interaction experiments	Ryan Murphy	thu, 11:20
CT236	Modeling the reversible transition of glioblastoma cells to resistant-senescent states via transport equations	José Antonio Romero Rosales	thu, 11:20
CT263	Evaluating the Public Health Programs during the 2010 Haiti Cholera Outbreak: Disease Dynamics and Epidemic Response	Vijay Pal Bajiya	thu, 11:20
CT300	Vitamin C as a nitrosation inhibitor: a modelling study across dietary patterns and water quality	Gordon R. McNicol	thu, 11:20
CT330	Modelling tissue damage in destructive lung disease	Alice Peyraut	thu, 11:20
MS101-07	Mechanisms for strong symmetry-breaking in coupled fast-slow oscillators	Yangyang Wang	thu, 11:20
MS105-03	From Food Webs to Fluxes: A New Decomposition of Ecosystem Networks	Caner Kazanci	thu, 11:20
MS126-03	Multi-modal data integration and individual cell-based modelling to infer viral spread and innate immune dynamics in human epithelia	Pascal Lukas	thu, 11:20
MS133-04	Probabilistic Cellular Automaton Modeling for Monolayer Degradation in Biosensors	Wes Caldwell	thu, 11:20
MS137-03	Patterned biodiversity responses to disturbance severity in competition-governed metacommunities	Daniel Bearup	thu, 11:20
MS14-03	A Cell–Cell Adhesion Model: Local Well Posedness	Anna Zhigun	thu, 11:20
MS142-01	Pattern formation in a phenotype-structured Shigesada-Kawasaki-Teramoto model	Davide Cusseddu	thu, 11:20
MS161-04	Membrane Transport and Metabolic Patterns: Mathematical Building Blocks and Data-Driven Insights	Adelle Coster	thu, 11:20
MS26-03	Chemical Reaction Networks with Stochastic Switching Behavior and Machine Learning Applications	Hye-Won Kang	thu, 11:20
MS45-02	Multi-omics Investigation Reveals Molecular Determinants of Cancer Cell Evolution on Soft Extracellular Matrix	Sarthak Sahoo	thu, 11:20
MS46-07	Self-intersection local times for Volterra Gaussian processes: Applications to polymer models	Olga Izyumtseva	thu, 11:20
MS60-03	A geometric surface PDE model for cell–nucleus translocation through confinement	Francesca Ballatore	thu, 11:20
MS72-06	Oncolytic Virotherapy: Mathematical Analysis and Optimization	Negar Mohammadnejad	thu, 11:20
MS75-03	Using a mathematical model to predict and optimize response to chemo- and radiation therapy in glioma cells	Reshmi J. S. Patel	thu, 11:20
MS79-01	Sideward contact tracing in an epidemic model with mixing groups	Dongni Zhang	thu, 11:20
MS82-07	Wait or Vaccinate? Behavioural Dynamics of Competing Vaccine Strategies in a Compartmental Epidemic Model with Prevalence-Dependent Hesitancy	Attila Denes	thu, 11:20
MS91-03	Theory and Algorithms for Constructing AI-Based Dynamic Virtual Cells	Peijie Zhou	thu, 11:20
MS93-06	Multitype Lambda-coalescents: Characterisation and duality	Noemi Kurt	thu, 11:20
MS99-07	1D/2D Coupling of HDG discretizations for the simulation of Cancer-on-chip devices	Silvia Bertoluzza	thu, 11:20
CT1335	A temperature-dependant spacial model to optimise sterile male releases for Drosophila suzukii	Jules Guilberteau	thu, 11:40
CT1513	Persistent homology analysis of human populations reveals topological signatures of admixture	Victoria May Mendoza	thu, 11:40
CT1596	Extended Pseudo-spectral Physics-inferred Neural Networks for phase field models	Callum Marsh	thu, 11:40
CT1684	Decoding Clonal Dynamics in MDS Through Adapted Mathematical Models of CHIP	Cameron Kerr	thu, 11:40
CT212	Stochastic-Deterministic Mathematical Models of Neuroblastoma Spatial Structure	Perla Mallouk	thu, 11:40
MS101-08	How slow rhythmic inputs induce spike-adding in neuronal model	Zahra Aminzare	thu, 11:40
MS105-04	A stochastic model of compartmentalized intracellular signalling	Romain Yvinec	thu, 11:40
MS126-04	Identifying Sex-Specific Immune during Primary Influenza Infection Using Mathematical Modeling	Jason E. Shoemaker	thu, 11:40
MS137-04	Response scenarios of the total population size in fluctuating environments	Daniel Franco	thu, 11:40
MS14-06	State-Dependent Sweeping Process Approach to Modeling of Networks with Softening	Ivan Gudoshnikov	thu, 11:40
MS142-02	A Starling resistor model for choroidal venous flow	Federica Vanone	thu, 11:40
MS161-03	The landscape reconsidered: non-equilibrium constraints on cell fate	Lucy Ham	thu, 11:40
MS26-05	Generative models for particle tracking microscopy	Jay Newby	thu, 11:40
MS45-06	Model Selection and Treatment Prediction in ODE Models for ER+ Breast Cancer	Kang-Ling Liao	thu, 11:40
MS46-08	Information and Energy in Stochastic Reaction Networks	Heinz Koeppl	thu, 11:40
MS60-04	Trait-structured chemotaxis: Exploring ligand-receptor dynamics and travelling wave properties in a Keller-Segel model	Viktoria Freingruber	thu, 11:40
MS72-04	Agent-Based Modeling of Tumor-Immune Interactions and Therapeutics in the Tumor Microenvironment	Kerri-Ann Norton	thu, 11:40
MS75-04	Incorporating Toxicity Constraints in Adaptive Cancer Therapy	Kathleen Wilkie	thu, 11:40

Code	Title	Speaker(s)	Date / time
MS79-03	How Within-Host Heterogeneity Shapes Population-Level Transmission: Insights From a Multiscale Model	Fernando Saldaña	thu, 11:40
MS82-08	A network model from sociodemographic heterogeneity to individual infection risk	Congjie Shi	thu, 11:40
MS91-04	Deep State Space Modeling for Real-World Biomedical Time-Series Analysis	Ryosuke Kojima	thu, 11:40
MS93-07	Multi-Type Birth-Death Processes with Mean-Field Interactions for B-cell Phylodynamics	Sebastian Hummel	thu, 11:40
MS99-05	UQ analysis for cancer-on-chip experiments	Laura Rinaldi	thu, 11:40
CT1220	The evolutionary fitness landscape of extrachromosomal DNA in cancer	Magnus Haughey	thu, 14:00
CT1265	Simulation-based inference of epidemic and phylodynamic models via neural posterior estimation	Francesco Pinotti	thu, 14:00
CT1273	Cancer-Associated Fibroblast Heterogeneity Predicts Cancer Progression and Treatment Response	Junho Lee	thu, 14:00
CT1274	Real time response modelling for high pathogenicity avian influenza	Chris Baker	thu, 14:00
CT128	Mathematical models for the treatment of leukemias with CAR-T cells alone and in combination with chemotherapy.	Aitana Moncho Espí	thu, 14:00
CT1282	Dynamics of a modified piece-wise linear Toivonen-Fromhage model	Ryusuke Kon	thu, 14:00
CT1296	On the stability of a non-local kinetic model for density-dependent cell migration	Francesco Maria Mori	thu, 14:00
CT1343	Identifying alternative explanations for population growth rate behaviour between ancestor and mutant strains of Chlamydomonas reinhardtii in stressful environmental conditions using multiple phenotype models	Adam Hillman	thu, 14:00
CT1397	Investigating the Relationship Between Angiogenesis and Tumour Carrying Capacity with Physics Informed Neural Networks	Morghan van Walsum	thu, 14:00
CT1401	A dynamical mechanism for intratumoral heterogeneity in breast cancer	Francisco Lopes	thu, 14:00
CT1448	A reaction-diffusion model of epithelial polarity dynamics in the endometrium over the menstrual cycle	Claire Miller	thu, 14:00
CT1462	Inferring CD8^{+} T Cell Differentiation Trajectories in Cancer through Data-Driven Distribution Modelling	Nissrin Alachkar	thu, 14:00
CT1483	Limiting resistance evolution through drug combinations	Christin Nyhoegen	thu, 14:00
CT1485	Simulation of Dll-4 and Notch Dynamics in a Blood Vessel with Celular Rearrangement	Tiago Gonçalves Pereira	thu, 14:00
CT1506	A mathematical framework for replication stress in cancer	Francisco Berkemeier	thu, 14:00
CT1534	First Explore, Then Settle: A Theoretical Analysis of Evolvability as a Driver of Adaptation	Juan Jiménez-Sánchez	thu, 14:00
CT1550	Gardner volumes and self-organization in a minimal model of complex ecosystems	Frederik Thomsen	thu, 14:00
CT181	Non-ergodicity in ecology and evolution	Teemu Kuosmanen	thu, 14:00
CT186	Toward single-cell control: noise-robust perfect adaptation in biomolecular systems	Dongju Lim	thu, 14:00
CT196	Application of Radial Basis Function Physics-Informed Neural Networks in Fractional Pharmacokinetics Modeling	Reza Mokhtari	thu, 14:00
CT233	Generation time for diseases with variable infectiousness before and after symptom onset: beyond geometric waiting times.	Jordi Ripoll	thu, 14:00
CT240	OP7, a promising influenza virus-derived interfering defective particle: multiscale modeling, role of the innate immune response and prediction of treatment windows	Daniel Rüdiger	thu, 14:00
CT246	Selective sweep probabilites in spatially expanding populations	Alexander Stein	thu, 14:00
CT255	Dissecting GLUT4 Trafficking: Structural Motifs, Insulin Responsiveness and Retention	Adelle Coster	thu, 14:00
CT306	Quantification of Information Flow by Dual Reporter System and Its Application to Bacterial Chemotaxis	Kento Nakamura	thu, 14:00
CT392	Spectral Parameterization of EEG Dynamics during Emergence from General Anesthesia	Sandra Widmann	thu, 14:00
CT393	Inferring Tissue Evolution Dynamics from DNA Methylation Profiles	Catherine Engert	thu, 14:00
CT397	A Systematic Framework for Inferring Stochastic Dynamics from Data	Katarina Bodova	thu, 14:00
CT1215	Novel methods for analyzing dynamics of functional brain networks: complex correlation patterns defining a robust functional architecture	Maria Ercsey-Ravasz	thu, 14:20
CT1216	Mathematical modelling of 5-aminolevulinic acid in glioblastoma	Charlotte Baxter	thu, 14:20
CT1281	Modeling Administration Time-Of-Day Effects in Clinical Research: A Simulation Study with Application to Cancer Chronotherapy	Sacha Revillon	thu, 14:20
CT1293	Identical ancestor points and maximal specieslike clusters	Samuel Alexander	thu, 14:20
CT1319	Genetic Switching Dynamics in a Spatial Branching Model of Cancer Metastasis	Lena Zuspann	thu, 14:20
CT1340	When Saboteur Bacteria Undermine Chemotherapy: A Mathematical Model of Cancer Treatment Dynamics	Anna Geretovszky	thu, 14:20
CT1341	Exploiting acid-dependent 'public bad' dynamics to develop normalizing adaptive therapies	Kevin Scibilia	thu, 14:20
CT135	Mechanisms of Synergy and Resistance: Modeling Dynamic PD-L1 Regulation in Combination Immunotherapy	Bruce Pell	thu, 14:20

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CT1350	Stability in asymmetric evolutionary games	Aradhana Narang	thu, 14:20
CT1441	Integrating Mechanistic Modeling and Machine Learning to Predict CAR-T Therapy Outcomes from CD4+/CD8+ Dynamics	Saranya Varakunan	thu, 14:20
CT1455	Modeling the Geospatial Dynamics of Lyme Disease in Maryland Under Projected Climate Scenarios	Salihu Musa	thu, 14:20
CT1470	Geometric analysis of an epidemiological model for Olive Quick Decline Syndrome (OQDS)	Keren Tapper	thu, 14:20
CT1511	Pharmacokinetic-Based Optimization of Dynamic PET Time Acquisition Protocols for Cancer Theranostics	Diego Samuel Rodrigues	thu, 14:20
CT1536	How group communication changes under competition	Gayashan Jayavilal	thu, 14:20
CT1562	A model of adipose cell population dynamics under variable energy balance	Aleksandra Tomaszek	thu, 14:20
CT16	Coupled Information-Infection Dynamics Produce Information-Driven Shifts in Epidemic Regimes via Competing Vaccine Narratives	Asma Azizi	thu, 14:20
CT1639	Understanding breakthrough infections following mpox vaccination: variation in the frequency of clinical characteristics	Anna Fairweather	thu, 14:20
CT182	Quantifying fitness, selection and survival in population trees with cell death	Arthur Genthon	thu, 14:20
CT191	Modeling circadian–contraceptive interactions: Simulations reveal daytime dosing as optimal for pill efficacy	Brenda Lyn Gavina	thu, 14:20
CT199	Interaction-based networks describe chemical pathways within proteins	Fabian Schuhmann	thu, 14:20
CT218	Opposites attract – the role of electrostatic polarisation in the sensory ecology of arthropods	Ryan Palmer	thu, 14:20
CT234	Eco-Epidemic Reaction–Diffusion Model with Fear and Carryover Effects: Stability and Pattern Formation	Namrata Mani Tripathi	thu, 14:20
CT279	Mathematical Modeling of Tertiary Lymphoid Structure Development and Stage Progression in Chronic Kidney Disease	Jinghao Chen	thu, 14:20
CT280	Optimal strategies for utilizing host plant distributions to slow the spread of plant pests	Adam Lampert	thu, 14:20
CT322	Semi-amortised simulation-based inference for stochastic nonlinear mixed-effects models	Henrik Häggström	thu, 14:20
CT341	Optimal control of symmetry-breaking in cell fate specification	Pearson Miller	thu, 14:20
CT347	Control of electrophysiology experiments in open and closed loop	Melvyn Tyloo	thu, 14:20
CT398	Invariant Image Reparameterisation for Identifiability Analysis of Individual-Based Models	Oliver Maclaren	thu, 14:20
CT1227	The trade-off between fractal morphology and function of Drosophila neurons	Ildikó-Beáta Márton	thu, 14:40
CT1232	A Nonlinear ODE Model of Butyrate-Tumour-Immune Cell Dynamics in Colorectal Cancer	Alexandra Lawryshyn	thu, 14:40
CT1240	PINNs for structured population dynamics inference: Application to oocyte dynamics in fish ovaries	Louis Fostier	thu, 14:40
CT1258	An agent-based modelling approach to investigate the impact of gender on tuberculosis transmission in Uganda	James Doran	thu, 14:40
CT1321	Lightning Laplace methods for floral-arthropod electrostatics	Samuel Harris	thu, 14:40
CT1352	Intraspecific trait variation and niche choice in predator-prey dynamics	Anastasiia Enne	thu, 14:40
CT1357	Modeling of glioblastoma recurrence based on local microenvironment characteristics	Hermann Frieboes	thu, 14:40
CT1361	Agent-based modeling of multiple myeloma in the aged bone microenvironment	Riley Hall	thu, 14:40
CT1406	Spatial remodeling of the tumor-immune microenvironment by neoadjuvant chemoradiotherapy in NSCLC	Zuzanna Nowicka-Kaszkowiak	thu, 14:40
CT1454	A mathematical model of osteocyte network growth	Jack Fenwick	thu, 14:40
CT1458	Mesoscale community organization governs epidemic onset and spread in metapopulations	Malbor Asllani	thu, 14:40
CT1512	TopROI: Topological partition and analysis of colorectal cancer tissue architecture	Sergio Serrano de Haro Iváñez	thu, 14:40
CT153	From clustering to dispersal: collective invasion driven by adhesion and motility	Josue Manik Nava	thu, 14:40
CT1546	Global well-posedness and stability analysis of a degenerate system for a prey-predator and natural enemy interaction	Duc-Anh Nguyen	thu, 14:40
CT185	Predicting the fate of mutation rate modifiers that shift the mutation bias	Diego Tenoch Morales Lopez	thu, 14:40
CT206	Explaining the dynamical consequences of cis binding in a mathematical model of Notch signaling.	Specioza Nambooze	thu, 14:40
CT294	Optimising booster vaccination against SARS-CoV-2 variants: when should a variant-adapted vaccine be developed?	Abbie Evans	thu, 14:40
CT304	Eco-evolutionary dynamics of proliferation: from a community model to tumor growth	Farnoush Farahpour	thu, 14:40
CT310	Regimes and mechanisms of inflammation described by reaction-diffusion systems	Wissam El Hajj	thu, 14:40
CT324	Using Approximate Bayesian Computation for Model Selection in a Dual-Layer Cell Migration Model	Tracy Stepien	thu, 14:40
CT344	Threshold-Driven Collapse and Recovery in a Simplified Wasp-Waist Ecosystem Model	Sam Subbey	thu, 14:40
CT352	In vivo spatial dynamics of actomyosin oscillations	Jochen Kursawe	thu, 14:40
MS103-03	Cross-border transmission, vaccination heterogeneity, and structural inequity during the 2025 measles outbreak in Chihuahua, Mexico	Jorge Velasco	thu, 15:10

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MS105-06	Mathematical modeling to infer signaling bias: kinetics and spatial compartmentalization	Romain Yvinec	thu, 15:10
MS126-05	Regularized estimation in high-dimensional mechanistic models: Application to vaccine development	Lisa Crépin	thu, 15:10
MS131-01	Mathematical of pre-exposure prophylaxis and the quest to end the HIV epidemic	Abba Gumel	thu, 15:10
MS14-08	Recent Results on Sweeping Processes with Asymmetric Continuity Properties	Vincenzo Recupero	thu, 15:10
MS142-04	Active shape evolution in tissue mechanics	Naoufel Cresson	thu, 15:10
MS161-05	What would a smart yeast do? Mapping biosynthetic resource allocation to explain prevalence of the Crabtree effect in yeasts	Pranas Grigaitis	thu, 15:10
MS162-01	Unseen but not unstoppable: Quantifying the impact of human compliance and mass screening and treatment on cryptic asymptomatic malaria transmission	Arnaja Mitra	thu, 15:10
MS165-01	Mechanistically Grounded Digital Twins for Relapse Forecasting in Acute Lymphoblastic Leukemia Using Multi-Omic Data	Sergio Márquez-Díaz	thu, 15:10
MS177-04	Retinal processing of natural scenes	Olivier Marre	thu, 15:10
MS26-04	From Blackboard to Bedside: Translating Mathematical Sleep Models into Samsung Galaxy Watch	Jae Kyoung Kim	thu, 15:10
MS45-04	How Random Forces Help Rather Than Hinder Dynein Cargo Transport	John Dallon	thu, 15:10
MS65-01	Rigorous and Automated Non-dimensionalisation, Preprocessing, and Reduction of Biochemical Reaction Networks	Georgio Hawi	thu, 15:10
MS72-01	A Multiscale Mathematical Framework for Modeling Ductal Deformation in Early Pancreatic Neoplasia	Daniel Bergman	thu, 15:10
MS75-05	Controlling drug-induced phenotypic plasticity via model-informed treatment scheduling	Einar Bjarki Gunnarsson	thu, 15:10
MS79-04	Genuine and spurious bistability in a simple epidemic model with waning immunity	Francesca Scarabel	thu, 15:10
MS82-02	Leveraging machine learning algorithms to model health behavior in network-based epidemic models	Joshua Chevalier	thu, 15:10
MS91-05	Data and Modelling for Medical Inverse Problems	Adam Stinchcombe	thu, 15:10
MS99-03	A Thermodynamically Consistent Multiphysics Model of Irreversible Electroporation for Cutaneous Melanoma: Coupling GENERIC Thermo-Poromechanics with Tissue Electroporation	Davide Baroli	thu, 15:10
MS126-06	Modeling how memory CD8 T cells may effect post-treatment control of HIV infection	Narendra Dixit	thu, 15:30
MS131-02	Failed rescue effect in metapopulations: when increased dispersal results in total collapse	Frank Hilker	thu, 15:30
MS14-07	Variational Modeling of AFM Indentation of Living Cells with a Pericellular Coat	Ivan Argatov	thu, 15:30
MS142-05	Modelling the emergence of connective tissue architecture	Pauline Chassonnery	thu, 15:30
MS161-06	How Do T Cells Migrate Through Collagen? Inferring Subcellular Forces from Fiber-Resolved Extracellular Matrix Deformations	Jeremy Worsfold	thu, 15:30
MS162-02	Decision-Support Modeling for One Health Pathogens: Using Mechanistic Models for Surveillance and Forecast Design	Joshua Macdonald	thu, 15:30
MS165-02	Co-expression Networks in Cancer	Britta Daub	thu, 15:30
MS26-06	Toward Predictive Digital Twins for Precision Oncology: A QSP Modeling Approach	Leili Shahriyari	thu, 15:30
MS65-02	Tikhonov-Fenichel Reductions: A Systematic Approach to Timescale Separation and its Application to Modelling Population Dynamics	Johannes Apelt	thu, 15:30
MS72-07	Mechanistic learning for spatio-temporal brain tumor response predictions	Sarah Brueningk	thu, 15:30
MS75-06	Agent-based modeling of intratumor heterogeneity coordination	Michela Prunella	thu, 15:30
MS79-05	Network effect and behavioral feedback in epidemics	Martina Alutto	thu, 15:30
MS82-05	Trending on the use of Google mobility data in COVID-19 mathematical models	Daihai He	thu, 15:30
MS91-06	Multi-Omics–Driven Mathematical Framework for Integrating A Mechanistic Model and Data in Precision Medicine	Sungrim Seirin-Lee	thu, 15:30
MS99-04	A Data-Informed Agent-Based Model of Tuberculosis Granuloma-Like Structures	Davide Moretti	thu, 15:30
MS103-01	The effectiveness of non-pharmaceutical interventions against COVID-19	Jane Heffernan	thu, 15:50
MS105-05	Linking structure to behaviour in regulatory biochemical reaction networks through geometric and combinatorial methods	Ahmad Mokhtar	thu, 15:50
MS126-07	Quantitative modeling of proliferation and differentiation in cell populations	Shoya Iwanami	thu, 15:50
MS131-03	Changing Connectivity: A Spatial Understanding of Bistable Coral Dynamics	Jennifer Paige	thu, 15:50
MS14-04	On the Mathematical Analysis of Generalized Fractional Viscoelastic Models	Hiromichi Itou	thu, 15:50
MS142-06	A new size-dependant individual based model for epithelial tissue	Sophie Hecht	thu, 15:50
MS161-07	Agent-based Modelling of Molecular Dynamics in the Gram-negative Outer Membrane	Thomas Williams	thu, 15:50
MS162-03	Rethinking mosquito biting rates: exploring how disturbed blood-feeding shapes vectorial capacity	Kyle Dahlin	thu, 15:50

Code	Title	Speaker(s)	Date / time
MS165-03	Mathematical Modeling of Prostate Growth for Digital Twin–Based Monitoring in Active Surveillance	Valentin Schmid	thu, 15:50
MS177-01	A Mathematical Framework for Photoreceptor Dynamics in Health and Disease: Modeling Metabolic Mechanisms and Spatial Dependence	Danielle Brager	thu, 15:50
MS26-07	Exploration of structure learning in pediatric sickle cell data	Reginald McGee	thu, 15:50
MS45-03	How Intermediate Filaments Move: Experiments and Models	Stephanie Portet	thu, 15:50
MS65-03	Lumping of Reaction Networks: Generic and Critical Parameters	Santiago Schnell	thu, 15:50
MS72-08	Mechanistic computational modeling of clonal dynamics in the aging hematopoietic system	Thomas Stiehl	thu, 15:50
MS75-07	Mechanistic and Machine Learning Models for Dose Scheduling and Treatment Response Prediction	Mohammad Kohandel	thu, 15:50
MS79-06	Modelling information-dependent protective behaviour in response to mosquito-borne epidemic outbreaks	Simone De Reggi	thu, 15:50
MS82-06	A Filippov Model Describing the Effect of Social Distancing in Controlling Infectious Diseases	Stacey Smith?	thu, 15:50
MS91-07	Towards Reliable Single-Cell Foundation Models: A Fully Data-Driven Framework for Signal Detection and Clustering via Mathematical Theories	JaeKyoung Kim	thu, 15:50
MS99-02	An adaptive high-order polytopal method for modeling neuronal electrophysiology	Caterina B. Leimer Saglio	thu, 15:50
MS103-02	Infectious disease surveillance using deep learning models	Bouchra Nasri	thu, 16:10
MS105-07	Mapping the co-expression landscape: network sparsification and null models	Rosemary Braun	thu, 16:10
MS126-08	Identifying determinants of long-term viral suppression following broadly neutralizing antibody treatment against HIV-1	Tyler Cassidy	thu, 16:10
MS131-04	Mathematical Models for evolutionary phenomena during range expansion	Silas Poloni	thu, 16:10
MS14-02	Towards Robust Quantitative Photoacoustic Tomography	Andreas Hauptmann	thu, 16:10
MS142-07	PDE models for the growth of heterogeneous cell populations: travelling fronts, sharp interfaces, and concentration phenomena	Tommaso Lorenzi	thu, 16:10
MS161-08	Model-based integration of large-scale datasets	Jan Hasenauer	thu, 16:10
MS162-04	Variable impact of density-dependent life history traits on the success of mosquito population reduction strategies	Lauren Childs	thu, 16:10
MS165-04	Modeling resistance multiverse: from metabolic activity to immune evasion	Malgorzata Tyczynska Weh	thu, 16:10
MS177-02	A Dual-Intervention Optimal Control Framework for Photoreceptor Degeneration Using RdCVF and RdCVFL	Erika Camacho	thu, 16:10
MS26-08	Asthma-mediated control of optic glioma growth via T cell-microglia interactions.	Yangjin Kim	thu, 16:10
MS45-05	Senolytic Drugs Reduce Cancer Resistance to PD-1 Blockade	Nourridine Siewe	thu, 16:10
MS65-04	Total QSSA: Pharmacokinetic Applications and the Validity of Its Stochastic Extension	Yun Min Song	thu, 16:10
MS72-02	Digital Twins in (Radiation) Oncology	Heiko Enderling	thu, 16:10
MS75-08	Personalizing Radiotherapy in HPV+ Oropharyngeal Cancer with Systematic In Silico Trials	Mohammad Zahid	thu, 16:10
MS79-07	SEIR Models With Host Heterogeneity: Main Properties And Applications To Seasonal Influenza Dynamics	Tamas Tekeli	thu, 16:10
MS82-03	The influence of human interactions on waterborne diseases: a reaction-diffusion model	Maria Francesca Carfora	thu, 16:10
MS91-08	Equation Learning for Agent-Based Infectious Disease Models	Kevin Flores	thu, 16:10
MS99-06	HDG-Based Discretization of a Mathematical Model for Multiple Sclerosis	Micol Pennacchio	thu, 16:10
CT1195	Perception of infection risk drives backward bifurcation during outbreaks	Alexis Erich Almocera	thu, 17:00
CT1200	When does it pay to eat your friend?	Josephine Solowiej-Wedderburn	thu, 17:00
CT1280	Kron Reduction in Species-Reaction Graphs of Mass-Action Chemical Reaction Networks	Manvel Gasparyan	thu, 17:00
CT1317	Branching Process Models of Sub-Critical FMD Transmission: Inference, Control, and Surveillance Design in Karnataka, India	Glen Guyver-Fletcher	thu, 17:00
CT1356	Transmission dynamics of Mpox: Implications for Global Control from the 2024 Outbreak	Rehan Akber	thu, 17:00
CT1359	A phylodynamic model for within-host HCV infection integrating genomic sequences and time series biomarkers	Wei-Ting Chen	thu, 17:00
CT1560	Modelling the active coordination of cell adhesion and contractility in cells and tissues	Carina Dunlop	thu, 17:00
CT1577	Modeling PSA dynamics to predict resistance during intermittent androgen deprivation therapy	Minhye Kim	thu, 17:00
CT1616	How inertia affects autotoxicity-mediated vegetation pattern dynamics	Giancarlo Consolo	thu, 17:00
CT21	Can Cheating Go Viral? A Classroom-Based Social Contagion Model of AI Misuse	Donna Dyer	thu, 17:00
CT297	Discrepancies between discrete and continuum models: insights from oncolytic virotherapy	David Morselli	thu, 17:00
CT345	Mapping the spread of artemisinin resistance in Africa	Lucy Harrison	thu, 17:00
MS103-05	Qualitative impact of political and economic decisions on PrEP users under HIV epidemic control	Laurent Pujo-Menjouet	thu, 17:00

Code	Title	Speaker(s)	Date / time
MS107-04	Tracking the evolution, spread and reassortment patterns of avian influenza viruses using sequence data and phylodynamic modelling	Lu Lu	thu, 17:00
MS112-01	Existence of a unique non-degenerate solution to parametrized systems of generalized polynomial equations	Abhishek Deshpande	thu, 17:00
MS130-01	Modelling longitudinal vaccine-elicited immune profiles in people living with HIV	Chapin Korosec	thu, 17:00
MS143-01	Control treatments on surface-dependent formation and dissolution of amyloid plaques in Alzheimer's disease	Francesca Acotto	thu, 17:00
MS147-01	Going underground: roots and rhizosphere hydraulic processes impacting plant response to drought	Mutez Ahmed	thu, 17:00
MS156-01	Modeling mechanisms of neuronal microtubule dynamics and polarity	Anna Nelson	thu, 17:00
MS165-05	Modeling phenotypic switch in cancer drug response to identify new drug combinations	Marielle Pere	thu, 17:00
MS166-01	Modeling the role of oxidative stress in aging and its impact on female fertility	Ana Portillo	thu, 17:00
MS169-01	Optimizing HIV Antiretroviral Therapy Strategies: A Multi-Scale, Multi-Objective Framework for Reconciling Individual–Population Trade-offs	Stacey Smith?	thu, 17:00
MS172-01	Regulatory Motifs in C. elegans	Brandilyn Stigler	thu, 17:00
MS176-01	Digital Twins of Morphogenesis	Mark Alber	thu, 17:00
MS178-01	Transmission and early infection as key elements of multi-scale viral dynamics modelling	Daniel Coombs	thu, 17:00
MS44-01	Spatiotemporal dynamics of tumor - CAR T-cell interaction	Ivana Bozic	thu, 17:00
MS57-01	Exploring the connection between hypoxia and intra-tumour heterogeneity through mathematical modelling	Giulia Celora	thu, 17:00
MS68-04	Drug dosing design guided by response and resistance convexity	Jeffrey West	thu, 17:00
MS77-04	Stochastic Gene Expression: Modeling and Inference from Single-Cell Data	Ramon Grima	thu, 17:00
MS84-04	Modeling the Ocular Immune Response to Seasonal Allergic Conjunctivitis and the Effects of Treatment	Lucia Carichino	thu, 17:00
MS96-01	Alignment processes in cells: From individual interactions to collective behaviour	Angelika Manhart	thu, 17:00
CT1206	Stochastic foraging and resource accumulation with queueing theory	José Giral-Barajas	thu, 17:20
CT1208	Mathematical modeling and optimal control analysis of impact of information-induced vaccination and saturated treatment on disease prevalence	Anuj Kumar	thu, 17:20
CT1320	A Kinetic Model of Yeast Fermentation Incorporating Nitrogen Metabolism	Diego Troitiño-Jordedo	thu, 17:20
CT1322	Impact of infant monoclonal antibody protection on RSV dynamics: a stage-structured modelling study	Anna Autoriello	thu, 17:20

Code	Title	Speaker(s)	Date / time
CT1360	Inferring the rules of TCR–pMHC binding using synthetic co-evolution	Antonio Matas Gil	thu, 17:20
CT1367	Phylodynamic inference of the contributions of different contact types to infectious disease transmission	Don Klinkenberg	thu, 17:20
CT1554	From Classrooms to Communities: Modeling the Impact of School-Based Interventions for Future Pandemic Response	Zicheng Fang	thu, 17:20
CT1566	Towards a thermodynamic consistent framework for reversible-irreversible hysteresis in epigenetic landscapes	Jacobo Ayensa-Jiménez	thu, 17:20
CT1602	Effects of Cell Orientation on Formation of Ordered Arrangement of Photoreceptor Cells in the Zebrafish Retina: A Lattice Model Study	Keiichi Yamamoto	thu, 17:20
CT1634	Dynamics of Consumer–Resource Systems	Rui Li	thu, 17:20
CT307	Personalizing Cancer Therapy by Modeling the Evolutionary Dynamics of the Tumor Immune Microenvironment	Qingpeng Zhang	thu, 17:20
CT387	Designing a feedback-oriented outreach activity: insights from a trial and error approach	Christin Nyhoegen	thu, 17:20
MS103-04	From within-host to between-hosts models via a virtual cohort	Julien Arino	thu, 17:20
MS112-02	Unbounded oscillations in the Selkov model of glycolysis with Michaelis-Menten kinetics	Alan Rendall	thu, 17:20
MS130-02	Mathematical Modeling of HIV-Driven Accelerated Biological Aging via Long-Lived Cell Reservoirs	Esteban Hernandez-Vargas	thu, 17:20
MS143-02	Tonic-clonic seizure transitions as bifurcations in a neural field model	Roberto Barrio	thu, 17:20
MS147-02	Geometry hidden in plain stress, Exploring the relationship between pressure-induced stresses and geometry in plant tissues.	Olivier Ali	thu, 17:20
MS156-02	Three-Dimensional Modeling of Kinesin Mechanics in Optical Traps	Christel Hohenegger	thu, 17:20
MS165-06	From Black Board, to Plate, to Patient: Dissecting Resistance Dynamics to Personalise Cancer Treatment Scheduling	Maximilan Strobl	thu, 17:20
MS166-02	Stochastic modelling of telomere length dynamics in cell populations with ALT survivors	Juan Mardomingo-Sanz	thu, 17:20
MS169-02	The population-level implications of immune boosting and immunological priming	Nir Gavish	thu, 17:20
MS172-02	BoolForge: Controlled generation and analysis of Boolean functions and networks	Claus Kadelka	thu, 17:20
MS176-02	A Patient-Specific Digital Twin for Adaptive Radiotherapy of Non-Small Cell Lung Cancer	Jun Deng	thu, 17:20
MS178-02	Stochastic Process and PDE Models for Cross-Scale Evolutionary Dynamics	Daniel Cooney	thu, 17:20
MS44-02	Predicting CAR T-cell Therapy Outcome and Cytokine Release Syndrome Through Data-Driven Mathematical Models	Luciana Barros	thu, 17:20

Code	Title	Speaker(s)	Date / time
MS57-02	The role of hypoxic memory on tumor invasion under cyclic hypoxia	Gopinath Sadhu	thu, 17:20
MS84-08	Modeling the impact of venous collapsibility on retinal blood flow	Tajkera Khatun	thu, 17:20
MS96-02	A mathematical framework for coupled mechanics and mechanosensing in adherent cells	Gordon R. McNicol	thu, 17:20
CT1214	Far-from-equilibrium patterns in a cross-diffusion vegetation-autotoxicity model	Frits Veerman	thu, 17:40
CT1219	Improving Parameter Identifiability in Household-Transmission Models Using Genomic Data	Thomas House	thu, 17:40
CT1323	Linking aggrephagy and mTOR signaling in amyotrophic lateral sclerosis: a minimal ODE model	Alex De Nardi	thu, 17:40
CT1339	The elasticity of SARS-CoV-2 transmission across the demographic landscape	Rey Audie Escosio	thu, 17:40
CT1372	Modeling the Heterogeneous Efficacy of Broad-Spectrum Medical Countermeasures: A Hybrid Approach from Outbreak Containment to Disease Burden Mitigation	Youngsuk Ko	thu, 17:40
CT1377	A Stochastic Germinal Center Model in Antigenic Space for Cross-Variant Antibody Responses to SARS-CoV-2 Vaccination	Rodolfo G. Blanco-Rodriguez	thu, 17:40
CT1405	Community-Engaged Learning in an Interdisciplinary Math-Biology Course	Brynja Kohler	thu, 17:40
CT1556	The Effectiveness of Contact Tracing on Clustered Networks: An Event-Driven Network Simulation Study	Martin Bootsma; Oluwapelumi Stephen Awolude	thu, 17:40
CT1595	A topological framework for the analysis of complex cellular interactions	MARIA JOSE JIMENEZ	thu, 17:40
CT1624	How little is enough? Can low-complexity parameters discriminate wakefulness and anesthetized states?	Sandra Widmann	thu, 17:40
CT1635	Effects of Fitness Gradient	Jakub Svoboda	thu, 17:40
CT313	Mathematical modelling of combination treatment of polycythaemia vera explains observed synergy of drugs	Tobias Idor Boklund	thu, 17:40
MS103-06	A Basic Reproduction Number for Pair Formation Models with Co-Infection	Iain Moyles	thu, 17:40
MS107-01	A modelling assessment of the impact of control measures on highly pathogenic avian influenza transmission in poultry in Great Britain	Edward Hill	thu, 17:40
MS112-03	Instabilities and pattern formation in distributive double phosphorylation	Carsten Conradi	thu, 17:40
MS130-03	A stochastic model of HIV viral rebound after treatment interruption	Jasmine Kreig	thu, 17:40
MS143-03	A Functional Network Method for Monitoring State Changes in Neural Activity	Richard Bertram	thu, 17:40
MS147-03	Fluid mechanics of the plant cell wall: from cellulose reorientation to twisting growth	Galane Luo	thu, 17:40
MS156-03	Analyzing Partially Observed Stochastic Epidemic Networks	Grzegorz Rempala	thu, 17:40
MS165-07	How Modulation of the Tumor Microenvironment Drives Cancer Immune Escape Dynamics	Pujan Shrestha	thu, 17:40
MS166-03	Stochastic Modeling of Telomere Shortening and Reconstruction and Connections with Carcinogenesis	Marek Kimmel	thu, 17:40
MS169-03	A Hybrid Networked SEIR Model with Generative-AI Driven Agents for Behavioral Heterogeneity in Epidemics	Jia Zhao	thu, 17:40
MS172-03	Towards Complement-based Therapeutics: Opportunities for (Discrete) Mathematical Modeling	Daniel Cruz	thu, 17:40
MS176-03	Machine Learning-Enabled Digital Twins for Personalized Modeling of Alzheimer's Disease	Wenrui Hao	thu, 17:40
MS178-03	Modelling the relationship between circulating levels of respiratory syncytial virus and health risk to infants	Alireza Tafreshi	thu, 17:40
MS44-03	Quantifying target antigen-dependent CAR T-cell performance against AML	Philipp Altrock	thu, 17:40
MS57-03	Population-level and agent-based models for a quantitative understanding of the tumor-immune interaction	Jason Thomas George	thu, 17:40
MS68-01	Mathematical modeling of tissue morphogenesis and regeneration	Diane Peurichard	thu, 17:40
MS77-01	Clone wars: understanding clonal diversity in resistance to CAR-T cell immunotherapy	Adriana Zanca	thu, 17:40
MS84-03	Untangling abnormal retinal blood vessels: Integrating computational modelling, image analysis and in vivo imaging	Katie Bentley	thu, 17:40
MS96-03	A geometric surface PDE framework to model cell migration	David Hernández-Aristizábal	thu, 17:40
CT1226	Evaluating infectious disease forecasts in a cost-loss situation	Philip Gerlee	thu, 18:00
CT1330	Mechanistic modeling of extracellular vesicle–mediated immune evasion in Candida albicans	Anastasia Solomatina	thu, 18:00
CT1349	Time Until Disease Detection in a Stochastic Epidemic Model with Imperfect Vaccination	Diana Taipe Hidalgo	thu, 18:00
CT1378	The evolution of latency in tuberculosis	Alexander Beams	thu, 18:00
CT1481	What happens when an epidemiologist gets refractory giardiasis? Within-host modelling of duodenal infection dynamics and oral treatment	David Haw	thu, 18:00
CT1516	Population dynamics of gene drives with density-dependent fitness costs	Adam Lampert	thu, 18:00
CT1559	Mathematical Modeling of Tuberculosis Transmission in Comorbid Populations: A Six-Compartmental Stability Analysis	Abigael Dangate	thu, 18:00

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CT1597	Deep reinforcement learning driven evolutionary therapy design for non-small cell lung cancer	Laura Jansen-Storbacka	thu, 18:00
CT1640	A reference panel assisted geometric structure non-negative matrix factorization for the deconvolution of bulk tissue RNA-seq data	Shaoyu Li	thu, 18:00
CT1645	Forcing-Driven Nonlinear Dynamics of Satellite-Informed Agro–Forestry Socio-Ecological Systems	Gerard Olivar-Tost	thu, 18:00
CT318	Investigating the impact of infusion time-of-day on the antitumour efficacy of immune checkpoint inhibitors	Marie Haghebaert	thu, 18:00
MS103-07	Instabilities in a disease model incorporating compliance	Jacques Bélair	thu, 18:00
MS107-02	Modelling H9N2 AIV transmission along poultry production and distribution networks in Bangladesh	Francesco Pinotti	thu, 18:00
MS112-04	Asymptotic stability of delayed complex balanced reaction networks with general kinetics	Gábor Szederkényi	thu, 18:00
MS130-04	Investigating Heterogeneity in HIV Viral Rebound Times	Jessica Conway	thu, 18:00
MS143-04	Modeling the formation of perinuclear crowns made of agglutinated ATM proteins observed in fibroblasts from patients affected by Alzheimer's disease	Laurent Pujo-Menjouet	thu, 18:00
MS147-04	The biomechanical basis of fixed handedness coiling in Cardamine fruit valves	Gabriella Mosca	thu, 18:00
MS156-04	Generative models for particle tracking microscopy	Jay Newby	thu, 18:00
MS165-08	Integrating glioblastoma plasticity into combination therapy strategies to overcome therapeutic resistance: a quantitative systems pharmacology approach	Thibault Delobel	thu, 18:00
MS166-04	Mathematical modeling and parameter inference of telomere length regulation in yeast	Viviana Gavilanes Guerrero	thu, 18:00
MS169-04	The young shield: Mathematical modeling of the impact of age-targeted and dose-structured vaccination on malaria dynamics	Arnaja Mitra	thu, 18:00
MS172-04	Prioritizing Control Strategies in Boolean Networks Using Mutation Scores and Network Modularity	David Murrugarra	thu, 18:00
MS176-04	Integrating Multimodal Patient Data with Biophysical Models for Predicting Patient-Specific Tumor Progression	Parag Katira	thu, 18:00
MS178-04	Cross-Scale Drivers of Antimicrobial Resistance: Competition, Trade-offs, and Landscape Structure	Jessica Hite	thu, 18:00
MS44-04	Synergistic interplay of morphology and metabolic activity rule response to CAR T-cells in B-cell lymphomas	Soukaina Sabir	thu, 18:00
MS57-04	Growth and adaptation mechanisms of tumour spheroids with time-dependent oxygen availability	Ryan Murphy	thu, 18:00
MS68-02	Leveraging spatial data analysis to provide robust, model-agnostic, and accurate measurements of pattern formation	Duncan Martinson	thu, 18:00
MS77-02	Detection Noise Renormalizes Apparent Kinetic Rates in Stochastic Gene Regulatory Networks	Iryna Zabaikina	thu, 18:00
MS84-06	Mathematical modelling to explore the role of metabolic dysfunction in retinal disease	Paul A. Roberts	thu, 18:00
MS96-04	Modeling active cell migration in complex confined environments: The interplay between nuclear mechanics, chemotaxis, and obstacle geometry	Francesca Ballatore	thu, 18:00
PLT-07	Estimating tipping points in climate	Susanne Ditlevsen	fri, 08:30
CT1218	A Novel Immunological Therapeutic Strategy Based on Morphology-Defined Endotypes of Skin Disease	Ying Xie	fri, 09:30
CT1225	Pattern Selection in mechanochemical model of pattern formation	Szymon Cygan	fri, 09:30
CT1279	Existence of periodic traveling waves in neural fields with direction preference	Maren Bråthen Kristoffersen	fri, 09:30
CT1291	Mechanistic mathematical modeling of primary myelofibrosis pathophysiology and progression	Qiujie Wang	fri, 09:30
CT1299	Information degradation across data transformations affect causal inference	Henrik Høiberg Jessen	fri, 09:30
CT1306	A distance-space framework for inferring selection from time-resolved trait data	Farnoush Farahpour	fri, 09:30
CT1324	Soil Biodiversity and Ecological Niches Under Climate Change: Modelling Insights from the BIOservicES Project	Rajneesh Kumar	fri, 09:30
CT1368	Phenotypical Characterization and Regional Morphometric Variation of the Invasive Indian Crow (Corvus splendens) in the Sultanate of Oman	Qais Abdullah SULIAMAN ALRawahi	fri, 09:30
CT1411	Mathematical Modelling and Optimisation of Combined Oncolytic Virotherapy and Anti-PD-L1 Immunotherapy	Hongfei Qiao	fri, 09:30
CT1434	Self-Organization in Stochastic Multiscale Systems with Conflict Resolution Dynamics	Richard Kollar	fri, 09:30
CT1465	Treatment Scheduling Modulates Vascular Normalization, Interstitial Fluid Pressure, and Drug Delivery in Solid Tumors	Defne Yilmaz	fri, 09:30
CT1477	Machine Learning–Based Classification of Microsatellite Instability in Colorectal Carcinoma Using PCR Fragment Length Analysis	Isabel Gernand	fri, 09:30
CT1478	From Checkerboards to Synchrony: How Mobility Alters Coupled Oscillator Dynamics	Tilmann Glimm	fri, 09:30
CT1486	Replicator Dynamics as a Framework for Invasion in Microbial Communities	Tomas Freire	fri, 09:30
CT1490	Mathematical Modelling and Numerical Simulations for Lipolysis on Lipid Droplets	Reymart Salcedo Lagunero	fri, 09:30
CT1571	Standing Wave Formation in In-Vitro Ultrasound Cell Culture Systems: Multiphysics Modeling and Experimental Investigation	Farshad Moradi Kashkooli	fri, 09:30

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CT1613	Total progeny analysis for infectious clusters within epidemics, with applications to COVID-19 in Albania.	Liam Critcher	fri, 09:30
CT197	Heavy-tailed dormancy and the role of cancer cell death in metastatic reactivation: a data-driven mathematical modelling study	Nikolaos Sfakianakis	fri, 09:30
CT204	Molecular noise modulates transitions in the cell-fate differentiation landscape	Yujing Liu	fri, 09:30
CT221	A hybrid response surface–ODE framework for multiscale predictions of microalgae growth dynamics	Mélanie Pietri	fri, 09:30
CT253	Mind the Gaps: How the Spiral of Silence Drives Partisan Misperception in the United States Political System	Daniel Levy	fri, 09:30
CT326	Exploring reaction-diffusion equations: Modelling wildfires and resistance development	Luca Madita Nieding	fri, 09:30
CT327	HORIZON: Hierarchical Optimization for Residual Inference with Zero-drift Ornstein-Uhlenbeck Noise	Yuhong Liu	fri, 09:30
CT1245	Filtered Schemes for a Continuum Model of DNA Transcription	Lisa Davis	fri, 09:50
CT1262	Can we hear the shape of a neural network?"	Giovanni Naldi	fri, 09:50
CT1287	Dynamical systems analysis of a model coupling hematopoiesis, hematological malignancy and inflammation.	Morten Andersen	fri, 09:50
CT1304	Macaque social network structure beyond rank assortativity	Diogo Pires	fri, 09:50
CT1325	Mathematical modelling of actin polymerisation in biological condensates	Fynn Wolf	fri, 09:50
CT1327	Inferring Cell-Cell Interaction Dynamics from Cell Trajectory Data Using Deep Attention Networks, and the Mechanisms Underlying T Cell Behavioural Heterogeneity in the Response to Cancer	James Boyle	fri, 09:50
CT1355	Robust regimens predicted using a virtual murine cohort of bladder cancer with experimental validation	Hannah Anderson	fri, 09:50
CT1415	Multiple observations in blood cancer dynamics of AML, CML, and MPNs are comprehensively explained by the unifying Multiple Clone Cancitis model.	Johnny Ottesen	fri, 09:50
CT1430	Macroscopic Models for Hard Anisotropic Particles	Carmela Moschella	fri, 09:50
CT1437	A mathematical model for a therapeutic approach for atherosclerosis	Nader El Khatib	fri, 09:50
CT1456	Habitat fragmentation promotes spatial scale separation under resource competition	Malbor Asllani	fri, 09:50
CT1472	Extended Kalman Filter for Real-Time Estimation of Hidden Bone Marrow Dynamics from Peripheral Blood Measurements	Haralampos Hatzikirou	fri, 09:50
CT1488	Structural Identifiability of Stoichiometric Matrix in Mass-Action Biochemical Reaction Networks	Shivam Yadav	fri, 09:50
CT1497	Spectral Fragility in Biological Spatial Networks under Geometric Vertex Noise	Ben Cardoen	fri, 09:50
CT1541	Phase-Field Model of Red Blood Cell Morphology in Sickle-Cell Anaemia	João Maria Loureiro de Oliveira Costa Santos	fri, 09:50
CT1544	Hexagons of Health: Robust Liver Detoxification Emerges from Lobular Geometry	Daniel Sanchez-Taltavull	fri, 09:50
CT1575	Connecting Vertex Based and Centroid Based Epithelial Monolayer Models	Patrick Grant	fri, 09:50
CT1609	Some measures for epidemic decision-making under limited resources in a stochastic SVIR model	María Gamboa Pérez	fri, 09:50
CT207	Coupled plasticity axes give rise to coordinated cellular behaviour and differentially aggressive attractor states in breast cancer	Ritesh Kumar Meena	fri, 09:50
CT242	Structural identifiability of stochastic processes: from single-particle trajectories to total particle density data	Arianna Ceccarelli	fri, 09:50
CT323	Population modelling under limited data: using expert knowledge in model calibration	Sarah Vollert	fri, 09:50
CT328	Efficient Integration of Binary Qualitative Data into ODE Model Estimation	Domagoj Doresic	fri, 09:50
CT338	Interferon-mediated antiviral protection limits Influenza spread: A spatial agent-based model	Rodolfo Blanco-Rodriguez	fri, 09:50
MS107-03	Co-circulation of low and highly pathogenic avian influenza viruses in poultry farms: an integrated experimental and modeling framework	Hadi Taghvafard	fri, 10:40
MS112-05	Automatic initial estimates of reaction rate coefficients	János Tóth	fri, 10:40
MS130-05	Tracking and predicting the dynamics of HIV-1 epidemics in France using virus genomic data	Louis Colliot	fri, 10:40
MS131-05	How the Tulips get their Stripes	Thomas Hillen	fri, 10:40
MS143-05	A Multimodal Mathematical Model of Iron and Amyloid-β in Alzheimer's Disease	Ilaria Stura	fri, 10:40
MS147-05	Three-dimensional multiscale modelling of plant tissue mechanobiology	João R. D. Ramos	fri, 10:40
MS156-05	Parameter Estimation for ODEs, Curve Registration, and Particle Filtering.	John Fricks	fri, 10:40
MS169-05	Optimal release of sterile males in the sterile insect technique for Anopheles mosquitoes	Clarke Safsten	fri, 10:40
MS170-01	Data Driven Modeling of Limb Bud Growth and Morphogenesis	Tim Liebisch	fri, 10:40
MS172-05	Reproducible Boolean model analyses and simulations with the CoLoMoTo software suite	Denis Thieffry	fri, 10:40
MS176-05	Developing a Digital Twin of the Maternal Brain	Sarah Kushne	fri, 10:40

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MS35-01	Tumor growth and immune response: on the impact of the space-structuration of the tumor microenvironment	Christian Tayou-Fotso	fri, 10:40
MS55-06	Multi-scale Models Arising in Chemical Reaction Networks	Lea Popovic	fri, 10:40
MS58-01	Characterization of macrophage phenotypes using mathematical modelling	Soujanya Barik	fri, 10:40
MS77-03	Neural approximations for models of transcriptional dynamics enable genome-wide parameter inference	Maria Carilli	fri, 10:40
MS96-05	From epithelial architecture to chemotaxis and polarity: multiscale mechanical modeling of cell behavior with CompuCell3D	James A. Glazier	fri, 10:40
MS107-05	Avian Flu Modelling in poultry farms: Case study of H5N1 HPAI in northern Italy in 2021–2022	Mattia Sensi	fri, 11:00
MS112-06	Turing instability in reaction networks with one diffusing species	Maya Mincheva	fri, 11:00
MS130-06	Elucidating the mechanism of synergy between latency reversal agents and broadly neutralizing antibodies for HIV remission	Narendra Dixit	fri, 11:00
MS131-07	Impact of Network Variation on Metapopulation Dynamics	Zhisheng Shuai	fri, 11:00
MS143-06	Folded node canard dynamics as a mechanism for delay in astrocytic evoked-Ca2+ responses	Matteo Martin	fri, 11:00
MS147-06	Impact of cellular geometry on the effective mechanics of growing plant tissues	Annamaria Kiss	fri, 11:00
MS169-06	Incorporating thermal performance curves into population dynamic models for the West Nile vector, Culex pipiens	Ben Bruncati	fri, 11:00
MS170-02	Oscillator coupling separates morphogenesis and patterning into two developmental modules	James Hammond	fri, 11:00
MS176-06	The mechanobiology of blood vessel formation: Towards a Digital Twin of Zebrafish intersegmental vessel formation	Roeland Merks	fri, 11:00
MS35-02	Modelling the efficacy of CIK cell immunotherapy targeting MET-expressing mesothelioma cells	Federica Padovano	fri, 11:00
MS55-07	Making Sense of Reaction Networks via Atoms and Inheritance	Murad Banaji	fri, 11:00
MS58-02	Dissecting immune-cell communication networks in chronic inflammation and cancer	Kevin Thurley	fri, 11:00
MS77-06	The Impact of the Cell Cycle on Cellular Decision Making	Charli Austin	fri, 11:00
MS96-06	Guidance by followers: inference of microscopic mechanical cell coordination through quantitative modelling	Jörn Starruß	fri, 11:00
MS107-06	Ecological and environmental predictors of highly pathogenic avian influenza risk in wild birds across Europe: a Bayesian machine learning approach	Joe Hilton	fri, 11:20
MS112-07	Buffering Structures and Factorization of the Jacobi Determinant in Biochemical Reaction Networks	Máté László Telek	fri, 11:20
MS130-07	Can we PrEP Our Way Out of the HIV Epidemic?	Salman Safdar	fri, 11:20
MS131-08	When Systems Tip: Predicting Critical Transitions and Uncovering Frequency-Induced Tipping	Shan Gao	fri, 11:20
MS143-07	How neuromodulation shapes breathing: from cellular dynamcis to network rhythmogenesis	Yangyang Wang	fri, 11:20
MS147-07	Busse balloon Barbapapa	Eric Siero	fri, 11:20
MS156-07	Stochastic fountain dynamics and associated challenges for inference	Scott McKinley	fri, 11:20
MS169-08	The effect of co-infection on disease transmission in plants with multiple transmission pathways	Hannah Hirsch	fri, 11:20
MS170-03	Image-based modelling explains variability in morphogen diffusion measurements	Yi Ting Loo	fri, 11:20
MS172-06	Using Canalizing Boolean Functions to Model Regulation in Mammalian Cell Division	Elena Dimitrova	fri, 11:20
MS176-08	The impact of ephaptic coupling and ionic electrodiffusion on arrhythmogenesis in the heart	Ning Wei	fri, 11:20
MS35-03	Tumour–Immune Dynamics: Patterns, Trade-offs, and Evasion in a Continuous Modelling Framework	Giulia Chiari	fri, 11:20
MS55-01	Bifurcations of Equilibria in Mass-action Systems	Balázs Boros	fri, 11:20
MS58-03	When data tells the story: Uncovering transcriptional control landscapes in cancer systems using data-driven model inference	Malvina Marku	fri, 11:20
MS77-07	Simulation-free approximate Bayesian computation for stochastic reaction networks and its applications on scRNA-seq data	Zekai Li	fri, 11:20
MS96-07	A Multiscale Model of Contact-Guided Cell Migration Incorporating Cell Morphodynamics	Nadia Loy	fri, 11:20
MS107-07	The role of ducks in detecting Highly Pathogenic Avian Influenza in small-scale backyard poultry farms	Steve Wu	fri, 11:40
MS112-08	Autocatalysis in Reaction Networks with Explicit Catalysis	Richard Golnik	fri, 11:40
MS130-08	Binding mediated clearance of anti-HIV-1 broadly neutralizing antibodies in viremic individuals	Tyler Cassidy	fri, 11:40
MS143-08	A Chemical Reaction Network-based approach to mTOR signaling dysregulation in drug-resistant epilepsy	Silvia Berra	fri, 11:40
MS147-08	Understanding the Biomechanics of Plant Tissues: the consequences of cell division, shape and arrangement	Euan Smithers	fri, 11:40
MS170-04	Collisions, Shape and Stigmergy: Modelling the Collective Organisation of ECM	Omar El Oakley	fri, 11:40
MS172-07	Structural Coherence Drives Cooperative Dynamics in Gene Networks	Pradyumna Harlapur	fri, 11:40
MS176-07	Virtual dynamics of spatiotemporal single-cell interactions	Peijie Zhou	fri, 11:40
MS35-04	Mechanistic modeling of the combined dynamics of CAR T-cell therapy and standard care in malignant gliomas	Matteo Italia	fri, 11:40
MS55-02	QSDs and Reaction Networks	Carsten Wiuf	fri, 11:40

Code	Title	Speaker(s)	Date / time
MS58-04	From Cells to Tissues: Multiscale Systems Biology on the Road to Digital Twins	Arnau Montagud	fri, 11:40
MS96-08	A purely mechanical model with bending bacteria for early morphogenesis of micro-colony growth	Sophie Hecht	fri, 11:40
PLT-08	The Geometry of Decision-Making	Iain Couzin	fri, 14:00
MS50-02	The polyhedral structure of the disguised toric locus	Diego Rojas La Luz	TBD

Poster Presentations

Code	Title	Speaker(s)	Date / time
PP226	How do bee's smell? The multi-physics of honeybee olfaction	Ryan Palmer	tue, 18:30
PP136	ABM–Deep Gaussian Process Virtual Patient Population Reveal Interferon-Dependent Efficacy of Sirolimus in SARS-CoV-2 Infection	Henrique de Assis Lopes Ribeiro	tue, 19:30
PP1197	Structural Gradients in Excess Mortality During and After the COVID-19 Pandemic: Economic Development, Preparedness, and Cross-National Differences, 2020–2024	Daeil Jang	tue, 20:10
PP120	DNA Strand Displacement Sequential Release Circuit for Scheduled Therapeutic Interventions	Alivia Behnke	tue, 20:10
PP1202	Systems-level Analysis of CAF Signaling Networks Identifies MASTL-driven Angiogenic Regulation	Jae-Young Kim	tue, 20:10
PP1209	Development of a Generalized Real-Time Early Detection Model based on Bootstrap Clustering	Yeonsu Lee	tue, 20:10
PP1224	Emergent patterns in systems coupling diffusive and non-diffusive components	Finn Münnich	tue, 20:10
PP1228	A parallel asymmetric particle Gaussian mixture filter for state-space estimation of highly nonlinear oscillators	San Kim	tue, 20:10
PP1230	PK-PD Model-Based Control Design for Anesthesia	Alexander Edthofer	tue, 20:10
PP1238	Fluctuations of plasmid copy numbers in a population	Valentine Brulard	tue, 20:10
PP1260	A non-autonomous continuous mathematical model for the Alzheimer biomarker cascade	Antonio Baeza	tue, 20:10
PP1264	Early Detection of Malaria Outbreaks Using Incubation Period Distributions and Machine Learning	Jeonghwa Seo	tue, 20:10
PP1268	Inferring Incubation-Related Temporal Structure in Korean Malaria Incidence Using Attention-Based Time-Series Models	Seongyu Yu	tue, 20:10
PP1272	Self-Organizing Neural Tissue Simulation Platform for Inferring and Validating Developmental Mechanisms	Jaekyoung Lee	tue, 20:10
PP1276	Sex-Specific Temporal Dynamics of Human Sleepiness	Hyunji Jeong	tue, 20:10
PP1285	Exploring the Neuroblastoma Microenvironment Using Cellular Automata: Insights into Celyvir Therapy.	José García Otero	tue, 20:10
PP1289	A Mathematical Approach for Enhancing Tumor Drug Delivery via Collagen Normalization	Onur Kerem Özmen	tue, 20:10
PP1292	Extending the Next Generation Matrix method outside of Mathematical Epidemiology	Andrei-Dan Halanay	tue, 20:10
PP1298	Molecular dynamics simulations of epilepsy- associated SCN1A variants for guiding pharmacological treatments	Mistre Bekele	tue, 20:10
PP1300	Improving Parameter Identifiability of Stochastic Reaction Network Systems Using Approximate Bayesian Computation	Matthew Brown	tue, 20:10
PP1309	Multiscale Simulation of Ovarian Follicle Dynamics: Linking PDE and ODE Descriptions	Edilbert Christhuraj	tue, 20:10
PP1312	Topological Data Analysis Reflects Bifurcation Structure in a Spatial SIR Agent-Based Model with a Locality Parameter	Miriam Van de Water	tue, 20:10
PP1313	The Nuclear Mechanome: A Stochastic Dynamical Characterization of Leukemic Cell Nuclei	Carlos del Pozo Rojas	tue, 20:10
PP1338	Extracting Hidden Cellular Dynamics: A Bayesian Approach to Estimating Time-varying Growth Rates from Noisy Microscopy Data	Vishvas Ranjan	tue, 20:10
PP1342	Genotoxic Effects of Bacteria and Drugs: Investigation of Hidden Interactions with a Differential Equation-Based Approach	Anna Geretovszky	tue, 20:10
PP1344	Evolution of the cell transcriptome during differentiation	Julien Blohm	tue, 20:10
PP1345	Inferring Intercellular Interaction Rules for HER2+ Breast Cancer Cell Motility Using Weak Form Inference	Allison Introne	tue, 20:10
PP1354	Parameter estimation in infectious disease models from infected case data using metaheuristic-tuned PINN	Arrianne Crystal Velasco	tue, 20:10
PP1364	Advance Prediction of Immunotherapy Outcomes Using Deep Learning	Juliette Sinnott	tue, 20:10
PP1369	A Mechanistic ODE Framework for Hormetic Dose–Response: Formulation, Identifiability, and the Limits of Static Data	Aini Fitriyah	tue, 20:10
PP1370	Perturbing MECOM self-activation reveals multistable hematopoietic cell fate landscapes with cusp and elliptic umbilic-like catastrophes	Jonathan Martinez	tue, 20:10
PP1373	Doubly Structured Models of Tumour-Immune Cell Reactions	Fenora David	tue, 20:10
PP1379	A Scalable Network-Based Parameterization of Positive Steady States for Dynamical Analysis of Biochemical Systems	Exequiel Jun Villejo	tue, 20:10
PP1383	Impact of Influenza Antiviral Treatment on the Risk of Household Transmission and the Serial Interval	Jihyeon Kim	tue, 20:10
PP1386	Wavelet-Based Machine Learning Prediction of Norovirus Detection Rates in South Korea	Giphil Cho	tue, 20:10
PP1392	Bacterial Attachment to Medical Implants: How can Mathematics Help us Understand and Prevent Biofilm Formation?	Otilia Casuneanu	tue, 20:10
PP1400	Mathematical Models for the Mechanics of Soft Tissues: From Linear Elasticity to Morpho-Visco-Poroelasticity	Sabia Asghar	tue, 20:10
PP1407	A Water Transfer Experiment Linking Physical Systems and Mathematical Models	Brynja Kohler	tue, 20:10
PP1410	Applying applied maths: mathematical biology in health economics	Joe Hilton	tue, 20:10

Code	Title	Speaker(s)	Date / time
PP1414	A Dynamical Model of Pandemic Dynamics with Healthcare Capacity Constraints	Carmen Martí Raga	tue, 20:10
PP1423	T-cell Motility in Confined Geometry: a Statistical Analysis and Model Comparison	Lorenzo Huang	tue, 20:10
PP1425	Development of a computational microscopy pipeline for the inference of the subcellular and population-scale mechanome of red blood cells	Jorge Barcenilla González	tue, 20:10
PP1439	Linking Social Signals from Large Language Models to Epidemic Transmission Dynamics through Physics-Informed Neural Networks	Dasom Lee	tue, 20:10
PP1442	Dynamical System Model Discovery from Sparse Data with GFlowNets and PINNs	Saranya Varakunan	tue, 20:10
PP1453	An investigation of The Role of Light in Sleep-Wake Dynamics based on Mathematical Model of Sleep pressure and Circadian Rhythm	Seunghun Lee	tue, 20:10
PP1469	Large-Scale Mechanistic Modeling for Patient-Specific Drug Recommendation and Organoid Testing in Colorectal Cancer	Moritz Richter	tue, 20:10
PP1474	The role of phenotypic plasticity in evolution of coexistence.	Vishnu Venugopal	tue, 20:10
PP1484	Learning Optimal Adaptive Therapies in Space and Time: Dynamic Threshold Therapy for Prostate Cancer	Yunli Qi	tue, 20:10
PP1489	Numerical Eigenvalue Analysis of an ODE Model Representing the Endocrine System	Clara Horvath; Marie-Sophie Kohlmayer	tue, 20:10
PP1504	Decoding Nonequilibrium Dynamics in Biological Systems with Horizontal Visibility Graphs	Lucía Benito Barca	tue, 20:10
PP1519	Sensitivity analysis for biological differential equation models with many parameters	Dilan Pathirana	tue, 20:10
PP1523	Modelling the Range Change Dynamics of Host-Macroparasite Systems under Climate Change using Reaction-Diffusion Equations	Haley Morris	tue, 20:10
PP1527	MICAL2 expression promotes invasion and metastasis by cell autonomous and non-cell autonomous mechanisms in Pancreatic Cells	Katherine Simms	tue, 20:10
PP1537	Threshold Dynamics in a Time-Delayed Logistic Model of Cell Populations	Villő Glavosits	tue, 20:10
PP1545	A Mathematical Framework for Per-Read and Per-Sequence Error Characterization in Single-Molecule Sequencing	Pranjal Srivastava	tue, 20:10
PP1549	User-friendly Modeling and Simulation of Multiscale Biological Processes using Morpheus	Lutz Brusch	tue, 20:10
PP1553	Contrastive Self-Supervised Learning for Decoding Physical Parameters in Stochastic Gene Expression Dynamics	Xi (Ian) Yang	tue, 20:10
PP1590	Mathematical Modelling of Antibiotic Diffusion in Bacterial Biofilms	Florence WILLIAMS	tue, 20:10
PP1591	An agent-based model to investigate the dynamics of HIF, TGF-α, and EGFR signalling under hypoxic conditions in cancer spheroids	Vaishnudebi Dutta	tue, 20:10
PP1594	Analysis of the Role of Network-Plasticity-Related Proteins in Molecular Networks	Mark Kerestely	tue, 20:10
PP1605	Mathematical model of adipose cell size and number	Aloïs Dauger	tue, 20:10
PP1620	Assessing the Safety of Pulsed Field Ablation in the presence of Implantable Medical Devices	Bakhtawar bibi	tue, 20:10
PP1622	Characterizing and Modeling Age and Drug Related EEG Waveform Asymmetry during Anesthesia	Marie Steil	tue, 20:10
PP1626	Benchmarking temporal causal discovery for fully and partially observed biochemical kinetic models	Holly Chambers	tue, 20:10
PP1632	A combined mathematical and experimental approach to understanding tristetraprolin regulation in inflammation	Teri Parsonage	tue, 20:10
PP1648	A mathematical framework for quantifying T cell expansion in acute and chronic regulatory contexts	Joanna Schnorr	tue, 20:10
PP1649	Reconstruction of neuromorphic dynamics from a single scalar time series using variational autoencoder and neural network map for cell culture data	Nataliya Stankevich	tue, 20:10
PP1667	What Survival Reveals and Misses: Dose-response inference in a preclinical immuno-oncology study	Teresa Ackerl	tue, 20:10
PP194	Quantifying serial killing via multiscale Bayesian comparison of models for sequential NK cell responses	Elephes Sung	tue, 20:10
PP200	Reconstructing High-Resolution Tau Distributions from Regional tau-PET Data Using Implicit Neural Representations	Ashish Raj	tue, 20:10
PP223	Modeling tumor dynamics under PROTAC therapy: a mathematical model for resistance analysis	Cecilia Torboli	tue, 20:10
PP268	Physics-Informed Neural Networks for Inferring Tumor Glucose–Lactate Metabolic Dynamics	Shadi Vandvajdi	tue, 20:10
PP286	From adaptation to collapse: a model of thermoregulation under prolonged cold stress	Pavel Morcinek	tue, 20:10
PP288	One item per disorder: a cross-disorder optimization framework for simultaneous prediction of six psychiatric conditions	Myna Lim	tue, 20:10
PP298	Quantifying Relative Contributions of Driving Factors of ILI Incidence Dynamics across Europe	Pauline Bakker	tue, 20:10
PP305	Inferring the Interaction Between Circadian Rhythms and Heart Rate Dynamics from Wearable Data	Kangmin Lee	tue, 20:10
PP311	A mathematical framework to accurately reconstruct cell lineage from single cell transcriptomics on barcoded cells: application for therapeutics optimization	Bence Hajdu	tue, 20:10
PP315	Same Calories, Different Outcomes: the Dynamics of Fasting in Cancer	Andrés Méndiz Fernández	tue, 20:10

Code	Title	Speaker(s)	Date / time
PP325	Three-delay model for glucose–insulin–glucagon regulation	Michelle Ann Abhijit	tue, 20:10
PP332	Protein-Ligand Affinity Prediction via Topologically-Aware Graph Neural Network	Perry Beamer	tue, 20:10
PP349	Comparing systems of ODEs to structured models for modeling heterogeneity of neural stem cells	Carolin Lindow	tue, 20:10
PP355	Linking tumour microenvironment dynamics to PET radiomic signatures: a computational modelling framework	Sahar Jafari Nivlouei	tue, 20:10
PP365	Investigating the roles of prior chemotherapy and risk type in bi-specific T-cell engager therapy against B-ALL via mathematical modeling and Bayesian inference	Yifan Chen	tue, 20:10
PP374	Modeling Adult Neural Stem Cells in Zebrafish With Feedback	Theo Andre	tue, 20:10
PP380	Identifying Key Patient-Specific Parameters of the Schnider PK-PD Model using Population Kinetics	Christina Huber	tue, 20:10
PP389	Machine Learning for Gene Calling: Neural Network Integration for BBTools' CallGenes	Brandon Imstepf	tue, 20:10
PP391	Multi-scale modeling of embryonic organoids	Marielle Péré	tue, 20:10
PP59	Considerations on Model Complexity with respect to Parameter Sensitivity and Identifiability: A case study of cell invasion	Veronika Hofmann	tue, 20:10
PP292	Evaluating and developing models for survival extrapolation	Maiya Tebäck	thu, 18:30
PP1194	Hopf Bifurcations Unravel Complex Antibody Dynamics in COVID-19 Patients	Alexis Erich Almocera	thu, 18:50
PP335	Developing a digital twin of 3D vascular systems to study haemorrhagic viral diseases	Jiale Miao	thu, 19:10
PP228	FEM Simulation of STING Pathway Activation for Glioblastoma Immunotherapy	Minjong Kim	thu, 19:30
PP247	A forecasting pipeline for triple-negative breast response to neoadjuvant chemotherapy: combining MRI and PK/PD-informed modeling with a mechanical constraint on proliferation.	Thomas Gallagher Romero	thu, 19:50
PP1201	Reconstructing Influenza Incidence from Sentinel Surveillance Data Using a Latent INGARCH Model	Changdae Son	thu, 20:10
PP1203	Codifying the rules of microbial community physiology for biotechnology designs: fighting natural biochemistry with its own sword	Pranas Grigaitis	thu, 20:10
PP1205	Ladderpath: A Compression-Based Hierarchical Framework for Biological Sequence Analysis	Yu Liu	thu, 20:10
PP1207	Quantifying Variant-Specific Transmissibility and Forecasting Summer COVID-19 Outbreaks in Korea	Yoonah Kang	thu, 20:10
PP1217	Deconstructing Pathogen Transmission: Cumulative Edge Flux in Empirical Temporal Networks	Nathan Millen	thu, 20:10
PP1222	Benchmarking of the robustness of mechanistic generative models in cellular trajectory and gene regulatory network joint inference	Yann Maugé	thu, 20:10
PP1223	Mathematical Modelling of the Tumour Microenvironment and $\gamma\delta$ T cells in Ovarian Carcinoma	Cedrik Neber	thu, 20:10
PP1253	Reaction Kinetics of Ryanodine Receptors in a Closed–Open–Inactivated Model	Anna Hlubinová	thu, 20:10
PP1263	Asymptotic problems for the Keller-Segel system with density cut-off	Mingyue Zhang	thu, 20:10
PP1267	Fluctuating Trait-Switching Opportunities Generate Bistable Cultural Dynamics in the Absence of Conformist Bias	Rinto Yoshizaki	thu, 20:10
PP1271	Thermodynamically Consistent Mechanochemical Modelling of Actin Dynamics	Volkan Ozcoban	thu, 20:10
PP1275	Bridging Reaction–Diffusion Models and Empirical Data: Reparameterization for Transporter Kinetics	Se Jun Ahn	thu, 20:10
PP1284	Antigenic drift and therapy resistance in CAR T-cell treatment: insights from a cellular automata model	Pablo Sanz Galarreta	thu, 20:10
PP1295	Fluctuation analysis of neural stem cell dynamics	Filip Klawe	thu, 20:10
PP1302	Mathematical Modelling of Plant Cell Polarity Induced by SOSEKI Proteins	Annika Brockhaus	thu, 20:10
PP1308	Adaptive Finite Element method for reconstructing dielectric properties of malign melanoma in 3D	Georg Kyhn	thu, 20:10
PP1310	Reaction-diffusion modelling of siderophore-mediated iron uptake and cross-feeding in bacteria communities.	Tosin Samuel Osikoya	thu, 20:10
PP1316	Turing Instability in a Reaction-Diffusion Predator-Prey Model with Prey Infection and Intraspecific Predator Competition	Mark Lois Fortin	thu, 20:10
PP1331	Topological Data Analysis for Unsupervised Feature Selection in Large Scale Spatial Omics Data Sets	James Boyle	thu, 20:10
PP1337	Dynamics and stability of microbial communities under community-level selection	Sofia Almirante	thu, 20:10
PP1346	A Time-Dependent Analysis of Protein Homeostasis Stability	Kaylie Green	thu, 20:10
PP1351	Risk-aware mechanistic framework for early detection of post-radiotherapy biochemical relapse in prostate cancer	Miguel Anxo Vicente Pardal	thu, 20:10
PP1358	Dynamical paths to depolarization block and back	Marisa Saggio	thu, 20:10
PP1371	Optimizing Dengue Vaccination Strategies in Brazil: The Role of Regional Immune Profiles and Age-Targeted Allocation	Youngsuk Ko	thu, 20:10
PP1387	Physics-Informed Neural Networks for Mathematical Models on Alzheimer's Disease Dynamics	Samira Breitling	thu, 20:10
PP1393	Mantis: A Foundation Model for Mechanistic Disease Forecasting	Carson Dudley	thu, 20:10

Code	Title	Speaker(s)	Date / time
PP1398	Quantifying End-of-Outbreak Probability of Ebola via Stochastic Models	Sumin Park	thu, 20:10
PP1402	Closed-Loop Control of Anesthesia with Propofol Using Reinforcement Learning on Clinical Data	Madlen Martinek	thu, 20:10
PP1408	Percolation theory and Voronoi geometry for modeling protein-ligand interactions	Miranda Lynch	thu, 20:10
PP1424	Reconstruction of Nosocomial Infectious Disease Outbreaks Using Bayesian Inference	Oskar Holmstedt	thu, 20:10
PP1426	Graph-Based Nonequilibrium Analysis of RNA Polymerase Transcription from Optical Tweezers Time Series	Diego Herraez Aguilar	thu, 20:10
PP1431	Quantifying the influence of inflammation on spiral artery dynamics through the expansion of a mechanistic endometrial model	Alexandria Johnson	thu, 20:10
PP1435	Real time modeling of the 2025 foot-and-mouth disease outbreak in the Hungary-Slovakia border region	Olivér Régaisz	thu, 20:10
PP1447	Clonal dynamics deviate from neutral drift in zebrafish spermatogenesis	Connor Shrader	thu, 20:10
PP1457	Real-time detection of aberrant physiological changes from wearable data using Kalman Filtering and Autoencoder	Yejoon Wang	thu, 20:10
PP1467	From Flickering to Dynamical States: A Data-Driven Analysis of Nonequilibrium Dynamics in Red Blood Cells	Irene Alférez Gómez	thu, 20:10
PP1471	Multiscale Analysis of Gut Dysbiosis Identifies Time-Adaptive Immune Augmentation as an Inflammatory Flare Suppression Strategy	Haya Mayoof	thu, 20:10
PP1473	Mechanistic Integration of Longitudinal RNASeq Data to Reveal Novel Biomarkers in Parkinson's Disease	Cyrille Lorenz-Dittmar	thu, 20:10
PP1476	Learning Population Dynamical Models in Systems Biology with Mixed-Effect Gaussian Process ODEs	Julien Martinelli	thu, 20:10
PP1487	Analysis of the Association Between COVID-19 Prevalence and Wastewater Viral Concentrations Using Survival Dynamical System	Sookyung An	thu, 20:10
PP1517	An initial stress-based Arruda-Boyce model for soft-solids and its FE implementation to study surface instabilities	Srikanth Krishnakumar	thu, 20:10
PP1520	Modelling Disease Progression through Manifold Approximation	Andreas Körner	thu, 20:10
PP1522	Non-Periodic Steady State in a Closed Ferroin-Catalyzed Belousov–Zhabotinsky Reaction induced by High Stirring Rates	Mihnea Hristea	thu, 20:10
PP1533	Social information under restricted observations	Andrew Bate	thu, 20:10
PP1535	Modelling scenarios to mitigate the potential spread of smallpox in Great Britain	Luke Webb	thu, 20:10
PP1548	Spatial and Stochastic Microtubule Dynamics Model in the Neuronal Dendrite	Meghan Kwon	thu, 20:10

Code	Title	Speaker(s)	Date / time
PP1569	Mathematical modeling of drug delivery by intra-vaginal rings, with application to trans-species ring designs	Bhavana Morankar	thu, 20:10
PP1574	Optimizing cell-cycle–targeted drug combinations for high-grade serous ovarian cancer: An integrated approach	Agata Xella	thu, 20:10
PP1588	Accurate noise models and their impact on parameter inference for mathematical models of collective cell behaviours	Marjorie Watts	thu, 20:10
PP1599	Cooperative motility emerges in crowds of T cells but not neutrophils	Inge Wortel	thu, 20:10
PP1615	Stationary and oscillatory corrosion patterns in extended Barkley models	Giancarlo Consolo	thu, 20:10
PP1627	The Curse of Black Sigatoka: A Backward Bifurcation Perspective	Bernard Asamoah Afful	thu, 20:10
PP1633	Inverse modelling of mechanochemically coupled tumour growth under paclitaxel treatment and hypoxic stress	Maryam Alka	thu, 20:10
PP1657	An individual-based model of Glioblastoma cell migration and PDGF-Driven recruitment	Gerda Suchodolskaite	thu, 20:10
PP1658	Integrating spatial statistics and agent-based modelling to study tertiary lymphoid structures in cancer	Nicholas Lai	thu, 20:10
PP1665	Message-passing graph neural network models to predict protein-protein interactions	Dan Mark Orapa	thu, 20:10
PP1666	Estimating the Distribution of Methylation Entropy in Genomic Data	Monika Kurpas	thu, 20:10
PP1691	Stem cell competition in the pre-neoplastic intestine under immunosurveillance	Birgit Tschiatschek	thu, 20:10
PP1692	Decoupling density- and adhesion-driven rigidity transitions in embryonic tissues	Elisa Floris	thu, 20:10
PP209	ON A MODIFIED LESLIE-GOWER MODEL WITH PREY DEFENSE AND PREDATOR CANNIBALISM	Gloria Botchway	thu, 20:10
PP252	SymScore: Machine Learning Accuracy Meets Transparency in a Symbolic Regression-Based Clinical Score Generator	Olive Cawiding	thu, 20:10
PP261	Catalyst: Fast and flexible modeling of reaction networks	Torkel Loman	thu, 20:10
PP275	Mathematical model of excitation and contraction in cardiomyocytes for simulation of in vitro systems for Heart Failure drug development	Jacob Bendsen	thu, 20:10
PP282	Optimal control approach to obesity reduction	Olga Vasilieva	thu, 20:10
PP287	Optimizing enzyme inhibition analysis: precise estimation with a single inhibition concentration	Hyeong Jun Jang	thu, 20:10
PP291	Epidemiological model under vaccination regime and non-permanent immunity driven by time-changed Lévy noise	Nenad Šuvak	thu, 20:10
PP296	The role of individual capillaries for the brain's oxygen supply and the consequences of their occlusion	Gaia Stievano	thu, 20:10
PP302	Modelling biochemically coupled viscoelastic systems: an open-source computational framework	Gordon McNicol	thu, 20:10

Code	Title	Speaker(s)	Date / time
PP303	Modelling pattern formation in a spatially discrete plant cell signalling system	Patience Aba Sakyi	thu, 20:10
PP308	AI-based multiomics profiling reveals complementary omics contributions to personalized prediction of cardiovascular disease	Qingpeng Zhang	thu, 20:10
PP309	Beyond Markovian Assumptions: A History-Dependent Framework for Epidemic Inference	Sunhwa Choi	thu, 20:10
PP314	Challenging the standard protocol: Mechanistic mathematical model for ^{177}Lu-PSMA therapy optimization	Silvia Bordel-Vozmediano	thu, 20:10
PP316	Multi-Scale Mathematical Modelling of Prostate Cancer Response to Iron-Supplemented Ferroptosis	Eisha Arif	thu, 20:10
PP339	Global Dynamics of Two-prey-one-predator Models with Direct Competition between the Prey:The Biological parts	MING CHIEH HUANG	thu, 20:10
PP340	Quantifying the Impact of Healthcare Accessibility on Non-COVID Excess Mortality During the COVID-19 Pandemic in South Korea	Soyoung Kim	thu, 20:10
PP343	Global Dynamics of Two-prey-one-predator Models with Direct Competition between the Prey : The Mathematical Parts	Tinghui Yang	thu, 20:10
PP369	Structural inference over reaction network spaces	Elijah Foo	thu, 20:10
PP373	Metabolic Bacterial Game	Oleksandr Cherednichenko	thu, 20:10
PP379	Parameter Optimization of a Thalamocortical Network Model for Simulation of EEG under Propofol	Olga Lazic	thu, 20:10
PP385	Decoding Cis-Regulatory Information Underlying Gene Expression in Forest Trees Using Field Transcriptomes from Natural Environments	Shuichi Kudo	thu, 20:10
PP390	Mathematical and Mouse Models Identify T Regulatory Cell Influx as A Key Determinant of Acquired Resistance to PD-1 Immunotherapy	Rachel Sousa	thu, 20:10
PP396	Bifurcation analysis of a distributed-delay Wilson–Cowan model for sleep spindle dynamics	Anca Stanoev	thu, 20:10

Chapter 2

Plenary Talks

PLT-01 — Mathematical modeling of spatial evolution with applications to biomedical systems

SPEAKERS Natalia L. Komarova (University of California, San Diego)
AUTHORS Natalia L. Komarova (University of California, San Diego)
SCHEDULE Monday, 13 July 2026; 09:20–10:10

Abstract

Evolutionary dynamics permeates life and life-like systems. Mathematical methods can be used to study evolutionary processes, such as selection, mutation, and drift, and to make sense of many phenomena in life sciences. Mass-action (or mean-field) evolutionary dynamics have been studied over the last 100 years, and produced an enormous wealth of useful results. In this talk, however, I will discuss how spatial interactions may change the laws of evolution, giving rise to a number of interesting and counterintuitive findings. I will discuss both explicitly spatial systems and metapopulations, and demonstrate a number of scaling laws that describe production and spread of disadvantageous, neutral, and advantageous mutants. Applications of these laws to bacterial growth and carcinogenesis will be discussed.

PLT-02 — Self-organisation in mouse development

SPEAKERS Takashi Hiiragi (Hubrecht Institute, Royal Netherlands Academy of Arts and Sciences (KNAW))
AUTHORS Takashi Hiiragi (Hubrecht Institute, Royal Netherlands Academy of Arts and Sciences (KNAW))
SCHEDULE Monday, 13 July 2026; 14:00–14:50

Abstract

Embryo patterning coordinates cell differentiation, tissue growth and morphogenesis. Understanding how precision is achieved despite the inherent developmental variabilities remains a challenge. Using early mammalian embryos as a model system, we aim to understand the design principle of multi-cellular organisms. Our studies show that feedbacks between cell fate, polarity and cell/tissue mechanics underlie the robustness in development. I will discuss our recent works.

PLT-03 — Impact of horizontal gene transfer on population evolutionary dynamics

SPEAKERS Sepideh Mirrahimi (CNRS, Institut de mathématiques de Toulouse)
AUTHORS Sepideh Mirrahimi (CNRS, Institut de mathématiques de Toulouse)
SCHEDULE Tuesday, 14 July 2026; 08:30–09:20

Abstract

The evolutionary adaptation of populations arises from the interplay of several fundamental biological processes, including heredity, natural selection, mutation, competition, and the mixing of gene pools through horizontal gene transfer or sexual reproduction. Horizontal gene transfer, defined as the exchange of genetic material between individuals outside vertical (parent-to-offspring) inheritance, plays a major role in the evolution and adaptation of many organisms. It is particularly well documented in the emergence of bacterial virulence and antibiotic resistance. However, despite its recognized importance, the impact of horizontal gene transfer on the phenotypic distribution and density of populations remains poorly understood. In this talk, I will present some integro-differential models from evolutionary biology. I will introduce an asymptotic approach designed to study such models in a regime of small mutational effects. By applying this method to a model that incorporates a type of horizontal gene transfer, I will show how that horizontal gene transfer may dramatically alter evolutionary dynamics, giving rise to novel adaptive trajectories as well as fundamentally different evolutionary outcomes.

PLT-04 — An intro to shape spaces - from differential geometry to the drosophila testis

SPEAKERS Benedikt Wirth (University of Münster)
AUTHORS Benedikt Wirth (University of Münster)
SCHEDULE Tuesday, 14 July 2026; 09:20–10:10

Abstract

Shape is a central principle in biology, linking form to function across scales. It determines ecological fit at organism level, assists physiological function at the organ level, and drives macroscopic form through growth and patterning at the (sub-)cellular level. Quantifying and comparing shape or morphology may therefore provide a powerful tool to understand phylo- and ontogenesis as well as biological organization in general. I will give an introduction into the mathematics of shape spaces and the theoretical and computational tools that allow to perform statistical and regression analyses of shapes, touching upon recent mathematical developments as well as applications.

PLT-05 — Behind the barrier: mathematical modelling of the neuroimmune system and multiple sclerosis

SPEAKERS Adrianne Jenner (Queensland University of Technology)
AUTHORS Adrianne Jenner (Queensland University of Technology)
SCHEDULE Thursday, 16 July 2026; 08:30–09:20

Abstract

The brain is protected by a complex and highly coordinated network of immune cells, regulated in part by the blood–brain barrier. When this tightly controlled system becomes dysregulated, the consequences can be profound, contributing to neurological disorders such as multiple sclerosis. Despite rapid advances in experimental neuroimmunology, fundamental questions remain unanswered: How do immune cells coordinate their activity within the brain? What mechanisms govern their response to infection or injury? And how do these interactions give rise to the fluctuating symptoms observed in chronic diseases such as multiple sclerosis? Our group is using mathematical modelling to interrogate the neuroimmune system across the many scales. Using everything from ODEs, to PDEs and even ABMs, we investigate how microglia — the brain's resident immune cells — respond to bacteria stimuli, how interactions between

immune populations may generate oscillatory disease dynamics in multiple sclerosis, and how mechanistic models can be leveraged to explore therapeutic strategies beyond current treatments. Mathematical neuroimmunology is an exciting frontier in mathematical biology. In this talk, I will share some of our early work that explores how mathematics may help us understand the neuroimmune system and its role in disease.

PLT-06 — A theory, just a theory, or not even a theory? Perspectives on the roles of mathematical modelling

SPEAKERS Caroline Colijn (Simon Fraser University)
AUTHORS Caroline Colijn (Simon Fraser University)
SCHEDULE Thursday, 16 July 2026; 09:20–10:10

Abstract

In this talk I'll take a birds-eye view of mathematical modelling. What is it? Is a model "a theory"? Is it "just a theory"? What can it accomplish, and how can it fail? Attitudes to modelling range from considering models to be hardly useful at all (especially without data or collaboration with end users), to considering them highly valuable in and of themselves, even capable of predicting (or forecasting) the future. These polarized views affect collaboration, public engagement, careers and funding outcomes over a range of fields in mathematical and theoretical biology. In this talk, I will outline the many roles modelling plays, including some lesser-known uses of modelling, rooted in experiences during the pandemic. I will also describe some weaknesses of modelling, as I see them, such that modelling can fail to be good science, and can even do harm. I will propose that the onus is at least partly on modellers to prevent this, and I will comment on how this might be done. I will conclude by inviting debate about how to interpret and assess modelling-based research.

PLT-07 — Estimating tipping points in climate

SPEAKERS Susanne Ditlevsen (University of Copenhagen)
AUTHORS Susanne Ditlevsen (University of Copenhagen)
SCHEDULE Friday, 17 July 2026; 08:30–09:20

Abstract

In recent years there has been an increasing awareness of the risks of collapse or tipping points in a wide variety of complex systems, ranging from human medical conditions, pandemics, ecosystems to climate, finance and society. Even in systems where governing equations are known, such as the atmospheric flow, predictability is limited by the chaotic nature of the system and by the limited resolution in observations and computer simulations. These phenomena are naturally modelled by strongly nonlinear stochastic processes, which permit a statistical description. In this talk I will present methods to model and analyze data from such complex systems, with application to an important tipping element in the climate, the Atlantic Meridional Overturning Circulation.

PLT-08 — The Geometry of Decision-Making

SPEAKERS Iain Couzin (Max Planck Institute of Animal Behavior Center for the Advanced Study of Collective Behaviour)
AUTHORS Iain Couzin (Max Planck Institute of Animal Behavior Center for the Advanced Study of Collective Behaviour)
SCHEDULE Friday, 17 July 2026; 14:00–14:50

Abstract

Running, swimming, or flying through the world, animals are constantly making decisions while on the move—decisions that allow them to choose where to eat, where to hide, and with whom to associate.

Despite this most studies have considered only on the outcome of, and time taken to make, decisions. Motion is, however, crucial in terms of how space is represented by organisms during spatial decision-making. Using a combination of modeling, automated tracking, computational reconstruction of sensory information, and immersive 'holographic' virtual reality (VR) experiments with ants, fruit flies, locusts, and zebrafish, I will demonstrate that this time-varying representation results in the emergence of new and fundamental geometric principles that considerably impact decision-making. Specifically, we find that the brain spontaneously reduces multi-choice decisions into a series of abrupt ('critical') binary decisions in space-time, a process that repeats until only one option—the one ultimately selected by the individual—remains. Due to the critical nature of these transitions (and the corresponding increase in 'susceptibility') even noisy brains are extremely sensitive to very small differences between remaining options (e.g., a very small difference in neuronal activity being in "favor" of one option) near these locations in space-time. This mechanism facilitates highly effective decision-making, and is shown to be robust both to the number of options available, and to context, such as whether options are static (e.g. refuges) or mobile (e.g. other animals). In addition, we find evidence that the same geometric principles of decision-making occur across scales of biological organization, from neural dynamics to animal collectives, suggesting they are fundamental features of spatiotemporal computation.

Chapter 3

Minisymposia

MS18 — PDE aspects of neural assemblies

TRACK Neuroscience and Neural Systems
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 15.12 - HS
ORGANISERS Benoit Perthame (Sorbonne Université); Delphine Salort (Sorbonne universite)

Minisymposium abstract

Mean-field limits of neural assemblies lead to nonlinear partial differential equations (PDEs) that have attracted considerable interest in recent years; see, for instance, the recent survey [CR25]. This interest stems from the fact that these equations raise several highly challenging mathematical questions within an unconventional formal framework, including the existence and uniqueness of solutions, finite-time blow-up, periodic solutions, and, more generally, long-time behavior and pattern formation. Beyond these theoretical issues, this important subject is also motivated by the investigation of qualitative properties of neural systems, including the emergence of rhythmic activity and synchronization, as well as by comparisons with experimental observations.

Four outstanding speakers have agreed to present their recent results: P. Roux (Lyon) [Ike+22], D. Salort (Paris) [PRS25], S. Solem (Oslo) [CHS22], Z. Zhou (Westlake) [DZ25]. They have contributed recently with several original contributions covering a wide range of subjects in the field of PDEs for neuroscience.

References

[CHS22] José A. Carrillo, Helge Holden, and Susanne Solem. “Noise-driven bifurcations in a neural field system modelling networks of grid cells”. In: *J. Math. Biol.* 85.4 (2022). Id/No 42, p. 30. ISSN: 0303-6812. DOI: 10.1007/s00285-022-01811-6.

[CR25] José A. Carrillo and Pierre Roux. “Nonlinear partial differential equations in neuroscience: from modeling to mathematical theory”. In: *Math. Models Methods Appl. Sci.* 35.2 (2025), pp. 403–584. ISSN: 0218-2025. DOI: 10.1142/S0218202525400044.

[DZ25] Xu’an Dou and Zhennan Zhou. “Dilating blow-up time: a generalized solution of the NNLIF neuron model and its global well-posedness”. In: *Commun. Math. Sci.* 23.4 (2025), pp. 1081–1138. ISSN: 1539-6746. DOI: 10.4310/CMS.250412104353.

[Ike+22] Kota Ikeda et al. “Theoretical study of the emergence of periodic solutions for the inhibitory NNLIF neuron model with synaptic delay”. In: *Math. Neurosci. Appl.* 2 (2022), Art. No. 4, 37.

[PRS25] Benoît Perthame, Clément Rieutord, and Delphine Salort. “Strongly nonlinear age-structured equation, time-elapsed model and large delays: B. Perthame et al.” In: *Journal of Mathematical Biology* 91.5 (2025), p. 65.

MS18-01 — Qualitative properties for certain PDEs derived from neuroscience

SPEAKERS Delphine Salort (Laboratoire Jacques Louis Lions)
AUTHORS Delphine Salort (Laboratoire Jacques Louis Lions)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

In recent years, the qualitative study of models derived from neuroscience has experienced significant progresses, particularly within the communities of probabilist and PDE specialists. The aim is to use certain models to understand the emergence of complex dynamics observed within interacting neural networks. In this regard, numerous PDE models have been proposed, presenting structures, and therefore mathematical difficulties, that vary greatly depending on the modeling choices made. We will begin with an overview of the main issues and existing results for PDE models derived from neuroscience that are now relatively well understood, in both linear and non-linear regimes. We will then focus on a particular framework where modeling involves equations with degenerate diffusion, for which, even in the simplest (linear) case, several questions remain open.

MS18-02 — Disentangling pulse-coupled oscillators in the mean-field regime through the pseudo-inverse in a dilated timescale

SPEAKERS Pierre Roux (Centrale Lyon)
AUTHORS Jose Antonio Carrillo (Mathematical Institute, University of Oxford); Pierre Roux (Centrale Lyon); Xu'an Dou (Beijing International Center for Mathematical Research, Peking University); Zhennan Zhou (Westlake University)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Systems of pulse-coupled oscillators model synchronization through singular interactions occurring at discrete times, when particles reach a specific firing phase. They have numerous applications in physics, biology and engineering, for example to cardiac cells, neurons and fireflies. They could also represent a stepping stone for the understanding of networks of voltage-conductance neurons at the mesoscopic scale. In the mean-field limit, the probability density in phase of the population of oscillators satisfies a singular continuity equation prone to finite-time blow-up, for which very few theoretical results are available. With José A. Carrillo, Xu'an Dou and Zhennan Zhou [Car+24], we have introduced a reformulation of the mean-field system based on the inverse distribution function seen in a dilated timescale. It allows to show a hidden contraction/expansion mechanism and to propose simple and rigorous proofs of the long-time behaviour, the existence of steady states, the rates of convergence and the occurence of finite time blow-up for a large class of monotone phase response functions.

References

[Car+24] José Antonio Carrillo et al. “Classical solutions of a mean field system for pulse-coupled oscillators: long time asymptotics versus blowup”. In: *arXiv preprint arXiv:2404.13703* (2024).

MS18-03 — Navigational-enabling settings of a PDE modelling noisy grid cell activity

SPEAKERS Suzanne Solem (Norwegian University of Life Sciences)
AUTHORS Suzanne Solem (Norwegian University of Life Sciences)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Grid cells, with their striking hexagonal firing patterns, are neurons which play a key role in the internal navigational system of mammals. This talk will concern a nonlocal Fokker–Planck-like PDE which emerged in a pursuit to better understand the effects of noise on grid cell activity. When this model produces hexagonal network activity which persists when translated in accordance with the mammal's movement in physical space, the model is in a setting which enables the mammal's ability to orientate itself. Through aspects concerning the existence and stability of particular solutions to the PDE, we explore for which parameter settings such activity could emerge.

Based on collaborations with: José Antonio Carrillo, Mathematical Institute, University of Oxford, Oxford OX2 6GG, UK Maren Brathen Kristoffersen, Department of Mathematics, Norwegian University of Life Sciences, 1433 As, Norway Pierre Roux, Institut Camille Jordan, Ecole Centrale de Lyon, 69134 Ecully, France

MS18-04 — Resolving the Blow-Up: A Time-Dilated Numerical Framework for Multiple Firing Events in Mean-Field Neuronal Networks

SPEAKERS Zhennan Zhou (Westlake university)
AUTHORS Louis Tao (Center for Bioinformatics and Center for Quantitative Biology, Peking University); Xu'an Dou (Beijing International Center for Mathematical Research); Zhe Xue (Institute for Theoretical Sciences, Westlake University); Zhennan Zhou (Westlake university); Zhennan Zhou (Institute for Theoretical Sciences, Westlake University)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

In large-scale excitatory neuronal networks, rapid synchronization manifests as multiple firing events (MFEs), mathematically characterized by a finite-time blow-up of the neuronal firing rate in the mean-field Fokker-Planck equation. Standard numerical methods struggle to resolve this singularity due to the divergent boundary flux and the instantaneous nature of the population voltage reset. In this work, we propose a robust multiscale numerical framework based on time dilation. By transforming the governing equation into a dilated timescale proportional to the firing activity, we desingularize the blow-up, effectively stretching the instantaneous synchronization event into a resolved mesoscopic process. This approach is shown to be physically consistent with the microscopic cascade mechanism underlying MFEs and the system's inherent fragility. To implement this numerically, we develop a hybrid scheme that utilizes a mesh-independent flux criterion to switch between timescales and a semi-analytical "moving Gaussian" method to accurately evolve the post-blowup Dirac mass. Numerical benchmarks demonstrate that our solver not only captures steady states with high accuracy but also efficiently reproduces periodic MFEs, matching Monte Carlo simulations without the severe time-step restrictions associated with particle cascades.

MS34 — Phenotypic Plasticity in Tumor Progression

TRACK Mathematical Oncology
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 62.01 - HS
ORGANISERS Francisco Lopes (Universidade Federal do Rio de Janeiro)

Minisymposium abstract

Phenotypic plasticity,the dynamic switching of cancer cells between phenotypes, is a fundamental, yet poorly quantified, driver of metastasis, therapy resistance, and relapse. A deep understanding of this process requires the integration of experimental data with advanced mathematical frameworks to move beyond description to prediction. This symposium brings together world-leading experts at this interface to present a cohesive view of plasticity across biological scales. Talks will span from the molecular circuitry of cell-fate decisions, patient-specific models of tumor evolution, to theoretical frameworks quantifying state transitions and network dynamics. By bridging mechanistic models, clinical data, and evolutionary theory, the session will highlight how mathematical and theoretical biology is uniquely positioned to uncover the principles governing plasticity. The goal is to foster discussions on inferring dynamical rules from data, predicting emergent tumor behavior, and ultimately informing novel therapeutic strategies that target the adaptable nature of cancer. This topic is of central importance to the ECMTB community, uniting theoreticians, computational biologists, and experimentalists.

MS34-03 — Dynamical modeling of phenotypic plasticity in melanoma reveals strategies of drug-induced adaptation

SPEAKERS Sarthak Sahoo (Indian Institute of Science)
AUTHORS Mohit Kumar Jolly (Indian Institute of Science); Sarthak Sahoo (Indian Institute of Science)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Phenotypic heterogeneity of melanoma cells contributes to drug tolerance, increased metastasis, and immune evasion in patients with progressive disease. However, the dynamics of the co-existence and interconversion among these different phenotypes remains unclear. Here, we integrate dynamical systems modeling with transcriptomic data analysis at bulk and single-cell levels to investigate underlying mechanisms behind phenotypic plasticity in melanoma and its impact on adaptation to different therapies. We construct a minimal core gene regulatory network involving the transcription factors implicated in this process and identify the multiple 'attractors' in phenotypic landscape enabled by this network. The emergent dynamics of this regulatory network comprising MITF, SOX10, SOX9, JUN and ZEB1 can recapitulate experimental observations about the co-existence of diverse phenotypes and reversible cell-state transitions among them, including in response to targeted therapy and immune checkpoint inhibitors. Our model predictions about changes in proliferative to invasive transition and PD-L1 levels as melanoma cells evade targeted therapy and immune checkpoint inhibitors were also validated in multiple RNA-seq data sets from in vitro and in vivo experiments. Our calibrated dynamical model offers a platform to test combinatorial therapies against phenotypic plasticity and provide rational avenues for treating melanoma.

MS34-04 — Critical assessment of Waddington's Epigenetic Landscape and its modern interpretations

SPEAKERS Eric Fanchon (CNRS - Université Grenoble Alpes - TIMC laboratory)
AUTHORS Eric Fanchon (CNRS - Université Grenoble Alpes - TIMC laboratory)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 62.01 - HS
TRACK Cellular and Developmental Biology

Abstract

Waddington's epigenetic landscape (EL) famously represents development as a ball rolling down a branching surface whose valleys correspond to cell fates, while the landscape itself is shaped by pegs and ropes representing gene interactions. Despite its enduring influence, which continues today in fields such as stem cell biology, aging and single-cell omics, the conceptual meaning of this metaphor remains ambiguous. In this work we propose a **systematic critical analysis** of both Waddington's original EL metaphor and its modern theoretical reinterpretations. At the metaphor level, a basic difficulty lies in the status of the "ball", which is supposed to represent the evolving cell, while the landscape is shaped by the "ropes" (gene products). This creates a visual and **conceptual separation** between the **cell** and the

regulatory system. At the theoretical level, several mathematical frameworks have been proposed to formalize the landscape idea, including geometric approaches based on dynamical systems and bifurcation theory [Cis+25], potential–flux decompositions of non-equilibrium dynamics [New15] and stochastic quasi-potentials derived from large-deviation theory [Wan15]. We analyze the relevance of their underlying assumptions and show that these frameworks differ in essential ways from Waddington's vision. Clarifying these conceptual differences helps assess the scope and limitations of the EL metaphor in contemporary theoretical biology

References

[Cis+25] Dillon J. Cislo et al. "Reconstructing Waddington's landscape from data". en. In: *Proceedings of the National Academy of Sciences* 122.49 (Dec. 2025), e2521762122. ISSN: 0027-8424, 1091-6490. DOI: 10.1073/pnas.2521762122. URL: https://pnas.org/doi/10.1073/pnas.2521762122 (visited on 05/10/2026).

[New15] Jay Newby. "Bistable switching asymptotics for the self regulating gene". In: *Journal of Physics A: Mathematical and Theoretical* 48.18 (May 2015), p. 185001. ISSN: 1751-8113, 1751-8121. DOI: 10.1088/1751-8113/48/18/185001. URL: https://iopscience.iop.org/article/10.1088/1751-8113/48/18/185001 (visited on 05/10/2026).

[Wan15] Jin Wang. "Landscape and flux theory of non-equilibrium dynamical systems with application to biology". en. In: *Advances in Physics* 64.1 (Jan. 2015), pp. 1–137. ISSN: 0001-8732, 1460-6976. DOI: 10.1080/00018732.2015.1037068. URL: http://www.tandfonline.com/doi/full/10.1080/00018732.2015.1037068 (visited on 05/10/2026).

MS34-02 — Analyzing the impact of proliferation and treatment parameters on low-grade glioma growth using mathematical models

SPEAKERS Maria Vela Pérez (Universidad Complutense de Madrid)
AUTHORS Marek Bodnar (University of Warsaw); Maria Vela Pérez (Universidad Complutense de Madrid)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Low-grade gliomas (LGGs) are slow-growing, infiltrative brain tumors for which complete surgical resection is often unfeasible, necessitating the use of adjunctive therapies. In this work [BVT25], we present and analyze a mathematical model for LGG growth and response to chemotherapy, validated against clinical patient data. We prove the existence and uniqueness of a global non-negative solution and rigorously analyze the stability of steady states, establishing conditions for global tumor-free equilibrium under both constant and periodic treatment regimens. Sensitivity analysis identifies the tumor proliferation rate as the key parameter influencing outcomes. Finally, numerical simulations are conducted to further assess the robustness and stability of the model fitting process.

References

[BVT25] M. Bodnar, M. Vela-Pérez, and A. Tryniecka-Maciążek. "Analyzing the impact of proliferation and treatment parameters on low-grade glioma growth using mathematical models". en. In: *Physica D: Nonlinear Phenomena* 472 (Feb. 2025), p. 134491. ISSN: 01672789. DOI: 10.1016/j.physd.2024.134491. URL: https://linkinghub.elsevier.com/retrieve/pii/S016727892400441X (visited on 04/08/2026).

MS34-01 — Uncovering underlying physical principles and driving forces of cancer from single-cell transcriptomics

SPEAKERS Jin Wang (Department of Chemistry, Stony Brook University)
AUTHORS Jin Wang (Department of Chemistry, Stony Brook University)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 62.01 - HS

TRACK Mathematical Oncology

Abstract

Cancer has been a serious disease for human health. Genetic mutations have often been thought to be mainly responsible for the cancer formation. More evidences have been accumulated that cancer emergence is not just caused by individual gene perturbation but also from the whole network or state of the system. This shift of thinking demands global quantification and physical understanding of the underlying mechanisms for cancer formation. Here, we develop cancer models from the underlying gene regulatory networks either based on the available low throughput or the recent high throughput sequence experimental studies [ZW24b; Zhu+24; ZW24a].

Cancer landscape can be quantified and cancer can be revealed as attractors representing cancer states. The landscape barrier and switching time become the measures of how difficult to transform from normal to cancer state. Furthermore, the cancer formation process can be quantified by the optimal paths between the normal state and cancer state. Due to the presence of the curl flux as the nonequilibrium driving force, the forward path and backward paths for cancer formation and normal state restoration are distinctly different. The global sensitivity analysis based on the landscape topography and nonequilibrium driving forces identify key genes and regulations responsible for the cancer formation. We further identify the nonequilibrium indicators through the curl flux, entropy production and time irreversibility as the early warning signals for the cancer formation. This helps to design practical strategy for cancer prevention and treatment.

References

[ZW24a] Ligang Zhu and Jin Wang. *Deciphering physical mechanism of cancer from single cell transcriptomics and lineage-tracing*. Submitted. 2024.

[ZW24b] Ligang Zhu and Jin Wang. "Quantifying Landscape-Flux via Single-Cell Transcriptomics Uncovers the Underlying Mechanism of Cell Cycle". In: *Advanced Science* 11 (2024), p. 2308879.

[Zhu+24] Ligang Zhu et al. "Uncovering underlying physical principles and driving forces of cell differentiation and reprogramming from single-cell transcriptomics". In: *Proceedings of the National Academy of Sciences of the United States of America* 121.34 (2024), e2401540121.

MS36 — Meta-Perspectives on Mathematical Biology

TRACK Education and Outreach
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 15.11 - HS
ORGANISERS Sara Hamis (Uppsala University, Sweden); Linnéa Gyllingberg (University of California, Los Angeles (UCLA), USA)

Minisymposium abstract

As the role of mathematical biology in society continues to expand, so too does the importance of reflecting on and understanding the field itself. In this minisymposium, we bring together meta-perspectives on mathematical biology that examine how the field interacts with both the scientific community and broader society. Our four speakers will address key interfaces of these interactions: policy-making [Ger+25], education [VisualPDE], scientific publishing [Pug+25], and inclusive science communication [SSE]. As such, this minisymposium will provide a forum in which to learn and discuss how mathematical biology is evolving in both scientific and societal contexts, and the ways in which we – as researchers, educators and science communicators – contribute to and shape this development.

References

[Ger+25] Philip Gerlee et al. "Handbook of mathematical modelling of infectious diseases for decision-making". In: (2025). URL: https://research.chalmers.se/en/publication/549809 (visited on 01/16/2026).

[Pug+25] Kira Pugh et al. “A bibliometric study on mathematical oncology: interdisciplinarity, internationality, collaboration and trending topics”. In: *Bulletin of Mathematical Biology* 87.12 (Dec. 2025), p. 174. DOI: 10.1007/s11538-025-01544-9. URL: https://link.springer.com/10.1007/s11538-025-01544-9.

[SSE] *Sensational SciFest Event*. URL: https://scientistsinc.org/sensational-scifest (visited on 01/16/2026).

[VisualPDE] *VisualPDE*. URL: https://visualpde.com/ (visited on 01/16/2026).

MS36-01 — Developing a handbook of mathematical modelling of infectious diseases for decision-making

SPEAKERS Philip Gerlee (Chalmers University of Technology, Sweden)
AUTHORS Anders Tegnell (Public Health Agency of Sweden, Stockholm, Sweden); Lisa Brouwers (Public Health Agency of Sweden, Stockholm, Sweden); Oscar Björnham (wedish Defence Research Agency, Umeå Sweden); Philip Gerlee (Chalmers University of Technology, Sweden); Torbjörn Lundh (Mathematical Sciences, Chalmers University of Technology and University of Gothenburg, Gothenburg, Sweden)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 15.11 - HS
TRACK Education and Outreach

Abstract

Decision-making during public health crises, such as pandemics or epidemics, often relies on evidence from mathematical models produced by public health authorities. In such crises, e.g. the recent COVID-19 pandemic, government and public health authorities in many countries were assisted by academic researchers. However, the practices of modelling for decision-making are different from that of academia, and this clash of cultures hampered the contributions made by academic modellers. In order to improve on this situation and increase preparedness for future crises we have composed a document titled “Handbook of mathematical modelling of infectious diseases for decision-making”. The handbook describes the process of modelling infectious diseases in a chronological order going from preparatory work, implementation, to communication of model results and lastly management of models. The target audience are academic modellers and junior public health officials, and we believe that this handbook has the potential to strengthen collaboration between government authorities and academia, ultimately leading to improved preparedness.

References

[Ger+21] Philip Gerlee et al. “Predicting regional COVID-19 hospital admissions in Sweden using mobility data”. In: *Scientific reports* 11.1 (2021), p. 24171.

[Ger+24] Philip Gerlee et al. “Evaluation and communication of pandemic scenarios”. In: *The Lancet Digital Health* 6.8 (2024), e543–e544.

[Ger+25] Philip Gerlee et al. *Handbook of Mathematical Modelling of Infectious Diseases for Decision-Making*. Report. Accessed: 2026-03-03. Chalmers University of Technology; Public Health Agency of Sweden; Swedish Defence Research Agency; Swedish Armed Forces, 2025. URL: https://research.chalmers.se/publication/549809.

[Had+25] Liza Hadley et al. “How does policy modelling work in practice? A global analysis on the use of epidemiological modelling in health crises”. In: *PLOS global public health* 5.6 (2025), e0004675.

MS36-02 — Interactive education and interdisciplinary communication via VisualPDE

SPEAKERS Benjamin Walker (University College London, UK)
AUTHORS Adam K. Townsend; Andrew L. Krause; Benjamin Walker (University College London, UK)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 15.11 - HS
TRACK Education and Outreach

Abstract

Partial differential equations (PDEs) underpin mathematical models across science and engineering, yet their complexity presents a significant barrier to students and researchers outside mathematics. In this talk, I will present VisualPDE (a free, open-source, browser-based platform for real-time interactive simulation of PDE systems) and discuss its role as both a pedagogical tool and a medium for interdisciplinary communication.

VisualPDE requires no installation or coding knowledge and runs instantly in any web browser. Simulations can be shared via a URL, making them trivially embeddable in lecture notes, papers, and outreach materials. This immediacy transforms the teaching of abstract concepts, rather than presenting bifurcations and instabilities as static equations, students can explore them by direct manipulation, such as by tuning parameters, painting initial conditions, and probing boundary effects in real time.

Beyond the classroom, VisualPDE serves as a communication bridge between mathematical modellers and domain scientists. Shareable, interactive simulations allow collaborators in biology, ecology, and beyond to build genuine intuition about model behaviour without engaging directly with the underlying mathematics. In an era of open science, we hope that interactive simulations like these can be first-class outputs of mathematical research designed to broaden impact and deepen understanding.

MS36-03 — Using bibliometrics to analyse trends in mathematical biology

SPEAKERS Kira Pugh (Uppsala University, Sweden)
AUTHORS Kira Pugh (Uppsala University, Sweden)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 15.11 - HS
TRACK Education and Outreach

Abstract

Quantitatively understanding a research field is important for funding allocation, teaching and curriculum development, research organisation, and scientific communication. One way to achieve this understanding is through bibliometric analysis, which involves using statistical methods to examine patterns in scientific publication metadata and citation networks. We present two case studies that use bibliometric analysis. First, we obtain a comprehensive overview of mathematical oncology as a research field. Our analysis shows that since the 1960s, mathematical oncology has become more globally connected, and more interactive with external disciplines, with larger research teams and evolving research topics [Pug+25]. Moreover, our results quantitatively demonstrate that mathematical oncology benefits both mathematics and the life sciences. Second, we investigate the cross-disciplinary exchange in biological agent-based modelling. Our findings suggest limited cross-talk between disciplines, with citations largely self-contained. We discuss the potential advantages and disadvantages of research fields being isolated versus those that are more interconnected.

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MS36-04 — A Sensational Approach to Inclusive STEM outreach

SPEAKERS Parmvir Kaur Bahia (Artha Science Media)
AUTHORS Parmvir Kaur Bahia (Artha Science Media)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 15.11 - HS
TRACK Education and Outreach

Abstract

“Science is not finished until it is communicated”. This quote attributed to Sir Mark Walport, the UKs Chief Scientist in 2013, has become a rallying cry for the science communication (SciComm) community and those involved in informal STEM education, to encourage researchers to communicate their science beyond

the academe. While there are no precise numbers, it is clear that more scientists, physicians, journalists and educators are choosing to get involved in engaging non-expert audiences with STEM subjects. This is reflected in the increasing number of communities dedicated to supporting SciComm, greater avenues for professional development, presence on social media platforms, and even in the requirements for funding bodies such as the NSF, to discuss the "Broader Impacts" of research beyond scientific advancement. Science outreach connects research to communities, inspires future scientists, and strengthens public trust in evidence-based decision-making. However, outreach that overlooks inclusion, limits who benefits and who participates in STEM. This talk uses the example of a sensory-friendly science festival to argue that inclusive outreach — designed to reach diverse learners — is essential for equitable opportunity, improved innovation, and more resilient scientific systems.

MS38 — Multiscale Modeling of Stochastic Reaction–Diffusion Systems in Biology

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 02.01 - HS
ORGANISERS Stefanie Winkelmann (Zuse Institute Berlin)

Minisymposium abstract

Stochastic reaction–diffusion systems provide a fundamental framework for modeling spatially structured biochemical processes such as gene regulation, neurotransmission, and protein clustering. This minisymposium focuses on efficient modeling and simulation approaches that bridge interacting particle-based descriptions and coarse-grained models, including compartment-based reaction–diffusion master equations and stochastic or deterministic partial differential equations. Topics cover coarse-graining limits, multiscale and hybrid methods, accurate numerical schemes, and biologically motivated applications in realistic cellular geometries.

MS38-01 — Field theories and quantum methods for multiscale complex systems

SPEAKERS Mauricio del Razo Sarmina (Zuse Institute Berlin)
AUTHORS Mauricio del Razo Sarmina (Zuse Institute Berlin)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Many real-world complex systems — from biochemical networks and ecological dynamics to epidemics and socioeconomic models — share a common mathematical structure: agents or particles that move, interact, and change in number. Stochastic reaction-diffusion processes provide a natural and unifying framework to model this broad class of systems, yet their standard probabilistic formulations become unwieldy due to combinatorial complexity arising from nonlinear interactions and varying particle numbers. In this talk, I will show how quantum-inspired methods — second quantization, Fock space, creation and annihilation operators, and path integrals — offer an elegant and powerful alternative. Originally developed for quantum field theory, these tools can be brought into the classical domain of stochastic systems, where they handle combinatorial bookkeeping automatically and enable systematic derivations of emergent behavior at multiple scales. I will present a unifying field theory representation that encompasses previous formulations (Doi, Doi-Peliti, RDME) as special cases, and demonstrate how it yields consistent multiscale numerical schemes and parameter relations across scales. Crucially, while the framework is grounded in reaction-diffusion systems, its structure is sufficiently general to extend to a wide range of complex systems — including population dynamics, neuroscience, and social systems — wherever agents interact and change in number, opening new avenues for multiscale modeling across disciplines.

MS38-02 — Multi-resolution simulations of intracellular processes

SPEAKERS Radek Erban (Oxford University)
AUTHORS Radek Erban (Oxford University)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

All-atom and coarse-grained molecular dynamics [ET23], Langevin dynamics [EV24], Brownian dynamics [ZE26; EC20], and compartment-based stochastic reaction–diffusion models [EC20; EW25] are computational methodologies that have been applied to the spatio-temporal modelling of numerous intracellular processes. They differ in the level of biological detail they can capture and in the computational intensity of the resulting simulations. In some cases, it is possible to establish rigorous connections between these modelling frameworks by deriving a macroscopic (coarse-grained) description from the detailed one. I will discuss these connections, with a focus on the development, analysis, and applications of multi-resolution methods, which combine detailed (high-resolution) simulations in localized regions of particular interest (where accuracy and microscopic detail are crucial) with a coarser stochastic model in other regions where accuracy may be traded for computational efficiency. I will illustrate applications of multi-resolution methodologies to modelling intracellular calcium dynamics, actin dynamics, and DNA dynamics.

References

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[ZE26] J. Zhang and R. Erban. "From Brownian dynamics to Poisson-Nernst-Planck equations: multi-resolution simulations of ions". In: *arXiv preprint arXiv:2506.19738* (2026).

MS38-03 — Adding Drift to Stochastic Reaction-Diffusion Processes

SPEAKERS Samuel Isaacson (Boston University)
AUTHORS Samuel Isaacson (Boston University)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Particle-based stochastic reaction-diffusion processes are widely used in modeling biological processes. There is now a well-developed set of results detailing their basic properties, how they rigorously relate to macroscopic reaction-diffusion PDE models, and a variety of numerical simulation methods for their accurate and efficient approximation. In contrast, adding drift to these models introduces a number of open questions. In this talk, I will describe our recent work on particle-based stochastic reaction-drift-diffusion models in which drift arises from one- and two-body interactions. I will first introduce the physical formulation of these models, illustrating how satisfying detailed balance of reaction fluxes at equilibrium constrains reactive interaction functions for reversible reactions. I will then summarize our work proving the rigorous mean-field large-population limit of such particle models, and outline which types of reaction-drift-diffusion PDEs arise in this limit.

MS38-04 — Coarse-Grained Modeling of Particle Clustering Dynamics

SPEAKERS Stefanie Winkelmann (Zuse Institute Berlin)
AUTHORS Stefanie Winkelmann (Zuse Institute Berlin)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Particle-based models with pairwise interactions provide a natural framework to describe clustering phenomena in biological systems, but their simulation and analysis become computationally demanding at large scales. In this talk, I will present two complementary approaches for efficient coarse-grained modeling of particle clustering dynamics. First, I will show that stochastic partial differential equations (SPDEs) can accurately reproduce spatiotemporal clustering behavior, including cluster formation and merging, beyond the reach of deterministic mean-field models [Weh+25]. Second, I will introduce a data-driven reduction of transfer operators, yielding low-dimensional Markov models that capture metastable cluster configurations and transition pathways [Weh+26]. Together, these approaches provide interpretable and computationally efficient descriptions of clustering dynamics, enabling the analysis of large systems and long time scales.

References

[Weh+25] Nathalie Wehlitz et al. “Approximating particle-based clustering dynamics by stochastic PDEs”. In: *SIAM Journal on Applied Dynamical Systems* 24.2 (2025), pp. 1231–1250.

[Weh+26] Nathalie Wehlitz et al. “Data-driven Reduction of Transfer Operators for Particle Clustering Dynamics”. In: *Communications in Applied Mathematics and Computational Science* (2026). Accepted for publication. Available at arXiv:2601.02932.

MS40 — Mathematical models of vector-borne diseases

TRACK Mathematical Epidemiology
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 15.03 - HS
ORGANISERS Roberto Kraenkel (UNESP); claudia ferreira (UNESP)

Minisymposium abstract

This mini-symposium explores the mathematical modeling of vector-borne diseases and their connection to potential epidemic drivers. Vector-borne diseases pose significant challenges to modeling approaches, as the ecological effects of the vector—intertwined with climatic drivers—play critical roles. In the case of dengue fever, which is caused by four different serotypes with incomplete knowledge of cross-immunity effects, complex immunological dynamics also plays a significant role.

The talks will center on the control of epidemic dynamics using techniques from dynamical systems theory and machine learning. We will discuss the following topics: dose-dependent transmission models in general and as applied to dengue fever epidemics; multi-serotype immune-epidemiological models for dengue and serotype invasion; machine learning techniques to study the main drivers of dengue epidemics and their relations to vector population dynamics; and models for vector-borne diseases with behavioral/awareness effects and the control of epidemic onset. Mathematical methods are derived mainly from the theory of dynamical systems and involve both analytical and computational approaches.

MS40-01 — A Stage-Structured Seasonal Model for the Spread of Flavescence Dorée in Vineyards

SPEAKERS Attila Denes (University of Szeged)
AUTHORS Attila Denes (University of Szeged); Nigussie Abeye Shifera (Bolyai Institute, University of Szege); Ábel Garab (Bolyai Institute, University of Szeged)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Flavescence dorée is one of the most severe phytoplasma diseases affecting grapevines, posing an increasing threat to vineyards worldwide. The bacterial agent causing the disease is spread by the American grapevine leafhopper. We present a compartmental model for the spread of flavescence dorée incorporating stage structure in the vectors. To account for seasonal weather variations and the behaviour of the vectors, we consider periodic transmission, birth and death rates. We calculate the basic reproduction number as the spectral radius of a linear operator and show that it serves as a threshold parameter for disease persistence. Finally, we provide numerical simulations to assess the effect of varying key model parameters.

MS40-02 — On Multiscale Modeling of Vector-Borne Diseases

SPEAKERS Jorge Velasco (UNAM)
AUTHORS Jorge Velasco (UNAM)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

The transmission of infectious diseases involves complex interactions across multiple biological scales, from within-host immunological processes to between-host transmission dynamics. We develop a multiscale epidemic model linking host–vector population-level transmission dynamics to within-host and within-vector pathogen dynamics. Our model captures key features of within-vector viral progression and allows bidirectional coupling between within-host and between-host processes. Our results underscore the importance of carefully selecting coupling functions and provide guidance on when multiscale modeling is essential for understanding and managing vector-borne diseases.

MS40-03 — Underlying Complexity in Dengue Reinfection Dynamics: Backward Bifurcation and Serotype Invasion in a Multi-Serotype Model

SPEAKERS Mostafa Adimy (INRIA)
AUTHORS Charlotte Dugourd-Camus (INRIA, Centre Lyon - France); Mostafa Adimy (INRIA); Ruben Taieb (INRIA, Centre Lyon - France)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Reinfection plays a central role in dengue dynamics, as secondary infection with a different serotype may lead to severe forms such as Dengue Hemorrhagic Fever (DHF) through Antibody-Dependent Enhancement (ADE). We propose a multi-scale nested immuno-epidemiological model coupling within-host immune dynamics and between-host transmission. The model accounts for primary and secondary infections, capturing the interplay between immunity and epidemiological spread [ADT26]. We show the occurrence of backward bifurcation, implying that the basic reproduction number alone does not ensure disease eradication. We also derive an invasion reproduction number characterizing the ability of a new serotype to invade a population where another is endemic. These results highlight the underlying complexity of dengue reinfection dynamics and the role of ADE in shaping transmission and disease severity.

References

[ADT26] M. Adimy, C. Dugourd-Camus, and R. Taieb. “Multi-serotype nested immuno-epidemiological model for dengue hemorrhagic fever involving backward bifurcation and serotype invasion”. In: *Discrete and Continuous Dynamical Systems - B* (2026). DOI: 10.3934/dcdsb.2026028.

MS40-04 — Modelling West Nile spread in a habitat heterogeneous in space and time

SPEAKERS Andrea Pugliese (University of Trento)
AUTHORS Andrea Pugliese (University of Trento); Cinzia Soresina (University of Trento, Italy); Luca Gerardo Giorda (Johannes Kepler University Linz); Michela Sabbatino (University of Trento); Giovanni Marini (Fondazione Edmund Mach)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

The epidemic dynamics of West Nile Virus in Northern Italy (where it has been endemic since 2012) has been analysed in several studies, from modelling studies elucidating the main factors allowing for its persistence [Mar+20; De +25], to genetic and epidemiological analysis identifying the most likely transmission routes [Men+23; Gob+25]. Here, we extend the existing modelling studies to include spatial spread over the whole of Northern Italy. The model considers spatial and temporal heterogeneity of mosquito population dynamics, due especially to temperature-dependent demographic rates. By a qualitative comparison with available surveillance data, we discuss whether spatial spread can better be explained by constant short-range diffusion or longer-range movements at migration times.

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MS55 — Past, Present, and Future of Reaction Networks Theory

TRACK Systems Biology and Biochemical Networks
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 11.02 - HS, 15.04 - HS
ORGANISERS Gheorghe Craciun (University of Wisconsin-Madison); Hye-Won Kang (University of Maryland, Baltimore County)

Minisymposium abstract

Abstract: This minisymposium, part of the Reaction Network subgroup, brings together leading experts to provide an accessible overview of the past, present, and future of chemical reaction network theory for a general SMB/ECMTB audience. Since its origins in the early 1970s, the field has aimed to understand chemical and biochemical systems through their network structure and species abundances. Foundational work has explored qualitative behavior, stability, mass-action and non-mass-action reaction kinetics, and network reduction using tools from dynamical systems, algebraic and graphical methods, and stochastic modeling. The session will highlight ongoing research and emerging directions, fostering discussion and collaboration among participants. Topics include mathematical and dynamical analysis of reaction networks. Modeling approaches will cover deterministic, stochastic, multiscale, and spatial frameworks. The session will also address network structure and reduction techniques. Dynamical phenomena such as oscillations, bifurcations, multistationarity, and quasi-steady-state behavior will be discussed. Applications in computational biology, population genetics, and microbial ecology will also be introduced.

MS55-05 — An Invitation to Reaction Networks

SPEAKERS Gheorghe Craciun (University of Wisconsin-Madison)
AUTHORS Gheorghe Craciun (University of Wisconsin-Madison)
SCHEDULE Monday, 13 July 2026; 10:40–11:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

This is an introductory talk, and does not assume that you have any previous knowledge about the theory of reaction networks.

We will introduce and discuss reaction networks and reaction systems, especially as they are used in Mathematical Biology.

We will also emphasize the history of the development of key results and ideas about reaction systems, starting with ideas from thermodynamics and the Boltzmann equation from the 19th century, and followed by steady progress which culminates in 1972 with three key achievements: the stability of vertex balanced systems by Horn and Jackson, the deficiency zero theorem of Horn and Feinberg, and the connection between deterministic and stochastic models by Thomas Kurtz.

Among the more recent developments we will mention results on existence and uniqueness of positive steady states, as well as persistence (i.e., non-extinction), and global stability (i.e., the existence of globally attracting states).

Many examples will illustrate these ideas and results.

MS55-04 — From Continuous-Time Markov Chains to Spatial Stochastic Simulation of Biochemical Reaction Networks

SPEAKERS Hye-Won Kang (University of Maryland, Baltimore County)
AUTHORS Hye-Won Kang (University of Maryland, Baltimore County)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In this talk, I will introduce stochastic models for biochemical reaction networks based on continuous-time Markov chains. These models can be formulated using Kurtz's random time change representation and simulated exactly using Monte Carlo algorithms, such as Gillespie's stochastic simulation algorithm. Through several biological examples, we will explore how stochasticity gives rise to important phenomena, including stochastic extinction and random switching between distinct states. I will then extend these ideas to spatial biochemical reaction networks. We will discuss stochastic modeling and simulation approaches that incorporate spatial heterogeneity, including compartment-based models and spatial Gillespie algorithms. Finally, I will introduce hybrid stochastic methods that couple different modeling regimes to efficiently simulate multiscale biological systems.

MS55-03 — Stochastic Reaction Networks: Definition, Classical Scaling and Long-Term Behavior

SPEAKERS Daniele Cappelletti (Politecnico di Torino)
AUTHORS Daniele Cappelletti (Politecnico di Torino)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Stochastic reaction networks, modelled as continuous-time Markov chains, provide a principled framework for capturing the inherent randomness in biochemical and population systems. In this overview, I will introduce them and present the classical scaling limit, which establishes convergence to the deterministic

reaction network model on compact time intervals as the system size grows. A natural question then arises: does this approximation persist over long-time horizons? The study of limit distributions is motivated both by the analysis of biological models over long-time intervals and by multiscale models, where fast subsystems are approximated by their stationary regime. I will discuss notable connections and discrepancies between the stochastic and deterministic long-term dynamics, highlighting the case of complex-balanced models. Time permitting, I will close with some related open problems.

MS55-06 — Multi-scale Models Arising in Chemical Reaction Networks

SPEAKERS Lea Popovic (Concordia University)
AUTHORS Lea Popovic (Concordia University)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 15.04 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Biochemical reaction networks are characterized by varying orders of abundance for different species types, as well as varying orders of magnitude for different reactions. This feature allows one to reduce a complex model to a simpler one without losing the salient features of the dynamics. The model reduction can also lead to novel models that incorporate both stochastic and deterministic dynamics. I will present some examples of both finite-dimensional and infinite-dimensional (spatial) systems where new processes arise in the multi-scale limit of reaction networks.

MS55-07 — Making Sense of Reaction Networks via Atoms and Inheritance

SPEAKERS Murad Banaji (Lancaster University)
AUTHORS Murad Banaji (Lancaster University)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 15.04 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Let's imagine "CRN space", the set of all finite chemical reaction networks (CRNs), which can be visualised as an infinite set of digraphs. We can stratify CRN space in various ways: by molecularity, by number of species or reactions, by rank, by various equivalences, and so forth. Much of CRN theory consists of theorems linking network structure and network dynamics. Many classical and modern results tell us that, under some kinetic assumptions, some dynamical behaviour is permitted or forbidden by a given set of networks. In this sense, CRN theory provides tools which bring order to CRN space. The theory of "inheritance" provides another powerful tool for this purpose: it tells us how dynamics *propagates through* CRN space. Its results provide partial orders on CRN space, with $A \leq B$ implying that some behaviour of network A must also occur in network B. The theory is most useful when combined with results on "atoms" - minimal networks with a given behaviour. Each atom immediately defines an upper set of networks which must have this behaviour and a lower set of networks which forbid this behaviour. I'll describe some elements of this story, and what I see as the most important missing pieces.

This talk is based on joint work with Balázs Boros (University of Szeged), Josef Hofbauer (University of Vienna), and Casian Pantea (West Virginia University).

References

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MS55-01 — Bifurcations of Equilibria in Mass-action Systems

SPEAKERS Balázs Boros (University of Szeged)
AUTHORS Balázs Boros (University of Szeged)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 15.04 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Multistability and oscillations are ubiquitous in nature, appearing in contexts ranging from biochemical reaction networks and cellular regulation to ecological and chemical processes. These phenomena are often associated with qualitative changes in system dynamics as parameters vary, making bifurcation analysis a fundamental tool for understanding the mechanisms that generate such behaviors.

In this talk, we give an overview of the recent developments in the systematic study of local bifurcations of equilibria of small mass-action systems, including fold, cusp, Andronov-Hopf, Bautin, and Bogdanov-Takens bifurcations. The intensive study of nontrivial dynamical properties in small reaction networks is justified and motivated by recent advances in inheritance theory, a mathematical tool that allows us to lift nondegenerate behaviors from smaller networks to larger, more realistic ones.

Joint work with Murad Banaji (Lancaster University, United Kingdom) and Josef Hofbauer (University of Vienna, Austria).

MS55-02 — QSDs and Reaction Networks

SPEAKERS Carsten Wiuf (University of Copenhagen)
AUTHORS Carsten Wiuf (University of Copenhagen)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 15.04 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

This talk is concerned with quasi-stationary distributions (QSDs) of continuous-time Markov chains (CTMCs) with extinction in a setting that embraces reaction networks. QSDs describe the long-term behavior of such stochastic systems conditioned not to go extinct.

QSDs have a long history in probability theory, and this talk draws from this rich history. The focus is on CTMCs on the non-negative integers with a local jump structure. This local jump structure enforces (among other things) a recursive characterization of QSDs, the existence of an extremal QSD, and the

existence of a sequence of Karlin-McGregor-type polynomials of increasing degree that characterizes Kingman's parameter. The local jump structure is naturally implied by the structure of reaction networks. Some of these results and their importance will be explained in a (presumably) non-technical way.

Furthermore, the results will be illustrated by examples from reaction network theory, and the intuition of the audience will be tested, and why our intuition might fail, will be discussed.

MS61 — Mathematical Modelling for Alzheimer's Disease

TRACK Cutting Edge Research Areas
SCHEDULE Monday, 13 July 2026; 10:40-12:00; 01.15 - HS
ORGANISERS Germana Landi (University of Bologna); Lucilla Corrias (Université Evry Paris-Saclay); Maria Carla Tesi (University of Bologna)

Minisymposium abstract

Mathematical modelling is an important tool for understanding a complex pathology such as Alzheimers disease (AD). Quantitative models enable to formalize hypotheses about key factors such as the dynamics of proteins ($A\beta$ and τ) and inflammation, providing insights difficult to obtain through experiments alone. To this purpose many different and possibly complementary approaches can be adopted. The aim of this proposal is to favour interaction between researchers using different mathematical and computational tools to study similar issues. To model the spread of proteins, a very convenient mathematical setting is given by networks, in particular by connectomes, which are weighted graphs constructed from clinical data [Ber+23; BG25]. By integrating data from neuroimaging, computational models can help elucidate disease progression patterns across brain networks [Lan+26]. Mechanistic models also support the identification of critical transition points in the preclinical phase of AD, improving early detection strategies [Far+23]. Interesting results have also been obtained with analytical methods from semigroups theory and homogenisation [FL24]. Overall, mathematical modelling provides a rigorous framework to integrate multi-scale AD data, generate testable predictions, and ultimately accelerate the development of effective clinical, possibly personalized strategies. They therefore represent a major challenge nowadays in a cutting-edge research area.

References

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MS61-01 — Deciphering Alzheimer's Disease Dynamics: Modelling Protein Pathology, Inflammation, and Therapeutic Strategies

SPEAKERS Georgia Brennan (Oxford–GSK Institute of Molecular and Computational Medicine, University of Oxford; Mathematical Institute, University of Oxford)

AUTHORS Alain Goriely (Mathematical Institute, University of Oxford); Anna Sher (GlaxoSmithKline, USA); David Gavaghan (Department of Computer Science, University of Oxford); Georgia Brennan (Oxford–GSK Institute of Molecular and Computational Medicine, University of Oxford; Mathematical Institute, University of Oxford); Marissa Renardy (GlaxoSmithKline, USA)

SCHEDULE Monday, 13 July 2026; 10:40–11:00; 01.15 - HS

TRACK Cutting Edge Research Areas

Abstract

By 2030, an estimated 78 million people will be living with Alzheimer's disease (AD). Despite decades of research, clinical trials continue to face failure rates exceeding 95%, highlighting the need for improved mechanistic understanding of disease progression and therapeutic response. Mathematical modelling provides a framework to integrate biological processes across scales and enable safe, cost-effective in silico experimentation.

AD is characterised by the aggregation of proteins into toxic species that propagate through the brain in a prion-like manner. These processes interact with biological pathways, including clearance mechanisms and neuroinflammatory responses, ultimately driving neurodegeneration.

In this talk, I present a mathematical framework for the spatiotemporal dynamics of AD that integrates protein aggregation, network-mediated transport, clearance, and therapeutic interventions. Reaction–diffusion models on human brain networks describe the propagation of protein pathology together with clearance and inflammation. Complementing this approach, models of aggregation are formulated as spatially extended nucleation–elongation–fragmentation systems. These models are integrated with quantitative systems pharmacology frameworks describing drug–target interactions and biomarker responses, allowing therapeutic interventions to be incorporated directly into disease dynamics. Calibrated to human and clinical data, this multiscale framework can reveal mechanistic links between clearance, protein propagation, and neurodegeneration. Analytical and computational results aim to provide a quantitative basis for identifying mechanisms whose modulation may slow AD progression.

MS61-02 — Spreading of pathological proteins through brain networks

SPEAKERS Samira Breitling (University of Bologna)

AUTHORS Arianna Scaravelli (University of Bologna); Claudia Testa (University of Bologna); Germana Landi (University of Bologna); Maria Carla Tesi (University of Bologna); Samira Breitling (University of Bologna)

SCHEDULE Monday, 13 July 2026; 11:00–11:20; 01.15 - HS

TRACK Cutting Edge Research Areas

Abstract

Mathematical models can be used to verify medical hypotheses and quantify the mechanisms of the progression of neurological pathologies like Alzheimer's disease. In this work, we are interested in elucidating the spread of misfolded tau protein, a critical hallmark of Alzheimer's disease, alongside amyloid β protein, while taking the synergistic interaction between the two proteins into account [Bia+24]. We analyze a model consisting of a set of ordinary differential equations defined on brain networks derived from human connectomes, where brain regions are connected by edges representing fiber tracts. In particular, we consider several modeling choices, all employing network frameworks for protein evolution, differentiated by the network architecture and diffusion operators employed. By carefully comparing the

model results against clinical tau concentration data [Pet+10], gathered through advanced multimodal analysis techniques, we can identify values for the parameters of the mathematical model such that it can reproduce the disease progression. Moreover, we show that certain models better replicate the protein's dynamics and that the mathematical setting must be chosen with great care if the comparison with clinical data is considered decisive [Lan+26].

References

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MS61-03 — A mathematical model for tau aggregation and diffusion: An approach via two-scale homogenization of the Smoluchowski equation with transmission boundary conditions

SPEAKERS Silvia Lorenzani (Politecnico di Milano)
AUTHORS Bruno Franchi (University of Bologna); Silvia Lorenzani (Politecnico di Milano)
SCHEDULE Monday, 13 July 2026; 11:20-11:40; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Pathological accumulations of hyperphosphorylated tau protein aggregates, known as neurofibrillary tangles, are detected in several neurodegenerative tauopathies, including Alzheimer's disease [GS17]. Tau is a highly soluble, natively unfolded protein which is predominantly located in the axons of neurons of the central nervous system. Here, its physiological function is to support assembly and stabilization of axonal microtubules. Under pathological conditions, tau can assume abnormal conformations. In particular, hyperphosphorylation has a negative impact on the biological function of tau proteins, since it inhibits the binding to microtubules, compromising their stabilization and axonal transport, and promotes self-aggregation. Recent evidences have demonstrated that the progression of tau pathology reflects cell-to-cell propagation of the disease, achieved through the release of tau into the extracellular space and the uptake by surrounding healthy neurons [Yam17]. In this work, we prove a two-scale homogenization result for a set of diffusion-coagulation Smoluchowski-type equations with transmission boundary conditions. This system is meant to describe the aggregation and diffusion of pathological tau proteins inside the axons and in the extracellular space. We prove the existence, uniqueness, positivity and boundedness of solutions to the model equations derived at the microscale (that is, the scale of single neurons). Then, we study the convergence of the homogenization process to the solution of a macro-model asymptotically consistent with the microscopic one [FL24].

References

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[GS17] Michel Goedert and Maria Grazia Spillantini. "Propagation of tau aggregates". In: *Mol. Brain* 10.1 (2017), pp. 18–26.

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MS61-04 — Linking Astrocyte Morphology to Metabolic Dysfunction in Alzheimer's Disease: A Multiscale Modelling Approach

SPEAKERS Sofia Farina (University of Bern)
AUTHORS Alexander Skupin (University of Luxembourg); Sofia Farina (University of Bern); Stéphane Bordas (University of Luxembourg)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Astrocytes are glial cells essential for brain homeostasis, acting as metabolic mediators that couple cerebral blood flow and nutrient uptake to neuronal energy demands. In Alzheimer's disease (AD), progressive neurodegeneration is accompanied by profound alterations in astrocyte morphology and metabolism. Reactive transformation leads to significant structural remodeling and metabolic changes [Esc+21], yet how these morphological alterations contribute to astrocytic metabolic dysfunction remains unclear. Here, we investigate the relationship between AD-related morphological changes and metabolic function using a multiscale mathematical modelling framework [Far+21; Far+23; Pap+24]. The model integrates intracellular energy metabolism with realistic three-dimensional astrocyte morphologies reconstructed from post-mortem human AD and age-matched control. By coupling spatially resolved metabolic pathway dynamics with cellular geometry, we systematically analyse how disease-associated alterations affect energy production, intracellular distribution, and neuronal support capacity. Simulations show that AD-related metabolic dysfunction reduces energetic output and compromises astrocytic support to neurons. Energy deficits arise from the cumulative impairment of multiple pathways rather than a single defect. Notably, morphological remodeling appears to partially compensate for metabolic deficits, suggesting a potential protective response in AD.

References

[Esc+21] Carole Escartin et al. "Reactive astrocyte nomenclature, definitions, and future directions". In: *Nature neuroscience* 24.3 (2021), pp. 312–325.

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MS62 — Newtonian and non-Newtonian Biofluidmechanics: Integrating Theory, Experiments, Modeling, and Simulations

TRACK Miscellaneous
SCHEDULE Monday, 13 July 2026; 10:40–18:20; 05.12 - HS
ORGANISERS Sookkyung Lim (University of Cincinnati); Sarah Olson (Worcester Polytechnic Institute); Owen Lewis (University of New Mexico)

Minisymposium abstract

Biological fluid–structure interactions arise in environments ranging from Newtonian liquids to complex non-Newtonian media such as mucus, cytoplasm, and extracellular matrices. This minisymposium focuses on Newtonian and non-Newtonian biofluid mechanics, integrating theoretical analysis, experimental observations, mathematical modeling, and numerical simulations. Topics include microscale locomotion and transport, fluid–structure interactions, rheological effects on biological function, and multiscale biomechanical phenomena. By bringing together diverse methodological perspectives, the minisymposium

aims to foster cross-disciplinary dialogue and advance our understanding of biological flows in complex fluid environments.

MS62-12 — Biofilm Rheology: Properties, Applications, and Parameter Estimation

SPEAKERS Nicholas Cogan (Florida State University)
AUTHORS Nicholas Cogan (Florida State University)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 05.12 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Biofilms—communities of bacteria encased in a self-produced extracellular matrix—exhibit unique mechanical properties that are essential to their formation, persistence, and adaptation to environmental conditions. These properties emerge dynamically as biofilms interact with their physical and chemical surroundings. Recent advances have provided deeper insights into the structural composition of the biofilm matrix and the genetic regulation of its polymeric constituents. In this talk, we will examine longitudinal rheological observations of mutant biofilm strains to elucidate the role of specific polymer types within the matrix. Additionally, from a mathematical perspective, we will explore parametric uncertainty and forward uncertainty propagation, discussing their implications for previous studies and future research directions.

MS62-09 — Modeling of biofouling dynamics in membrane filtration systems

SPEAKERS Vincenzo Luongo (University of Napoles Federico II)
AUTHORS Vincenzo Luongo (University of Napoles Federico II)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 05.12 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Microfiltration is a widely used technology for water production and wastewater treatment. The major limitation associated with this process is the formation of fouling layers on membrane surfaces, primarily composed of organic matter such as bacteria and extracellular polymeric substances (EPS). The dynamics of these deposits leads to an increase in the hydraulic resistance and a consequent decline in permeate flux during membrane operation. In water treatment systems, biofouling constitutes a significant operational cost due to the energy consumption and chemical usage required for membrane cleaning procedures. Since biofouling is intrinsically connected to biofilm formation and development, the ability to predict biofilm growth and its temporal evolution on membrane surfaces is essential for optimizing membrane reactor performance and designing effective backwashing strategies under different filtration conditions. To address this issue, a one-dimensional PDE mathematical model has been developed and formulated as a free boundary value problem, describing biofilm dynamics and EPS production during microfiltration. The proposed framework combines classical filtration theory in series—based with a multispecies biofilm growth model. The proposed model provides a mathematical framework for describing backwashing effects and biofouling kinetics during microfiltration processes.

MS62-11 — Mixing and transport in the Drosophila melanogaster oocyte

SPEAKERS David Stein (Flat Iron Institute)
AUTHORS David Stein (Flat Iron Institute); Olenka Jain (Princenton University)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 05.12 - HS
TRACK Miscellaneous

Abstract

In the oocyte of the common fruit fly, large scale cytoplasmic flows appear in the mid-to-late stages of oogenesis. Proteins, synthesized in adjoining nurse cells, and yolk, endocytosed through the cell cortex,

are transported and mixed throughout the oocyte, presumably accelerated by the cytoplasmic flows. While a biophysical mechanism has been proposed which explains the onset of cytoplasmic streaming flows [Ste+21; Dut+24], the flows thereby produced are poor at mixing and typically orient themselves orthogonal to the required transport direction [Jai+25]. In this talk, we explore further mechanisms which accelerate cytoplasmic mixing and transport.

References

[Dut+24] Sayantan Dutta et al. "Self-organized intracellular twisters". In: *Nature Physics* 20.4 (2024), pp. 666–674.

[Jai+25] Olenka Jain et al. "Geometric effects in large scale intracellular flows". In: *PRX Life* 3.2 (2025), p. 023007.

[Ste+21] David B Stein et al. "Swirling instability of the microtubule cytoskeleton". In: *Physical review letters* 126.2 (2021), p. 028103.

MS62-10 — Modeling and Optimization of Microfluidic Spheroid-on-Chip Systems Using Computational Fluid Dynamics and Neural Network–Based Bayesian Optimization

SPEAKERS Mohsen Rezaeian (Department of Applied Mathematics, University of Waterloo, Canada)

AUTHORS Erich Alexandro Márquez Galarz (Facultad de Ciencias Químicas, Universidad Autónoma de San Luis Potosí, San Luis Potosí, Mexico); Biran Ingalls (Department of Applied Mathematics, University of Waterloo, Canada); Mohsen Rezaeian (Department of Applied Mathematics, University of Waterloo, Canada)

SCHEDULE Monday, 13 July 2026; 11:40–12:00; 05.12 - HS

TRACK Mathematical Oncology

Abstract

Microfluidic spheroid-on-chip platforms are widely used for high-throughput drug screening, providing controlled microenvironments for studying tumor–drug interactions. However, drug delivery to tumor spheroids in these devices is governed by complex fluid flow and mass transport processes, making device design challenging. Consequently, their design has often relied on time- and resource-intensive experimental trial-and-error approaches. Here, we develop a computational framework for modeling drug transport in multi-well spheroid-on-chip systems. A computational fluid dynamics model is constructed to simulate fluid flow and drug transport within a microfluidic array, capturing key mechanisms including advection, diffusion, and binding. The model is validated against published experimental data. Simulation results reveal how device geometry, fluid velocity, and tumor- and drug-related parameters influence drug distribution and local shear stress within the wells. Importantly, the simulations predict device-induced heterogeneity in drug exposure and shear stress across spheroids in different wells. To address the need for well-to-well consistency in drug screening platforms, simulation data from parametric sweeps are used to train a neural network surrogate model. The surrogate model is then integrated into a Bayesian optimization framework to identify device configurations that minimize variability in drug exposure across wells, which is essential for reliable drug screening.

MS62-14 — From Langevin Functions to Macro Closures: Multiscale Modeling of Viscoelastic Biofluids

SPEAKERS Paula Vasquez (University of South Carolina)

AUTHORS Michael Cromer (Rochester Institute of Technology); Paula Vasquez (University of South Carolina)

SCHEDULE Monday, 13 July 2026; 15:00–15:20; 05.12 - HS

TRACK Miscellaneous

Abstract

Viscoelastic behavior in biological fluid arises from the stretching of polymeric structures. Capturing finite extensibility is essential for predicting phenomena like strain hardening and elastic instabilities. This work develops a multiscale framework linking the statistical mechanics of freely jointed chains, expressed through

the inverse Langevin function (ILF), to stochastic microscale simulations and continuum constitutive models.

Using the Brownian Configuration Field approach [Hvv97], we compare stochastic dumbbell dynamics across three ILF approximations: the classical FENE model and the more accurate Pade-based approximations of Cohen and Rickaby–Scott (RS). While models coincide in weak and fully stretched limits, significant differences emerge in the moderate extension regime critical to biofluids. FENE consistently underestimates chain stretch and stress. Linear stability analysis shows that the ILF choice influences eigenvalue growth, impacting numerical stiffness.

To enable large-scale simulations, we derive corresponding macroscopic closures—FENE-P, FENE-CR, and new Cohen/RS-based variants—which preserve Pade-based accuracy while maintaining computational efficiency. Validation against stochastic data demonstrates enhanced prediction of strain hardening, oscillatory responses, and thinning dynamics.

References

[Hvv97] M.A Hulsen, A.P.G van Heel, and B.H.A.A van den Brule. "Simulation of viscoelastic flows using Brownian configuration fields". In: *Journal of Non-Newtonian Fluid Mechanics* 70.1 (1997), pp. 79–101. ISSN: 0377-0257. DOI: https://doi.org/10.1016/S0377-0257(96)01503-0. URL: https://www.sciencedirect.com/science/article/pii/S0377025796015030.

MS62-15 — Dynamic deformation and positioning of nucleus

SPEAKERS Yuan-Nan Young (New Jersey Institute of Technology)
AUTHORS Alex Barnett (Flatiron Institute); Libin Liu (Flatiron Institute); Michael Shelley (Flatiron Institute); Reza Farhadifar (Flatiron Institute); Yuan-Nan Young (New Jersey Institute of Technology)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 05.12 - HS
TRACK Miscellaneous

Abstract

We investigate the coupled dynamics of centrosomes and the cell nucleus under microtubule-mediated pulling within a viscous cytoplasmic environment. A coarse-grained, stoichiometric framework [You+25] is developed to capture the interactions between microtubules and force generators (FGs) distributed on the nuclear envelope and cortex. The model integrates hydrodynamic coupling, nuclear envelope elasticity, FG transport along the nuclear envelope, and FG binding kinetics, enabling quantitative predictions of force balance and shape evolution. Using a boundary-integral formulation benchmarked against analytical limits, we examine how envelope stiffness, permeability, and FG mobility control the stability and positioning of the centrosome nucleus complex. Simulations in spherical and spheroidal geometries reveal that enhanced FG mobility or weakened envelope stiffness amplifies nuclear deformation and destabilizes centrosomal organization conditions reminiscent of pathological nuclear softening. Parameter sweeps across FG number and mechanical moduli delineate the transition from stable to misaligned configurations. This framework establishes a unified mechanical description linking molecular-scale force transduction to mesoscale nuclear morphology, providing mechanistic insight into how cytoskeletal dysregulation and envelope integrity jointly govern centrosome nucleus coupling.

References

[You+25] Yuan-Nan Young et al. "Geometric model for dynamics of motor-driven centrosomal asters". In: *Physical Review Research* 7.1 (2025), p. 013004.

MS62-16 — Monte Carlo simulations of membrane microfiltration for water purification

SPEAKERS Francesca Bernardi (Worcester Polytechnic Institute)
AUTHORS Abigail Drumm (Worcester Polytechnic Institute); Francesca Bernardi (Worcester Polytechnic Institute)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 05.12 - HS
TRACK Miscellaneous

Abstract

Microfiltration is a water purification method used by municipal facilities to produce potable water that relies primarily on a size-exclusion mechanism. When a contaminated solvent passes through a membrane filter, unwanted contaminants with diameter larger than that of the membranes pores, such as suspended solids, large bacteria, and proteins, are retained on the membrane. This foulant build-up occludes the area open for fluid flow, impairing the efficiency of filtration operations by decreasing the flux through the membrane over time. Backwashing is a strategy to restore filtration wherein clean water is processed backward through the membrane to dislodge attached foulants. The overarching goal of this work is to determine the optimal backwashing parameters (duration, frequency, and flux) to ensure sufficient membrane recovery without sacrificing more clean water than is needed.

We developed a 2D Monte Carlo model to simulate forward filtration and backwashing through constant pressure, dead-end, at-sheet membranes, and benchmarked it against lab-scale experiments [DB25]. Then, we extended our model to approximate the more complicated hollow-fiber membrane geometry and were able to qualitatively capture constant-flux filtration operations in such systems. Our next step aims to incorporate long-term fouling mechanisms into our model to capture foulant build-up during cake filtration which can lead to biofilm formation. This research is funded by NSF CBET-2211001.

References

[DB25] Abigail Rose Drumm and Francesca Bernardi. “Monte Carlo simulations of two-dimensional flat-sheet membrane filters for constant-pressure water purification”. In: *Physical Review E* 112.5 (2025), p. 055501.

MS62-13 — A spheroidal squirmer model for euglenids

SPEAKERS Henry Shum (University of Waterloo, Canada)
AUTHORS Henry Shum (University of Waterloo, Canada)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 05.12 - HS
TRACK Miscellaneous

Abstract

Euglenids are flagellated microorganisms whose elongated, flexible bodies enable rich swimming dynamics in confined environments. Focusing on their flagellated mode of locomotion rather than body shape deformations, we construct a simplified hydrodynamic model in which the euglenid is represented as a rigid prolate spheroid and surface slip velocities are imposed over a portion of the body to model the action of the flagellum at one pole. Using a boundary element method, we numerically compute the flow field and resulting swimmer dynamics driven by the squirming activity. We investigate the behavior of these swimmers in both straight and wavy channels, focusing on how confinement and channel geometry influence trajectories, orientations, and migration patterns. Systematic variations of swimmer aspect ratio, the active fraction of the body length, and channel parameters allow us to identify key geometric and hydrodynamic mechanisms governing swimmer–channel interactions. Our results highlight the strong coupling between swimmer geometry and environmental structure, demonstrating that the body shape and flagellar activity can be tuned to promote transport through particular structures. This framework provides a physically grounded and computationally efficient model for euglenid motility and offers insights relevant to microorganism transport in complex microfluidic and biological environments.

MS62-19 — Seeing new depths: Three-dimensional flow of a free-swimming alga

SPEAKERS Sookkyung Lim (University of Cincinnati, USA)
AUTHORS Gregorius Pradipta (University of Minnesota); Wanho Lee (National Institute for Mathematical Sciences, Republic of Korea); Van Tran (University of Minnesota, USA); Kyle Welch (University of Minnesota, USA); Santosh Sankar (University of Minnesota, USA); Yongsam Kim (Chung-Ang University, Seoul, Republic of Korea); Satish Kumar (University of Minnesota, USA); Xin Yong (University at Buffalo, USA); Jiarong Hong (University of Minnesota, USA); Sookkyung Lim (University of Cincinnati, USA); Xiang Cheng (University of Minnesota, USA)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 05.12 - HS
TRACK Miscellaneous

Abstract

A swimming microorganism stirs the surrounding fluid, creating a flow field that governs not only its locomotion and nutrient uptake, but also its interactions with other microorganisms and the environment. Despite its fundamental importance, capturing this flow field and unraveling its biological implications remains a challenge. Here, we report the first direct, time-resolved measurements of the 3D flow field generated by a single, free-swimming microalga, Chlamydomonas reinhardtii, a model organism for microbial locomotion and flagellar dynamics. Supported by hydrodynamic modeling and simulations, our measurements resolve how established 2D flow features such as in-plane vortices and the stagnation point emerge from and shape the 3D structure of the algal flow. More importantly, we reveal unexpected low-Reynolds-number flow phenomena including micron-sized vortex rings and periodically recurring translating vortices and uncover topological changes in the underlying fluid structure associated with the puller-to-pusher transition of an alga. Biologically, access to the 3D flow field enables rigorous quantification of the alga's energy expenditure, as well as its swimming and feeding efficiency, improving the precision of these key physiological metrics. Our study demonstrates rich vortex dynamics in inertialess flows and shows their influence on microbial motility. The work also introduces a new method for mapping the fluid environment sculpted by beating flagella.

MS62-17 — Swimming-limited aggregation of Escherichia coli bacteria in liquid crystals

SPEAKERS Maria Tătulea-Codrean (University of Amsterdam, Netherlands)
AUTHORS Guillaume Sintès (Université Paris Sciences et Lettres, France); Martyna Goral (Université Paris Sciences et Lettres, France); Teresa Lopez-Leon (Université Paris Sciences et Lettres, France); Anke Lindner (Université Paris Sciences et Lettres, France); Maria Tătulea-Codrean (University of Amsterdam, Netherlands)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 05.12 - HS
TRACK Miscellaneous

Abstract

Liquid crystals serve as model systems for structured environments and represent a broader class of anisotropic, non-Newtonian fluids encountered by bacteria, such as host mucus [Fig+19] and extracellular polymeric substances involved in biofilm formation [Rep+22]. In this study, we investigated the collective swimming of fluorescently labelled Escherichia coli in nematic liquid crystals and observed the emergence of long-lived chains of bacteria swimming along the nematic director, similar to those observed for Proteus mirabilis by Mushenheim et al. [Mus+14]. Remarkably, we found that longer chains swim faster, contrary to predictions from fundamental force-balance models and observations of merging bacterial pairs. To explain this counterintuitive behaviour and identify the physical mechanism driving bacterial aggregation in liquid crystals, we combined experiments with agent-based simulations and minimal theoretical modeling. By incorporating the intrinsic speed distribution of individual bacteria, our simulations revealed a positive correlation between chain length and swimming speed, consistent with experimental observations. A minimal aggregation model, based on encounter probabilities between a bacterium and its two nearest neighbours, further supports our interpretation that longer chains swim faster because they are more likely to contain faster-swimming individuals, which meet and merge with their neighbours in less time.

References

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[Mus+14] Peter C Mushenheim et al. “Dynamic self-assembly of motile bacteria in liquid crystals”. In: *Soft Matter* 10.1 (2014), pp. 88–95.

[Rep+22] Andrii Repula et al. “Biotropic liquid crystal phase transformations in cellulose-producing bacterial communities”. In: *Proceedings of the National Academy of Sciences* 119.24 (2022), e2200930119.

MS62-20 — Parallel computing methods for large-scale simulations of flagellar dynamics

SPEAKERS Weifan Liu (Beijing Forestry University, China)
AUTHORS Weifan Liu (Beijing Forestry University, China); Ruixiang Huang (University of Washington-Seattle, USA)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 05.12 - HS
TRACK Miscellaneous

Abstract

The Method of Regularized Stokeslets (MRS) provides a robust, mesh-free framework for resolving the fluid-structure interactions of these filaments [Cor01]. It has two critical bottlenecks: the spatial complexity of pairwise hydrodynamic calculations and the temporal stiffness arising from the material properties of the rod. To address these challenges, we first discuss a parallel-in-time approach based on the Parareal algorithm [LMT01], in which we construct a novel coarse solver using a data-driven method based on Dynamic Mode Decomposition in place of a classic time marching scheme. Then we will present a heterogeneous CPU–GPU computing framework that synergistically addresses the challenges in space and time complexity to enable scalable, high-fidelity simulations of flagellar dynamics.

References

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MS62-07 — Multi-timescale dynamics of microswimmers

SPEAKERS Sara Drummond-Curtis (University College London)
AUTHORS Benjamin Walker (University College London, UK); Mohit Dalwadi; Sara Drummond-Curtis (University College London)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 05.12 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Microswimmers typically undergo rapid motion to propel themselves through fluid. When considering the long-time swimmer dynamics of interest, the effect of the rapid motion is often assumed to average out without significantly affecting the trajectories. However, recent work has shown that this is not always the case. In this talk, we consider how the interaction between rapid propulsive motion and environmental factors influences swimmer trajectories. Touching upon examples of boundaries and external flows, we exploit the separation of timescales through a systematic multiscale analysis to derive effective equations for the long-time dynamics. As a result of our analysis, qualitatively different predictions emerge, highlighting the role of fast-time motion on the long-time swimmer dynamics as they navigate their environment.

MS62-21 — Swimming Strategies of Bipolar Flagellated Bacteria

SPEAKERS Jeungeun Park (State University of New York at New Paltz, USA)
AUTHORS Jeungeun Park (State University of New York at New Paltz, USA); Yongsam Kim (Chung-Ang University, Republic of Korea); Wanho Lee (National Institute for Mathematical Sciences, Republic of Korea); Sookkyung Lim (University of Cincinnati, USA)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 05.12 - HS
TRACK Miscellaneous

Abstract

Flagellated bacteria navigate complex environments by modulating flagellar rotation. The properties of the flagella, together with the cell body shape, govern the dynamics of bacterial motion and resulting swimming strategies. While the motility of many flagellated bacteria is well understood, comparatively little is known about bipolar flagellated species. In this talk, we present a mathematical model for the motility of Campylobacter jejuni, which drives a flagellum at each pole to move through the viscous mucosa of its host's gastrointestinal tract [Coh+20]. We investigate how the interplay between the helical body shape and spatially non-uniform rigidities of the flagella gives rise to distinct swimming modes. In particular, we highlight the wrapping mode, in which the leading flagellum wraps around the cell body.

References

[Coh+20] Eli J Cohen et al. "Campylobacter jejuni motility integrates specialized cell shape, flagellar filament, and motor, to coordinate action of its opposed flagella". In: *PLoS pathogens* 16.7 (2020), e1008620.

MS62-22 — Modeling the Transposition of Deformable Bacteria Through Membrane Pores

SPEAKERS Abigail Drumm (Worcester Polytechnic Institute, USA)
AUTHORS Abigail Drumm (Worcester Polytechnic Institute, USA); Francesca Bernardi (Worcester Polytechnic Institute, USA)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 05.12 - HS
TRACK Miscellaneous

Abstract

From research to clinical development and production, Mycoplasma are well-recognized and widespread contaminants in biopharmaceutical manufacturing. Their successful proliferation despite the ultrafiltration performed to eliminate them has been attributed in part to the flexibility of their cell walls.

How does this bacterium manage to squeeze through pores that are, in some cases, one-third of its size? We build on a force-balance approach to quantify a threshold transmembrane pressure that determines retention versus passage, then use semi-analytical modeling based on elasticity relations, Stokes equations, and the method of reflections to explore the deformation and motion of a bacterium once it has trespassed into a pore.

This work aims to identify the maximum flow conditions at which membrane filtration remains a viable strategy for the filtering out of Mycoplasma from permeate to effectively inform the processes and materials employed to eradicate it. This research is funded by NSF CBET-2211001.

MS62-24 — Morphology and dynamics of chemotacting droplets

SPEAKERS Giulia Celora (University of Oxford)
AUTHORS Giulia Celora (University of Oxford); Benjamine Walker (University College London); Mohit Dalwadi (University of Oxford); Philip Pearce (University College London)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 05.12 - HS
TRACK Miscellaneous

Abstract

Collective cell migration is ubiquitous amongst multicellular communities and contributes to many phenomena, e.g., morphogenesis and cancer metastasis. Nonetheless, it is still poorly understood how cells coordinate to control the emergent collective motion of cell groups (or swarms). Recent experimental data suggests that physical interactions between cells within the swarms can result in emergent fluid-like properties. In this talk, I will discuss our recent developments in modelling the spatiotemporal dynamics of cell swarms' collective and their emergent fluid mechanics. Combining numerics and analysis, our results reveal how the interplay between physical interactions, cell proliferation and cell motion shape the morphology and migration dynamics of cell collectives.

MS62-23 — Efficient Immersed Boundary method for flows with rigid objects

SPEAKERS Brittany Leathers (University of Hawai'i at Mānoa)
AUTHORS Brittany Leathers (University of Hawai'i at Mānoa)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 05.12 - HS
TRACK Miscellaneous

Abstract

The Immersed Boundary (IB) method is widely used for problems that involve fluid-structure interactions or complex geometries. However, when adapted to flows with rigid objects, the IB method typically involves either using penalty forces, which only approximately satisfy boundary conditions, or they are formulated as constraint problems that suffer from the need to solve a linear system that is poorly conditioned. These types of fluid-structure interactions are important for many biological applications, including swimming organisms and flows with rigid particles. Here, we present the Immersed Boundary Double Layer (IBDL) method for boundary value problems. It relies on a reformulation of the IB method that corresponds to a regularized second-kind integral equation. The linear solve is then well-conditioned and can therefore be accomplished with a small number of iterations of a Krylov method without preconditioning. Therefore, this method is highly beneficial for time-dependent problems for which this solve is done at every time step. We present the method formulation and recent developments.

MS62-01 — The Immersed Boundary Single-Layer (IBSL) method for reaction-advection-diffusion systems

SPEAKERS Owen Lewis (University of New Mexico)
AUTHORS Brittany Leathers (University of Hawaii at Manoa); Owen Lewis (University of New Mexico); Robert Guy (University of California Davis)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 05.12 - HS
TRACK Miscellaneous

Abstract

The Immersed Boundary Method (IBM) is widely used in the simulation of systems involving fluid-structure interaction [Pes02]. The ability to simulate problems involving complex, moving geometry using a simple, Eulerian grid has led to the use of the IBM for the study of many biomechanical and biophysical systems. However, the study of chemical systems often necessitates the ability to impose Neumann or Robin boundary conditions at irregular locations on the Eulerian grid. Here, we present a method for imposing Neumann and Robin boundary conditions within an IBM framework. The method leverages the single-layer formulation of boundary integral equations, and we therefore refer to it as the Immersed Boundary Single Layer (IBSL) method. It requires solving a larger, augmented linear system. However, this system is well-conditioned and can be solved with a small number of iterations of a standard Kylov method without preconditioning. We also present some convergence results, limitations, and use cases of our methodology.

References

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MS62-02 — Suppression of upstream swimming in shear flows by memory-mediated chemotaxis

SPEAKERS Smitha Maretvadakethope (Imperial College London)
AUTHORS Ruben Perez-Carrasco (Imperial College); Sahil Rai (Imperial College); Smitha Maretvadakethope (Imperial College London); Smitha Maretvadakethope (Imperial College)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 05.12 - HS
TRACK Miscellaneous

Abstract

Elongated microswimmers such as E. coli exhibit run-and-tumble dynamics that bias motion in response to chemical gradients. In confined pressure-driven flows, elongated swimmers also reorient along Jeffery orbits, spending extended periods oriented near the upstream or downstream direction. Near boundaries, this alignment leads to upstream swimming, contributing to contamination and surface colonisation [BH15; KK12].

In this work, we investigate the competition between shear-induced reorientation and finite memory chemotactic responses in confined flows. We show that strong chemotactic sensitivity amplifies temporal comparisons in chemical signalling, fundamentally altering the orientation phase space associated with shear-induced dynamics of near-wall swimmers. In particular, strong chemorepulsive cues at channel surfaces lead to the loss of longer upstream trajectories, inducing a transition from positive rheotaxis to downstreamdominated transport.

Chemotactic memory, when coupled with chemorepulsive surface cues, inhibits upstream contamination by progressively reducing the number of swimmers occupying upstream-oriented states. These results identify a general mechanism by which memory-driven chemotactic responses can be used to control microswimmer transport in flow, offering new design principles for limiting surface colonisation in microfluidic and biomedical environments, complementing existing studies of microswimmer–surface interactions and suspension dynamics [Mar+23; RGS14; ES15].

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[KK12] Tolga Kaya and Hur Koser. “Direct upstream motility in Escherichia coli”. In: *Biophysical journal* 102.7 (2012), pp. 1514–1523.

[Mar+23] Smitha Maretvadakethope et al. “The interplay between bulk flow and boundary conditions on the distribution of microswimmers in channel flow”. In: *Journal of Fluid Mechanics* 976 (2023), A13.

[RGS14] Roberto Rusconi, Jeffrey S Guasto, and Roman Stocker. “Bacterial transport suppressed by fluid shear”. In: *Nature physics* 10.3 (2014), pp. 212–217.

MS62-03 — Mechanical Interactions between a Contact Lens and the Eye

SPEAKERS Lucia Carichino (Rochester Institute of Technology)
AUTHORS Kara Maki (Rochester Institute of Technology); Lucia Carichino (Rochester Institute of Technology); Riley Supple (Rochester Institute of Technology)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 05.12 - HS

TRACK Miscellaneous

Abstract

Contact lenses are worn by millions of people worldwide to correct vision impairments. The mechanical interactions between the lens and the ocular surface are difficult to access in the clinic. We developed a mathematical model of the mechanical interactions between the lens and the eye to predict the ocular stress load due to contact lens wear.

The lens is modeled as a thin, axially symmetric, linear elastic material that conforms to the eye [RMH16]. The eye is modeled as an axially symmetric, linear elastic material with spatially varying material properties. The eye and lens models are coupled non-linearly via the suction pressure under the lens [Car+25]; we assume the thin tear film layer between the lens and the eye has a constant thickness. The coupled problem is solved by an iterative numerical algorithm using finite difference methods to approximate the lens mechanics and f inite element methods to approximate the eye. We have extended the model to account for the effect of intraocular pressure (IOP) on lens-eye interactions.

The model predicts that the center of the cornea (center of the eye) is negatively displaced (toward the inside of the eye), while the edge of the cornea is positively displaced and experiences the highest stresses. The final aim of this work is to improve contact lens design and the fitting process by predicting ocular deformations and ocular stress load before wearing a lens.

References

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MS62-04 — An immersed boundary model for fluid membrane interaction in the cochlea

SPEAKERS John Stockie (Rochester Institute of Technology)
AUTHORS John Stockie (Rochester Institute of Technology)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 05.12 - HS
TRACK Miscellaneous

Abstract

The mammalian ear has a remarkable ability to distinguish sounds that differ only slightly in frequency, while at the same time amplifying these signals so that they can be converted into neural impulses. This fine-frequency tuning phenomenon is commonly attributed to some form of mechanical resonance within the fluid-filled cochlea (or inner ear), and more specifically to resonant oscillations in the basilar membrane (BM) that runs along the cochlear duct [Hud97]. We extend a well-known immersed boundary model for fluid-structure interaction in the cochlea [Bey92; LPL88] by incorporating a small-amplitude periodic internal forcing due to contractions of outer hair cells that are embedded within the BM structure and induce parametric resonance through periodic variations in the BM stiffness. A Floquet stability analysis demonstrates the existence of resonant (unstable) solutions for physical parameters typical of mammalian cochleas, and also exhibits travelling wave solutions that are consistent with other models and experiments [KS15]. We then describe more recent efforts to include the influence of Reissner’s membrane, which is another much more flexible elastic structure that is typically ignored in other cochlear models. Numerical simulations validate the analytical results and support the hypothesis that fluid-mediated resonance may be a significant contributing factor in the active process that drives cochlear mechanics [RH14].

References

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[RH14] Tobias Reichenbach and A James Hudspeth. “The physics of hearing: fluid mechanics and the active process of the inner ear”. In: *Reports on Progress in Physics* 77.7 (2014), p. 076601.

MS62-05 — Drag on Microtubule Asters

SPEAKERS Sarah Olson (Worcester Polytechnic Institute, USA)
AUTHORS Dana Ferranti (Worcester Polytechnic Institute); Sarah Olson (Worcester Polytechnic Institute, USA)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 05.12 - HS
TRACK Miscellaneous

Abstract

Microtubule asters–dynamic microtubules anchored at a centrosome–form the spindle poles during cell division. Utilizing the method of regularized Stokeslets, we prescribe the motion of microtubule asters in a confined geometry and compute the resulting hydrodynamic drag on the aster. Moving beyond classical asymptotic approximations, we characterize the drag on a single aster inside a spherical cell as a function of its positions and microtubule distribution. We then extend this to study drag on several interacting asters, corresponding to cellular aster configurations prior to division.

MS62-06 — How do glioma cells infiltrate the narrow gap between astrocytes endfeet and blood vessel through ion-exchange and physical deformation for the critical cellular infiltration?

SPEAKERS Yangjin Kim (Konkuk University)
AUTHORS Sangkwon Kim (Korea University); Sookkyung Lim (University of Cincinnati); Wanho Lee (National Institute for Mathematical Sciences); Yangjin Kim (Konkuk University)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 05.12 - HS
TRACK Miscellaneous

Abstract

Malignant gliomas are devastating, aggressive tumours that frequently kill patients with a low survival rate (1 year after diagnosis). Diffuse invasion of glioma cells after surgical resection in brain tissue is a major obstacle for effective therapy. Glioma cells use the tortuous extracellular routes of migration, using blood vessels as guides. These cells manipulate ion channels in the local microenvironment to dynamically adjust their cell volume to accommodate to narrow spaces and breach the blood-brain barrier through disruption of astrocytic endfeet, that support blood vessels. We investigated the cell-mechanical aspect of glioma cell migration along the blood vessels by using an immersed boundary method without volume convervation. This involves many intercellular interactions and biochemical reactions. We illustrate that the viscoelastic properties of the glioma cell and ion-induced reduction in volume enable them to penetrate the narrow gap. The unique biology of glioma invasion provides unexplored brain-specific therapeutic targets for this devastating disease with little effective treatment options.

References

[Cud+14] Vishnu Anand Cuddapah et al. “A neurocentric perspective on glioma invasion”. In: *Nature Reviews Neuroscience* 15.7 (2014), pp. 455–465.

MS62-08 — The Method of Regularized Stokeslets as an Introduction to Fluid-Structure Interactions

SPEAKERS Amy Buchmann (University of San Diego)
AUTHORS Amy Buchmann (University of San Diego); Eva Strawbridge (James Madison University); Longhua Zhao (Case Western Reserve University)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 05.12 - HS
TRACK Miscellaneous

Abstract

Mathematical and computational modeling plays an important role in understanding biological fluid mechanics. Even though the fluid motion is governed by partial differential equations, there are many projects in this field that can be made accessible to undergraduate students. The Method of Regularized Stokeslets (MRS) [Cor01] is a popular technique used in modeling low Reynolds number flows. In this talk, I discuss the development of an introductory guide to the MRS designed for undergraduate students with a background in linear algebra and multivariable calculus. This framework can be adapted for high-impact research projects for undergraduates at any level.

References

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MS62-18 — Thin-film modelling and parameter optimisation for biofilms

SPEAKERS Alex Tam (Adelaide University)
AUTHORS Alex Tam (Adelaide University); Daniel Netherwood (Adelaide University); Jennifer Gardner (Adelaide University); Jin Zhang (Adelaide University); Campbell Gourlay (The University of Kent); Vladimir Jiranek (The University of Southampton); Benjamin Binder (Adelaide University); Edward Green (Adelaide University)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 05.12 - HS
TRACK Miscellaneous

Abstract

Yeast colonies exhibit a wide range of patterns and growth modes, making them a fruitful source of mathematical modelling problems. Our group aims to better understand yeast growth using agent-based, reaction–diffusion, and continuum mechanical models, and collaborates with experimental yeast biologists based in Australia and the UK. Biofilms are a form of growth characterised by communities of cells residing within a self-produced protective viscous matrix. They cause an estimated 80% of all microbial infections and are difficult to remove, giving them particular biomedical importance.

I will discuss our recent work [Tam+26] that applies an extensional-flow thin-film model to model how agar density affects biofilm growth in lab experiments. The mathematical model contains 5 unknown parameters, requiring multiple experimental measurements (biofilm size, shape, and composition) and numerical optimisation to estimate parameters effectively. Parameter optimisation reveals that higher density agar increases biofilm–substratum adhesion strength, which favours vertical over horizontal expansion.

References

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MS69 — Spatial plasticity and heterogeneity in cancer: from niches to therapy

TRACK Mathematical Oncology
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 15.04 - HS
ORGANISERS Juan Jiménez-Sánchez (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla-La Mancha (UCLM)); Simon Syga (Technische Universität Dresden)

Minisymposium abstract

Tumour cell plasticity and intratumour heterogeneity are key determinants of cancer progression and treatment failure [Cor+24; Sef+23]. In particular, spatial heterogeneity in the tumor microenvironment (hypoxia, acidity, nutrient limitation, immune/stromal organisation, and mechanical constraints) shapes phenotypic states and their transitions [PBB23]. Mathematical modeling can give qualitative insight into these processes. Yet, many modelling efforts neglect spatial tumor heterogeneity, e.g., by assuming a homogeneous tumor microenvironment. This MS will place spatial heterogeneity at the centre of cancer plasticity modelling, aiming to integrate eco-evolutionary ideas with spatially resolved data and clinically relevant treatment contexts. We will bring together researchers working on phenotype switching in spatially structured tumours, hybrid multiscale models linking intracellular regulatory networks to tissue-level patterns, and data-driven mechanistic inference from spatial imaging and multi-omics. Particular attention will be given to (i) evolutionary dynamics underlying intratumor heterogeneity, (ii) multi-compartment plasticity involving tumour, immune and stromal populations, and (iii) spatial aspects of therapy-driven state transitions, including combination therapies. The MS will complement existing sessions on cancer evolution and digital twins by emphasizing explicit spatial plasticity as a unifying mechanistic theme.

References

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MS69-01 — Spatial immune-tumor ecology in the multiple myeloma bone niche: an agent-based modeling approach

SPEAKERS David Basanta (Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, USA)
AUTHORS Conor C. Lynch (Department of Tumor Microenvironment and Metastasis, H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, USA); David Basanta (Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, USA); Matthew Froid (Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, USA)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Cancer progression can be understood as the disruption of tissue homeostasis by tumor cells that co-opt their microenvironment [BA17]. In multiple myeloma (MM), this disruption unfolds within the bone marrow, where stromal cells, osteoclasts, osteoblasts, and immune populations form spatially heterogeneous

niches. We have previously shown that integrated computational models of the bone ecosystem can capture how spatial microenvironmental structure shapes cancer-bone interactions [Ara+14] and how environment-mediated drug resistance (EMDR) in stroma-proximal niches facilitates relapse and clonal heterogeneity [BMF+24]. Separately, our work on stromal protection in breast cancer demonstrated that stroma-augmented proliferation indirectly drives chemoresistance by accelerating tumor recovery between treatment cycles [Mir+23]. Here, we extend our MM bone ecosystem ABM by introducing cytotoxic T-cell and regulatory T-cell (Treg) agents whose activity depends on local microenvironmental conditions. We investigate how spatial niche structure shapes effective anti-myeloma immunity, how Treg-mediated suppression generates local immunosuppressive refugia, and how these dynamics influence evolutionary trajectories under therapy. Our results suggest that spatially homogeneous assumptions about immune function can substantially mischaracterize treatment response, underscoring that niche-specific immune ecology is critical for understanding resistance in MM.

References

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MS69-02 — Integrating longitudinal MRI and clinical data into a biomechanistic tumor growth model for spatial forecasting of prostate cancer aggressiveness

SPEAKERS Guillermo Lorenzo (Department of Mathematics, University of A Coruña, Spain)
AUTHORS Guillermo Lorenzo (Department of Mathematics, University of A Coruña, Spain)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

The Gleason score (GS) is a key predictor of prostate cancer (PCa) aggressiveness and survival, yet treatment decisions rely on biopsies that incompletely sample highly heterogeneous tumors. As a result, clinically relevant spatial variations in tumor aggressiveness may remain undetected. To address this clinically unresolved issue, I present a personalized modelling framework for pointwise prediction of PCa aggressiveness across the 3D tumor domain that is informed by routine clinical and imaging data. The approach combines physics-based and data-driven components in a three-step pipeline. First, a biomechanistic model of PCa growth is personalized using longitudinal MRI and serum PSA data. Second, the model generates spatial maps of mechanistic biomarkers (e.g., reflecting tumor proliferation activity, cell density, and growth dynamics). Third, machine-learning classifiers use these spatial features to infer local tumor aggressiveness across the 3D tumor geometry. Preliminary results using a reaction-diffusion biomechanistic model in a cohort of n=16 PCa cases in active surveillance show that a logistic classifier based on tumor proliferation activity and density achieved an AUC under the ROC curve of 0.96, with sensitivity of 86.4% and specificity of 90.7% for prediction of GS. Of note, model-based predictions anticipated the emergence of higher-risk disease more than one year earlier than standard monitoring, thereby showing promise for guiding clinical decision-making.

References

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MS69-03 — How evolvability dynamics affect tumor growth and treatment response

SPEAKERS Jill Gallaher (Department of Integrated Mathematical Oncology, Moffitt Cancer Center, Tampa, FL)

AUTHORS Alexander R. A. Anderson (Department of Integrated Mathematical Oncology, Moffitt Cancer Center, Tampa, FL); Jill Gallaher (Department of Integrated Mathematical Oncology, Moffitt Cancer Center, Tampa, FL)

SCHEDULE Monday, 13 July 2026; 11:20–11:40; 15.04 - HS

TRACK Mathematical Oncology

Abstract

Drug resistance is an ongoing problem for maintaining a treatment response in advanced cancers, which are often more heterogeneous and evolvable. Evolvability may be beneficial if lesions can easily respond to large shifts in the microenvironment by modifying their traits to survive, like how metastases can survive a new environment and even thrive despite treatment applications. However, evolvability may also be a detriment. With too much deviation from the parental phenotype, cells lose important functions necessary to survive. So, is there an optimal rate of evolvability for tumors to grow and survive treatment that can be exploited therapeutically?

We use an off-lattice agent-based model to investigate how the rate of change through proliferation-migration phenotype space affects tumor growth and response to treatment. During growth, both proliferation and migration are advantageous traits. Increasing migration allows cells to distribute spatially, which allows more proliferation by lessening the spatial competition, and increasing proliferation allows for faster turnover and more mutation. More evolvability leads to more heterogeneity and faster recurrence under treatment. But when evolvability is costly, tumor survival depends on the rate and jump size of heritable changes to transiently lose proliferation fitness selected for during growth and gain resistance for survival. We consider how to design treatment strategies based on a tumor's evolvability dynamics.

MS69-04 — Quantifying Tissue Architecture in the Tumour Microenvironment with Multi-Parameter Persistent Homology

SPEAKERS Kylie Savoye (1) School of Mathematics, College of Engineering and Physical Sciences, University of Birmingham, Birmingham, UK; 2) School of Physics and Astronomy, College of Engineering and Physical Sciences, University of Birmingham, Birmingham, UK)

AUTHORS Alaide Morcavallo (In Vivo Pharmacology, Oncology Targeted Discovery, Oncology, AstraZeneca, UK); Arthur Lewis (Pathology, Clinical Pharmacology & Safety Sciences, R&D BioPharmaceuticals, AstraZeneca, UK); Azam Hamidinekoo (Pathology, Clinical Pharmacology & Safety Sciences, R&D BioPharmaceuticals, AstraZeneca, UK); Fabian Spill (School of Mathematics, College of Engineering and Physical Sciences, University of Birmingham, Birmingham, UK); Kylie Savoye (1) School of Mathematics, College of Engineering and Physical Sciences, University of Birmingham, Birmingham, UK; 2) School of Physics and Astronomy, College of Engineering and Physical Sciences, University of Birmingham, Birmingham, UK); Semiramis Popova (Discovery Bioanalysis Spatial & Quantitative Omics, Clinical Pharmacology & Safety Sciences, R&D BioPharmaceuticals, AstraZeneca, UK)

SCHEDULE Monday, 13 July 2026; 11:40–12:00; 15.04 - HS

TRACK Mathematical Oncology

Abstract

Spatial transcriptomics has revolutionised our ability to measure gene expression while preserving tissue architecture. Yet extracting meaningful patterns from the complex interaction of spatial organisation and

molecular profiles remains challenging, particularly in the heterogeneous tumour microenvironment (TME). Here we apply Multi-Parameter Persistent Homology (MPH), a topological data analysis framework that can simultaneously track cellular organisation patterns across spatial proximity and gene expression levels, to reveal disease-relevant tissue architecture in cancer that may be invisible to conventional methods.

MPH constructs two-parameter filtrations combining spatial distance with gene expression gradients, enabling quantitative characterisation of topological features as they emerge and persist across both parameters. This approach captures how cells organise relative to both their neighbours and their molecular states, which provides a unified signature of tissue structure well suited to characterise the spatial and molecular heterogeneity central to tumour plasticity.

We demonstrate MPH's capabilities in a cancer cell line derived from colorectal cancer tumour, revealing spatial immune and stromal compartmentalisation patterns relevant to understanding TME organisation. Notably, we find differences in topological signatures across fibroblast populations that are not observed in macrophage populations, suggesting that stromal heterogeneity in the TME may have structure beyond purely spatial organisation. We are currently extending this framework to additional human cancer datasets, with ongoing data collection and methods development aimed at broadening its applicability to clinical contexts.

MPH's quantitative characterisation of tissue architecture offers potential for identifying pathology-specific spatial patterns that may inform treatment outcome prediction and disease progression monitoring. Topological approaches like MPH provide a bridge between molecular measurements and the architectural context essential for understanding tumour plasticity, stromal remodelling, and therapeutic response.

MS70 — Emergence of collective behaviour across biological scales

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 02.11 - HS
ORGANISERS Dimitrios Katsaounis (RWTH Aachen University); Viktoria Freingruber (TU Delft)

Minisymposium abstract

Collective behaviour in biological systems often manifests through large-scale, observable patterns. These macroscopic phenomena, however, typically arise from interactions and dynamics of numerous particles occurring at much smaller spatial and temporal scales. Mathematical modelling provides a rigorous framework for elucidating how microscopic interactions within complex, heterogeneous biological systems give rise to coordinated behaviour on the population level, offering insights into fundamental biological processes ranging from cell migration to tissue organization.

This mini-symposium highlights recent advances in mathematical modelling approaches that explicitly incorporate detailed microscopic descriptions. Such approaches account, for example, for dynamic particle size [Dou+25], cell-cell adhesion mediated by molecular bonds [ZR24], or phenotypic heterogeneity [Thi+25; LC25] within populations. By retaining these mechanistic features, the resulting models capture how individual-level properties shape collective dynamics. Within this multiscale perspective, one can study phase transitions and other qualitative changes on the population-level [DFL15] in a mathematically rigorous yet biologically grounded way. By bringing together researchers from different countries and at different stages of their academic careers, this mini-symposium aims to showcase recent advancements in multiscale modelling of collective phenomena.

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MS70-01 — A cell-cell adhesion model: a multiscale derivation

SPEAKERS Anna Zhigun (Queen's University Belfast)
AUTHORS Anna Zhigun (Queen's University Belfast); Mabel Lizzy Rajendran (University of Birmingham)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cell-cell adhesion is a key organiser of tissue structure, in both healthy and cancerous environments, and plays a crucial role in regulating cancer cell migration. In this talk, we introduce a multiscale modelling framework for the dynamics of a moving self-adhesive cell population [ZR24]. The approach links a detailed microscopic description of deterministic adhesion-driven motion with a standard mesoscopic representation of a stochastic velocity-jump process, leading to a kinetic transport equation that features several nonlocal terms. Passing to the macroscopic scale yields continuum equations that couple nonlocal adhesion with myopic diffusion. The framework lends itself conveniently to representing the underpinning binding dynamics of adhesion molecules such as cadherins. When these microscopic effects are translated to the macroscopic level, they generate a novel nonlinear integral equation coupled to the cell-density equation. For a rigorous mathematical analysis of models of this type, we refer to [RZ25] and the talk "A cell-cell adhesion model: local well-posedness" in this conference.

References

[RZ25] Mabel Lizzy Rajendran and Anna Zhigun. "Local Well-Posedness for a Novel Nonlocal Model for Cell-Cell Adhesion via Receptor Binding". In: *SIAM Journal on Mathematical Analysis* 57.4 (2025), pp. 4016–4067. DOI: 10.1137/24M1667518. eprint: https://epubs.siam.org/doi/pdf/10.1137/24M1667518. URL: https://epubs.siam.org/doi/abs/10.1137/24M1667518.

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MS70-02 — Scaling limits for a population model with growth, division and cross-diffusion

SPEAKERS Diane Peurichard (INRIA Paris)
AUTHORS Diane Peurichard (INRIA Paris); Marc Hoffmann (University Paris-Dauphine); Marie Doumic-Jauffret (Inria and Ecole polytechnique); Sophie Hecht (Sorbonne Universite)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Motivated by the modeling of bacteria microcolony morphogenesis across multiple scales, we explore in this talk models for a spatial population of interacting, growing and dividing particles. Starting from a microscopic stochastic model, we first write the corresponding stochastic differential equation satisfied by the empirical measure, and rigorously derive its mesoscopic (mean-field) limit. We then take an interest in the so-called localization limit, to reach a macroscopic (large-scale) model. The scaling consists in

assuming that the range of interaction between individuals is very small compared to the size of the domain. In proving the localization limit using compactness arguments, the difficulties are twofold: first, growth and division render the system non-conservative, preventing the use of energy estimates. Second, the size of the particles, being a continuous trait, leads to new difficulties in obtaining compactness estimates. We first show rigorously the localization limit in the case without growth and fragmentation, under smoothness and symmetry assumptions for the interaction kernel. We then perform a thorough numerical study in order to compare the three modeling scales and study the different limits in situations not covered by the theory yet. These works provide a better understanding of the link between the micro- meso- and macro- scales for interacting particle systems.

MS70-03 — Spatial phase transition in collective dynamics

SPEAKERS Léo Meyer (University of Vienna)
AUTHORS Léo Meyer (University of Vienna); Pierre Degond (IMT, Université Paul-Sabatier); Sara Merino-Aceituno (University of Vienna)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

I will present the result of a collaboration with Pierre Degond and Sara Merino-Aceituno. We study the emergence of band patterns in the Vicsek model, a minimal agent-based model of alignment dynamics with noise. Agent-based simulations on periodic domains display coexisting ordered (high-density, aligned) bands and disordered (low-density, non-aligned) regions, a phenomenon not explained by classical parameter-driven phase transitions. We review prior kinetic and macroscopic results that identify an spatial phase transition [DFL15] : depending on the local density ρ relative to a critical threshold ρ_c, different PDEs govern different spatial regions—Self-Organized Hydrodynamics (SOH, [DM08]) in the ordered regime ($\rho > \rho_c$) and a degenerate diffusive correction (at order ε) in the disordered regime ($\rho < \rho_c$). Building on this framework, we propose a model that couples the ordered and disordered macroscopic equations to simulate the continuum dynamics [MN11], with the goals of reproducing band formation at the macroscopic level, exploring pattern formation, and connecting the linear stability properties of the coupled model with those of SOH.

References

[DFL15] Pierre Degond, Amic Frouvelle, and Jian-Guo Liu. “Phase Transitions, Hysteresis, and Hyperbolicity for Self-Organized Alignment Dynamics”. In: *Archive for Rational Mechanics and Analysis* 216.1 (Apr. 2015), pp. 63–115. ISSN: 0003-9527, 1432-0673. DOI: 10.1007/s00205-014-0800-7.

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[MN11] Sebastien Motsch and Laurent Navoret. “Numerical Simulations of a Nonconservative Hyperbolic System with Geometric Constraints Describing Swarming Behavior”. In: *Multiscale Modeling & Simulation* 9.3 (July 2011), pp. 1253–1275. ISSN: 1540-3459, 1540-3467. DOI: 10.1137/100794067.

MS70-04 — Emergent modes of collective cell invasion: a multiscale perspective

SPEAKERS Martina Conte (Politecnico di Torino)
AUTHORS Martina Conte (Politecnico di Torino)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Collective cell invasion in many biological systems emerges from the complex interplay between individual cell behaviour and environmental heterogeneity, mediated by processes acting across multiple spatial and temporal scales. Cells continuously sense and respond to external cues, such as chemical gradients or physical constraints, while undergoing intrinsic dynamics including proliferation, adaptation, and phenotypic variability [CL23; CS21]. Understanding how these mechanisms integrate across scales is essential to explain the emergence of coordinated population-level dynamics. In this talk, we propose a novel multiscale modelling framework that connects cell-level responses with macroscopic descriptions of collective invasion. We investigate how external cues influence movement through taxis mechanisms, and how their interplay with internal processes—including cell growth—may shape large-scale behaviour. Particular emphasis is placed on the coupling between scales, showing how the integration of microscopic and macroscopic dynamics can lead to qualitatively distinct collective behaviours. The proposed approach is flexible and can be adapted to various biological contexts, providing insight into how multiple processes combine to regulate migration and organization in complex systems.

References

[CL23] Martina Conte and Nadia Loy. "A non-local kinetic model for cell migration: a study of the interplay between contact guidance and steric hindrance". In: *SIAM Journal on Applied Mathematics* 84.3 (2023), S429–S451.

[CS21] Martina Conte and Christina Surulescu. "Mathematical modeling of glioma invasion: acid- and vasculature mediated go-or-grow dichotomy and the influence of tissue anisotropy". In: *Applied Mathematics and Computation* 407 (2021), p. 126305.

MS86 — Mathematical Modelling and Automatic Treatment of the HPT Complex and Thyroid Diseases

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 15.05 - HS
ORGANISERS Andreas Körner (TU Wien); Markus Reichhartinger (TU Graz)

Minisymposium abstract

The endocrine system is a highly complex feedback loop, making it difficult to model in order to gain insights into biological and medical processes, which are important for basic research in this field, the treatment of diseases and the optimal use of medication. This minisymposium will present methods for mathematically describing the hormonal processes of the Hypothalamus-Pituitary-Thyroid (HPT) axis, thyroid-related diseases and the interaction between the HPT axis and radioimmune therapy. Contributions will cover modelling and analysis of the HPT axis, estimation of hormones, optimal drug dosing and the response of thyroid disease to radioiodine therapy. Contributions range from modelling to supporting the treatment of thyroid diseases and cancer. Some contributions deal with mathematical modelling, model analysis and model calibration using data, while others are related to controlling optima in relation to drug dosing and cancer therapy.

MS86-01 — Data-Based Calibration and Fast–Slow Decomposition of a Patient-Specific Hypothalamus–Pituitary–Thyroid Axis Model

SPEAKERS Clara Horvath (TU Wien)
AUTHORS Andreas Körner (TU Wien); Clara Horvath (TU Wien); Marie-Sophie Kohlmayer (TU Wien)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The hypothalamic-pituitary-thyroid (HPT) axis regulates endocrine feedback mechanisms essential for metabolic homeostasis. Mathematical models based on ordinary differential equations provide a framework

for studying hormonal regulations. Nevertheless, the integration of physiological models with patient-specific clinical data continues to present significant challenges.

In this study, a dynamic model of the HPT axis [PMB14] for the simulation of hypothyroidism is calibrated using clinical hormone measurements and analyzed with respect to its intrinsic fast–slow structure [Kue15]. The estimation of model parameters is achieved through the utilization of multiple objective functions, whose outcomes are subsequently compared to align simulated hormone dynamics with clinical data from the General Hospital of Vienna.

The calibrated system is studied using methods from singular perturbation theory [LW26]. The separation of physiological time scales can be exploited through the implementation of a fast-slow decomposition, which results in a reduced model. We examine whether this reduced formulation preserves the essential regulatory dynamics consistent with the Tikhonov–Fenichel theory [Tik52].

The reduced model reveals the dynamical organization of endocrine feedback mechanisms and thereby facilitates the analysis of stability and long-term behavior. Future work includes bifurcation analysis within the parameter ranges obtained from the data-driven calibration.

References

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[LW26] Timothy Earl Figueroa Lapuz and Martin Wechselberger. *Coordinate-independent model reductions of chemical reaction networks based on geometric singular perturbation theory*. 2026. arXiv: 2508.03304 [math.DS]. URL: https://arxiv.org/abs/2508.03304.

[PMB14] Balamurugan Pandiyan, Stephen J. Merrill, and Salvatore Benvenga. "A patient-specific model of the negative-feedback control of the hypothalamus-pituitary-thyroid (HPT) axis in autoimmune (Hashimoto's) thyroiditis." In: *Mathematical medicine and biology: a journal of the IMA* 31 3 (2014), pp. 226–58.

[Tik52] A. N. Tikhonov. "Systems of Differential Equations Containing a Small Parameter Multiplying the Derivative". In: *Matematicheskii Sbornik* 31 (1952), pp. 575–586.

MS86-02 — Mathematical modelling of thyroid response to incidental radiotherapy exposure

SPEAKERS Isabel Gonzalez Crespo (TU Wien)
AUTHORS Isabel Gonzalez Crespo (TU Wien)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The thyroid gland is an organ at risk (OAR) during head-and-neck radiotherapy (RT) due to its proximity to the treatment field. Ionising radiation can damage thyroid tissue, disrupt hormonal regulation by the hypothalamus-pituitary-thyroid (HPT) axis, and lead to endocrine dysfunction. Clinical studies report that 40-50% of patients develop hypothyroidism as a late RT-induced toxicity [Roo+23]. In nasopharyngeal carcinoma (NPC) patients, hypothyroidism peaks 24 months post-RT, coinciding with a reduction of 40% in thyroid volume that subsequently stabilises [Lin+18].

This work presents a mechanistic model of ordinary differential equations (ODEs) to simulate RT-induced changes in thyroid volume and T4-TSH dynamics. Thyroid volume reduction is modelled as a dose-dependent fraction of the initial volume derived from the radiobiological linear-quadratic (LQ) formalism. The HPT feedback loop is described using Michaelis-Menten kinetics, with T4 production expressed as a function of thyroid volume [PMB14]. Model parameters were calibrated using longitudinal clinical data from NPC patients [Lin+18].

The model reproduces observed thyroid volume reduction and long-term T4-TSH clinical levels. It provides a baseline for developing a predictive tool to estimate patient-specific risk of RT-induced

hypothyroidism. Future work will focus on validating the model and evaluating its predictive performance using individualised patient data.

References

[Lin+18] Zhixiong Lin et al. "Pattern of radiation-induced thyroid gland changes in nasopharyngeal carcinoma patients in 48 months after radiotherapy". In: *PLoS ONE* 13.7 (2018), e0200310. DOI: 10.1371/journal.pone.0200310.

[PMB14] Balamurugan Pandiyan, Stephen J. Merrill, and Salvatore Benvenga. "A patient-specific model of the negative-feedback control of the hypothalamus-pituitary-thyroid (HPT) axis in autoimmune (Hashimoto's) thyroiditis". In: *Mathematical medicine and biology* 31 (2014), pp. 226–58. DOI: 10.1093/imammb/dqt005.

[Roo+23] Michael K. Rooney et al. "Hypothyroidism following radiotherapy for head and neck cancer: A systematic review of the literature and opportunities to improve the therapeutic ratio". In: *Cancers* 15 (2023), p. 4321. DOI: 10.3390/cancers15174321.

MS86-03 — Hormone level estimation in the pituitary-thyroid axis with infrequent sampling

SPEAKERS Seth Siriya (Leibniz University Hannover)

AUTHORS Seth Siriya (Leibniz University Hannover); Tobias M. Wolff (Institute of Automatic Control, Leibniz University, Hannover); Isabelle Krauss (Institute of Automatic Control, Leibniz University, Hannover); Victor G. Lopez (Institute of Automatic Control, Leibniz University, Hannover); Matthias Müller (Institute of Automatic Control, Leibniz University, Hannover)

SCHEDULE Monday, 13 July 2026; 11:20–11:40; 15.05 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Control techniques that rely on a known model of the hypothalamic-pituitary-thyroid axis, such as model predictive control, have recently been explored for systematically recommending medication dosages to treat thyroid diseases like hypo- and hyperthyroidism [WDM22; Sha+24; Wol+25]. When implemented based on a high-fidelity model, they require knowledge of internal hormone concentrations of thyroid stimulating hormone, thyroxine, and triiodothyronine inside the pituitary and thyroid glands [WDM22; Wol+25]. However, only peripheral concentrations can be measured from blood tests, often at irregular and sparse sampling times, preventing the direct use of these techniques. We address this by implementing a sample-based moving horizon estimation scheme for the HPT axis based on [KLM25], that estimates the internal concentrations from irregular measurements of peripheral concentrations. Robust stability of the estimator is demonstrated across multiple sampling schemes in simulation. Frequent sampling allows the tracking of intraday behavior and hence better estimation performance, but less-frequent sampling still captures slower behavior which is clinically relevant.

References

[KLM25] Isabelle Krauss, Victor G. Lopez, and Matthias A. Müller. "Sample-based moving horizon estimation". In: (2025). DOI: 10.48550/ARXIV.2510.24191. URL: https://arxiv.org/abs/2510.24191.

[Sha+24] Ravi Sharma et al. "Model predictive controller-based levothyroxine dosing strategy for patients suffering from hashimoto's thyroiditis". en. In: *IFAC-PapersOnLine* 58.24 (2024), pp. 275–280. ISSN: 24058963. DOI: 10.1016/j.ifacol.2024.11.049. URL: https://linkinghub.elsevier.com/retrieve/pii/S2405896324021761.

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[Wol+25] Tobias M. Wolff et al. “Modeling and predictive control for the treatment of hyperthyroidism”. en. In: *IFAC-PapersOnLine* 59.1 (2025), pp. 235–240. ISSN: 24058963. DOI: 10.1016/j.ifacol.2025.03.041. URL: https://linkinghub.elsevier.com/retrieve/pii/S2405896325002587.

MS86-04 — Human-Readable Tabular Reinforcement Learning for Dosing Decisions in Graves' Disease

SPEAKERS Thomas Benninger (Graz University of Technology)
AUTHORS Markus Reichhartinger (Graz University of Technology); Thomas Benninger (Graz University of Technology)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Graves' disease is an autoimmune thyroid disorder causing hyperthyroidism, with a lifetime risk of 3% in women and 0.5% in men, see [SH16]. It is commonly treated with antithyroid drugs where current dosing guidelines still provide limited support for optimal dose selection as it is reported e.g. in [Kah+18; Ben+23]. Strong inter-individual variability poses a major challenge for conventional computer-based dosing approaches. Among machine learning paradigms, reinforcement learning is particularly well suited for managing sequential treatment decisions (see e.g. [Jay+24]) as required in Graves' disease. We developed a reinforcement learning based agent, which approximates its policy using a neural network. It was evaluated on a validated simulation platform and outperformed existing algorithmic approaches as well as experienced endocrinologists. Since the decisions of neural network-based agents are opaque to clinicians, we additionally developed fully human-readable, inherently interpretable tabular reinforcement learning agents. Contrary to common expectations, these transparent agents performed better than the neural network–based approach. This result suggests that transparent, tabular reinforcement learning may be applicable to a broader class of cyclic treatment settings in which drug doses are iteratively adjusted based on measurable physiological parameters, as it is the case in Graves' disease.

References

[Ben+23] Thomas Benninger et al. “Hyperthyroidism therapy: What can decision support systems already achieve?” In: *Journal für Klinische Endokrinologie und Stoffwechsel* 16.4 (2023), pp. 122–131. ISSN: 1998-7781.

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MS87 — Uncovering life's equations: hybrid AI for biological dynamics learning

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 15.06 - HS
ORGANISERS Carles Falco (University of Oxford); Rebecca Crossley (University of Oxford)

Minisymposium abstract

Understanding biological systems requires models that are both grounded in empirical observations and mechanistically interpretable. Recent advances in equation learning provide powerful new tools to infer governing equations directly from biological data, bridging modern machine learning with classical mathematical biology. This minisymposium brings together leading and emerging researchers developing state-of-the-art approaches for learning biological dynamics from data. The session will feature

neural-network–based methods, including universal differential equations and biologically-informed or physics-informed neural networks, which enable learning of full dynamical models or unknown components within mechanistic frameworks. Speakers will present recent advances demonstrating how these techniques can uncover interpretable biological structure while maintaining predictive power. Complementing these approaches, regression-based and probabilistic methods – such as SINDy variants and Bayesian equation learning – will be highlighted through work that has delivered pioneering results across cell biology applications. By bringing together researchers at different career stages and showcasing complementary methodologies, this session aims to provide a broad and balanced perspective on current equation-learning techniques in mathematical biology, fostering discussion and future collaborations.

MS87-01 — Data-Driven Modelling of Cell Cycle Regulation

SPEAKERS Luke De Bretton-Gordon (University of Oxford)
AUTHORS Luke De Bretton-Gordon (University of Oxford)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Understanding how local crowding regulates progression through the cell cycle is central to explaining tissue growth and contact inhibition. We develop a novel model of density-dependent cell-cycle progression using Universal Differential Equations (UDEs), in which transition rates between stages of the cell-cycle are represented by neural networks. This approach preserves key mechanistic features, including the Brownian motion of individual cells and conservation of mass between successive cell-cycle phases, while allowing experimental data of expanding epithelial monolayers to determine how local crowding influences progression through the cell cycle.

MS87-02 — Using inference to obtain models for biological systems

SPEAKERS Marta Sales Pardo (Universitat Rovira i Virgili)
AUTHORS Marta Sales Pardo (Universitat Rovira i Virgili)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

I will discuss how to use Bayesian inference approaches to obtain models for biological data in different contexts. In particular, I will discuss two examples: First, I will describe how we can use inference approaches to obtain the underlying differential equations that govern a specific biological process and apply it to the case of bacterial growth. Second, I will describe how we can use inference to develop a statistical generative model for neural connectomes of a developing animal using C. elegans as an example.

MS87-03 — Making PINNs ready for parameter inference from noisy data

SPEAKERS Roman Vetter (ETH Zurich)
AUTHORS Dagmar Iber (ETH Zürich); Marius Almanstötter (ETH Zürich); Roman Vetter (ETH Zurich)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

A common inverse problem in systems biology, biophysics and related disciplines is the inference of model parameters from limited measured data. For cases where the underlying dynamics follow a set of known (or assumed) differential equations, Physics-Informed Neural Networks (PINNs) have been put forward as decent approximators of the inverse equations, offering an alternative way to estimate parameters to classical methods. However, naive application of PINNs for parameter inference suffers from uncontrolled

overfitting and convergence problems when the available data is noisy. Here we show that these problems stem from inadequate loss function design and training. We introduce PINNverse, a recently proposed reinterpretation of the PINN training paradigm as a constrained optimization problem, which overcomes these limitations. PINNverse combines the advantages of PINNs with the Modified Differential Method of Multipliers and enables convergence to any point on the Pareto front. Based on a few classical ODE and PDE problems, we demonstrate that PINNverse accurately infers model parameters even under high levels of noise in sparse data. Finally, some potential future applications are discussed.

MS87-04 — Towards Inferring Biological Mechanisms with Physics-Informed Neural Networks

SPEAKERS William Lavery (Uppsala University)
AUTHORS William Lavery (Uppsala University)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Physics-Informed Neural Networks (PINNs) have developed into a flexible framework for embedding mechanistic knowledge within data-driven models. Our work provides two complementary studies using PC9 lung cancer cell microscopy data. First, we apply a Biologically-Informed Neural Network (BINN) to spatiotemporal (2D+t) data under a reaction–diffusion model, where diffusion and growth are treated as unknown functions of cell density. We augment this architecture with symbolic regression (SR) to recover interpretable analytical expressions for the diffusion and growth functions. Second, we explore Bayesian PINNs for inverse problems with noisy, sparse biological data. We use an ordinary differential equation model of population dynamics and compare fully Bayesian inference using Hamiltonian Monte Carlo with approximate Bayesian approaches. We further examine the universal PINN (UPINN) framework, highlighting trade-offs between flexibility and mechanistic constraints, and propose an iterative UPINN–SR strategy to infer governing structure when prior knowledge is limited. Together, these contributions offer practical, interpretable, and user-friendly workflows for applying physics-informed learning to biological systems.

MS88 — Mathematical modeling to propose optimized immunotherapies for chronic disease

TRACK Immunobiology & Infection
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 02.21 - HS
ORGANISERS Anne Talkington (University at Buffalo); Jordana O'Brien (University at Buffalo)

Minisymposium abstract

We propose a minisymposium highlighting recent advances in mathematical and computational modeling of the immune response to therapy in the context of chronic diseases. Disease-associated chronic antigen exposure induces an immune response characterized by sustained activation followed by progressive functional decline of immune cells. Therapeutic interventions must therefore overcome not only pathological persistence but also a dysregulated immune environment marked by reduced effector function, impaired cytokine secretion, and expanded regulatory or suppressive gene expression. The complexity of this problem makes chronic disease an ideal context for mechanistic and data-driven modeling approaches aimed at exploring and optimizing treatment strategies. The four speakers in our proposed single 80-minute session will present mathematical models built from principles of quantitative systems pharmacology, omics, and systems biology to interrogate the tightly coupled processes that dictate immune dysfunction, treatment efficacy, and disease fate. Topics of discussion will include immune cell activation and exhaustion dynamics, treatment strategies and predictors of outcome, and disease resistance. Additionally, our minisymposium will highlight different modeling strategies employed in computational immunology, including the application of QSP models and hybrid agent-based models.

MS88-01 — Predicting outcomes of combination treatment of Tuberculosis using a multi-scale quantitative systems pharmacology model

SPEAKERS Christian Michael (University of Michigan)
AUTHORS Christian Michael (University of Michigan)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Understanding how to safely shorten antibiotic treatment for tuberculosis (TB), caused by infection with Mycobacterium tuberculosis (Mtb), is a critical step towards eradicating the world's leading cause of death by single infectious pathogen. Many patients need three months of antibiotic treatment or less, though shortening the recommended treatment duration is dangerous as we cannot predict who will have recurrent TB. The first confounding factor is the substantial heterogeneity exhibited during TB, both between and within patients. Second is data paucity, as our understanding must reconcile the limited-resolution human datasets and corresponding experimental murine and non-human primate animal models. To synthesize our knowledge toward more principled predictions of TB treatment outcomes we extended HostSim, our recent whole-host model of Mtb infection and treatment. HostSim bridges datasets for antibiotic treatment of TB and the potential for post-treatment relapse via a detailed representation of within-host pharmacokinetics, pharmacodynamics, and host-immune interactions. We have now added the ability to track in silico recreations of diagnostic tests that clinically and experimentally establish disease states. Our simulations reproduce the relative efficacy of multiple TB treatments, predict regimen-specific rates of misdiagnosed cure, and articulate how experimental and clinical study design may subtly vary what mechanisms underpin post-treatment relapse.

MS88-02 — A mathematical model of TCR-T cell therapy

SPEAKERS Doron Levy (University of Maryland)
AUTHORS Doron Levy (University of Maryland)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Engineered T cell receptor T cells (TCR-T) are intended to drive strong anti-tumor responses upon recognition of the specific cancer antigen, resulting in rapid expansion in the number of TCR-T cells and enhanced cytotoxic functions, causing cancer cell death. Although TCR-T cell therapy against cancers has shown promising results, it remains difficult to predict which patients will benefit from such therapy. We develop a mathematical model to identify mechanisms associated with an insufficient response in a mouse cancer model. Using mathematical modeling, we show that certain parameters, such as increasing the cytotoxicity of effector TCR-T cells and modifying the number of TCR-T cells, play important roles in determining outcomes. This is a joint work with Zuping Wang, Heyrim Cho, Noriko Sato, and Peter Choyke.

MS88-03 — Illuminating antigen presentation dynamics in the pancreatic cancer microenvironment with omics-informed agent-based modeling

SPEAKERS Daniel Bergman (University of Maryland Baltimore)
AUTHORS Daniel Bergman (University of Maryland Baltimore); Jeanette Johnson (University of Maryland)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

By coupling omics-derived cell states to ABMs of PDAC ecosystems, this work develops a new in silico framework that can systematically investigate the implication of candidate antigen presentation and T

cell activation mechanisms in the microenvironment from PDAC spatial multi-omics data, allowing us to better understand the interplay of pro-activation and pro-tolerance signals experienced by T cells in the PDAC microenvironment. This framework supports in silico identification of microenvironmental features that favor control over progression, and allows us to forecast putative outcomes of rational combination therapy strategies in PDAC on a per-tissue basis. In the context of PDAC, this will pave the way for prevention studies in future work adapting this framework to pancreatic precancer and to understanding the sequential microenvironment transformations that enable lesion progression. Moreover, the modeling framework and TME cell types selected here are generic to be used for cell behavior and therapy modeling across tumor atlases, providing a pipeline for modeling antigen presentation and immune recognition in any solid tumor microenvironment.

MS88-04 — Towards Optimization of Monoclonal Antibody Therapeutics Using a 3D Model of Cancer-Immune Interaction

SPEAKERS Jordana O'Brien (University at Buffalo)
AUTHORS Jordana O'Brien (University at Buffalo)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

T cell exhaustion is a dysfunctional state that develops after prolonged antigen exposure, in which T cells progressively lose effector function. Exhaustion is marked by the sustained expression of inhibitory receptors which transmit suppressive signals that limit immune activity. Recent studies indicate that the 24 hour period after initial antigen exposure is a critical window in the determination of T cell fate. Monoclonal therapeutic antibodies that target inhibitory receptors boost immune function by disrupting suppressive signaling pathways and have been shown to aid in the recovery of cytotoxic function. In this project, we explore trajectories of early phase T cell exhaustion and the impact of immune checkpoint intervention within the first 24 hours of antigen exposure by constructing a multiscale, off-lattice 3D model of the tumor microenvironment. We couple an agent-based model of cell–cell interactions with a reaction–diffusion model for antibody transport and track exhausting interactions between cancer and effector T cells. We assess parameter sensitivity by computing first-order and total-order Sobol indices. We highlight key factors that influence T cell migration and progression into an exhaustive state. Our analysis provides insights into how microenvironmental and therapeutic parameters influence T cell function and may inform future modeling strategies for immunotherapy optimization.

MS124 — Advanced Progresses in Population Models Driven by Natural and/or Artificial Intelligence

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Monday, 13 July 2026; 10:40–18:20; 03.01 - HS
ORGANISERS Zhisheng Shuai (University of Central Florida)

Minisymposium abstract

Population models have long been grounded in natural intelligence: the human-driven theoretical frameworks that include nonlinear dynamics, bifurcation theory, PDEs, structured population models, and stochastic processes. These classical approaches remain indispensable for explaining underlying mechanisms, generating deep insight, and ensuring interpretability. In parallel, artificial intelligence provides powerful computational strategies that enable rapid calibration, enhanced forecasting, and the identification of hidden structure in complex datasets.

This minisymposium will explore the synergy between these complementary approaches and highlight recent advances in population modeling arising from both pillars of scientific inquiry. By bringing together these perspectives, the session aims to showcase cutting-edge developments in traditional theory-based

modeling and AI-empowered methodologies, illustrating how their integration drives new understanding of population dynamics in ecology, epidemiology, and evolutionary biology.

MS124-02 — Empirical and mechanistic modeling of SARS-CoV-2 transmission in the United States to design or evaluate and possibly improve mitigation measures

SPEAKERS John Glasser (Emory University)
AUTHORS John Glasser (Emory University)
SCHEDULE Monday, 13 July 2026; 10:40–11:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Effective reproduction numbers, $\mathcal{R}_E$, provide a means of evaluating the effectiveness of interventions to control the transmission of pathogens in host populations. They can be estimated empirically from reported infections via a renewal equation or analytically via next generation matrices derived from mechanistic models. Both approaches have advantages and disadvantages, with one being more amenable to implementation via artificial and other natural intelligence. The main problems with empirical estimates are delayed, under- and biased reporting of infections and absence of heterogeneity, while analytical estimates depend on assumptions, which are too rarely evaluated. The main advantage of mechanistic models is their ability to assist in designing programs for deploying mitigation measures. To compare approaches, we used a metapopulation SEIR model of SARS-CoV-2 transmission and control in the United States that includes relevant biological characteristics of COVID-19 and whose parameters we either estimated from first principles or, with one exception, sourced from the literature. After ensuring that predictions matched accurate independent observations, we compared empirical estimates of $\mathcal{R}_E$ using simulated infections as input time-series to weekly estimates via the next generation matrix. We conclude not only that analytical results from reliable mechanistic models are more informative than empirical ones, but that artificial intelligence is not yet capable of such modeling.

Joint work with Troy Day (Queen's University) and Zhilan Feng (National Science Foundation).

MS124-01 — Long-range forecasting of seasonal influenza vaccine uptake using web search data

SPEAKERS Edward Thommes (University of Guelph and Sanofi)
AUTHORS Edward Thommes (University of Guelph and Sanofi)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The population-level burden of an influenza season depends strongly on the proportion of people vaccinated beforehand. Being able to predict whether a given season is on track to have low, high or average uptake would provide a critical piece of highly actionable intelligence for all stakeholders involved: Governments, public health authorities, healthcare providers, vaccines manufacturers, and not least, the population itself. In particular, sufficiently early advance warning provides the opportunity for interventions—such as increased investment in awareness programs—to proactively boost participation and avert a projected shortfall. We present an ensemble forecast model which utilizes a panel of Google web search queries to make meaningful predictions about US national-level total seasonal vaccine uptake as early as the beginning of the year in which the season begins.

This work was supported by a National Sciences and Engineering Research Council (NSERC) Alliance grant co-funded by Sanofi.

EWT is an employee of Sanofi and may hold shares and/or stock options.

MS124-04 — Modelling disease transmission with game theoretic determinants: a look at MPOX modelling

SPEAKERS Monica Cojocaru (University of Guelph)
AUTHORS Monica Cojocaru (University of Guelph)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Mathematical modelling is a valuable tool in assessing disease dynamics and interventions. Models may be deterministic, stochastic, data driven, network-based, or hybrid, combining multiple approaches. Advances in computing power, data collection, and simulation frameworks have made it possible to create dynamic models that integrate individual behaviour, environmental context and policy scenarios. The agent-based framework was chosen in this work because of its ability to simulate person-to-person behavioural characteristics, personality traits, and individual decision-making.

For a disease such as MPOX, with a strong behavioural component influencing transmission dynamics, we investigate the possible transmission from a small subpopulation to a larger one, by accounting for differing individual decisions leading to differentiated contacts among population members. We then employ calibration methods and scenario testing to investigate the cross-over spread of the disease and possible control strategies. The talk will illustrate our ideas in the case of MPOX transmission with a signaling game.

This talk is joint work with: Bridgette Amoako, Ed Thommes.

MS124-05 — Integrating Machine Learning Techniques in Mathematical Models of Dengue Transmission Dynamics

SPEAKERS Naveen Vaidya (San Diego State University)
AUTHORS Naveen Vaidya (San Diego State University)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

About half of the world's population, about 4 billion people, live in areas with a risk of dengue infection. Recent evolutionary adaptations of dengue-transmitting mosquitoes to colder regions, such as the Himalayas of Nepal, have raised severe public health concerns about dengue pandemics. In this talk, I will demonstrate how machine-learning techniques and mathematical models can be combined to develop valuable tools for describing dengue transmission dynamics. Using dengue and climate data from Nepal and Taiwan, I will present methods for computing dengue-virus transmission reproduction numbers using mathematical models integrated with machine learning. Our methods provide a valuable approach to combining mathematical models and real-time data in a machine-learning framework to identify effective strategies for preventing dengue outbreaks.

MS124-06 — A dimension-reduction technique for modelling evolution in structured populations

SPEAKERS Troy Day (Queen's University)
AUTHORS Troy Day (Queen's University)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Natural populations exhibit complex class structures that shape evolutionary trajectories. While evolutionary demography provides a formal framework to predict adaptation using invasion fitness, the high mathematical dimensionality of these models often precludes interpretable analytical solutions. We introduce two complementary tools to simplify complex life cycles. First, we formulate the 'invasion

determinant,' an algebraic method that yields a direct scalar condition for mutant invasion. Second, we develop the Projected Next-Generation Matrix (PNGM), which structurally compresses life-cycle graphs by eliminating secondary classes. We demonstrate that this reduction is mathematically equivalent to separating dynamical timescales, explicitly preserving Fisher's reproductive values for the retained focal classes. We will illustrate with diverse ecological examples.

Joint work with Ryosuke Iritani.

MS124-07 — Modeling the invasion of novel SARS-CoV-2 variants and their co-existence with or replacement of ancestral variants in the United States

SPEAKERS Zhilan Feng (National Science Foundation)
AUTHORS Zhilan Feng (National Science Foundation)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

To better understand SARS-CoV-2 variant succession during the COVID-19 pandemic, we developed multi-variant transmission models, derived conditions for novel variants to invade and coexist with or replace ancestral ones, and explored phenomena that might explain observed patterns. To invade, novel variants require reproduction numbers greater than unity when ancestral ones are at their endemic equilibria. Replacement occurs when one variant can invade at another's endemic equilibrium, but not vice versa, and coexistence occurs when both variants can invade at the other's endemic equilibrium. As transitions among successive Omicron variants almost certainly involved immune escape, we explored three hypotheses for the transitions from Alpha to Beta and Beta to Omicron, greater reproduction numbers, shorter generation times, and immune escape. We found that, while greater reproduction numbers always are advantageous, Beta may also have had a shorter generation time than Alpha by virtue of infecting cells in the upper versus lower respiratory tract. But neither was advantageous while the vaccination of healthcare and other essential workers reduced transmission, nor was immune escape advantageous until susceptible hosts were limiting. Consequently, whether replacement of Beta by Omicron was caused or facilitated by immune escape is unclear. Developing, evaluating, and improving these models increased our understanding of phenomena affecting transitions among SARS-CoV-2 variants.

Joint work with Troy Day (Queen's University) and John W Glasser (Emory University).

MS124-08 — Waning immunity and the critical vaccination threshold

SPEAKERS Jane Heffernan (York University)
AUTHORS Jane Heffernan (York University)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Measles is currently affecting many countries globally. In recent studies we have shown that measles vaccine induced immunity can wane over time. In this talk we will determine the control reproduction number and the critical vaccination threshold under these conditions. We will then present a case study for measles infection in Ontario, Canada and determine the effective size of the population of the outbreak.

MS124-09 — Spatial Pattern Formation and the Evolution of Cooperative Behavior

SPEAKERS Daniel Cooney (University of Illinois Urbana-Champaign)
AUTHORS Daniel Cooney (University of Illinois Urbana-Champaign)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Social dilemmas featuring tension between the individual incentive to cheat and a collective goal to maintain cooperative behavior arise across a range of natural and social systems, from the origins of multicellular life to the sustainable manage of shared natural resources. Evolutionary game theory provides a helpful analytical framework for describing this conflict between individual and collective interests, exploring mechanisms that can help the emergence of cooperative behaviors. In this talk, we discuss several PDE models for evolutionary games featuring diffusion of individuals and directed motion towards either increasing payoff or improved environmental quality. We show that biased motion of cooperators can promote the formation of spatial patterns featuring regions with greater population density and increased average payoffs and environmental quality in regions in which cooperators have aggregated. However, by measuring the average payoff of the population or the average level of environmental quality across the population, we see that these pattern-forming mechanisms can actually decrease the overall success of the population, relative to the equilibrium outcome in the absence of spatial motion. This suggests that payoff-driven and environmental-driven motion can produce a kind of spatial social dilemma, in which biased motions towards more beneficial regions can produce emergent patterns featuring a worse overall environment for the population.

MS124-10 — Rotating waves driven by asymmetric cognitive map in nonlocal aggregation-diffusion model driven by spatial cognitive map

SPEAKERS Junping Shi (College of William & Mary)
AUTHORS Junping Shi (College of William & Mary)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Nonlocal aggregation-diffusion models, when coupled with a spatial map, can capture cognitive and memory-based influences on animal movement and population-level patterns. A reaction-diffusion-aggregation system coupled with a separate dynamically updating map is proposed to describe the animal population movement. We show that when an asymmetric cognitive map influences instantaneously, a rotating movement pattern emerges.

MS124-11 — Are the mathematical biological models governed by nonlinear first order Caputo fractional differential equations solvable?

SPEAKERS Kunquan Lan (Toronto Metropolitan University)
AUTHORS Kunquan Lan (Toronto Metropolitan University)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Consider the first order Caputo fractional differential equation (FDE)

$$(D^{1-\alpha}_{C,a^+}u)(x) := (I^{\alpha}_{a^+}u')(x) = f(x,u(x)) \quad \text{for almost every } x \in [a,b],$$

where $\alpha \in (0,1)$, $I^{\alpha}_{a^+}$ is the Riemann-Liouville fractional integral, u' is the traditional first-order derivative and $f : [a,b] \times [0,\infty) \to \mathbb{R}$ is a function. The Caputo FDE can be a single equation or a system of equations.

It was claimed in some previous papers that if f satisfies the locally Lipschitz condition in the second variable, then the Caputo FDE has a unique solution. However, the result would be incorrect, see the open question below.

The above result has been widely used in the literature to obtain the existence and uniqueness of solutions of a variety of models such as disease models and predator-prey models published, for example in *Scientific

Reports*, *PLoS One*, *Epidemics*, *Communications in Nonlinear Science and Numerical Simulation*. However, these previous results which applied the above claimed result to obtain the existence and uniqueness of solutions of the models would not be correct unless one can prove that the locally Lipschitz condition implies the necessary condition for the Caputo FDE to have solutions:

$$Fu \in I_{a^+}^{\alpha}(L^1[a,b]) \quad \text{for all } u \in S,$$

where S is a ball in $C_+[a,b]$ and $(Fu)(x) = f(x,u(x))$ for almost every $x \in [a,b]$.

The open question is under what conditions on the nonlinearity f , does the above necessary condition hold?

It is noted that if the nonlinearity f satisfies the locally Lipschitz condition in the second variable or is infinitely differentiable, it is unknown whether f satisfies the necessary condition.

MS124-12 — Evolutionary Escape in Antibiotic Cycling Therapies

SPEAKERS Esteban Hernandez Vargas (University of Idaho)
AUTHORS Esteban Hernandez Vargas (University of Idaho)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Collateral sensitivity, where resistance to one antibiotic induces increased sensitivity to another, has been proposed as a strategy to combat antimicrobial resistance. Predicting the evolutionary outcomes of sequential antibiotic exposure, however, remains a major challenge. In this work we develop a mathematical framework that integrates collateral sensitivity networks with switched dynamical systems describing bacterial population dynamics. Within this framework, antibiotic therapies correspond to switching policies acting on phenotype populations.

Using invariant sets and network theory, we characterize therapeutic windows in which treatment schedules can maintain the infection within controllable regions of the state space. The approach embeds collateral interactions into a network of switched systems and provides a computational algorithm to identify effective sequential therapies. By leveraging experimental interaction data for *Pseudomonas aeruginosa*, the model predicts scenarios in which antibiotic cycling prevents evolutionary escape and maintains the population within a bounded containment region.

These results provide a control-theoretic interpretation of evolutionary therapy and illustrate how invariant-set analysis can guide the design of sequential treatment strategies to mitigate antimicrobial resistance.

MS127 — Vertex Models in Tissue Mechanics and Morphogenesis: Bridging Cell-Scale Forces to Multicellular Dynamics

TRACK Cellular and Developmental Biology
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 02.23 - HS
ORGANISERS Natalia Briñas-Pascual (Universidad Carlos III de Madrid); Pilar Guerrero (Universidad Carlos III de Madrid)

Minisymposium abstract

Vertex models have emerged as a central computational and theoretical framework for understanding how mechanical interactions at the level of individual cells give rise to collective behaviours in developing and homeostatic tissues [Bri+24; AGS17]. In these models, epithelial and other confluent tissues are represented as tilings of polygons or polyhedra whose vertices encode cell–cell interfaces, tensions, and forces, enabling mechanistic investigation of key biological processes such as force-mediated cell rearrangements, rigidity transitions, tissue fluidity, and morphogenetic shape changes [Bi+14]. Recent advances have extended classical 2D vertex models to three-dimensional geometries, incorporated mechanochemical feedback and

active cellular processes, and developed statistical inference strategies linking model predictions with experimental data. Applications range from embryonic development and wound healing to cancer invasion and organoid dynamics, highlighting both the strengths and limitations of current formulations.

This minisymposium invites contributions on theoretical developments, computational innovations, and applications of vertex models to complex biological phenomena, with emphasis on mechanical principles, multiscale integration, and quantitative comparison with experimental systems. By stimulating dialogue across disciplines, we aim to chart future directions for vertex-based modelling in mathematical and theoretical biology.

References

[AGS17] Silvanus Alt, Poulami Ganguly, and Guillaume Salbreux. "Vertex models: From cell mechanics to tissue morphogenesis". In: *Philosophical Transactions of the Royal Society B: Biological Sciences* 372 (Mar. 2017), p. 20150520. DOI: 10.1098/rstb.2015.0520.

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[Bri+24] Natalia Briñas-Pascual et al. "No Country for Old Frameworks? Vertex Models and Their Ongoing Reinvention to Study Tissue Dynamics". In: *Biophysica* 4 (Nov. 2024), pp. 586–603. DOI: 10.3390/biophysica4040039.

MS127-01 — Mechanosensitive Feedback Organizes Cell Shape and Motion During Hindbrain Neuropore Morphogenesis

SPEAKERS Fernanda Pérez Verdugo (Institute of Science and Technology Austria)

AUTHORS Eirini Maniou (Developmental Biology and Cancer Department, UCL GOS Institute of Child Health, 30 Guilford Street, London, WC1N 1EH, UK); Fernanda Pérez Verdugo (Institute of Science and Technology Austria); Gabriel L. Galea (Developmental Biology and Cancer Department, UCL GOS Institute of Child Health, 30 Guilford Street, London, WC1N 1EH, UK); Shiladitya Banerjee (Department of Physics, Carnegie Mellon University, Pittsburgh, PA, USA)

SCHEDULE Monday, 13 July 2026; 10:40–11:00; 02.23 - HS

TRACK Cellular and Developmental Biology

Abstract

Neural tube closure in mammals requires the sealing of the hindbrain neuropore (HNP) gap. Failure in this critical process results in fatal birth defects. Yet, the physical forces orchestrating this morphogenetic event at the cellular and tissue level have remained elusive. Here, we combine live and fixed imaging of mouse embryos with cell-based computational modeling to investigate the mechanisms underlying mouse HNP closure. We find that two force-generating mechanisms drive HNP closure: a high-tension actomyosin purse-string acting along the gap edge and directional cell migration. While together these reproduce gap-level dynamics, they are insufficient to explain the reproducible pattern of cell elongation along the gap border. We show that a mechanical feedback loop between shear stress and cytoskeletal organization, where the purse-string serves as a mechanical cue, generates the observed morphological pattern around the gap. Over time, cell neighbor exchanges stall and the tissue solidifies, helping preserve cell shapes and their relative arrangements even away from the high-tension cue. This induces mechanical memory, leading to rostro-caudal midline cell elongation, consistent with observations in both the cranial and spinal mouse regions. We validate this feedback model by comparing mouse to chick embryos, which naturally lack the purse-string.

References

[Man+21] Eirini Maniou et al. "Hindbrain neuropore tissue geometry determines asymmetric cell-mediated closure dynamics in mouse embryos". en. In: *Proceedings of the National Academy of Sciences* 118.19 (May 2021), e2023163118. ISSN: 0027-8424, 1091-6490. DOI: 10.1073/pnas.2023163118. URL: https://pnas.org/doi/full/10.1073/pnas.2023163118 (visited on 05/11/2026).

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MS127-02 — Linking vertex models with experiments in tissue morphogenesis

SPEAKERS Gonca Erdemci-Tandogan (Western University)
AUTHORS Gonca Erdemci-Tandogan (Western University)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Collective cell movements play a critical role in guiding embryonic development, wound repair, and disease progression, such as cancer metastasis. The coordination of these movements is strongly influenced by mechanical forces. Biological tissues can be viewed as soft, out-of-equilibrium systems whose constituent cells continuously generate forces and undergo rearrangements. During development, tissue material properties can change drastically, reminiscent of rigidity transitions in physics. Measuring the impact of these transitions on cell behaviour, or identifying how to control them, remains experimentally challenging in developing organisms. In this talk, I will present our work developing vertex models in close collaboration with experimentalists, where model predictions and in vivo measurements inform one another iteratively. By integrating experimental measurements into our models and generating testable predictions, we investigate the interplay between tissue material properties, cell-scale forces, and tissue boundary formation across a range of morphogenetic processes. Together, these efforts yield mechanistic insights with implications for understanding congenital disorders and cancer.

MS127-03 — Cell-cell heterogeneities in vertex models for tissue mechanics

SPEAKERS Oliver Drozdowski (Institute of Science and Technology Austria, Klosterneuburg, Austria)
AUTHORS Oliver Drozdowski (Institute of Science and Technology Austria, Klosterneuburg, Austria)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Vertex models have been incredibly successful in describing the mechanics of epithelia. However, many models do not systematically consider heterogeneities in the cellular mechanical and biochemical properties. Using three-dimensional force inference in intestinal organoids, we show that cells indeed have inhomogeneous tissue tensions. In particular, outer basal surface tensions at topological defects are increased in organoids to stabilize cell shapes against mechanical instabilities. Using a three-dimensional bubbly vertex model, we show how such geometry-dependent tensions—which could arise from mechanosensitive feedback—allow for ideal cell shape stabilization superior to other tissue-scale strategies [DS24]. Further, incorporating into the vertex model both such mechanosensation through intracellular mechanochemical coupling and cellular heterogeneities, we uncover the complex modulation of mechanochemical waves in two-dimensional epithelia. Consequently, our work underlines the importance of moving beyond homogeneous mean-field descriptions of tissues.

References

[DS24] Oliver Drozdowski and Ulrich Schwarz. “Cell bulging and extrusion in a three-dimensional bubbly vertex model for curved epithelial sheets”. In: (Nov. 2024). DOI: 10.48550/arXiv.2411.07141.

MS127-04 — A quantitative vertex model of neural tube closure informed by imaging data

SPEAKERS Rubén Perez-Carrasco (Imperial College London)
AUTHORS Gabriel L. Galea (Developmental Biology and Cancer Department, UCL GOS Institute of Child Health, 30 Guilford Street, London, WC1N 1EH, UK); Natalia Briñas-Pascual (Universidad Carlos III de Madrid); Rubén Perez-Carrasco (Imperial College London)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Tissue morphogenesis emerges from the collective mechanical behaviour of epithelial cells, where gene expression, cytoskeletal dynamics and cell cycle progression combine to drive coordinated tissue-scale deformations. Neural tube closure is a paradigmatic example whose failure gives rise to severe congenital conditions including spina bifida. Yet quantitative links between gene-level perturbations, single-cell mechanics and tissue-scale closure outcomes remain missing. Here we develop a data-driven vertex model that bridges these scales, parameterised directly from quantitative imaging across developmental stages. Incorporating cell cycle dynamics and apical constriction as mechanistically grounded inputs, the model recapitulates tissue-scale deformation during closure and correctly predicts neuropore widening under pharmacological inhibition of contractility.

This provides a framework in which genetic perturbations altering cytoskeletal tension or cell cycle progression can be mapped directly onto closure outcomes offering a quantitative route to understanding how mutations disrupting neuroepithelial mechanics lead to neural tube defects.

MS145 — Cell Migration Across Scales – exploring different mathematical frameworks

TRACK Cellular and Developmental Biology
SCHEDULE Monday, 13 July 2026; 10:40–16:20; 01.22 - HS
ORGANISERS Annachiara Colombi (Politecnico di Torino)

Minisymposium abstract

Cell migration plays a central role in key physiological and pathological contexts, including embryonic development, immune responses, tissue repair, and tumour invasion. It arises from the coordinated interplay of mechanisms acting across multiple spatial and temporal scales: from subcellular events - cytoskeletal remodelling, nuclear deformation, polarization, intracellular signalling - to cell-cell and cell-matrix interactions, and collective behaviours at tissue or population levels.

The multiscale nature of migration poses challenges for quantitative understanding and predictive modelling. Mathematical models can integrate experimental data, test biological hypotheses, and reveal emergent properties inaccessible to single-scale descriptions. A wide variety of frameworks – discrete, continuum and hybrid - has been developed, each capturing complementary aspects of migratory behaviour and enabling the study of phenomena ranging from intracellular force generation to large-scale pattern formation and collective motion.

This mini-symposium brings together contributions on mathematical modelling for cell migration across subcellular, cellular, and population scales, focusing on different aspects of the biological process. The session will provide an integrated overview of current modelling strategies and foster discussion on how multiscale frameworks can enhance our understanding of cell migration in complex biological systems.

MS145-01 — From Cell Alignment to Axonal Pathfinding: Mechanics as a Guidance Cue

SPEAKERS Chiara Giverso (Politecnico di Torino)
AUTHORS Annachiara Colombi (Politecnico di Torino); Chiara Giverso (Politecnico di Torino); Giulio Lucci (Sapienza University of Rome); Luigi Preziosi (Politecnico di Torino)
SCHEDULE Monday, 13 July 2026; 10:40–11:20; 01.22 - HS
TRACK Cellular and Developmental Biology

Abstract

Cells are highly sensitive to mechanical cues in their microenvironment, which play a fundamental role in regulating orientation, cytoskeletal organization, and growth dynamics. In particular, cells and neurons seeded on cyclically stretched substrates have been shown to reorient toward well-defined equilibrium angles with respect to the principal strain direction [Giv+23] Cell reorientation can be mathematically described by a linear viscoelastic model that captures the coupled evolution of intracellular stress and orientation under periodic stretching [LGP21]. The model predicts oscillatory reorientation dynamics converging toward a stable equilibrium angle, which can be interpreted as the minimizer of a general orthotropic elastic energy. Bifurcation analysis and numerical simulations reveal a transition in reorientation dynamics governed by the interplay between stretching frequency and the characteristic turnover time of cytoskeletal and adhesion components, with faster reorientation occurring at higher frequencies. Building on this approach, the modeling framework is extended to neurons by coupling a viscoelastic reorientation model of the growth cone with a moving-boundary description of tubulin-driven neuron growth [CBG25]. Numerical simulations across a range of stretching frequencies and strain amplitudes reproduce experimentally observed equilibrium orientations and demonstrate that both the directionality and speed of axonal growth are strongly modulated by mechanical stimulation. In particular, higher stretching frequencies and amplitudes lead to faster reorientation and more directed pathfinding. Overall, the proposed models highlight common physical principles underlying mechanosensitive orientation in cells and neurons and provide a quantitative link between substrate mechanics, viscoelastic cellular response, and long-term growth behavior.

References

[CBG25] Annachiara Colombi, Andrea Battaglia, and Chiara Giverso. “A Mathematical Model for Neuron Reorientation and Axonal Growth on a Cyclically Stretched Substrate”. In: *Studies in Applied Mathematics* 155.3 (2025), e70103.

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MS145-03 — Bayesian parameter inference of stochastic two-population cell movement models with spatial statistics

SPEAKERS Duncan Martinson (The Francis Crick Institute)
AUTHORS Duncan Martinson (The Francis Crick Institute)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 01.22 - HS
TRACK Cellular and Developmental Biology

Abstract

Linking models to experimental data is a common challenge in mathematical biology. Several techniques have been developed to parameterize theoretical frameworks, including maximum likelihood estimation and Approximate Bayesian Computation. Previous work established that combining these approaches with spatial data analysis, specifically with statistics known as pairwise correlation functions (PCFs) that quantify the relative degree of clustering or exclusion among cells across multiple length scales, can recover parameters in models of cell movement in homogeneous populations. Yet it remains unclear whether PCFs are equally useful at parameterizing models in cases with multiple cell types. I will describe a simplistic mathematical framework created to address this gap, which is based on in vitro experiments of immune cell infiltration into tumor-dense space. The model tracks individual cell positions using an overdamped version of Newton’s law, with forces arising from short-range repulsion and long-range attraction between cells. I create multiple synthetic datasets in which tumor and immune cells interact with varying length scales and strengths of attraction, and apply Approximate Bayesian Computation-Sequential Monte Carlo

to determine how well the posterior parameter distribution recovers the "ground truth". I demonstrate that the attractive forces between cells are practically identifiable, even when their underlying parameters may not be.

MS145-04 — A Physiologically Based Pharmacokinetic (PBPK) Model of Whole-Body CAR T-Cell Trafficking in Humans

SPEAKERS Elio Campanile (University of Trento)
AUTHORS Elio Campanile (University of Trento); Federico Reali (Fondazione the Microsoft Research, University of Trento Centre for Computational and Systems Biology, Rovereto, Italy); Soukaina Sabir (UC3M)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 01.22 - HS
TRACK Cellular and Developmental Biology

Abstract

Chimeric antigen receptor (CAR) T-cell therapy has shown remarkable clinical success in the treatment of B-cell malignancies. Numerous mathematical models have been developed to describe the interactions between CAR T cells and malignant B cells at the tumor site [Leó+21; Sab+25]; however, these approaches often neglect the systemic trafficking of CAR T cells across physiological compartments, which plays a crucial role in linking intravenously administered doses to quantitative measurements at the tumor site [Zhu+96]. In this work, we develop a physiologically based pharmacokinetic (PBPK) framework for CAR T-cell trafficking in humans, comprising both a full whole-body model and a sensitivity-analysis-based minimal PBPK model. The full model describes organ-level transport processes, intra-tissue kinetics, and interactions with malignant B cells within lymph nodes [Sab+25], and is used for parameter calibration and validation against clinical data from more than 100 patients. The minimal model is used to perform equilibria and stability analyses, providing insight into biologically uncertain mechanisms such as organ uptake. Our results highlight the importance of migration dynamics for a quantitative understanding of CAR T-cell therapy and support the use of mechanistic whole-body models for dose optimization and personalized treatment planning through simulation-guided approaches.

References

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MS145-02 — Multiscale modelling of cancer invasion and metastasis: the effects of tumour heterogeneity

SPEAKERS Dimitrios Katsaounis (RWTH Aachen University)
AUTHORS Dimitrios Katsaounis (RWTH Aachen University); Mark Chaplain (University of St Andrews); Nikolaos Sfakianakis (University of St Andrews); Pierre Degond (IMT, Université Paul-Sabatier)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 01.22 - HS
TRACK Cellular and Developmental Biology

Abstract

Cancer invasion and metastasis are multiscale processes driven by complex interactions between cancer cells and the tumour microenvironment. A key mechanism underlying cancer heterogeneity is the Epithelial-to-Mesenchymal Transition (EMT), through which proliferative epithelial-like cancer cells (ECCs), forming the bulk of solid tumours, progressively acquire migratory and invasive mesenchymal-like traits. Mesenchymal-like cancer cells (MCCs) can actively invade surrounding tissue and disseminate to distant organs via the vasculature. At secondary sites they may undergo the reverse Mesenchymal-to-Epithelial Transition (MET), enabling metastatic growth. Importantly, EMT is a continuous process that generates intermediate hybrid phenotypes with progressively increasing invasive potential. In this talk, we will explore a variety of models that incorporate phenotypic transitions for cancer invasion across mathematical and biological scales. Firstly, I will introduce a novel 3D hybrid multiscale model which couples individual-based representations of migrating cancer cells with continuum descriptions of tumour growth. The model illustrates how EMT-driven phenotypic changes shape macroscopic invasion patterns within a multi-organ framework. Furthermore, I will present a phenotype-dependent individual based model and its corresponding macroscopic model, which incorporates continuous transitions along the epithelial–mesenchymal spectrum, providing a tractable framework to study the emergence and maintenance of phenotypic heterogeneity in cancer.

MS145-08 — The biomechanics of the single cell-ECM interaction during migration

SPEAKERS Nicolas Louviaux (Université Grenoble Alpes)
AUTHORS Angélique Stéphanou (Univ. Grenoble Alpes, CNRS, UMR 5525, VetAgro Sup, Grenoble INP, TIMC, 38000 Grenoble, France); Arnaud Chauvière (Univ. Grenoble Alpes, CNRS, UMR 5525, VetAgro Sup, Grenoble INP, TIMC, 38000 Grenoble, France); Nicolas Louviaux (Université Grenoble Alpes)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 01.22 - HS
TRACK Cellular and Developmental Biology

Abstract

Cell migration is involved in many developmental process such as embryogenesis, morphogenesis, angiogenesis or cancer invasion and critically depends on the mechanical properties of the extra-cellular matrix (ECM). Indeed, in the mesenchymal migration mode, cells tend to follow ECM stiffness gradient. This durotaxis [Lo+00] is explained by the ability of the cell to probe its environment thanks to mechano-sensors [Plo+12] (filopodia). Moreover, the migration process relies on the ability of the cell to self propel through a combination of events comprising cell anchoring to the ECM through maturation of focal adhesions and contractions of the actin cytoskeleton to generate a traction force strong enough to translocate the cell nucleus. The cell-ECM interactions during cell migration is thus a bidirectionnal process : the cell traction force on the matrix generate ECM deformation that in return can be perceived by neighbouring cells through mechano-sensors. This leads to the question of the existence and importance of inter-cellular communication through matrix deformation to favour cell-cell encouter in processes such as tumoral angiogenesis. In this talk a single cell motility model [Lou+25] mechanically coupled to a continuous deformable substrate will be presented. The model is designed to incorporate minimal a priori assumptions. In that sense, no mechanism of cell polarization nor stiffness dependent traction force is implemented. The main objective is to evaluate if those behaviours can emerge solely from cell-ECM mechanical interactions and probing. Particular attention will be given on the range and magnitude of cell induced ECM deformations that the model can reproduce. Reciprocally the influence of imposed ECM deformations on the migratory patterns will be considered and compared to those obtained on a rigid substrate. In conclusion the effective impact of the mechanical cues on the cell encounter will be assessed.

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MS145-06 — Heterogeneous elastic wires: a toy model for biological membranes

SPEAKERS Gaspard Jankowiak (University of Graz)

AUTHORS Anna Dall'Acqua (University of Ulm); Christian Schmeiser (University of Vienna); Fabian Rupp (University of Vienna); Gaspard Jankowiak (University of Graz); Katharina Brazda (University of Vienna); Leonie Langer (University of Ulm); Ulisse Stefanelli (University of Vienna)

SCHEDULE Monday, 13 July 2026; 15:40–16:00; 01.22 - HS

TRACK Cellular and Developmental Biology

Abstract

Motivated by variational models for heterogeneous biological membranes such as those described by the Canham-Helfrich functional, we study closed planar elastic curves whose bending stiffness depends on an additional scalar density. The resulting energy can be seen as the one-dimensional analogue of 2D membrane models.

Previous work on the static problem include [Bra+23], where the energy minimization under fixed length and total mass constraints is performed, followed by a bifurcation analysis around the trivial circular state is carried out. In [DLR24], the authors study a related time-dependent problem, i.e. the associated L^2-gradient flow. Local well-posedness, global existence, and full convergence of the flow to a stationary solution are established, using a version of the Łojasiewicz–Simon gradient inequality.

This talk focuses on the qualitative properties of the L^2-gradient flow solutions, like the (non)preservation of convexity, embeddedness, positivity of the density, and rotational or axial symmetry of the initial datum. In particular, we identify conditions on the model parameters and the bending stiffness under which the asymptotic limit can be explicitly characterized as a homogeneous elastica, i.e., an elastica with constant density. Some numerical experiments will also be shown.

References

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MS145-07 — Towards optimal scaffolds for cell migration

SPEAKERS Luigi Preziosi (Politecnico di Torino)

AUTHORS Chiara Giverso (Politecnico di Torino); Chiara Lonati (Politecnico di Torino); Luigi Preziosi (Politecnico di Torino); Marco Scianna (Politecnico di Torino)

SCHEDULE Monday, 13 July 2026; 16:00–16:20; 01.22 - HS

TRACK Cellular and Developmental Biology

Abstract

Cell-extracellular matrix interaction and the mechanical properties of the cell nucleus have been demonstrated to play a fundamental role in cell movement across fibrous networks, micro-channels, basal membranes, and confining environment in general. So, their study is important on one hand in oncology to understand the spread of cancer metastases and on the other hand in tissue engineering to build artificial scaffolds that are able to promote cell re-population. It is known that key ingredients are the geometrical properties of the environment, the stiffness of the cell nucleus and the ability of cell to exxert traction forces. This talk will review previous results, merging the outcome of some continuum mechanics models with individual cell-based models that allow to identify an invasion criterium and optimal characteristics for cell migration. Such criteria will be used to identify the geometric characteristics that some triply periodic porous structures need to have to optimize cell migration.

MS146 — Metabolic Foundations of Microbial Community Interactions: From Theory to Practice

TRACK Systems Biology and Biochemical Networks
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 11.03 - HS
ORGANISERS Jürgen Zanghellini (Department of Analytical Chemistry, University of Vienna); Stefan Müller (University of Vienna, Faculty of Mathematics)

Minisymposium abstract

Microbial communities play a central role in ecosystems, biotechnology, and human health. Understanding how these communities grow, interact, and respond to environmental change requires a strong theoretical foundation that can bridge the gap between genome-scale experimental data and community-level behavior. This minisymposium aims to bring together researchers working on the theory of microbial community growth, with an emphasis on metabolic modeling and ecological interaction types, highlighting how theoretical frameworks help interpret large-scale experimental findings and clarify emergent community dynamics.

By fostering dialogue between mathematicians, theoretical biologists, and experimentalists, the minisymposium will showcase recent advances in theory [Mül+26; VAH25], illustrate how metabolic modeling can guide the interpretation of large-scale experimental data [Car+25; Bes+25], and identify open challenges at the interface of applied mathematics and microbiology of communities.

References

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MS146-01 — Microbial community metabolic models of the human gut microbiome: From objectives to validation

SPEAKERS Christian Diener (Diagnostic & Research Institute of Hygiene, Microbiology and Environmental Medicine, Medical University of Graz)
AUTHORS Christian Diener (Diagnostic & Research Institute of Hygiene, Microbiology and Environmental Medicine, Medical University of Graz)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

From the first hours of life until the last, the human body is colonized by microbes that consume and produce a wide array of metabolites, predominantly within the gastrointestinal tract. These microbial metabolites are intimately linked to inflammation and immune signaling. Molecules such as short-chain fatty acids (SCFAs) and indoles have been shown to attenuate host inflammatory responses, making the gut microbiome a promising target for clinical interventions. However, metabolic output within microbial communities results from a complex interplay between hundreds of species, host factors, and dietary intake. Consequently, metabolic function cannot be accurately predicted from microbiome composition alone. Microbial Community Metabolic Models (MCMMs) offer a mechanistic solution to this challenge, yet their utility has been hindered by poorly defined optimization objectives and a lack of robust validation. Here, we demonstrate that ecological two-step objectives significantly enhance the predictive accuracy of MCMMs, rendering them suitable for clinical intervention modeling. We validate our flux predictions using large-scale ex vivo microbiome cultivation and engraftment studies. Our results establish MCMMs as a potent, mechanism-based strategy for predicting personalized intervention effects within the human gut microbiome.

MS146-02 — Thermodynamic Feasibility in Genome-Scale Microbial Consortia

SPEAKERS Jürgen Zanghellini (Department of Analytical Chemistry, University of Vienna)
AUTHORS Jürgen Zanghellini (Department of Analytical Chemistry, University of Vienna)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Systematically mapping species interactions in microbial communities remains a central challenge. Community-level elementary flux modes (cEFMs) provide a complete description of community metabolism, but are intractable at genome-scale, necessitating optimization-based approaches. However, the latter introduce thermodynamically infeasible cycles (TICs) across organisms, leading to spurious cross-feeding predictions.

Here, we show that blocking external metabolite inflow allows cEFMs to fully characterize all TICs in a community model. We leverage this insight to derive simple activity-based constraints that yield cycle-free solutions at genome-scale, when integrated into a mixed-integer linear program.

The resulting computational framework links metabolic function, species interactions, and thermodynamic feasibility for the analysis and design of microbial consortia.

MS146-03 — Elementary vectors reveal minimal interactions in microbial communities

SPEAKERS Stefan Müller (University of Vienna, Faculty of Mathematics)
AUTHORS Jürgen Zanghellini (Department of Analytical Chemistry, University of Vienna); Stefan Müller (University of Vienna, Faculty of Mathematics)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Understanding microbial communities is crucial for advancing fields like ecology, biotechnology, and human health. Recently, constraint-based metabolic models of individual organisms have been combined in various ways to study microbial consortia. In this work, we present a comprehensive geometric approach to characterize all feasible microbial interactions. First, we project community models onto the relevant variables for interaction, namely exchange fluxes and community compositions. Next, we compute 'elementary' compositions/exchange fluxes, thereby extending the concept of minimal metabolic pathways from single species to entire communities. Every feasible community is a combination of these elementary compositions/exchange fluxes, and, surprisingly, every elementary vector represents a fundamental ecological interaction (such as specialization, commensalism, or mutualism). Hence, our geometric approach allows us to decode the metabolic interactions underlying microbial cooperation. Moreover, it provides a foundation for rational community design. Since it treats exchange fluxes and community compositions equally, we can directly apply existing constraint-based methods and algorithms.

MS146-04 — Automated Discovery of Mechanistic Models for Microbial Communities

SPEAKERS Artai Rodríguez Moimenta (Biosystems and Bioprocess Engineering Bio2Eng (iim-CSIC))
AUTHORS Artai Rodríguez Moimenta (Biosystems and Bioprocess Engineering Bio2Eng (iim-CSIC)); Oana Teodora Chis (Biosystems & Bioprocess Engineering Bio2Eng (iim-CSIC)); Patricia González Gómez (Universidad de A Coruña (UDC)); Eva Balsa Canto (Biosystems & Bioprocess Engineering Bio2Eng (CSIC))
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 11.03 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Microbial communities play key roles in biotechnology, environmental systems, and human health. Understanding and predicting the dynamics of interacting microbial populations is essential for both advancing fundamental knowledge and enabling the rational design and optimization of these systems. Mechanistic models provide a powerful framework for this purpose, enabling hypothesis testing and predictive simulation and control. However, formulating such models remains challenging due to biological complexity, limited experimental data, and the number of possible interaction mechanisms.

We present a framework to automate the formulation of dynamic mechanistic models. The approach considers multiple formulations compatible with the observed microbial community behavior enabling data fitting and discrimination among candidate structures by solving a mixed-integer dynamic optimization (MIDO) problem.

The framework is illustrated through a case study involving a microbial community of three interacting species under realistic time-resolved experimental data. A grammar of mechanisms describing bacterial growth, inhibition, and substrate consumption is used to generate alternative model structures. The MIDO problem is solved using a self-adaptive cooperative multimethod algorithm. Results show the method successfully recovers the true system even in the presence of experimental noise, highlighting its potential as a scalable tool for automated model discovery in complex systems.

MS163 — Modelling Ocular Disease: From Mechanisms to Treatment

TRACK Cutting Edge Research Areas
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 11.01 - HS
ORGANISERS Jessica Crawshaw (Queensland university of Technology); Neda Khodabakhsh Joniani (The University of Sydney)

Minisymposium abstract

Vision loss and ocular disease constitute major global health challenges. In recent years, mathematical ophthalmology has experienced substantial growth and increasing acceptance within the clinical and biomedical communities, driven by advances in ocular imaging, experimental measurement, and the availability of high-quality clinical and preclinical data.

This mini-symposium will highlight recent mathematical advances in understanding ocular disease mechanisms and progression, as well as developments in modelling approaches that inform and optimise treatment strategies. Ocular disease involves inherently multi-physics processes, and consequently, its modelling spans a broad range of mathematical methodologies. Contributions will include, but will not be limited to, continuum and discrete frameworks, multiscale and multi-physics approaches, and data-informed models. These methods will be applied across diverse problem areas in ophthalmology, including ocular biomechanics, fluid dynamics, electrophysiology, disease progression, and drug delivery within the eye.

By bringing together researchers working across a range of ocular diseases, the mini-symposium aims to foster focused discussion and cross-fertilisation of ideas, provide a forum for the presentation of recent research to the mathematical biology community, and highlight emerging directions in the field. The session will also support the development of new collaborations.

MS163-01 — The whole baby PK/PD model of anti-VEGF therapy that big pharma doesn't want you to see

SPEAKERS Jessica Crawshaw (Queensland university of Technology)
AUTHORS Jessica Crawshaw (Queensland university of Technology)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 11.01 - HS
TRACK Cutting Edge Research Areas

Abstract

Anti-VEGF therapy has recently been approved by the FDA to prevent the progression of retinopathy of prematurity (ROP; a leading cause of childhood blindness) and preserve vision in preterm newborns. Following injection, it is known that these drugs leave the eye and enter the systemic circulation; however, from there, the distribution of these drugs to the developing organs is unknown. Whilst uptake of these growth-suppressing drugs into the developing organs is denied by the financially interested parties, emerging evidence from animal studies shows that anti-VEGF agents enter the brain, lungs, and heart, and restrict the growth of these organs. Clinicians currently have no quantitative way to predict these risks or adjust treatment accordingly, let alone make an informed decision on whether anti-VEGF therapy is the best treatment option.

In this talk, I will present a data-driven organ-level pharmacokinetic/pharmacodynamic model of intravitreal aflibercept therapy in growing preterm newborns with ROP to predict the drug load experienced by key developing organs. I use a Bayesian approach to validate the model against clinical data and predict a physiologically plausible parameter space in preterm newborns. We then use this model to predict guild dosing, timing and monitoring.

MS163-03 — How Mechanical Forces Shape Stratification and Wound Healing Dynamics in the Corneal Epithelium

SPEAKERS Neda Khodabakhsh Joniani (The University of Sydney)
AUTHORS Neda Khodabakhsh Joniani (The University of Sydney)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 11.01 - HS
TRACK Cutting Edge Research Areas

Abstract

The corneal epithelium is a self-renewing tissue maintained with remarkable precision. Its regeneration is driven by limbal epithelial stem cells (LESCs), which reside at the corneal periphery and give rise to transit amplifying cells (TACs) that migrate centripetally toward the centre. These TACs continually replenish the tissue and, together with vertical delamination between layers, sustain the five to seven stratified layers of the epithelium. Despite this highly coordinated renewal process, the mechanical mechanisms regulating epithelial stratification remain poorly understood.

In this talk, I present simulation results demonstrating that stratification is strongly coupled to TAC proliferation, whereas LESC activity remains largely unchanged, consistent with their slow-cycling

behaviour. Weakening cell–substrate adhesion increases epithelial turnover without the need for external growth factor stimulation. In addition, increased surface shedding promotes both cell division and delamination, while excessive shedding induces mechanical compensation in the form of cell stretching in the upper epithelial layer.

The model further predicts a direct relationship between the shedding rate and the centripetal velocity of clonal expansion, reflecting a wound-healing-like acceleration of epithelial migration. Overall, these results highlight how intercellular mechanical forces coordinate cell size, migration, and turnover to maintain and rapidly restore epithelial integrity. Finally, I introduce an extension of the model to a five-layer epithelial structure and demonstrate the resulting wound healing dynamics.

MS163-04 — Estimating effective diffusion in the vitreous: A profile likelihood approach

SPEAKERS Patricia Lamirande (Université de Montréal)
AUTHORS Patricia Lamirande (Université de Montréal)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 11.01 - HS
TRACK Cutting Edge Research Areas

Abstract

Wet age-related macular degeneration is a chronic ocular disease treated by repeated intravitreal injections. The relatively short intravitreal half-life of these therapeutics leads to frequent administration, creating a substantial treatment burden. Accurate estimation of the effective diffusion coefficient is central to predicting intravitreal half-life and to the rational design of longer-lasting treatments.

Quantitative investigation of intraocular drug transport is challenging: experimental access to the eye is limited, and the vitreous humour is a structurally complex, heterogeneous medium. Classical approaches estimate diffusion coefficients in substitute materials or extracted samples, which do not faithfully represent the intact vitreous. Recently, collaborators developed a method to measure diffusion in intact ex vivo vitreous. We analyse these data for molecules of varying sizes using a profile likelihood framework to estimate effective diffusion coefficients, assess practical identifiability, derive confidence intervals, and investigate the influence of molecular size and charge on transport. Comparison with synthetic data evaluates parameter recoverability under the experimental protocol. Our analysis shows partial practical identifiability, with confidence intervals dominated by measurement uncertainty and potential bias for highly charged molecules, indicating the need for experimental refinement and improved modelling of charge-dependent transport.

MS163-02 — Heterogeneity, Bias, and Translation Bottleneck in Age-Related Macular Degeneration

SPEAKERS Moussa Zouache (University of Utah)
AUTHORS Moussa Zouache (University of Utah)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 11.01 - HS
TRACK Cutting Edge Research Areas

Abstract

Age-related macular degeneration (AMD) is a primary cause of irreversible vision loss worldwide, causing years of progressive visual decline and reduced quality of life that may culminate in severe visual impairment or blindness. Available therapeutic options are limited to late-stage disease, when irreversible damage to ocular tissues has already occurred and patients experience significant vision loss. Moreover, these treatments often suffer from limited efficacy and a high treatment burden. With an estimated attrition rate of 85-90%, the translation pipeline for AMD therapeutics faces a persistent bottleneck, hindering the development of effective alternative treatments. In this talk, we demonstrate that this translational bottleneck is driven largely by substantial variability in disease presentation and clinical progression among affected individuals. This population-level heterogeneity propagates bias across multiple stages of the translational pipeline, from clinical study design and patient recruitment to molecular analyses and biomarker validation. Integrative analyses across genetics, transcriptomics, proteomics, and clinical

phenotyping suggest that this heterogeneity arises from a lack of integration among three core disease dimensions: age, genetic risk profile, and staging of AMD severity. We argue that frameworks combining these axes are required to identify knowledge gaps, clarify disease mechanisms, and support effective therapy development.

MS164 — Modelling heterogeneity in infectious disease transmission and control

TRACK Mathematical Epidemiology
SCHEDULE Monday, 13 July 2026; 10:40–12:00; 15.02 - HS
ORGANISERS Emma Fairbanks (University of Manchester); Luzia Nora Felber (Swiss TPH)

Minisymposium abstract

Heterogeneity is a defining feature of infectious disease systems, shaping transmission patterns, intervention effectiveness and prospects for elimination. Yet capturing relevant sources of variation remains a central challenge for mathematical modellers. This mini-symposium brings together researchers developing novel approaches to incorporating heterogeneity across scales.

Talks will examine how different forms of heterogeneity influence disease dynamics and intervention design. We explore spatial heterogeneity in mosquito movement using random walk models that capture fine-scale dispersal patterns and their consequences for local transmission intensity. The symposium addresses heterogeneity in epidemiological settings, particularly the distinct challenges arising in elimination contexts where residual transmission becomes increasingly focal and stochastic. Speakers will also present frameworks for optimising control measures that account for heterogeneity in intervention costs, effectiveness and policy objectives.

A unifying theme is how mathematical approaches can move beyond population-average assumptions to better represent the variation inherent in real-world disease systems. By bringing together perspectives on vector biology, elimination dynamics and adaptive control, the symposium highlights how explicitly modelling heterogeneity can improve predictions and support more effective, targeted interventions for infectious disease control programmes.

MS164-01 — Mosquito biting heterogeneity and the community impact of targeted malaria interventions

SPEAKERS Emma Fairbanks (University of Manchester)
AUTHORS Emma Fairbanks (University of Manchester)
SCHEDULE Monday, 13 July 2026; 10:40–11:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Heterogeneity in mosquito biting rates is a key driver of malaria transmission dynamics and strongly influences the distribution of cases within a population. We develop a stochastic compartmental model describing malaria transmission between humans and mosquitoes, incorporating variation in biting exposure. We quantify the level of biting heterogeneity required to generate different Pareto fractions in malaria case distributions, from relatively homogeneous transmission to scenarios where a small proportion of individuals account for a large proportion of cases. We then investigate the impact of targeted control strategies that prioritise individuals based on biting exposure. Our results show that targeting highly bitten individuals can substantially reduce overall transmission. However, protecting specific individuals may initially lead to temporary increases in cases among non-targeted individuals due to shifts in transmission dynamics. Over longer time horizons, targeted interventions generally reduce transmission across the entire community, including those not directly receiving control measures. These findings highlight the importance of accounting for exposure heterogeneity when designing malaria control strategies and demonstrate the potential long-term community-wide benefits of targeted interventions.

MS164-02 — Simulating Mosquito Flight Paths with a Random Walk Model

SPEAKERS Luzia Nora Felber (Swiss TPH)
AUTHORS Luzia Nora Felber (Swiss TPH); Mathurin Fatou (Institut Pasteur); Beatrice H. Bredt (Swiss TPH); Luis García-Naranjo (University of Padua); Pie Müller (Swiss TPH); Thomas A. Smith (Swiss TPH); Helmut Harbrecht (University of Basel); Nakul Chitnis (Swiss TPH)
SCHEDULE Monday, 13 July 2026; 11:00–11:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Many animal species have been shown to use search patterns described by Lévy flight instead of particle-like Brownian motion [Hum+12; Rey+07; Sim+08]. To investigate which type of random walk describes the search pattern of mosquitoes, we analyse flight tracks obtained by infrared recordings of *Anopheles* mosquitoes in a screened cage by looking at the distribution of their step lengths. On the other hand, we set up and simulate different types of random walk models to extract those step lengths. To determine which random walk model fits the flight behaviour of mosquitoes and to build a final model, we compare the distributions of the step lengths yielded by the simulations to the distribution of the step lengths given by the recorded flight path data. In a next step, we include stimuli into the simulations and scale up the model to the size of a village. The flight path simulation builds the foundation to model the dispersal of mosquitoes and hence helps to estimate the effect of interventions targeting the mosquito population to fight disease transmission, for example releasing mosquitoes infected with *Wolbachia* bacteria or genetically modified mosquitoes, or distributing tools such as spatial emanators, odour-baited traps for adult mosquitoes, attractive targeted sugar baits (ATSB), or oviposition traps to assess their impact. The goal is to use the model to suggest the optimal placement of interventions.

References

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MS164-03 — Immunity-driven biases in malaria incidence: a study for Senegal and The Gambia

SPEAKERS Miguel Garrido Zornoza (Abdus Salam International Centre for Theoretical Physics)
AUTHORS Miguel Garrido Zornoza (Abdus Salam International Centre for Theoretical Physics); Nakul Chitnis (Swiss TPH); Cyril Caminade (Abdus Salam International Centre for Theoretical Physics); Adrian M. Tompkins (Abdus Salam International Centre for Theoretical Physics)
SCHEDULE Monday, 13 July 2026; 11:20–11:40; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Pronounced demographic heterogeneity in immunity is a defining feature of malaria; slowly acquired through repeated and sustained exposure, yet vanishing when this ceases. While compartmental models offer computational efficiency and may capture population-level trends, they often simplify the complex, cumulative nature of acquired immunity against the disease. Furthermore, varying levels of acquired immunity may differentially modify the sensitivity of the disease to climate, thus creating systematic spatially-dependent biases unaccounted for by population-average modelling approaches. We present

VECTRI-ABM, a novel grid-based mechanistic framework where the climate-sensitive vector ecology model, VECTRI, is integrated with an agent-based model (ABM) of human health, replacing its original compartmental SEIR module to explicitly resolve individual-level demographic and immunity traits.

We applied this framework to Senegal and The Gambia - a region characterized by a pronounced north-south rainfall gradient, with diverse transmission intensity regimes. Through counterfactual experiments, we quantify the systematic biases in modelled incidence that arise when individual exposure history is neglected. We show how these biases shift as a function of the rainfall regime and host age, identifying the demographic and climatic thresholds where population-level averages fail to capture malaria dynamics. Finally, we discuss how identifying these systematic biases supports the design of more effective, targeted interventions for malaria prevention and elimination programs.

MS164-04 — Identifying robust triggers for controlling epidemic waves in real-time

SPEAKERS Nathan Doyle (University of Warwick)
AUTHORS Nathan Doyle (University of Warwick); Robin Thompson (University of Oxford); Michael Tildesley (University of Warwick)
SCHEDULE Monday, 13 July 2026; 11:40–12:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Effectively assessing outbreak control strategies in real-time remains a substantial public health challenge. During the COVID-19 pandemic, public health and social measures (PHSMs) were often implemented and relaxed under a ”tiered” system to balance the benefits and costs associated with broad societal measures. To guide decision making, modelling studies designed frameworks for tiered PHSMs which typically assumed the movement between tiers was triggered by an incidence or prevalence metric (for example, the number of COVID-19 patients occupying beds in hospital). However, the epidemiological delay from transmission events to clinical outcomes leads to a substantial epidemic peak overshoot upon imposing ”lockdown”, which is difficult to infer from measurements of current incidence or prevalence alone. We therefore introduce a framework that combines scenario modelling of tiered PHSMs with explicit short-term forecasts to estimate the epidemic peak in real-time. As we show, optimal hospital occupancy thresholds for imposing lockdown can be inferred using this framework, accounting for a range of sources of uncertainty. We demonstrate that metrics characterising the epidemic speed (in particular, the growth rate and the serial interval) are crucial for imposing a timely and effective lockdown to prevent healthcare systems from becoming overwhelmed.

MS11 — Immune attack on the nervous system: mathematical models of multiple sclerosis

TRACK Neuroscience and Neural Systems
SCHEDULE Monday, 13 July 2026; 15:00–18:00; 15.12 - HS
ORGANISERS Adrianne Jenner (Queensland University of Technology); Thomas Hillen (University of Alberta)

Minisymposium abstract

Multiple sclerosis (MS) is a chronic autoimmune disease affecting almost 3 million people worldwide and causing substantial physical and cognitive disability. In MS, dysregulated immune responses target myelin—the protective coating of neurons—leading to neurodegeneration and progressive neurological impairment. There is currently no cure for MS, and existing treatments are only partially effective, often slowing disease progression or alleviating symptoms in only a subset of patients. The increasing incidence of MS, particularly among women, underscores the urgent need for new quantitative approaches to understand its underlying mechanisms.

This minisymposium brings together eight mathematical researchers at the forefront of MS disease modelling to showcase how mathematical and computational methods can provide mechanistic insight into MS pathophysiology. Using a diverse range of approaches, including ordinary and partial differential equations and agent-based models, speakers will demonstrate how mathematics can capture the complex

immune dynamics and non-trivial spatial patterns of lesion formation and spread within the brain. With growing interest in mathematical immunology driven by applications in cancer, COVID-19, HIV, and other diseases, this minisymposium aims to foster cross-disciplinary exchange and highlight opportunities for applying and extending these techniques to the modelling of MS.

MS11-01 — Computational Approaches to Multiple Sclerosis: Immune Dynamics and Data-Driven Lesion Modeling

SPEAKERS Barbara Quintela (Universidade Federal de Juiz de Fora)
AUTHORS Barbara Quintela (Universidade Federal de Juiz de Fora); Gustavo G Silva (Federal University of Juiz de Fora (UFJF)); Luan C Silva (Federal University of Juiz de Fora (UFJF)); Marcelo Lobosco (Federal University of Juiz de Fora (UFJF)); Philippe Neumann (Federal University of Juiz de Fora (UFJF))
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Multiple Sclerosis (MS) is a chronic autoimmune disease affecting approximately 1.8 million people worldwide and demands improved tools for diagnosis and disease understanding. This talk presents two complementary computational models addressing distinct challenges in MS research and care. The first focuses on medical imaging and builds upon an MS lesion segmentation dataset [Gua+25]. To address data scarcity, we investigate generative deep learning for artificial data augmentation. A two-stage pipeline is employed: a Variational Autoencoder (VAE) generates synthetic lesion masks, followed by a Conditional Generative Adversarial Network (cGAN) that synthesizes realistic brain textures conditioned on these masks. We evaluate the impact of augmented data on MRI lesion segmentation performance. The second model targets disease mechanisms through an in silico representation of Experimental Autoimmune Encephalomyelitis (EAE) [Sil+20]. The model captures key immunological dynamics of EAE progression, enabling simulation of disease evolution and therapeutic strategies while reducing costs and ethical constraints of animal experimentation. Together, these approaches demonstrate how computational modeling advances both clinical applications and mechanistic understanding of MS.

References

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[Sil+20] Luan Cristian da Silva et al. “A new lipophilic amino alcohol, chemically similar to compound FTY720, attenuates the pathogenesis of experimental autoimmune encephalomyelitis by PI3K/Akt pathway inhibition”. In: *International Immunopharmacology* 84 (2020), p. 106553.

MS11-03 — Mass-conserving boundary motion in a model of invasion and recession

SPEAKERS Georgia Weatherley (Queensland University of Technology)
AUTHORS Adrianne Jenner (Queensland University of Technology); Georgia Weatherley (Queensland University of Technology); Michael Dallaston (Queensland University of Technology)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

In multiple sclerosis patients, immune cells attack myelinated nerve axons, creating demyelinated regions called lesions. MRI observations show that individual lesions can grow or shrink over time. These lesion dynamics provide important insights into disease progression, as well as the efficacy of treatment. We develop a moving boundary model to represent lesion boundaries as sharp interfaces that can advance or recede. Existing moving boundary extensions of reaction–diffusion equations, such as the Fisher–KPP

equation, typically use a Stefan-like condition in which boundary motion is determined by the flux of cells. Instead, we model the boundary velocity as a function of local cell density, allowing us to represent biological processes where cells degrade or deposit material to move a boundary without being consumed. We observe a variety of behaviours depending on the parameterisation of the boundary velocity function, including regimes supporting multiple invading and receding travelling waves, unstable travelling waves, and receding solutions with population blow-up.

MS11-05 — Exploring immune pathways and remyelination in Multiple Sclerosis

SPEAKERS Maria Carmela Lombardo (University of Palermo)
AUTHORS Maria Carmela Lombardo (University of Palermo)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

In this talk, I review a class of mathematical models for the early stages of Multiple Sclerosis (MS). A central immunological question concerns the trigger of the immune cascade initiating MS pathology. We compare two scenarios: one based on local microglia activation, recruitment of systemic immune responses, and oligodendrocyte apoptosis [Bar+16; Gar+24; KC07; Lom+17]; the other on cytokine-mediated modulation of macrophage activation [MF21]. We focus on a reaction-diffusion-chemotaxis system for activated macrophages, cytokines, and oligodendrocytes. By varying the parameter governing the effect of cytokines on macrophage activation, the model captures both mechanisms within a unified framework.

Combining analytical results and numerical simulations, we show that the model generates a variety of demyelination patterns. For biologically realistic parameter values, its asymptotic states reproduce pathological features consistent with MRI observations. Bifurcation analysis highlights marked differences between the two scenarios. The innate-immunity mechanism leads to highly aggressive pathology, with strong focal inflammation and rapid progression, whereas the cytokine-mediated mechanism yields milder inflammation and much slower disease evolution.

Finally, I discuss ongoing extensions including endogenous repair mechanisms active in acute inflammation, with the aim of describing remyelination processes observed in early MS and their failure in chronic lesions.

References

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[Lom+17] M.C. Lombardo et al. “Demyelination patterns in a mathematical model of multiple sclerosis”. In: *Journal of Mathematical Biology* 75.2 (2017), pp. 373–417.

[MF21] N. Moise and A. Friedman. “A mathematical model of the multiple sclerosis plaque”. In: *Journal of Theoretical Biology* 512 (2021), p. 110532.

MS11-06 — Derivation from kinetic theory of chemotaxis models for Multiple Sclerosis

SPEAKERS Marzia Bisi (University of Parma)
AUTHORS Maria Groppi (University of Parma); Marzia Bisi (University of Parma); Romina Travaglini (University of Pavia)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

We present the derivation of a class of reaction-diffusion models for Multiple Sclerosis starting from kinetic equations for the distribution functions of the cell populations involved in the biological processes underlying the evolution of the disease. The kinetic setting for the cell distributions is outlined, detailing interaction operators that account for conservative and non-conservative processes. Under suitable hypotheses of multiple scale processes, an asymptotic diffusive limit of kinetic equations is performed, leading to a system of reaction-diffusion equations for population densities, with general diffusivity and growth functions for some kinds of cells. The Turing instability analysis of such macroscopic system provides necessary conditions for the emergence of spatial patterns in a two-dimensional domain; the shape and stability of such patterns are discussed through a weakly nonlinear analysis, and some numerical simulations are presented in order to confirm theoretical results.

MS11-07 — A New Paradigm for the Mathematical Modelling of Multiple Sclerosis

SPEAKERS Thomas Hillen (University of Alberta)
AUTHORS Adrianne Jenner (Queensland University of Technology); Thomas Hillen (University of Alberta)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

The mathematical modelling of MS so far has focussed on a few aspects of the disease, but an overall modelling framework is missing. Here we propose a paradigm for the mathematical modelling of MS. Based on biological principles we propose six consecutive modelling levels. We develop models on Level 1,2, and 3, and test if these models can describe known effects related to MS. We first show that periodic disease outbreaks are possible in this framework. We show that presence of Epstein-Barr virus infections can initiate the disease, low levels of estrogen and vitamin D can alleviate it, mutations in the HLA-DR gene can promote MS, and we find that memory B-cells play a dominant role in the disease progression.

MS11-04 — Mathematical Model of Multiple Sclerosis

SPEAKERS Mo'tassem Al-arydah (Khalifa University)
AUTHORS Mo'tassem Al-arydah (Khalifa University)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 15.12 - HS
TRACK Immunobiology & Infection

Abstract

We develop and analyze a nonlinear mathematical model for autoimmune-mediated demyelination in multiple sclerosis that captures interactions between healthy myelin, autoreactive immune cells, regulatory immune cells, and neuronal functional capacity. Immune activation is driven by myelin damage through a saturating response function, while regulatory cells suppress pathogenic activity and therapeutic intervention contributes to immune clearance. A fast–slow structure reflects disparate physiological timescales and permits quasi-steady-state reduction to a lower-dimensional immune subsystem. We establish biological well-posedness by proving positivity and global existence of solutions and identifying invariant regions, and we derive a threshold quantity governing autoimmune invasion via linearization about the disease-free equilibrium. The existence and stability of steady states are characterized, including parameter regimes associated with chronic inflammation, remission, and oscillatory immune dynamics, and sufficient conditions ensuring uniform boundedness of immune populations are obtained and interpreted biologically. Numerical simulations illustrate long-term disease trajectories and quantify the impact of therapeutic control on immune regulation and neuronal preservation, providing a mathematically tractable and biologically grounded framework for investigating autoimmune progression and evaluating treatment strategies in multiple sclerosis.

MS13 — Mathematical Endocrinology: Models of Regulation, Disease and Dynamics

TRACK Cutting Edge Research Areas
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 01.15 - HS
ORGANISERS Ami Radunskaya (Pomona College); Harsh Jain (University of Minnesota Duluth)

Minisymposium abstract

Hormones play a central role in regulating physiological systems and maintaining homeostasis, yet the multiscale, feedback-rich nature of endocrine function poses major challenges for understanding and treating disorders spanning metabolic, mood, and hormone-responsive disorders. This mini-symposium brings together researchers developing mathematical and computational frameworks to interrogate endocrine processes across biological systems and clinical contexts. Topics include modeling hormonal dysregulation in diseases such as diabetes, depression, and cancer; investigating gut/brain and neuroendocrine signaling through neurotransmitter pathways like serotonin and dopamine; and analyzing hormone-driven biological rhythms and life-stage transitions. Embracing systems-level thinking, the talks will highlight mechanistic modeling, data integration, and network-based approaches with relevance to personalized medicine. Our speakers span a range of career stages from graduate students to faculty and industry scientists and reflect a diversity of identities and perspectives. By bridging disciplines and amplifying emerging voices, this session showcases how mathematical modeling can accelerate both fundamental understanding and clinical insight in endocrinology.

MS13-01 — A harmful cycle: understanding the roles of inflammation, serotonin and the blood brain barrier in neurological development through mathematical models.

SPEAKERS Ami Radunskaya (Pomona College)
AUTHORS Ami Radunskaya (Pomona College); Luisa Gianuca (Claremont Graduate University)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Experimental results have highlighted the role of serotonin in fetal brain development, and have indicated that inflammation can cause a cyclical disruption to the blood-brain barrier permeability, leading to long-term neurological disorders. In this talk I will present a mathematical model of the tryptophan-serotonin pathway in the placenta that includes modulation by maternal inflammation. In particular, we explore the hypothesis that blood brain barrier disruption due to gestational maternal immune activation can initiate a cycling of immune activation and BBB dysfunction that persists into adulthood. The model has the potential to help us understand a possible mechanism for the development of mental disorders.

MS13-02 — Mathematical Modeling of the Gut-Brain Axis

SPEAKERS Cornelia Mihaila (St Michael's College)
AUTHORS Cornelia Mihaila (St Michael's College)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

In this talk I will present a mathematical model that describes the interaction of gut and brain through the presence of serotonin and tryptophan. The neurotransmitter serotonin is produced in the brain and the gut from the amino acid tryptophan via an enzymatic reaction. However, tryptophan is not produced by the body, but is obtained from food. While serotonin cannot cross the blood-brain barrier, tryptophan can, and does. Since serotonin regulates digestion, mood, sleep, and other physical processes in response to stress, we build a model that describes the kinetics of its production and absorption. Because both tryptophan and serotonin are present in the gut and the brain, the tryptophan-serotonin pathway is a major communication route between the gut and the brain. This model extends previous models of

serotonin and tryptophan focused in the brain. This work is a collaboration with Noah Avery Hughes, Harsh Jain, Steven R Lippold, Ami Radunskaya, Susmita Sadhu, and Sundar Tamang.

MS13-03 — The Four Ds of Ovulation: Dynamics, Dysfunction, Disparities, and Dosing

SPEAKERS Erica Graham (Bryn Mawr College)
AUTHORS Erica Graham (Bryn Mawr College)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Endocrine physiology is a complex system of crosstalk between hormones in various tissues. Tight regulation of reproductive hormones is essential for ovulatory function, but dysregulation can lead to infertility and may be exacerbated by other endocrine–especially metabolic–abnormalities and/or racial and ethnic disparities. Further, inter-individual differences may alter clinical outcomes under both physiological and pathological circumstances. Here we discuss a series of mathematical models describing ovulatory dynamics with applications to polycystic ovary syndrome, endometriosis, metabolism, and oral contraceptives.

MS13-04 — Bridging Structured PDEs and ODE Models in Ovarian Follicle Dynamics

SPEAKERS Claudio Iuliano (Anhalt University of Applied Sciences)
AUTHORS Alexander Lange (Anhalt University of Applied Sciences); Claudio Iuliano (Anhalt University of Applied Sciences); Edilbert Christhuraj (Anhalt University of Applied Sciences)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 01.15 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Ovarian follicle development is commonly described either by compartmental models, in which each developmental stage is represented by a discrete compartment (e.g. [HHS14]), or by physiologically structured population models (PSPMs), formulated as transport PDEs, where each follicle is treated as a cell population distributed along a continuous maturation variable (e.g. [Mon+16]). The former reproduces the follicular and hormonal dynamics of a full menstrual cycle but does not account for multiscale structure or explicit follicular competition. The latter captures multiscale behaviour and competition at the level of total cell populations, similarly to the ODE system in [FR22], but lacks a direct link with compartmental models, so that the extension to a full-cycle description remains open.

We introduce a class of ODE systems obtained as finite-dimensional reductions of PSPMs via a perturbative moment closure. These systems provide backbone approximations of the structured dynamics: they retain the leading nonlinear mechanisms governing recruitment, selection, and atresia while projecting the cell maturation onto macroscopic variables. This establishes an explicit correspondence between PSPMs and compartmental models, leading to a tractable multiscale description of follicle development under different physiological and pathological scenarios.

References

[FR22] Sophie Fischer-Holzhausen and Susanna Röblitz. “Hormonal regulation of ovarian follicle growth in humans: Model-based exploration of cycle variability and parameter sensitivities”. In: *Journal of Theoretical Biology* 547 (2022), p. 111150. ISSN: 0022-5193. DOI: https://doi.org/10.1016/j.jtbi.2022.111150. URL: https://www.sciencedirect.com/science/article/pii/S0022519322001485.

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MS13-05 — Models of Regulation and Disease Dynamics in Ovarian Cancer Invasion

SPEAKERS Leili Shahriyari (University of Massachusetts Amherst)
AUTHORS Leili Shahriyari (University of Massachusetts Amherst)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

The ovarian tumor microenvironment is shaped by dynamic interactions among cancer, stromal, and immune cells, but the drivers of progression remain unclear. In ovarian cancer, we found that immune balance is more prognostic than absolute immune cell abundance: CD8/Treg and CD8/CD4 ratios were more strongly associated with survival than CD8+, CD4+, or Treg levels alone. Macrophage state was also critical. Tumors enriched in M2 macrophages were linked to vascular invasion, persistent disease, and worse survival, whereas higher M0 macrophage levels predicted better outcomes; M1 macrophages showed little prognostic value. Neutrophil infiltration, though less common, was likewise associated with poor survival. Unsupervised clustering identified four immune-defined subtypes, with the worst outcomes in tumors enriched for M2 macrophages and CD4+ T cells and depleted in M0 macrophages. To investigate the dynamics of some of these players mechanistically, we pair patient-derived observations with a three-dimensional tumoroid model of ovarian cancer invasion and a baseline ODE model describing the coupled dynamics of ovarian cancer cells, stromal cells, macrophages, and mesothelial invasion. This experimental–mathematical framework allows us to examine how these components interact and how those interactions may be leveraged to reduce tumor invasion.

MS13-06 — Modeling PSA Dynamics to Characterize Prostate Cancer Treatment Response

SPEAKERS Renee Brady-Nicholls (Moffitt Cancer Center)
AUTHORS John Palacios (Moffitt Cancer Center); Renee Brady-Nicholls (Moffitt Cancer Center)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Prostate cancer (PCa) remains a major global health burden, with more than 1.5 million new cases diagnosed annually worldwide. In the United States alone, over 300,000 men are expected to be diagnosed in 2026, with more than 36,000 deaths. Prostate-specific antigen (PSA) is widely used as a surrogate marker of tumor burden, and its temporal dynamics are closely associated with disease progression. Improving our understanding of patient-specific PSA kinetics and the underlying drivers of progression is critical for advancing PCa treatment. In this study, we developed and analyzed two simple kinetic pharmacodynamic models of PSA dynamics, a latent variable and transit compartment linking to PSA dynamics. We evaluated each model’s ability to describe longitudinal PSA data from 55 patients undergoing hormone therapy. Model performance was assessed using goodness-of-fit metrics, diagnostic plots, and predictive accuracy for patient-specific trajectories. Both models were able to capture key features of PSA dynamics, describing both responsive and progressive patient dynamics. However, the transit model demonstrated superior predictive performance and greater stability in estimating patient-specific parameters. These findings highlight the potential of simple mechanistic models to characterize disease progression and support personalized treatment strategies in prostate cancer.

MS13-07 — Longitudinal Biomarkers and Alzheimer’s Disease in Down Syndrome

SPEAKERS Samuel Handelman (Aerska)
AUTHORS Samuel Handelman (Aerska)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 01.15 - HS

TRACK Cutting Edge Research Areas

Abstract

Background: Down syndrome (Trisomy 21) includes triplication of the APP gene, leading to excess amyloid precursor protein and early amyloid plaque formation. Individuals with Down syndrome typically develop plaques in their 40s, and Alzheimer's disease is their leading cause of mortality. APP-directed interventions may therefore provide substantial clinical benefit in Down syndrome. However, Down Syndrome also involves immunologic and metabolic comorbidities. **Methods:** The Alzheimer's Disease Biomarkers-Down Syndrome (ABC-DS) consortium has recruited a population study of over 100 Down Syndrome patients. Participants undergo cognitive testing and multimodal biomarker profiling, including inflammatory, metabolic, and PET-based amyloid measures. **Results:** PET-derived amyloid measures are the strongest predictors of cognitive decline. Observed accumulation rates indicate that trials with approximately 50 participants per arm should be powered. However, immunologic and metabolic comorbidities may introduce heterogeneity that complicates trials or reveals subgroups requiring additional interventions. **Discussion:** Interventions targeting amyloid accumulation in Down Syndrome are urgently needed. In addition to reporting these results, we will also pose forward-modeling challenges related to immunologic and metabolic dysfunction in Down syndrome.

MS13-08 — Analyzing (Sub-phenotyping) the Longitudinal Progression to Type 2 Diabetes using Machine Learning and Physiological Simulations

SPEAKERS Vijaya Subramanian (Johns Hopkins University)
AUTHORS Vijaya Subramanian (Johns Hopkins University)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Type 2 diabetes (T2D) diabetes disease progression is associated with genetic susceptibility coupled with risk factors like overweight/obesity and prior incidence of gestational diabetes. Impaired insulin sensitivity coupled with alpha and beta cell dysregulation is observed in the progression to T2D. What is unclear is why some individuals progress to T2D while others never transition. In our previous model of disease progression [Sub+25], we showed that mild alpha cell dysregulation could be beneficial as it leads to robust compensatory insulin secretion in the milieu of impaired insulin sensitivity leading to long term glycemic stability. Here we analyze longitudinal data on at risk individuals from the DPP/DPPOS study [Gro+09] using Machine Learning tools coupled with simulations of the previously developed physiological model. We generated fine-grained sub-phenotyping of individuals, based on the relative influence of the different underlying dysregulations in disease progression. We first performed multivariate dynamic time warping (DTW)-based hierarchical clustering of longitudinal trajectories using a composite dissimilarity measure that combined path-length-normalized dynamic time warping distance with penalties for differences in participants' baseline levels at study entry and mean longitudinal levels over follow-up. The clustering revealed groups of individuals who showed not only long-term stability but also periods of improvement in glycemia. The simulations showed that mild alpha cell dysregulation is likely to develop and plateau in these individuals leading to this behavior. This work highlights the power of integrating physiological modeling with machine learning tools to deliver precision medicine.

References

[Gro+09] Diabetes Prevention Program Research Group et al. "10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study". In: *The Lancet* 374.9702 (2009), pp. 1677–1686.

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MS22 — Recent perspectives on mathematical-biology education

TRACK Education and Outreach
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 15.11 - HS
ORGANISERS Michael Kelly (Transylvania University); Punit Gandhi (Virginia Commonwealth University); Rebecca Everett (Haverford College); Veronika Zarnitsyna (Emory University School of Medicine)

Minisymposium abstract

The minisymposium brings together educators to examine current challenges and innovations that highlight the impact of integrating mathematics and biology in the classroom. The Education minisymposium has long been a cornerstone of SMB annual meetings emphasizing the essential role of education in translational science and in cultivating the next generation of mathematical biologists. A strong commitment to diversity underlies this session, with speakers representing a broad range of disciplinary backgrounds, instructional contexts, identities, and scholarly perspectives.

MS22-01 — Universal design for learning applied to the development of a new a second-year undergraduate statistics course

SPEAKERS Adriana Zanca (The University of Melbourne)
AUTHORS Adriana Zanca (The University of Melbourne); Paul Fijn (The University of Melbourne)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 15.11 - HS
TRACK Education and Outreach

Abstract

Universal Design for Learning (UDL) is an educational framework that provides students with avenues for access to course material. The core principles of UDL are to provide learners with multiple means of engagement, representation and action and expression. In the context of the design of a new second-year undergraduate statistics course, we have developed course materials, including assessment, that prioritise accessibility and agency for students. In this talk I will discuss what are the 'low hanging fruit' that you may be able to apply to your context, as well as the more challenging aspects of applying UDL.

CAST (2024). CAST Universal Design for Learning Guidelines version 3.0. []

References

[] *Cast 2024*. URL: https://udlguidelines.cast.org.

MS22-02 — Mentoring First-Year STEM Students Through Collaborative Research in the Haynes Scholars Program

SPEAKERS Alex Capaldi (James Madison University)
AUTHORS Alex Capaldi (James Madison University)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 15.11 - HS
TRACK Education and Outreach

Abstract

The Haynes Scholars Program at James Madison University is an academic residential learning community for a cohort of first-year STEM majors that emphasizes early research and fostering a sense of belonging. Over the past year, we co-taught the program's sequence of two research courses, where small groups of first-year students worked on accessible projects in mathematical biology with a unified application theme of white-nose syndrome in bats. Our aim was to introduce them to the process of doing applied mathematical and statistical research: reading the literature, asking questions, exploring models, and presenting their results. We will share how we structured the experience, including scaffolding the background material and pacing the research steps, while also giving the students ownership of their

projects. We'll also reflect on what worked well, what was challenging, and how this kind of early research experience helps build confidence and community for students just beginning their STEM journey.

MS22-03 — Structured Onboarding of Undergraduate Researchers in Mathematical Biology

SPEAKERS Ashley Ford Versypt (University of Buffalo)
AUTHORS Ashley Ford Versypt (University of Buffalo)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 15.11 - HS
TRACK Education and Outreach

Abstract

The Ford Versypt Lab uses mathematical biology methods to study tissues, treatments, and toxicology. We introduce new undergraduate students to a suite of techniques for these topics in mathematical systems biology. To onboard students in a semester or summer research experience, we use a structured approach with two phases: the training phase and the research phase. We have crafted a series of computational notebooks (MATLAB and Python) arranged as assignments that introduce applying conservation balances to populations of cells and amounts of chemical species in living organisms and numerically solving systems of ODEs. Then we provide guidance for an open exploration period for the students to investigate topics of their interest that use the techniques and computational notebooks for the remainder of the academic term. They receive guidance on searching the literature. They are tasked with finding two mathematical biology papers that involve ODE models with different equations for the selected biomedical topics. By the end of the term, they must use MATLAB or Python to replicate the two models, write a report detailing their progress and their topic, and present their work. By using MATLAB or Python templates provided by the lab, students focus on the learning goals related to the research concepts instead of being hindered by programming or analytical mathematics proficiency or deficiency. Senior students also appreciate that the templates enable them to quickly make progress towards using advanced techniques. This approach has been used with one high school student and dozens of college undergraduate students across all levels and majors including applied mathematics, chemical engineering, biomedical engineering, and biology.

MS22-04 — Putting Math Bio in its Place

SPEAKERS Kyle Dahlin (Virginia Tech)
AUTHORS Kyle Dahlin (Virginia Tech); Stacy Potes (University of Hawai'i at Mānoa)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 15.11 - HS
TRACK Education and Outreach

Abstract

Place-based mathematics pedagogy can increase student engagement and persistence in the classroom, at a time when maintaining student attention is increasingly difficult. By focusing on relevance, this pedagogical framework enables students to connect their lived experiences to mathematics, thereby supporting engagement and persistence in the classroom. Mathematical biologists often focus on topics of particular interest to students of this generation, such as public health, sustainability, and environmental change. Mathematical biology, therefore, has the unique potential to bring "place" into the classroom in a way that other areas of theoretical and applied mathematics cannot. In this talk, I will share my experience working to integrate place-based pedagogy, mathematical biology, and culturally relevant teaching practices with educators in Hawai'i. I will discuss practical ways of incorporating place-based practices into your own math classrooms.

MS22-05 — Advances in Accessibility of Undergraduate Numerical Methods

SPEAKERS Laura Ellwein Fix (Virginia Commonwealth University)
AUTHORS Laura Ellwein Fix (Virginia Commonwealth University); Punit Gandhi (Virginia Commonwealth University); Reed Ogrosky (Virginia Commonwealth University)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 15.11 - HS
TRACK Education and Outreach

Abstract

Many mathematical problems that students encounter in typical undergraduate mathematics courses are designed so that an exact solution can be found without using a computational tool. While this is beneficial for learning fundamental mathematics, large-scale "real-world" applications that students may encounter after college, especially in the life sciences, frequently require complex and sophisticated computational algorithms. In undergraduate Numerical Methods, students learn how to build – and when to implement – algorithms to approximate solutions to common mathematical problems. This course therefore provides a critical foundation for students as they translate mathematical theory into future STEM careers. An undergraduate numerical methods course is uniquely challenging because it requires treatment of concepts from both theoretical and practical perspectives, and a solid knowledge of several prequisites. In addition, many textbooks and programming languages used in this course are costly, further impacting its accessibility. To address these challenges, we worked to create a set of open-access ancillary materials to supplement free textbooks and coordinate with the introduction of Python as the primary programming language. These included computer programming activities with built-in scaffolding, a theoretical problem bank, a standard set of course notes, a review packet for prerequisite material, and a curated library of external multimedia resources. The work of creating these materials also led to increased conversations among the primary faculty and improved pedagogy. In this talk we will share some early results from development and implementation of these materials over the previous academic year.

MS22-06 — Learning Differential Equation Modeling through Student-Driven Projects

SPEAKERS Rebecca Everett (Haverford College)
AUTHORS Rebecca Everett (Haverford College)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 15.11 - HS
TRACK Education and Outreach

Abstract

Open-ended evaluated class projects provide an opportunity for students to take ownership of mathematical ideas, develop persistence, and enhance skills in communicating mathematics. By allowing students to choose project topics, they can engage with material that is relevant to their lives and interests. In this talk, I will reflect on my experiences in working with undergraduate students on student-driven projects, presenting some examples from an upper-level mathematical modeling course.

MS22-07 — Building Modeling Workshops at Biology Conferences

SPEAKERS Angela Peace (Virginia Tech University)
AUTHORS Angela Peace (Virginia Tech University); Lauren Childs (Department of Mathematics, Virginia Tech); Michael Robert (Virginia Tech)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 15.11 - HS
TRACK Education and Outreach

Abstract

Workshops embedded within biologically focused conferences can be effective ways to bring new people into mathematical biology. They create accessible entry points for experimentalists, field biologists, and early-career researchers to learn modeling, while also giving mathematicians a chance to teach, build collaborations, and connect with real data and systems. In this talk, we share our experiences designing and running hands-on modeling workshops alongside domain-specific meetings, including one at the Global Amphibian and Reptile Disease (GARD) Conference and a recent workshop held just prior to the Ecology and Evolution of Infectious Diseases (EEID) 2026 conference. These particular workshops focus on approachable introductions to dynamical systems and epidemiological modeling, with a mix of short lectures and guided coding activities centered on simulation, parameterization, and working with data. We'll also point to a growing set of publicly available resources, including curricula, code, and organizational tools, that make it much easier to develop and run these kinds of workshops. Overall, we

highlight how these efforts can spark new collaborations, lower barriers to entry, and create meaningful connections between mathematical and biological communities.

MS22-08 — Does classroom-flipping improve analytical skills? Results from a cross-sectional comparison between undergraduate calculus exams

SPEAKERS Stacey Smith? (University of Ottawa)
AUTHORS Stacey Smith? (University of Ottawa)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 15.11 - HS
TRACK Education and Outreach

Abstract

In a flipped life-sciences classroom, base concepts are presented asynchronously and students spend instruction time actively applying their problem-solving skills with the expert guidance of the teacher. Educational theory suggests that emphasizing application and analysis skills may benefit students, particularly in the advent of Artificial Intelligence as a learning tool for undergraduates. We analyzed the individual exam answers from three classrooms of MAT1332 students, one of which was flipped, to determine how flipped-classroom methods impacted student success. We explored what differences in question format, course sub-topic, final grade and question difficulty could reveal about student achievement on individual questions, and thus each section's methods for approaching the final exam. Students in the flipped classroom found greater success on analytical questions but less success on rote-application questions than the other two sections. Very low-achieving students ($<45\%$ final exam grade) and very high-achieving students ($>80\%$ final exam grade) were less affected by classroom flipping than mid-range students. Students in the flipped classroom performed worse on early-semester content, suggesting that an adjustment period to a flipped-classroom style may be necessary. Classroom-flipping seems to benefit life-sciences students by equipping them with the problem-solving-focused skills necessary to succeed on a final exam in the sciences. Mid-achieving students appear to see the greatest benefits of a classroom emphasis on problem-solving once they adapt to the novel learning method.

MS29 — Novel Tools and Methodologies for Epidemiological Models

TRACK Mathematical Epidemiology
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 15.03 - HS
ORGANISERS Lauren Childs (Virginia Tech); Madison Pratt (University of Tennessee, Knoxville); Olivia Prosper Feldman (University of Tennessee, Knoxville)

Minisymposium abstract

Epidemiological models are often used to address real-world policy and intervention questions for which it is challenging to collect large-scale and precise data. As a result, such models frequently involve the use of noisy or sparse data. Traditional modeling and parameter estimation methods used in the wrong context can lead to erroneous conclusions about the impact of interventions, or our understanding of the underlying mechanisms. For example, parameter estimation is often used as a first step before simulating different control scenarios, or performing sensitivity analyses to identify effective targets for public health interventions; if those initial estimates are inaccurate, the subsequent analyses are likely also invalid. In order to mitigate this challenge and appropriately incorporate this noisy or sparse data often encountered in epidemiology, it is key to consider model identifiability and parameter uncertainty as well as work with innovative modeling types. This mini-symposium brings together researchers working on model identifiability, parameter uncertainty and novel modeling structures (e.g., integral projection models) that integrate empirical data, with applications to infectious disease dynamics. It will showcase cutting edge methodologies to assess these factors as well as the applicability of such models to biological questions involving the evaluation and control of infectious disease outbreaks.

MS29-01 — The impact of sojourn distribution assumptions on malaria parasite transmission dynamics: a flexible within-host modeling framework to accommodate empirically driven distributions

SPEAKERS Jordan Pellett (Grand Valley State University)
AUTHORS Jordan Pellett (Grand Valley State University); Olivia Feldman (Prosper) (University of Tennessee, Knoxville)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Many compartmental infectious disease models assume that sojourn times (the time spent in each state) are exponentially distributed; that is, the probability of exiting a particular state at an instant in time is independent of time already spent in that state. While this simplifying assumption may be sufficient in certain contexts, for complex diseases like malaria where control and elimination efforts are hindered by drug resistance, relaxing this assumption to enhance biological realism and better facilitate multi-scale modeling is particularly advantageous. In this talk, we present a flexible PDE model framework of within-host malaria parasite dynamics that allows for non-exponential distributions for the sojourn times for each red blood cell and parasite life cycle stage. We discuss how our framework accommodates empirically driven distributions and demonstrate that different distribution assumptions can substantially impact malaria transmission probability estimates.

MS29-02 — Forecasting Malaria using test positivity and reported case rates

SPEAKERS Katie Gurski (Howard University)
AUTHORS Kathleen Hoffman (University of Maryland Baltimore County); Katie Gurski (Howard University)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Parameters are fundamental components of biological models and play a critical role in determining model behavior. While some parameters can be estimated directly from experimental or observational data, many remain difficult to measure or infer. This work investigates the role of parameters in a malaria transmission model when the available data consist only of the number of malaria tests performed, the number of positive test results, and the population size. We examine parameter identifiability by analyzing the relationship between model parameters and observable quantities, both in the presence and absence of measurement noise. To assess the influence of parameters on model outputs, we compute Sobol sensitivity indices, which quantify how variations in parameter values contribute to changes in the model predictions. Finally, we apply data assimilation techniques to perform forward prediction and to estimate parameters that cannot be determined from experimental data alone.

MS29-03 — Modeling Insecticide Resistance and Management of Malaria Mosquitoes

SPEAKERS Lihong Zhao (Kennesaw University)
AUTHORS Christina Edholm (Scripps College); Jing Li (California State University Northridge); Lihong Zhao (Kennesaw University); Omar Saucedo (Virginia Tech); Silvie Huijben (Arizona State University); Summer Atkins (The University of Alabama in Huntsville)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

With over 1 million deaths annually across the globe due to mosquito-borne diseases, insecticide resistance is a major public health concern. Insecticide-resistant mosquitoes experience both positive and negative selective forces, depending on the resistance genotype, the type and dosage of insecticide applied, and the fitness costs associated with the resistance mutations they carry. Understanding how these forces interact with each other is critical in determining optimal resistance management strategies. We develop and analyze an ODE-based model to examine the impact of resistance management strategies on the evolution of insecticide resistance in mosquito populations and explore optimal control options.

MS29-04 — Likelihood-Free Confidence Regions for Stochastic Epidemic Models via CTMC Simulation and Neural Networks

SPEAKERS Madison Pratt (University of Tennessee, Knoxville)
AUTHORS Madison Pratt (University of Tennessee, Knoxville); Olivia Feldman (Prosper) (University of Tennessee, Knoxville)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Estimating confidence regions for parameters in stochastic epidemic models is challenging when the likelihood is intractable. In continuous-time Markov chain (CTMC) formulations of compartmental models such as SIR, the likelihood cannot be evaluated directly due to the intractability of the Kolmogorov forward equations. Existing approaches rely on simplifying assumptions, such as Gaussian approximations or ad hoc noise models, which can lead to inaccurate inference. We develop a fully frequentist framework for constructing confidence regions without explicit likelihood evaluation. Our method uses empirical moments from CTMC simulations and Monte Carlo approximations of a Mahalanobis-type test statistic, producing confidence sets with nominal coverage. However, the computational burden of Monte Carlo simulation limits dense exploration of the parameter space. To address this, we introduce a neural network–based pipeline that learns the mapping from parameters to the mean and covariance of the CTMC and estimates key quantiles (0.68, 0.80, 0.90, 0.95) using coverage-based stopping criteria. We apply our method to SIR and SEIR models and additionally test the models on the English boarding school influenza dataset. Results show that our approach yields narrow, well-calibrated confidence regions when data are modeled as stochastic realizations of CTMC dynamics rather than observations with additive noise.

MS29-05 — New approaches to old problems: data-driven models of dengue in emergent and endemic environments

SPEAKERS Michael Robert (Virginia Tech)
AUTHORS Michael Robert (Virginia Tech)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Dengue, a mosquito-borne disease, is rapidly expanding its global distribution, emerging in previously naïve populations, while also causing more intense and more frequent outbreaks in endemic areas. There is an increasingly urgent need for innovative approaches for studying drivers of dengue emergence and spread, forecasting outbreaks, and determining how environmental suitability for dengue will change in the face of short- and long-term changes in climate patterns. In this talk, I will discuss recent work harnessing new approaches for modeling dengue emergence and spread in both the city of Córdoba, Argentina, where dengue first appeared in 2009, and the Dominican Republic, where dengue has long been endemic. Among the models I will discuss are machine learning and neural network models developed to study relationships between dengue cases climate with the goal of improving forecasts of dengue transmission. I will discuss our findings from these models in the context of developing early warning systems for predictions for future dengue outbreaks and working with local public health and vector control stakeholders to harness model results to improve dengue surveillance and control.

MS29-06 — Can We End the HIV Epidemic in the U.S.? A Multiscale Model Linking Clinical and Surveillance Data

SPEAKERS Necibe Tuncer (Florida Atlantic University)
AUTHORS Leila Kohan (Florida Atlantic University); Maia Martcheva (University of Florida); Necibe Tuncer (Florida Atlantic University)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

The human immunodeficiency virus (HIV) epidemic remains a pressing public health challenge in the United States, despite major advances in treatment and prevention. Achieving the national Ending the HIV Epidemic (EHE) goals by 2030 requires a deeper understanding of the interplay between individual-level viral dynamics and population-level transmission. To address this, we develop and analyze a nested multiscale immuno-epidemiological HIV model that explicitly links within-host processes to epidemic outcomes. The model incorporates viral load–dependent transmission and treatment effects, allowing us to capture how immune responses and therapeutic interventions shape epidemic trajectories. We derive the basic reproduction number and establish threshold conditions for the existence and stability of the disease-free and endemic equilibria. We fit the multiscale model to within-host clinical data from 80 HIV-infected individuals and to national CDC surveillance data on incidence, diagnoses, and AIDS classifications. Our results show that reducing transmission from diagnosed individuals and increasing diagnosis rates could lead to epidemic declines consistent with EHE targets. This study highlights the importance of multiscale modeling in identifying intervention pathways and underscores the need for strengthened strategies to accelerate epidemic control.

MS29-07 — From Bird Viremia to Bird Surveillance: Identifiability in a Multiscale Vector-Borne Model of Usutu Virus Infection

SPEAKERS Quiyana Murphy (University of Michigan)
AUTHORS Quiyana Murphy (University of Michigan)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Usutu virus is an emerging mosquito-borne flavivirus, maintained through an enzootic cycle involving wild birds and mosquitoes, with occasional spillover to humans. Understanding how interactions across these biological scales shape transmission dynamics is essential for predicting outbreaks and improving surveillance strategies. In this study, we developed a multiscale vector-borne model of Usutu virus infection that links within-host viral kinetics in birds, the per-bite probability of mosquito infection, and population-level mosquito–bird transmission dynamics. Model parameters were validated using two laboratory datasets collected under an optimally designed experimental framework and one surveillance dataset from wild bird populations. Structural and practical identifiability analyses were conducted to evaluate parameter robustness under varying levels of measurement noise. We found that simultaneous multiscale fitting to integrated datasets improved parameter identifiability and robustness. These results highlight the importance of combining microscale and macroscale data to enhance the predictive reliability of vector-borne disease models and demonstrate the broader utility of multiscale modeling frameworks for understanding the transmission dynamics of emerging arboviruses.

MS29-08 — An immuno-epidemiological model with consideration for symptom score

SPEAKERS Summer Atkins (University of Alabama, Huntsville)
AUTHORS Binod Pant (Northeastern University); Hana Dobrovolny (Texas Christian University); Summer Atkins (University of Alabama, Huntsville)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

The incorporation of human behavior into epidemiological models has been quite a popular topic as of late, and there are many effective ways to mathematically introduce behavior into such models. In this talk, we incorporate behavior into an epidemiological model for an upper respiratory infectious disease based upon the following assumption: “The more sick a person is the more likely they will reduce their contacts”. Our methodology in capturing this behavioral assumption will be through a nested-multiscale modeling approach. The within-host dynamics of a symptomatic individual is represented by a system of ODEs in which one of the compartments measures the symptom score of the infected individual. This model is then

linked to an aged-structured between-host model (where age represents time since infection) by having a reduction in contact rate of symptomatic individuals be a function that depends on the individual's symptom score. In this talk, we present a flowchart of the model, some of the analytical results, and some simulations. In our simulations, we vary parameters of the linking function to investigate how the spread of the infection changes based on how reactive an individual is to their symptoms.

MS31 — Progresses in Mathematical and Computational Immunobiology and Infections

TRACK Immunobiology & Infection
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 02.21 - HS
ORGANISERS Alan Perelson (Los Alamos National Laboratory); Montana Ferita (University of Utah); Tin Phan (Los Alamos National Laboratory)

Minisymposium abstract

This minisymposium showcases concrete progress in understanding immunobiology and infections using mathematical and computational methods under real experimental and clinical constraints. The speakers work at the boundary of immunology, medicine, and quantitative science, combining mechanistic modeling with modern computational and statistical tools to provide insights into complex biological processes. Across talks, we emphasize testing hypothesis-driven models against data to improve what we can quantify and predict about infection and immune response.

MS31-01 — Revealing the influence of cell motility on immune processes within complex tissue environments

SPEAKERS Frederik Graw (Department of Medicine 5, Hematology and Oncology, University Hospital Erlangen, Friedrich-Alexander-Universität Erlangen-Nürnberg)
AUTHORS Frederik Graw (Department of Medicine 5, Hematology and Oncology, University Hospital Erlangen, Friedrich-Alexander-Universität Erlangen-Nürnberg); Laeschkir Würthner (Developmental Biology Unit, European Molecular Biology Laboratory)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Cell motility represents an important hallmark of immune processes governing cell interactions, communication and function. Determining how external factors, such as chemokine gradients and structural elements, influence immune cell motility is therefore of critical importance to understand the development of immune responses and their impact on pathological changes within complex tissue environments. Here, by combining individual cell-based models using the cellular Potts modelling (CPM) framework with theoretical analyses, we could reveal how the mode of cell motility is shaped by porous environments, as represented by extracellular matrices (ECM). Geometrical properties of these structures define characteristic cell motility regimes, with spatial heterogeneities in tissue environments effectively guiding cell movement and leading to nonhomogeneous cell distributions that can determine cellular interactions. By developing computationally efficient graph-based modelling approaches, we are able to combine them with time-lapse microscopy data on cell dynamics to infer and predict how host-pathogen interactions are shaped by complex tissue environments. Our analyses, as well as analyses of experimental data, illustrate the necessity to account for spatial interactions when aiming to determine the key processes governing disease progression and tissue pathology.

MS31-02 — Beyond MIC: Defining a Minimum Inhibitory Dose ("MID") for Periodic Antibiotic Treatments

SPEAKERS Jessica Conway (Penn State, USA)
AUTHORS Jessica Conway (Penn State, USA); Leah Childers (Penn State); Pia Abel zur Wiesch (Norway Institute of Public Health, University of Tromso)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Rising antimicrobial resistance drives the search for dosing strategies that maximize bacterial eradication while minimizing unnecessary drug exposure. Standard indices, such as the minimum inhibitory concentration (MIC) or the time spent above MIC ($\%T_{>MIC}$), are static snapshots that often fail to capture the time-varying nature of periodic dosing. To address this gap, we introduce the minimum inhibitory dose (MID), a dynamic MIC analogue defined as a threshold dose size, above which bacterial populations show net negative growth from one dose to the next. Using Floquet theory on a coupled bacterial-growth and antibiotic pharmacokinetic/pharmacodynamic (PK/PD) model, we calculate the MID as a function of the dose schedule and PK/PD properties. Our framework recapitulates established clinical guidelines: minimizing the dose period-normalized MID identifies frequent dosing as optimal for time-dependent antibiotics such as ampicillin and infrequent dosing for concentration-dependent antibiotics such as rifampin. Applying this framework to N. gonorrhoeae treated with ceftriaxone, the MID accurately predicts historical dose size adjustments driven by shifts in MIC and fitness costs. These results position the MID as a unifying, mechanism aware metric to guide future rational antibiotic dosing strategies.

MS31-03 — Hemogenic endothelium-derived multipotent progenitors shape prenatal hematopoiesis

SPEAKERS Connor Shrader (University of Utah)
AUTHORS Anna Beaudin (University of Utah); Connor Shrader (University of Utah); Frederick Adler (University of Utah); Nicole Tseng (University of Utah)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Hematopoiesis is the process by which blood and immune cells are formed. During fetal development, hematopoietic stem cells emerge from intra-embryonic aortic hemogenic endothelium (HE). We hypothesize that fetal hematopoietic stem cells (HSCs) and multipotent progenitors (MPPs) arise separately from HE, and that fetal MPPs contribute independently to postnatal blood production. However, the specific roles of these heterogeneous populations to fetal and postnatal hematopoiesis remain unclear. We used an inducible endothelial fate-mapping approach to mark cells emerging from the HE in mice at embryonic day (E)10.5 and track their contributions to hematopoiesis across time. HE-labeling was higher in MPPs compared to HSCs, suggesting that some fetal MPPs emerge independently from HE. To evaluate this hypothesis, we developed a mathematical model of HSC emergence, labeling, and proliferation in the developing mouse fetus. Our model lends support to the theory that MPPs emerge independently from HSCs. This method enables us to study how the timing of emergence from the HE affects hematopoietic cell fate. We will then use modeling and experiments to investigate the lasting impacts of prenatal factors, such as maternal infection, on hematopoiesis.

MS31-04 — Uncovering the T cell differentiation pathway

SPEAKERS Mason Lacy (Queensland University of Technology)
AUTHORS Adrianne Jenner (Queensland University of Technology); Mason Lacy (Queensland University of Technology); Pascal Buenzli (Queensland University of Technology (QUT)); Yoav Binenbaum (John A. Paulson School of Engineering and Applied Sciences, Harvard University)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Adoptive T cell therapy is a promising immunotherapy for treating cancer, leveraging the T cell's natural ability to kill cancer cells. A crucial step in this therapy involves the expansion of T cells ex vivo, however, it can be difficult to balance the desired amounts of memory, effector and dysfunctional T cell subtypes. Mathematical modelling of T cell expansion is a powerful tool that can be used to optimise expansion, assuming that the differentiation (to memory and effector) and dysfunction pathways are well-understood. Unfortunately, there is a large amount of uncertainty and disagreement in the literature describing the ways in which T cells differentiate to memory and effector cells, or become dysfunctional. I present one of the first large-scale mathematical investigations that aims to answer fundamental questions regarding

T cell differentiation and dysfunction. Using observations from biological literature, we constructed a list of potential model features which inform the structure of an ordinary differential equation model. We compare all possible models (7,280 unique pathways) to data describing the ex vivo expansion of T cell subtypes, and we determine the most plausible features of the T cell differentiation and dysfunction pathways. This framework allows for a deeper understanding of T cell behaviour during immune responses which may be exploited to improve the administration of adoptive cell therapy.

MS31-05 — Modelling the HBV immune response: insights from virtual humanised mouse cohorts

SPEAKERS Melanie Prague (Université de Bordeaux, Bordeaux Population health Inserm U1219, Inria, SISTM, Vaccine Research Institute)
AUTHORS Bastien Reyne (Université de Bordeaux, Bordeaux Population health Inserm U1219, Inria, SISTM, Vaccine Research Institute); Helene Strick-Marchand (Institut Pasteur, Inserm U1223); Linda Wittkop (Université de Bordeaux, Bordeaux Population health Inserm U1219, Inria, SISTM, Vaccine Research Institute); Melanie Prague (Université de Bordeaux, Bordeaux Population health Inserm U1219, Inria, SISTM, Vaccine Research Institute)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Hepatitis B infections become chronic in approximately 5% of adult infections and 80-90% of childhood infections. Once HBV has become chronic, no cure exists yet. Current clinical research aims to reach a functional cure, defined as the sustained loss of HBV surface antigens (HBsAg). Lately, the GLP-26, a capsid assembly modulator leading to the release of uninfectious empty particles, has shown a decrease in HBV DNA, HBsAg, and covalently closed circular DNA (cccDNA) in humanised mice. Based on two cohorts (comprising 27 mice in total), we have HBV DNA and HBsAg dynamics for durations ranging from 18 to 24 weeks. We propose using non-linear mixed effect mechanistic models to reproduce the observed data and quantify the treatment effectiveness and the intensity of the immune-induced cytotoxic response. Using virtual mouse cohorts generated with the mechanistic model, we explore alternative treatment strategies and explain the mechanisms leading to clearance in our simulations. Our model can reproduce the observed HBV DNA and HBsAg dynamics, suggesting the underlying mechanisms are well captured. GLP-26 inhibits the HBV replication cycle and reduces the production of HBsAg. Our results suggest that an increase in treatment duration would also increase the proportion of mice controlling their infections. While not reaching a 100% response, a key emergent property is that the treated mice that controlled the infection present a strong adaptive immune response. A possible explanation is that GLP-26 could act as a facilitator by decreasing circulating HBsAg, allowing the immune system to neutralise infected hepatocytes. This work consist as a proof of concept on the methodology to use to build a mechanistic virtual mice cohort.

MS31-06 — Mechanistic mathematical modeling of PD-1/IL-10 co-blockade predicts post-ART SIV control

AUTHORS Alan Perelson (Theoretical Biology and Biophysics, Los Alamos National Laboratory); Mirko Paiardini (Division of Microbiology and Immunology, Emory National Primate Research Center); Pedro Gerum (Operations & Supply Chain Management, Cleveland State University); Rafick Sekaly (Pathology Advanced Translational Research Unit, Department of Pathology and Laboratory Medicine, Emory University School of Medicine); Ruian Ke (Theoretical Biology and Biophysics, Los Alamos National Laboratory); Ruy Ribeiro (Theoretical Biology and Biophysics, Los Alamos National Laboratory); Susan Pereira-Ribeiro (Pathology Advanced Translational Research Unit, Department of Pathology and Laboratory Medicine, Emory University School of Medicine); Tin Phan (Theoretical Biology and Biophysics, Los Alamos National Laboratory); Zachar Strongin (Division of Microbiology and Immunology, Emory National Primate Research Center)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Dual IL-10/PD-1 blockade in ART-treated, SIVmac239-infected rhesus macaques (RMs) produced durable control of viral rebound after analytical treatment interruption (ATI). We analyzed the longitudinal data from 28 RMs randomized to vehicle (n=8), anti–IL-10 (n=10), or anti–IL-10+anti–PD-1 (n=10): plasma viremia, cell-associated vRNA/vDNA, intact proviral DNA (IPDA), and multiple immune markers were

assayed. We compared different model classes by the corrected Bayesian Information Criteria. We used in-silico cohorts to simulate trial-level outcomes (controller frequency, viremia, CA-DNA/IPDA). Machine learning analyses of simulated trajectories identified a minimal predictive signature and linked pre-ATI immune markers to key parameters. Our models captured plasma viremia and CA-vRNA trajectories and predicted CA-DNA and IPDA dynamics across all RMs. Anti–IL-10 increased infected-cell loss rates by 29–80%, while anti–PD-1 enhanced effector cell exhaustion reversal by 2.5–4.7-fold. Parameter distributions showed that viremia control was mainly driven by treatment effects rather than baseline differences. A four-parameter classifier learned from the in-silico cohorts achieved 90% accuracy in predicting controller status. Mechanistic modeling indicates IL-10/PD-1 co-blockade synergistically enhances effector-cell function and reduces rebound risk, explaining high controller frequency after ATI.

MS31-07 — Uncovering the mechanisms of SHIV dynamics in rhesus macaques undergoing immune therapy

SPEAKERS Stanca Ciupe (Virginia Tech)
AUTHORS Stanca Ciupe (Virginia Tech)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Understanding the mechanisms underlying post-therapy control of simian–human immunodeficiency virus (SHIV) is critical for the development of functional cure strategies. In this study, we develop viral kinetic models and validate them using viral load data from four cohorts of SHIV-infected nonhuman primates: untreated controls, animals receiving HuAd5 SIV Gag/Tat vaccination, animals treated with antibody therapy, and animals receiving combination therapy. Each model is fitted to longitudinal viral load measurements to quantify how different interventions alter viral dynamics. Using bifurcation analysis and numerical simulations, we identify parameter regimes in which therapy drives viral loads below the limit of detection. Furthermore, we characterize the conditions that enable sustained viral control after cessation or waning of immune-based interventions.

MS31-08 — Multiscale modelling identifies a predictive relationship between circulating biomarkers and antiviral efficacy of new treatments for chronic hepatitis B virus infection

SPEAKERS Tyler Cassidy (University of Leeds)
AUTHORS Tyler Cassidy (University of Leeds)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Chronic hepatitis B virus (HBV) infection is responsible for approximately one million deaths per year, worldwide. While existing therapies effectively block the production of new virus, viral rebound occurs rapidly if treatment is stopped. As a result, no cure for chronic HBV infection is currently available. Patients therefore typically receive treatment indefinitely with corresponding life-long treatment burden. Consequently, there is pressing need for new treatment options that block crucial steps in the viral life cycle. I'll show how multiscale mathematical modelling can illuminate biological mechanisms that would otherwise be inaccessible during typical clinical trials, identify circulating biomarkers of drug effectiveness, and help understand new treatments as possible functional cures for HBV. I'll show how this multiscale modelling can identify a simple predictive relationship between circulating biomarkers and drug efficacy.

MS31-09 — Accurate estimation of the frequency of asymptomatic SARS-CoV-2 infections from serosurveys

SPEAKERS Narendra M Dixit (Indian Institute of Science)
AUTHORS Narendra M Dixit (Indian Institute of Science)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Asymptomatic SARS-CoV-2 infections have contributed substantially to disease transmission during the COVID-19 pandemic. Estimating their frequency, denoted ψ, is important for understanding SARS-CoV-2 epidemiology and optimizing. Current estimates of ψ are inaccurate. Two-part serosurveys are used to estimate ψ. They employ assays to detect infection and then a questionnaire to ascertain symptoms. Of the individuals who test positive, the fraction declaring symptoms is taken as an estimate of ψ. Infection tests, however, are not perfect. Based on their sensitivity and specificity, they yield false positives and negatives. Further, the symptoms elicited by COVID-19 are not unique. They are also elicited by a host of other conditions, including influenza and common cold. Thus, serosurveys may misclassify individuals experiencing symptoms from other conditions as symptomatic for COVID-19, underestimating ψ. No formalisms have been proposed to correct for these confounding effects, despite the hundreds of serosurveys reported during the pandemic. We developed such a formalism. We obtained an analytical expression that employs data from serosurveys and yields an accurate estimate of ψ. We verified the formalism against synthetic datasets. Applying it to 50 COVID-19 serosurveys, we found that ψ was substantially larger (median 60%) than previously reported (40%). Further, our formalism showed that ψ became more consistent across surveys within nations and better associated with age. Our findings help better understand COVID-19 epidemiology and inform efforts to unravel origins of asymptomatic infections.

MS31-10 — Modeling the Interferon Responses to Influenza A Virus Infection

SPEAKERS Cailan Jeynes-Smith (Department of Pediatrics, University of Tennessee Health Science Center)
AUTHORS Amanda Smith (Department of Pediatrics, University of Tennessee Health Science Center); Amber Smith (Department of Pediatrics, University of Tennessee Health Science Center); Cailan Jeynes-Smith (Department of Pediatrics, University of Tennessee Health Science Center); Hai-Hsin Huang (Department of Mathematics, Virginia Military Institute); Ivan Ramirez-Zuniga (Department of Pediatrics, University of Tennessee Health Science Center); Meagan Herald (Department of Pediatrics, University of Tennessee Health Science Center); Nicole Bruce (Department of Pediatrics, University of Tennessee Health Science Center)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Influenza A virus infections are a major cause of human morbidity and mortality worldwide. A key aspect of the immune response, bridging innate and adaptive immunity, is the role of interferons. Here, we present experimental data identifying the primary producers of Type II interferon and providing novel insights into production rates via the integrated Median Fluorescence Intensity. This approach offers a new modelling opportunity, allowing us to quantify production rates at the population level and uncover production and binding mechanisms that would likely be overlooked from population data alone. Building on these insights, we present a novel viral dynamics model that incorporates Type II interferon production and binding. Using this framework, we investigate how interferon responses may be associated with infection severity and consider potential binding mechanisms that could contribute to this relationship.

MS31-11 — Towards a generalized framework for validating biological fidelity in immunobiological virtual patients

SPEAKERS Chapin Korosec (University of Guelph)
AUTHORS Chapin Korosec (University of Guelph)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

High-fidelity synthetic data is a critical frontier for disease modelling, yet few generalized methods exist to quantify whether generated virtual patients maintain physiological resemblance. Building on our previous work in machine learning-enabled immune profiling [Kor+26], we introduce a robust framework to assess the quality of synthetically generated immunobiological datasets. We deploy supervised and unsupervised generative methods, such as conditional variational autoencoders (cVAEs) and Gaussian

Mixture Models, to generate virtual patients across multiple medical and immunobiological datasets and develop an algorithm to assess synthetic data fidelity. To determine 'physiological resemblance' between synthetic and real data we implement a series of tests, including a discriminator-as-evaluator layer in the algorithm. In this approach, random forest (RF) classifiers are trained to distinguish real from synthetic data, with classifier performance serving as a proxy for generative success. When the classifier performs near chance, the synthetic dataset is effectively indistinguishable from the real data. Beyond straightforward validation, our framework provides diagnostic feedback by identifying specific physiological deviations, such as shifts in statistical properties or multidimensional interdependencies, thereby allowing for the iterative refinement towards high-fidelity virtual patients and ensuring transparency in synthetic data applications.

References

[Kor+26] Chapin S. Korosec et al. "Modeling of longitudinal immune profiles reveals distinct immunogenic signatures following five COVID-19 vaccinations among people living with HIV". In: *Patterns* 7.3 (2026), p. 101474. ISSN: 2666-3899. DOI: https://doi.org/10.1016/j.patter.2025.101474.

MS31-12 — Novel regulators found by network perturbation of sex-specific influenza A infection in clinical samples

SPEAKERS Jason E. Shoemaker (Department of Chemical and Petroleum Engineering, University of Pittsburgh)
AUTHORS Jason E. Shoemaker (Department of Chemical and Petroleum Engineering, University of Pittsburgh); Pariksheet Nanda (Department of Chemical and Petroleum Engineering, University of Pittsburgh)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Cellular protein interactome data are fundamental to determine mechanisms of pathogens, infection progression, and potential drug targets. Performing network analysis provides a data-driven middle ground between differential gene expression analysis, which assumes that gene expression is largely independent, and highly detailed mathematical models, which require careful measurements or estimation of parameters from training data. In the absence of detailed mathematical behaviors, network analysis has been shown to outperform differential gene expression analysis when gene interactions cannot largely be assumed to be independent. Here we reanalyze influenza A infection of patient-derived epithelial cell lines that include treatment with a sex hormone, estradiol. We show that network analysis results are largely divergent from differential analysis; network analysis enriches for cell cycle and estrogen signaling pathways whereas differential gene expression enriches for methylation, programmed cell death and meiosis pathways. Half the transcription factors we uncover were previously unstudied in viral infection and only one was previously been implicated in coronavirus replication. Finally, our network analysis significantly identifies relevant host factors in viral replication screens. Thus, we reveal the role of hormones in influenza A viral infection by identifying key signaling proteins and 9 transcription factors using our network modeling approach.

MS31-13 — Inside the wavering mind of an NK cell: Mathematical modeling of NK cell activation in health and disease

SPEAKERS Montana Ferita (Department of Mathematics, School of Biological Sciences)
AUTHORS Frederick Adler (Department of Mathematics, School of Biological Sciences); Montana Ferita (Department of Mathematics, School of Biological Sciences)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

The paradigm of NK cell activation is the "Missing-Self Hypothesis"; NK cells kill cells that lack MHC. When considering a single interaction between an NK cell and a target cell, the "Missing-Self Hypothesis" offers a plausible explanation for NK cell activation. However, an NK cell's interaction with a target cell is not an isolated event. Experimental studies have shown that NK cells in an MHC-deficient environment fail to lyse cells lacking MHC. Curiously, a study by the Yokoyama lab (Washington University in St. Louis) found that when 10% of cells in a well-mixed environment lacked MHC, the NK cells failed to lyse most of the cells. Our hypothesis is that target cell recognition is less about the balance of the presence or absence of markers, and more so about detecting outliers in an environment. We developed mathematical models of how NK cells assess and learn from their environment under static and dynamic MHC suppression. Our results suggest NK cells may be vulnerable to "slow growing danger" which makes detecting outliers challenging. We are collaborating with the Weis lab (Huntsman Cancer Institute) to experimentally test our model predictions.

MS31-14 — From binding to neutralization: A mathematical model for flavivirus antibody responses

SPEAKERS Nathanael Hoze (Institut national de la santé et de la recherche médicale)
AUTHORS Nathanael Hoze (Institut national de la santé et de la recherche médicale)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Flaviviruses such as Dengue, Zika, Yellow Fever and West Nile virus represent major global public health threats. Envelope (E) proteins on the viral surface mediate host-cell attachment and membrane fusion. These 180 E proteins are arranged in a highly ordered geometry that shapes epitope accessibility and antibody binding.

Neutralization of flaviviruses is primarily mediated by monoclonal antibodies (mAb) targeting the E protein in an interplay between mAb affinity, epitope accessibility, and the number and organization of bound mAb.

We present preliminary results on a mathematical model of seroneutralization. We model virus–antibody interactions as a stochastic binding process that accounts for epitope spatial organization and occupancy on the viral surface. We include conformational transitions of the E protein and viral "breathing" dynamics, which modulate epitope accessibility and constrain binding kinetics. By introducing these structural and dynamical constraints, our approach aims to provide a mechanistic interpretation of neutralization curves grounded in the geometry and physics of the virion.

In parallel, we have developed a database of seroneutralization data for many measurements from mAbs, cells and flaviviruses. Using this resource, we explore mechanistic links between microscopic binding parameters and the shape of neutralization curves. Our framework aims to account for key features observed experimentally, including partial neutralization and neutralization by antibody mixtures.

This approach is designed to bridge quantitative modeling with practical questions in public health, including antibody-dependent enhancement (ADE) and the interpretation of polyclonal immune responses.

MS31-15 — Mathematical and Mouse Models Identify T Regulatory Cell Influx as A Key Determinant of Acquired Resistance to PD-1 Immunotherapy

SPEAKERS Rachel Sousa (Institute for Immunology, University of California Irvine)
AUTHORS Rachel Sousa (Institute for Immunology, University of California Irvine)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

The immune system can eradicate cancer, but various immunosuppressive mechanisms active within a tumor curb this beneficial response. However, unraveling the effects of multimodal interactions between

tumor and immune cells and their contributions to tumor control using an experimental approach alone is time- and resource-intensive. To identify the critical immunological features associated with tumor control and escape, we built a mechanistic, structurally identifiable mathematical model of the interactions between CD8+ T cells, Tregs, DCs, and tumor cells deeply rooted in current biological concepts. A distinguishing feature of our model is that it captures Treg accrual occurring after checkpoint blockade immunotherapy. After successfully fitting the mathematical model to experimental data from an immunogenic melanoma mouse model that shows acquired resistance to PD-1 immunotherapy and ensuring that the model was practically identifiable, we generated hundreds of parameter sets, each of which fits the data well and represents a unique 'virtual mouse' to capture variability across individuals. Our model indicates that the initial tumor and immune conditions instruct cancer control or progression and that there exist optimal initial ratios of immune cells that result in improved tumor control. The model further predicts that the Treg influx into the tumor is a key determinant of resistance to PD-1 immunotherapy. We validated all of these predictions experimentally. Overall, this integrated approach of modeling and experimental validation identified crucial determinants of resistance to PD-1 immunotherapy and can be used to guide the development of more effective therapeutic strategies.

MS31-16 — Decoding Influenza Transmission from Ferret Contact and Viral Dynamics

SPEAKERS Veronika Zarnitsyna (Emory University School of Medicine)
AUTHORS Veronika Zarnitsyna (Emory University School of Medicine)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

The determinants of influenza transmission at the level of individual contacts remain poorly understood. We analyze a unique dataset of controlled ferret transmission experiments in which one or two infected donors interact with four susceptible recipients for varying durations (typically 1–4 hours), combining high-resolution video and longitudinal viral load measurements to link time-resolved behavior with infection outcomes.

Our approach integrates machine learning–based behavioral tracking using Social LEAP Estimates Animal Poses (SLEAP) with statistical and mechanistic modeling to quantify how contact patterns influence infection risk. We reconstruct individual-level exposure histories and relate them to infection outcomes to estimate associations between specific contact patterns and transmission, and to explore parsimonious transmission-kernel formulations.

The primary objective is to determine which features of behavior and infectiousness are identifiable from these data, and which are fundamentally confounded. More broadly, this work asks a general question: given realistic behavioral and biological data, what can we actually learn about transmission mechanisms?

MS39 — Structural Approaches to the Dynamics of Chemical Reaction Networks

TRACK Systems Biology and Biochemical Networks
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 11.03 - HS
ORGANISERS Ankit Gupta (ETH Zürich); Atsushi Mochizuki (Kyoto University); Polly Yu (University of Illinois Urbana-Champaign)

Minisymposium abstract

Biological functions emerge from the dynamics of complex chemical reaction networks comprising of multiple interacting biomolecules within cells. Understanding the dynamics of such chemical reaction systems is therefore central to elucidating biological function. However, the inherent complexity of these systems has made rigorous analytical characterisation of their dynamical behaviour extremely challenging.

In recent years, a range of mathematical frameworks has been developed to analyse chemical reaction systems based solely on the structural properties of their underlying networks. These approaches draw on linear algebraic and graph-theoretic concepts, enabling qualitative and quantitative insights into system behavior based on their network structure, in a parameter-independent and model-independent manner. Such structural theories not only open new frontiers in mathematical science but also offer promising avenues for advancing our understanding of complex biological systems.

This mini-symposium brings together leading researchers investigating the dynamics of chemical reaction networks using these and related mathematical methods. It aims to foster close interdisciplinary discussions from both mathematical and biological perspectives. We anticipate that this mini-symposium will stimulate progress in interdisciplinary research and contribute to advances in both mathematics and the life sciences.

MS39-01 — Manifold robust perfect adaptation and structural limits on steady-state reachability

SPEAKERS Ankit Gupta (ETH Zürich)
AUTHORS Ankit Gupta (ETH Zürich); Yuji Hirono (University of Tsukuba); Mustafa Khammash (ETH Zurich)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Biochemical reaction networks often display robust responses to persistent perturbations despite high dimensionality, feedback, and uncertain kinetics. Classical robust perfect adaptation (RPA) captures one important mechanism: after sustained input changes, a designated output returns to a fixed steady state independent of perturbation magnitude. However, many cellular systems do not relax to a single adapted point. Instead, their steady states lie on lower-dimensional sets such as lines or planes, indicating regulation of relations between species rather than fixed absolute levels.

We introduce manifold robust perfect adaptation (manifold RPA), a framework in which persistent perturbations drive the system to an invariant manifold in steady-state concentration space. We show that manifold RPA can arise as a structural property of biochemical reaction networks and derive sufficient conditions for it without assuming specific kinetic forms. This yields a rigorous characterisation of adaptive behaviour that preserves ratios or linear relations among molecular species while allowing absolute concentrations to vary.

Our results extend the classical notion of adaptation and reveal structural constraints on steady-state reachability imposed by network architecture. This provides a principled explanation for non-pointwise steady-state responses in metabolism and signalling, and suggests a design principle for synthetic biological circuits requiring adaptive behaviour under relational, rather than fixed, targets.

MS39-02 — Biological functions and functional modules originated in structure of chemical reaction network.

SPEAKERS Atsushi Mochizuki (Kyoto University)
AUTHORS Atsushi Mochizuki (Kyoto University); Yuhei Yamauchi (Kyoto University)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In living cells, numerous chemical reactions are interconnected by sharing substrates and products, forming a reaction network. Various functions of cells emerge from dynamics of such interconnected system. Cells regulate amount of key chemicals by controlling amount/activity of enzymes, thereby achieving control of cellular functions. However, in such an interconnected system, can different chemicals responsible for different biological functions be controlled independently? We mathematically demonstrate that “modularity”, where parts of a system are controlled independently of others, arises solely from network topology. We found that the qualitative response of chemical concentrations to changes in enzyme amount/activity are localized to finite ranges in a network, and each range is determined by a

subnetwork called a "buffering structure", that is defined by the equation $\chi \vDash -(\#\text{ of chemicals})+(\#\text{ of reactions})-(\#\text{ of cycles})+(\#\text{ of conserved quantities})= 0$ from local topology of a network. Using the cell cycle system as an example, we show that buffering structures actually exist in living organisms, performing important roles. In the cell cycle, the G1-S and G2-M transitions are strictly controlled by distinct protein complexes, requiring the activation of different complexes at different phases. Analysis of the cell cycle network revealed that the two complexes belong to different buffering structures. Moreover, by comparing theoretical predictions with experimental verification, we theoretically predict the necessity of an unknown reaction and experimentally confirm it.

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MS39-06 — The structural concept(s) of cores in reaction networks

SPEAKERS Nicola Vassena (University of Leipzig)
AUTHORS Alex Blokhuis (IMDEA Nanoscience Madrid); Nicola Vassena (University of Leipzig); Peter F. Stadler (University Leipzig)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In recent years, there have been a few uses of the word 'core' in the context of chemical reaction networks, all related to some idea of minimality. Based on stoichiometric considerations alone (i.e. no dynamics nor kinetic considerations involved), Alex Blokhuis with co-authors identified autocatalytic cores as minimal structures carrying autocatalysis, a fundamental biochemical concept related to self-amplification. To effectively connect stoichiometric intuitions to dynamical conclusions, I introduced with Peter Stadler the framework of parameter-rich kinetics, which allows disentangling parametric dependence from the structural analysis of the Jacobian matrix. Widely used kinetics in biochemistry, such as Michaelis–Menten or generalized mass action, are naturally parameter-rich. Within this framework, we investigated minimal stoichiometric structures whose presence guarantees the possibility of dynamical instability (that is, the capacity for a locally unstable steady state). Since the autocatalytic cores sensu Blokhuis form a strict subset of these structures, we named them unstable cores. Finally, with both Peter Stadler and Alex Blokhuis, we addressed periodic oscillations, introducing the concept of oscillatory cores, i.e. minimal structures that guarantee periodic oscillations in any network containing them, again under parameter-rich kinetics. In this talk, I will give an overview of these and related results.

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MS39-04 — Cycling Reaction Network Robustness Analysis

SPEAKERS Franco Blanchini (University of Udine)
AUTHORS Erica Salvato (University of Trieste); Franco Blanchini (University of Udine)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Cycling reaction networks are ubiquitous in nature, underpinning key processes such as the Krebs and Kelvin cycles. In this talk, we analyze the robustness of such networks. Under the regularity assumption—that all supplied species are subject to degradation—we show that the dynamics converge to a consensus state, where all external and internal flows equalize while species concentrations remain bounded. Our proof relies on a suitably constructed piecewise–linear Lyapunov function in the reaction rates. The non-regular case is substantially more delicate, since concentrations of external species may diverge. We show that all internal reaction rates still converge to a consensus value. If the concentration of the minimal-input node remains bounded, the consensus value coincides with the minimal input. Otherwise, the rates converge to a common value that does not exceed it

MS39-05 — Structural properties and biological oscillators: ideas from strongly 2-cooperative systems

SPEAKERS Giulia Giordano (University of Trento)
AUTHORS Giulia Giordano (University of Trento); Michael Margaliot (Tel Aviv University); Rami Katz (Tel Aviv University)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Fundamental properties and emergent behaviours of biochemical systems often depend exclusively on the system structure (the graph topology along with qualitative information), regardless of parameter values. We first provide an overview of the parameter-free assessment of important properties, including the stability of equilibria and the sign of steady-state input-output influences. Then, we focus on the emergence of sustained oscillations via convergence to periodic orbits, a complex question with important applications in systems biology, including the understanding of biomolecular oscillators that rule cell life cycle and metabolism, as well as circadian rhythms in hormone secretion, body temperature and metabolic functions. The study of sustained oscillations in a dynamical system requires first showing that at least one periodic orbit exists and then assessing the stability of periodic orbits and characterising the initial conditions from which the solutions converge to periodic trajectories. For a class of strongly 2-cooperative nonlinear dynamical systems, leveraging results from the theory of cones, the spectral theory of totally positive matrices and Perron-Frobenius theory, we show that every solution emanating from an explicit set of initial conditions of positive measure converges to a periodic orbit. The result applies to well-known

biological systems, including the n-dimensional Goodwin oscillator and biological oscillators based on RNA-mediated regulation.

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MS39-03 — Monomial parametrizations and absolute concentration robustness in reaction networks

SPEAKERS Elisenda Feliu (University of Copenhagen)
AUTHORS Beatriz Pascual Escudero (Universidad Politécnica de Madrid); Elisenda Feliu (University of Copenhagen); Oskar Henriksson (Max Planck Institute)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In this talk I will share recent results on how to detect whether a reaction network with mass-action kinetics admits a monomial parametrization or displays absolute concentration robustness. When requiring that either property arises in an open subset of parameter space, both problems can be approached by an initial check using linear algebra, and a second check where a more detailed analysis of the steady state variety is required. The linear algebra check provides a necessary condition, and therefore lack of both properties can be easily detected in many cases.

MS39-07 — Non-monotonic dose-response curve in biochemical systems

SPEAKERS Polly Yu (University of Illinois Urbana-Champaign)
AUTHORS Eduardo Sontag (Northeastern University); Polly Yu (University of Illinois Urbana-Champaign)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

A response curve measures the output of a biological system at equilibrium against an input parameter u, which could be a rate constant, or amount of a stimulus (poison, drug, ligand, etc.). Of interest is the shape of a response curve: does it have a plateau region (homeostasis); is it monotonic? In T-cell's response to antigen, a non-monotonic (i.e., biphasic) response has been observed. For systems where u directly affects one variable, we previously proved that either an incoherent feedforward loop, or a combination of positive and negative feedback loops, is necessary for biphasic response. In this talk, we consider the case where u impacts multiple variables, for example if u is the rate constant of a reaction.

Finally, we comment more generally on translating motifs between the influence diagram and the reaction network.

MS39-08 — A Structural Approach to Identifying Indicator Species in Chemical Reaction Networks

SPEAKERS Yong-Jin Huang (Kyoto University)
AUTHORS Atsushi Mochizuki (Institute for Life and Medical Sciences, Kyoto University); Takashi Okada (Institute for Life and Medical Sciences, Kyoto University; Interdisciplinary Theoretical and Mathematical Sciences Program (iTHEMs), Riken); Yong-Jin Huang (Kyoto University)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Cellular phenotypes exhibit remarkable diversity, reflecting the complex functional states of individual cells. Although phenotypic diversity has traditionally been assessed at the transcriptomic level, recent advances in single-cell technologies have shifted attention toward metabolomic phenotyping, which provides a more direct reflection of cellular function. Nonetheless, the high dimensionality of the metabolome poses significant challenges for both measurement and computational classification. A fundamental question therefore arises: which subset of species in a reaction network suffices to represent the system's overall state? To address this, we develop a novel theory that relies solely on the structural topology of chemical reaction networks to identify indicator species—those whose concentrations uniquely determine all others and thereby distinguish multistable equilibria. An efficient algorithm implementing this theory is applied to biochemical pathway databases. Numerical experiments show that phenotypic classification using only these indicator species achieves accuracy that matches or exceeds that of the full metabolite set, while demonstrating superior robustness to measurement noise. These results establish a rigorous, topology-based foundation for indicator species identifications, advancing metabolomic phenotyping and biomarker discovery.

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MS41 — Methods in applied population dynamics

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Monday, 13 July 2026; 15:00–16:20; 15.06 - HS
ORGANISERS Claudia Ferreira (UNESP)

Minisymposium abstract

In this minisymposium, we will explore a range of mathematical and computational approaches for modeling population dynamics. Topics will include agent-based models, differential equations, qualitative analysis, and evolutionary dynamics, with an emphasis on applications to real-world biological and ecological systems, particularly in the context of insect pest management.

MS41-01 — Bifurcation Structures in Low-Dimensional Models of Ant–Coffee Berry Borer Dynamic

SPEAKERS Gerard Olivar-Tost (UCM)
AUTHORS Carlos Andrés Trujillo-Salazar (Universidad del Quindío, Colombia); Deissy Milena Sotelo-Castelblanco (Universidad Nacional de Colombia); Gerard Olivar-Tost (UCM)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 15.06 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Ant communities play a key role in the natural regulation of agricultural pests, acting through collective behavior and non-linear feedback at the ecosystem level. In coffee agroecosystems, interactions between ant populations and the Coffee Berry Borer (CBB) provide a paradigmatic example of biologically mediated pest suppression driven by low-dimensional dynamics rather than large-scale complexity. In this talk, we present a class of minimal dynamical systems that describe ant–CBB interactions, focusing on the qualitative structure of the models. Using piecewise-smooth systems of ordinary differential equations, we analyze equilibrium configurations, stability properties, and bifurcation structures in response to changes in key ecological parameters. Our results reveal multiple dynamical regimes, including pest persistence, effective biological control, and bistability between controlled and outbreak states. Transitions between these regimes are governed by bifurcations that provide a mechanistic explanation for changes in pest pressure observed in managed coffee systems. The analysis highlights how collective ant behavior can induce nonlinear threshold effects leading to robust pest suppression. This work illustrates how low-dimensional dynamical models can capture essential features of complex agroecosystems and provides a theoretical framework for understanding biologically driven control strategies from a mathematical biology perspective [ALS23; Di +08; DF08; TOS23].

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MS41-02 — SIT-based control of Aedes aegypti mosquitoes featuring spatial heterogeneity

SPEAKERS Olga Vasilieva (Universidad Del Valle)
AUTHORS Olga Vasilieva (Universidad Del Valle); Oscar E. Escobar-Lasso (Universidad del Valle); Reinhard Racke (Universität Konstanz); Stefan Frei (Universität Konstanz)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 15.06 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The Sterile Insect Technique (SIT) is an environmentally friendly strategy for suppressing mosquito populations that transmit diseases such as dengue, Zika, and Chikungunya. We investigate SIT-based control of *Aedes aegypti* mosquitoes using a spatially explicit reaction–diffusion model describing the dynamics of wild males, wild females, and released sterile males. The model incorporates mosquito dispersal, together with a biologically motivated Ricker-type density-dependent recruitment function that

captures the regulation of mosquito reproduction due to competition and resource limitations at earlier aquatic stages, and allows for spatially heterogeneous releases of sterile males. Numerical simulations show how the magnitude, spatial distribution, and timing of sterile male releases affect suppression outcomes. The results confirm that the critical release threshold previously identified in an ODE model without spatial mosquito dispersal [Bli+19] persists in the spatially explicit setting, separating two long-term regimes: persistence of the wild mosquito population at a reduced equilibrium or its eventual elimination. Spatial heterogeneity strongly influences the dynamics. Uniformly distributed releases tend to produce faster suppression, whereas poorly located release sites may allow mosquitoes to persist locally. These findings highlight the importance of spatially informed release strategies for effective SIT programs in heterogeneous environments.

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MS41-03 — About the Combination of Control Tools Against the Oriental Fruit Fly Using a Metapopulation Approach

SPEAKERS Yves Dumont (CIRAD, AMAP, University of Pretoria)
AUTHORS Manon de la Tousche (Sorbonne Université, AMAP, EPC MUSCLEES); Pierre-Alexandre Bliman (Sorbonne Université, EPC MUSCLEES); Yves Dumont (CIRAD, AMAP, University of Pretoria)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 15.06 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The oriental fruit fly, Bactrocera dorsalis, is a major invasive pest and classified as a quarantine pest by the European Union. First recorded in La Réunion in April 2017, it is now established and causes serious damage, particularly in mango orchards. Due to the high biodiversity of La Réunion, chemical control is highly restricted, so only non-chemical tools can be used, such as the Male Annihilation Technique (using Methyl-eugenol traps) and soil entomopathogen fungi applied via irrigation systems to target the pupal and adult stages. Additionally, the Sterile Insect Technique (SIT) has been under investigation since 2020. It consists of mass rearing, irradiation, and release of sterilized insects to compete with wild populations, reducing reproduction rates.

All these control approaches have been studied using mathematical models [BDD25; BTD25; Dum25]. The aim of this talk is to present new mathematical results, illustrated with simulations, where the combination of these control tools is analyzed to derive optimal strategies in space and time.

The authors acknowledge the support of the European Regional Development Fund (ERDF). YD acknowledges the support of the BactroTIS project, grant number ANR-25-ECOM-0031-01, and the DST/NRF M3B2 SARChI Chair at the University of Pretoria (Grant 82770).

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MS41-04 — Modelling Movement and Stage-Specific Habitat Preferences of a Polyphagous Insect Pest

SPEAKERS claudia ferreira (UNESP)
AUTHORS claudia ferreira (UNESP)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 15.06 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The feeding preferences of *Diabrotica speciosa* (Coleoptera: Chrysomelidae) generate a parent-offspring conflict, as selecting the optimal host for offspring development can negatively affect adult survival and fecundity. Understanding this conflict is essential for developing effective pest-management strategies. We investigated the foraging behavior of *D. speciosa* using an individual-based model in two scenarios. In an intercropping scenario, we simulated parent-offspring conflict with adults exploiting two crops (corn and soybean) providing distinct nutritional advantages for each life stage. Three adult dispersal strategies were compared under continuous oviposition: simple diffusion, attraction to a fixed host, and alternating between hosts with a foraging period τ per crop. Two behavioral principles were explored: "mother knows best" (adults foraging on corn during oviposition) and "optimal bad motherhood" (adults foraging on soybean to maximize their own fitness), including pre-oviposition effects. In a landscape scenario, population dynamics were simulated across four crop plots with temporal changes. Results indicated that crop-alternating dispersal near an optimal τ maximized population performance, and the "mother knows best" strategy supported higher population growth than "optimal bad motherhood." Landscape heterogeneity, fallow periods, and reduced soybean monocultures lowered insect density. Our findings suggest that alternating crop foraging enhances population fitness, while spatial and temporal crop management can effectively mitigate *D. speciosa* infestations [Gar+20].

References

[Gar+20] Adriano G Garcia et al. "Modelling movement and stage-specific habitat preferences of a polyphagous insect pest". In: *Movement Ecology* 8.1 (2020), p. 13.

MS42 — Insights into cell-tissue interactions via mathematical modelling and computational simulations

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 02.01 - HS
ORGANISERS Qiyao (Alice) Peng (Lancaster University); Raluca Eftimie (University of Franche-Comte); Dumitru Trucu (University of Dundee)

Minisymposium abstract

Cell-tissue interactions, including both chemical and mechanical interactions, are essential and crucially important for various biological processes, such as functioning organs, regenerating tissues and regulating cellular behaviours. In the past years, mathematical modelling has shown its great potential to further understand the biology and then predict the behaviours of cells and tissue, which is particularly beneficial to study diseases such as wound healing, cancer cell metastasis and tumour growth. Considering the difference of scales between the cell and tissue, typically multiscale and multiphysics modelling is required to describe the biological phenomenon.

In this mini-symposium, we aim to bring researchers working in the field of modelling cell-tissue interactions, with applications in cell migration, tissue morphogenesis and diffusion of cytokines etc. The contributed talks will cover various modelling frameworks, ranging from lattice-based to off-lattice, and from agent-based to continuum models.

MS42-01 — Spatial organization of adhesion-deficient cells governs multiscale rigidity transitions in epithelial tissues

SPEAKERS Pilar Guerrero (Universidad Carlos III de Madrid)
AUTHORS Natalia Briñas-Pascual (Universidad Carlos III de Madrid); Pilar Guerrero (Universidad Carlos III de Madrid); Victor Manso (Institut d'investigació biomèdica de Bellvitge (IDIBELL))
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Epithelial tissues maintain structural integrity through a balance between cell-cell adhesion and cortical contractility. Disruption of E-cadherin-mediated adhesion is a hallmark of metastatic progression, yet the physical mechanisms by which local molecular defects escalate into global tissue destabilization remain poorly understood. Traditional theoretical studies have focused on the density of defective cells, while largely neglecting their spatial organization—a critical factor in clonal expansion.We employ a 2D stochastic vertex model to investigate how the spatial arrangement of adhesion-deficient cells dictates tissue-scale rigidity transitions. By comparing random and clustered distributions, we identify a distinct mechanical response across scales. Our results show that while isolated defects are rapidly eliminated via T_2 extrusion events with minimal impact on tissue architecture, clustered defects significantly delay extrusion kinetics. This "shielding effect" fosters the emergence of local fluidization zones characterized by a high cellular shape index ($q > 3.81$). Furthermore, we demonstrate that clustered defects leave a "topological scar" in the wild-type matrix, lowering the global threshold for the unjamming transition. By linking local molecular dysfunction to macroscopic fluidization, our framework provides new insights into how clonal clustering facilitates tissue invasion and metastasis.

References

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[BMG24] N. Briñas-Pascual, V. Manso, and P. Guerrero. "Spatial organisation of adhesion-deficient cells controls epithelial rigidity transitions". In: *Preprint / In preparation* (2024).

[Ma16] T. Mestre and et al. "Quantification of topological features in cell meshes to explore E-cadherin dysfunction". In: *Scientific Reports* 6 (2016), p. 25101.

[Pa15] J.-A. Park and et al. "Unjamming and cell shape in the asthmatic airway epithelium". In: *Nature Materials* 14 (2015), pp. 1040–1048.

MS42-02 — Modelling single and collective cell migration through confined non-isotropic environments using geometric surface PDEs

SPEAKERS Anotida Madzamuse
AUTHORS Anotida Madzamuse
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In this talk, I will introduce a modelling approach for single and collective cell migration through confined non-isotropic environments using geometric surface partial differential equations. By assuming that cell migration is driven by cell surface biochemical processes and surface mechanics, the evolution law of the cell and nuclear envelope is modelled through a force balance equation posed at each surface material point in the normal direction. The force balance equation naturally encodes most of the biophysical properties of energetic closed surfaces such as surface tension, bending energy, surface

area/volume constraint/conservation as well as taking into account intra- and extra-cellular forces, cell-to-cell interactions, cell-to-environment interactions, and so forth. This modelling approach leads to 4th-order geometric surface partial differential equations which are solved efficiently by employing an operator-splitting approach within an evolving surface finite element method. Numerical simulations demonstrate the generality, applicability and predictive power of this modelling approach; it offers a new and robust modelling formalism that bridges the gap between experiments and theory of single and collective cell migration through biologically relevant non-isotropic and confined environments.

MS42-03 — Mathematical Models for the Mechanics of Soft Tissues: From Linear Elasticity to Morpho-Visco-Poroelasticity

SPEAKERS Sabia Asghar (Hasselt University)
AUTHORS Sabia Asghar (Hasselt University); Qiyao (Aliec) Peng (Lancaster University); Etelvina Javierre (University of Zaragoza, Spain); Fred Vermolen (HASSELT UNIVERSITY)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Biological tissues are often subjected to forces. In many cases, such as tumor growth or skin contraction, it is crucially important to model the state of tissues that are exposed to forces in order to improve or optimize therapies for different pathologies. The simplest models use linear elasticity as a constitutive law. This linearity enables the use of the superposition principle and the use of fundamental solutions to analyze the influence of multiple points of action of forces. A clear illustration of this principle is the immersed interface method [RDB20]. In this presentation, we discuss this principle in terms of convergence properties using the singularity removal principle [GKN19].

However, in real-life tissues, the use of linear elasticity is too restrictive due to the presence of moisture and the porous structure of biological tissues. Furthermore, in various biomedical cases, the microstructure of the tissue changes due to cellular activity. For this reason, we construct and use a model that consists of elasticity, porosity and microstructural changes. The mathematical framework is referred to as morpho-visco-poroelasticity [Hal08]. This framework is original and for this reason, we analyze this framework in terms of stability around equilibria [AV25]. Since numerical solutions can be characterized by spurious oscillations, we provide conditions for monotonicity by mathematical analysis. Furthermore, we propose a numerical stabilization method to avoid spurious oscillations on forehand.

References

[AV25] Sabia Asghar and Fred J Vermolen. “Stability of Equilibria in a One Dimensional Model for Morpho–Poroelasticity for Soft Tissues”. In: *Numerical Mathematics and Advanced Applications ENUMATH 2023, Volume 2*. Ed. by Adélia Sequeira et al. Cham: Springer Nature Switzerland, 2025, pp. 468–476. ISBN: 978-3-031-86169-7. DOI: 10.1007/978-3-031-86169-7_48.

[GKN19] Ingeborg G. Gjerde, Kundan Kumar, and Jan M. Nordbotten. “A singularity removal method for coupled 1D–3D flow models”. In: *Comput. Geosci.* 24.2 (Dec. 2019), pp. 443–457. ISSN: 1573-1499. DOI: 10.1007/s10596-019-09899-4. URL: http://dx.doi.org/10.1007/s10596-019-09899-4.

[Hal08] C L Hall. “Modelling of some biological materials using continuum mechanics”. PhD thesis. Queensland University of Technology, 2008.

[RDB20] Somnath Roy, Ashoke De, and Elias Balaras. *Immersed boundary method*. Computational Methods in Engineering & the Sciences. Singapore: Springer, 2020. DOI: https://doi.org/10.1007/978-981-15-3940-4.

MS42-04 — Contact guidance and ECM architecture jointly regulate cancer invasion

SPEAKERS Fabian Spill
AUTHORS Fabian Spill; Margherita Botticelli (University of Birmingham); John Metzcar (University of Minnesota); Paul Macklin (Indiana University); Pradeep Keshavanarayana (University College London)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cancer invasion emerges from coupled interactions between tumour cells and the extracellular matrix (ECM), where fibre architecture, remodelling, and microenvironmental cues jointly regulate migration and growth. Building on our earlier PhysiCell framework for collagen-density-dependent spheroid invasion, we present a hybrid discrete-continuous model that incorporates fibre orientation, anisotropy, contact guidance, chemotaxis, cell-front ECM sensing, matrix displacement/degradation, and proliferation regulated by oxygen and mechanical pressure. Simulations show that invasion is enhanced when ECM fibres provide coherent directional cues: perpendicular or radial organisation promotes outward invasion, whereas parallel or tangential organisation suppresses it. Crucially, fibre anisotropy determines whether aligned architectures are effectively translated into directed motion. We further show that ECM remodelling has context-dependent effects: increasing reorientation can rescue invasion in initially restrictive matrices, but diminish invasion when the initial architecture is already favourable. The model reproduces experimentally motivated qualitative trends and yields mechanistic predictions on how tumour cells reshape and exploit ECM structure during collective invasion. These results provide a computational framework for dissecting tumour–ECM feedbacks and for designing experiments on matrix-guided invasion.

References

[Bot+25a] Margherita Botticelli et al. “A hybrid computational model of cancer spheroid growth with ribose-induced collagen stiffening”. In: *Frontiers in Bioengineering and Biotechnology* Volume 13 - 2025 (2025). ISSN: 2296-4185. DOI: 10.3389/fbioe.2025.1515962. URL: https://www.frontiersin.org/journals/bioengineering-and-biotechnology/articles/10.3389/fbioe.2025.1515962.

[Bot+25b] Margherita Botticelli et al. “The role of contact guidance and ECM remodelling in cancer invasion: a computational study”. In: *bioRxiv* (2025), pp. 2025–09.

MS42-05 — Emergence of Cellular Stress and Metabolic Dynamics within GBM progression

SPEAKERS Dumitru Trucu (University of Dundee)
AUTHORS Dumitru Trucu (University of Dundee)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cancer cells in the GBM microenvironment often experience stress due to multiple sources, primarily hypoxia (low nutrient availability, i.e., low oxygen and other nutrients components) and therapeutic interventions such as chemotherapy and radiotherapy. Indeed, while cellular death is the main intended outcome of therapeutic strategies, this also occurs within excessive hypoxic regions (such as necrotic zones) even in the absence of therapy. However, both of these two means that lead to cell death, turn out to be major sources of stress for the GBM cells ecosystem, triggering phenotypic cell adaptations, including the development of resistance to therapy. To address this from a modelling point of view, in this work we propose a multiscale moving boundary framework to explore the cellular stress state within the tumour cell population and its implications in terms of tumour mutations and emerging resistant cells subpopulations. This modelling is then extended to account for the broader metabolic profile of the GBM tumour microenvironment.

MS42-08 — Modelling the dynamics of focal adhesions in cell migration using geometric surface PDEs

SPEAKERS Sofie Verhees (University of British Columbia)
AUTHORS Sofie Verhees (University of British Columbia); Anotida Madzvamuse (University of British Columbia); Miranda Holmes-Cerfon (University of British Columbia); Daniel Bouvard (CNRS); Marion Lautier (CNRS)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cell migration is a complex biological process underlying phenomena such as wound healing, tissue morphogenesis, and cancer invasion. A key component of this process is the formation and turnover of focal adhesions, which provides a mechanical coupling between the cytoskeleton and the extracellular matrix. Actin filaments anchored at focal adhesions generate forces on the cell membrane, driving the formation of protrusions that regulates cell movement.

In this work, we develop a mathematical framework based on geometric surface partial differential equations (GS-PDEs) to investigate the role of focal adhesions in cell migration. GS-PDE models have shown strong potential in describing cell shape evolution and migratory behaviour. Building on this approach, we incorporate the maturation and dynamics of focal adhesions and examine their interplay with membrane forces generated by actin polymerisation at the leading edge. Our model enables us to study how focal adhesion growth and turnover influence protrusion formation and overall cell motility. This framework provides new insight into the mechanical processes governing cell migration.

MS42-07 — Multiphase cross-diffusion models for tissue structures

SPEAKERS Cordula Reisch (Roskilde University)
AUTHORS Ansgar Jügel (TU Wien); Cordula Reisch (Roskilde University); Sara Xhahysa (TU Wien)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Motivated by a mechanical model of tumor encapsulation, we derive a generalized volume-filling cross-diffusion system for the components of tissue structures. The equations are formally derived from mass conservation laws and force balances in a multiphase approach. The model provides a general framework for studying tissue structures, and we show the analytical well-posedness of the system in general settings. The existence of solutions is proven by using entropy methods, and the conditions for the existence of entropy structures are discussed from a modeling perspective. Numerical simulations showcase the solution behavior beyond the entropy regime.

MS42-06 — Upscaling between an agent-based model (smoothed particle approach) and a continuum-based model for cellular forces

SPEAKERS Qiyao (Alice) Peng (MARS, School of Mathematical Sciences, Lancaster University, Lancaster, UK; Mathematical Institute, Leiden University, Leiden, The Netherlands)
AUTHORS Fred Vermolen (Computational Mathematics Group, Discipline group Mathematics and Statistics, Faculty of Science, Hasselt University); Qiyao (Alice) Peng (MARS, School of Mathematical Sciences, Lancaster University, Lancaster, UK; Mathematical Institute, Leiden University, Leiden, The Netherlands)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cells exert forces in their direct environment and cause the deformation of the tissue, which can result in severe consequences in some diseases such as contractures in burn injuries. Various mathematical models — particularly in various scales — have been developed aiming to understand the biomechanics. In most of our work, we used the immerse boundary approach based on a superposition of Dirac Delta

functions to describe the forces exerted by individual cells, which results in a finite-element solution that is not in H1. In [PV22c; PV22b], we developed the smoothed particle approach as a replacement of the immersed boundary approach to improve the accuracy of the solution. The smoothed particle approach is categorized as agent-based model, in which cells are considered as individuals. However, once the number of cells is in the order of thousands and the wound scale is large, these models become too expensive from a computational perspective. For the larger scales, continuum-based models are used which are expressed as partial differential equations. We investigated the connections and consistency between these two types of models, regarding the momentum balance equation [PV22a]. In one dimension, we establish the consistency between these approaches in both analytical solutions and finite-element method solutions. In the multi-dimensional case, we have only computationally shown the consistency between the continuum-based and agent-based models.

References

[PV22a] Q. Peng and F. J. Vermolen. "Upscaling between an agent-based model (smoothed particle approach) and a continuum-based model for skin contractions". In: *Journal of Mathematical Biology* 85.3 (Sept. 2022). ISSN: 1432-1416. DOI: 10.1007/s00285-022-01770-y.

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[PV22c] Q. Peng and F.J. Vermolen. "Point forces in elasticity equation and their alternatives in multi dimensions". In: *Mathematics and Computers in Simulation* 199 (Sept. 2022), pp. 182–201. ISSN: 0378-4754. DOI: 10.1016/j.matcom.2022.03.021.

MS50 — Dynamical Analysis of Biochemical Reaction Networks

TRACK Systems Biology and Biochemical Networks
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 11.02 - HS
ORGANISERS Balázs Boros (University of Szeged); Matthew Johnston (Lawrence Technological University); Polly Yu (University of Illinois Urbana-Champaign)

Minisymposium abstract

Biochemical reaction networks provide a powerful and flexible mathematical framework for modelling the dynamics of complex biochemical systems. Such models, which tend to be nonlinear and have unknown parameter values, can exhibit diverse and interesting behavior, including bistability, oscillations, and even chaos. Existing methods for analyzing such models draw on not only dynamical systems theory but also algebra and graph theory. Previous efforts have motivated new directions in mathematical biology and have opened new mathematical questions. This mini-symposium highlights the latest progress in reaction network theory with an emphasis on approaches that link network structure to qualitative system behavior.

MS50-06 — Biochemical reaction networks: systematic design, limit cycles and properties of Hilbert-like numbers

SPEAKERS Radek Erban (University of Oxford)
AUTHORS Radek Erban (University of Oxford)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

I will discuss two types of mathematical models of biochemical reaction networks: (i) deterministic models described by reaction-rate equations, i.e., ordinary differential equations (ODEs) for the concentrations

of the involved biochemical species [CE25; EK23], and (ii) stochastic models described by the Gillespie stochastic simulation algorithm, which provides more detailed information about the simulated system than ODEs [EEK21; Ple+18; EC20]. I will present methods for the systematic design of relatively simple reaction systems with prescribed dynamical behaviours, including systems with multiple oscillating solutions (limit cycles). We will focus on chemical reaction systems with two species, which under mass-action kinetics are described by planar autonomous ODEs whose right-hand sides are polynomials. The Hilbert number $H(n)$ is defined as the maximum number of limit cycles of a planar autonomous system of ODEs whose right-hand sides are polynomials of degree at most n. I will discuss analogues of the Hilbert number $H(n)$ for several classes of chemical reaction systems, including systems with reactions up to the n-th order and systems with up to n-molecular chemical reactions. Lower bounds on the modified Hilbert numbers will be presented [CE25].

References

[CE25] G. Craciun and R. Erban. "Planar chemical reaction systems with algebraic and non-algebraic limit cycles". In: *Journal of Mathematical Biology* 90.6 (2025), p. 64.

[EEK21] G. Enciso, R. Erban, and J. Kim. "Identifiability of stochastically modelled reaction networks". In: *European Journal of Applied Mathematics* 32.5 (2021), pp. 865–887.

[EC20] R. Erban and S. J. Chapman. *Stochastic Modelling of Reaction-Diffusion Processes*. Cambridge University Press, 2020. ISBN: 9781108498128. DOI: 10.1017/9781108628389.

[EK23] R. Erban and H. Kang. "Chemical systems with limit cycles". In: *Bulletin of Mathematical Biology* 85.8 (2023), p. 76.

[Ple+18] T. Plesa et al. "Noise control for molecular computing". In: *Journal of the Royal Society Interface* 15.144 (2018).

MS50-03 — A bimolecular reaction network with chaotic dynamics

SPEAKERS Josef Hofbauer (University Vienna)
AUTHORS Josef Hofbauer (University Vienna)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

I plan to give a rigorous proof that the Willamowski-Roessler reaction network

$$X \leftrightarrow 2X, \quad X+Y \leftrightarrow 2Y, \quad Y \leftrightarrow 0, \quad X+Z \leftrightarrow 0, \quad Z \leftrightarrow 2Z$$

has chaotic dynamics.

MS50-04 — Stability and Robustness in Biochemical Networks with Bifunctional Enzymes

SPEAKERS Matthew Johnston (Lawrence Technological University)
AUTHORS Badal Joshi (California State University San Marcos); Matthew Johnston (Lawrence Technological University); Tung Nguyen (UCLA)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Recent work has revealed that biochemical networks with bifunctional enzymes can display remarkably rich dynamics, including ultrasensitivity, switch-like responses, concentration robustness, and even species exhaustion. This talk presents a dynamical systems analysis of such networks, shedding light on the subtle architectural differences that produce these vast differences in functional behavior. We outline and employ the next-generation matrix method—only recently adapted to biochemical reaction networks—to characterize previously incomputable thresholds for the stability of boundary steady states. These

thresholds are critical for determining when a mechanism will proceed or shut down. Using bifurcation analysis, we further establish conditions for multistationarity, showing how multiple positive steady states can arise within a single stoichiometric compatibility class—a property theorized to underlie toggle-switch behavior in genetic networks. Finally, we consider the capacity of such networks for absolute concentration robustness (ACR)—an essential feature of metabolic regulation—and explore the interplay between robustness and boundary stability.

MS50-05 — Alternative equations for studying bifurcations in mass action networks

SPEAKERS Murad Banaji (Lancaster University)
AUTHORS Balázs Boros (University of Szeged); Elisenda Feliu (University of Copenhagen); Josef Hofbauer (University Vienna); Murad Banaji (Lancaster University)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

I'll outline some recent results on the geometry of the positive equilibrium sets of mass action networks. We obtain useful parameterisations of equilibria and bounds on the number of (positive, nondegenerate) equilibria a mass action network can admit on any stoichiometric class. The techniques also lead to new approaches to studying bifurcations in mass action networks. Sometimes, via relatively simple calculations, we are able to guarantee or rule out particular bifurcations in a network or class of networks. Moreover, in some cases, the theory gives us tools to obtain explicit parameterisations of bifurcation sets.

References

[BBH24] Murad Banaji, Balázs Boros, and Josef Hofbauer. "Bifurcations in planar, quadratic mass-action networks with few reactions and low molecularity". In: *Nonlinear Dynamics* 112.23 (2024), pp. 21425–21448.

[BF25] Murad Banaji and Elisenda Feliu. "Positive equilibria in mass action networks: geometry and bounds". In: (2025). arXiv:2409.06877. arXiv: 2409.06877.

MS50-01 — Structural classification of chemical reaction networks via visual and algebraic decompositions and its implications to steady state analysis

SPEAKERS Bryan Hernandez (University of the Philippines Diliman)
AUTHORS Bryan Hernandez (University of the Philippines Diliman); Eduardo Mendoza (De La Salle University, Manila, Philippines / Center for Natural Sciences and Environmental Research, De La Salle University, Taft Avenue, Manila 0922, Philippines / Max Planck Institute of Biochemistry, Martinsried near Munich, Germany)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Biochemical and environmental modeling utilizes reaction networks to represent complex transformations, yet the standard Linkage Class Decomposition (LCD), which is based purely on visual connectivity, frequently fails to capture the algebraic properties that govern long-term system dynamics. This work introduces a structural classification based on the finest decompositions of chemical reaction networks, which bridges this gap by mapping the hierarchical relationships between the LCD and two critical algebraic structures: the Finest Independent Decomposition (FID) and the Finest Incidence-Independent Decomposition (FIID). They act as the fundamental building blocks for characterizing general and complex-balanced equilibria, respectively. By the partial order of "coarsens to," between such decompositions, we categorize reaction networks into six distinct classes, three subclasses of Independent Linkage Classes (ILC) and three subclasses of Dependent Linkage Classes (DLC). To facilitate this classification, we introduce two discriminants: the Deficiency Difference, measuring the variance between total and subnetwork deficiencies, and Common Complexes Cardinality. We further establish the properties of these subclasses

and provide illustrative examples. By separating ILC and DLC networks, the framework reveals alignment between structural connectivity and kinetic attributes that could offer insights in the steady state analysis of biochemical and environmental systems.

MS50-07 — Exploring the effect of parametric model reduction on the structural identifiability of linear models

SPEAKERS Sabina Haque (University of Michigan - Ann Arbor)
AUTHORS Sabina Haque (University of Michigan - Ann Arbor)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Structural identifiability answers a theoretical question about which parameter combinations of a mathematical model are uniquely recovered from ideal, noise-free input–output data. Identifiability has been long studied on linear compartment models, and is closely related to the algebraic structure of the input-output equation obtained from the model. In this work, we investigate how structural identifiability is affected under singular limits in which a single parameter of a model is sent to infinity. Such limits induce a collapse of the underlying linear compartment graph that resembles an edge contraction and results in a reduced model. Here, we show that the transfer function of the original model converges to the transfer function of the collapsed model. From this result, we can identify which parameter combinations survive the singular limit and characterize the resulting loss of identifiability. We illustrate these results on several families of linear compartment models, including mammillary, catenary, and cyclic networks, and discuss connections to model reduction and the geometry of parameter space under asymptotic limits.

MS50-08 — Simple chemical systems with chaos

SPEAKERS Tomislav Plesa (University of Cambridge)
AUTHORS Julien Clinton Sprott (University of Wisconsin-Madison); Tomislav Plesa (University of Cambridge)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Systems of N first-order autonomous ordinary-differential equations with polynomials of at most degree n on the right-hand side, called N-dimensional n-degree polynomial dynamical systems (DSs), can display a rich set of solutions whose complexity increases with N and n. When $N \geq 3$ and $n \geq 2$, polynomial DSs can exhibit chaos — aperiodic long-term behavior that is sensitive to initial conditions.A number of simple three-dimensional quadratic DSs are reported in the literature that display chaos with various properties, containing as few as 5 or 6 monomials. However, none of these simple systems are chemical dynamical systems (CDSs) — a special subset of polynomial DSs that model the dynamics of mass-action chemical reaction networks. In this talk, I will present some properties of chaotic CDSs, and a systematic method for their design. Using both analytic and computational approach, I will present a number of simple three-dimensional CDSs with chaos, containing as few as 4 or 5 chemical reactions.

References

[Ple25] Tomislav Plesa. “Chemical systems with chaos”. In: (2025). DOI: 10.48550/ARXIV.2511.15554. URL: https://arxiv.org/abs/2511.15554.

MS50-02 — The polyhedral structure of the disguised toric locus

SPEAKERS Diego Rojas La Luz (University of Wisconsin-Madison)
AUTHORS Balázs Boros (University of Szeged); Diego Rojas La Luz (University of Wisconsin-Madison); Gheorghe Craciun (University of Wisconsin-Madison); Jiaxin Jin (University of Louisiana at Lafayette); Oskar Henriksson (Max Planck Institute)
TRACK Systems Biology and Biochemical Networks

Abstract

Polynomial dynamical systems arise in many applications (e.g., biochemistry, population dynamics) but are hard to analyze because they can display multistability, oscillations, and chaos. Mass-action systems, and in particular complex-balanced (toric) systems, are remarkably stable: they admit a unique attracting equilibrium and rule out oscillations and chaos. We study the set of rate constants for which a mass-action system is dynamically equivalent to a complex-balanced system, the disguised toric locus, and introduce a flux-based toolkit for its analysis and computation. We prove the disguised toric locus is homeomorphic to a prism over the disguised toric flux locus, a polyhedral cone with rich combinatorial structure. This leads to new theoretical results on the geometry of the disguised toric locus: that is a contractible manifold with boundary. This prism/flux viewpoint also brings practical consequences: an explicit computational strategy that, for the first time, computes the full disguised toric locus for many networks of interest. Based on joint work with Boros, Craciun, Jin, and Henriksson.

References

[Bor+26] Balázs Boros et al. *A flux-based approach for analyzing the disguised toric locus of reaction networks*. 2026. arXiv: 2510.03621 [math.DS]. URL: https://arxiv.org/abs/2510.03621.

MS52 — Universal Differential Equations in Mathematical Biology

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 15.05 - HS
ORGANISERS Sebastian Persson (Francis Crick Institute); Torkel Loman (University of Oxford)

Minisymposium abstract

Mechanistic differential equation models are widely used in biology and medicine, but have limited ability to handle incomplete system knowledge. Hybrid modelling approaches (also termed "Scientific Machine Learning") aim to address this by combining mechanistic differential equations with data-driven machine learning components. This minisymposium focuses on a conceptually simple, yet powerful, such approach: universal differential equations (UDEs). UDEs embed neural networks (or other universal function approximators) directly into differential equations and can be fitted using familiar parameter estimation workflows. By learning from data, the embedded network can capture unknown or highly complex dynamics that are difficult to represent with purely mechanistic models. In their simplest form, UDEs extend classical parameter estimation from fitting unknown parameters to learning unknown functions, such as protein production rates as functions of a transcription factor concentration. In more complex settings, they can represent substantial components of unknown system dynamics.

This minisymposium highlights the rapidly developing field of UDEs and their applications in biology. Following a brief introductory overview, it will feature presentations spanning systems biology, epidemiology, and ecology. The program also covers emerging software tools for training UDEs, as well as methodological advances including identifiability analysis and effective model-training strategies.

MS52-06 — PEtab-SciML: The missing layer for accessible SciML modelling

SPEAKERS Sebastian Persson (Francis Crick Institute)
AUTHORS Branwen Snelling (The Francis Crick Institute); Dilan Pathirana (Life and Medical Sciences (LIMES) Institute, University of Bonn, Bonn, Germany, Bonn Center for Mathematical Life Sciences, University of Bonn, Bonn, Germany); Fabian Fröhlich (The Francis Crick Institute); Maren Philipps (University of Bonn); Sebastian Persson (Francis Crick Institute)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mechanistic ordinary differential equation (ODE) models are a powerful tool for studying biological systems. However, their predictive power is constrained by gaps, biases, and inconsistencies in the literature. They typically also require quantitative time-lapse data for training, which is time-consuming to collect. While training could benefit from integrating other modalities such as omics and patient metadata, doing so remains an open challenge. Conversely, machine-learning models lack interpretability and require large datasets. Hybrid scientific machine learning (SciML) models aim to address these shortcomings by combining mechanistic and data-driven modules.

Despite this promise, adoption of SciML modeling in biology remains limited by insufficient infrastructure. Dedicated software packages are needed because implementing end-to-end differentiable SciML workflows for state-of-the-art gradient-based training (parameter estimation) is technically challenging. In addition, model exchange is hindered by the absence of a standardized, reproducible format for specifying SciML training problems, analogous to the PEtab standard for ODE models. To address these gaps, we developed the PEtab-SciML extension to the PEtab format and implemented support in PEtab.jl and AMICI. We here present the PEtab-SciML format, show how it enables efficient training strategies such as curriculum learning and multiple shooting, and report benchmark results comparing training approaches.

MS52-03 — Universal Differential Equations Trained on Remotely Sensed Datasets Can Identify Tipping Points in Large-Scale Ecosystems

SPEAKERS Jack Buckner (Oregon State University)
AUTHORS Jack Buckner (Oregon State University); James Watson (Oregon State University, College of Earth Oceans and Atmospheric Sciences); Mark Novak (Oregon State University, Department of Integrative Biology); Will White (Oregon State University, Fisheries Wildlife and Conservation Science)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Identifying tipping points in spatially distributed ecosystems is critical for conservation but remains challenging due to the complexity of nonlinear dynamics, spatial connectivity, and limited observational data. We present a framework combining Universal Differential Equations (UDEs) with a novel dynamic gradient matching algorithm to learn ecosystem dynamics from large-scale remotely sensed time series. Dynamic gradient matching extends existing gradient matching approaches by simultaneously fitting linear spline smoothing functions and UDE parameters via a joint loss function, eliminating the need for ODE solvers during training while accounting for both process and observation error. This enables efficient scaling to datasets with many state variables. We evaluate four UDE model formulations on simulated spatially distributed kelp forest dynamics with emergent Allee effects, testing their ability to detect and quantify tipping points. The models achieve low false positive rates (0.5–6.2%) and moderate to high true positive rates (58.5–91.5%) for threshold detection, with performance depending on the proximity of the system to the tipping point and spatial correlation of environmental forcing. We further demonstrate the approach on satellite-derived kelp abundance data from central California using the Kelp Watch database, incorporating sea surface temperature as a covariate and estimating dispersal kernels. Results highlight the potential of UDE-based approaches for data-driven identification of critical transitions in spatially extended ecosystems.

MS52-05 — Guiding the optimal use of hybrid mechanistic and data-driven models for individual prediction of platelet dynamics

SPEAKERS Marie Steinacker (Leipzig University)
AUTHORS Marie Steinacker (Leipzig University); Markus Scholz (Leipzig University); Yuri Kheifetz (Leipzig University)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Drug-induced damage to the blood-forming system, also called hematotoxicity, is a frequent side effect of cytotoxic chemotherapy. Due to high patient heterogeneity, it remains difficult to predict individual treatment responses. Mechanistic models describing thrombopoiesis provide some physiological interpretability but often fail to capture individual irregular patient trajectories. Here, we investigate hybrid mechanistic and data-driven approaches for individualized prediction of platelet dynamics during chemotherapy. For this purpose, we consider hybrid models that combine mechanistic myelosuppression models with neural networks in a universal differential equation framework. In addition, we present a purely data-driven alternative, based on nonlinear auto-regressive exogenous models with gated recurrent units. We systematically compare the approaches with several mechanistic models across a range of real patient scenarios with varying levels of toxicity risk, data availability and data sparsity. Our results show that data-driven models substantially improve predictive accuracy if sufficient longitudinal data are available, particularly for high-risk patients with irregular platelet dynamics. In contrast, mechanistic and hybrid approaches outperform purely data-driven models in sparse-data regimes. These findings provide practical guidance on modeling choices for different individual scenarios to support clinical decision-making in chemotherapy management.

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MS52-04 — Theme and Variations: Conditional Universal Differential Equations for Biological Heterogeneity

SPEAKERS Max de Rooij (Eindhoven University of Technology)
AUTHORS Max de Rooij (Eindhoven University of Technology); Natal A.W. van Riel (Eindhoven University of Technology); Shauna D. O'Donovan (indhoven University of Technology)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Biological data often exhibit substantial heterogeneity between individuals. While part of this variability reflects random biological variation, systematic differences may arise from physiological diversity or disease. To reflect this diversity in mechanistic models, we often use the same mathematical equations, while individuals differ in parameter values that govern system dynamics. Capturing this structure, without disregarding the relevant physiological variability remains challenging for regular universal differential equations (UDEs).

In regular UDEs, mechanistic ordinary differential equations are combined with neural networks to learn a single population-level relationship, and therefore struggle to represent systematic heterogeneity between individuals. To address this limitation, we propose conditional universal differential equations (cUDEs). Instead of learning a single function, cUDEs learn a parameterized family of functions conditioned on a latent variable, allowing the model to capture individual differences while preserving a shared mechanistic structure.

We demonstrate this approach by modelling postprandial C-peptide production in a mixed population of healthy individuals and individuals with type 2 diabetes. Combining cUDEs with symbolic regression enables recovery of interpretable mechanistic relationships while accounting for population heterogeneity.

MS52-08 — Functional Identifiability for Universal Differential Equations

SPEAKERS Torkel Loman (University of Oxford)
AUTHORS Ruth Baker (University of Oxford); Torkel Loman (University of Oxford)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Parameter fitting workflows, in which model parameter values are recovered from data, are well established in mathematical biology. The introduction of universal differential equations (UDEs) extends this framework by enabling the estimation of not only scalar parameters but also unknown functions. Examples include learning a protein's production rate as a function of its transcription factor concentration, or an infectious disease's transmission rate as a function of the number of infected individuals. Within parameter fitting, the identifiability concept describes our ability to recover true parameter values from data. That is, identifiability analysis determines whether alternative parameter sets can produce equally good fits to observations. The absence of such alternatives suggests that the inferred parameters reflect the true system dynamics.

Here, we extend the concept of identifiability from scalar parameters to unknown functions. We demonstrate how to assess both structural functional identifiability (i.e. whether a function is fundamentally recoverable, even with perfect data) and practical functional identifiability (i.e. whether a function is recoverable given available data). In both cases, we show how to handle additional sources of nuisance that are not present for standard parametric identifiability. Finally, we show that UDE identifiability can be decomposed into parametric and functional components (with the parametric component assessable using classical parameter identifiability methods) together yielding a complete characterisation of UDE identifiability.

MS52-02 — Elucidating Regional and Global Epidemiological Trends Using Neural Network Model-form Error Corrections

SPEAKERS Erin Acquesta (Sandia National Laboratories)
AUTHORS Erin Acquesta (Sandia National Laboratories); Kyle Nguyen
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The field of scientific machine learning (SciML) seeks to fuse traditional mathematical modeling with advances in machine learning to balance mechanist equations with data-driven inference, resulting in computational models that preserve scientific knowledge while readily adapting to the unknown through data-driven discovery. These advancements are setting the foundation for which SciML surrogates provide novel diagnostics that decompose global and local behavior inherent to high fidelity stochastic models and real-world data. This presentation will introduce neural network (NN) function approximations of model-form error to close the gap between ordinary differential equations (ODE) to an epidemiological agent-based model (ABM). This universal differential equations approximation to the ABM allows us to preserve the foundational ODE that represents the global disease dynamics and couples it with the NN for function approximations of nonlinear state transition dynamics. Equipped with an approximation to the ABM, we further our investigation to account for variability in contact patterns in real-world data by analyzing county level disease dynamics within the state of New Mexico U.S. and compare the result to trends observed in aggregation for the total state dynamics. Ultimately, we will introduce a novel diagnostic to elucidate the regional and global epidemiological trends to fundamentally understanding the impact of regional versus global trends on disease transmission.

MS52-01 — Sensitivity analysis for biological differential equation models with many parameters

SPEAKERS Dilan Pathirana (University of Bonn)
AUTHORS Dilan Pathirana (University of Bonn)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 15.05 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mathematical models of biology commonly use differential equation formulations. Certain application areas, such as signal transduction modeling or scientific machine learning, involve models that contain many parameters. Efficient training of these models requires sensitivity analysis that scales well as the number of parameters grows. Hence, adjoint sensitivity analysis (ASA) is typically employed, instead of forward sensitivity analysis (FSA).

In this work, we derive a new sensitivity analysis method that has similar scaling properties to ASA but, unlike ASA, can be solved in the forward direction. This provides some computational efficiency gains in terms of memory and complexity, especially for the stiff systems that are common when modeling biology. Furthermore, higher-order sensitivities are cheaper to compute with the new method. A drawback is that, when the parameter size is small or the state size is large, then the FSA or ASA methods, respectively, can naïvely outperform the new method.

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MS52-07 — Symbolic regression enables coarse-grained model discovery for cancer-relevant signalling dynamics

SPEAKERS Theodore de Pomereu (Francis Crick Institute)
AUTHORS Fabian Fröhlich (The Francis Crick Institute); Theodore de Pomereu (Francis Crick Institute)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cells respond to their environment through protein networks often dysregulated in cancer, making predictive modelling crucial. Because experiments capture only limited observables, coarse-graining is needed to uncover low-dimensional descriptions. Yet classical approaches rely on idealised assumptions, leaving it unclear when partial experimental observations support reduced system dynamics. Here we show that symbolic regression (SR) provides a principled framework to probe the existence of coarse-grained dynamics and, when they exist, infers mechanistically interpretable models. In synthetic enzyme systems, SR recovers Michaelis–Menten kinetics for two- and three-step mechanisms, but not for a four-step dimerisation model. As data quality is degraded, SR simplifies toward effective kinetic laws while preserving correct theoretical limits. Applied to published time-resolved ERK phosphorylation data, SR identifies compact non-linear ERK rate laws in selected cancer-relevant overexpression contexts, yielding interpretable kinetic effects when models are predictive, and otherwise failing alongside our Neural ODE baseline, indicating missing observations. Together, these findings establish a data-driven approach to identifying coarse-grained models of signalling. More broadly, they establish symbolic regression as a framework for guiding experiment design by generating hypotheses where reduced laws emerge and motivating new measurements where they do not.

MS53 — State of the art methods in modeling for cell and developmental biology

TRACK Cellular and Developmental Biology
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 02.23 - HS
ORGANISERS Alessandra Bonfanti; Anna Nelson; Giulia Celora; Keisha Cook (Clemson University); Kelsey Gasior; Qixuan Wang

Minisymposium abstract

Modern cell and developmental biology increasingly relies on the integration of mechanistic modeling, data-driven inference, and advanced computational techniques to generate predictive insight and guide experimental design. State-of-the-art approaches now combine differential equations and stochastic models with modern data analysis and data science methodologies, including machine learning, to address the complexity, heterogeneity, and multiscale nature of biological systems. These tools enable parameter inference, model discovery, uncertainty quantification, and sensitivity analysis from high-dimensional experimental data. This session will highlight recent advances in modeling and inference across cell and developmental biology, medicine, medical research, and bioengineering, and their role in cell growth, intracellular transport, cell differentiation, cell migration, and tissue development. Speakers will present methods that integrate experimental data with mechanistic and statistical models to study regulatory networks and emergent multicellular behaviors, as well as translational applications relevant to disease modeling and therapeutic design. The speakers will discuss current challenges and open directions in combining mathematical modeling, experimental data, stochastic processes, and machine learning to advance biological understanding and medical innovation.

MS53-01 — The role of transient crosslinks in the chromatin search response to DNA damage

SPEAKERS Caitlin Hult (Gettysburg College)
AUTHORS Caitlin Hult (Gettysburg College); Andrew Atanasiu (UNC-Chapel Hill); Daniel Kolbin (University of Wyoming); Greg Forest (UNC-Chapel Hill); Kerry Bloom (UNC-Chapel Hil)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Homology search is a means through which DNA double-strand breaks (DSBs) explore the genome for sequences of lossless repair. As this search process is fundamental to the relationship between DNA damage and disease, a better understanding the underlying mechanisms is crucial. In this work, we use an effective entropic bead-spring polymer chain model to simulate the spatiotemporal dynamics of the yeast genome during interphase. Through a combination of mathematical modeling and experimental work, we explore the effects of inducing damage within a chromosome segment that, previous to the damage event, occupies a cross-linked region organized by transient structural maintenance of chromosome (SMC) complexes. Using novel computational and visualization techniques, we investigate the particular role of these transient crosslinks in driving local chromatin dynamics, repair mechanisms, and substructure.

MS53-02 — Harnessing Discrete, Multiscale Modeling and Topological Data Analysis to Forecast Stem Cell Behavior

SPEAKERS Daniel Cruz (California Polytechnic State University, San Luis Obispo)
AUTHORS Eunbi Park (National Cancer Institute); Daniel Cruz (California Polytechnic State University, San Luis Obispo); Jack Toppen (Massachusetts Institute of Technology); Elena Dimitrova (California Polytechnic State University, San Luis Obispo); Melissa Kemp (Georgia Institute of Technology & Emory University)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Pluripotent stem cells have the capacity to differentiate into all the primary germ layers represented in embryos. Individual cells process information from their environment through biophysical and biochemical cues in order to determine their cell fate (i.e., the germ layer into which they will differentiate). However, cell autonomous decision-making does not fully account for the organizational features associated with developmental patterning. In this work, we investigated the role of intercellular/intracellular signaling and morphogen-based chemotaxis in the context of cell fate decisions. Along with in vitro experiments, we developed an ensemble of multiscale, agent-based models which represent key mechanisms (e.g., signaling and cell division) using discrete mathematical models (e.g., Boolean networks and Markov

chains, respectively). The goal of this type of modeling is to connect the local interactions with the population-level patterning that we observed in microscopy images. We also used persistent homology to generate multiscale, topological descriptors (i.e., persistence landscapes) from both microscopy images and model simulations. By comparing descriptors from in vitro and in silico experiments, we were able to perform model selection and to improve model predictions of emergent organization under a variety of differentiation conditions.

MS53-03 — Learning collective multicellular dynamics with an interacting mean field neural SDE model

SPEAKERS Lin Wan (Chinese Academy of Sciences)
AUTHORS Qi Jiang (Chinese Academy of Sciences); Longquan Li (Chinese Academy of Sciences); Lei Zhang (Tongji University); Lin Wan (Chinese Academy of Sciences)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

The advent of temporal single-cell RNA sequencing (scRNA-seq) data has enabled in-depth investigation of dynamic processes in heterogeneous multicellular systems. Despite remarkable advancements in computational methods for modeling cellular dynamics, integrating cell-cell interactions (CCIs) into these models remains a major challenge. This is particularly true when dealing with high-dimensional gene expression profiles from large populations of interacting cells, where the intricate interplay between cells can be obscured by data complexity. In this talk, I will present scIMF, a deep generative Interacting Mean Field model that learns collective multicellular dynamics directly from temporal scRNA-seq data. Built on the McKean-Vlasov stochastic differential equation (MV-SDE) framework, scIMF models each cell's dynamics as a function of both its own gene expression state and the empirical distribution of the entire cell population. A cell-wise Transformer attention mechanism parameterizes the interaction term, enabling efficient inference of nonlocal and asymmetric CCIs. Benchmarked against state-of-the-art methods across zebrafish embryogenesis, mouse fibroblast reprogramming, and pancreatic β-cell differentiation datasets, scIMF achieves superior gene expression reconstruction at unobserved time points and more accurate cellular velocity inference. Furthermore, scIMF uncovers biologically interpretable, non-reciprocal interaction patterns of cells, providing a principled framework for studying complex, particularly non-equilibrium biological systems.

MS53-04 — Multi-scale regulation of organ growth through a gene-regulatory network that drives a transient proliferative signal

SPEAKERS Marcos Nahmad (Centre for Research and Advanced Studies (Cinvestav))
AUTHORS Erik Alejandro Ruiz-Perez (Centre for Research and Advanced Studies (Cinvestav)); Hugo Ariel Alvarez (Conicet-UNLP); Marcos Nahmad (Centre for Research and Advanced Studies (Cinvestav))
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Organ growth during development is orchestrated by a combination of patterning, cell proliferation, and morphogenesis, but the extent in which these contributions are integrated into a multi-scale process is largely unknown. The developing wing of the fruit fly, *Drosophila melanogaster*, offers an excellent experimental model to address this question because there is a broad knowledge of the molecular players that drive patterning and growth. Previous work has revealed that the gene regulatory network that drives the expansion of the gene that determines cell fate, also involves the nuclear translocation of the Hippo effector, Yorkie (Yki), which has been extensively reported as a source of driving an excess of cell proliferation in overexpression conditions. However, it is unclear if wild-type Yki levels participate in normal developmental growth. We found that nuclear Yki levels moderately and transiently increase prior to wing cell differentiation. We then used mathematical modelling to show that this result is consistent with the gene network of the system and built a multi-scale molecular/tissue model to investigate the

possible role of this transient nuclear Yki as a mechanism that transiently contribute to organ growth. This modelling-driven hypotheses-generation approach uncovers a key role of transient effects that are often neglected when averaging over space and time in classical developmental studies.*emphasized text*

MS53-05 — Geometric control and mechanics in growing biological tissues

SPEAKERS Pascal Buenzli (Queensland University of Technology)
AUTHORS Pascal Buenzli (Queensland University of Technology)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Biological tissues grow under strong mechanical constraints, leading to curvature control of their rate of growth. However, elucidating how this emergent control arises from dynamic cellular processes such as cell proliferation, cell migration, and cell mechanics remains a major challenge. In this talk, I will present recent advances from cell-based mathematical models and their continuum limit, that help disentangle how curvature dependences of tissue growth emerge from collective crowding effects and individual cellular processes. These models suggest two main mechanisms by which cells at a tissue interface may sense large-scale geometric features: a dynamic mechanism, related to changes in a cell's tangential stress state, and a static mechanism, related to a cell's normal stress state. The continuum limits of these models provide evolution equations of tissue stress that help shed new light on the continuum mechanics of biological tissue growth.

MS53-06 — Uncovering noisy dynamics during cell growth using biologically-informed neural networks

SPEAKERS Rebecca Crossley (University of Oxford)
AUTHORS Rebecca Crossley (University of Oxford)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Neural ordinary differential equation frameworks, such as Biologically-Informed Neural Networks (BINNs), have shown strong potential for learning mechanistic laws from sparse biological data. However, most existing approaches assume homoscedastic Gaussian noise, overlooking biologically meaningful variability arising from cell-to-cell heterogeneity and experimental measurement processes. In this work, we extend the BINN framework by introducing a learnable noise model that enables the identification of additive, multiplicative, or mixed noise structures directly from data. Using population growth systems motivated by cell proliferation assays, we demonstrate that the approach accurately recovers underlying noise types, captures state-dependent variability, and produces well-calibrated uncertainty estimates. These results highlight the importance of modelling structured noise for interpreting biological dynamics and provide a general framework for integrating data-driven uncertainty into neural ODE models, with applications to developmental and cellular systems.

MS53-07 — First-passage times and queueing behavior of stochastic search with dynamic redundancy and mortality

SPEAKERS Samantha Linn (Imperial College London)
AUTHORS Samantha Linn (Imperial College London)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Stochastic search is ubiquitous in cell biology, from the propagation of action potentials via synaptic transmission to the spatial regulation of patterning during tissue development via cytoneme-based

morphogenesis. In dynamic systems like these, the number of 'searchers' is rarely constant: new agents may be recruited while others can abandon the search. Despite the ubiquity of these dynamics, their combined influence on search times remains largely unexplored. In this talk we will introduce a general framework for stochastic search in which agents progressively join and leave the process, a mechanism we term 'dynamic redundancy and mortality'. Under minimal assumptions on the underlying search dynamics, our framework yields the exact distribution of the first-passage time to a target region and further reveals surprising connections to stochastic search with stochastic resetting, wherein a single searcher is randomly 'reset' to its initial state. We will then treat the target region as a queue, which we show has interarrival times governed by a nonhomogeneous Poisson process. Altogether this work provides a rigorous foundation for studying stochastic search processes with a fluctuating number of searchers. This work is in collaboration with Dr. Aanjaneya Kumar (Santa Fe Institute) and Jose Giral-Barajas (Imperial College London).

MS53-08 — Plant Cortical Microtubules: Curvature Sensing by Elastic Curves on Smooth Surfaces

SPEAKERS Tim Tian (University of British Columbia)
AUTHORS Tim Tian (University of British Columbia); Colin Macdonald (University of British Columbia); Eric Cytrynbaum (University of British Columbia); Geoffrey Wasteneys (University of British Columbia)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

The self-organization of microtubule (MT) polymers along the inner surface (cortex) of the plant cell membrane is an essential element in facilitating directional cell growth. The key questions are: what gives rise to the ordering and orientation of MT patterns? Mathematical and computational modelling has proven successful in providing insights: the process is distilled into a system of interacting curves on a 2D surface. There has been interest in the role of cell geometry in this process. Our recent work revisited a common assumption, that MT shapes are described by geodesics. More realistically, MTs are relatively rigid filaments and should seek to minimizing bending, resulting in elastic curves. Our model of elastic curves on cylindrical cells has shown that the curvature influence on MTs should orient MTs in directions opposite to what is biologically favourable. I present our work in generalizing this model to other geometries: we solve for elastic curves on various surfaces to show bifurcations and diverse curves resulting from non-local curvature sensing. This opens the field to realistic models on complex geometries. Our model indicates that there must be additional processes involved to overcome geometric influences. The identity of these processes is the subject of active debates, with many hypotheses being proposed. Lastly, I present the current state of the field and the ongoing effort to overcome the associated modelling challenges.

MS74 — Personalized forecasting in oncology informed by multiscale multimodal data

TRACK Mathematical Oncology
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 62.01 - HS
ORGANISERS Chengyue Wu (The University of Texas MD Anderson Cancer Center, US); Guillermo Lorenzo (Universidade da Coruña, Spain); Pirmin Schlicke (University of Salzburg, Austria)

Minisymposium abstract

Cancer is a highly heterogeneous disease group characterized by diverse biological mechanisms, interactions, and evolution across multiple spatial and temporal scales. Advances in imaging, omics, and clinical biomarkers have enabled improved characterization of tumor behaviors from molecular to tissue scale at diagnosis, during disease monitoring, and for treatment selection. However, these data are typically discrete snapshots available at the time of measurement and are often correlated with clinical outcomes of interest (e.g., pathological response and progression, or treatment resistance) through statistical and AI methods. Despite these advances, the approaches inherently lack explainability and often suffer from limited generalizability to more diverse datasets, rare diseases or clinical objectives. Conversely,

biomechanistic models are rooted in mathematical formulations representing the underlying biological mechanisms driving disease dynamics and have demonstrated potential to enable personalized prediction of tumor growth and treatment response across multiple scales while considering various data types. This minisymposium will connect researchers that have been advancing multiscale and multimodal biomechanistic modeling of cancer by leveraging various data types, establishing multiscale connections through multimodal measurements, and hybridizing mechanistic and AI approaches to accommodate these diverse data types to enhance clinically relevant tumor forecasting.

MS74-07 — Feeding Less, Modeling More: Can Mathematics Optimize Intermittent Fasting in Cancer?

SPEAKERS Víctor M. Pérez-García (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain)
AUTHORS Alba E. Simo (Fundación Instituto Valenciano de Oncología); Andrés Méndiz (University of Castilla-La Mancha); Beatriz Ocaña-Tienda (Spanish National Cancer Research Center); Estanislao Arana (Fundación Instituto Valenciano de Oncología); Gabriel F. Calvo (University of Castilla-La Mancha); Juan Jimenez-Sanchez (University of Castilla-La Mancha); Julián Pérez-Beteta (University of Castilla-La Mancha); Philip K. Maini (University of Oxford); Víctor M. Pérez-García (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain)
SCHEDULE Monday, 13 July 2026; 15:00–15:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

A growing body of experimental and clinical evidence indicates that intermittent fasting can exert beneficial effects in cancer prevention and therapy. Preclinical studies consistently show that fasting cycles can slow tumor growth, enhance tumor cell sensitivity to chemotherapy and radiotherapy, and protect normal tissues. Early-phase clinical trials further suggest that intermittent fasting is feasible, safe, and may reduce treatment-related toxicity while improving therapeutic response. These benefits should not be conflated with chronic caloric restriction, which does not confer therapeutic advantage. Thus, the potential value of intermittent fasting in oncology lies in its structured, time-limited metabolic modulation rather than generalized nutritional deprivation [Cli+21].

Current evidence remains limited by small sample sizes, heterogeneous protocols, and an incomplete mechanistic understanding of metabolic–tumor interactions, limitations that may be addressed through mathematical modeling.

Metabolic scaling laws provide a mechanistic link between tumor growth dynamics and energetic balance [Pér+20]. In this talk, I describe a minimal conceptual model revealing an Allee effect driven by tumor energetic availability. I then present more detailed nutritional models incorporating glucose, insulin, and glucagon dynamics. Theoretical results are complemented by in silico digital twin simulations calibrated with tumor growth and patient metabolic data, leading to optimized fasting schemes for different primary cancers. Our findings illustrate the potential of mathematics to guide nutritional strategies in oncology.

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MS74-01 — From patient vascular data to radiotherapy outcome prediction: a multiscale mechanistic framework

SPEAKERS Cristina Macaluso (Politecnico di Milano, Italy)
AUTHORS Cristina Macaluso (Politecnico di Milano, Italy); Luca Possenti (Fondazione IRCCS Istituto Nazionale dei Tumori); Paolo Zunino (Politecnico di Milano); Piermario Vitullo (Politecnico di Milano); Tiziana Rancati (Fondazione IRCCS Istituto Nazionale dei Tumori)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Radiotherapy (RT) efficacy in solid tumours is critically shaped by the microvascular environment (MVE); hypoxic niches confer radioresistance, while RT-induced phenotypic selection enriches residual tumours in cancer stem cells (CSCs), driving recurrence. We present a multiscale computational framework integrating three components: (i) a phenotype-structured PDE model for tumour cell dynamics with oxygen-dependent proliferation, necrosis, plasticity, and radiobiological response [CBK23]; (ii) a 3D–1D coupled model for microvascular oxygen transport [Pos+21]; and (iii) patient-specific vascular networks generated from capillary density data of head and neck cancer cohort [Mat+25], subjected to vessel damage to simulate RT-induced MVE degradation. A reduced order model based on proper orthogonal decomposition and mesh-informed neural networks enables real-time oxygen field evaluation [Vit+24]. Simulating fractionated (FRT) and ultrahypofractionated (UHFRT) protocols on healthy and 50%-pruned networks reveals that vascular architecture—not dose alone—is the primary determinant of outcome. Although hypoxia-induced dedifferentiation shifts the residual tumor composition to radioresistant stem-like states, vascular geometry compromises the efficacy of RT, rendering dose escalation essentially pointless. UHFRT outperforms FRT in well-perfused settings, but both fail under severe MVE damage. These patient-informed results provide mechanistic insights to guide RT planning, paving the way for patient-specific digital twins.

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MS74-05 — Multimodality Stacking with Blockwise missing values and application to the PIONeeR biomarkers study for prediction of resistance to immunotherapy

SPEAKERS Mohamed Boussena (INRIA, France)
AUTHORS Mohamed Boussena (INRIA, France); Sebastien Benzekry (INRIA)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Integrating multimodal datasets in clinical oncology is frequently hindered by high dimensionality and blockwise missingness, where entire data sources are unavailable for specific patient subsets. Standard survival models often struggle with these gaps, leading to biased results or patient exclusion. We introduce Multimodality Stacking with Blockwise missing values (MSB), a late-fusion framework for survival analysis that independently models modalityspecific features before aggregating predictions via a cross-validated stacking meta-learner. MSB was validated on the PIONeeR study ($n = 443$ patients, 378 biomarkers across eight heterogeneous sources) to predict progression-free survival in advanced non-small cell lung cancer patients receiving immunotherapy. MSB yielded higher predictive performance (C-index) than baseline algorithms. Improvements varied by baseline strength: linear models showed a 15.9% increase ($p < 0.001$ for the Wilcoxon signed-rank test, consistent across 15 cross-validation folds), random survival forests gained 5.4% ($p = 0.002$), and gradient boosting methods improved by 2.1% ($p = 0.030$). Beyond discrimination, MSB reduced the generalization gap (train-test difference in 5 folds cross-validation repeated 3 times: 0.055 vs 0.380 for linear models). Permutation importance analysis identified routine laboratory markers, clinical features, and PD-L1 expression as primary predictive drivers. Missing block indicators showed negligible importance, suggesting the model learned from biomarker values rather than data availability patterns. MSB provides a statistically validated framework for multimodal survival prediction with blockwise missingness. By enabling systematic biomarker evaluation without requiring complete data, MSB offers a practical tool for predictive modeling in biomedical research, pending external validation.

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MS74-02 — NHOC: A PET imaging biomarker for survival forecasting in oncology

SPEAKERS Jesús J. Bosque (Universidad Politécnica de Madrid, Mathematical Oncology Laboratory (MOLAB))
AUTHORS Jesús J. Bosque (Universidad Politécnica de Madrid, Mathematical Oncology Laboratory (MOLAB))
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Human cancers are biologically and morphologically heterogeneous, exhibiting complex spatiotemporal dynamics during tumor growth. Imaging with positron emission tomography (PET) enables visualization of metabolic activity and its intratumor heterogeneity. Using continuous and discrete mathematical models of tumor growth, we showed that the location of the metabolic activity hotspot drifts from the tumor center toward the periphery. This observation led to the definition of NHOC, the normalized distance from the PET hotspot to the tumor centroid. We applied this metric to cohorts of patients with breast

cancer and non–small-cell lung cancer (NSCLC). In these datasets NHOC was shown to be a strong predictor of survival [Jim+21]. NHOC is therefore a biomarker inspired by mechanistic modeling that enables personalized forecasting in patients. Since the publication of the original study, this concept has gained significant traction in the medical community. NHOC has been applied and extended by nuclear medicine groups and validated in independent cohorts [Hov+24; Che+25]. Moreover, we have recently extended its application to a cohort of high-grade gliomas, where it continues to demonstrate prognostic value [Bos+26]. These results highlight the generalizability of the biomarker across tumor types. In this talk, I will review the mathematical foundations of this metric, its application as an imaging biomarker, and its impact on enabling personalized forecasting.

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MS74-04 — Forecasting Patient Specific Treatment Response by Leveraging a Mathematical Model and Patient-Reported Outcomes

SPEAKERS Diya Upadhyaya (Moffitt Cancer Center)
AUTHORS Diya Upadhyaya (Moffitt Cancer Center); Renee Brady-Nicholls (Moffitt Cancer Center)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Computed tomography scans remain a common modality for measuring tumor burden in cancer patients. However, diagnostic imaging can be burdensome or invasive for patients due to enclosed environments and clinical discomfort. Tumor volume data is also collected infrequently. Patient-Reported Outcomes (PROs) offer a less intrusive avenue for gauging cancer progression through measurements regarding the quality of life and symptomology. Past studies have shown that certain PROs, such as insomnia, are highly correlated with changes in tumor volume in non-small cell lung cancer (NSCLC). We analyzed PROs and tumor volumes collected from 80 NSCLC patients undergoing immunotherapy to determine how PRO dynamics could inform when volumetric treatment progression would occur. We calibrated the tumor growth inhibition (TGI) model to patient-specific tumor volume dynamics and evaluated its predictive performance. We then analyzed changes in PROs to assess how they correlated with model parameters and could be used to improve predictive ability. The TGI model can accurately describe patient-specific volume dynamics. Predictions using only the TGI model yielded a true positive rate of 62.5% and an overall accuracy of 64.7%. We found that incorporating changes in insomnia into the model increased the true positive rate to 72.2%, with an accuracy of 71.3%. Additionally, using this innovative framework, we can predict precisely when progression occurs, which may aid clinical decision-making.

MS74-03 — From measurement to decision: a tissue-aware digital-twin platform for CAR T cell dosimetry

SPEAKERS Luciana Luque (CRUK Scotland Institute)
AUTHORS Luciana Luque (CRUK Scotland Institute)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Chimeric antigen receptor (CAR) T cell therapy has transformed the treatment of certain haematological malignancies, yet relapse and primary resistance remain common. Although CAR T cells circulate systemically, their ability to activate, persist and eliminate target cells varies across tissues, suggesting that microenvironmental context plays a key role in therapeutic outcome. Understanding these dynamics requires integrating experimental observations with mechanistic modelling across biological scales. Experimental systems provide essential measurements of CAR T behaviour, but many mechanistic hypotheses and treatment strategies cannot be directly tested in vivo. Agent-based models (ABMs) offer a complementary approach by representing cell-level interactions and emergent population dynamics, enabling controlled exploration of tissue contexts, dosing strategies and treatment schedules while supporting the principles of the 3Rs (Replace, Reduce, Refine). Here I present an integrative framework linking multiscale experiments with mechanistic modelling to study CAR T dynamics across anatomical sites. Central to this effort is the development of an organ-to-organ atlas capturing tissue-specific CAR T behaviour across multiple organs. Integrated with an ABM as a mechanistic engine, these data enable calibration of tissue-aware virtual experiments and lay the groundwork for patient-specific digital twins capable of forecasting treatment responses in silico.

MS74-08 — Data-efficient early prediction of treatment response using mechanistic tumor growth models

SPEAKERS Yi Jiang (Georgia State University, US)
AUTHORS Haozhe Yao (Georgia State University); Somiya Rauf (Georgia State University); Yi Jiang (Georgia State University, US); Ziheng Qiu (Georgia State University)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Personalized forecasting in oncology requires models that are both mechanistically interpretable and effective with limited data. I will present recent unpublished results on predicting treatment response in mouse tumor models using a library of mechanistic growth and treatment models, including 1D formulations (exponential, logistic, weak Allee, strong Allee, and piecewise exponential growth) and 2D models with sensitive/resistant subpopulations under related growth structures, plus a frequency-dependent evolutionary game model. These models are applied to two longitudinal datasets: a chemotherapy dataset from NSG mice and a radiotherapy dataset from WT and SIRPα-deficient mice. Models are evaluated by both goodness of fit and ability to predict outcome (relapse versus durable control). Prediction performance is high across both datasets. For chemotherapy, strong Allee model parameters are highly informative: using only one measurement before and one after treatment, prediction achieves balanced accuracy 0.946 and AUC 1.0. For radiotherapy, the same sparse design yields balanced accuracy 0.874 and AUC 0.964. These results suggest that low-dimensional mechanistic models, combined with machine learning, can provide early, data-efficient forecasts of treatment response and identify interpretable biomarkers for adaptive oncology.

MS90 — Clinically Focused, Translational Modeling of Cancer

TRACK Mathematical Oncology
SCHEDULE Monday, 13 July 2026; 15:00–16:20; 15.04 - HS
ORGANISERS Jonathan Rodriguez (University of California, Irvine); Rachel Sousa (University of California, Irvine)

Minisymposium abstract

Mathematical modeling has proven to be useful for studying many biological fields and the increased focus on translational models has been a major boon to clinical research. Models benefit each stage of clinical research and have resulted in significant improvements to disease monitoring, treatment development, and the scheduling of treatment regimes [Sci+25]. Clinically focused models have identified novel cell-cell interactions and biomarkers that inform clinicians of disease development and progression. Further exploration of cellular interactions and biomarkers through modeling allows expedited investigation of new therapeutic options. Models allow newly developed therapies to be evaluated through virtual clinical trials such as with digital twins representing individual patients or synthetic patient cohorts. Additional testing of treatment scheduling with modeling has revealed new insights allowing clinicians to reduce strain on patients while more effectively targeting treatment resistance and improving patient outcome. In this minisymposium we will highlight research that bridges traditional mathematical modeling with translational research. In particular, we will showcase math models that are developed with clinical data or in collaboration with clinicians for clinically applicable models with real world utility.

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MS90-01 — Phase i trials in cancer: From board to bench to bedside and back again

SPEAKERS Alexander Anderson (Moffitt Cancer Center)
AUTHORS Alexander Anderson (Moffitt Cancer Center)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Cancers are complex evolving systems that adapt to therapeutic intervention through a suite of resistance mechanisms, therefore whilst the maximum tolerated dose (MTD) therapies generally achieve impressive short-term responses, they unfortunately give way to treatment resistance and tumor relapse. The importance of evolution during cancer treatment is becoming more widely accepted. However, MTD treatment strategies continue to dominate precision oncology. Adaptive therapy is an evolutionary therapy that aims to slow down the emergence of drug resistance by controlling tumor burden through competition between drug sensitive and resistant cell populations. This approach was developed through mathematical model driven insights and has been shown to work in preclinical animal models (prostate, ovarian, melanoma, breast) and in pilot clinical trials (NCT02415621; NCT05189457; NCT03543969). In this talk we will discuss how mathematical models and machine learning can be used to optimize treatment strategies [Sci+25], including adaptive therapy, and drive Phase i (imaginary) trials [Kim+16]. We will highlight how mathematical model driven digital twins can: (i) Integrate patient variability; (ii) Bridge between bench and bedside; (iii) Be calibrated from historic clinical data; (iv) Drive Phase i trials; (v) Stratify and optimize treatment; (vi) Predict novel trial outcomes.

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MS90-02 — Tumours, risk, and waves: learning from multi-modal data to predict cancer outcomes

SPEAKERS Chay Paterson (University of Manchester)
AUTHORS Chay Paterson (University of Manchester); David Wedge (University of Manchester); Vivien Holmes (University of Manchester)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 15.04 - HS
TRACK Mathematical Oncology

Abstract

In this talk, I will describe my group's ongoing research into designing mathematical models that can learn from different data modalities simultaneously: such as clinical reports, whole-genome sequencing, and medical images. We apply these to describe risk stratification, patient outcomes, and subclonal evolution for several different pre-cancerous conditions. In my lab, we draw from both stochastic processes, especially compound birth-death processes, and differential equations and dynamical systems, especially hyperbolic PDEs. We recently succeeded in developing a numerical integration scheme for directly computing survival probabilities of a first-order birth-death process on an arbitrary directed graph, without the use of stochastic simulations: ten million times faster than the state of the art. I will explain how this has let us study the genomics and growth of vestibular schwannoma, a type of of slow-growing brain tumour, in unprecedented detail, and how our research is informing real treatment decisions.

Although I work primarily on applications, I am also interested in "theory". All the models we have studied have shown emergent wave-like behaviour, even models without any obvious spatial dependence: I will try to explain why this happens, and point out questions for future research, both theoretical and applied and in nature.

MS90-03 — A Multiscale Physiologically Based Pharmacokinetic Model for mRNA-Encoded Therapeutics: Preclinical Predictions and Translational Perspectives

SPEAKERS Elio Campanile (University of Trento)
AUTHORS Elio Campanile (University of Trento)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

mRNA-encoded therapeutics are emerging as a promising strategy for cancer treatment, yet the quantitative link between lipid nanoparticle (LNP) delivery, intracellular mRNA processing, and systemic protein exposure remains poorly defined. We present a multiscale physiologically based pharmacokinetic (PBPK) model that integrates a parsimonious LNP–mRNA trafficking and translation layer with a mechanistic antibody PBPK framework based on [Sep+19], including FcRn recycling and two-pore tissue exchange [Cam+25]. The model was calibrated and validated in mice using five literature datasets covering multiple mRNA-encoded anticancer therapeutics with diverse molecular weights, Fc properties, and LNP-mRNA formulations. It accurately reproduced plasma concentration-time profiles across single- and multi-dose regimens, while structural identifiability analysis supported robust estimation of the mRNA-related parameters [CBB11]. These results support the use of the model as a general platform to compare mRNA-LNP systems, investigate delivery and expression kinetics, and optimize dose scheduling for mRNA-encoded therapeutics. The talk will present the model, its validation across diverse case studies, and will address the opportunities and current limitations of extending this parsimonious preclinical framework to non-human primates and humans, toward predictive tools for optimizing therapeutic kinetics and supporting individualized mRNA treatment strategies [MWJ09].

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MS90-04 — Identifying population interactions in multiple myeloma immunotherapy with SINDy

SPEAKERS Ryan Woodall (City of Hope)
AUTHORS Ryan Woodall (City of Hope)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Introduction: Multiple myeloma (MM) is managed with complex multi-agent immunotherapy, including daratumumab (dara), a CD38 antibody, and BCMA-targeted CAR-T cell therapy for relapse. As the treatment landscape for MM is broad, it is important to identify prognostic biomarkers to help personalize treatment for individuals. To investigate immune interactions and response to immunotherapy, we used sparse identification of nonlinear dynamics (SINDy).

Methods: Patient's immune populations were measured longitudinally via cytometry in two City of Hope trials: (1) dara maintenance post-stem cell transplant and (2) BCMA-targeted CAR-T therapy. Cell type abundances were input into SINDy to identify patient-specific interaction dynamics.

Results: Under dara maintenance therapy, SINDy discovered multiple 1st order interaction terms between immune populations which distinguished responders from non-responders, including NK cell-mediated stimulation of CD4+ T cells (AUC=0.90), and CD8+ T-cell mediated stimulation of NK cells (AUC=0.88). Further, the dynamic stability of the identified system predicted response (AUC=0.89). In BCMA CAR-T treatment, CAR-T cells contributed towards dynamic system stability. Interactions with CD8+ T-cells (Spearman $R = 0.87$, $p < 0.001$) and B-cells (Spearman $R = 0.94$, $p < 0.0001$) were strongly correlated with the individual's immune system stability.

Conclusion: SINDy reveals interpretable immune interaction structures that separate treatment responders from non-responders and characterize system-level stability, offering a data-driven framework for personalizing immunotherapy in multiple myeloma.

MS121 — Bridging Structure and Dynamics in Biological Networks

TRACK Systems Biology and Biochemical Networks
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 11.01 - HS
ORGANISERS Matthew Wheeler (University of Florida); Venkata Sai Narayana Bavisetty

Minisymposium abstract

Understanding how the structure of a network constrains and drives its dynamics is a central challenge in complex biological systems. While network topology is known to influence phenomena such as multistability, robustness, synchronization, and spreading behavior, the precise mechanisms linking structure to dynamics remain only partially understood. Many real world networks display hallmark properties of complex

systems such as feedback loops, modularity, and nonlinear interactions that make it difficult to predict dynamical outcomes from connectivity alone.

This minisymposium showcases recent advances aimed at closing this gap. The talks highlight new mathematical and computational approaches that reveal how patterns of interaction shape collective behavior in biological, social, and engineered networks. By integrating theoretical insights with application driven examples, the session seeks to illuminate unifying principles that govern the structure–dynamics relationship and to inspire future work in this rapidly developing area.

MS121-07 — A Research Program on Modularity in Biological Systems

SPEAKERS Reinhard Laubenbacher (University of Florida)
AUTHORS Reinhard Laubenbacher (University of Florida)
SCHEDULE Monday, 13 July 2026; 15:00–15:40; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

This talk will outline a research program aimed to develop a formal foundation for research on the concept of modularity that links structure and function of networks. The program is based on an April 2026 workshop on this topic at the National Institute for Theoretical and Mathematical Biology.

MS121-06 — Design principles of complex cellular decision-making networks in cancer

SPEAKERS Mohit Kumar Jolly (Indian Institute of Science)
AUTHORS Mohit Kumar Jolly (Indian Institute of Science)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Elucidating the design principles of regulatory networks driving cellular decision-making is of fundamental importance in mapping and controlling cellular behaviour. Despite their size and complexity, large biological regulatory networks often lead to a limited number of cell-states/phenotypes. How this canalization is achieved remains largely elusive. Here, we investigated multiple different networks governing cell-state transition during cancer metastasis, and identified a latent design principle in their topology that limits their phenotypic repertoire – the presence of two ”teams” of nodes engaging in a mutually inhibitory feedback loop. These ”teams” are specific to these networks and directly shape the phenotypic landscape and consequently the cell-fate trajectories. Our analysis reveals that network topology alone can contain information about phenotypic distributions it can lead to, thus obviating the need to simulate them. We present experimental evidence of such ”teams” in transcriptomic datasets across many contexts (cancer cell plasticity in breast cancer, melanoma, lung cancer etc.). Overall, we propose these ”teams” as a network design principle that drive cell-fate canalization in diverse decision-making processes, and drastically reduce the dimensionality of the phenotypic space.

MS121-01 — Biconnected components and structural robustness in directed networks

SPEAKERS Byungjoon Min (Chunbbuk National University)
AUTHORS Byungjoon Min (Chunbbuk National University); Minsoo Yang (Chungbuk National University)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Strongly connected components (SCCs) are essential for identifying modular structures in directed networks. However, they are inherently fragile, as the removal of even a single node can fragment the component and compromise its functionality. To address this limitation and better capture structural stability, we study

strongly bi-connected components (SBCs), subgraphs where every pair of nodes is linked by at least two vertex-independent directed paths in both directions. Using a generating function formalism, we develop an analytical framework to estimate the size of the largest SBC in directed random graphs. Our analysis reveals that while the largest SBC emerges at the same threshold as SCC, they grow more gradually due to stricter connectivity requirements. These findings suggest that SBCs offer a more robust framework for modularity, with implications for failure-tolerant design and biological network dynamics.

MS121-02 — Attractors are less stable than their basins: Canalization creates a coherence gap in gene regulatory networks

SPEAKERS Claus Kadelka (Iowa State University)
AUTHORS Claus Kadelka (Iowa State University); Matthew Wheeler (University of Florida); Venkata Sai Narayana Bavisetty
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Waddington's epigenetic landscape has long served as a central metaphor for cellular differentiation, depicting mature cell types as stable valley floors. Boolean networks, introduced by Kauffman in 1969, provide a mathematical formalization in which attractors represent phenotypes and basins correspond to developmental valleys. Traditional stability measures assess robustness via perturbations of arbitrary states, although biological systems typically reside at attractors. Here we formalize and analyze attractor coherence – a stability measure Kauffman envisioned but never rigorously developed – which quantifies the likelihood that perturbations of attractor states induce phenotype switching. Across 122 curated biological Boolean models, we uncover a paradox: attractors representing mature cell types are consistently less stable than the trajectories leading to them. Simulations of random networks show that this coherence gap arises from canalization, where specific genes dominate regulation. While canalization increases overall stability, it disproportionately stabilizes transient states, placing attractors near basin boundaries. The gap is almost perfectly predicted by network bias, itself shaped by canalization. These results revise Waddington's landscape: canalization creates robust developmental valleys while flattening ridges near attractors, enabling phenotypic plasticity.

MS121-03 — Principles of dynamical modularity in biological regulatory networks

SPEAKERS Erzsebet Regan (The College of Wooster)
AUTHORS Erzsebet Regan (The College of Wooster)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Biological systems are thought to be hierarchically modular, such that small semi-autonomous modules work together to create larger modules, each responsible for function at a different scale of organization. We thus expect that understanding each module in isolation and putting them together tells us how the whole works. When it comes to cellular regulation, however, a registry of biological modules and their behaviors do not appear to be not sufficient to decipher their coordinated response. Here we ask: are there are general rules by which cellular functions are coordinated in health and disease? We hypothesize that distinct phenotype-combinations are generated by interactions among several multistable regulatory switches, each in control of a discrete set of phenotypic outcomes. To test whether this organization sets apart regulatory networks from random ones, we define measures that quantify whether a) a Boolean network's dynamics can be accurately described via combinations of module-autonomous dynamics, b) modules preserve dynamical autonomy while coupled to others, and c) switches at all scales of the hierarchy show robustness in their phenotype-choice control. Comparing a modular mammalian cell cycle model to its randomized counterparts, we formulate three general principles that govern the way coupled switches coordinate their function. These principles can guide construction of large Boolean regulatory models that reproduce a broad range of observed cell behaviors.

MS121-04 — Network dynamical stability analysis of age-related health

SPEAKERS Glen Pridham (Weizmann Institute of Science)
AUTHORS Andrew Rutenberg (Dalhousie University); Glen Pridham (Weizmann Institute of Science); Uri Alon (Weizmann Institute of Science)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Nearly every physiological function declines with age, as risk of death, disability and chronic disease rises exponentially. Whereas many specific diseases have well-characterized biomarkers and diagnostic thresholds, age-related decline is difficult to quantify and often appears as small, ambiguous changes across many biomarkers. We hypothesized that these changes reflect collective, pathological behaviours that push health systems away from stability, i.e. homeostasis. We used linear order eigen-analysis to characterize these deviations in terms of stability and fixed point drift. We then analyzed associations with adverse outcomes including death and chronic disease – that we surmise occur after deviations reach a tipping point.

We used longitudinal health data from multiple species and datasets [PR23; Pri+24; PR24] to estimate interaction networks to linear order. Stability analysis using the eigen-decomposition consistently revealed that health biomarker data are stable, and demonstrate two characteristic behaviours. The position of the fixed point drifts with age, a phenomenon we call "mallostasis", and variance accumulates along weakly-stable dimensions, a phenomenon we call "stochastic accumulation". Each network has only a few such dimensions but they dominate risk of adverse outcomes. Analysis of these dimensions shows that each dimension shares similarities to a different medical syndrome. We discuss the dynamical behaviour and utility of eigen-states for characterizing overall health.

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MS121-05 — From self-sustained intracellular oscillations to whole-cell network organization in Physarum polycephalum

SPEAKERS Linnéa Gyllingberg (University of California, Los Angeles (UCLA), USA)
AUTHORS Abid Haque; Jason Graham (Scranton University); Linnéa Gyllingberg (University of California, Los Angeles (UCLA), USA); Simon Garnier (New Jersey Institute of Technology)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Across living systems, oscillations support coordination, information flow, and decision making, from neural rhythms to calcium signaling in single cells. The unicellular slime mold Physarum polycephalum is a striking example: despite lacking a nervous system, it exhibits decision-like behaviors including maze

solving, network formation, and exploration–exploitation trade-offs. However, existing models focus either on large-scale network adaptation or local mechanochemical oscillations, leaving open how intracellular dynamics propagate through the organism to shape collective behavior. We present a mechanistic model linking intracellular oscillations to network-scale dynamics by coupling self-sustained calcium oscillations to active pressure, fluid flow, and morphology. In one spatial dimension, reaction–diffusion dynamics drive pressure and tube radius changes, reproducing contraction waves and stimulus-induced symmetry breaking. We extend the model to two spatial dimensions using a phase-field formulation in which calcium-regulated tension drives cell deformation and migration. The model reproduces key cell-level behaviors including exploration–exploitation trade-offs and efficient transport network formation. More broadly, the results show how feedback between oscillatory dynamics and evolving morphology constrains information flow in a living transport network, illustrating how network structure and nonlinear dynamics jointly generate complex behavior in biological systems.

MS148 — Multiscale modeling in bioelectromagnetics

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 02.11 - HS
ORGANISERS Clair Poignard (Inria Research Centre at Rennes University); Emil Timlin (Chalmers University of Technology); Irina Pettersson (Chalmers University of Technology and Gothenburg University)

Minisymposium abstract

Multiscale modeling has become an essential tool for understanding complex biological systems, where processes at the cellular or subcellular level influence tissue- and organ-scale behavior. This minisymposium brings together researchers working on mathematical, computational, and biophysical approaches to bridge scales in bioelectric phenomena. Topics of interest include homogenization techniques, effective medium models, electrical properties of tissues, and numerical methods for efficiently coupling micro- and macroscales. Applications span a wide range of systems, including electropermeabilization in heterogeneous tissues, where cell-level responses shape bulk tissue conductivity, and cardiac electrophysiology, where multiscale models link ionic currents in single cells to organ-level electrical activity and arrhythmia dynamics. The session aims to foster cross-disciplinary discussion on challenges in multiscale modeling, validation against experimental data, and integration of theoretical and computational methods. By highlighting both methodological advances and applied examples, this minisymposium seeks to promote collaboration between applied mathematicians, computational biologists, and experimental electrophysiologists, advancing predictive modeling in bioelectromagnetics and related fields.

MS148-01 — Multiscale mathematical modeling of pulsed field cardiac ablation

SPEAKERS Annabelle Collin (Nantes Université)
AUTHORS Annabelle Collin (Nantes Université); Clair Poignard (AIMOKA: Inria, Assistance Publique-Hôpitaux de Paris, Université Sorbonne-ParisNord, Rennes); Guido Caluori (IHU Liryc, Fondation Bordeaux Université); Michael Leguèbe (Centre Inria de l'Université de Bordeaux; Université de Bordeaux; IHU Liryc, Fondation Bordeaux Université); Simon Bihoreau Duchemin (Centre Inria de l'Université de Bordeaux; Université de Bordeaux)
SCHEDULE Monday, 13 July 2026; 15:00–15:40; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cardiac ablation is a key procedure for treating arrhythmias, one of the leading causes of death worldwide. While radiofrequency ablation (RFA), based on thermal injury, has long been the clinical standard, pulsed field ablation (PFA) has recently emerged as a promising non-thermal alternative. PFA relies on irreversible electroporation, a microscopic phenomenon in which strong electric fields disrupt the cell membrane, leading to cell death.

Modeling PFA is essential to understand how these microscopic effects translate to the tissue scale and to improve clinical guidance. In this talk, we present a physiologically relevant model specific to cardiac

tissue, going beyond the classical Poisson framework with nonlinear conductivity. Our approach is based on the periodic homogenization of a nonlinear microscopic bidomain model, where electroporation is described as a voltage-dependent increase in membrane conductance. The associated two-scale expansion is derived and rigorously justified.

From its leading terms, we obtain an effective macroscopic model and introduce relevant quantities to identify ablated regions. We then investigate clinically relevant scenarios, highlighting the impact of fiber orientation and pulse repetition, and propose an extension accounting for conductivity memory effects between pulses.

MS148-05 — Computational assessment of wireless powering of a pulmonary artery intravascular sensor via volume conduction

SPEAKERS Mar Gadea Saez (Universitat Pompeu Fabra)
AUTHORS Antony Ivorra (Department of Engineering, Universitat Pompeu Fabra, Barcelona, Spain); Laura Becerra-Fajardo (Department of Engineering, Universitat Pompeu Fabra, Barcelona, Spain); Mar Gadea Saez (Universitat Pompeu Fabra)
SCHEDULE Monday, 13 July 2026; 15:40–16:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Remote monitoring of heart failure patients using intravascular implants may enable early detection of disease worsening and reduce hospitalizations [MK21]. Within the FORESEE project, a multiparametric pulmonary artery sensor is being developed for wireless powering and communication through volume conduction [Bec+24], [Gar+22]. In this approach, harmless high-frequency current bursts (6.78 MHz) are applied across the torso using textile electrodes, and the implant is powered through two pick-up electrodes located at its ends. The feasibility of this powering strategy was assessed by means of computational modeling. A three-dimensional adult human torso model, including the pulmonary artery, was used for electromagnetic simulations (COMSOL Multiphysics). The influence of implant electrode exposed area, implant length, and implant position was analyzed while ensuring compliance with electrical safety criteria (SAR $\leq$ 10 W/kg). Additional simulations considered physiological variability, different respiratory cycle states and adipose tissue thickness. The results showed that more than 5 mW can be safely delivered to the implant, sufficient to power the sensor electronics. Although adipose tissue thickness and implant alignment were identified as critical factors, the study supports the feasibility of safely powering a pulmonary artery intravascular sensor using volume conduction.

References

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MS148-03 — Pulsed Field Ablation: electroporation based cardiac ablation

SPEAKERS Damijan Miklavčič (University of Ljubljana)
AUTHORS Damijan Miklavčič (University of Ljubljana)
SCHEDULE Monday, 13 July 2026; 16:00–16:20; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Pulsed field ablation (PFA) – an electroporation-based ablation - is being rapidly adopted as a safer and more efficient alternative to conventional thermal ablation methods for the treatment of cardiac arrhythmias, particularly atrial fibrillation. Although electroporation is a phenomenon occurring on the cell membrane level, its effects require modeling on several levels. The basic biophysics of PFA, focusing on electroporation at the membrane, cellular, and tissue levels will be presented providing mechanistic explanations for the observed clinical outcomes and potential adverse events. Drawing on decades of electroporation research in other biomedical fields, modeling attempts/efforts will challenge current understanding of PFA. Cell membrane electroporation is based on long standing "pore formation" theory leading to transient membrane permeabilization [Kot+19]. Although non-thermal, when applied in vivo, high electric currents increase temperature, which can be significant [Cor+20]. Cell membrane electroporation is usually related to membrane electroporation which depends on local electric field. Electric field distribution in the tissue depends on tissue conductivity, which is inhomogeneous and anisotropic, and further increases locally with cell membrane electroporation and temperature increase. Mass transport, electrochemistry and cellular and tissue effects like cell death [BPM21], interstitial fluid pressure [PM00] and effects on blood perfusion [Jar+10] are only sparsely addressed although they may be critical in predicting treatment outcome.

References

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MS148-02 — A multiscale cardiac pulsed field ablation model: from cellular electroporation effects to lesion formation prediction

SPEAKERS Argyrios Petras (Johann Radon Institute for Computational and Applied Mathematics)
AUTHORS Argyrios Petras (Johann Radon Institute for Computational and Applied Mathematics); Aurel Neic (NumeriCor GmbH, Graz, Austria); Edward J. Vigmond (Liryc Heart Rhythm Disease Institute, Fondation Bordeaux University; CNRS, Bordeaux INP, IMB, University

of Bordeaux); Gernot Plank (Division of Biophysics and Medical Physics, Gottfried Schatz Research Center, Medical University of Graz); Luca Gerardo-Giorda (Johann Radon Institute for Computational and Applied Mathematics (RICAM), Austrian Academy of Sciences, Linz, Austria; Institute for Mathematical Methods in Medicine and Data-Based Modelling, Johannes Kepler University, Linz, Austria); Luca Pasini (Johann Radon Institute for Computational and Applied Mathematics (RICAM), Austrian Academy of Sciences, Linz, Austria)

SCHEDULE Monday, 13 July 2026; 17:00–17:20; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cardiac pulsed field ablation (PFA) is an emerging non-thermal ablation modality that employs high-intensity, short-duration electric pulses to induce irreversible electroporation in cardiac cells. In contrast to conventional thermal techniques, PFA selectively disrupts cell membranes while largely preserving the extracellular matrix and surrounding critical structures, thereby reducing collateral damage. This selectivity, combined with rapid energy delivery, enabled the quick adoption PFA for the treatment of many types of cardiac arrhythmia. In this work, we present a multiscale modeling framework that links cellular electroporation dynamics to tissue-level lesion formation in cardiac PFA. At the cell level, the model captures pore generation, membrane oxidation, and the resulting changes in cellular permeability and electrical conductivity in response to the evolution throughout the temporal progression of the electric pulses. At tissue level, these cellular responses are integrated into a bidomain formulation to predict lesion development. The proposed time-dependent multiscale framework accounts for physiological tissue responses during and after the delivery of the pulses, and is capable of simulating PFA-induced lesion formation along time.

MS148-06 — Multiscale modelling, analysis and simulation of cancer invasion processes

SPEAKERS Mariya Ptashnyk (Heriot-Watt University, Edinburgh, Scotland, UK)
AUTHORS Chandrasekhar Venkataraman (University of Sussex, UK); Mariya Ptashnyk (Heriot-Watt University, Edinburgh, Scotland, UK)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Invasion, one of the hallmarks of cancer, is a complex process involving numerous interactions between cancer cells and the extracellular matrix, facilitated by matrix degrading enzymes (MDEs). It was demonstrated experimentally that there are two important types of MDEs, membrane-bound and diffusible metalloproteinases, involved in cancer invasion. To analyse the impact of those two types of MDEs on cancer invasion, we formulate a microscopic cell-scale model for the degradation of the extra-cellular matrix by matrix degrading enzymes produced by cancer cells. Using tools from the theory of homogenisation we propose a macroscopic tissue-level model for cancer cell invasion into the extra-cellular matrix mediated by bound and soluble MDEs. For the macroscopic model we prove the well-posedness result and propose a finite element method for the numerical approximation. Simulation results illustrate the role of the bound and soluble enzymes in cancer invasion processes.

MS148-07 — Homogenization of a phenomenological electropermeabilization model

SPEAKERS Tobias Gebäck (Chalmers University of Technology and University of Gothenburg)
AUTHORS Ioanna Motschan-Armen (Chalmers University of Technology and University of Gothenburg); Irina Pettersson (Chalmers University of Technology and University of Gothenburg); Tobias Gebäck (Chalmers University of Technology and University of Gothenburg)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In this work, we study the homogenization of the phenomenological electropermeabilization model introduced by Kavian et al (2014) in a periodic tissue subject to an applied electric field. We introduce a small parameter epsilon and derive the most relevant scaling of the equations in epsilon through dimension

analysis. Asymptotic expansions yield a macroscopic model, where we are able to define an effective conductivity of the medium in terms of solutions to cell problems on the microscopic scale. The effective conductivity agrees qualitatively with experimental data from real tissue and depends non-linearly both on time and the applied electric field. Due to the non-linearities in the equations, the macroscopic and microscopic problems do not fully decouple, and the effective conductivity exhibits memory effects. Still, we are able to prove two-scale convergence of the solutions as epsilon tends to zero using monotonicity arguments. Our results therefore provide a rigorous mathematical coupling between cell-scale properties in the tissue and the observed macroscopic conductivity dynamics. The talk will provide an overview of the above results. Some numerical results will also be shown, varying parameters including size and shape of the biological cells.

MS148-04 — Multiscale Modeling of Electroporation Capturing Long-Term Membrane Permeability

SPEAKERS Emil Timlin (Chalmers University of Technology)
AUTHORS Clair Poignard (AIMOKA: Inria, Assistance Publique-Hôpitaux de Paris, Université Sorbonne-ParisNord, Rennes); Emil Timlin (Chalmers University of Technology); Irina Pettersson (Chalmers University of Technology and University of Gothenburg); Tobias Gebäck (Chalmers University of Technology and University of Gothenburg)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

We derive a tissue-scale model of electroporation through periodic asymptotic analysis, based on a cell-scale model introduced by Leguèbe et al. (2014). In the cell-scale model, electroporation is described by coupling the transmembrane voltage to phenomenological variables representing the degree of membrane poration and oxidative effects in the lipid bilayer. An important biological feature of electroporation is that cell membranes can remain permeable to molecules for minutes after the electric pulse has ceased, even after pore resealing. This effect is accounted for by an additional phenomenological variable governed by a reaction–diffusion equation posed on the cell membranes. Using periodic homogenization, we rigorously upscale the coupled system from the cellular level to an effective tissue-scale description. In the homogenization limit, the membrane reaction–diffusion dynamics persist and give rise to macroscopic equations with diffusion effects inherited from the microscale structure. This multiscale framework provides a basis for analyzing and optimizing pulse protocols in applications such as drug delivery and electrochemotherapy, where both immediate and long-term permeability effects are crucial.

MS173 — Advances in Modeling Human Behavior and Infectious Disease Spread: A cross-disciplinary perspective

TRACK Mathematical Epidemiology
SCHEDULE Monday, 13 July 2026; 15:00–18:20; 15.02 - HS
ORGANISERS Lauren Childs (Virginia Tech); Navid Ghaffarzadegan (Virginia Tech); Chadi Saad-Roy (University of British Columbia)

Minisymposium abstract

Recent infectious disease outbreaks highlighted the critical role of human behavior and social processes in shaping infectious disease dynamics. Individual decisions—such as whether to wear a mask, get vaccinated, or work remotely—are not made in isolation, but are strongly influenced by the current state of the disease and individuals' perceptions of risk. Such behavioral responses are also not fixed and change over the course of a pandemic. As these behavior changes accumulate across individuals, they scale up to shape population-level outcomes, including the timing, magnitude, and geographic spread of outbreaks. In order to curtail disease spread with minimal impact to a population, governmental interventions to control epidemics must therefore account for individual decision-making and, particularly, noncompliance or even defiance of recommended or even mandated policies. Modeling techniques developed to account for such dynamic human responses are therefore essential for understanding and managing infectious disease spread.

This minisymposium will feature researchers from a range of disciplines, from theoretical to applied, and talks will focus on recent advances in modeling human behavior as it relates to the spread of infectious diseases, encompassing several disease applications.

MS173-01 — The role of human behavior in shaping infectious disease dynamics

SPEAKERS Chris Soria (University of California, Berkeley)
AUTHORS Audrey Dorelien (University of Washington); Dennis Feehan (University of California, Berkeley); Soren Larsen (University of California, Berkeley); Ayesha Mahmud (University of California, Berkeley); Chris Soria (University of California, Berkeley)
SCHEDULE Monday, 13 July 2026; 15:00–15:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Demography and social structures shape many aspects of an infectious disease outbreak in a population – from host susceptibility and exposure to transmission and health outcomes. However, the social forces that shape human behavior are difficult to quantify and often omitted from mathematical epidemiological models. In this talk, I will discuss some recent work using data from surveys to parameterize infectious disease models to capture heterogeneities in disease outcomes.

References

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[Sor+26] Chris Soria et al. "Partisan differences in health behaviors can impact respiratory disease dynamics". In: *medRxiv* (2026), pp. 2026–01.

MS173-02 — Social Dilemma of Disease Control

SPEAKERS Feng Fu (Dartmouth College)
AUTHORS Feng Fu (Dartmouth College)
SCHEDULE Monday, 13 July 2026; 15:20–15:40; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

This talk will focus on the social dilemma aspects of disease control, and in particular on the "hysteresis" effect in bottom-up public health behavior dynamics that is responsible for resistance to top-down public health recommendations, ranging from mask hysteria, distancing disobedience, and vaccine hesitancy. Synergistic integration of top-down and bottom-up perspectives is required to increase the awareness and preparedness of epidemics of disease.

MS173-03 — Behavioral Adaptation to Novel Pandemics: Learning to Move from Mobility Restrictions to Less Costly Measures

SPEAKERS Binod Pant (Northeastern University)
AUTHORS Binod Pant (Northeastern University); Ann Osi (Merck); Mauricio Santillana (Machine Intelligence Group for the Betterment of Health and the Environment, Northeastern University, Boston, MA, USA; Network Science Institute, Northeastern University, Boston, MA, USA;

Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, MA, USA); Leah LeJeune (Virginia Tech); Lauren Childs (Virginia Tech); Navid Ghaffarzadegan (Virginia Tech)

SCHEDULE Monday, 13 July 2026; 15:40–16:00; 15.02 - HS

TRACK Mathematical Epidemiology

Abstract

During the COVID-19 pandemic, individuals initially responded to epidemic risk through high-cost interventions such as mobility reduction. However, empirical data reveal that mobility reductions became less pronounced in later waves despite persistent death rates. This declining responsiveness likely reflects economic constraints, psychological fatigue, and learning about disease risk that shift individuals toward alternative cost-effective protective measures (e.g., mask wearing). Existing aggregate behavioral feedback models fail to capture this reallocation of protective behavior across interventions. In this study, we formalize this micro-level behavioral adaptation by incorporating an explicit learning mechanism, driven by accumulated pandemic experience, that shifts individuals from high-cost interventions (such as mobility reduction) toward lower-cost measures (such as mask wearing) within a risk-responsive epidemic model.

MS173-04 — SIR model with the distribution of preventive behaviors in a community

SPEAKERS Zhiqiong Fu (Tohoku University)

AUTHORS Hiromi Seno (Tohoku University); Zhiqiong Fu (Tohoku University)

SCHEDULE Monday, 13 July 2026; 16:00–16:20; 15.02 - HS

TRACK Mathematical Epidemiology

Abstract

We consider a mathematical model of differential equations for the epidemic dynamics with heterogeneity in preventive behaviors among individuals. Our focus is on how the distribution of preventive behaviors influences the epidemic consequence in a community. The preventive behavior determines the level of caution to the disease transmission. We assume that the community could be categorized into n classes according to their caution level about a spreading disease. The caution level affects both the susceptibility of susceptible individuals and the transmissibility of infected individuals. Susceptible individuals of low caution level are more likely to be infected than those of high caution level. Infected individuals of low caution level contribute more to the disease transmission. Based on this social structure, we present an SIR-type model with n classes of different caution levels. We investigate how the distribution of classes could affect the final epidemic size, which is defined as the total number of individuals who experienced the disease transmission, and discuss how the severity of the disease outbreak depends on the nature of the distribution.

MS173-05 — Extending an SEIR behavioural model with structures for supply chain efficiency and vaccine allocation

SPEAKERS Jim Duggan (University of Galway)

AUTHORS Jim Duggan (University of Galway)

SCHEDULE Monday, 13 July 2026; 17:00–17:20; 15.02 - HS

TRACK Mathematical Epidemiology

Abstract

A key challenge in infectious disease modelling is to extend beyond classical epidemiological structures to model the interplay between the spread of a disease and how people respond to an outbreak [LeJ+25]. Recent research highlights the potential of incorporating a human behaviour feedback loop into existing model structures as a promising way to advance disease forecasting, and provide better methods for decreasing the negative impact of disease countermeasures on society [GPV25; RXG22]. This talk presents an age-cohort infectious disease spread model, and builds upon recent contributions for exploring pandemic response strategies in the context of limited resources [And+24] in three ways. First, the it incorporates a negative feedback structure to capture human behaviour which moderates disease spread; second, it adds

a supply chain structure that determines the availability of countermeasures; and third, it implements allocation policies to allow for prioritisation of specific age cohorts for vaccine distribution. The scenario results are framed based on the overall aNack rate and hospitalisation rate, and the conventional scenario analysis is extended to encompass techniques from the discipline of statistical learning – namely explanatory model analysis [BB21], which can be deployed to highlight influential model parameters.

References

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[LeJ+25] Leah LeJeune et al. "Formulating human risk response in epidemic models: Exogenous vs endogenous approaches". In: *European Journal of Operational Research* 324.1 (2025), pp. 246–258.

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MS173-06 — Not Everyone Panics Alike: When Heterogeneous Behavioral Responses Change Epidemic Outcomes

SPEAKERS Navid Ghaffarzadegan (Virginia Tech)
AUTHORS Navid Ghaffarzadegan (Virginia Tech); Haniyeh Fattahpour (George Mason University); Lauren Childs (Virginia Tech)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Compartmental epidemic models increasingly capture demographic and contact heterogeneities, yet behavioral responses are typically treated as uniform: as cases or deaths rise, an average person perceives greater risk and increases compliance with non-pharmaceutical interventions (e.g., masking), reducing transmission. But is treating societal behavior as a single average feedback loop a safe simplification? We first develop a two-group compartmental behavioral epidemic model in which groups differ in infection fatality ratio, susceptibility, and contact rates, generating distinct behavioral responses to the same epidemic signals. We show increased variation in mortality risk alters dynamics: homogeneous assumptions lead to underestimated prevalence and overestimated fatality. Heterogeneity in susceptibility alone reduces cumulative cases and deaths, and differences in mixing patterns amplify these effects. A counterintuitive result emerges: a lower-risk group responding weakly reaches herd immunity early, indirectly shielding a more cautious, higher-risk group. We further extend the model to eight age groups reflecting COVID-19 risk variation, examining how behavioral heterogeneity shapes outcomes at a finer scale. Together, these findings clarify when explicitly representing heterogeneous behavioral responses is essential for reliable epidemic modeling.

MS173-07 — Optimizing infectious disease mitigation under dynamic conditions

SPEAKERS Laura Mueller (Max-Planck-Institute for Dynamics and Self-Organization; Institute for the Dynamics of Complex Systems, University of Göttingen)
AUTHORS Fabio Sartori (Max-Planck-Institute for Dynamics and Self-Organization; Karlsruhe Institute of Technology); Jonas Dehning (Max-Planck-Institute for Dynamics and Self-Organization; Institute for the Dynamics of Complex Systems, University of Göttingen); Laura Mueller (Max-Planck-Institute for Dynamics and Self-Organization; Institute for the Dynamics of Complex Systems, University of Göttingen); Maximilian F. Eggl (Institute of Neuroscience, CSIC-UMH; Institute of Experimental Epileptology and Cognition Research,

University of Bonn Medical Center); Viola Priesemann (Max-Planck-Institute for Dynamics and Self-Organization; Institute of Experimental Epileptology and Cognition Research, University of Bonn Medical Center)

SCHEDULE Monday, 13 July 2026; 17:40–18:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Mitigation measures are essential for controlling the spread of infectious diseases during pandemics and epidemics, but they impose considerable societal, individual, and economic costs. We developed a general optimization framework to balance costs related to infection and to mitigation [Mül+25]. Optimizing the trade-off between mitigation and infection cost, we identify three key effects: First, assuming a constant reproduction number, the optimal response to an infectious disease requires either strict mitigation or none at all, depending on disease severity; an intermediate mitigation level is never optimal. Second, under seasonal variations, optimal mitigation is stricter during winter. Interestingly, a single wave of infections still arises in spring with 3 months delay to the seasonal peak of infectivity, replacing the autumn/winter waves known for classical influenza. Third, during steady vaccination campaigns, even optimal mitigation can result in transient infection waves. Finally, we quantify the cost of delayed mitigation onset and show that even short delays can substantially increase total costs—if the disease is severe.

References

[Mül+25] Laura Müller et al. “Optimizing infectious disease mitigation under dynamic conditions”. In: *arXiv preprint arXiv:2512.11454* (2025).

MS173-08 — Measles and mandates: A mathematical assessment of Florida’s proposed school-entry vaccination mandate removal

SPEAKERS Alice Oveson (University of Maryland)
AUTHORS Abba Gumel (University of Maryland); Alice Oveson (University of Maryland)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Recent measles outbreaks in the United States and the United Kingdom have renewed public health concern over a disease once considered eliminated in these countries. Declining vaccination coverage, driven in part by changing public attitudes and policy discussions, has increased the risk of sustained transmission in previously protected populations. In particular, proposed changes to school-entry vaccination requirements in Florida raise important questions about the potential epidemiological consequences of reduced immunization. In this work, we develop a compartmental framework stratified by age and vaccination status to derive and analyze conditions for disease persistence or eradication, such as the basic and control reproduction numbers, and the vaccine-induced herd immunity threshold. We incorporate Florida-specific demographic data, vaccination coverage among kindergarteners, and empirically estimated contact matrices to represent population mixing to explore how shifts in vaccination policy may influence transmission dynamics. This framework is extended to provide a flexible foundation for assessing the population-level impact of policy changes, possible pharmaceutical and non-pharmaceutical interventions, and identifying conditions under which measles resurgence is most likely.

MS27 — Delay differential equation models in mathematical biology

TRACK Miscellaneous
SCHEDULE Monday, 13 July 2026; 17:00–18:20; 01.22 - HS
ORGANISERS Jan Haskovec (KAUST)

Minisymposium abstract

Abstract. Delay differential equations (DDEs) have become an indispensable tool in mathematical biology and the life sciences. Time delays often arise naturally in processes such as gestation, immune response, epidemiological transmission, and neural dynamics, reflecting the time lag between cause and effect. This minisymposium brings together researchers and practitioners to discuss recent advancements in the theory and application of DDEs. Topics include stability analysis, bifurcation theory, numerical methods, and their implications for understanding biological rhythms, collective dynamics, and various types of feedback mechanisms. By highlighting both theoretical innovations and real-world applications, the symposium aims to foster interdisciplinary collaboration and inspire novel insights into complex biological systems. Participants will gain a deeper appreciation of how delays shape the dynamics of living systems and learn about cutting-edge techniques to tackle these challenges.

MS27-02 — Reaction-transmission delays in flocking models

SPEAKERS Elisa Continelli (University of Padova)
AUTHORS Elisa Continelli (University of Padova); Jan Haskovec (KAUST); Cristina Pignotti (University of L'Aquila)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 01.22 - HS
TRACK Miscellaneous

Abstract

Multiagent systems have attracted the attention of many researchers in recent years. Among them, there are the celebrated Hegselmann-Krause opinion formation model and its second-order version, the Cucker-Smale flocking model. Typically, for such systems, one is interested in investigating the asymptotic behavior of their solutions, namely the convergence to consensus for the Hegselmann-Krause model and the asymptotic flocking for the Cucker-Smale model. In such models, it is important to introduce time delay effects since time delays unavoidably appear as times needed to receive some information or reaction times. In this talk, we focus on a Cucker-Smale type model in presence of transmission-reaction delays. Unlike the existing related literature, the reaction delay and the transmission delay are allowed to have different sizes. Under suitable smallness conditions on the sizes of the time delays and on the initial conditions, we prove exponential flocking for the considered model.

MS27-03 — On the local stability of the elapsed-time model in terms of the transmission delay and interconnection strength

SPEAKERS Nicolás Torres Escorza (Université Côte d'Azur)
AUTHORS Jose Canizo (University of Granada); Maria Caceres (University of Granada); Nicolás Torres Escorza (Université Côte d'Azur)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 01.22 - HS
TRACK Miscellaneous

Abstract

he elapsed-time model describes the behavior of interconnected neurons through the time since their last spike. It is an age-structured non-linear equation in which age corresponds to the elapsed time since the last discharge, and models many interesting dynamics depending on the type of interactions between neurons. We investigate the linearized stability of this equation by considering a discrete delay, which accounts for the possibility of a synaptic delay due to the time needed to transmit a nerve impulse from one neuron to the rest of the ensemble. We state a stability criterion that allows to determine if a steady state is linearly stable or unstable depending on the delay and the interaction between neurons. Our approach relies on the study of the asymptotic behavior of related Volterra-type integral equations in terms of theirs Laplace transforms. The analysis is complemented with numerical simulations illustrating the change of stability of a steady state in terms of the delay and the intensity of interconnections.

References

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MS27-04 — Models with Memory and Delay in Molecular, Cellular, and Population Biology

SPEAKERS Radek Erban (Oxford University)
AUTHORS Radek Erban (Oxford University)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 01.22 - HS
TRACK Miscellaneous

Abstract

Memory and delay naturally arise in mathematical models across physical scales. In molecular simulations, the generalized Langevin equation is a stochastic integro-differential equation describing a molecule (or a degree of freedom) subjected to colored (time-correlated) noise and a non-Markovian friction term [Erb20; EC20]. In chemotaxis, cells respond to extracellular signals through intracellular signal transduction networks, and this internal dynamics naturally introduces delays and ‘chemical memory’ in signal processing [Erb26; EC20]. In collective animal behaviour, systems of interacting individuals base their observations and responses to stimuli not only on their present state but also on the system’s past history, with models accounting for transmission delays and reaction delays to signals [EH26; EC20]. In this talk, I will discuss how the parameters of delay equations relate to those of more detailed, non-delayed models and how population-level properties can be established for mathematical biology systems described by coupled delay equations.

References

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MS27-05 — An Exact Solution and Amplitude Enhancement in a Non-Autonomous Delay Differential Equation

SPEAKERS Toru Ohira (Nagoya University)
AUTHORS Hideki Ohira (Graduate School of Informatics, Nagoya University); Kenta Ohira (Graduate School of Informatics, Nagoya University); Toru Ohira (Nagoya University)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 01.22 - HS
TRACK Miscellaneous

Abstract

Delays in feedback mechanisms are well known to generate complex behavior, including oscillations. Understanding delay effects is therefore important in fields such as biology, mathematics, economics, and engineering. Delay differential equations (DDEs) provide a natural framework for analyzing such systems, yet exact analytical solutions are rare.

We present an exact solution of a simple non-autonomous DDE, which to our knowledge is the first analytical result of this type. Using this solution, we study a coupled system of two such equations. For suitable parameter values, the oscillation amplitude is dramatically enhanced - up to about 10 to the 8 times larger than in the uncoupled case.

Large collective oscillations are often assumed to require many interacting units. For example, the sinoatrial node, the heart's primary pacemaker, contains thousands to tens of thousands of cells. Our results indicate that the presence of delay can produce strong amplification even in systems with only a small number of units.

References

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MS71 — Novel Approaches in Mathematical Biology

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Monday, 13 July 2026; 17:00–18:20; 15.06 - HS, 15.05 - HS
ORGANISERS Kyle Nguyen; Lena Podina (University of Waterloo); Tin Phan (Los Alamos National Laboratory)

Minisymposium abstract

The intersection of mathematical methods, machine learning, and scientific machine learning is paving the way for innovative solutions to complex biological challenges. This mini-symposium aims to showcase cutting-edge research that leverages these interdisciplinary approaches to enhance our understanding of biological systems and improve health outcomes. The session will explore transformative applications of artificial intelligence (AI) and mathematical methods in biological research, including the integration of advanced bioinformatics techniques with machine learning, the use of generative AI for enhanced data generation and cell tracking, and the application of large language models (LLMs) to generate behavioral states in network modeling for infectious diseases. Additionally, we will discuss how mechanistic models can improve predictability in biological contexts. Through these interdisciplinary strategies, the symposium will highlight the potential of AI and mathematical methods to drive advancements in biological understanding and public health.

MS71-09 — Scientific Machine Learning Methods for Extracting ODE Models of EBV-Driven B-Cell Fate Trajectories from scRNA-seq Data

SPEAKERS Melanie Sadecki (North Carolina State University)
AUTHORS Cliburn Chan (Duke University); Grace Maclaughlin (Duke University); Kevin Flores (Department of Mathematics, North Carolina State University); Melanie Sadecki (North Carolina State University); Micah Lutfig (Duke University)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Epstein-Barr virus (EBV) infection drives a coordinated cascade of B-cell state transitions underlying both primary infection and EBV-associated malignancies [SoR+22]. scRNA-seq provides high-dimensional snapshots of these transitions across thousands of individual cells, yet current clustering-based approaches capture only a fraction of the quantitative information available. We present an equation learning pipeline that extracts the underlying dynamical structure directly from EBV-driven B-cell scRNA-seq data. First, the raw data is processed via UMAP-based dimensionality reduction and clustered to identify discrete cell states, then mapped onto trajectories using pseudotime. These trajectories serve as input to a hybrid scientific machine learning method in which a structured candidate library encodes known biological relationships alongside neural network terms that capture unknown dynamics. SINDy is applied to recover a parsimonious ODE system whose structure reflects both the data and the underlying biology of EBV-induced B-cell differentiation [WMM25]. The learned system can identify known and unknown cell state transitions and generate mechanistic hypotheses. This method bridges single-cell transcriptomics and scientific machine learning, offering a principled framework for extracting mechanistic structure from high-dimensional omics data and advancing our understanding of EBV-driven B-cell fate decisions.

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MS71-07 — Generation of Model-Based Virtual Patient Cohorts Using Neural Networks

SPEAKERS Kathleen Wilkie (Toronto Metropolitan University)
AUTHORS Kathleen Wilkie (Toronto Metropolitan University); Salim Rezvani (Toronto Metropolitan University)
SCHEDULE Monday, 13 July 2026; 17:20–17:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Limitations of human data in cancer research poses significant challenges for accurately predicting tumor growth and treatment outcomes. To address this, virtual patient cohorts are created to facilitate in silico exploration of new treatments. We propose a novel framework that integrates mathematical modeling, statistical data augmentation, noise reduction, and neural network-based trajectory tailoring to create a virtual patient cohort. Starting from 10 initial simulated patients, we apply a bootstrap technique with added noise to generate a cohort of 200 candidate patients. We then apply denoising techniques and process the candidate patients through a neural network, to sculpt the tumor growth trajectories to match the statistics of our simulated data. To benchmark our method, we compare it against cohort generation via Bayesian inference and sampling of posteriors, demonstrating that our framework is not only more computationally efficient but also more robust in handling noisy data. Finally we apply the pipeline to a dataset for oncolytic virotherapies and recapture the Kaplan Meier survival curves accurately. Our framework effectively handles noisy data, produces suitable virtual patient cohorts, and offers a scalable, computationally efficient solution for virtual patient cohort generation.

MS71-11 — Predicting the impact of gene therapy in preventing HIV transmission

SPEAKERS Steffen Docken (Infection Analytics Program, Kirby Institute, University of New South Wales Sydney)
AUTHORS Christian Boutwell (Ragon Institute of MGH, MIT and Harvard); Daniel Claiborne (Vaccine and Immunotherapy Center, The Wistar Institute); Deborah Cromer (Infection Analytics Program, Kirby Institute, University of New South Wales Sydney); Miles Davenport (Infection Analytics Program, Kirby Institute, University of New South Wales Sydney); Steffen Docken (Infection Analytics Program,

Kirby Institute, University of New South Wales Sydney); Todd Allen (Ragon Institute of MGH, MIT and Harvard); Zachary Detwiler (Wisconsin Institute for Discovery, University of Wisconsin-Madison)

SCHEDULE Monday, 13 July 2026; 17:40–18:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

To date, the only instances of HIV 'cure' were the result of transplantation with stem cells containing mutated genes for the CCR5 coreceptor, a cell-surface protein required by most HIV strains to enter and infect a cell. Thus, researchers are pursuing different gene therapy methods to reduce CCR5 expression without the need for a stem cell transplant, but it is unclear what fraction of target cells must be gene edited to achieve HIV cure or prevent transmission. To answer this question, 73 mice underwent human stem cell transplantation with varying mixtures of CCR5 intact and knock-out stem cells. Animals were subsequently inoculated weekly with HIV, and the time-to-infection was monitored. The observed risk of infection per inoculation decreased from 28% to 0% in animals receiving only intact CCR5 vs. only CCR5 knock-out stem cells. To mechanistically characterize the effect of CCR5 knock-out on infection risk, we developed a probabilistic model of the expected number of infected cells as a function of the fraction of CCR5 knock-out cells. We then predicted the impact of editing rate on human HIV infection by shifting the parameters of this model to values relevant to the human context. We estimated that to sufficiently suppress replication to prevent transmission would require an editing rate of 88%. This rate is beyond the capabilities of current technologies, although rates of up to 80% have been reported in gene therapy clinical trials for other diseases.

MS71-12 — 3D reaction-diffusion model-based biopsy simulation for dynamic tumor growth parameter estimation

SPEAKERS Veronika Hofmann (School of Computation, Information and Technology, Technical University of Munich)
AUTHORS Christina Kuttler (School of Computation, Information and Technology, Technical University of Munich); Heiko Enderling (Department of Radiation Oncology, The University of Texas MD Anderson Cancer Center); Jan Zawallich (School of Computation, Information and Technology, Technical University of Munich); Pirmin Schlicke (Department of Radiation Oncology, The University of Texas MD Anderson Cancer Center); Veronika Hofmann (School of Computation, Information and Technology, Technical University of Munich)

SCHEDULE Monday, 13 July 2026; 18:00–18:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Once diagnosed, cancer requires a fast, inexpensive and reliable assessment of the current state and potential progression of the disease. A new method for estimating tumor cell diffusivity D and proliferation rate γ from single-point-in-time routine biopsies aims to deliver just that, and the ratio of its estimates D/γ is a promising candidate for a new biomarker for risk-stratification in radiotherapy. Here, we extend the findings of the researchers at MD Anderson Cancer Center [Pas+24], who developed the method, by providing a simulation-based validation. The method is applied to *in silico* biopsies which are generated by solving the three-dimensional reaction-diffusion (RD) equation for different growth terms (exponential and logistic) with a Dirac-Delta initial condition, and transforming the continuous results into spatial point patterns via a form of reverse coarse-graining.

First results of this validation process have been presented at SMB 2025, which were performed using a less realistic two-dimensional RD equation and an inferior reverse coarse-graining algorithm.

References

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MS71-01 — Hybrid Mechanistic-Machine Learning Frameworks for Personalized Oncology

SPEAKERS Alvaro Köhn-Luque (Oslo Centre for Biostatistics and Epidemiology, Faculty of Medicine, University of Oslo)
AUTHORS Alvaro Köhn-Luque (Oslo Centre for Biostatistics and Epidemiology, Faculty of Medicine, University of Oslo)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mathematical modeling in oncology frequently encounters a trade-off between the structural interpretability of mechanistic equations and the flexible predictive power of data-driven algorithms. While mechanistic models provide essential biological constraints, they are often subject to structural misspecification when faced with the high dimensionality of clinical heterogeneity. Conversely, purely statistical machine learning (ML) approaches lack the inductive biases necessary to generalize from the sparse and noisy datasets typical of clinical practice. This work presents a methodological framework that treats mechanistic models and ML as complementary components of a unified architecture. The use of ML to learn mechanistic model residuals is discussed, thereby augmenting the predictive accuracy of first-order mechanistic approximations. Hierarchical Bayesian modeling is examined as a robust approach for parameter estimation across patient cohorts, facilitating the personalization of mechanistic dynamics. Operator learning is investigated to map high-dimensional clinical data to patient-specific dynamics. Finally, the integration of reinforcement learning (RL) with mechanistic simulations is demonstrated, where the latter provides a biologically grounded environment for optimizing sequential treatment policies. By formalizing these hybrid methodologies, this work aims to contribute to the ongoing development of decision support tools in oncology.

MS71-02 — Deciphering antibody repertoire evolution using protein language models and B cell lineage inference

SPEAKERS Daphne van Ginneken (Center for Translational Immunology, University Medical Center Utrecht)
AUTHORS Alexander Yermanos (Center for Translational Immunology, University Medical Center Utrecht); Anamay Samant (Department of Biosystems Science and Engineering, ETH Zurich); Andreas Agrafiotis (Department of Biosystems Science and Engineering, ETH Zurich); Daphne van Ginneken (Center for Translational Immunology, University Medical Center Utrecht); Evgenios Kladis (Department of Biosystems Science and Engineering, ETH Zurich); Karlis Daga-Krumins (Center for Translational Immunology, University Medical Center Utrecht); Lucas Stalder (Department of Biosystems Science and Engineering, ETH Zurich); Sai Reddy (Department of Biosystems Science and Engineering, ETH Zurich); Sophie Bakker (Center for Translational Immunology, University Medical Center Utrecht); Tudor-Stefan Cotet (Department of Biosystems Science and Engineering, ETH Zurich); Valentijn Tromp (Center for Translational Immunology, University Medical Center Utrecht); Wiona Glänzer (Department of Biosystems Science and Engineering, ETH Zurich)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

B cell selection and evolution are key processes in regulating successful adaptive immune responses. Recent advances in single-cell sequencing and deep learning strategies have unlocked new potential to study affinity maturation of B cells at unprecedented scale and resolution. To unravel the complex dynamics of B cell repertoire evolution during immune responses and to facilitate Protein Language Model (PLM)-guided antibody engineering, we created the R package AntibodyForests [Van+25a]. AntibodyForests encompasses pipelines to infer B cell lineages, quantify inter- and intra-antibody repertoire evolution, and analyze somatic hypermutation (SHM) using PLMs and protein structure. Using AntibodyForests, we explore how general and antibody-specific PLM-generated likelihoods relate to features of in vivo B cell selection, evolution, antigen specificity and binding affinity [Van+25b]. We find that PLM likelihoods correlate with biologically relevant features including isotype and V-gene usage, mutational load, and SHM patterns. Additionally, we observed that mutating residues along evolutionary trajectories tend to have lower PLM likelihoods than conserved residues. These results indicate that PLMs could predict to what amino acid SHM will most likely mutate and at which position. Interestingly, our findings challenge in vitro observations [Hie+24] by revealing a negative correlation between PLM likelihoods and antigen binding

affinity in in vivo repertoires. In our exploitation of these discoveries using six different PLMs and varying sequence regions, we uncovered that the region of antibody sequence (Complementarity-Determining Region (CDR3) or full-length VDJ) provided to the PLM, as well as the type of PLM used, influences the resulting likelihoods. These comparisons emphasize the importance of PLM long-range interaction, potential training data biases, and pairing heavy and light chains. Together, these studies highlight the power of combining repertoire-wide phylogenetic inference with PLMs to better understand the principles governing antibody evolution and selection, and offer new tools for therapeutic antibody discovery and engineering.

References

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MS71-03 — Establishing a Taxonomy of Computational Modeling as the Foundation Towards Certifying Trustworthy Artificial Intelligence

SPEAKERS Erin Acquesta (Sandia National Laboratories)
AUTHORS Erin Acquesta (Sandia National Laboratories); Jake Nichol (Sandia National Laboratories); Michael Darling (Sandia National Laboratories)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

There is a 30-year history and leadership in verification, validation, and uncertainty quantification (VVUQ) established by the U.S. Department of Energy National Nuclear Security Administration (NNSA) Labs to evaluate the credibility of computational models used in high consequence applications. Today, the U.S. National Institute of Standards and Technology has established the building blocks for trustworthy artificial intelligence (AI) in response to the advancements in machine learning (ML). In this presentation we describe ongoing work to certify the trustworthiness of ML models by establishing the credibility evidence for an epidemiology example that leverages hybrid models, that include neural network model-form error corrections, as a novel diagnostic to elucidate global versus local trends in complex dynamical systems. We will introduce the variability in trustworthiness evidence and credibility in ML models is based on their datasets, inference goals, and training algorithms. As new emerging AI technology continues to revolutionize advancements in scientific discovery and computational modeling, we articulate the importance in establishing a taxonomy of computational models. Prioritizing a taxonomy of AI algorithms, we establish the principles of AI trustworthiness for a class of algorithms that are well suited to the application context and provide a framework for establishing a certification process.

MS71-04 — Exploring the Spatial Dynamics of the Immune Response to Viral Infections

SPEAKERS Fatemeh (Azade) Beigmohammadi (University og Montreal)
AUTHORS Fatemeh (Azade) Beigmohammadi (University og Montreal)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Respiratory viruses, such as influenza and SARS-CoV-2, pose significant global health threats. Mathematical models have been instrumental in understanding epidemiological spread and within-host viral dynamics. These are typically compartmental models that neglect spatial structure. In contrast, agent-based models (ABMs) enable the representation of localized, single-cell interactions and spatial structure within infected tissues. We developed a multiscale, spatial agent-based model in PhysiCell to investigate viral spread and immune dynamics within the lungs following influenza infection. In our model, cells are represented as off-lattice agents capable of migrating, proliferating, and exchanging substrates within the microenvironment. Viral propagation and immune interactions are resolved at single-cell resolution, with diffusive transport coupled through BioFVM. We coupled viral dynamics to the spatiotemporal dynamics of immune cells, including macrophages, neutrophils, CD8+ and CD4+ T cells, dendritic cells, and key inflammatory mediators (i.e., cytokines, chemokines, IFN, ROS), to quantify infection control and tissue damage. Using this framework, we characterized how inhaled virus spreads within lung tissue and how immune responses shape spatial infection patterns. By integrating spatial structure and cellular-level interactions within a multiscale ABM framework, this work advances mechanistic understanding of within-host viral dynamics. Overall, spatially resolved modeling provides a computational platform for dissecting infection control mechanisms and supporting preparedness against emerging respiratory viruses, with potential applications in the pre-clinical evaluation of therapeutic strategies.

MS71-05 — Curriculum Learning for Patient-Specific Parameters in Glioblastoma Multiforme

SPEAKERS Erica Rutter (University of California, Merced)
AUTHORS Erica Rutter (University of California, Merced); Masato Terasaki (University of California, Merced)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Glioblastoma Multiforme is an aggressive primary brain tumor with poor prognosis. Understanding the patient-specific phenotype of the disease (e.g., proliferative vs diffusive) can be crucial for treatment planning. However, learning patient-specific parameters in a reaction-diffusion equation of glioma growth is difficult due to the (1) lack of time-series information available for a single patient, (2) magnetic resonance images (MRI) delineating existence of tumor but not encoding tumor cell density, and (3) lack of publicly available datasets. In this talk, I will present recent work on using curriculum learning to recover patient specific parameters. Curriculum learning is a machine learning technique in which we train on examples of increasing complexity. Using synthetic data, we first learn on dense time-series before annealing to recovering our parameters from two time points and fine-tuning at that data sparsity level. We demonstrate the efficacy of curriculum learning over baseline models especially in regions of increased noise and fewer training samples.

MS71-06 — Experimental and theoretical investigations of rotating algae biofilm reactors (RABRs): Areal productivity, nutrient recovery, and energy efficiency

SPEAKERS Jia Zhao (University of Alabama)
AUTHORS Gerald Benjamin Jones (Utah State University); Jia Zhao (University of Alabama); Ronald Sims (Utah State University)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Microalgal biofilms have been shown to recover nutrients from wastewater and serve as biomass feedstocks for bioproducts. In this talk, I present a platform to quantitatively describe microalgal biofilm production, providing guidance and insights for improving biomass areal productivity and nutrient uptake efficiency in a rotating algae biofilm reactor (RABR). Furthermore, I will present a novel mathematical model to accurately capture the growth dynamics of algae biofilms within RABR.

MS71-10 — Multivariate drivers of the heterogeneous within-host immune response during influenza

SPEAKERS Nicole Bruce (University of Tennessee Health Science Center)
AUTHORS Nicole Bruce (University of Tennessee Health Science Center)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Influenza infections exhibit substantial heterogeneity in viral shedding and immune responses, along with varying widely in severity and outcome. While viral load dynamics and certain immune responses are often assumed to correlate with disease severity, experimental observations suggest that these relationships are not always straightforward and can change across the duration of infection. To investigate the multivariate sources of this variability and how it relates to severity, we turn to mechanistic modeling. We apply a system of differential equations describing influenza viral replication and CD8+ T cell dynamics to human challenge data. Model parameters are estimated across individuals to characterize meaningful variability that governs infection kinetics. Through model-based analysis and clustering of infection trajectories, we identify multivariate drivers that contribute to heterogeneous infection courses. We further extend the model to incorporate symptom development and a wide range of data sources, including human clinical data as well as additional immune cell and cytokine data. These results highlight the value of mechanistic modeling for disentangling the biological processes underlying variable immune responses and provide a framework for improving the predictability of influenza infection dynamics.

MS71-08 — Equation Learning and Distributional Inference for Modeling Mitochondrial Inheritance in Yeast

SPEAKERS Kevin Flores (Department of Mathematics, North Carolina State University)
AUTHORS Kevin Flores (Department of Mathematics, North Carolina State University); Orlando Arguello-Miranda (Department of Plant and Microbial Sciences, North Carolina State University)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mitochondrial inheritance during cell division is a fundamental biological process that ensures daughter cell viability, yet its governing mechanisms remain incompletely understood. In budding yeast (Saccharomyces cerevisiae), experimental observations reveal substantial variability in how mitochondrial content is partitioned between mother and daughter cells, raising key questions about the relative roles of deterministic regulation and stochasticity. In particular, we investigate whether inheritance can be explained by simple proportional rules or whether it depends on a higher-dimensional cellular state immediately prior to division. To address these questions, we develop a data-driven modeling framework that combines equation learning with distributional inference. We analyze high-resolution live-cell imaging data that track mitochondrial content, cell size, and additional molecular markers (e.g., Ime1, Erg6, Vma1) across thousands of cell division events, enabling the reconstruction of lineage relationships and pre-division cellular states. We first apply equation learning techniques, including sparse regression and biologically informed neural networks, to infer interpretable relationships between pre-division cellular features and post-division mitochondrial allocation. To account for inter-cellular variability, we further incorporate a distributional calibration framework based on the Prohorov metric, which enables comparison between empirical and model-generated distributions of inheritance outcomes. Together, these methods provide a unified framework for learning interpretable models of mitochondrial inheritance. More broadly, this work illustrates how combining equation discovery with distributional inference can be used to investigate mechanistic hypotheses in complex biological systems where both structure and population heterogeneity play essential roles.

MS117 — Mathematical Models of Tumour-Immune Interactions and Cancer Evolution

TRACK Mathematical Oncology

SCHEDULE Monday, 13 July 2026; 17:00–18:20; 15.04 - HS
ORGANISERS David Morselli (University College London); Elio Campanile (University of Trento)

Minisymposium abstract

Cancer exhibits a remarkable capacity to adapt to therapeutic and immune pressures, posing major challenges to long-term treatment efficacy. The evolutionary dynamics of the tumour complicate immune recognition, limiting the effective activation of Natural Killer (NK) and T cells, and eventually leading to immune escape. On the other hand, the immune responses can be enhanced through interactions with antigen-presenting cells (such as dendritic cells) and oncolytic viruses. Mathematical and computational models provide a rigorous framework to explore unobservable mechanisms and assess therapeutic strategies through integration with experimental data.

This minisymposium focuses on mathematical modelling approaches for cancer immunotherapy, addressing how immune-mediated tumour control emerges from processes acting across molecular, cellular, and tissue scales. Contributions include the prediction of heterogeneous tumour responses to TRAIL-mediated NK cytotoxicity, the role of TCR-pMHC recognition in shaping immune targeting, and the enhancement of T-cell activation through dendritic cells and oncolytic viruses. By combining mechanistic, stochastic, and agent-based models, we aim to identify determinants of durable immune control and inform rational designs of immunotherapeutic strategies.

We seek to foster discussion by bringing together a diverse, gender-balanced group of researchers from various institutions across different countries.

MS117-01 — Spatial dynamic modelling to understand how dendritic cell clustering affects T cell activation

SPEAKERS Domenic Germano (University of Melbourne)
AUTHORS Domenic Germano (University of Melbourne); Federico Frascoli (Swinburne University of Technology); Peter P. Lee (Beckman Research Institute, City of Hope); Peter S. Kim (University of Sydney); Robyn Araujo (University of Melbourne)
SCHEDULE Monday, 13 July 2026; 17:00–17:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

The coordination of the immune system is essential for maintaining health. Recent clinical studies show breast cancer patients with high dendritic cell (DC) clustering in tumour-draining lymph nodes have improved survival outcomes, when compared to those with a lower degree of DC clustering. However, the mechanistic basis for this spatial organization effect remains unclear. We develop a spatially dynamic model of T cells interacting with Dendritic cells within the lymph node. We present a probabilistic agent-based model (ABM) of T cells, and use it to derive the deterministic, phenotypically structured partial differential equation (PS-PDE) of T cell activation and motion. Using the PS-PDE, we derive an analytic approximation of the expected level of T activation, based on the topology of a given Dendritic cell population. Our analytic approximation enables us to identify T cell characteristics that benefit most from Dendritic cell clustering, to result in an enhanced stimulation distribution. We perform a sensitivity analysis with our models, to identify T cell characteristics that result in desirable T cell activation. Our key findings show that T cells with an intermediate level of stimulation uptake benefit most from higher levels of Dendritic cell clustering, activating with a comparable or greater abundance, and greater heterogeneity, when compared to T cells of a similar characteristic but with a lower level of Dendritic cell clustering.

MS117-02 — TRAIL sensitivity: Decision boundaries in a continuous cell-state landscape with implications for NK-mediated cytotoxicity

SPEAKERS Giada Fiandaca (OMPutational pharmacology and clinical Oncology (COMPO) and Cancer Research Center of Marseille)
AUTHORS Giada Fiandaca (OMPutational pharmacology and clinical Oncology (COMPO) and Cancer Research Center of Marseille); Marielle Péré (Aix-Marseille Université); Kelian Bonhomme (Université Côte d'Azur); Madalena Chaves (Université Côte d'Azur and Institut Hospitalo-Universitaire (IHU) RespirERA); Jérémie Roux (Université Côte d'Azur and Institut Hospitalo-Universitaire (IHU) RespirERA)

SCHEDULE Monday, 13 July 2026; 17:20–17:40; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Natural Killer (NK) cells mediate tumor cell killing through mechanisms such as granzyme–perforin release and death ligand–induced activation of the extrinsic apoptosis pathway [Pra+19]. Among them, the TNF-related apoptosis-inducing ligand (TRAIL) plays a central role and has been widely explored as a therapeutic agent. However, its efficacy is consistently limited by fractional killing, whereby a subset of cells remains tolerant even at high doses [Rou+15]. To investigate the origin of this heterogeneity, we develop a mechanistic model of the TRAIL-induced apoptosis pathway [Fia+25]. By fitting the model to single-cell time resolved FRET trajectories monitoring apoptosis commitment across multiple doses, we infer latent, cell-specific protein abundances together with key kinetic parameters, recapitulating the full diversity of observed responses. These inferred parameters define a continuous state space describing each cell's underlying biochemical configuration. By linking each trajectory to early signaling dynamics, we identify a dose-dependent decision boundary within this space that separates apoptotic from tolerant regions, indicating that cell fate is largely determined by a cell's position in this state space at the time of treatment. Taken together, these results provide a quantitative basis for understanding heterogeneous sensitivity to TRAIL–induced apoptosis and suggests new strategies to modulate response in NK-based therapies.

References

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[Pra+19] I. Prager et al. "Target cell–induced NK cell dysfunction limits cytotoxic activity". In: *Science Immunology* 4.33 (2019), eaav6122. DOI: 10.1126/sciimmunol.aav6122.

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MS117-03 — The co-evolution of a tumour's genomic landscape and TCR repertoire

SPEAKERS Isabella Sodi (University College London)
AUTHORS Isabella Sodi (University College London); Gayathri Nageswaran (University College London); Suzanne Byrne (University College London); Trupti Gore (University College London); Selvaraju Veeriah (Francis Crick Institute); Andrew Pyo (Stanford University); Daniel Fisher (Stanford University); Andreas Tiffeau-Mayer (University College London); TRACERx Consortium; Mariam Jamal-Hanjani (Francis Crick Institute); Charles Swanton (Francis Crick Institute); Benny Chain (University College London)
SCHEDULE Monday, 13 July 2026; 17:40–18:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Cancer–immune co-evolution shapes tumour progression and immunotherapy response, but quantitatively linking immune selection pressure to genomic intratumour heterogeneity and to the tumour-infiltrating T-cell receptor (TCR) repertoire remains challenging. Central to this challenge is recognising that immune selection drives a dynamic process in which the TCR repertoire both shapes and is shaped by the evolving tumour genomic landscape.

We set out to identify immune evasion signatures in one of the largest sets of TCR repertoires from non-small cell lung cancer (NSCLC). We performed bulk TCR sequencing on multi-region tumour samples from 265 NSCLC patients in the TRACERx 421 cohort, comprising 1.27 million unique TCRα and 2.5 million TCRβ chains.

A conceptual expectation of cancer–immune co-evolution predicts that the number of mutations is correlated with the number of expanded T-cell clones, previously shown in a smaller subset of the cohort [Jos+19]. Using data analysis alongside an agent-based model of tumour–immune co-evolution, we

investigate the contexts in which this relationship holds. The model links mutation accumulation to T-cell clonal expansion and allows us to explore how immune evasion mechanisms, such as MHC loss and checkpoint mediated immune suppression, perturb this relationship [Put+24; Mar+23]. The analysis of the TRACERx 421 genomic, transcriptional and TCR repertoire data reveals foundational principles for the relationship between tumour evolution and host immune control.

References

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MS117-04 — Optimizing combined oncolytic virotherapy and chemotherapy for neuroblastoma: a mathematical approach

SPEAKERS Matteo Italia (University of Castilla-La Mancha)
AUTHORS José García Otero (University of Castilla-La Mancha); Juan Belmonte-Beitia (University of Castilla-La Mancha); Mariusz Bodzioch (University of Warmia and Mazury Olsztyn); Matteo Italia (University of Castilla-La Mancha)
SCHEDULE Monday, 13 July 2026; 18:00–18:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Neuroblastoma is the most common extracranial solid tumor in children, where high-risk cases often face poor prognosis. We propose an ordinary differential equation model to investigate optimal combined dynamics between chemotherapy (Cyclophosphamide) [Gar+21] and oncolytic virotherapy (Celyvir, mesenchymal stem cells carrying the adenovirus ICOVIR-5) [Mel+16], combining our previous works on chemotherapy [Ita+23] and virotherapy [GBB24].

The mechanistic model incorporates interactions between tumor, immune system, virus, and drug. Calibrated using published data, we perform mathematical analysis to identify threshold values for treatment administrations required to eliminate the tumor. Through sensitivity analysis, we determine key parameters driving treatment outcomes, serving as potential biomarkers.

We formulated an optimal control problem with a nonlinear objective functional aimed at minimizing tumor burden and treatment toxicity [Pon18]. We show that periodic bang-bang control regimes optimize the delivery of Celyvir, while singular control can characterizes the chemotherapy regimen. By conducting in silico trials on virtual patient cohorts, we assessed the effects of patient heterogeneities on optimal solutions. Our results demonstrate that personalized scheduling, informed by patient-specific data, can significantly optimize combined treatment outcomes, providing a mechanistic-based foundation for clinical decision support systems.

References

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MS33 — Mathematics and the Life-Sciences: dedicated to 65th Birthday of Angela Stevens

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 02.11 - HS
ORGANISERS Benoit Perthame (Sorbonne Université); Mariya Ptashnyk (Heriot-Watt University, Edinburgh)

Minisymposium abstract

Mathematical analysis plays a crucial role in advancing the field of mathematical biology. By providing rigorous frameworks and tools, it enables the development of consistent mathematical models for complex biological systems, the analysis of underlying mechanisms, and predictions of long-term dynamics. This minisymposium will present several modelling and analysis results addressing different aspects of the development and function of biological systems. It will cover a variety of methods and approaches, such as biomechanics, networks, stochastic particle systems, and PDEs. Along with discussions of exciting novel research results, it will also give us the opportunity to celebrate Angela Stevens' birthday and her significant contributions to the field of mathematical biology.

MS33-01 — Kinetic equations and mathematical epidemiology

SPEAKERS Angela Stevens (University of Muenster)
AUTHORS Angela Stevens (University of Muenster); Stephan Luckhaus (Leipzig University)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In 1914 McKendrick came up with a method inspired by L. Boltzmann to describe the evolution of statistical distributions in an - in principle continuous - parameter space, also for the life sciences and the social sciences.This method leads to partial differential equations. Nevertheless, the Kermack-McKendrick models are often misinterpreted solely as the well-known SIR ODE-system for the dynamics of susceptibles, infectious and removed during an epidemic.

But McKendricks equations are by far more general. For instance the hydrodynamic limit of a stochastic epidemiological model, where two infection scenarios alternate, namely a) infections in separated groups of finite size; b) and infections at meeting places of finite capacity, where individuals meet randomly, also results in such a type of McKendrick system with polynomial infection force.

For this system of kinetic equations we derive invariants which uniquely determine the outcome of the model epidemics. Such kinetic equations allow to link global data of an epidemic with not so easily observable local rate dependencies.

References

[LS23a] Stephan Luckhaus and Angela Stevens. "A free boundary problem-in time-for the spread of Covid-19". In: *J. Math. Biol.* 86.3 (2023), Paper No. 45, 17. ISSN: 0303-6812,1432-1416. DOI: 10.1007/s00285-023-01881-0. URL: https://doi.org/10.1007/s00285-023-01881-0.

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MS33-02 — Bacterial movement by run and tumble: the flux-limited Keller-Segel equation

SPEAKERS Benoit Perthame (Laboratoire J.-L. Lions, Sorbonne Université)
AUTHORS Benoit Perthame (Laboratoire J.-L. Lions, Sorbonne Université)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

At the individual scale, bacteria as *E. coli* move by performing so-called run-and-tumble movements. This means that they alternate a jump (run phase) followed by fast re-organization phase (tumble) in which they decide of a new direction for run. For this reason, the population is described by a kinetic-Botlzmann equation of scattering type. Nonlinearity occurs when one takes into account chemotaxis, the release by the individual cells of a chemical in the environment and response by the population.

These models can explain experimental observations, fit precise measurements and sustain various scales. They also allow to derive, in the diffusion limit, macroscopic models (at the population scale), as the Flux-Limited-Keller-Segel system, in opposition to the traditional Keller-Segel system, this model can sustain robust traveling bands as observed in Adler's famous experiment.

Furthermore, the modulation of the tumbles, can be understood using intracellular molecular pathways. Then, the kinetic-Boltzmann equation can be derived with a fast reaction scale. Long runs at the individual scale and abnormal diffusion at the population scale, can also be derived mathematically.

MS33-03 — Oncogenic transformation of tubular epithelial ducts: how mechanics affects morphology

SPEAKERS Davide Ambrosi (Polytechnic University of Turin)
AUTHORS Davide Ambrosi (Polytechnic University of Turin)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Epithelial tissues at a pre-tumoral stage exhibit morphological changes: in particular epithelial ducts depart from the cylindrical shape, showing invaginations and evagination in the regions of the surface with malignant cells. Experiments report that at the inner and outer boundary of the epithelial sheets are

concentrated molecular motors able to generate a surface active tension, that can vary between healthy and tumor cells. The mechanical origin of such morphology can be mathematically tackled by a continuum mechanical model [Amb+26] able to relate, also quantitatively, the role of the impaired surface tensions.

The mathematical model, derived from first principles, accounts for the competition between the bulk elasticity of the epithelium and the surface tension of the apical and basal boundaries. The variation of the energy functional yields the Euler-Lagrange equations to be numerically integrated. The numerical results reproduce a variety of morphological shapes, from invagination to evagination, depending on the ratio between bulk and surface energy at variance of the length of the section. In particular, using parameters independently measured, we are able to reproduce experimental data reported for a ring partially made of transformed cells.

The numerical results obtained with a mathematical model that accounts, in a suitable way, for the thickness of the epithelial wall, prompt us to a deeper mathematical characterization that we address exploiting the Euler Elastica. In this framework we study the variety of possible shapes that a planar inextensible closed rod can take because of a piecewise inhomogeneity in its natural curvature. On the basis of numerical simulations, perturbation analysis and geometrical arguments, we are able to devise three morphological regimes and we provide a first order approximation of the curves that separate the shape regimes in the (k, s_0) plane, k being the jump in natural curvature in the relative curvilinear coordinate s_0. Our perturbation analysis, supported by geometrical arguments, compares well with the numerical results based on the fully nonlinear theory.

References

[Amb+26] D. Ambrosi et al. “Oncogenic transformation of tubular epithelial ducts: How mechanics affects morphology”. In: *European Journal of Mechanics - A/Solids* 117 (2026), p. 105984.

MS33-04 — Minimal Principles for Dynamics in Reaction Networks

SPEAKERS Nicola Vassena (Leipzig University)
AUTHORS Nicola Vassena (Leipzig University)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Biochemical networks are notoriously large and complex and involve many unknown parameters. As a consequence, various reduction methods for their analysis have been proposed. A common critique of this approach is that biochemical complexity is intrinsic and that any “reduced” model is therefore doomed to miss essential features. However, minimal models can be particularly valuable to illustrate/investigate/-suggest general and fundamental qualitative mechanistic principles, rather than to provide quantitative predictions or detailed explanations of specific phenomena.

In this spirit, I will present a few minimal models that produce characteristic dynamical behavior, such as the emergence of periodic oscillations or multistability arising from symmetry breaking. The examples are taken from joint work with Alex Blokhuis and Peter Stadler (oscillations), and with Angela Stevens (symmetry breaking and multistability).

MS47 — Quantitative Models of Immune Regulation and Therapeutic Response in Cancer

TRACK Mathematical Oncology
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 62.01 - HS
ORGANISERS Fatemeh (Azade) Beigmohammadi (Université de Montréal/Sainte-Justine University Hospital Research Centre); Mia Brunetti (Université de Montréal/Sainte-Justine University Hospital Research Centre); Morgan Craig (Université de Montréal/Sainte-Justine University Hospital Research Centre)

Minisymposium abstract

This session will bring together leading researchers to discuss quantitative methods for studying immune-related therapies and tumor-specific mechanisms in cancer to support the development of more effective treatments. Our objective is to highlight immune-focused treatments and models and their significance to cancer research, from immune-mediated treatment optimization, mechanisms underlying differential responses to immunotherapy, tumour-specific mechanisms, and antibody transfer processes. This minisymposium will explore how immune mechanisms can improve the efficacy of radiation therapy for brain metastases; the complex role of PD-L1 in both activating and suppressing immune responses; insights into improving therapies for acute myeloid leukemia (AML) by establishing predictive models of preleukemia; and the movement of therapeutic antibodies throughout the body. Through the integration of diverse perspectives and approaches, this session will advance immune-focused cancer research and the use of mechanistic modelling to shepherd the development of more effective treatment strategies.

MS47-01 — Modelling monoclonal antibody transfer across the human placenta

SPEAKERS Eleanor Doman (University of Manchester)
AUTHORS Eleanor Doman (University of Manchester)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Monoclonal antibody (mAb) therapies, although widely established in cancer treatment, are increasingly being developed and repurposed for the long term management of chronic autoimmune and inflammatory conditions such as multiple sclerosis, rheumatoid arthritis, and inflammatory bowel disease. Many of these conditions disproportionately affect women, raising important questions about fetal exposure when mAbs are taken during pregnancy, whether inadvertently or as part of maternal disease control. Most therapeutic mAbs are engineered from human immunoglobulin G (IgG) and therefore interact with the same pathways as endogenous IgG antibodies. This includes the pathways which allow maternal IgGs to pass to the fetus through the placenta. In this work, we present a mathematical model of mAb transport across the human placenta, calibrated using experimental data from ex vivo placental perfusion studies. The human placenta contains two circulations, maternal and fetal, that come into close contact to allow for the transfer of oxygen and nutrients, however, are anatomically separate. Using ODEs we model the placental compartments, FcRn expression, and associated transport processes to explore how these factors influence fetal exposure to mAbs. Understanding placental mAb transfer is essential for informing regulatory decisions on mAb use in pregnancy, and our model provides a mechanistic framework to predict fetal exposure and quantify risks.

MS47-02 — Modelling Hematopoietic Dynamics in H3K27M-Driven Preleukemia and Implications for AML Therapy

SPEAKERS Mia Brunetti (Université de Montréal/Sainte-Justine University Hospital Research Centre)
AUTHORS Mia Brunetti (Université de Montréal/Sainte-Justine University Hospital Research Centre)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Acute myeloid leukemia (AML) is an aggressive blood cancer driven by genetic and epigenetic alterations that disrupt normal hematopoiesis. Among these, the H3K27M mutation, originally identified in pediatric high-grade gliomas, reshapes gene repression programs by reducing global H3K27 trimethylation. Although rare, H3K27M mutations have been detected in preleukemic hematopoietic stem cells (HSCs) of AML patients, suggesting its role in early leukemogenesis and identifying it as a promising therapeutic target. Here, we investigated how H3K27M alters hematopoiesis using a longitudinal xenotransplantation model of human HSCs carrying either H3-K27M or wild-type H3. To quantify hematopoietic dynamics, we developed a collection of mathematical models representing alternative lineage hierarchies and fitted them to our longitudinal experimental data. Using information criteria and global sensitivity analysis, we

identified the model that best capture blood dynamics in each condition. Our results show that H3K27M promotes the expansion of HSCs, common myeloid progenitors, and megakaryocyte-erythroid progenitors while inducing a delayed block in erythroid differentiation. By stimulating HSC proliferation, H3-K27M dynamics allowed us to distinctly characterize mouse HSC subpopulations (HSC1 and HSC2). Together, our results provide the first quantitative framework to reveal how H3K27M influences hematopoietic hierarchy roadmap and alters blood cell dynamics in preleukemia.

MS47-03 — How much biological realism is enough? An application to ovarian cancer treatment response

SPEAKERS Patricia Lamirande (Université de Montréal)
AUTHORS Patricia Lamirande (Université de Montréal)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Developing a mathematical model requires balancing biological realism against model tractability. We investigate this question through a case study of mechanistic models of ovarian cancer tumour growth and treatment response, assessing which biological hypotheses can be addressed at different levels of model complexity. We use data from Paffenholz et al. (2022), who developed mouse models of high-grade serous ovarian cancer to better understand therapeutic responses to platinum-based chemotherapy (i.e., cisplatin), ICB, and their combinations in homologous recombination (HR)-deficient and HR-proficient immunocompetent mice.

Identifying and interpreting mechanisms of response in these scenarios can be challenging because of the overlapping and nonlinear interactions among tumour, immune, and treatment dynamics. To this end, we developed a hierarchy of ordinary differential equation (ODE) models with increasing biological complexity, progressing from simple exponential tumour growth to mechanistic models incorporating tumour and immune cell populations. Comparing these models reveals that some treatment-response patterns can be captured using relatively simple formulations, whereas others require explicit representation of immune-mediated mechanisms, highlighting how the appropriate level of biological realism depends on the hypothesis being investigated.

References

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MS47-04 — Modeling immune-mediated effects of RAGE inhibition and radiotherapy in malignant gliomas

SPEAKERS Miguel Perales-Patón (University of Castilla-La Mancha)
AUTHORS Miguel Perales-Patón (University of Castilla-La Mancha)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

The dynamics of malignant neoplasms of the central nervous system, such as malignant gliomas and brain metastases, are strongly shaped by the blood–brain barrier and by interactions between tumor cells and immune populations within the tumor microenvironment. In particular, tumor-associated myeloid populations—including microglia and macrophages—can promote tumor growth through immunosuppressive and pro-tumoral signaling pathways. Among the molecular regulators involved in these interactions, the receptor for advanced glycation end-products (RAGE) has emerged as a key mediator linking inflammation, immune dysfunction, and tumor progression. Azeliragon (AZG), a small-molecule RAGE inhibitor, has recently shown promising preclinical results in combination with radiotherapy (RT), and clinical trials are currently underway to evaluate its therapeutic potential (e.g., NCT05635734 and

NCT05789589). Motivated by these findings, we developed an ordinary differential equation model of malignant gliomas that explicitly incorporates tumor–immune interactions and the combined effects of RT and AZG. This framework enables the quantitative exploration of treatment schedules through in silico trials and provides a mechanistic tool to investigate how RAGE inhibition may reshape the tumor immune microenvironment and enhance radiotherapy efficacy. The proposed framework also provides a quantitative basis to investigate similar immune-mediated treatment strategies in other intracranial malignancies, where activation of the S100A9–RAGE signaling axis has been identified as a driver of radiotherapy resistance. I will discuss the theoretical foundations of the model, and its validation with clinical data as well as the extension of the concepts to brain metastases, a more frequent malignancy where similar biological processes are also present.

MS66 — Mechanical models of collective cell dynamics

TRACK Cellular and Developmental Biology
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 02.23 - HS
ORGANISERS Kai Li (Adelaide University); Ryan Murphy (Adelaide University)

Minisymposium abstract

Understanding collective cell dynamics is fundamental to the study of key problems in cell biology, such as tumour growth, biofilm expansion, and wound healing. A key aspect of collective cell dynamics that is often underappreciated is the role of cellular mechanics. Cellular mechanics plays a role in dynamics at all scales, from subcellular cytoskeletal forces acting over seconds to drive cell shape, to cell-cell mechanical interactions driving tissue-level outcomes on the order of hours, days, and weeks. Mathematical modelling provides a powerful framework to study the multi-scale nature of mechanical interactions. In this session, we will feature a series of talks from experts in modelling the role of mechanics at and across various scales. Together, this session will present a broad integrated understanding of how mechanics influences biological systems.

MS66-01 — Mechanical regulation of the G1/S checkpoint in collective cell migration: a two-stage age-structured model

SPEAKERS Stéphanie Abo (University of Oxford)
AUTHORS Ruth Baker (University of Oxford); Stéphanie Abo (University of Oxford)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Cell proliferation and migration are tightly regulated by mechanical cues, particularly crowding through contact inhibition. A central control point is the G1/S restriction point (R-point), an irreversible checkpoint in late G1 where mammalian cells integrate environmental signals, such as cell density, with intrinsic regulatory cues to commit to DNA replication and division. We introduce a two-stage age-structured model of collective cell migration that resolves the G1 and S/G2/M phases, with both density-dependent and age-dependent regulation acting at the G1/S transition. We investigate the checkpoint-mediated coupling between cell-cycle progression and crowding, and determine how contact inhibition shapes wave speed, front structure, and the age distribution of migrating cell populations.

MS66-02 — From discrete models of cell mechanics to continuum descriptions of tissue growth

SPEAKERS Shahak Kuba (Queensland University of Technology)
AUTHORS Matthew Simpson (Queensland University of Technology); Pascal Buenzli (Queensland University of Technology); Shahak Kuba (Queensland University of Technology)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

The rate at which biological tissues grow is regulated by the interplay between geometry, cell mechanics, and cellular processes. In scenarios where tissue growth occurs primarily at the surface of a confined environment – such as bone remodelling, wound healing, and tissue growth within engineered scaffolds – cells compete for space as they deposit new material. This competition leads to cell crowding or spreading depending on substrate curvature and generates mechanical stresses that may influence cellular processes including proliferation, differentiation, and survival. We present a discrete mathematical model for simulating tissue growth in confined geometries. The tissue interface is represented as a chain of mechanically interacting cells (modelled as springs) that simultaneously generate new tissue material. To more accurately capture cell population dynamics during tissue growth, we incorporate cell proliferation, death, and embedment as stochastic processes. To describe the collective behaviour of the cell population, we derive a continuum limit by representing each cell with m subcellular mechanical components and taking the limit as $m \to \infty$. This derivation yields a reaction–diffusion partial differential equation governing the evolution of cell density along a moving interface parameterised by arc length.

MS66-03 — Toward a unified theory of curvature-guided epithelial migration: ER remodeling, cytoskeletal polarity, and cell mechanics

SPEAKERS Fabian Spill (University of Birmingham)
AUTHORS Amirreza Khodadadian (Keele University); Andrew Hazel (University of Manchester); Fabian Spill (University of Birmingham); Maryam Parvizi (University of Birmingham)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Curvature-dependent epithelial migration is usually described at the level of actin and adhesion, but recent experiments reveal a central role for organelle mechanics. We combine new experimental evidence on wound-edge geometry with a unified variational model of single-cell migration to argue that the endoplasmic reticulum (ER) acts as a mechanotransducer linking curvature, cytoskeletal forces and migration mode. In epithelial gaps, convex edges promote ER tubules, perpendicular focal adhesions and lamellipodial crawling, whereas concave edges promote ER sheets, parallel adhesions and purse-string-like contraction. Building on these findings, we formulate a thermodynamically consistent framework that couples ER sheet–tubule remodeling, membrane curvature elasticity, actin turnover, microtubule reorganization, intracellular flow, and cell polarity within a diffuse-interface description. The model explains how geometry and force bias intracellular organization through strain-energy minimization and predicts how perturbations of ER structure reshape polarity and motility. Together, this work provides a route toward a predictive theory of epithelial migration in which intracellular architecture is not a passive readout of mechanics, but an active regulator of cell movement.

References

[Raw+25] Simran Rawal et al. “Edge curvature drives endoplasmic reticulum reorganization and dictates epithelial migration mode”. In: *Nature Cell Biology* 27.10 (2025), pp. 1660–1675.

MS66-04 — Modelling bulk mechanical effects in a planar cellular monolayer

SPEAKERS Natasha Cowley (Department of Mathematics, University of Manchester)
AUTHORS Natasha Cowley (Department of Mathematics, University of Manchester); Oliver Jensen (Department of Mathematics, University of Manchester); Sarah Woolner (Division of Cell Matrix Biology & Regenerative Medicine, University of Manchester)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

The 2D vertex model is successful in capturing many phenomena observed in epithelia but does not consider out-of-plane mechanical effects. To understand how 3D effects can be captured in 2D, we introduce a model for a monolayer of columnar cells where the energy includes terms relating to volume, surface area, lateral adhesion and cortical tension. When reduced to a 2D formulation, bulk effects due to volume and surface area introduce coupling between the apical area and perimeter. This coupling is not captured in the standard 2D model and leads to a more complex phenomenology. The reduced 2D model has five independent parameters, each of which can lead to a rigidity transition when varied; these are the target apical perimeter as used in the standard 2D model, as well as parameters controlling the strength of adhesion, cortical line tension, total area tension, and constrictive forces at the apical cortex. This reveals multiple potential mechanisms by which tissues can lose rigidity. We also find a sharp continuous squamous to columnar transition in the fixed volume limit, where a floppy region connects the two rigid states. In the rigid regime, the model shows how lateral crowding in a disordered isolated monolayer can lead to cell elongation towards the monolayer centre.

References

[CWJ25] Natasha Cowley, Sarah Woolner, and Oliver E Jensen. “Modeling bulk mechanical effects in a planar cellular monolayer”. In: *Physical Review E* 112.5 (2025), p. 054401.

MS80 — Stochastic Chemical Reaction Networks

TRACK Systems Biology and Biochemical Networks
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 11.01 - HS
ORGANISERS Philippe Robert (INRIA)

Minisymposium abstract

This set of talks is devoted to the investigation of the mathematical properties of chemical reaction networks described as Markov processes in a finite dimensional state space. The stochastic component of dynamics of the chemical reaction networks of this mini-symposium play a very important role for each of the examples investigated. Several properties of stochastic chemical reaction networks are investigated in these talks: stability properties (positive recurrence), characterization of the invariant distribution and functional limit theorems: law of large numbers and averaging principles.

MS80-01 — Stability of reaction networks with randomly switching parameters

SPEAKERS Aidan Howels (Polytechnico Torino)
AUTHORS Aidan Howels (Polytechnico Torino); Chuang Xu; Daniele Cappelletti (Politecnico di Torino)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Since the dawn of stochastic chemical reaction network theory over 50 years ago, there have been many general results about (positive) recurrence, especially in the case of mass-action kinetics. One less-explored area is that of mass-action models whose rate constants, rather than being static, are themselves stochastic. Such models have relevance in applications, since biomolecular systems rarely exist in isolation and their rates often depend on time-changing quantities.

This talk will present matrix conditions for positive recurrence and transience in the case where the system is switching between finitely many possible choices of rate constants. These conditions will depend on the specific choice of parameters for the model, which makes it possible to uncover phase transitions where the stability behavior of the model varies. We will see that the speed at which the rate constants are changing plays an important role, with the model behaving as one with averaged rate constants when this speed is high and behaving as though the rate constants were unaveraged when this speed is low. This talk is based on joint work with Daniele Cappelletti and Chuang Xu.

MS80-02 — Weakly Reversible Chemical Reaction Networks Are Recurrent in 2d

SPEAKERS Andrea Agazzi (Bern University)
AUTHORS Andrea Agazzi (Bern University)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

We prove that, for weakly reversible chemical reaction networks with stochastic mass-action kinetics in two species, the associated continuous-time Markov chain is positive recurrent on each closed irreducible communicating class. Equivalently, the process returns to finite sets infinitely often with finite expected return times, and it possesses an invariant probability measure supported on the class. The proof is based on a Foster–Lyapunov argument. Exploiting weak reversibility together with the geometric constraints of the two-dimensional state space, we construct a Lyapunov function allowing to establish, using pathwise large deviations estimates, sufficient asymptotic dissipation of the given process.

MS80-03 — Sensitivity of dynamics in Autocatalytic Reaction Networks of Togashi-Kaneko type

SPEAKERS Lea Popovic (Concordia University)
AUTHORS Grzegorz Rempala (Ohio State); Hye-Won Kang (University of Maryland, Baltimore County); Lea Popovic (Concordia University); Ruth Williams; Wasiur Rahman Khuda Bukhsh (University of Nottingham)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

The Togashi–Kaneko (TK) model is a prototypical example of an auto- catalytic reaction network exhibiting dramatic switching behavior that is a result of the stochastic dynamics at small volumes. I will present a study of the TK model with additional mutations, using a stochastic averaging principle to make use of the multi-scale feature of its dynamics. I will demonstrate a sensitivity of the model to even slight departures from symmetry in the autocatalytic reactions, using a detailed analysis of the stationary distribution of the fast process when the state of the slow process is fixed. This is joint work with Yi Fu, HyeWon Kang, Wasiur Khudabukhsh, Greg Rempala and Ruth Williams.

MS80-04 — Noise induced stabilization in stochastic chemical reaction network.

SPEAKERS Lucie Laurence (Bern University)
AUTHORS Andrea Agazzi (Bern University); Lucie Laurence (Bern University)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Chemical reaction networks (CRNs) are commonly analyzed through deterministic or stochastic models that track molecular populations over time. In regimes with large molecule counts, stochastic dynamics are typically approximated by deterministic mass-action kinetics. We present a CRN that defies this expectation: while the deterministic system is unstable, exhibiting finite-time blow-up of trajectories within the interior of the state space, its stochastic counterpart is positive recurrent.

MS83 — Automated discovery of dynamical digital twins from time series

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 15.05 - HS
ORGANISERS Annabelle Ballesta (Institut Curie, INSERM U1331, Cancer Systems Pharmacology team); Martin Rosalie (Université de Perpignan Via Domitia, France)

Minisymposium abstract

Dynamical digital twins stand out as powerful mechanistic tools to advance biomedical challenges. However, their design involves the integration of numerous multimodal time-resolved datasets calling for a change of practice, from manual design to automated discovery. The development of data-driven model learning approaches is constrained by the realities of biological data: scarcity and heterogeneity of time-resolved datasets available across multiple conditions, partial observability, and large candidate network spaces (1). This minisymposium aims to provide a coherent overview of modern strategies for inferring reaction networks' structure and dynamics under these constraints, clarifying how modeling assumptions and prior knowledge affect identifiability and robustness. Across four talks, we will cover a broad range of modeling formalisms, including Bayesian dynamical-system learning (2), sparse identification for tochastic delay differential equations (3), and equation discovery in complex systems with chaotic regimes (4). Critically, we will emphasize lessons from real-data applications, and discuss benchmarking practices needed to make automated digital-twin inference reliable and reproducible.

MS83-02 — Sparse identification of renewal equations with application to disease transmission dynamics

SPEAKERS Dimitri Breda (University of Udine, Italy)
AUTHORS Dimitri Breda (University of Udine, Italy); Jianhong Wu; Muhammad Tanveer; Xue Zhang
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Data-driven model discovery has become a powerful approach for identifying governing equations of dynamical systems using temporal data. The Sparse Identification of Nonlinear Dynamics (SINDy) algorithm, initially developed for ordinary differential equations (ODEs) [BPK16], has been extended to more general classes of problems, recently including also deterministic and stochastic delay differential equations with discrete delays [BBT24; Bre+26; Pec+25]. However, its application to systems with distributed delays and renewal equations remains unexplored. Distributed delays, at the core of renewal-type integral equations, involve integration over a continuum of past states. Related models are prevalent in biological and ecological applications to describe, e.g., structured populations and epidemics. They portrait memory-dependent dynamics but are challenging to identify due to the inherent complexity of delay kernels and renewal processes. Building on the integral formulation of SINDy for ODEs, we propose a novel extension of the SINDy framework to recover the (possibly nonautonomous) kernel of distributed delays through the use of quadrature formulas. As such the new approach aims at providing a sparse interpretable model rather than just a black-box right-hand side. We demonstrate the efficacy of the new method first on academic examples and then by applying it to real data of the transmission dynamics of Severe Fever with Thrombocytopenia Syndrome (SFTS) [Zha+25], an emerging tick-borne disease.

References

[BBT24] E. Bozzo, D. Breda, and M. Tanveer. "Sparse identification of time delay systems via pseudospectral collocation". In: *IFAC-PapersOnLine* 58.27 (2024), pp. 108–113.

[Bre+26] D Breda et al. "Sparse identification of Nonlinear Dynamics for Stochastic delay differential equations". In: *Journal of Computational and Applied Mathematics* 479 (2026), p. 117247.

[BPK16] S L Brunton, J L Proctor, and J N Kutz. "Discovering governing equations from data by sparse identification of nonlinear dynamical systems". In: *PNAS* 113.15 (2016), pp. 3932–3937.

[Pec+25] A Pecile et al. "Data-driven methods for delay differential equations". In: *J. Comput. Appl. Math.* 461 (2025), p. 116439.

[Zha+25] Xue Zhang et al. "Patterns of Transmission Dynamics of Severe Fever with Thrombocytopenia Syndrome Virus in Dalian: Roles of Systemic, Co-Feeding, and Transovarial Routes". In: *CSIAM Transactions on Life Sciences* 1.2 (2025), pp. 299–319. ISSN: 3006-2721. DOI: https://doi.org/10.4208/csiam-ls.SO-2025-0001.

MS83-01 — Discovering Dynamical Digital Twins from Time Series through Bayesian Sparse Regression

SPEAKERS Clémence Métayer (Institut Curie, INSERM U1331, Cancer Systems Pharmacology team)
AUTHORS Annabelle Ballesta (Institut Curie, INSERM U1331, Cancer Systems Pharmacology team); Clémence Métayer (Institut Curie, INSERM U1331, Cancer Systems Pharmacology team)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The automated discovery of dynamical digital twins from time series data, known as model learning, is a central challenge in systems biology, particularly in the presence of noise, partial observability, and limited data availability.

We recently conducted a comprehensive review of available methods for data-driven discovery of dynamical systems, and identified 117 algorithms based on either symbolic or sparse regression, which we evaluated across eight key biological and methodological challenges [MBM26].

We then propose a novel method based on sparse Bayesian inference that jointly estimates the structure and parameters of dynamical models while incorporating biological prior knowledge. The approach is specifically designed to integrate multi-condition data, as commonly encountered in biological experiments.

The method was first validated on simulated systems, where it exhibits robust performances in recovering interactions under multiple noise levels. It was then applied to circadian gene expression data from human lung cells to automatically infer the dynamical interactions between the clock and the innate immune receptor NLRP3. The ambition is to provide interpretable representations of the underlying biological processes and enable the exploration of system perturbations.

Overall, this work contributes to the development of automated pipelines for constructing biologically grounded digital twins, with potential applications in personalized medicine.

References

[MBM26] Clémence Métayer, Annabelle Ballesta, and Julien Martinelli. “Data-driven discovery of digital twins in biomedical research”. In: *Briefings in Bioinformatics* 27.1 (2026).

MS83-03 — Bayesian discovery of biochemical reaction networks from time-course data with projection predictive inference

SPEAKERS Julien Martinelli (Aalto University)
AUTHORS Julien Martinelli (Aalto University)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mechanistic ordinary differential equation models are central to systems biology, pharmacology and emerging dynamical digital twins, but they are still commonly built by hand through extensive literature review, manual specification of reaction mechanisms, and repeated parameter fitting. As biological datasets become larger and more diverse, this process becomes difficult to scale, motivating automated approaches to mechanistic model discovery.

We present a Bayesian framework for discovering biochemical reaction networks directly from time-resolved concentration data under mass-action kinetics. Starting from a library of candidate reactions, we infer parsimonious networks and rate parameters with full posterior uncertainty, using a projection-predictive search strategy in trajectory space that favors compact reaction sets while preserving predictive dynamics. We demonstrate the approach on synthetic benchmarks and real biological time-course data, where it recovers interpretable reaction networks, while quantifying structural uncertainty.

This work contributes a biologically grounded route toward automated, uncertainty-aware inference of reaction networks from limited experimental data.

MS83-04 — Global modelling of algae blooms from time series: comparison of chaotic attractors

SPEAKERS Martin Rosalie (Université de Perpignan Via Domitia, France)
AUTHORS Martin Rosalie (Université de Perpignan Via Domitia, France)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Global modelling such as GPoM [Man+12] is a tool for constructing systems of polynomial differential equations from time series. We present one use of this tool for variable selection. We study the bloom of the toxic algae (*Ostreopsis* cf. *ovata*) [Fab+24] and look for strong relationships between the alga and its environment (from COPERNICUS datasets) through the lens of dynamics. Using time series, we have highlighted other links that exist between the proliferation of this algae and salinity, oxygen, or nutrient concentrations.

The second part of the talk is dedicated to the chaotic dynamics emerging from the numerous ordinary differential equations systems generated with GPoM. The comparison of models is performed using topological characterization of the chaotic attractors [Ros16; RM25]. It enables to reduce the number candidates to a smaller set of systems that are able to reproduce dynamics observed in the data. Structures of the polynomial models of ordinary differential equations systems will be discussed as well.

References

[Fab+24] S. Fabri-Ruiz et al. “Harmful Ostreopsis cf. ovata blooms could extend in time span with climate change in the Western Mediterranean Sea”. In: *Science of The Total Environment* 947 (Oct. 2024), p. 174726. ISSN: 0048-9697. DOI: 10.1016/j.scitotenv.2024.174726. URL: http://dx.doi.org/10.1016/j.scitotenv.2024.174726.

[Man+12] S. Mangiarotti et al. “Polynomial search and global modeling: Two algorithms for modeling chaos”. In: *Phys. Rev. E* 86 (4 Oct. 2012), p. 046205. DOI: 10.1103/PhysRevE.86.046205. URL: https://link.aps.org/doi/10.1103/PhysRevE.86.046205.

[Ros16] M. Rosalie. “Templates and subtemplates of Rössler attractors from a bifurcation diagram”. In: *Journal of Physics A: Mathematical and Theoretical* 49.31 (2016), p. 315101. DOI: 10.1088/1751-8113/49/31/315101.

[RM25] M. Rosalie and S. Mangiarotti. “Structure analysis of the Lorenz-84 chaotic attractor”. In: *Chaos: An Interdisciplinary Journal of Nonlinear Science* 35.10 (Oct. 2025). ISSN: 1089-7682. DOI: 10.1063/5.0287725. URL: http://dx.doi.org/10.1063/5.0287725.

MS89 — Collective dynamics at multiple scales

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 03.01 - HS
ORGANISERS Christopher Strickland (University of Tennessee, Knoxville)

Minisymposium abstract

The dynamics and emergent behavior of collectives is a rich area of study in mathematical biology, and this session will focus on contributions to the field ranging from the molecular level all the way up to that of agricultural fields. In chemical theories of nucleation, growth, and nanoparticle formation, “collective behavior” refers to phenomena where many interacting species (monomers, clusters, ligands, solvent, fields) produce emergent kinetics, structures, or pathways that cannot be reduced to independent-particle events. Our session will consider such behavior in plant pigment systems. In ocean systems at the meter-scale,

plankton form a foundational layer of the food web as they interact with larger, filter feeding organisms. We will hear results regarding novel numerical methods for understanding this relationship and how active behavior may affect outcomes. Finally, at the landscape level, locust hopper bands are a well-known example of collective behavior with important implications for food security. Our session will present results on a new data assimilation and model selection pipeline allowing collective behavior models to be parameterized and compared using video data of locusts in the field. Apart from communicating results about ongoing research, a goal of this minisymposium is to start conversations about how the different perspectives and methods from these varying spatial scales may inform each other and generate new ideas and avenues for research.

MS89-01 – How perception and neural weighting can drive the geometry of decision-making in target pursuit

SPEAKERS Christopher Strickland (University of Tennessee, Knoxville)
AUTHORS Andrew Bernoff (Harvey Mudd College); Christopher Strickland (University of Tennessee, Knoxville)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Recent research, grounded in experiments (notably by Iain Couzin and collaborators), has connected neural ring models of vision to how animals navigate a complex landscape of attractive targets. In this talk we investigate the mathematical and biological implications of a three-stage model where animals pre-process visual stimuli to identify a discrete set of targets, process this input to select the dominant targets, and then post-process this information to navigate the landscape. Incorporating finite target sizes and a neural density allows consistent target acquisition and pursuit reflecting what is seen in nature. Mathematically, we show this model corresponds to an energy minimization problem, simplifying both its analysis and numerical implementation. Biologically, we argue that the model reproduces experimental observations and presents a fairly direct pathway to decoding neural geometry via empirical data.

MS89-02 – Small Organism Collective Behavior in Fluid Environments

SPEAKERS Margaret Knight (University of Tennessee, Knoxville)
AUTHORS Margaret Knight (University of Tennessee, Knoxville); Nicholas Battista (The College of New Jersey); Koee Chapman (University of Tennessee, Knoxville); Tyler Hall (University of Tennessee, Knoxville); Ani Harsha (University of Tennessee, Knoxville); Christopher Strickland (University of Tennessee, Knoxville)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The movement and behavior of small organism collectives can often play a key role in ecosystem function. One example is marine larval plankton which are critical for the health of filter feeders such as coral reefs and jellyfish. However, holistic modeling of scenarios like these can be a difficult multiscale problem involving individual locomotion dynamics within larger-scale flows. To address this problem, Dr. Christopher Strickland has developed an open-source, agent-based modeling library in the Python programming language called Planktos. It is targeted at collective behavior in 2D and 3D fluid environments with immersed structures and readily interacts with computational fluid dynamics data generated externally. In this talk, I present mathematical models for collective behavior and then apply them to novel and biologically interesting scenarios involving fluid flows and immersed boundaries- all within the Planktos framework.

MS89-03 – Pattern formation through collective behavior in plant pigment systems

SPEAKERS Patrick Shipman (University of Arizona)
AUTHORS Patrick Shipman (University of Arizona); Stephen Thompson (Colorado State University)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Anthocyanins are glycosidic flavonoid pigments responsible for most of the red, purple, and blue colors in flowers, fruits, leaves, stems, bracts, seeds, and pollen. These multifunctional secondary metabolites reside primarily in epidermal vacuoles and act as antioxidants, providing photoprotection, contributing to wound healing, and offering freeze protection. In the crowded vacuolar environment (pH 3–6), anthocyanins undergo a complex series of molecular transformations governed by pH, metal ions, copigments, and concentration. Color is determined by kinetic and thermodynamic relationships among anthocyanin species, as well as by concentration-dependent associative self-assembly. Microscopic analysis of live epidermal cells reveals multiple anthocyanic phases with sizes ranging from 1–10 μm. Self-assembly is driven by intra- and intermolecular π–π stacking, hydrogen bonding, ion pairing, and charge-shift interactions, leading to amphiphilic micelle formation. These associations can generate spatial patterning in petals. In our mathematical framework, pattern formation results from collective behavior due to emergent structures and pathways arising from interactions among many coupled species. We have also developed microfluidic, self-reporting spectroscopic methods to study these phase behaviors.

MS89-04 — Data Assimilation and Model Selection for Collective Motion in Locust Swarms

SPEAKERS Rebecca Everett (Haverford College)
AUTHORS Rebecca Everett (Haverford College); Jasper Weinburd (University of St. Thomas); John Maclean (Adelaide University); Christopher Strickland (University of Tennessee, Knoxville); J. Edward Green (Adelaide University); Maryann Hohn; Andrew Bernoff (Harvey Mudd College)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

In a striking example of collective behavior, swarms of locusts march in an apparently coordinated direction. Various swarm morphologies emerge in these groups that aid in feeding or migration, depending on the environment through which the swarm moves. However, unlike eusocial insects (such bees or ants) locusts have no social structure (or queen) to facilitate this directed motion. Instead, agent-based models with identical individuals are a natural lense with which to study the collective motion of locusts. In this talk, we will introduce a framework for evaluating the appropriateness of several models for individual interaction. The work is made possible by recent advances in the availability and magnitude of trajectory data on individual locusts within a swarm. We formulate a Bayesian particle filter to estimate parameters of a given model for individual interaction based on these data. We next conduct a simulation study using the given model with parameters from the posterior distribution. Finally, we compare quantities aggregated from the simulated data and the empirical data to determine if the model recreates similar collective motion.

MS92 — Modeling, Forecasting, and Management of Public Health and Ecological Threats

TRACK Mathematical Epidemiology
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 15.03 - HS
ORGANISERS Chidozie Williams Chukwu (Georgia Southern University, Statesboro Georgia, USA); Folashade Agusto (University of Kansas, USA)

Minisymposium abstract

This mini-symposium centers on the development and analysis of mathematical models arising from public health and ecological threats, including infectious diseases, substance-use–related mortality, and biological invasions. The talks emphasize the formulation and study of coupled, multi-scale, and non-autonomous dynamical systems, with attention to qualitative properties such as stability, asymptotic behavior, and sensitivity to perturbations. Additional focus is placed on the integration of analytical insight with forecasting and control strategies, illustrating how rigorous mathematical approaches can support intervention design and management of complex population-level systems.

This session is aimed at researchers and students in mathematical biology and applied mathematics interested in the analysis of dynamical systems arising in epidemiology and ecology. It will appeal particularly to those studying coupled, multi-scale models, asymptotic behavior, and mathematically grounded forecasting and control.

MS92-01 — Mathematical Models for Understanding and Managing Biological Invasions

SPEAKERS Ousmane Ousmane Seydi (Université Le Havre Normandie, France)
AUTHORS Ousmane Ousmane Seydi (Université Le Havre Normandie, France)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Invasive species pose major challenges for biodiversity and ecosystem management. In this talk, I present mathematical models to understand how biological invasions emerge, spread, and can be controlled. I first focus on early invasion stages, where stochastic effects are important, and discuss extinction probabilities and invasion thresholds. I then address invasion dynamics at larger spatial and temporal scales, with an application to the invasion of black cherry (Prunus serotina) in France, using an age-structured and spatially explicit model. I conclude with perspectives on spatial control strategies and coordinated management.

MS92-02 — A Hybrid Modeling Framework for Forecasting the 2025 Texas Measles Outbreak and Evaluating Vaccination Strategies

SPEAKERS Chidozie Williams Chukwu (Georgia Southern University, Statesboro Georgia, USA)
AUTHORS Chidozie Williams Chukwu (Georgia Southern University, Statesboro Georgia, USA)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

In this talk, we discuss a hybrid modeling framework to analyze measles transmission and vaccination strategies during the 2025 Texas outbreak. A deterministic model incorporating single- and double-dose vaccination with vaccine efficacy was calibrated to reported Texas measles data from January 20 to May27, 2025. To complement the mechanistic analysis, time-series forecasting methods, Facebook Prophet and a Gated Recurrent Unit are applied to predict daily new cases. The Prophet model outperforms the GRU based on standard performance metrics. Both models project continued low-level transmission, with an estimated average of 1–2 daily cases (95% CI: 1–2) through August 30, 2025, in the absence of additional interventions. These results highlight the importance of maintaining high vaccination coverage to prevent future outbreaks and reduce the risk of measles endemicity in Texas.

MS92-03 — Coupled Dynamics of COVID-19 and Drug Overdose Deaths

SPEAKERS Folashade Agusto (University of Kansas, USA)
AUTHORS Folashade Agusto (University of Kansas, USA)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

The simultaneous escalation of COVID-19 and drug overdose deaths motivates the development of mathematical frameworks capable of capturing interacting population-level processes across disparate time scales. We propose a coupled dynamical systems formulation that represents the joint evolution of an infectious disease process and a mortality process associated with substance use. The framework allows interactions to be encoded through coupling operators, time-dependent forcing, and feedback mechanisms arising from behavioral and structural drivers. The formulation is amenable to analytical investigation,

including questions of well-posedness, stability, and sensitivity to perturbations, and provides a basis for integration with data assimilation. This work establishes a general mathematical foundation for studying the dynamics of co-occurring public health crises.

MS92-04 — Associations of spatial dynamics of COVID-19 transmissions with social distancing measures on household visits in the Netherlands

SPEAKERS Ward Spee (National Institute for Public Health and the Environment (RIVM), the Netherlands)
AUTHORS Jantien Backer (National Institute for Public Health and the Environment (RIVM), the Netherlands); Jacco Wallinga (National Institute for Public Health and the Environment (RIVM), the Netherlands); Ward Spee (National Institute for Public Health and the Environment (RIVM), the Netherlands)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

During the COVID-19 pandemic, various social distancing measures were implemented to mitigate epidemic spread. To evaluate the spatial effects of these measures, mobility data were often used as a proxy for disease transmission [Sch+20; Gib+21]. Here, we analyzed the spatial patterns of 141,881 self-reported transmission pairs that occurred during household visits in the Netherlands from September 2020 until May 2021. We assessed whether the implementation and lifting of restrictions on household visits aligned with observed changes to spatial transmission patterns. In particular, we focused on transmissions with a long geographical distance between the residences of the infector and infectee, because they were a critical determinant of the speed of the epidemic. We found that long-distance transmissions reduced disproportionately at the start of the second COVID-19 wave (Sep. - Oct. '20), which supposedly slowed down epidemic spread. Interestingly, we did not observe similar reductions during the third (Dec. '20) and fourth wave (Mar. - Apr. '21). Overall, we found no robust association between the strictness of household visit restrictions and the proportion of long-range transmissions. Our findings demonstrated the usefulness of transmission data for evaluating social distancing measures, and suggested that policy changes did not always translate to changes in spatial transmission patterns.

References

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MS95 — Molecular Computing: Theory and Implementations

TRACK Systems Biology and Biochemical Networks
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 11.02 - HS
ORGANISERS Badal Joshi (California State University San Marcos); JINSU KIM (POSTECH); Tung Nguyen (UCLA)

Minisymposium abstract

Computational tasks can be implemented using biochemical species, such as DNA, RNA, lipids, and proteins, that interact through specially designed reaction networks. This research area encompasses biocomputing, molecular computing, and computing with reaction networks. The long-term vision is seamless integration of living biological systems, nanorobotics, and computing, with immediate applications in

autonomous nanocomputing, localized sensing and response, and pathogen detection. This minisymposium focuses on realistic biological constraints and opportunities, as well as design principles that effectively leverage them. The field has been shaped by close collaboration between mathematical theory and bioengineering to develop efficient future technologies. The minisymposium brings together mathematicians and bioengineers to foster synergies and advance collaborative research at this interdisciplinary frontier.

MS95-01 — From CRN to CNN: Deep Learning with Stochastic Chemical Reaction Networks

SPEAKERS Bernie Daigle (The University of Memphis)
AUTHORS Bernie Daigle (The University of Memphis)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Advances in deep neural networks (DNNs) have revolutionized our ability to model complex data via the capacity to approximate continuous functions with arbitrary accuracy. DNN models have been used to achieve or surpass human-level performance in tasks such as image classification, generative modeling, and scientific discovery. Recent studies of chemical reaction networks (CRNs) suggest similar potential; for example, it has been shown that stationary distributions of particular stochastic CRNs can closely approximate any distribution on the nonnegative integer lattice [Cap+20].

To explore this potential, we develop well-mixed stochastic CRN models with mass-action kinetics whose equilibrium behavior enables accurate image classification and realistic image generation. We construct our models as multilayer regulatory cascades with network motifs implementing standard neural network operations such as convolution, pooling, and nonlinear activation. To efficiently calibrate and evaluate these models, we apply the linear noise approximation (LNA), which provides a second-order approximation of the system size expansion of the chemical master equation (CME). We benchmark the ability of our models to classify and generate realistic images of handwritten digits based on the classic MNIST dataset, showing performance competitive with that of DNNs. Our results underscore the ability of CRNs to be universal approximators and suggest new methodological innovations for modeling complex data.

References

[Cap+20] Daniele Cappelletti et al. “Stochastic chemical reaction networks for robustly approximating arbitrary probability distributions”. In: *Theoretical Computer Science* 801 (Jan. 2020), pp. 64–95. ISSN: 0304-3975. DOI: 10.1016/j.tcs.2019.08.013. URL: https://www.sciencedirect.com/science/article/pii/S030439751930502X (visited on 03/21/2026).

MS95-02 — Buffered Chemical Reaction Networks for Reusable Computation

SPEAKERS Dominic Scalise (Washington State University)
AUTHORS Dominic Scalise (Washington State University)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Synthetic chemical reaction networks (CRNs) typically operate in closed systems with finite reactants, limiting most circuits to a single round of computation. This constraint prevents sustained dynamics such as repeated execution of Boolean logic.

Here a simple motif, called a CRN buffer, is described that enables repeated circuit operation. CRN buffers consist of high concentration reversible reactions, analogous to acid-base pH buffers, and can maintain the availability of species by reversible activation and deactivation. As long as the buffer concentration remains high, circuits continue to operate over multiple cycles.

We show theoretically how this motif extends a range of chemical computations from single-use to multi-cycle operation, enabling reusable digital logic, sequential logic, sustained analog arithmetic, robust oscillators, and stable reaction-diffusion systems that can maintain spatial patterns over extended durations.

We then present preliminary experimental implementations using DNA strand-displacement (DSD) reactions, including a simple DSD reaction that operates for ten cycles. Our experimental results incorporate recent advances in circuit preparation that reduce leak in high-concentration regimes, enabling reliable operation under the conditions required for buffering.

Buffered reaction networks provide a general strategy for sustaining chemical computation, enabling chemical circuits to operate for many cycles in closed systems.

MS95-03 — Event-based reservoir computing via chemical reaction networks

SPEAKERS Jinsu Kim (Pohang University of Science and Technology)
AUTHORS Jinsu Kim (Pohang University of Science and Technology)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

We investigate a reservoir computing framework based on unimolecular (linear) chemical reaction networks. While the mean-field dynamics are linear, stochastic dynamics can induce richer input–output behaviour through long-time statistics. Using tools from ergodic theory and reaction network structure, we study how such systems generate feature representations and assess their expressive potential under simple readouts, with preliminary indications of universal approximation under suitable conditions. These observations suggest that stochasticity and long-time behaviour can enable nonlinear computation within structurally simple biochemical systems.

MS95-04 — Heterochiral DNA for intracellular computing

SPEAKERS Matthew Lakin (University of New Mexico)
AUTHORS Anshika Mishra (University of New Mexico); Matthew Lakin (University of New Mexico); Tracy Mallette (University of New Mexico)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Many societal challenges, including the diagnosis and treatment of disease, can be tackled by harnessing the unique capabilities of biology. A key goal in biomedical engineering is to improve human health via personalized biomedicine, which promises targeted treatment guided by the patient's physiology. To this end, I work to engineer and build programmable biological systems for autonomous sensing, decision-making, and response in living organisms, combining experimental validation with formal computational methods for the specification, compilation, and verification of designs. In this talk, I will outline some of my contributions, focusing on the use of heterochiral DNA, which combines both naturally occurring D-DNA and chiral mirror image L-DNA, to build robust nanodevices for sensing and decision-making in the harsh environment of a living cell. My work has shown that DNA strand displacement reactions can be used to translate nucleic acid signals between chiralities [Mal+20], and that L-DNA can be used to protect D-DNA molecular components from degradation in biological environments [ML22; MML26]. I will also discuss some preliminary work on the transfection of heterochiral components into living mammalian cells [MLL24].

References

[ML22] Tracy L. Mallette and Matthew R. Lakin. "Protecting heterochiral DNA nanostructures against exonuclease-mediated degradation". In: *ACS Synthetic Biology* 11.7 (2022), pp. 2222–2228. DOI: 10.1021/acssynbio.2c00105.

[MLL24] Tracy L. Mallette, Diane S. Lidke, and Matthew R. Lakin. "Heterochiral modifications enhance robustness and function of DNA in living human cells". In: *ChemBioChem* 25.5 (2024), e202300755. DOI: 10.1002/cbic.202300755.

[MML26] Tracy L. Mallette, Anshika Mishra, and Matthew R. Lakin. "On degradation-induced leak in heterochiral DNA strand displacement cascades". In: *ACS Synthetic Biology* 15.2 (2026), pp. 599–609. DOI: 10.1021/acssynbio.5c00680.

[Mal+20] Tracy L. Mallette et al. "Robust heterochiral strand displacement using leakless translators". In: *ACS Synthetic Biology* 9.7 (2020), pp. 1907–1910. DOI: 10.1021/acssynbio.0c00131.

MS106 — GLIMPRINT Minisymposium: From Mechanistic Multiscale Immune Models to Digital Twins

TRACK Immunobiology & Infection
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 02.21 - HS
ORGANISERS James Doran (University of Bath); James Glazier (Indiana University); Lorenzo Veschini (Indiana Uniersity)

Minisymposium abstract

GLIMPRINT (the Global Alliance for Immune Prediction and Intervention) is a community advancing mechanistic modeling methods for infection and immune response, with the long-term goal of enabling immune digital twins—computational representations that connect biological mechanism to patient- and tissue-relevant predictions. This minisymposium highlights complementary multiscale approaches spanning sepsis and acute inflammation, fungal lung infection dynamics, T cell regulation and functional responses, and influenza infection with spatially explicit tissue-scale modeling. Across these applications, speakers emphasize principled links from molecular and cellular processes to tissue and organism-level outcomes, integration of experimental and clinical observations, and practical workflows for using models to interrogate disease trajectories and evaluate candidate interventions. Together, the talks illustrate how mechanistic multiscale immune modeling is maturing into actionable digital-twin approaches across diverse infectious and inflammatory contexts.

References

[AC24] Gary An and Chase Cockrell. *A design specification for Critical Illness Digital Twins (CIDTs) to cure sepsis: responding to the National Academies of Sciences, Engineering and Medicine Report "Foundational Research Gaps and Future Directions for Digital Twins"*. arXiv:2405.05301v2. 2024. arXiv: 2405.05301.

[LSG21] Reinhard Laubenbacher, James P. Sluka, and James A. Glazier. "Using digital twins in viral infection". In: *Science* 371.6534 (2021), pp. 1105–1106. DOI: 10.1126/science.abf3370.

[Rib+22] Henrique Al Ribeiro et al. "Multi-scale mechanistic modelling of the host defence in invasive aspergillosis reveals leucocyte activation and iron acquisition as drivers of infection outcome". In: *Journal of the Royal Society Interface* 19.189 (2022), p. 20210806. DOI: 10.1098/rsif.2021.0806.

[Wer+21] Kenneth Y. Wertheim et al. "A multi-approach and multi-scale platform to model $CD4^+$ T cells responding to infections". In: *PLoS Computational Biology* 17.8 (2021), e1009209. DOI: 10.1371/journal.pcbi.1009209.

MS106-02 — Introductions + Junior member mini-presentation

SPEAKERS Lorenzo Veschini (Indiana Uniersity)
AUTHORS Lorenzo Veschini (Indiana Uniersity)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Introductions Lorenzo Veschini We will outline GLIMPRINT activities and introduce the speakers to the audience.

Junior member mini-presentation James Doran, Bath University, UK

Equation Learning for multiscale models of infectious diseases

Tuberculosis (TB) is an airborne disease caused by the pathogen Mycobacterium tuberculosis. In 2023, according to the World Health Organization, it "probably" replaced COVID-19 as the leading cause of death from an infectious agent globally; in the nineteenth century, one in seven of all humans deaths were as a result of tuberculosis. More than 10 million people are diagnosed with TB every year. The majority of cases in adults occur in males (62.5% of all global adult cases in 2023, compared to 37.5% in females). The main reasons for males suffering from a higher burden of global TB cases, compared to females, is likely to be a combination of within-host factors, such as differences in immune response, and population-scale factors, such as likelihood of completing treatment. To investigate the impact different scales have in determining this higher TB burden in males, we have developed a gender/sex-stratified multiscale framework. We have learnt ordinary differential equations (ODEs) to capture the average output of an agent-based within-host model, and used the resulting equations to describe the within-host scales of the multiscale framework. We evolve the population demographics at the between-host scale using ODEs and link the scales with stochastic coupling functions. We have considered counterfactual scenarios to elucidate the impact of sex and gender on the infectious disease dynamics of TB. This paper is intended to provide a proof-of-concept for the development and implementation of the presented multiscale framework

MS106-03 — Multiscale modeling of the innate immune response to respiratory pathogens

SPEAKERS Reinhard Laubenbacher (University of Florida)
AUTHORS Reinhard Laubenbacher (University of Florida)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

This talk will describe agent-based modeling of the early immune response to fungal and viral respiratory pathogens and some applications.

MS106-04 — A Large-Scale Logic-Based Model of the Human Immune System as a Foundation for Mechanistically Interpretable Clinical Prediction and Immune Digital Twins

SPEAKERS Tomas Helikar (University of Nebraska, Lincoln)
AUTHORS Tomas Helikar (University of Nebraska, Lincoln)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Predicting patient-specific immune outcomes requires frameworks that are mechanistically grounded, scalable across disease contexts, and capable of producing interpretable clinical predictions. We present an integrated pipeline built around a large-scale logic-based mechanistic model of the human immune system. The model encodes Boolean regulatory logic derived from 449 human experimental publications, representing 88 immune cell types across innate and adaptive compartments, 37 secretory factors, and 11 disease environments connected through 1,450 regulatory interactions. Validation against independent human data confirmed the model's capacity to reproduce pathogen-specific cytokine signatures and cell dynamics across nine pathogens, with agreement rates of 75–90%. We leverage this validated model to generate synthetic datasets of in silico patient profiles by systematically varying immune state activity across the full spectrum from healthy to severely dysregulated. Machine learning models trained on

these data identify patient-specific biomarkers predictive of pathogen clearance, hospitalization, and ICU admission across IAV, SARS-CoV-2, CMV, and Plasmodium falciparum. Critically, because each biomarker is a node in the underlying mechanistic model, its predictive weight can be traced directly to causal regulatory logic, addressing the interpretability gap that limits purely data-driven tools. This pipeline provides a generalizable approach to mechanistically grounded biomarker discovery and a methodological foundation for personalized immune digital twins.

MS106-01 — Using an Agentic AI Critical Illness Digital Twin to rescue from immune exhaustion in sepsis

SPEAKERS Gary An (University of Vermont)
AUTHORS Gary An (University of Vermont)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Critical illness (CI) represents a highly significant healthcare issue, not only because of the resources required for its acute management in ICUs, but also in terms of the lingering health effects as a chronic disease on survivors. A central hallmark of CI is immune dysfunction, with both early hyperactivation leading to organ dysfunction and subsequent immunocompetency leading to increased susceptibility to infection and shortened life. The effective control of CI requires personalized precision medicine, which requires the capabilities provided by digital twins compliant with industrial standards and consistent with the definition put forth by the National Academies of Science, Engineering and Medicine (NASEM) in 2024. The Critical Illness Digital Twin (CIDT) is a proposed cyberphysical system compliant with the NASEM report that melds mechanistic models with machine learning and artificial intelligence and intrinsically incorporates control via an ongoing bidirectional sense-actuate connection to a real-world individual patient. We propose embedding the CIDT in an agentic-AI system that can evolve patient state space characterization, forecast capability and control policy optimization with the goal of rescuing ICU patients with immune exhaustion. Key points are the necessary integration of the virtual assets with sensor/assay technology and control modalities flexible enough to accomplish the stated goal.

MS108 — Mechanisms of Brain Development, Structure, and Metabolic Impacts

TRACK Neuroscience and Neural Systems
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 15.12 - HS
ORGANISERS Julianna Capece (Department of Mathematics, Florida State University); Monica K. Hurdal (Department of Mathematics, Florida State University)

Minisymposium abstract

Neural systems depend on both cellular mechanisms and organization to execute functioning in the brain. Understanding these processes requires bridging the gap across cellular and organismal levels. This minisymposium explores mechanisms that contribute to brain physiology and regulation, from the micro to the macro, using novel mathematical modeling approaches. By presenting research investigating developmental, structural, and metabolic impacts, this session aims to describe the biological underpinnings governing brain structure and function. Topics include biomechanical and biochemical mechanisms that drive brain development, axonal and brain microstructure estimates to capture underlying biophysical processes, and how type 2 diabetes can give rise to neurodegeneration and Alzheimer's disease. Theoretical frameworks in conjunction with experimental data, qualitative comparisons to imaging data, and numerical simulations lead to insights that improve our understanding of the brain.

MS108-01 — Sub-diffusion Compartmental Model for Diffusion MRI to Probe Brain Microstructural Information: Insights from a Simulation Study of White Matter

SPEAKERS S M Erfanul Kabir Chowdhury (School of Mathematical Sciences, Queensland University of Technology, Australia)

AUTHORS S M Erfanul Kabir Chowdhury (School of Mathematical Sciences, Queensland University of Technology, Australia); Qianqian Yang (School of Mathematical Sciences, Queensland University of Technology, Australia); Tim Moroney (School of Mathematical Sciences, Queensland University of Technology, Australia); Adrianne Jenner (School of Mathematical Sciences, Queensland University of Technology, Australia)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Axon radius and extra-axonal volume fraction are fundamental descriptors of white matter microstructure, critically shaping conduction velocity and the efficiency of neural communication. Although existing diffusion MRI models have attempted to estimate these parameters, their limited sensitivity and constrained assumptions often result in unstable and biased estimates. Such bias typically arises from inappropriate compartmental modelling that can not capture the underlying biophysical processes accurately, and from confounding effects among the fitted model parameters. To overcome these limitations, we propose a novel sub-diffusion compartmental model that achieves accurate estimation of axon radius and extra-axonal volume fraction in axon substrates with both uniform and gamma-distributed radii. Using numerical simulations of white matter microstructure with varying axon radii, diffusion times, and noise levels, we demonstrate that the proposed model yields improved stability of extracellular volume fraction and axon radius estimates compared to a mono-exponential extracellular diffusion model. These results suggest that incorporating sub-diffusive extracellular dynamics can mitigate diffusion time-dependent bias in compartmental DW-MRI modelling.

MS108-02 — Energy Constraints and Neural Strategy Transitions in Alzheimer's: A Game-Theoretic Model

SPEAKERS Irina Kareva (Department of Biomedical Engineering, Northeastern University, U.S.A.)
AUTHORS Irina Kareva (Department of Biomedical Engineering, Northeastern University, U.S.A.); Georgy Karev (National Center for Biotechnology Information, National Institutes of Health, U.S.A., retired)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

While many mechanisms have been proposed to drive Alzheimer's disease, particularly the accumulation of amyloid plaques and hyperphosphorylation of tau proteins, emerging evidence suggests that they may be the byproducts of earlier damage rather than initiating events. Instead, metabolic dysfunction and the inability of neural cells to support their energetic demands may be a more plausible trigger for subsequent pathological cascade (the neuron energy crisis hypothesis). Here we highlight how type 2 diabetes (T2D) can contribute to neurodegeneration by impairing brain energy metabolism. We present a game-theoretic framework, where neurons face trade-offs between energy efficiency and information fidelity. We show that under metabolic stress, neural networks can evolve toward smaller group sizes that prioritize energy efficiency over information quality, which may underlie the observed collapse of cognitive capacity during neurodegeneration. We conclude with a discussion of interventions, ranging from antidiabetic drugs to cognitive engagement and sensory stimulation, aimed at reducing metabolic stress and preserving cognitive function.

MS108-03 — Biomechanical Forces and Tensions that Contribute to Cortical Folding in the Brain

SPEAKERS Julianna Capece (Department of Mathematics, Florida State University)
AUTHORS Julianna Capece (Department of Mathematics, Florida State University); Monica K. Hurdal (Department of Mathematics, Florida State University)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

The outermost layer of the brain's surface, the cerebral cortex, consists of gyri (hills) and sulci (valleys). In humans, cortical folding begins around the 16th week of gestation, and the most obvious cortical folding changes occur during the 26th week of gestation. The mechanism by which the folding pattern develops remains unknown; however, several hypotheses suggest that cortical folding occurs through biochemical or biomechanical mechanisms. The axonal tension hypothesis in particular claims that folding is caused by mechanical tension in axons. Based on a previous model that uses the location and magnitude of stress-strains to simulate cortical folding, our current model simulates biomechanical forces that contribute to cortical deformations over a series of time steps. We represent our model cortex with a semicircular mesh discretized into linear, quadrilateral elements, and a finite element method process is used to numerically compute the updated nodal displacements at each time step. We test our model with ferret MR imaging data, and our model is used to anticipate the biomechanical forces observed. In modeling the deformations, we gain insight into how sulci develop through the evolution of time.

MS108-04 — A Biomchemical Model of Cortical Folding via Turing Patterns

SPEAKERS Monica K. Hurdal (Department of Mathematics, Florida State University)
AUTHORS Monica K. Hurdal (Department of Mathematics, Florida State University)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Turing pattern formation has been used to model many biological patterns including animal coat, fish, and butterfly patterns. We apply a Turing reaction-diffusion system to a prolate spheroid domain representing the developing cortex, to study cortical folding pattern formation. The brain's cerebral cortex has complex folds with hills (gyri) and valleys (sulci). While the mechanism for the development of cortical folds is not understood, one proposed theory suggests that patterning of a non-uniform distribution of intermediate progenitor (IP) cells during neurodevelopment correlates with cortical folds. We model IP cell patterning via the activator morphogen of the Turing system. Patterns generated by our Turing system represent prepatterns of self-amplification of IP cells. Our results demonstrate that cortical folding patterns are influenced by the shape of the domain, growth rate, and genetic control. Using these parameters, our Turing models of cortical folding can be used to explain diseases of cortical folding pattern formation, such as lissencephaly and polymicrogyria.

MS114 — Multiscale modelling and simulation of stochastic gene regulation

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 15.06 - HS
ORGANISERS Susanna Röblitz (University of Bergen, Norway); Maryam Yousefian (University of Bergen, Norway)

Minisymposium abstract

Gene expression is inherently stochastic and controlled at multiple stages, including epigenetic and transcriptional regulation. These processes are complex and often result in multimodal distributions. For example, the presence or absence of DNA methylation at a given genomic location exhibits a generally bimodal distribution, which can be linked to transcriptional variability. Likewise, gene regulatory networks give rise to multimodal protein distributions, with random transitions between the modes contributing to cell fate decisions. Advances in single-cell sequencing and the availability of large epigenomic datasets have increased the interest in utilizing mathematical models to understand these processes. However, standard approaches for stochastic chemical kinetics have drawbacks, such as long simulation times for the Stochastic Simulation Algorithm (SSA), especially for multimodal distributions, or impractically large system sizes for the Chemical Master Equation (CME). This minisympsium highlights methods that have been developed to overcome these drawbacks. This includes (1) an efficient mean-field CME approximation, which quantitatively recapitulates methylation bimodality [Bon+24], (2) a hybrid discrete/continuous model for transcriptional bursting [Fou+25], (3) a domain decomposition approach for SSA [You+25a], and (4) neural networks for learning the macroscopic dynamics [You+25b].

References

[Bon+24] Kwadwo A Bonsu et al. “A Tunable, Ultrasensitive Threshold in Enzymatic Activity Governs the DNA Methylation Landscape”. In: *bioRxiv* (2024), pp. 2024–06.

[Fou+25] Clémence Fournié et al. “Cell Trajectory Inference based on Schrödinger Problem and a Mechanistic Model of Stochastic Gene Expression”. In: *bioRxiv* (2025), pp. 2025–11.

[You+25a] Maryam Yousefian et al. “Efficient construction of Markov state models for stochastic gene regulatory networks by domain decomposition”. In: *BMC Bioinformatics* 26.1 (2025), p. 147.

[You+25b] Maryam Yousefian et al. “Exploring Metastable Dynamics of Gene Regulatory Networks with ISOKANN”. In: *International Conference on Computational Methods in Systems Biology*. Springer. 2025, pp. 126–149.

MS114-01 — The role of DNA methylation and DNA sequence in epigenetic plasticity

SPEAKERS Elizabeth Read (University of California, Irvine)

AUTHORS Elizabeth Read (University of California, Irvine); Kwadwo A. Bonsu (University of California, Irvine); Nandor Laszik (University of California, Irvine); Timothy L. Downing (University of California, Irvine)

SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 15.06 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

DNA methylation is a ubiquitous epigenetic mark that plays important yet disparate roles in gene regulation. On the one hand, genome-wide methylation patterns help establish and maintain distinct cell types, and these patterns are stably maintained. On the other hand, patterns in some loci are dynamic, facilitating nimble cellular responses to environmental stimuli. This talk will present our efforts combining discrete stochastic mathematical modeling with data-driven statistical inference approaches to shed light on enzymatic mechanisms that enable the mammalian DNA methylation system to accomplish both stability and responsiveness [Bon+26]. Integration with sequencing data is enabled by a novel mean-field approximation that can efficiently handle collective behavior among multiple genomic sites. We show how local genetic and non-genetic factors control a sensitive DNA methylation switch. As DNA methylation works in concert with other epigenetic mechanisms, our results could contribute to improved models of gene regulatory networks and epigenetic landscapes.

References

[Bon+26] Kwadwo A. Bonsu et al. “ATunable, Ultrasensitive Threshold in Enzymatic Activity Governs the DNA Methylation Landscape”. In: *Biophysical Journal* (2026). ISSN: 0006-3495. DOI: https://doi.org/10.1016/j.bpj.2026.03.013. URL: https://www.sciencedirect.com/science/article/pii/S0006349526001852.

MS114-02 — Joint trajectory and gene regulatory network inference using a mechanistic optimal transport-based framework

SPEAKERS Elias Ventre (INRIA Centre d’Université Côte d’Azur)

AUTHORS Elias Ventre (INRIA Centre d’Université Côte d’Azur); Yann Mauge (ENS Lyon)

SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 15.06 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

A key challenge in inferring gene regulatory networks (GRNs) governing cellular processes, such as differentiation and reprogramming, from experimental data lies in the impossibility of directly observing protein trajectories at the single-cell level, which prevents establishing causal relationships between regulator activity and target responses. In this talk, we present CardamomOT, a new algorithm that uses temporal

snapshots of scRNA-seq data to calibrate a mechanistic model of gene expression [MV26]. The method reconstructs both the GRN and the unobserved protein trajectories using an innovative mechanistic optimal transport framework. We present some results on both in silico and experimental datasets, demonstrating the ability to accurately recovers velocity fields driving cellular trajectories and unobserved protein levels, alongside reliable GRN structures. We finally show that the calibrated mechanistic model can be used as a generative model to predict cellular responses to unseen perturbations.

References

[MV26] Yann Mauge and Elias Ventre. "CardamomOT: a novel mechanistic optimal transport-based framework for joint gene regulatory network and trajectory inference". In: *bioRxiv* (2026), pp. 2026–13.

MS114-03 — A domain decomposition approach to the construction of Markov state models for stochastic gene regulatory networks

SPEAKERS Maryam Yousefian (University of Bergen (UiB))
AUTHORS Anna-Simone Frank (University of Bergen, Norway); Marcus Weber (Zuse Institute Berlin (ZIB)); Maryam Yousefian (University of Bergen (UiB)); Susanna Röblitz (University of Bergen, Norway)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Within a cell, gene expression levels are governed by molecular regulators interacting with each other in gene regulatory networks (GRNs). In this talk, we present an efficient computational framework for analyzing stochastic GRNs, in which cellular behavior is characterized by multiple metastable phenotypes and rare transitions between them. The dynamics of stochastic GRNs is described by the chemical master equation (CME), whose high dimensionality makes direct analysis computationally prohibitive. To address this, a domain decomposition approach (DDA) has been introduced [You+25], in which the state space is discretized using Voronoi tessellations and reduced to a Markov state model (MSM) [Chu+17]. In this setting, adaptive stochastic sampling combined with spectral clustering in terms of PCCA+ [FSR24] enables the identification of metastable states and their transition dynamics without relying on high-performance computing. Evaluation on two biological models, one for the genetic toggle switch and one for macrophage polarization, shows that the method accurately detects metastable phenotypes, estimates transition probabilities, and provides uncertainty quantification. Our findings highlight that accuracy is mainly driven by the number of Voronoi cells, while uncertainty is controlled by the sampling effort. The approach is computationally efficient and easily parallelizable, offering a practical tool for studying complex stochastic cellular dynamics and advancing the understanding of gene regulation and phenotype switching.

References

[Chu+17] Brian K Chu et al. "Markov state models of gene regulatory networks". In: *BMC Systems Biology* 11.1 (2017), p. 14.

[FSR24] Anna-Simone Frank, Alexander Sikorski, and Susanna Röblitz. "Spectral clustering of Markov chain transition matrices with complex eigenvalues". In: *Journal of Computational and Applied Mathematics* 444 (2024), p. 115791.

[You+25] Maryam Yousefian et al. "Exploring Metastable Dynamics of Gene Regulatory Networks with ISOKANN". In: *International Conference on Computational Methods in Systems Biology*. Springer. 2025, pp. 126–149.

MS114-04 — From Simulation to Macroscopic Dynamics: Learning Reaction Coordinates and Sampling Rare Transitions in Stochastic Gene Regulatory Networks

SPEAKERS Alexander Sikorski (Freie Universität Berlin)
AUTHORS Alexander Sikorski (Freie Universität Berlin); Luca Donati (Zuse Institute Berlin (ZIB)); Marcus Weber (Zuse Institute Berlin (ZIB)); Maryam Yousefian (University of Bergen (UiB)); Susanna Röblitz (University of Bergen, Norway)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Rare transitions between metastable states in stochastic gene regulatory networks are difficult to resolve in practice, as direct stochastic simulations require large amounts of data to capture these events reliably. In this talk, we present a neural network–based approach (ISOKANN) to learn low-dimensional reaction coordinates that describe the slow dynamics of such systems [Sik+25; You+25; SRW24]. These coordinates provide both a coarse-grained, dynamical view of the system and a basis for adaptive sampling. Starting from unbiased simulations, computational effort is iteratively focused on transition regions by resampling along the learned coordinate. This substantially reduces the amount of simulation time required while improving the resolution of transition pathways. At the same time, the learned representation captures the dominant slow processes in a dynamically consistent way. We illustrate the approach on prototypical stochastic gene regulatory models and discuss how combining representation learning with adaptive sampling enables an efficient analysis of rare event dynamics. The emphasis is on intuition and practical insights rather than technical details.

References

[SRW24] Alexander Sikorski, Enric Ribera Borrell, and Marcus Weber. “Learning Koopman eigenfunctions of stochastic diffusions with optimal importance sampling and ISOKANN”. In: *Journal of Mathematical Physics* 65.1 (2024).

[Sik+25] Alexander Sikorski et al. “Capturing the macroscopic behavior of molecular dynamics with membership functions”. In: *Mathematical Optimization for Machine Learning: Proceedings of the MATH+ Thematic Einstein Semester 2023*. Berlin, Boston: De Gruyter, 2025, pp. 41–58. ISBN: 9783111376776. DOI: doi:10.1515/9783111376776-004. URL: https://doi.org/10.1515/9783111376776-004.

[You+25] Maryam Yousefian et al. “Exploring Metastable Dynamics of Gene Regulatory Networks with ISOKANN”. In: *International Conference on Computational Methods in Systems Biology*. Springer. 2025, pp. 126–149.

MS138 — Modelling the interplay between radiotherapy, cell metabolism, and tumor dynamics

TRACK Mathematical Oncology
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 15.04 - HS
ORGANISERS Angélique Stéphanou (CNRS); Botao Dai (Grenoble-Alpes University); Damian Bimbenet (Swansea University); Mathilde Badoual (CNRS IN2P3/Université Paris Cité); Nahum Puebla-Osorio (University of Texas MD Anderson Cancer Center); Sarah Brueningk (University of Bern)

Minisymposium abstract

More than 50% of cancer patients undergo radiotherapy. However, radioresistance too often leads to tumor recurrence and poor prognosis. Recent studies (for reviews, see [Gao+25]) have highlighted the key role of glycolytic reprogramming in modulating tumor cell responses to radiation. In tumors, under hypoxic and nutrient-limited conditions, cells enhance glycolysis and lactate production in order to maintain high proliferation rates. This metabolic adaptation enhances resistance to radiotherapy by modulating oxidative stress and facilitating DNA repair pathways.

Beyond intracellular mechanisms, radiotherapy responses are characterized by strong inter-patient heterogeneity and complex tumor volume dynamics over time, often observed under clinically realistic right-censored follow-up conditions. Accurately modeling these longitudinal tumor trajectories is therefore essential to better understand treatment response, resistance emergence, and to guide personalized therapeutic strategies.

In this minisymposium, we will explore recent advances in mathematical and data-driven modeling approaches that capture the multiscale interplay between cellular metabolism, radiation response, and tumor growth dynamics. In particular, we will discuss hybrid mechanistic models and geometric adversarial learning frameworks for reconstructing censored tumor evolution directly from clinical data, offering new perspectives for uncertainty-aware prediction of radiotherapy outcomes.

References

[Gao+25] Shuaining Gao et al. "Glucose Metabolism Modulation as a Strategy to Enhance Cancer Radiotherapy". en. In: *Metabolites* 15.12 (Dec. 2025), p. 793. ISSN: 2218-1989. DOI: 10.3390/metabo15120793. URL: https://www.mdpi.com/2218-1989/15/12/793 (visited on 05/11/2026).

MS138-01 — Quantifying the effect of initial cell density on hypoxia-induced radioresistance: experimental analysis and compartmental modeling of cell state dynamics

SPEAKERS Botao Dai (CNRS, Grenoble-Alpes University, TIMC , Paris Saclay University, Paris Cité University, IJCLab, France)

AUTHORS Botao Dai (CNRS, Grenoble-Alpes University, TIMC , Paris Saclay University, Paris Cité University, IJCLab, France); Delphine Crépin (CNRS, Paris Saclay University, Paris Cité University, IJCLab, France); Stéphane Plaszczynski (CNRS, Paris Saclay University, Paris Cité University, IJCLab, France); Olivier Seksek (CNRS, Paris Saclay University, Paris Cité University, IJCLab, France); Mathilde Badoual (Paris Cité University, CNRS, Paris Saclay University, IJCLab, France)

SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 15.04 - HS

TRACK Mathematical Oncology

Abstract

More than 50% of patients with cancer receive radiotherapy to kill tumor cells. Several mathematical models predicting post-irradiation cell survival are used clinically to plan treatment; however, they often neglect the tumor microenvironment, which can modulate radiotherapy response. A key driver of radioresistance in solid tumors is hypoxia. In this project, we focus on the effect of initial cell density on hypoxia-induced radioresistance. Our experiments are designed to quantify how reduced oxygen levels before and after irradiation and initial cell density affect survival and post-treatment dynamics. We show that the hypoxia-induced radioresistance is stronger for high initial cell densities. We also present a biologically motivated compartmental model to analyze the dynamic transitions of cells between distinct states (e.g., repaired, senescent, and unrepaired subpopulations) under different conditions. By fitting time-course data, the model estimates key transition rates and incorporates oxygen dependence, enabling accurate simulation of cellular responses under hypoxic conditions. This work provides insight into the oxygen-dependent responses of cancer cells to radiation, informing more effective and context-aware therapeutic strategies.

MS138-02 — Exploring how metabolism comes into play with radiations: a mechanistic approach

SPEAKERS Damian Bimbenet (1. Univ. Grenoble Alpes, CNRS, UMR 5525, VetAgro Sup, Grenoble INP, TIMC, 38000 Grenoble, France 2. Department of Mathematics, Swansea University, Swansea SA1 8EN, UK)

AUTHORS Angélique Stéphanou (Univ. Grenoble Alpes, CNRS, UMR 5525, VetAgro Sup, Grenoble INP, TIMC, 38000 Grenoble, France); Damian Bimbenet (1. Univ. Grenoble Alpes, CNRS, UMR 5525, VetAgro Sup, Grenoble INP, TIMC, 38000 Grenoble, France 2. Department of Mathematics, Swansea University, Swansea SA1 8EN, UK); Gibin Powathil (Department of Mathematics, Swansea University, Swansea SA1 8EN, UK)

SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 15.04 - HS

TRACK Mathematical Oncology

Abstract

The reciprocal interactions between ionising radiation (IR) and glucose metabolism in mammalian cells have gained interest over these last decades. While reactive oxygen species (ROS) and HIF-1α emerge as key mediators in these interactions, the influence of ROS on HIF-1α following irradiation is still being debated. Different types of intermediate entities between ROS and HIF-1α have been proposed, each with different modes of action. In this study we propose to identify potential intermediates through their mode of action for several cell types studied in the literature. This is realized by reproducing the measured dynamics of key metabolic proteins following IR, specifically the LDH protein involved in glycolysis or ATP dynamics experimentally measured in cell cultures. These findings are then transposed in a tissue scale model – the spheroid – that allows to address the metabolic interactions between cells. To that end, a hybrid multiscale model based on the PhysiCell framework has been developed. It integrates individual cell metabolic states – previously defined - and incorporate cell-cell interactions in relation to the evolving environmental conditions for the spheroid exposed to IR. In light of the identified ROS-HIF relationships, our model aims to gain insight into the metabolic dynamics within an irradiated spheroid - dynamics which could be observed in future experiments.

MS138-03 — Geometric Adversarial Learning for Reconstruction of Right-Censored Tumor Dynamics

SPEAKERS Nahum Puebla-Osorio (University of Texas MD Anderson Cancer Center)

AUTHORS Nahum Puebla-Osorio (University of Texas MD Anderson Cancer Center); Ari Barnett (Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center, Tampa, FL, USA); Pirmin Schlicke (Department of Radiation Oncology, Institute for Data Science in Oncology (IDSO), UT MD Anderson Cancer Center, Houston, TX, USA); Mohammad U. Zahid (Department of Radiation Oncology, Institute for Data Science in Oncology (IDSO), UT MD Anderson Cancer Center, Houston, TX, USA); Sarah Brüningk (Department of Radiation Oncology, Inselspital, Bern University Hospital and University of Bern, Switzerland); Heiko Enderling (Department of Radiation Oncology, Institute for Data Science in Oncology (IDSO), UT MD Anderson Cancer Center, Houston, TX, USA)

SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 15.04 - HS

TRACK Mathematical Oncology

Abstract

To predict radiotherapy patient outcomes under clinically realistic right-censoring, we model longitudinal gross tumor volume (GTV) dynamics using a geometric adversarial learning framework rather than predefined mathematical growth equations. In this setting, patient trajectories are observed only up to a given follow-up time, after which tumor evolution continues but remains unobserved. Instead of assuming a specific functional form (e.g., exponential, logistic, or Gompertz growth), our approach (GAN- GAF framework) first transforms observed GTV trajectories into Gramian Angular Fields (GAFs), two-dimensional representations that encode pairwise temporal correlations across the entire observation window, and then learns to complete these correlation manifolds using adversarial training (GAN). Thus, right-censoring is implemented by withholding the terminal portion of each patient's trajectory, with the generator inferring plausible continuations by restoring missing correlation structure in GAF space. This enables uncertainty-aware reconstruction of post-observation tumor dynamics that preserves amplitude, trend, and long-range temporal dependencies reflective of individual treatment response heterogeneity, without requiring mechanistic assumptions or parameter fitting. The GAN-GAF framework operates directly on real-time clinical data, enforces global geometric consistency, and provides a data-driven alternative to classical mathematical modeling for inference under right-censored radiotherapy follow-up.

MS138-04 — A new metric for functional drug screening: combing radiation with systemic therapies

SPEAKERS Sarah C. Brüningk (University of Bern)

AUTHORS Sarah C. Brüningk (University of Bern); Ana Castro Marquez (1. Center for AI in Radiation Oncology, Department of Radiation Oncology, Inselspital and University of Bern, Bern, Switzerland ; 2. Department of Digital Medicine, University of Bern, Bern, Switzerland); Bettina Kritzer (Children's Research Center, University Children's Hospital Zurich, University of Zurich, Zurich, Switzerland); Sandra Laternser (Children's Research Center, University Children's Hospital Zurich, University of Zurich, Zurich, Switzerland); Micaela Freitas (PreComb Therapeutics AG, Hombrechtikon, Switzerland); Jens M. Kelm (PreComb Therapeutics AG, Hombrechtikon, Switzerland); Javad Nazarian (Children's Research Center, University Children's Hospital Zurich, University of Zurich, Zurich, Switzerland)

SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

We will present a modelling study to provide a longitudinal response quantification of efficacy for single and combination therapies of 11 compounds and radiation (at different doses). This is based on the design of a flexible ODE model accounting for different mechanisms of cell death/modes of action induced by the different treatments, paired with a modelling of synergies and scheduling. We will also motivate possible alternative combination strategies regarding order/schedule based on these models. The results are presented across two cell lines, with an emphasis on the modeling approach, and motivation of a new response metric that disentangles intrinsic efficacy from dynamic response observation.

MS144 — A Co-production Approach to Epidemic Modelling of Infectious Diseases

TRACK Mathematical Epidemiology
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 15.11 - HS
ORGANISERS Elizabeth Fearon (University College London); Thomas House (The University Manchester); Zviiteyi Chazuka (University College London)

Minisymposium abstract

The last half-century has experienced ecological change and greater global connectivity, leading to an increase in emerging and pandemic infections alongside existing endemic pathogens. Epidemic impacts on health and well-being have been large and unequal. There has been a growing demand for modelling of infectious diseases to support epidemic decision-making, enabling the exploration of interactions between biological, social and behavioural processes that underpin transmission patterns. Decisions made in modelling epidemics often contain implicit and unrecognised assumptions affecting model realism and framing of trade-offs, and impacting the utility and representativeness of the advice given to policy-makers. However, unlike other public health fields that benefit from established patient and public involvement, the epidemic modelling field is still developing clear guidance and tools for meaningfully involving affected communities. Co-producing infectious disease models with the community, patients, clinicians, researchers and policymakers is one approach to involving the public in model development to enhance quality and influence on public health policy. This minisymposium will feature talks from the COMMET and MEMVIE projects focused on integrating community voices into the modelling process. We will share insights from mathematical modellers, social scientists and the co-producers themselves on how this can enhance public health interventions during disease outbreaks.

References

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MS144-02 — Involving communities in epidemic modelling: an introduction to co-production principles and tensions

SPEAKERS Lucy Rycroft-Smith (University of Cambridge); Sarah Barnes (University College London)
AUTHORS Beth Malory (University College London); Caroline Jay (University of Manchester); Curtis Ochan Ashok; Ela Portnoy; Elio Yague Raguz; Elizabeth Fearon (University College London); Emily Jay Nicholls (University College London); Jasmine Folz (University of Manchester); Lucy Rycroft-Smith (University of Cambridge); Niccola Hutchinson-Pascal (University College London); Sarah Barnes (University College London); Shema Tariq (University College London); Thomas House (University of Manchester); Zviiteyi Chazuka (University College London)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

Co-production, defined as collective knowledge making across different groups of stakeholders, has been suggested as the most effective strategy for mobilising evidence in policy and practice contexts [BAL23]. While co-production is becoming an increasingly popular term in research, it is not always evident what counts as co-production: what is being produced, under what circumstances, and with what implications for participants [FRM17].

With co-production becoming more of a priority, its place in non-clinical research can be difficult to navigate. We will explore the arguments for co-production within modelling, taking into account the national and international context and co-production's place in the wider knowledge exchange agenda.

We will use the COMMET (co-produced Mathematical Modelling of Epidemics Together) project as a case study to examine processes of co-production as way of making better decisions in epidemic modelling, since co-production is increasingly recognised as a way of ensuring that models reflect the realities and values of those most affected and avoiding harm. We will share examples of how co-production has been put into practice in the case study of the COMMET project and present our experiences as a co-producer and facilitator of co-production respectively.

References

[BAL23] Justyna Bandola-Gill, Megan Arthur, and Rhodri Ivor Leng. "What is co-production? Conceptualising and understanding co-production of knowledge and policy across different theoretical perspectives". In: *Evidence & Policy* 19.2 (2023), pp. 275–298.

[FRM17] Angela Filipe, Alicia Renedo, and Cicely Marston. "The co-production of what? Knowledge, values, and social relations in health care". In: *PLoS biology* 15.5 (2017), e2001403.

MS144-03 — Neat Models, Messy Lives: Social Science as a Bridge to Lived Experience

SPEAKERS Shema Tariq (University College London)
AUTHORS Beth Malory (University College London); Caroline Jay (University of Manchester); Curtis Ochan Ashok; Ela Portnoy; Elio Yague Raguz; Elizabeth Fearon (University College London); Emily Jay Nicholls (University College London); Jasmine Folz (University of Manchester); Lucy Rycroft (University of Cambridge); Niccola Hutchinson-Pascal (University College London); Sarah Barnes (University College London); Shema Tariq (University College London); Thomas House (University of Manchester); Zviiteyi Chazuka (University College London)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

Mismatches between modelled and real-world outcomes frequently arise from misplaced assumptions about how people live, interact, and respond to infectious disease threats and interventions. Co-production is increasingly recognised as a way of ensuring that models reflect the realities of those most affected. Without it, models risk overlooking context, perpetuating inequities, and causing inadvertent harm, particularly among marginalised groups.

Meaningful co-production requires more than collaboration; it depends on methods, frameworks and values that enable the systematic integration of diverse forms of knowledge into the modelling process. Social science disciplines and methods that generate evidence on behaviour, social structure and wider cultural and political context provides a foundation for this work.

In my talk, I will introduce key social science approaches and frameworks including qualitative and participatory methods, and assumptions about knowledge showing how they can facilitate co-production. I will argue that social science helps create the conditions for inclusive co-production, supports its integration across all stages of modelling, and enables its evaluation. Drawing on examples from literature and from our own team's work, I will demonstrate how social science strengthens both process and input, helping to ground models in the messy realities of the lives they seek to represent.

MS144-04 — How Close is Too Close? Modelling Sexual and Non-sexual Transmission Pathways of Mpox in the UK

SPEAKERS Zviiteyi Chazuka (University College London)
AUTHORS Beth Malory (University College London); Caroline Jay (University of Manchester); Curtis Ochan Ashok; Ela Portnoy; Elio Yague Raguz; Elizabeth Fearon (University College London); Emily Jay Nicholls (University College London); Jasmine Folz (University of Manchester); Lucy Rycroft-Smith (University of Cambridge); Niccola Hutchinson-Pascal (University College London); Sarah Barnes (University College London); Shema Tariq (University College London); Thomas House (University of Manchester); Zviiteyi Chazuka (University College London)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

The 2022 mpox outbreak disproportionately affected gay, bisexual, and other men who have sex with men (GBMSM) in the United Kingdom, prompting the need for models that reflect both the epidemiological realities of transmission and the lived experiences of affected communities. We initially develop a dynamic network model capturing sexual and non-sexual close contact transmission dynamics. Central to this work is a co-production process with team members bringing lived experience and alongside broader community engagement workshops to bring modellers and GBMSM community members together to jointly refine assumptions, clarify behavioural patterns, and shape research priorities. These collaborative sessions are aimed at improved transparency, strengthened trust, and ensuring that the final model realistically represents diverse sexual partnerships and patterns and types of sexual behaviour, and community informed understandings of non-sexual exposure risks. Through this co-production approach we aim to demonstrate how lived experiences can meaningfully shape the model structure, improve the relevance of transmission scenarios, and support more effective and equitable public health responses. In this talk I explain our modelling process and illustrate how co-production has influenced its development so far.

MS144-01 — The MEMVIE public involvement framework: Developing a framework for public involvement in epidemiological and health economic modelling to bring dynamism to vaccination policy recommendations

SPEAKERS Edward Hill (University of Liverpool)
AUTHORS Edward Hill (University of Liverpool); Sophie Staniszewska (University of Warwick); Tinevimbo Shiri (Johnson & Johnson Innovative Medicine); Samik Datta (Irish Marine Institute); Stavros Petrou (University of Oxford); Matt Keeling (University of Warwick); Richard Grant; Jarina Porter; Sue Tulip; Jane Whitehurst; Claire Wright
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

The Mathematical and Economic Modelling for Vaccination and Immunisation Evaluation project (MEMVIE) involves the construction of epidemiological models and evaluating the cost-effectiveness of different vaccination interventions to help inform vaccination policy in the UK.

A contributor to the MEMVIE project is our MEMVIE public involvement group. The MEMVIE public involvement group and academic contributors are exploring, capturing and supporting the potential contribution of the public to epidemiological and health economic modelling.

This talk overviews the MEMVIE public involvement framework that has been iteratively co-produced through this process. The MEMVIE public involvement framework identifies points of collaborative public contribution to modelling and supports its implementation [Sta+21].

References

[Sta+21] Sophie Staniszewska et al. "Developing a framework for public involvement in mathematical and economic modelling: bringing new dynamism to vaccination policy recommendations". In: *The Patient-Patient-Centered Outcomes Research* 14.4 (2021), pp. 435–445.

MS150 — Making cells dance: modelling gene regulation and cell fate from transcriptomics

TRACK Systems Biology and Biochemical Networks
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 11.03 - HS
ORGANISERS Meichen Fang (Imperial College London); Philipp Thomas (Imperial College London); Zekai Li (Imperial College London)

Minisymposium abstract

Understanding how gene regulation gives rise to diverse cell states and cell fates during differentiation is a central question in biology. Single-cell RNA sequencing (scRNA-seq), as a rapidly developing and powerful methodology, provides both new insights into this problem and new challenges for mathematical modeling. While scRNA-seq enables measurement of genome-wide gene expression at single-cell resolution, it provides only snapshot data. This makes inference of dynamical processes and regulatory mechanisms difficult. How to fully exploit the rich gene expression information in the data while overcoming these inherent limitations remains a challenging open problem.

This minisymposium presents recent work that tackles these challenges from complementary perspectives. We will cover stochastic methods that utilize time-resolved single-cell transcriptomics [VST24] and perturbation [CGP24] to shed light on gene regulation, as well as approaches for inferring cell differentiation dynamics based on drift–diffusion Markov processes and deep learning models that encode gene regulatory relationships [Wan+24]. These cutting edge methodologies provide distinct magnifying lenses on cellular dynamics. Their synergy through a shared emphasis on mathematical modeling and statistical inference will foster a timely discussion of both the limitations and opportunities in scRNA-seq–based modeling of gene regulation and cell fate.

References

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[VST24] Dimitris Volteras, Vahid Shahrezaei, and Philipp Thomas. "Global transcription regulation revealed from dynamical correlations in time-resolved single-cell RNA sequencing". In: *Cell Syst.* 15 (2024), 694–708.e12. DOI: 10.1016/j.cels.2024.07.002.

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MS150-01 — Inferring RNA processing dynamics across cell states and fates from single-cell snapshot data

SPEAKERS Tara Chari (Eric and Wendy Schmidt Center at the Broad Institute of MIT and Harvard)
AUTHORS Cameron Smith (Department of Pathology, Harvard Medical School, and Molecular Pathology Unit and Krantz Family Center for Cancer Research, Massachusetts General Hospital); Gennady Gorin (Fauna Bio, Emeryville, California, United States of America); Lior Pachter (Division of Biology and Biological Engineering, California Institute of Technology, Pasadena, California, United States of America); Tara Chari (Eric and Wendy Schmidt Center at the Broad Institute of MIT and Harvard)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

With single-cell genomics datasets, it is possible to assay the transcriptome in thousands to millions of cells as they undergo development, differentiation and external perturbation. These data are ripe for investigation into how regulation of RNA processing governs the myriad cell states comprising these systems.

However, despite our ability to assay these states, single-cell data is sparse and noisy, a result of both innate biological features of the RNA molecules and technical effects in RNA capture and sequencing. Standard analyses often rely on heuristic application of dimensionality reductions to remove noise, and focus on mean-level (observational) changes in gene expression to define cell states.

Here we demonstrate how stochastic, biophysical models of RNA processing can be applied to snapshot single-cell data to reveal underlying mechanisms of observed gene expression patterns across heterogenous cell states. By explicitly realizing the biophysical and technical processes underlying the sparse, discrete molecular data, we can reveal altered dynamics of bursty transcription, splicing and degradation between populations of cells. We can additionally resolve heterogeneous regulation within these populations, and propose combinatorial strategies of regulation guiding these distinct fates. Together this work enables hypothesis-driven experimentation by proposing key points of transcriptional regulation underlying higher-level changes in gene expression and noise.

MS150-02 — Inferring Cellular Dynamics through Ordered Diffusion Kernels

SPEAKERS Jack Soulsby (Imperial College London)
AUTHORS Jack Soulsby (Imperial College London)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Single-cell RNA-sequencing (scRNA-seq) data provides a detailed view into the gene regulatory landscape that cells traverse during differentiation from pluripotent to mature cell types. However, analysing these datasets remains challenging due to their sparsity, high dimensionality, lack of temporal information, and high levels of technical noise, necessitating the development of numerous specialised analytical methods.

We introduce and apply Ordered Diffusion Kernels (ODKs) to model cellular differentiation as a drift-diffusion Markov process by biasing transition probabilities with an ordering function from which we can compute velocity estimates, terminal states, stationary distributions, passage times and trajectory inference. Furthermore, we propose transforming snapshot scRNA-seq data into a pseudo-trajectory dataset with ODKs for purpose of applying powerful tools from the time series analysis literature to perform equation discovery and infer gene regulatory networks.

MS150-03 — Inferring causal gene regulatory relationships from time-resolved single-cell transcriptomics

SPEAKERS Dimitris Volteras (The Francis Crick Institute)
AUTHORS Dimitris Volteras (The Francis Crick Institute)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Deciphering gene regulatory networks is central to understanding how cells orchestrate gene expression programmes. Single-cell RNA sequencing (scRNA-seq) has enabled the investigation of genome-wide regulatory relationships through pairwise gene correlations. Yet, interpreting these correlations is challenging due to confounding factors such as biological sources of covariation or technical noise. Time-resolved scRNA-seq with metabolic RNA labelling provides access to transcription dynamics, offering opportunities to address these challenges. We introduce a modelling framework integrating mechanistic models of gene regulation with machine learning to analyse regulatory relationships in time-resolved scRNA-seq data. We build stochastic models of causal gene regulatory relationships, including direct regulation and co-regulation, capturing key confounders such as extrinsic noise, cell cycle coupling and technical noise, to simulate temporal summary statistics. A neural network supervised by these simulations learns to classify regulatory scenarios, mapping observed correlation patterns to underlying causal models, while leveraging gene-specific priors inferred from the data. Applied to real time-resolved scRNA-seq data, the framework predicts causal gene–gene relationships while quantifying uncertainty. Overall, our approach shows that mechanistically informed machine learning enables interpretable gene regulation inference from single-cell data.

MS150-04 — Decoding gene regulatory networks and cellular dynamics

SPEAKERS Weixu Wang (ICB, Helmholtz Munich)
AUTHORS Weixu Wang (ICB, Helmholtz Munich)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

RNA velocity has emerged as a popular approach for modeling cellular change along the phenotypic landscape but routinely omits regulatory interactions between genes. Conversely, methods that infer gene regulatory networks (GRNs) do not consider the dynamically changing nature of biological systems. To integrate these two currently disconnected fields, we present RegVelo, an end-to-end dynamic, interpretable, and actionable deep learning model. RegVelo learns a joint model of splicing kinetics and gene regulatory relationships and allows us to perform in silico perturbation predictions. When applied to datasets of the cell cycle, human hematopoiesis, and murine pancreatic endocrinogenesis, RegVelo provides reliable predictive power for terminal states, gene interaction and perturbation simulations. To leverage RegVelo's full potential, we studied the dynamics of zebrafish neural crest development and underlying regulatory mechanisms using deep full-transcript-length Smart-seq3 dataset and shared gene expression and chromatin accessibility measurements. Using RegVelo's in silico perturbation predictions, supported by CRISPR/Cas9-mediated knockout and single-cell Perturb-seq, we establish transcription factor tfec as an early driver and elf1 as a novel regulator of pigment cell fate. Together, RegVelo provides a powerful framework for quantitatively bridging gene regulation and cell fate decisions.

MS158 — Methods to integrate -omics data into mechanistic models

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 01.15 - HS
ORGANISERS Claire Miller (Auckland Bioengineering Institute, the University of Auckland); Hilary Hunt (Queensland University of Technology)

Minisymposium abstract

The prevalence of -omics data is steadily increasing within the biological and ecological sciences, and it is widely accepted within the community that data is published open access alongside journal articles. Consequently, this provides mechanistic modellers with a wealth of accessible data with which to parameterise and validate models. However, understanding the complexities of this data and how to process and make use of it with mechanistic models is not straightforward. In this minisymposia, we will hear from

researchers across a range of biological mathematics application areas who are making use of this data within their research activities.

The goal of this minisymposia is twofold. Firstly, to provide an opportunity for those who already use, or are starting to use, -omics data with mechanistic models to make connections with other established researchers in the area. Secondly, to provide anyone who has an interest in the use of this data an understanding of the considerations required for its use. We ask each speaker to cover both their approach to processing the data and how they then use it for their application in order to best facilitate these two goals.

MS158-01 — Informing Macrophage phenotypic change over the endometrial cycle using omics data

SPEAKERS Domenic Germano (University of Melbourne)
AUTHORS Domenic Germano (University of Melbourne); Claire Miller (Auckland Bioengineering Institute, the University of Auckland)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 01.15 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The endometrial cycle is known to be mediated via the endocrine system, with both estradiol and progesterone hormones heavily implicated in menstruation. Throughout this cycle, the tissue undergoes vast changes in tissue and cellular-level function, including phagocytosis and apoptosis, cell proliferation, both tissue and vascular remodelling, and extracellular matrix construction. Macrophages are a key component of the regulation of these vastly different function, and have traditionally thought of as either M1-like (pro-inflammatory) phenotype, or M2-like (anti- inflammatory) phenotype. However, a paradigm shift is underway, where macrophages are increasingly observed to exhibit characteristics of each phenotype simultaneously. This is referred to as the "continuum spectrum of Macrophage phenotypes".

In this work, we present a mathematical model of a continuous macrophage phenotype. We utilise openly available, single cell transcriptomics data, and the fact that the endocrine system is a driver of endometrial cycle dynamics, to validate our model. This validated models allows us to understand how the macrophages change their phenotype along this continuous spectrum throughout the hormonal cycle. We then use our validated model for hypothesis generation, in the context of immune dysfunction and its potential consequences, for example for the uterine disease endometriosis.

MS158-02 — Transcriptome-informed flux predictions revealed temporal metabolic reprogramming and branch points mediating cold stress-growth trade-offs in rice

SPEAKERS Fatemeh Soltani (Institute for Plant Sciences, Cluster of Excellence on Plant Sciences (CEPLAS), University of Cologne)
AUTHORS Fatemeh Soltani (Institute for Plant Sciences, Cluster of Excellence on Plant Sciences (CEPLAS), University of Cologne); Jan-Niklas Weder (Institute for Plant Sciences, Cluster of Excellence on Plant Sciences (CEPLAS), University of Cologne); Tiago Moreira Machado (Institute for Plant Sciences, Cluster of Excellence on Plant Sciences (CEPLAS), University of Cologne); Nadine Töpfer (Institute for Plant Sciences, Cluster of Excellence on Plant Sciences (CEPLAS), University of Cologne)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 01.15 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Rice (Oryza sativa) is highly sensitive to cold stress, imposing major constraints on its productivity and geographical distribution. Elucidating cold-induced metabolic reprogramming is essential for understanding the underlying mechanisms of rice adaptive responses.

Constraint-based metabolic modeling is a powerful framework for capturing metabolic interactions. A key challenge, however, is selecting an appropriate objective function, a decision that is critical for model predictions. In this study, we integrated transcriptomics data with a genome-scale rice metabolic model using the E-Flux algorithm to construct a temporal model of cold stress response. We analyzed the resulting model via two approaches: (i) random sampling of the transcriptome-informed solution space, and

(ii) Pareto frontier analysis quantifying trade-offs between biomass production and proline accumulation, a well-studied stress marker.

Pareto analysis revealed carbon flux redistribution to support increased substrate availability for proline biosynthesis. We identified branch points where proline accumulation competes with growth, which can serve as potential engineering targets for improving cold resilience without yield penalties. Machine learning-assisted pathway enrichment analysis of the sampling data highlighted temporal metabolic transitions across time points. These findings offer a pathway-level view of cold-induced metabolic reprogramming in rice and targets for crop improvement.

References

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MS158-03 — From genome sequencing to metabolic modelling and dynamic flux balance analysis extensions

SPEAKERS Thomas Munn (University of Birmingham)
AUTHORS Thomas Munn (University of Birmingham); David Tourigny (University of Birmingham)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 01.15 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Genomic sequencing data can be used to identify gene-protein-reaction (GPR) rules for microbial species, which determine how the genes corresponding to enzymes catalyse specific metabolic reactions. GPRs can thus be combined to create genome-scale metabolic models (GEMs) for any organism for which a genome sequence is available. Mathematically, GEMs are large stoichiometric matrices that can be used to predict which reactions are vital for their metabolism. GEMs are then used to construct kinetic models, which are often very large systems of ODEs. In this talk, I will describe how GEMs are constructed and how flux balance analysis (FBA) is used to calculate a biologically feasible combination of fluxes for an organism at steady state. Next, I will extend FBA to consider dynamic environments (dFBA) to describe scenarios such as growth in a bioreactor where resources are finite. This requires a quasi-steady-state approximation where the intracellular metabolism is assumed to be at steady state. dFBA has been applied with much success to model microbial metabolism, both in industry and research. In the remainder of this talk, I will present my own work on extending dFBA to consider variable objective functions, including how an organism's cellular objective might change in response to a dynamic environment. Notably, this will include an algorithm for solving these extended problems efficiently, minimising the number of times the LP problem must be solved.

MS158-04 — Inferring mass isotopomer distribution dynamics from partial isotopic labeling data: Implications for flux estimation

SPEAKERS Anika Küken (Bioinformatics, Institute of Biochemistry and Biology, University of Potsdam)
AUTHORS Illia Terpylo (Bioinformatics, Institute of Biochemistry and Biology, University of Potsdam); Anika Küken (Bioinformatics, Institute of Biochemistry and Biology, University of Potsdam)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 01.15 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Isotopically non-stationary metabolic flux analysis (INST-MFA) enables the estimation of intracellular fluxes from time-resolved labeling data but remains limited to medium-scale networks due to experimental constraints on metabolite coverage. Extending INST-MFA toward large-scale models with high confidence in flux estimates requires extensive labeling datasets. Here, we employ a neural network approach to infer complete mass isotopomer distribution (MID) dynamics from partial ^{13}C-isotopic labeling data. The model is trained on synthetic time-resolved MID datasets generated from a large-scale network of Chlamydomonas central metabolism. We show that MID trajectories can be accurately reconstructed for a broad set of metabolites from limited observable inputs. Furthermore, we apply the approach to labeling data from different green algae and investigate to what extent predicted complete MID datasets can support downstream flux estimation by comparison to previously published flux estimates derived using scale-free constrained regression and a state-of-the-art tool for INST-MFA, INCA [KTN23]. Together, our findings highlight the potential of neural network based MID prediction as a tool toward precise large-scale flux estimation from isotopic labeling data. By augmenting incomplete labeling datasets with predicted MID dynamics, this approach provides a pathway to overcome current limitations in metabolite coverage and improve the reliability of flux estimates in INST-MFA.

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MS171 — Methodological Advances in Modeling Human Behavior and Infectious Disease Spread

TRACK Mathematical Epidemiology
SCHEDULE Tuesday, 14 July 2026; 10:40–12:00; 15.02 - HS
ORGANISERS Binod Pant (Northeastern University, USA); Kyle Dahlin (Virginia Tech)

Minisymposium abstract

The COVID-19 pandemic highlighted the critical need for improved formulations that incorporate human behavior into epidemiological models. Individual actions, manifested through behaviors such as mask-wearing or changes in mobility patterns, are influenced by public risk perception and, in turn, alter infectious disease spread locally and across the population. Furthermore, individual actions impact the evaluation of interventions, which are typically decided at a governmental level, without necessarily considering the diverse social, psychological, and contextual factors that shape human behavior. Given the high variability of human behavior, novel and innovative implementations in epidemic models are vital. This minisymposium showcases researchers working on cutting-edge formulations that incorporate human behavior into epidemiological models and how those choices affect projected outcomes and understanding.

MS171-01 — Information delays, network structure and adaptive human behavior jointly shape epidemic dynamics

SPEAKERS Claus Kadelka (Iowa State University)
AUTHORS Claus Kadelka (Iowa State University); Md Shahriar Mahmud (Iowa State University)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Human behavior strongly shapes epidemic dynamics, yet responses to risk are typically based on spatially aggregated and temporally delayed information. While behavioral adaptation, spatial structure, and delays have been studied separately, their combined effects on networks remain poorly understood. We investigate these effects using stochastic epidemic simulations on small-world contact networks with behavior driven by delayed, spatially aggregated risk perception. We find a robust and non-monotonic dependence of epidemic size and outbreak probability on the spatial scale of information, with intermediate scales minimizing burden. This reflects a fundamental trade-off: highly local information is precise but loses predictive value under delays, whereas highly aggregated information obscures heterogeneity and delays behavioral activation. As delays increase, behavioral responses become increasingly misaligned with epidemic growth, and differences across spatial scales diminish, yielding dynamics similar to homogeneous mixing. Increased network connectivity further reduces spatial heterogeneity, weakening the benefits of localized information. A complementary multi-patch model exhibits qualitatively similar patterns, supporting the generality of these findings. Overall, our results show that the effectiveness of behavior-driven epidemic control depends critically on the alignment of information scale and timing with underlying transmission dynamics.

MS171-02 — Coupled Dynamics: How Genes Shape Epidemics (and Epidemics Shape Genes)

SPEAKERS Joan Ponce (Arizona State University)
AUTHORS Joan Ponce (Arizona State University); Elizabeth Ghartey (University of Arizona, Tucson, AZ, United States); Gautam Rai (Arizona State University, Tempe, AZ, United States); Dasha Selivonenko (Rice University, Houston, TX, United States); Rachel Wissenbach (Arizona State University, Tempe, AZ, United States)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Host genetic structure can significantly alter disease transmission dynamics and long-term disease outcomes. Past work by Beck, Keener, Hoppensteadt, Feng, and others has shown that when pathogen transmission interacts with evolving host traits—such as susceptibility, recovery, or disease-induced mortality—the resulting coupled system can exhibit novel dynamics. These models demonstrated that genetic composition within a host population can shift during an epidemic, and conversely, infection pressures can reshuffle genetic frequencies, producing true feedback between genes and epidemics. In this talk, I will discuss a specific example of this phenomenon, focusing on the interaction between Plasmodium vivax and the Duffy antigen, a host genetic trait that confers partial protection against infection.

MS171-03 — Models that ignore behavior underestimate the basic reproduction number

SPEAKERS Marko Lalovic (Network Science Institute, Northeastern University London, London, UK)
AUTHORS Binod Pant (Machine Intelligence Group for the Betterment of Health and the Environment, Northeastern University, Boston, MA, USA; Network Science Institute, Northeastern University, Boston, MA, USA); István Kiss (Network Science Institute, Northeastern University London, London, UK; Department of Mathematics, Northeastern University, Boston, MA, USA); Marko Lalovic (Network Science Institute, Northeastern University London, London, UK); Mauricio Santillana (Machine Intelligence Group for the Betterment of Health and the Environment, Northeastern University, Boston, MA, USA; Network Science Institute, Northeastern University, Boston, MA, USA; Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, MA, USA)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Behavioral responses during epidemics alter transmission, yet most epidemiological models assume constant contact rates. We assessed the resulting bias by fitting a baseline SEIR model with fixed transmission and three behavioral variants, in which transmission declines with increasing mortality, to COVID-19 mortality data from 30 U.S. states during the first wave (March-July 2020).

Behavioral models fit the data better in 28 of 30 states, with Bayes factors giving substantial to decisive support in those same states. Ignoring behavioral feedback produced consistent inferential errors: baseline models systematically underestimated the basic reproduction number (R_0) while simultaneously overestimating the final epidemic size. Posterior R_0 estimates were higher under behavioral models across all states, yet baseline models predicted substantially larger cumulative infection burdens.

Synthetic-data experiments showed that these discrepancies are caused by model misspecification rather than noise or data limitations. Analytically, we show that for fixed R_0, models without behavioral feedback overestimate epidemic size whenever mortality reduces transmission. Explicit behavioral modeling is therefore essential for reliable epidemic inference and forecasting.

References

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MS171-04 — Highlights of projects funded through the Mathematical Biology program and other initiatives at the US NSF

SPEAKERS Zhilan Feng (National Science Foundation)
AUTHORS Zhilan Feng (National Science Foundation)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

The Mathematical Biology program and other programs in the Directorate of Mathematical and Physical Sciences at the US National Science Foundation have funded many high impact research projects on various topics including mathematical modeling of infectious diseases. Some highlights will be presented.

MS174 — Multicellular Modelling and Simulation Tools - The OpenVT Project

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Tuesday, 14 July 2026; 10:40–16:20; 02.01 - HS
ORGANISERS James Glazier (Indiana University); James Osborne (University of Melbourne); Lutz Brusch (TU Dresden)

Minisymposium abstract

Multicellular simulations have become indispensable in understanding complex biological phenomena, from tissue development to disease progression. However, the diversity in simulation methods, ranging from agent-based models to lattice-free models and stochastic particle simulations, poses challenges in terms of reproducibility, modularity, reusability, and integration within multi-scale simulations. The Open VT (Virtual Tissues) project is an engaged international community of researchers - modelers and software engineers who use and develop open source multicellular modeling frameworks. These frameworks allow virtual experiments (simulations) of cell behaviors within tissues and organs.

This minisymposia is part of a pair which aim to present the variety of multicellular simulations being used by the community, along with the efforts to make these simulations replicable and reproducible.

In this part we present the myriad of modelling frameworks and simulation tools available. We present seven simulation frameworks who are part of the Open VT project. We also present the winner of the 2026 OpenVT Reproducible Agent-Based Multicellular Model Competition.

MS174-08 — A Data-Driven Model of Polarity Reversal in Migrating Cells

SPEAKERS Jupiter Algorta (University of British Columbia)
AUTHORS Jupiter Algorta (University of British Columbia)
SCHEDULE Tuesday, 14 July 2026; 10:40–11:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

This talk is for the winner of the 2026 OpenVT Reproducible Agent-Based Multicellular Model Competition.

We study how motile cells can reverse their polarity when exposed to changing stimuli, using mathematical modeling alongside extensive experimental data from optogenetic assays carried out by our collaborators in the Orion Weiner lab (UCSF). These experiments revealed an unexpected phenomenon: when a localized input is followed by a global stimulus, cells often reverse their direction of turning. To explain this, our collaborators hypothesized the existence of a slow-acting, locally produced inhibitor downstream of Rac, a signaling molecule known to promote actin assembly and front-edge protrusion. We test this idea by adapting an existing reaction diffusion model that, under certain conditions, produces a stable spatial pattern: a polarized distribution with a clear front and back. This modeling framework, often referred to as wave-pinning, has not previously been fitted directly to experimental data. Here, we calibrate the model's reaction terms to time-dependent Rac activity data, introducing a novel approach that embraces cellular heterogeneity by fitting a distribution of parameters across multiple cells. While the Rac-inhibitor circuit captures several key features of the response, it fails to reproduce reversal. Incorporating PIP3, an upstream regulator of Rac, allows the model to recover reversal dynamics and reproduce the full range of observed behaviours. In this presentation, we will show the development of our modeling framework from its earliest steps, including how data fitting informed model refinement. Our results validate the experimental hypothesis and yield new predictions about the molecular timing and feedback logic underlying flexible polarity control.

MS174-04 — Chaste: a computational framework for multicellular modelling

SPEAKERS James Osborne (University of Melbourne)
AUTHORS James Osborne (University of Melbourne)
SCHEDULE Tuesday, 14 July 2026; 11:00–11:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Problems in biology are intrinsically multi-scale, with processes occurring on many disparate spatial and temporal scales. Here we present a multiscale framework for the mathematical modelling of biological systems. Utilising the natural structural unit of the cell, the framework consists of three main scales: the tissue level (macro-scale); the cell level (meso-scale); and the sub-cellular level (micro-scale), with interactions occurring between all scales. The cell level is central to the framework and cells are modelled as discrete interacting entities using one of a number of possible modelling paradigms, including lattice based models (cellular automata and cellular Potts) and off-lattice models (cell centre and vertex based representations). The sub-cellular level concerns numerous metabolic and biochemical processes represented by interaction networks rendered stochastically or into ODEs. The outputs from such systems influence the behaviour of the cell level affecting properties such as adhesion and also influencing cell mitosis and apoptosis. Tissue level behaviour is represented by field equations for nutrient or messenger concentration, with cells functioning as sinks and sources. This modular approach enables more realistic behaviour to be considered at each scale.

The multi-scale framework is implemented in an open source software library known as Chaste (https://chaste.github.io/). This software library consists of object orientated C++, developed using an agile development approach. All software is tested, robust, reliable and extensible. The library enables general simulations to be undertaken and includes tools to atomically curate and store simulation results expediting model development. In this talk we introduce the framework and give some key examples of its use.

MS174-01 — Vivarium: a compositional framework for multiscale modeling

SPEAKERS Eran Agmon (University of Connecticut)
AUTHORS Eran Agmon (University of Connecticut)
SCHEDULE Tuesday, 14 July 2026; 11:20–11:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Multicellular biological systems span many spatial and temporal scales, yet models of these systems are often developed in isolation, using different assumptions, algorithms, and software frameworks. Vivarium is an open-source compositional framework designed to make these models interoperable by allowing independently developed components to be connected through standardized interfaces and shared state variables. Rather than building monolithic simulations, Vivarium enables researchers to assemble complex models by composing modular processes representing different biological functions and modeling formalisms, including agent-based models, differential equations, flux balance analysis, and spatial simulators. In this talk, I will introduce the core ideas behind Vivarium and show how this compositional approach supports multiscale simulations of microbial and multicellular systems while enabling integration with existing modeling tools. By focusing on modularity, explicit interfaces, and reusable components, Vivarium aims to lower the barrier to composing large biological simulations and to support a more collaborative ecosystem for building and connecting models across the community.

MS174-02 — Bringing biology to the browser with Artistoo

SPEAKERS Inge Wortel (Radboud University)
AUTHORS Inge Wortel (Radboud University)
SCHEDULE Tuesday, 14 July 2026; 11:40–12:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

The cellular Potts model (CPM) is a powerful in silico method for simulating biological processes at tissue scale. Their inherently graphical nature makes CPMs very accessible in theory, but in practice, they are mostly implemented in specialised frameworks users need to master before they can run simulations. Artistoo (Artificial Tissue Toolbox) is a JavaScript library for building 'explorable' CPM simulations where viewers can change parameters interactively, exploring their effects in real time. Simulations run directly in the web browser and do not require third-party software, plugins, or back-end servers. The JavaScript implementation imposes no major performance loss compared to frameworks written in C++ and remains sufficiently fast for interactive, real-time simulations.

In this talk, I will discuss the design philosophy behind Artistoo and show how it provides an opportunity to unlock models for a broader audience: interactive simulations can be shared via a web page in a zero-install setting, but models can also run from the command line for large-scale scientific applications. I will discuss applications in research, science dissemination, open science, and education, and demonstrate how the modular design of Artistoo makes it easy for users to plug in their own model terms when needed.

MS174-03 — Compucell3D: Python-scripted open-source multiscale modeling framework

SPEAKERS James Glazier (Indiana University)
AUTHORS James Glazier (Indiana University); Joel Vanin (Indiana University Bloomington)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

CompuCell3D is an open-source multiscale modeling environment for multicellular systems that combines biological realism, computational flexibility, and ease of learning. Based on the cellular Potts/Glazier-Graner-Hogeweg formalism, it supports explicit dynamic cell shape, adhesion, motility, growth, elastic solid-like junctions, filopodia, extracellular transport, and coupling to intracellular regulatory and signaling models encoded in SBML, Antimony, CellML, and MaBoSS. CompuCell3D has also evolved into a Python library, enabling interoperability with other Python-accessible modeling tools, and includes a Vivarium wrapper that allows it to serve as a component in the Vivarium ecosystem. Its high-level model specification, interactive tools, and multithreaded execution on multicore CPUs lower barriers to entry while supporting sophisticated multiscale applications.

Over the past two decades, CompuCell3D has been used in a broad range of applications, including embryonic development, morphogenesis, cancer, angiogenesis, immune response, viral infection, regeneration, tissue engineering, and toxicology. I will present the framework's core ideas and distinctive capabilities, highlight representative applications, and describe current OpenVT efforts to create reference models supporting reproducibility and output comparability across CPM-based platforms such as CompuCell3D, Morpheus, Artistoo, and CHASTE. This work underscores the central role of open sharing, FAIR and CURE principles, and reproducible model exchange in building a robust community ecosystem for multicellular systems biology.

MS174-05 — Morpheus: User-friendly multiscale modeling

SPEAKERS Jörn Starruß (TU Dresden)
AUTHORS Jörn Starruß (TU Dresden)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Collaborative modeling and simulation become increasingly important to study complex developmental processes across molecular to organism scales. To support interdisciplinary workflows in quantitative multicellular biology we designed the extensible open-source modeling framework Morpheus. It is centered

around the cellular Potts model (cell behavior), allows for tight integration with intra-cellular regulation models (e.g. SBML) and fluently couples cells to neighboring cells and their extracellular environment (1,2,3). A user-friendly GUI allows to formulate the multiscale models in an accessible modular modeling language (MorpheusML), rendering Morpheus applicable for a broad scientific community, beyond skilled programmers. Numerical simulations are automatically composed by extracting the graph of model components from the MorpheusML model definitions and scheduling suitable solvers in the simulator. The associated FitMultiCell toolbox supports parameter estimation for stochastic Morpheus models (4,5). MorpheusML models can easily be exchanged, versioned, extended and shared through a FAIR model repository (6). It already presents more than 100 hundred models in valuable detail, the majority published in peer-reviewed journals. (1) Starruß et al. (2014). Bioinformatics 30, 1331. (2) Morpheus homepage: https://morpheus.gitlab.io (3) Open source code: https://gitlab.com/morpheus.lab/morpheus (4) Alamoudi et al. (2023). Bioinformatics 39, btad674. (5) FitMultiCell toolbox: https://gitlab.com/fitmulticell/fit (6) Model repository: https://morpheus.gitlab.io/models

MS174-06 — PhysiCell: multiscale modeling with a grammar

SPEAKERS Randy Heiland (Indiana University)
AUTHORS Randy Heiland (Indiana University)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

PhysiCell (physicell.org) is an open source physics-based multicellular modeling framework. A cell is defined by a (off-lattice) centroid and volume and several phenotypic parameters that define its behavior (cell cycle, death, mechanics, motility, secretion, etc). It is bundled together with the BioFVM diffusion solver for cell secretion/uptake. PhysiCell has many predefined (but modifiable) model parameter values and several sample models bundled with the (C++/OpenMP and XML) software. A notable, recent addition is a simple English grammar that lets a modeler write rules that define cells' behaviors based on one or more signals. PhysiCell also allows for intracellular models as addons, e.g., libRoadrunner for ODEs in SBML, and PhysiBoSS for boolean networks. PhysiCell Studio is a desktop GUI (written in Python) that makes it easier to build models and visualize simulation results. The Studio is also available as an interactive tool on the Web-based Galaxy platform. In this talk, I plan to show a live demo of immunology dynamics.

MS174-07 — PolyHoop & SimuCell3D: Open-source tools for high-resolution cell dynamics

SPEAKERS Roman Vetter (ETH Zurich)
AUTHORS Roman Vetter (ETH Zurich)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cellular tissue simulations can be computationally demanding, which restricts them to simplified cell geometries and low spatial resolution. Here I present two recently introduced cell-based simulation programs written in C++, PolyHoop and SimuCell3D, which are designed to efficiently model cellular tissues with detailed subcellular resolution. PolyHoop simulates cellular monolayers in 2D using flexible hoops to represent individual cell membranes, while SimuCell3D models cell membranes, nuclei, and extracellular matrices in 3D using triangulated surfaces and an automatic cell polarization algorithm. Both tools are highly efficient, enabling simulations with many thousand deformable cells. They are open source, freely available, and easy to use. In this talk, I will give a short live demo that shows the tools in action in a typical scenario featuring a proliferating tissue.

MS28 — Population level models of bacterial processes and interaction

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 03.01 - HS
ORGANISERS Blessing Emerenini (RIT); Hermann Eberl (University of Guelph)

Minisymposium abstract

Bacterial populations can be well-mixed free swimming planktonic communities, or as microbial aggregates with spatial structure, including biofilms, granules or flocks, single cell layers, and microbial biozones with increased biomass density relative to their bulk environment, etc. In applications (e.g environmental, biotechnological), such populations often consist of a large number of cells, such that a continuum description is appropriate. The complexity of bacterial processes is rendered by the mathematical complexity of models describing them. Usually the models also depend on limiting nutrients, inhibitory stressors, signal molecules, etc. When no spatial structure needs to be accounted for, this often leads to ODE models (the simplest scenario, but possibly with many parameters and state variables). When spatial effects play a role as well, also terms describing bacterial movement and substrate transport need to be included. For example, in biofilm reactor models one often has a system of ODEs for the reactor itself, coupled to a free boundary value problem for a 1st order nonlocal nonlinear hyperbolic balance equation for the biofilm, coupled in turn to a semilinear two point boundary value problem for dissolved substances. Other models contain nonlinear degenerate diffusion terms, or complicated interactions with the physical environment. In this minisymposium the focus will be on novel applications of such modeling frameworks for bacterial populations.

MS28-02 — Toward Sustainable Corrosion Control: Identifying tuning parameters associated with Biofilm-Induced corrosion Inhibition in a mathematical framework

SPEAKERS Blessing Emerenini (RIT)
AUTHORS Blessing Emerenini (RIT); Vedat Kurtay (Rochester Institute of Technlogy)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Microorganisms play a pivotal role in corrosion processes, exerting profound effects on the integrity of metallic surfaces across agricultural machinery, transportation infrastructure, and energy systems, leading to substantial economic losses and environmental risks. Depending on the species and environmental context, microbial activity can either accelerate or inhibit corrosion, making their role complex and multifaceted. Understanding the interactions between biofilms, hydrogels, and metallic surfaces is essential for elucidating the mechanisms driving corrosion and for developing effective mitigation strategies. In this work, we introduce a mathematical biology framework for the study of Microbiologically Influenced Corrosion (MIC), based on a spatiotemporal modeling approach. This framework enables systematic investigation of how environmental factors influence microbial activity and corrosion dynamics, while also providing a platform for evaluating potential strategies to inhibit or control MIC. The approach integrates mechanistic modeling with predictive analysis, offering new insights into corrosion processes and informing targeted interventions.

MS28-01 — A Predictive Model of Subaerial Biofilms: Insights into Daily Dynamics and Environmental Changes

SPEAKERS Alberto Tenore (Univ Naples)
AUTHORS Alberto Tenore (Univ Naples); B Buttaro (Temple University); Fabiana Russo (University of Naples Federico II); Isaac Klapper (Temple University); J. Jacob (US National Park Service); J.D. Grattepanche (Temple University)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Subaerial biofilms (SABs) are microbial communities on air-exposed surfaces that play key roles in biogeochemical cycling and deterioration of built heritage. Their activity and persistence strongly depend on environmental conditions, particularly moisture and carbon availability. A mathematical framework is presented to investigate SAB dynamics and ecology. The model describes SABs as thin mixed biofilm-water layers on stone surfaces and is based on ODEs governing the dynamics of key components - including cyanobacteria and heterotrophs - together with intracellular pools of carbon, nitrogen, and energy. These components interact through dominant metabolic pathways constrained by biotic and abiotic factors. Daily cycles of temperature, humidity, light, and CO_2 are incorporated to capture effects on water availability and metabolism.

Simulations explore SAB dynamics in a real-world context and reveal distinct daily windows of microbial activity primarily controlled by water availability and light. Heterotrophs contribute to SAB stability by recycling organic matter and regulating pH. The model assesses long-term stability under future environmental scenarios. While temperature increases alone produce limited effects on SABs, elevated CO_2 may enhance carbon fixation and productivity. Relative humidity emerges as the key factor regulating SAB viability through water availability.

MS28-05 — Mathematical Modeling of Microbial-Induced Corrosion of Bioplastic in Marine Environments

SPEAKERS Luigi Frunzo (Univ Naples)
AUTHORS Alberto Tenore (Univ Naples); Fabiana Russo (University of Naples Federico II); Luigi Frunzo (Univ Naples); Maria Rosaria Mattei (Univ Naples); Vincenzo Lungo (Univ Naples)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Microbial-induced corrosion (MIC) and biodegradation in marine environments are driven by complex microbial consortia whose metabolic activity controls chemical transformations at material surfaces. In this presentation, we present a mathematical framework describing the interaction among microbial functional groups and their role in degradation processes. The model is applied to the biodegradation of biodegradable plastics in deep-sea conditions, accounting for microbial attachment, biofilm formation, enzyme secretion, polymer breakdown, and uptake of soluble compounds.

The system is formulated as a set of nonlinear differential equations, combining hyperbolic equations for microbial populations, parabolic equations for enzymes and degradation products, and an ordinary equation for biofilm thickness. To capture spatial effects, a one-dimensional two-layer diffusion model with double free boundaries is introduced, describing the interaction between biofilm growth and corrosion layer formation.

Numerical simulations highlight the key role of microbial metabolism in regulating degradation rates and shaping microbial community dynamics. The results provide insights into MIC mechanisms and offer a predictive tool for assessing bioplastic fate in marine environments.

MS28-04 — Towards a mechanistic model of the mercury methylation process

SPEAKERS Grace D'Agostino (University of Guelph)
AUTHORS Grace D'Agostino (University of Guelph); Harry Gaebler (Falkirk Environmental Consultants and University of Guelph); Hermann Eberl (University of Guelph)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Mercury methylation is a microbially mediated process that occurs in anaerobic soils, sediments, and at the water-sediment interface. Sulfur-reducing bacteria (SRB) uptake substrates, namely sulfate and dissolved organic carbon for growth, and transform inorganic mercury into methylmercury. Methylmercury is the

most abundant and toxic organic mercury compound that bio-accumulates in tissues and bio-magnifies in the food chain. It is a neurotoxin that is a threat to all trophic levels and all stages of human life.

We present a model of the mercury methylation process that considers the governing chemical reactions; the production of methylmercury by SRB and the inhibition by sulfide, the product of the redox reaction that supports SRB proliferation, at high enough concentrations. In a first study, the model is studied in a chemostat setting. We perform model analysis and validation, and numerical simulation experiments to explore the system dynamics.

MS28-06 — Bacterial Biofilm Growth Modelling in a Counter-Diffusion System, Coupled with Biozone Formation in the Aqueous Phase by Planktonic Bacteria, driven by Directed Movement.

SPEAKERS Rachana Mandal (Univ. Guelph)
AUTHORS Hermann Eberl (University of Guelph); Rachana Mandal (Univ. Guelph)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

In marine environments, bacterial biofilm formation occurs on the surface of plumes of marine snow that serve as moving nutrient hotspots. We develop a mathematical model on bacterial biofilm study that accounts for biomass growth, surface attachment-detachment, diffusive and directed movement of planktonic bacteria and perform a numerical simulation study. The biomass density controls the spatial expansion of biofilm, whereas biomass growth depends on the concentration of two counter-diffusive substrates, carbon and oxygen. Carbon diffuses from surface of marine snow into the domain, while oxygen enters from the opposite boundary. Planktonic bacteria, driven by directed movement, accumulate in regions with favorable growth conditions. The system is described by a one-dimensional set of four highly nonlinear PDEs. The flux-conservative finite volume method is used for space discretization of transport terms corresponding to biomass in biofilm and suspension. Substrate equations are discretized and numerically solved using a time-adaptive method from 'ReacTran' library in 'R'. Simulation results show biofilm expansion toward the aqueous phase and dynamic migration of suspended bacteria toward optimal nutrient zones. A comparative study on uniform and non-uniform grid refinement using the Shishkin mesh and graded mesh is conducted. The interplay between directed movement, attachment-detachment and counter-diffusion is shown to significantly influence biofilm maturation dynamics.

MS28-03 — Aspects of Nonlinear Dynamics, Spatial Effects and Cellular Memory in Bacterial Quorum Sensing

SPEAKERS Christina Kuttler (TU Munich)
AUTHORS Christina Kuttler (TU Munich)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Many bacterial species employ quorum sensing as a communication mechanism to coordinate collective behaviours such as pathogenicity or major lifestyle transitions. Gene regulatory networks govern these processes, that typically involve coupled positive and negative feedback loops, giving rise to nonlinear dynamics and, under suitable conditions, bi- or multistability. Additionally, intervention strategies, including classical antibiotics as well as quorum quenching, may play a role. The diffusion of signalling molecules introduces an additional layer of complexity. In particular, the coupling of nonlinear reaction kinetics with diffusion processes naturally leads to reaction–diffusion systems, in which spatial inhomogeneities can induce pattern formation, travelling waves, or spatially heterogeneous switching between stable states. Beyond these effects, increasing evidence suggests that bacterial cells can exhibit memory-like behavior, where prior exposure to signals or environmental conditions influences current responses. From a modeling perspective, such effects can be incorporated e.g. via additional dynamical variables or delayed terms in the underlying nonlinear system. Our aim is to analyse these mechanisms by combining dynamical systems and reaction–diffusion approaches and to discuss the biological meaning behind.

MS28-07 — Modelling accumulation of toxic substances in bacterial cells: a route to suppressing antimicrobial resistance

SPEAKERS Sandeep Shirgill (University of Birmingham)
AUTHORS Sandeep Shirgill (University of Birmingham); Sara Jabbari (Univ Birmingham)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The global rise in antimicrobial resistance levels, coupled with the downturn in discovery of new antibiotics, has resulted in an urgent need for novel ways to tackle bacterial infections. Most antibiotics work by accumulating inside bacterial cells, but bacteria have evolved mechanisms to prevent prolonged intracellular exposure to toxic substances including by limiting the permeability of their membrane (to prevent substances entering the cell) and activating efflux pumps (to secrete substances outside of the cell). Understanding how these processes are regulated under different conditions presents a route for us to manipulate them for therapeutic gain. We will present a variety of mathematical modelling approaches, integrated with experimental data where possible, to investigate the dynamics of toxic substance accumulation in bacteria cells. This computational framework enables the generation of experimentally-testable predictions on the optimisation of accumulation.

MS28-08 — Ecological interactions and spatial dynamics in microbial aggregates: A novel modelling framework

SPEAKERS Viktoria Freingruber (TU Delft)
AUTHORS Havva Yoldas (TU Delft); Rebeca Gonzalez-Cabaleiro (TU Delft); Viktoria Freingruber (TU Delft)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

We present a mathematical model based on a system of partial differential equations (PDEs) with cross-diffusion and reaction terms to describe ecological interactions between multiple bacterial species and substrates within microaggregates, where bacteria proliferate in response to substrate availability and undergo passive dispersal driven by population pressure gradients. The ecological interactions include interspecific competition for shared substrates, and commensalism, whereby one species benefits from the metabolic by-products of another. The main motivation comes from individual-based models (IBMs) of microbial aggregates, where simulations reveal that substrate-limited conditions can give rise to rich spatial patterns. Our numerical experiments demonstrate that our PDE-based model captures the key qualitative features of three verification scenarios that have previously been investigated with IBMs. Moreover, we formally derive a competition system from an on-lattice biased random walk, and establish local well-posedness for a parameter-symmetric subcase of it. We then formally analyse the travelling wave behaviour of this case in one spatial dimension and compare the minimal travelling wave speed with the wave speed measured in the simulations.

MS30 — Introduction to chemical reaction network modelling and simulation with Catalyst.jl

TRACK Miscellaneous
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 01.22 - HS
ORGANISERS Samuel Isaacson (Boston University); Torkel Loman (University of Oxford); Sebastian Persson (Francis Crick Institute)

Minisymposium abstract

This workshop introduces Catalyst.jl, a feature-rich Julia library for modeling chemical reaction networks (CRNs) arising in epidemiology, systems biology, ecology, and beyond. In Catalyst, symbolic reaction

systems can be constructed using chemical reaction notation or generated programmatically, and can be coupled to non-reaction ODEs, algebraic equations, and events. Catalyst models automatically convert to symbolic ModelingToolkit.jl representations of reaction rate equations (ODEs), chemical Langevin equations (SDEs), stochastic chemical kinetics (jump processes), or hybrid models. These are then compiled to optimized Julia functions, enabling access to a broad variety of solvers and integration with higher-order tools (such as for sensitivity analysis, parameter estimation, and bifurcation analysis).

Catalyst's symbolic representation enables a variety of CRN analysis, including detection and elimination of conservation laws, calculation of network properties (such as deficiency), automatic calculation of dependency graphs for Gillespie methods, and algebraic simplifications that significantly speed up computations.

The first half of this workshop will focus on model creation and simulation. The second half will demonstrate tools for model analysis and inverse problems, including symbolic computation of network properties (e.g., deficiency indices), steady-state analysis via homotopy continuation, bifurcation analysis, and parameter estimation via PEtab.jl.

MS30-01 — Introduction to Catalyst.jl and Applications in Chemical Reaction Network Modeling

SPEAKERS Samuel Isaacson (Boston University); Torkel Loman (University of Oxford)
AUTHORS Samuel Isaacson (Boston University); Torkel Loman (University of Oxford)
SCHEDULE Tuesday, 14 July 2026; 15:00–16:20; 01.22 - HS
TRACK Miscellaneous

Abstract

Modern mathematical biology requires moving seamlessly from model construction to highly optimized simulation and deep mathematical analysis. In this first of two sessions, we introduce Catalyst.jl, a domain-specific language and symbolic modeling platform within the Julia SciML ecosystem designed for the rapid, intuitive modeling of complex biological networks. With a special focus on the newly released Catalyst 16, attendees will learn how to construct systems and automatically generate ordinary differential equations (ODEs), stochastic differential equations (SDEs), and jump processes models. We will highlight new Catalyst 16 capabilities such as adding coupled jump-diffusion equations, enabling the simulation of advanced systems like stochastic neuron models and reaction networks that include dynamic, fluctuating, cell volumes. We will also present performance optimization strategies for speeding up simulations.

Complementing basic model construction and simulation, we will demonstrate how Catalyst easily integrates with packages throughout the broader Julia ecosystem. First, we will consider how HomotopyContinuation.jl and Catalyst enable finding all of a model's steady states. Next, we will show how to compute bifurcation diagrams using BifurcationKit.jl. Finally, we will show how such packages can leverage Catalyst's network analysis functionality, including the automatic detection and computation of conservation laws.

MS30-02 — Parameter Identifiability and Inference with Catalyst.jl and PEtab.jl

SPEAKERS Sebastian Persson (Francis Crick Institute); Torkel Loman (University of Oxford)
AUTHORS Sebastian Persson (Francis Crick Institute); Torkel Loman (University of Oxford)
SCHEDULE Tuesday, 14 July 2026; 17:00–18:20; 01.22 - HS
TRACK Miscellaneous

Abstract

In this second part of the session on Catalyst.jl, we focus on inverse problems, i.e. estimating parameters in models via fitting to data. Using Catalyst models as examples, we will demonstrate how Julia packages can be composed to generate complete model fitting pipelines.

First, we will show how structural identifiability can be assessed for any model using StructuralIdentifiability.jl. Next, we will demonstrate how Catalyst.jl integrates with PEtab.jl to define and solve inverse

problems by fitting models to time-series data. This includes showing how PEtab.jl can be used to create parameter problems with a wide range of features, such as events/callbacks, simulation conditions (where the model is simulated under different control parameters for different measurements), complex observable models linking model output to data, and steady-state initialization. We will then show how to fit models using robust global optimization methods such as multistart parameter estimation, and how to assess practical identifiability using profile likelihood analysis.

Finally, we will cover how to define and perform parameter estimation for scientific machine learning (SciML) models that combine mechanistic and data-driven components using Catalyst.jl and PEtab.jl. As these models are often challenging to train, we will also demonstrate how to leverage state-of-the-art training strategies implemented in PEtab.jl to improve parameter estimation performance.

MS48 — Models and Methods for the Analysis of Cancer Treatment

TRACK Mathematical Oncology
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 62.01 - HS
ORGANISERS Kathleen Wilkie (Toronto Metropolitan University)

Minisymposium abstract

This minisymposium brings together speakers that will highlight advances in the modelling, simulation, and analysis of various treatments for cancer. Novel approaches are required for new drug developments, including bi-specific molecules, nanoparticles immunotherapies, drug combinations and synergies, adaptive therapy, virtual clinical trials, and so on. Speakers in this session will discuss recent mathematical work aimed at improving understanding of cancer treatment through the simulation and analysis of models. There are numerous factors to consider in the biological response of a treated tumour within the human body. Here we will highlight many of these factors, and the diverse modelling approaches needed to describe and analyze the motivating complex system.

MS48-02 — Spatial Mechanistic Modelling of T-Cell Engagers: Insights into Trimer Formation and its effect on Cancer Killing

SPEAKERS Gibin Powathil (University of Swansea)
AUTHORS Gibin Powathil (University of Swansea); Jonathan Davies (Swansea University); Liam Brown (AstraZeneca); Mark Jones (Swansea University); Mark Penney (AstraZeneca)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

The rapid expansion of immunotherapies in Oncology has increased the need for quantitative models that can understand their mechanisms and limitations. T cell engagers (TCEs) are an emerging therapeutic class that bind both T cell receptors and tumour associated antigens to promote immunological synapse formation and targeted cytotoxicity. Despite growing clinical success, their efficacy is constrained by challenges such as treatment limiting toxicities, T cell exhaustion, and the hook effect. Mathematical and computational modelling offers a powerful means to understand these processes and support the rational optimisation of TCE design. We present an agent-based multiscale modelling framework using PhysiCell to investigate how binding kinetics, receptor expression, and spatial heterogeneity within the tumour microenvironment influence TCE function. By integrating mechanistic binding rules with spatially explicit cell–cell interactions, the model enables systematic exploration of trimer formation dynamics, the emergence of the hook effect, and their combined impact on tumour cell killing.

MS48-01 — From Image to Simulations: Integrating Imaging Biopsy Data into Agent-Based Models to Understand the Impact of Tumour-Microenvironment Spatial Heterogeneity on Cancer Treatment

SPEAKERS Blanche Mongeon (Sainte-Justine University Hospital Research Centre, Université de Montréal)
AUTHORS Blanche Mongeon (Sainte-Justine University Hospital Research Centre, Université de Montréal); Morgan Craig (Sainte-Justine University Hospital Research Centre, Université de Montréal)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Glioblastoma is the most common and deadliest primary brain tumour in adults, with a median survival of 15 months under the current standard of care [TGS20]. Its tumour microenvironment has been shown to be highly heterogeneous [Kar+23], meaning the magnitude of cell-cell interactions might differ due to spatial differences. While ordinary differential equation models can be useful to analyze novel treatment strategies and run preliminary virtual clinical trials [MC25], they do not easily allow for describing this spatial cell heterogeneity and its impact on treatment outcomes. To that end, new single-cell imaging modalities like imaging mass cytometry can be leveraged and integrated to computational agent-based models [Sur+23]. However, to effectively capture intra-tumoral dynamics, we have shown that care must be put into this integration and that model initialization must be done with a sufficient number of cells to provide biologically relevant predictions [Mon+24]. Here, we propose a mechanistic reconstruction of homogeneous and heterogenous imaging mass cytometry data that captures first- and second-order spatial summary statistics. We then generate larger synthetic datasets from patient-specific glioblastoma biopsy samples using our algorithm and integrate them into an agent-based model to study the effect of tumour micro-environment spatial heterogeneity on glioblastoma treatment outcomes. Though our focus is on the incorporation of imaging mass cytometry data into an ABM, our technique has applications beyond this case study to other multi-type homogeneous and heterogeneous and point patterns.

References

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MS48-03 — Mechanistic Modeling of Heterogeneous Tumor Response to Anti-NUPR1 Therapy in Docetaxel-Resistant Prostate and Pancreatic Cancers

SPEAKERS Harsh Jain (University of Minnesota Duluth)
AUTHORS Harsh Jain (University of Minnesota Duluth)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Resistance to the anti-mitotic drug docetaxel is a major challenge in the treatment of solid tumors, including prostate and pancreatic cancers. The stress-response protein NUPR1 has been identified as a key molecular driver of docetaxel resistance, suggesting that inhibition of NUPR1 may restore treatment sensitivity and enable effective combination therapy. In this talk, I develop mathematical models that incorporate the mechanisms of action of docetaxel and relevant NUPR1 pathways to study tumor response to combined docetaxel and NUPR1-targeted therapy. The models are informed by preclinical data, including in vitro prostate cancer cell-line experiments and in vivo pancreatic cancer xenografts, and capture tumor growth dynamics and treatment-induced cell death. Using these calibrated models, we quantify potential synergy between therapies and extend the analysis to heterogeneous virtual populations that represent variability in tumor growth and drug response. This framework enables systematic exploration of treatment schedules and patient heterogeneity, allowing us to assess when NUPR1-targeted combination therapy is likely to be successful and to identify subpopulations that may benefit most. These results illustrate how mechanistic modeling and virtual populations can support the evaluation and design of combination therapies aimed at overcoming chemotherapy resistance.

MS48-04 — Model-supported patient stratification using multi-objective synergy optimization in combination therapy

SPEAKERS Irina Kareva (Quercus Insights)
AUTHORS Irina Kareva (Quercus Insights)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

The challenge of stratifying patients for combination therapy is both technically demanding and clinically crucial. We build upon our previous framework for identifying Pareto optimal doses, reconciling competing definitions of synergy through multi-objective optimization to balance synergy of efficacy and potency (a measure of toxicity) such that no further improvement is possible in one without detriment to the other, and extend it to address interpatient heterogeneity. We apply the methodology to our previously developed model of a combination of an immune checkpoint inhibitor and an antiangiogenic agent and extend it to address interpatient heterogeneity. We demonstrate that depending on patient-specific drug sensitivities, different regimens may be Pareto optimal for different subgroups. We also highlight that there exist subpopulations for whom no meaningfully efficacious combination exist, suggesting that these individuals are not good candidates for this drug combination. In situations where measurable biomarkers are unavailable, we propose an initiation protocol with explicit, practical criteria that permit the estimation of patient-specific sensitivities to both monotherapies and to the combination therapy. Taken together, the proposed approach provides a potential way to find the right combination therapy for a patient, and to find the right patient for existing therapy.

MS48-05 — Assessing the Role of Model Complexity in Virtual Clinical Trial Outcomes

SPEAKERS Jana Gevertz (The College of New Jersey)
AUTHORS Jana Gevertz (The College of New Jersey); Joanna Wares (University of Richmond)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Virtual clinical trials (VCTs) hold significant promise for improving the drug development process, yet their predictive reliability depends on design decisions that remain poorly understood. This presentation investigates how model complexity, prior parameter distributions, and virtual patient (VP) inclusion criteria interact to shape VCT outcomes.

Using oncolytic virotherapy in murine tumors as a case study, we compared three models of varying complexity using different parameter priors and methods for including/excluding a parameterization in a virtual population. Our results demonstrate that while the simplest model inadequately spans the feasible trajectory space, potentially missing critical interpatient heterogeneity, there are diminishing returns beyond intermediate complexity. Both intermediate and complex models captured similar ranges of patient responses across various dosing protocols.

Further, we show that methods that simply reject "out-of-bound" parameterizations can generate posterior distributions that overly resemble the chosen prior, artificially reducing variability in treatment responses. In contrast, patient generation methods that instead perturb "out-of-bound" VPs to make them feasible produced results less sensitive to prior assumptions. These findings suggest that VCT design should prioritize intermediate-complexity models to capture key biological mechanisms, paired with perturbation-based inclusion criteria that prevent unrealistic prior assumptions from overconstraining the virtual population. [Joint with Dr. Joanna Wares]

MS48-06 — Cellular kinetics of drug response: are they conserved when altering experiment and biological context?

SPEAKERS Matt Faria (University of Melbourne, Pneumatica Bio)
AUTHORS Aria Ahmed-Cox (The University of Melbourne, Children's Cancer Institute Australia); Hyeon Hae Lee (The University of Melbourne); Matt Faria (University of Melbourne, Pneumatica Bio); Stuart Johnston (The University of Melbourne, Pneumatica Bio)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Sometimes! Gleaning insight from a model independent of data is (understandably) rarer and rarer in mathematical biology, and determining parameters from biological data has become an established practice of modern mathematical modelling. Once parameters are estimated (ideally with bounds), an important question remains: whether (and to what extent) are biological parameters conserved? For example: do cells grow at the same rate in a dish and in an organoid? Are enzyme kinetics in a beaker the same as within a cell? Is the dose-response curve the same for a cell in and out of the body?

Often, biological parameters are implicitly assumed to be conserved - but this is an assumption we should examine and grapple with. In this talk, I will present our recent work examining conservation of biological parameters that describe the kinetics of cellular response to targeted therapeutics (i.e., drugs that have been engineered to preferentially interact or avoid specific types of cell) – specifically, to lipid nanoparticles (LNPs). We have investigated to what extent these cellular kinetics are conserved when changing experimental details or biological contexts. This work may be of particular interest to those developing targeted therapeutics or lipid nanoparticles, or to those interested in mathematical oncology (as one of the primary expected uses of targeted therapeutics is in the treatment of cancer).

MS48-08 — An Agent Based Modelling Framework for the Role of Tumour-Stroma Dynamics in Metastatic Colorectal Cancer

SPEAKERS Sam Oliver (University of Oxford)
AUTHORS Helen Byrne (University of Oxford); Joshua Moore (University of Oxford); Sam Oliver (University of Oxford); Simon Leedham (University of Oxford)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Metastasis to the liver remains a leading cause of mortality in colorectal cancer patients, due largely to the difficulty of treating established metastatic lesions. Spatial transcriptomic (ST) imaging provides highly detailed, spatially resolved data on the cellular composition and interactions within these lesions, offering new insights into how metastases are established and evolve in the liver. Upon arrival, tumour cells interact with hepatic cells and actively remodel the surrounding tissue to form a supportive metastatic niche [Li+25]. These early processes, which can be resolved through spatial analysis of ST images, determine whether lesions can successfully form and expand. Here, we develop an agent-based multiscale model using a PhysiCell framework to investigate colorectal metastases in the liver [Gha+18; GFM16]. To bridge experimental data and model development, we employ a spatial analysis pipeline using tools such as Muspan [Bul+24] to quantify cellular neighbourhoods, interactions, and tissue architecture from the ST images. This approach enables the construction of a data-driven, spatially informed agent-based model that captures the mechanistic processes underlying metastatic colonisation. By validating the model against experimental observations, we establish a framework to predict disease progression and potentially improve future therapeutic strategies.

References

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MS48-07 — Mechanistic Learning of Engineered Immune Cell-Tumor Cell Interactions and Heterogeneity

SPEAKERS Philipp Altrock (Cancer Modeling & Evolution UKSH Campus Kiel)
AUTHORS Philipp Altrock (Cancer Modeling & Evolution UKSH Campus Kiel)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Chimeric Antigen Receptor (CAR) T-cell therapy has emerged as a promising option for relapsed or refractory lymphoma patients and acute leukemias. Mathematical modeling offers a valuable tool for investigating the interactions among these living drugs, tumors, and heterogeneous patients’ immune or inflammatory context. Using longitudinal data from experiments and the clinic, we examine how diverse CAR T cell phenotypes interact with heterogeneous tumors, how these interactions can be inferred, and how they impact outcomes.

MS67 — Epidemiological-behavioural modelling to address health challenges

TRACK Mathematical Epidemiology
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 15.03 - HS
ORGANISERS Edward Hill (University of Liverpool); Matthew Ryan (Commonwealth Scientific and Industrial Research Organisation (CSIRO))

Minisymposium abstract

The behavioural element in the transmission dynamics of infectious diseases is very influential; infection risk affects behaviour while behaviour affects individual and communal infection risk.

There are several challenges recognised amongst the epidemiology and infectious disease modelling communities on incorporating the dynamics of behaviour into models of infectious disease dynamics [Fun+15; Hil+24]. Challenges arise due to a lack of readily translatable quantitative behavioural science models that might capture the changing of relevant behaviours, societal norms and policy directives across individuals and/or populations, particularly in novel social contexts. These challenges are not confined to public health; veterinary and plant health researchers also strive to integrate infectious disease and behavioural dynamics [Hid+18; MHC22]. The COVID-19 pandemic highlighted the deficiencies in availability of both suitable data and of disease outbreak models to reasonably incorporate data-driven and/or theoretical knowledge regarding the behavioural response to a pandemic, including the drivers of voluntary behaviour changes [Bro+23].

With that motivation, we share research examples tackling interdisciplinary problems in epidemiology and infectious disease modelling that involve the dynamics of behaviour. Application areas cut across public health, veterinary health and plant health.

References

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MS67-02 — Integrating human behaviour and epidemiological modelling: unlocking the remaining challenges

SPEAKERS Edward Hill (University of Liverpool); Matthew Ryan (Commonwealth Scientific and Industrial Research Organisation (CSIRO))
AUTHORS Edward Hill (University of Liverpool); Matthew Ryan (Commonwealth Scientific and Industrial Research Organisation (CSIRO))
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

The SARS-CoV-2 pandemic highlighted that epidemic models fail to incorporate data-driven and theoretical knowledge of behavioural response to pandemics. This gap is partially driven by the lack of quantitative models that can predict the adoption of behaviours across individuals and populations, particularly in new social contexts. Hence, there is a need to improve behavioural realism in integrated "epidemiological-behavioural" models.

We will first summarise our involvement in related events around epidemiological-behavioural modelling. That includes an upcoming Isaac Newton Institute Satellite programme on 'Maths of Human Behaviour: modelling sociality, mobility and protectionism' taking place during 20 July – 14 August 2026 at the University of Nottingham, UK (https://www.newton.ac.uk/event/mhb/).

Ed will discuss a perspective paper [Hil+24] highlighting: (i) the key role of interdisciplinary collaboration for integrating dynamic human behaviour into epidemiological models; (ii) interdisciplinary challenge areas in epidemiological-behavioural modelling; (iii) recommendations to make progress in each of the challenge areas.

Matt will then present work that investigates behavioural trends in testing behaviour for COVID-like illness [Rya+25]. Matt will briefly describe how dynamic testing behaviour can be included into compartmental models and highlight key results from this modelling.

References

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MS67-01 — Epidemics Don't Just Spread - People React: Modelling Behaviour in Real Time

SPEAKERS David Haw (University of Liverpool, UK)
AUTHORS David Haw (University of Liverpool, UK)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Standard epidemic models tend to assume that human behaviour is fixed, rational, or slow to change. Reality is messier. During COVID-19 and beyond, behaviour has proven fast-moving, socially contagious, and often emotionally driven. People respond to risk, to each other, and to policy - sometimes amplifying interventions, sometimes undermining them. We present a modelling framework that treats behaviour as part of the epidemic system itself, not an external input. Transmission dynamics are coupled to time-varying behavioural indices (e.g. trust, adherence), which evolve through feedback from incidence, peer influence, and bounded policy responses. This creates a two-way interaction: epidemics shape behaviour, and behaviour reshapes epidemics. Using structured perturbations of behavioural drivers under realistic "policy budgets," we generate ensembles of counterfactual scenarios and quantify uncertainty in outcomes such as peak incidence and healthcare demand. Even small shifts in behavioural response can produce large and nonlinear differences in epidemic trajectories, particularly around critical periods such as pre-peak intervention timing. The key message is simple: identical policies can lead to very different

outcomes, depending on how people respond. By embedding behaviour directly into mechanistic models, we move towards a more realistic and policy-relevant understanding of epidemic dynamics - one where human response is not noise, but signal.

MS67-03 — Modelling social influence through discussion-based contacts in coupled behaviour-epidemic models

SPEAKERS Elisabetta Colosi (Bocconi University, Italy)
AUTHORS Alessia Melegaro (Bocconi University, Italy); Chiara Chiavenna (Bocconi University, Italy); Claudia Cozzolino (Sorbonne Université, INSERM, France); Duilio Balsamo (Bocconi University, Italy); Elisabetta Colosi (Bocconi University, Italy); Francesco Bonacina (Bocconi University, Italy); Lorenzo Lucchini (Bocconi University, Italy); Vittoria Colizza (Sorbonne Université, INSERM, France); Vittoria Offeddu (Bocconi University, Italy)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Peer influence can act as an invisible force that promotes or hinders the adoption of health-protective behaviours. While traditional epidemiological models focus on physical contacts driving transmission, they often overlook the social interactions through which opinions and behaviours spread.

We use contact data from a multinational survey of over 22,000 respondents across six European countries to construct age-stratified matrices for disease transmission and social contagion [Off+25], distinguishing physical from discussion-based interactions. We develop a stochastic SIR model in which adoption and abandonment of protective behaviours are mediated by discussion contacts and modulated by infection and recovery levels through non-linear responses.

Incorporating empirical discussion mixing into the model produces an earlier and sharper epidemic peak due to rapid behavioural uptake, followed by heterogeneous relaxation across age groups that generates a secondary wave absent under fixed adoption. Assuming the age structure of physical contacts for discussion yields epidemic trajectories that generally diverge from the empirical case, with more pronounced effects in some countries than others.

This data-driven framework captures peer-mediated behavioural contagion and shows how age-specific social influence can reshape both adoption and epidemic dynamics, underscoring its importance in fostering the uptake of protective behaviours.

References

[Off+25] Vittoria Offeddu et al. “Advancing coupled behavioural-epidemic models: An interdisciplinary framework for the collection of empirical data”. In: *medRxiv* (2025), pp. 2025–10.

MS67-05 — Feedback loops in multi-season influenza vaccinations strategies

SPEAKERS Joel Winterton (University of Cambridge, UK)
AUTHORS Joel Winterton (University of Cambridge, UK); Julia Gog (University of Cambridge, UK)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Annual vaccination remains the most effective intervention in reducing the burden of seasonal influenza epidemics. Considering vaccination strategies over multiple seasons could allow the identification of more effective interventions than those designed for a single season. The effect of vaccination depends on and changes both the immunological memory of the host population and the evolutionary pressure driving antigenic drift, creating a multi-season feedback loop in which current interventions influence future disease dynamics and vaccine performance.

In this talk, we explore such a multi-season feedback loop by considering how vaccination strategies targeting groups with different social contact patterns perform across multiple seasons. We explore if and when the dependence of vaccine efficacy on prior infection and vaccination history can change the relative effectiveness of targeting each group, as well as some structural trade-offs that emerge in long-term vaccination dynamics.

MS67-04 — Modelling the adoption of Integrated Pest Management (IPM) by UK crop growers

SPEAKERS Elliot Vincent (University of Warwick, UK)
AUTHORS Edward Hill (University of Liverpool, UK); Elliot Vincent (University of Warwick, UK); Stephen Parnell (University of Warwick, UK)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Reducing the harmful impact of pesticides is a key challenge faced by the agricultural industry globally. In many countries, policies have been introduced to discourage the use of pesticides, while encouraging the use of alternative methods of crop disease control. One such method is Integrated Pest Management (IPM), a set of holistic and sustainable measures for the prevention, monitoring and control of crop diseases [UK 25]. While many IPM measures are acknowledged to offer effective control, their implementation has thus far been limited in the UK.

To explore the potential impact of policy on the adoption of IPM by crop growers in the UK, we developed a joint epidemiological-behavioural model [VHP25; VPH26]. Within a simulated outbreak of Septoria tritici blotch (STB), growers could choose to switch between two disease control strategies based on their relative profitability: IPM, and a conventional fungicide regime. In these simulations, we found that the projected rate of IPM adoption would not meet the UK's pesticide reduction targets for 2030. We also found that policies addressing behavioural barriers among crop growers had the greatest potential to increase adoption trends, compared to other forms of incentive.

References

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MS67-06 — Memory mechanisms in Bayesian behavioural change epidemic models

SPEAKERS Rob Deardon (University of Calgary, Canada)
AUTHORS Yicheng Mao (University of Calgary); Rob Deardon (University of Calgary, Canada); Lorna Deeth (University of Guelph, Canada)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Accurately capturing epidemic dynamics requires accounting for how individuals adjust behaviour in response to perceived infection risk. Recently models have been developed to incorporate behavioural feedback [WDS23], but they currently rely on simplified, ad hoc representations of memory. For instance, many emphasize only recent case counts, neglecting the lasting influence of earlier epidemic experiences on current risk perception. Here, we introduce a framework of Memory Mechanism Enhanced Behavioural

Change (MEBC) models within a Bayesian SIR setting. Five memory formulations – memoryless, sliding window, power-law, exponential, and reciprocal – are considered, each reflecting a distinct way past epidemic information shapes present behaviour. A fully Bayesian data-augmented MCMC approach jointly estimates transmission and behavioural parameters while accounting for uncertainty in infectious periods. Simulation results show that the MEBC framework provides accurate parameter recovery and remains robust under misspecified memory assumptions. Applications to the early COVID-19 outbreak in Miami-Dade County and the 2023–2024 influenza season in Manitoba demonstrate that incorporating an interpretable memory mechanism significantly improves model fit, underscoring the importance of collective memory in behavioural adaptation and disease transmission.

References

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MS67-07 — Oscillatory epidemic dynamics driven by non-linear behavioural feedback

SPEAKERS Ryosuke Omori (Hokkaido University, Japan)
AUTHORS Koichi Ito (Doshisha University, Japan); Ryosuke Omori (Hokkaido University, Japan)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Spontaneous behavioural changes in response to an outbreak can significantly alter transmission dynamics. Recent studies [Omo+24] have explored how the human perception of risk, and the subsequent reduction in contact rates, can be modelled as a function of epidemiological indicators. This study aims to provide a qualitative understanding of how such perception-based feedback shapes the emergence and characteristics of multi-wave epidemics.

We analyse an SIR-type model where the transmission rate depends on a non-linear function of past infection levels, reflecting the diminishing sensitivity of human behaviour to increasing incidence. To evaluate the oscillatory potential of the system, we employ a quasi-stationary approximation for the susceptible population. By linearizing the dynamics using logarithmic perturbations around the quasi-equilibrium, we derive the characteristic equation and analytically identify the conditions under which the interaction between the disease and behaviour leads to self-sustained oscillations. Our findings elucidate how the non-linear scaling of risk perception, combined with information lag, governs the fundamental frequency and damping of epidemic waves.

This analysis offers a theoretical basis for understanding the mechanisms by which behavioural-epidemiological interactions generate complex, multi-modal epidemic trajectories.

References

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MS67-08 — Predicting contact rates in response to control measures: a validated protocol for generating contact matrices relevant to transmission modelling of respiratory diseases

SPEAKERS Willem Frederiks (National Institute for Public Health and the Environment (RIVM), The Netherlands)
AUTHORS Willem Frederiks (National Institute for Public Health and the Environment (RIVM), The Netherlands); Ka Yin Leung (The Netherlands Institute for Social Research, The Netherlands); Jantien Backer (National Institute for Public Health and the Environment (RIVM), The Netherlands); Marijn de Bruin (Radboud University Medical Center & Radboud Institute for Health Sciences, The Netherlands); Jos

de Haan (The Netherlands Institute for Social Research, The Netherlands); Jacco Wallinga (National Institute for Public Health and the Environment (RIVM), The Netherlands); Don Klinkenberg (National Institute for Public Health and the Environment (RIVM), The Netherlands)

SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Transmission models are used to predict the course of epidemics and inform policy makers. For respiratory diseases, many models incorporate rates of contacts within and between age groups, summarized in contact matrices. Contact rates may change due to control measures. To forecast transmission, contact matrices should reflect this impact before measures are introduced. We present a protocol to predict future contact matrices using data on time-use, contacts, and demographics, collected before an epidemic.

For each set of control measures, we identified activities on which less time will be spent. The protocol assumes that reductions in time lead to proportional reductions in numbers of contacts made during these activities. School and work time were stratified by educational level and profession based on demographics. We validated the protocol by applying it to measures against COVID-19, comparing predicted to observed matrices.

Predicted matrices agreed with observed matrices. By age, predicted contact rates matched observed rates, especially for contacts involving adults. For child-child contacts, predicted rates were lower than observed rates, with observed school contacts likely overreported. Predicted workplace contact rates matched office occupancy data.

The protocol uses pre-epidemic data, providing consistent and transparent contact matrices for transmission models. It is suited for future epidemics when data are periodically collected.

MS73 — Cell Growth & Division: Connecting Phenomenology to Mechanisms

TRACK Cellular and Developmental Biology
SCHEDULE Tuesday, 14 July 2026; 15:00–16:20; 02.23 - HS
ORGANISERS Ethan Levien (Dartmouth College); Farshid Jafarpour (Utrecht University)

Minisymposium abstract

Cell growth and division are fundamental processes that shape cellular physiology and population dynamics across diverse organisms. Over the past century, substantial progress has been made in identifying the molecular components governing these processes. Yet growth and division are inherently complex, and bridging mechanistic descriptions with the statistical patterns observed in cell-size variability and gene-expression fluctuations remains challenging. Furthermore, the two processes are often treated as separate subfields, leaving open fundamental questions about how they are dynamically coupled and their involvement in cell size control.

Recent phenomenological models treat growth and division as stochastic processes and have shown promise in explaining empirical patterns. However, the deeper issue—how such coarse-grained models constrain or reflect underlying mechanistic architectures—remains largely unresolved. This minisymposium brings together researchers in mathematical modeling and experimental biology to advance this connection and to share recent progress in linking microscopic mechanisms to stochastic descriptions.

MS73-01 — Effects of molecular noise on bacterial size control

SPEAKERS Andrew Mugler (University of Pittsburgh)
AUTHORS Andrew Mugler (University of Pittsburgh)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Exponentially growing cells employ control strategies to maintain a stable size in the presence of noise. Phenomenological models provide important insights into these strategies, revealing classes such as ”timer”, ”adder”, and ”sizer” control. However, these models necessarily ignore the molecular mechanisms needed to implement the strategy. I will describe our work showing that incorporating these mechanisms can change the model conclusions in important ways. For example, the sizer strategy is thought to minimize the noise in cell size. But using a mechanistic model where division is triggered at a molecular abundance threshold, we find that the adder strategy minimizes noise in cell size. The reason is that cell size noise inherits the molecular noise of the division mechanism. We derive a lower bound on size noise that agrees with publicly available data from six microfluidic studies on Escherichia coli bacteria. Our work connects molecular mechanisms to phenomenological modeling and reveals the consequences of noise propagating across scales.

MS73-02 — Cell growth under high antibiotic concentrations drastically reshapes the bacterial transcriptome

SPEAKERS Daniel Schultz (Geisel School of Medicine)
AUTHORS Daniel Schultz (Geisel School of Medicine)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Bacteria exhibit remarkable phenotypic heterogeneity upon antibiotic exposure, induced by complex feedback mechanisms mediated by interactions between drug action, metabolism, gene regulation, and expression of resistance. However, how global transcriptional programs are rewired under drug stress in these different phenotypes remains poorly understood. In Escherichia coli, exposure to the translation inhibitor tetracycline was shown to produce distinct phenotypes with a range of growth rates, whose underlying transcriptional states have not been fully characterized. Here, we use RNA sequencing across nutrient and drug conditions to show that increasing tetracycline concentration leads to a progressive breakdown of transcriptional organization and the emergence of a disordered gene expression state. We find that exposure to intermediate tetracycline concentrations induce coordinated transcriptional changes consistent with the maintenance of homeostasis under the drug. In contrast, high drug levels trigger widespread dysregulation marked by increased transcriptional entropy, and the upregulation of stress and resistance functions at the expense of metabolism and growth. This work combines environmental perturbations with bulk transcriptomics to develop models explaining the progression of heterogeneous cellular states, providing insight into how bacteria persist in growth-limited, clinically relevant conditions.

MS73-03 — Cell Size Regulation from Bacteria to Mammalian Cells

SPEAKERS Kuheli Biswas (Weizmann)
AUTHORS Kuheli Biswas (Weizmann)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

How cells regulate their size remains an open question. Cell-size regulation is commonly characterized by the Pearson correlation between birth and division sizes, with the corresponding regulation parameter α defined as one minus this correlation coefficient. Single-cell experiments provide generation-resolved measurements of birth and division sizes along individual lineages, typically across many lineages of varying lengths. The parameter α is usually inferred either by estimating it separately for each lineage and averaging across lineages, or by fitting a single effective parameter to the pooled data across lineages. These two approaches are affected by distinct systematic errors: the lineage-wise estimator is biased for finite lineages, whereas the pooled estimator is biased by lineage-to-lineage variability arising from extrinsic noise. Moreover, both estimators are affected by different kinds of measurement errors. Here, we quantify the above-mentioned effects and develop a Bayesian framework to overcome these biases. Applied

to synthetic and experimental cell-size data, this approach yields more reliable estimates from short and heterogeneous lineages and provides a principled way to distinguish among different size regulation strategies.

MS73-04 — Essential Role of Extrinsic Noise in Models of E. Coli Division Control

SPEAKERS Mattia Corigliano (IFOM)
AUTHORS Mattia Corigliano (IFOM)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Our understanding of cell division control still relies largely on interpreting correlations between phenomenological variables, with limited connection to the underlying molecular mechanisms.

In this talk, I present an analytically tractable stochastic threshold–accumulation model in which a size-dependent divisor protein triggers division upon reaching a noisy, autocorrelated threshold. This framework disentangles, within a unified theory, the roles of intrinsic and extrinsic noise, as well as key mechanistic features such as threshold reset and threshold memory. We show that these ingredients generate a much richer spectrum of behaviors than the commonly assumed adder, spanning continuously from timer to sizer-like strategies while modulating size fluctuations.

Comparison with single-cell E. coli data indicates that extrinsic noise and additional mechanistic ingredients are required to reproduce the observed variability. However, mechanisms that control fluctuations typically reshape the underlying division strategy. Strikingly, we identify a regime in which size fluctuations can be tuned independently of division control, preserving adder-like behavior even in the presence of extrinsic noise. This regime emerges from a balance between threshold correlations and threshold reset, extending the prevailing view that full reset is required for adder control. Altogether, this provides a direct mechanistic link between molecular noise and the emergent division laws observed in data.

MS76 — Mathematical and AI-enhanced computational models for sexually transmitted diseases and other public health threats

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 15.06 - HS
ORGANISERS Qi Deng (York University); Edward Thommes (University of Guelph and Sanofi); Jianhong Wu (York University)

Minisymposium abstract

Sexually transmitted diseases (STDs) and other communicable diseases remain a major global public health challenge due to complex transmission dynamics, heterogeneous contact patterns, and strong social, behavioral, and biological interactions. Mathematical and computational modeling is essential for understanding STD dynamics and evaluating prevention, screening, and intervention strategies.

Advances in surveillance and information technologies have enabled large-scale data collection across health, pathogen, social, environmental, and behavioral domains. Strategic use of these data is critical for disease prevention, health promotion, and reducing disparities. Precision Public Health (PPH) addresses these challenges by applying artificial intelligence and advanced mathematical and statistical methods to assess disease dynamics, exposures, behaviors, and population-level susceptibilities, while promoting evidence-informed policies and targeted, equitable interventions.

This mini-symposium focuses on computational and data-driven modeling approaches for STDs and other communicable diseases, emphasizing public health relevance and AI integration. Contributions include mechanistic transmission models, network and agent-based models, stochastic and multiscale approaches, as well as machine learning and hybrid AI–mechanistic models informed by epidemiological and surveillance data.

MS76-01 — Development of agent-based modelling of STI's: Can we use AI methods for improving implementation of partnership dynamics?

SPEAKERS Mirjam Kretzschmar (University Medical Center Utrecht)
AUTHORS Alexandra Teslya (University Medical Center Utrecht, The Netherlands); Ganna Rozhnova; Jacob Roberts; Janneke van de Wijgert; Joshua Chevalier (UMC Utrecht); Mirjam Kretzschmar (University Medical Center Utrecht)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

With the recent increase in computing power, agent-based simulations have become progressively more complex. There is a growing tendency to include greater detail of sexual networks, disease progression, and targeted interventions into STI models to answer relevant public health questions. However, increasing model complexity also amplifies data requirements, and often necessitates the integration of data from different sources for model parameterization. One key challenge in STI transmission modeling is the accurate representation of the dynamic partnership network. Formation and separation of partnerships depend on many factors, including demographics (age, sex), partnership history, infection status, and an individual's perceived risk of acquiring infection. AI models offer a promising avenue to better integrate data analysis with agent-based modelling to link partnership dynamics directly to underlying determinants. In a first step to explore such a hybrid model, we integrated an XGBoost algorithm—trained on behavioural survey data—into a static network model to inform decisions of agents to engage in protective behaviour during an outbreak of a respiratory infection. We find that epidemic trajectories are influenced by underlying network structure as well as the adaptive behaviour of agents. For agent-based models of STI our approach may provide a flexible way to incorporate determinants of partnerships, predicting their formation or separation from behaviour surveys.

MS76-02 — Early-warning signals for infectious diseases with a social-media compartment

SPEAKERS Stacey Smith? (University of Ottawa)
AUTHORS Francisca Olajide; Stacey Smith? (University of Ottawa)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Early-warning signals (EWSs) are crucial tools for anticipating disease emergence and guiding public-health responses, but uncertainties in transmission and incomplete data can limit their reliability. Additionally, the performance of EWSs is rarely evaluated when disease emergence is delayed, and their use in the context of interactions between disease transmission and communication through social-media platforms has largely not been considered. We evaluate the relative performance of EWSs in predicting disease emergence under varying noise conditions and explore the use of EWSs with social-media dynamics to predict disease emergence. We develop a mechanistic model coupling infectious disease and social-media dynamics, introduce stochasticity and generate simulated time series. We detect changepoints, quantify delays relative to the bifurcation point and assess the performance of EWSs across different segments of the time series. The "reporting" infected compartment proves most reliable, and variance outperforms autocorrelation in high-noise scenarios. However, in the social-media compartment, variance and autocorrelation have weak predictive power. Our work provides a framework to advance understanding of how EWSs can be applied to forecasting disease emergence, contributing to improved disease preparedness and response.

MS76-03 — Mathematical Modeling of Post-Infection Mortality and Transmission Structure: Implications for Persistent Infections and Public Health Dynamics

SPEAKERS Zhisheng Shuai (University of Central Florida)
AUTHORS Zhisheng Shuai (University of Central Florida)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Post-infection mortality is an important yet often overlooked factor in epidemiological modeling, as it directly influences both disease prevalence and long-term population structure. In this work, we revisit and analyze compartmental models that explicitly incorporates post-infection mortality along with partial immunity, and we investigate how disease outcomes depend on the choice of incidence function. Under mass-action incidence, the inclusion of post-infection mortality can generate complex dynamics, including bifurcations that lead to recurrent outbreaks. In contrast, when standard incidence is used, such oscillatory behavior is greatly reduced or eliminated, and the system typically converges to a stable endemic state. Our results show that modeling post-infection mortality in combination with different transmission assumptions leads to markedly different qualitative and quantitative outcomes, underscoring its significance for understanding persistent infections and for guiding more realistic epidemiological predictions.

MS76-05 — Modelling COVID-19 vaccine breakthrough infections in highly vaccinated Israel—The effects of waning immunity and third vaccination dose

SPEAKERS Daihai He (The Hong Kong Polytechnic University)
AUTHORS Daihai He (The Hong Kong Polytechnic University)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In August 2021, a major wave of the SARS-CoV-2 Delta variant erupted in the highly vaccinated population of Israel. The transmission advantage of the Delta variant enabled it to replace the Alpha variant in approximately two months. The outbreak led to an unexpectedly large proportion of breakthrough infections (BTI)–a phenomenon that received worldwide attention. Most of the Israeli population, especially those aged 60+, received their second dose of the vaccination four months before the invasion of the Delta variant. Hence, either the vaccine induced immunity dropped significantly or the Delta variant possesses immunity escaping abilities, or both. In this work, we model data obtained from the Israeli Ministry of Health, to help understand the epidemiological factors involved in the outbreak. We propose a mathematical model that captures a multitude of factors, including age structure, the time varying vaccine efficacy, time varying transmission rate, BTIs, reduced susceptibility and infectivity of vaccinated individuals, protection duration of the vaccine induced immunity, and the vaccine distribution. We fitted our model to COVID-19 cases among the vaccinated and unvaccinated, for <60 and 60+ age groups, and quantified the transmission rate, the vaccine efficacy over time and the impact of the third dose booster vaccine. The peak transmission rate of the Delta variant was found to be 2.14 times higher than that of the Alpha variant. The two-dose vaccine efficacy against infection dropped significantly from >90% to 40% over 6 months. We further performed model simulations and quantified counterfactual scenarios examining what would happen if the booster had not been rolled out. We estimated that approximately 4.03 million infective cases (95%CI 3.19, 4.86) were prevented by vaccination overall, and 1.22 million infective cases (95%CI 0.89, 1.62) averted by the booster.

MS76-06 — An agent-based platform for simulating the impact of chlamydia vaccine in the US population

SPEAKERS Qi Deng (York University); Edward Thommes (University of Guelph and Sanofi)
AUTHORS Qi Deng (York University); Edward Thommes (University of Guelph and Sanofi)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Chlamydia trachomatis (CT) remains the most reported bacterial sexually transmitted infection in the United States (US), underscoring the urgent need for an effective vaccine. An agent-based model calibrated to the US National Health and Nutrition Examination Survey (NHANES) datasets was developed and used to study the impact of vaccine on the CT and all-cause pelvic inflammatory disease (PID) burden.

Model simulations predicted a conventional vaccine with 50% efficacy and 60% coverage can achieve a 64.2% [95%CI: 43.0–68.1] reduction in chlamydia prevalence and a 14.3% [95%CI: 10.1–29.4] reduction in the prevalence of women with one or more lifetime episodes of all-cause PID. In addition, in some countries, the testing policy for asymptomatic chlamydia has been changed. In Netherlands, from 2025, CT testing at Centers for Sexual Health is indicated only for individuals with symptoms or partner notification. No test means no diagnosis and no treatment. However, studies have shown that around 2%-5% untreated CT will develop PID. Following PID, around 15%-20% individuals will develop infertility. Thus we also applied the ABM platform to simulate the natural infection history of Chlamydia infection and testing the impact of removing asymptomatic tests, as well as the impact of limiting the screening to patients with symptoms on the incidence of CT, the incidence of PID, the incidence of longer-term sequelae, and on transmission.

MS76-07 — Decision-making, epidemiological dynamics, individuals, and societies

SPEAKERS Chadi Saad-Roy (University of British Columbia)
AUTHORS Chadi Saad-Roy (University of British Columbia)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The COVID-19 pandemic highlighted the importance of individual decision-making for adherence to an intervention. In this talk, I will use simple mathematical models to examine the dynamics of individual adherence to an intervention, and examine when tensions might arise between individuals and societies. I will also examine the effects of adherence to a nonpharmaceutical intervention on epidemiological dynamics (and vice-versa).

MS76-04 — Modular arithmetic as an alternative to model seasonal time-varying transmission rates: Influenza as a case study

SPEAKERS Woldegebriel Assefa Woldegerima (York University, Canada)
AUTHORS Nickson Golooba (York University, Canada); Woldegebriel Assefa Woldegerima (York University, Canada)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.06 - HS
TRACK Mathematical Epidemiology

Abstract

Existing established alternative methods for modeling seasonality use sinusoidal forcing functions, flexible splines, etc. In this talk, I outline a novel method for incorporating seasonality into the SIRS model using modular arithmetic. The study proposes a modular arithmetic approach to model seasonal variations in influenza transmission rates, with smooth transitions between them. Unlike sinusoidal models, which struggle with sharp transmission shifts, the modular approach captures abrupt changes linked to school terms and behavioral changes. Statistical methods like periodic splines achieve accuracy but often lack interpretability; the modular framework balances interpretability and data requirements. The study develops this framework as a theoretical tool, demonstrating its capacity to generate realistic, recurring seasonal outbreaks under plausible parameter assumptions. Then the model was calibrated using real-world influenza data from Ontario, Canada, from 2014 to 2019. Smoothing methods, such as a Gaussian kernel, are applied to avoid unrealistic jumps in transmission rates. A systematic optimization using a coarse grid search followed by stochastic refinement calibrates the model to the observed data. The study highlights the potential of modular arithmetic in enhancing the understanding of seasonal infectious diseases.

MS78 — Game theory in ecology and evolution

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 15.11 - HS

ORGANISERS Alexander Browning (University of Melbourne); Maria Kleshnina (Queensland University of Technology); Sara Hamis (Uppsala University)

Minisymposium abstract

Game theory, a field that models strategic interactions, is seeing a resurgence throughout the theoretical biology and ecology communities. Well-established techniques are being brought to new applications, and traditional modelling approaches expanded through game-theoretic ideas, including evolutionary games, stochastic games, and learning in interacting populations. These tools provide a unifying language to study behaviour, adaptation, and feedbacks between ecological and evolutionary processes. As approaches to modelling ecology and evolution become more diverse, the need to reconcile and recognise existing approaches becomes more important.

This mini-symposium presents a spectrum of researchers ranging from game theorists to traditional mathematical biologists, facilitating discussion between communities that may not otherwise interact. The session will open with an accessible perspectives talk providing a broad introduction to, and overview of, game theory as a modelling framework. This perspective will stimulate discussion and demonstrate what game theory has to offer mathematical biology. We proceed through a diverse spectrum of applications, ranging from the evolution of cooperation and social behaviour to eco-evolutionary dynamics and strategic interactions in structured populations, to models that leverage competition and strategic decision-making to improve outcomes in management-relevant applications such as ecosystem conservation and cancer treatment.

MS78-02 — Evolutionary game theory and the emergence of noisy learning

SPEAKERS Christian Hilbe (Interdisciplinary Transformation University)
AUTHORS Christian Hilbe (Interdisciplinary Transformation University)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:40; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

This talk has two complementary aims. The first part provides a self-contained and accessible introduction to (evolutionary) game theory, focusing on some key tools used to describe the dynamics of strategies in adaptive populations. In the second part, I explore a counterintuitive question: why might evolutionary processes favour seemingly suboptimal learning rules? To this end, I present a model showing how occasional selection of low-payoff strategies can, under certain conditions, enhance long-term success.

MS78-01 — Direct reciprocity under constraints: information and evolutionary dynamics

SPEAKERS Nikoleta Glynatsi (RIKEN Center for Computational Science)
AUTHORS Nikoleta Glynatsi (RIKEN Center for Computational Science)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Direct reciprocity is a central mechanism for the evolution of cooperation, yet most theoretical results rely on simplifying assumptions about information, interaction structure, and evolutionary dynamics. In this talk, I revisit direct reciprocity by analysing how relaxing these assumptions affects evolutionary outcomes. Building on recent work, I study settings in which agents have access to limited payoff information and where alternative evolutionary processes govern strategy updates. Under such constraints, the set of viable strategies changes, and classical reciprocity mechanisms can lose their effectiveness. In particular, I show that evolutionary outcomes depend sensitively on how agents update their behaviour, highlighting the role of modelling assumptions in shaping cooperation.

MS78-03 — The role of structure and mobility in the evolution of cooperation

SPEAKERS Diogo Pires (University of Copenhagen)
AUTHORS Diogo Pires (University of Copenhagen)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Human and non-human animals form structured social systems in which individuals interact preferentially within groups shaped by environmental structure, movement, and social preferences. To study how structure and mobility affect collective behaviour and the evolution of cooperation, we consider a model in which individuals move across nodes of a spatial network representing interaction sites (e.g., resource patches or social hubs). Those at the same node engage in a multiplayer game capturing conflict between individual and collective interests arising from resource production and shared use, and their behaviour evolves through selection. This work identifies community structure and conditional movement as two distinct yet complementary mechanisms, in which network structure and evolution interplay differently to sustain cooperation. First, under limited independent movement, populations form separate communities, leading to a nested evolutionary process. Although defection holds an advantage within communities, cooperation spreads effectively between them. Consequently, small communities sustain cooperation, independent of network topology. Second, under conditional (Markov) movement, individuals move based on satisfaction with current group composition. Cooperation co-evolves with high mobility, as cooperators find each other while avoiding defectors. In contrast to the first mechanism, less network degree heterogeneity and low movement costs are key drivers.

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MS78-04 — Exploiting evolutionary trade-offs in Anaplastic Large Cell Lymphoma though optimal targeted therapy

SPEAKERS Franco Pradelli (Moffitt Cancer Center)
AUTHORS Franco Pradelli (Moffitt Cancer Center)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Anaplastic Large Cell Lymphoma (ALCL) is the most common peripheral lymphoma in children, with 95% of pediatric cases driven by oncogenic ALK mutations [LW25; Bru+09]. Although ALK inhibitors initially reduce tumor burden, continuous therapy (CT) leads to natural selection of resistant populations. Critically, this process in ALCL produces a strong reduction in cell viability in drug-free conditions (a phenomenon known as ”drug addiction”), introducing an evolutionary trade-off that could be exploited with intermittent therapy (IT) [Ami+15]. However, this concept has found limited translation into clinical practice. In this work, we used patient-derived cell lines (PDCLs) and mathematical modeling to design an optimal treatment strategy based on evolutionary steering of drug resistance and addiction in ALCL. Using a 180-day dose-escalation protocol, we confirm that PDCLs evolve resistance and addiction in vitro. We leverage the measure of the dose-response curve in time to calibrate a mathematical model recapitulating this process, and we simulate over 500 possible treatment schedules. An optimal strategy combining induction (60 days CT) followed by IT extended tumor control beyond one year, whereas CT alone predicted relapse within 150 days. To account for parameter uncertainty, we generated a virtual cohort by sampling 1000 parameter sets from estimated distributions. Across this cohort, the proposed

schedule reduced tumor growth four-fold compared with CT, supporting the robustness of the strategy. In conclusion, these results illustrate how integrating experiments with evolutionary modeling can identify treatment schedules that exploit adaptive trade-offs to prolong tumor control.

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MS78-05 — Stackelberg evolutionary game theory: how to manage evolving systems

SPEAKERS Alexander Stein (Universite Libre de Bruxelles & VIB-KU Leuven)
AUTHORS Alexander Stein (Universite Libre de Bruxelles & VIB-KU Leuven)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Stackelberg evolutionary game (SEG) theory combines classical and evolutionary game theory to frame interactions between a rational leader and evolving followers. In some of these interactions, the leader wants to preserve the evolving system (e.g. fisheries management), while in others, they try to drive the system to extinction (e.g. pest control). Often the worst strategy for the leader is to adopt a constant aggressive strategy (e.g. overfishing in fisheries management or maximum tolerable dose in cancer treatment). Taking into account the ecological dynamics typically leads to better outcomes for the leader and corresponds to the Nash equilibria in game-theoretic terms. However, the leader's most profitable strategy is to anticipate and steer the eco-evolutionary dynamics, leading to the Stackelberg equilibrium of the game. In this presentation, we first introduce the concepts underlying SEG theory and follow with applications to fisheries management and cancer treatment, illustrating the benefit of taking eco-evolutionary dynamics into consideration.

MS78-06 — When policy shapes selection: anticipating evolutionary feedbacks in conservation

SPEAKERS Maria Kleshnina (Queensland University of Technology)
AUTHORS Maria Kleshnina (Queensland University of Technology)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Evolutionary change is rapid, ubiquitous, and increasingly documented across managed ecological systems. Yet management decisions are still largely formulated as if ecological systems were static, with targets focused on abundance, habitat area, or harvest rates while evolutionary responses are treated as background processes or unintended side effects. This mismatch generates predictable outcomes: resistance to control, harvest-induced life-history shifts, and trait erosion under habitat simplification. Such responses are not anomalies but consequences of altered selection pressures created by policy itself. We argue that conservation should be reframed explicitly as an evolutionary decision problem in which management actions reshape fitness landscapes and populations adapt accordingly. Whether adaptation ultimately leads to persistence (evolutionary rescue) or collapse (evolutionary suicide) depends on how trade-offs are structured and how interventions modify ecological constraints. Through conceptual and mathematical

examples, we show that policies designed to stabilise populations can inadvertently reduce evolutionary resilience, and that relocating evolutionary costs across demographic parameters can qualitatively shift system outcomes, revealing eco-evolutionary tipping points. Framing conservation as a hierarchical, leader–follower interaction between policymakers and evolving systems provides a principled way to anticipate adaptive feedbacks, identify extinction boundaries, and design interventions that balance short-term ecological stability with long-term evolutionary robustness. Integrating such eco-evolutionary foresight into conservation practice is essential for managing adaptive systems in a rapidly changing world.

MS78-07 — Evolutionary Therapy in Non-Small Cell Lung Cancer

SPEAKERS Kailas Honasoge (Delft University of Technology)
AUTHORS Kailas Honasoge (Delft University of Technology)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Resistance to targeted therapies remains a challenge in the treatment of metastatic non-small cell lung cancer (NSCLC). Standard Maximum Tolerated Dose (MTD) protocols often accelerate the competitive release of drug-resistant cell populations, leading to treatment failure. Evolutionary therapy (ET) offers an alternative approach, seeking to forestall resistance by exploiting density- and frequency-dependent competition between drug-sensitive and drug-resistant cells. The aggressive nature of NSCLC and clinical toxicity considerations motivate the exploration of dynamic dosing strategies. In this study, we explore the theoretical efficacy of various treatment strategies in NSCLC using a data-driven mathematical modeling framework. Building on two-population ordinary differential equation (ODE) models validated against longitudinal tumour-burden data from NSCLC patients treated with Osimertinib, a tyrosine kinase inhibitor used as a first-line therapy, we compare different static and dynamic dosing protocols. Specifically, we focus on adaptive protocols where drug dosages are dynamically adjusted based on relative changes in tumour volume observed between clinical assessments. We also analyze how different clinical parameters, such as inter-test intervals and baseline doses, influence the Time to Progression (TTP).

MS81 — Modeling of protein dynamics with applications to Neurodegenerative Diseases

TRACK Neuroscience and Neural Systems
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 15.12 - HS
ORGANISERS Suzanne Sindi (University of California, Merced); Laurent Pujo-Menjouet (Lyon 1 University)

Minisymposium abstract

The theme of this mini-symposium focuses on the mathematical modeling of protein dynamics in the context of neurodegenerative diseases such as Alzheimer's, Creutzfeldt–Jakob, and Parkinson's diseases. The proposed approaches span multiple biological scales, ranging from protein–protein interactions to cellular dynamics (protein–cell interactions), and up to tissue-level phenomena, including the spatial spread of pathology across brain regions and the formation of amyloid plaques. The mathematical tools discussed may include ordinary and delay differential equations, partial differential equations describing aggregation, fragmentation, diffusion, or advection processes, with various structural features such as size, spatial, or combined size–space dependencies. Data-driven approaches, including image analysis and data augmentation using neural networks, are also within the scope of this mini-symposium. Contributions from all relevant fields and at all scales are welcome. By bringing together diverse modeling strategies and methodologies, this mini-symposium aims to advance our understanding of the complex mechanisms underlying these often fatal diseases, which continue to pose many open and challenging questions.

MS81-01 — Mathematical models of brain networks: propagation of diseases and of brain activity

SPEAKERS Ashish Raj (University of California, San Francisco)
AUTHORS Ashish Raj (University of California, San Francisco); Benjamin Sipes (UCSF Dept of Radiology); Nuutti Barron (UCSF Dept of Radiology); Robin Sandell (UCSF Dept of Radiology)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

My talk will focus on developing mathematical and computational models that use the brain's structural connectivity to predict the development of neurodegenerative diseases like Alzheimer's. I will first describe our original proposal that Alzheimer's and other dementias are underpinned by misfolded pathologies that spread in the brain's structural connectome. This process can be mathematically captured by the so-called "Network Diffusion Model". Several examples from AD, ALS, Huntington's, Parkinson's, and other dementias will be demonstrated. I will then present new extensions of this model in many meaningful ways, incorporating protein aggregation, clearance, active axonal transport, and mediation by external genes, cells, and neuroinflammation. Recent work on interactions between microglia, inflammatory signals, and cytokines will be presented. Deep neural network implementations of these complex and computationally prohibitive models will be motivated, and exciting new work on physics-informed neural networks that utilize neural operator learning will be presented. I will also briefly describe recent work in modeling brain electrophysiology using similar graph spectral models. All above models centrally involve the brain's complex network Laplacian eigen-spectrum and "graph harmonics." Through this work, we have found significant differences in the model's parameters that relate healthy brains to Alzheimer's disease, sleep, epilepsy, and infant brain maturation. The related papers will be briefly highlighted.

MS81-02 — Non-Linear Dynamics and Catalytic Exchange Govern Prion Assembly and Spreading

SPEAKERS Human Rezaei (INRAE)
AUTHORS Angélique Igel (Université Paris-Saclay, INRAe, UVSQ, VIM, 78350 Jouy-en-Josas, France); Basile Fornara (Université Paris-Saclay, INRAe, UVSQ, VIM, 78350 Jouy-en-Josas, France); Davy Martin (Université Paris-Saclay, INRAe, UVSQ, VIM, 78350 Jouy-en-Josas, France); Human Rezaei (INRAE); Laurent Pujo-Menjouet (Lyon 1 University); Pierre Sibille (Université Paris-Saclay, INRAe, UVSQ, VIM, 78350 Jouy-en-Josas, France); Vincent Béringue (Université Paris-Saclay, INRAe, UVSQ, VIM, 78350 Jouy-en-Josas, France)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Prion fibril assemblies exhibit a strong structural and dynamical heterogeneity that cannot be described by classical linear polymerization models. Using single-assembly imaging and bulk kinetic measurements, we show that fibrillar and oligomeric subpopulations coexist and continuously exchange material through catalytically mediated processes. At the population level, this exchange generates spontaneous depolymerization, damped oscillations, and multi-timescale relaxation dynamics, indicating that prion assemblies evolve far from equilibrium and form a non-linear dynamical system with multiple interacting species. These experimental observations provide quantitative constraints for a minimal kinetic framework based on catalytic feedback and exchange between subpopulations. Embedded in a reaction–diffusion setting, this framework was used to explore how non-linear replication dynamics shape prion spreading in tissue. The model reveals the emergence of attractor states, strain-dependent propagation regimes, and non-trivial strain interference and co-propagation, arising solely from kinetic non-linearities and feedback rather than tissue-specific templating. Overall, this work illustrates how experimentally grounded non-linear dynamics at the assembly level can control large-scale spreading and strain behavior.

MS81-03 — Modelling Optimal Anti-Inflammatory Regimens and Outcomes in Alzheimer's Disease

SPEAKERS Wissam El Hajj (University of Rennes)
AUTHORS Laurent Pujo-Menjouet (Lyon 1 University); Léon Matar Tine; Vitaly Volpert (University Claude Bernard Lyon 1); Wissam El Hajj (University of Rennes)

SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

The application of non-steroidal anti-inflammatory drugs (NSAIDs) for Alzheimer's disease is considered to be a promising therapeutic approach. Epidemiological studies suggest potential benefits of NSAIDs; however, these findings are not consistently supported by clinical trials. This long-standing discrepancy has persisted for decades and remains a significant barrier to developing effective treatment strategies. To assess the efficacy of NSAIDs in Alzheimer's disease, we have developed a mathematical model based on a system of ordinary differential equations. The model captures the dynamics of key players in disease progression, including Aβ-monomers, oligomers, pro-inflammatory mediators (M1 microglial cells and pro-inflammatory cytokines), and anti-inflammatory mediators (M2 microglial cells and anti-inflammatory cytokines). The effects of NSAIDs are modeled through a reduction in the production rate of inflammatory cytokines (IC). While a single NSAID administration temporarily reduces IC levels, their concentration eventually returns to baseline due to drug elimination. The return time depends on the drug dose, resulting in a patient-specific return time function. By analyzing this function, we propose an optimal treatment regimen and identify conditions under which NSAID treatment is most effective in reducing IC levels. Our results suggest that NSAID efficacy in Alzheimer's disease is influenced by the stage of the disease (with earlier intervention being more effective), patient-specific parameters, and the treatment regimen.

MS81-04 — ATM-ApoE crown formation in Alzheimer's disease

SPEAKERS Felipe Olivares (Lyon 1 University)
AUTHORS Felipe Olivares (Lyon 1 University); Laurent Pujo-Menjouet (Lyon 1 University); Maxime Estavoyer (CNRS, Centre de Physique Theorique, Marseille, France); Nicolas Foray (Institut National de la Santé et de la Recherche Médicale (INSERM) U1296 Unit "Radiation: Defense, Health, Environment", Lyon, France)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Alzheimer's disease (AD) is a neurodegenerative disorder, with current therapies primarily focused on symptom management. Recent studies have proposed the Radiation-Induced ATM NucleoShuttling (RIANS) response as a mechanism that involves the formation of a perinuclear crown (PC) and may impair DNA repair. We present a mathematical framework to investigate this process. First, an ordinary differential equation (ODE) model is developed to analyze PC dynamics under constant and periodic oxidative stress. This model is used to simulate the effects of combined radiation and antioxidant/statin treatments. Second, we formulate a spatial model using partial differential equations (PDEs) that incorporates protein diffusion and advection. For this system with quadratic reactions, we prove the global existence and uniqueness of a non-negative solution, extending well-posedness results for non-autonomous reaction-diffusion-advection systems. Numerical simulations in two dimensions illustrate the behavior of the model.

MS81-05 — Modeling the Prion Aggregation Process During Polymerization Experiments Using Delay Differential Equations

SPEAKERS Theo Loureaux (University of California, Merced)
AUTHORS Human Rezaei (INRAE); Laurent Pujo-Menjouet (Lyon 1 University); Paul Lemarre (Université Claude Bernard Lyon 1, Institut Camille Jordan, Lyon, France, Department of Applied Mathematics, University of California Merced, Merced, California, United States of America, Nova In Silico, Lyon, France); Suzanne Sindi (University of California, Merced); Theo Loureaux (University of California, Merced); Thomas de Mondesir (Université Claude Bernard Lyon 1, Institut Camille Jordan, Lyon, France)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Prion proteins are notorious for their ability to induce neurodegenerative diseases by forming long fibrillar aggregates that accumulate in the brain. While the aggregation of these proteins and their fragmentation by oligomeric species are central to disease progression, the underlying mechanisms remain poorly understood. To better interpret experimental data, mathematical models have been developed to translate the key chemical reactions governing this process. In this talk, I present a novel modeling approach based on delay differential equations (DDEs), designed to capture the time-dependent features of prion polymerization dynamics. I will demonstrate how this framework aligns with experimental observations from polymerization assays in which prion monomers are thermally induced to aggregate. The model not only fits the data well but also suggests an alternative perspective on the interplay between aggregation and fragmentation, offering a new theoretical lens on prion dynamics.

MS81-06 — An optimal control problem for anti-inflammatory treatments of Alzheimer's disease

SPEAKERS Nicolás Torres Escorza (Université Côte d'Azur)
AUTHORS Emilio Molina; Laurent Pujo-Menjouet (Lyon 1 University); Nicolás Torres Escorza (Université Côte d'Azur)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

We present and analyze an optimal control problem to model anti-inflammatory treatment strategies for Alzheimer's disease, using a system of differential equations that captures interactions between ABeta-peptides, microglial cells, interleukins, and neurons. These interactions operate through mechanisms such as protein polymerization, inflammation processes, and neural stress responses. In particular, inflammation is highlighted as a key factor in the onset and progression of Alzheimer's disease, driven by a hysteresis effect related to the degradation rate d of monomers and the initial concentration of interleukins. This implies a critical inflammation threshold that determines whether the disease persists over the long term. The optimal control problem we propose seeks to minimize the concentration of toxic oligomers by modulating interleukin production and degradation rates, representing potential anti-inflammatory treatment effects. Under natural constraints on treatment dose efficacy and cumulative exposure, our goal is to assess whether it is possible to shift the system from a persistent disease state to a disease-free equilibrium. We provide the necessary conditions of the optimal solution and supplement our theoretical findings with numerical simulations, which illustrate the system's behavior under different parameter settings and the imposed constraints of the optimal control problem.

MS81-07 — Machine learning accelerated modeling of tau protein spread with the Network Transport Model

SPEAKERS Brandon Imstepf (University of California, Merced)
AUTHORS Ashish Raj (Department of Radiology and Biomedical Imaging, University of California, San Francisco); Brandon Imstepf (University of California, Merced); Emilia Cozzolino (Istituto per le Applicazioni del Calcolo, National Research Council of Italy (CNR)); Justin Torok (Radiology, University of California, San Francisco); Nuutti Barron (Department of Radiology and Biomedical Imaging, University of California, San Francisco); Suzanne Sindi (University of California, Merced); Veronica Tora (Istituto per le Applicazioni del Calcolo, National Research Council of Italy (CNR))
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Computational models provide a biophysically grounded framework to study tau protein spread in Alzheimer's disease and other tauopathies. However, model parameter inference, a key step in analyzing empirical data, is often intractable due to the slow speed and high computational burden of generating simulations.

Here, we introduce a machine learning framework to accelerate the Network Transport Model (NTM), which models tau spread on the brain's structural connectome graph. We replaced the computationally expensive graph edge partial differential equation (PDE) solver subcomponent with three classes of surrogate models: linear regression, symbolic regression, and multilayer perceptrons (MLP). The MLP

approach achieved the highest fidelity, attaining $R^2 > 0.99$ against ground-truth numerical solvers, while symbolic regression yielded nontrivial analytical relationships between tau spread and kinetic NTM parameters. Crucially, these approaches reduced simulation time from hours to minutes (approx 60x speed-up), enabling the thousands of forward simulations required for data-driven parameter inference.

MS81-08 — Study of a bi-monomeric Becker-Döring-type model

SPEAKERS Mathieu Mezache (INRAE)
AUTHORS Juan J. L. Velázquez (University of Bonn, Institute for Applied Mathematics, Endenicher Allee 60, D-53115 Bonn, Germany); Klemens Fellner (Institute of Mathematics and Scientific Computing, University of Graz, Graz 8010, Austria); Marie Doumic (Sorbonne Universités, Inria, Université Paris-Diderot, CNRS, Laboratoire Jacques-Louis Lions, Paris, France and Wolfgang Pauli Institute, C/O University of Vienna, Austria); Mathieu Mezache (INRAE)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

In order to provide an explanation for the damped oscillations surprisingly observed in Prion depolymerization experiments, a bi-monomeric variant of the seminal Becker-Döring system is proposed. In this talk, we look in detail at the mechanisms leading to these oscillations. We characterize the dynamics of the system in different kinetic phases: from the initial phase of high amplitude oscillations to progressive damping and convergence towards the unique stationary solution. This result is based on quantitative approximations of the main quantities of interest: the period of oscillations, the damping of oscillations corresponding to an energy loss, and the number of oscillation cycles characterizing each kinetic phase.

MS84 — Mathematical and Computational Ophthalmology

TRACK Miscellaneous
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 15.02 - HS, 01.22 - HS
ORGANISERS Paul A. Roberts (City St George's, University of London)

Minisymposium abstract

Mathematical Ophthalmology concerns the use of mechanistic mathematical models (e.g., ODE/PDE models) to derive insight into the mechanisms underpinning the structure and function of the eye in health, development and disease, and to predict the effects of existing and putative treatments. Computational Ophthalmology involves the application of advanced computational techniques, including artificial intelligence, to the big data being generated in ophthalmology, to identify pathology and predict disease progression. It is an exciting time in Mathematical and Computational Ophthalmology (MaCO). Though a relatively small field, we are reaching a tipping point as the importance of quantitative tools in ophthalmology becomes more widely recognised and the number of those working in the field reaches a critical mass. In this minisymposium we will showcase research in MaCO to the broader Mathematical Biology community, while building and strengthening connections and collaborations within the MaCO community.

MS84-07 — Drug Release Dynamics from a Three-Layer Composite Contact Lens

SPEAKERS Rayanne A. Luke (George Mason University)
AUTHORS Daniel M. Anderson (George Mason University); Rayanne A. Luke (George Mason University)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 15.02 - HS
TRACK Miscellaneous

Abstract

Eye drops are typically used to deliver ophthalmic drugs that treat both acute and chronic eye disorders such as glaucoma. However, drugs delivered by this method quickly leave the tear film due to blinking

and drainage. In contrast, drug-eluting contact lenses allow the drug to remain in the tear film much longer, thus serving as a potential drug delivery vehicle. Recently, such lenses have been designed by encapsulating drug-polymer films in contact lens hydrogels, where the drug-laden region takes an annular shape when viewed from the top down. Informed by in vivo data, we design a coupled partial differential equation three-layer model of contact lens drug release to mathematically investigate the effect of lens design characteristics on the time to 50% drug release (t50) in the vial, blister pack, and eye settings. In the eye setting, we incorporate our prior model that considers the effect of many blinks on the pre- and post-lens tear film drug concentrations. We simulate drug cumulative release profiles and study the variability of t50 across: (1) the ratio of the hydrogel to drug-polymer film diffusion coefficients, (2) the centerline of the polymer within the hydrogel, and (3) the polymer thickness. This work may help medical professionals better understand the mechanics of contact lens drug delivery and predict targeted tissue transport of drug.

References

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[Ros+19] Amy E Ross et al. “Topical sustained drug delivery to the retina with a drug-eluting contact lens”. In: *Biomaterials* 217 (2019), p. 119285.

MS84-01 — A mathematical model of fluid and solute transport across the corneal endothelium

SPEAKERS Federica Vanone (Gran Sasso Science Institute)

AUTHORS Federica Vanone (Gran Sasso Science Institute); Alexander J.E. Foss (Nottingham University Hospitals NHS Trust); Francesco Viola (Gran Sasso Science Institute); Rodolfo Repetto (University of Genoa); Mariia Dvoriashyna (University of Edinburgh)

SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 15.02 - HS

TRACK Miscellaneous

Abstract

The cornea is the transparent tissue in front of the eye, composed of three main layers: endothelium (a monolayer of cells towards the anterior chamber), stroma and epithelium (towards the tear film). The cornea is kept at the correct hydration, essential for its transparency, by the endothelium pumping water out of the stroma. In our previous work [Van+25], we showed that local osmosis (water flux generated by the buildup of a concentration gradient in the channel between adjacent endothelial cells, called cleft) is the main driver of the endothelial pump. In this work, we refine our model to investigate another possible mechanism hypothesized in the literature, accounting for the presence of the lactate ion [Bon12].

The model domain includes a well-mixed 0D compartment representing the cell and a 2D compartment representing the cleft. We consider 7chemical species (sodium, potassium, chloride, bicarbonate, carbon dioxide, protons, lactate) and a chemical reaction. We write equations for fluid and ion balance, accounting for membrane channels and transporters, and for the tight junction closing the cleft on the anterior chamber side. Asymptotic analysis leads to a system of algebraic equations and ODEs, solved iteratively in MATLAB. We parametrize the model by optimizing the membrane permeability to ions against literature values.

With this model, we show the link between transendothelial lactate and water flux, and we investigate the role of bicarbonate [LKB16] in their transport.

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MS84-02 — V-Cornea: A Multi-Scale In Silico Framework for Ocular Toxicology and IVIVE

SPEAKERS Joel Vanin (Indiana University Bloomington)
AUTHORS Joel Vanin (Indiana University Bloomington); Michael Getz (Indiana University Bloomington); Catherine Mahony (Procter & Gamble Technical Centre); Thomas Knudsen (Indiana University Bloomington); James Glazier (Indiana University Bloomington)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 15.02 - HS
TRACK Miscellaneous

Abstract

Predicting injury severity and time-to-recovery after ocular chemical exposure is critical for chemical classification and risk assessment. Time-resolved in vitro data from localized injury on reconstructed corneal epithelium reveal concentration-dependent lesion evolution, including secondary propagation likely driven by necrotic cell rupture, yet these assays cannot alone predict tissue-level recovery. To address this gap, we extend V-Cornea, a published multi-scale agent-based model of epithelial homeostasis and recovery from slight to mild injuries [Van+25], with stochastic Boolean networks governing cell-fate decisions between survival, apoptosis, and necrosis [CTF+10] as functions of local chemical concentration. This extended framework serves as an in silico NAM and engine for in vitro to in vivo extrapolation (IVIVE). Leveraging the model as a computational hypothesis generator, we performed systematic sweeps of concentration and exposure duration, producing phase diagrams that map exposure conditions to outcome regimes ranging from containment to catastrophic spread. Without immune-mediated debris clearance, the damage loop lacks an off-switch, mirroring in vitro observations but diverging from in vivo recovery. This divergence defines the IVIVE boundary and motivates the introduction of immune dynamics to provide the missing recovery signals, enabling extrapolation to in vivo predictions of injury severity and recovery time.

References

[CTF+10] Laurence Calzone, Laurent Tournier, Simon Fourquet, et al. "Mathematical Modelling of Cell-Fate Decision in Response to Death Receptor Engagement". In: *PLOS Computational Biology* 6.3 (2010), e1000702. DOI: 10.1371/journal.pcbi.1000702.

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MS84-05 — A coupled fluid-dynamics-heat transfer model for 3D simulations of the aqueous humor flow in the human eye

SPEAKERS Marcela Szopos (Université Paris Cité)
AUTHORS Marcela Szopos (Université Paris Cité); Thomas Saigre (Inria); Christophe Prud'homme (Université de Strasbourg); Vincent Chabannes (Université de Strasbourg)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 15.02 - HS
TRACK Miscellaneous

Abstract

Understanding the behavior of the human eye is challenging due to the complex interactions between various physical phenomena, such as heat transfer, fluid dynamics, and tissue deformation. Although medical data can offer valuable insights into ocular physiopathology, the available information can be scarce and noisy. Moreover, in experimental studies, multiple factors come into play and it is difficult to isolate the contributions of individual mechanisms. Therefore, developing a robust and accurate model for ocular applications can enhance our understanding of this complex system, by integrating governing mechanisms and data variability.

This talk presents our ongoing efforts in this direction, focusing on the thermo-fluid dynamics governing aqueous humor flow and its coupling with heat transfer throughout the eyeball [Sai+26]. Our methodology involves developing a comprehensive mathematical and computational model based on the finite element method, rigorously validated against experimental data and numerical studies. To facilitate real-time feedback, we derived a reliable reduced-order model using the certified reduced basis method. We performed forward uncertainty quantification studies with the reduced model, utilizing experimental based stochastic inputs, and conducted global sensitivity analysis to address variability and noise [SPS24]. We will illustrate these developments, discuss their applications, and address the remaining challenges.

References

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MS84-04 — Modeling the Ocular Immune Response to Seasonal Allergic Conjunctivitis and the Effects of Treatment

SPEAKERS Lucia Carichino (Rochester Institute of Technology)
AUTHORS Lucia Carichino (Rochester Institute of Technology); Kara L. Maki (Rochester Institute of Technology); Narshini D. Gunputh (Rochester Institute of Technology)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 01.22 - HS
TRACK Miscellaneous

Abstract

Seasonal allergic conjunctivitis (SAC) affects up to 40% of the population; however, the complex immune signaling processes that drive ocular inflammation are not fully understood. In this work, we present a quantitative mechanistic model of the cellular immune response during SAC. A system of 23 coupled ordinary differential equations is developed to represent interactions among allergens, immune cells, cytokines, chemokines, antibodies, and histamine in the conjunctiva.

To evaluate the model uncertainty, a local and global sensitivity analyses was conducted to identify the most influential parameters. Model validation was performed by comparing simulated cytokines concentrations to experimental cytokines concentrations in tears. Predicted histamine levels were further compared with clinical symptom scores, demonstrating a strong correlation.

The model highlights which pathways may be the most effective for therapeutic targets. We are currently working on extending the model to account for treatment of SAC using antihistamine drugs administered via eye drops or released by a pre-loaded contact lens. This modeling approach demonstrates potential applications in optimizing drug dosing, evaluating new therapeutic strategies, and predicting patient responses.

MS84-08 — Modeling the impact of venous collapsibility on retinal blood flow

SPEAKERS Tajkera Khatun (Indiana University Indianapolis)
AUTHORS Tajkera Khatun (Indiana University Indianapolis); Brendan Fry (Metropolitan State University of Denver); Brent Siesky (Icahn School of Medicine at Mount Sinai Hospital); Alice Verticchio (Icahn School of Medicine at Mount Sinai Hospital); Alon Harris (Icahn School of Medicine at Mount Sinai Hospital); Julia Arciero (Indiana University Indianapolis)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 01.22 - HS
TRACK Miscellaneous

Abstract

Glaucoma is a leading cause of blindness worldwide that is characterized by irreversible vision loss. In addition to elevated intraocular pressure (IOP), impairments in retinal blood flow and oxygenation have been shown to contribute to the progression of glaucoma. We extend our existing model of the retinal vasculature, which includes blood flow and oxygen transport mechanisms, to incorporate venous collapsibility. Venules are modeled as Starling resistors, allowing collapse when external pressure exceeds intravascular pressure. Retinal blood flow and tissue oxygenation are predicted as intraluminal pressure and IOP are varied. At baseline IOP, blood flow remains relatively constant across a physiological pressure range (autoregulation plateau). Elevated IOP shifts this plateau to higher pressures, reducing autoregulatory capacity. At elevated IOP, the oxygen extraction fraction decreases sharply as arterial pressure increases. This suggests that venous collapse alters microvascular flow distribution and limits effective oxygen utilization despite increased perfusion pressure. Ultimately, the inclusion of venous collapsibility in the model yields more accurate predictions of the impact of IOP on autoregulation and hemodynamic dysfunction in glaucoma. This model framework will allow for future comparisons to sectorial-specific clinical data to assess the role of impaired blood flow regulation in ocular disease.

References

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MS84-03 — Untangling abnormal retinal blood vessels: Integrating computational modelling, image analysis and in vivo imaging

SPEAKERS Katie Bentley (The Francis Crick Institute and Kings College London)
AUTHORS Katie Bentley (The Francis Crick Institute and Kings College London); Alexandru Toma (The Francis Crick Institute, London); Markus Koerbel (The Francis Crick Institute, London and EMBL Heidelberg); Zuzanna Dzieniak (UCL Institute of Ophthalmology and Moorfields Eye Hospital); Andreia Pena (Catolica Biomedical Research Centre, Lisbon); Markus Fruttiger (UCL Institute of Ophthalmology and Moorfields Eye Hospital); Marinko Sarunic (UCL Institute of Ophthalmology and Moorfields Eye Hospital); Cathy Egan (UCL Institute of Ophthalmology and Moorfields Eye Hospital); Senth Selvam (UCL Institute of Ophthalmology and Moorfields Eye Hospital); Claudio Franco (Catolica Biomedical Research Centre, Lisbon); Colin Chu (UCL Institute of Ophthalmology and Moorfields Eye Hospital)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 01.22 - HS
TRACK Miscellaneous

Abstract

Abnormal retinal neovascularisation is a hallmark of many retinopathies, including diabetic retinopathy (DR), Age-related macular degeneration (AMD) and retinopathy of prematurity (ROP). It is still unclear how the altered conditions in these diseases, drive cells to construct these vessels, which exacerbate disease progression and in some cases sight loss. Current therapies focus on anti-vegf or laser to limit further damage, but complications, risks and a large proportion of poor responders mean greater understanding of the disease mechanism is required to reveal new strategies.

We are developing integrated computational methods to integrate with state-of-the-art retinal imaging modalities in preclinical mouse, patient and post-mortem human eyes to reveal the dynamic mechanisms governing abnormal vessel growth towards identifying new/adapted therapeutic strategies to improve patient outcomes.

I will give an overview of our tools development and insights to date on 1) our 3D image analysis tool to quantify abnormal retinal vessel and cell-scale morphology and signalling patterns and 2) our predictive cell-based mechanistic simulations integrating with imaging at Moorfields Eye Hospital.

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MS84-06 — Mathematical modelling to explore the role of metabolic dysfunction in retinal disease

SPEAKERS Paul A. Roberts (City St George’s, University of London)
AUTHORS Paul A. Roberts (City St George’s, University of London); James B. Hurley (The University of Washington); Philip J. Luthert (University College London); François Paquet-Durand (University of Tübingen)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 01.22 - HS
TRACK Miscellaneous

Abstract

The retina is the light-detecting tissue layer which lines the back of the eye. There is an increasing awareness of the central importance of metabolic dysfunction in driving a range of currently incurable blinding retinal conditions. The retinal metabolic network is highly nonlinear with multiple feedback mechanisms, necessitating a mathematical modelling approach to accompany ongoing experimental studies for full understanding. We developed a novel mathematical model of retinal metabolism to explore how the retina maintains healthy metabolic homeostasis and how this breaks down in disease.

An ordinary differential equation (ODE) model was formulated to describe and predict the evolving concentrations over time of respiratory metabolites in outer retinal cells. The model accounts for the passage and transformation of metabolites through key respiratory pathways. The model was parameterised using data from the literature and through fitting to data from ongoing experimental studies. The full model was solved computationally, while reduced models were solved analytically.

Our model demonstrates behaviour in agreement with experimental studies, showing metabolites moving through the metabolic pathways in the established fashion. Further, simulations predict key metabolites and processes for maintaining metabolic homeostasis, indicating potential treatment strategies where this breaks down in disease.

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MS94 — Mathematical Foundations of Biochemical Computing

TRACK Systems Biology and Biochemical Networks
SCHEDULE Tuesday, 14 July 2026; 15:00–18:00; 11.02 - HS
ORGANISERS Badal Joshi (California State University San Marcos); Jinsu Kim (Pohang University of Science and Technology); Tung Nguyen (University of California Los Angeles)

Minisymposium abstract

Biomolecules can be repurposed to build programmable chemical computers, either in vitro or in living cells. A particularly promising technology is DNA strand displacement, which has been shown capable of implementing arbitrary systems of chemical reactions. Since chemical reactions can perform computation, this raises fundamental mathematical questions: What types of computation can chemistry-based dynamical systems perform, and with what efficiency, reliability, and robustness?

This minisymposium showcases recent research breakthroughs contributing to the theoretical foundations of this emerging field. Speakers will discuss novel chemical modules designed for diverse computational tasks, including fundamental arithmetic operations, transcendental functions, parameter search, and machine learning algorithms. The talks will address key theoretical questions such as computability, computational speed, and programmability within the constraints of emerging technologies and available resources. Through the minisymposium and related discussions, we aim to foster collaboration within a community of mathematicians and experimentalists working at the interface of chemistry and computation.

MS94-01 — Computing with reaction networks at input-independent speed

SPEAKERS Badal Joshi (California State University San Marcos)
AUTHORS Badal Joshi (California State University San Marcos); David Anderson (University of Wisconsin-Madison); Tung Nguyen (UCLA)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Can a test tube compute? And if so, can it compute with the same efficiency over a wide range of inputs? Recent advances in synthetic biology have made it possible to deploy chemical reactions that implement computation inside a cell. Chemical reactions function as analog computers — they are ideally suited for solving differential equations and interface naturally with real-world analog signals. However, exact arithmetic on an analog computer presents novel challenges: in existing approaches the computation time can depend on the input, in some cases resulting in impractically slow computation.

We will discuss novel algorithms that address this issue. First, we present algorithms that perform arithmetic operations — identification, inversion, addition, multiplication, absolute difference, rectified subtraction, and nth roots — at input-independent speed, with the striking property that computation time does not scale with the number of elementary steps, a consequence of the inherently parallel nature of chemical computation. We then extend this program to transcendental functions, constructing reaction network modules for the exponential and logarithm that achieve arbitrary accuracy at input-independent speed without relying on truncated power series — the logarithm presenting significantly deeper mathematical challenges. Finally, we present a fractional (ratio) representation of numbers under which arithmetic becomes instantaneous — the encoded output is correct at every positive time.

MS94-02 — Molecular Machines and the EM algorithm

SPEAKERS Carsten Wiuf (University of Copenhagen)
AUTHORS Abhinav Singh (UM-DAE Centre for Excellence in Basic Sciences); Abhishek Behera (Indian Institute of Technology Bombay); Carsten Wiuf (University of Copenhagen); Manoj Gopalkrishnan (Indian Institute of Technology Bombay)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

The implementation of abstract dynamical systems with molecular systems has gained scientific interest. Automated theoretical schemes can compile formal reaction networks into DNA oligonucleotide sequences; thereby providing a potential molecular implementation of the dynamics of the formal reaction network. In this context, we propose a novel algorithm for learning parameters of Hidden Markov Model (HMM), a flexible statistical framework widely used in Bioinformatics, Machine Learning and Data Science to model an underlying hidden structure.

Our algorithm is specified by a network of chemical reactions, and mimics the Baum-Welch algorithm which is the standard learning algorithm for HMMs. The Baum-Welch algorithm is an iterative Expectation-Maximization (EM) algorithm where one step is performed at a time in a prescribed sequence. The reaction network scheme is divided into four subnetworks that correspond to the forward, backward, expectation, and maximization steps of the Baum-Welch algorithm. Each subnetwork describes a system of ordinary differential equations that might be run separately, exactly mimicking the steps of the Baum-Welch algorithm, or simultaneously, thereby obtaining a variant on the Baum-Welch algorithm.

Promising areas of application of this work come from cellular biology. In the future, a molecule-based HMM device might learn a molecular environment within an organism by sensing and interacting with the environment at the molecular level. It might take action according to the learning outcome, for example, choosing among different drug options, or a molecule-based HMM might be used as a building block in an artificial cell or population of cells, enabling cooperative behavior among cells or facilitating various tasks.

MS94-04 — Analog computation with transcriptional networks

SPEAKERS David Soloveichik (University of Texas at Austin)
AUTHORS David Soloveichik (University of Texas at Austin)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Transcriptional networks represent one of the most extensively studied types of reaction networks in synthetic biology. While transcriptional networks typically rely on cooperativity and highly non-linear behavior of transcription factors to regulate production of proteins, they are often modeled with simple linear degradation terms. In contrast, general analog computation requires both non-linear positive as well as negative terms, seemingly necessitating control over not just transcriptional (i.e., production) regulation but also the degradation rates of transcription factors. Surprisingly, we prove that controlling transcription

factor production (i.e., transcription rate) without explicitly controlling degradation is mathematically complete for analog computation, achieving equivalent capabilities to systems where both production and degradation are programmable. We demonstrate our approach on several examples including oscillatory and chaotic dynamics, analog sorting, memory, PID controller, and analog extremum seeking. Our results provide a systematic methodology for engineering novel analog dynamics using synthetic transcriptional networks without the added complexity of degradation control and informs our understanding of the capabilities of natural transcriptional networks.

MS94-05 — Limits of Equilibrium Computation in DNA Seesaw and Dimerization Networks

SPEAKERS Ho-Lin Chen (National Taiwan University)
AUTHORS Erik Winfree (California Institute of Technology); Ho-Lin Chen (National Taiwan University)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

DNA strand displacement systems have enabled the construction of molecular circuits capable of performing complex computations, typically relying on irreversible reactions and non-equilibrium dynamics for signal amplification and restoration. An alternative paradigm is equilibrium computation, where outputs are determined by the steady-state concentrations of molecular species.

In this talk, we investigate the computational limits of equilibrium seesaw networks. We prove that, under constraints on species concentrations, the influence of an input signal decays exponentially with its distance from the input. This result implies that signal amplification is impossible and that large-scale equilibrium circuits inherently suffer from vanishing signal propagation. Extending our analysis, we connect seesaw networks to broader classes of equilibrium systems, including ligand–receptor and dimerization networks, and discuss how similar limitations arise in these models.

Overall, our results identify a fundamental barrier to scalable equilibrium computation in molecular systems, highlighting the necessity of non-equilibrium mechanisms or carefully engineered regimes for robust information processing at scale.

MS94-06 — Designing optimal computational networks: a case study from maximum likelihood estimation

SPEAKERS Oskar Henriksson (Max Planck Institute)
AUTHORS Carlos Améndola (Technical University of Berlin); Jose Israel Rodriguez (University of Wisconsin–Madison); Oskar Henriksson (Max Planck Institute); Polly Yu (University of Illinois Urbana-Champaign)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

A fundamental question in the field of molecular computation is what computational tasks biochemical systems are capable of carrying out. In this talk, we will see that chemical reaction networks can do maximum likelihood estimation of log-affine models in the following sense: Given a basis for the kernel of the design matrix of a given model, we construct a detailed-balanced network such that the MLE can be read off from the unique equilibrium when the initial concentrations are set to the observed distribution. Interestingly, the choice of basis for the kernel in the construction has a large influence on the dynamical properties and chemical complexity of the network. The desire to make this choice ”optimally” (in a number of different senses of the word) leads to several interesting questions at the crossroads between dynamics, chemistry, statistics, and algebra. This is based on the paper [Hen+25].

References

[Hen+25] Oskar Henriksson et al. “Maximum likelihood estimation of log-affine models using detailed-balanced reaction networks”. English. In: *J. Math. Biol.* 91.4 (2025), p. 30. ISSN: 0303-6812. DOI: 10.1007/s00285-025-02262-5.

MS94-07 — Recurrent neural chemical reaction networks: a versatile way of generating complex dynamics in chemical systems

SPEAKERS Tom Ouldridge (Imperial College)
AUTHORS Alexander Dack (Imperial College London); Benjamin Qureshi (Imperial College London); Tom Ouldridge (Imperial College); Tomislav Plesa (University of Cambridge)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

A common goal in the theory of Chemical Reaction Networks (CRNs) is to design systems that reproduce or approximate a desired dynamics. This theory supports ongoing efforts in synthetic biology and molecular nanotechnology to emulate the functional molecular networks seen in nature. Here, we propose a molecular version of a recurrent artificial neural network, the RNCRN, which we prove is able to approximate arbitrary dynamics, and demonstrate that functionality for systems that exhibit multi-stability, oscillations and chaos. The neural net nature of the RNCRN means that it is particularly well suited to extrapolating a CRN, defined for all concentrations of the executive species, from a relatively small region of defined target behaviour. We show that this property allows the RNCRN to approximate exotic limit cycles that are not easily expressed as an attractor of a simple dynamical system. Similarly, the RNCRN can interpolate between two defined regions of qualitatively different target behaviour, automatically generating an appropriate bifurcation.

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MS102 — Reaction Networks in Modern Mathematical Biology

TRACK Systems Biology and Biochemical Networks
SCHEDULE Tuesday, 14 July 2026; 15:00–18:20; 11.03 - HS
ORGANISERS Stefan Müller (University of Vienna, Faculty of Mathematics); Wasiur Rahman Khuda Bukhsh (University of Nottingham)

Minisymposium abstract

Reaction networks form a foundational mathematical framework for modelling complex biological systems, from intracellular biochemical interactions to multiscale dynamics at the ecological scale. Recent advances in stochastic and deterministic theory, nonequilibrium thermodynamics, and multiscale analysis have significantly deepened our understanding of noise-driven phenomena, biochemical regulation, and energetic constraints in living systems. This mini-symposium gathers leading experts to present recent theoretical and applied developments that highlight how reaction network models [MFS22] inform core questions in modern mathematical biology. Topics include applications to (population) genetics [BB21], systems biology [Lim+25], chemical graph theory, and thermodynamics [AFE23]. By bringing together complementary perspectives from probability, dynamical systems, statistical physics, and biological modelling, the session will showcase how reaction network theory continues to shape modern mathematical biology and foster new insights into biological complexity.

Keynote speaker: Ellen Baake (University of Bielefeld)

References

[AFE23] Francesco Avanzini, Nahuel Freitas, and Massimiliano Esposito. "Circuit Theory for Chemical Reaction Networks". In: *Physical Review X* 13.2 (June 2023). ISSN: 2160-3308. DOI: 10.1103/physrevx.13.021041. URL: http://dx.doi.org/10.1103/PhysRevX.13.021041.

[BB21] Ellen Baake and Michael Baake. "Ancestral lines under recombination". In: *Probabilistic structures in evolution*. EMS Ser. Congr. Rep. EMS Press, Berlin, [2021] ©2021, pp. 365–382. ISBN: 978-3-98547-005-1. DOI: 10.4171/ECR/17-1/17. URL: https://doi.org/10.4171/ECR/17-1/17.

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MS102-04 — Thermodynamics of Chemical Reaction Networks: From Circuits to Metabolism

SPEAKERS Massimiliano Esposito (University of Luxembourg)
AUTHORS Massimiliano Esposito (University of Luxembourg)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

After a brief overview of the thermodynamic framework for open chemical reaction networks (CRNs) [RE16], I will introduce a circuit-theoretic approach that provides thermodynamically consistent schemes for coarse-graining CRNs [AFE23]. I will then illustrate how this framework can be used to analyse energy transduction in metabolic networks, emphasising how network structure constrains pathways of free-energy conversion and shapes their efficiency and regulation in biological systems [WRE22; BE25b; BE25a].

References

[AFE23] Francesco Avanzini, Nahuel Freitas, and Massimiliano Esposito. "Circuit Theory for Chemical Reaction Networks". In: *Physical Review X* 13.2 (June 2023). ISSN: 2160-3308. DOI: 10.1103/physrevx.13.021041. URL: http://dx.doi.org/10.1103/PhysRevX.13.021041.

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MS102-05 — Hypergraph-based models of random chemical reaction networks: Conservation laws, connectivity, and percolation

SPEAKERS Nahuel Freitas (University of Buenos Aires)
AUTHORS Massimiliano Esposito; Nahuel Freitas (University of Buenos Aires); Shesha Gopal Marehalli Srinivas
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Random graph models have been instrumental in characterizing complex networks, but chemical reaction networks (CRNs) are better represented as hypergraphs. Traditional models of random CRNs often reduce CRNs to bipartite graphs, representing species and reactions as distinct nodes, or simpler derived graphs, which can obscure the relationship between the statistical properties of these representations and the physical characteristics of the CRN. We introduce a straightforward model for generating random CRNs that preserve their hypergraph structure and atomic composition, enabling the direct study of chemically relevant features. Notably, our approach distinguishes two notions of connectivity that are equivalent in graphs but differ fundamentally in hypergraphs. These notions exhibit percolation-like phase transitions, which we analyze in detail. The first type of connectivity has relevance to steady-state synthesis and transduction, determining the effective reactions an open CRN can perform at steady state. The second type is suitable to identify which species can be produced from a given initial set of species in a closed CRN. Our findings highlight the importance of hypergraph-based modeling for uncovering the complex behaviors of CRNs.

MS102-06 — Positive algebraic geometry for mathematical biology

SPEAKERS Stefan Müller (University of Vienna, Faculty of Mathematics)
AUTHORS Abhishek Deshpande (Indian Institute of Technology, Hyderabad); Georg Regensburger (Johannes Kepler University Linz); Stefan Müller (University of Vienna, Faculty of Mathematics)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Power-law dynamical systems are widely used as models in chemistry and biology (e.g., in ecology and epidemiology), as well as in economics and engineering. We study positive solutions to parametrized systems of generalized polynomial equations (with real exponents) in abstract terms. In particular, we identify the relevant geometric objects: the coefficient polytope, the monomial difference, and the monomial dependency subspaces.

Using only linear algebra and polyhedral geometry, we rewrite these polynomial equations and inequalities in terms of binomial equations. The resulting solution sets are then studied using analytical methods; for example, we characterize unique existence for all parameters using Hadamard's theorem. Furthermore, we revisit mass-action systems that are decomposable and essentially univariate, providing an extension of the classical deficiency one theorem.

MS102-07 — Widespread biochemical reaction networks enable Turing patterns without imposed feedback

SPEAKERS Tian Hong (UT Dallas)
AUTHORS Shibashis Paul (National Institute Of Technology Sikkim, India); Tian Hong (UT Dallas)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Understanding self-organized pattern formation is fundamental to biology. In 1952, Alan Turing proposed a pattern-enabling mechanism in reaction-diffusion systems containing chemical species later conceptualized as activators and inhibitors that are involved in feedback loops. However, identifying pattern-enabling

regulatory systems with the concept of feedback loops has been a long-standing challenge. To date, very few pattern-enabling circuits have been discovered experimentally. This is in stark contrast to ubiquitous periodic patterns and symmetry in biology. In this work, we systematically study Turing patterns in 23 elementary biochemical networks without assigning any activator or inhibitor. These mass action models describe post-synthesis interactions applicable to most proteins and RNAs in multicellular organisms. Strikingly, we find ten simple reaction networks capable of generating Turing patterns. While these network models are consistent with Turing's theory mathematically, there is no apparent connection between them and commonly used activator-feedback intuition. Instead, we identify a unifying network motif that enables Turing patterns via regulated degradation pathways with flexible diffusion rate constants of individual molecules. Our work reveals widespread biochemical systems for pattern formation, and it provides an alternative approach to tackle the challenge of identifying pattern-enabling biological systems.

MS102-02 — Genetic recombination, deterministic reaction systems, stochastic partitioning, and Markov embedding

SPEAKERS Ellen Baake (Bielefeld University)
AUTHORS Ellen Baake (Bielefeld University); Michael Baake (Bielefeld University)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

We consider the system of differential equations describing genetic recombination, which is well known to be equivalent to the law of mass action of a chemical reaction network [MH15; Alb21]. It is equally well known that this nonlinear system can be solved via its dual process backward in time (see [BB21] for a review). This dual process is a (linear) Markov chain in continuous time, which describes how an individual's genes are partitioned across its ancestors when looking back into the past.

We find an explicit representation of the semigroup of the partitioning process, and thus an explicit solution of the recombination equation, both for the simple case of single crossovers and for general recombination distributions. The latter works by representing the realisations of the partitioning process as trees and establishing an inclusion-exclusion principle for the decomposition of these trees into subtrees.

Based on the semigroup, we attack the embedding problem for recombination in discrete time, where the partitioning is described by a discrete-time Markov chain. The embedding problem of Markov transition matrices into Markov semigroups is a classic problem that goes back to Kingman [Kin62]; the question is whether a discrete-time Markov chain may be represented as the semigroup of a continuous-time Markov process. We solve the problem for short gene sequences and give an outlook to the general case.

References

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[Kin62] J. F. C. Kingman. "The imbedding problem for finite Markov chains". In: *Z. Wahrscheinlichkeitsth. verw. Geb.* 1 (1962), pp. 14–24.

[MH15] S. Müller and J. Hofbauer. "Genetic recombination as a chemical reaction network". In: *Math. Model. Nat. Phenom.* 10 (2015), pp. 84–99.

MS102-01 — A Graph grammar view on chemical reaction networks

SPEAKERS Christoph Flamm (University of Vienna)
AUTHORS Christoph Flamm (University of Vienna)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

I will introduce a graph grammar-based formalism to handle (bio)chemical reaction networks. The formalism provides a set of powerful techniques to specify, construct and analyze chemical reaction spaces. A chemical space is defined by a set of compounds and a ”reaction chemistry” defined as a collection of graph transformation rules. This algebraic model of chemical transformation in combination with mathematical optimization techniques makes it possible to explore the bio-synthetic design space in a systematic manner. Questions such as ”Does a specified chemical space harbor multiple, possibly competing, routes to a target molecule or harbors reaction motifs of interest?” is rephrased in the mathematical language of integer hyperflows on hyper-graphs offering an efficient way to identify functional networks in the chemical space, such as auto-catalysis, that conform to a formal flow specification. I will briefly touch the question of randomization of chemical reaction networks, and what needs to be preserved to remain during randomization in the realm of chemical reaction networks. If appropriate thermodynamic and kinetic parameterization is available, questions concerning the cost of maintaining pathways that run at a constant throughput can be asked. Finally, rule-base stochastic simulations of closed and open reactive system can be performed, allowing to discover mechanistic ideas how an event of interest, e.g. the construction of a particular molecule is achieved by the concurrent dynamic system.

MS102-03 — From Mean Control to Variability Control in Biological Systems

SPEAKERS JaeKyoung Kim (KAIST)
AUTHORS JaeKyoung Kim (KAIST)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Biological systems must maintain stable functions despite environmental perturbations and intrinsic molecular noise. Robust perfect adaptation (RPA) enables biological networks to restore their output to a desired level after disturbances. While antithetic integral feedback can guarantee adaptation of the mean output, it often amplifies stochastic fluctuations and therefore fails to control single-cell variability. In this talk, I present a mathematical framework that achieves noise-robust perfect adaptation, enabling simultaneous control of both the mean and variability of molecular outputs. The key idea is to combine a classical mean controller with a newly developed noise controller that regulates the second moment of the output species. This architecture allows stochastic biochemical networks to maintain both their mean and noise levels even after perturbations. We demonstrate how this framework stabilizes gene-expression dynamics and reduces failure rates in a model of the DNA damage response in E. coli. These results illustrate how mathematical modeling and stochastic control theory can provide new principles for regulating cellular variability in biological systems.

MS139 — Computational and Theoretical Advances in cfDNA and ctDNA Modeling

TRACK Mathematical Oncology
SCHEDULE Tuesday, 14 July 2026; 15:00–16:20; 15.04 - HS
ORGANISERS Madhav Nair (Texas A&M University); Pujan Shrestha (Texas A&M University)

Minisymposium abstract

Circulating tumor DNA (ctDNA) and cell-free DNA (cfDNA) are increasingly used to monitor tumor burden, treatment response, and therapeutic resistance. A central challenge, however, is that liquid

biopsy signals reflect multiple biological processes operating across scales, including stochastic biomarker shedding, clearance dynamics, and coupling to tumor–immune interactions. As a result, linking measured ctDNA and cfDNA trajectories to latent tumor states benefits from explicit mathematical structure.

This minisymposium brings together complementary modeling approaches that address these challenges using tools from stochastic processes, dynamical systems, and computational inference. The talks span hybrid ODE–SDE frameworks connecting tumor–immune dynamics to stochastic ctDNA release, mechanistic models of cfDNA fragmentation and clearance that shape observable signal distributions, and joint models of tumor kinetics and cell-free DNA dynamics developed to study and predict immunotherapy resistance. Disease-specific applications are also presented, including ctDNA modeling in HPV-associated solid tumors calibrated to longitudinal clinical data to track progression and therapeutic response.

Collectively, these contributions highlight how mathematically grounded, mechanistic models can improve quantitative interpretation of liquid biopsy data and help bridge biological complexity and clinical observables in translational oncology.

MS139-01 — Mechanistic modeling of joint size-dependent cell-free DNA concentrations and tumor kinetics for immunotherapy resistance prediction

SPEAKERS Linh Nguyen Phuong (INRIA)

AUTHORS Audrey Boutonnet (Adelis Technologies, Labège, France); Frédéric Fina (ID-Solutions Oncology); Frédéric Ginot (Adelis Technologies, Labège, France); Jean-Laurent Deville (Assistance Publique-Hôpitaux de Marseille, Timone Hospital, Aix Marseille University, Marseille, France); Laurent Greillier (Centre Inria d'Université Côte d'Azur, Computational pharmacology and clinical Oncology Department); Linh Nguyen Phuong (INRIA); Pascale Tomasini (Multidisciplinary Oncology & Therapeutic Innovations Department, Assistance Publique-Hôpitaux de Marseille, Nord Hospital, Aix Marseille University, Marseille, France); Sébastien Benzekry (COMPutational pharmacology and clinical Oncology, Centre Inria d'Université Côte d'Azur, Marseille, France); Sébastien Salas (COMPutational pharmacology and clinical Oncology, Centre Inria d'Université Côte d'Azur, Marseille, France)

SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 15.04 - HS

TRACK Mathematical Oncology

Abstract

Plasma cell-free DNA (cfDNA) shows promise as a predictive biomarker of treatment resistance in cancer patients. However, the mechanisms governing its production, fragmentation, elimination, and their relationships with tumor burden and disease progression, remain poorly understood. We developed a mechanistic model jointly describing the dynamics of short (75-<580 bp) and long (≥580-1650 bp) cfDNA fragments alongside tumor kinetics in 112 advanced cancer patients receiving immune checkpoint inhibition, using a population modeling approach, to characterize inter-individual variability in cfDNA kinetic parameters. The model captured complex longitudinal cfDNA patterns, including early treatment-associated spikes. Analysis revealed substantial inter-patient variability and demonstrated a 7.4-fold higher shedding rate for short versus long fragments. A model-derived parameter estimated at 6 weeks of treatment—reflecting increased release or reduced elimination of short fragments—was significantly associated with progression-free survival (PFS, HR=1.6, 95% CI: 1.2–2.2, $p = 0.001$). Incorporating this parameter to baseline clinical variables significantly improved PFS prediction (C-index: from 0.78 [95% CI: 0.73-0.89] to 0.80 [95% CI: 0.74-0.90], $p < 0.0001$). This framework provides quantitative insights into cfDNA biology and offers a noninvasive approach for early prediction of treatment resistance, with implications for adaptive therapeutic strategies from longitudinal liquid biopsy.

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MS139-02 — ctDNA precedes imaging: A predictive model for real-time treatment adaptation in HPV-associated anal squamous cell carcinoma

SPEAKERS Phebe Havor (Moffitt Cancer Center)
AUTHORS Brandon Huffman; James Cleary; Jamie Jabalee; Phebe Havor (Moffitt Cancer Center); Renee Brady-Nicholls
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Background: Real-time treatment response assessment in HPV-associated anal squamous cell carcinoma (ASCC) remains challenging. Traditional tumor volume measurements require serial imaging that is costly, time-intensive, and delays clinical decisions. Circulating tumor DNA (ctDNA) offers a more accessible, real-time alternative biomarker, yet its predictive value for guiding treatment adaptation remains undefined. Methods: We developed a mechanistic mathematical model of tumor volume-ctDNA dynamics using longitudinal data from 32 HPV-associated ASCC patients receiving pembrolizumab (every 3 weeks, up to 2 years). The model was fit across three scenarios: simultaneous measurements (8 patients), volume preceding ctDNA (14 patients), and ctDNA preceding volume (2 patients). Results: ctDNA showed strong positive correlation with tumor burden and predicted clinical response within 4 weeks of treatment initiation. ctDNA kinetics preceded volume changes in multiple patients, providing early response signal before imaging confirmation. The model robustly captured heterogeneous patient dynamics across all scenarios. Conclusions: ctDNA functions as a leading indicator biomarker for early treatment response identification in HPV-associated ASCC. Our framework translates this biomarker into actionable clinical predictions for real-time treatment escalation, maintenance, or de-escalation—replacing burdensome imaging with accessible blood-based monitoring, particularly impactful for underserved populations. Future studies will prospectively validate this model for personalized, adaptive treatment strategies.

MS139-03 — Coupled SDE-ODE Modeling of Tumor-Immune Dynamics to Infer Biomarker Release

SPEAKERS Pujan Shrestha (Texas A&M University)
AUTHORS Jason T. George (Department of Biomedical Engineering, Texas A&M University and Translational Medical Sciences, Texas A&M Health Science Center and Center for Theoretical Biological Physics, Rice University and Hematopoietic Biology and Malignancy, MD Anderson Cancer Center); Pujan Shrestha (Texas A&M University); Yijia Fan (Department of Biomedical Engineering, Texas A&M University, College Station, TX 77843; Translational Medical Sciences, Texas A&M Health Science Center, Houston, TX 77030)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Tumor–immune interactions are central to cancer progression and treatment response, driving cell death through immune-mediated killing and resource-limited competition. In early-stage disease or following effective treatment, cancer populations are often small and difficult to observe directly. Disease monitoring therefore relies on biomarkers such as circulating tumor DNA (ctDNA) as noisy proxies for tumor size. Existing approaches lack robust frameworks to infer tumor burden from these signals near detection thresholds.

We present a coupled deterministic–stochastic framework linking tumor–immune dynamics to biomarker release. A two-prey, one-predator Lotka–Volterra model captures interactions between immune cells and competing tumor subpopulations under shared resource constraints. Biomarker production is modeled via stochastic differential equations driven by tumor cell death from immune-mediated apoptosis and competition-induced necrosis. We incorporate both square-root (CIR-type) noise, capturing count-limited

fluctuations near detection, and multiplicative (geometric-type) noise, representing proportional variability at higher concentrations. We derive analytical expressions for biomarker trajectories and first-passage statistics, including mean detection times. Our results show how tumor heterogeneity, immune pressure, and stochastic variance structure jointly shape biomarker detectability.

MS139-04 — Linking cfDNA clearance to fragment length with mechanistic models

SPEAKERS Thomas Rachman (Carnegie Mellon University)

AUTHORS Adrian V. Lee (Women's Cancer Research Center, UPMC Hillman Cancer Center, Pittsburgh, PA, USA; Department of Pharmacology and Chemical Biology, University of Pittsburgh, Pittsburgh PA, USA; Institute for Precision Medicine, University of Pittsburgh and UPMC, Pittsburgh, PA); Daisong Liu (Women's Cancer Research Center, UPMC Hillman Cancer Center, Pittsburgh, PA, USA; School of Medicine, Tsinghua Medicine, Tsinghua University, Beijing China); David L. Bartlett (Allegheny Health Network Cancer Institute, Pittsburgh, PA, USA); Julia Foldi (Women's Cancer Research Center, UPMC Hillman Cancer Center, Pittsburgh, PA, USA); Marija Balic (Women's Cancer Research Center, UPMC Hillman Cancer Center, Pittsburgh, PA, USA); Oana Carja (Ray and Stephanie Lane Department of Computational Biology, School of Computer Science, Carnegie Mellon University,Pittsburgh, PA, USA); Patrick L. Wagner (Allegheny Health Network Cancer Institute, Pittsburgh, PA, USA); Patti Petrosko (Allegheny Health Network Cancer Institute, Pittsburgh, PA, USA); Phillip H. Gallo (Allegheny Health Network Cancer Institute, Pittsburgh, PA, USA); Rahul Kumar (Women's Cancer Research Center, UPMC Hillman Cancer Center, Pittsburgh, PA, USA; Department of Pharmacology and Chemical Biology, University of Pittsburgh, Pittsburgh PA, USA); Russell Schwartz (Ray and Stephanie Lane Department of Computational Biology, School of Computer Science, Carnegie Mellon University,Pittsburgh, PA, USA); Steffi Oesterreich (Women's Cancer Research Center, UPMC Hillman Cancer Center, Pittsburgh, PA, USA; Department of Pharmacology and Chemical Biology, University of Pittsburgh, Pittsburgh PA, USA; Institute for Precision Medicine, University of Pittsburgh and UPMC, Pittsburgh, PA); Thomas Rachman (Carnegie Mellon University); William A. LaFramboise (Allegheny Health Network Cancer Institute, Pittsburgh, PA, USA)

SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 15.04 - HS

TRACK Mathematical Oncology

Abstract

Liquid biopsy studies consistently report both elevated circulating cell-free DNA (cfDNA) concentrations and shortened fragment lengths in cancer. These features are often attributed to tumor-specific processes, despite tumor-derived cfDNA frequently constituting less than 1% of the total. Here, we consider an alternative explanation: Saturation of cfDNA clearance, which prolongs cfDNA circulation time, increases exposure to plasma nucleases, causing fragments to shorten. By combining a mechanistic model of cfDNA fragmentation with analyses of two independent cancer patient cohorts, and publicly available clearance-perturbation experiments, we show that elevated cfDNA levels in cancer patients are accompanied by a characteristic leftward shift in fragment length distributions consistent with impaired hepatic clearance. This signature is highly correlated with cfDNA concentration, independent of circulating tumor DNA (ctDNA) fraction, and independently prognostic of patient survival. In contrast to hepatic clearance reduction, DNA-protecting antibodies cause a systematic rise in short cfDNA fragments. Our model allows us to directly identify the parameter governing fragmentation kinetics and predict the effect of antibody priming on cfDNA concentration. Together, these results identify saturating clearance as a central determinant of cfDNA abundance and fragment length. More broadly, they highlight the value of mechanistic modeling of clearance processes in extracting clinically meaningful signals from cfDNA fragmentation data.

MS160 — Stochastic Modelling for Inference with Gene Expression data: Methods and Applications

TRACK Systems Biology and Biochemical Networks

SCHEDULE Tuesday, 14 July 2026; 15:00–18:00; 11.01 - HS

ORGANISERS Andrew Nicoll (University of Edinburgh); Muhan Ma (University of Edinburgh)

Minisymposium abstract

Biological functions emerge from biomolecular interactions that are inherently stochastic, heterogeneous across cells, and only partially observable. This has motivated the development of stochastic models in mathematical biology that link mechanistic hypotheses to data and enable principled inference and prediction. In the context of gene expression, modern single-cell and live-cell measurement technologies have made this stochastic variability explicit, highlighting gene expression as a noisy and variable process governed by underlying biochemical reaction networks.

This mini-symposium brings together recent work on stochastic modelling and computational approaches for inference with gene expression data, spanning methodological developments [Pop+26], data-driven case studies, and complementary techniques such as stochastic simulation, differential equation–based models, and machine-learning-assisted inference [MG25]. We encourage contributions addressing inference from stochastic dynamics—including parameter estimation, uncertainty quantification, model selection, and identifiability—and examining how model structure, data resolution, and computational constraints shape what can be learned from experiments [FGP25]. By connecting theory, computation, and data, this symposium aims to explore how current and developing methods can be used to infer interpretable and biologically meaningful gene expression dynamics from experimentally accessible measurements.

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MS160-03 — Integrating Single-Cell Experiments and Stochastic Models to Infer a Predictive, Mechanistic Understanding for Glucocorticoid Receptor Transport and DUSP1 mRNA Expression Dynamics

SPEAKERS Brian Munsky (Colorado State University)
AUTHORS Alex Popinga (School of Biomedical and Chemical Engineering, Colorado State University); Brian Munsky (Colorado State University); Eric Ron (School of Biomedical and Chemical Engineering, Colorado State University)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:40; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Biochemical assays have made outstanding progress to elucidate how cells sense and respond to stimuli, but mechanistic and parametric uncertainties preclude quantitative predictions for the full spatial, temporal and heterogeneous responses of signal-activated gene expression. Stochastic models use random noise as an abstraction to account for unknown or uncertain dynamics. When inferred from appropriate single-cell experiments, such as smFISH or immunocytochemistry (ICC), these models can quantitatively predict complex biological responses in new environments. However, many smFISH/ICC experiments are possible for different induction levels, measurement times, or observables, and each may be time consuming, expensive, or subject to labeling, imaging, or data processing errors. We introduce the Finite State Projection based Fisher Information Matrix (FSP-FIM) as a rigorous guide for the design of single-cell experiments [Pop+26]. We extend the FSP-FIM with empirical probabilistic distortion operators to account for unavoidable measurement errors. By analyzing different combinations of models, experiment designs, and data distortions, we discover practical working principles to simplify single-cell experiments while allowing for the use of inexpensive ‘crappy’ imaging conditions. We validate the FSP-FIM approach

in HeLa cells using ICC data for glucocorticoid receptor transport and smFISH data for DUSP1 gene regulation upon stimulation with a synthetic corticosteroid.

References

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MS160-05 — When Trajectory Inference Fails: The Challenges of Inferring Dynamical Models from Single-Cell RNA-seq Data

SPEAKERS Meichen Fang
AUTHORS Gennady Gorin (Fauna Bio, Emeryville, California, United States of America); Lior Pachter (Division of Biology and Biological Engineering and Department of Computing and Mathematical Sciences, California Institute of Technology, Pasadena, California, United States of America); Meichen Fang
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Single-cell RNA sequencing (scRNA-seq) enables inference of cellular trajectories from snapshots of differentiating cells. However, in many cases the inferred "pseudotime" does not have a clear mechanistic interpretation. We developed a principled trajectory inference framework based on biophysical models that can estimate interpretable parameters including process time and transcriptional rates [FGP25]. Based on this model, we demonstrate that trajectory inference from scRNA-seq data faces fundamental limitations. Through systematic analysis of simulated and real datasets, we characterise specific failure scenarios where insufficient dynamical information is embedded in the data. Key challenges include unmatched time scales, high measurement noise, and sparse sampling of intermediate states—limitations inherent to the static nature of scRNA-seq measurements. Our findings reveal critical gaps between the promise of trajectory inference and its practical limitations, emphasising the need for careful experimental design and rigorous model assessment.

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MS160-06 — Deep learning for the Analysis and Reconstruction of Transcriptional dynamics from live-cell imaging data

SPEAKERS Muhan Ma (University of Edinburgh)
AUTHORS Muhan Ma (University of Edinburgh); Ramon Grima (School of Biological Sciences, University of Edinburgh)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Quantifying transcriptional bursting from live-cell imaging data is critical for understanding stochastic gene regulation. Here, we present DART (Deep learning for the Analysis and Reconstruction of Transcriptional dynamics) [MG25], a deep learning framework that infers promoter-state trajectories from fluorescence intensity traces, enabling the estimation of activation and inactivation rates and the selection of the most appropriate promoter-switching model. Using extensive synthetic datasets spanning a wide range of transcriptional bursting levels, we demonstrate that DART outperforms current binarization methods, including conventional and augmented hidden Markov models, in both accuracy and robustness. Furthermore, a reanalysis of published experimental data using DART reveals a strong linear coupling between

activation and inactivation rates, contradicting previous claims of independence. By integrating machine learning with stochastic modelling, DART provides a powerful and generalizable tool for quantitative analysis of transcriptional kinetics from live-cell imaging data.

References

[MG25] Muhan Ma and Ramon Grima. "DART: Deep learning for the Analysis and Reconstruction of Transcriptional dynamics from live-cell imaging data". In: *bioRxiv* (2025), pp. 2025–09.

MS160-04 — Statistical inference of chromatin- and transcription dynamics in living cells

SPEAKERS Christoph Zechner (SISSA)

AUTHORS Anders S Hansen (Massachusetts Institute of Technology, Biological Engineering, Cambridge, MA); Christoph Zechner (SISSA); Christos Katsifis (Columbia University, Biological Sciences, New York, NY); Henrik D Pinholt (Massachusetts Institute of Technology, Physics, Cambridge, MA); Jack Toppen (Massachusetts Institute of Technology, Biological Engineering, Cambridge, MA); Jacob K Hansen (Massachusetts Institute of Technology, Biological Engineering, Cambridge, MA); James Jusuf (Massachusetts Institute of Technology, Biological Engineering, Cambridge, MA); Jin H Yang (Massachusetts Institute of Technology, Biological Engineering, Cambridge, MA); Leonid A Mirny (Massachusetts Institute of Technology, Institute for Medical Engineering and Science, Cambridge, MA); Miles K Huseyin (Massachusetts Institute of Technology, Biological Engineering, Cambridge, MA)

SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 11.01 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

Recent live-cell microscopy techniques allow the simultaneous tracking of distal genomic elements and transcription activity, offering new ways to study chromatin dynamics and gene regulation. However, drawing robust conclusions from such data is statistically challenging due to substantial technical noise, intrinsic fluctuations and limited time resolution. In this talk, I will present a method to infer the statistical relationship between transcription activation and enhancer-promoter distance from live-cell measurements. The problem is formulated as a generalized state-space model, accounting for chromatin dynamics, stochastic transcription activation as well as technical noise. Based on this model, we develop a path-space variational inference scheme, which reveals posterior densities over gene promoter states, polymerase loading events and enhancer-promoter distance. This allows us to quantify the spatiotemporal relationship between enhancer-promoter interactions and gene activation events. We applied the approach to experimental data in mESCs where enhancer-promoter distance and nascent transcription have been quantified across a broad range of conditions. I will conclude the talk by discussing potential implications and future work.

MS160-07 — Quantifying the impact of cell division and size control on stochastic auto-regulatory gene expression

SPEAKERS Abigail Kushnir (University of Edinburgh)

AUTHORS Abigail Kushnir (University of Edinburgh); Nikola Popovic (University of Edinburgh); Ramon Grima (School of Biological Sciences, University of Edinburgh)

SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 11.01 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

Gene expression is inherently stochastic, leading to fluctuations in protein levels that can influence cellular function. Negative autoregulatory feedback is a common regulatory motif that can suppress these fluctuations and stabilize gene expression, but its effects can depend strongly on additional cellular processes. We investigate how cell-cycle dynamics influence the noise-reduction properties of negative feedback loops by first considering a framework where cell growth and division are modeled through the effective dilution of proteins. We then extend this framework by introducing an explicit model of the cell cycle that accounts for cell growth, division, and molecular partitioning. Our results demonstrate that explicitly modelling the cell cycle can qualitatively alter noise behavior: depending on parameter

regimes, the cell cycle can either amplify or further suppress fluctuations compared to the effective model. These results highlight how model structure influences the relationship between underlying biochemical mechanisms and measurable variability, with direct implications for inference from gene expression data. Finally, we explore how different cell-size regulation strategies—such as sizer, timer, and adder mechanisms—affect noise in protein expression.

MS160-02 — Predicting Single-Cell RNA Expression Variability from DNA Sequence and Epigenetics

SPEAKERS Andrew Nicoll (University of Edinburgh)
AUTHORS Andrew Nicoll (University of Edinburgh); Diego Oyarzún (School of Biological Sciences and School of Informatics, University of Edinburgh, Edinburgh, UK); Guido Sanguinetti (SISSA, Trieste); Patrik Kojanec (Faculty of Computer and Information Science, University of Ljubljana, Ljubljana, Slovenia); Ramon Grima (University of Edinburgh); Tomaž Curk (Faculty of Computer and Information Science, University of Ljubljana, Ljubljana, Slovenia)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Single-cell technologies have unearthed vast heterogeneities in gene expression across cell populations. Understanding these cell-to-cell differences is essential for determining how DNA sequence specifies cellular function and drives phenotypic diversity. Recent advances in machine learning and AI have enabled the development of DNA sequence-to-expression prediction models. These models are typically trained on bulk expression data—how well they predict single-cell gene expression remains unclear. Here, we develop a joint machine learning and inference framework to parameterise stochastic models of transcription using biological features extracted from sequence-to-expression models and regress them against statistics derived from single-cell RNA-seq data. We further integrate epigenetic measurements, including DNA methylation and chromatin accessibility, to assess whether combining sequence and epigenetic information improves prediction of single-cell variability. We investigate the effects of technical sequencing noise and extrinsic biological variability on model performance, evaluating how well these approaches explain observed heterogeneity in single-cell RNA expression. This framework enables investigation of how mutations in DNA sequence influence regulatory dynamics in single-cell populations.

MS162 — Dynamics of Vector Populations and Pathogen Transmission

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Tuesday, 14 July 2026; 15:00–16:30; 02.11 - HS, 03.01 - HS
ORGANISERS Alex Safsten (University of Maryland); Joan Ponce (Arizona State University)

Minisymposium abstract

Vector-borne diseases pose major challenges to global health, driven by nonlinear interactions between vector population dynamics, host behavior, pathogen biology, and intervention strategies. Recent advances in mathematical and theoretical biology have enabled increasingly realistic models that move beyond homogeneous assumptions and incorporate heterogeneity such as vaccination, seasonality, and data-driven parameterization. This mini symposium focuses on recent progress in the mathematical modeling of vector populations and pathogen transmission, with applications to diseases such as malaria and dengue. Topics include models incorporating heterogeneous mosquito biting behavior [DRC25], biologically and genetically modified vector control strategies [Pan+26], and within-host immune dynamics that feed back into population-level transmission, including antibody-dependent enhancement [MG23] and vaccination effects [Qu+25]. These frameworks give rise to high-dimensional, nonlinear systems that require analytical and computational approaches to ensure identifiability, robustness, and biological interpretability. The session brings together speakers at multiple career stages, including senior researchers and early-career scientists, to highlight new models motivated by real-world vector-borne disease challenges, foster cross-disciplinary collaboration, and provide a platform for emerging researchers to present innovative theoretical and applied work.

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MS162-05 — Could malaria mosquitoes be controlled by periodic releases of transgenic mosquitocidal Metarhizium pingshaense fungus? A mathematical modeling approach.

SPEAKERS Salman Safdar (University Of Maryland, College Park)

AUTHORS Abba Gumel (University of Maryland); Abdoulaye Diabaté; Arnaja Mitra (University of Maryland); Binod Pant (Northeastern University, USA); Etienne Bilgo; Raymond St. Leger; Salman Safdar (University Of Maryland, College Park)

SCHEDULE Tuesday, 14 July 2026; 15:00–15:20; 02.11 - HS

TRACK Population Dynamics, Ecology & Evolution

Abstract

Insect pathogenic fungi present a promising alternative to chemical insecticides for controlling insecticide-resistant mosquitoes. One proposed method involves releasing male Anopheles mosquitoes contaminated with transgenic Metarhizium pingshaense (Met-Hybrid) to lethally infect females during mating. This study presents a novel deterministic mathematical model to evaluate the impact of this control approach in malaria-endemic areas. The model incorporates two fungus transmission pathways: mating-based transmission and indirect transmission through contact with fungus- colonized mosquito cadavers. We found that the fungus cannot establish in the mosquito population without transmission from infected cadavers (in this scenario, the reproduction number of the model is zero). However, if transmission from colonized cadavers is possible, the fungus can persist in the local mosquito population when the reproduction number exceeds one. Simulations of periodic releases of infected male mosquitoes, parameterized using Met-Hybrid-exposed mosquito data from Burkina Faso, show that an 86% reduction in the local female mosquito population can be achieved by releasing 10 Met-Hybrid- exposed male mosquitoes per wild mosquito every three days over six months. This matches the efficiency of some genetic mosquito control approaches. However, a 90% reduction in the wild mosquito population requires, for instance, daily releases of the fungal-treated mosquitoes in a 6:1 ratio for about 5 months, which proves less efficient than some genetic approaches. This study concludes that fungal programs with periodic releases of infected males may complement other methods or serve as an alternative to genetic-based mosquito control methods, where regulatory, ethical, or public acceptance concerns restrict genetically-modified mosquito releases.

MS162-06 — Modeling the Geospatial Dynamics of Lyme Disease in Maryland Under Current and Projected Climate Change Scenarios

SPEAKERS Salihu Musa (Department of Mathematics, University of Maryland, College Park, MD, 20742, USA)

AUTHORS Abba Gumel (University of Maryland); Salihu Musa (Department of Mathematics, University of Maryland, College Park, MD, 20742, USA)
SCHEDULE Tuesday, 14 July 2026; 15:20–15:40; 02.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Lyme disease is the most prevalent vector-borne illness in the United States, with incidence rising across Maryland over recent decades. Transmission of *Borrelia burgdorferi* by *Ixodes scapularis* ticks is highly sensitive to climatic conditions, making it essential to understand how ongoing and projected warming will shape future disease dynamics. In this presentation, I will introduce a climate-driven epidemiological model developed to investigate the spatiotemporal dynamics of Lyme disease across Maryland. I will describe how the framework integrates fine-scale ecological and epidemiological data with temperature-dependent processes governing tick development, host interactions, and pathogen transmission. I will then present simulation results under current and future warming scenarios, specifically RCP 4.5 and 8.5, highlighting how projected warming influences tick population dynamics, seasonal activity shifts, and the geographic distribution of transmission risk. I will identify regions of heightened vulnerability under each warming scenario and discuss the implications of these spatial shifts for public health planning. Finally, I will evaluate the potential of targeted interventions, including environmental management and rodent-focused tick suppression, to mitigate transmission risk, and I will present coverage thresholds at which such strategies can meaningfully reduce disease persistence. Together, these findings highlight the need for climate-adaptive approaches to vector-borne disease prevention. The modeling framework presented offers a transferable tool for informing evidence-based public health strategies in regions facing similar climate-driven challenges.

MS162-07 — Investigating the Impact of Novel Transmission-Blocking Anti-malarial Drugs: A Mathematical Modeling Approach

SPEAKERS Woldegebriel Assefa Woldegerima (York University, Canada)
AUTHORS Jacek Banasiak (Department of Mathematics and Applied Mathematics, University of Pretoria, South Africa); Rachid Ouifki (Department of Mathematics and Applied Mathematics, Northwest University, South Africa); Woldegebriel Assefa Woldegerima (York University, Canada)
SCHEDULE Tuesday, 14 July 2026; 15:40–16:00; 02.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Most available drugs kill malaria as it gets established in the liver or after it has infected red blood cells, but cannot tackle it once the parasite is released from the cells as gametocytes, which is when it is transmissible to other people via mosquito bites. Recently, promising clinical advances have been made in developing novel antimalarial drugs that block parasite transmission, cure the disease, and have prophylactic effects, called transmission-blocking drugs (TBDs). Our main aim is to explore the potential effects of such TBDs on malaria transmission in the effort to control and eliminate the disease using mathematical models to ascertain how the presence of TBDs can mitigate the transmission of malaria parasites on both asymptomatic and symptomatic carriers in a defined hotspot of malaria. Our special focus was on the effects of the treatment coverage and the efficacy of TBDs along with the protective effect and waning effect of TBDs. For this, we propose and analyze a mathematical model for malaria transmission dynamics that extends the SEIRS-SEI type model to include a class of humans undergoing treatment with TBDs and a class of those protected because of successful treatment. The mathematical and epidemiological implications of TBDs are assessed using different approaches. Furthermore, we fit the model to malaria data using the library ”lmfit” in Python and use the validated model to explore the model’s predictions under various scenarios. Results from our analysis show that the effect of treatment coverage rate on reducing reproduction number depends on other key parameters such as the efficacy of the drug. The projections of the validated model show the benefits of using TBDs in malaria control in preventing new cases and reducing mortality. We find that treating 35% of the population of Sub-Saharan

Africa with a 95% efficacious TBD from 2021 will result in approximately 82% reduction on the number of malaria deaths by 2035.

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MS162-08 — Mathematics of Wolbachia-based biocontrol of mosquito-borne diseases

SPEAKERS Abba Gumel (University of Maryland)
AUTHORS Abba Gumel (University of Maryland)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:20; 02.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The release of Wolbachia-infected mosquitoes into the population of wild mosquitoes is one of the promising biological control methods for combating the population abundance of mosquitoes that cause deadly diseases, such as dengue. In this lecture, I will present a two-sex mathematical model for the population ecology of dengue mosquitoes and disease, and use the model to assess the potential impact of the periodic release of Wolbachia-infected mosquitoes (into the wild mosquito population in the community) on the population ecology of the dengue mosquitoes and disease.

MS162-01 — Unseen but not unstoppable: Quantifying the impact of human compliance and mass screening and treatment on cryptic asymptomatic malaria transmission

SPEAKERS Arnaja Mitra (University of Maryland)
AUTHORS Abba Gumel (University of Maryland); Arnaja Mitra (University of Maryland); Salihu Musa (Department of Mathematics, University of Maryland, College Park, MD, 20742, USA)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Abstract: Malaria, a mosquito-borne disease, is transmitted to humans by the bite of an infectious female Anopheles mosquito and remains a major global public health burden. As of 2024, malaria accounted for an estimated 282 million cases and 610,000 deaths worldwide. In malaria transmission dynamics, asymptomatic individuals play an important role. Although such individuals do not exhibit clinical symptoms, they may still carry malaria parasites in their blood and, hence, serve as a hidden reservoir of infection. Because they often do not seek treatment due to the absence of symptoms, they can remain infectious for relatively long periods, thereby allowing susceptible mosquitoes to acquire the parasite during blood feeding and subsequently transmit it to susceptible humans. Consequently, asymptomatic infections can sustain community-level malaria transmission, particularly in endemic regions, and thereby complicate

malaria control and elimination efforts. In this talk, I will present a new deterministic mathematical model, formulated as a nonlinear system of differential equations, which incorporates the effects of human behavioral change and the detection and treatment of asymptomatic infections in assessing malaria transmission control.

MS162-02 — Decision-Support Modeling for One Health Pathogens: Using Mechanistic Models for Surveillance and Forecast Design

SPEAKERS Joshua Macdonald (Johns Hopkins University)
AUTHORS Anna Jolles (Oregon State University); Brianna Beechler (Oregon State University); Emma Thomson (U of Glasgow); Joshua Macdonald (Johns Hopkins University); Leah Owen (U of Glasgow); Marina Kugler (U of Glasgow); Shirin Ashraf (U of Glasgow); Stella Atim (U of Glasgow)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

We present an in development simulation-based framework for evaluating inference and decision procedures in multi-host, One Health infectious disease systems. The framework integrates surveillance design, forecasting, and scenario analysis within a unified pipeline built around mechanistic transmission models that serve as synthetic data-generating processes and known ground truth. Structured multi-population differential equation models represent coupled human, animal, and vector dynamics, incorporating demographic turnover, trait structure, and spatial heterogeneity. Empirical estimates and expert elicitation inform parameterization, while a stochastic observation layer maps latent states to synthetic surveillance data through explicit reporting and sampling processes. This generative environment enables systematic comparison of mechanistic, statistical, and machine-learning approaches under controlled observation error, partial observability, and model misspecification, with performance assessed using robustness and decision-relevant metrics. We illustrate the framework using a multi-host ODE-PDE hybrid model of Crimean–Congo hemorrhagic fever virus, a tick-born orthonirovirus, in Uganda, demonstrating the utility our our framework for studying identifiability, forecasting, and surveillance optimization across heterogeneous one-health systems.

MS162-03 — Rethinking mosquito biting rates: exploring how disturbed blood-feeding shapes vectorial capacity

SPEAKERS Kyle Dahlin (Virginia Tech)
AUTHORS Clément Vinauger (Virginia Tech); Kyle Dahlin (Virginia Tech); Lauren Childs (Department of Mathematics, Virginia Tech); Michael Robert (Virginia Tech)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Mosquito-borne diseases are increasing in incidence and geographic range, renewing interest in how mosquito behavior shapes disease transmission. One unresolved issue is how disturbed feeding affects transmission. Many models assume that mosquito biting can be represented by a single, constant contact rate, implicitly treating feeding as instantaneous and always successful. In reality, defensive behaviors interrupt blood-feeding, causing a mosquito to leave without completing its blood meal. In this case, the mosquito may need to feed on additional hosts, thereby increasing the number of contacts per reproductive cycle.

Combining laboratory experiments with mathematical modeling, this project revisits the common modeling assumption that biting should be represented by a single, constant contact rate. With video tracking, we measure how disturbance affects the duration and success of different feeding stages in Aedes aegypti. We track whether mosquitoes persist in feeding, abandon it, or otherwise change their behavior in response to disruption. The resulting measurements are used to parameterize a model that treats blood-feeding as a sequence of behaviors rather than as a single event. By focusing on variation in feeding behavior

among individual mosquitoes, this study explores how disturbed feeding may influence vectorial capacity and outbreak risk. The study aims to connect individual-level behavior to population-level patterns of mosquito-borne disease transmission.

MS162-04 — Variable impact of density-dependent life history traits on the success of mosquito population reduction strategies

SPEAKERS Lauren Childs (Virginia Tech)
AUTHORS Lauren Childs (Virginia Tech); Michael Robert (Virginia Tech); Zhuolin Qu (University of Texas, San Antonio)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Population growth is often mediated by density-dependent regulation, which can impact different life history traits, such as reproduction, development, or survival. The way and extent that density dependence alters these traits can substantially change population dynamics, which may have important consequences for control efforts. We develop an ordinary differential equations model including density dependence in various life history traits – reproduction of juvenile mosquitoes, development of juveniles to adults, and mortality of adult mosquitoes. We then examine how these various incorporations of density dependence affect the success of population reduction strategies mediated by the endosymbiont bacteria, Wolbachia. We use a combination of analysis and numerical simulations to show that when density dependence impacts the development of juvenile mosquitoes, Wolbachia-based control strategies may have the unintended effect of increasing population size. In contrast, when density dependence only impacts mortality or reproduction, Wolbachia-based control behaves as expected, reducing the mosquito population. Our results demonstrate that understanding how and to what extent density dependence alters various mosquito life history traits is crucial to unraveling the impacts of control efforts.

MS1644 — Symposium in memory of Professor Béla Novák (1956-2025)

TRACK Miscellaneous
SCHEDULE Tuesday, 14 July 2026; 15:00–16:15; 01.15 - HS
ORGANISERS Denis Thieffry (Ecole Normale Supérieure); John Tyson (VirginiaTech)

Minisymposium abstract

This symposium will feature contributions from several of Béla Novak's collaborators and colleagues, with the aim of commemorating his fundamental contributions to the field of computational systems biology, in particular regarding the deciphering and predictive modelling of the complex molecular network controlling the eucaryotic cell cycle, as well as his exceptional scientific mentoring qualities.

MS1644-03 — Thirty Years of Cell Cycling with Bela

SPEAKERS John Tyson (VirginiaTech, VA, USA)
AUTHORS John Tyson (VirginiaTech, VA, USA)
SCHEDULE Tuesday, 14 July 2026; 15:00–15:15; 01.15 - HS
TRACK Miscellaneous

Abstract

In 1993, Novak & Tyson published a comprehensive model of M-phase control in frog egg extracts (J. Cell Sci. 106, 1153-1168). The model, soundly based on known molecular mechanisms, provided a unified understanding of cell-cycle transitions, mitotic oscillations, checkpoints, and wave propagation in frog eggs, yeast cells and fruit fly embryos. It made many non-intuitive predictions that were later confirmed experimentally. Later work by Novak, Tyson and their students and collaborators led to a general 'dynamical perspective for molecular cell biology,' based on detailed numerical simulations and

generic bifurcation diagrams. In this talk, I will give a brief survey of how the theory developed over the intervening 30+ years.

MS1644-01 — Unlike networks, molecules are dreams - the early years of systems biology with Bela Novak.

SPEAKERS Andrea Ciliberto (IFOM)
AUTHORS Andrea Ciliberto (IFOM)
SCHEDULE Tuesday, 14 July 2026; 15:15–15:30; 01.15 - HS
TRACK Miscellaneous

Abstract

In this presentation, I will start from my own experience in the lab of Bela Novak to discuss some results on cell cycle regulation, remember his figure as a mentor, and I will discuss the importance of doing basic science slowly and in depth. In particular, I will discuss the last stretch of 'hungarian years' in Bela's work (early 2000s up to 2007), when he had the freedom to do research in relative isolation and with little external pressure. This mostly theoretical work laid the ground for the very fruitful interactions with experimental biologists that took place in Oxford, which brought to Bela an even wider recognition among experimentalists.

MS1644-04 — Modeling the Cell Cycle: Reflections Inspired by Béla Novák

SPEAKERS Laurence Calzone (Institute Curie)
AUTHORS Laurence Calzone (Institute Curie)
SCHEDULE Tuesday, 14 July 2026; 15:30–15:45; 01.15 - HS
TRACK Miscellaneous

Abstract

My scientific path has been deeply shaped by my collaboration with Béla Novák and John Tyson. Working on the early embryonic cycles of Drosophila melanogaster, I was introduced to a way of thinking that seeks simple, robust principles behind complex biological phenomena. Beyond the specific results, Béla Novák's approach, which combines conceptual clarity with biological insight, left a lasting mark on how I approach modeling. This influence continues to guide my work at Institut Curie, where I continue to explore the cell cycle in cancer. There, I have moved from deterministic ODE models toward a stochastic Boolean framework to better capture the heterogeneity and variability of tumor systems. This presentation is both a reflection on our early work and a personal tribute to Béla's enduring impact on our scientific thinking.

MS1644-02 — Positive feedback + negative feedback = "bloody complicated"

SPEAKERS Attila Czikasz-Nagy (Pázmány Péter Catholic University Faculty of Information Technology and Bionics / Cytocast)
AUTHORS Attila Czikasz-Nagy (Pázmány Péter Catholic University Faculty of Information Technology and Bionics / Cytocast)
SCHEDULE Tuesday, 14 July 2026; 15:45–16:00; 01.15 - HS
TRACK Miscellaneous

Abstract

I started working with Béla as an undergraduate and remained in his lab for more than a decade, continuing to learn from him even until he left us. Together with John, he taught me how to analyse biological regulatory systems with both positive and negative feedback loops. The combination of these two can lead to fascinating dynamical behaviours. As Béla used to say: "it is bloody complicated" to understand what type of spatial and temporal pattern such combined systems might show. I will present biological examples showing how the combined effects of these two feedback types explain the observed behaviour.

MS1644-05 — A systems biology study of autophagy induction playing a key role in survival upon cellular stress

SPEAKERS Orsolya Kapuy (Semmelweis University)
AUTHORS Orsolya Kapuy (Semmelweis University)
SCHEDULE Tuesday, 14 July 2026; 16:00–16:15; 01.15 - HS
TRACK Miscellaneous

Abstract

As a PhD student, I began working with Prof. Béla Novák, and I learned virtually everything about the theoretical biology approach from him. The years I spent in his group at Oxford truly showed me how to combine theoretical biology techniques with molecular biology methods.

Later, I tried to apply this knowledge when I began studying autophagy induction at Semmelweis University. Although autophagy is traditionally classified as a cell-death mechanism, many scientific results have revealed that autophagy has an essential role in cellular survival upon various stress events (such as starvation or bacterial infection).

Our goal is to identify the essential regulatory motifs of autophagy control network and to explore their roles in cellular stress-induced life-and-death decision directly focusing on the inter- and intra-molecular-crosstalks of the system. By discovering the dynamics of the key feedback loops using both molecular and theoretical biological techniques, we try to understand how autophagy-dependent cellular survival can be extended.

MS68 — Biology at the Interfaces: Data-Informed Multiscale Modelling

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Tuesday, 14 July 2026; 17:00–18:20; 02.23 - HS, 02.11 - HS
ORGANISERS Giulia Chiari (University of Oxford / BCAM (Basque Center for Applied Mathematics)); Luciana Luque (CRUK Scotland Institute)

Minisymposium abstract

Biology is no longer limited by ideas; it is limited by our ability to connect them. This minisymposium spotlights cross-disciplinary research that turns wet-lab and clinically derived measurements into mechanistic, testable mathematical and computational models — and back again. We will showcase studies where experiments and modelling are co-designed: data inform the model, the model makes falsifiable predictions, and new experiments close the loop.

Across diverse applications (from cell–cell interactions and tissue mechanics to immune dynamics and therapy response), speakers will demonstrate how theory can denoise data, expose hidden constraints, quantify uncertainty, reveal emergent behaviours that cannot be read off directly from components, and propose efficient next experiments — saving time, animals, and resources while improving biological insight and translational relevance. Alongside success stories, we will discuss practical strategies for collaboration: building shared vocabularies across fields, reproducible pipelines, and validation standards that make models trustworthy and reusable beyond a single lab.

Expect punchy talks, real datasets, and a clear message: the most powerful biology happens at the interfaces.

MS68-03 — Integrating multiscale multimodal imaging-based and PK/PD-informed models to predict triple negative breast cancer response to neoadjuvant therapy

SPEAKERS Guillermo Lorenzo (University of A Coruña)
AUTHORS Guillermo Lorenzo (University of A Coruña)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 02.23 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Neoadjuvant therapy (NAT) is a standard initial treatment for triple-negative breast cancer (TNBC), yet early prediction of treatment response remains challenging. This capability would enable adjustment of therapeutic plans to optimize outcomes and minimize toxicities. To this end, I will present a mechanistic, multiscale mathematical model of TNBC response to NAT that integrates in vivo longitudinal MRI with time-resolved in vitro drug-response data to identify key mechanisms driving tumor dynamics. The model describes tumor cell density evolution through mechanically-constrained mobility and net proliferation, coupled to a pharmacokinetic/pharmacodynamic (PK/PD) representation of NAT drug regimens. To facilitate clinical applicability, a global sensitivity analysis (GSA) is carried out by using Sobol's method on 3D MRI-based tissue domains from well and poorly-perfused tumors. The parameter space combines prior in silico estimates informed by in vivo imaging with in vitro measurements capturing drug-induced proliferation changes across TNBC cell lines. The GSA reveals a small subset of dominant parameters governing treatment response, enabling construction of a parsimonious surrogate model that preserves the dynamics of the full formulation. Building on these results, I will briefly outline a personalized tumor forecasting pipeline in which the reduced model is calibrated to early-treatment MRI data and used to obtain patient-specific forecasts of tumor response to NAT.

References

[Lor+22] Guillermo Lorenzo et al. "Quantitative in vivo imaging to enable tumour forecasting and treatment optimization". In: *Cancer, complexity, computation*. Springer, 2022, pp. 55–97.

[Lor+24] Guillermo Lorenzo et al. "A global sensitivity analysis of a mechanistic model of neoadjuvant chemotherapy for triple negative breast cancer constrained by in vitro and in vivo imaging data". In: *Engineering with computers* 40.3 (2024), pp. 1469–1499.

[Mey+19] Christian T Meyer et al. "Quantifying drug combination synergy along potency and efficacy axes". In: *Cell systems* 8.2 (2019), pp. 97–108.

[Pat+25] Reshmi JS Patel et al. "MRI-Based Mathematical Modeling to Predict the Response of I-SPY 2 Patients with Breast Cancer to Neoadjuvant Therapy". In: *Clinical Cancer Research* 31.22 (2025), pp. 4846–4856.

MS68-05 — Linking Primary and Metastatic Growth: Testable Predictions from a Shared-Carrying-Capacity Model

SPEAKERS Pirmin Schlicke (University of Salzburg)

AUTHORS Heiko Enderling (Department of Radiation Oncology, Institute for Data Science in Oncology, UT MD Anderson Cancer Center, Houston, TX, U.S.A.); Pirmin Schlicke (University of Salzburg)

SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 02.23 - HS

TRACK Multiscale and Multiphysics Modelling

Abstract

To inform decisions rather than replicating noise, low-dimensional and identifiable models calibrated with multiscale data can serve as reliable tools through separating host-level constraints from lesion-level properties.

As an example for a simple, yet structured approach, we present a mechanistic model formulating systemic cancer dynamics that links primary and metastatic growth through a shared carrying capacity. Building on Gompertzian growth, lesions are coupled via a host-level constraint on total tumor burden and extend the same idea to a metastatic size-distribution framework, enabling joint calibration to longitudinal primary-tumor measurements and metastatic nodule data. Model selection identifies the most parsimonious formulation that captures cross-lesion interdependence from more classic alternatives. Identifiability analysis shows that key drivers can be practically constrained from limited datasets, supporting robust inference with few parameters. Counterfactual simulations result in competitive metastatic release after primary tumor resection, generating testable hypotheses for prospective experiments. Beyond oncology, the work illustrates a general theoretical-biology principle: simple, yet structured models can denoise

multiscale data by separating host- from lesion-level traits, yielding computational biomarkers and efficiently informing experimental designs across interacting population systems.

MS68-06 — Data-driven reconstruction of phenotypic transition networks in colorectal cancer

SPEAKERS Rodrigo Garcia-Tejera (Institute of Genetics and Cancer)
AUTHORS Rodrigo Garcia-Tejera (Institute of Genetics and Cancer)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 02.23 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cancer cells exhibit a remarkable ability to adopt different phenotypic states in response to cell-intrinsic programs and environmental cues, a phenomenon known as cancer cell plasticity. Plasticity underlies tumour heterogeneity, treatment resistance, and metastasis. To move therapeutic approaches beyond pathway and enrichment analyses comparing early and late disease, we need to understand the principles governing the evolution of phenotypic composition and spatial organisation in cancerous tissue. Mechanistic modelling may be a great asset in this direction, allowing analysis of in vitro studies and in vivo spatial omics data from a complex systems perspective, linking local rules of cell division and state transition to emergent tissue-level patterns. Here, I will present our work dissecting cell-intrinsic plasticity in colorectal cancer organoids. Perturbation experiments reveal ATRX, a chromatin remodelling enzyme, as a key regulator of plasticity. Using Markov-chain models, we leverage time-course cell sorting experiments to infer phenotypic transition networks following ATRX deletion and identify key transitions driving heterogeneity. I will discuss how to integrate these observations with in vivo spatial transcriptomics measurements, where plasticity is driven by environmental cues, and how analysing the spatial structure with complex systems methods further reveals tissue dynamics.

References

[Cam+25] Patrizia Cammareri et al. “Loss of colonic fidelity enables multilineage plasticity and metastasis”. In: *Nature* 644.8076 (2025), pp. 547–556.

MS68-07 — A data-integrative approach to building ABMs for TNBC metastasis initiation and growth in the lungs

SPEAKERS Tatiana Miti (Moffitt Cancer Center)
AUTHORS Amar Omar; David Basanta; Tatiana Miti (Moffitt Cancer Center); Thordur Oskarsson
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 02.23 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

We developed a data-integrative framework to parametrize and validate an agent-based model (ABM) of triple-negative breast cancer (TNBC) metastasis in the lung, focusing on the emergent dynamics of tumor-microenvironment interactions. The model represents tumor cells, fibroblasts, macrophages, and endothelial cells as interacting agents, governed by probabilistic rules for proliferation, death, migration, and phenotypic transitions. Parameterization leverages complementary datasets across scales. Quantitative histology (IHC/IF) provides information on cell type, spatial distribution, and temporal growth dynamics with further constraints on phenotypic composition from flow cytometry. In vitro/vivo assays quantify activation rates underlying tumor–stroma cross-talk, while bulk growth curves constrain system-level dynamics. These heterogeneous datasets are integrated using Approximate Bayesian Computation to infer parameter distributions consistent with observed multiscale behaviors. We calibrate the model against joint observables, including population growth, phenotypic proportions, and temporal evolution. Validation is performed using chemotherapy and pathway inhibition experiments to assess predictive capacity under intervention. This approach enables identification of constrained interaction rules that reproduce observed metastatic dynamics and provides a principled framework for linking experimental measurements to mechanistic models of metastatic progression.

MS68-04 — Drug dosing design guided by response and resistance convexity

SPEAKERS Jeffrey West (Moffitt Cancer Center)
AUTHORS Jeffrey West (Moffitt Cancer Center)
SCHEDULE Thursday, 16 July 2026; 17:00–17:40; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

We explore the practicality of guiding treatment scheduling based on the convexity (or concavity) of dose-response curves, which provides a straightforward comparison of continuous treatment and high-dose / low-dose alternatives. Concave dose-response functions predict that the daily administration of a dose of x may be less efficacious than a regimen that switches equally between 120% of x and 80% of x (every other day). Convex dose-response provide the opposite prediction (high / low dosing is best). However, treatment fails due to the evolution of resistance, indicating that dose-response is changing in time with mutation or plasticity driven resistance mechanisms. Drug holidays have been suggested as re-sensitization method for tumors. Using mathematical modeling integrated with in vivo data, we predict the dose-dependent rate of resistance onset for targeted therapy, which we show is a concave function of dose. Our integrative modeling framework predicts a trade-off between maximizing response (continuous protocols) and maintaining drug sensitivity (high / low protocols), suggesting re-sensitization is possible. Thus, we propose alternative switching treatment protocols to balance this trade off: continuous followed by high / low (or vice versa), subsequently validated in a non-small cell lung cancer in vivo model. This convexity-based approach to treatment scheduling illustrates the effectiveness of incorporating principles of convexity into protocol design.

MS68-01 — Mathematical modeling of tissue morphogenesis and regeneration

SPEAKERS Diane Peurichard (INRIA Paris)
AUTHORS Diane Peurichard (INRIA Paris)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In this talk, we investigate how organs acquire functional structure and rebuild after injury using Individual Based Models (IBM) confronted with experimental data. We first study simple 2D and 3D models for architecture emergence, with cells appearing and growing in a dynamic network of cross-linked fibers. Cells and fibers interact via mechanical repulsion. When applied to adipose tissue, the model reproduces experimental structures and suggests that cell clusters could spontaneously emerge from simple cell–fiber interactions. By implying that vasculature could be secondary to tissue architecture, this simple model therefore proposes a new view of tissue development. In the second part of the talk, we extend the model to account for tissue repair, exploring mechanisms of adipose tissue regeneration. The model successfully generates regeneration or scar formation as functions of few key parameters and indicates that injury outcomes largely depend on ECM rigidity. Via a combined in vivo/in silico approach, the model enables to identify a therapeutically validated target enabling regeneration in mouse adipose tissue. Altogether, these studies point to the essential role of mechanics in tissue structuring and regeneration, and bring a comprehensive view on the role of ECM crosslinking on tissue architecture emergence and reconstruction.

MS68-02 — Leveraging spatial data analysis to provide robust, model-agnostic, and accurate measurements of pattern formation

SPEAKERS Duncan Martinson (The Francis Crick Institute)
AUTHORS Duncan Martinson (The Francis Crick Institute)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Understanding how groups of cells robustly coordinate their behavior represents a key question in developmental biology. Mathematical modeling helps to address this problem by enabling researchers to investigate hypotheses in an abstract setting, yet it remains challenging to link these theoretical frameworks to experimental data. Here, we present a novel computational pipeline that addresses this issue and illustrate its application to the case of zebrafish stripe pattern formation. The pipeline generates labeled point clouds from experimental and theoretical images and extracts information about their spatial patterns by applying tools from spatial data analysis. We demonstrate that statistics which measure spatial organization across multiple length scales are robust to experimental and synthetic replicates, even when synthetic data are sampled from a nearly spatially uniform initial state. Unsupervised clustering of images based on pipeline-derived statistics yields biologically interpretable clusters based on the presence of stripes, spots, or maze-like structures. We envision that our spatial analysis pipeline, which is agnostic to data source and not limited to models of zebrafish pattern formation, will enable future researchers to robustly and accurately link dynamic models of spatially heterogeneous phenomena to experimental data.

MS116 — Complexity Science for Biological and Medical Problems

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Tuesday, 14 July 2026; 17:00–18:20; 02.11 - HS
ORGANISERS Javier Galeano (E.T.S.I.A.A.B., Universidad Politécnica de Madrid); Jesús J. Bosque (Universidad Politécnica de Madrid, Mathematical Oncology Laboratory (MOLAB))

Minisymposium abstract

Complexity science and mathematical biology are closely related disciplines in terms of goals and approach. However, due to their independent historical development, the two fields largely follow parallel paths, with relatively limited interaction in conferences. Complexity science focuses on the use of mathematical models to understand global phenomena emerging from the low-level interactions among multiple components. The insights provided by these mathematical tools, together with increasingly available data, have led to a wide range of applications in biology and medicine.

This minisymposium brings together researchers addressing biological and medical problems from a complexity science perspective. This year, the session will centre on modelling multiple interacting cell populations and the implications of their ecological structure. The four speakers will cover diverse applications, including the prediction of microbial ecosystem dynamics using generalised Lotka-Volterra models, Bayesian inference algorithms to reconstruct interaction-driven probability distributions, co-evolution and the emergence of mutualism in interacting populations, and the use of these methods to better quantify cancer response to chemotherapy.

Building on the strong interest that the minisymposium generated at ECMTB 2024 (Toledo, Spain), this new edition aims to continue fostering interaction and collaboration between mathematical biologists and complexity science researchers.

References

[Cam+24] José Camacho-Mateu et al. “Sparse species interactions reproduce abundance correlation patterns in microbial communities”. In: *Proceedings of the National Academy of Sciences* 121.5 (2024), e2309575121.

[Cas+25] Mario Castro et al. “Scarce data, noisy inferences and overfitting: the hidden flaws in ecological dynamics modelling”. In: *Journal of the Royal Society Interface* 22.231 (2025).

[Lam+24] Aniello Lampo et al. “Structural dynamics of plant–pollinator mutualistic networks”. In: *PNAS nexus* 3.6 (2024), pgae209.

[Sán+25] Marta Sánchez-Díez et al. “Assessment of cell viability in drug therapy: IC50 and other new Time-Independent indices for evaluating chemotherapy efficacy”. In: *Pharmaceutics* 17.2 (2025), p. 247.

[Stu+20] Luciano Stucchi et al. “A general model of population dynamics accounting for multiple kinds of interaction”. In: *Complexity* 2020.1 (2020), p. 7961327.

MS116-01 — Simplicity in complexity: A probabilistic view microbial ecology

SPEAKERS Mario Castro (Instituto de Investigación Tecnológica (IIT), Universidad Pontificia Comillas de Madrid)
AUTHORS Mario Castro (Instituto de Investigación Tecnológica (IIT), Universidad Pontificia Comillas de Madrid)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 02.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Combining RNAseq data and models in microbial ecology aims to reveal species interactions and improve health outcomes. However, data is often noisy, and inferred ”interactions” are merely model-dependent correlations rather than direct biological mechanisms. This raises a crucial question: when inferring models from data, are complex models better, or is simplicity more effective?

We argue that minimal models generally outperform complex ones. Using information geometry and Bayesian inference, we demonstrate that simple models maximize reliable information extraction, making them information-theoretically optimal [Cas+25]. Furthermore, many widely reported microbial macroecological patterns may simply result from data aggregation [CC] or lack the robustness required to be genuine laws [Cam+25].

References

[Cam+25] José Camacho-Mateu et al. “Microbial populations hardly ever grow logistically and never sublinearly”. In: *Physical Review E* 111.4 (2025), p. 044404.

[CC] Mario Castro and Jose A Cuesta. *In preparation.*

[Cas+25] Mario Castro et al. “Scarce data, noisy inferences and overfitting: the hidden flaws in ecological dynamics modelling”. In: *Journal of the Royal Society Interface* 22.231 (2025).

MS116-02 — An evolutionary approach to ecological transitions in population dynamics

SPEAKERS Luciano Stucchi (Universidad del Pacífico)
AUTHORS Luciano Stucchi (Universidad del Pacífico)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 02.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Population dynamics has traditionally focused on the study of what occurs in ecological regimes that only change their relationships quantitatively. However, populations that transition from one ecological regime to another, such as predation or parasitism shifting to mutualism, or vice versa, have been extensively documented in diverse ecological contexts. When population dynamics has addressed those transitions, it usually follows ad hoc mechanisms or uses different dynamics for each regime. Here, we present a model that combines adaptive dynamics with a generalized population dynamics model [Stu+20] in an integrated way, which allows the transition between ecological regimes. We use a microbial toy-model that despite its simplicity shows that such transitions might occur naturally [Stu+22] for different set of parameters and serves as a proof of concept. We also show a few simulations that resemble empirical cases of ecological transitions driven by evolution.

References

[Stu+20] Luciano Stucchi et al. “A general model of population dynamics accounting for multiple kinds of interaction”. In: *Complexity* 2020.1 (2020), p. 7961327.

[Stu+22] Luciano Stucchi et al. “Prevalence of mutualism in a simple model of microbial coevolution”. In: *Physical Review E* 106.5 (2022), p. 054401.

MS116-03 — Species interactions do matter in microbial dynamics

SPEAKERS Aniello Lampo (Departamento de Matemáticas, Universidad Carlos III de Madrid)
AUTHORS Aniello Lampo (Departamento de Matemáticas, Universidad Carlos III de Madrid)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 02.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

During the last decades, macroecology has identified broad-scale patterns of abundances and diversity of microbial communities and put forward some potential explanations for them. However, these advances are not paralleled by a full understanding of the dynamical processes behind them. In particular, abundance fluctuations of different species are found to be correlated, both across time and across communities in metagenomic samples. Reproducing such correlations through appropriate population models remains an open challenge. The present paper tackles this problem and points to species interactions as a necessary mechanism to account for them. Specifically, we discuss several possibilities to include interactions in population models and recognize Lotka–Volterra constants as a successful ansatz [Cam+24b; Cam+24a]. For this, we design a Bayesian inference algorithm to extract sets of interaction constants able to reproduce empirical probability distributions of pairwise correlations for diverse biomes. Importantly, the inferred models still reproduce well-known single-species macroecological patterns concerning abundance fluctuations across both species and communities. Endorsed by the agreement with the empirically observed phenomenology, our analyses provide insights into the properties of the networks of microbial interactions, revealing that sparsity is a crucial feature.

References

[Cam+24a] José Camacho-Mateu et al. “Nonequilibrium microbial dynamics unveil a new macroecological pattern beyond Taylor’s law”. In: *Physical Review E* 110.4 (2024), p. 044402.

[Cam+24b] José Camacho-Mateu et al. “Sparse species interactions reproduce abundance correlation patterns in microbial communities”. In: *Proceedings of the National Academy of Sciences* 121.5 (2024), e2309575121.

MS116-04 — Robust time-independent metrics for evaluating drug efficacy

SPEAKERS Marta Sánchez-Díez (Center for Biomedical Technology, Universidad Politécnica de Madrid)
AUTHORS Carmen Ramírez Castillejo (CTB (CTB-UPM) Centro de Tecnología Biomédica, Universidad Politécnica de Madrid, 28223 Pozuelo de Alarcón, Spain); Juan Manuel Pastor (Grupo de Sistemas Complejos, Universidad Politécnica de Madrid, 28040 Madrid, Spain); Marta Sánchez-Díez (Center for Biomedical Technology, Universidad Politécnica de Madrid)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 02.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Traditional drug efficacy assessment relies on cell viability and the IC_{50} index. This index represents the concentration of drug required for cell viability to be 50%, i.e. the population of treated cells is half that of the control population. However, since early-stage cell populations follow Malthusian growth, viability is inherently time-dependent. This makes IC_{50} a transient metric that fails to distinguish between different proliferation dynamics.

In this work, we propose a robust mathematical framework to analyze cell viability assays by focusing on effective growth rates rather than endpoint populations. Assuming exponential proliferation, we employ bootstrap resampling method to achieve precise exponential fits. This approach allows for the

discovery of two new time-independent parameters: IC_{r0}, corresponding to a zero-growth rate, and IC_{rmed},corresponding to a growth rate half that of the control [Sán+25].

Both indices provide a more biologically meaningful evaluation of drug efficacy. This methodology offers a standardized, mathematically based alternative to classical pharmacodynamics, ensuring that efficacy measurements reflect the real impact of a drug on cell proliferation regardless of the experimental timeframe.

References

[Sán+25] Marta Sánchez-Díez et al. "Assessment of cell viability in drug therapy: IC50 and other new time-independent indices for evaluating chemotherapy efficacy". In: *Pharmaceutics* 17.2 (2025), p. 247.

MS151 — Modeling Collective Dynamics in Heterogeneous Cell Populations

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Tuesday, 14 July 2026; 17:00–12:30; 02.01 - HS
ORGANISERS Giulia Celora; Martina Conte (Politecnico di Torino); Negar Mohammadnejad (University of Alberta)

Minisymposium abstract

Heterogeneity in cell behaviour plays a central role in the spatiotemporal organisation of cell populations, such as in the formation of bacterial biofilms, tissues, and tumour invasion. Mathematical modelling provides a framework for understanding how emergent collective behaviours arise from dynamic interactions within heterogeneous cell populations. The nature of this heterogeneity, as well as the specific collective function of interest, often necessitates different modelling approaches. In this minisymposium, we aim to showcase a range of mathematical approaches for studying cellular heterogeneity across scales, from gene regulatory networks to phenotypically structured population models and their applications. By fostering interactions among speakers with diverse expertise, applications, and backgrounds, we aim to promote the integration of these approaches, helping to overcome their individual limitations and to reveal how heterogeneity promotes robustness or drives disruption in the physiological functions of cell collectives across spatial and temporal scales.

MS151-01 — Radiotherapy as an evolutionary force: modeling phenotypic heterogeneity and treatment response in tumors

SPEAKERS Lara Schmalenstroer (Bioinformatics and Computational Biophysics, University of Duisburg-Essen)
AUTHORS Farnoush Farahpour (Bioinformatics and Computational Biophysics, University of Duisburg-Essen); Russell Rockne (Professor & Chair, Department of Computational and Quantitative Medicine Beckman Research Institute, City of Hope.); Lara Schmalenstroer (Bioinformatics and Computational Biophysics, University of Duisburg-Essen)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 02.01 - HS
TRACK Mathematical Oncology

Abstract

Tumors exhibit phenotypic heterogeneity in proliferation rates, affecting growth, therapy response, and relapse risk. Faster-proliferating cells dominate untreated growth but face fitness costs like increased susceptibility to DNA damage or metabolic stress. Proliferation capacity is a core tumor characteristic, and capturing its heterogeneity is key to understanding tumor evolution and treatment outcomes. We introduce a novel mathematical framework using a continuous phenotype-structured PDE to model proliferation heterogeneity after radiotherapy. Parameterized with experimental data from irradiated murine lung tumors, the model captures tumor growth, phenotypic drift, and selection pressures. Radiotherapy is modeled with a trait-dependent killing term, reflecting the biological premise that slower-proliferating cells are more resistant to radiation-induced damage. Simulations show radiotherapy exerts strong selection pressure, enriching slow-proliferating, radioresistant cells, rather than merely reducing tumor volume. This

compositional shift reduces the population-wide mean proliferation rate, slowing post-treatment regrowth. Depending on dose, residual tumors show distinct phenotypic distributions, such as bimodal structures with coexisting slow- and fast-proliferating subpopulations, revealing divergent evolutionary paths. Our findings emphasize the importance of integrating continuous heterogeneity for accurate modeling and designing evolutionarily informed therapies.

MS151-02 — Multi-phase field approach towards modelling mechanical heterogeneity in tissues

SPEAKERS Aleksandra Ardaševa (Institute of Biosciences, École polytechnique fédérale de Lausanne and Niels Bohr Institute, University of Copenhagen)

AUTHORS Aleksandra Ardaševa (Institute of Biosciences, École polytechnique fédérale de Lausanne and Niels Bohr Institute, University of Copenhagen); Amin Doostmohammadi (Niels Bohr Institute, University of Copenhagen); Benoit Ladoux (Max Planck Zentrum Physics and Medicine, FAU Erlangen-Nürnberg University and Institute Jacques Monod, CNRS, University Paris City); Lakshmi Balasubramaniam (Gurdon Institute, University of Cambridge and Institute Jacques Monod, CNRS, University Paris City); Siavash Monfared (Niels Bohr Institute, University of Copenhagen)

SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 02.01 - HS

TRACK Multiscale and Multiphysics Modelling

Abstract

Biological tissues exhibit heterogeneity across multiple scales, from genetic to non-genetic. In epithelial layers, variability in mechanical properties such as adhesion and motility, together with differences in cell–cell interactions, strongly influences collective dynamics and tissue organization.

We present a 3D multiphase-field model [MAD25] of confluent cell monolayers in which each cell is described by a phase field whose evolution is governed by motility, interfacial tension, adhesion, and volume constraints. Mechanical heterogeneity is introduced through cell-dependent parameters controlling passive and active contributions. We apply this framework to investigate cell extrusion in epithelial monolayers [Bal+25]. Combining simulations with experiments on epithelial cells with different levels of E-cadherin–mediated adhesion, we show how mechanical properties determine whether extrusion is apoptotic or live and whether it occurs apically or basally, leading to cell invasion into soft collagen gels.

Hence, by incorporating cell heterogeneity at various levels, our model provides a more comprehensive framework for understanding the role of mechanical heterogeneity on collective behaviour in tissues.

References

[Bal+25] Lakshmi Balasubramaniam et al. “Dynamic forces shape the survival fate of eliminated cells”. In: *Nature physics* 21.2 (2025), pp. 269–278.

[MAD25] Siavash Monfared, Aleksandra Ardaševa, and Amin Doostmohammadi. “Multiphase-field models of tissues”. In: *Annual Review of Condensed Matter Physics* 17 (2025).

MS151-03 — Phenotype structuring in collective cell migration: a tutorial of mathematical models and methods

SPEAKERS Chiara Villa (CNRS & MAP5 (UPC))

AUTHORS Chiara Villa (CNRS & MAP5 (UPC)); Kevin J. Painter (Politecnico di Torino); Tommaso Lorenzi (Politecnico di Torino)

SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 02.01 - HS

TRACK Multiscale and Multiphysics Modelling

Abstract

The coexistence of diverse phenotypic traits within a population - such as variations in cell movement, growth, or signalling - can profoundly shape collective dynamics of cell populations. To capture these complexities, classical PDE models for cell migration can be extended to include phenotypic structuring, giving rise to a powerful class of non-local models: phenotype-structured partial integro-differential equations (PS-PIDEs). In this talk, I will present a tutorial-style review paper dedicated to this growing

field [LPV25], offering both a pedagogical foundation for teaching and a roadmap for advancing research. We will first explore the current state of the art of how PS-PIDEs can be formally derived from agent-based models, analysed with semi-classical asymptotic methods, and solved numerically, in order to investigate the emergence of spatial sorting of the population at the tissue-scale due to the interplay between cell adaptive dynamics and environmental feedback. Finally, we will discuss open mathematical and interdisciplinary challenges expected to shape the future of this field.

References

[LPV25] Tommaso Lorenzi, Kevin J Painter, and Chiara Villa. "Phenotype structuring in collective cell migration: a tutorial of mathematical models and methods". In: *Journal of Mathematical Biology* 90.6 (2025), p. 61.

MS151-04 — A multiscale discrete-to-continuum framework for structured population models

SPEAKERS Eleonora Agostinelli (University of Oxford)
AUTHORS Eleonora Agostinelli (University of Oxford); Helen Byrne (Mathematical Institute, University of Oxford and Ludwig Institute for Cancer Research, University of Oxford); Keith Chambers (Ludwig Institute for Cancer Research, University of Oxford); Mohit Dalwadi (Mathematical Institute, University of Oxford)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Population models commonly use discrete structure classes to capture trait heterogeneity among individuals (e.g. age, size, phenotype, intracellular state). Upscaling these discrete models into continuum descriptions can improve analytical tractability and scalability of numerical solutions. Common upscaling approaches based solely on Taylor expansions may, however, introduce ambiguities in truncation order, uniform validity and boundary conditions. To address this, we introduce a discrete multiscale framework to systematically derive continuum approximations of structured population models. Using multiscale asymptotic methods applied to discrete systems, we identify regions of structure space for which a continuum representation is appropriate. The leading-order dynamics are governed by nonlinear advection in the bulk, with diffusive boundary-layer corrections near wavefronts and stagnation points. We also derive discrete descriptions for regions where a continuum approximation is fundamentally inappropriate. This multiscale framework can be applied to other heterogeneous systems with discrete structure to obtain appropriate upscaled dynamics with asymptotically consistent boundary conditions.

MS151-05 — Age-structured models for cell-cycle synchrony

SPEAKERS Benjamin Stephens (Mathematical Institute, University of Oxford)
AUTHORS Benjamin Stephens (Mathematical Institute, University of Oxford); Carles Falco (University of Oxford); Helen Byrne (University of Oxford); Ruth Baker (University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cell-proliferation dynamics shape the spatiotemporal organisation of developing and regenerating tissues, yet collective growth often emerges from heterogeneous cell-cycle behaviour at the single-cell level. In early animal embryos, initially synchronous divisions progressively lose synchrony after a well-characterised number of cycles, providing a setting to study how individual variability shapes population-level dynamics. We compare stochastic branching-process models of cell division with deterministic age-structured partial differential-equations formulations. We identify the assumptions of each framework and clarify how model choice affects biological interpretation, tractability, and suitability for different data types. We then introduce extensions incorporating cell-cycle variability and spatial structure. These spatial models explain proliferation patterns observed in large epithelial expansions visualised with FUCCI, and allow

us to estimate the length scale over which confluent cell cultures can be meaningfully described as synchronous.

MS151-06 — Understanding the role of space in modulating cellular heterogeneity

SPEAKERS Paras Jain (Department of Bioengineering, Indian Institute of Science)
AUTHORS Jason T. George (Department of Biomedical Engineering, Texas A&M University and Translational Medical Sciences, Texas A&M Health Science Center and Center for Theoretical Biological Physics, Rice University and Hematopoietic Biology and Malignancy, MD Anderson Cancer Center); Madhav B Nair (Department of Biomedical Engineering, Texas A&M University); Mohit Kumar Jolly (Department of Bioengineering, Indian Institute of Science); Paras Jain (Department of Bioengineering, Indian Institute of Science)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

The control of gene expression by epigenetic factors, along with gene expression noise, results in a distribution of cell states amongst genetically identical cells. Previous studies have explored the role of gene expression in proliferation and vice versa, which, in turn, shapes cellular heterogeneity within a population. However, in these studies, the population was assumed to be well-mixed. As crowding, migration, local interactions, and other factors are key features of spatial population dynamics, their consideration has important implications for cell growth and migration. Thus, space introduces additional complexity into the feedback between gene regulation and the population's growth rate, modulating cellular heterogeneity within a population with respect to non-spatial contexts. We have developed spatial agent-based models in which cells divide and migrate, along with non-spatial models as a control. The division and migratory rates of cells are governed by their cell states, and an underlying gene regulatory network models the dynamics of cell states. With the developed framework, we compare the roles of regulation and stochasticity in shaping population-level heterogeneity in spatial and non-spatial contexts, and highlight how non-genetic phenotypic selection operates differently in these contexts.

MS151-07 — A novel cellular automaton approach for modeling genotypic and phenotypic heterogeneity in cell systems

SPEAKERS Simon Syga (Center for Interdisciplinary Digital Sciences, TUD Dresden University of Technology)
AUTHORS Andreas Deutsch (Center for Interdisciplinary Digital Sciences, TUD Dresden University of Technology); Josué Manik Nava-Sedeño (Department of Mathematics, Universidad Nacional Autónoma de México); Simon Syga (Center for Interdisciplinary Digital Sciences, TUD Dresden University of Technology)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cellular automaton models have long been used to study cellular processes, but may be challenging for incorporating heterogeneity, migratory, and high-density effects. In this work, we introduce an extension of the classic lattice-gas cellular automata, a framework which allows to consider changes in cell numbers, cell–cell interactions, migration, and evolution of genotypic and phenotypic heterogeneity. To demonstrate the utility of our approach, we consider a growing population of cells whose genotype—passed on at birth from the mother cell and subject to stochastic mutations—determines their individual proliferation rates. Using spatial simulations, we show that the model exhibits traveling-wave invasion patterns, where the fastest-growing cells accumulate at the leading edge, accelerating population expansion. We predict this behavior using a mathematical analysis based on a mean-field assumption [SND26].

References

[SND26] Simon Syga, Josué Manik Nava-Sedeño, and Andreas Deutsch. "A novel cellular automaton approach for modeling genotypic and phenotypic heterogeneity in cell systems". In: *The European Physical Journal Special Topics* (2026). ISSN: 1951-6401. DOI: 10.1140/epjs/s11734-026-02186-1. URL: https://doi.org/10.1140/epjs/s11734-026-02186-1.

MS151-08 — A mathematical model of cell-state dynamics in melanoma

SPEAKERS Charlotte Taylor Barca (University of Manchester)
AUTHORS Charlotte Taylor Barca (University of Manchester); Gareth Wyn Jones (University of Manchester); Kerrie L. Marie (University of Manchester); Oliver E. Jensen (University of Manchester); Rotem Leshem (University of Manchester); Sarah Woolner (University of Manchester); Vishaka Gopalan (National Institutes of Health)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 02.01 - HS
TRACK Mathematical Oncology

Abstract

Melanoma cells can transition between cell states, contributing to therapy resistance and immune evasion. These state changes involve dynamic and reversible shifts in gene expression, making it essential to understand the underlying regulatory mechanisms for developing effective therapies. We present a mathematical model of a minimal gene regulatory network comprising key transcription factors associated with melanoma cell states. Using deterministic temporal and spatio-temporal differential equation models, we analyse gene expression dynamics and classify stable states in a biologically meaningful way. We exploit an approximation, based on cooperative binding of transcription factors, in which the models are piecewise smooth. At the population level, we use a naïve model of intercellular communication to explore how cells within a tumour can exhibit coordinated behaviour through travelling waves of gene expression. Additionally, we propose a method for deriving a condition that determines the final state of a population of communicating cells. This model provides a framework for better understanding some of the mechanisms driving gene expression dynamics and to inform and validate experimental hypotheses. Motivated by these results, we discuss a mechanical model of collagen-encased capsule formation in tumours.

MS155 — Computational Modeling of Cerebrovascular Dynamics

TRACK Cardiovascular Modelling
SCHEDULE Tuesday, 14 July 2026; 17:00–18:20; 01.15 - HS
ORGANISERS Laura Ellwein Fix (Virginia Commonwealth University)

Minisymposium abstract

Regulation of the dynamics of blood flow and pressure to, from, and within the brain is one of the most critical parts of sustaining human life. Disordered regulation in the cerebral (brain) vasculature has been associated with small vessel disease, stroke, brain tumors, traumatic brain injury, pharmaceutical use, and normal aging. In lieu of major illness, people with cerebrovascular disorders may still suffer from symptoms such as dizziness, syncope, and breathing instability that can affect quality of life. Accurately measuring cerebrovascular dynamics and surrogates of related morbidity continues to be a focus of clinical and experimental work, though it is inherently limited by practical and ethical concerns. As such the factors driving cerebrovascular dynamics, its regulation, and their manifestations are still not well understood. Computational modeling coupled with clinical data can help determine the factors most influencing dynamics and ultimately help predictions at the individual level, while providing a critical means to study fragile populations and address scenarios with a scarcity of data. This minisymposium will focus on recent mathematical and computational models of cerebrovascular dynamics that may help uncover possible mechanisms underlying various conditions.

MS155-04 — Eye2Heart: A reduced mathematical model bridging cardiovascular and ocular hemodynamics

SPEAKERS Marcela Szopos (Université Paris Cité)
AUTHORS Alon Harris (Icahn School of Medicine at Mount Sinai); Faith Hughes (University of Maine); Giovanna Guidoboni (University of Maine); Lorenzo Sala (Universite Paris-Saclay); Marcela Szopos (Université Paris Cité); Mohamed Zaid (Foresite Healthcare LLC); Sergey Lapin (Washington State University); Virginia Huxley (University of Missouri School of Medicine)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 01.15 - HS
TRACK Cardiovascular Modelling

Abstract

The cardiovascular and ocular systems are intricately connected, with hemodynamic interactions playing a crucial role in both physiological regulation and pathological conditions. However, existing models often treat these systems separately, thus limiting the understanding of their interdependence.

This talk presents the Eye2Heart model, which is a novel closed-loop mathematical framework that integrates cardiovascular and ocular dynamics [Sal+26]. Using an electrical-hydraulic analogy, the model describes the interactions between the heart and retinal circulation through a nonlinear system of ordinary differential equations. The model is tested against clinical and experimental data, thus demonstrating its ability to reproduce key cardiovascular parameters (e.g., stroke volume, cardiac output) and ocular hemodynamics (e.g., retinal blood flow). Additionally, we explore in silico the effects of intraocular pressure and left ventricular compliance on both local ocular and global systemic circulation, thus revealing critical dependencies between cardiovascular and ocular health. The results highlight the model's potential for studying cardiovascular diseases with ocular manifestations and support emerging research in oculomics by providing a mechanistic basis to interpret ocular biomarkers within a systemic context.

References

[Sal+26] Lorenzo Sala et al. "Eye2Heart: A reduced mathematical model bridging cardiovascular and ocular hemodynamics". In: *Mathematical Biosciences and Engineering* 23.2 (2026), pp. 421–448.

MS155-01 — An agent-based model of atherosclerotic plaque angiogenesis grounded in human spatial and single-cell data

SPEAKERS Katrin Schröder
AUTHORS Katrin Schröder
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 01.15 - HS
TRACK Cardiovascular Modelling

Abstract

Atherosclerotic plaques with similar luminal narrowing carry markedly different rupture risk, depending on fibrous-cap thickness, necrotic-core size, and the origin and maturity of intraplaque microvessels. We present a spatial agent-based model of a plaque-bearing arterial cross-section that couples the following mechanisms:

(i) hypoxia-driven foam-cell necrosis with variable efferocytic clearance, forming a necrotic core;

(ii) hypoxia-driven tip-cell/stalk-cell microvessel sprouting from adventitial vasa vasorum or luminal endothelium, where anastomosis confers perfusion; and

(iii) pericyte recruitment onto perfused vessels, which stabilizes the vessels and controls intraplaque hemorrhage.

(iv) A fibrous cap evolves from the balance of collagen synthesis and MMP degradation, and two-phase Glagov remodeling allows the plaque to narrow the lumen.

We calibrate cell composition and test the model's assumptions against human data: Visium spatial transcriptomics and two single-cell atlases provide healthy-versus-plaque cell proportions and show that the plaque core is uniquely hypoxic and pericyte-depleted. Simulations were used to track hypoxia, tip-cell, and pericyte-loss signatures in disease progression.

We found that efferocytosis controls necrotic-core content independently of cap thickness. Further our data suggest that pericyte coverage determines whether angiogenesis stabilizes or destabilizes a plaque and that luminal stenosis is decoupled from rupture vulnerability.

MS155-02 — Modelling Cerebral Autoregulation at the Microvascular Scale: From Health to Post-Stroke Dysfunction

SPEAKERS Chryso Lambride (University of Bern)
AUTHORS Chryso Lambride (University of Bern); Franca Schmid (University of Bern); Mohamad El Amki (University of Zurich); Susanne Wegener (University of Zurch)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 01.15 - HS
TRACK Cardiovascular Modelling

Abstract

Cerebral autoregulation stabilizes cerebral blood flow despite changes in blood pressure through adjustments in arterial diameter. However, the precise roles of individual vessels and impaired autoregulation in outcomes after ischemic stroke remain poorly understood. We present a computational framework incorporating static myogenic and endothelial regulation in large semi-realistic microvascular networks derived from mouse cortical vasculature (200,000 vessels). Arteries actively regulate their diameter, whereas capillaries and veins behave passively according to a pressure–area relationship. Changes in transmural pressure and shear stress modulate vascular wall stiffness and compliance, enabling active arterial diameter adaptation and linking local haemodynamic stimuli to vessel-level flow regulation. Autoregulatory responses were evaluated under three conditions: healthy regulation, post-occlusion reperfusion with altered vascular reactivity, and chronic autoregulatory dysfunction. Under healthy conditions, the simulations reveal the dominant role of surface arteries in buffering pressure changes. During reperfusion, impaired myogenic reactivity emerges as a key driver of post-stroke hyperperfusion. Progressive loss of autoregulation in arteries within the previously ischemic territory disrupts capillary perfusion. These results provide mechanistic insight into how microvascular structure and vessel-level regulation shape cerebral perfusion in health and after stroke.

MS155-03 — Mechanistic modeling of closed-loop cerebrovascular reactivity

SPEAKERS Laura Ellwein Fix (Virginia Commonwealth University)
AUTHORS Helen Harris (Virginia Commonwealth University); Laura Ellwein Fix (Virginia Commonwealth University)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 01.15 - HS
TRACK Cardiovascular Modelling

Abstract

Impaired cerebrovascular reactivity (CVR), the ability of cerebral blood vessel tone to respond to stimuli for regulating blood flow and metabolism in the brain, has been linked to many acute and chronic cardiovascular and neurological diseases. In one mechanism of cerebrovascular reactivity, the cerebral blood vessels dilate to lower resistance in response to increased arterial carbon dioxide (CO2), thereby increasing blood flow to wash out the CO2. However, the integration of this with other regulatory processes and the implications for the systemic circulation are still not fully understood. Since the ability to measure the cerebral vasculature is inherently limited, a computational model that incorporates multiple possible factors underlying cerebrovascular blood flow regulation with patient-specific noninvasive data can help determine what most influences dynamics and regulation at the individual level. We present a mechanistic representation of cerebrovascular resistance as a function of partial pressure of CO2 together with a first-order control equation within a closed-loop whole-body circulation framework. We also incorporate functional representations of additional systemic and cerebral responses to CO2 and pressure. The current model is calibrated against experimental blood flow, pressure, and end-tidal CO2 from a cohort of control subjects and patients during a CO2 rebreathing protocol. These model adaptations will improve understanding of the system-level integration of mechanisms underlying CVR.

MS157 — Integrating machine learning into mathematical oncology

TRACK Mathematical Oncology
SCHEDULE Tuesday, 14 July 2026; 17:00–18:20; 15.04 - HS

ORGANISERS Daniel Camacho-Gomez (Moffitt Cancer Center); Alexander R.A. Anderson (Moffitt Cancer Center)

Minisymposium abstract

Cancer is a complex multiscale disease characterized by heterogeneous cellular behaviors, nonlinear dynamics, and interactions across scales. Computational models provide a formal framework to study cancer progression, predict treatment response, and support translational research. However, significant challenges remain due to spatial and temporal scales, nonlinear behavior, and the high-dimensional, heterogeneous data that are difficult to integrate into mechanistic frameworks. Machine learning approaches easily integrate multi-modal data across diverse spatial and temporal scales to make classifications and predictions but often this comes at the cost of interpretability that is standard in mathematical oncology. This minisymposium aims to bring together researchers to discuss recent advances in integrating machine learning with mechanistic models for cancer research in (1) hybrid approaches combining mechanistic models and machine learning; (2) machine learning methods for calibration and adaptation of mechanistic models; (3) mechanistic learning approaches to improve interpretability, robustness, and generalization; and (4) machine learning models for knowledge discovery, pattern extraction, and guidance of new mechanistic models. Applications span in vitro, in vivo, and patient-specific digital twins for personalized cancer progression prediction, and treatment planning.

References

[Roc+26] Russell C. Rockne et al. "The future of mathematical oncology in the age of AI". en. In: *npj Systems Biology and Applications* 12.1 (Jan. 2026), p. 22. ISSN: 2056-7189. DOI: 10.1038/s41540-026-00656-9. URL: https://www.nature.com/articles/s41540-026-00656-9 (visited on 05/19/2026).

MS157-01 — LLM-Guided Mechanistic Model Discovery in Population Pharmacometrics

SPEAKERS Sébastien Benzekry (1. COMPutational pharmacology and clinical Oncology, Centre Inria d'Université Côte d'Azur 2. Cancer Research Center of Marseille, Institut Paoli-Calmettes, Inserm UMR1068, CNRS UMR7258, Aix Marseille University UM105, Marseille, France)

AUTHORS R. Ferrara (1. COMPutational pharmacology and clinical Oncology, Centre Inria d'Université Côte d'Azur 2. Cancer Research Center of Marseille, Institut Paoli-Calmettes, Inserm UMR1068, CNRS UMR7258, Aix Marseille University UM105, Marseille, France); M. Soucail (1. COMPutational pharmacology and clinical Oncology, Centre Inria d'Université Côte d'Azur 2. Cancer Research Center of Marseille, Institut Paoli-Calmettes, Inserm UMR1068, CNRS UMR7258, Aix Marseille University UM105, Marseille, France 3. Quantitative Pharmacology, Translational Medicine, Servier, France); Sébastien Benzekry (1. COMPutational pharmacology and clinical Oncology, Centre Inria d'Université Côte d'Azur 2. Cancer Research Center of Marseille, Institut Paoli-Calmettes, Inserm UMR1068, CNRS UMR7258, Aix Marseille University UM105, Marseille, France)

SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.04 - HS

TRACK Mathematical Oncology

Abstract

Structural model selection for pharmacokinetics (PK) and tumor dynamics (TD) is iterative and expert-driven, requiring ODE formulation, nonlinear mixed-effects fitting, and biological plausibility assessment. We present an LLM-agent framework for automated population ODE model discovery, fit locally via SAEM (Monolix).

The workflow iterates: a builder agent proposes candidate ODE systems in MLXTRAN; a diagnostic agent interprets goodness-of-fit metrics (BICc, RSEs, IWRES); a reflection agent selects the best structure. No patient data are sent to the LLM.

For PK discovery (synthetic benchmark, n=10), 7/10 ground-truth structures were identified, including 4/4 one-compartment models. On real clinical data (Theophylline, Warfarin, Docetaxel, Irinotecan), AI-selected models matched or outperformed published references.

For TD (synthetic and real preclinical/clinical datasets), canonical growth models (exponential, logistic, Gompertz) were recovered alongside drug-effect structures. Exact model recovery occurred in 50% of six PK-TD scenarios; biologically plausible alternatives emerged otherwise.

Runtimes remained under 20 minutes. This framework offers a tractable, reproducible assistant for mechanistic model discovery in mathematical oncology, complementing expert judgment.

MS157-02 — Deep Reinforcement Learning for Sequential Decision-Making in a Polytherapeutic Cancer Landscape

SPEAKERS Alvaro Köhn-Luque (1 Oslo Centre for Biostatistics and Epidemiology, Faculty of Medicine, University of Oslo 2 Department of Medical Genomics, Oslo University Hospital)

AUTHORS Alvaro Köhn-Luque (1 Oslo Centre for Biostatistics and Epidemiology, Faculty of Medicine, University of Oslo 2 Department of Medical Genomics, Oslo University Hospital)

SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.04 - HS

TRACK Mathematical Oncology

Abstract

Drug resistance remains a primary obstacle in oncology, transforming clinical management into a complex, sequential decision-making problem. While Reinforcement Learning (RL) has shown promise in optimizing adaptive dosing for single agents, its application to large polytherapeutic panels—where clinicians choose from numerous drugs with overlapping resistance profiles—remains underexplored. This gap is exacerbated by the lack of mechanistic models capturing how drug sensitivity is shared across diverse agents due to cell subpopulation overlap.

In this talk, I will present a deep RL framework designed to optimize treatment strategies within a broad therapeutic panel. Inspired by the cyclic response-and-relapse dynamics of multiple myeloma, a mathematical model of tumor evolution is developed to track the competition between sensitive and resistant subpopulations. Crucially, a statistical model is integrated to encode correlations in drug sensitivity profiles, offering a conceptual approach to account for the biological reality of cross-resistance. Using these environments, a Proximal Policy Optimization (PPO) agent is trained to navigate the high-dimensional decision landscape. The agent learns to select drug sequences that adaptively respond to the evolving virtual tumor, aiming to minimize long-term tumor burden. Results demonstrate that RL can identify non-intuitive sequencing strategies that outperform standard clinical heuristics, providing a promising proof-of-concept for the future of AI-driven personalized medicine.

MS157-03 — Towards personalized glioblastoma radiotherapy using growth modeling and recurrence prediction

SPEAKERS Lucas Zimmer (1AI for Image-Guided Diagnosis and Therapy, Technical University of Munich, Germany)

AUTHORS Lucas Zimmer (1AI for Image-Guided Diagnosis and Therapy, Technical University of Munich, Germany); Jonas Weidner (1AI for Image-Guided Diagnosis and Therapy, Technical University of Munich, Germany 2Munich Center for Machine Learning (MCML)); Michal Balcerak (3Department of Quantitative Biomedicine, University of Zurich, Switzerland); Florian Kofler (3Department of Quantitative Biomedicine, University of Zurich, Switzerland 4Helmholtz AI, Helmholtz Zentrum München, Germany); Mara Krupa (1AI for Image-Guided Diagnosis and Therapy, Technical University of Munich, Germany); Ivan Ezhov (5AI in Healthcare and Medicine, Technical University of Munich, Munich, Germany); Santiago Cepeda (6Department of Neurosurgery, Río Hortega University Hospital, Valladolid, Spain 7Specialized Group in Biomedical Imaging and Computational Analysis (GEIBAC), Instituto de Investigación Biosanitaria de Valladolid (IBioVALL), Valladolid, Spain); Ray Zhang (8Department of Mathematical Sciences, Worcester Polytechnic Institute, USA); John Lowengrub (9Department of Mathematics, University of California, Irvine, USA 10Department of Biomedical Engineering, University of California, Irvine, USA); Bjoern Menze (3Department of Quantitative Biomedicine, University of Zurich, Switzerland); Benedikt Wiestler (1AI for Image-Guided Diagnosis and Therapy, Technical University of Munich, Germany 2Munich Center for Machine Learning (MCML))

SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.04 - HS

TRACK Mathematical Oncology

Abstract

Glioblastoma remains one of the greatest challenges in oncology, with near-universal recurrence largely driven by diffuse tumor infiltration beyond radiologically visible tumor margins, yet current radiotherapy planning relies on uniform geometric expansions that ignore patient-specific tumor biology and anatomy.

Computational growth models and machine learning approaches have the potential to estimate these invisible tumor extensions and guide personalized radiotherapy planning, but their clinical translation has been limited by a lack of standardized benchmarking datasets and evaluation frameworks.

In this talk, I present current approaches to tumor growth modeling and recurrence prediction, including a novel U-Net-based model, and compare their performance against the current standard of care for radiotherapy planning. To this end, I introduce PREDICT-GBM, an end-to-end platform and curated dataset for evaluating computational models of glioblastoma growth and recurrence prediction. The results demonstrate that both biophysical and deep-learning approaches significantly outperform standard-of-care protocols in predicting future recurrence. Finally, I discuss what these comparisons reveal about the strengths and limitations of biophysical and data-driven approaches for guiding personalized radiotherapy, and outline the next steps toward clinical integration.

MS157-04 — Image-based prediction of skin cancer evolution for clinical decision support through machine learning

SPEAKERS Daniel Camacho-Gomez (Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center, Tampa, FL 33612)
AUTHORS Daniel Camacho-Gomez (Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center, Tampa, FL 33612); Alexander R.A. Anderson (Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center, Tampa, FL 33612)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Skin cancer is among the most prevalent cancers worldwide, and current monitoring relies on biweekly image-based follow-ups that visually compare lesion changes over time. Although effective, this strategy is reactive and offers limited ability to anticipate future lesion behavior. To address this gap, we propose a predictive framework that leverages routinely collected clinical images to forecast lesion evolution. The approach combines a convolutional autoencoder, which encodes lesion images into a compact latent representation capturing key visual features, with a machine learning model that models temporal changes within this latent space. The decoder reconstructs images from latent vectors, enabling generation of future lesion appearances. Analysis of the latent space revealed structured, interpretable representations of lesion dynamics. From these, quantitative metrics such as trajectory length, progression speed, and cluster transition frequency were derived. Notably, these descriptors effectively differentiated stable lesions from rapidly evolving ones and captured patient-specific progression patterns. Lesions with higher latent speeds and more frequent cluster transitions showed greater morphological change over time. Overall, this framework provides a quantitative basis for anticipating lesion progression, supporting more proactive and personalized treatment decisions, including therapy initiation, adjustment, and response prediction.

MS177 — Mathematical and Experimental Approaches to Retinal Degeneration and Visual Restoration

TRACK Miscellaneous
SCHEDULE Tuesday, 14 July 2026; 17:00–16:30; 15.02 - HS, 01.22 - HS
ORGANISERS Erika Camacho (The University of Texas at San Antonio); Stephen Wirkus (The University of Texas at San Antonio)

Minisymposium abstract

The retina is a complex neural network that transforms incoming light into neural signals that are transmitted to the brain. Understanding retinal function in normal and pathological conditions provides insights for intervening in degenerative processes and restoring vision. In recent years, mathematical approaches have come to play an increasingly prominent part in efforts to understand how the retina responds in diseased conditions, evaluate proposed treatments, and predict outcomes following novel interventions. This minisymposium considers a range of approaches to investigate retina function: analyses of photoreceptor decline as seen in inherited and age-related conditions; explorations of how metabolic

changes and intracellular feedback might influence survival or loss of these cells; and practical modeling that informs the development of therapies. The session shows how mathematical modeling and experiments can inform each other, clarify complex biological interactions, and ultimately add to the strategies available for preserving vision.

MS177-03 — Numerical Modelling for Glucose Metabolization in the Retina

SPEAKERS Kohl James (Saginaw Valley State University)
AUTHORS Atanaska Dobreva (Augusta University); Erika Camacho (The University of Texas at San Antonio); Kohl James (Saginaw Valley State University)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.02 - HS
TRACK Miscellaneous

Abstract

The buildup of toxic reactive oxygen species (ROS) is a significant contributor to retinal degeneration and cellular dysfunction. It has been shown that the glutathione (GSH) antioxidant system and nicotinamide adenine dinucleotide phosphate (NADPH) play an important role in the detoxification of ROS. Existing models describe these dynamics with system of ordinary differential-algebraic equations. However, ROS diffuses across retinal cells, which is not addressed in existing models. Understanding the spatial behavior of ROS could inform treatment strategies, but doing so requires the use of more advanced numerical tools. In this presentation, we address challenges and strategies for domain meshing, temporal discretization, and computational expense.

MS177-05 — Mathematical Models of Retinal Drug Delivery

SPEAKERS Paul Roberts (City St George's, University of London)
AUTHORS Chloe N. Thomas (University of Birmingham); Gabriel Bellamy Plaice (University of Birmingham); James Andrews (University of Birmingham); James. Roberts (University of Birmingham); Lisa Hill (University of Birmingham); Marie-Christine Jones (University of Birmingham); Paul Roberts (City St George's, University of London)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.02 - HS
TRACK Miscellaneous

Abstract

Wet age-related macular degeneration (AMD) causes vision loss when vascular endothelial growth factor (VEGF) stimulates blood vessel growth into the light-sensitive retina. Anti-VEGF treatments such as ranibizumab are currently administered to treat wet AMD via intravitreal injections, which are unpleasant, expensive and risk complications. We explored the efficacy of topically administered ranibizumab, with cell penetrating peptides (CPPs).

Ex vivo pig eyes were divided into 3 groups and treated with 1. topical or 2. intravitreal ranibizumab and CPP, or 3. intravitreal ranibizumab. ELISAs measured ranibizumab and VEGF concentrations in the aqueous and vitreous at 20 min, 40 min, 1 hr and 3.5 hr (n = 3, per group). An ordinary differential equation model was formulated to describe the evolving concentrations of ranibizumab, VEGF and their compounds in the tear, aqueous and vitreal compartments.

CPP allows topical ranibizumab to penetrate the cornea but reduces ranibizumab availability and efficacy in neutralising VEGF for intravitreal treatment. Topical treatment may provide sustained, moderate suppression of vitreal VEGF levels, while intravitreal treatment provides strong suppression which lessens between treatments. Combined intravitreal/topical treatment presents a promising approach. Treatment efficacy would be enhanced if ranibizumab's rate of binding to VEGF or tear residence time could be increased.

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MS177-06 — Mathematical analysis of photoreceptor changes in conditions of separation from the underlying retinal pigment epithelium

SPEAKERS Stephen Wirkus (The University of Texas at San Antonio)
AUTHORS Stephen Wirkus (The University of Texas at San Antonio); Erika Camacho (The University of Texas at San Antonio); Miriam Goldman (University of California-San Francisco); Lucia Gonzalez-Buendia (Puerta de Hierro-Majadahonda University Hospital); Daniel Maidana (Illinois Eye and Ear Infirmary, University of Illinois Chicago); Demetrios Vavvas (Department of Ophthalmology Harvard Medical School)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.02 - HS
TRACK Miscellaneous

Abstract

Photoreceptors (PR) are responsible for absorbing and converting light into electrical signals necessary to create vision. To accomplish that, they are in an intimate relationship and attached to the underlying retinal pigment epithelium (RPE). Separation of the PR from the underlying RPE is seen in many retina disorders and leads to PR cell death and subsequent vision loss. Separation of PR from the RPE (detachment) interferes with the normal phagocytosis and recycling of PR outer segments by the RPE, and disrupts nutrient and metabolite delivery, including glucose. All these factors contribute to cell dysfunction and eventual cell death. In this work, we sought to understand the importance of different factors in PR cell loss after detachment using mathematical modeling. We used known information of photoreceptor interactions in the healthy retina from the literature, and datasets for rod and cone degeneration after detachment. We also included three additional published datasets of PR cell death kinetics. A mathematical sensitivity analysis examined the impact of the parameters on the system over a detachment of 150 days and found that the parameters that significantly impact the rod and the cone population at the stages where "early intervention" happens (3-7 days after detachment) are not always the same ones that significantly impact the populations at 150 days. Additionally, some of the parameters negatively affected one population while having the opposite effect on the other. An increase in nutrient availability and efficiency of rod energy uptake were the only parameters that did not have a negative effect on either population. Similar results were obtained for reattachment. The interplay between these variables indicates that effective photoreceptor neuroprotection in retinal detachment may require multiple strategies. The prediction of these impactful parameters over time can be further assessed in experimental models and may provide guidance on the most effective ways to improve PR survival after their separation from RPE.

MS177-07 — Predicting the efficacy of optogenetic strategies for vision restoration

SPEAKERS Chiara Boscarino (Institut de la vision)
AUTHORS Chiara Boscarino (Institut de la vision); Marco Rucli (Institut de la vision); Olivier Marre (Institut de la vision); Ulisse Ferrari (Institut de la vision)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.02 - HS
TRACK Miscellaneous

Abstract

Optogenetic gene therapy enabled partial functional vision restoration in patients with retinal degeneration, but the achieved visual acuity remains below the threshold of legal blindness. Various modifications to these therapies have been proposed to improve acuity. However, existing reports typically quantify the light sensitivity of reactivated retinas, but rarely provide corresponding estimates of visual resolution, making it difficult to predict which strategies are most suitable for clinical translation. To address this gap, we developed a computational framework that translates electrophysiological recordings from optogenetically treated retinas into predictions of achievable visual resolution. Starting from a single-cell model fitted to

MEA recordings of treated rd1 mouse retinas, we build a digital retinal population and simulate visual acuity tests. Maximum-likelihood decoding then provides an upper bound on achievable acuity for each strategy. This framework enables systematic comparison across optogenetic strategies, quantifying the impact of opsin kinetics on acuity and the potential benefit of bipolar-cell targeting.

MS177-04 — Retinal processing of natural scenes

SPEAKERS Olivier Marre (Institut de la vision)
AUTHORS Baptiste Lorenzi (Institut de la vision); Matias Goldin (Institut de la vision); Olivier Marre (Institut de la vision); Rémi Baroux (Institut de la vision); Samuele Virgili (Institut de la vision); Simone Azeglio (Institut de la vision)
SCHEDULE Thursday, 16 July 2026; 15:10–15:50; 01.22 - HS
TRACK Miscellaneous

Abstract

While a great deal is known about how neurons of the early visual system respond to simple stimuli, our understanding of how they process natural stimuli is still limited. Machine learning models have been invaluable to predict how these neurons respond to natural stimuli. However, the increasing complexity of these models make them difficult to interpret, and their ability to generalize to new stimuli is limited. I will describe recent works from my lab where we address these issues at the level of the retina. We developed a new perturbative approach to probe the selectivity of individual neurons during natural scenes, and to understand the features they extract. We use this method to characterize and model how short-term adaptation impact how the retina processes natural scenes. Our results show that adaptive mechanisms are not just here to normalize the response to the average luminance and contrast: during natural scene stimulation it also reshapes the selectivity of specific types of ganglion cells. Finally, we show that the ability of artificial neural networks to generalize can be dramatically enhanced in specific ganglion cell types by incorporating in the model a new geometry constraint derived from a natural hunting behavior. Together our results suggest that scaling up model size may not be enough: including knowledge about the biological basis of visual processing in artificial neural network models might be necessary to understand sensory processing.

MS177-01 — A Mathematical Framework for Photoreceptor Dynamics in Health and Disease: Modeling Metabolic Mechanisms and Spatial Dependence

SPEAKERS Danielle Brager (District of Columbia Public Schools)
AUTHORS Danielle Brager (District of Columbia Public Schools); Erika Camacho (The University of Texas at San Antonio); Stephen Wirkus (The University of Texas at San Antonio); Anthony Kearsley (National Institute of Standards and Technology); Daniel Anderson (George Mason University)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 01.22 - HS
TRACK Miscellaneous

Abstract

Retinitis Pigmentosa (RP) is a group of genetically heterogeneous retinal diseases characterized by the progressive loss of rod and cone photoreceptors, leading to irreversible blindness. While genetic mutations in RP primarily affect rods, secondary cone degeneration inevitably follows. This phenomenon is linked to the loss of rod-derived cone viability factor (RdCVF), a rod-secreted protein that stimulates aerobic glycolysis in cones to support the metabolic demands of outer segment (OS) renewal. To understand these complex mechanisms, we use mathematical models to investigate the dynamics governing photoreceptor dynamics. We first developed a system of nonlinear ordinary differential equations to model RP progression. Through stability, global sensitivity, and bifurcation analyses, we identified key parameters driving the transition from health to blindness. Our results interpret model equilibria as distinct degenerative states and reveal that stable limit cycles emerge at critical junctions, suggesting specific windows for therapeutic intervention. We find that the balance between OS shedding and renewal is vital for cone preservation. Next, we developed a spatially-dependent model to include photoreceptor density distributions and nutrient diffusion. The model was validated against experimental data, accurately predicting OS length

and regrowth. These findings establish a foundation for exploring various retinal pathologies and spatial treatment strategies.

MS177-02 — A Dual-Intervention Optimal Control Framework for Photoreceptor Degeneration Using RdCVF and RdCVFL

SPEAKERS Erika Camacho (The University of Texas at San Antonio)
AUTHORS Erika Camacho (The University of Texas at San Antonio); Kathryn Wifvat (University of the Cumberlands); Matthias Kawski (Arizona State University); Thierry Léveillard (Institut de la vision); Stephen Wirkus (The University of Texas at San Antonio)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 01.22 - HS
TRACK Miscellaneous

Abstract

Rod and cone photoreceptors are among the most metabolically active cells in the human body, relying on tightly regulated redox homeostasis to maintain function. In retinal degeneration resulting from chronic blue light exposure or inherited degenerative conditions such as retinitis pigmentosa (RP), the progressive loss of photoreceptors disrupts this balance, leading to an accumulation of reactive oxygen species (ROS) that accelerates further cell death. Two proteins produced by rod photoreceptors have emerged as promising therapeutic targets. The rod-derived cone viability factor (RdCVF) promotes cone survival by enhancing glucose uptake and supporting aerobic glycolysis. Its long isoform (RdCVFL) protects both rods and cones by regulating the redox environment. While the therapeutic potential of each protein has been investigated independently, their combined effect has not yet been examined in a unified mathematical framework. RdCVF and RdCVFL act on distinct but coupled biological processes, targeting metabolic support and oxidative stress regulation, respectively. A dual-intervention strategy may therefore offer substantially greater neuroprotection than either treatment alone. In this work, we develop and analyze an optimal control model incorporating both RdCVF and RdCVFL as co-administered treatments. This provides a mathematical basis for evaluating the synergistic potential of this dual-intervention approach in slowing or halting photoreceptor degeneration arising from chronic blue light exposure, RP, and related retinal diseases.

MS12 — Inference, Calibration, and Sensitivity in Agent-Based Models: A Data-Centric View

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 15.06 - HS
ORGANISERS Harsh Jain (University of Minnesota Duluth); Tracy Stepien (University of Florida)

Minisymposium abstract

Agent-based models (ABMs) are a natural platform to capture the complexity inherent in multiscale biological systems. However, the impact of mathematical modeling with ABMs remains limited by persistent challenges in model calibration, sensitivity analysis, and uncertainty quantification. Difficulties in integrating experimental data with models and the computational cost of simulating ABMs leads to challenges in rigorous comparison and prediction. This minisymposium will highlight an assortment of methods for analyzing data-driven ABMs applied to various biological settings ranging from cancer growth, ecological population dynamics, and epidemiology. Our speakers span a range of career stages, from graduate students to full professors, and reflect a diversity of identities, perspectives, and geographical locations.

MS12-01 — From Sparse Data to Smart Decisions: Surrogate-Assisted Agent-Based Modeling for Regional Outbreak Response

SPEAKERS Carson Dudley (University of Michigan)

AUTHORS Carson Dudley (University of Michigan); Daniel Bergman (University of Maryland Baltimore); Erica Rutter (University of California, Merced); Harsh Jain (University of Minnesota Duluth); Kerri-Ann Norton (Bard College); Trachette Jackson (University of Michigan)

SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.06 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Effective disease outbreak response requires actionable, region-specific guidance, but most modeling tools rely on detailed surveillance or strong assumptions, such as random mixing. Agent-based models (ABMs) allow us to capture key heterogeneity in contact patterns and intervention mechanisms, but linking these models with data is often computationally intractable, particularly at the larger scales needed for decision-making (e.g., county- or state-level). We present a simulation-based framework that combines ABMs with surrogate modeling to infer key transmission and severity parameters using only routine case and hospitalization data. This enables local health agencies to evaluate candidate interventions while explicitly accounting for uncertainty. Applied to COVID-19 in Michigan counties, our method recovers core parameters (transmissibility, latent period, asymptomatic transmissibility, underreporting, hospitalization risk, and duration) that align with empirical estimates, while demonstrating regional variation linked to age and comorbidity patterns. We find that intervention effectiveness cannot be reliably predicted from simple demographic predictors such as age structure, population density, or workforce participation. While school closures aligned with child population in some settings, other interventions showed weak or counterintuitive relationships with demographics. Traditional ODE models with random mixing assumptions cannot capture how interventions target specific contact networks, making it impossible to assess whether demographic proxies predict intervention success. Our framework addresses this gap by explicitly modeling intervention mechanisms within heterogeneous contact structures. Solely using routine case and hospitalization data, our method enables practical, uncertainty-aware decision support for local health agencies facing COVID-19, influenza, RSV, or future novel pathogens.

MS12-02 — Quantifying Uncertainty of ABM Parameters Using Explicitly Formulated Surrogate Models

SPEAKERS Kerri-Ann Norton (Bard College)

AUTHORS Daniel Bergman (University of Maryland Baltimore); Harsh Jain (University of Minnesota Duluth); Kerri-Ann Norton (Bard College); Trachette Jackson (University of Michigan)

SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.06 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Agent-based models (ABMs) have become valuable tools for understanding complex systems in biology and medicine. In order to evaluate the robustness and accuracy of the model predictions, uncertainty quantification using global sensitivity analysis should be performed. Unfortunately, most global sensitivity analyses are computational prohibitive for complex ABMs. By leveraging explicitly formulated surrogate models, we develop an efficient and flexible framework for inferring global sensitivity. We evaluate our methodology on two AMBs, one a simple 2D in vitro cell proliferation assay model and a second, more complex ABM of 3D vascular tumor growth. In these models, we simulate cells in their environment as decision making “agents” that have their own individual properties and interactions between agents, both temporally and spatially. In this talk, we show that our method for uncertainty quantification is comparable with other methods in accuracy, such as Morris one-at-a-time method or eFAST, but substantially speeds up the time it takes to complete the analysis from days to minutes. In addition, our method is able to estimate sensitives of ABM parameters that are not explicitly included in the surrogate model.

MS12-03 — Parameter-wise predictions and sensitivity analysis for random walk models in the life sciences

SPEAKERS Matthew Simpson (Queensland University of Technology)

AUTHORS Matthew Simpson (Queensland University of Technology)

SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.06 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Sensitivity analysis characterizes input–output relationships for mathematical models and has been widely applied to deterministic models across many applications in the life sciences. In contrast, sensitivity analysis for stochastic models has received less attention, with most previous work focusing on well-mixed, non-spatial problems. For explicit spatiotemporal stochastic models, such as random walk models (RWMs), sensitivity analysis has received far less attention. Here we present a new type of sensitivity analysis, called parameter-wise prediction, for two types of biologically motivated and computationally expensive RWMs. To overcome the limitations of directly analyzing stochastic simulations, we employ continuum-limit partial differential equation (PDE) descriptions as surrogate models, and we link these efficient surrogate descriptions to the RWMs using a range of physically motivated measurement error models. Our approach is likelihood-based, which means that we also consider likelihood-based parameter estimation and identifiability analysis along with parameter sensitivity. Our workflow illustrates how different process models can be combined with different measurement error models to reveal how each parameter impacts the outcome of the expensive stochastic simulation. Open-access software to replicate all results is available on GitHub.

MS12-04 — Probabilistic Estimation of Agent Based Models using Hamiltonian Monte Carlo

SPEAKERS Benjamin Olsson (Uppsala University)
AUTHORS Benjamin Olsson (Uppsala University); Måns Magnusson (Uppsala University); Sara Hamis (Uppsala University)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Agent-based models (ABMs) are increasingly used to study complex systems in biology, enabled by advances in computing and the growing availability of high-resolution data tracking individual agents. However, fitting ABMs to data remains challenging because their likelihood functions are typically intractable, making standard statistical methods such as maximum likelihood estimation or Markov chain Monte Carlo difficult to apply. Likelihood-free inference approaches, including approximate Bayesian computation and synthetic likelihood [Woo10; Pri+18], address this issue by relying on model simulations. Recent work has explored replacing these simulations with learned emulators [JC24; MW14]. In this work, we exploit the differentiability of such an emulator within the synthetic likelihood framework, enabling efficient parameter inference using Hamiltonian Monte Carlo with the No-U-Turn Sampler [HG14]. Using toy models with known likelihoods, we demonstrate the empirical accuracy of our approach. We further show that it improves parameter inference in complex ABMs compared to common likelihood-free inference methods that do not provide a gradient with respect to the parameters, and we investigate the asymptotic accuracy of the proposed emulator.

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MS15 — Mathematical Modeling of Oncolytic Viral Therapy in Solid Tumors

TRACK Mathematical Oncology
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 15.04 - HS
ORGANISERS Arwa Baabdulla (United Arab Emirates University)

Minisymposium abstract

Oncolytic virotherapy has emerged as a promising modality for treating solid tumors by exploiting viruses that selectively infect and lyse cancer cells while stimulating antitumor immune responses. Mathematical modeling plays a pivotal role in understanding these multiscale and nonlinear biological dynamics. This minisymposium will focus on recent advances in predictive mathematical and computational modeling of oncolytic viral infection in tumor tissues. Key topics include the quantitative analysis of immune–virus–tumor interactions, spatial modeling of viral spread in heterogeneous tumor microenvironments, and data-driven approaches for parameter estimation, treatment planning, and optimal dosing strategies. By integrating theoretical analysis with experimental and clinical findings, these models can guide treatment design, improve dosing strategies, and identify key mechanisms influencing therapeutic success. This session aims to foster collaboration across mathematics, oncology, and virology, and to highlight how model-based predictions can accelerate the translation of oncolytic virotherapy into effective clinical applications. The thematic track for this minisymposium is Mathematical Oncology.

MS15-01 — Mathematical Modeling and Analysis of Oncolytic Virus and CAR-T Cell Combination Therapy via Switched Optimal Control

SPEAKERS Arwa Baabdulla (United Arab Emirates University)
AUTHORS Abdessamad Tridane (United Arab Emirates University); Arwa Baabdulla (United Arab Emirates University); Udhayakumar Kandasamy (United Arab Emirates University)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.04 - HS
TRACK Mathematical Oncology

Abstract

The integration of oncolytic virotherapy and chimeric antigen receptor T-cell (CAR-T) therapy has emerged as a promising strategy for enhancing anti-tumor efficacy in the treatment of solid tumors. Oncolytic viruses selectively infect and destroy cancer cells while simultaneously modifying the tumor microenvironment, potentially improving CAR-T cell infiltration and activity. Understanding the complex interactions among tumor cells, oncolytic viruses, and CAR-T cells is essential for designing effective treatment protocols.

In this work, we develop and analyze a mathematical model describing the dynamics of tumor progression under combined oncolytic virus and CAR-T cell therapy. The model incorporates key biological mechanisms, including viral infection and replication, tumor cell lysis, and CAR-T cell expansion and persistence. To optimize therapeutic outcomes while minimizing treatment burden and adverse effects, we formulate a switched optimal control problem in which treatment interventions are administered according to time-dependent switching strategies.

MS15-02 — Resistance and immune response during oncolytic virotherapy

SPEAKERS Darshak Bhatt (University of Groningen Medical Center, University of Groningen)
AUTHORS BN Hoogeboom (University Medical Center Groningen, University of Groningen); Darshak Bhatt (University of Groningen Medical Center, University of Groningen); F.J. Weissing (University of Groningen); L. Horvathov (University Medical Center Groningen, University of Groningen); N. van Montfoort (Leiden University Medical Center); P. Kinderman (Leiden University Medical Center); T. Daemen (University Medical Center Groningen, University of Groningen); T. Janzen (University of Groningen)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Oncolytic viruses interact dynamically with cancer cells, healthy stromal cells, and immune cells in the tumor microenvironment. Therapeutic success therefore depends on the balance between viral spread, infection resistance, and immune activation. In this talk, I will present insights into these interactions using a spatiotemporal computational modeling framework. Our simulations reveal that therapy failure can arise through multiple mechanisms. For example, even when infection-resistant cancer cells are initially rare, spatial competition can lead to their rapid expansion and tumor persistence. I also find that fast killing of infected cells can paradoxically reduce efficacy by clearing the virus before it spreads, while infection-resistant stromal cells, like macrophages, can further inhibit infection and protect the tumor. Additionally, incorporating immune responses uncovers a fundamental trade-off. Virus-induced signals can enhance T cell-mediated cytotoxicity, but may simultaneously suppress viral amplification by eliminating infected cells too quickly. This leads to stochastic and sometimes counterintuitive outcomes, where treatment timing and delivery strongly affect success. Finally, I will show that macrophages, despite limiting infection, do not necessarily prevent effective therapy. Instead, rationally-engineered viruses to stimulate T cell responses can induce robust anti-tumor immunity even when infection remains spatially constrained. Together, these results highlight how resistance and immune dynamics jointly shape virotherapy outcomes.

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MS15-03 — Cell evolution in the presence of viruses

SPEAKERS Dominik Wodarz (University of California, San Diego)
AUTHORS Dominik Wodarz (University of California, San Diego)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Viral infection can substantially alter the selection of mutant cells, particularly in spatially structured settings. Understanding how infection shapes evolutionary dynamics is relevant both for cancer cells targeted by oncolytic viruses and for bacteria subject to bacteriophage infection. While cellular resistance to infection is one important dimension of this problem, I focus here on advantageous and deleterious mutants that remain equally susceptible to infection, such as drug-resistant cancer cell mutants.

I first quantify how selection is modified in spatially structured cell populations subject to virus infection, using mutant fixation probabilities as the key metric. I show that infection substantially weakens selection: advantageous mutants become less advantageous, and deleterious mutants become less deleterious. This effect is driven by spatial patterns that emerge from the infection process itself. I then turn to expanding cell colonies and show that infection can either increase or decrease the number of mutants in the expanding population, depending on assumptions about mutant fitness.

Together, these results illuminate how viral infection reshapes evolutionary dynamics in spatially structured cell populations , with direct implications for understanding how the presence of a virus alters the clonal composition of a tumor and its subsequent response to chemotherapy or targeted therapies.

MS15-04 — Computational investigation of the interactions between oncolytic viruses and tumour-associated macrophages

SPEAKERS Raluca Eftimie (University of Franche-Comte)
AUTHORS Raluca Eftimie (University of Franche-Comte)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.04 - HS
TRACK Mathematical Oncology

Abstract

The interactions between oncolytic viruses (OVs) and innate immunity are crucial for the success of oncolytic therapies. Here we focus on a heterogeneous and plastic population of innate immune cells, the tumour-associated macrophages, that can be involved in the elimination of OVs, as well as in the replication of OVs and their spread across tumour tissue. Computational approaches are used to investigate the importance of different biological mechanisms in the faster spread of OVs, as helped/hindered by different macrophage phenotypes.

MS23 — Optimal Control and Numerical Simulation Methods for Disease Tracking, Diagnosis and Treatment

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 15.05 - HS
ORGANISERS Anthony Kearsley (National Institute of Standards and Technology)

Minisymposium abstract

Mathematical modeling and numerical simulation are essential tools for understanding disease dynamics and treatment strategies. Biological systems underlying the important tasks of disease detection, predicting transmission and treatment are inherently complex, spanning multiple spatial and temporal scales and requiring advanced computational strategies as well as deep area-specific understanding in fields such as physics, engineering, medicine, public health, and mathematical biology. This minisymposium will highlight recent advances in applied mathematical biology modeling and simulation, including studies of antibody kinetics from structured populations, multiscale modeling frameworks for optimizing cancer therapies, and medical device optimization for personalized medicine with results of interest to applied mathematicians, computational scientists, and biomedical researchers.

MS23-01 — Modeling of DNA origami electrochemical signaling for optimal biosensor design

SPEAKERS Allison Carson (National Institute of Standards and Technology)
AUTHORS Allison Carson (National Institute of Standards and Technology)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Biological field effect transistors (Bio-FETs) have shown great promise in revolutionizing diagnostic testing, enabling the development of inexpensive and portable biosensors with a very low limit of detection that are capable of providing point-of-care diagnostics within minutes. A recent biosensor design using DNA origami nanostructures has shown unique potential, as the nanostructure's strong negative charge and low relative permittivity is able to greatly affect the electric field within the device to control sensor signaling. This talk will develop a mathematical model and simulation of this electrochemical process, in pursuit of determining optimal nanostructure designs. The model is posed as a modification to the Poisson-Boltzmann equation and a finite difference method approximates its solution. Parameter studies quantifying the effect of numerical and physical variations in the system are used to inform choices on computational parameters. Three different nanostructure designs are studied and their capacity for signaling compared, with optimal bulk salt concentrations suggested.

MS23-02 — Restoring immune regulation in cancer: a multifaceted optimization problem

SPEAKERS Anne Talkington (University of Buffalo)
AUTHORS Anne Talkington (University of Buffalo)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The maintenance of healthy immune function requires a complex set of checkpoints to promote a response to genuine threats (such as cancer or infection) while recognizing benign actors to avoid chronic systemic inflammation. This process leads to a delicate balance in which stimulatory and inhibitory receptors compete to dictate a cell's fate. Cancer cells notoriously ramp up inhibitory signaling, resulting in a lack of immune response. We explore the process of T cell exhaustion, or loss of ability to respond to a threat. Notably, we characterize the progression of T cells through their initial stimulus, activation, and subsequent exhaustion upon the earliest stages of solid tumor infiltration. We quantify, through experimental data and mechanistic modeling, activation and exhaustion scores for T cells upon encountering cancer cells. Introducing macrophages to the system presents an additional layer of complexity due to their highly plastic phenotypes, i.e., their ability to take on both an immunostimulatory and an immunosuppressive role. By characterizing T cell and macrophage activity at high resolution, we identify potential perturbations to leverage in optimizing treatment strategies for restoring a dysregulated anti-tumor immune response to a functional therapeutic state.

MS23-03 — Optimal and Predictive Control of Acute Myeloid Leukemia with Drug Resistance

SPEAKERS Pauline Mazel (Université Côte d'Azur, Inria, INRAE, CNRS, Macbes Project-Team)
AUTHORS Frédéric Grognard (Université Côte d'Azur, Inria, INRAE, CNRS, Macbes Project-Team); Thomas Stiehl (RWTH Aachen University); Pauline Mazel (Université Côte d'Azur, Inria, INRAE, CNRS, Macbes Project-Team); Walid Djema (Université Côte d'Azur, Inria, INRAE, LOV (CNRS - Sorbonne University), GreenOwl Project-Team)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.05 - HS
TRACK Mathematical Oncology

Abstract

Acute myeloid leukemia (AML) is one of the most aggressive forms of leukemia, driven by uncontrolled proliferation of abnormal stem cells in the bone marrow, which disrupts normal blood cell production.

Through mathematical modeling and analysis, we aim to better understand the dynamical behaviors underlying leukemia progression and to derive insights for optimal treatment design. Starting from the AML model in [KSS24], we extend the framework by incorporating chemotherapy dosage as a control variable [Maz+26]. We study the system dynamics and formulate an optimal control problem balancing leukemic cell reduction with toxicity. Our analysis reveals a turnpike phenomenon: the optimal therapy maintains a constant intermediate dose over most of the treatment horizon. This turnpike behavior occurs near a bifurcation point linked to a manifold of equilibria.

However, prolonged dosing at specific levels may favor clonal heterogeneity and drug resistance. To address this, we introduce a refined framework incorporating sensitive and resistant subpopulations. In this setting, the turnpike behavior persists, but with a richer structure featuring two manifolds of equilibria and a turnpike value shifted to the lowest bifurcation threshold.

Finally, we propose a Model Predictive Control approach that adapts dynamically to observed cell evolution, ensuring effective regulation even when resistance mechanisms are partially unknown, thereby enhancing therapeutic success.

References

[KSS24] Rohit Kumar, Sapna Ratan Shah, and Thomas Stiehl. "Understanding the Impact of Feedback Regulations on Blood Cell Production and Leukemia Dynamics Using Model Analysis and Simulation of Clinically Relevant Scenarios". In: *Applied Mathematical Modelling* 129 (2024), pp. 340–389. DOI: 10.1016/j.apm.2024.01.048.

[Maz+26] Pauline Mazel et al. "Modeling, Analysis, and Optimal Control of Leukemic Cell Population Dynamics under Therapy". In: *Bulletin of Mathematical Biology* (2026). Under review.

MS23-04 — Probabilistic Modeling of Antibody Kinetics Post Infection and Vaccination

SPEAKERS Rayanne Luke (George Mason University)
AUTHORS Rayanne Luke (George Mason University)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

There is a significant gap in the theoretical and experimental understanding of the time- dependent, person-specific viral response to infections and vaccinations. Such events pose considerable modeling challenges, and a mathematical characterization of antibody kinetics is crucial for tackling future questions related to herd immunity and optimal decision-making regarding vaccination policy. We address the important task of tracking the antibody response to multiple infections or vaccinations or combinations of the two immune events, which was previously unresolved. We describe event-to-event transitions for post-infection or post-vaccination antibody kinetics by employing a novel combination of probability distribution models with a time-inhomogeneous Markov chain framework. This approach is ideal to model sequences of infections and vaccinations and predict missed events. We validate our work using SARS-CoV-2 antibody measurements from two datasets with different measurement systems. This work paves the way to future frameworks that can analyze the protective power of natural immunity or vaccination, predict missed immune events at an individual level, and inform booster timing recommendations for both emerging and endemic diseases.

MS46 — Stochastic models and methods in mathematical biology

TRACK Miscellaneous
SCHEDULE Wednesday, 15 July 2026; 11:10–12:00; 01.22 - HS
ORGANISERS Arnab Ganguly (Louisiana State University); Olga Izyumtseva (University of Nottingham); Wasiur Rahman Khuda Bukhsh (University of Nottingham)

Minisymposium abstract

Biology has been often touted as the new physics for mathematics with the promise of new theorems that are inspired by biological investigations. The proposed mini-symposium will discuss new theoretical results and open problems in probability theory and stochastic processes, which are inspired by models in biology. In particular, the session will provide an overview of stochastic models arising from polymers, fragmentation processes, epidemics on networks, mean-field limits connecting macroscopic behaviour to microscopic interactions, neuroscience, systems biology and reaction networks. The two-part mini-symposium will

showcase a variety of probabilistic and analytic tools that provide rigorous insights into the mechanisms driving complex biological systems.

The invited speakers all of whom have confirmed to participate and speak in the mini-symposium should the proposal be accepted are carefully chosen to ensure diversity of not only areas of biological systems but also career stages and backgrounds.

MS46-01 — Branching annihilating random walk models for self-regulating populations

SPEAKERS Alice Callegaro (Technische Universität München)
AUTHORS Alice Callegaro (Technische Universität München)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 01.22 - HS
TRACK Miscellaneous

Abstract

We study a branching-annihilating random walk in which particles evolve on the lattice in discrete generations. Each particle produces a Poissonian number of offspring which independently move to a uniformly chosen site within a fixed distance from their parent's position. Whenever a site is occupied by at least two particles, all the particles at that site are annihilated. This models a population living in demes under a very strong form of local competition. For certain ranges of the parameters of the model, we show that the system dies out almost surely, or on the other hand survives with positive probability. In an even more restricted parameter range, we strengthen the survival results to convergence to a non-trivial invariant measure upon survival, and we show that the population invades space with a linear spreading speed. A central tool in the proof is comparison with oriented percolation on a coarse-grained level, using carefully tuned density profiles which expand in time and are reminiscent of discrete travelling wave solutions.

References

[Bir+24] Matthias Birkner et al. "Survival and complete convergence for a branching annihilating random walk". In: *The Annals of Applied Probability* 34.6 (Dec. 2024). ISSN: 1050-5164. DOI: 10.1214/24-aap2105. URL: http://dx.doi.org/10.1214/24-AAP2105.

MS46-02 — A spectral Koopman framework for stochastic reaction networks

SPEAKERS Ankit Gupta (ETH Zürich)
AUTHORS Ankit Gupta (ETH Zürich); Mustafa Khammash (ETH Zurich)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 01.22 - HS
TRACK Miscellaneous

Abstract

Stochastic reaction networks (SRNs) are a general class of continuous-time Markov jump processes used to model a wide range of systems, including biochemical dynamics in single cells, ecological and epidemiological populations, and queueing or communication networks. Yet analyzing their dynamics remains challenging because these processes are high-dimensional and their transient behavior can vary substantially across different initial molecular or population states. Here we introduce a spectral framework for the stochastic Koopman operator that provides a tractable, low-dimensional representation of SRN dynamics over continuous time, together with computable error estimates. By exploiting the compactness of the Koopman operator, we recover dominant spectral modes directly from simulated or experimental data, enabling efficient prediction of moments, event probabilities, and other summary statistics across all initial states. We further derive continuous-time parameter sensitivities and cross-spectral densities, offering new tools for probing noise structure and frequency-domain behavior. We demonstrate the approach on biologically relevant systems, including synthetic intracellular feedback controllers, stochastic oscillators, and inference of initial-state distributions from high-temporal-resolution flow cytometry. Together, these results establish spectral Koopman analysis as a powerful and general framework for studying stochastic dynamical systems across the biological, ecological, and computational sciences.

MS46-03 — Stochastic ordering tools for reaction network models

SPEAKERS Giulio Cuniberti (Politecnico di Torino)
AUTHORS Daniele Cappelletti; Giulio Cuniberti (Politecnico di Torino); Paola Siri
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 01.22 - HS
TRACK Miscellaneous

Abstract

Stochastic reaction networks are mathematical models with a wide range of applications in biochemistry, ecology, and epidemiology, and are often complex to analyze. Except for some special cases, it is generally difficult to predict how the abundances of all considered species evolve over time. A possible approach to address this issue is to develop tools to compare the model under study with a similar one whose behavior is better understood. The main contribution of our work is to provide direct and computable conditions that can be used to ensure the existence of an ordered coupling between two stochastic reaction networks and to identify which parameter changes in a given model lead to an increase or decrease in the count of certain species. We also make an algorithm available that implements our theory and we illustrate it with several applications.

MS46-04 — Remodelling selection: What is it?

SPEAKERS Gabriela Gomes (University of Strathclyde)
AUTHORS Gabriela Gomes (University of Strathclyde)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 01.22 - HS
TRACK Miscellaneous

Abstract

Every population consists of individuals who vary in their traits, and each trait may, or may not, be associated with frailty or fitness. Variation in frailty and fitness traits makes population studies prone to selective depletion bias (SDB). The issue is widespread across fields. When an ageing cohort exhibits declining mortality, is it individuals becoming healthier or selective depletion of the frail? In an epidemic, when growth in cumulative infections decelerates, is it individuals cautiously changing behaviour or selective depletion of the most susceptible? In microbial populations, when mutations increase population vulnerability to stress, it is individuals becoming more vulnerable or mutant populations having higher variance in fitness? In each case, the first explanation invokes individuals changing, while the second recognises that populations change due to selection on pre-existing variation. While the former are intuitive and widely adopted, explanations that rely on selective depletion are more neutral and less commonly considered due to cognitive biases and challenges in estimating all variation that matters. We propose remodelling selection (ReMS) as a general strategy of study design and analysis to address the SDB problem. We have been using case studies to illustrate how it works but it remains a challenge to formalise ReMS in abstract terms that are broad enough to represent the entire domain of its applicability. Let's discuss!

MS46-05 — Mean field limit of non exchangeable interacting diffusions on co-evolutionary networks

SPEAKERS Julian Cabrera-Nyst (University of Granada)
AUTHORS Julian Cabrera-Nyst (University of Granada)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 01.22 - HS
TRACK Miscellaneous

Abstract

Traditional models of interacting particle systems often assume a fixed network of connections, which simplifies analysis but fails to capture the dynamics of many real-world phenomena. Consequently, co-evolutionary network models, where the network structure and particle states evolve in mutual influence, are increasingly recognised as essential in diverse fields. For instance, in neuroscience, where learning is

encoded through the strengthening and weakening of synaptic connections (synaptic plasticity). Similarly, in social sciences, the co-evolution of opinions and social ties is a key driver of social dynamics.

This presentation discusses the rigorous derivation of the mean-field limit for systems of interacting diffusions on co-evolutionary networks. While previous research has primarily addressed continuum limits or systems with linear weight dynamics, our work overcomes these restrictions. The main challenge arises from the coupling between the network weight dynamics and the agent states, which results in non-Markovian dynamics where the system's future depends on its entire history. Consequently, the mean-field limit is not described by a partial differential equation, but by a non-Markovian stochastic integrodifferential equation.

MS46-06 — The effects of individual versus community-influenced isolation on SIS epidemic persistence on finite random graphs

SPEAKERS Matthew Wascher (Case Western Reserve University)
AUTHORS David Sivakoff (The Ohio State University); Matthew Wascher (Case Western Reserve University); Shirshendu Chatterjee (CUNY)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 01.22 - HS
TRACK Miscellaneous

Abstract

The contact process, or SIS epidemic, is a continuous-time Markov process used to model the spread of infection on a graph. Each vertex is either healthy or infected, and each infected vertex independently infects each of its healthy neighbors at rate λ and recovers at rate 1. We study the contact process in the presence of additional intervention measures by introducing a third possible state for vertices, which we call isolated. Vertices may enter the isolated state either because of individual decisions or due to community-influenced decisions, which leads to two distinct models that we call the isolation model and the vigilance model, respectively. In the isolation model, infected vertices self-isolate at rate α. In the vigilance model, each healthy vertex causes each of its infected neighbors to isolate at rate α. Unlike the usual contact process, these models lack the key features of attractiveness and existence of a dual, which makes analyzing them more challenging. We study the persistence times of the infection on large, finite, degree-heterogeneous random graphs. We show that the infection in the isolation model persists for at least stretched exponential time in the size of the graph for all values of α and λ. By contrast, in the vigilance model, for every fixed α the persistence time of the infection exhibits a phase transition in λ: for small λ the infection persists for at most a linear time in the size of the graph, while for large λ the infection persists exponentially long. This contrast demonstrates that individual versus community-influenced isolation can substantially affect the persistence of an epidemic.

MS46-07 — Self-intersection local times for Volterra Gaussian processes: Applications to polymer models

SPEAKERS Olga Izyumtseva (University of Nottingham)
AUTHORS Olga Izyumtseva (University of Nottingham); Wasiur Rahman Khuda Bukhsh (University of Nottingham)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 01.22 - HS
TRACK Miscellaneous

Abstract

Self-intersection local times of a random process are random variables describing how much time the process spends in small neighbourhoods of points where the trajectory intersects itself a multiple number of times. Le Gall's classical result on the asymptotic expansion of the planar Wiener sausage (1990) shows that self-intersection local times are geometric characteristics of random processes describing its topological complexity. It is widely used for the construction of continuous polymer models helping to define polymer measures penalising trajectories with many self-intersections highlighting the excluded volume effect of real polymers.

In this talk, self-intersection local times are discussed for Volterra Gaussian processes. Volterra Gaussain process is defined as a stochastic integral of a deterministic Volterra kernel with respect to a Wiener process. The existence of self-intersection local times for Volterra Gaussian processes is discussed in terms of conditions on Volterra kernels generating processes. Moreover, the aymptotics of conditional moments for self-intersection local times of some classes of Volterra Gaussian processes is described given the end-to-end distance tends to infinity.

MS46-08 — Information and Energy in Stochastic Reaction Networks

SPEAKERS Heinz Koeppl (TU Darmstadt)
AUTHORS Heinz Koeppl (TU Darmstadt)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 01.22 - HS
TRACK Miscellaneous

Abstract

Biological information processing is constrained by energetic costs, making it natural to treat information flow and dissipation jointly. We develop a unified framework for continuous-time chemical reaction networks (CRNs) that couples trajectory-level mutual and directed information between disjoint species sets with process-based stochastic thermodynamics for open, multi-reservoir systems. The formulation covers causal conditioning and indistinguishable reactions at the subnetwork level arising from multiple reservoir coupling or projection. We also give a conversion from species–reaction graphs to local-independence graphs on reactions and link local independence to causally conditioned directed information.

MS51 — Cancer-Immune Ecology

TRACK Mathematical Oncology
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 62.01 - HS
ORGANISERS Philipp M. Altrock (University Hospital Schleswig-Holstein, Kiel University, Germany); Yifan Chen (University Hospital Schleswig-Holstein, Kiel University, Germany)

Minisymposium abstract

The mathematical models of cancer onset and progression increasingly reflect the emerging view of tumors and the cancer-immune system as complex ecosystems. In combination with empirical evidence and statistical approaches, mathematical models of ecological and eco-evolutionary dynamics of cancer have gained increased importance for both theorists and experimentalists.

This minisymposium focuses on mathematical and computational models of cell population dynamics and systems biology in cancer and tumor systems, and on the resulting complex dynamical systems. These approaches encompass tumor-immune interactions, immunotherapy systems, and cancer ecological processes. The four speakers in this session will present recent advances in mathematical modeling and integrative theory-data modeling, ranging from differential equation models and statistical inference to agent-based and stochastic frameworks. These frameworks aim to deepen our understanding of phenomena in cancer evolution, ecology, and immunotherapy. Topics include immune activation and exhaustion, tumor microenvironment ecology, treatment scheduling and optimization respecting complex interactions, and the integration of experimental or clinical data with theoretical models.

MS51-01 — Modelling early immune selection and evasion in tumour evolution

SPEAKERS Eszter Lakatos (Chalmers University of Technology, Sweden)
AUTHORS Eszter Lakatos (Chalmers University of Technology, Sweden)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 62.01 - HS
TRACK Mathematical Oncology

Abstract

A growing tumour is an evolving system: during tumour development, cancer cells randomly acquire mutations that may provide them with a beneficial phenotype. However, such alterations can also give rise to neoantigens – novel cancer-specific peptides presented on the cell surface that trigger host immune responses. Successfully navigating interactions with the microenvironment and overcoming immune elimination is therefore a crucial step in cancer development. In recent work on colorectal cancer, we analysed the relative timing and evolutionary dynamics of subclones bearing immune-relevant alterations, observing a "Big Bang" pattern in which mutations acquired early in tumour growth shaped subsequent tumour-immune evolution [Lak+25].

Here, we investigate under which biological and sampling regimes such patterns are expected – or not expected –; and how immunogenicity metrics or sequencing strategies capture various modes of tumour-immune co-evolution. Building on our previous stochastic branching process-based model of tumour evolution and neoantigen acquisition [Lak+20], we simulate sequencing data from multi-region biopsies to explore the distribution of neoantigen burden and diversity under different levels of ongoing immune selection and immune escape prevalence. Our simulations span from near-neutral evolution to strong early immune pressure selecting for parallel escape, illustrating how distinct regimes manifest in sequencing data patterns.

References

[Lak+20] Eszter Lakatos et al. "Evolutionary dynamics of neoantigens in growing tumors". In: *Nature Genetics* 52.10 (Oct. 1, 2020), pp. 1057–1066. DOI: 10.1038/s41588-020-0687-1. URL: https://doi.org/10.1038/s41588-020-0687-1.

[Lak+25] Eszter Lakatos et al. "Epigenetically driven and early immune evasion in colorectal cancer evolution". In: *Nature Genetics* 57.12 (Dec. 1, 2025), pp. 3039–3049. DOI: 10.1038/s41588-025-02349-1. URL: https://doi.org/10.1038/s41588-025-02349-1.

MS51-02 — Deciphering systemic immune cell dynamics to inform immunotherapeutic approaches

SPEAKERS Frederik Graw (Friedrich-Alexander University of Erlangen-Nuremberg, University Hospital Erlangen, Deutsches Zentrum Immuntherapie, Bavarian Cancer Research Centre, Erlangen, Germany)

AUTHORS Frederik Graw (Friedrich-Alexander University of Erlangen-Nuremberg, University Hospital Erlangen, Deutsches Zentrum Immuntherapie, Bavarian Cancer Research Centre, Erlangen, Germany)

SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 62.01 - HS

TRACK Mathematical Oncology

Abstract

Immunotherapeutic approaches that exploit and manipulate cellular immune responses have increased our ability to treat various malignant diseases. However, these approaches still have their limitations and tend to fail in numerous patients, requiring a more mechanistic and quantitative understanding about the complex, intermingled dynamics of cell migration, differentiation and turnover that regulate immune responses. Developing multi-scale population dynamics models and applying them to time-resolved murine and human data on immune dynamics in response to vaccination, as well as immunotherapeutic treatment by CAR-T cell therapies in patients suffering from diffuse large B-cell lymphoma (DLBCL), we determined how individual processes of cell migration, proliferation and differentiation interact to shape cellular immune responses. Our analyses suggest the existence of optimal time windows for therapeutic interventions and are able to recapitulate the dynamics of responders and non-responders to CAR-T cell therapy, suggesting the relation of individual T cell subset kinetics to therapy outcome and toxic side effects. This illustrates the necessity for a mechanistic and quantitative understanding of the individual processes that shape cellular immune responses on a systemic level in order to improve immunotherapeutic strategies.

MS51-03 — Mathematical modelling of cytokine dynamics in myeloproliferative neoplasm patients during treatment

SPEAKERS Johanne Gudmand-Høyer (Roskilde University, Denmark)
AUTHORS Christina Ellervik (University of Copenhagen, Zealand University Hospital, Denmark, Boston Children's Hospital, Harvard Medical School, USA); Christina Schjellerup Eickhardt-Dalbøge (Zealand University Hospital, University Hospital of Region Zealand, Denmark); David Kent (University of York, United Kingdom); Hans C. Hasselbalch (Zealand University Hospital, Denmark); Johanne Gudmand-Høyer (Roskilde University, Denmark); Johnny T. Ottesen (Roskilde University, Denmark); Jordan Andrew Snyder (Roskilde University, Denmark); Lasse Kjær (Zealand University Hospital, Denmark); Morten Andersen (Roskilde University, Denmark); Morten Kranker Larsen (Zealand University Hospital, Denmark); Nina Friesgård Øbro (Rigshospitalet, Denmark); Sophie Davies (University of York, United Kingdom); Thomas Stiehl (Roskilde University, Denmark, RWTH Aachen University, Germany); Tobias Idor Boklund (Roskilde University, Denmark); Trine Alma Knudsen (Zealand University Hospital, Denmark); Vibe Skov (Zealand University Hospital, Denmark)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 62.01 - HS
TRACK Mathematical Oncology

Abstract

The Philadelphia-chromosome-negative myeloproliferative neoplasms (MPNs) are slowly developing haematological malignancies characterised by an overproduction of blood cells. Chronic inflammation is associated with the diseases and suggested to play an important role in disease initiation and progression as well as being a consequence of the diseases.

In the DALIAH trial, treatment-naïve MPN patients were treated with either Hydroxyurea (HU) or low dose interferon-α (IFN-α) and followed for up to five years [Knu+23]. Both HU and IFN-α are commonly used treatments for MPN. On top of their cytoreductive effects, IFN-α is a potent immunostimulatory cytokine, while HU has been shown to decrease the level of pro-inflammatory cytokines and the neutrophil-to-lymphocyte ratio in sickle cell disease patients [Zah+20].

With a mechanism-based, nonlinear ordinary differential equation model, we investigate the role of inflammation in MPN disease progression and response to treatment. The model includes stem cell, progenitor cell, and mature cell compartments of wild-type and mutated hematological cells with a nonlinear negative feedback from the mature to the stem cell compartments.

By comparing the mechanism-based model to longitudinal measurements of mature blood cell counts, variant allele frequency of the MPN driver mutation *JAK2*V617F, and cytokines of the patients enrolled in the DALIAH trial, we aim to identify a signature of inflammatory mediators with prognostic impact.

References

[Knu+23] Trine Alma Knudsen et al. "Final Analysis of the Daliah Trial: A Randomized Phase III Trial of Interferon-α Versus Hydroxyurea in Patients with MPN". en. In: *Blood* 142.Supplement 1 (Nov. 2023), pp. 746–746. ISSN: 0006-4971, 1528-0020. DOI: 10.1182/blood-2023-173127. (Visited on 03/12/2026).

[Zah+20] Asmaa M. Zahran et al. "Effect of Hydroxyurea Treatment on the Inflammatory Markers Among Children With Sickle Cell Disease". en. In: *Clinical and Applied Thrombosis/Hemostasis* 26 (Jan. 2020), p. 1076029619895111. ISSN: 1076-0296, 1938-2723. DOI: 10.1177/1076029619895111. (Visited on 03/12/2026).

MS51-04 — A Bayesian ODE model for CAR T-cell kinetics in non-Hodgkin lymphoma

SPEAKERS Yifan Chen (University Hospital Schleswig-Holstein, Kiel University, Germany)
AUTHORS Philipp Altrock (Cancer Modeling & Evolution UKSH Campus Kiel); Yifan Chen (University Hospital Schleswig-Holstein, Kiel University, Germany)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Non-Hodgkin lymphoma (NHL) is a heterogeneous group of hematological malignancies arising from lymphoid cells. In relapsed or refractory cases, chimeric antigen receptor (CAR) T-cell therapy offers a potentially curative treatment by engineering patient-derived T-cells to target tumor-associated antigens. Despite promising outcomes, treatment response remains variable across patients. Lactate dehydrogenase (LDH) is a serum marker that reflects tumor burden, cell turnover, and tissue damage. It is routinely measured prior to lymphodepletion and during CAR T-cell therapy and may provide insight into the immunological context at treatment onset. We developed a mechanistic ordinary differential equation (ODE) model that studies the relationship between CAR T-cell kinetics, tumor burden, and treatment response. By integrating individual patients' longitudinal CAR T-cell and LDH measurements of NHL patients within a Bayesian inference framework, we aim to infer latent tumor burden trajectories, linking tumor-immune interactions to treatment outcome.

MS93 — Stochastic agent- and particle-based models in biology: methods and analytical insights

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Wednesday, 15 July 2026; 11:10–12:00; 05.12 - HS
ORGANISERS Chunyi Gai (University of Northern British Columbia); Ellen Baake (Bielefeld University); Eric Foxall (University of British Columbia)

Minisymposium abstract

Individual-based modelling has applications in biology ranging from the molecular and cellular level up to the scale of population and movement ecology, and is typically implemented with stochastic processes. This diversity of applications is underpinned by a common toolbox of analytical techniques for studying mean behaviour, fluctuations and rare events related to particle trajectories, interactions and genealogies. In this session, we explore and share insights revealed by these techniques, with a view towards broadening their efficacy and scope in existing and novel areas of application.

MS93-01 — Pattern Formation and Spike Dynamics in the Presence of Noise

SPEAKERS Chunyi Gai (University of Northern British Columbia)
AUTHORS Chunyi Gai (University of Northern British Columbia)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Noise plays a crucial role in the formation and evolution of spatial patterns in various reaction-diffusion systems in mathematical biology and ecology. In this talk, I give two examples where noise significantly influences spatial patterning. The first example describes how patterned states can provide a refuge and prevent extinction under stressed conditions. It also illustrates the importance of not only the absolute level of climate change, but also the speed with which it occurs. The second example studies the effect of noise on dynamics of a single spike pattern for the classical Gierer–Meinhardt model on a finite interval.

MS93-08 — Recurrent disease outbreaks and their frequency

SPEAKERS Theodore Kolokolnikov (Dalhousie University)
AUTHORS Theodore Kolokolnikov (Dalhousie University)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

We derive the frequency and distribution of outbreaks in an SIR model with noise. This involves techniques from matched asymptotics combined with stochastic ODEs and random maps.

MS93-03 — Structure of family trees in finite-population exchangeable models

SPEAKERS Eric Foxall (University of British Columbia)
AUTHORS Eric Foxall (University of British Columbia)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

We give conditions for an exchangeable genealogy model, with an arbitrary sequence of population and litter sizes, to have either a unique infinite path, or a unique lineage whose descendants eventually dominate the population. Conditions relate naturally to the coalescent time scale: the second property holds iff infinite time passes on this scale, while the first property holds if a truncated version of this time scale diverges. We make use of the well-known lookdown representation, also giving a more intuitive derivation of the lookdown via the complementary notions of forward and backward neutrality. We also discuss connections of the first property to the question of identifiability of the lookdown labelling from the unlabelled tree.

MS93-04 — An inverse problem for the modern Hopfield network

SPEAKERS Linh Huynh (Dartmouth College)
AUTHORS Linh Huynh (Dartmouth College)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

A modern Hopfield network can be viewed as an agent-based model as each neuron (or memory pattern) behaves like an individual agent following local update rules, and their interactions collectively produce the network's emergent dynamics. In this talk, I will discuss an inverse problem for the modern Hopfield network.

MS93-02 — Genealogical structures under interactive neutral reproduction: Ancestral influence graph and Frankenstein process

SPEAKERS Ellen Baake (Bielefeld University)
AUTHORS Ellen Baake (Bielefeld University)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Collaborators: Hannah Dopmeyer (Bielefeld University, Germany), Fernando Cordero (BOKU University, Vienna, Austria).

Abstract: We consider the two-type Moran model of population genetics with frequency-dependent neutral reproduction. Relying on the model's graphical representation in terms of a particle system, we establish a (factorial) moment dual. Moment duals are usually related to the genealogy of the population, but in this case, the connection is mysterious. This is because the natural ancestral graph of the model (the so-called ancestral influence graph, AIG) exhibits a complicated hierarchical structure, whereas the moment dual is a simple density-dependent branching process. We solve this mystery by starting from the fact that moment duality is a property in expectation; it need not hold pathwise. This provides the freedom to construct what we call the *Frankenstein process* by tracing back sample configurations and piecing them together across different realisations of the AIG. This leads to the dual process and resolves the mystery.

MS93-05 — Multi-type logistic branching processes with selection: frequency process and genealogy for large carrying capacities

SPEAKERS Marta Dai Pra (Humboldt University Berlin)
AUTHORS Marta Dai Pra (Humboldt University Berlin)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

We present a model for growth in a multi-species population. We consider two types evolving as a logistic branching process with mutation, where one of the types has a selective advantage. We first study the frequency of the disadvantageous type, once the population approaches the carrying capacity. Adapting techniques from [Kat91], we show that this process converges to a Gillespie-Wright-Fisher diffusion process. We then study the dynamics backward in time: we fix a time horizon at which the population is at carrying capacity and we study the ancestral relations of a sample of individuals. We prove that, provided that the advantageous and disadvantageous branching measures are stochastically ordered, this ancestral line process converges to the moment dual of the limiting diffusion. This talk is based on [PK25].

References

[Kat91] G. S. Katzenberger. "Solutions of a Stochastic Differential Equation Forced Onto a Manifold by a Large Drift". In: *The Annals of Probability* 19.4 (1991), pp. 1587–1628. DOI: 10.1214/aop/1176990225. URL: https://doi.org/10.1214/aop/1176990225.

[PK25] Marta Dai Pra and Julian Kern. *Multi-type logistic branching processes with selection: frequency process and genealogy for large carrying capacities*. 2025. arXiv: 2507.12601 [math.PR]. URL: https://arxiv.org/abs/2507.12601.

MS93-06 — Multitype Lambda-coalescents: Characterisation and duality

SPEAKERS Noemi Kurt (Goethe University Frankfurt)
AUTHORS Noemi Kurt (Goethe University Frankfurt)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Lambda-coalescents, or multiple merger coalescents, have been extensively studied since their introduction in 1999, and were interpreted as genealogies of populations with skewed offspring distribution. More recently, multitype versions of such coalescents have garnered attention. However, it turned out that generalising the characterisation of multiple merger coalescents in terms of a finite measure on [0,1] isn't quite as straightforward a task as one might guess. In particular, the notion of asynchronity of mergers, or transitions, needs to be carefully treated when characterising multitype multiple merger coalescents.

In this talk, we discuss how multitype multiple merger coalescents are related to continuous state branching processes via duality, and we provide a characterisation as a class of partially exchangeable Markov coalescent processes.

This is joint work with Adrián González Casanova (Arizona State University), Imanol Nunez Morales (CIMAT, Guanajuato) and José Luis Pérez (CIMAT, Guanajuato).

MS93-07 — Multi-Type Birth-Death Processes with Mean-Field Interactions for B-cell Phylodynamics

SPEAKERS Sebastian Hummel (BOKU University Vienna)
AUTHORS Ella Hiesmayr (CNRS, ENS Lyon); Sebastian Hummel (BOKU University Vienna); Steven N. Evans (University of California, Berkeley); William S. DeWitt (University of Washington)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 05.12 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Antibody binding affinity maturation is a crucial process of the adaptive immune system. Motivated to model this process, we formulate a system of multi-type birth-death processes that can interact through their empirical distribution. We show that the empirical distribution process of the system of birth-death processes converges to a deterministic probability measure-valued flow as the system size tends to infinity. In this limit, a focal process evolves as a multi-type birth-death process with rates governed by the probability measure-valued flow, which is, in turn, the flow of the one-dimensional marginal distribution of the focal process. Individual processes become independent in the limit, which suggests inference to be feasible for this model.

References

[DeW+24] William S DeWitt et al. "Mean-field interacting multi-type birth–death processes with a view to applications in phylodynamics". In: *Theoretical Population Biology* 159 (2024), pp. 1–12.

MS101 — Advances in multiple timescale dynamics in neurons and related excitable systems

TRACK Neuroscience and Neural Systems
SCHEDULE Wednesday, 15 July 2026; 11:10–12:00; 15.12 - HS
ORGANISERS Jonathan Rubin (University of Pittsburgh); Morten Gram Pedersen (University of Padova)

Minisymposium abstract

Biological systems often feature aspects that evolve on highly disparate timescales. While substantial theoretical work has led to the development of extensive mathematical theory about dynamics of multi-timescale systems, open frontiers remain. This minisymposium will explore recent advances on several of these fronts in the context of problems arising in electrically excitable systems, predominantly in neuroscience. Directions to be discussed include dynamic effects arising in three-timescale systems, links between bifurcations and functionally relevant dynamic events and patterns, dynamics induced by forcing in multi-timescale systems, and challenges arising from a lack of clear separation between timescales across the dynamic range at which a system operates. Applications span topics such as neurotransmitter release for neuronal communication, generation of rhythmic activity, synchronization for hormone release, and pathological states such as spreading depression.

MS101-01 — Timescale separation anxiety in capturing neuronal activity patterns in biophysically detailed models

SPEAKERS Jonathan Rubin (University of Pittsburgh)
AUTHORS Anna Thomas (University of Pittsburgh); Jonathan Rubin (University of Pittsburgh); Ryan Phillips (Seattle Children's Hospital); Sushmita John (University of Pittsburgh)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

We consider multi-dimensional, Hodgkin-Huxley type models for single neurons in respiratory and motor components of the brainstem. Such models allow for the analysis of how specific ion currents contribute to the generation and control of a variety of complicated temporal voltage patterns that are observed experimentally. For this analysis to proceed, we nondimensionalize the original models to try to extract the timescales on which model components evolve and group these into distinct classes to which methods of geometric singular perturbation theory (GSPT) can be applied; however, in the models we consider, this classification is not clear-cut. We show how in both cases, GSPT tools such as bifurcation theory and averaging can explain the dynamical mechanisms underlying observed activity, but achieving these results requires us to impose different timescale groupings in different regions of phase space.

References

[JPR24] Sushmita R John, Ryan S Phillips, and Jonathan E Rubin. "A novel mechanism for ramping bursts based on slow negative feedback in model respiratory neurons". In: *Chaos: An Interdisciplinary Journal of Nonlinear Science* 34.6 (2024).

[TR26] Anna Kishida Thomas and Jonathan E Rubin. "Multiple timescale dynamics of conductance-based models of brainstem locomotor neurons". In: *arXiv preprint arXiv:2603.11467* (2026).

MS101-02 — Unfolding neuronal excitability: dynamical paths to depolarization block during spreading depolarization events

SPEAKERS Marisa Saggio (Aix-Marseille University)
AUTHORS Marisa Saggio (Aix-Marseille University)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Fast mechanisms responsible for action potential generation are modulated by slower processes that can lead to complex sequences of firing regimes. We use a dataset that provides an unusual window into a progression of transitions in neural activity, from baseline excitability to depolarization block and back. The dataset consists of whole-cell patch-clamp recordings of neurons during potassium-induced spreading depolarization events, which are waves of depolarization that spread across the cortex and involve massive changes in ion concentrations. The peculiarity is that these events are partial: they do not reach all cortical layers, creating a gradient in the perturbation to neural activity.

We apply a phenomenological approach to show how this progression arises naturally from minimal dynamical constraints. On the fast timescale, the neuronal excitability class interpreted through the lens of Unfolding Theory provides a generic bifurcation diagram of essential neuronal dynamics. On the slow timescale, simple homeostatic rules and their interaction with external perturbations (such as the potassium waves) steer the neuron through the diagram, giving rise to the experimentally observed progression. The model predicts other transition sequences, which we identified in the experimental and modeling literature. This frames depolarization block as a nuanced dynamical phenomenon, with implications for modeling choices and experimental design.

MS101-03 — Multiscale Organization in a novel Cartwheel Interneuron Model: From averaging to folded node dynamics

SPEAKERS Matteo Martin (University of Padova)
AUTHORS Jonathan Rubin (University of Pittsburgh); Matteo Martin (University of Padova); Morten Gram Pedersen (University of Padova)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

A recent eleven-dimensional cartwheel cell (CWC) model [MRP25] has provided insight into the evolution of the electrical activity of this interneuron. Specifically, CWCs transit from bursting, via continuous spiking, to complex spiking, as the applied current (IApp) increases [KT07]. Reductions in the maximal conductance of BK (gBK) or L-type Ca2+ (gCaL) channels lead to the loss of continuous spiking, and loss of bursting and complex spiking, respectively. Here, we provide a mathematical explanation of these transitions by analyzing a six-dimensional reduction of the model. The reduced system has three timescales with one fast, three slow, and two super-slow variables. We find that studying the super-slow dynamics, specifically the existence and the stability of the super slow equilibrium point through averaging theory, is informative for understanding the mechanism underlying the transitions between activity patterns as IApp, gBK, and gCaL change. Complementary results are obtained by considering the system as a two-time scale problem where the slow and the super-slow variables are all treated as slow. This approach

reveals that the small-amplitude oscillations seen in bursting and complex spiking originate from a folded node structure. The associated geometric features provide an alternative perspective on the regulation of the electrical activity. Overall, by integrating two- and three-timescale analyses, this work provides a coherent framework for understanding CWC dynamics.

References

[KT07] Yuil Kim and Laurence O. Trussell. "Ion Channels Generating Complex Spikes in Cartwheel Cells of the Dorsal Cochlear Nucleus". In: *Journal of Neurophysiology* 97 (2 Feb. 2007), pp. 1705–1725. ISSN: 0022-3077. DOI: 10.1152/jn.00536.2006.

[MRP25] Matteo Martin, Jonathan E. Rubin, and Morten Gram Pedersen. *On the role of L-type Ca ²⁺ and BK channels in a biophysical model of cartwheel interneurons*. Aug. 2025. DOI: 10.1101/2025.08.01.668076.

MS101-04 — Slow–fast dynamics in a neurotransmitter release model: Delayed response to a time-dependent input signal

SPEAKERS Mattia Sensi (Università di Trento)
AUTHORS Mathieu Desroches (MathNeuro Team, Inria Branch at the University of Montpellier, Université de Montpellier); Mattia Sensi (Università di Trento); Serafim Rodrigues (Basque Center for Applied Mathematics)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

We provide a short introduction to the SNARE-SM model proposed in [Rod+16] to describe the release of neurotransmitters in synapses. We highlight the use of slow-fast dynamics to recreate experimental data exhibiting delay between an input stimulus and the corresponding (asynchronous) neurotransmitter release. In particular, we analyse the possible scenarios in a 2D singularly perturbed system of ODEs in standard form, which serves as a building block for the full 6D system. Then, we introduce a generalization [SDR23] of the aforementioned model, which manages to realize more complex transient behaviours. This is achieved by increasing the complexity of the critical manifold of the planar system of singularly perturbed ODEs. We showcase the corresponding entry-exit phenomenon and a variety of canard orbits.

References

[Rod+16] S. Rodrigues et al. "Time-coded neurotransmitter release at excitatory and inhibitory synapses". In: *Proceedings of the National Academy of Sciences* 113.8 (2016), E1108–E1115.

[SDR23] M. Sensi, M. Desroches, and S. Rodrigues. "Slow–fast dynamics in a neurotransmitter release model: Delayed response to a time-dependent input signal". In: *Physica D: Nonlinear Phenomena* 455 (2023), p. 133887.

MS101-05 — Multimodal Synchronization of Pancreatic Islets

SPEAKERS Richard Bertram (Florida State University)
AUTHORS James Thornham (Florida State University); Michael Roper (Florida State University); Richard Bertram (Florida State University)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

The activity of insulin-secreting beta-cells within pancreatic islets of Langerhans is oscillatory, with a period of approximately 5 min. There are hundreds of islets in the mouse pancreas and hundreds of thousands in the human pancreas, and they are physically isolated from one another. Yet somehow that exhibit a great deal of synchrony. In addition, there is often an ultradian rhythm in blood insulin levels, with a period close to one hour. How does the islet synchrony occur? What is the mechanism for

the ultradian rhythm? We will present a unified explanation for both the synchronozation of the fast oscillations and the generation of the slow ultradian rhythm. This explanation is explored using computer simulations and a reduced mathematical model, and tested using a hybrid approach utilizing islets studied in a microfluidic device.

MS101-06 — Cardiomyocyte Models: Bifurcations and Fast-Slow Decomposition

SPEAKERS Roberto Barrio (University of Zaragoza)
AUTHORS Hiroyuki Kitajima (Kagawa University); M. Angeles Martinez (Universidad de Zaragoza); Roberto Barrio (University of Zaragoza); Sergio Serrano (University of Zaragoza); Toru Yazawa (Kagawa University)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Early afterdepolarizations (EADs) are abnormal behaviors that can lead to heart failure and even cardiac death. In this presentation, we review recent results and we mathematically investigate the occurrence and development of these phenomena in two realistic ventricular myocyte models: the rabbit model of Sato (2009) and the human model of O'Hara (2011). These models are of high dimension, 27 and 41 respectively, so a mix of techniques must be used in their study. We connect the results with a reduced low-dimensional model, the Luo-Rudy cardiomyocyte model (1991). The combined use of analytical and numerical techniques allows us to propose a global conjecture of a mathematical mechanism of EAD creation in low- and high-dimensional models. By examining the bifurcation structure of the model, we elucidate the dynamical elements associated with these patterns and their transitions. Using a fast-slow analysis, we explore the emergence and evolution of EAD in the low-dimensional model and develop new methodologies for fast-slow decomposition for the realistic high-dimensional O'Hara model. This decomposition has allowed us to propose some new theoretical techniques for the control of prearrhythmia situations.

References

[Bar+23] R. Barrio et al. "Mathematical birth of Early Afterdepolarizations in a cardiomyocyte model". In: *Mathematical Biosciences* 366 (2023), p. 109088. DOI: https://doi.org/10.1016/j.mbs.2023.109088. URL: https://www.sciencedirect.com/science/article/pii/S0025556423001281.

[Bar+21] Roberto Barrio et al. "Dynamical analysis of early afterdepolarization patterns in a biophysically detailed cardiac model". In: *Chaos: An Interdisciplinary Journal of Nonlinear Science* 31.7 (2021), p. 073137. DOI: 10.1063/5.0055965.

[Bar+22] Roberto Barrio et al. "Dynamical mechanism for generation of arrhythmogenic early afterdepolarizations in cardiac myocytes: Insights from in silico electrophysiological models". In: *Phys. Rev. E* 106 (2 2022), p. 024402. DOI: 10.1103/PhysRevE.106.024402.

[KYB24] Hiroyuki Kitajima, Toru Yazawa, and Roberto Barrio. "Fast–slow analysis and bifurcations in the generation of the early afterdepolarization phenomenon in a realistic mathematical human ventricular myocyte model". In: *Chaos: An Interdisciplinary Journal of Nonlinear Science* 34.12 (2024), p. 123108. DOI: 10.1063/5.0230834.

MS101-07 — Mechanisms for strong symmetry-breaking in coupled fast-slow oscillators

SPEAKERS Yangyang Wang (Brandeis University)
AUTHORS Yangyang Wang (Brandeis University)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Two identical oscillators with mutual inhibition provide a conceptual framework for modeling a latching mechanism in cell cycle regulation. In this talk, we study two such coupled oscillator models and investigate mechanisms of symmetry-breaking. In both models, inhibitory coupling induces stable alternating large-amplitude oscillations corresponding to the normal cell cycle. However, the systems also exhibit strong symmetry-breaking states in which the two oscillators display qualitatively different rhythms. In one case, this corresponds to endocycles, in which only one of the two oscillators undergoes large-amplitude oscillations. Using bifurcation analysis and geometric singular perturbation theory, we identify and characterize two distinct strong symmetry-breaking mechanisms: one arising through a homoclinic bifurcation and the other through a novel type of symmetric folded singularity.

MS101-08 — How slow rhythmic inputs induce spike-adding in neuronal model

SPEAKERS Zahra Aminzare (University of Iowa)
AUTHORS Pake Melland (Oregon Institute of Technology); Rodica Curtu (Michigan Technological University); Zahra Aminzare (University of Iowa)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Bursting is a common firing pattern in neurons, characterized by alternating active (spiking) and silent phases. While the transition from tonic spiking to bursting has been widely studied, the mechanisms underlying spike-adding within a burst remain less understood. We investigate spike-adding in a three-dimensional neuronal model with three distinct timescales. Using the FitzHugh–Nagumo system with slow periodic forcing of the voltage equation as a prototypical example, we show that decreasing the forcing frequency and increasing its amplitude induce transitions from single-spike responses to complex bursting with spike-adding. We relate these transitions to folded node and folded saddle singularities in the underlying fast–slow structure. Finally, we demonstrate that similar spike-adding mechanisms arise in the more physiologically realistic Morris–Lecar model.

This work is joint with Pake Melland (Oregon Institute of Technology) and Rodica Curtu (Michigan Technological University) [MCA25].

References

[MCA25] Pake Melland, Rodica Curtu, and Zahra Aminzare. “Spike-adding Mechanisms in a Three-timescale System: Insights from the Fitzhugh–Nagumo Model with Periodic Forcing”. In: *SIAM Journal on Applied Dynamical Systems* 24.4 (2025), pp. 2977–3011. DOI: 10.1137/24M1706980. eprint: https://doi.org/10.1137/24M1706980. URL: https://doi.org/10.1137/24M1706980.

MS113 — Inferring and designing stochastic biochemical processes at the single-cell level

TRACK Systems Biology and Biochemical Networks
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 11.02 - HS
ORGANISERS Alexandra Loudieres (Université Paris-Saclay); Eve Bousquet-Gautherat (Inria Saclay); Jakob Ruess (Inria Saclay); Juan David Marmolejo Lozano (University of Edinburgh); Valentine Brulard (Inria Saclay); William Hilton (Imperial College London)

Minisymposium abstract

Biochemical processes inside cells are fundamentally stochastic. Inferring parameters of stochastic models describing these processes from collected single-cell data poses mathematical and computational problems. This minisymposium brings together international speakers who will present recent methodological advances on modeling and inference with different types of single-cell data. Juan David Marmolejo-Lozano (Uni. of Edinburgh) will present a general stochastic framework showing how DNA replication–induced gene dosage changes shape the timing and variability of threshold-crossing events in transcriptional

networks, with implications for single-cell inference. Valentine Brulard (Inria Saclay) will present an age-structured model that connects stochastic intracellular plasmid replication and segregation dynamics to population-level plasmid copy number distributions. William Hilton (Imperial College London) will focus on inferring gene regulatory networks from single-cell RNA sequencing data by utilizing moment equations of interacting random telegraph models. Alexandra Loudieres (Paris-Saclay Uni.) will propose a model of phage mediated communication between bacteria that shows the importance of stochastic modeling for the design of bacterial communication systems [Puj+25b; Puj+25a; Cho+22]. Together, the talks address important challenges in learning and modeling stochastic processes at the single-cell level and propose innovative mathematical solutions to deal with computational challenges in stochastic modeling and statistical inference.

References

[Cho+22] Da-Jung Cho et al. "Distributed computation with continual population growth". In: *Distributed Computing* 35.6 (2022), pp. 547–569.

[Puj+25a] Abhinav Pujar et al. "An M13 phagemid toolbox for engineering tuneable DNA communication in bacterial consortia". In: *bioRxiv* (2025), pp. 2025–06.

[Puj+25b] Abhinav Pujar et al. "Phage-mediated intercellular CRISPRi for biocomputation in bacterial consortia". In: *Nucleic Acids Research* 53.3 (2025).

MS113-01 — Stochastic modeling of horizontal gene transfer via M13 bacteriophages in E. coli populations

SPEAKERS Alexandra Loudieres (Université Paris-Saclay)
AUTHORS Alexandra Loudieres (Université Paris-Saclay)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Horizontal gene transfer mediated by bacteriophages is a critical mechanism for bacterial genome plasticity, among others, responsible for the diffusion of antibiotic resistance. We develop a stochastic Chemical Reaction Network (CRN) model capturing phage-mediated infection in E. coli populations, where M13 bacteriophages transport genetic sequences, including antibiotic resistance genes. Our model integrates several scales: cell growth kinetics, infection dynamics, and population-level interactions under antibiotic selection. Its main features are modeling of infection phases, growth-burden-associated phage production, and the impact of antibiotic selection. The stochastic CRN considers individual cell variability and rare infectious events, which are essential when phage concentrations are low. This model allows to quantitatively describe resistance transfer efficiency under different conditions such as phage proportions and antibiotic selection.

References

[Cho+22] Da-Jung Cho et al. "Distributed computation with continual population growth". In: *Distributed Computing* 35.6 (2022), pp. 547–569.

[Puj+25a] Abhinav Pujar et al. "An M13 phagemid toolbox for engineering tuneable DNA communication in bacterial consortia". In: *bioRxiv* (2025), pp. 2025–06.

[Puj+25b] Abhinav Pujar et al. "Phage-mediated intercellular CRISPRi for biocomputation in bacterial consortia". In: *Nucleic Acids Research* 53.3 (2025), gkae1256.

MS113-02 — Bridging single-cell and population dynamics of plasmids with an age-structured model

SPEAKERS Valentine Brulard
AUTHORS Valentine Brulard
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Plasmids are extrachromosomal DNA molecules widely used in synthetic biology to implement gene circuits and control protein production. At the single-cell level, plasmids replicate stochastically and segregate at division, generating copy-number variability, while at the population level cell growth and division shape plasmid distributions. Existing models typically focus on either intracellular or population dynamics and fail to capture their coupling. We propose an age-structured model linking intracellular plasmid dynamics to population-level distributions. We investigate the long-time behavior of the model in both low-copy and high-copy plasmid regimes. Our analysis reveals how intracellular stochasticity propagates to population-level copy-number distributions and how cell-cycle variability influences plasmid copy-number dynamics and loss. Finally, we calibrate the model against single-cell measurements of plasmid copy numbers and obtain quantitative agreement across distinct plasmid regimes.

MS113-03 — DNA replication as a hidden driver of single-cell timing variability

SPEAKERS Juan David Marmolejo Lozano (University of Edinburgh)
AUTHORS Juan David Marmolejo Lozano (University of Edinburgh)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Gene expression models often treat the cell cycle as mere background, overlooking the transient gene-dosage shifts introduced by DNA replication. We ask how these dosage changes reshape the time it takes single cells to cross regulatory thresholds—a key currency for decision-making in transcriptional networks. Using a general stochastic framework that captures cell-to-cell variability without relying on system-specific details, we examine how replication-induced changes in effective synthesis rates modulate timing distributions. We find that replication timing can systematically advance or delay threshold crossings and, depending on network context, either sharpen or broaden variability across cells. These effects are most consequential for circuits that gate events requiring precise schedules, such as division checkpoints or developmental transitions. Our analysis highlights qualitative design principles for buffering or leveraging replication-driven noise and suggests inference strategies to disentangle replication from other sources of stochasticity. By placing DNA replication back into the picture, we reveal a general mechanism by which the cell cycle sculpts single-cell temporal coordination.

MS113-04 — Unravelling causal interactions of coding and non-coding RNA from genome wide single-cell data

SPEAKERS William Hilton (Imperial College London)
AUTHORS William Hilton (Imperial College London)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Inferring gene regulatory interactions from transcriptomic data is crucial for understanding gene networks. It is made difficult by technical noise, cell size and other confounding factors which introduce spurious correlations and inference errors. Focusing on regulation via coding and non-coding RNA interactions, we generalise correlation tests in the presence of technical noise to allow accurate inference. Moment estimates from single cell RNA sequencing data are combined with moment equations from interacting telegraph models to form a computationally tractable optimization problem including semidefinite moment constraints, which can be efficiently solved using a cutting plane approach. We demonstrate the approach using total-RNA-sequencing in single cells, which allows us to identify and model interactions between non-coding RNA and their targets. The resulting network across the non-coding genome recovers causal relations that underlie the true gene-gene correlations in the data.

MS118 — Modelling applications in the pharmaceutical industry (Pharmacometrics Subgroup)

TRACK Cutting Edge Research Areas
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 01.15 - HS
ORGANISERS Marissa Renardy (GSK)

Minisymposium abstract

In drug development, mathematical models and quantitative tools are essential to driving efficiency and innovation. Such tools include quantitative systems pharmacology (QSP) models, disease progression models, pharmacokinetics and pharmacodynamics (PKPD) models, virtual populations, digital twins, and in silico clinical trials. These are used to assist in decision making across all stages of drug development, informing target selection, design of preclinical experiments, dose selection, clinical trial design, and more. In this session, we feature modelers across the pharmaceutical industry who will showcase their innovative work and real-world impact through compelling case studies. Join us to discover how cutting-edge quantitative science is driving informed decisions, reducing risk, and accelerating the delivery of new, life-changing therapies to patients.

Speakers: Inmaculada Sorribes (Merck): “Fit-for-Purpose Modeling in Immuno-Oncology: A Practical Guide to Data and Quantitative Tools Across the Pipeline, featuring a Case Study on novel T Cell Engagers” Gianluca Selvaggio (Pharmetheus): “Mathematical modelling as a tool to investigate bell-shaped dose-response relationships in drug development” Hoai-Thu Thai (Sanofi): “Joint modeling of longitudinal data and survival outcome to improve decision making in oncology drug development” Felix Jost (Sanofi): “Feature based prediction of preclinical pharmacokinetic profiles using machine learning and compartmental modeling”

MS118-01 — Feature-based prediction of preclinical pharmacokinetic profiles using machine learning and compartmental modeling

SPEAKERS Felix Jost (Sanofi)
AUTHORS Christoph Grebner (Sanofi); Clemens Giegerich (Sanofi); Felix Jost (Sanofi); Hans Matter (Sanofi); Henrik Cordes (Sanofi)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

For healthcare and drug discovery, particularly during the earlier stages of target identification and hit finding, recent advancements in data availability, computational power, and methods (machine learning (ML) and artificial intelligence (AI)) have shown great promise to further rationalized the drug development pipeline initiating a transition from a “data generation and triaging” to a ”result prediction and verification” pipeline. We present different feature-based concepts of predicting in vivo PK profiles for chemical and biological identities derived from their chemical structure or amino acid sequences with physics informed neural networks. The first example evaluates the performance of different state of the art (hybrid) methods for predicting PK profiles based on chemical structures and benchmark their performance on a common data set for pre-clinical species. Next, a method for predicting non-rodent PK profiles for using allometric AI approach is introduced. The last concept utilizes protein large language models and sequence derived features to predict in vivo clearance and PK profile in pre-clinical species.

MS118-02 — Mathematical modelling as a tool to investigate bell-shaped dose-response relationships in drug development

SPEAKERS Gianluca Selvaggio (Pharmetheus)
AUTHORS Gianluca Selvaggio (Pharmetheus)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

In traditional pharmacology, dose-response relationships have long been the gold standard for selecting optimal therapeutic doses. Typically, these relationships are monotonic. However, in recent years hormetic, bell-shaped relationships have become more common. This is the case of, for example, bispecific T-cell engager (TCE) drugs, treatments where anti-drug antibodies (ADAs) may act as cross-linkers, or multi-valent molecular targets. These non-monotonic "hook effects" arise from the unique requirement for complex active structures, such as ternary or higher-order complexes, which are highly sensitive to stoichiometric imbalances. This work demonstrates how mechanistic mathematical modelling can decode the origins of these responses by formulating the kinetics of complex formation through systems of ordinary differential equations. We explore how different molecular architectures, drive self-inhibition at high concentrations. By simulating the drug-target synapsis, we show that the peak of the bell-shaped curve is a predictable function of the relative concentrations and binding affinities of the molecular species involved. The non-linear nature of these interactions, including non trivial hormetic behaviour, impose a shift towards mechanistic modelling, which can powerfully define the underlying biological drivers rather than describing the observed output, providing an important tool for drug development.

MS118-03 — Joint modeling of longitudinal data and survival outcome to improve decision making in oncology drug development

SPEAKERS Hoai-Thu Thai (Sanofi)
AUTHORS Antoine Pitoy (Sanofi); Hoai-Thu Thai (Sanofi); Marc Cerou (Sanofi); Nathalie Fagniez (Sanofi); Semiond Dorothée (Sanofi); Sylvain Cartot-Cotton (Sanofi)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Drug development in oncology faces high failure rates and significant costs due to methodological and operational challenges. Modeling and simulation approaches increasingly support decision-making by integrating biological knowledge and existing data throughout development programs. Nonlinear joint modeling of longitudinal and survival data characterizes the dynamic relationship between biomarker evolution and event occurrence, enabling precise parameter estimation and reduced bias in treatment effect predictions [Des+15]. Increasingly adopted in oncology, it supports trial simulations to explore dosing regimens, identify prognostic factors, or support regulatory decisions [Tha+23]. We illustrate the impact of joint PK/PD modeling through four case studies. Joint modeling of serum M-protein (surrogate of tumor burden) and progression-free survival (PFS) supported isatuximab dose selection in relapsed refractory multiple myeloma patients and regulatory approval in combination with dexamethasone in Japan, avoiding a dedicated trial [Tha+23]. Remarkably, phase 3 PFS outcomes were accurately predicted using joint models built on early phase 1–2 data alone. This predictive capability was shown across multiple indications: multiple myeloma with isatuximab-pomalidomide-dexamethasone [Pit+24], breast cancer with amcenestrant [Cer+24], and lung cancer with tusamitamab ravtansine [Cer+26]. Beyond efficacy, we developed a novel framework to assess benefit-risk balance, accounting for efficacy-safety interactions and the impact of dose modifications due to adverse events [Cer+26]. These case studies demonstrate how joint modeling enhances decision-making in drug development. Future applications include prospective implementation with virtual patient approaches to inform trial designs.

References

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MS118-04 — Fit-for-Purpose Modeling in Immuno-Oncology: A Practical Guide to Data and Quantitative Tools Across the Pipeline, featuring a Case Study on novel T Cell Engagers

SPEAKERS Inmaculada Sorribes (Merck)
AUTHORS Inmaculada Sorribes (Merck)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

The development of T cell engagers (TCEs) in immuno-oncology can leverage fit-for-purpose modeling strategy that aligns quantitative tools with the key decisions guiding the development pipeline. In preclinical and first-in-human stages, mechanistic PKPD models translate in vitro and in vivo data into predictions of safe, biologically active starting doses by characterizing trimolecular synapse formation [Che+24]. As programs progress toward a recommended Phase 2 dose, population PK modeling coupled with exposure–response (ER) analyses continue to be widely used to support monotherapy. However, TCE efficacy is primarily driven by trimer formation, so Quantitative Systems Pharmacology (QSP) models that explicitly predict trimer dynamics provide additional mechanistic insight. QSP integrates drug properties, tumor microenvironment, and the immune system to predict interactions, optimize dosing regimens and schedules, and identify determinants of safety and efficacy. Modern QSP platforms—often including ’digital twin’ virtual patients—simulate these multiscale dynamics and apply global sensitivity analysis to reveal parameters governing cytokine peaks, attenuation dynamics, bell shaped ER relationships, and efficacy plateaus [Sin+24]. These insights enable CRS risk mitigation, rational step-up dosing, and dose escalation, as illustrated with mosunetuzumab or elranatamab [Poe+25]. For late stage (Phase 2b/3), QSP can support dose and regimen optimization, predicts combination and safety outcomes, guides enrichment and trial design, and supports regulatory interactions by translating mechanistic data into decision ready simulations. QSP frameworks also guide combination therapy dose selection by quantifying synergy and therapeutic index boundaries [Jaf+23]. Other techniques, such as Physiologically based pharmacokinetic (PBPK) models, further support development by evaluating drug–drug interaction risk. Overall, regulatory agencies increasingly recognize these model-informed approaches as supportive in dose justification and benefit–risk evaluation [Mus+24]. This talk will highlight these methods, the decisions they enable, and key agency feedback through case studies of novel T cell engagers.

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MS119 — Spatial Ecology: Analysis of population distributions and spatial patterns

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 03.01 - HS
ORGANISERS Laura Storch (Bates College); Leah Shaw (William & Mary)

Minisymposium abstract

Explicit consideration of a population's distribution across space introduces added complexity to modeling and data analysis. However, explicit consideration of spatial distribution is important for many ecological processes such as dispersal, invasion, and resilience. Here, we highlight research studying population distributions and patterns with differing techniques to answer a range of ecological questions.

MS119-01 — Community-Level distribution modelling of endangered pelagic bycatch species: a joint modelling approach

SPEAKERS Clara Lereboug (Institut de Recherche pour le Développement)
AUTHORS Clara Lereboug (Institut de Recherche pour le Développement); David Kaplan (Institut de Recherche pour le Développement); David March Morla (Universitat de València, University of Exeter); Philippe Sabarros (Institut de Recherche pour le Développement)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The study of the spatial distribution of marine species has traditionally relied on single-species distribution models (SDMs), which implicitly assume independence among taxa. However, marine species evolve within structured communities whose dynamics are governed by complex biotic interactions. To account for this complexity, we use joint species distribution models (JSDMs) to model the distribution of endangered species bycaught by tropical tuna purse seine fisheries in the Atlantic Ocean. Unlike classical SDMs, JSDMs allow for the simultaneous modelling of multiple binary variables (species presence-absences) by isolating shared environmental effects from residual biotic interactions. By capturing the residual covariance not explained by abiotic variables alone, this methodology allows us to identify the actual interdependencies between species. This approach is particularly interesting in the context of bycatch of pelagic species, which has many rare, but sensitive, species and relatively low predictive power of classic SDMs. The objective of this presentation is to demonstrate how statistical inference on species dependencies improves the accuracy of spatial predictions and provides a robust tool for managing critical biodiversity areas, where traditional approaches fail to capture the ecological reality of marine communities.

MS119-02 — Restoring oyster reefs by optimizing metapopulation connectivity

SPEAKERS Leah Shaw (William & Mary)
AUTHORS Leah Shaw (William & Mary); Romuald Lipcius (Virginia Institute of Marine Science)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 03.01 - HS

TRACK Population Dynamics, Ecology & Evolution

Abstract

Oyster populations in Chesapeake Bay in the eastern United States have dropped to a few percent of historic levels. Restoration requires building artificial reefs as substrate for oysters to grow on. I will present a metapopulation model for oyster reefs that are coupled by transport of larvae. The model consists of ordinary differential equations for juveniles, adults, dead shell/reef substrate, and sediment at each location. I will first discuss results for up to two reefs. There is a tradeoff between spending more money to start constructed reefs with better initial conditions versus spending more time by restoring different sites in stages over multiple years. Next, I will discuss heuristics for selecting the best locations from a larger set when resources permit restoration of only some of the sites. I will use a realistic connectivity matrix between sites based on water flow and will consider both the total population and the resilience to disturbances. Finally, I will add harvest to the model and consider which reefs should be protected or harvested.

MS119-04 — Computational topology methods for spatial population pattern quantification

SPEAKERS Sarah Day (William & Mary)
AUTHORS Alethea Callahan (William & Mary); Laura Storch (Bates College); Sarah Day (William & Mary)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

This talk focuses on the use of cubical homology and topological persistence as a framework for pattern quantification and image processing. As illustration, we will discuss a study that applies these methods to noisy spatial population data generated by a coupled-patch model to mimic grey scale satellite images of vegetation. Early work shows promise in detecting early warning signals of collapse as well as automating image processing techniques for noise reduction.

MS119-03 — Prediction of spatial population collapse with coarse grain population distribution pattern data

SPEAKERS Laura Storch (Bates College)
AUTHORS Fatou Sanogo (Bates College); Laura Storch (Bates College); Sarah Day (William & Mary)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Populations globally are experiencing undue stress due to climate change, habitat destruction and overharvesting. Predicting impending dynamical change or collapse is challenging due to the complex spatiotemporal dynamics natural populations display and the difficulty in obtaining ecological time series. When we explicitly consider a population's distribution over space, we can quantify population spatial distribution patterns and how they change during a dynamical transition (such as collapse). Here, we quantify population distribution patterns with computational topology and use this coarse grain information from a very limited number of time steps in a population time series to predict the future state. Using supervised machine learning methods, we find that coarse grain (in space and time) topological information has a high success rate of classifying populations at risk of collapse.

MS125 — Mathematical Modelling of Tuberculosis and Nontuberculous Mycobacterial Infections: From Model Synthesis to Vaccination and Whole-Host Immune Dynamics

TRACK Immunobiology & Infection
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 02.21 - HS

ORGANISERS Aminat Yetunde Saula (University of Bath); James Doran (University of Bath); Roselyn Kaondera-Shava (University of Bath); Ruth Bowness (University of Bath)

Minisymposium abstract

Tuberculosis (TB) and other mycobacterial infections remain major global health challenges due to their complex, multiscale interactions with the host immune system. Within-host mathematical modelling provides a principled framework for integrating experimental and clinical data and generating mechanistic insight. This Mini symposium will open with a synthesis of within-host mathematical models of TB, reviewing common modelling frameworks, biological assumptions, data sources, and key insights. This introductory contribution identifies open challenges and emerging directions and establishes a unifying conceptual foundation for the session. Building on this synthesis, subsequent talks present mechanistic models of Bacillus Calmette-Guerin (BCG) vaccination and its impact on Mycobacterium tuberculosis dynamics in mice. Further contributions focus on whole-host TB models that link immune responses, bacterial burden, and disease progression. Additionally, the session will include modelling studies of nontuberculous mycobacterial (NTM) infections, in particular, an agent-based model of NTM infection in human lungs examines spatially resolved host-pathogen dynamics and identifies mechanistic correlates of disease progression. Together, the contributions in this session integrate model synthesis, vaccination modelling, whole-host immune dynamics, and agent-based approaches to advance predictive understanding and inform future modelling and intervention strategies.

MS125-01 — Whole-host modeling of immune-pathogen interactions during Tuberculosis infection

SPEAKERS Christian Michael (University of Michigan)
AUTHORS Christian Michael (University of Michigan)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Pulmonary tuberculosis (TB) remains a dire concern for countries across the globe, caused by inhaling Mycobacterium tuberculosis (Mtb). Granulomas, tissue-scale nodules that form within lungs of Mtb infected individuals, are hallmarks of human TB and are a central factor that complicates predictions of TB outcomes. Granulomas form in both lungs and lymph nodes (LNs) of pulmonary TB patients, and exhibit heterogeneity within an organ, between organs, and between patients. Experiments implicate both lung and LN granulomas as key drivers of disease outcomes, but the complex interaction of multiple simultaneous infections within an individual makes predicting outcomes challenging. To understand how multiple granulomas within lungs and lymph nodes impact patient outcomes, we have developed two agent-based hybrid models HostSim and LymphSim. HostSim captures formation and maintenance of multiple lung granulomas within a virtual host in the presence of a dynamically-priming immune response including coarse grain blood and LN models. LymphSim is a fine-grained model tracking formation and maintenance of granulomas within lung draining LNs, and the downstream effects to providing adaptive immune response to damage, and hence impairment in that can arise. Using each, we are able to capture heterogeneity of hosts, granulomas and bacterial dynamics observed in experimental studies, and probe the most influential mechanisms governing host outcome using sensitivity analyses.

MS125-02 — Agent-Based Modeling of Nontuberculous Mycobacteria Infection In Vivo with Multi-Modal Patient Data Integration

SPEAKERS Kali Konstantinopoulos (Purdue University)
AUTHORS Balaji Pathakumari (Mayo Clinic); Elsje Pienaar (Purdue University); Kali Konstantinopoulos (Purdue University); Kelly M Pennington (Mayo Clinic); Nianqiao Ju (Dartmouth College); Patricio Escalante (Mayo Clinic); Pedro Arias Sanchez (Mayo Clinic)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Nontuberculous mycobacteria (NTM) lung infection is increasing in prevalence globally and remains difficult to treat in part due to varied disease presentation and prognosis. Some patients experience stable symptoms and require observation and bronchial hygiene treatment; other patients will demonstrate disease progression symptomatically and radiographically, requiring antibiotic therapy that may cause intolerable side effects. Predicting patient prognosis to inform therapeutic decisions remains a challenge. To better understand the biological mechanisms of NTM disease progression and help identify patient prognostic factors, we developed an agent-based model of a pulmonary NTM granuloma. These virtual granulomas are initialized to represent existing chronic infection and are informed by histological studies. Peripheral immune cell data from NTM patients and lung resection immunohistochemical staining inform model initialization, parameters and calibration. The model captures multiple trajectories of granuloma development, recapitulating the heterogeneity in granuloma bacterial burden and infection progression observed in animal models of mycobacterial infection. The model demonstrates the feasibility of simulating in vivo NTM granulomas representing chronic infection. Patient data integration increases model translatability, with the potential for predicting patient prognosis and simulating patient-specific granulomas.

MS125-03 — Within-Host Mathematical Models of Tuberculosis: Foundations, Progress, and Future Directions

SPEAKERS Roselyn Kaondera-Shava (University of Bath)
AUTHORS Roselyn Kaondera-Shava (University of Bath); Aminat Yetunde Saula (University of Bath)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Within host mathematical modelling has become central to understanding the complex, multiscale biology of Mycobacterium tuberculosis infection. This talk will synthesise the major modelling frameworks that have shaped the field, from early ordinary differential equation models to agent based, hybrid, and multiscale approaches linking molecular to systemic processes. We highlight core assumptions, data sources, and the mechanistic insights they offer into host-pathogen interactions, immune dynamics, lesion heterogeneity, and treatment response. We highlight rapidly developing areas including omics-driven personalisation, systems pharmacology integration, virtual clinical trials for regimen optimisation, and modelling of clinically relevant coinfections and comorbidities such as HIV and diabetes. To provide a unified synthesis across this diverse landscape, we present quantitative thematic heatmaps that map modelling approaches to the biological and clinical questions they address. We conclude by identifying key open challenges and outline emerging directions that position within-host modelling as a cornerstone for precision therapy and global tuberculosis control.

MS125-04 — Mathematical modeling of impact of BCG vaccination on Mycobacterium tuberculosis dynamics in mice

SPEAKERS Vitaly Ganusov (Texas Biomedical Research Institute)
AUTHORS Vitaly Ganusov (Texas Biomedical Research Institute)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

The BCG vaccine remains the only licensed vaccine against tuberculosis (TB), yet its protective efficacy is highly variable, and the mechanisms behind BCG-induced protection remain poorly understood. Plumlee et al. (PLOS Pathogens 2023) infected over 1,000 mice, half of which were vaccinated with BCG, with an ultra low dose (ULD) of Mycobacterium tuberculosis (Mtb); the authors found that BCG vaccination resulted in fewer infected mice, lower CFU lung burden, and more frequent unilateral lung infection. We have developed several mathematical models of Mtb dynamics and dissemination between murine lungs and fit these models to the CFU data from unvaccinated or BCG-vaccinated mice. Alternative mathematical models incorporating either direct (lung-to-lung) or indirect (lung-intermediate-tissue-lung)

dissemination pathways fit the unvaccinated data equally well, suggesting multiple plausible routes of Mtb spread. Yet, irrespective of the dissemination route, the models predicted rapid Mtb replication during early infection, transient control within 1–2 months after infection, and continued bacterial growth in the chronic phase. Fitting models to the data from BCG-vaccinated animals revealed that BCG reduces the rate of Mtb dissemination by 89% while having a more modest effect on the replication rate within the lung, reducing it by 9%. Stochastic simulations incorporating random infection and dissemination between the right lung and left lung explained the observed variability in experimentally measured CFUs well. We used our parameterized mathematical models to calculate the number of mice needed to detect the efficacy of a hypothetical vaccine on the probability of Mtb clearance or dissemination between murine lungs that extends previously provided estimates. Taken together, our novel mathematical modeling-based framework provides a rigorous way for quantifying vaccine efficacy in ULD-infected mice, paving the way for the pre-clinical evaluation of next-generation TB vaccines.

MS134 — Novel Data Streams for Disease Modeling: Challenges and Opportunities

TRACK Mathematical Epidemiology
SCHEDULE Wednesday, 15 July 2026; 11:10–12:10; 15.03 - HS
ORGANISERS Bruce Pell (Lawrence Technological University); Fuqing Wu (UTHealth Houston School of Public Health)

Minisymposium abstract

The integration of novel data streams with mathematical models plays an increasingly important role in understanding disease dynamics and informing intervention strategies. This minisymposium focuses on challenges and opportunities arising from limited observability, data quality, and model complexity in biological and epidemiological systems. Topics include inference and identifiability under partial observation, detection thresholds, and the use of indirect or mobility-based data sources to study within-host and population-level dynamics.

MS134-01 — A Minimal Model Framework for Robust CAR-T Cell and Oncolytic Virus Combination Therapy

SPEAKERS Aisha Tursynkozha (Astana IT University)
AUTHORS Aisha Tursynkozha (Astana IT University); Yang Kuang (Arizona State University)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Glioblastoma remains one of the most lethal brain cancers. Combination therapy using CAR-T cells and oncolytic viruses shows promise, yet the mechanisms underlying their synergy remain poorly understood. We develop mathematical models to analyze IL-13Rα2-targeting CAR-T cells and the oncolytic virus C134 using patient-derived glioblastoma data. We propose a minimal model framework for predicting outcomes of combination immunotherapy. By applying timescale separation between rapid viral dynamics and slower cellular processes, we derive quasi-steady-state (QSS) approximations that reduce model complexity while maintaining predictive accuracy. The QSS model contains nine parameters compared with eleven in the full model and achieves comparable fits to the data. Model comparisons using the Akaike Information Criterion indicate that the QSS model is generally favored, particularly for oncolytic virus monotherapy and several combination therapy conditions. These results demonstrate that simplified QSS formulations effectively capture viral dynamics and provide a practical framework for analyzing and optimizing combination immunotherapies.

References

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[Pel+25] Bruce Pell et al. “Dynamic PD-L1 regulation shapes tumor immune escape and response to immunotherapy”. In: *Cancers* 17.23 (2025), p. 3803.

MS134-02 — Using Naive Bayes to uncover probabilistic maps of SIV tissue spread

SPEAKERS Chapin Korosec (University of Guelph)
AUTHORS Chapin Korosec (University of Guelph); Jessica Conway (Pennsylvania State University)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Systemic HIV infection is typically established following mucosal exposure, but the earliest events stretching from viral dissemination from those mucosal tissues to body-wide (systemic) infection remain incompletely defined. Using tissue-level SIV RNA data from rhesus macaques (an animal model for HIV) we broadly aim to map paths of infection through tissues as infection is established, to better inform HIV prevention strategies. We will discuss how we transformed our data into a cohort-wide map of viral occupancy and co-occurrence across organs and lymphatic compartments. We converted tissue measurements into presence/absence, and introduced a probabilistic framework using a Naive Bayes classifier to estimate posterior probabilities of SIV tissue spread through time, yielding an interpretable, directed dissemination network. Building on the network, we implement Monte Carlo random walk simulations to explore likely dissemination pathways and quantify mean first-passage step counts to plasma (systemic infection). Probabilistic maps reveal an SIV tissue-hierarchy leading to systemic infection that identifies specific lymphatic tissues, and the spleen, as dominant conduits. Thus we generate hypotheses on modes of establishment of systemic HIV infection. This work also demonstrates how machine learning and network simulation can transform novel biological data into quantitative models of within-host disease spread.

MS134-04 — Inferring Mobility Distributions in Heterogeneous Epidemic Models

SPEAKERS Weiqi Chu (University of Massachusetts)
AUTHORS Ning Jiang (University of Massachusetts Amherst); Weiqi Chu (University of Massachusetts); Yao Li (University of Massachusetts Amherst)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Classical compartmental models often overestimate the size of a pandemic due to the assumption of a homogeneous population. At the early stage of an outbreak, individuals with higher mobility are more likely to become infected, resulting in an inflated estimate of the final pandemic size. In this talk, we introduce a heterogeneous compartmental model in which each individual has different mobility levels. We develop a deep-learning framework to infer the mobility distribution from partial observations of epidemic dynamics. We also present theoretical results showing that these partial observations uniquely determine the mobility distribution, which establishes that the corresponding inverse problem is well-posed.

References

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MS149 — Coordination at Tip-Growing Cell Interfaces: Cell Polarity, Membrane Flow, Wall Growth, and Morphogenesis

TRACK Cellular and Developmental Biology
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 02.23 - HS
ORGANISERS Min Wu (Worcester Polytechnic Institute); Rholee Xu (Worcester Polytechnic Institute)

Minisymposium abstract

Recent work across diverse model systems has advanced our understanding of growth and morphogenesis in walled tip-growing cells, including pollen tubes, root hairs, and fungal hyphae. While cell extension is mechanically constrained by the growth and extension of the cell wall, wall dynamics are tightly coupled to exocytosis, membrane trafficking, and, in some systems, cellulose synthesis and spatially varying mechanical properties on the cell wall.

This minisymposium brings together researchers studying tip growth from complementary perspectives—wall growth and mechanics, membrane flow, and polarity regulation—to present recent advances and foster cross-disciplinary dialogue. The goal is to better understand the coordinated pipeline and feedback loops that regulate the spatiotemporal patterning of growth and shape formation and maintenance.

MS149-01 — Oscillations in the growing tip of the filamentous brown alga Ectocarpus

SPEAKERS Nelson Serre
AUTHORS Benedicte Charrier (CNRS Station Biologique Roscoff); Nelson Serre
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Ectocarpus is a filamentous brown alga whose cells are surrounded by a cell wall [Cha+08]. Using a visco-plastic model, we previously demonstrated that the polarised apical growth of its prostrate, uniserial filaments is regulated by changes in cell wall thickness, independent of alterations to the material's mechanical properties [Rab+19]. Apical growth is accompanied by transverse cell divisions, generating a new growing apical cell and an 'inert' subapical cell. More recently, time-lapse observations at various timescales have demonstrated the following: 1) growth occurs according to two patterns of cell elongation rate oscillation; 2) cell divisions are synchronised with these oscillations; and 3) the filament's growth trajectory proceeds in undulations, the changes in direction of which are synchronised with the oscillations. We are now investigating the functional relationships between these three biological processes: growth oscillations, cell divisions, and changes in growth direction. To do so, we are using a bank of *Ectocarpus* mutants that exhibit alterations in apical growth (e.g. the etoile mutant [Le +11; Neh+11]), as well as pharmacological treatments to dissociate these three parameters. The results will feed a multi-component cellular growth model and identify the molecular players involved in controlling these growth processes in brown algae. As algae diverged from other multicellular eukaryotes over 1.2 billion years ago, the results of this study will enable us to compare the various tip growth strategies that have evolved within the tree of life.

References

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MS149-02 — Membrane flows for cell patterning and resilience in fission yeast

SPEAKERS Dimitrios Vavylonis (Lehigh University)
AUTHORS Dimitrios Vavylonis (Lehigh University)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Polarized growth and division in tip-growing cells require tight coordination between membrane trafficking, in-plane membrane flows, and biochemical patterning. Using *Schizosaccharomyces pombe*, we combine mathematical and computational modeling with experiments by collaborators to study the physical mechanisms of membrane flows in cell polarity, cytokinesis, and stress adaptation. We developed a continuum hydrodynamic model of the plasma membrane during cell division, treating it as a compressible fluid coupled to cell wall mechanics and osmotic pressure. The model reveals how contractile ring constriction and spatially regulated endo- and exocytosis generate membrane tension and flows, and identifies regimes where trafficking is essential to maintain membrane integrity during division. We also examined how secretion-driven flows during tip growth interact with the Cdc42 polarity system. A stochastic reaction–diffusion model with membrane area conservation, constrained by experimental FRAP measurements and measured rates of endocytosis and exocytosis, shows how membrane flows deplete low-mobility GAPs from regions of growth, enabling robust polarization. Polarization persists even when Cdc42 mobility is reduced, though with broader and weaker activity zones, highlighting the interplay between protein mobility and flow-driven redistribution. Finally, studies of membrane expansion under protoplast hypoosmotic stress show how rapid lipid delivery, including non-vesicular transfer, buffers delays in exocytosis and helps preserve membrane integrity. These results highlight membrane flows as active regulators of pattern formation and mechanical robustness in walled cells.

MS149-03 — Emergence of polar cell growth in plants: A quantitative perspective.

SPEAKERS Zhenbiao Yang (Shenzhen University of Advanced Technology)
AUTHORS Zhenbiao Yang (Shenzhen University of Advanced Technology)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

The pollen tube expands rapidly (up to 1 cm/h) by the extreme form of polar growth known as tip growth, which is shared by many cells such as fungal hyphae. Experimental work supports a molecular framework underpinng pollen tube growth: An apical cap of active ROP1 GTPase defines the apical growth domain and is dynamically formed generated via a feed-forward loop and and a feedback loop linked by ROP1-dependent exocytosis. Mathematical modeling suggests that this framework is sufficient for the generation of the apical ROP1 cap and tip growth and also underscores pollen tube guidance to a gradient of attractants. To allow further quantitative dissection of the dynamic molecular mechanism controlling tip growth, we developed a Python-based automated tool for quantitative analysis of the apical structures and signaling components in growing pollen tubes. The broader application of this tool will be discussed.

MS149-04 — Mathematical modeling of cell wall elasticity, loosening, and feedback mechanisms on tip-growing morphogenesis

SPEAKERS Rholee Xu (Worcester Polytechnic Institute)
AUTHORS Luis Vidali (Worcester Polytechnic Institute); Min Wu (Worcester Polytechnic Institute); Rholee Xu (Worcester Polytechnic Institute)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 02.23 - HS

TRACK Cellular and Developmental Biology

Abstract

Plant development and adaptation are highly dependent on cell morphology and growth. High turgor pressure in plants causes stress on the cell wall, followed by cell extension. In tip-growing cells, the localization of vesicles and cytoskeleton components has been well studied. However, there has been a lack of attention to the spatial profile of mechanical properties, specifically the cell wall elasticity. Furthermore, the contributions of exocytosis and cell wall rearrangement to the morphogenesis of the cell are still not well understood. In this study, we first introduce a new surface morphology-based method to measure the elasticity of the cell wall in tip-growing cells. Through a parameter sensitivity study on simulated cells, we were able to verify a gradient of cell wall elasticity in tip-growing cells in the moss *Physcomitrium patens*. To explore the intricate dynamics of tip growth, we next developed a cell wall model tracking the dynamics of cell wall thickness, exocytosis, and cell wall remodeling. Especially since cell rupture can be caused by cell wall thinning, we aimed to understand the conditions that this might occur. Our system is a combined dynamical and mechanical system, where each cell growth step has contributions from the cell elastic deformation, and the actions of the cellular processes. All together, we present findings that will provide a more comprehensive view of tip-growing cell mechanics and help inform future experimental directions.

MS152 — Boolean models in Systems Biology

TRACK Systems Biology and Biochemical Networks
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 11.03 - HS
ORGANISERS B V Harshavardhan (Indian Institute of Science); Elisabeth Remy (CNRS Marseille); Laurence Calzone (Institute Curie); Paul Ruet (CNRS Paris); Samuel Pastva (Masaryk University); Tomas Gedeon (Montana State University)

Minisymposium abstract

Every year new experimental technologies in cell biology continue to generate more data about the cellular responses to different conditions and in different environments. As sophisticated as these data are, they still provide only partial snapshots of a behavior of a living cell in their natural environment. For the data to yield understanding and, ultimately, control of cellular processes in the context of medical interventions, the data need to be interpreted by mathematical models. Intracellular interactions of chemical species are represented by directed signed networks. Boolean models are used to model network dynamics when the first principle stochastic or ODE models are too under-parameterized or too large to analyze. In this session we gather the leading experts in Boolean models spanning the range from practical applications to theoretical analysis of their behavior

MS152-04 — A Boolean Approach to Invasion and Mesenchymal–Epithelial Transition (MET)

SPEAKERS Laurence Calzone (Institute Curie)
AUTHORS Laurence Calzone (Institute Curie)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Understanding cancer invasion requires linking intracellular regulatory processes with multicellular dynamics. Boolean modelling provides a powerful framework to describe phenotypic transitions such as the mesenchymal–epithelial transition (MET), capturing how signalling networks and environmental cues regulate cell plasticity and state switching.

At a larger scale, intracellular logical models can be integrated with spatial, cell-based simulations to study tumour invasion. By coupling regulatory states with cell–matrix interactions, these models reproduce diverse invasion patterns and offer insight into how individual decision-making processes give rise to emergent collective behaviours.

MS152-01 — Biological Battle Royale: Structural Constraints of Networks Supporting Multi-Fate Cellular Decisions Using Monotone Boolean Models

SPEAKERS Harshavardhan BV (Indian Institute of Science, Bengaluru)
AUTHORS Harshavardhan BV (Indian Institute of Science, Bengaluru)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Cell fate decisions are driven by gene regulatory networks (GRNs). While the mutually inhibitory toggle switch effectively models binary fate decisions, fully connected inhibitory networks with more than two nodes fail to capture multi-fate decisions due to the low prevalence of ”single-high states”, where only a single master regulator is highly expressed. In this study, we use monotone Boolean models to derive structural constraints necessary for an n-node network to support n phenotypes. We find that the only network that maximizes the prevalence of single-high states is completely disconnected. However, since biological networks typically require connectivity, we further investigate the requirements for equipotency, where a network structure supports all single-high states with equal prevalence. Finally, we characterize the requirements for multistability across all single-high states, finding that it is possible only in networks in which each node either has self-activations or inhibitions with every other node. Our findings provide a theoretical framework for understanding the topological design principles required for master regulator networks to support simultaneous differentiation into multiple distinct cell types.

MS152-02 — Long-lived sets: The missing oscillatory phenotypes of Boolean networks

SPEAKERS Samuel Pastva (Masaryk University)
AUTHORS Samuel Pastva (Masaryk University)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In Boolean networks, biological phenotypes are traditionally mapped to system attractors. However, attractors are often sensitive to the chosen update scheme and the granularity of the influence graph. Applying a more permissive update scheme or adding mediator nodes can easily disrupt plausible attractors, forcing modelers to carefully tune model properties to achieve biologically relevant outcomes.

In this talk, we introduce long-lived sets, a robust generalization of attractors. Long-lived sets are strongly connected sets of states that can be escaped, but never by value percolation (updating a single system variable across all states of the set). Theoretically, long-lived sets are immune to disruptions caused by more permissive update schemes or refinements of the influence graph. Practically, we demonstrate their utility in real-world models: they successfully capture known oscillatory behaviors that standard attractors miss. Furthermore, the number of long-lived sets in a model is often close to the number of attractors, even in models with millions of other ”short-lived” strongly connected components. This makes long lived sets not only robust, but highly specific generalization of known attractor-based phenotypes.

MS152-03 — How well do Boolean model capture continuous dynamics of regulatory networks?

SPEAKERS Tomas Gedeon (Montana State University)
AUTHORS Tomas Gedeon (Montana State University)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Regulatory networks in cell biology specify monotonicity structure of interactions between genes and proteins, but do not specify the interactions between multiple inputs. We describe several classes of models compatible with a given regulatory network:

(**A**) a collection of all monotone Boolean networks (MBF) whose influence graph matches the network;

(**B**) a collection of MBFs where influence graph is a subgraph of the network;

(**C**) a collection of all ODE models with monotone steep nonlinearities.

We show that these collection of models are strict subsets of each other

$$(\mathbf{A}) \subsetneq (\mathbf{B}) \subsetneq (\mathbf{C})$$

and describe precise embedding of each smaller class into the larger class. While the dynamics of protein and mRNA abundance is stochastic, it is usually well approximated by dynamics that is continuous in time and space generated i.e. by models in class (**C**). We illustrate on the set of examples which dynamics is reliably captured by the smaller class(es)of models, and where the set of dynamics of the larger class(es) is significantly richer than that of a smaller class(es).

Our results begin to illuminate the gap between dynamics observed by monotone Boolean models in class (**A**) and continuous dynamics of ODE models in class (**C**).

MS154 — Advanced Topics in Stochastic Chemical Reaction Networks

TRACK Systems Biology and Biochemical Networks
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 11.01 - HS
ORGANISERS Aidan Howells (Politecnico di Torino); Daniele Cappelletti (Politecnico di Torino)

Minisymposium abstract

A Chemical Reaction Network (CRN) is a set of possible chemical species, along with a finite set of chemical transformations that the species can undergo. These models are frequently used in biochemistry both for investigating existing systems and for designing new synthetic biological circuits, and also see use in other areas of biology like ecology and epidemiology. There is a rich history of study of stochastic models of CRNs, dating back to the introduction of stochastic mass-action kinetics over 50 years ago. This minisymposium will explore various recent mathematical advances in the study of these stochastic CRN models, with a focus on multiscale systems and stability.

The stability talks in this minisymposium will focus on methods for determining the existence and form of any limiting distribution for the models, whereas research in multiscale networks seeks to understand situations where some of the chemical reactions are much faster than the others. While each of the talks in this minisymposium will be theoretical and mathematical in nature, these topics have been seen to be important for understanding the behavior of real-world reaction networks.

MS154-01 — The Dummy species extension of the Chemical reaction networks; Adding additional species to derive its stationary distribution.

SPEAKERS Hoejin Kim (POSTECH)
AUTHORS Hoejin Kim (POSTECH)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Stochastic chemical reaction networks (CRNs) provide a fundamental framework for modeling stochastic dynamics in systems biology, population dynamics, and chemistry. A stationary distribution of stochastic CRN describes its long-term behavior. An analytic formula for a stationary distribution can be obtained for only in limited cases, linear or finite-state systems.

Interestingly, the analytic form of stationary distribution can be obtained when the underlying network satisfies topological conditions, specifically weak reversibility and deficiency zero, and kinetic conditions

such as mass-action or kinetics satisfying factorizability. Moreover, other studies resolved certain violations of topological conditions with the network translations. In this talk, I will introduce a "dummy species extension" framework to overcome violations of kinetic conditions. By adding an additional species, originally non-factorizable networks are transformed into factorizable systems within this framework, enabling the analytic derivation of their stationary distribution. I will demonstrate its applications with several examples from biological network systems.

MS154-02 — Bang-bang population control with reduced fluctuations: lessons from bacteria

SPEAKERS Jinsu Kim (Pohang University of Science and Technology)
AUTHORS Jinsu Kim (Pohang University of Science and Technology)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

A bang–bang control in a reaction system refers to rapid switching between two extreme behavioral regimes of a species in order to optimize an objective, such as population growth. Because such transitions occur abruptly, intrinsic stochasticity typically induces large fluctuations in the system. We have recently identified a class of bacteria that employs a remarkable strategy to suppress these fluctuations while achieving population dynamics equivalent to those generated by bang–bang control. In this talk, we examine how biological systems mitigate noise while reproducing bang–bang–like behavior. To this end, we compare two reaction network models describing bacterial division mechanisms, each formulated as a continuous-time Markov chain. Although both models yield qualitatively similar mean population dynamics, their fluctuation levels differ substantially: one exhibits significantly reduced noise. This lower-variance regime corresponds to the so-called size-control mechanism, which is supported by experimental data reported in prior studies.

MS154-03 — On the abelian structure of noncompetitive chemical reaction networks.

SPEAKERS Louis Faul (Université de Fribourg)
AUTHORS Christian Mazza (University of Fribourg); Louis Faul (Université de Fribourg); Marie Bétrisey (University of Fribourg); Xavier Richard (University of Applied Sciences of Western Switzerland)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Chemical reaction networks (CRNs) are foundational models for describing complex biochemical processes. We study noncompetitive CRNs, a class of networks whose static states are rate-independent, and that can implement ReLU neural networks. A central contribution of this work is that noncompetitive CRNs are special instances of Abelian networks (ANs)—a well-established framework for self-organized criticality. CRNs of interest in biochemistry and systems biology are embedded in complex networks so that local CRNs have to respond to internal and environmental cues. We describe the network's response to such perturbations using a Markov chain whose state space is the set of CRN's static states, from where no reaction is possible. The addition of molecules to a static state induces reactions that move the system into a new static state. For noncompetitive CRNs of finite state space, we use AN theory to get that only a fraction of the static states are recurrent, suggesting that these states might be the biologically relevant configurations.

We obtain furthermore that the set of recurrent states is in one to one correspondence with the critical group of the AN. Overall, this work establishes a unified algebraic and probabilistic framework for analyzing the long-term behavior of noncompetitive CRNs.

References

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[Vas+22] M. Vasic et al. “Programming and training rate-independent chemical reaction networks”. In: *PNAS* 24 (2022), e2111552119.

MS154-04 — On the Stability Properties of Stochastic Chemical Reaction Networks with Phosphorylation

SPEAKERS Philippe Robert (INRIA)
AUTHORS Lucie Laurence (University of Bern); Philippe Robert (INRIA)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

We investigate a class of chemical reaction networks models associated to a phosphorylation mechanism with three steps. This is an important mechanism in many biological cells. A chemical species, the substrat has three possible configurations: $\mathcal{S}_1$, $\mathcal{S}_2$ and $\mathcal{S}_3$. There are transformations by two types of chemical species (enzymes) $\mathcal{A}$ and $\mathcal{B}$:

$$\&A+S_1 \underset{\alpha_1^-}{\overset{\alpha_1^+}{\rightleftarrows}} AS_1 \xrightarrow{\lambda_1} A+S_2 \underset{\alpha_2^-}{\overset{\alpha_2^+}{\rightleftarrows}} AS_2 \xrightarrow{\lambda_2} A+S_3$$

$$\&B+S_1 \xleftarrow{\mu_2} BS_2 \underset{\beta_2^+}{\overset{\beta_2^-}{\rightleftarrows}} B+S_2 \xleftarrow{\mu_1} BS_3 \underset{\beta_1^+}{\overset{\beta_1^-}{\rightleftarrows}} B+S_3.$$

In this work, in a Markovian setting, we assume that the initial total number of copies of substrat is N and that the total number of copies of each type of enzymes is proportional to N. We investigate the asymptotic behavior, when N gets large, of the concentrations of the chemical species $(\mathcal{S}_i)$. The dependence of the possible asymptotic regimes on the reaction rates is investigated. It turns out that there are significant differences with deterministic models of the literature. The detailed stability properties of a deterministic dynamical system in $\mathbb{R}^4_+$ plays an important role in several of our scaling results.

Joint work with Lucie Laurence (University of Bern)

MS159 — Agent-based models, networks and machine-learning methods for epidemiological modelling

TRACK Mathematical Epidemiology
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 15.02 - HS
ORGANISERS Jasmina Panovska-Griffiths (Pandemic Sciences Institute, University of Oxford, Oxford, UK)

Minisymposium abstract

In the first presentation, Jasmina Panovska-Griffiths from University of Oxford, will discuss how ABMs can be combined with digital technology, such as machine-learning methods and large-language models, to better understand transmission of both respiratory and sexually-transmitted diseases. Jasmina will discuss recent work by her team to integrate digital technology to generate realistic networks for ABMs as well as challenges in calibration of ABMs, noting her team’s recently developed machine-learning and data-drive methods for more efficient and more robust calibration of these models. In the second presentation, Fabian Sturman from University of Oxford will introduce STARsim agent-based modelling framework,

and illustrate it with discussing a subset model of the framework: HPVsim, an ABM for transmission of Humanpapilloma Virus. The talk will also illustrate HPVsim application to explore the impact of different cervical screening and HPV vaccination strategies in the UK. In the third presentation, Ganna Rozhnova from the Utrecht University will discuss an ABM-modelled Omicron transmission across secondary schools in Netherlands, using the model to quantify the SARS-CoV-2 health burden and the cost-benefit (number of prevented infected students per absent student) for reactive quarantine interventions across different stages of the epidemic. Finally, in the fourth presentation, by Tom Britton from Stockholm University, will explore whether accounting for heterogeneity in social contacts (by splitting age groups into more and less socially active) affects two epidemic transmissibility metrics: the reproduction number R and the final epidemic size.

The talks within this minisymposium will be combined into a commentary piece that the authors plan to submit to the Journal of Theoretical Biology in late 2026.

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MS159-01 — MS159-1:Agent-based models, networks and machine-learning models as tools for pandemic preparedness

SPEAKERS Jasmina Panovska-Griffiths (Pandemic Sciences Institute, University of Oxford, Oxford, UK)
AUTHORS Jasmina Panovska-Griffiths (Pandemic Sciences Institute, University of Oxford, Oxford, UK)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

In the introductory talk of this mini-symposium, Jasmina Panovska-Griffiths from University of Oxford, will discuss how ABMs can be combined with digital technology, such as machine-learning methods and large-language models, to better understand transmission of both respiratory and sexually-transmitted diseases, and as tools for pandemic preparedness. As well as introducing the minisymposium, Jasmina will discuss recent work by her team to integrate digital technology to generate realistic networks for ABMs as well as challenges in calibration of ABMs, noting her team's recently developed machine-learning and data-drive methods for more efficient and more robust calibration of these models.

MS159-02 — Starsim: A fast, flexible agent-based disease modeling framework

SPEAKERS Fabian Sturman (University of Oxford)
AUTHORS Fabian Sturman (University of Oxford); Jasmina Panovska-Griffiths (Pandemic Sciences Institute, University of Oxford, Oxford, UK)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

In this talk we will introduce the Starsim suite of agent-based models- originally developed on the back of the Covasim, COVID-19 model and widely used to advise policy during COVID-19 across worldwide settings. Recent developments of the Starsim framework is covering areas as diverse as HIV, typhoid, tuberculosis, rift valley fever, family planning, influenza and pneumococcal transmission. We will introduce Starsim and showcase how the Starsim's community-driven approach to software development has the potential to strengthen capacity and improve access to fit-for-purpose modelling tools, and thus contribute to better model-informed decisions. To illustrate the application of Starsim, we will discuss the development and application of HPVsim - a Starsim model for the transmission of HPV and able to evaluate different immunisation and screening strategies across settings. We recently developed an HPVsim model for England and we will discuss the findings of this model related to the impact on cervical cancer outcomes 2026-2050 of incorporating vaccine status and vaccine eligibility into screening strategies in England.

MS159-03 — Comparative evaluation of HIV testing interventions for men who have sex with men in the Netherlands: insights for a low-incidence setting

SPEAKERS Ganna Rozhnova (University Medical Center Utrecht)
AUTHORS Ganna Rozhnova (University Medical Center Utrecht)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Background: Despite declining HIV diagnoses among men who have sex with men (MSM) in the Netherlands, a recent plateau and the high proportion of late-stage diagnoses (29% in 2024) indicate ongoing transmission and delayed detection. Infections introduced through immigration are increasingly relevant in this low-incidence setting, motivating a reassessment of testing guidelines, which we evaluate in this study.

Methods: We used an agent-based model of HIV transmission among MSM in the Netherlands, incorporating domestic and imported infections. For 2024–2040, we simulated targeted testing strategies, including testing at immigration, increased testing among resident MSM, and combined approaches.

Results: Testing immigrating MSM at entry averted up to 94 infections over 15 years (95% QI 128–328) with $\geq$50% uptake. Increasing testing to every 7 months among general resident MSM achieved the largest reduction, with 508 infections averted (95% QI 292–900), followed by targeting MSM with > 5 partners within the previous six months (340; 95% QI 132–592). Combining entry testing with 7-monthly testing among resident MSM yielded the greatest impact (534; 95% QI 308–884).

Conclusions: Combining HIV testing at entry for immigrating MSM with 7-monthly testing among resident MSM can substantially reduce infections. The greater impact of population-wide versus risk-targeted testing suggests that current risk-based testing criteria may be too narrow.

MS159-04 — Improving the use of social contact studies in infectious disease modelling

SPEAKERS Tom Britton (Stockholm University)
AUTHORS Tom Britton (Stockholm University)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Up until now, the main use of social contact studies in epidemic modelling has been to create a next generation matrix containing mean number of contacts between different age groups, in order to capture heterogeneity in contacts between age groups. However, empirical evidence show that the majority of variation in contacts is not between age-groups, but within age groups, and also within individuals (between different days). We propose methods for improving the analysis taking also this variation into account.

MS168 — Mathematical Insights into Ageing, Evolution, and Cell Dynamics

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 15.11 - HS
ORGANISERS Marie Doumic-Jauffret (Inria and Ecole polytechnique); Sarah Kaakai (Sorbonne Paris-Nord)

Minisymposium abstract

This mini-symposium gathers four researchers to explore the mathematical foundations of ageing, evolutionary selection, and cell physiology, bridging theory and biological applications. Sarah Kaakai investigates how age-structured populations could cross fitness valleys. Sylvie Méléard presents a scenario for the evolutionary selection of ageing, using a life-history trait model to demonstrate the convergence of fertility and senescence. Her work suggests that ageing could be an adaptive force, improving genetic variability and evolutionary success. Luce Breuil introduces a coupled system of age-structured partial differential equations to model two-phase ageing in D. melanogaster. By analyzing well-posedness, stability, and global asymptotic behavior, she explores the evolutionary advantages of discontinuous ageing and its implications for population dynamics. Alexandre Perrin shifts focus to E. coli, employing mathematical models to study DNA replication, cell division, and antibiotic response.

References

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[Per+25] Alexandre Perrin et al. “Deciphering the Replication-Division Coordination in E. coli: A Unified Mathematical framework for Systematic Model Comparison”. In: *bioRxiv* (2025), pp. 2025–07.

[Rog+24] Tristan Roget et al. “A scenario for an evolutionary selection of ageing”. In: *Elife* 13 (2024), RP92914.

MS168-01 — DNA replication and division cycles coordination in Escherichia coli

SPEAKERS Alexandre Perrrin
AUTHORS Alexandre Perrrin; Marie Doumic-Jauffret (Inria and Ecole polytechnique); Meriem El Karoui (ENS Saclay); Sylvie Méléard (Ecole polytechnique)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Despite extensive research, the quantitative principles that govern the coordination between DNA replication and cell division in bacteria remain debated. Multiple theoretical models have been proposed, some postulating that a single regulatory process is sufficient to ensure replication–division coordination, while others argue that two concurrent processes are required for robust control. To enable the comparison of these approaches, we developed a unifying mathematical framework within which models can be consistently formulated and quantitatively compared. Through theoretical analysis, this talk will present the necessary and sufficient conditions under which independent replication and division cycles recover

physiological cell behaviours. Beyond the correlation-based analyses extensively used to date, this talk will discuss within a comprehensive statistical framework that double-process models more accurately recapitulate experimental data across all growth conditions. Finally, a novel model will be introduced that robustly captures the replication-division coordination in every growth regime, and ensures the homeostasis of the cell DNA content.

MS168-04 — A scenario for an evolutionary selection of ageing: from biological observations to mathematical modelling

SPEAKERS Sylvie Méléard
AUTHORS Michael Rera; Sylvie Méléard; Tristan Roget
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Ageing's sensitivity to natural selection has long been discussed because of its apparent negative effect on an individual's fitness. Thanks to the recently described (Smurf) 2-phase model of ageing (data of Michael Rera) we propose a fresh angle for modeling the evolution of ageing. Indeed, by coupling a dramatic loss of fertility with a high-risk of impending death—amongst other multiple so-called hallmarks of ageing—the Smurf phenotype allowed us to consider ageing as a couple of sharp transitions. The birth–death model we describe here is a simple life-history trait model where each asexual and haploid individual is described by its fertility period x_b and survival period x_d. We show that x_b and x_d converge during evolution to configurations $x_b - x_d \approx$ constant in finite time. To do so, we built an individual-based stochastic model which describes the age and trait distribution dynamics of such a finite population.Then we rigorously derive the adaptive dynamics models, which describe the trait dynamics at the evolutionary time-scale. We extend the Trait Substitution Sequence with age structure and study the limiting behaviour of this jump process when mutations are small. We show that the limiting behaviour is described by a differential equation for (x_b, x_d). It's a joint work with Michael Réra and Tristan Roget.

MS168-02 — 2-phase model of ageing : Stability analysis of coupled non-linear partial differential equations with age structure

SPEAKERS Luce Breuil (Ecole polytechnique)
AUTHORS Luce Breuil (Ecole polytechnique)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Recent biological evidence suggests the presence of a 2-phase ageing process in D. Melanogaster. Following these discoveries, we first introduce a stochastic individual-based model and estimate from data the rates of transition between phases. We then consider a deterministic approximation of the individual model to model an interacting population. We introduce a system of coupled age-structured partial differential equations to model the evolution of a wild population with two distinct age phases. The goal of this work is to try and understand the evolutionary implications of a discontinuous ageing process, and the behaviour of such a model. The model includes birth, death, transition between phases and non linearities due to competition. We study general properties of the system such as well-posedness, positivity and boundedness under few conditions and using general relative entropy methods. We also propose some particular cases of the system, for which we study the existence and stability of steady states with the semi-group approach. By comparing with classical one-phase ageing models, we motivate the presence of a discontinuity from an evolutionary perspective. Finally, we introduce further assumptions and study the global asymptotic behavior of our system without assuming weak non-linearities.

MS168-03 — Crossing a fitness valley in age-structured populations

SPEAKERS Sarah Kaakai (Sorbonne Paris-Nord)
AUTHORS Manuel Esser (Ecole polytechnique); Marie Doumic-Jauffret (Inria and Ecole polytechnique); Sarah Kaakai (Sorbonne Paris-Nord); Sylvie Méléard (Ecole polytechnique)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The presence of older individuals in natural populations raises critical evolutionary questions: what influence do these individuals exert on population evolution? An example is the so-called Lansing effect, a transgenerational mechanism in which offspring born to older parents have shorter lifespans than those born to younger parents. This phenomenon has been observed across multiple species, but its underlying mechanisms remain debated. From an adaptive dynamics perspective, the persistence of such an effect, which apparently decreases individual fitness, is puzzling. However, if individuals carrying the Lansing mechanism can evolve more rapidly under intermediate mutation rates of power-law order, they could reach a fitter trait faster than those without it, hence ensuring the persistence of this strategy. This scenario is reminiscent of fitness valley crossing, where a population must pass through an intermediate state of reduced fitness to reach a trait of higher fitness.

In this talk, I will extend the framework and results of Bovier, Coquille and Shadi (2019) on fitness valley crossing to an age-structured population dynamics, where individuals are characterized by both their age and a discrete trait. In particular, I will show how accounting for age structure introduces new mathematical challenges, requiring fine results for age-structured populations and renewal equations.

MS175 — Multicellular Reference Models and Applications - The OpenVT Project

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Wednesday, 15 July 2026; 11:10–12:30; 02.11 - HS
ORGANISERS James Glazier (Indiana University); James Osborne (University of Melbourne); Lutz Brusch (TU Dresden)

Minisymposium abstract

Multicellular simulations have become indispensable in understanding complex biological phenomena, from tissue development to disease progression. However, the diversity in simulation methods, ranging from agent-based models to lattice-free models and stochastic particle simulations, poses challenges in terms of reproducibility, modularity, reusability, and integration within multi-scale simulations. The Open VT (Virtual Tissues) project is an engaged international community of researchers - modelers and software engineers who use and develop open source multicellular modeling frameworks. These frameworks allow virtual experiments (simulations) of cell behaviors within tissues and organs.

This minisymposia is part of a pair which aim to present the variety of multicellular simulations being used by the community, along with the efforts to make these simulations replicable and reproducible.

In this part, we will demonstrate the need for standardization and the importance of sharing and reusing models. Within a 40 minute keynote, we present the Open VT projects work on developing a set of Reference Models for multicellular simulations. We then present two scientific applications of multicellular modelling and simulation which demonstrate the potential uses of the developing standards.

MS175-01 — Using reference models to compare multicellular simulation frameworks and tools

SPEAKERS Inge Wortel (Radboud University); Lorenzo Veschini (Indiana Uniersity); Roman Vetter (ETH Zurich)
AUTHORS Inge Wortel (Radboud University); Lorenzo Veschini (Indiana Uniersity); Roman Vetter (ETH Zurich)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:50; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In this round-table discussion, we will examine how much variation can be accepted when the same spatial model is implemented in different software tools, or when similar biological systems are represented using different modelling formalisms. Using examples such as tissue monolayer growth and a basic angiogenesis model, we will discuss where differences arise, how they should be interpreted, and whether full reproducibility is a realistic goal. A central topic will be the choice of comparison metrics. We will ask whether simple summary measures are sufficient to detect meaningful discrepancies, or whether they can hide important differences in spatial behaviour. We will also discuss what level of agreement is reasonable in practice, both across different modelling approaches and across tools based on the same formalism. The goal of this session is to stimulate discussion on practical standards for comparing spatial models and to clarify what reproducibility should mean in the context of spatiotemporal simulation.

MS175-02 — Modeling Blastocyst and Organoid Morphogenesis Towards a Virtual Embryo in Morpheus

SPEAKERS Lutz Brusch (TU Dresden)
AUTHORS Lutz Brusch (TU Dresden)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Multicellular models integrating cell-cell signaling, gene regulation, proliferation and tissue mechanics are needed to unravel the organizational principles of embryo morphogenesis. We() here demonstrate, using FAIR and OpenVT principles, how a subcellular element-based blastocyst model can be reproduced in a cellular Potts model (CPM) framework such as Morpheus []. The open-source framework Morpheus keeps the model definition strictly separated from the simulation code []. It uses the domain-specific language MorpheusML to define multicellular models in a modular manner through a user-friendly GUI []. A numerical simulation is then composed by parsing the MorpheusML model definition and automatic scheduling of predefined components in the simulator. These principles enable sharing and maintenance of the blastocyst model through the MorpheusML model repository []. Extending the blastocyst model, we analyze how cells dynamically polarize and differentiate in response to signals from their environment and how polarized fluid transport controls amniotic cavity formation in a human embryoid culture experiment. () in collaboration with J. Algorta and L. Edelstein-Keshet (UBC, Canada)

References

[] *Blastocyst model*. URL: https://identifiers.org/morpheus/M9999.

[] *Model repository*. URL: https://morpheus.gitlab.io/models.

[] *Morpheus homepage*. URL: https://morpheus.gitlab.io.

[] *MorpheusML language*. URL: https://doi.org/10.25504/FAIRsharing.78b6a6.

MS175-03 — Born or Made? Modeling the Origins of Leader Cells in Collective Cancer Invasion

SPEAKERS Sheriff Akeeb (Georgia State University)
AUTHORS Sheriff Akeeb (Georgia State University)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Collective cancer invasion often displays a distinct spatial hierarchy, with leader cells at the tips of invasive chains and follower cells trailing behind. Experiments using the Spatiotemporal Genomic and Cellular Analysis (SaGA) platform have shown that these subpopulations differ in motile, signaling, and proliferative behavior. But a fundamental question remains unresolved: what makes a leader? Are leader cells a fixed intrinsic subtype, or do leader-like states emerge from local context, biophysical interactions, and phenotypic plasticity? If leaders are removed, can other cells replace them? Here, we develop a computational analogue of the SaGA protocol using a two-dimensional Cellular Potts Model to

address these questions. Each tumor cell is initialized with a randomly assigned combination of adhesion strength, migration coefficient, and proliferative probability, drawn from distributions representing four evolutionary scenarios. Leader- and follower-like behaviors are not imposed a priori, but emerge from the collective invasion dynamics and are classified post hoc by spatial position within the evolving tumor. We systematically investigate how the emergence, stability, and replaceability of leader-like cells depend on four biologically motivated scenarios: an unconstrained random baseline, heritable trait transmission with stochastic perturbation, clonally grouped initialization mimicking discrete sublineages, and biophysical trade-offs among adhesion, motility, and proliferation. By asking when leader cells arise, whether they persist, and whether they can be replaced, this model provides a mechanistic framework for distinguishing fixed cellular identity from context-dependent invasive state. It also offers a platform to study how heterogeneity, plasticity, and trade-offs regulate collective invasion and metastatic potential.

MS14 — Viscoelastic and multifunctional modelling and applications in biology (on the occasion of the 60th birthday of Victor A. Kovtunenko)

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 02.11 - HS
ORGANISERS Andreas Hauptmann (University of Oulu); Hiromichi Itou (Chuo University); Victor Kovtunenko (University of Graz)

Minisymposium abstract

The mechanical models describe many functions of populations, organisms and cells including their motility, proliferation, and morphogenesis. The mathematical modeling is crucial for description of biological cells, soft tissues, and multifunctional materials in various applications stemming from mechanical and environmental engineering, biological and medical sciences. For example, we refer to tumor growth and invasion, nanoindentation and creep relaxation tests, computed tomography scanning and other nondestructive methodologies used for imaging in the applications. From the mathematical point of view, such models are characterized by nonlinear evolutionary PDE and ODE systems with hysteresis, hereditary integrals with memories, fractional derivatives, and nonlocal effects such as adhesion. From a mechanical viewpoint, the most of biological tissues are viscoelastic. Viscoelastic response of cells and tissues represents a class of implicit constitutive relations between the histories of the stress and the relative deformation gradient. Their analysis needs non-standard theoretical methods like hemivariational inequalities, nonlinear inclusions, sweeping processes, maximal monotone graphs for implicit relations, and measure-theoretical tools. It should be supported by advanced numerical techniques, multiscale modelling at different length scales and AI-based computer simulations. Contributions to the fields in a broad scope are highly welcome.

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MS14-05 — Analytical and Numerical Optimization Methods for Solution of Non-smooth Dynamic Impact Problems

SPEAKERS Victor Kovtunenko (University of Graz)
AUTHORS Victor Kovtunenko (University of Graz)
SCHEDULE Thursday, 16 July 2026; 10:40–11:20; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

We are motivated by solution of dynamic variational inequalities appearing in the context of non-smooth optimization subjected to inequality constraints. The use of Lagrange multipliers and merit functions leads to semi-smooth Newton and primal-dual active-set numerical techniques. In the variational framework, we solve dynamic contact and impact problems with discontinuous velocities stemming from multifunctional applications in engineering, geo- and bio-mechanics.

MS14-03 — A Cell–Cell Adhesion Model: Local Well Posedness

SPEAKERS Anna Zhigun (Queen's University Belfast)
AUTHORS Anna Zhigun (Queen's University Belfast); Mabel Lizzy Rajendran (University of Birmingham)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

We present a result on local well posedness for a highly nonlocal nonlinear diffusion–adhesion system [RZ25]. Macroscopic systems of this type were previously obtained through upscaling [ZR24] and can account for the effect of microscopic receptor binding dynamics in cell–cell adhesion. The system couples an integro PDE featuring degenerate diffusion of porous medium type and nonlocal adhesion with a novel nonlinear integral equation. The approach is based on decoupling the system and using Banach's fixed point theorem to solve each of the two equations individually and subsequently the full coupled system. A key challenge lies in identifying a suitable functional setting. One of the main results is the local well posedness of the integral equation with a Radon measure as parameter. The analysis of this equation employs the Kantorovich–Rubinstein norm, marking what appears to be the first use of this norm in the study of a nonlinear integral equation. For details of a multiscale derivation of models of this kind, we refer to [ZR24] and to the talk "A cell–cell adhesion model: a multiscale derivation" in this conference.

References

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MS14-06 — State-Dependent Sweeping Process Approach to Modeling of Networks with Softening

SPEAKERS Ivan Gudoshnikov (Institute of Mathematics, Czech Academy of Sciences)
AUTHORS Ivan Gudoshnikov (Institute of Mathematics, Czech Academy of Sciences)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Bone material can be seen as porous and fiberous, exhibiting the quasi-brittle type mechanical response, with some degree of plasticity. Softening plasticity and fracture mechanics lead to ill-posed mathematical problems due to the loss of monotonicity. Multiple co-existing solutions are possible when softening elements are coupled together, and solutions cannot be continued beyond the point of complete failure of a material. Moreover, spatially continuous models with softening suffer from localization of strains and stresses to measure-zero submanifolds.

We formulate a problem of quasistatic evolution of elasto-plastic spring networks (Lattice Spring Models, suitable for fiberous materials) with a plastic flow rule that allows for softening. The fundamental kinematic and static characteristics of the network are described by the rigidity theory and structural mechanics. The assumptions on the network itself allow for inhomogeneous, disordered or porous structures, such as bone material.

To solve the evolution problem we convert it to a type of a differential quasi-variational inequality known as the state-dependent sweeping process. The existence of solution to the associated time-stepping problem (implicit catch-up algorithm) can be shown, and the associated estimates imply the existence of a solution to the (time-continuous) sweeping process.

Using numerical simulations of regular grid-shaped networks with softening we demonstrate the development of non-symmetric plastic slips. At the same time, in toy examples it is easy to analytically derive multiple co-existing solutions, appearing in a bifurcation which happens when the parameters of the networks continuously change from hardening through perfect plasticity to softening.

MS14-08 — Recent Results on Sweeping Processes with Asymmetric Continuity Properties

SPEAKERS Vincenzo Recupero (Politecnico di Torino)
AUTHORS Vincenzo Recupero (Politecnico di Torino)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Sweeping processes are a class of evolution problems with unilateral constraints which were originally introduced by J.J. Moreau, motivated by problems in elastoplasticity and nonsmooth mechanics. Later they have then found applications in several diverse disciplines: economic theory, electrical circuits, crowd motion modeling, biology.

In his work, Moreau considered moving convex constraints having suitable continuity properties with respect to the asymmetric Hausdorff distance, also called *excess*, which is very natural for evolution problems. His results were later generalized by several authors to non-convex prox-regular constraints, but in the simpler framework of the symmetric Hausdorff distance.

In this talk I will present some recent results where for the first time sweeping processes are driven by non-convex prox-regular constraints having suitable continuity properties with respect to the asymmetric Hausdorff distance. Some of these results were obtained in collaboration with Federico Stra (Politecnico di Torino, Italy).

References

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MS14-07 — Variational Modeling of AFM Indentation of Living Cells with a Pericellular Coat

SPEAKERS Ivan Argatov (Malmö University)
AUTHORS Ivan Argatov (Malmö University)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Qualitative and quantitative variations in mechanobiological markers, such as extracellular matrix stiffness, cell adhesion, and cellular Young's modulus, are strongly correlated with physiological state and frequently associated with pathological conditions. Atomic force microscopy (AFM) enables mechanical testing at the single-cell level, but interpreting indentation data is challenging due to contact nonlinearity and complex cell surface structure. We develop a heuristic variational framework for unilateral indentation in AFM probing of living cells. Interpreting the augmented Lagrangian formulation of quasi-variational inequalities as a Winkler-type compliant coating, we propose a variational model for the indentation of an elastic substrate covered by a nonlinearly deforming, brush-like layer that represents the pericellular coat. Its compressive response follows the Alexander–de Gennes model, capturing strong nonlinearity. Building on Itou, Kovtunenko, and Rajagopal's general solution for viscoelastic substrates with non-increasing contact area, we derive explicit displacement–force relations for monomial (axisymmetric) and self-similar (non-axisymmetric, e.g., Berkovich pyramidal) indenters. The proposed formulation provides a mathematically consistent route to separating bulk cellular mechanics from pericellular coat effects, thereby improving the interpretation of AFM measurements in mechanobiology.

MS14-04 — On the Mathematical Analysis of Generalized Fractional Viscoelastic Models

SPEAKERS Hiromichi Itou (Chuo University)
AUTHORS Hiromichi Itou (Chuo University); Masahiro Yamamoto (The University of Tokyo); Victor Kovtunenko (University of Graz)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Viscoelastic materials appear in a wide range of fields: engineering, biology, geophysics, etc., such as synthetic polymers, biological tissues and concrete. In this talk we discuss a mathematical model of nonlinear fractional viscoelastic materials within the context of infinitesimal strain theory under quasi-static situation. Constitutive relations of such a generalized fractional viscoelastic models (shortly GFV models) are given by Volterra hereditary integrals with creep functions described by Prony series replaced with a Mittag-Leffler function. For a boundary value problem of the GFV model, we show the existence of a solution. Moreover, we also perform a numerical simulation for one-dimensional creep test under isotropic expansion, analyzing the effects of the fractional derivative order and non-linearity on material behavior in each of three stages: loading, maintaining, and release stages of the creep test.

MS14-02 — Towards Robust Quantitative Photoacoustic Tomography

SPEAKERS Andreas Hauptmann (University of Oulu)
AUTHORS Andreas Hauptmann (University of Oulu)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 02.11 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In photoacoustic tomography, biological tissue is illuminated with a short laser pulse of near infrared light. The absorbed energy creates a local pressure increase that propagates through the tissue, governed by the acoustic wave equation and can be measured on the boundary. From this measured time-series the initial pressure in the tissue is reconstructed, providing valuable information on local structures. Subsequently, it is possible to recover the quantitative optical parameters of absorption and scattering. Correct recovery of the optical parameters would provide valuable functional and biological information. Solving both the acoustic and optical inverse problem comes with challenges. From limited-view geometries to modelling errors and uncertainties. The above challenges can be effectively mitigated by training a learned reconstruction method, but three crucial ingredients are necessary: a learned method with good generalisability for out-of-distribution data, a computationally fast model to allow for feasible training and inference times, and finally reference data for the training procedure. Here, we use a learned model-based iterative reconstruction. A novel fast FFT based approach to solve the acoustic problem in circular geometries. And finally, training and evaluation using a digital twin providing a link between experimental and simulated data. Reconstructions are presented for experimental data and the digital twin.

References

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MS26 — MBI Community Gathering: Emerging Methods and Mathematical Models Arising from Biology

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 02.23 - HS
ORGANISERS Farrah Sadre-Marandi (qPharmetra, LLC); Wenrui Hao (Penn State University)

Minisymposium abstract

This minisymposium brings together members of the Mathematical Biosciences Institute (MBI) community to highlight the latest mathematical methods, models, and applications inspired by biological questions. The session showcases the breadth of mathematical biology, including stochastic and continuum models of molecular and cellular processes, network dynamics in infectious diseases and ecological systems, and data-informed modeling in biomedical applications. Speakers will present research that integrates innovative mathematical techniques with real-world biological problems, reflecting both methodological development and biological insight. The session also celebrates the ongoing impact of MBI by featuring former postdoctoral fellows and visitors whose research, collaborations, and career trajectories have been shaped by their experience at the Institute. Overall, this minisymposium emphasizes methodological innovation, interdisciplinary collaboration, and community building within mathematical biology, with a focus on integrating mathematical modeling and biological insight to foster discussions that inspire

new directions in the field. This minisymposium aims to foster inclusive and respectful environment for scientific exchange, promote cross-disciplinary interaction, and encourage new collaborations across diverse communities engaged in mechanistic and data-driven biological modeling.

MS26-01 — Entropy-Based Information Sharing for Uncertainty Quantification in Multi-Layer Complex Systems

SPEAKERS Grzegorz Rempala (Ohio State)
AUTHORS Grzegorz Rempala (Ohio State)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

We propose an entropy-based framework for uncertainty quantification in settings involving multiple, heterogeneous data sources. The central idea is to represent each empirical layer through an entropy-induced probability measure, allowing information to be shared and propagated across layers in a principled and consistent manner. This approach provides a natural mechanism for reconciling uncertainty arising from observational, experimental, and model-based components, while enabling interpretable variance–covariance decompositions analogous to ANOVA.

As an illustrative example, we reference recent work on random-measure-based sensitivity analysis in randomized controlled trials (Bastian, Rabitz, and Rempala, 2025), where entropy-consistent measures are used to quantify and decompose uncertainty across treatment and outcome spaces. While arising in a clinical context, this example highlights the broader applicability of entropy-driven information sharing for uncertainty quantification in complex, multi-layer systems.

MS26-02 — Algebraic, Geometric, and Combinatorial Approaches to Modeling Gene Regulatory Networks

SPEAKERS Brandilyn Stigler (Southern Methodist University)
AUTHORS Brandilyn Stigler (Southern Methodist University)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The challenge of developing predictive models for gene regulatory networks has motivated a multitude of approaches, ranging from the discrete to the continuous, the deterministic to the probabilistic. In many settings, however, available experimental data do not determine a unique model: time series or input-output data typically admit multiple network structures or dynamical rules that are equally consistent with the observations and are computationally indistinguishable. This ambiguity raises a fundamental question at the interface of biology and mathematics: how can one design experiments that more effectively discriminate among competing models?

In this talk, I will highlight algebraic, geometric, and combinatorial methods for modeling gene regulatory networks and focus on reducing model uncertainty through improved experimental design. The central idea is to use structural properties of model classes to identify data that are maximally informative for model selection. This perspective has led to new questions and results involving identifiability, algebraic characterization of candidate models, and the geometry of data.

MS26-03 — Chemical Reaction Networks with Stochastic Switching Behavior and Machine Learning Applications

SPEAKERS Hye-Won Kang (University of Maryland, Baltimore County)
AUTHORS Hye-Won Kang (University of Maryland, Baltimore County)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In this talk, we examine switching behavior in stochastic reaction networks, where molecular copy numbers fluctuate between multiple distinct states. Such switching occurs when intrinsic noise drives transitions between multiple stable or quasi-stable states, rather than through deterministic oscillations. Stochastic switching has been observed in various biological phenomena, including gene expression, cell fate decisions, and biochemical oscillators with noise-induced transitions. We focus on two models exhibiting this behavior, emphasizing that while both display state switching, their underlying mechanisms differ significantly. Using stochastic simulations, we analyze and compare the dynamics of the two models, tuning parameters so that their trajectories appear similar. From these oscillatory time series, we extract key features and apply several measures and classification techniques to determine whether the models can be distinguished. Additional preliminary results, including extensions to other network architectures and parameter regimes, will also be discussed. This work is conducted jointly with Dongli Deng at UMBC.

MS26-05 — Generative models for particle tracking microscopy

SPEAKERS Jay Newby (University of Alberta)
AUTHORS Jay Newby (University of Alberta)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

I will discuss the use of diffusion models for particle tracking microscopy images. The goal is to have a fully integrated generative model that connects stochastic models of particle motion to microscopy image data. Unfortunately, the observation likelihood function is too complex to explicitly model, and we do not know this function. Learning the likelihood function from a suitably large image set is the primary purpose of so-called diffusion models. We discuss the application of these neural network models for evaluating the log likelihood of image sets given particle positions.

MS26-04 — From Blackboard to Bedside: Translating Mathematical Sleep Models into Samsung Galaxy Watch

SPEAKERS Jae Kyoung Kim (KAIST)
AUTHORS Jae Kyoung Kim (KAIST)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Standard recommendations of 7–9 hours of sleep do not reliably guarantee daytime alertness. Using wearable-derived sleep-wake data, we infer two latent physiological states—homeostatic sleep pressure and circadian phase—via a mathematical model, and generate personalized sleep-wake schedules aligned with each individual's circadian rhythm. In two prospective clinical trials, adherence to model-based schedules significantly improved alertness, with circadian alignment proving a far stronger predictor than total sleep time alone. These findings led to deployment of our algorithm across all Samsung Galaxy Watch devices—the first mathematical biology model adopted by a major tech company. Extending this framework, we show that mathematically derived circadian features from sleep-wake data accurately predict mood episodes in bipolar disorder, outperforming models that rely on richer but more invasive data. Finally, we introduce HADES-NN, a neural network method for optimizing circadian models under real-world discontinuous light signals, enabling future model personalization at scale.

MS26-06 — Toward Predictive Digital Twins for Precision Oncology: A QSP Modeling Approach

SPEAKERS Leili Shahriyari (UMASS Amherst)
AUTHORS Leili Shahriyari (UMASS Amherst)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In this talk, I present a computational approach to precision oncology that combines mechanistic modeling with modern machine learning to build predictive digital twins; patient- and subgroup-specific models that forecast tumor progression and response to therapy. Our foundation is quantitative systems pharmacology (QSP) modeling, where tumor–immune dynamics are represented as systems of differential equations. We parameterize these models using patient observations and introduce a clustered inference strategy that stratifies individuals by immune profiles and estimates mechanistic parameters within each cluster. This reduces heterogeneity, improves identifiability, and yields interpretable subgroup phenotypes that link immune state to dynamical behavior. A distinguishing aspect of our evaluation is out-of-treatment generalization: we calibrate tumor growth and immune dynamics without fitting treatment response data, then use the resulting mechanistic parameters to predict outcomes under therapy. This design sharpens the scientific test of the model and targets the real-world challenge of predicting under new regimens. I then describe a digital twin platform that operationalizes this pipeline.

MS26-07 — Exploration of structure learning in pediatric sickle cell data

SPEAKERS Reginald McGee (Haverford College)
AUTHORS Reginald McGee (Haverford College)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Pain episodes are a defining feature of sickle cell disease and can lead to reduced quality of life. Statistical analyses by Valrie et al. (2021) examined interactions between physiological (like pain severity and sleep quality) and psychosocial variables (like positive and negative affect) in pediatric sickle cell patients. Clinical datasets can present challenges for continuous dynamical systems modeling as there may be a mix of continuous data and categorical data (for instance, whether a patient took a nap, disease genotype, or the type of medication taken) recorded within the dataset, and there may not be obvious biophysical relationships to guide modeling the variables. In this work-in-progress talk, we consider probabilistic graphical models (or Bayesian networks) which are flexible enough to address the mix of categorical and continuous data types present in a clinical dataset. We will explore the learned model structures between variables for subpopulations of patients defined by age.

MS26-08 — Asthma-mediated control of optic glioma growth via T cell-microglia interactions.

SPEAKERS Yangjin Kim (Konkuk University)
AUTHORS Yangjin Kim (Konkuk University)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 02.23 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Optic glioma, a slow-growing tumor, is associated with Neurofibromatosis type 1 (NF1) mutations and increased midkine (MDK) production. A connection between asthma and optic glioma has previously been observed, but the mechanisms are unclear. To elucidate the role of asthma in the regulation of glioma formation, we investigated the role of T cells and the subsequent pathways in the regulation of microglia, a key player in the glioma tumor microenvironment (TME). While asthma is often linked to chronic inflammation, our mathematical analysis and experimental evidence suggest that inflammation can play a key role in suppressing the proliferation of optic glioma cells via immune reprogramming of T cells and the delicate control of signaling networks in microglia. Through a mathematical model, we investigate the complex interactions between tumor and immune cells in optic glioma. Our results indicate that asthma-induced T cell reprogramming inhibits tumor growth by promoting the release of decorin and a subsequent suppression of CCR8 and the intercellular binding kinetics in microglia, followed by blocking of CCL5 production in TME via suppression of NFκB. We also developed anti-cancer strategies by leveraging this asthma-induced immune regulation. Midkine intracellular dynamics also suggest a

critical link between stromal cells and glioma cells. Our study based on hybrid multi-scale models suggests that the critical role of asthma control can perturb TME near optic glioma, leading to a critical cue on development of multi-organ anti-cancer therapeutics in optic glioma.

MS45 — Data-driven modeling in biology and medicine

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 15.06 - HS
ORGANISERS Kang-Ling Liao (University of Manitoba, Winnipeg, MB, Canada); Wenrui Hao (Pennsylvania State University, University Park, PA, USA)

Minisymposium abstract

Mathematical modelling and computation allow for quantitative testing of proposed hypotheses and estimation of important physical and biological parameters. Combining experiments with mathematical modelling allows a rigorous validation of model hypotheses, thereby providing a powerful investigation tool in biology and medicine. The focus of this session will be on applications of mathematics and modelling to the understanding of experiments in biological sciences and medicine.

MS45-07 — Parameter identifiability for the Data-Driven Modeling

SPEAKERS Wenrui Hao (Department of Mathematics, Pennsylvania State University, University Park, PA, USA)
AUTHORS Wenrui Hao (Department of Mathematics, Pennsylvania State University, University Park, PA, USA)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Integrating high-dimensional biological data into mechanistic models requires practical identifiability for reliable inference. We develop a computational framework that reveals scaling laws for identifiability via asymptotic analysis, combining Fisher information with perturbed Hessians to quantify identifiability and uncertainty in non-identifiable subspaces. Validated on several benchmark data-driven models, the framework uncovers key mechanisms and provides principled scaling laws for data-driven mechanistic modeling and digital twins.

MS45-01 — Effects of Cellular Memory and Adaptation Cost on Optimal Survival in Fluctuating Environments

SPEAKERS Paras Jain (Indian Institute of Science)
AUTHORS Jason T. George (Department of Biomedical Engineering, Texas A&M University and Translational Medical Sciences, Texas A&M Health Science Center and Center for Theoretical Biological Physics, Rice University and Hematopoietic Biology and Malignancy, MD Anderson Cancer Center); Mohit Kumar Jolly (Indian Institute of Science); Paras Jain (Indian Institute of Science)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cells invariably encounter unpredictable changes in their microenvironment and adapt by orchestrating substantial alterations in their molecular states. The timescale of molecular adaptation depends on how quickly a cell loses its molecular memory of past environmental encounters through the degradation rate of proteins unfavorable to the current environment. Concurrently, de novo synthesis of favorable biomolecules during adaptation imposes an energetic cost that impacts cellular fitness. Here, by developing a phenomenological model of intracellular processing of environmental signals and associated cell-state switching, we study the dynamical implications of cellular memory and adaptation cost on cellular responses to a changing environment. We find that while increasing cellular memory reduces cell fitness in periodic environments, counterintuitively, increasing adaptation cost can improve growth by minimizing mismatches between the environment and the cell state. Similarly, we observed a variable role of memory capacity

and adaptation cost for stochastic correlated environments. Lastly, we show that cellular memory and population heterogeneity in adaptation cost explained reported experimental observations in melanoma: increased population survival during drug treatment when the population was either enriched for rare cells expressing resistance marker genes or primed with low-dose drug treatment before exposure to high-dose treatment. Overall, this work establishes a foundational model for cellular adaptation under different environmental conditions and explain complex cellular behaviors.

MS45-02 — Multi-omics Investigation Reveals Molecular Determinants of Cancer Cell Evolution on Soft Extracellular Matrix

SPEAKERS Sarthak Sahoo (Department of Bioengineering, Indian Institute of Science, Bengaluru, India)
AUTHORS Irtisha Singh (Texas A&M University, College Station, TX, USA); Mohit Kumar Jolly (Indian Institute of Science, Bengaluru, Karnataka, India); Sarthak Sahoo (Department of Bioengineering, Indian Institute of Science, Bengaluru, India); Sejal Khanna (Indian Institute of Science, Bengaluru, Karnataka, India); Tanmay P. Lele (Texas A&M University, College Station, TX, USA); Ting-Ching Wang (Texas A&M University, College Station, TX, USA)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cancer cell adaptation to their physical tumor microenvironment is a key driver of malignancy. Recent experimental evolution experiments show that the soft extracellular matrix (ECM) can impose a selection pressure on genetically variable tumor populations. Over months of sustained culture, the selection pressure leads to enrichment of specific genetic variants with high fitness, but the mechanisms underlying the high fitness of these soft-selected clones are not fully understood. Here, we used a combination of RNA-seq, ATAC-seq, and RRBS-seq to compare soft-selected populations with non-selected ancestral populations cultured on soft ECM. We demonstrate that ancestral populations grown on soft ECM for short durations are characterized by a low fitness cell state marked by cell cycle arrest whereas sustained culture selects for a robust proliferative phenotype. Mechanistically, selected cells exhibit a silenced ancestral stress response through epigenetic modifications, with reduced chromatin accessibility and de-novo DNA methylation. This repressive landscape supports a high-fitness state defined by elevated MYBL2 and FAK levels. An in-silico mechanism-based model shows that these molecular differences, together with high YAP1 nuclear localization in soft-selected cells, are key features of genetic clones capable of FAK upregulation. These findings uncover a coordinated genetic and epigenetic mechanism driving cancer cell evolution in mechanically soft substrates.

MS45-06 — Model Selection and Treatment Prediction in ODE Models for ER+ Breast Cancer

SPEAKERS Kang-Ling Liao (Department of Mathematics, University of Manitoba, Winnipeg, MB, Canada)
AUTHORS Adam J. Wieler (University of Waterloo, Waterloo, ON, Canada); Kang-Ling Liao (Department of Mathematics, University of Manitoba, Winnipeg, MB, Canada)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In this talk, I will introduce four systems of ODEs to identify the most plausible model and the key reactions governing the dynamics of the tumor microenvironment (TME) in estrogen receptor–positive (ER+) breast cancer. All models quantitatively fit the experimental data for radiation therapy (RT) and endocrine therapy fulvestrant (Fulv) in TC11 ER+ cells, as well as RT and immune checkpoint inhibitor (anti-PD-1/anti-PD-L1) treatments in 4T1-HA and MCF-7 breast tumor cells. The Akaike information criterion analysis suggests that interactions among tumor cells, $CD8^+$ T cells, and estrogen are the primary drivers of the dynamics in the TC11 and MCF-7 cell lines, whereas interactions involving dendritic cells (DCs) and M2 macrophages are required to capture the dynamics in the 4T1-HA cell line. Global sensitivity analysis and identifiability analysis are then conducted to evaluate the uniqueness of parameter values in model calibration and to suggest additional data points that may improve model

reliability. Furthermore, numerical simulations and model comparisons indicate the optimal treatment protocols for different cell lines and provide guidance for additional experimental designs to identify the most plausible model.

MS45-04 — How Random Forces Help Rather Than Hinder Dynein Cargo Transport

SPEAKERS John Dallon (Department of Mathematics, Brigham Young University, Provo UT, USA)
AUTHORS Chris Grant (Brigham Young University, Provo UT, USA); Emily Evans (Brigham Young University, Provo UT, USA); John Dallon (Department of Mathematics, Brigham Young University, Provo UT, USA); Stephanie Portet (University of Manitoba, Winnipeg, MB, Canada)
SCHEDULE Thursday, 16 July 2026; 15:10–15:50; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Recent experimental work has shown that dynein transports cargo faster in vivo than in vitro, although counter intuitive, the results are consistent with other experimental data [Tor+25]. Typically, it is assumed that the cytoplasm, a more viscous and complex environment, should slow down transport compared to in vitro experiments. It is postulated that the catch bond nature of dynein allows this counter intuitive result. In this talk I will discuss a stochastic tug-of-war model of dynein transport that sheds light on this phenomena.

References

[Tor+25] Takayuki Torisawa et al. “Active fluctuations of cytoplasmic actomyosin networks facilitate dynein-driven intracellular transport along microtubules”. In: *iScience* 28.114096 (2025).

MS45-03 — How Intermediate Filaments Move: Experiments and Models

SPEAKERS Stephanie Portet (Department of Mathematics, University of Manitoba, Winnipeg, MB, Canada)
AUTHORS John C. Dallon (Brigham Young University, Provo, Utah, USA); Leticia Arias (University Claude Bernard Lyon 1, Lyon, France); Pascale Bomont (University Claude Bernard Lyon 1, Lyon, France); Sandrine Etienne-Manneville (Institut Pasteur, Paris, France); Stephanie Portet (Department of Mathematics, University of Manitoba, Winnipeg, MB, Canada); Youngmin Park (University of Florida, Gainesville, Florida, USA)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Intermediate filaments are key components of the cytoskeleton, playing essential roles in cell mechanics, signaling and migration. Their spatiotemporal organization, which supports these functions, results from the interplay between assembly and disassembly processes, as well as retrograde actin flow and motor protein–driven transport. Investigating the different mechanisms involved in the intracellular transport of intermediate filaments requires a combination of experimental setups and complementary biological and mathematical models. In this talk, several examples illustrating these approaches will be presented.

MS45-05 — Senolytic Drugs Reduce Cancer Resistance to PD-1 Blockade

SPEAKERS Nourridine Siewe (School of Mathematics and Statistics, Rochester Institute of Technology, Rochester, NY, USA)
AUTHORS Nourridine Siewe (School of Mathematics and Statistics, Rochester Institute of Technology, Rochester, NY, USA)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Resistance to immune checkpoint inhibitors (ICI), including anti-PD-1 and anti-PD-L1 therapies, remains a major limitation in cancer treatment, with most patients relapsing despite initial responses. Previous studies have shown that inhibition of TNF-alpha or TGF-beta overcome resistance to anti-PD-1. Here,

we develop a mathematical model to investigate a mechanism of ICI resistance driven by age-associated senescence of CD8+ T cells. In the model, senescent T cells secrete VEGF, thereby promoting tumor growth through enhanced vascular support. To counteract this effect, we incorporate the senolytic drug navitoclax (ABT-263), which selectively eliminates senescent T cells, and assess the efficacy of combined senolytic-ICI therapy in extending tumor remission. Model predictions are validated against independent experimental data from murine studies involving navitoclax and anti-PD-L1 treatment. Using the validated framework, we conduct in silico clinical trials simulating multi-cycle anti-PD-1 treatment in young and old patient cohorts with different timings of senolytic administration. The results demonstrate that the optimal timing of navitoclax depends on immune age: maximal remission extension is achieved when senolytic therapy is initiated in the second treatment cycle in older patients, whereas earlier administration is most effective in younger patients. These findings highlight immune senescence as a key determinant of response to cancer immunotherapy and suggest that age-adapted scheduling of senolytic drugs may improve the durability of ICI-based treatments.

MS60 — Identifying the origin and consequences of non-genetic cell-to-cell variability

TRACK Cellular and Developmental Biology
SCHEDULE Thursday, 16 July 2026; 10:40–12:00; 11.02 - HS
ORGANISERS Chiara Villa (CNRS & MAP5, Université Paris Cité); Kaan Öcal (University of Melbourne)

Minisymposium abstract

The complexity of biological systems resides in the stochastic fluctuations and interactions between their many components. Even among genetically identical cells, there exists significant heterogeneity in the expression of mRNAs and proteins. This non-genetic cell-to-cell variability plays a central role in shaping population- and tissue-scale dynamics, influencing proliferation, survival, movement, and spatial organisation. Understanding how such variability arises and propagates across scales is therefore key to linking cellular-level noise with emergent collective behaviour. Mathematical and computational models, informed by experimental data, provide a framework to identify effective sources of variability and to quantify their impact on collective dynamics. Variability in transcriptional activity, receptor expression, and cellular mechanical properties can lead to heterogeneous proliferation rates, distinct motility behaviours, and diverse responses to environmental cues. Although the classical view of noise is that it is detrimental to cellular function, phenotypic noise and plasticity can have significant benefits, including robustness and adaptability in variable environments. This mini-symposium brings together speakers studying how non-genetic variability influences emergent dynamics across biological scales, and how its sources can be identified using mathematical models informed by data.

MS60-01 — Identifiability of phenotypic adaptation from low-cell-count experiments and a stochastic model

SPEAKERS Alexander Browning (University of Melbourne)
AUTHORS Adrianne Jenner (Queensland University of Technology); Alexander Browning (University of Melbourne); Chiara Villa (CNRS & MAP5 (UPC)); Philip Maini (Mathematical Institute, Oxford University); Rebecca Crossley (University of Oxford); Sara Hamis (Uppsala University); Tyler Cassidy (University of Leeds)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 11.02 - HS
TRACK Cellular and Developmental Biology

Abstract

In this talk, we develop a stochastic individual-based model of phenotypic adaptation through a continuously structured phenotype space. Probabilistically, our model corresponds to common partial differential equation models of phenotypic heterogeneity, allowing us to formulate a likelihood that captures the intrinsic noise ubiquitous to low-cell-count proliferation assays. We apply our framework to study the identifiability of key model parameters relating to the adaptation velocity and cell-to-cell heterogeneity. Significantly, we find that cell-to-cell heterogeneity is practically non-identifiable from both cell count and proliferation marker data, implying that population-level behaviours may be well characterised by homogeneous ordinary differential equation models.

References

[Bro+25] Alexander P Browning et al. “Identifiability of phenotypic adaptation from low-cell-count experiments and a stochastic model”. In: *PLoS Computational Biology* 21.6 (2025), e1013202.

MS60-02 — From noisy cell size control to population growth: When variability can be beneficial

SPEAKERS Arthur Genthon (Max Planck Institute for the Physics of Complex Systems)
AUTHORS Arthur Genthon (Max Planck Institute for the Physics of Complex Systems)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 11.02 - HS
TRACK Cellular and Developmental Biology

Abstract

Single-cell experiments revealed substantial variability in generation times, growth rates, birth and division sizes between genetically identical cells. Understanding how these fluctuations determine the fitness of the population, i.e. its long-term growth rate, is necessary in any quantitative theory of evolution. In this talk, I will present a biologically relevant agent-based model of population dynamics which accounts for single-cell stochasticity. I will derive expressions for the population growth rate and mean birth size in the population in terms of single-cell fluctuations. Allowing division sizes to fluctuate reveals how the mechanism of cell size control (timer, sizer, adder, ...) influences population growth. Surprisingly, we find that fluctuations in single-cell growth rates can be beneficial for population growth when slow-growing cells tend to divide at smaller sizes than fast-growing cells. Our framework is not limited to exponentially growing cells like Escherichia coli, and we derive similar expressions for cells with linear and bilinear growth laws, such as Mycobacterium tuberculosis and fission yeast Schizosaccharomyces pombe, respectively.

MS60-03 — A geometric surface PDE model for cell–nucleus translocation through confinement

SPEAKERS Francesca Ballatore (Université Côte d’Azur, Laboratoire J. A. Dieudonné)
AUTHORS Anotida Madzamuse (LJAD CNRS, University of British Columbia, University of Zimbabwe, University of Pretoria); Cécile Jebane Payet (Aix-Marseille Université, CNRS, CINAM); Emmanuèle Helfer (Aix-Marseille Université, CNRS, CINAM); Francesca Ballatore (Université Côte d’Azur, Laboratoire J. A. Dieudonné); Rachele Allena (LJAD CNRS, Institut Universitaire de France)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 11.02 - HS
TRACK Cellular and Developmental Biology

Abstract

Understanding how cells migrate through confined environments is crucial for elucidating fundamental biological processes, including cancer invasion, immune surveillance, and tissue morphogenesis. The nucleus, as the largest and stiffest cellular organelle, often limits cellular deformability, making it a key factor in navigating narrow pores or highly constrained spaces. In this talk, I will present a novel geometric surface partial differential equation (GS-PDE) framework in which the cell plasma membrane and the nuclear envelope are modeled as evolving energetic closed surfaces governed by force-balance equations. To validate the model, we replicate a biophysical experiment using a microfluidic device that imposes compressive stresses on cells driven through narrow microchannels under a controlled pressure gradient. I will discuss the results of our parametric sensitivity analysis, which highlights the dominant influence of specific parameters, such as surface tension and confinement geometry, as key determinants of translocation efficiency. Finally, I will show how this framework, while tailored to a specific experimental setup for validation, provides a robust, flexible, and generalizable tool for investigating the broader interplay between cell mechanics and confinement, laying the groundwork for integrating more complex biochemical processes like active migration.

MS60-04 — Trait-structured chemotaxis: Exploring ligand-receptor dynamics and travelling wave properties in a Keller-Segel model

SPEAKERS Viktoria Freingruber (TU Delft)
AUTHORS Kevin J. Painter (Politecnico di Torino, Torino, IT); Mariya Ptashnyk (Heriot-Watt University, Edinburgh, Scotland, UK); Tommaso Lorenzi (Politecnico di Torino); Viktoria Freingruber (TU Delft)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 11.02 - HS
TRACK Cellular and Developmental Biology

Abstract

A novel trait-structured Keller-Segel model that explores the dynamics of a migrating cell population guided by chemotaxis in response to an external ligand concentration is derived and analysed. Unlike traditional Keller-Segel models, this framework introduces an explicit representation of ligand-receptor bindings on the cell membrane, where the percentage of occupied receptors constitutes the trait that influences cellular phenotype. The model posits that the cell's phenotypic state directly modulates its capacity for chemotaxis and proliferation, governed by a trade-off due to a finite energy budget: cells highly proficient in chemotaxis exhibit lower proliferation rates, while more proliferative cells show diminished chemotactic abilities. The model is derived from the principles of a biased random walk, resulting in a system of two non-local partial differential equations, describing the densities of both cells and ligands. Using a Hopf-Cole transformation, we derive an equation that characterises the distribution of cellular traits within travelling wave solutions for the total cell density, allowing us to uncover the monotonicity properties of these waves. Numerical investigations are conducted to examine the model's behaviour across various biological scenarios, providing insights into the complex interplay between chemotaxis, proliferation, and phenotypic diversity in migrating cell populations.

MS72 — SMB MathOnco Subgroup Mini-Symposium: Emerging Themes in Mathematical Oncology

TRACK Mathematical Oncology
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 15.04 - HS
ORGANISERS Jana Gevertz (The College of New Jersey); Rebecca Bekker (The University of Texas MD Anderson Cancer Center); Thomas Hillen (University of Alberta)

Minisymposium abstract

This mini-symposium highlights emerging themes in mathematical oncology, focusing on recent methodological advances and clinical applications. Topics include emerging methodologies in spatiotemporal modeling, optimization, immune dynamics, and mechanistic learning. These methodologies will be explored in the context of the physical tumor microenvironment, therapeutic design, and the development of patient-specific digital twins. Collectively, these talks highlight how rigorous modeling can both deepen biological insight and support the development of predictive, translational tools in oncology.

MS72-03 — A quantitative and phenotype-resolved model for melanoma cell populations

SPEAKERS Keith Chambers (University of Oxford)
AUTHORS Keith Chambers (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Melanoma is an aggressive skin cancer driven by a phenotypically heterogeneous cell population. While a full mechanistic understanding is currently lacking, the leading micropthalmia-associated transcription factor (MITF) rheostat theory asserts that the downstream activity of MITF regulates transitions between differentiated, proliferative and invasive states. The population dynamics implied by the MITF rheostat

and the conditions for which these dynamics are consistent with published data are currently unknown. To address this, we have developed a phenotype-structured model for melanoma cell populations in vivo. In this talk, I first introduce a subcellular SDE system that illustrates how stochastic fluctuations in MITF RNA and protein concentrations propagate to a downstream phenotype variable. By exploiting a timescale separation, we reduce the coupled SDEs to an effective phenotype flux that informs the full structured population model. Numerical solutions indicate that the model population transitions from a balanced exponential growth phase with mainly differentiated and proliferative cells to either a steady state or limit cycle. Both long-term behaviours exhibit a substantial proportion of invasive cells. Parameter value calibration via Bayesian inference indicates that the "window" of proliferative phenotypes must be small for consistency with data. Together, these results clarify the role of MITF in shaping the phenotype heterogeneity of melanoma cell populations.

MS72-05 — Spin Glass Active Inference for Cancer Stochastic Switching Systems

SPEAKERS Linh Huynh (Dartmouth College)
AUTHORS Linh Huynh (Dartmouth College)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Stochastic switching systems arise in a wide range of applications in which agents adapt to dynamically changing environments. Two seemingly distinct yet analogous applications are cancer cells adapting to drug environments and large language models adapting to human interaction environments. In this work, I present a collective active inference framework for such systems, in which agents make optimal decisions by quantifying uncertainty from partially observable data. This framework is related to sampling from the stationary distribution of a spin glass model known as a Boltzmann machine. More broadly, this work establishes a new direction for applying spin glass theory to cancer evolution and artificial intelligence.

MS72-06 — Oncolytic Virotherapy: Mathematical Analysis and Optimization

SPEAKERS Negar Mohammadnejad (University of Alberta)
AUTHORS Negar Mohammadnejad (University of Alberta)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.04 - HS
TRACK Mathematical Oncology

Abstract

This talk focuses on the mathematical modeling of oncolytic virotherapy (OVT), based on joint work with T. Hillen. We begin with a qualitative analysis of an extended model that captures the interactions between tumor cells, viruses, and the immune system. Building on this foundation, we investigate strategies to improve treatment efficacy, showing through analysis and simulations that combination therapies and optimized protocols can significantly outperform monotherapy approaches. A central theme of this work is the emergence and role of travelling wave solutions, which describe the spatial spread of infection and its interaction with both the tumor and the immune response. We present numerical evidence of complex wave dynamics and establish their existence rigorously using analytical techniques.

MS72-04 — Agent-Based Modeling of Tumor-Immune Interactions and Therapeutics in the Tumor Microenvironment

SPEAKERS Kerri-Ann Norton (Bard College)
AUTHORS Kerri-Ann Norton (Bard College)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

The tumor microenvironment is a complex system involving cross-talk between tumor cells, stromal cells, and therapeutics in the microenvironment. One major avenue of my research has been the interactions between cancer cells, immunotherapy, and immune cells. In order to examine this, we developed an agent-based model that examines the interplay between cancer cells and their surrounding host environment, including blood vessels, hypoxia, therapeutics, and immune cells. This talk will look at the various computational models that examine tumor therapeutics and the tumor microenvironment. This will include monotherapies such as anti-angiogenesis therapies, salinomycin, T-cell therapy, and macrophage polarity as well as combination therapies. This type of modeling allows us to predict under which conditions immunotherapy would be most successful under different assumptions.

MS72-01 — A Multiscale Mathematical Framework for Modeling Ductal Deformation in Early Pancreatic Neoplasia

SPEAKERS Daniel Bergman (University of Maryland Baltimore)
AUTHORS Daniel Bergman (University of Maryland Baltimore)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Pancreatic cancer precursor lesions (PanINs and IPMNs) generate pronounced ductal deformations, arising from reciprocal interactions between epithelial mechanics, basement membrane integrity, and stromal remodeling. Yet the dynamic feedback loops governing this structural evolution remain poorly characterized. We present a multiscale mathematical modeling framework that couples ductal epithelial cells, the basement membrane, and cancer associated fibroblasts (CAFs). The model integrates mechanistic rules for cell-matrix adhesion, epithelial mechanical responses, and CAF driven matrix remodeling to characterize how specific parameter combinations generate the ductal morphologies observed in precursor lesions. Simulations reveal how variations in epithelial stiffness, basement membrane degradation kinetics, and CAF remodeling rates produce canonical deformation modes consistent with histological sections from resected human pancreas tissues. Current work extends the framework to enable predictive exploration of lesion progression, identifying parameter regimes that shift lesions toward increased morphological irregularity and potential invasiveness. This modeling approach provides a mathematically rigorous platform for quantifying multicellular interactions and uncovering mechanistic drivers of early pancreatic neoplastic evolution.

MS72-07 — Mechanistic learning for spatio-temporal brain tumor response predictions

SPEAKERS Sarah Brueningk (University of Bern)
AUTHORS Sarah Brueningk (University of Bern)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Magnetic resonance imaging (MRI) is central to diagnosis, longitudinal monitoring, and response assessment in brain tumors, motivating predictive models that operate not only on volumetric trajectories but directly in the imaging domain. While mechanistic models capture individualized tumor dynamics and treatment effects, they compress the spatial and anatomical information contained in radiographic data; conversely, generative AI solutions such as guided denoising diffusion models, enable anatomically coherent prediction of future MR images and tumor growth probability maps, but require temporal structure that is often difficult to learn from limited follow-up data. Hybrid mechanistic-learning approaches are therefore particularly suitable in this application, where longitudinal imaging is sparse and irregularly sampled, especially in rare pediatric tumors. By coupling biologically plausible ordinary differential equation models of tumor growth with guided denoising diffusion models, these frameworks combine interpretable estimates of future tumor burden with patient-specific image synthesis and spatially resolved progression maps. This integration supports biologically informed, anatomically meaningful prediction of spatio-temporal

tumor evolution and provides a foundation for improved response assessment, treatment monitoring, and radiotherapy planning.

MS72-08 — Mechanistic computational modeling of clonal dynamics in the aging hematopoietic system

SPEAKERS Thomas Stiehl (RWTH Aachen University)
AUTHORS Thomas Stiehl (RWTH Aachen University)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Blood cell formation is maintained throughout the lifespan of an organism by a small population of hematopoietic (blood-forming) stem cells (HSCs). HSCs sustain their population through self-renewal while simultaneously giving rise to more differentiated offspring, referred to as progenitors and precursors, that mature into the various blood cell types. Over time, HSCs accumulate mutations that can confer growth advantages and trigger clonal expansion. This leads to a condition known as clonal hematopoiesis of indeterminate potential (CHIP). The incidence of clonal hematopoiesis increases with age, however, the underlying mechanisms are not fully understood. As CHIP can eventually transform into severe blood cancers, it is crucial to understand and quantitatively predict its dynamics. Evidence suggests that the interplay between mutation accumulation and age-related systemic changes, such as chronic inflammation, altered growth factor levels, and changes in the bone marrow micro-environment, contributes to CHIP evolution. We propose mechanistic mathematical models that account for stem and progenitor cell self-renewal and differentiation, nonlinear feedback regulation, chronic inflammation, and changes in the stem cell niche to understand how these processes influence clonal dynamics. The models are informed by data on steady-state blood cell formation and clonal expansion after stem cell transplantation.

MS72-02 — Digital Twins in (Radiation) Oncology

SPEAKERS Heiko Enderling (MD Anderson Cancer Center)
AUTHORS Heiko Enderling (MD Anderson Cancer Center)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 15.04 - HS
TRACK Mathematical Oncology

Abstract

To personalize cancer radiation therapy, we must give the right dose and dose fractionation, at the right time, dynamically adapted, to best harness the radiobiological effects of radiation as well as synergy with the patient's immune system. I present the latest developments in the mathematical and computational modelling in radiation oncology to develop digital twins – constructs that mimic the structure and behavior of the patient and the tumor to make predictions and inform decisions that realize value. I present different simple approaches to build predictive pipelines and how to integrate those into clinical decision making towards the concept of real-time adaptive personalized radiation treatments. I will discuss past, present, and future clinical trials of such model-guided treatments.

MS75 — From dose to response: advances in modelling treatment efficacy and toxicity in tumor forecasts

TRACK Mathematical Oncology
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 62.01 - HS
ORGANISERS Guillermo Lorenzo (Universidade da Coruña, Spain); Maximilian Strobl (Imperial College & The Institute of Cancer Research, UK); Thomas E. Yankeelov (The University of Texas at Austin, US)

Minisymposium abstract

Recent efforts to improve cancer treatment focus on identifying new therapeutic targets, enhancing delivery methods, and optimizing treatment combinations and sequencing to address the pronounced spatial and temporal heterogeneity of tumors. Mathematical models of cancer progression and therapeutic response have emerged as powerful tools for personalized tumor forecasting, adaptive treatment design, and optimization of dosing schedules to maximize efficacy while limiting adverse effects. However, many current modeling frameworks emphasize tumor response, treating toxicity through simplified formulations and relying on coarse pharmacokinetic/pharmacodynamic (PK/PD) descriptions. Nonetheless, the PK/PD literature offers a rich set of mathematical models that could substantially enhance tumor forecasting by more accurately capturing drug delivery, efficacy, and toxicity. Moreover, the growing availability of longitudinal, multimodal clinical data (from omics to imaging) calls for refined modeling and data assimilation pipelines to support reliable digital twins for clinical decision-making and experimental discovery. This minisymposium brings together a selected group of researchers advancing mathematical approaches to modeling treatment effects and toxicity across cancer sites and therapeutic modalities, highlighting recent progress and key open problems for the next generation of predictive cancer models.

MS75-01 — Efficacy-toxicity and toxicity-toxicity trade-offs in head and neck cancer treated with radiotherapy

SPEAKERS Daniel Glazar (Moffitt Cancer Center, US)
AUTHORS Daniel Glazar (Moffitt Cancer Center, US); Niema B. Razavian (Moffitt Cancer Center); Renee Brady-Nicholls (Moffitt Cancer Center); Ryan Werthmann (Moffitt Cancer Center)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Radiotherapy (RT) is an effective localized therapy used to treat 75% of head and neck cancer (HNC) patients. However, delivery to surrounding normal tissues induce toxicities that exacerbate patient symptoms. Motivated by a published dataset of longitudinal patient reported outcomes (PROs) in HNC patients treated with RT, we developed a mathematical model to capture both on-target tumor response and off-target toxicities. The classical linear-quadratic model was employed to describe tumor response to RT. To model off-target toxicities, we introduce a novel concept of radiation exposure analogous to drug exposure. We then employed a Markov chain model with radiation exposure as a time-varying covariate to describe PRO dynamics. While efficacy-toxicity trade-offs were sensitive to RT dose and use concurrent chemotherapy, toxicity-toxicity trade-offs were more sensitive to RT plan (sparing vs. non-sparing). We also derived minimum efficacious dose (MED) and maximum tolerable dose (MTD) for various tumor response and toxicity endpoints. Overall, this model offers adaptable, data-informed treatment decisions by integrating both tumor control and quality of life considerations. Future iterations of the model could aid clinicians in RT dose-finding and selecting a RT plan that will optimize tumor control and patients' goals of care.

MS75-02 — Beyond Dose, What Really Drives Radiation-Induced Brain Necrosis?

SPEAKERS Ibrahim Chamseddine (Harvard Medical School, US)
AUTHORS Ibrahim Chamseddine (Harvard Medical School, US)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Brain necrosis after brain and head & neck radiotherapy presents a fundamental inference problem since by the time a lesion is visible on MRI, it has already expanded, remodeled, and erased the evidence of where and why it began. Behind this expansion lies a spatially dynamic process governed by brain architecture and patient-specific biology, which are not captured in clinical dose thresholds. Dosimetric indices showed no consistent voxel-level correlation with necrosis in our cohort, leading us to hypothesize that the dose-outcome relationship is masked by patient heterogeneity. Decomposing this heterogeneity

through an expert-augmented Bayesian network revealed that brain anatomy, particularly proximity to ventricles and white matter, governs necrosis risk more strongly than dose alone. Building on this, a 3D cellular automaton incorporating MRI-derived vascular density maps reproduced the anisotropic spatial progression of lesions with AUC 0.87-0.95. Inverting the model's rules allowed backward-in-time simulation to localize lesion initiation sites invisible to standard imaging. A multi-channel Vision Transformer model was developed to integrate 3D dose distributions with crucial nondosimetric factors to predict necrosis risk. Disentangling the biological, anatomical, and dosimetric drivers of brain necrosis aims to redefine biological effective radiation dose to personalize treatment planning.

MS75-03 — Using a mathematical model to predict and optimize response to chemo- and radiation therapy in glioma cells

SPEAKERS Reshmi J. S. Patel (The University of Texas at Austin)
AUTHORS Reshmi J. S. Patel (The University of Texas at Austin); Ernesto A. B. F. Lima (The University of Texas at Austin); David A. Hormuth (The University of Texas at Austin); Chengyue Wu (The University of Texas MD Anderson Cancer Center); Patrik Parker (The University of Texas at Austin); Casey E. Stowers (The University of Texas at Austin); Thomas E. Yankeelov (The University of Texas at Austin)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Over 75% of cancer patients receive chemo- and/or radiation therapy, but treatment schedules are not optimized for the individual patient. Optimized schedules may lead to improved outcomes. We aim to (1) collect confluence time courses for glioma cells treated over a wide range of chemo- and radiation therapy schedules, (2) calibrate a biology-based math model to the data, and (3) apply optimal control theory to the calibrated model to identify and test theoretically optimal schedules. As it is highly cost-intensive to test all schedules in vivo, we are collecting data in vitro and optimizing in silico. Using C6 glioma cells, we tested 64 unique schedules for chemotherapy (temozolomide), 72 for radiation therapy, and 24 for combination chemoradiation. Using Incucyte live-cell imaging, phase contrast and confluence data were acquired every four hours. Our math model accounts for the temporal change in tumor cell confluence as a function of logistic proliferation with treatment-induced suppression of the proliferation rate. Fitting our model to the 120 unique treatment schedules complete at time of submission yielded a mean (±SD) R2 value of 0.87 (±0.12). Early analyses identified conditions in which treating with half the dose on a different schedule or the same dose with more rest yielded similar outcomes. These initial results suggest a potential to optimize dose and timing for chemo- and/or radiation therapy. Ongoing work seeks to identify and validate the optimal regimens.

MS75-04 — Incorporating Toxicity Constraints in Adaptive Cancer Therapy

SPEAKERS Kathleen Wilkie (Toronto Metropolitan University)
AUTHORS Eduardo Sontag (Northeastern University); Harsh Jain (University of Minnesota Duluth); Irina Kareva (Northeastern University); Jana Gevertz (The College of New Jersey); Joel S. Brown (Moffitt Cancer Center); Kathleen Wilkie (Toronto Metropolitan University); Mark Davies (Cardiff University); Vladimir Vinogradov (University of Toronto Scarborough); Yitong Pepper Huang (Smith College)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Adaptive cancer therapy is a new paradigm of treatment for non-curative disease that aims to prolong emergence of resistance, and thus treatment failure. Here we use a mathematical model to explore how incorporating treatment toxicity into the protocol of adaptive therapy can be beneficial by both extending time to treatment failure and improving the quality of life for the patient [Gev+26]. Our mathematical framework implements treatment-pausing adaptive therapy with a tumour comprised of sensitive and resistant cancer cells. We show that the degree of competition between these populations critically modulates the impact of toxicity feedback, and treatment breaks. We explore circumstances

where these breaks provide benefit both at our baseline parameterization and across heterogeneous virtual populations.

References

[Gev+26] Jana L Gevertz et al. “Delaying cancer progression by integrating toxicity constraints in a model of adaptive therapy”. In: *npj Systems Biology and Applications* (2026).

MS75-05 — Controlling drug-induced phenotypic plasticity via model-informed treatment scheduling

SPEAKERS Einar Bjarki Gunnarsson (Division of Applied Mathematics, Science Institute, University of Iceland)
AUTHORS Einar Bjarki Gunnarsson (Division of Applied Mathematics, Science Institute, University of Iceland)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Anti-cancer drug treatment can induce tumor cells to adopt reversible drug-tolerant phenotypic states, setting the stage for the evolution of resistance and eventual treatment failure. This challenges conventional maximum tolerated dose (MTD) regimens by creating a trade-off between short-term tumor reduction and long-term tumor control. Here, we show how mathematical model-informed treatment scheduling can be used to balance this trade-off. Across a family of simple models of drug-induced tolerance, we characterize when and why intermittent treatment is preferable to continuous treatment and low doses are preferable to high doses. On the one hand, our results reveal distinct classes of optimal strategies depending on how the drug affects cell proliferation and phenotypic state transitions, indicating a need for patient-specific approaches. On the other hand, we identify a handful of robust schedules that outperform MTD treatment across a broad range of possible dynamics, suggesting that outcomes can be improved even when the tolerance-inducing effects of the drug are only partially understood.

MS75-06 — Agent-based modeling of intratumor heterogeneity coordination

SPEAKERS Michela Prunella (Politecnico di Bari, Italy)
AUTHORS Mariagrazia Dotoli (Politecnico di Bari); Michela Prunella (Politecnico di Bari, Italy); Nicola Altini (Politecnico di Bari); Nicola Mignoni (Politecnico di Bari); Raffaele Carli (Politecnico di Bari); Vitoantonio Bevilacqua (Politecnico di Bari)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Deciphering intratumor spatial configuration of cell communities is fundamental for mechanistically understanding how heterogeneity in tumor phenotypes impacts the effectiveness of treatments. Such dynamic interplay in the tumor microenvironment determines a continuum of transition stages, having different levels of compliance to therapy [Pru+25]. Scheduling and sequencing of two or more treatments starting from routinely available H&E-stained Whole Slide Images could hence be improved by considering also longitudinal interactions between cancerous and immune cells. Agent-based modeling is a computational modeling framework that deploys dynamic cell-to-cell inter-actions within a drug-modulated selective environment, and can provide a time-resolved quantitative approach for more informed and adaptive treatment [Kat+17] selection. The temporal dimension of the model allows not only simulating under fixed behavioral rules, but also includes the cumulative effects of drug exposure over time [Sur+23]. This enables the representation of plastic interactions between cells and therapeutic agents, whereby cellular behavior can dynamically change as a function of prior treatment history. Such a framework allows the simulation of clinically relevant phenomena frequently observed in oncology, including the emergence and selection of therapy-resistant clones.

References

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[Sur+23] Anudeep Surendran et al. “Agent-based modelling reveals the role of the tumor microenvironment on the short-term success of combination temozolomide/immune checkpoint blockade to treat glioblastoma”. In: *The Journal of Pharmacology and Experimental Therapeutics* 387.1 (2023), pp. 66–77.

MS75-07 — Mechanistic and Machine Learning Models for Dose Scheduling and Treatment Response Prediction

SPEAKERS Mohammad Kohandel (University of Waterloo, Canada)
AUTHORS Mohammad Kohandel (University of Waterloo, Canada); Shalev Manor (University of Waterloo)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 62.01 - HS
TRACK Mathematical Oncology

Abstract

The integration of mechanistic models with machine learning is becoming increasingly important for predicting treatment response and optimizing dose schedules in cancer therapy. Mechanistic models based on differential equations capture biological processes such as tumor growth and drug dynamics, while machine learning provides flexible tools for learning unknown components from data. However, two major challenges arise in this integration: identifiability of model components and the computational cost of repeated inverse problems. In this talk, I present recent work addressing both challenges. First, we develop a framework that establishes conditions for identifiability when simultaneously inferring unknown parameters and functional terms in differential equation models, improving the interpretability and reliability of hybrid mechanistic–ML approaches. Second, we introduce efficient inverse modeling methods based on physics-informed neural networks that enable rapid parameter inference through an offline-online decomposition. Together, these approaches provide practical tools for integrating mechanistic knowledge with data-driven learning to support treatment response prediction and exploration of personalized dosing strategies.

MS75-08 — Personalizing Radiotherapy in HPV+ Oropharyngeal Cancer with Systematic In Silico Trials

SPEAKERS Mohammad Zahid (The University of Texas MD Anderson Cancer Center, US)
AUTHORS Heiko Enderling (Department of Radiation Oncology, The University of Texas MD Anderson Cancer Center); Mohammad Zahid (The University of Texas MD Anderson Cancer Center, US)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 62.01 - HS
TRACK Mathematical Oncology

Abstract

RT for HPV+ oropharyngeal cancer has high cure rates, but this is often associated with significant toxicity. Despite broad interest in de-intensifying RT in this context, there isn’t a reliable biomarker to identify individual patients for safe de-escalation without sacrificing cure. We address this by creating a virtual cohort of head and neck cancer. The virtual cohort is based on two mathematical models: (1) a model of RT-response that simulates tumor volume dynamics during RT; (2) a model of tumor regrowth that simulates disease recurrence from post-treatment viable tumor burden dynamics. The virtual cohort’s RT response parameters are calibrated to weekly CT images from a cohort of 39 head and neck cancer patients that received fractionated RT. The recurrence/regrowth parameters are calibrated to 5-year locoregional recurrence data abstracted from a large cooperative group clinical trial that tested both standard and hyperfractionated RT (RTOG 9003). This outputs a calibrated virtual cohort of HNC patients that has the same recurrence patterns as its real counterparts in RTOG 9003, i.e. a digital twin of the trial. We then ran systematic in silico trials on this virtual cohort to determine patient-specific

adaptive RT schedules that maximize locoregional control rates and minimizes both total RT dose and number of RT fractions.

MS79 — Heterogeneity in epidemic modelling

TRACK Mathematical Epidemiology
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 15.03 - HS
ORGANISERS Elisa Paparelli (Sorbonne Université); Mattia Sensi (Università di Trento)

Minisymposium abstract

Understanding infectious disease dynamics increasingly requires models that can capture forms of heterogeneity which are invisible to standard population-level compartmental frameworks. Real-world data reveal substantial variation across individuals and groups (e.g., in terms of spatial distribution, age structure, viral-load trajectories, contact patterns, risk exposure, and behavioural responses) that cannot be adequately represented by homogeneous models.

This minisymposium aims to explore the role and impact of such heterogeneities on epidemic spread, and how they can be incorporated through mathematically rigorous modelling approaches. These include network-based ODE models, renewal equations, and continuously structured systems such as PDEs or integro-differential equations. We are interested both in theoretical insights arising from these models and in methodologies that allow heterogeneous structures to be inferred from, or compared to, data.

Given the diverse audience of ECMTB, ranging from theoretical mathematicians to researchers working closely with real-world datasets, our session aims at bridging rigorous theoretical insight from mathematical modelling with data-driven perspectives, fostering dialogue between experts from heterogeneous backgrounds.

MS79-02 — Estimating epidemic risk in spatially and behaviorally structured populations

SPEAKERS Eugenio Valdano (INSERM)
AUTHORS Eugenio Valdano (INSERM)
SCHEDULE Thursday, 16 July 2026; 10:40–11:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Assessing epidemic risk, both the probability that pathogen introductions lead to major outbreaks and the trajectory of ongoing circulation, often relies on population-level indicators derived from compartmental models. These frameworks typically assume homogeneous mixing within epidemiological compartments, yet real populations are structured by space, behavior, and heterogeneous exposure. Such heterogeneities complicate both the description of epidemic dynamics and the implementation of public health interventions. How should these different sources of heterogeneity be handled for mathematical models to inform public health policymaking? In this talk I discuss when heterogeneity must be explicitly represented, when it can be simplified, and when it may mislead intervention design. Different forms of heterogeneity play different roles. Variation in individual exposure risk can shape transmission patterns but does not necessarily justify targeted interventions: broad non-selective distribution of preventive measures may outperform strategies aimed at high-risk groups, as shown for HIV prevention. Even when heterogeneity is present, it may sometimes be handled through aggregation, for instance by combining spatial data with surveillance information to improve epidemic monitoring for respiratory pathogens. Together these examples highlight which forms of heterogeneity matter for estimating epidemic risk and how they can be incorporated into models used for policy.

MS79-01 — Sideward contact tracing in an epidemic model with mixing groups

SPEAKERS Dongni Zhang (Linköping University)
AUTHORS Dongni Zhang (Linköping University); Martina Favero
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

We consider a stochastic epidemic model with sideward contact tracing. Infection is assumed to occur through mixing events, that is, gatherings of two or more individuals [BN22]. When an infective is diagnosed, each person infected at the same event is traced with a given probability. Instead of tracing who infected a diagnosed person or whom they later infected, sideward tracing identifies people who were infected at the same event. This makes it especially relevant in settings such as large gatherings and potential superspreading events. Assuming a small number of initial infectives in a large population, the early phase of the epidemic can be approximated by a branching process with sibling dependencies. To address these dependencies, we group together individuals infected at the same event and treat them as macro-individuals, leading to a corresponding macro-branching process. This allows us to derive an effective reproduction number that acts as an epidemic threshold, with critical value 1. The results show that the sideward tracing becomes more effective when more infections occur within the same event. At the same time, if gatherings are very large, sideward tracing alone may not be sufficient to bring the effective reproduction number below 1.

References

[BN22] F. Ball and P. Neal. “An epidemic model with short-lived mixing groups”. In: *Journal of Mathematical Biology* 85.6 (2022), p. 63.

MS79-03 — How Within-Host Heterogeneity Shapes Population-Level Transmission: Insights From a Multiscale Model

SPEAKERS Fernando Saldaña (INRAE Nantes)
AUTHORS Fernando Saldaña (INRAE Nantes); Gaelle Gonzalez; Hélène Cecilia; Pauline Ezanno; Quirine ten Bosch
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Host heterogeneity is a key driver of epidemic dynamics, yet most epidemic models assume homogeneous hosts and represent transmission and recovery using constant rates. In many infections, however, variation in within-host pathogen dynamics generates substantial differences in infectiousness, disease progression, and infection outcomes across individuals. Understanding when such heterogeneity must be represented explicitly remains an important challenge in epidemic modeling. We investigate this question using West Nile virus (WNV), a mosquito-borne pathogen maintained in transmission cycles between mosquitoes and a diverse community of avian hosts that exhibit strong heterogeneity in viral load dynamics and infectiousness. We develop a stochastic individual-based multiscale model that explicitly represents within-host viral dynamics for multiple bird species and mechanistically links viral load to mosquito infection probability. This framework captures biologically grounded heterogeneity in infectiousness, latent and infectious periods, and disease-induced mortality. By comparing the resulting epidemic dynamics with those predicted by an equivalent single-scale model parameterized using mean epidemiological traits, we assess when average transmission parameters suffice and when explicitly representing within-host heterogeneity is necessary to capture epidemic behavior.

MS79-04 — Genuine and spurious bistability in a simple epidemic model with waning immunity

SPEAKERS Francesca Scarabel (University of Leeds)
AUTHORS Francesca Scarabel (University of Leeds); Harry Coldwell; Tyler Cassidy
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

We analyse an infection-age structured epidemic model where both infectivity and immunity loss depend on time since infection [SCC26]. The model can be formulated as a nonlinear renewal equation for the incidence or as a PDE for the infected population. Using ODE approximations and numerical bifurcation analysis, we study gamma-distributed infection durations and show that distribution shape critically affects endemic equilibrium stability, even when R_0 and the mean infectious period are fixed. We identify regions of bistability, where a stable endemic state coexists with a stable periodic orbit—providing, to our knowledge, the first example of such behaviour in models with waning immunity alone. Our analysis also shows how standard compartmental models, which impose implicit assumptions on infection duration, may yield to spurious dynamical outcomes.

References

[SCC26] F. Scarabel, H. Coldwell, and T. Cassidy. "Genuine and spurious bistability in a simple epidemic model with waning immunity". In: *Mathematical Biosciences* 396 (2026), p. 109671. ISSN: 0025-5564. DOI: https://doi.org/10.1016/j.mbs.2026.109671. URL: https://www.sciencedirect.com/science/article/pii/S0025556426000611.

MS79-05 — Network effect and behavioral feedback in epidemics

SPEAKERS Martina Alutto (KTH)
AUTHORS Fabio Fagnani; Francesca Parise; Giacomo Como; Leonardo Cianfanelli; Martina Alutto (KTH)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

In recent years, there has been renewed interest in the modeling, analysis and control of epidemics. The classic SIR model presents limitations due to its assumption of fully mixed and homogeneous population. It is essential to consider network effects to account for heterogeneity in susceptibility, infectivity and interactions. In addition, different behavioral changes of the individuals may influence the epidemic. This talk first focuses on analyzing the SIR model on a network of interacting subpopulations. These subpopulations consist of homogeneously mixed individuals with differing activity rates, disease susceptibility and infectivity. In contrast to the classic SIR model, we show that infection curves can be multimodal in the single node. With rank-1 interaction matrix, we provide sufficient conditions for this multimodality and establish an upper bound on the number of monotonicity changes in the node-level infection curve. Additionally, we characterize the system's asymptotic behavior, with explicit expressions for limit equilibrium points and conditions for their stability. The second part of the talk analyzes a deterministic SIR model in which the transmission rate depends on the system state, reflecting behavioral adaptations in response to the epidemic. Under general conditions, we prove that the infection curve is unimodal. We then solve an optimal control problem to minimize intervention costs while keeping the infection curve below a desired threshold.

MS79-06 — Modelling information-dependent protective behaviour in response to mosquito-borne epidemic outbreaks

SPEAKERS Simone De Reggi (Università di Trento)
AUTHORS Andrea Pugliese; Cinzia Soresina; Mattia Sensi; Simone De Reggi (Università di Trento)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

We present a model for a mosquito-borne epidemic outbreak in which human hosts may adopt protective behaviour against mosquito bites according to the information they receive about disease prevalence. In line with the information index approach [dM09], we assume that individuals react to this information according to a memory kernel that is continuously distributed over the past. Assuming that mosquitoes can also feed on noncompetent hosts, i.e. hosts that do not contribute to disease transmission, we revisit existing results [Dem+18; MDH16], showing that behaviour-driven protection may either decrease or increase the reproduction number of the epidemic depending on several factors. Furthermore, assuming that behavioural changes occur much faster than epidemic dynamics, it is possible to separate the timescales of the two mechanisms. By applying methods from Geometric Singular Perturbation Theory [Fen79], we derive an epidemic model with information index for a homogeneous host population. The reduced system enables a detailed investigation of the impact of information-induced behavioural feedback on the transient dynamics of the epidemic, including scenarios in which protective measures lead to outbreaks with low attack rates [And+26]. Our analysis shows that behavioural responses may facilitate epidemic control, but may also prolong disease persistence, potentially generating recurrent damped epidemic waves. Numerical simulations are provided to illustrate and support the analytical findings.

References

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MS79-07 — SEIR Models With Host Heterogeneity: Main Properties And Applications To Seasonal Influenza Dynamics

SPEAKERS Tamas Tekeli (University of Szeged)
AUTHORS Tamas Tekeli (University of Szeged)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Population heterogeneity strongly shapes epidemic dynamics, beyond age, space, or contact patterns. We focus on host susceptibility differences within the SEIR framework, using the renewal equation approach of Diekmann and Inaba. Analytical results of SEIR models with heterogeneous susceptibility show that greater susceptibility variation reduces final epidemic size.

MS82 — Social dynamics and behavioural interactions in infectious disease modeling

TRACK Mathematical Epidemiology
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 15.02 - HS

ORGANISERS Congjie Shi (Department of Mathematics and Statistics, York University, Toronto, Canada; Bolyai Institute, University of Szeged, Szeged, Hungary)

Minisymposium abstract

In a pandemic, alongside biological factors, societal interactions, cognitive behaviours, and personal attitudes can also influence the progression of an epidemic. For instance, people's compliance to vaccination or non-pharmaceutical measures rely on their social links as well as their individual opinions. How people connect with each other and their clustering structure also adds heterogeneity and complexity to disease dissemination. Thus, modeling frameworks should explicitly incorporate these aspects to enhance realism, accuracy, and predictive power. The key challenges exist in how to formulate and develop such models, rigorously validate and evaluate them, as well as feeding appropriate data to support public health strategies. Innovative modeling efforts may draw on a wide range of mathematical approaches, incorporating differential equations models to capture collective behaviours, network models to represent realistic contact patterns or structures, and agent-based models to simulate local feedback, or integrations thereof. Recently, there has been an increasing interest in combining social characteristics, cognitive choices, and epidemic spread. This mini symposium will catalyze cross-methodological conversations and provide a platform to showcase and highlight how novel approaches, techniques, and findings in this field may jointly inform robust and actionable responses for curbing infectious diseases.

MS82-01 — Non-monotonic attack rates arising from risk-aware and imitation-driven behaviours during epidemics

SPEAKERS Gergely Röst (Department of Applied and Numerical Mathematics, University of Szeged, Hungary)
AUTHORS Gergely Röst (Department of Applied and Numerical Mathematics, University of Szeged, Hungary); Seyed M Moghadas (Agent-Based Modelling Laboratory, York University, Toronto, Ontario, Canada)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Human behaviour can profoundly alter disease dynamics and the impact of public health interventions. Recognising this socio-epidemiological interplay, recent modelling efforts have increasingly incorporated behavioural responses triggered by perceived risk, disease outcomes, policy recommendations, or conformity pressure. We present a mathematical model that integrates two types of behavioural responses—reversible and irreversible— to investigate the effect of risk-based and imitation-driven mechanisms on the proportion of population infected (i.e., the attack rate). Reversible behaviours—such as adopting non-pharmaceutical measures—are represented using threshold-like functional responses that capture sharp increases in protective behaviour once disease prevalence exceeds a critical level. Irreversible behaviours—such as vaccination—are modelled through a dynamic imitation process influenced by population-level adoption. Extending previous approaches, we demonstrate that behavioural feedbacks can generate non-monotonic relationships between the attack rate and disease transmissibility for a broad range of parameters representing behavioural effectiveness or adoption strength. We observed that, for sufficiently high imitation rates, distinct intrinsic transmission rates can yield the same attack rate, even though the corresponding epidemic durations and peak prevalence levels may differ substantially.

MS82-04 — Behaviour-driven epidemic phenomena in heterogeneous populations and networks

SPEAKERS Márton Karsai (Department of Network and Data Science, Central European University, Vienna, Austria; HUN-REN Rényi Institute of Mathematics, Budapest, Hungary)
AUTHORS Márton Karsai (Department of Network and Data Science, Central European University, Vienna, Austria; HUN-REN Rényi Institute of Mathematics, Budapest, Hungary)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

While realistic approaches have become increasingly important in epidemic modelling, behavioral factors and individual differences have historically been overlooked due to the lack of high-resolution data and appropriate mathematical methods. This gap became particularly evident during the recent pandemic, highlighting the need for large-scale data collection on individual-level epidemic-related behaviors across representative populations. These advancements have revealed several new and interesting spreading phenomena that challenge our previous understandings. In this talk, we will focus on two examples of such new insights, derived from data-driven and behavior-informed epidemic models. We will explore input-output inequalities in spreading dynamics [MKK24; Man+24], and the paradoxical effects of awareness-driven adaptive behavior on epidemic outcomes [Kol+25]. These findings highlight the role of behavioral factors, offering a more accurate understanding and modelling of real-world epidemic scenarios.

References

[Kol+25] Cs. Kolok et al. “Epidemic paradox induced by awareness driven network dynamics”. In: *Physical Review Research* 7.1 (2025), p. L012061.

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MS82-07 — Wait or Vaccinate? Behavioural Dynamics of Competing Vaccine Strategies in a Compartmental Epidemic Model with Prevalence-Dependent Hesitancy

SPEAKERS Attila Denes (Bolyai Institute, University of Szeged, Szeged, Hungary)

AUTHORS Attila Denes (Bolyai Institute, University of Szeged, Szeged, Hungary); Sarkhel Akbar Mahmood (Bolyai Institute, University of Szeged, Szeged, Hungary)

SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.02 - HS

TRACK Mathematical Epidemiology

Abstract

As emerging pathogens frequently share biological similarities with previously circulating diseases, pre-existing vaccines may provide valuable yet imperfect protection during the early stages of an outbreak, before disease-specific vaccines become available.

In this work, we present a compartmental model that integrates two types of vaccines: a partially protective one, and a more efficient onw which only becomes available after a given time corresponding to vaccine development. Our model includes a hesitant population consisting of individuals who lack confidence in the newly developed vaccine. Both hesitancy and vaccine uptake are included as a function of disease prevalence.

In the case of constant coefficients, we show that the disease-free equilibrium is globally asymptotically stable when $R_0 < 1$, while the disease persists when $R_0 > 1$. A transcritical forward bifurcation is shown to occur at $R_0 = 1$.

Numerical simulations are provided for scenario analysis considering various situations differing in severity of the new disease, efficacy of the old vaccine and the time needed for development of the new vaccine.

Critically, delayed introduction of a superior vaccine even with a high efficacy allows uncontrolled viral proliferation during the waiting period, producing a markedly higher infection peak and greater cumulative mortality compared to immediate deployment of a moderately effective vaccine. Elevated hesitancy amplifies transmission and delays epidemic control, whereas declining hesitancy enhances vaccination campaign effectiveness.

MS82-08 — A network model from sociodemographic heterogeneity to individual infection risk

SPEAKERS Congjie Shi (Laboratory for Industrial and Applied Mathematics, York University, Toronto, ON, Canada; Bolyai Institute, University of Szeged, 6720 Szeged, Hungary)

AUTHORS Congjie Shi (Laboratory for Industrial and Applied Mathematics, York University, Toronto, ON, Canada; Bolyai Institute, University of Szeged, 6720 Szeged, Hungary); Csegő Balázs Kolok (HUN-REN Alfréd Rényi Institute of Mathematics); Gergely Röst (University of Szeged); Jianhong Wu (York University); Júlia Koltai (ELTE Centre for Social Sciences); Márton Karsai (Central European University)

SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.02 - HS

TRACK Mathematical Epidemiology

Abstract

Social interactions and disease transmission are tightly intertwined, with behavioural responses and risk perception evolving during an epidemic. Capturing these feedbacks remains a major challenge in epidemic modeling. In social networks, the “ego” denotes the focal individual, while “alters” are individuals directly connected to the ego. We propose a type-configuration algorithm based on node attributes, including demographic and contact information, to assess individual infection risk in a network. Individual infection risk is defined as a function of epidemiological parameters – relative susceptibility, infectiousness, contact frequency, and baseline disease transmission rate, with values depending on social factors such as education level, residence area, gender, and age. Model inputs are derived from sociodemographic data from a representative Hungarian population sample and contact surveys. Our framework captures the heterogeneous impact of sociodemographic variables on individual risk during epidemics. This data-driven approach reveals how social characteristics and contact patterns shape the local vulnerability landscape, providing insights on both individual- and population-level risk. Ultimately, individuals can use this framework as a personalized risk predictor by providing sociodemographic and contact information, allowing the algorithm to assign a type-configuration and compute an interpretable estimate of infection risk under a given epidemiological scenario.

MS82-02 — Leveraging machine learning algorithms to model health behavior in network-based epidemic models

SPEAKERS Joshua Chevalier (UMC Utrecht)

AUTHORS André Calero Valdez (University of Lübeck); Beate Jahn (UMIT TIROL-University for Health Sciences and Health Technology); Florian van Daalen (Maastricht University); Joshua Chevalier (UMC Utrecht); Leonard Stellbrink (University of Lübeck); Lilian Kojan (University of Lübeck); Lisanne Steijvers (GGD Zuid Limberg); Mickaël Hiligsmann (Maastricht University); Mirjam Kretzschmar (UMC Utrecht); Nannan Li (Maastricht University); Nicole Dukers-Muijrers (GGD Zuid Limberg); Rik Crutzen (Maastricht University); Senne Wijnen (Maastricht University); Uwe Siebert (UMIT TIROL-University for Health Sciences and Health Technology)

SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 15.02 - HS

TRACK Mathematical Epidemiology

Abstract

Individuals adapt to epidemics, creating a feedback loop between disease spread and human behavior. Incorporating this dynamic into infectious disease models is critical for evaluating interventions against future pandemic threats. We embedded an XGBoost algorithm, trained on real-world survey data (Imperial College London YouGov COVID-19 Behavior Tracker; Netherlands, Jun 2020–Jan 2021; N=5,611), directly into an agent-based model to predict contact restricting behavior. Agent attributes (age, gender, perceived severity, willingness to isolate, working outside the home) combined with population-level hospitalizations drove behavioral predictions, with adopted behavior reducing agent contacts (edge-masking; 47% reduction). The model was parameterized to wildtype COVID-19 (R0=3.28) and run on three synthetic networks (small world, random, preferential attachment; 10,000 nodes; 100 simulations). Additional mechanisms captured local epidemic awareness spread, behavioral fatigue, and prevalence-dependent re-adoption. Behavior was least effective at slowing epidemic spread on preferential attachment and random networks (median durations: 87 and 145 days; final sizes: 65–70%) versus baseline (87–92%), while the small world network showed substantial curve flattening (403 days; final size: 71%). Integrating machine learning

algorithms trained on real data into agent-based models offers a principled framework for modeling behavioral feedback and evaluating pandemic interventions.

MS82-05 — Trending on the use of Google mobility data in COVID-19 mathematical models

SPEAKERS Daihai He (Department of Applied Mathematics, The Hong Kong Polytechnic University)
AUTHORS Daihai He (Department of Applied Mathematics, The Hong Kong Polytechnic University)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Google mobility data has been widely used in COVID-19 mathematical modeling to understand disease transmission dynamics. This review examines the extensive literature on the use of Google mobility data in COVID-19 mathematical modeling. We mainly focus on over a dozen influential studies using Google mobility data in COVID-19 mathematical modeling, including compartmental and metapopulation models. Google mobility data provides valuable insights into mobility changes and interventions. However, challenges persist in fully elucidating transmission dynamics over time, modeling longer time series and accounting for individual-level correlations in mobility patterns, urging the incorporation of diverse datasets for modeling in the post-COVID-19 landscape.

MS82-06 — A Filippov Model Describing the Effect of Social Distancing in Controlling Infectious Diseases

SPEAKERS Stacey Smith? (Department of Mathematics and Faculty of Medicine, The Univeristy of Ottawa, Ottawa, ON K1N 6N5, Canada)
AUTHORS Stacey Smith? (Department of Mathematics and Faculty of Medicine, The Univeristy of Ottawa, Ottawa, ON K1N 6N5, Canada); Aili Wang (Xi'an University of Technology, China); Yinjiao Gong (Baoji University of Arts and Sciences, China); Duo Bai (Xi'an University of Technology, China); Weike Zhou (Northwest University, China)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Social distancing is now a familiar strategy for managing disease outbreaks, but it is important to understand the interaction between disease dynamics and social behaviour. We distinguished the fully susceptible from social-distancing susceptibles and proposed a Filippov epidemic model to study the effect of social distancing on the spread and control of infectious diseases. The threshold policy is defined as follows: once the number of infected individuals exceeds the threshold value, social-distancing susceptibles take more stringent social-distancing practices, resulting in a decreasing infection rate. The target model exhibits novel dynamics: in addition to the coexistence of two attractors, it also demonstrates the coexistence of three attractors. In particular, bistability of the regular endemic equilibrium and the disease-free equilibrium occurs for the system; multistability of the regular endemic equilibrium, pseudo-equilibrium and the disease-free equilibrium occurs for the system. Discontinuity-induced bifurcations, including boundary node, focus and saddle-node bifurcations, occur for the proposed model, which reveals that a small change in threshold values would significantly affect the outcome. Our findings indicate that for a proper threshold value, the infections can be ruled out or contained at the previously given level if the initial infection is relatively small.

MS82-03 — The influence of human interactions on waterborne diseases: a reaction-diffusion model

SPEAKERS Maria Francesca Carfora (Istituto per le Applicazioni del Calcolo, Consiglio Nazionale delle Ricerche, 80131 Napoli, Italy)
AUTHORS Isabella Torcicollo (Istituto per le Applicazioni del Calcolo, Consiglio Nazionale delle Ricerche, 80131 Napoli, Italy); Maria Francesca Carfora (Istituto per le Applicazioni del Calcolo, Consiglio Nazionale delle Ricerche, 80131 Napoli, Italy)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Waterborne diseases continue to pose a major global public health concern, particularly in areas lacking adequate water infrastructure. During outbreaks, changes in human behavior often play a crucial—sometimes dominant—role in shaping disease transmission. We introduce a reaction–diffusion model that accounts for varying patterns of human mobility and behavioral responses within a spatially heterogeneous environment to better understand the disease dynamics and control strategies. The model incorporates both direct and indirect transmission pathways, and assumes a cubic growth for bacterial intrinsic dynamics to highlight more interesting and complicated dynamics. The existence and uniqueness of biologically meaningful equilibria is investigated, along with their stability analysis. The effects of diffusion on the stability of the equilibria are also considered. Numerical simulations are used to explore how environmental heterogeneity, mobility, and behavioral adaptation influence the final epidemic size and, consequently, the spread of the disease.

MS91 — Data Science × Mathematical Modeling for Transforming Quantitative Life and Medical Sciences

TRACK Cutting Edge Research Areas
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 01.15 - HS
ORGANISERS Sungrim Seirin-Lee (Kyoto University); Jae Kyoung Kim (KAIST); Kevin Flores (North Carolina State University)

Minisymposium abstract

Quantitative experimental methods in the life sciences have advanced rapidly in recent years. The emergence of multi-omics, organoid systems, and in vivo live imaging has placed data at the center of modern biological discovery, driving the rapid evolution of data science for extracting biologically meaningful insights.

In parallel, mathematical modeling in the life sciences is shifting from primarily qualitative approaches toward data-driven and data-integrated paradigms. A key challenge is how to effectively fuse structure-based, mechanistic models with high-dimensional and often unstructured biological data, which is essential for achieving a deeper, system-level understanding of complex biology and medicine.

Importantly, this problem cannot be solved by conventional modeling frameworks alone. Instead, it demands a genuine and seamless integration of two mathematical sciences: mechanistic modeling grounded in biological principles, and data-centric methodologies that leverage modern statistical and computational techniques. Their full convergence is essential for next-generation discovery in the life sciences.

This mini-symposium aims to share and discuss cutting-edge research that tackles these challenges, highlighting novel frameworks, methodologies, and applications at the interface of mathematical modeling, data science, and quantitative life and medical science.

MS91-01 — How choices in quantifying data affect parameter identifiability in agent-based models of pattern formation

SPEAKERS Yue Liu (Purdue University)
AUTHORS Yue Liu (Purdue University)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Pattern formation arising from the collective behaviour of autonomous agents occurs across many areas of biology, including skin patterns. Agent-based models provide a natural framework for describing such systems. However, the high-dimensional nature of the data and model stochasticity pose significant challenges for parameter inference and identifiability analysis. To help address this challenge, researchers often rely on lower-dimensional summaries of model output, such as cell number or stripe width. However, it remains unclear which quantitative summaries are most informative for inference. In this work, we

compare topological signatures derived from Vietoris–Rips and sweeping-plane filtrations with classical statistical summaries, such as pair correlation functions. We evaluate the effectiveness of these different quantitative approaches within a Bayesian pipeline for parameter inference, and show how the choice of method for summarizing pattern data impacts parameter identifiability.

MS91-02 — Cancer systems immunology reveals the tumor-immune dynamics that determine responses to novel combination therapies in metastatic tumor microenvironments

SPEAKERS Adam Maclean (University of Southern California)
AUTHORS Adam Maclean (University of Southern California)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Tumors grow and metastasize through intricate interactions among the diverse signals and cell types composing the tumor microenvironment (TME). Cancer systems immunology combines mathematical modeling with analysis of high-dimensional multi-modal data and machine learning to gain insight into the complex ecosystems created by the immune system in and around a tumor. Immunotherapies such as checkpoint inhibition can provide durable responses in a subset of metastatic breast cancer patients, yet the mechanisms governing intrinsic resistance and response remain poorly understood. Here, via cancer systems immunology methods we integrate single-cell genomics and spatial transcriptomics with mathematical modeling to dissect metastatic breast cancer TMEs. We developed methods to identify the cell circuits mediating responses with treatment in specific TMEs. In the mouse lung TME, using cell circuits we discovered that treatment with entinostat (a histone deacetylase inhibitor) modulates chemokine and adhesive signaling pathways. We revealed that the benefits of this treatment with checkpoint inhibitors are mediated by activating B cells and decreasing immunosuppressive myeloid cell—T cell interactions. We validated these predictions using mathematical modeling and analysis of clinical samples from a phase 1b trial. We further developed mathematical models of the tumor-immune dynamics of tumors at different sites of metastasis and fit them to RECIST clinical response data via Bayesian parameter inference. In doing so, we derived mechanistic explanations for differential patient outcomes and generated testable predictions for therapeutic interventions. Together, this work offers a generalizable framework for decomposing complex TMEs, inferring multiscale interaction networks, and modeling their dynamics to guide therapeutic strategy.

MS91-03 — Theory and Algorithms for Constructing AI-Based Dynamic Virtual Cells

SPEAKERS Peijie Zhou (Peking University)
AUTHORS Peijie Zhou (Peking University)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Artificial Intelligence Virtual Cell (AIVC) is increasingly emerging as a frontier in the interdisciplinary integration of biology and artificial intelligence. Its core vision is to construct digital twins capable of simulating and predicting the dynamic evolution of cellular states, thereby providing computational support for experimental design and mechanistic analysis. We have explored a unified modeling framework that integrates generative artificial intelligence methods and dynamical systems theory. By combining mathematical theories such as Optimal Transport, Schrödinger Bridge, and differential geometry with generative AI techniques like Flow Matching and diffusion models, this framework effectively infers continuous dynamic processes of complex state transitions—such as cell proliferation, apoptosis, differentiation, migration, and interactions—from static, heterogeneous single-cell omics temporal snapshots. Compared to black-box methods, this approach not only exhibits strong generative and generalization capabilities but also offers improved mechanistic interpretability. It enables the generation of cell state data across

temporal scales and spatial structures, providing a promising direction for constructing dynamic virtual cell models with interpretability, predictive power, and the ability to integrate biological priors.

MS91-04 — Deep State Space Modeling for Real-World Biomedical Time-Series Analysis

SPEAKERS Ryosuke Kojima (RIKEN/Kyoto University)
AUTHORS Ryosuke Kojima (RIKEN/Kyoto University)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Real-world biomedical time-series data, including clinical records and measurements of complex biological phenomena, contain valuable insights into disease progression and underlying mechanisms. However, such data are often noisy, irregularly sampled, and partially observed, posing significant challenges for analysis. Extracting latent temporal structures from these observations is therefore crucial for advancing our understanding of dynamic biological systems. In this talk, we introduce a framework based on deep state space models (DSSMs) with variational inference to capture complex temporal dependencies in such data. The proposed approach infers latent trajectories corresponding to unobserved disease states or physiological processes, enabling interpretable analysis of system dynamics. As a real-world example, we demonstrate the application of DSSMs to electronic health record data, highlighting their ability to uncover meaningful patient representations. Furthermore, we present recent advances in constrained state space modeling, where prior knowledge—such as stability and physiological consistency—is incorporated into latent dynamics through structured constraints. These techniques improve identifiability, robustness to noise, and consistency with known biological principles. Overall, this framework provides a powerful and principled approach for analyzing real-world biomedical time-series data.

MS91-05 — Data and Modelling for Medical Inverse Problems

SPEAKERS Adam Stinchcombe (University of toronto)
AUTHORS Adam Stinchcombe (University of toronto)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Electrical impedance tomography (EIT) is a medical imaging technique that uses electric currents and potential measurements on the surface of the body to infer the electrical conductivity within the body. To improve the reconstructed image, our models of biological tissues incorporate anisotropic conductivity, the electrophysiology of electrically active tissues, and the physics of ionic solutions. These partial differential equation models are solved numerically using boundary integral equation methods as well as biologically-informed neural networks. We take two parallel approaches to this medical inverse problem: numerical optimization and deep learning. Our approaches are validated against a large experimental dataset collected from our own EIT device. A second medical inverse problem is cardiography. The cells of the heart are electrically active and synchronized, which make measurement of the electrical activity of the heart readily observable in electrocardiograms (EKG). We model the electrical activity of the heart using the mono-domain model with detailed ionic current models and patient-specific heart geometry. We employ our simulation and vast collection of EKG recordings to pursue a variety of applications: a) visualize the shape of the heart as an indication of congestive heart failure; b) visualize the electrical conduction pathway within the heart to understand conduction disorders; and c) study circadian rhythms in the electrical activity of the heart.

MS91-06 — Multi-Omics-Driven Mathematical Framework for Integrating A Mechanistic Model and Data in Precision Medicine

SPEAKERS Sungrim Seirin-Lee (Kyoto University)
AUTHORS Sungrim Seirin-Lee (Kyoto University)

SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Complex disease mechanisms in medical research are often inferred from limited patient samples, posing fundamental challenges in capturing inter-individual variability and selecting optimal therapeutic strategies. To address this limitation, I propose a new methodological framework, termed a multi-omics methodological framework, that systematically integrates mechanistic mathematical models with spatial transcriptomics data from patient biopsy samples, clinical trial outcomes, and virtual patients. Through the iterative integration of in silico simulations and data analysis based on the pathophysiology of virtual patients, this approach enables systematic analysis of disease mechanisms, therapeutic effects, and biomarker identification. As an application, I focus on inflammatory skin diseases to demonstrate how this framework uncovers latent structures underlying disease heterogeneity, particularly in terms of immune regulatory dynamics, and links them to variability in therapeutic responses.

MS91-07 — Towards Reliable Single-Cell Foundation Models: A Fully Data-Driven Framework for Signal Detection and Clustering via Mathematical Theories

SPEAKERS JaeKyoung Kim (KAIST)
AUTHORS JaeKyoung Kim (KAIST)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

The reliability of single-cell RNA sequencing (scRNA-seq) analysis is often hindered by subjective parameter tuning and stochastic inconsistencies, which pose significant challenges for the reproducibility of large-scale studies. To overcome these limitations and establish a rigorous foundation for single-cell foundation models, we propose a fully data-driven analysis framework based on mathematical and physical theories. Centrally, we introduce scLENS, which utilizes Random Matrix Theory (RMT) to distinguish biological signals from random noise. By employing RMT-based noise filtering and a signal robustness test, scLENS enables the objective determination of signal dimensions without manual intervention, ensuring high-fidelity feature extraction even in sparse datasets. Complementing this, scICE evaluates clustering consistency through the Inconsistency Coefficient (IC), providing a scalable and efficient way to identify stable cellular identities across tens of thousands of cells. By replacing subjective heuristics with robust, theory-based signal detection and evaluation, this integrated approach provides the essential high-quality, standardized data processing required to train and deploy reliable, large-scale single-cell foundation models in the era of artificial intelligence.

MS91-08 — Equation Learning for Agent-Based Infectious Disease Models

SPEAKERS Kevin Flores (North Carolina State University)
AUTHORS Kevin Flores (North Carolina State University)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 01.15 - HS
TRACK Cutting Edge Research Areas

Abstract

Agent-based models (ABMs) capture heterogeneous contacts, stochastic transmission, and complex interventions in infectious disease dynamics but are computationally expensive, limiting parameter inference and policy analysis. We develop an equation-learning framework that derives interpretable ordinary differential equation (ODE) surrogates directly from stochastic ABM simulations. Using the COVID-19 ABM Covasim, we construct a 12-compartment ODE system incorporating testing and contact tracing while treating key epidemiological rates as state-dependent functions rather than constants. These functions are learned from ABM-generated trajectories using Biologically Informed Neural Networks (BINNs), which denoise state dynamics while enforcing epidemiological constraints. Sparse regression

is then applied to obtain symbolic representations of the learned parameter functions to enable model interpretability. To quantify uncertainty arising from ABM stochasticity, we integrate Approximate Bayesian Computation to infer posterior distributions over model coefficients. The resulting ODE surrogates accurately reproduce ABM dynamics under constant and time-varying interventions and provide a fast, interpretable framework for analyzing complex epidemic models.

MS99 — Differential modelling and numerics for human diseases

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 15.05 - HS
ORGANISERS Gabriella Bretti (CNR - IAC); Micol Pennacchio (CNR - IMATI); Silvia Bertoluzza (CNR - IMATI)

Minisymposium abstract

Mathematical models are a key tool for describing physiological processes across multiple scales (organs, tissues, cells, molecules). Within the framework of precision medicine, differential models and their numerical (i.e. in silico) realizations can tackle complex biological phenomena with the goal of improving the treatment of human diseases. As a core component of digital twins, in silico models can indeed provide valuable support to biologists and clinicians in tasks such as designing biological experiments, predicting disease progression, and selecting drugs for novel therapies. In this framework, advances in data analytics and cutting-edge HPC technologies, leading to ever increasing availability of large datasets and computational resources, can be leveraged to enhance and improve such models by integrating real data within a multiphysics approach where an increasingly important role is also played by artificial intelligence.

Here, we discuss current challenges and future perspectives involving different mathematical techniques applied to human diseases.

MS99-01 — Global sensitivity-driven simplification and calibration of Cancer-on-Chip nonlocal integro-differential model

SPEAKERS Annachiara Colombi (Politecnico di Torino)
AUTHORS Annachiara Colombi (Politecnico di Torino); Elio Campanile (University of Trento); Gabriella Bretti (CNR - IAC)
SCHEDULE Thursday, 16 July 2026; 10:40–11:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cancer-on-Chip experiments reproduce complex biological environments to study the immune response to cancer and test the effect of therapies. Following a digital-twin approach, mathematical models reproducing Cancer-on-Chips dynamics have the potential to be able to produce in-silico different scenarios and to be largely economically convenient. However, a critical aspect in the employment of mathematical model outcomes in biomedical research is the necessity of a careful calibration of the model parameters, to ensure reliable outcomes. In this context, we focus on a nonlocal integro-differential model for Cancer-on-Chip experiments where chemotherapy-treated tumour cells release chemical signals activating the immune response. Model reliability is assessed through a Global Sensitivity Snalysis to capture parameter influence and nonlinear effects. The impact of 13 parameters is evaluated over a region of the parameter space using 11 outputs describing immune cell spatial distribution and temporal dynamics. To address computational costs, a two-step approach is adopted: the Morris method first ranks parameter importance, identifying six key parameters affecting all outputs; the extended Fourier Amplitude Sensitivity Test (eFAST) then quantifies their contributions. Results highlight the feasibility of the parameter space and identify parameters related to the chemical field and cell–substrate adhesion as dominant. These findings suggest model simplifications—such as neglecting cell–cell alignment in the absence of experimental evidence—and emphasize the need for additional data to reduce uncertainty in the most influential parameters and improve predictive accuracy.

MS99-07 — 1D/2D Coupling of HDG discretizations for the simulation of Cancer-on-chip devices

SPEAKERS Silvia Bertoluzza
AUTHORS Gabriella Bretti (CNR - IAC); Micol Pennacchio (CNR - IMATI); Silvia Bertoluzza
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

We present a framework for the simulation of Cancer-on-chip devices where independent software codes handling respectively the 2d chambers and the 1d channels are coupled by the approach proposed in [BB26]. The 2D and 1D codes, that the coupling mechanism treats as black boxes and that can be therefore be individually replaced by the user's preferred code, are presently both based on Hybridized Discontinuous Galerkin (HDG) discretizations [Ber+23]. For the 2D chambers, an "in house" matlab code is used, while the 1D channels are handled using BioNetFlux, a python library for the simulation of biological fluxes in complex one-dimensional networks, that we developed in the framework of the "Digital Driven Diagnostics, prognostics and therapeutics for sustainable Heath care (D34Health)".

This work is realized with the support of the Italian Ministry of Research, under the complementary action NRRP "D34Health - Digital Driven Diagnostics, prognostics and therapeutics for sustainable Health care" (Grant #PNC0000001).

References

[BB26] Silvia Bertoluzza and Erik Burman. *An abstract framework for heterogeneous coupling: stability, approximation and preconditioning*. 2026. arXiv: 2312.11733 [math.NA]. URL: https://arxiv.org/abs/2312.11733.

[Ber+23] Silvia Bertoluzza et al. "A HDG Method for Elliptic Problems with Integral Boundary Condition: Theory and Applications: S. Bertoluzza et al." In: *Journal of Scientific Computing* 95.1 (2023), p. 6.

MS99-05 — UQ analysis for cancer-on-chip experiments

SPEAKERS Laura Rinaldi (CNR - IMATI)
AUTHORS Gabriella Bretti (CNR - IAC); Laura Rinaldi (CNR - IMATI); Lorenzo Tamellini (CNR - IMATI); Pietro Zanotti (Università di Milano); Silvia Bertoluzza; Vittoria Bianchi
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In this talk, we will present our work to predict the dynamics of cancer-on-chip experiments using data-informed differential models [Ber+26].

We will consider a complex one-dimensional network along which tumor and immune cells evolve in response to chemotactic signals. This model is managed by coupled partial differential equations and solved by a Hybridized Discontinuous Galerkin method [Ber26].

Synthetic data will be used to tune the parameters of the governing equations employing Bayesian inversion techniques. After this, we will assess the uncertainty on the predictions of the dynamics due to the uncertainty remaining on the parameters after the tuning procedure (i.e., their posterior distribution).

Finally, we will focus on surrogate models to make the analysis computationally affordable. Especially, we will exploit surrogates to approximate the mapping of the uncertain parameters to the quantities of interest of the system (e.g. the concentration of immune and tumor cells or the total amount of chemoattractant).

References

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MS99-03 — A Thermodynamically Consistent Multiphysics Model of Irreversible Electroporation for Cutaneous Melanoma: Coupling GENERIC Thermo-Poromechanics with Tissue Electroporation

SPEAKERS Davide Baroli
AUTHORS Argyrios Petras (RICAM); Davide Baroli; Luca Gerardo-Giorda (RICAM)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Irreversible electroporation (IRE) is a promising non-thermal ablation technique for cutaneous melanoma, where high-voltage, short-duration electric pulses induce permanent membrane permeabilisation and cell death. We present a coupled multiphysics framework integrating a thermo-poromechanical model of skin with a nonlinear electroporation model. The tissue response is described within the GENERIC (General Equation for Non-Equilibrium Reversible-Irreversible Coupling) framework, providing a thermodynamically consistent formulation. The skin is modelled as a uid-saturated porous medium governed by energy and entropy functionals through Hamiltonian and Onsager operators satisfying a noninteraction condition ensuring energy conservation and entropy production. The electroporation component uses a model where tissue conductivity increases nonlinearly with the local electric eld via a sigmoid law. The coupling arises naturally: Joule heating enters the poromechanical thermal balance, while the heterogeneous temperature eld inuences conductivity and the electric eld distribution, yielding spatially varying temperature proles critical for delineating the ablation zone. In this preliminary study, we consider an idealised geometry with a skin slab and planar needle electrodes, solved via the nite element method. Results show that neglecting the poromechanical response leads to errors in the predicted ablation volume. Future work will incorporate realistic morphologies from photonics-based imaging.

References

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MS99-04 — A Data-Informed Agent-Based Model of Tuberculosis Granuloma-Like Structures

SPEAKERS Davide Moretti (CNR - IAC)
AUTHORS Alessandro Stamilla (Università di Padova); Aurora D’Alessio (Università Roma Tre); Davide Moretti (CNR - IAC); Enrico Mastrostefano (CNR - IAC); Greta Segafreddo (Università di Pavia)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Tuberculosis (TB) remains a major global health challenge, with nearly 10 million new cases annually and increasing drug resistance.

As part of the ERA4TB initiative, which aims to advance new treatment regimens, multiple research institutes collaborated to develop and characterise in vitro granuloma-like structures (GLSs), aggregates of human PBMCs that exhibit key features of tuberculosis granulomas [Pui+04].

While physiologically more relevant than standard cell line models, GLS experiments remain costly, and studying granuloma biology in vivo is challenging. Host-directed therapies, which target the host immune response rather than the pathogen directly, remain relatively underexplored and represent an area where GLS could provide valuable information.

Computational modelling can complement GLS experiments by reducing cost and providing mechanistic insight [Mic+24]. We developed an agent-based model (ABM) of GLS dynamics using PhysiCell, a scalable and extensible platform for simulating multicellular systems [Gha+18].

The model was calibrated using a multi-laboratory dataset comprising CFU time series obtained from drug-free and drug-treated conditions, together with quantitative measures of GLS size and number, the latter extracted from microscopy images, both via expert annotation and automated machine learning analysis.

Given the inherent biological variability, this parameter estimation was performed using Approximate Bayesian Computation (ABC), yielding a posterior distribution for a subset of parameters [Tav+97].

References

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MS99-02 — An adaptive high-order polytopal method for modeling neuronal electrophysiology

SPEAKERS Caterina B. Leimer Saglio (Politecnico di Milano)
AUTHORS Caterina B. Leimer Saglio (Politecnico di Milano); Paola F. Antonietti (Politecnico di Milano); Stefano Pagani (Politecnico di Milano)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Traveling wave phenomena are central in many biological processes, including electrical activity in neural and cardiac tissues. Their simulation is challenging due to sharp, fast-moving wavefronts that demand high spatial and temporal resolution, resulting in high computational costs. Brain electrophysiology at the tissue level is a key example: the transmembrane potential exhibits steep wavefronts propagating along preferential axonal directions. Modeling these dynamics involves multiple scales and complex, anisotropic domains. We employ a high-order discontinuous Galerkin method on polygonal meshes (PolyDG) that remains computationally demanding. To address this, we propose a p-adaptive algorithm that exploits

the localized nature of traveling waves: strong variations are confined to small regions, while the rest of the domain is nearly stationary. We design a posteriori error indicators to detect wavefronts and locally adjust the polynomial degree. Numerical results show that the method accurately captures wave dynamics and effectively simulates epileptic events in heterogeneous brain tissues, reducing degrees of freedom and computational cost while preserving high-order accuracy.

References

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MS99-06 — HDG-Based Discretization of a Mathematical Model for Multiple Sclerosis

SPEAKERS Micol Pennacchio (CNR - IMATI)
AUTHORS Christophe Prud'homme (Université de Strasbourg); Gabriella Bretti (CNR - IAC); Micol Pennacchio (CNR - IMATI); Silvia Bertoluzza
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Multiple sclerosis is a complex neurodegenerative disorder whose progression can be described through nonlinear mathematical models accounting for both inflammatory and degenerative processes. In this work, we investigate the application of the Hybridized Discontinuous Galerkin (HDG) method to the numerical approximation of such models. The HDG framework retains the flexibility of discontinuous Galerkin schemes while significantly reducing the number of globally coupled degrees of freedom, thereby enhancing computational efficiency without sacrificing accuracy. We consider the model introduced in [Des+22], derive the corresponding HDG discretization, and provide a rigorous convergence analysis of the proposed method. The theoretical findings are validated by numerical experiments confirming the predicted convergence behavior. Overall, the results highlight the suitability of HDG methods for the simulation of multiple sclerosis dynamics and support their use as reliable tools for the numerical approximation of complex biomedical models. This work is realized with the support of the Italian Ministry of Research, under the complementary action NRRP "D34Health - Digital Driven Diagnostics, prognostics and therapeutics for sustainable Health care" (Grant #PNC0000001).

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MS105 — Applications of reaction networks

TRACK Systems Biology and Biochemical Networks
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 11.03 - HS
ORGANISERS Bryan Hernandez (University of the Philippines Diliman); Noel Fortun (De La Salle University, Manila, Philippines)

Minisymposium abstract

Reaction networks provide a framework for representing the structural form of dynamical systems. While traditionally used in systems biology and biochemistry to describe molecular interactions, the mathematical principles of reaction networks are increasingly applied across various fields such as cell biology, ecology, epidemiology, and environmental science. By viewing entities—be they molecules, organisms, or system components—as species within a formal interaction network, one can apply rigorous kinetic and stochastic methods to identify system properties. Through a range of applications, this minisymposium demonstrates how the formal language of species and its interactions provides a unifying approach for understanding the stability and behavior of complex systems.

MS105-01 — CRNT approaches in modelling carbon dioxide removal through direct ocean capture

SPEAKERS Al Jay Lan Alamin (Institute of Mathematics, University of the Philippines Diliman, 1101, Quezon City, Philippines)

AUTHORS Al Jay Lan Alamin (Institute of Mathematics, University of the Philippines Diliman, 1101, Quezon City, Philippines); Bryan Hernandez (Institute of Mathematics, University of the Philippines Diliman, 1101, Quezon City, Philippines); Eduardo Mendoza (Department of Mathematics and Statistics, De La Salle University, Taft Avenue, Manila 0922, Philippines / Center for Natural Sciences and Environmental Research, De La Salle University, Taft Avenue, Manila 0922, Philippines / Max Planck Institute of Biochemistry, Martinsried near Munich, Germany); Melquezedec James Cruz (Institute of Mathematics, University of the Philippines Diliman, 1101, Quezon City, Philippines)

SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 11.03 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

The mitigation of climate change has stood at the forefront of interest in the interface of mathematics, biology, and chemistry. Recently, direct carbon dioxide removal strategies have been examined which include direct ocean capture, as well as direct air capture. However, the structural and dynamical properties of these expanded carbon cycle models have yet to be explored. We apply methods in chemical reaction network theory (CRNT) to analyze the kinetic properties of these systems. Beyond identifying steady states, we also look at possibilities for multistationarity signaling crucial tipping points in the carbon cycle. We examine the conditions for independence of steady states to starting values and the capability for carbon concentration reduction. Finally, through the methods of flux balance analysis, we present a linear programming technique to show the capability of the carbon cycle to reduce overall free carbon dioxide concentration in both atmosphere and oceans. Our results do not only propose mechanisms to mitigate climate change, but give a consistent approach to analyze other carbon dioxide removal techniques in a general carbon cycle system.

MS105-02 — Spatiotemporal modeling of signaling pathways: impact of endosomal compartmentalization and application to gonadotropin receptors

SPEAKERS Chloe Weckel (BIOS, PRC, UMR CNRS, Université de Tours, INRAE, INRAE Centre Val de Loire 37380 Nouzilly, France / MUSCA, Université Paris-Saclay, Inria, Centre Inria de Saclay, 91120 Palaiseau, France)

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SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 11.03 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

Cells communicate by sending extracellular ligands such as hormones. Once recognized by their plasma membrane receptors, these ligands trigger intracellular signaling cascades. G Protein-Coupled Receptors (GPCRs) can activate such cascades both at the plasma membrane and, once internalized, from endosomal compartments. Signal kinetics and spatial organization are key determinants of cellular responses. Receptor trafficking (internalization, recycling, endosomal dynamics) thus plays a crucial role in signaling pathways, yet it remains underexplored in theoretical GPCR models. We developed a mathematical framework incorporating receptor trafficking and signal compartmentalization into generic GPCR models. Using a compartmentalized system of ordinary differential equations, we analyzed how internalization and recycling affect receptor-induced responses. This framework addresses two questions: (i) How does trafficking influence signaling? and (ii) How does signaling feedback affect trafficking? We show that trafficking can enhance or reduce ligand action depending on membrane versus endosomal signaling and that feedback mechanisms can generate multi-stability. Applied to the Follicle-Stimulating Hormone Receptor (FSHR), our model calibrated with kinetic data improves ligand characterization and understanding of FSHR signaling.

MS105-03 — From Food Webs to Fluxes: A New Decomposition of Ecosystem Networks

SPEAKERS Caner Kazanci (University of Georgia, USA)
AUTHORS Caner Kazanci (University of Georgia, USA)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Ecosystems are commonly represented as directed graphs describing flows of energy or biomass among species. While these models highlight compartments and flows, neither provides a fundamental dynamical unit. We introduce fluxes as elementary processes that serve as building blocks of ecosystem networks. Inspired by flux balance analysis and metabolic control analysis, a flux represents the smallest process that can theoretically sustain itself, such as a material cycle or a simple food chain embedded within a larger food web. Mathematically, fluxes define a unique network decomposition: any ecological network can be represented as a linear combination of its fluxes. Because this decomposition preserves all network connections, it maintains system-wide properties of the original ecosystem. For example, the total amount of material cycling in the ecosystem equals the sum of cycling occurring within individual fluxes, independent of network size or complexity. More generally, several global ecosystem properties are conserved under this decomposition. We demonstrate the mathematical structure and ecological interpretation of this approach using EcoNet (http://eco.engr.uga.edu), a freely available online platform for ecosystem network analysis.

MS105-04 — A stochastic model of compartmentalized intracellular signalling

SPEAKERS Romain Yvinec (UMR PRC, INRAe Val-de-Loire, 37175, Nouzilly, France / MUSCA, Inria Saclay-Ile-de-France, 91120, Palaiseau, France)
AUTHORS Léo Darrigade (UMR BOA, INRAe Val-de-Loire, 37175, Nouzilly, France); Romain Yvinec (UMR PRC, INRAe Val-de-Loire, 37175, Nouzilly, France / MUSCA, Inria Saclay-Ile-de-France, 91120, Palaiseau, France)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

We study the long-time behaviour of a stochastic model for compartmentalised receptor signalling motivated by the trafficking of G protein-coupled receptors. The model describes the evolution of several chemical species distributed between the plasma membrane and a random population of endosomes. Chemical reactions follow deterministic dynamics inside each compartment, while stochastic events create endosomes by internalisation and recycle them back to the membrane. This leads to a piecewise deterministic Markov process with a variable number of compartments.

Using techniques inspired by the theory of Markov switching systems, we analyse the stability properties of the process. Under suitable contraction assumptions on the reaction dynamics, we prove the existence

and uniqueness of a stationary distribution and establish exponential convergence in Wasserstein distance by a coupling argument. We also investigate exponential convergence in total variation using Harris theorem and identify structural conditions ensuring the existence of accessible Doeblin points.

MS105-06 — Mathematical modeling to infer signaling bias: kinetics and spatial compartmentalization

SPEAKERS Romain Yvinec (UMR PRC, INRAe Val-de-Loire, 37380, Nouzilly, France / Musca, Inria, Université Paris-Saclay, Inria Saclay-Ile-de-France, Palaiseau, 91120, France)

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SCHEDULE Thursday, 16 July 2026; 15:10–15:50; 11.03 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

An active area of research in pharmacology and drug discovery applies to biased signaling: the ability of a ligand to selectively activate some signal transduction pathways as compared to the native ligand acting at the same receptor. At the practical level, experimentalists seek to quantify ligand bias in order to classify ligands according to their selectivity. One popular method uses the so-called operational model to fit dose-response curves.

In this presentation, I will review the main limitations of this methodology, recently pointed out by our group and others. Our objective is then to design a method that fully take into account the kinetic nature of signaling pathways and as well as their possible cross-talks. Kinetic experiments, that measure the activity of several downstream effectors of a receptor after ligand binding with respect to time, are now widely available. I will explain how one can exploit such data and dynamical reaction network modeling with suitable statistical framework to provide a complete “bias map” of a ligand, compared to the native ligand, that successfully answer to our objective.

Going further, we can extend this methodology to take into account recently discovered compartmentalised signaling, which can eventually lead to spatial or location signaling bias. We have thus build a dynamical model that incorporate endocytosis/recycling event and spatial specificity, helping to quantify kinetic experiments under various receptor trafficking perturbations. This new methodology is formulated as either partial differential equations (PDE) or piecewise deterministic Markov processes (PDMP), which calls for interesting new developments in applied mathematics.

MS105-05 — Linking structure to behaviour in regulatory biochemical reaction networks through geometric and combinatorial methods

SPEAKERS Ahmad Mokhtar (The University of Melbourne)

AUTHORS Ahmad Mokhtar (The University of Melbourne); Haripriya Sridharan (The University of Melbourne); Robyn Araujo (The University of Melbourne)

SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 11.03 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

Chemical reaction networks are mathematical tools used to model the dynamics and steady-states of intricate biochemical systems. One major obstacle in analyzing biochemical reaction networks is a lack of

precise knowledge of system parameters such as reaction rates. Often, even the structure of the entire network is not accurately known and therefore simulation methods may not be sufficient in getting insight into how biochemical systems will behave. In this presentation, I will talk about how techniques of tropical geometry and matroid theory can help unveil the structural sources of robustness in regulatory mechanisms. As an application, I will use these techniques to determine Absolute Concentration Robustness in several biochemical reaction networks. This is joint work with Robyn Araujo and Haripriya Sridharan.

MS105-07 — Mapping the co-expression landscape: network sparsification and null models

SPEAKERS Rosemary Braun (Northwestern University, Evanston, IL 60208, USA / Santa Fe Institute, Santa Fe, NM 87501, USA)
AUTHORS Eliza Duvall (Northwestern University, Evanston, IL 60208, USA); Rosemary Braun (Northwestern University, Evanston, IL 60208, USA / Santa Fe Institute, Santa Fe, NM 87501, USA)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Network reconstruction from high-dimensional omics data remains an open challenge. While one can calculate gene-gene co-expression statistics for every pair of genes, these complete weighted graphs must then be sparsified to obtain an interpretable network. Common sparsification approaches (such as thresholding) can lead to an excessively fragmented network, masking the relationship between genes and neglecting the ”strength of weak ties,” wherein a critical but weak relationship may serve as a vital link between two subnetworks. Here we present Network Skeleton Extraction (NSE), a co-expression network generation method using spectral sparsification to sparsify co-expression statistics into minimal co-expression graphs. Spectral sparsification has the advantage of maintaining connections among genes in a manner that preserves the coarse-grained structure and dynamical properties of the input graph. This yields networks that are highly sparse while still being predictive of gene expression. We also present a probabilistic model to generate a null distribution of networks with similar spectral properties, against which inferred networks can be compared. We illustrate the method by applying it to Xenopus transcriptome data across six developmental stages (from pluripotency to lineage commitment), identifying networks whose structure changes over the course of development to give rise to cell-type specific networks.

MS126 — Immunobiology and Infection Subgroup Minisymposium 2026

TRACK Immunobiology & Infection
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 02.21 - HS
ORGANISERS Daniel Reeves (Fred Hutchinson Cancer Center); Jason E. Shoemaker (1. Department of Chemical and Petroleum Engineering, University of Pittsburgh; 6. McGowan Institute for Regenerative Medicine, University of Pittsburgh; 7. Department of Computational and Systems Biology, University of Pittsburgh; 8. Department of Bioengineering, University of Pittsburgh); Morgan Craig (CHU Sainte-Justine Research Centre / Université de Montréal); Tin Phan (Los Alamos National Laboratory)

Minisymposium abstract

The Immunobiology and Infection Subgroup focuses on modelling applied to the various aspects of immunity and immune responses, including host-pathogen interactions. Our subgroup’s aim is to unite mathematical biologists, clinicians, and wet lab researchers working on within-host infectious disease dynamics, host immune responses, causes and effects of inflammation, disease progression and outcome, integration of experimental and clinical data into models, and model-driven experimental design to advance our understanding of the immune system. In our 2026 minisymposium, we are highlighting some of the excellent research in our subgroup focused on a variety of immune-related applications, from new methods to novel applications of models.

MS126-01 — Spatial Heterogeneity in Tumours: How Tumour-Immune Spatial Relationships Impact Immunotherapy Outcomes in Glioblastoma

SPEAKERS Blanche Mongeon (University of Montreal)
AUTHORS Blanche Mongeon (University of Montreal); Morgan Craig (CHU Sainte-Justine Research Centre / Université de Montréal)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Glioblastoma is the most common and deadliest primary brain tumour in adults, with a median survival of 15 months under the current standard of care [TGS20]. Its tumour microenvironment has been shown to be highly heterogeneous [Kar+23], meaning the magnitude of cell-cell interactions differ across space due to spatial differences. Agent-based models (ABMs) are well-suited for describing this spatial cell heterogeneity. When initialized with synthetic data obtained from single-cell imaging modalities, like imaging mass cytometry data, ABMs can capture realistic intra-tumoral dynamics [Mon+24]. In this talk, we show how spatial relationships between immune and cancer cells shape tumour-immune dynamics and influence predictions of immunotherapy outcomes. To this end, we build a glioblastoma-immune system ABM and include in our new spatial model immunotherapies previously modeled with an ordinary differential equation (ODE) model [MC25]. Unlike ABMs, ODE models have a very low computational cost. However, they assume cell populations are well-mixed. Our work lays the foundations for combining ABMs and ODE models to leverage both the spatial realism of the former and the computational efficiency of the latter in describing the overall tumour progression.

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MS126-02 — Granger-causality analysis reveals defective viral genomes with antiviral potential from longitudinal infection data

SPEAKERS Tanja Laske (Leibniz Institute of Virology, Viral Systems Modeling, Hamburg, Germany)
AUTHORS Giulia Saresini (Leibniz Institute of Virology, Viral Systems Modeling, Hamburg, Germany); Jan Baumbach (University of Hamburg, Institute for Computational Systems Biomedicine, Hamburg, Germany); Jens Lohmann (University of Hamburg, Institute for Computational Systems Biomedicine, Hamburg, Germany); Mia Le (Bernhard Nocht Institute for Tropical Medicine (BNITM), Hamburg, Germany); Nils Petersen (Bernhard Nocht Institute for Tropical Medicine (BNITM), Hamburg, Germany); Sara Grundel (Leipzig University of Applied Sciences (HTWK), Mathematisch-Naturwissenschaftliches Zentrum (MNZ), Leipzig, Germany); Sophie Duraffour (Bernhard Nocht Institute for Tropical Medicine (BNITM), Hamburg, Germany); Tanja Laske (Leibniz Institute of Virology, Viral Systems Modeling, Hamburg, Germany)

SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Defective viral genomes (DVGs) interfere with infectious standard virus (STV) replication and are considered promising antiviral agents. In longitudinal influenza A virus (IAV) infections, DVG accumulation drives oscillatory virus dynamics (von-Magnus effect [Von54]). Experimental evaluation of individual DVGs is resource-intensive, motivating computational prioritization. We aim to identify DVGs influencing STV titers using Granger-causality analysis of sequencing data from a longitudinal IAV infection [Pel+21]. For 1,968 DVGs, two ordinary least squares models were compared: a restricted model trained on STV titers, and a full model including DVG trajectories. Upon evaluation of goodness of predictions, DVGs were classified as Granger-causing (DVG predicts STV titers), Granger-caused (STV titers predict DVG), Granger-bi-directional (mutual predictive influence), or non-related [Gra69; Lüt05]. We found that 109 DVGs significantly influenced STV titers. A previously validated DVG that reduced STV titers by five orders of magnitude in experiments was classified as Granger-bi-directional, whereas a candidate reducing STV titers by only three orders of magnitude was categorized as non-related. These results indicate that Granger causality-based classification reflects antiviral efficacy and enables prioritization of potent DVGs. This proof-of-concept shows that Granger-causality allows systematic identification of DVGs with antiviral potential, reducing experimental effort.

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MS126-03 — Multi-modal data integration and individual cell-based modelling to infer viral spread and innate immune dynamics in human epithelia

SPEAKERS Pascal Lukas (Department of Internal Medicine 5, Haematology and Oncology, Friedrich-Alexander-Universität Erlangen-Nürnberg and Universitätsklinikum Erlangen)

AUTHORS Aurélien Gibeaud (CIRI, Centre International de Recherche en Infectiologie, Team VirPath, Université de Lyon, INSERM U1111, Université Claude Bernard Lyon 1, CNRS); Clarisse Schumer (Université Paris Cité and Université Sorbonne Paris Nord); Frederick Graw (Department of Internal Medicine 5, Haematology and Oncology, Friedrich-Alexander-Universität Erlangen-Nürnberg and Universitätsklinikum Erlangen; Deutsches Zentrum Immuntherapie (DZI); Bavarian Cancer Research Centre (BZKF)); Jonas Arruda (Bonn Center for Mathematical Life Sciences & Life and Medical Sciences Institute, University of Bonn); Jérémie Guedj (Université Paris Cité and Université Sorbonne Paris Nord); Olivier Terrier (CIRI, Centre International de Recherche en Infectiologie, Team VirPath, Université de Lyon, INSERM U1111, Université Claude Bernard Lyon 1, CNRS); Pascal Lukas (Department of Internal Medicine 5, Haematology and Oncology, Friedrich-Alexander-Universität Erlangen-Nürnberg and Universitätsklinikum Erlangen)

SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Understanding the mechanisms that govern viral spread within human tissues remains a major challenge, especially for identifying and quantifying key factors that influence viral transmission and innate immune responses. Although mathematical models and experimental advances have provided valuable insights, revealing the complex spatio-temporal interactions of infection and immune processes at the tissue level has remained elusive. Here, we present a novel workflow that combines multimodal experimental data and individual cell-based modeling to allow the inference of viral and immune kinetics within tissues. While standard inference methods typically require custom summary statistics and resourceful re-fitting procedures for individual data sets, our workflow relies on simulation-based inference using BayesFlow, a framework for neural posterior estimation that allows for amortized inference of multimodal data. Validating our approach with synthetic data, we showed that integrating spatial information is essential for reliably inferring viral transmission kinetics and innate immune interactions within human airway epithelium, with subsequent application to experimental data on SARS-CoV-2 infection indicating local transmission as the dominant mode of viral spread. Our method can be readily adapted to various respiratory viral infections, helping to investigate co-infection and treatment scenarios, and presents a general framework for analysing viral infections at tissue level.

MS126-04 — Identifying Sex-Specific Immune during Primary Influenza Infection Using Mathematical Modeling

SPEAKERS Jason E. Shoemaker (1. Department of Chemical and Petroleum Engineering, University of Pittsburgh; 6. McGowan Institute for Regenerative Medicine, University of Pittsburgh; 7. Department of Computational and Systems Biology, University of Pittsburgh; 8. Department of Bioengineering, University of Pittsburgh)

AUTHORS Amie J. Eisfeld (2. Department of Pathobiological Sciences, Influenza Research Institute, University of Wisconsin-Madison); Asim Biswas (2. Department of Pathobiological Sciences, Influenza Research Institute, University of Wisconsin-Madison); Jason E. Shoemaker (1. Department of Chemical and Petroleum Engineering, University of Pittsburgh; 6. McGowan Institute for Regenerative Medicine, University of Pittsburgh; 7. Department of Computational and Systems Biology, University of Pittsburgh; 8. Department of Bioengineering, University of Pittsburgh); Tatum S. Liparulo (1. Department of Chemical and Petroleum Engineering, University of Pittsburg); Yoshihiro Kawaoka (2. Department of Pathobiological Sciences, Influenza Research Institute, University of Wisconsin-Madison; 3. Department of Virology, Institute of Medical Science, University of Tokyo; 4. The Research Center for Global Viral Diseases, National Center for Global Health and Medicine Research Institute; 5. The University of Tokyo Pandemic Preparedness, Infection and Advanced Research Center (UTOPIA), University of Tokyo)

SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 02.21 - HS

TRACK Immunobiology & Infection

Abstract

In humans, differences in immune response between males and females influence influenza infection outcomes. During the 2009 H1N1 pandemic, Eshima et al., 2011 found that adult females were 40% more likely to be hospitalized than their male age-matched counterparts. The innate immune response has been implicated as a factor of these sex differences. Together with collaborators at UW Madison, we have completed experiments on male and female mice infected with CA04-H1N1 influenza. These experiments show that female mice have increased viral production at 36 hours post infection and early, excessive innate immune activation characterized by proinflammatory cytokine profiles, and critical differences in macrophage counts. Histopathology shows lesions are present in the alveolar region of female, but not male, mice, indicating influenza virus penetrates more deeply in female lungs. While experimental data identifies key immune components linked to severity, mathematical modeling enables detection of sex-specific mechanistic differences. We developed a mathematical model of influenza infection to identify mechanisms responsible for observed increases in disease severity in female mice. We have identified two models with significant evidence using Bayesian model selection (AICc), where each model has a different parameter subset with individual male and female values. These parameters are sex-specific and each model points to a unique mechanism that could be targeted for regulating severe influenza disease.

MS126-05 — Regularized estimation in high-dimensional mechanistic models: Application to vaccine development

SPEAKERS Lisa Crépin (Université de Bordeaux, Bordeaux Population Health Inserm; Inria Bordeaux SISTM, Vaccine Research Institute)
AUTHORS Boris Hejblum (Université de Bordeaux, Bordeaux Population Health Inserm; Inria Bordeaux SISTM, Vaccine Research Institute); Cecile Prous-Lima (Université de Bordeaux, Bordeaux Population Health Inserm); Lisa Crépin (Université de Bordeaux, Bordeaux Population Health Inserm; Inria Bordeaux SISTM, Vaccine Research Institute); Melanie Prague (Université de Bordeaux, Bordeaux Population Health Inserm; Inria Bordeaux SISTM, Vaccine Research Institute); Morgan Craig (CHU Sainte-Justine Research Centre / Université de Montréal); Rodolphe Thiebaut (Université de Bordeaux, Bordeaux Population Health Inserm; Inria Bordeaux SISTM, Vaccine Research Institute)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Objectives: Mechanistic models describe biological processes over time, but they often rely on few observed compartments and sparse longitudinal data. They may therefore be too simple to capture complex processes, such as post-vaccination immune dynamics, or may suffer from identifiability issues, especially in nonlinear mixed-effects models based on differential equations. At the same time, longitudinal high-throughput data, including transcriptomics and proteomics, are increasingly available and may help inform unobserved biological processes. Integrating such high-dimensional data into mechanistic models with latent compartments remains difficult.

Methods: We hypothesize that observed omics biomarkers can inform the dynamics of unobserved immune compartments. We propose a regularized estimation method for mechanistic models with latent compartments measured indirectly through high-dimensional longitudinal biomarkers. Relevant biomarkers are selected by regularizing the parameters linking them to latent compartments while estimating population mechanistic parameters. The algorithm alternates between a regularization step, based on penalized log-likelihood derivatives, and a mechanistic inference step using SAEM in Monolix.

Results: We evaluated the method in simulations and applied it to immune responses after Pfizer/BioNTech COVID-19 vaccination in 15 infection-naïve individuals (Rinchai et al.). Daily blood samples were collected for 9 days after each dose, with 8,172 gene expression measurements grouped into 34 pathways, and serology at baseline, day 7, and day 14. Inflammation, neutrophils, interferon, and type I interferon were most strongly associated with short-term immune response dynamics.

Conclusion: Transcriptomic data improved identifiability of latent compartments. Limitations include the assumed linear biomarker-compartment relationship and high computational cost. The method is implemented in the REMixed R package on CRAN.

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MS126-06 — Modeling how memory CD8 T cells may effect post-treatment control of HIV infection

SPEAKERS Narendra Dixit (Indian Institute of Science)
AUTHORS Narendra Dixit (Indian Institute of Science)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Antiretroviral therapy (ART) for HIV-1 infection is not curative. Treatment interruption typically results in viral rebound and progressive disease, necessitating lifelong ART. However, a small fraction of people living with HIV achieve long-term post-treatment control (PTC) of the virus, offering hope that strategies may be devised that obviate the need for lifelong treatment. While mechanisms underlying PTC remain to be understood, recent studies have identified early treatment initiation as a key factor and suggested a role for CD8 T cells. Here, we developed a within-host mathematical model to elucidate the associated mechanisms. We hypothesized, based on immunological studies, that upon infection, virus-specific CD8 T cells accumulate 'antigenic experience' through continual exposure to the antigen, which compromises their survival. Early ART initiation limits this experience, preserving memory CD8 T cells and enabling better response to rebounding virus post-ART. We fit the model to longitudinal data from a recent macaque study using a nonlinear mixed effects approach. The model exhibited bistability. One stable steady state had low viral load and high memory cell levels, and the other vice versa, recapitulating PTC and progressive disease, respectively. Early treatment initiation made the control state more accessible. The model predicted treatment initiation times and the associated memory CD8 T cell pool sizes for maximizing PTC, informing interventions.

MS126-07 — Quantitative modeling of proliferation and differentiation in cell populations

SPEAKERS Shoya Iwanami (Interdisciplinary Biology Laboratory, Graduate School of Science, Nagoya University, Japan)
AUTHORS Shoya Iwanami (Interdisciplinary Biology Laboratory, Graduate School of Science, Nagoya University, Japan)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Cell populations consist of heterogeneous cells and are maintained through cell production via complex differentiation processes. To quantitatively understand these intricate dynamics, we employed mathematical modeling across two distinct biological domains: the hematopoietic system and oncogenic proliferation. Regarding the differentiation dynamics of hematopoietic stem cells (HSCs), we performed a mathematical analysis of experimental tracking data from blood cell production following transplantation. This allowed us to quantify which production pathways play a dominant role in long-term hematopoietic maintenance, thereby identifying the importance of pathways within a hierarchical differentiation system. In parallel, we modeled the cancer proliferation process driven by extrachromosomal DNA (ecDNA) harboring driver mutations. Our model elucidates how the unequal segregation mechanism of ecDNA during cell division generates profound genetic heterogeneity within the population, and how this heterogeneity contributes to overall growth rates and environmental adaptability. Through these two studies, we discuss the broader outlook for the quantitative understanding of cell population dynamics. By formulating these processes mathematically, we aim to provide a comprehensive theoretical foundation for manipulating cell fates in regenerative medicine and forecasting therapeutic resistance in oncology.

MS126-08 — Identifying determinants of long-term viral suppression following broadly neutralizing antibody treatment against HIV-1

SPEAKERS Tyler Cassidy (University of Leeds)
AUTHORS Tyler Cassidy (University of Leeds)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Due to their long circulating half-life, high neutralization potency, and large breadth of coverage, broadly neutralizing antibodies are increasingly studied for the treatment of HIV-1 infection. Recent phase I clinical studies of antibody treatment have demonstrated robust and durable antiviral effects in viremic participants and in participants undergoing analytical treatment interruption. Consequently, these antibodies are an attractive novel treatment candidate for long-term, antiretroviral therapy-free, viral control. However, these early-stage trials are small, time consuming, and expensive. I'll show how mechanistic mathematical

modelling can identify clinically actionable determinants of treatment response and uncover the evolutionary dynamics driving viral rebound. Further, I'll show how combining mechanistic modelling and virtual population approaches can predict the duration of viral suppression in both monotherapy and combination clinical trial results. The resulting virtual population platform provides mechanistic insight that identifies clinically relevant biomarkers that are predictive of bnAb response and individualize bnAb combination therapy against HIV-1 infection. Further, this computational approach can quantify the increased antiviral effect of recently developed bnAb variants.

MS133 — Stochastic and Deterministic Methods in Mathematical Biology: From Theory to Computation

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Thursday, 16 July 2026; 10:40–11:40; 15.11 - HS
ORGANISERS Luis Melara (Shippensburg University of Pennsylvania); Ryan Evans (National Institute of Standards and Technology)

Minisymposium abstract

Stochastic methods have recently been used to solve important problems in mathematical biology, from modeling degradation of protective layers in biosensors to separating signal from noise in diagnostic instruments. Deterministic tools like integrodifferential and partial differential equations remain powerful for modeling phenomena like reaction-diffusion processes and tumor growth. In these applications, questions of existence and uniqueness provide the rigorous foundation for reliable simulation. This mini-symposium brings these approaches together through a series of presentations that reveal how theoretical guarantees inform computational methods, and how stochastic models complement deterministic frameworks. Topics span analytical foundations to numerical simulation, connecting theory to experiment across different biological applications.

MS133-01 — Optimal Bandwith Selection in Biological Field Effect Transistor Measurements

SPEAKERS Luis Melara (Shippensburg University of Pennsylvania)
AUTHORS Luis Melara (Shippensburg University of Pennsylvania)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.11 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Instruments known as biological field effect transistors (BioFETs) have potential to offer affordable and highly sensitive medical diagnostics that can be administered point of care. Signal is separated from noise in these instruments with stochastic regression, a technique that involves modeling the signal with a linear deterministic drift term and a white noise term that captures stochastic behavior. Both of these terms have coefficients that are estimated from data or simulation with local weighted regression and maximum likelihood estimation. Crucially, these coefficient estimation techniques depend on an averaging function–known as a kernel function–and an averaging window, the size of which is governed by variable known as the bandwidth parameter. In this talk, we determine optimal bandwidth parameters associated with an experimental BioFET measurement and simulation data. Cross validation is performed with respect to different instrument aspect ratios. Results show optimal badwidth parameters are surprisingly consistent across aspect ratios, and suggest a choice of kernel function.

MS133-03 — A Numerical Method for a Singular Integrodifferential Equation Model of Biosensor Dynamics

SPEAKERS Ryan Evans (National Institute of Standards and Technology)
AUTHORS Ryan Evans (National Institute of Standards and Technology)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.11 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Biological field effect transistors are portable and highly sensitive biosensors that show promise as medical diagnostic instruments. During a typical experiment, chemical reactants from solution diffuse onto a surface to bind with receptors that are confined to the surface. This produces a time series signal that may be used to analyze the reaction of interest. In experimentally relevant parameter regimes, a model for these experiments takes the form of a nonlinear and logarithmically singular integrodifferential equation. A numerical method based on a Nystrom discretization and specialized quadrature will be presented, and it will be demonstrated that this method achieves second-order accuracy. Numerical simulation compares favorably with experiment.

MS133-04 — Probabilistic Cellular Automaton Modeling for Monolayer Degradation in Biosensors

SPEAKERS Wes Caldwell (Johns Hopkins University)
AUTHORS Anthony Kearsley (National Institute of Standards and Technology); Ryan Evans (National Institute of Standards and Technology); Wes Caldwell (Johns Hopkins University)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.11 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

A novel chip-scale electrochemical biosensor is being developed to detect biomolecules with high sensitivity. Unlike traditional methods such as qPCR that require specialized equipment and trained personal, these cost-effective and portable chip-scale sensors are designed to administer tests at the point of care. Target molecules of interest are immobilized on the surface, and specific interactions with molecules that diffuse onto the surface from solution produces an electrochemical signal. To prevent non-specific interactions that may contaminate the signal, the sensor surface is coated with a thin passivating self-asssembled monolayer (SAM). The SAM degrades over time, which exposes sensor's bare metal surface and can lead to non-specific interactions. Understanding and predicting SAM degradation is key to producing reliable biosensors. A probabilistic cellular automaton (PCA) model for SAM degradation is given and solved explicitly in one dimension. A continuous-time limit is computed for the case of Poisson distributed probabilities.

MS137 — Population Dynamics in Disturbed Landscapes

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Thursday, 16 July 2026; 10:40–12:00; 03.01 - HS
ORGANISERS Christian Wiewelhove (University of British Columbia - Okanagan); Frank Hilker (Osnabrück University)

Minisymposium abstract

Ecosystems all around the world are affected by disturbances. These disturbances can be natural (e.g., wildfires, floods, droughts, pest outbreaks, landslides), anthropogenic (e.g. agriculture and farming, forestry, urbanization, fishing, deforestation, global warming, pollution, introduction of alien invasive species), or a combination of both. They are known to affect life history traits such as reproduction and survival, how populations interact, or how individuals move in landscapes. Population dynamics play a key role in many areas of ecology including conservation, livestock and wildlife management, as well as natural resource management. It is therefore imperative to understand how disturbances affect population dynamics. The minisymposium introduces a broad range of perspectives and approaches to study questions related to population dynamics and disturbed landscapes. It will feature mathematical modelling methods such as partial differential equations, networks, or metapopulation-type models. The presentations will showcase latest research on modelling the interaction of aspects such as population dynamics, different forms of movement, spatial heterogeneity, and disturbance regimes.

MS137-01 — Goshawk-Squirrel Predator-Prey Dynamics in Patchy Forests Subject to Fire and Forestry Disturbances

SPEAKERS Christian Wiewelhove (University of British Columbia - Okanagan)
AUTHORS Christian Wiewelhove (University of British Columbia - Okanagan); Michael Noonan (University of British Columbia - Okanagan); Rebecca Tyson (University of British Columbia - Okanagan)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Our work focuses on understanding predator-prey dynamics in a complex landscape that is constantly changing due to forest growth and disturbances such as wildfire or logging. Climate change is increasing the frequency and severity of wildfires, causing additional stress to the forest ecosystem on top of existing anthropogenic activity. We seek to understand how these landscape disturbances affect the persistence of resident predator-prey populations. In particular, how changes to disturbance frequency and magnitude alter ecosystem stability. As a practical example of this interconnected system, we focus on the goshawk-squirrel predator-prey system in Canadian coniferous forests. Most predator-prey models that consider patchy landscapes, keep patch properties constant over time. We include time-varying patch properties, ac- counting for the disturbance-regrowth cycle. An additional complicating factor we take into account is the territoriality of squirrels. We present a mathematical model for this system and show how species persistence is affected by a chang- ing disturbance regime. Our results show that changes in disturbance frequency, along with patch sizes affects species densities and their persistence. Ultimately, we hope that the results and the model will inform conversa- tion efforts for the goshawk and for predator-prey systems that rely on mature forests.

MS137-02 — Starvation-driven cell patterning: integrating lab experiments and mathematical modelling

SPEAKERS Cinzia Soresina (University of Trento)
AUTHORS Anna Bernardi-D'Agostini (CNR IMATI, Milano, Italy); Cinzia Soresina (University of Trento); Giovanni Cappello (LIPhy, CNRS and Université Grenoble Alpes)); Simone Pezzuto (University of Trento, Italy)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

We present a reaction–diffusion model describing the interactions among cells, nutrients, and growth factors, aimed at capturing the emergence of starvation-driven cell pattern formation, a phenomenon recently observed in laboratory experiments under nutrient-limited growth conditions. Experiments and modelling were developed in parallel, enabling progressively more targeted experimental design while enhancing the biological realism of the model. This interdisciplinary feedback loop led to the formulation of new hypotheses and enabled the estimation of several key parameters. Numerical simulations show that the model reproduces pattern formation in both one- and two-dimensional spatial domains. To provide theoretical support for these findings, we performed a Turing instability analysis to investigate the potential for diffusion-driven instability. The analysis indicates that the observed patterns are not driven by chemotaxis; rather, they arise naturally under starvation conditions and display structural similarities to the Klausmeier model for vegetation pattern formation in semi-arid environments, suggesting the robustness of the underlying mechanism across biological scales.

MS137-03 — Patterned biodiversity responses to disturbance severity in competition-governed metacommunities

SPEAKERS Daniel Bearup (University of Leicester)
AUTHORS Daniel Bearup (University of Leicester)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Whether static, e.g. habitat destruction, or dynamic, e.g. environmental fluctuations, disturbance of a landscape has typically been expected to degrade its resident ecosystems. However, recent modelling studies [Zha+23; Zha+25] have predicted that community biodiversity can oscillate, both rising and falling, as disturbance severity increases. Similar patterns have been found by re-examination of empirical data collected in disturbed ecosystems. While the overall trend is for biodiversity to decline with disturbance severity, these responses could mislead ecosystem managers as to the impact of ongoing climate change and the effectiveness of conservation interventions.

In this talk, I will outline the general mechanism that gives rise to these responses; specifically, the effect of a tradeoff in growth rate and competitive ability. I will then explore why identical results are obtained for apparently different types of disturbance.

References

[Zha+23] Helin Zhang et al. "Complex nonmonotonic responses of biodiversity to habitat destruction". In: *Ecology* 104.12 (2023), e4177. DOI: https://doi.org/10.1002/ecy.4177.

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MS137-04 — Response scenarios of the total population size in fluctuating environments

SPEAKERS Daniel Franco (Universidad Nacional de Educación a Distancia, Spain)
AUTHORS Daniel Franco (Universidad Nacional de Educación a Distancia, Spain); J.A. Cid (University of Vigo, Spain); P. Amster (University of Buenos Aires, Argentina)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 03.01 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Habitat fragmentation, driven by human activities, poses a major threat to biodiversity by isolating populations and disrupting ecological processes. Metapopulation models are essential for understanding these dynamics, but they often assume temporally homogeneous environments. This assumption overlooks the reality that abiotic and biotic factors, such as temperature or resource availability, fluctuate over time, altering growth and competition. In this talk, we will show that such temporal heterogeneity can alter the impact of dispersal on total population size, leading to new response patterns not observed in constant environments using a discrete-time two-patch model.

MS142 — Mechanistic insights from in-vivo and in-vitro data: modelling tissue physiology and pathology away from equilibrium

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 02.01 - HS
ORGANISERS Luca Alasio (INRIA Paris & LJLL); Marcela Szopos (Université Paris Cité)

Minisymposium abstract

Tissues in health and disease are inherently dynamic, adaptive, and driven by processes that operate away from classical equilibrium assumptions. Integrating mechanistic models with experimental data (spanning in-vitro systems, organoids, live imaging, and in-vivo measurements) is key to unraveling how physiological function emerges and how dysregulation leads to pathology. However, traditional modelling frameworks often fail to capture the transient nature of biological regulation, stochastic fluctuations, and feedback mechanisms that shape tissue behaviour over multiple scales. This mini-symposium will bring together researchers advancing mathematical and computational models that are informed by experimental datasets concerning tissue dynamics in physiology and disease, with the goal of fostering an integrated view

from theory to applications. The main topics will include (i) active tissue response and shape variation, (ii) strategies to integrate heterogeneous experimental data into predictive models, (iii) computational techniques for analysing transient behaviours in complex biological tissues, and (iv) multiscale formalisms that link cell-level events to emergent tissue responses.

MS142-03 — Collective cell dynamics driven by active boundaries and cell turnover: a multi-scale approach.

SPEAKERS Laurent Navoret (Université de Strasbourg)
AUTHORS Laurent Navoret (Université de Strasbourg)
SCHEDULE Thursday, 16 July 2026; 10:40–11:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

During morphogenesis, hundreds of cells undergo large-scale movements, and the biological or mechanical regulatory factors involved are still poorly understood, despite intensive research. To better characterize the emergence of these movements, we propose a microscopic Vicsek-type model, combining the alignment of polarities and contact forces, and have studied the dynamics in confined environments. A first study has raised the key role of active boundaries in triggering rotational motion. Cells are then confined within an annular region bounded by self-generated elastic cables and comparisons between numerical simulations of the model and experiments show quantitative agreement. In a second study, we have investigated apoptosis, the spontaneous death of cells, which plays an ambiguous role in these dynamics: we have thus added reorientation of polarities near apoptotic cells in the model. In this case, numerical simulations allow us to highlight the influence of apoptosis in congested situations. Through a statistical description of the dynamics, we then propose the derivation of fluid-type models (Self-Organized Hydrodynamics) under different parameter regimes, enabling a better understanding of the process.

These works are the result of strong collaborations with Simon Lo Vecchio, Daniel Riveline, Roxana Sublet and Marcela Szopos and recent developments are also in close cooperation with all the members of the ANR Mapeflu project (ANR-22-CE45-0028).

MS142-01 — Pattern formation in a phenotype-structured Shigesada-Kawasaki-Teramoto model

SPEAKERS Davide Cusseddu (https://orcid.org/0000-0002-6882-9486)
AUTHORS Davide Cusseddu (https://orcid.org/0000-0002-6882-9486)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Shigesada, Kawasaki, and Teramoto showed that introducing cross-diffusion into the spatial Lotka–Volterra competition model can destabilise homogeneous equilibria and generate spatial patterns. In this talk, I will consider populations with phenotype heterogeneity and introduce a general framework in which individuals' movements and interactions are phenotype-dependent. Motivated by cellular plasticity, I study a regime of rapid phenotype switching and derive conditions for cross-diffusion- and phenotype-driven instabilities. I conclude with numerical simulations illustrating the role of the phenotype distribution and its impact on competitive outcomes. This is a joint work with Tommaso Lorenzi and Gaetana Gambino.

MS142-02 — A Starling resistor model for choroidal venous flow

SPEAKERS Federica Vanone (Gran Sasso Science Institute)
AUTHORS Federica Vanone (Gran Sasso Science Institute)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

The choroid is a densely vascularised layer of tissue of the eye, lying between the retina and the sclera (outer layer of the eye), which carries the majority of ocular blood flow. Vortex veins drain blood out of the eye (to the orbit), crossing the sclera from the choroidal circulation. Animal experiments have shown that pressure in these veins is tightly linked to intraocular pressure (IOP) [Mäe92]. Partial collapse of the vortex veins in the sclera has been hypothesised to be responsible for venous pressure control in the choroid [Bil62; Spa+22]. In this work, we formulate a Starling resistor model of a vortex vein, investigating whether it can explain the observed pressure behaviour. We model the vein as a collapsible tube, with a segment within the choroid subjected to IOP, and a segment crossing the sclera in which the external pressure linearly decreases from IOP to the orbital tissue pressure. In order to fully reproduce the experimental results [Bil62], we extend the model to account for more than one vortex vein. We show that, under physiological conditions, the experimentally observed behaviour of the choroidal venous outflow can be reproduced by the model, if the vortex vein is collapsed in the scleral segment. In this case, increasing IOP results in an increase of choroidal venous pressure of almost the same amount. In summary, the model suggests a mechanical explanation for passive control of choroidal venous pressure by vortex vein collapse within the sclera.

Co-authors: - Peter Stewart, School of Mathematics & Statistics, University of Glasgow, Glasgow, UK - Alexander J. E. Foss, Department of Ophthalmology, Nottingham University Hospitals NHS Trust, Nottingham, UK - Jennifer Tweedy, Mathematical Sciences, University of Bath, Bath, UK - Rodolfo Repetto, Department of Civil, Chemical and Environmental Engineering, University of Genoa, Genoa, Italy.

References

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MS142-04 — Active shape evolution in tissue mechanics

SPEAKERS Naoufel Cresson (INRIA, LJLL)
AUTHORS Naoufel Cresson (INRIA, LJLL)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Since D’Arcy Thompson, the interplay between mechanics and shape in living organisms has been a central question in mathematical biology. In tissue mechanics, shape variation has been studied through growth, development, and wound healing with various modeling approaches and objectives ([Mir+25; PC24]).

We develop a variational approach to active deformable materials where shape evolution is characterized by its dissipative behavior in response to internal activity. Inspired by Generalized Standard Materials’ extension to tissue mechanics ([Tli+15; Xi+18]), the tissue is described by an Eulerian velocity field determined at each instant by minimizing a functional combining energy dissipation and active energy input. The tissue boundary evolves as a free boundary carried by this velocity — making shape a dynamical consequence of the material’s internal processes rather than a prescribed input.

We will discuss the mechanistic insights of this modeling approach for tissue mechanics, its mathematical properties, and its biological interpretation in physiology and pathology.

This is joint work with L. Alasio (INRIA & LJLL) & M. Szopos (UPCité & MAP5).

References

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MS142-05 — Modelling the emergence of connective tissue architecture

SPEAKERS Pauline Chassonnery (BRGM Orléans, Bureau de recherches géologiques et minières)
AUTHORS Pauline Chassonnery (BRGM Orléans, Bureau de recherches géologiques et minières)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Mathematical models present numerous advantages when studying biological issues, mainly because they enable us to explore hypotheses that cannot be tested in vitro and even less in vivo, such as evolutionary scenarii, possible alternative mechanisms or arbitrary setting of physiological parameters.

In this talk, we will use a 3D agents-based model to explore the question of how biological tissues acquire their 3D functional architecture. The spatial organization of any living tissue has a deep impact on this tissue's functionality. We focus on the case of white adipose tissue (WAT), which can represent up to 50% of the body weight and is now recognized to be an important endocrine organ involved in all physiological functions. Mature WAT consists of lobular clusters of adipocyte cells surrounded by an organized collagen fiber network. However, tridimensional observations show that the cells clusters are not as well separated as what 2D imaging could lead us to believe, but are instead organized into connected macro-structures [Dic+19]. Hence, a fully tridimensional investigation of how WAT acquires its functional structure is needed.

Our hypothesis is that the emergence of WAT structuration could be explained by simple mechanical interactions between adipocyte cells and collagen fibers [Peu+17]. To test it, we developed an agent-based model featuring cells represented as spheres appearing and growing in a dynamical 3D network of cross-linked spherocylinders (modelling the collagen fibers) [Cha+24]. Using state-of-the-art 3D data visualization and segmentation tools, we will show that this model is able to produce cell structures morphologically similar to those observed in experimental images [Cha+]. Through fine parametrical analysis, we will identify the key parameters of the model and study their influence on the simulation outcome. We will show that this simple model can give insights on how complex 3D cell and fiber structures could spontaneously emerge as a result of simple mechanical interactions between cells and fibers, highlighting the key role of mechanics in tissue structuration.

List of co-authors: Pauline Chassonnery, Jenny Paupert, Anne Lorsignol, Childérick Séverac, Marielle Ousset, Pierre Degond, Louis Casteilla, Diane Peurichard.

References

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MS142-06 — A new size-dependant individual based model for epithelial tissue

SPEAKERS Sophie Hecht (Sorbonne Universite)
AUTHORS Sophie Hecht (Sorbonne Universite)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Epithelial tissues are densely packed, confluent systems whose organization and mechanics are commonly described mathematically using vertex models. While these approaches successfully capture membrane dynamics and cell–cell junction remodeling, they inherently lack the ability to explicitly account for intercellular attraction and/or repulsion forces. As a result, key aspects of tissue organization driven by such interactions remain insufficiently explored. In this work, we introduce a novel individual-based model in which cells are represented as radius-dependent spheres. This framework explicitly incorporates both attractive and repulsive interactions between cells, enabling a more direct description of mechanical cell coupling within the tissue. A central feature of our model is the ability of cell size to dynamically evolve, allowing the system to adapt to the balance of forces and reach mechanically consistent configurations. We analyze the stationary states of this system and characterize the resulting tissue organization across parameters regimes. Finally, we validate our approach by comparing model predictions with experimental data from retinal pigment epithelium.

MS142-07 — PDE models for the growth of heterogeneous cell populations: travelling fronts, sharp interfaces, and concentration phenomena

SPEAKERS Tommaso Lorenzi (Politecnico di Torino)
AUTHORS Tommaso Lorenzi (Politecnico di Torino)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In this talk, PDE models for the growth of heterogeneous cell populations will be considered. Both models with discrete phenotype states, which consist of coupled systems of nonlinear PDEs, and models wherein the phenotype enters as a continuous structuring variable, which are formulated as non-local PDEs, will be examined. Focusing on scenarios where cells with different phenotypes are spatially segregated across invading fronts, travelling wave solutions that exhibit sharp interfaces, for the first class of models, and concentration phenomena, for the second class of models, will be studied. The insights into the mechanisms that underpin collective cell migration generated by these mathematical results will be briefly discussed.

MS161 — Whole-cell modelling: progress and perspectives

TRACK Systems Biology and Biochemical Networks
SCHEDULE Thursday, 16 July 2026; 10:40–16:30; 11.01 - HS
ORGANISERS Thomas Williams (University of Melbourne)

Minisymposium abstract

The landmark first whole-cell model (WCM) – of Mycoplasma genitalium – was published in 2012, offering insight into cellular biology at unprecedented scale and detail [Kar+12]. The years since have seen WCMs developed by a variety of groups for a range of organisms, and major strides have been made in improving the biological fidelity and analytical power of these models. The rapid development of computing hardware and machine-learning methods has opened further possibilities for whole-cell modelling. However, challenges remain: how can WCMs be rigorously validated by experimental data? How can structural aspects of the cell, including spatial organisation, be accommodated in a WCM framework? What biological insights can be gleaned from WCMs which are not possible with simpler models? This minisymposium will showcase exciting work at the cutting edge of WCM research from a range of countries and institutions, on topics ranging from cellular metabolism to membrane dynamics, to gene perturbation and beyond, unified by a common goal of holistic, comprehensive modelling of cells to fuel biological discovery.

References

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MS161-01 — Producing quantitative protein function data at scale to enable mechanistic simulation - and protein design

SPEAKERS Cyrus Knudsen (Stanford University)
AUTHORS Cyrus Knudsen (Stanford University); Zane Chan (Stanford University); Markus Covert (Stanford University)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

To this day, there is still no existing quantitative model that can simulate the reaction rates and concentration changes in the comprehensive biochemical reaction network of a cell. Such a simulation model would be useful for understanding the nature of living cells.

A primary barrier is the lack of accurate parameters. Key biochemical parameters are notoriously difficult to measure, with published values for the same reaction often varying by orders of magnitude. This uncertainty severely limits the predictive power of mechanistic models. Furthermore, while advances in machine learning have enabled protein structure prediction, predicting quantitative functional properties (e.g., catalytic constants) remains a major hurdle, largely due to the scarcity of high-quality in vivo data for model training.

Here, we present a framework that overcomes this limitation. Using convex optimization, we integrate mechanistic constraints with high-throughput measurements, and demonstrate that this approach yields large-scale estimates of protein functional parameters that are consistent with in vivo phenotypes and diverse experimental data. These parameters enable accurate mechanistic simulation of cellular metabolism and provide a rich, quantitative dataset of biomolecule function, creating a valuable resource for training next-generation predictive models for protein design.

MS161-02 — Thermodynamically Consistent Mechanochemical Modelling of Actin Dynamics

SPEAKERS Volkan Ozcoban (University of Melbourne)
AUTHORS Volkan Ozcoban (University of Melbourne); Michael Pan (University of New South Wales Sydney); Vijay Rajagopal (The University of Melbourne)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Actin assembly drives force generation and membrane remodelling (protrusion, budding, tethering). Many actin kinetic models omit explicit energy accounting with force production [VYO05; Dit+09], limiting reliable coupling to multiscale models of whole-cell remodelling. Prior efforts linking actin kinetics to continuum mechanics often approximate energy flows and omit explicit nucleotide states [MO96; GPR25]. Here, we present a modular, thermodynamically consistent actin model combining nucleation, polymerisation, depolymerisation, ATP/ADP states, and force production. Built using a bond graph approach (thermodynamically consistent ODEs) [GPR25; GC14; Raj+22], the technique preserves energy accounting and provides modular ports for mechanical coupling. The model is currently being used to explore sensitivity and parameter identifiability, with a particular focus on how ATP/ADP state changes influence conservation of thermodynamic consistency, reproduce canonical behaviours (growth regimes, force-velocity relations, nucleotide turnover), and how Gibbs free energies tune relationships. More broadly, this thermodynamically consistent scaffold lays a foundation for predictive multiscale studies of energy consumption during cell migration and protrusion generation.

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MS161-04 — Membrane Transport and Metabolic Patterns: Mathematical Building Blocks and Data-Driven Insights

SPEAKERS Adelle Coster (University of New South Wales Sydney)
AUTHORS Adelle Coster (University of New South Wales Sydney); Catheryn Gray (University of New South Wales Sydney); Michael Pan (University of New South Wales Sydney)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Understanding nutrient transport is essential for mathematically modelling bacterial functions because transport processes govern the rates at which cells acquire substrates that fuel all downstream metabolic activity. Nutrient uptake directly shapes intracellular metabolite levels and thereby constrains metabolic pathway fluxes.

Some data driven mathematical approaches to model membrane nutrient transport and metabolic flow are explored. These model building blocks need to be scoped for different levels of biophysical specificity to enable future hybrid modelling and incorporation into whole cell models.

Techniques to identify inconsistencies between models, observations, and the data itself are discussed, and illustrate how mathematical modelling can guide experimental interpretation.

MS161-03 — The landscape reconsidered: non-equilibrium constraints on cell fate

SPEAKERS Lucy Ham (The University of Melbourne)
AUTHORS Lucy Ham (The University of Melbourne); Augustinas Sukys (University of Melbourne); Leo Diaz (University of Melbourne); Stephen Zhang (University of Melbourne); Michael P. H. Stumpf (University of Melbourne)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Cells don't roll downhill. Waddington's epigenetic landscape has shaped our thinking about cell fate for decades, inspiring rich mathematical frameworks such as quasi-potential methods, catastrophe theory and energy landscapes. These frameworks share a hidden assumption: that cellular dynamics are gradient systems, derived from a scalar potential. Whole-cell models (WCMs) allow us to test this assumption directly. We prove that chemical reaction networks with mass-action kinetics are gradient systems if and only if they satisfy detailed balance, the hallmark of thermodynamic equilibrium. But living cells are not at equilibrium. ATP hydrolysis, ion gradients, and irreversible regulatory cascades generically violate detailed balance as a result. This extends to genome-scale metabolic models, showing that flux balance analysis captures non-equilibrium steady states that lie outside the gradient framework. The landscape picture is therefore fundamentally incompatible with the biochemistry that WCMs describe.

Rather than simply rejecting landscape ideas, this perspective clarifies when gradient approximations remain useful. It points instead toward a new class of non-equilibrium descriptions, where cell fate is shaped not by descent along a potential, but by fluxes, cycles, and dynamics far from equilibrium.

MS161-05 — What would a smart yeast do? Mapping biosynthetic resource allocation to explain prevalence of the Crabtree effect in yeasts

SPEAKERS Pranas Grigaitis (Karlsruhe Institute of Technology)
AUTHORS Pranas Grigaitis (Karlsruhe Institute of Technology); Julius Battjes (Vrije Universiteit Amsterdam); Frank J. Bruggeman (Vrije Universiteit Amsterdam); Bas Teusink (Vrije Universiteit Amsterdam)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Genome-scale metabolic models (GEMs) are computable knowledge bases containing information of all biochemical reactions in an organism of interest. Typically, Flux Balance Analysis (FBA) is used to predict intracellular flux distributions corresponding to limiting substrate-efficient metabolic phenotypes. For fast-growing microbes (*Escherichia coli*, yeasts), alternative efficiency calculi (e.g., proteome efficiency) might govern their physiology in different growth regimes – inaccessible to classical FBA. Extensions of GEMs, called proteome-constrained GEMs (pcGEMs), on the contrary, capture complex metabolic phenotypes based on optimal biosynthetic resource allocation to the enzymatic machinery operating the metabolic network. We have constructed pcGEMs for yeasts *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe* to identify constraints ruling the respiration-fermentation switch in aerobic growth, also known as Crabtree effect. Both yeasts started fermenting glucose into ethanol after hitting the mitochondrial protein capacity constraint. Contrary to Crabtree+ yeasts, this constraint was never hit in a pcGEM of a Crabtree- yeast *Pichia kluyveri*. Contrasting quantitative proteomics data with pcGEM predictions hinted to *P. kluyveri* possessing more catalytically efficient electron transport chain proteins, sustaining a respiratory phenotype even at fast growth.

MS161-06 — How Do T Cells Migrate Through Collagen? Inferring Subcellular Forces from Fiber-Resolved Extracellular Matrix Deformations

SPEAKERS Jeremy Worsfold (University of New South Wales Sydney)
AUTHORS Jeremy Worsfold (University of New South Wales Sydney)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 11.01 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

Cell migration through extracellular matrix (ECM) is fundamental to many biological processes, including development, immune responses, and cancer metastasis. However, this migration is governed by mechanical interactions that occur at the subcellular scale of individual collagen fibres, and conventional traction-force methods treat the ECM as a continuum. To move beyond this, we introduce a framework that accounts for the complex global architecture of the fibrillar network to infer the subcellular-scale forces imparted by a cell as it migrates through collagen in 3D.

Our approach combines high-resolution imaging with deep-learning models for collagen-fibre skeletonization, cell segmentation, and displacement tracking. Using a mechanical model of the fibre skeleton that incorporates extensional tension, torsional stiffness at junctions, and drag forces, we solve a physics-based inverse problem to infer the fibre tensions, torques, and forces applied by the cell. This discrete network formulation naturally captures non-local, long-range mechanical interactions that existing continuum approximations cannot recover.

This framework reveals how migrating cells coordinate forces across multiple fibres to navigate complex 3D environments. More broadly, it provides new mechanistic insight into cell movement and force transmission across diverse biological contexts.

MS161-07 — Agent-based Modelling of Molecular Dynamics in the Gram-negative Outer Membrane

SPEAKERS Thomas Williams (University of Melbourne)

AUTHORS Thomas Williams (University of Melbourne); James Osborne (University of Melbourne); KJ Goh (Monash University); Trevor Lithgow (Monash University); Jennifer Flegg (University of Melbourne)

SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 11.01 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

The outer membrane of Gram-negative bacteria is a crucial defensive structure, conferring - among other properties - substantial protection against antibiotics. Decades of high-resolution molecular dynamics modelling has provided powerful insights into the structures of the chemical species that reside in the Gram-negative outer membrane and their chemical and physical interactions. However, the extraordinary complexity of these models limits their use to microseconds of simulation time. In this talk, we introduce a coarse-grained, agent-based modelling approach which can provide insights into membrane dynamics on the seconds-to-minutes time scale of cell growth. The model reveals that spatial constraints play a key role in the growth dynamics of the Gram-negative outer membrane, demonstrates the emergence of characteristic spatial structures, and challenges the current biological dogma of how the Gram-negative outer membrane grows.

MS161-08 — Model-based integration of large-scale datasets

SPEAKERS Jan Hasenauer (Bonn Center for Mathematical Life Sciences, University of Bonn)

AUTHORS Jan Hasenauer (Bonn Center for Mathematical Life Sciences, University of Bonn)

SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 11.01 - HS

TRACK Systems Biology and Biochemical Networks

Abstract

Mechanistic mathematical models are powerful tools in modern life sciences. Similar to experimental techniques, mechanistic models enable the investigation of biological processes and hypothesis testing. Furthermore, they allow the integrative analysis of multiple datasets as well as the prediction of latent variables and future experimental outcomes.

In this talk, I will outline how mechanistic modeling can support the analysis and integration of large-scale datasets. First, I will present tailored computational methods for training large-scale mechanistic models of cellular pathways using multi-omic data. This includes recent advances in combining mechanistic modeling with machine learning approaches to enable scalable inference and the handling of qualitative measurements. Second, I will introduce methods for handling heterogeneous data types and and population-level variability. In particular, I will discuss approaches for the analysis of cell-to-cell variability using amortized inference techniques, which enable efficient inference in complex stochastic and multi-cellular models.

MS65 — Algorithmic Approaches to Biochemical Network Reduction

TRACK Systems Biology and Biochemical Networks
SCHEDULE Thursday, 16 July 2026; 15:10–16:30; 11.02 - HS
ORGANISERS Georgio Hawi (University of Sydney)

Minisymposium abstract

Biochemical reaction networks (BCRNs) form the backbone of systems biology. Effectively modeling complex biological systems requires a delicate balance between complexity and accuracy, motivating the need for rigorous, principled BCRN reduction. This minisymposium addresses the demand for systematic and algorithmic, model-independent reduction methods. Featured topics include lumping, quasi-steady-state approximations (QSSA), and Tikhonov-Fenichel reductions, with a specific focus on the parametrisation method for systems exhibiting multiple timescales.

Participants will present algorithms to non-dimensionalise and convert large reaction systems into forms amenable to formal mathematical analysis. The session explores how lumping aggregates subsets of state variables into new ‘lumped’ variables to enable exact dimensionality reduction while retaining dynamical fidelity. Furthermore, the minisymposium highlights how algebraic singular perturbation theory and coordinate-independent geometric singular perturbation theory (GSPT) provide a robust framework for reducing multiscale BCRNs without relying on rigid, heuristic timescale separations. Finally, the minisymposium examines the total quasi-steady-state approximation (tQSSA) for deterministic and stochastic networks, alongside applications in pharmacokinetics. By replacing intuition with algorithmic rigor, these methods yield mathematically sound reductions that enhance both analytical insight and computational tractability.

MS65-01 — Rigorous and Automated Non-dimensionalisation, Preprocessing, and Reduction of Biochemical Reaction Networks

SPEAKERS Georgio Hawi (University of Sydney)
AUTHORS Georgio Hawi (University of Sydney); Martin Wechselberger (University of Sydney)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Mathematical models provide a powerful framework for analysing the dynamics of biochemical reaction networks (BCRNs); however, effective modelling of complex biological systems requires balancing complexity and accuracy. As such, the rigorous and principled reduction of BCRNs via systematic, model-independent methods, while ensuring that key dynamics are preserved, is of vital importance.

In this talk, we discuss how to automate the fast yet rigorous non-dimensionalisation, preprocessing, and reduction of BCRNs, without being limited to low-dimensional models, as many current approaches are. Using the parametrisation method, the constructive counterpart to Tikhonov–Fenichel theory, together with techniques from algebraic geometry and linear algebra, we automate the end-to-end, principled reduction of a wide class of BCRNs without dimensional constraints. Importantly, the true power of the parametrisation method lies in its ability to deal with systems exhibiting multiple timescales, where

standard geometric singular perturbation theory approaches can fail. We demonstrate the approach by rigorously reducing numerous well-known and real-world BCRNs, many of which we reduce for the first time, establishing its utility and showing that even low-order reduced models can achieve high fidelity and accuracy.

MS65-02 — Tikhonov-Fenichel Reductions: A Systematic Approach to Timescale Separation and its Application to Modelling Population Dynamics

SPEAKERS Johannes Apelt (Universität Greifswald)
AUTHORS Johannes Apelt (Universität Greifswald)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Modelling biological systems imposes the challenge to balance realism and complexity. Precise mechanistic models are often difficult to analyse and thus yield little insight, whereas simpler conceptual models tend to lack justification, as they rely on implicit descriptions of the underlying mechanisms. Model reduction via timescale separation can mitigate this trade-off by allowing to derive models of the latter type from the former while essential properties remain unchanged.

Tikhonov-Fenichel reduction theory developed by Goeke and Walcher [GW14] allows us to find all timescale separations of rates (i.e. slow-fast separations of processes) for polynomial ODE systems algorithmically and to compute the corresponding reductions [GWZ15]. Therefore this is a systematic, coordinate-free approach to geometric singular perturbation theory, which can be readily applied using the Julia package TikhonovFenichelReductions.jl [Ape26].

Following [AL25], the starting point of this talk is a detailed "super model" describing the population dynamics of mutualistic partners. From this we consider embedded conceptual models derived via Tikhonov-Fenichel reduction theory. The focus lies on the relationship between the mathematical formalism and the underlying biology resulting in good interpretability of the reductions. This for instance enables us to answer the question under which circumstances mutualism may break down.

References

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MS65-03 — Lumping of Reaction Networks: Generic and Critical Parameters

SPEAKERS Santiago Schnell (Department of Mathematics, Dartmouth College, Hanover, NH, USA)
AUTHORS Justin Eilertsen (Mathematical Reviews, American Mathematical Society, Ann Arbor, MI, USA); Santiago Schnell (Department of Mathematics, Dartmouth College, Hanover, NH, USA); Sebastian Walcher (Fachgruppe Mathematik, RWTH Aachen, Germany); Valery G. Romanovski (Faculty of Electrical Engineering and Computer Science, University of Maribor, Slovenia)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Lumping methods reduce biochemical reaction networks by aggregating species concentrations via algebraic conditions that hold globally. We study linear lumping for parameter-dependent mass action systems, asking how lumpability depends on rate constants. Our first result shows that for generic parameters—those ranging over a non-empty open subset of parameter space—exact linear lumping yields only "obvious" reductions: elimination of non-reactant species or projections along stoichiometric first integrals [LR89]. This characterization extends to product-form kinetics, including Michaelis–Menten and Hill-type rate laws, and structurally explains why parameter-independent approaches such as CLUE [Ovc+21] often fail to find nontrivial reductions.

Beyond generic parameters, we develop an algorithmic approach to identify critical parameter sets—algebraic subvarieties where non-trivial lumpings become available. Determining critical parameters reduces to solving finitely many polynomial systems, and extends naturally to constrained lumping. For proper lumpings of quadratic systems, covering all networks with at most bimolecular reactions, we provide a complete characterization via block-structured Jacobian conditions. Applications to a self-replicator model [GP20] and a two-pathway enzyme mechanism show that critical parameters reveal hidden conservation laws, with approximate lumpings available near these values [Leg+24].

References

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MS65-04 — Total QSSA: Pharmacokinetic Applications and the Validity of Its Stochastic Extension

SPEAKERS Yun Min Song (Institute for Basic Science)
AUTHORS Yun Min Song (Institute for Basic Science)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

For over a century, the Michaelis–Menten (MM) equation has provided the foundation for modeling enzyme kinetics in biochemistry and pharmacology. Despite its remarkable success, MM relies on assumptions that may break down in physiologically relevant regimes. In this talk, we revisit these limitations and introduce the total quasi-steady-state approximation (tQSSA) as a principled generalization of MM. We show that this framework enables more accurate quantitative predictions in pharmacokinetic applications, including enzyme-mediated drug–drug interaction modeling relevant to regulatory practice. We further discuss how this deterministic reduction extends to stochastic simulations. By clarifying the validity conditions of its stochastic extension, we identify when tQSSA reliably captures intrinsic biochemical noise and propose alternative stochastic reaction functions when it does not.

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MS103 — Behaviour and Individuality in Population Level Epidemiological Models

TRACK Mathematical Epidemiology
SCHEDULE Thursday, 16 July 2026; 15:10–18:20; 15.11 - HS
ORGANISERS Iain Moyles (York University); Jacques Bélair (Université de Montréal)

Minisymposium abstract

Classic epidemiological models typically assume that, at a population level, infection is proportional to incidence of disease and the probability of contact between those infected and those susceptible. However, human behaviour can drastically change the contact and infectious pathways of diseases altering the types of outbreaks that models can predict. Additionally, the way that individuals interact and spread diseases can alter the structure of these models, further impacting the solution structure. This session will examine how the inclusion of behaviour and the dynamics of individuals can impact disease burden and case estimates epidemiological population models.

MS103-03 — Cross-border transmission, vaccination heterogeneity, and structural inequity during the 2025 measles outbreak in Chihuahua, Mexico

SPEAKERS Jorge Velasco (UNAM)
AUTHORS Jorge Velasco (UNAM)
SCHEDULE Thursday, 16 July 2026; 15:10–15:50; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

In early 2025, a measles outbreak emerged in West Texas, USA, and spread into northern Mexico, with Chihuahua becoming the epicenter of Mexico’s largest measles resurgence in decades. The outbreak affected Mennonite colonies, Indigenous Rarámuri (Tarahumara) communities in the Sierra Tarahumara, and highly mobile agricultural worker populations. We analyzed municipality-level confirmed case counts in Chihuahua using a generalized logistic growth model to estimate early epidemic growth rates and characterize deviations from exponential growth. Uncertainty was quantified by bootstrap resampling under a negative binomial observation model. We linked epidemic dynamics to structural deprivation using hierarchical clustering based on poverty municipal indicators. To conceptualize cross-border coupling under heterogeneous immunization, we summarize a two-population vaccination model.

MS103-01 — The effectiveness of non-pharmaceutical interventions against COVID-19

SPEAKERS Jane Heffernan (York University)
AUTHORS Jane Heffernan (York University)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

We have developed and age and immunity-structured model of COVID-19 infections and interventions. We have employed this model to assess the effectiveness of vaccination and various non-pharmaceutical interventions (school closure, work-from-home, closing of public places) in reducing COVID-19 spread and the healthcare demand. In this presentation, we will share our results for Canada (Ontario), USA (Utah, Washington, New York), Sweden, Hungary, and Italy.

MS103-02 — Infectious disease surveillance using deep learning models

SPEAKERS Bouchra Nasri (Université de Montréal)
AUTHORS Bouchra Nasri (Université de Montréal)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

Social media data has become increasingly used to monitor infectious diseases. This presentation will showcase case studies on Mpox and Lyme disease, demonstrating how social media data can support the prediction of cases and other disease-related characteristics.

MS103-05 — Qualitative impact of political and economic decisions on PrEP users under HIV epidemic control

SPEAKERS Laurent Pujo-Menjouet (Lyon 1 University)
AUTHORS Abdennasser Chekroun (University of Tlemcen); Grégoire Ranson (université Claude Bernard Lyon 1, Institut Camille Jordan, Laboratory for Industrial and Applied Mathematics, Department of Mathematics and Statistics, York University); Jianhong Wu (York University); Julien Molina (université Claude Bernard Lyon 1, Institut Camille Jordan,); Laurent Pujo-Menjouet (Lyon 1 University); Mostafa Adimy (Inria Lyon, Institut Camille Jordan, Lyon, France)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

The HIV/AIDS epidemic remains a major global health challenge, with no definitive cure currently available. Following the World Health Organization's 2014 recommendations, a preventive treatment known as Pre-Exposure Prophylaxis (PrEP) has been adopted worldwide, introduced in France in 2016, to curb HIV transmission. We propose here a new compartmental epidemiological model that explicitly accounts for the limited duration of PrEP's protective effect. The PrEP compartment is described by an age-structured hyperbolic equation, while initiation of PrEP use is governed by a differential equation. Together, these lead to a nonlinear differential–difference system with discrete delays. We analyze both local stability and global dynamics of the system. Using numerical simulations informed by data from the French population of men who have sex with men (MSM), we validate the accuracy of our model. In particular, we show that combining logistic time dynamics with a Hill-type function yields an excellent fit to the available data. The model is further extended by incorporating a variable force of infection among PrEP users, influenced not only by the size of the infected population but also by political and economic factors. This reflects the reality that socioeconomic constraints can restrict access to PrEP for part of the population. Finally, we investigate epidemic dynamics under a non-monotonic recruitment rate. Altogether, these results provide new insights into the potential trajectory of the HIV epidemic in France, under the assumption of sustained PrEP uptake within the population.

MS103-04 — From within-host to between-hosts models via a virtual cohort

SPEAKERS Julien Arino (University of Manitoba)
AUTHORS Julien Arino (University of Manitoba)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

I will present a methodology that maps within-host dynamics of infection into an age-of-infection model. The one-directional link is achieved using a virtual cohort of individuals through which we gather information about disease characteristics at the population level namely, age-of-infection dependent transmission, recovery and death rates.

MS103-06 — A Basic Reproduction Number for Pair Formation Models with Co-Infection

SPEAKERS Iain Moyles (York University)
AUTHORS Iain Moyles (York University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

The basic reproduction number is a standard tool in disease modelling for determining the conditions under which a disease free equilibrium goes unstable and produces an epidemic. One popular procedure for determining this is the next generation matrix method. When this method is applied to models with pair formation, it is able to determine the stability threshold for the disease free equilibrium, but does not accurately compute the basic reproduction number. In this talk we show how to reconcile this by adapting the method to pair formation models. We extend this to the case where there are multiple diseases to consider via co-infection. We demonstrate that our modified next generation method recovers the basic reproduction number in a mathematical model of coinfection with mpox and HIV.

MS103-07 — Instabilities in a disease model incorporating compliance

SPEAKERS Jacques Bélair (Université de Montréal)
AUTHORS Jacques Bélair (Université de Montréal)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.11 - HS
TRACK Mathematical Epidemiology

Abstract

Management of the COVID-19 pandemic required in its early stages the deployment of non pharmaceutical interventions (NPIs) [social isolation, physical distancing, mask-wearing, hand-washing]. We analyse a simple model to illustrate the consequences, for the evolution of the disease, of variable, dynamic individual compliance to these measures: the model divides a population in uninformed, informed-compliant and informed-noncompliant subpopulations. Conditions for the existence of multiple stable equilibria will be discussed as well as the consequences for the control of the infection.

MS131 — Recent mathematical discoveries in Population Dynamics, Ecology and Evolution

TRACK Population Dynamics, Ecology & Evolution
SCHEDULE Thursday, 16 July 2026; 15:10–11:40; 15.12 - HS, 15.11 - HS
ORGANISERS Christopher Heggerud (University of Manitoba); Judith Miller (Georgetown University); Daniel Cooney (University of Illinois at Urbana-Champaign); Shan Gao (University of Alberta)

Minisymposium abstract

Supported by the SMB Population Dynamics, Ecology and Evolution subgroup, this mini-symposium will bring together a diverse set of researchers developing and analyzing mathematical models that address timely questions in evolutionary dynamics, population biology, ecological systems, and related areas at their interfaces. This session aims to provide a collegial forum for presenting new results, encouraging discussion, and catalyzing collaboration across institutions and career stages, from early-career to senior researchers.

MS131-01 — Mathematical of pre-exposure prophylaxis and the quest to end the HIV epidemic

SPEAKERS Abba Gumel (University of Maryland)
AUTHORS Abba Gumel (University of Maryland)
SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 15.12 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The use of pre-exposure prophylaxis (PrEP), where approved antivirals are administered to uninfected high-risk individuals, is universally regarded as a promising strategy to prevent susceptible high-risk individuals from acquiring HIV infection from their infected partners. A number of antiviral drugs (and their combinations) have been developed and are being used as prophylaxis against the HIV epidemic here in the U.S. and globally. We will first present a risk-structured mathematical model for assessing the population-level impact of PrEP in an MSM (men who have sex with men) population. An extended model, which considers several high-risk populations, will also be presented and used to assess the potential spillover effect, where the administration of PrEP to individuals in one risk group induces a reduction of disease burden in other risk group(s). The central aim is to determine whether the use of the aforementioned strategies can aid the End the HIV Epidemic initiative, aimed at eliminating the disease in the U.S. by 2030.

MS131-02 — Failed rescue effect in metapopulations: when increased dispersal results in total collapse

SPEAKERS Frank Hilker (Osnabrück University)
AUTHORS Carolin Grumbach (Osnabrück University); Frank Hilker (Osnabrück University)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 15.12 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

The rescue effect is a well-known process in spatial ecology where immigration from a subsidizing subpopulation prevents a vulnerable subpopulation from going extinct. It has become a cornerstone of metapopulation theory and provides a conceptual basis for conservation management.

In this talk, we challenge the prevailing optimism surrounding dispersal. We uncover the phenomenon of the failed rescue effect, where increased dispersal drives both the source and target subpopulations toward joint extinction. We demonstrate this in a two-patch metapopulation model with explicit local population dynamics that are subject to a strong Allee effect. The failed rescue effect occurs when staggering emigration depletes the source patch while the resulting immigration remains insufficient to lift the target patch above its critical threshold. Furthermore, we refine the typology of successful rescue effects, distinguishing between sustenance rescue, which maintains a vulnerable subpopulation at low abundance, and super rescue, which boosts the subpopulation beyond its Allee threshold to a high-abundance stable state.

By integrating local non-linear dynamics with spatial coupling, our analysis reveals that metapopulations with Allee effects may be far more fragile than previously assumed, offering a novel perspective on the risks of connectedness in spatial ecology.

MS131-03 — Changing Connectivity: A Spatial Understanding of Bistable Coral Dynamics

SPEAKERS Jennifer Paige (University of California, Davis)
AUTHORS Alan Hastings (University of California, Davis; Santa Fe Institute); Ariel Greiner (University of New Hampshire); Jennifer Paige (University of California, Davis)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 15.12 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Modeling coral ecosystems in a theoretical framework typically focuses on tradeoffs between coral, algae, and grazing fish populations, highlighting its bistable dynamics; however, spatial understanding of this system is often suppressed. Analysis of models with spatial features is crucial, as degraded, patchy reef systems become more common, and the size, clustering, or overall connectivity of these patches play a key role in the persistence and the emergent dynamics. More broadly, the implications of spatial heterogeneity and connectivity in systems with alternative stable states are complex and underexplored. In this work, we explore the long term dynamics, through both numerical and mathematical analysis, of a spatial model of a coral ecosystem, extended to multiple reef patches. Our results explore the "mixed-blessing" that connectivity can have on coral metacommunities.

MS131-04 — Mathematical Models for evolutionary phenomena during range expansion

SPEAKERS Silas Poloni (Roskilde University)
AUTHORS Frithjof Lutscher (University of Ottawa); Mark Lewis (University of Victoria); Silas Poloni (Roskilde University)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 15.12 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

It has become increasingly evident that evolution can take place in ecological time scales. One such example is that of evolution of dispersal during range expansion. As individuals with higher motility get to the leading edge faster, if these individuals benefit from low competition therein, they reproduce, and their offspring are then equally able to colonize unexplored regions. Over time, individuals at the leading edge become much better at dispersal than their conspecifics at the range core, leading to faster spread of the population. This has been observed in nature for some species, most notoriously the cane toads in north Australia and red-shouldered soapberry bugs in Texas, USA, but also tested experimentally in bacteria, mites, bean bugs and thale cress. In light of such observations and the challenge they present in our understanding of range expansions, mathematical perspectives that once relied on describing population dispersal based on average phenotypes began to account for population variability in dispersal. In this talk, I plan to go over some of such models and some of our contributions, and show how evolution changes population asymptotic spreading speeds and leading edge population trait distributions during range expansion.

MS131-05 — How the Tulips get their Stripes

SPEAKERS Thomas Hillen (University of Alberta)
AUTHORS Thomas Hillen (University of Alberta)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Tulips have captivated human interest for centuries, with their vibrant colors and unique shapes. Particularly striped tulips have been highly popular, leading to the "tulipomania" in the Dutch Golden Age. But how do the tulips get their stripes? Maybe Turing can help?

MS131-07 — Impact of Network Variation on Metapopulation Dynamics

SPEAKERS Zhisheng Shuai (University of Central Florida)
AUTHORS Zhisheng Shuai (University of Central Florida)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Spatiotemporal population dynamics are often modeled using reaction–diffusion equations, yet analytical insight into the role of movement and network structure remains limited. In this talk, we present a metapopulation framework based on coupled ordinary differential equations on networks, focusing on regimes where dispersal is faster than local growth. Using tools from matrix analysis and perturbation theory, including new rank-one results for group-invertible matrices, we quantify how local changes in movement affect the dominant eigenvalue, which governs population persistence. In particular, we show that increasing symmetric connectivity in weight-balanced networks can inhibit growth under fast dispersal.

MS131-08 — When Systems Tip: Predicting Critical Transitions and Uncovering Frequency-Induced Tipping

SPEAKERS Shan Gao (University of Alberta)
AUTHORS Shan Gao (University of Alberta)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 15.11 - HS
TRACK Population Dynamics, Ecology & Evolution

Abstract

Tipping points (critical transitions) are abrupt, often irreversible shifts in dynamical systems that can be triggered by small changes. This talk tackles two questions: how to predict tipping early and why tipping occurs. In this talk, I will demonstrate how combining tipping-point ideas with machine learning can enhance early-stage outbreak prediction using limited epidemic data. I will then present a new mechanism, frequency-induced tipping, where a system can be driven to a critical transition by subthreshold periodic forcing at particular frequencies, even in the absence of noise and without crossing a static bifurcation threshold.

MS165 — Multiscale perspectives on cancer resistance: from intracellular networks to population dynamics

TRACK Mathematical Oncology
SCHEDULE Thursday, 16 July 2026; 15:10–18:20; 05.12 - HS
ORGANISERS Giada Fiandaca (Centre Inria d'Université Côte d'Azur - Team COMPO); Joon-Hyun Song (Stony Brook University); Junho Lee (Korea Institute of Science and Technology); Linh Nguyen Phuong (INRIA)

Minisymposium abstract

Cancer development and therapeutic resistance emerge from adaptive mechanisms acting across multiple biological scales, ranging from intracellular regulatory and metabolic processes to interactions among heterogeneous tumor cell populations and population-level system dynamics involving the immune system and the tumor microenvironment. This minisymposium brings together **early-career researchers** (PhD students and postdoctoral fellows) who combine mechanistic modeling with quantitative experimental and pre-clinical data to investigate cancer progression and therapy response. Contributions are organized into two sessions addressing complementary biological scales: (1) intracellular and cellular mechanisms, and (2) population-level and microenvironmental dynamics. At the **cell-intrinsic level**, models characterize regulatory, metabolic, and gene-network processes shaping cellular response. At the **intercellular level**, interactions among tumor subpopulations and phenotypic plasticity are explored as key drivers of resistance. At the **population level**, models incorporate immune dynamics and tumor microenvironment modulation

to study therapy-induced resistance, immune escape, and treatment scheduling. By showcasing data-driven, multi-scale modeling approaches explicitly confronted with experiments, this minisymposium aims to foster dialogue between theory and experiments and to promote integrative quantitative perspectives on cancer dynamics and therapeutic strategies.

MS165-01 — Mechanistically Grounded Digital Twins for Relapse Forecasting in Acute Lymphoblastic Leukemia Using Multi-Omic Data

SPEAKERS Sergio Márquez-Díaz (MOLAB (Mathematical Oncology Laboratory))

AUTHORS Carlos Torroja (National Center for Cardiovascular Research Carlos III (CNIC), Madrid 28029, Spain.); David G. Aragonés González (Mathematical Oncology Laboratory (MOLAB), Department of Mathematics, University of Castilla-La Mancha, Spain); Diego Herráez (Computational Biophysics and Biological Data Analysis, Institute for Biosanitary Research, Faculty of Experimental Sciences, Francisco de Vitoria University (UFV), Pozuelo de Alarcón, 28223 Madrid, Spain.); Elena Garcia-Sánchez (Oncohematology Unit, Niño Jesús University Children'sHospital, Madrid, Spain.); Francisco Monroy (Department of Physical Chemistry, Complutense University of Madrid, Ciudad Universitaria, 28040 Madrid, Spain.); Gabriel Calvo (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain); Manuel Ramírez-Orellana (Oncohematology Unit, Niño Jesús University Children'sHospital, Madrid, Spain.); Ramón Ramírez Torija (Mathematical Oncology Laboratory (MOLAB), Department of Mathematics, University of Castilla-La Mancha, Spain); Sergio Márquez-Díaz (MOLAB (Mathematical Oncology Laboratory))

SCHEDULE Thursday, 16 July 2026; 15:10–15:30; 05.12 - HS

TRACK Mathematical Oncology

Abstract

Relapse in acute lymphoblastic leukemia (ALL) remains a clinical challenge, affecting 15% of pediatric patients despite effective therapies. Understanding the population dynamics underlying treatment response and resistance is central in mathematical oncology.

Within the LEUKODOMICS project, we develop a stochastic population framework to model leukemic cell dynamics under multi-drug therapy. The model is a discrete probabilistic process describing proliferation, natural death, and drug-induced cytotoxicity, incorporating multiple leukemic subpopulations with distinct resistance profiles. Resistance to different therapeutic agents can evolve independently during treatment, following the SEHOP-PETHEMA 2013 pediatric protocol.

This approach enables simulation of heterogeneous clonal dynamics and exploration of relapse as an evolutionary and stochastic process. The framework integrates clinical and molecular information, supporting patient-specific digital twins capable of reproducing individual disease trajectories.

Mechanomic variables from chromatin motion analysis provide insights on nuclear organization and cellular mechanical behavior. These features, alongside transcriptomic, proteomic, and clinical data, are being evaluated to refine model parameterization.

Overall, stochastic multi-population models combined with multi-scale data support predictive modeling of leukemia progression and the development of digital twins for precision pediatric oncology.

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MS165-02 — Co-expression Networks in Cancer

SPEAKERS Britta Daub (MAX-PLANCK-INSTITUT)
AUTHORS Bernadette Stolz (Max-Planck-Institut for Biochemestry, Am Klopferspitz 18, 82152 Martinsried, Germany); Britta Daub (MAX-PLANCK-INSTITUT); Daniel Roggenkamp (Max-Planck-Institut for Mathematics in the Sciences, Inselstraÿe 22, 04103 Leipzig, Germany)
SCHEDULE Thursday, 16 July 2026; 15:30–15:50; 05.12 - HS
TRACK Mathematical Oncology

Abstract

Recent advancements in high throughput RNA sequencing technologies have generated unprecedented amounts of high-dimensional genomic data, enabling more detailed analysis. Techniques from network science are widely used to analyze such data, but face significant challenges. One shortcoming of these methods is that many of them use thresholding. The lack of consensus regarding the method for choosing the threshold value leads to substantial variability in downstream results and biological interpretations. Furthermore, many networks are constructed based on pairwise relationships between genes, disregarding potential contributions of higher order interactions. We explore Persistent Homology, a key method from Topological Data Analysis that quantifies topological features across multiple scales and joint cumulants, that capture higher order dependencies, in order to address these limitations. We investigate advantages of using hypergraphs as a framework for modeling higher order interactions and the concept of Structural Balance as a method for incorporating edge sign information. We will present preliminary results.

MS165-03 — Mathematical Modeling of Prostate Growth for Digital Twin-Based Monitoring in Active Surveillance

SPEAKERS Valentin Schmid (Heidelberg Institute for Theoretical Studies HITS gGmbH)
AUTHORS Isabel Gernand (Heidelberg Institute for Theoretical Studies (HITS), Germany); Valentin Schmid (Heidelberg Institute for Theoretical Studies HITS gGmbH); Vincent Heuveline (Heidelberg Institute for Theoretical Studies (HITS) and Engineering Mathematics and Computing Lab, IWR, Heidelberg University)
SCHEDULE Thursday, 16 July 2026; 15:50–16:10; 05.12 - HS
TRACK Mathematical Oncology

Abstract

Active surveillance is a common management strategy for patients with low-risk prostate cancer, aiming to delay or avoid invasive treatment while monitoring disease progression. However, interpreting longitudinal observations is challenging because the prostate itself can undergo significant physiological growth due to aging or benign prostatic hyperplasia. These baseline changes may obscure early indicators of clinically relevant disease progression. We present a mathematical framework for modeling prostate organ growth as a first step toward patient-specific digital twins for monitoring prostate cancer under active surveillance. Longitudinal magnetic resonance imaging (MRI) data are processed using automated segmentation methods to extract prostate geometries and estimate organ volumes at multiple time points. These imaging-derived measurements are combined with clinical variables such as prostate-specific antigen (PSA) levels. To describe the temporal evolution of the prostate, we employ a growth model based on partial differential equations that captures the gradual expansion of prostate tissue. Model parameters are estimated from longitudinal imaging data to characterize patient-specific growth trajectories. By explicitly accounting for benign organ growth, the model provides a baseline against which potential tumor-related changes can be assessed, supporting improved interpretation of surveillance data and the development of predictive digital twins.

MS165-04 — Modeling resistance multiverse: from metabolic activity to immune evasion

SPEAKERS Malgorzata Tyczynska Weh (MD Anderson Cancer Center)
AUTHORS Malgorzata Tyczynska Weh (MD Anderson Cancer Center)
SCHEDULE Thursday, 16 July 2026; 16:10–16:30; 05.12 - HS
TRACK Mathematical Oncology

Abstract

The words “resistance” and “relapse” are used interchangeably, and statements such as “treatment resistance remains a critical limitation to the success of cancer therapy” are flooding the introductions of research papers in (mathematical) oncology. Is this practice correct and appropriate? Does it matter in the clinic?

In this talk, I will contrast my preclinical research studies in targeted therapy (Non-Small Cell Lung Cancer) and radioimmunology (HPV+ Head & Neck cancer) to demonstrate that the distinction between resistance and relapse is not just semantics. A clear, measurable definition of resistant disease and how it’s different from therapy failure can change the disease management plan and outcomes.

Central to my message are the definitions of the terms “treatment resistance” and “treatment failure”. I will show how different mathematical and statistical models skew our perception of cancer treatment modeling, and what can be done to address such limitations. My conclusion is the word of caution and advocacy for a combination - a multiverse - of imperfect, yet complementary models to address the most urgent clinical needs in oncology.

MS165-05 — Modeling phenotypic switch in cancer drug response to identify new drug combinations

SPEAKERS Marielle Pere (Marseille Medical Genetics)
AUTHORS Benjamin Bian; Bernard Mari; George Vassaux; Jérémie Roux; Ludovic Peyre; Marielle Pere (Marseille Medical Genetics); Marina Moureau-Barbato; Mickael Meyer; Walid Djema
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 05.12 - HS
TRACK Mathematical Oncology

Abstract

The emergence of drug-tolerant cells in clonal cancer populations is the main cause of reduced cytotoxic drug efficacy. But these tolerant cells can also be used as a specific target to design drug combinations. In this study [Pey+25], we investigate the response of clonal cancer cells to pro-apoptotic and pro-necroptotic drugs. In both cases, fractional killing increases upon repeated administration. However, transcriptomic analysis reveals that pro-apoptotic-tolerant cells resemble pro-necroptotic-sensitive cells, suggesting that alternating treatments could mitigate this effect, which we validate experimentally. We then present a population model of drug response phenotypic switch, based on ODEs, to understand the re-sensitization of tolerant cells. The model is calibrated independently for each drug then coupled into a unified system simulating transitions between the tolerant, apoptosis-sensitive and necroptosis-sensitive states. We demonstrate that cells adapt to treatment by modulating drug retention time via regulation of drug degradation rates and reveal that the majority of pro-apoptotic-tolerant cells are in fact sensitive to necroptosis; pro-apoptotic treatment only accelerating the transition from the tolerant state toward necroptotic sensitivity.

References

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MS165-06 — From Black Board, to Plate, to Patient: Dissecting Resistance Dynamics to Personalise Cancer Treatment Scheduling

SPEAKERS Maximilan Strobl (The Institute of Cancer Research and Imperial College London)
AUTHORS Dagim Tadele (Department of Medical Genetics, Oslo University Hospital, Oslo, Norway); Jacob Scott (Department of Genomic Medicine, Cleveland Clinic Research, Cleveland, USA); Jinling Wu (Department of Genomic Medicine, Cleveland Clinic Research, Cleveland, USA); Maximilan Strobl (The Institute of Cancer Research and Imperial College London); Mina Dinh (Department of Genomic Medicine, Cleveland Clinic Research, Cleveland, USA); Ruben Perez-Carrasco (Department of Life Sciences, Imperial College, London, UK); Trevor Graham (Institute of Cancer Research)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 05.12 - HS
TRACK Mathematical Oncology

Abstract

Systemic therapies have revolutionised our ability to treat metastatic cancer. However, improvements are all too often temporary due to compounding toxicity and the emergence of drug-resistance. Most drugs are given according to a one-size-fits all approach and only changed upon toxicity or progression. In this talk, I will present work across two different spatial-temporal scales in which we ask: can we improve outcomes by personalising treatment scheduling? In the first part, I will discuss integration of in vitro experiments and mathematical modelling to study the impact of drug scheduling on cancer evolution. By treating fluorescent co-cultures of sensitive and resistant cells with four different treatment schedules (Continuous Therapy, Intermittent Therapy, Low-Dose Continuous Therapy), we show that intermittent scheduling can slow drug resistance and that cell plasticity plays an important role in shaping the treatment dynamics. In the second part, I will present recent work in which we are asking what we can learn about resistance dynamics in patients from routinely collected tumour burden data. I will present a Bayesian model selection framework with which we dissect the response dynamics of metastatic colorectal cancer patients by comparing different evolutionary hypotheses. Optimising drug scheduling is a key aim of mathematical oncology, and I hope to convince you of the opportunities – and importance - of studying this problem at multiple scales.

MS165-07 — How Modulation of the Tumor Microenvironment Drives Cancer Immune Escape Dynamics

SPEAKERS Pujan Shrestha (Texas A&M University)
AUTHORS Jason T. George (Texas A&M University, Houston, TX, USA ; Rice University, Houston, TX, USA); Pujan Shrestha (Texas A&M University); Zahra S. Ghoreyshi (Department of Biomedical Engineering, Texas A&M University, College Station, TX 77843 Translational Medical Sciences, Texas A&M Health Science Center, Houston, TX 77030)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 05.12 - HS
TRACK Mathematical Oncology

Abstract

Metastatic disease is the leading cause of cancer-related death, despite recent advances in therapeutic interventions. Prior modeling approaches have accounted for the adaptive immune system's role in combating tumors, which has led to the development of stochastic models that explain cancer immunoediting and tumor-immune co-evolution. However, cancer immune-mediated dormancy, wherein the adaptive immune system maintains a micrometastatic population by keeping its growth in check, remains poorly understood. Immune-mediated dormancy can significantly delay the emergence (and therefore detection) of metastasis. An improved quantitative understanding of this process will thereby improve our ability to identify and treat cancer during the micrometastatic period. Here, we introduce a generalized stochastic model that incorporates the dynamic effects of immunomodulation within the tumor microenvironment on T cell-mediated cancer killing. This broad class of nonlinear birth-death model can account for a variety of cytotoxic T cell immunosuppressive effects, including regulatory T cells, cancer-associated fibroblasts, and myeloid-derived suppressor cells. We develop analytic expressions for the likelihood and mean time of immune escape. We also develop a method for identifying a corresponding diffusion approximation applicable to estimating population dynamics across a wide range of nonlinear birth-death processes. Lastly, we apply our model to estimate the nature and extent of immunomodulation that best explains the timing of disease recurrence in bladder and breast cancer patients. Our findings quantify the effects

that stochastic tumor-immune interaction dynamics can play in the timing and likelihood of disease progression. Our analytical approximations provide a method of studying population escape in other ecological contexts involving nonlinear transition rates.

MS165-08 — Integrating glioblastoma plasticity into combination therapy strategies to overcome therapeutic resistance: a quantitative systems pharmacology approach

SPEAKERS Thibault Delobel (Institut Marie Curie)
AUTHORS Ahmed Idbaih (Sorbonne Université, AP-HP, Institut du Cerveau - Paris Brain Institute - ICM, Inserm, CNRS, Hôpitaux Universitaires La Pitié Salpêtrière - Charles Foix, DMU Neurosciences, Service de Neurologie 2-Mazarin, France); Annabelle Ballesta (Institut Curie, INSERM U1331, Cancer Systems Pharmacology team); Maité Verreault (Paris Brain Institute, Inserm UMR 1127, Hopital Pitié Salpetrière AP-HP, France); Sergio Corridore (Servier, data science and data management unit, France); Thibault Delobel (Institut Marie Curie)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 05.12 - HS
TRACK Mathematical Oncology

Abstract

Glioblastoma (GBM) is the most aggressive primary brain tumor in adults, with a median survival between 14-21 months and no curative treatment currently available. To investigate resistance mechanisms to temozolomide (TMZ), the standard-of-care chemotherapy, we generated perturbed proteomic data (with and without TMZ) for 12 patient-derived cell lines (PDCLs). Pathways enrichment and independent component analysis revealed a high inter-patient heterogeneity. Proteins linked to TMZ response were identified and matched to pharmacological compounds using a new pipeline, leading to 40 promising drugs from an initial screening. These candidates are currently evaluated in a second screening to assess synergistic effects. The next step is to build a digital twin for each PDCL, enabling personalized prediction of combination therapies. A previously developed quantitative systems pharmacology (QSP) model of TMZ pharmacokinetics-pharmacodynamics serves as a foundation. To initiate model individualization, we developed a method to personalize model parameters using publicly available multi-omics and TMZ cytotoxicity data. Current work focuses on integrating proteomics-derived key species into the core model to obtain PDCL-specific digital twins and infer personalized treatment by combining QSP and machine learning. This integrative approach, combining data analysis, network reconstruction, and mechanistic modeling, opens the path for efficient patient specific therapies in GBM.

MS44 — Data-Driven Modeling of CAR T-Cell Dynamics in cancer

TRACK Mathematical Oncology
SCHEDULE Thursday, 16 July 2026; 17:00–18:20; 15.04 - HS
ORGANISERS LUCIANA RODRIGUES CARVALHO BARROS (Center for Translational Research in Oncology, Hospital das Clínicas Fmusp, Brazil); Soukaina Sabir (UC3M)

Minisymposium abstract

Chimeric Antigen Receptor (CAR) T-cell therapy has emerged as a transformative approach in cancer treatment, particularly for hematological malignancies, and is increasingly explored for solid tumors. Patient responses, however, are highly variable, reflecting the complexity of tumor-immune interactions, antigen heterogeneity, immune exhaustion, and microenvironmental influences.

This minisymposium focuses on mathematical and data-driven modeling approaches that aim to understand, predict, and optimize CAR T-cell therapy. Mechanistic and computational models, informed by clinical and experimental data, provide quantitative insights into tumor and CAR T-cell dynamics, the emergence of resistance, and the factors that influence therapeutic efficacy. By integrating theoretical and empirical perspectives, these models offer guidance for treatment design, biomarker identification, and personalized therapy strategies.

Attendees will gain a comprehensive view of how mathematical and data-driven modeling can support the development and optimization of CAR T-cell therapy, bridging the gap between quantitative theory and clinical application.

MS44-01 — Spatiotemporal dynamics of tumor - CAR T-cell interaction

SPEAKERS Ivana Bozic (University of Washington)
AUTHORS Ivana Bozic (University of Washington)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

The success of chimeric antigen receptor (CAR) T-cell therapy in treating hematologic malignancies has generated widespread interest in translating this technology to solid cancers. However, issues like tumor infiltration, the immunosuppressive tumor microenvironment, and tumor heterogeneity limit its efficacy in the solid tumor setting. Recent experimental and clinical studies propose local administration directly into the tumor or at the tumor site to increase CAR T-cell infiltration and improve treatment outcomes. We develop a simplified spatiotemporal model for CAR T-cell treatment of solid tumors and demonstrate that the model can reproduce tumor and CAR T-cell data from small imaging studies of local administration of CAR T cells in mouse models. Our results suggest that locally administered CAR T cells will be most successful against slowly proliferating, highly diffusive tumors. In our simulations, low average tumor cell density is a better predictor of treatment success than total tumor burden or volume doubling time. These findings affirm the clinical observation that CAR T cells will not perform equally across different types of solid tumors, and suggest that measuring tumor density may be helpful when considering the feasibility of CAR T-cell therapy and planning dosages for a particular patient.

MS44-02 — Predicting CAR T-cell Therapy Outcome and Cytokine Release Syndrome Through Data-Driven Mathematical Models

SPEAKERS Luciana Barros (Universidade de São Paulo)
AUTHORS Luciana Barros (Universidade de São Paulo)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Chimeric Antigen Receptor (CAR) T-cell therapy represents a frontier in treating leukemias and lymphomas, with recent clinical approvals worldwide. Despite its potential, therapeutic outcomes remain heterogeneous due to factors such as limited in vivo persistence, impaired migration, exhausted phenotypes, and tumor-mediated immunosuppression. Mathematical modeling offers a robust framework to predict these outcomes and elucidate biological mechanisms that are otherwise inaccessible through direct measurement. Our group has developed models incorporating diverse CAR T-cell phenotypes—including effector, exhausted, and memory cells—calibrated with data from public clinical trials. Furthermore, our models account for the roles of healthy B-cells and macrophages in clinical outcomes and the development of Cytokine Release Syndrome (CRS). Finally, we analyze antigen expression dynamics as a proxy for predicting antigen-positive and antigen-negative relapses. Overall, our multiscale models integrate in vitro and in vivo data to enhance the predictive understanding of CAR T-cell therapy in hematological malignancies.

MS44-03 — Quantifying target antigen-dependent CAR T-cell performance against AML

SPEAKERS Philipp Altrock (Cancer Modeling & Evolution UKSH Campus Kiel)
AUTHORS Philipp Altrock (Cancer Modeling & Evolution UKSH Campus Kiel)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.04 - HS
TRACK Mathematical Oncology

Abstract

Antigen Receptor (CAR) T-cell therapy has transformed cancer immunotherapy by genetically engineering T-cells to target tumor antigens. Acute myeloid leukemia (AML) presents unique challenges due to resistance mechanisms, especially in patients with TP53 loss mutations. The complex dynamics of CAR T-cell expansion remain poorly understood. The field lacks validated quantitative frameworks to systematically evaluate different CAR T-cell target constructs, such as CD33, CD123, and CD371, against resistant AML variants. We address this gap by combining mathematical modeling with in vitro assay data and Bayesian inference. We select, train, and validate a two-compartment deterministic mathematical model that describes the nonlinear dynamics of target AML and CAR T cells, accounting for expansion, killing, and exhaustion. Using Bayesian inference, we train and select the best-performing functional form for CAR T expansion, and then validate it on unseen data. Our framework selects a model that accounts for handling time and T-cell self-interference. Thus, expansion is a complex and dynamic process in which the handling time of target cells and T-cell crowding negatively affect T-cell expansion. Analysis of posterior parameter distributions reveals target-antigen-specific responses against TP53-deficient AML. For instance, CD33-targeting CARs have reduced attack rates against TP53-deficient cells, while CD123- and CD371-targeting CARs show moderately increased attack rates; however, the former exhibit higher death rates, and the latter have increased handling times, impeding efficacy. This target-dependent form of resistance challenges the assumption of uniform performance and reveals a unifying nonlinear expansion model for integrated, yet antigen-specific, experimental and theoretical predictions of efficacy.

MS44-04 — Synergistic interplay of morphology and metabolic activity rule response to CAR T-cells in B-cell lymphomas

SPEAKERS Soukaina Sabir (UC3M)
AUTHORS Soukaina Sabir (UC3M)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.04 - HS
TRACK Mathematical Oncology

Abstract

CAR T-cell therapy, based on genetically engineered T-cells, has demonstrated significant potential in treating hematological malignancies, including B-cell lymphomas. This treatment has complex longitudinal dynamics due to the interplay of different T-cell phenotypes (e.g. effector and memory), the expansion of the drug and the cytotoxic effect on both normal and cancerous B-cells, the exhaustion of the immune cells, the tumor immunosupressive environments, and more. Thus, the outcome of the therapy is not yet well understood leading to a variety of responses ranging from sustained complete responses, different types of partial responses, or no response at all. We developed a mechanistic model for the interaction between CAR T- and cancerous B-cells, accounting for the role of the tumor morphology and metabolic status. The simulations showed that lesions with irregular shapes and high proliferation could contribute to long term progression by potentially increasing their immunosuppressive capabilities impairing CAR T-cell efficacy. We analyzed 18F-FDG PET/CT imaging data from 63 relapsed/refractory diffuse large B-cell lymphoma receiving CAR T-cells, quantifying radiomic features including tumor sphericity and lesion aggressiveness through standardized uptake values (SUV). Statistical analyses revealed significant correlations between these metrics and progression-free survival (PFS), emphasizing that individual lesions with complex morphology and elevated metabolism play a critical role in shaping long-term treatment outcomes. We demonstrated the potential of using data-driven mathematical models in finding molecular-imaging based biomarkers to identify lymphoma patients treated with CAR T-cell therapy having higher risk of disease progression.

MS57 — Temporal Fluctuations in the Tumor Microenvironment and Their Influence on Cancer Progression

TRACK Mathematical Oncology
SCHEDULE Thursday, 16 July 2026; 17:00–18:20; 62.01 - HS

ORGANISERS Gopinath Sadhu (Indian Institute of Science Bangalore); Jason Thomas George (Texas A&M University)

Minisymposium abstract

Tumor microenvironmental factors change over time, and these factors include hypoxia and the extracellular matrix (ECM). Experimental studies demonstrated that oxygen levels fluctuate between normoxic and hypoxic levels, a phenomenon known as cyclic hypoxia. This fluctuation facilitates intratumoral cellular heterogeneity. However, how cyclic hypoxia shapes the landscape of tumor invasion is not yet fully understood. The ECM also plays a crucial role in tumor invasion and the infiltration of immune cells. Numerous studies reported that ECM density evolves as a dynamic property. It is essential to investigate how the temporal heterogeneity of the ECM influences tumor progression and immune infiltration. Mathematical models and other computational tools have become an essential tool for describing the evolutionary dynamics of cancer, from agent-based models to partial differential equations. Assessing how temporal variations in tumor microenvironmental factors influence phenotypic switching, invasion dynamics, and immune infiltration is crucial for understanding the complete picture of cancer progression.

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MS57-01 — Exploring the connection between hypoxia and intra-tumour heterogeneity through mathematical modelling

SPEAKERS Giulia Celora
AUTHORS Giulia Celora
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

In solid tumours, the presence of regions of abnormally low oxygen levels (i.e., hypoxia) is recognised as a major driver of tumour progression and therapeutic resistance. Yet, how exactly oxygen levels shape cancer cell responses and disease dynamics in vivo remains poorly understood. While in vitro models of hypoxia exist, they often fail to capture the complex oxygenation dynamics of real tumours. In this talk, I will discuss how mathematical modelling can be used to address the gap between in vitro and in vivo experimental conditions. As an example, I will present our work on cell-cycle dysregulation in cancer cells exposed to fluctuating oxygen environments – namely cyclic hypoxia. In this work, we have integrated mechanistic mathematical modelling and cell culture experiments to explore and predict cell responses to a wide range of (cyclic) hypoxia conditions. Our results uncover the possible multifaced role of hypoxia in shaping the heterogeneous composition of vascularized tumours.

MS57-02 — The role of hypoxic memory on tumor invasion under cyclic hypoxia

SPEAKERS Gopinath Sadhu (Indian Institute of Science Bangalore)
AUTHORS Gopinath Sadhu (Indian Institute of Science Bangalore)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Tumor growth and angiogenesis drive complex spatiotemporal variation in micro-environmental oxygen levels. Previous experimental studies have observed that cancer cells exposed to chronic hypoxia retained a phenotype characterized by enhanced migration and reduced proliferation, even after being shifted to normoxic conditions, a phenomenon which we refer to as hypoxic memory. However, because dynamic hypoxia and related hypoxic memory effects are challenging to measure experimentally, our understanding of their implications in tumor invasion is quite limited. Here, we propose a novel phenotype-structured partial differential equation modeling framework to elucidate the effects of hypoxic memory on tumor invasion along one spatial dimension in a cyclically varying hypoxic environment. We incorporated hypoxic memory by including time-dependent changes in hypoxic-to-normoxic phenotype transition rate upon continued exposure to hypoxic conditions. Our model simulations demonstrate that hypoxic memory significantly enhances tumor invasion without necessarily reducing tumor volume. This enhanced invasion was sensitive to the induction rate of hypoxic memory, but not the dilution rate. Further, shorter periods of cyclic hypoxia contributed to a more heterogeneous profile of hypoxic memory in the population, with the tumor front dominated by hypoxic cells that exhibited stronger memory. Overall, our model highlighted the complex interplay between hypoxic memory and cyclic hypoxia in shaping heterogeneous tumor invasion patterns.

MS57-03 — Population-level and agent-based models for a quantitative understanding of the tumor-immune interaction

SPEAKERS Jason Thomas George (Texas A&M University)
AUTHORS Jason Thomas George (Texas A&M University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Cancer populations evolve in response to dynamic microenvironments, and the adaptive immune system responds by attempting to recognize and eliminate cancer cells by targeting tumor-associated antigens. The interplay between an evading cancer and recognizing immune compartment is quite dynamic and results in cancer elimination or ultimate immune escape preceded by a period of sustained equilibrium. In this talk, I will discuss our group's efforts at understanding the role of the adaptive immune system, surrounding extracellular matrix, and alterations in immune activity in contributing to ultimate tumor escape or elimination. This work introduces both population dynamics and agent-based modeling frameworks to better understand the resultant co-evolution of cancer populations.

MS57-04 — Growth and adaptation mechanisms of tumour spheroids with time-dependent oxygen availability

SPEAKERS Ryan Murphy (Adelaide University)
AUTHORS Ryan Murphy (Adelaide University)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Tumours are subject to external environmental variability. However, in vitro tumour spheroid experiments, used to understand cancer progression and develop cancer therapies, have been routinely performed for the past fifty years in constant external environments. Furthermore, spheroids are typically grown in ambient atmospheric oxygen (normoxia), whereas most in vivo tumours exist in hypoxic environments. Therefore,

there are clear discrepancies between in vitro and in vivo conditions. We explore these discrepancies by combining tools from experimental biology, mathematical modelling, and statistical uncertainty quantification. Focusing on oxygen variability to develop our framework, we reveal key biological mechanisms governing tumour spheroid growth. Growing spheroids in time-dependent conditions, we identify and quantify novel biological adaptation mechanisms, including unexpected necrotic core removal, and transient reversal of the tumour spheroid growth phases.

MS77 — Mechanistic Model Inference for Stochastic Single-Cell Dynamics

TRACK Systems Biology and Biochemical Networks
SCHEDULE Thursday, 16 July 2026; 17:00–11:40; 11.02 - HS
ORGANISERS Augustinas Šukys (The University of Melbourne); Kaan Öcal (The University of Melbourne); Lucy Ham (The University of Melbourne)

Minisymposium abstract

Modern single-cell technologies measure gene expression with unprecedented resolution, yet this data reflect multiple overlapping sources of variation. Separating technical noise from biologically meaningful fluctuations to reveal the underlying transcriptional dynamics remains a central challenge.

Mechanistic models offer a framework for proposing hypotheses about the molecular processes generating observed data. Unlike purely statistical or machine learning approaches, these models connect measurable parameters to specific biological mechanisms, enabling us to move from describing patterns to explaining their biophysical origins.

Applying such models to single-cell data, however, presents significant obstacles. Snapshot measurements provide limited information for parameter estimation, making it difficult to distinguish between competing mechanisms. Emerging temporal, multiomic and spatial profiling technologies offer new opportunities to resolve these ambiguities, but introduce model complexity and computational challenges.

This mini symposium will explore recent advances that address these challenges. Speakers will present new experimental designs, inference methods and modelling frameworks that improve identifiability, exploit structured single-cell datasets and enable robust validation of mechanistic conclusions. Our goal is to move the field toward a framework where single-cell data routinely yield quantitative, mechanistic understanding of cellular regulation.

MS77-04 — Stochastic Gene Expression: Modeling and Inference from Single-Cell Data

SPEAKERS Ramon Grima (The University of Edinburgh)
AUTHORS Ramon Grima (The University of Edinburgh)
SCHEDULE Thursday, 16 July 2026; 17:00–17:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Gene expression is intrinsically stochastic, leading to substantial cell-to-cell variability in mRNA and protein levels, now routinely quantified with single-cell technologies. In this talk, I will discuss extensions of the classical two-state telegraph model to incorporate salient features of single-cell biology, including cell division, DNA replication, mRNA maturation, gene dosage compensation, growth-dependent transcription, cell-size control strategies and cell-cycle duration variability. I will also present our statistical inference and machine learning approaches for fitting both classical and complex gene-expression models to single-cell data (smFISH, live-cell imaging, and scRNA-seq). These frameworks provide principled ways to separate biological from technical noise, estimate transcriptional parameters, and infer the mechanisms most compatible with observed transcriptional dynamics.

MS77-01 — Clone wars: understanding clonal diversity in resistance to CAR-T cell immunotherapy

SPEAKERS Adriana Zanca (The University of Melbourne)
AUTHORS Adriana Zanca (The University of Melbourne); Augustinas Sukys (University of Melbourne); Elijah Foo (University of Melbourne); Jamie Kuzich (Peter MacCallum Cancer Centre); Lucy Ham (The University of Melbourne); Mark Dawson (Peter MacCallum Cancer Centre); Michael P. H. Stumpf (University of Melbourne)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Chimeric Antigen Receptor (CAR)-T cell therapy is revolutionising immunotherapy, and has shown significant efficacy in cancers of the blood. Experimental collaborators have observed that patterns of clone-agnostic and clone-specific resistance change sharply with immunologic pressure. We have leveraged mathematical modelling to determine which mechanisms can give rise to the experimentally observed clonal resistance profiles. We develop our models under a branching process framework, considering features such as CAR-T expansion, cytotoxic action, tumour competition, and stochastic extinction. Our modelling suggests that the observed clonal distributions are only possible in the presence of cellular competition. Calibrating the models to experimental data has presented numerous challenges. We conclude by sharing our ambitions for model calibration and highlighting the value of mathematical modelling as a framework for exploring mechanisms in the absence of explicit model calibration.

MS77-02 — Detection Noise Renormalizes Apparent Kinetic Rates in Stochastic Gene Regulatory Networks

SPEAKERS Iryna Zabaikina (Comenius University)
AUTHORS Iryna Zabaikina (Comenius University); Ramon Grima (The University of Edinburgh)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Imperfect molecular detection in single-cell experiments introduces technical noise that can distort the observed dynamics of gene regulatory networks, hence complicating the inference of the true kinetic parameters from single-cell data. We extend binomial capture models from simple gene-expression systems to general, possibly time-dependent, regulatory networks, using both chemical master equation and piecewise-deterministic Markov process descriptions. Our main result is that the effects of technical noise can be absorbed into the renormalization of a subset of the kinetic rates. This occurs when transcription factor abundance is not too small. In this regime, imperfect capture leads to apparently smaller mean burst sizes of gene products and apparently larger transcription factor binding rates. Together, these results provide a systematic framework for interpreting noisy single-cell measurements.

References

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MS77-03 — Neural approximations for models of transcriptional dynamics enable genome-wide parameter inference

SPEAKERS Maria Carilli (Caltech)
AUTHORS Gennady Gorin (Fauna Bio, Emeryville, California, United States of America); Lior Pachter (Division of Biology and Biological Engineering, California Institute of Technology, Pasadena, California, United States of America); Maria Carilli (Caltech); Tara Chari (Eric and Wendy Schmidt Center at the Broad Institute of MIT and Harvard); Yongin Choi (University of California, Davis: Davis, California, US)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

The advent of high-throughput transcriptomics provides an opportunity to advance mechanistic understanding of transcriptional processes and their connections to cellular function at an unprecedented, genome-wide scale. These transcriptional systems, which involve discrete stochastic events, are naturally modeled using chemical master equations (CMEs), which can be solved for probability distributions to fit biophysical rates that govern system dynamics. While CME models have been used as standards in fluorescence transcriptomics for decades to analyze single-species RNA distributions, there are often no closed-form solutions to CMEs that model multiple species, such as nascent and mature RNA transcript counts. This has prevented the application of standard likelihood-based statistical methods for analyzing high-throughput, multi-species transcriptomic datasets using biophysical models. We show how neural networks and statistical understanding of system distributions can produce accurate approximations to a steady-state bivariate distribution of the bursty model of transcription, bypasses intensive numerical solving techniques and reducing likelihood evaluation time by several orders of magnitude. This method can be incorporated into existing machine learning architectures to enable broad exploration of parameter space of transcriptional burst sizes, RNA splicing rates, and mRNA degradation rates from experimental transcriptomic data.

References

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MS77-06 — The Impact of the Cell Cycle on Cellular Decision Making

SPEAKERS Charli Austin (The University of Edinburgh)
AUTHORS Charli Austin (The University of Edinburgh); Nikola Popovic (University of Edinburgh); Ramon Grima (The University of Edinburgh)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Gene regulatory networks (GRNs) underpin many of the most important processes in cellular biology – including cell fate, patterning and adaptation. Whilst the biological realism of GRN models has improved in recent years, there has been little consideration of cell cycle effects on the dynamics of these models. For the classic, bistable toggle switch, we find that the inclusion of cell growth and division can significantly alter the basins of attraction of the two stable states in the model as a result of a shift in the separatrix that divides those basins in the phase plane. Given the highly non-linear nature of the original toggle switch model, we formulate a Boolean modelling framework which allows us to derive analytical expressions for the separatrices in an approximate Boolean model, both with and without division. These expressions allow us to describe ”regions of disagreement” in the phase plane in which attraction to the opposite steady state ensues when the cell cycle is incorporated into the Boolean toggle switch. Furthermore, we assess the discrepancies between these regions in the original toggle switch model and our Boolean approximation in dependence of system parameters such as synthesis rates and Hill exponents.

MS77-07 — Simulation-free approximate Bayesian computation for stochastic reaction networks and its applications on scRNA-seq data

SPEAKERS Zekai Li (Imperial College London)
AUTHORS Philipp Thomas (Imperial College London); Zekai Li (Imperial College London)

SCHEDULE Friday, 17 July 2026; 11:20–11:40; 11.02 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In single-cell biology, stochastic reaction networks (SRNs) model molecular production, degradation, and interactions, and inferring their structure and parameters from data is central to understanding underlying biological mechanisms. Approximate Bayesian computation (ABC) offers a flexible Bayesian approach with posterior uncertainty quantification, but its reliance on extensive stochastic simulations results in high computational cost even for small systems, despite advances in sampling schemes. In this work, we propose a simulation-free approximate Bayesian computation (SFABC) framework that avoids explicit simulation of system dynamics. The method instead exploits theoretical constraints derived from the governing equations and uses convex optimization to evaluate whether a candidate parameter set is consistent with the observed data. Using synthetic benchmarks, we show that SFABC is compatible with different sampling schemes and achieves performance comparable to standard ABC algorithms, while substantially reducing computational cost that does not scale with sample size. Applied to steady-state scRNA-seq datasets, SFABC unravels transcriptional bursting kinetics through full posterior inference beyond point estimates. Using metabolic labelling data, we compare snapshot and time resolved models. Extending SFABC to model selection, we distinguish competing models of cell cycle–dependent transcriptional regulation using scRNA-seq data with cell cycle reporters.

MS96 — Modelling the mechanical behaviour of cells

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 03.01 - HS
ORGANISERS Sofie Verhees (University of British Columbia); Jupiter Algorta (University of British Columbia)

Minisymposium abstract

Understanding how cells sense, generate, and respond to physical forces is central to explaining a wide range of biological processes, from collective migration and tissue morphogenesis to tumour invasion and intracellular signalling [Rom+21]. Mathematical and computational models that integrate the mechanical properties of cells and/or their environment have become essential tools for uncovering the principles underlying these phenomena [Che+17; CR17]. Such mechanochemical approaches provide new insights into how physical forces interact with signalling pathways, offering improved understanding of cell function, development, and disease progression.

This mini-symposium brings together researchers developing state-of-the-art theoretical, mechanochemical, and simulation-based frameworks that explicitly incorporate the physical properties of cells and their microenvironment. The talks will showcase diverse modelling approaches—ranging from continuum and agent-based descriptions to multiscale and hybrid methods—aimed at exploring how force generation, deformation, and cell shape drive cellular behaviour.

References

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MS96-01 — Alignment processes in cells: From individual interactions to collective behaviour

SPEAKERS Angelika Manhart (University of Vienna)
AUTHORS Angelika Manhart (University of Vienna); Vivienne Leech (University College London); Mohit Dalwadi (University of Oxford); Brian Stramer (King's College London)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 03.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

How do many groups of organisms, ranging in size from micro-meter scale bacteria, to meter scale birds, form highly co-ordinated groups? One commonly observed dynamic is that of directional alignment of individuals. In this talk I will focus on alignment of elongated cells, with a focus on fibroblasts, which show particularly strong alignment in certain type of scars (keloid scars). To obtain a mechanistic, interpretable, model for the dynamics of cell position, cell orientation and cell shape we use an energy minimization approach. We analyse the resulting dynamics from two points of view: Computationally via a collective dynamics simulation, and analytically in a two cell setting. The combined insights shed light on the role of cell speed, cell deformability and supracellular actin cables in alignment dynamics.

References

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[Lee+24] Vivienne Leech et al. "Derivation and simulation of a computational model of active cell populations: How overlap avoidance, deformability, cell-cell junctions and cytoskeletal forces affect alignment". In: *PLoS computational biology* 20.7 (2024), e1011879. ISSN: 1553-7358. DOI: 10.1371/journal.pcbi.1011879.

MS96-02 — A mathematical framework for coupled mechanics and mechanosensing in adherent cells

SPEAKERS Gordon R. McNicol (University of Waterloo)
AUTHORS Gordon R. McNicol (University of Waterloo); Peter S. Stewart (University of Glasgow)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 03.01 - HS
TRACK Cellular and Developmental Biology

Abstract

Cells sense and respond to their local environment through an integrated network of subcellular structures that convert mechanical and biochemical cues into coordinated biological responses. Understanding how these components interact to regulate cellular mechanosensing and signalling is central to controlling cell behaviour, with applications in tissue engineering and regenerative medicine. Here, we develop a bio-chemo-mechanical model that captures the formation and maturation of two key mechanosensing structures: focal adhesions (FAs) and stress fibres (SFs) in non-motile eukaryotic cells cultured *in vitro*. We formulate a two-dimensional axisymmetric model that couples cytoplasmic and adhesion mechanics with cell biochemistry, represented by a system of reaction-diffusion-advection equations. The model incorporates bidirectional feedback mediated by Rho signalling and explicitly includes both the plasma membrane and a stiff elastic nucleus. Our simulations demonstrate that the nucleus is a critical regulator of adhesion localisation, striation patterns, and intracellular signalling, whereas the plasma membrane exerts negligible influence. Using the model, we also show that well-adhered cells sense distant extracellular perturbations primarily through their adhesions, with a mechanosensing range spanning several cell lengths. Furthermore, we demonstrate how selective inhibition of actomyosin pathways induces targeted disassembly of distinct mechanosensing components.

MS96-03 — A geometric surface PDE framework to model cell migration

SPEAKERS David Hernández-Aristizábal (Université Côte d'Azur, LJAD UMR CNRS 7351, Nice, France)
AUTHORS David Hernández-Aristizábal (Université Côte d'Azur, LJAD UMR CNRS 7351, Nice, France); Francesca Ballatore (Université Côte d'Azur, LJAD UMR CNRS 7351, Nice, France); Anotida Madzvamuse (The University of British Columbia, Mathematics Department, 1984 Mathematics Road, Vancouver, V6T 1Z2, Canada; Department of Mathematics and Computational Sciences, University of Zimbabwe, Mt Pleasant, Harare, Zimbabwe; Department of Mathematics and Applied Mathematics, University of Pretoria, Pretoria, 0132, South Africa; Applied Mathematics, University of Johannesburg, PO Box 524 Auckland Park Johannesburg, 2006, South Africa); Frédéric Luton (Université Côte d'Azur, IPMC UMR CNRS 7275, INSERM U1323, Valbonne, France); Rachele Allena (Université Côte d'Azur, LJAD UMR CNRS 7351, Nice, France; Institut Universitaire de France, Paris, France)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 03.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Cell migration is a ubiquitous and complex process in biology, spanning embryonic development to cancer metastasis. During migration, cells undergo high deformation as they explore the medium or as they traverse narrow pores. Over the last decades, there has been an increasing interest in mathematical models for cell migration, with the particular challenge of integrating dynamic bio-chemo-mechanical processes within an evolving domain. Here, we present a biophysical model of cell migration grounded in the theory of curvature-elastic energetic biomembranes. We develop the model systematically, progressing from equilibrium in the absence of external loads to migration through confined environments. We study diverse migration scenarios, emphasising the mechanical coupling between the cell plasma membrane and the nuclear envelope, and the role of size constraints in cell squeezing. These size constraints arise from the finite availability of lipids to constitute the plasma membrane and the nuclear envelope, combined with osmotic pressure, together restricting surface area and volume. While the elastic stiffness of the nucleus has been thought to be the main obstacle for cell migration in confinement, our results suggest that size constraints and the membrane-to-nucleus mechanical coupling also play a critical role. This suggestion aligns with recent experimental studies tracking the evolution of the nucleus during cell migration.

MS96-04 — Modeling active cell migration in complex confined environments: The interplay between nuclear mechanics, chemotaxis, and obstacle geometry

SPEAKERS Francesca Ballatore (Laboratoire Jean Alexandre Dieudonné, CNRS UMR7351, Nice, France)
AUTHORS Francesca Ballatore (Laboratoire Jean Alexandre Dieudonné, CNRS UMR7351, Nice, France); David Hernández-Aristizábal (Laboratoire Jean Alexandre Dieudonné, CNRS UMR7351, Nice, France); Anotida Madzvamuse (Mathematics Department, University of British Columbia, Vancouver, Canada; Department of Mathematics and Computational Sciences, University of Zimbabwe, Mt Pleasant, Harare, Zimbabwe; Department of Mathematics and Applied Mathematics, University of Pretoria, Pretoria, 0132, South Africa; Applied Mathematics, University of Johannesburg, PO Box 524 Auckland Park Johannesburg, 2006, South Africa); Rachele Allena (Laboratoire Jean Alexandre Dieudonné, CNRS UMR7351, Nice, France; Institut Universitaire de France, France)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 03.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Understanding how cells navigate through dense and heterogeneous microenvironments is a fundamental challenge in biomechanics, with direct implications for cancer metastasis, immune surveillance, and tissue morphogenesis. In confined spaces, the nucleus, the largest and stiffest cellular organelle, acts as the primary physical bottleneck, limiting deformability and determining the success of migration through narrow gaps.

In this work, we present an advanced computational framework based on geometric surface partial differential equations (GS-PDE) to simulate active cell migration. Our approach treats the plasma membrane and the nuclear envelope as evolving, energetic closed surfaces governed by force-balance equations, integrated with a chemotactic signalling mechanism that drives autonomous motion. We investigate various migration scenarios by systematically varying the mechanical properties of the cell and its nucleus, as well as the geometric features of the surrounding environment. Our results highlight how

the interplay between nuclear properties and obstacle morphology dictates the efficiency of the migration process. In general, this framework provides a robust and flexible tool for dissecting the biophysical strategies cells employ to bypass physical barriers, offering new insights into the mechanobiology of invasive cellular behaviour.

MS96-05 — From epithelial architecture to chemotaxis and polarity: multiscale mechanical modeling of cell behavior with CompuCell3D

SPEAKERS James A. Glazier (Indiana University, Department of Intelligent Systems Engineering and Biocomplexity Institute, Bloomington, IN, 47408, USA)

AUTHORS James A. Glazier (Indiana University, Department of Intelligent Systems Engineering and Biocomplexity Institute, Bloomington, IN, 47408, USA); Joel Vanin (Indiana University, Department of Intelligent Systems Engineering and Biocomplexity Institute, Bloomington, IN, 47408, USA)

SCHEDULE Friday, 17 July 2026; 10:40–11:00; 03.01 - HS

TRACK Multiscale and Multiphysics Modelling

Abstract

Mechanical behavior affects multiple biological scales, from tissue organization to cell migration to subcellular patterning. I will show how CompuCell3D can model different aspects of cell mechanics within a common cell-based framework. CC3D cells are extended, deformable objects with explicit interfaces and subcellular structure, allowing linkage of adhesion, shape, force balance, polarization, and motion to emergent biological form and function.

I will illustrate this multiscale perspective with three examples. The organization and disorganization of epithelial sheets, showing how variation in cell-cell contact energies can reproduce subtype-specific morphologies in lung adenocarcinoma [Zha+24]. Second, a three-dimensional model of cell migration in a chemical field, where chemotaxis emerges from the interplay of directional sensing, polarity reorientation, protrusive activity, and migration [Dal+25]. Third, a subcellular model of planar cell polarity in which intracellular reorganization of polarization components and their intercellular interactions generate tissue-scale alignment, showing how coherent polarity can arise from local rules in the absence of a tissue-wide morphogen gradient [TB26]. Together, these examples highlight how to integrate mechanical and regulatory processes in a unified computational framework to study cell behavior across scales.

References

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MS96-06 — Guidance by followers: inference of microscopic mechanical cell coordination through quantitative modelling

SPEAKERS Jörn Starruß (TU Dresden)

AUTHORS Jörn Starruß (TU Dresden)

SCHEDULE Friday, 17 July 2026; 11:00–11:20; 03.01 - HS

TRACK Multiscale and Multiphysics Modelling

Abstract

Modeling and simulation become increasingly important to study complex developmental processes across scales and thus constitute an indispensable component of interdisciplinary workflows in quantitative multicellular biology.

Along the lines of a data driven study of collective migration in early zebrafish development, that revealed a novel concept termed 'guidance by followers' [Bou+22; GF22], I will recapitulate how the dynamic cell shape representation in the cellular Potts model allows us to account for membrane-local sensing and active behavior (e.g. motility). The fluent composability of the CPM with other paradigms simplified the development and implementation of multiple concurrent hypotheses about the underlying coordinating mechanisms, obscured in complex biological scenario. To infer the best mechanism we employed the FitMultiCell workflow: implement the concurrent models in the multi-scale modelling framework Morpheus and perform model selection for stochastic models using FitMultiCell and PyABC [Sta+14; Ala+23].

References

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MS96-07 — A Multiscale Model of Contact-Guided Cell Migration Incorporating Cell Morphodynamics

SPEAKERS Nadia Loy (Politecnico di Torino)
AUTHORS Nadia Loy (Politecnico di Torino)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 03.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

In this talk, I present a multiscale model for cell migration in fibrous extracellular matrix (ECM) structures. The microscopic dynamics account not only for the classical mechanism of contact guidance, but also for cell morphodynamics in response to the surrounding fibrous environment. Different migration modes and morphological adaptations are considered, together with their interplay. In particular, we recover key experimental observations concerning amoeboid versus mesenchymal morphologies and their dependence on the underlying fiber organization. By deriving kinetic (mesoscopic) models, we gain insight into the aggregate statistical behavior of cells. This provides a consistent framework for the formulation of macroscopic models describing the evolution of cell number density, in which contact guidance dynamics are modulated by cell morphology and internal states. We further discuss the role of morphology and polarization dynamics in determining cell persistence, and analyze how these factors influence migration patterns in spatially heterogeneous environments composed of unpolarized fibers, highlighting their impact on large-scale cell behavior and emergent transport properties, as observed, for example, in carcinoma cell invasion into aligned collagen matrices.

MS96-08 — A purely mechanical model with bending bacteria for early morphogenesis of micro-colony growth

SPEAKERS Sophie Hecht (Sorbonne Universite)
AUTHORS Sophie Hecht (Sorbonne Universite)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 03.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Bacteria colonize surfaces to settle sessile colonies called biofilms. Understanding the formation of these biofilm is of crucial interest for industrial processes, in animal health and in biotechnological applications. Rod-shaped bacteria such as Escherichia coli and Pseudomonas aeruginosa have been extensively studied and modeled in-silico using rigid spherocylinders. However, experimental data have shown that bacteria can significantly bend in order to maximize cell-cell interactions in the colony. We develop an individual-based model with bending bacteria composed of a minimal set of heuristic mechanical rules to identify the key factors required to recover realistic structured patterns observed in bacterial populations. With a throughout parameter analysis we investigate the influence of bacteria bending on four-cell array structure, colony elongation, bacterial density, etc. To validate our model assumption we then confront numerical simulations to experimental data.

MS107 — Modeling Avian Influenza Dynamics

TRACK Mathematical Epidemiology
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 15.03 - HS
ORGANISERS Mattia Sensi (Università di Trento)

Minisymposium abstract

Avian influenza remains a major threat to animal health, food security and human health due to its capacity for cross-species transmission and rapid evolutionary change. Recent outbreaks in Europe and globally highlight the urgent need for quantitative frameworks that integrate epidemiological, ecological and evolutionary processes across multiple host species.

This minisymposium aims to bring together researchers developing mathematical and theoretical approaches to model, understand, predict and control avian influenza. The session will showcase a range of modeling techniques, including compartmental and network models, stochastic and spatial approaches, and phylodynamic and eco-evolutionary frameworks. By fostering dialogue across a broad spectrum of mathematical modellers, this minisymposium aims to stimulate new collaborations and advance the role of mathematical biology in addressing the ongoing challenges posed by avian influenza.

MS107-04 — Tracking the evolution, spread and reassortment patterns of avian influenza viruses using sequence data and phylodynamic modelling

SPEAKERS Lu Lu (Roslin Institute, University of Edinburg)
AUTHORS Lu Lu (Roslin Institute, University of Edinburg)
SCHEDULE Thursday, 16 July 2026; 17:00–17:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Genetic reassortment, the exchange of gene segments between distinct influenza A virus genomes, is a key mechanism driving the emergence of novel variants. In avian influenza viruses, reassortment are extremely frequent and accompanies phenotypic changes. Since 2020, clade 2.3.4.4b H5 high pathogenicity avian influenza viruses (HPAIVs) have driven a global panzootic, causing mass mortality in wild birds, poultry and repeated spillover infections in a variety of mammalian species. This resurgence of H5 HPAIV has coincided with a dramatic increase in the number of circulating reassortant strains; however, the scale,

impact and drivers of these reassortants remain unknown. Here, we combined statistical and phylodynamic modelling to reconstruct the global evolutionary dynamics of H5Nx viruses across four epizootic seasons. We identified over 200 genetically distinct reassortants, stratified into three transmission categories based on their phylogenetic and epidemiological profiles. Statistical modelling revealed that reassortant success was strongly shaped by ecological factors, including circulation in specific wild bird orders and the ability to infect a wider range of host niches. Collectively, our findings reveal reassortment dynamics in H5 HPAIVs and identify key virological and ecological. These insights support the importance of enhanced surveillance to track evolution of H5 HPAIV and identify traits relevant for consideration in pandemic risk assessment.

MS107-01 — A modelling assessment of the impact of control measures on highly pathogenic avian influenza transmission in poultry in Great Britain

SPEAKERS Edward Hill (University of Liverpool)
AUTHORS Chris Davis (UK Health Security Agency \& University of Warwick); Chris Jewell (Lancaster University); Edward Hill (University of Liverpool); Kristyna Rysava (University of Warwick); Mike Tildesley (University of Warwick); Robin Thompson (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Since 2020, highly pathogenic avian influenza (HPAI) H5N1 outbreaks in Great Britain have resulted in substantial poultry mortality and economic losses. There are additional concerns of increased HPAI circulation leading to a viral reassortment that causes zoonotic spillover. However, the mechanisms driving transmission between poultry premises and the impact of potential control measures in Great Britain, such as vaccination, are not fully understood. To enhance understanding of the disease transmission process and the potential measures that could be used to avert future infections, there is a need for mathematical models to be calibrated to outbreak data. I will present a spatial transmission model for the spread of HPAI in poultry premises calibrated using Markov chain Monte Carlo to infected premises data for the 2022–23 influenza season in Great Britain. The model formulation adapts existing spatial individual poultry premises-based models - such as Jewell et al. [Jew+09] and Hill et al. [Hil+17; Hil+18] - for Great Britain. I will then overview model simulations investigating the potential epidemiological impacts of reactive enhanced control strategies that reduce susceptibility of poultry (e.g. enhanced biosecurity measures and/or vaccination). Our findings highlight thatenhanced control measures could limit the future impact of HPAI on the poultry industry and reduce the risk of broader health threats [Dav+26].

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MS107-02 — Modelling H9N2 AIV transmission along poultry production and distribution networks in Bangladesh

SPEAKERS Francesco Pinotti (INRAE, VetAgro Sup, UMR EPIA, Université de Lyon)

AUTHORS Francesco Pinotti (INRAE, VetAgro Sup, UMR EPIA, Université de Lyon)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

H9N2 avian influenza viruses (AIVs) are endemic in Bangladesh and are consistently detected at high prevalence within live bird markets (LBMs). Despite its low pathogenic status, H9N2 AIV can hamper poultry meat and egg production. Moreover, its sheer prevalence in LBMs is concerning due to the associated risks of zoonotic spillover and viral reassortment with other co-circulating AIV subtypes (e.g. H5N1). In this presentation, I will first summarise recent modelling efforts leveraging observational data from a large, collaborative project to characterise H9N2 AIV epidemiology in LBMs. In particular, I will discuss the main drivers of virus persistence and diversity within LBMs. I will then show that within-LBM AIV dynamics cannot be fully understood without considering the rest of the production and distribution network (PDN) where LBMs are the terminal nodes. Indeed, upstream activities like poultry farming and transport are found to provide opportunities for the transmission and dissemination of infectious agents along the PDN. Finally, I will introduce a novel agent-based modelling framework to describe AIV transmission at the scale of the entire PDN. The model involves multiple actors and settings, including farms, mobile traders and LBMs, and uses extensive survey data to inform their behaviour. Our results demonstrate the importance of accounting for the entire PDN structure to implement more effective veterinary public health measures to reduce the burden of H9N2 AIV.

MS107-03 — Co-circulation of low and highly pathogenic avian influenza viruses in poultry farms: an integrated experimental and modeling framework

SPEAKERS Hadi Taghvafard (Wageningen Bioveterinary Research)
AUTHORS Hadi Taghvafard (Wageningen Bioveterinary Research); Jose L. Gonzales (Wageningen Bioveterinary Research); Nancy Beerens (Wageningen Bioveterinary Research); Rene Heutink (Wageningen Bioveterinary Research)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Emergence of high-pathogenicity avian influenza virus (HPAIV) H5 or H7 variants, following infection with low-pathogenicity avian influenza (LPAIV) of several poultry flocks or directly after infection of a single of few flocks within a poultry farm, emphasizes the need for understanding co-circulation dynamics of LPAIV and HPAIV in poultry farms to develop adequate control strategies. Although the transition from LPAIV to HPAIV is a well-documented phenomenon, limited knowledge exists on the epidemiological processes leading to selection of the HPAIV variant following its emergence in a flock. We performed transmission experiments in chickens to assess transmission of field sample containing a mix of both LPAIV and HPAIV and experiments with purified LPAIV and HPAIV variants. We derived quantitative estimates of key epidemiological parameters and developed a mechanistic stochastic Susceptible-Infectious-Recovered model, based on these estimates. We found that if HPAIV randomly emerges within an LPAIV infected chicken within the first 10 days following the start of LPAIV outbreak in a flock, a large fraction of chickens in the flock will become infected with the HPAIV ("major outbreak") with a probability of 0.9, followed by a gradual decline over subsequent days, resulting in a probability of less than 0.1 at 17 days post-mutation. We discuss the implications for control and surveillance measures of the HPAIV in poultry farms, and how these findings can support policymakers.

MS107-05 — Avian Flu Modelling in poultry farms: Case study of H5N1 HPAI in northern Italy in 2021–2022

SPEAKERS Mattia Sensi (Dipartimento di Matematica, Università degli Studi di Trento)
AUTHORS Andrea Pugliese (Dipartimento di Matematica, Università degli Studi di Trento); Mattia Sensi (Dipartimento di Matematica, Università degli Studi di Trento); Paolo Mulatti (Istituto Zooprofilattico Sperimentale delle Venezie); Sara Sottile (Deparment of Medical and Surgical Sciences, University of Bologna)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 15.03 - HS

TRACK Mathematical Epidemiology

Abstract

We provide an overview of the mechanisms behind the spreading of avian flu in farms, focusing in particular on the H5N1 Highly Pathogenic Avian Influenza (HPAI) epidemic which occurred in northern Italy in 2021–2022 [IZSVe].

We present unpublished data on the spread of HPAI among a network of hundreds of farms, visualizing the possible infection routes of the epidemic. We then define a stochastic SEUDR (Susceptible – Exposed – Undetected – Detected – Removed) model, based in part on [Dor+10].

We identify three main sources of infection (wildlife, same poultry company, and geographical distance), and estimate the parameters of our probabilistic model through the available data. We then proceed to the identification of the most likely date and source of infection for each farm involved in the epidemic wave under analysis, and provide a model which can be adapted to any other avian flu epidemic which involves both wildlife and domestic poultry.

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MS107-06 — Ecological and environmental predictors of highly pathogenic avian influenza risk in wild birds across Europe: a Bayesian machine learning approach

SPEAKERS Joe Hilton (University of Manchester)
AUTHORS Joe Hilton (University of Manchester)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Highly pathogenic avian influenza (HPAI) represents a serious threat to animal and human health, with the ongoing H5N1 outbreak within the H5 2.3.4.4b clade being one of the largest on record. Although wild birds are known to be a key reservoir of HPAI, the factors driving prevalence within this reservoir remain poorly understood. In this study we use Bayesian additive regression trees, a machine learning method designed for probabilistic modelling of complex nonlinear phenomena, to construct species distribution models (SDMs) for HPAI presence and identify factors driving geospatial patterns of infection in wild birds across Europe. Our models are time-stratified to capture both seasonal changes in risk and shifts in epidemiology associated with the succession of earlier strains by H5N1 within the clade. While previous studies aimed to model HPAI presence from physical geography, we explicitly consider wild bird ecology by including estimates of bird species richness, abundance of specific taxa, and "abundance indices" describing total abundance of birds with high-risk behavioural traits. Our model projections indicate a shift in persistent, year-round risk towards cold, low-lying regions of northwest Europe associated with H5N1. Methodologically, we demonstrate that while most variation in risk can be explained by climate and physical geography, adding host ecology is a valuable refinement to SDMs of HPAI.

MS107-07 — The role of ducks in detecting Highly Pathogenic Avian Influenza in small-scale backyard poultry farms

SPEAKERS Steve Wu (University of Warwick)
AUTHORS Steve Wu (University of Warwick)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 15.03 - HS
TRACK Mathematical Epidemiology

Abstract

Previous research efforts on highly pathogenic H5N1 avian influenza (HPAI) suggest that different avian species exhibit a varied severity of clinical signs after infection. Waterfowl, such as ducks or geese, can be asymptomatic and act as silent carriers of H5N1, making detection harder and increasing the risk of further transmission, potentially leading to significant economic losses. For backyard hobby farmers, passive reporting is a common HPAI detection strategy. We aim to develop a computational, mechanistic model to quantify the effectiveness of this strategy by simulating the spread of H5N1 in a mixed-species, small-population backyard flock. Quantities such as detection time and undetected burden of infection in various scenarios are compared. Our results indicate that the presence of ducks can lead to a higher risk of an outbreak and a higher burden of infection. If most ducks within a flock are resistant to H5N1, detection can be significantly delayed. We find that within-flock infection dynamics can heavily depend on the species composition in backyard farms. Ducks, in particular, can pose a higher risk of transmission within a flock or between flocks. Our findings can help inform surveillance and intervention strategies at the flock and local levels.

MS112 — Reaction networks: Mathematical structures and concrete biochemical systems

TRACK Systems Biology and Biochemical Networks
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 11.03 - HS
ORGANISERS Carsten Conradi (Hochschule für Technik und Wirtschaft Berlin); Maya Mincheva (Northern Illinois University); Stefan Müller (University of Vienna, Faculty of Mathematics)

Minisymposium abstract

Mathematical aspects in the analysis of reaction networks range from graph theory and dynamical systems (ODEs and PDEs) to positive algebraic geometry and inverse problems. In recent years, substantial progress has been made in the mathematics of reaction networks, and the proposed minisymposium will bring together leading experts to present both theoretical advances and analyses of concrete biochemical systems.

Dynamical systems arising from reaction network models are typically nonlinear and involve unknown parameters. This often makes direct simulation challenging and highlights the need for structural and qualitative approaches. A central goal is therefore to understand how different parameter regimes give rise to qualitatively distinct dynamical behavior, as well as to identify structural properties of reaction networks that are independent of parameter values.

Topics of the minisymposium include species extinction [Aga+26], delay systems [VS24], and the identification of autocatalytic subnetworks [Gol+25]. Applications range from Turing instabilities in distributive double phosphorylation systems [CM24] to classical models such as the Selkov model of glycolysis [BR18].

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MS112-01 — Existence of a unique non-degenerate solution to parametrized systems of generalized polynomial equations

SPEAKERS Abhishek Deshpande (International Institute of Information Technology, Hyderabad)
AUTHORS Abhishek Deshpande (International Institute of Information Technology, Hyderabad); Stefan Müller (University of Vienna, Faculty of Mathematics)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

We consider solutions to parametrized systems of generalized polynomial equations (with real exponents) in n positive variables, involving m monomials with positive parameters; that is, $x \in \mathbb{R}^n_>$ such that $A\,(c \circ x^B) = 0$ with coefficient matrix $A \in \mathbb{R}^{l\times m}$, exponent matrix $B \in \mathbb{R}^{n\times m}$, and parameter vector $c \in \mathbb{R}^m_>$ (and with componentwise product $\circ$).

We demonstrate that the existence of a unique nondegenerate solution for all parameters is equivalent to a specific moment map being a diffeomorphism. We characterize this property using Hadamard’s global inversion theorem and establish sufficient conditions based on the sign vectors of the underlying geometric objects. This result represents a multivariate generalization of Descartes’ rule of signs for exactly one nondegenerate solution.

MS112-02 — Unbounded oscillations in the Selkov model of glycolysis with Michaelis-Menten kinetics

SPEAKERS Alan Rendall (Johannes Gutenberg University Mainz)
AUTHORS Alan Rendall (Johannes Gutenberg University Mainz)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Glycolysis, the process by which energy is extracted from sugar, is known to exhibit oscillations and it has been shown experimentally that these arise in a reaction catalysed by the enzyme phosphofructokinase. Selkov introduced a simple model for this phenomenon which is a system of two ODE with mass action kinetics (in what follows the MA system). It is derived by a limiting process from a system with Michaelis-Menten kinetics (in what follows the MM system). Selkov claimed that the MA system has solutions with unbounded oscillations but that these are an artefact of the limiting process and are not present in the MM model. With Pia Brechmann we gave a rigorous proof of Selkov’s first claim. In this talk I present evidence that his second claim is not correct. A key step in the existence proof was showing that the MA system admits a heteroclinic cycle at infinity. I present a proof that the MM system also admits a heteroclinic cycle at infinity. I then explain what is needed to pass from this statement to the statement that there exist unbounded oscillations in the MM system.

References

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MS112-03 — Instabilities and pattern formation in distributive double phosphorylation

SPEAKERS Carsten Conradi (Hochschule für Technik und Wirtschaft Berlin)
AUTHORS Carsten Conradi (Hochschule für Technik und Wirtschaft Berlin); Hannes Uecker (University of Oldenburg); Maya Mincheva (Northern Illinois University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Reaction-diffusion equations provide a framework for understanding how spatial patterns emerge from biochemical networks. In quantitative biology, however, parameter values are frequently accompanied by large confidence intervals due to measurement uncertainty and limited experimental repetitions. This necessitates the study of entire families of parametrized PDEs to determine their qualitative behavior.

In this talk, we focus on a reaction-diffusion model of distributive double phosphorylation with unknown parameters. As numerical exploration of high-dimensional parameter spaces is often computationally prohibitive, we present an analytical approach to identify regions of the parameter space where the system exhibits Turing-like instabilities.

We derive a polynomial inequality — depending only on the catalytic constants and the diffusion constants of the enzyme-substrate complexes — that guarantees the existence of positive eigenvalues in the linearization of the PDE system, regardless of the values of the remaining constants. Notably, this description is structurally similar to the semi-algebraic conditions characterizing multistationarity in the corresponding ODE setting.

This is joint work with Maya Mincheva and Hannes Uecker.

MS112-04 — Asymptotic stability of delayed complex balanced reaction networks with general kinetics

SPEAKERS Gábor Szederkényi
AUTHORS Gyula Molnár; Gábor Szederkényi; Mihály A. Vághy
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Time delays are often present in natural and technological processes, and can be essential for the precise understanding and description of important phenomena. On the other hand, chemical reaction networks (CRNs) provide a general framework for describing general nonnegative nonlinear dynamics. It is known that complex balance is a property of fundamental importance, which guarantees a strong robust stability for CRNs. It was proved in the 2010’s that delayed complex balanced CRNs with mass action kinetics are asymptotically stable for arbitrarily large discrete delays and also for any practically meaningful distributed delay. In this contribution, these results are extended to CRNs with general non-mass-action kinetics having a product structure. The applicable reaction rates include e.g., Michaelis-Menten and Hill kinetics. It is shown that within each positive stoichiometric compatibility class there is a unique positive equilibrium that is locally asymptotically stable relative to the class. The stability of the equilibria is shown using appropriate logarithmic Lyapunov–Krasovski functionals both in the discrete and the

distributed delay case. The results further underline the importance of complex balance in the theory of general dynamical systems.

MS112-05 — Automatic initial estimates of reaction rate coefficients

SPEAKERS János Tóth (Eötvös Loránd University Budapest)
AUTHORS János Tóth (Eötvös Loránd University Budapest); Tamás Ladics; Tibor Nagy
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Fitting functions to data with nonlinear parameter dependence is challenging, particularly for differential equations common in chemical kinetics, due to the need for good initial estimates. We propose a method to automatically generate robust initial estimates for mass-action kinetic ODEs (where the right-hand side is linear in parameters). Illustrative examples show these estimates often yield highly accurate final fits.

References

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MS112-06 — Turing instability in reaction networks with one diffusing species

SPEAKERS Maya Mincheva (Northern Illinois University)
AUTHORS Carsten Conradi; Hannes Uecker; Maya Mincheva (Northern Illinois University)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Conditions for Turing instability in reaction networks involving two interacting species are well understood and typically require self-activation or autocatalysis. However, general criteria for the emergence of Turing instabilities in large-scale reaction networks are less studied. We consider a reaction network with an arbitrary number of species, in which only a single species diffuses. We establish a novel condition for Turing instability, which arises when a spatially homogeneous equilibrium loses stability via a pair of complex conjugate eigenvalues.

MS112-07 — Buffering Structures and Factorization of the Jacobi Determinant in Biochemical Reaction Networks

SPEAKERS Máté László Telek (Budapest University of Technology)
AUTHORS Arne Kuhrs (Paderborn University); Máté László Telek (Budapest University of Technology); Nicola Vassena (University of Leipzig)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In the bifurcation analysis of biochemical reaction networks, the determinant of the Jacobian of the corresponding ODE system plays a crucial role. When network parameters are treated symbolically, computing this determinant becomes challenging, even for moderately sized systems. This computation can be simplified if one decomposes the network into subnetworks such that the Jacobi determinant factorizes into a product of determinants associated with these subnetworks. One such known decomposition is based on so-called buffering structures. In this talk, we show that buffering structures are not only sufficient but also necessary for a factorization of the Jacobi determinant. Furthermore, we present an effective computational method for detecting buffering structures in a given reaction network.

MS112-08 — Autocatalysis in Reaction Networks with Explicit Catalysis

SPEAKERS Richard Golnik (University Leipzig)
AUTHORS Richard Golnik (University Leipzig); Nicola Vassena (University Leipzig); Peter F. Stadler (University Leipzig); Thomas Gatter (University Leipzig)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 11.03 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In the practical analysis of chemical reaction networks, it is often assumed that no explicit catalysts exist, i.e., that species appear both as a reactant and a product in the same reaction. In this case, the stoichiometric matrix uniquely identifies the corresponding reaction network (RN), and methods from matrix theory naturally apply. However, catalysis plays an important role, in particular for biochemical reactions. We, therefore, adapt here the concepts derived for autocatalytic cores, the minimal units accounting for the emergence of autocatalysis, to RNs with explicitly catalyzed reactions. In this setting, we confirm that an inspection of the stoichiometric matrix alone is inconclusive concerning the presence and number of autocatalytic cores, and that a more delicate algebraic analysis is required. Nevertheless, this generalization demonstrates that, up to certain subtleties, both the graph and matrix representations of autocatalytic cores are preserved. We additionally show that in the common case of unit stoichiometries (0 and 1), autocatalytic cores containing explicitly catalyzed reactions always have a spanning subgraph that is composed of a single loop with a simple metabolite-to-reaction chord.

MS130 — Prospectives in HIV

TRACK Immunobiology & Infection
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 02.21 - HS
ORGANISERS Jasmine Kreig; Macauley Locke

Minisymposium abstract

The HIV-1 epidemic has been ongoing for over 40 years and has prompted a large interdisciplinary effort involving experimentalists, mathematicians, epidemiologists, and public health officials. Together, these communities have worked not only to understand the virus and its modes of transmission, but also to develop therapies, interventions, and educational strategies aimed at reducing the global HIV-1 burden. Despite this substantial progress, a definitive cure remains elusive. In this session, we examine perspectives on HIV-1 across time and across disciplines within mathematical biology, highlighting how new modeling approaches and emerging experimental data are being integrated to advance our understanding. We aim to encourage discussion of emerging directions in HIV-1 research and mathematical modeling. To support this goal, we have assembled a diverse group of speakers spanning multiple research areas – including epidemiology, viral dynamics, and within-host evolution—and career stages, from graduate students and postdoctoral researchers to established faculty.

MS130-01 — Modelling longitudinal vaccine-elicited immune profiles in people living with HIV

SPEAKERS Chapin Korosec (University of Guelph)
AUTHORS Chapin Korosec (University of Guelph)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

The immune response to vaccination is highly heterogeneous and arises from a dynamic interplay of immune components. In this talk, I will discuss how we employed random forests (RFs) to classify differences in immunogenicity between older people with HIV (PWH) on ART and age-matched controls who received up to five SARS-CoV-2 vaccinations. Harnessing machine learning (ML) to learn immune interdependencies offers the potential to decode immune signatures linked to a specified comorbidity, and further reveal individualized patterns laying the groundwork for precision-guided vaccination and targeted clinical follow-up. Our data set contains an extensive range of immune features, including serum and saliva IgG and IgA responses, ELISpot IFNg and IL2 responses to SARS-CoV-2 spike peptides, ACE2 receptor displacement, and SARS-CoV-2 neutralization capacity; all tracked longitudinally up to 104 weeks in each individual following SARS-CoV-2 vaccine doses 1 through 5. In this biomarker space, RFs identify highly important and unimportant combinations of features that distinguish PWH from controls, and further reveal a subset of PWH whose immune signatures resemble controls, suggesting near-complete immunologic restoration from a vaccine perspective in these individuals. Our results highlight the effectiveness in utilizing RFs to identify complex immunological interdependencies.

MS130-02 — Mathematical Modeling of HIV-Driven Accelerated Biological Aging via Long-Lived Cell Reservoirs

SPEAKERS Esteban Hernandez-Vargas (University of Idaho,)
AUTHORS Esteban Hernandez-Vargas (University of Idaho,)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Suppressive antiretroviral therapy (ART) has been shown to only partially mitigate biological aging by plasma proteomic clocks in people living with HIV (PLWH). Despite effective viral suppression, treated individuals exhibit a measurable increase in biological age relative to uninfected controls, with more pronounced effects reported in younger populations. In this talk, an eight-compartment ordinary differential equation (ODE) model is presented, in which a three-stage HIV viral dynamics sub-model is coupled with a long-lived cell (LLC) reservoir. This reservoir comprises tissue-resident macrophages and memory CD4+ T cell populations and is further linked to chronic inflammation, cellular senescence, and cumulative biological age advancement, denoted by $\Delta(t)$. Mathematical analysis indicates that post-ART inflammatory decay is governed by the LLC reservoir half-life and that residual biological age accumulation is directly proportional to the reservoir size at treatment initiation. Under this framework, ART initiation at year 2 results in an estimated age advancement of $\Delta = 1.37$ years, and initiation at year 4 yields $\Delta = 3.81$ years. These results quantify the cumulative biological cost associated with delayed treatment initiation.

MS130-03 — A stochastic model of HIV viral rebound after treatment interruption

SPEAKERS Jasmine Kreig
AUTHORS Jasmine Kreig
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Human Immunodeficiency Virus (HIV) infections can be effectively controlled with the use of antiretroviral therapy (ART), which keep viral loads below detectable levels. Currently, individuals with HIV must

adhere to treatment for the rest of their lives to manage the virus. This is due to the existence of the HIV reservoir – a population of cells that are latently infected by HIV – which can reactivate and cause viral rebound in individuals who stopped ART. Interestingly, the time to viral rebound is variable from weeks (in most individuals) to years. Mechanisms behind viral rebound or factors that could influence the timing of viral rebound remain largely misunderstood. We have developed a simplified model that simulates the seeding of the reservoir and viral rebound after treatment interruption. Using this model, we explore different factors that could be associated with extending the time to viral rebound.

MS130-04 — Investigating Heterogeneity in HIV Viral Rebound Times

SPEAKERS Jessica Conway (Penn State, USA)
AUTHORS Jessica Conway (Penn State, USA)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Antiretroviral therapy (ART) effectively controls HIV infection, suppressing HIV viral loads. While typically suspension of therapy is rapidly followed by rebound of viral loads to high, pre-therapy levels, there is an important nuance: in a small fraction of cases, rebound may be delayed by months, years, or even possibly, permanently. We will discuss modeling to investigate that heterogeneity in outcome of treatment suspension, focusing on time to viral rebound. We will first discuss our data-validated, mechanistically-motivated survival function for time-to-rebound using time-inhomogeneous branching processes. We show good agreement with data for both rapid and significantly delayed viral rebound. We will then use this model to characterize the impact of covariates such as treatment initiation time and pre-ART drug regimen on time to rebound.

MS130-05 — Tracking and predicting the dynamics of HIV-1 epidemics in France using virus genomic data

SPEAKERS Louis Colliot
AUTHORS Louis Colliot
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Understanding the dynamics of HIV epidemics is important to control them effectively. Classical methods that mainly rely on occurrence data are limited by the fact that an unknown part of the epidemic eludes sampling. Since the early 2000s, phylodynamic methods have enabled the estimation of key epidemiological parameters from virus genetic sequence data. These methods have the advantage of being less sensitive to sampling bias and to track the epidemic history even before the date of the first samples. In this study, we analysed 2,205 HIV sequences from the French ANRS PRIMO C06 cohort. We identified and were able to reconstruct the history of two large clades that reflect key features of the HIV-1 epidemics in the country. Using Bayesian phylodynamic inference models, we found that the first clade, from subtype B, originated in the end of 1970s, grew rapidly during the 80s before decreasing from 2000 to 2015 and stagnating since then. The second clade, from CRF02_AG, emerged and spread in the 80s, grew again in the early 2000s, before declining slightly. Finally, using numerical simulations, we investigate prospective scenarios for future epidemic trends. This study is one of the first to analyse the HIV epidemic in France using phylodynamics methods. It demonstrates the historical and public health value of routine HIV sequence data in epidemiological surveillance.

MS130-06 — Elucidating the mechanism of synergy between latency reversal agents and broadly neutralizing antibodies for HIV remission

SPEAKERS Narendra Dixit (Indian Institute of Science)
AUTHORS Narendra Dixit (Indian Institute of Science)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 02.21 - HS

TRACK Immunobiology & Infection

Abstract

Recent studies have shown that the administration of combinations of latency reversal agents (LRAs) and broadly neutralizing antibodies (bNAbs) at the time of antiretroviral therapy (ART) cessation significantly enhances the chances of eliciting long-term control of HIV post treatment over that with ART alone. Surprisingly, neither LRAs nor bNAbs succeed independently, implying strong synergy between them. The mechanism(s) underlying the synergy remain unknown, precluding optimal deployment of the drug combinations. Here, we posit a mechanism of synergy and evaluate it using mathematical modeling. When ART is successful, productive viral replication is nearly completely halted. Under these circumstances, LRAs reactivate latently infected cells, triggering bursts of viral production. bNAbs bind to these virions and enhance antigen presentation to immune cells, stimulating HIV-specific CD8 T-cells. The combination, administered towards the end of ART, thus leaves a reduced latent reservoir and a primed CD8 T-cell population. The smaller reservoir size suppresses the magnitude of the viral rebound post ART. The primed CD8 T-cell population proves adequate to exert lasting control of the rebounding virus. Our model, based on the above mechanism of synergy, recapitulated clinical data of long-term remission following the LRA/bNAb combination therapy. The model offered insights into the role of CD8 T-cells in eliciting and maintaining viremic control and presented a route to identifying optimal dosages of LRAs and bNAbs for achieving this control, informing ongoing clinical trials.

MS130-07 — Can we PrEP Our Way Out of the HIV Epidemic?

SPEAKERS Salman Safdar (University Of Maryland, College Park)
AUTHORS Salman Safdar (University Of Maryland, College Park)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 02.21 - HS
TRACK Mathematical Epidemiology

Abstract

Abstract: The use of pre-exposure prophylaxis (PrEP), where approved antivirals are administered to uninfected high-risk individuals, is universally regarded as a promising strategy to prevent susceptible high-risk individuals from acquiring HIV infection from their infected partners. A number of antiviral drugs (and their combinations) have been developed and are being used as prophylaxis against the HIV epidemic here in the U.S. and globally. We will first present a risk-structured mathematical model for assessing the population-level impact of PrEP in an MSM (men who have sex with men) population. An extended model, which considers several high-risk populations, will also be presented and used to assess the potential spillover effect, where the administration of PrEP to individuals in one risk group induces a reduction of disease burden in other risk group(s). The central aim is to determine whether the use of the aforementioned strategies can aid the End the HIV Epidemic initiative, aimed at eliminating the disease in the U.S. by 2030.

MS130-08 — Binding mediated clearance of anti-HIV-1 broadly neutralizing antibodies in viremic individuals

SPEAKERS Tyler Cassidy (University of Leeds)
AUTHORS Tyler Cassidy (University of Leeds)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 02.21 - HS
TRACK Immunobiology & Infection

Abstract

Due to their long-half life and breadth of coverage, broadly neutralizing antibodies (bnAbs) are increasingly studied for both treatment and prevention of HIV-1 infection. Recent clinical studies have shown potent neutralization with corresponding multiple log-declines following single or multiple administrations of bnAbs in viremic participants. While bnAb concentrations typically exhibit biphasic decline during these trials, bnAb pharmacokinetics significantly differ between HIV-1 positive and negative individuals during phase I trials. These differences are hypothesized to be driven by nonlinear interactions between bnAb

and circulating virus that result in increased clearance of bnAb-virus complexes. I'll discuss a series of mechanistic models that quantify these nonlinear effects across three phase I clinical trials of distinct bnAbs. I'll show how the dynamics of these bnAb-virus complexes can explain nonlinear viral dynamics during early bnAb treatment and implicate the immune system in the viral dynamics following bnAb treatment.

MS143 — Modeling of neural dynamics and neurodegeneration

TRACK Neuroscience and Neural Systems
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 15.12 - HS
ORGANISERS Iulia Martina Bulai (University of Torino)

Minisymposium abstract

Understanding brain function requires integrating processes across multiple spatial and temporal scales from neuronal and synaptic dynamics to large neural networks. Mathematical and theoretical modeling is crucial for linking these scales and for providing mechanistic insight into how normal brain function arises and how it is disrupted in disease.

This minisymposium focuses on mathematical models of brain function and neural systems. Particular attention will be given to how these models can be used to study pathological alterations of brain function, with Alzheimer's disease serving as a key application and motivating example rather than the exclusive focus.

Contributions may address deterministic and stochastic models of neuronal activity, network and mean-field approaches, multiscale modeling, and data-informed or computational frameworks. Applications may include changes in synaptic function, network connectivity, and large-scale brain dynamics associated with cognitive decline and neurodegeneration.

The goal of the minisymposium is to bring together mathematicians and neuroscientists interested in fundamental questions of brain function, while illustrating how theoretical approaches can be applied to understand neurodegenerative processes. By emphasizing general principles of neural systems alongside disease-oriented applications, the session aims to foster interdisciplinary exchange and stimulate new modeling approaches relevant to both healthy and diseased brains.

MS143-01 — Control treatments on surface-dependent formation and dissolution of amyloid plaques in Alzheimer's disease

SPEAKERS Francesca Acotto (Dipartimento di Matematica "Giuseppe Peano", Università di Torino)
AUTHORS Francesca Acotto (Dipartimento di Matematica "Giuseppe Peano", Università di Torino); Iulia Martina Bulai (Dipartimento di Matematica "Giuseppe Peano", Università di Torino); Francesca Gladiali (Dipartimento di Scienze Chimiche, Fisiche, Matematiche e Naturali, Università di Sassari)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

The extracellular amyloid plaques, whose primary component is the amyloid-beta peptide (Aβ), are a key hallmark of Alzheimer's disease. A four-compartment mathematical model for the Aβ movements and aggregation processes has been proposed in [Fic+23]. The growth of the amyloid plaques is assumed to depend on their surface area, using an area-to-volume shape index. In this talk, at first, we refine the model proposed in Ficiarà 2023. In particular, we assume that also the possible dissolution of amyloid plaques is surface-dependent, analogously to their formation. Then, we incorporate terms modeling possible treatment actions to enhance Aβ disaggregation and reduce Aβ aggregation. Specifically, we take into account that a low Aβ level in the cerebrospinal fluid is considered a positive biomarker of Alzheimer's disease. In addition, we assume that treatments become increasingly inefficient as the disease

progresses. Finally, we reformulate the refined model as an optimal control problem to reduce the amyloid plaques and minimize treatment actions and costs.

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MS143-02 — Tonic-clonic seizure transitions as bifurcations in a neural field model

SPEAKERS Roberto Barrio (Department of Applied Mathematics, University of Zaragoza, Zaragoza, Spain)
AUTHORS Roberto Barrio (Department of Applied Mathematics, University of Zaragoza, Zaragoza, Spain); Oliver Cattell (School of Mathematical Sciences, University of Nottingham, Nottingham, UK); Ana Mayora-Cebollero (Department of Applied Mathematics, University of Zaragoza, Zaragoza, Spain); Reuben O'Dea (School of Mathematical Sciences, University of Nottingham, Nottingham, UK); Stephen Coombes (School of Mathematical Sciences, University of Nottingham, Nottingham, UK)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Epilepsy is a dynamic complex disease involving a paroxysmal change in the activity of millions of neurons, often resulting in seizures. Tonic-clonic seizures are a particularly important class of these and have previously been theorised to arise in systems with an instability from one temporal rhythm to another via a quasi-periodic transition. We show that a recently introduced class of next generation neural field models has a sufficiently rich bifurcation structure to support such behaviour. This is used to seed a more exhaustive numerical bifurcation analysis that highlights the preponderance of the model to generate torus bifurcations. Since the neural field model is derived from a biophysically meaningful spiking tissue model we are able to highlight the neurobiological mechanisms that can underpin tonic-clonic seizures as they relate to levels of excitability, electrical and chemical synaptic coupling, and the speed of action potential propagation.

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MS143-03 — A Functional Network Method for Monitoring State Changes in Neural Activity

SPEAKERS Richard Bertram (Florida State University)
AUTHORS Richard Bertram (Florida State University); Greg Owanga (Florida State University); Adam Dewan (Florida State University); Roberto Vincis (Florida State University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Our lab uses multi-electrode probes to monitor the electrical activity from populations of neurons in the gustatory cortex of the mouse as it drinks a liquid. We simultaneously measure when licks occur. Is it possible to use the neural recordings to determine when licks occur or what tastant is presented at various time points? We describe a new method for doing this using functional networks and analysis tools from network science. We demonstrate that this tool, NeuroSeq, is capable of determining dynamic state changes in the activity of the neural ensemble, and that many of these correspond to stimuli or mouse behavior. This functional network approach is an alternative to Hidden Markov Models that allows the user more control in determining what features of the data are used in the determination of states and state changes. It is generally applicable to the analysis of spike trains from any brain region.

MS143-04 — Modeling the formation of perinuclear crowns made of agglutinated ATM proteins observed in fibroblasts from patients affected by Alzheimer's disease

SPEAKERS Laurent Pujo-Menjouet (Université Claude Bernard Lyon 1, France)
AUTHORS Laurent Pujo-Menjouet (Université Claude Bernard Lyon 1, France); Nicolas Foray (Institut National de la Santé et de la Recherche Médicale (INSERM) U1296 Unit "Radiation: Defense, Health, Environment", Lyon, France); Maxime Estavoyer (CNRS, Centre de Physique Theorique, Marseille, France); Felipe Olivares (Université Claude Bernard Lyon 1, Institut Camille Jordan(ICJ), Lyon, France)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Alzheimer's disease is a progressive neurodegenerative disorder characterized by the irreversible loss of neurons. Under normal conditions, ATM proteins exist as inactive homodimers in the cytoplasm. When oxidative stress occurs, these dimers dissociate into monomers that migrate to the nucleus, where they detect and repair double-strand DNA breaks. However, recent findings indicate that in all cells from Alzheimer's patients, the ApoE protein, overexpressed and abnormally clustered around the nucleus, binds to ATM monomers. This interaction traps ATM–ApoE heterodimers at the nuclear envelope. As a consequence, the remaining ATM monomers, present at unusually high concentrations, rapidly re-dimerize before reaching the nucleus. These retained dimers accumulate at the perinuclear region, leading to the formation of a distinctive perinuclear crown. To better understand the mechanisms underlying this phenomenon and to explore potential therapeutic strategies, we developed two complementary modeling approaches: a compartmental model and a reaction–diffusion system that describes the physical and biochemical interactions between ATM and ApoE proteins. Both models integrate key processes such as protein transport, monomer aggregation, dimer and complex dissociation, and spatial constraints at the nuclear envelope. Using these models, we investigated how irradiation and antioxidant treatments influence the disintegration of the perinuclear crown. Our simulations show that while each strategy alone has a measurable effect, their combined application is significantly more effective in delaying or preventing the reformation of the crown. This synergy points toward a promising therapeutic direction for mitigating cellular dysfunction in Alzheimer's disease.

MS143-05 — A Multimodal Mathematical Model of Iron and Amyloid-β in Alzheimer's Disease

SPEAKERS Ilaria Stura (Dept. of Neurosciences, Università degli Studi di Torino, Italy)
AUTHORS Ilaria Stura (Dept. of Neurosciences, Università degli Studi di Torino, Italy); Eleonora Ficiarà (Dept. of Neurosciences, Università degli Studi di Torino, Italy); Carla D'Agostino (Dept. of Neurosciences, Università degli Studi di Torino, Italy); Fabrizio Pizzagalli

(Dept. of Neurosciences, Università degli Studi di Torino, Italy); Annamaria Vernone (Dept. of Neurosciences, Università degli Studi di Torino, Italy); Caterina Guiot (Dept. of Neurosciences, Università degli Studi di Torino, Italy)

SCHEDULE Friday, 17 July 2026; 10:40–11:00; 15.12 - HS

TRACK Neuroscience and Neural Systems

Abstract

Alzheimer's disease (AD) is a complex, multifactorial, and currently incurable neurodegenerative disorder. Existing treatments can only slow disease progression, highlighting the urgent need for reliable biomarkers for early diagnosis. Among the most promising candidates are iron and amyloid-beta 42 (Aβ42), which can be measured both in blood and cerebrospinal fluid (CSF). One of the hypothesized contributing mechanisms in AD is the formation of amyloid-beta plaques in the brain. However, this process remains difficult to observe and quantify in vivo. To address this limitation, several mathematical models have been proposed by our group to describe the temporal accumulation of iron and amyloid-beta in the brain (Ficiarà et al. 2022, 2023). Despite their relevance, these models are constrained by simplifying assumptions, most notably the representation of the brain as a single homogeneous compartment rather than a complex, spatially structured system. In this work, we propose an integrated mathematical framework that combines existing models of iron and Aβ dynamics into a more comprehensive description of their interaction and evolution. Model validation is performed using multimodal data from the Alzheimer's Disease Neuroimaging Initiative (ADNI), including PET and T2-weighted imaging, CSF measurements of iron and Aβ, and cognitive assessment scores such as the Mini-Mental State Examination (MMSE).

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MS143-06 — Folded node canard dynamics as a mechanism for delay in astrocytic evoked-Ca2+ responses

SPEAKERS Matteo Martin (University of Padova, Department of Information Engineering)

AUTHORS Alla Borisyuk (University of Utah, Department of Mathematics); Matteo Martin (University of Padova, Department of Information Engineering); Morten Gram Pedersen (University of Padova, Department of Information Engineering)

SCHEDULE Friday, 17 July 2026; 11:00–11:20; 15.12 - HS

TRACK Neuroscience and Neural Systems

Abstract

Astrocytic calcium responses under glutamate or ATP stimuli are highly heterogeneous, ranging from oscillatory dynamics to sustained plateau-like signals [Han+17]. In the case of multi-peaks-type responses, experimental observations consistently report a delay preceding the onset of large amplitude cytosolic calcium oscillations (LAO). This delay is captured by a novel astrocyte model based on [Tah+17], which exhibits a phase of small amplitude oscillations (SAO) before the onset of LAOs. Our computational results demonstrate that the duration of the delay depends on the number of SAOs, which is strongly modulated by the calcium concentration in the endoplasmic reticulum (ER): ER overload prolongs the delay, whereas depletion shortens it. To explain this behaviour, we analyse the model using slow–fast and canard theories, revealing its geometric structure and the presence of a folded node. The latter explains the origin of SAOs and provides a mechanistic explanation for discussing the duration of the

delay. Guided by this insight, we design an in silico experiment to reactivate SAOs by exploiting the identified folded node structure. Specifically, we suggest that the low-affinity calcium chelator TPEN could restore transient SAOs by elevating the ER calcium concentration following TPEN wash-out. Overall, this study illustrates how slow–fast and canard theories can uncover the origins of delayed calcium signalling in multi-peaks responses.

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MS143-07 — How neuromodulation shapes breathing: from cellular dynamcis to network rhythmogenesis

SPEAKERS Yangyang Wang (Brandeis University)
AUTHORS Yangyang Wang (Brandeis University)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 15.12 - HS
TRACK Neuroscience and Neural Systems

Abstract

Breathing is a vital, involuntary behavior that must remain robust while adapting to changing physiological demands. This poses a challenge for the control of breathing. The preBötzinger complex (preBötC) is a heterogeneous neuronal network responsible for driving the inspiratory rhythm. While neuromodulators such as norepinephrine (NE) allow it to be both robust and flexible for all living beings to interact with their environment, the basis for how neuromodulation impacts neuron-specific and network-level properties remains poorly understood. In this talk, I will present our recent work on neuromodulatory control of respiratory rhythms, with a focus on NE. By modeling NE effects on key intrinsic parameters, we show how it differentially modulates different neuronal subtypes. At the network level, we demonstrate that both the organization of intrinsic properties and synaptic interactions critically shape population activity. Together, these results provide a multiscale perspective on how neuromodulation regulates network state underlying respiratory rhythm generation in the preBötC.

MS143-08 — A Chemical Reaction Network-based approach to mTOR signaling dysregulation in drug-resistant epilepsy

SPEAKERS Silvia Berra (LISCOMP lab, IRCCS Azienda Ospedaliera Metropolitana Ospedale Policlinico San Martino, Genova, Italy and Dipartimento di Matematica, Università di Genova, Genova, Italy)
AUTHORS Silvia Berra (LISCOMP lab, IRCCS Azienda Ospedaliera Metropolitana Ospedale Policlinico San Martino, Genova, Italy and Dipartimento di Matematica, Università di Genova, Genova, Italy); Vincenzo Di Trani (LISCOMP lab, IRCCS Azienda Ospedaliera Metropolitana Ospedale Policlinico San Martino, Genova, Italy); Sara Pisini (Neuroscience and Human Genetics Department, Meyer Children's Hospital IRCCS, Firenze, Italy); Sara Sommariva (Dipartimento di Matematica, Università di Genova, Genova, Italy); Federico Benvenuto (Dipartimento di Matematica, Università di Genova, Genova, Italy); Franco Caviglia (Dipartimento di Matematica, Università di Genova, Genova, Italy); Michele Piana (LISCOMP lab, IRCCS Azienda Ospedaliera Metropolitana Ospedale Policlinico San Martino, Genova, Italy and Dipartimento di Matematica, Università di Genova, Genova, Italy); Matteo Lenge (Neuroscience and Human Genetics Department, Meyer Children's Hospital IRCCS, Firenze, Italy); Renzo Guerrini (Neuroscience and Human Genetics Department, Meyer Children's Hospital IRCCS, Firenze, Italy and Neurosciences, Psychology, Drug Research and Child Health (NEUROFARBA) Department, University of Florence, Florence, Italy); Valerio Conti (Neuroscience and Human Genetics Department, Meyer Children's Hospital IRCCS, Firenze, Italy)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 15.12 - HS

TRACK Neuroscience and Neural Systems

Abstract

Chemical Reaction Networks (CRNs) provide a rigorous mathematical framework to represent molecular interactions along signaling pathways by large systems of ordinary differential equations, exploiting the mass-action law. This framework enables a deeper understanding of the mechanisms underlying different diseases by analyzing the dynamics of the concentrations of the involved chemical species [SCP20], capturing the steady-state behaviors through specific accurate root-finding methods [Ber+24], and observing the perturbation effects induced by genetic mutations and pharmacological treatments by implementing their action on the network [Som+23]. We will present an application of this approach to the PI3K-AKT-mTOR axis to investigate the synergistic effect of two somatic activating mutations - MTOR p.S2215F and RPS6 p.R232H - documented in a patient with brain malformations and drug-resistant epilepsy [Pel+19]. Single and combined mutations are implemented as parameter perturbations within the CRN to capture how altered signaling dynamics propagate along the pathway and shift the system toward pathological steady states. Furthermore, we simulate pharmacological inhibition via mTOR inhibitors to explore whether and how their administration attenuates the dysregulation of key cellular pathways that may be involved in aberrant neuronal migration, proliferation, and electrical activity, potentially providing new therapeutic avenues for patients refractory to conventional anti-epileptic drugs.

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MS147 — Plant models: mechanics, development and environment

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 02.01 - HS
ORGANISERS Eva Deinum (Wageningen University, Netherlands); Lionel Dupuy (Department of Conservation of Natural Resources, Neiker & IKERBASQUE, Basque Foundation for Science, Bilbao, Spain); Mariya Ptashnyk (Heriot-Watt University, Edinburgh, Scotland, UK)

Minisymposium abstract

Plants now lie at the core of strategies aimed at addressing major global challenges, from ensuring food security and capturing atmospheric carbon to preserving soil quality and halting desertification. At the same time, plant production systems are becoming more vulnerable to the impacts of climate change, including rising temperatures, altered precipitation patterns, and increased frequency of extreme

events. Meeting such challenges is not possible without a fundamental understanding of the biological and physical mechanisms that regulate plant growth and development, as these processes determine how plants respond to environmental constraints and how effectively they can be optimized for future agricultural and ecological demands. The objective of this symposium is to showcase recent theories and tools to describe the physics of plant growth, from single cells to the development of complex shapes, and the interactions between plants and their environment.

MS147-01 — Going underground: roots and rhizosphere hydraulic processes impacting plant response to drought

SPEAKERS Mutez Ahmed (Root-Soil Interaction, School of Life Sciences, Technical University of Munich, Freising, Germany)
AUTHORS Asegidew Akale (Root-Soil Interaction, School of Life Sciences, Technical University of Munich, Freising, Germany); Efstathios Diamantopoulos (Chair of Soil Physics, University of Bayreuth, Bayreuth, Germany); Frederic Leuther (Chair of Soil Physics, University of Bayreuth, Bayreuth, Germany); Gaochao Cai (School of Agriculture and Biotechnology, Shenzhen Campus of Sun Yat-sen University, Shenzhen, China); Lara Kersting (Chair of Soil Physics, University of Bayreuth, Bayreuth, Germany); Mutez Ahmed (Root-Soil Interaction, School of Life Sciences, Technical University of Munich, Freising, Germany); Scott McAdam (Department of Botany and Plant Pathology, Purdue University, West Lafayette, USA); Shurong Liu (School of Agriculture and Biotechnology, Shenzhen Campus of Sun Yat-sen University, Shenzhen, China)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Plants respond to soil and atmospheric water deficits through strategies such as stomatal regulation and belowground adaptations. Root mucilage buffers erratic fluctuations in the rhizosphere water content, yet its influence on soil hydraulic properties, especially unsaturated hydraulic conductivity, and stomatal regulation remains unknown. We hypothesized that mucilage facilitates water uptake by attenuating the drop in matric potential at the root–soil interface during soil and atmospheric drying. We measured the impact of various maize (*Zea mays*) mucilage contents (0.0%, 0.05%, 0.2%, and 0.4%) on the water retention and hydraulic conductivity of a loamy soil. Leveraging a soil–plant hydraulic model, we investigated the effects of mucilage contents on transpiration and stomatal responses under soil drying and increased vapor pressure deficit (VPD). Higher mucilage contents prevented sharp declines in unsaturated hydraulic conductivity as soils dried. Simulations revealed that higher mucilage contents delayed the onset of hydraulic stress (the threshold transpiration rate beyond which a small increase in transpiration would result in a disproportionate decline in leaf water potential), broadened the hydroscape zone, and shifted stomatal behavior from isohydric to more anisohydric regulation, enabling plants to sustain stable transpiration and lower midday leaf water potentials under drought. The buffering effects on soil–plant hydraulics persisted across varying degrees of VPD, although high mucilage contents accelerated soil drying, indicating a trade-off between improved water uptake and faster moisture depletion during prolonged drought. Our findings underscore the important role of mucilage in modulating soil–plant water relations and stomatal regulation, offering insights into strategies for improving plant responses to soil and atmospheric drought.

MS147-02 — Geometry hidden in plain stress, Exploring the relationship between pressure-induced stresses and geometry in plant tissues.

SPEAKERS Olivier Ali (Laboratoire Reproduction et Développement des Plantes (RDP), Université de Lyon, ENS de Lyon, Inria)
AUTHORS Olivier Ali (Laboratoire Reproduction et Développement des Plantes (RDP), Université de Lyon, ENS de Lyon, Inria)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Mechanical stresses play a central role during the morphogenesis of multicellular structures. Not only do they generate tissue deformations but they also provide cells with signals triggering differentiation and pacing development. This is especially true in growing plant epithelia where turgidity generates

tremendous stresses within cell walls. From a systematic perspective, one can wonder what kind of signalling cues, turgor-induced stresses can provide growing cells with? In this presentation, we will first see how, in the case of symmetric and closed epithelia, such stresses can be related to a specific kind of geometrical descriptors: Killing vectors fields. We will then relax the symmetry assumption and evoke how this connection can be extended to pseudo Killing fields.

MS147-03 — Fluid mechanics of the plant cell wall: from cellulose reorientation to twisting growth

SPEAKERS Galane Luo (School of Mathematics, University of Birmingham, UK)
AUTHORS Galane Luo (School of Mathematics, University of Birmingham, UK); Rosemary Dyson (University of Birmingham); Tobias Baskin (Biology Department, University of Massachusetts)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Twisted shapes in plants, seen in helical roots, spiral grains and climbing vines, are both ubiquitous and consequential. They affect crop yield, impact lumber quality, and inspire biomimetic robotics. Understanding their mechanical origins begins at the single-cell level. The cell wall is a complex material: a pectin matrix reinforced by cellulose microfibrils that dynamically reorient during deformation. Under constant turgor pressure, anisotropic wall extension drives growth. We combine theories of transversely isotropic fluids, pressure-driven viscous sheets, and dynamic fibre-reorientation, developing a model for helical cell wall extension. The talk will present the model and semi-analytical solutions, including recent results on an internalised control mechanism for the growth and twist rates, and a generalised Lockhart equation that relates cell morphology to cellulose dynamics. This framework provides a key step towards explaining and controlling twisting morphology at the tissue and organ scales, which in turn underpins food security, sustainable development, and bio-inspired design.

MS147-04 — The biomechanical basis of fixed handedness coiling in Cardamine fruit valves

SPEAKERS Gabriella Mosca (Center for Plant Molecular Biology, University of Tuebingen, DE)
AUTHORS Friederike Brand (University of Tübingen); Gabriella Mosca (Center for Plant Molecular Biology, University of Tuebingen, DE)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

While the rest of the plant does not show any twisting or coiling, the valves of *Cardamine hirsuta* coil with fixed right-handedness when released from the rest of the fruit at the end of its maturation. Through multi-scale biomechanical modeling of the valve, supported by live confocal imaging data of cortical microtubules (CMT), we show how CMT dynamics in the final phases of valve maturation can explain the coiling process. By coupling organ-level to tissue-level behaviour, we show the effect of a multi-layered cell wall structure and which hypotheses are required on the cell wall mechanical properties to quantitatively fit the coiling. Finally, we investigate the range of forces in the cell wall and tension on individual CMTs while the epidermal cells of the valve undergo a dramatic cytoskeletal reorientation from transverse to longitudinal — pivotal for the entire process — speculating on the possible causes of the consistent angular tilting ultimately achieved by the CMT.

References

[Hof+16] Hugo Hofhuis et al. “Morphomechanical innovation drives explosive seed dispersal”. In: *Cell* 166.1 (2016), pp. 222–233.

[Mos+24] Gabriella Mosca et al. “Growth and tension in explosive fruit”. In: *Current Biology* 34.5 (2024), pp. 1010–1022.

MS147-05 — Three-dimensional multiscale modelling of plant tissue mechanobiology

SPEAKERS João R. D. Ramos (Mathematical Institute, Leiden University, NL)
AUTHORS João R. D. Ramos (Mathematical Institute, Leiden University, NL); Roeland M. H. Merks (Mathematical Institute, Leiden University)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

The genetic underpinnings that define tissue shape and function are still an open question. Part of the answer is in the biomechanics of cells and tissues and in the cross-talk between biomechanics and biochemistry. In particular, plants exhibit differential growth mediated by the viscous and plastic properties of the cell wall and the spatial distribution of chemical patterns. We are working on a 3D version of the plant modelling package VirtualLeaf, i.e., a quantitative multiscale three-dimensional vertex model of plant development. The final goal is to combine cell wall mechanics with known models of growth, genetic regulation, and water transport, to model plant tissues developing through regulated cell divisions and growth. We will present first results, in which we validate a simulation of uniaxial cell elongation with well-established models and solutions (the Lockhart equations). We will show that our model is capable of capturing the mechanical frustration arising naturally from models of plant cell growth, when accounting for spatial heterogeneity of mechanical parameters and multicellularity. We also explore the possible three-dimensional structures arising from different cells division rules, and whether the three-dimensionality and cell division rules change known relationships between cell volume, pressure and number of neighbours, typically measured only on the epidermis.

MS147-06 — Impact of cellular geometry on the effective mechanics of growing plant tissues

SPEAKERS Annamaria Kiss (RDP, ENS de Lyon, Claude Bernard University Lyon 1, CNRS, INRAE, INRIA, Lyon, France)
AUTHORS Arezki Boudaoud (LadHyX, CNRS, Ecole Polytechnique, IP Paris, Palaiseau, France); Annamaria Kiss (RDP, ENS de Lyon, Claude Bernard University Lyon 1, CNRS, INRAE, INRIA, Lyon, France); Mariya Ptashnyk (Department of Mathematics, Heriot-Watt University, The Maxwell Institute for Mathematical Sciences, Edinburgh, Scotland, UK)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

How morphogenesis at a tissue level depends on cellular properties is an active direction of research. We focus on mechanical models of growing plant tissues, where cells are pressurized, a rigid cell wall resists, and the resulting elastic strain or stress generates growth in the walls. In order to establish links between microscopic and macroscopic properties, we adopt here the results of a multiscale analysis that allows us to rigorously derive by homogenization the corresponding macroscopic tissue scale model. Here we consider isotropic cell walls in two dimensions and study the effect of cell shape and cell packing on tissue-level growth. In addition to the homogenized elasticity tensor, particular attention is given to the rôle of the tissue level second order tensor, which describes how turgor pressure in cells contributes to macroscopic stress. We use the macroscopic model to make predictions about several configurations of interest, both in stress-based and strain-based growth hypotheses.

References

[BKP23] Arezki Boudaoud, Annamaria Kiss, and Mariya Ptashnyk. “Multiscale Modeling and Analysis of Growth of Plant Tissues”. In: *SIAM Journal on Applied Mathematics* 83.6 (2023), pp. 2354–2389. DOI: 10.1137/23M1553315. URL: https://doi.org/10.1137/23M1553315.

MS147-07 — Busse balloon Barbapapa

SPEAKERS Eric Siero (Mathematical and Statistical Methods – Biometris, Wageningen University & Research, NL)
AUTHORS Eric Siero (Mathematical and Statistical Methods – Biometris, Wageningen University & Research, NL); Hannes Uecker (University of Oldenburg)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Spatial self-organisation arises in many contexts. A classical example is Rayleigh-Bénard convection: when the temperature between a heated bottom plate and a cooler top plate grows beyond a certain threshold, convection cells form. Busse balloons are a (graphical) representation of these patterns. A prominent example in ecology is dryland vegetation. If yearly precipitation falls below some critical value, vegetation patterns emerge with a wavelength of tens of meters. These patterns have paradoxically been interpreted as both 1) early-warning signals of complete desertification and 2) a way of the ecosystem to adapt to drought. In this talk, we will include grazing in a Klausmeier-type reaction-diffusion model and see how the shape of the corresponding Busse balloon changes. The shape of the Busse balloon is then used to infer ecosystem response to increased drought.

MS147-08 — Understanding the Biomechanics of Plant Tissues: the consequences of cell division, shape and arrangement

SPEAKERS Euan Smithers (Sainsbury Laboratory, University of Cambridge, UK)
AUTHORS Elise Laruelle (Sainsbury Laboratory, University of Cambridge); Euan Smithers (Sainsbury Laboratory, University of Cambridge, UK); Leo Serra (Sainsbury Laboratory, University of Cambridge); Mahwish Ejaz (Sainsbury Laboratory, University of Cambridge); Sarah Robinson (Sainsbury Laboratory, University of Cambridge)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 02.01 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

How would you build a plant? Where to place the cell walls? Does this even matter? Due to turgor pressure, plant cell walls must resist substantial tensile stresses, and if not managed properly, they can lead to structural damage or an ineffective use of resources. Since plant cells are rigidly connected to one another, to resist this mechanical stress, precise control over the placement of new cell walls is vital for developing effective and efficient tissues. To explore how plants manage these stresses, we use interdisciplinary methods, such as mechanical perturbations on live tissues using an extensometer and performing finite element inflation simulations of different deformations. For this purpose, we have built up and improved existing modelling software to simulate plant tissues in 3D efficiently and with multiple layers. Through such methods, we have investigated the consequences of cell shapes and tissue structure across multiple scales. Our findings offer new insights into the role of cell division patterns, the prevalence of 3-way junctions (staggered like bricks in a wall), and why plant cells take certain shapes like rectangles. This research advances our understanding of how plants sense and respond to mechanical forces in their environment.

MS156 — Stochastic Dynamics and the Realities of Experimental Observation

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Thursday, 16 July 2026; 17:00–11:40; 15.06 - HS
ORGANISERS John Fricks (Arizona State University); Scott McKinley (Tulane University)

Minisymposium abstract

Data collection for biological systems has undergone a revolution over the past few decades with electronic trackers for wildlife populations giving us minute to minute information about their locations and fluorescence data at the cellular level tracking the number and location of proteins. On the theory side,

we have well-known population models for the interaction of wildlife and also for intracellular interactions. A difficulty in practice is the divide between the collected data and the idealized models used to represent the relevant biological systems, models which can bring real insight into the biological mechanisms at play, but with the need to be calibrated to the data. The realities of experimental observation can include data on a coarser scale than the one of interest for the model, data collected at uneven time steps, or observational systems that can actually affect the system of interest, such as with laser traps tracking molecular motors. Some of this separation can be handled by improved computational tools, such as improvements in stochastic simulation and statistical tools that rely on stochastic simulation. However, unifying approaches would aid in linking the vagaries of experimental data to theoretical model; this mini-symposium will present some approaches to handling these complexities by bringing together researchers with complimentary perspectives across statistics, stochastic modeling, and machine learning.

MS156-01 — Modeling mechanisms of neuronal microtubule dynamics and polarity

SPEAKERS Anna Nelson
AUTHORS Anna Nelson
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Microtubules are protein polymers comprised of tubulin, which are polarized and facilitate intracellular transport. A stable microtubule structure is important to ensure the long-term survival of neurons. However, microtubules also need to be dynamic and reorganize in response to injury events, which results in an increase in microtubule dynamics. How this complex balance is achieved on different scales remains an open question. Using experimental data and a stochastic mathematical model that limits microtubule growth, we seek to understand how nucleation, or new microtubules, can impact microtubule dynamics in dendrites of fruit fly neurons. We develop a partial differential equation (PDE) model that describes microtubule growth and nucleation dynamics to gain analytical insight to our stochastic model, and we compare analytical results to results from the complex stochastic model. Insights from these models can then be used to understand how microtubules can organize into polarized structures in neurons, where several mechanisms have been hypothesized to regulate microtubule polarity organization. Guided by experimental results, we implement our stochastic model with spatial polarity mechanisms to understand the efficacy of proposed biological mechanisms at establishing and maintaining the microtubule polarity observed in healthy neurons. Finally, we utilize our framework to explore mechanisms important in injury, where microtubule polarity is known to reverse.

MS156-02 — Three-Dimensional Modeling of Kinesin Mechanics in Optical Traps

SPEAKERS Christel Hohenegger (University of Utah)
AUTHORS Christel Hohenegger (University of Utah)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mechanical properties of molecular motors such as kinesin are often measured using optical traps. We develop three-dimensional stochastic models based on force and torque balances for two experimental setups: the single-bead assay and the three-bead assay. Our goal is to provide insight into how modeling choices in force–velocity and force–detachment relationships influence motion along the microtubule, and how these choices may fail to accurately represent experimentally observed displacements. We identify key misconceptions, particularly relating to the detachment times that arises when out-of-plane components are neglected. Finally, we discuss the limitations of these models and their discrepancies with experimental data.

MS156-03 — Analyzing Partially Observed Stochastic Epidemic Networks

SPEAKERS Grzegorz Rempala (The Ohio State University)
AUTHORS Grzegorz Rempala (The Ohio State University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Understanding how epidemics spread on contact networks when the data are incomplete remains one of the central challenges in mathematical epidemiology. In this talk, I will describe a framework for analyzing stochastic epidemic models under partial observation, combining ideas from dynamical survival analysis (DSA), pairwise survival models, and recent exact closure results for SIR dynamics on random networks.

The starting point is to use DSA to estimate effective reproduction parameters directly from observed infection and recovery times, without requiring full knowledge of the underlying contact structure. These estimates are then combined with pairwise survival models, following Kenah and collaborators, to account for local dependence and correlations between connected individuals. To connect these approximations to large-network behavior, I will also discuss recent results on exact closure for configuration-model epidemics.

Taken together, these elements provide a tractable and interpretable approach to inference and uncertainty quantification in network-based epidemic models.

MS156-04 — Generative models for particle tracking microscopy

SPEAKERS Jay Newby (University of Alberta)
AUTHORS Jay Newby (University of Alberta)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

I will discuss the use of diffusion models for particle tracking microscopy images. The goal is to have a fully integrated generative model that connects stochastic models of particle motion to microscopy image data. Unfortunately, the observation likelihood function is too complex to explicitly model, and we do not know this function. Learning the likelihood function from a suitably large image set is the primary purpose of so-called diffusion models. We discuss the application of these neural network models for evaluating the log likelihood of image sets given particle positions.

MS156-05 — Parameter Estimation for ODEs, Curve Registration, and Particle Filtering.

SPEAKERS John Fricks (Arizona State University)
AUTHORS John Fricks (Arizona State University)
SCHEDULE Friday, 17 July 2026; 10:40–11:20; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

A common challenge in mathematical biology arises from data collected from a biological system for which we have a mechanistic model, such as an ODE model. Identifying the parameters of the model is often done through estimation schemes such as non-linear least squares, which presume errors in the amplitude of the data. Such estimation can be challenging, and other sources of error, such as deviations in phase, might also be considered. Relatedly, curve registration is a set of techniques in the statistical analysis of functions to address variations in phase across a collection of observed curves. In this talk, we will present techniques to apply methods of curve registration to estimation of parameters from ordinary differential equations models. We will show how particle filtering methods can facilitate such inference in a computationally tractable way and allow us to quantify the phase variability in such data. A brief

discussion of vaccine trials will be included to illustrate how characterizing such phase variability can give new insights into inter-subject biological variation.

MS156-07 — Stochastic fountain dynamics and associated challenges for inference

SPEAKERS Scott McKinley (Tulane University)
AUTHORS Scott McKinley (Tulane University)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 15.06 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In the last couple of years, I have noticed an emerging theme in my work. Across multiple biological systems, colleagues and I have articulated models that involve particles that (1) emerge at random times from a fixed source-location distribution; (2) move throughout a local environment randomly (either diffusing, or switching between deterministic states); and (3) are removed from the system due to state-switching or escape from some predefined region.

We have been tentatively calling these systems "stochastic fountains" (in the classic Markov chain literature these are called "open systems") and have been studying what these systems look like when you only have access to partial information. For example, what if you only have a snapshot of particles at one instant in time? Or, what happens if you can only observe particles at the moment they leave the domain? The associated inference problems arise naturally in mathematical biology applications, and I will give an overview of how they sit at an interesting intersection of stochastic processes, PDE-inverse theory, spatial point processes, and asymptotic statistics.

MS166 — Mathematical modelling of telomere dynamics

TRACK Cellular and Developmental Biology
SCHEDULE Thursday, 16 July 2026; 17:00–18:20; 02.23 - HS
ORGANISERS Coralie Fritsch (Inria); Marie Doumic-Jauffret (Inria and Ecole polytechnique)

Minisymposium abstract

This mini-symposium brings together researchers from different groups, from USA, Spain and France, all working on the mathematical modeling of telomere dynamics. It will give a unique opportunity to compare and discuss different approaches on close subjects : parameter inference of telomere length regulation in yeast (Viviana Gavilanes); stochastic models of telomere shortening and reconstruction, and their connections to carcinogenesis (Marek Kimmel) on the one hand, and on the emergence of ALT (Alternative Lengthening of Telomeres) survivors (Juan Mardomingo Sanz), a critical area for understanding cellular immortality on the other hand. Finally, Ana-María Portillo della Fuente will explore the role of oxidative stress in aging and its impact on female fertility, integrating modeling approaches with physiological outcomes.

References

[Ben+24] Athanase Benetos et al. "Stochastic branching models for the telomeres dynamics in a model including telomerase activity". In: *arXiv preprint arXiv:2407.11453* (2024).

[LK20] Kyung Hyun Lee and Marek Kimmel. "Analysis of two mechanisms of telomere maintenance based on the theory of g-Networks and stochastic automata networks". In: *BMC genomics* 21.Suppl 9 (2020), p. 587.

[PGV25] AM Portillo, JA García-Velasco, and E Varela. "Influence of oxidative stress on women's fertility: A model with a generational age Caputo's fractional derivative". In: *BioSystems* (2025), p. 105577.

[PVG23] Ana M Portillo, Elisa Varela, and Juan A García-Velasco. “Influence of telomerase activity and initial distribution on human follicular aging: Moving from a discrete to a continuum model”. In: *Mathematical Biosciences* 358 (2023), p. 108985.

[Rat+25] Anaïs Rat et al. “Mathematical model linking telomeres to senescence in Saccharomyces cerevisiae reveals cell lineage versus population dynamics”. In: *Nature Communications* 16.1 (2025), p. 1024.

MS166-01 — Modeling the role of oxidative stress in aging and its impact on female fertility

SPEAKERS Ana Portillo (University of Valladolid)
AUTHORS Ana Portillo (University of Valladolid); Elisa Varela; Juan Antonio García-Velasco
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Cellular aging associated with telomere shortening plays a crucial role in female fertility. Beyond the natural decline caused by the loss of telomeric repeats during cell division, additional factors such as oxidative stress (OS) can accelerate telomere erosion by inducing substantial telomeric damage. Despite its biological relevance, mathematical modeling of accelerated aging mechanisms leading to infertility remains limited in the literature.

An initial-boundary value problem based on a diffusion-advection equation has previously been proposed in [PVG23] to describe the evolution of a cell population experiencing a gradual reduction in proliferative potential due to the end-replication problem. Subsequently, we introduced a continuum model that accounts for random telomere shortening induced by OS in [PGV25] by replacing the advection term with a Caputo fractional derivative of order β, $0 < \beta < 1$, with respect to the generational age. The deviation of the fractional order from unity is interpreted as an oxidation parameter.

The model is applied to human follicular development from the preantral to the pre-ovulatory stage in both young and older women, allowing us to investigate the combined effects of oxidative stress and reduced telomerase activity. Our results show that increasing oxidative stress leads to an increase in the generational age of granulosa cells, indicating accelerated telomere aging.

References

[PGV25] AM Portillo, JA García-Velasco, and E Varela. “Influence of oxidative stress on women’s fertility: A model with a generational age Caputo’s fractional derivative”. In: *BioSystems* (2025), p. 105577.

[PVG23] Ana M Portillo, Elisa Varela, and Juan A García-Velasco. “Influence of telomerase activity and initial distribution on human follicular aging: Moving from a discrete to a continuum model”. In: *Mathematical Biosciences* 358 (2023), p. 108985.

MS166-02 — Stochastic modelling of telomere length dynamics in cell populations with ALT survivors

SPEAKERS Juan Mardomingo-Sanz (University of Lorraine)
AUTHORS Coralie Fritsch (Inria); Denis Villemonais (University of Strasbourg and IUF); François Laffont (CRCM-CNRS); Juan Mardomingo-Sanz (University of Lorraine); Marie-Noëlle Simon (CRCM-CNRS); Nicolas Champagnat (Inria)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Telomeres are repetitive sequences of DNA at the end of linear chromosomes. Their length decreases at each cell division, leading to replicative senescence in the absence of elongation mechanisms. In some unicellular organisms, as the yeast Saccharomyces Cerevisiae, telomere length homeostasis is maintained by the enzyme telomerase [MBT25]. In liquid dilution experiments of yeasts with inactivated telomerase,

emergence of cells presenting an alternative recombination-based mechanism (ALT) is observed after replicative senescence. The origin and dynamics of ALT cells are not yet well understood [KCM21] and the goal of this talk is to present a stochastic modelling approach to characterize the time of emergence of ALT cells and the variability of their telomere distribution. An asymptotic analysis under a relevant parameter scaling, inspired by [CMT21], allows us to account for experimental observations.

References

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[KCM21] Zachary W. Kockler, Josep M. Comeron, and Anna Malkova. "A unified alternative telomere-lengthening pathway in yeast survivor cells". In: *Molecular Cell* 81.8 (2021), 1816–1829.e5. ISSN: 1097-2765. DOI: https://doi.org/10.1016/j.molcel.2021.02.004. URL: https://www.sciencedirect.com/science/article/pii/S1097276521000903.

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MS166-03 — Stochastic Modeling of Telomere Shortening and Reconstruction and Connections with Carcinogenesis

SPEAKERS Marek Kimmel (Rice University)
AUTHORS Leonard Mauvernay; Marek Kimmel (Rice University); Marie Doumic-Jauffret (Inria and Ecole polytechnique); Teresa Teixeira (CNRS and Sorbonne Université); Yann Lustig (Sorbonne Université)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

We present stochastic models of growth of a cell population of cultured yeast cells with gradually decaying chromosome endings, called the telomeres. The model has the form of the age-dependent Markov branching process with doubly-denumerable type space, where the type of a cell is defined as the pair of integers representing the length of telomeres at both ends of a chromosome. We derive the forward and backward equations for the generating functions characterizing the process, and find that their general solutions. We derive monet equations and consider special cases with senescence and cell death. These computations as well as simulations suggest instability of telomere lengths close to their disappearance (cells' senescence).

We further consider models involving cell death and the ALT mechanism of telomere reconstruction, which include connections with slightly supercritical branching processes conditional on non-extinction. We illustrate the theory with examples from our own experiments as well as from recent literature. We further explain how models of the so-called "branching within branching" link telomere dynamics to human carcinogenesis.

MS166-04 — Mathematical modeling and parameter inference of telomere length regulation in yeast

SPEAKERS Viviana Gavilanes Guerrero (Sorbonne University)
AUTHORS Marie Doumic-Jauffret (Inria and Ecole polytechnique); Viviana Gavilanes Guerrero (Sorbonne University); Zhou Xu
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Telomere length regulation in *Saccharomyces cerevisiae* has been modeled through stochastic rules combining deterministic shortening and telomerase elongation. We provide a formulation of the single-telomere dynamics as a discrete-time Markov chain depending on parameters controlling shortening, telomerase activation and elongation. On a finite truncated state space, we prove ergodicity and obtain a unique stationary distribution with an explicit spectral characterization, yielding a deterministic forward map from parameters to observables. This approach replaces costly stochastic simulations by an efficient deterministic computation. Parameter identifiability is addressed through a generating-function identity expressing the stationary distribution in terms of the telomerase activation probability. Parameter estimation is performed by minimizing the 1-Wasserstein distance between theoretical and Nanopore empirical distributions, using the CMA-ES optimization algorithm. A discrete-to-continuous scaling limit leads to an integro-differential equation.

MS169 — Mathematical Modeling of Infectious Diseases: Classical Foundations to Contemporary Approaches

TRACK Mathematical Epidemiology
SCHEDULE Thursday, 16 July 2026; 17:00–11:40; 15.02 - HS
ORGANISERS Prashant Kumar Srivastava (Indian Institute of Technology Patna); Binod Pant (Northeastern University, USA); Meredith Greer (Bates College , USA)

Minisymposium abstract

Work within the mathematical epidemiology subgroup focuses on critical questions about the emergence, spread, and control of infectious diseases at multiple scales, and to study these questions, we must develop and implement a variety of tools. In this mini-symposium, we feature work across a broad spectrum of infectious disease modeling research and highlight work that members of the SMB Mathematical Epidemiology subgroup have been doing over the past year. This minisymposium will feature work addressing issues in vector borne diseases and their epidemiology, age-targeted vaccination, co-infection of plants and models with Generative-AI driven agents, among other important topics, and feature the work of a diverse group of mathematical biologists who are implementing traditional and novel methods to study questions in mathematical epidemiology.

The titles of each contributor is provided in the comment.

MS169-01 — Optimizing HIV Antiretroviral Therapy Strategies: A Multi-Scale, Multi-Objective Framework for Reconciling Individual–Population Trade-offs

SPEAKERS Stacey Smith? (University of Ottawa)
AUTHORS Stacey Smith? (University of Ottawa)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Existing single-scale HIV models often overlook conflicts between within-host and population-level treatment strategies. To resolve this, we develop a multi-scale model with infection-age structure to optimize antiretroviral therapy (ART) initiation timing and efficacy through multi-objective optimization. By reformulating initiation time as a control variable in the system, we establish the existence of Pareto-optimal solutions through variational analysis and provide a theoretical characterization of the optimal treatment schedule. Numerical analyses reveal that optimal ART strategies must dynamically adapt to evolving priorities between individual outcomes and population-level benefits: early high-efficacy ART maximizes individual benefits, while delayed initiation optimizes population outcomes under persistent post-treatment risks. Post-treatment behavioural shifts influence this balance: reduced sexual activity supports earlier initiation, while high-risk populations require precisely timed delayed administration to

balance transmission control and treatment costs. This framework provides quantitative principles for reconciling scale-specific treatment priorities.

MS169-02 — The population-level implications of immune boosting and immunological priming

SPEAKERS Nir Gavish (Technion Israel Institute of Technology)
AUTHORS Guy Katriel (Braude College, Israel); Nir Gavish (Technion Israel Institute of Technology); Zohar Rom Ramon (Technion Israel Institute of Technology, Israel)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Vaccine-induced immunity is rarely static; rather, an individual's immune state is fundamentally shaped by their unique history of pathogen exposures. Silent exposures to the pathogen may lead to "immune boosting," reinforcing immunity without causing symptomatic infection. Moreover, because a primed immune system can respond to a lower dose of antigen than a naive one, immune boosting can be triggered by exposures that are too weak to lead to productive infection. Recent analyses of pertussis dynamics suggest that such extensive immune priming has significant implications for population-level vaccine effectiveness. Nevertheless, the population-level implications of immune boosting and immunological priming are not well understood and remain largely unexplored.

In this work, we incorporate a 'strong' form of immune boosting and the mechanism of immunological priming into a leaky-vaccine SIR model. Our analysis reveals a fundamental relationship between the individual probability of boosting and the population-level impact of vaccination. As expected, we show that immune boosting enhances vaccine effectiveness in a wide range of scenarios. Surprisingly, we demonstrate that when immune priming is extensive, vaccine effectiveness can paradoxically decrease with increasing vaccine coverage. Furthermore, we show that in high-transmission settings, VE becomes independent of vaccine coverage. We elucidate these counterintuitive behaviors using a complementary analysis of repeated pathogen exposures and an analysis of direct and indirect effects. Our work sheds light on the complex interplay between individual immune history and herd immunity, emphasizing the importance of accounting for immunological priming when evaluating vaccine impact.

MS169-03 — A Hybrid Networked SEIR Model with Generative-AI Driven Agents for Behavioral Heterogeneity in Epidemics

SPEAKERS Jia Zhao (The University of Alabama, USA)
AUTHORS Jinming Wan (State University of New York at Binghamton); Md Obaidul Haque (University of Alabama); Kaitlin Ho (State University of New York at Binghamton); James Giuffre (State University of New York at Binghamton); Changqing Cheng (State University of New York at Binghamton); Jia Zhao (The University of Alabama, USA)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

In this paper, we develop a hybrid, networked SEIR framework that integrates generative AI-driven agents to capture individualized protective behavior. Each agent is characterized by demographic and socioeconomic attributes, and a large language model (LLM) generated daily willingness-to-comply scores from prompts that encode personal traits, occupation, and income, local and global epidemic conditions, social influence, and policy strength. These AI-generated behavioral states modulate edge-level infection risk on dynamic physical contact networks, thereby linking individual decision-making to population-level transmission outcomes. We further embed the same behavioral mechanism into empirically measured temporal contact networks from six real-world scenarios (conference, hospital, workplace, high school, primary school, and college campus).

MS169-04 — The young shield: Mathematical modeling of the impact of age-targeted and dose-structured vaccination on malaria dynamics

SPEAKERS Arnaja Mitra (University of Maryland)
AUTHORS Abba B. Gumel (University of Maryland); Arnaja Mitra (University of Maryland)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Malaria, a parasitic disease spread to humans via effective bite by an infectious adult female Anopheles mosquito, continues to exude a major burden in endemic areas (causing in excess of 600,000 deaths annually, mostly in children under the age of five). Much progress was made over the last two or three decades in the fight against malaria, largely due to the heavy and large-scale use of chemical insecticides (particularly in the form of long-lasting insecticidal nets and indoor residual spraying) to kill the malaria mosquito, promoting a renewed quest for malaria eradication. Unfortunately, such heavy use has also resulted in widespread Anopheles resistance to all the main chemical insecticides used in vector control, posing challenges to the eradication objective. New anti-malaria vaccines have been approved recently and are being deployed in a number of countries in sub-Saharan Africa. In this talk, I will present a new mathematical model, in the form of a system of delayed-differential equations, for assessing the population-level impact of one of the approved vaccines (R21/Matrix-M vaccine) in curtailing the disease burden in the targeted (vaccinated) population.

MS169-05 — Optimal release of sterile males in the sterile insect technique for Anopheles mosquitoes

SPEAKERS Clarke Safsten (University of Maryland)
AUTHORS Abba Gumel (University of Maryland); Clarke Safsten (University of Maryland)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

The sterile insect technique (SIT) is a promising strategy for controlling malaria-transmitting Anopheles mosquitoes, but its practical implementation hinges on identifying release protocols that are both effective and resource-efficient. In this talk, I present a mathematical framework for optimizing time-dependent releases of sterile male mosquitoes within a biologically structured population model incorporating nonlinear mating dynamics and life-cycle stages. A key feature of the model is the emergence of an Allee effect induced by mate-finding limitations, creating a threshold below which the wild population collapses naturally. Building on this, I formulate a free-horizon optimal control problem that minimizes the total number of sterile males released while ensuring the population is driven below this threshold. Unlike classical approaches with a fixed terminal time, our formulation uses a state-dependent stopping time corresponding to threshold crossing. I discuss analytical and numerical challenges arising from the geometry of the threshold manifold and the coupling between control intensity and time to elimination. Preliminary results suggest adaptive release strategies can substantially reduce total releases compared to standard protocols, and I explore how mechanisms such as introducing a competing species may further improve SIT efficiency.

MS169-06 — Incorporating thermal performance curves into population dynamic models for the West Nile vector, Culex pipiens

SPEAKERS Ben Bruncati (Virginia Tech, USA)
AUTHORS Ben Bruncati (Virginia Tech, USA); Helle Aronson (Virginia Tech); Chloé Lahondère (Virginia Tech); Michael Robert (Virginia Tech)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Culex mosquitoes are a growing concern in North America given their ability to transmit diseases such as West Nile Virus and adapt their thermal tolerances. *Culex pipiens*, one species found in temperate environments, is predicted to continue to spread north and south of the equator as environmental conditions become more favorable. As climate change persists, it is of high importance to understand how temperature influences *Culex pipiens* life-history traits and what impacts this might have on transmission of West Nile and other pathogens. In this work, we explore potential functional forms of thermal performance curves (TPCs) for key traits of *Culex pipiens* by fitting TPCs to temperature-dependent trait data. We found the best-fitting TPCs for the following traits: age-specific mortality, fecundity, juvenile development, and survival to adulthood. We then incorporated TPCs for each trait as a parameter function in a system of stage-structured differential equations of *Culex pipiens* population dynamics and we explored potential impacts on population dynamics of different TPC curves. Separately toggling both rates yielded notable differences in projected population counts and predicted peaks. We also compared the adult population projections from our model to surveillance data to find the best fitting model and see which fits were strongly supported by the data. Mathematical models such as ours are useful for predicting the potential population dynamics of *Culex pipiens* in specific geographic regions and for informing public health interventions. Further investigation is needed from additional data collection to explore other potential TPCs which may help to further improve these mathematical models.

MS169-08 — The effect of co-infection on disease transmission in plants with multiple transmission pathways

SPEAKERS Hannah Hirsch (Osnabrück University, Germany)
AUTHORS Frédéric M. Hamelin (Univ Rennes, France); Frank M. Hilker (Osnabrück University, Germany); Hannah Hirsch (Osnabrück University, Germany)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 15.02 - HS
TRACK Mathematical Epidemiology

Abstract

Co-infection of hosts by multiple pathogens can significantly increase disease prevalence and severity. In plant hosts, virus infection typically occurs via multiple transmission pathways. Here, we consider vertical transmission (via seeds, typically between growing seasons) and horizontal transmission (via insect vectors, typically within growing seasons). An open question is whether and how co-infection affects virus transmission, particularly seed transmission. We therefore investigate how the virus spread is affected by increased or decreased seed transmission of co-infected plants, compared to singly infected ones, in a semi-discrete model. A bifurcation analysis reveals that there can be two different types of bistability: (1) between a disease-free and a co-infection equilibrium, and (2) between the two boundary equilibria corresponding to singly infected plants. We interpret these results biologically and discuss their implications for plant disease management.

MS172 — Discrete frameworks for modeling biological systems

TRACK Systems Biology and Biochemical Networks
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 11.01 - HS
ORGANISERS Brandilyn Stigler (Southern Methodist University); Claus Kadelka (Iowa State University); David Murrugarra (University of Kentucky); Elena Dimitrova (Cal Poly)

Minisymposium abstract

As biological data grows in both scale and complexity, discrete modeling methods are valuable complements to classical continuous frameworks as they connect diverse areas such as algebra and probability theory to discrete approaches. This minisymposium explores recent developments and challenges in discrete formalisms including Boolean networks, Petri nets, and polynomial dynamical systems, with applications spanning inference, dynamics, and control.

MS172-01 — Regulatory Motifs in C. elegans

SPEAKERS Brandilyn Stigler (Southern Methodist University)
AUTHORS Brandilyn Stigler (Southern Methodist University); Imasha Saparamadu (Southern Methodist University)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

For more than four decades, the nematode C. elegans has served as a key model organism in aging research, particularly at the cellular and molecular level. Recently single-cell time series studies have shown that different types of cells age at different rates and through distinct regulatory mechanisms. Using that published data, we identified transcription factors that are co-expressed in the same cells and constructed cell-specific models. The models were built using so-called minimal sets and are represented as directed graphs. The original goal was to identify a universal regulatory subnetwork conserved across multiple cell types, that when augmented within a specific type of cell would give rise to cell-specific behavior. Instead our work revealed similar motifs across cell types: we observed coordinated regulatory pairs, although the specific transcription factors involved vary by cell type. This suggests that aging regulation in C. elegans may be organized not around a single conserved subnetwork, but around motifs differentiated by cell type. We discuss how these motifs may contribute to robustness and maintenance of cellular dynamics during aging.

MS172-02 — BoolForge: Controlled generation and analysis of Boolean functions and networks

SPEAKERS Claus Kadelka (Iowa State University)
AUTHORS Claus Kadelka (Iowa State University); Benjamin Coberly (Iowa State University)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Boolean networks are a widely used modeling framework in systems biology for studying gene regulation, signal transduction, and cellular decision-making. Empirical studies indicate that biological Boolean networks exhibit a high degree of canalization, a structural property of Boolean update rules that stabilizes dynamics and constrains state transitions. Despite its central role, existing software packages provide limited support for the systematic generation of Boolean functions and networks with prescribed canalization properties. In this talk, I introduce BoolForge, a Python toolbox for the random generation and analysis of Boolean functions and networks, with a particular focus on canalization. BoolForge enables users to (i) generate random Boolean functions with specified canalizing depth, layer structure, and related constraints; (ii) construct Boolean networks with tunable topological and functional properties; and (iii) analyze structural and dynamical features including canalization measures, robustness, modularity, and attractor structure. By enabling controlled generation alongside analysis, BoolForge facilitates ensemble-based investigations of structure-dynamics relationships, benchmarking of theoretical predictions, and construction of biologically informed null models for Boolean network studies.

MS172-03 — Towards Complement-based Therapeutics: Opportunities for (Discrete) Mathematical Modeling

SPEAKERS Daniel Cruz (Cal Poly)
AUTHORS Luis Sordo Vieira (University of Florida); Daniel Cruz (Cal Poly); Ivan Ramirez-Zuniga (University of Texas at Tyler); Carl Atkinson (Northwestern University); Borna Mehrad (University of Florida); Reinhard Laubenbacher (University of Florida)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

The complement system is a potent arm of the immune system, linking the adaptive and innate immune systems, and affecting many facets of human pathophysiology. Recent advances in the development of complement-specific drugs in several rare diseases have opened the gateways to apply complement

interventions toward a broader array of pathologies. However, this goal remains elusive due to the complexity and the multifaceted roles of the complement system. Mechanistic mathematical models can provide an effective tool to integrate the different mechanisms, features, and feedback loops into a framework that allows the simulation of complement dynamics under different conditions and the prediction of the effect of interventions. In this talk, we provide an overview of the complement system and discuss open problems related to complement biology. We focus on how mechanistic mathematical modeling of the complement system can potentially alleviate some of the challenges in complement biology. In particular, we discuss how discrete modeling has largely been absent from the field and how it might provide some unique opportunities.

MS172-04 — Prioritizing Control Strategies in Boolean Networks Using Mutation Scores and Network Modularity

SPEAKERS David Murrugarra (University of Kentucky)
AUTHORS David Murrugarra (University of Kentucky)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Control of biological networks is often achieved by targeting a small number of regulatory nodes, but identifying optimal control strategies remains challenging. Existing control methods frequently yield multiple alternative control sets that satisfy the same objective, making it difficult to select among them. Optimality is typically defined by minimality or by minimizing a cost function. This talk presents a framework for prioritizing control strategies using mutation scores and the modular structure of Boolean networks. Within a stochastic Boolean network setting, mutation scores are computed by simulating the effects of available control interventions. We apply this approach to a pancreatic cancer model, where candidate controls are identified through network modularity and ranked using mutation scores to select an optimal control set. We assess the feasibility of these controls and discuss their biological relevance. Finally, we outline key challenges and potential extensions of this approach to broader classes of biological networks.

MS172-05 — Reproducible Boolean model analyses and simulations with the CoLoMoTo software suite

SPEAKERS Denis Thieffry (Ecole Normale Superiore)
AUTHORS Denis Thieffry (Ecole Normale Superiore)
SCHEDULE Friday, 17 July 2026; 10:40–11:20; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

In order to address the pervasive reproducibility crisis, we combine Docker containers (or Conda packages) with Jupyter electronic notebooks, in order to develop and document dynamical analyses of logical models of complex biological molecular networks. The resulting Common Logical Modelling Tools (CoLoMoTo) environment currently encompasses over 20 tools, written in multiple languages, making them accessible with a single popular language, python. To illustrate how complementary tools can generate novel insights, we have revisited the analysis of a model of the regulatory network controlling mammalian cell proliferation, published by Sizek et al in PLoS Computational Biology in 2019. Using the CoLoMoTo environment, we could reproduce the main results and figures published in the original article, and further extend these results with the help of a selection of tools included in the CoLoMoTo suite. More precisely, we developped a notebook encompassing the visualisation of the network with the tool GINsim, an attractor analysis with bioLQM, the identification of synchronous attractors with BNS, the extraction of modules from the full model, stochastic simulations for the wild-type and selected mutant background with MaBoSS, and finally the computation of compressed probabilistic state transition graphs. The integration of all these analyses in an executable Jupyter notebook greatly eases their reproducibility, as well as novel extensions.

This notebook can further be used as a template and enriched with other ColoMoTo tools to enable comprehensive dynamical analyses of biological network models.

MS172-06 — Using Canalizing Boolean Functions to Model Regulation in Mammalian Cell Division

SPEAKERS Elena Dimitrova (Cal Poly)
AUTHORS Elena Dimitrova (Cal Poly); Brandilyn Stigler (Southern Methodist University); David Murrugarra (University of Kentucky); Claus Kadelka (Iowa State University)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

Discrete models are a natural framework for biological systems whose regulation is governed by switch-like interactions. In this talk, we will present a reverse-engineering approach to Boolean modeling of mammalian cell division through the regulation of the transcription factor E2F. Starting from partial biological knowledge about the roles of CycB, Rb, p27, and CycA, we will show how a prescribed canalizing-layer structure can be used to recover a family of nested canalizing Boolean functions consistent with the known regulatory logic. This framework makes it possible to encode dominant regulatory effects, distinguish variables acting at different hierarchical levels, and systematically infer admissible update rules from incomplete mechanistic information.

MS172-07 — Structural Coherence Drives Cooperative Dynamics in Gene Networks

SPEAKERS Pradyumna Harlapur (Indian Institute of Science)
AUTHORS Pradyumna Harlapur (Indian Institute of Science); Claus Kadelka (Iowa State University); Rahul Jagadeesan (Tampere University); Andre Sanches Ribeiro (Tampere University); Mohit Kumar Jolly (Department of Bioengineering, Indian Institute of Science)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 11.01 - HS
TRACK Systems Biology and Biochemical Networks

Abstract

A central challenge in systems biology is understanding how gene regulatory networks (GRNs) coordinate cellular decision-making within complex topological structures. This study introduces a framework to quantify the alignment of regulatory logic among interacting genes, a property defined here as structural coherence. By applying this metric, we identify ”teams”, functionally coupled gene sets that exhibit cooperative activation. We investigate the topological implications of structural coherence by characterizing how team size and composition scale with key network features. To link architecture with dynamical behavior, we employ a threshold Boolean model, specifically a probabilistic Ising model, to evaluate the relationship between structural coherence and gene expression coordination. Our results demonstrate that structurally coherent groups significantly influence the configuration and stability of network attractors. Furthermore, analysis of biological GRNs shows how hierarchical organization enables coherent decision-making to scale across large gene assemblies, even in the presence of localized incoherence. The structural coherence framework provides a robust, generalizable tool that integrates local interactions and global network architecture to explain the emergent regulatory coordination.

MS176 — Recent Development on Digital Twins for Biology and Biomedical Sciences

TRACK Numerical, Computational, and Data-Driven Methods
SCHEDULE Thursday, 16 July 2026; 17:00–12:00; 15.05 - HS
ORGANISERS Weitao Chen (University of California, Riverside); Uduak George (San Diego State University)

Minisymposium abstract

Mathematical modeling has long played a critical role in advancing our understanding of biological systems and disease processes, spanning scales from molecular interactions to population-level dynamics.

These approaches provide a principled framework for uncovering mechanisms, integrating heterogeneous data, and generating predictive insight. Recent advances in computational power, high-throughput sequencing, advanced imaging, electronic health records, and artificial intelligence have created unprecedented opportunities for mathematical and computational modeling to fundamentally reshape biomedical research.

This minisymposium brings together applied mathematicians and computational scientists working on diverse forms of digital twins, highlighting both methodological innovations and novel biomedical applications. Our objective is to catalyze new directions in mathematical biology that address pressing, real-world challenges requiring integrative and cross-disciplinary approaches. Through structured presentations and interactive discussion, the minisymposium aims to promote a cultural shift toward deeper, more systematic integration of mathematics into biomedical research and clinical practice.

MS176-01 — Digital Twins of Morphogenesis

SPEAKERS Mark Alber (University of California, Riverside)
AUTHORS Jeremiah Zartman (University of Notre Dame); Mark Alber (University of California, Riverside); Nan Hao (University of California San Diego); Weitao Chen (University of California, Riverside); Zhiliang Xu (University of Notre Dame)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The regulation and maintenance of a tissue's shape and structure is a major outstanding question in developmental biology and plant biology. In this talk, through iterations between experiments and multiscale model simulations that include a mechanistic description of interkinetic nuclear migration, we will show that the local curvature, height, and nuclear positioning of cells in the Drosophila wing imaginal disc are defined by the concurrent patterning of actomyosin contractility, cell-ECM adhesion, ECM stiffness, and interfacial membrane tension [Kum+24]. The biologically calibrated model describing both tissue growth and morphogenesis incorporates the spatial patterning of fundamental subcellular properties. Additionally, the model implements for the first time the dynamics of interkinetic nuclear migration within the simulated pseudostratified epithelium. This includes the basal to apical motion of the nucleus, mitotic rounding, and cell division dynamics. Key characteristics of global tissue architecture, such as the local curvature of the basal wing disc epithelium, cell height, and nuclear positioning, serve as metrics for model calibration. The experiments have shown how these physical features are jointly regulated through spatiotemporal dynamics in the localization of pMyoII, β-Integrin, and ECM stiffness. As the disc grows, there are progressive changes in the patterning of key subcellular features such as actomyosin contractility. The predictions made by the model simulations agree with the observed changes in contractility and cell-ECM adhesion during wing disc morphogenesis. AI techniques are incorporated in the model calibration to develop surrogate models to optimize model parameters. In the second half of the talk, aging of yeast cells will be discussed. Understanding the mechanisms of the cellular aging processes is crucial for attempting to extend organismal lifespan and for studying age-related degenerative diseases. Yeast cells divide through budding, providing a classical biological model for studying cellular aging. With their powerful genetics, relatively short cell cycle, and well-established signaling pathways also found in animals, yeast cells offer valuable insights into the aging process. A three-dimensional multi-scale chemical-mechanical model was developed and used to suggest and test hypothesized impacts of aging on bud morphogenesis [Tsa+24]. Experimentally calibrated model simulations showed that during the early stage of budding, tubular bud shape in one aging mode could be generated by locally inserting new materials at the bud tip, a process guided by the polarized Cdc42 signal. Furthermore, the aspect ratio of the tubular bud could be stabilized during the late stage as was also observed in experiments [Tsa+24]. The model simulation results suggest that the localization of new cell surface material insertion, regulated by chemical signal polarization, could be weakened due to cellular aging in yeast and other cell types, leading to the change and stabilization of the bud aspect ratio.

References

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MS176-02 — A Patient-Specific Digital Twin for Adaptive Radiotherapy of Non-Small Cell Lung Cancer

SPEAKERS Jun Deng (Yale University)
AUTHORS Anvi Sud (Yale University); Gregory R. Hart (University of Guam); Jialu Huang (Yale University); John Kim (Yale University); Jun Deng (Yale University); Lauren Tressel (Yale University)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In this talk, we introduce a patient-specific digital twin framework termed COMPASS (COMprehensive Personalized ASsessment System) for adaptive radiotherapy of non-small cell lung cancer (NSCLC) patients based on fractional PET/KVCT imaging, radiomics, dosiomics, and biologically equivalent dose (BED) kinetics. Specifically, eight NSCLC patients treated with biology-guided radiotherapy (BGRT) were modeled with 99 organ-fraction observations across 24 organ trajectories. Organ specific time-series features were derived to preserve spatial dose heterogeneity and biological response. A gated recurrent unit (GRU) autoencoder was used to learn compact latent representations of evolving dose–response trajectories for critical organs, which were subsequently classified using logistic regression to predict eventual CTCAE grade ≥ 1 toxicity. Despite the limited cohort size, COMPASS achieved an AUC of 0.90 with 80% sensitivity and 78% specificity. Importantly, elevated toxicity risks were predicted several fractions prior to clinical symptom onset, defining an actionable window for early intervention for patients. Incorporation of BED kinetics and spatial dose-texture features sensitively captured transient metabolic and dosimetric perturbations not reflected by regular metrics. COMPASS provides a physics- and biology-informed framework for adaptive radiotherapy where organ toxicity and tumor dynamics are continuously updated based on delivered dose and images to guide radiotherapy.

MS176-03 — Machine Learning-Enabled Digital Twins for Personalized Modeling of Alzheimer’s Disease

SPEAKERS Wenrui Hao (Penn State University)
AUTHORS Wenrui Hao (Penn State University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Digital twins are emerging as a powerful paradigm for modeling complex biological systems by integrating data-driven and mechanistic approaches. In this talk, we present recent developments in constructing machine learning-enabled digital twins for Alzheimer’s disease (AD), a highly heterogeneous neurodegenerative disorder characterized by diverse biomarker trajectories, progression rates, and treatment responses. We develop a unified framework that combines mechanistic mathematical modeling with data-driven learning to build patient-specific digital twins capable of simulating disease progression and predicting individualized outcomes. This approach enables the integration of multimodal clinical and biomarker data, providing a more comprehensive representation of disease dynamics. We demonstrate how these digital twins can be used to explore the underlying biomarker cascade, quantify patient variability, and evaluate personalized therapeutic strategies in silico. This work highlights the potential of digital twin technologies to advance precision medicine in AD and reduce the cost and time associated with clinical trials.

MS176-04 — Integrating Multimodal Patient Data with Biophysical Models for Predicting Patient-Specific Tumor Progression

SPEAKERS Parag Katira (San Diego State University)
AUTHORS Parag Katira (San Diego State University)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Tumor progression is shaped by a complex interplay between genomic heterogeneity, biophysical interactions, and the tissue microenvironment. Computational models of tumor growth have historically struggled to incorporate the full richness of patient-specific data — from genomics and transcriptomics to medical imaging and tissue mechanics. Here, I will present a multi-scale computational approach designed to bridge this gap. The centerpiece is a stochastic state-space modeling framework that discretizes the tissue microenvironment into spatially resolved voxels characterized by resource availability, mechanical properties, vascular state, and clonal composition of tumor cells. Multi-modal patient data — medical imaging, bulk genomics, and digital pathology — parameterize and personalize model behavior, while cell proliferation, death, migration, and metabolic state are governed by microenvironmental conditions. Applied to glioblastoma, the framework integrates brain-scale atlases of oxygen, glucose, and tissue stiffness with patient MRI and whole-genome sequencing to simulate how ploidy-dependent resource sensitivity shapes clonal competition and spatial patterns of tumor recurrence. To complement this meso- to macroscale approach, I will briefly introduce a pipeline connecting RNA sequencing data to biophysical models of single-cell migration through cytoskeletal signaling networks — a path toward directly informing cell migration parameters from patient transcriptomic data.

MS176-05 — Developing a Digital Twin of the Maternal Brain

SPEAKERS Sarah Kushne (University of California, Santa Barbara)
AUTHORS Nina Miolane (University of California Santa Barbara); Sarah Kushne (University of California, Santa Barbara)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The maternal brain undergoes significant anatomical change during pregnancy, yet the geometric principles governing these transformations remain poorly understood. Studying brain shape change is critical for identifying markers of maternal health, but prior work focuses on scalar volumes, masking subtle deformation. We develop a digital twin that integrates precision imaging with large datasets to map pregnancy trajectories of brain structures. For preprocessing, we conduct a quality control analysis to identify which segmentation and meshing tools provide the most accurate surface representations for the brain structures. Utilizing varifold geometry and multidimensional scaling (MDS), we create a shared latent space representing brain deformations. This merges a densely sampled longitudinal dataset including hormone metrics with a larger population study on postpartum health. Traditional linear statistical models (e.g., PCA) often fail in these paradigms because subcortical shapes exist on very high dimensional nonlinear manifolds, negating impact of Euclidean methods. To manage this, we train a multilayer perceptron to map latent MDS coordinates back to vertex positions, achieving a compact, expressive representation of 3D anatomy. Using regression models, our framework is able to predict 3D shape based on time and hormones. Our digital twin provides a foundational dynamic atlas, enabling personalized modeling to inform our current understanding of maternal brain health.

References

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MS176-06 — The mechanobiology of blood vessel formation: Towards a Digital Twin of Zebrafish intersegmental vessel formation

SPEAKERS Roeland Merks (Leiden University)

AUTHORS Erika Tsingos (Leiden University); Joaquin Abugattas (Leiden University); Koen Keijzer (Leiden University); Roeland Merks (Leiden University)

SCHEDULE Friday, 17 July 2026; 11:00–11:20; 15.05 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

To form new sprouts during angiogenesis, endothelial cells must coordinate their migration through biophysical and biomechanical signaling between each other and the micro-environment. A relatively simple model of angiogenesis is intersegmental vessel (ISV) formation in zebrafish. While various molecular, cellular, and mechanical factors coordinate ISV pathfinding between the somites, the specific contributions of ECM components remain incompletely understood. Here we hypothesize that guidance through ECM molecules laid down in the intersomitic space steers a process of self-organized pattern formation to reproducibly form blood vessels at stereotypic locations. We combine theoretical and experimental approaches in the zebrafish to investigate this hypothesis. Firstly, we developed a hybrid mathematical model to study the effect of ECM mechanics on a self-organized mechanism of endothelial network formation. We combined a previous, experimentally validated [VVM25] model of endothelial network formation based upon the Cellular Potts Model (CPM) with a molecular dynamics-based bead-spring model of the intersegmental ECM [Tsi+23; KTM25] and gradients of signaling molecules secreted by the somites. While in absence of the ECM, our model predicts that the endothelial cells form network-like patterns as they do in vitro [VVM25], in presence of the ECM, the model predicts the formation of intersomitic sprouts, consisting of endothelial cells that migrate along high concentrations of the ECM [Abu+26]. The model predicts that if the concentration of ECM were reduced in the intersomitic space, the endothelial cells should again organize into network-like structures. To investigate the contributions of the ECM to ISV formation in the zebrafish, we employed morpholino-mediated gene knockdown of ECM components in endothelial cell- and ECM-tagged zebrafish lines, coupled with high-resolution laser scanning confocal microscopy [Abu+26]. Although knock-down of Fibronectin-a and b or Laminin-a1 and a4 delays ISV sprouting during the first six hours, simultaneous knockdown of both ECM proteins resulted in aberrant vessel pathfinding, leading to disorganized, incomplete ISV development, resembling endothelial network patterns formed through self-organization by endothelial cells in silico and in vitro. Furthermore, we observed that endothelial cells interact with and migrate along laminin- and fibronectin-rich pathways, as revealed by zebrafish reporter lines, further underscoring the role of these ECM components in guiding vessel formation. Our simulations suggest that ECM stiffness may significantly influence endothelial cell migration, with cells potentially traveling further along VEGF gradients on stiff ECM compared to soft ECM under moderate VEGF sensitivity, providing a potential explanation for the observation in ECM component knockdowns. Our results highlight that ECM-regulated tip cell migration could be a key determinant of ISV growth speed and patterning. More generally, our study suggests an integrated, ‘mathematics-driven’ experimental approach, in which a digital twin is developed hand in hand with each next insight into the experimental system.

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MS176-08 — The impact of ephaptic coupling and ionic electrodiffusion on arrhythmogenesis in the heart

SPEAKERS Ning Wei (Purdue University)

AUTHORS Elena G Tolkacheva (University of Minnesota); Joyce Lin (Cal Poly San Luis Obispo); Ning Wei (Purdue University); Yoichiro Mori (University of Pennsylvania)

SCHEDULE Friday, 17 July 2026; 11:20–11:40; 15.05 - HS

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cardiac action potential (AP) propagation occurs through gap junction (GJ)-rich intercalated discs (IDs). However, recent experimental studies show that GJ knockout mice can still maintain heart structure and function, even when GJs are undetectable at IDs. Ephaptic coupling (EpC), an electric field effect across the narrow, tortuous IDs, provides an alternative mechanism for cell-to-cell communication when GJs are impaired. Given the current lack of direct experimental evidence for EpC, modeling studies are essential for understanding its physiological and pathological roles in the heart. Our research investigates how EpC and ionic electrodiffusion influence arrhythmogenesis in the hearts. We developed the first two-dimensional multidomain electrodiffusion model incorporating EpC. Our findings demonstrate that strong EpC suppresses the initiation of reentry, resulting in absent or nonsustained reentrant activity with a reduced maximum dominant frequency (DF), although it can also introduce transient instability and heterogeneity in cardiac dynamics. In contrast, weak EpC supports the initiation of sustained reentry, characterized by a stable rotor and high DF. Strong EpC terminates reentry through self-attenuation, while moderate EpC does so through self-collision. Additionally, we applied spectrum theory to analyze the impact of EpC on one-dimensional wave trains. Our results show that EpC reduces both the frequency and amplitude of wave trains.

References

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[WM25b] Ning Wei and Yoichiro Mori. “Role of ionic electrodiffusion and ephaptic coupling in cardiac dynamics”. In: *Biophysical Journal* 124.18 (2025), pp. 3060–3074.

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MS176-07 — Virtual dynamics of spatiotemporal single-cell interactions

SPEAKERS Peijie Zhou (Peking University)
AUTHORS Han Hou (Peking University); Jiantao Shen (Peking University); Jiaqi Han (Peking University); Peijie Zhou (Peking University); Qiangwei Peng (Peking University); QinXue Tian (Peking University); Qing Nie (University of California, Irvine); Tiejun Li (Peking University); Xueya Zhou (Peking University); Yuhao Sun (Peking University); Zhenyi Zhang (Peking University); Zihan Wang (Peking University)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 15.05 - HS
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Building virtual cells and virtual tissues that capture how interacting cell populations evolve in space and time is a central goal of modern biology, yet spatial omics experiments can only provide sparse snapshots of this process. Here, we introduce CytoBridge, a computational method that reconstructs continuous virtual cell dynamics from discrete spatial transcriptomic data. Starting from a joint expression–space manifold, CytoBridge learns stochastic trajectories of cell states, positions, and population sizes through an unbalanced mean-field Schrödinger bridge formulation that explicitly incorporates cell–cell interaction as a dynamical driver. A time-varying interaction graph built from spatial proximity and ligand–receptor priors allows the transition velocity to be decomposed into an interaction program, learned from within-system observables, and an intrinsic-context program that captures intrinsic regulatory processes. Applied to five datasets across three spatial transcriptomics platforms and spanning development, regeneration, and neurodegeneration, CytoBridge accurately generates tissue dynamics at unmeasurable time points, recovers known lineage trajectories and growth patterns, and reveals spatiotemporal ligand–receptor signalling axes not accessible from static analyses. CytoBridge provides a general method for turning heterogeneous spatiotemporal transcriptomic measurements into continuously evolving virtual tissues.

MS178 — Mathematical Modeling of Cross-Scale Biological Dynamics

TRACK Multiscale and Multiphysics Modelling
SCHEDULE Thursday, 16 July 2026; 17:00–18:20; 01.15 - HS
ORGANISERS Chadi Saad-Roy (University of British Columbia); Daniel Cooney (University of Illinois Urbana-Champaign)

Minisymposium abstract

Biological processes occur at many scales. To simplify mathematical analyses, the multiscale nature of these systems is often abstracted. However, processes at one scale can have important effects at other scales. To clarify these effects, detailed cross-scale modelling is needed. There are many approaches that have been developed by researchers in different sub-fields of mathematical biology. In this session, we will bring together researchers studying cross-scale biological dynamics across a range of natural systems. This will highlight how novel mathematical frameworks can be used to formulate rules for cross-scale forces and to discover complex emergent phenomena across levels of biological organization. Mathematically, the talks will draw on tools from stochastic processes, dynamical systems, game theory, and network science to explore cross-scale phenomena in many settings, including cancer, infectious disease, and cultural evolution.

MS178-01 — Transmission and early infection as key elements of multi-scale viral dynamics modelling

SPEAKERS Daniel Coombs (University of British Columbia, Vancouver, Canada)
AUTHORS Daniel Coombs (University of British Columbia, Vancouver, Canada)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 01.15 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Multi-scale mathematical models of viral infections obviously require two main models: a population-scale model of transmission between hosts, and an individual-scale model of the course of infection. However,

attempting to integrate these into a coherent multi-scale model leads us to confront their coupling at the moment of transmission, and during the first moments of the infection process. These parts of the overall process are generally poorly understood in comparison with the large-scale epidemic dynamics (population scale) and the large-scale viral dynamics (individual scale). In this talk, I will describe this problem, and then briefly summarize several projects where we closely examined transmission and early-time dynamics for viral infections, including human immunodeficiency virus (HIV), Epstein-Barr virus (EBV) and murine cytomegalovirus (MCMV). This talk represents joint work with many trainees and colleagues over the last twenty years and effusive acknowledgments will be given.

MS178-02 — Stochastic Process and PDE Models for Cross-Scale Evolutionary Dynamics

SPEAKERS Daniel Cooney (University of Illinois Urbana-Champaign)
AUTHORS Daniel Cooney (University of Illinois Urbana-Champaign)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 01.15 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Natural selection often operates simultaneously at multiple levels of biological organization, with evolutionary forces at each level potentially creating a tug-of-war between individual-level incentives to cheat and a collective incentive of maintaining cooperation. In this talk, we will discuss a stochastic framework for describing nested birth-death processes in group-structured populations with selection operating within and among competing groups, presenting simulations of the stochastic models and deriving ODE and PDE models that arise by successively taking the limit of an infinite number of groups and infinite group size. By comparing different possible update rules for individual-level and group-level replication events, we will be able to explore how forms of frequency-dependent competition at each level of selection can help shape the long-time support for cooperation by multilevel selection in both finite and infinite populations.

MS178-03 — Modelling the relationship between circulating levels of respiratory syncytial virus and health risk to infants

SPEAKERS Alireza Tafreshi (University of British Columbia, Vancouver, Canada)
AUTHORS Alireza Tafreshi (University of British Columbia, Vancouver, Canada)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 01.15 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Respiratory Syncytial Virus (RSV) is the leading cause of infant hospitalizations. During the SARS-CoV-2 pandemic, non-pharmaceutical measures (NPIs) such as masking and physical distancing resulted in very low circulating levels of RSV. When measures were relaxed, RSV-related hospitalizations surged among infants in many jurisdictions, far exceeding usual seasonal levels. Many have attributed this surge to the accumulation of susceptible infants during NPIs. However, this does not explain how total infections can decrease while pediatric intensive care hospitalizations increase, as was reported in Europe during the 2020-2021 season. We present an epidemiological model focused on placentally transferred maternal antibodies which protect infants from severe infections. During the pandemic, the reduction in general RSV circulation meant that fewer infants were born with maternal antibodies. Additionally, the time to first infection increased, leading to waning of maternal antibody protection before first infection. We show that decreasing RSV circulation among adults leads to an increase in the number of first-time infections without protective maternal antibodies. Temporal simulations show that infants were least protected from severe infection soon after pandemic-era public health policies were relaxed. We conclude that maternal antibodies play a major explanatory role in the dynamics of RSV-related hospitalizations. We further predict that general-population vaccination campaigns are likely to lead to an increase in higher-risk infections among infants. Importantly, treatments that are currently being widely implemented, such as maternal vaccination during pregnancy and monoclonal antibody treatment to infants, can effectively protect infants against the potential adverse effects of reduced RSV circulation.

MS178-04 — Cross-Scale Drivers of Antimicrobial Resistance: Competition, Trade-offs, and Landscape Structure

SPEAKERS Jessica Hite (University of Wisconsin – Madison)
AUTHORS Jessica Hite (University of Wisconsin – Madison)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 01.15 - HS
TRACK Multiscale and Multiphysics Modelling

Abstract

Antimicrobial resistance (AMR) is commonly framed as a genetic response to drug exposure, yet resistance reliably emerges from the ecological and spatial contexts in which microbes live. A growing body of theory and experiment suggests that competition can slow or even prevent the emergence of AMR, motivating intervention strategies that seek to enhance competitive interactions in clinical, agricultural, and environmental settings. However, our understanding of when and how competition could constrain resistance remains limited. Here, we develop a cross-scale mathematical framework linking genetic costs of resistance to population-level competition and landscape-scale spatial structure. Using a classical growth–efficiency trade-off from ecological theory, we examine competition between drug-sensitive and drug-resistant strains in which increasing resistance incurs a cost in reduced growth rate or resource efficiency. We ask how anthropogenic land-use change, particularly spatial and environmental homogenization, alters the ecological conditions under which these strains compete. By embedding AMR within a broader ecological theory of resource competition and life-history trade-offs, this work reframes resistance as an emergent property of coupled ecological and evolutionary dynamics rather than a purely pharmacological phenomenon. More broadly, the framework helps identify general principles governing when and why resistance genes emerge, persist, and spread.

MS35 — Deterministic Models to Describe and Analyse Tumour-Immune Interactions and Immunotherapy

TRACK Mathematical Oncology
SCHEDULE Friday, 17 July 2026; 10:40–12:00; 02.11 - HS
ORGANISERS Emma Leschiera (De Vinci Research Center); Federica Padovano (LJLL)

Minisymposium abstract

Tumour growth arises from a complex interplay between cancer cells and their microenvironment, often facilitated by failure of the immune system to effectively control malignant cells. Over the past decades, several immunotherapeutic approaches have been developed to enhance the anti-tumour immune response by harnessing the body's natural immune defences to fight cancer. However, the design and implementation of effective immunotherapeutic strategies remain challenging.

Mathematical models, and in particular deterministic models, provide mechanistic insights into tumour–immune interactions and enable the prediction of treatment outcomes. In this session we aim to bring together researchers who develop and study, both analytically and numerically, deterministic continuum models of tumour–immune interactions and immunotherapy. The presentations will range from the description of tumour–immune dynamics within the tumour-microenvironment to applications involving immunotherapeutic treatments, either alone or in combination with other therapies. This symposium will offer an opportunity to discuss how existing deterministic models can be improved to better understand, predict, and ultimately manipulate the immune response to cancer.

MS35-01 — Tumor growth and immune response: on the impact of the space-structuration of the tumor microenvironment

SPEAKERS Christian Tayou-Fotso (Université Côte d'Azur)

AUTHORS Fabienne Anjuère (Université Côte d'Azur, CNRS UMR7275, Inserm U1323, IPMC, Valbonne, France); Véronique M. Braud (Université Côte d'Azur, CNRS UMR7275, Inserm U1323, IPMC, Valbonne, France); Florence Hubert (Aix-Marseille Université, CNRS, Institut de Mathématiques de Marseille, France); Thierry Goudon (Université Côte d'Azur, CNRS, LJAD, France); Christian Tayou-Fotso (Université Côte d'Azur)

SCHEDULE Friday, 17 July 2026; 10:40–11:00; 02.11 - HS

TRACK Mathematical Oncology

Abstract

Understanding how the tumor microenvironment influences interactions between tumor cells and the immune system is a major question in mathematical oncology. In particular, the spatial organization of nutrient sources and immune cell recruitment may strongly affect tumor progression and immune control. In this work, we study this question using a continuum mixture model describing nutrient-dependent tumor growth, mechanical interactions within the tissue and the extracellular matrix, and the dynamics of anti- and pro-tumoral immune cell populations. External sources of nutrients and immune cells are included in the model, and their spatial location is treated as a key parameter. Using numerical simulations, we analyze how different spatial configurations of these sources affect tumor–immune dynamics. The results show a strong sensitivity of the system to the relative position of nutrient and immune sources. In particular, small changes in distance can significantly delay tumor growth, while some configurations lead to stable states where tumor mass and immune activity coexist. These results highlight the important role of spatial heterogeneity in the tumor microenvironment and show that its geometric organization can strongly influence the balance between tumor progression and immune control.

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MS35-02 — Modelling the efficacy of CIK cell immunotherapy targeting MET-expressing mesothelioma cells

SPEAKERS Federica Padovano (LJLL)

AUTHORS Federica Padovano (LJLL); Andrea Maria Lombardi (Candiolo Cancer Institute, Candiolo, Italy); Elisa Vigna (Candiolo Cancer Institute, Candiolo, Italy); Tommaso Lorenzi (Politecnico di Torino); Chiara Villa (CNRS & MAP5 (UPC)); Emma Leschiera (De Vinci Research Center)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 02.11 - HS
TRACK Mathematical Oncology

Abstract

Malignant pleural mesothelioma (MPM) is an aggressive tumour associated with poor prognosis and limited treatment options, motivating the investigation of novel therapeutic approaches. Among these, chimeric antigen receptor (CAR)–based adoptive cell immunotherapy has attracted considerable interest due to its potential to achieve durable clinical responses

We combine experimental and mathematical approaches to investigate the efficacy of a CAR-based immunotherapy using cytokine-induced killer (CIK) cells engineered with a MET-specific receptor targeting MET-overexpressing tumour cells. *In vitro* data from diverse MPM cell lines and CIK preparations are used to develop and calibrate a mathematical model describing the interaction dynamics between tumour cells, structured by their MET expression level, and CIK cells. The model successfully reproduces experimental observations and is used to predict treatment efficacy under previously untested conditions, which are validated with new data from MPM primary cell lines.

Our results elucidate the interplay between three key factors positively impacting treatment efficacy: the initial immune-to-tumour cell ratio, the percentage of CAR-MET-expressing CIK cells generated upon engineering, and the MET expression level of tumour cells. Together, these findings provide a quantitative basis for assessing CAR-CIK therapy effectiveness and initiate the development of a predictive framework that could support clinical decision making.

MS35-03 — Tumour–Immune Dynamics: Patterns, Trade-offs, and Evasion in a Continuous Modelling Framework

SPEAKERS Giulia Chiari (University of Oxford / BCAM (Basque Center for Applied Mathematics))
AUTHORS Giulia Chiari (University of Oxford / BCAM (Basque Center for Applied Mathematics)); Jose Antonio Carrillo (Mathematical Institute, University of Oxford); Marcello Edoardo Delitala (Politecnico di Torino)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 02.11 - HS
TRACK Mathematical Oncology

Abstract

We present a continuous model describing the interplay between tumour mass and the immune system. Building on [Car+19], we incorporate a nonlocal formulation that captures adhesion and chemoattraction forces at the microscopic level.

We investigate how the proliferation–immune susceptibility trade-off can be calibrated, identifying which balance between rapid growth and immune evasion/resistance leads to more favorable tumour outcomes under immune pressure. The model is designed to explore how the emergence of spatial patterns, arising from the interplay between nonlocal interactions and reaction terms, affects tumour evolution and its response to immune pressure. This perspective is connected to the classification of tumours based on T-cell infiltration (hot vs. cold tumours, see [OTS19]), which reflects their inflammatory and immunological state.

We first analyse homogeneous tumour populations, assessing how intrinsic proliferation–evasion properties affect both pattern formation and immune control. We then consider heterogeneous tumours characterised by a different phenotypic traits, examining the role of both quantitative composition (relative abundance) and spatial arrangement.

Overall, this framework highlights how spatial patterns and reaction dynamics jointly determine tumour behavior, providing insight into the emergence of resistant configurations and offering a theoretical basis for identifying optimal strategies in an immunotherapy context.

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MS35-04 — Mechanistic modeling of the combined dynamics of CAR T-cell therapy and standard care in malignant gliomas

SPEAKERS Matteo Italia (University of Castilla-La Mancha)

AUTHORS Dmitri Sinelshchikov ((Instituto Biofisika (UPV/EHU, CSIC), University of the Basque Country, Spain)); Juan Belmonte-Beitia (University of Castilla-La Mancha); Matteo Italia (University of Castilla-La Mancha)

SCHEDULE Friday, 17 July 2026; 11:40–12:00; 02.11 - HS

TRACK Mathematical Oncology

Abstract

Malignant gliomas (MGs) remain highly resistant to conventional treatments. We propose an ordinary differential equation model to investigate the potential synergistic dynamics between standard therapy (radiotherapy (RT) and temozolomide (TMZ)) [Stu+05] and chimeric antigen receptor (CAR) T-cell therapy [Bro+16]. While both TMZ and RT can be lymphotoxic, TMZ may also enhance CAR T efficacy by upregulating tumor antigens and inducing lymphodepletion, and RT can promote T-cell infiltration. Our model extends and combines our previous works [SBI25; Per+25], consisting of a nine-dimensional dynamical system describing MG subpopulations and treatment dynamics and incorporating impulsive inputs to simulate clinical administration. Mathematical analysis reveals parameter regimes under which constant treatment administration achieves tumor eradication. To bridge theory and clinical application, the model was validated using published trial data [Stu+05; Bro+16; Lai+10]. We generated cohorts of virtual patients through random sampling within biologically and clinically relevant ranges to conduct in silico trials. Numerical simulations evaluate various treatment combinations to identify optimal protocols and parameter-derived biomarkers at the population level. Our results provide new theoretical insights into treatment scheduling and offer a mechanistic tool to optimize and personalize MG therapy.

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MS58 — Multi-scale dynamical modeling of immune and cancer cell behaviour

TRACK Mathematical Oncology
SCHEDULE Friday, 17 July 2026; 10:40–12:00; 62.01 - HS
ORGANISERS Mohit Kumar Jolly (Indian Institute of Science); Soujanya Barik (PhD, IISc Bangalore, India)

Minisymposium abstract

Cancer progression is driven by uncontrolled proliferation and the ability of tumors to exploit the tumor microenvironment (TME), a heterogeneous ecosystem composed of cancer, immune and endothelial cells with extracellular matrix. Within the TME, reciprocal interactions between tumor and immune cells shape disease outcome. The balance between anti-tumor immune surveillance and tumor-promoting immune modulation is a dynamic and context-dependent process, varying across cancer types, stages, and microenvironmental conditions. The session aims to foster cross disciplinary dialogue and advance predictive, testable frameworks for cancer immune dynamics. This session focuses on multi-scale dynamical modeling of cancer immune interactions, bridging intracellular regulatory networks, intercellular signaling, and tissue-scale spatial organization. Topics and speakers include "Diffusion-limited cytokine signaling in T cell populations" (Kevin Thurley, University of Bonn), "Intracellular network dynamics governing macrophage polarization" (Soujanya Barik, Indian Institute of Science), "Uncovering transcriptional control landscapes in cancer systems using data-driven model inference" (Malvina Marku, Toulouse Cancer Research Center) and "From Cells to Tissues: Multiscale Systems Biology on the Road to Digital Twins" (Arnau Montagud, Institute for Integrative Systems Biology, CSIC). By integrating theory with data, these approaches enable mechanistic interpretability across scales.

References

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MS58-01 — Characterization of macrophage phenotypes using mathematical modelling

SPEAKERS Soujanya Barik (PhD, IISc Banglore, India)
AUTHORS Soujanya Barik (PhD, IISc Banglore, India)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Macrophages are innate immune cells with a wide range of functional capacities [Wu+21]. They have been broadly classified into M1 or M2 phenotypes based on environmental signals. Due to variation in such regulatory cues, these macrophages are thought to also show states that are intermediate within a spectrum that has M1 and M2 at its two extremes [KAR14]. However, across all these studies, characterization for hybrid macrophages is not consistent due to overlapping macrophage markers. How hybrid macrophages arise, how stable these states are, how easy is it for them to transition from, or to M0, M1, or M2 states, and how is their stability reinforced or attenuated by the distinct secretory states remains poorly understood. To address these set of problems, we define the hybrid macrophage phenotype based on gene regulatory network (GRN) information at intracellular scale. Our findings reveal a 'teams' structure as an emergent property of the macrophage GRN. Perturbation analysis was done with M1 and M2 as initial states and as two different cases. In one node perturbation, the system is unable to switch its phenotype in either case, but in some two and three-node combinations, the system can switch to hybrid phenotype. Furthermore, overexpression analysis was done, where STAT3 and STAT1 combination was specifically observed to give rise to the most hybrid populations. The phenotypic distribution obtained from the Boolean framework was then validated using RAndom CIrcuit PErturbation (RACIPE). The distribution of steady state in overexpression analysis using RACIPE was consistent with the Boolean framework, making the distribution of steady states a robust dynamical property of the network.

References

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MS58-02 — Dissecting immune-cell communication networks in chronic inflammation and cancer

SPEAKERS Kevin Thurley (University of Bonn)
AUTHORS Kevin Thurley (University of Bonn)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 62.01 - HS
TRACK Mathematical Oncology

Abstract

Immune responses are tightly regulated by a diverse set of interacting immune-cell populations, and immune-cell communication pathways are routinely targeted by immunotherapy in autoimmune diseases and cancer. However, a quantitative understanding of immune-cell regulation in vivo is only beginning to emerge [Ste+25]. We assessed cytokine reaction-diffusion dynamics using a 4D finite-element based modeling framework [Bru+24]. We found that spatial cytokine gradients robustly arise in physiological parameter regimes and are critical for effective paracrine signaling, and they do not arise by diffusion and uptake alone, but rather depend on properties of the cell population such as an all-or-none behavior of cytokine secreting cells. Next, we explored the effect of cytokine gradients in the context of motile cell populations in the lymph node and the tumor microenvironment, employing both cellular Potts models and a numerically efficient formulation of cytokine gradients based on a homogenization approach. Further, we developed a general mathematical framework for analysis of interactive immune-cell population dynamics, accounting for cell-cell interactions, cell-proliferation, and cell-differentiation by means of measurable response-time distributions [BT23]. We employed that framework to describe the onset of lupus nephritis, the renal manifestation of systemic lupus erythematosus (SLE) [Kre+26]. Starting from analysis of single-cell RNA-sequencing data on innate lymphoid cells, our mathematical model reconciles that experimental data with long-standing clinical observations such as elevated autoantibodies and interferons in individuals with genetic predisposition for SLE. Overall, our results from model simulations and data analysis highlight the complex dynamics imposed by cell-cell signaling networks in the immune system, with implications for therapeutic intervention.

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MS58-03 — When data tells the story: Uncovering transcriptional control landscapes in cancer systems using data-driven model inference

SPEAKERS Malvina Marku (Toulouse Cancer Research Center)
AUTHORS Malvina Marku (Toulouse Cancer Research Center)

SCHEDULE Friday, 17 July 2026; 11:20–11:40; 62.01 - HS
TRACK Mathematical Oncology

Abstract

A central challenge in systems oncology is understanding how the tumour microenvironment (TME) reconfigures the internal regulatory circuitry of cancer cells. While the reprogramming of immune cells within the TME is well-documented, the longitudinal regulatory dynamics of the cancer cells themselves, especially in response to immune interactions, remain elusive. In this work, we present a data-driven framework that bridges time-series transcriptomics and gene regulatory network (GRN) inference to map these temporal landscapes.

Using Chronic Lymphocytic Leukaemia (CLL) as a model, we integrate longitudinal expression data from patient-derived cells within a reconstituted in vitro TME [Hac+14]. By inferring GRNs based on transcription factor activity across multiple time points [Mar+26; MP23], we uncover a complex orchestration of cytokine signalling, metabolic shifts, and differentiation. Our analysis reveals that while immune-cell interactions significantly drive CLL activation and phenotypic plasticity, the long-term survival trajectories of these cells are governed by deeply ingrained intrinsic features [HG18]. This underscores a dual regulatory architecture where the environment sets the pace, but the internal network determines the destination. These insights provide a roadmap for identifying patient-specific regulatory nodes that could be targeted to disrupt cancer-immune co-evolution, which can then be used to study the long-term behaviour of the CLL cells through dynamical modelling.

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MS58-04 — From Cells to Tissues: Multiscale Systems Biology on the Road to Digital Twins

SPEAKERS Arnau Montagud (Institute for Integrative Systems Biology, CSIC)
AUTHORS Arnau Montagud (Institute for Integrative Systems Biology, CSIC)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 62.01 - HS
TRACK Mathematical Oncology

Abstract

This talk explores how we move from models toward clinically useful digital twins in biomedicine. It focuses on three pillars: multiscale integration of intracellular signalling with agent-based cell simulations [Est+26] , personalisation using patient-specific omics [Mon+22], and community standards for model validation [Nti+25]. We present PhysiBoSS1, a tool that couples stochastic Boolean network simulations with agent-based multicellular dynamics, demonstrating applications in drug screening and in predicting synergies in cancer treatments. Crucially, this modular architecture enables independent evolution of biological components, such as signalling logic and cell properties, supporting long-term maintainability and scalability. We will highlight that realistic tumour simulations will require better-curated data, novel algorithms and computer architectures, and, more importantly, careful methods to bridge very different

temporal and spatial scales [Pon+23]. A community benchmarking effort across several major agent-based tools shows that, despite similar core features, differences in underlying mathematics and numerics affect results, underscoring the need for shared interfaces, metrics, and open dissemination [Nti+25]. Finally, we show that patient-specific Boolean models, parameterised with genomic and transcriptomic data in breast and prostate cancer, can suggest actionable drug targets and are experimentally validated in cell lines, illustrating the potential of predictive, personalised digital twins [Mon+22].

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[Pon+23] Miguel Ponce-de-Leon et al. "PhysiBoSS 2.0: a sustainable integration of stochastic Boolean and agent-based modelling frameworks". In: *NPJ Syst. Biol. Appl.* 9.1 (Oct. 2023), p. 54.

MS170 — Mechanochemical modelling of patterning in developmental systems

TRACK Cellular and Developmental Biology
SCHEDULE Friday, 17 July 2026; 10:40–12:00; 02.23 - HS
ORGANISERS Yi Ting Loo (The Francis Crick Institute)

Minisymposium abstract

During embryonic development, cells differentiate and self-organise with remarkable robustness to form complex tissue structures. These processes arise from tightly coupled interactions between morphogen transport, biochemical signalling, cellular dynamics, and tissue mechanics. Despite significant progress, how these mechanisms act together to drive developmental patterning and morphogenesis remains an open problem.

This minisymposium will focus on mechanochemical modelling approaches for understanding developmental patterning, which involves the roles of tissue mechanics and geometry, cell shape and polarity, cell movement and rearrangements, and morphogen-mediated signalling. Emphasis will be placed on theoretical and computational frameworks that capture feedbacks between chemical and mechanical cues during pattern formation. The goal of the minisymposium is to bring together researchers developing and applying mechanochemical models to developmental systems, fostering discussion on emerging methods, biological insights, and open problems in the field. Example applications include gastrulation, somitogenesis, invagination, and other developmental processes.

MS170-01 — Data Driven Modeling of Limb Bud Growth and Morphogenesis

SPEAKERS Tim Liebisch (EMBL Barcelona)
AUTHORS James Sharpe (EMBL Barcelona); Tim Liebisch (EMBL Barcelona)
SCHEDULE Friday, 17 July 2026; 10:40–11:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Limb development is regulated by feedback between the tissue-scale mechanics and cellular decisions–migration, contraction, division, and death–implemented through gene-regulatory networks. The dynamically changing states of these cellular networks reflect the decisions being made. To integrate these interactions across 3 scales: genes, cells, and tissues, we develop a 3D cell-based model in which cellular decisions

are spatially regulated by morphogen gradients. By constraining parameters with empirical data and optimizing against wild-type limb-bud shape, we obtain a model that links signal distributions to emergent mechanical behaviors.

Multiple mechanisms have been proposed for limb-bud morphogenesis, including ectodermal mechanical constraints, proliferation gradients, oriented divisions, distally biased migration, motility gradients, and convergent extension. Yet which spatio-temporal combinations of these activities generate correct morphology remains unresolved.

We use the model to test whether convergent extension alone is sufficient to reproduce limb-bud outgrowth and shaping. We compare 2 distinct modes of convergence: (1) orthogonal to an AER/FGF8-associated proximo-distal cue, and (2) parallel to a surface-to-core (radial) cue consistent with Wnt3a. For each mode, we quantify how the strength and timing of convergent extension affects predicted shape trajectories and whether either mode–or a combination of both–can match wild-type morphology.

MS170-02 — Oscillator coupling separates morphogenesis and patterning into two developmental modules

SPEAKERS James Hammond (University of Cambridge)
AUTHORS Berta Verd (Department of Biology, University of Oxford); Callum Bucklow (Department of Biology, University of Oxford); James Hammond (University of Cambridge); James Malone (Department of Biology, University of Oxford, Rudolf Peierls Centre for Theoretical Physics, University of Oxford); Ruth Baker (Mathematical Institute, University of Oxford); Shannon Taylor (Department of Molecular Genetics, University of Hohenheim)
SCHEDULE Friday, 17 July 2026; 11:00–11:20; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

In the early embryo an intracellular oscillator known as the segmentation clock patterns the somites, blocks of mesoderm that give rise to vertebrae and skeletal muscle. Clock oscillations are coupled between cells of the pre-somitic mesoderm (PSM) via notch-delta signalling, synchronising differentiation of PSM cells into somites. While this is happening, PSM cells are rearranging and dividing, and are being replaced by new cells that ingress from surrounding tissues. The relative contribution of each of these processes to elongation of the PSM varies across species, facilitating the evolution of diverse body plans and ecological niches. However, processes like cell ingression and division disrupt synchrony of the clock and could affect somite patterning if these events occur too often. How then does the diversity of morphogenetic dynamics evolve without disrupting the clock, and how does this 'evolvability' depend on the clock itself? Using a phase oscillator model to describe clock coupling, and a point-based model to describe cell movement, we studied how clock dynamics react to varying morphogenesis of the PSM. We find that clock dynamics are robust to changing morphogenesis when oscillator coupling is of the correct magnitude and timescale. By experimentally quantifying coupling in Lake Malawi cichlids, we show that these conditions are satisfied across 34% of extant vertebrate species. This suggests that clock coupling has permitted the evolution of diverse body plans.

MS170-03 — Image-based modelling explains variability in morphogen diffusion measurements

SPEAKERS Yi Ting Loo (The Francis Crick Institute)
AUTHORS Yi Ting Loo (The Francis Crick Institute)
SCHEDULE Friday, 17 July 2026; 11:20–11:40; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Morphogens are intercellular signalling molecules that provide spatial information to cells in developing tissues by establishing long-range concentration gradients. Measurements of morphogen diffusivity vary considerably depending on the experimental approach, and these discrepancies have been used to question diffusion as a viable mechanism for morphogen gradient formation. Using particle-based modelling on realistic zebrafish brain images, we demonstrate that incorporating local tissue architecture together with

receptor binding is sufficient to reproduce experimentally observed morphogen dynamics measured by FRAP and FCS. Our model recapitulates a range of biological observations such as generating two distinct particle populations with slow and fast diffusion coefficients, as reported in vivo. We further show how interactions between these populations contribute to setting the length scale of the concentration gradient, and how differences in experimental measurements can arise from the complex dynamics of hindered diffusion-driven transport.

MS170-04 — Collisions, Shape and Stigmergy: Modelling the Collective Organisation of ECM

SPEAKERS Omar El Oakley (The Francis Crick Institute)
AUTHORS Katie Bentley (King's College London, The Francis Crick Institute); Malcolm Logan (King's College London); Omar El Oakley (The Francis Crick Institute)
SCHEDULE Friday, 17 July 2026; 11:40–12:00; 02.23 - HS
TRACK Cellular and Developmental Biology

Abstract

Radial dysplasia (RD) is a musculoskeletal disorder in which the anatomical structure of the forelimb is fundamentally altered - in extreme cases the radius bone is missing, along with a number of muscles. RD is a disorder of later development, that manifests through changes in the extracellular matrix (ECM) deposition behaviour of a population of fibroblast precursors. As muscle cells follow patterning cues in the ECM, this change in fibroblast behaviour and ECM organisation propagates in multiple complex ways to alterations in muscle cell alignment. In my project I am using predictive computational modelling to understand how these emergent changes at the tissue-level derive from collective cell interactions. To do this, I have acquired a rich dataset of collective cell motion timelapse data, designed a model of cellular collective motion incorporating fine-grained simulation of cell motion, and have implemented a novel analysis inspired by methods from information geometry and simulation-based inference to both fit this data and pursue global model reduction. Unexpectedly, I have found that greater collective organisation of cells due to nematic alignment and flocking behaviours can, when ECM feedback is allowed, derange the process of depositing globally organised ECM.

Chapter 4

Contributed Talks

CT273 — 3D-Analysis of tumor spheroids – a synergistic combination of mechanistic modeling and data-driven computational methods

SPEAKERS Anja Voss-Böhme (HTWD - University of Applied Sciences Dresden)
AUTHORS Anja Voss-Böhme (HTWD - University of Applied Sciences Dresden); Florian Franke (HTWD - University of Applied Sciences Dresden); Leoni Kunz-Schughart (Technische Universität Dresden); Steffen Lange (HTWD - University of Applied Sciences Dresden)

SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Three-dimensional (3D) tumor spheroids, reflecting avascular microregions within a tumor, are preclinical cell culture systems for assessing the impact of radio(chemo)therapy. However, spheroid experiments remain laborious, and determining long-term radio(chemo)therapy outcomes is challenging. Mathematical models of spheroid dynamics have the potential to enhance the informative value of experimental data, and can support study design. We present an effectively one-dimensional mathematical model based on the cell dynamics within and across radial spheres which fully incorporates the 3D dynamics of tumor spheroids by exploiting their approximate rotational symmetry and demonstrate that this radial-shell (RS) model reproduces experimental spheroid growth curves. In parallel, we develop a (semi-) automated spheroid analysis pipeline to evaluate thousands of microscopy images per treatment arm. The pipeline integrates automated spheroid segmentation and classification of therapeutic outcome using statistical and machine learning algorithms. Model integration into the data-driven computational pipeline enables the analysis of complex radiobiological data sets and facilitates biologically optimized, innovative solutions for radiation therapy.

CT1586 — A mathematical model of ultrasensitive sequencing protocol

SPEAKERS Eszter Lakatos (Chalmers University of Technology)
AUTHORS Alexey Lindo (University of Glasgow); Serik Sagitov (Chalmers University of Technology); Eszter Lakatos (Chalmers University of Technology); Anders Ståhlberg (University of Gothenburg); Firaol Tamiru Kebede (University of Gothenburg)

SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 11.34 - SR

SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Detecting extremely rare DNA variants is key in multiple clinical applications, such as circulating tumour DNA detection or transplant monitoring. A serious limitation of detection ability comes from polymerase-induced sequencing errors. Unique molecular identifiers (UMIs) help suppress these errors, but their efficiency depends on how many amplified copies each barcoded molecule yields - an outcome shaped by protocol design.

Here we present a stochastic branching process-based model that captures the key steps of ultrasensitive UMI-tagged sequencing: barcoding PCR, dilution, amplification PCR with saturation, and final sequencing. We quantitatively fit the model to data generated with the SiMSen-Seq protocol [Stå+17] using Approximate Bayesian Computation. We demonstrate that the model accurately reproduces the experimentally observed linear relationship between sequencing depth and average UMI cluster size, and suggests that even UMI-affecting polymerase error rates are fragment dependent. We then utilise the model to explore the effect of mid-protocol dilution on final UMI yields, and find negligible impact, indicating experimental flexibility without loss of sensitivity.

In summary, our model provides a computationally efficient framework for evaluating protocol modifications and understanding sources of variability in UMI-based ultrasensitive sequencing.

References

[Stå+17] Anders Ståhlberg et al. “Simple multiplexed PCR-based barcoding of DNA for ultrasensitive mutation detection by next-generation sequencing”. In: *Nature Protocols* 12.4 (Apr. 1, 2017), pp. 664–682. DOI: 10.1038/nprot.2017.006. URL: https://doi.org/10.1038/nprot.2017.006.

CT216 — Conflict-Gated Gradient Scaling: Resolving gradient pathology in physics-informed epidemiological models

SPEAKERS Woldegebriel Assefa Woldegerima (York University, Canada)
AUTHORS Nickson Golooba (York University, Canada); Woldegebriel Assefa Woldegerima (York University, Canada)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 03.01 - SR
SESSION Contributed Talks
TRACK Cutting Edge Research Areas

Abstract

Physics-Informed Neural Networks (PINNs) are increasingly used in mathematical epidemiology to bridge the gap between noisy clinical data and compartmental models. However, training these hybrid networks is often unstable due to competing optimization objectives. As established in recent literature on “Gradient Pathology,” the gradient vectors derived from the data loss and the physical residual often point in conflicting directions, leading to slow convergence or optimization deadlock. While existing methods attempt to resolve this by balancing gradient magnitudes or projecting conflicting vectors, we propose an efficient alternative: Conflict-Gated Gradient Scaling (CGGS). This method utilizes the cosine similarity between the data and physics gradients to dynamically modulate the penalty weight. Unlike standard annealing schemes that only normalize scales, CGGS acts as a geometric gate: it suppresses the physical constraint when directional conflict is high, allowing the optimizer to prioritize data fidelity, and restores the constraint when gradients align. We prove that this gating mechanism preserves the standard $O(1/T)$ convergence rate for smooth non-convex objectives, a guarantee that fails under fixed-weight or magnitude–balanced training when gradients conflict. We demonstrate that this mechanism autonomously induces a curriculum learning effect, improving parameter estimation in stiff epidemiological systems compared to magnitude-based baselines.

CT1229 — Decision-making in heterogeneous self-propelled systems

SPEAKERS Víctor Villegas-Morral (Polytechnic University of Catalonia)
AUTHORS Gissell Estrada-Rodriguez (Polytechnic University of Catalonia); Marie-Therese Wolfram (University of Wolfram); Nastassia Pouradier Duteil (Paris Sorbonne University); Víctor Villegas-Morral (Polytechnic University of Catalonia)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Inspired by empirical observations in animal swarming – particularly in schooling fish – we propose an opinion-swarming model for self-propelled particles in order to understand the effect of uninformed individuals in consensus formation [Cou+11]. Building on classical bounded-confidence opinion models [HK02] and self-propelled swarming models [DOr+06], we introduce a three-population framework that distinguishes between leaders, followers, and uninformed individuals. Particles are described by their position, velocity, and a continuous opinion variable; they interact through self-propulsion, alignment, attraction-repulsion forces, and opinion-based mechanisms. First, we derive and analyse an individual-based coupled model that integrates spatial swarming dynamics with the evolution of individual opinions [EVW25]. We perform an extensive numerical study of the relevant parameters and their effect on the dynamics. To study the long-term behaviour of the system, we rigorously derive the mean-field partial differential equations for this coupled individual-based model following [HL09]. Our analysis reveals that uninformed individuals, despite lacking any opinion bias, significantly influence group dynamics by diluting the effect of leaders and promoting more democratic decision-making. These findings support the role of uninformed agents in collective decision-making and provide the first analytical insights into leadership and decision-making in heterogeneous crowds.

References

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[DOr+06] Maria R D’Orsogna et al. “Self-propelled particles with soft-core interactions: patterns, stability, and collapse”. In: *Physical review letters* 96.10 (2006), p. 104302.

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[HK02] Rainer Hegselmann and Ulrich Krause. “Opinion dynamics and bounded confidence: models, analysis and simulation”. In: *Journal of Artifical Societies and Social Simulation* (2002).

CT1565 — Decomposing noise in RNA sequencing experiments of adaptive immune repertoires

SPEAKERS Matthew Cowley (University College London)
AUTHORS Matthew Cowley (University College London); Gemma Freeman (University College London); Joseph Santiapillai (University College London); Gayathri Nageswaran (University College London); Suzanne Byrne (University College London); Andreas Tiffeau-Mayer (University College London); Benny Chain (University College London)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 01.14 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

RNA sequencing is a vital technology for immune repertoire profiling, allowing us to understand how the human adaptive immune system is structured. This technique enables clonal lineages to be tracked spatially and longitudinally: important for quantifying immune responses and the impact of immunotherapies [Oak+17]. However, non-Poissonian sources of noise exist in the data that can make decomposing biological and technical signals challenging [Gie+15]. Here, we conduct a variance decomposition experiment with blood draws from healthy volunteers, creating replicates at the repertoire sampling, library preparation, and sequencing levels. This strategy allows us to identify the contributions to noise from biological variation, molecular biology techniques, and short-read sequencing technologies. We then build an interpretable hierarchical statistical model informed by biological sampling mechanisms which captures the variance behaviour observed. We use this model to simulate the impact that sample properties, such as cell count, have upon the molecular mechanisms of noise. Finally, we use our mechanistic understanding to investigate suitable methods to account for non-Poissonian mean-variance relationships in adaptive immune repertoire sequencing data.

References

[Gie+15] Marek Gierliński et al. "Statistical Models for RNA-seq Data Derived from a Two-Condition 48-Replicate Experiment". In: *Bioinformatics* 31.22 (Nov. 2015), pp. 3625–3630. ISSN: 1367-4811, 1367-4803. DOI: 10.1093/bioinformatics/btv425. (Visited on 03/16/2026).

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CT1440 — Estimating directed connectivity from fMRI and structural priors via network diffusion model

SPEAKERS Ashish Raj (University of California San Francisco)
AUTHORS Ashish Raj (University of California San Francisco); Fahimeh Arab (University of California San Francisco)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Estimating whole-brain directed connectivity from resting-state fMRI remains an open challenge. Dynamic Causal Modeling (DCM) is prohibitive at whole-brain scale, while lagged correlations conflate direct and indirect propagation with common-input artifacts. We recover directed connectivity by fitting a Network Diffusion Model (NDM) to temporal dynamics. The NDM [AVR14] describes propagation on connectome S via $d\mathbf{x}/dt = -\beta(I - S)\mathbf{x}(t)$. Replacing symmetric S with unknown directed E, model-predicted lagged covariances:

$$\mathrm{FC}(\tau; E) = e^{-\beta t(I-E)} \left(e^{-\beta(t+\tau)(I-E)}\right)^T$$

are asymmetric for $\tau > 0$, encoding directional flow. We estimate E by fitting empirical lagged covariances across multiple lags, with sigmoid regularization constraining edges to structural pathways while permitting asymmetry. Applied to resting-state fMRI from 300 HCP [Van+13] subjects (100 cortical [Sch+18] + 14 subcortical regions), the directed network was sparser than empirical lagged FC, with all edges supported by structural connections. Net flow revealed consistent hierarchical organization across subjects: subcortical structures, V1, and limbic areas as sources, and somatomotor and dorsal attention areas as sinks. These findings show that temporal precedence in lagged correlations can be misleading, and that modeling propagation through network topology recovers directed pathways grounded in anatomy.

References

[AVR14] Farras Abdelnour, Henning U. Voss, and Ashish Raj. "Network diffusion accurately models the relationship between structural and functional brain connectivity networks". In: *NeuroImage* 90 (2014), pp. 335–347. DOI: 10.1016/j.neuroimage.2013.12.039.

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CT1311 — Evaluating the impact of school measures on early SARS-CoV-2 transmission in Portugal: a retrospective study

SPEAKERS Ganna Rozhnova (University Medical Center Utrecht)
AUTHORS Ana Nunes (Faculdade de Ciências da Universidade de Lisboa); Benedetta Canfora (Faculdade de Ciências da Universidade de Lisboa); Ganna Rozhnova (University Medical Center Utrecht); Otilia Boldea (Tilburg School of Economics and Managemet, Tilburg University); Rey Audie Escosio (Faculty of Sciences, University of Lisbon)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

School closures were among the most contentious non-pharmaceutical interventions during the pandemic due to their socio-economic costs and uncertainty surrounding their population-level epidemiological impact. Given the role of schools as key transmission settings, we retrospectively assessed their effect on SARS-CoV-2 dynamics in Portugal prior to mass vaccination.

We used an age-structured transmission model fitted to hospitalization and seroprevalence data within a Bayesian framework. We then compared the observed trajectories against two counterfactuals: schools remaining open during the lockdown in spring 2020, and schools closing for in-person education following the summer holidays. Elasticity analyses quantified the contributions of each age group to transmission.

Our results show that the 2020 pandemic in Portugal was primarily adult-driven. Keeping schools open during the first lockdown would have driven hospitalizations far beyond observed levels, unless school contacts reached at most 50% of pre-pandemic levels. In contrast, closing schools after summer, when adult-driven transmission was already high, would not have prevented the autumn wave, though it could have mitigated its impact.

These findings underscore that the epidemiological impact of school closures is highly context-dependent, shaped by within-school mitigation and community transmission. Integrated strategies combining both are essential to effectively curb transmission during future pandemics.

CT232 — Markov Process based Mathematical Model of Vascular Stenosis Formation and Treatment

SPEAKERS Taiga Kadowaki (Kyushu university)
AUTHORS Atsushi Tero (Kyushu university); Taiga Kadowaki (Kyushu university)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 11.11 - SR
SESSION Contributed Talks
TRACK Cardiovascular Modelling

Abstract

Vascular stenosis, the pathological narrowing of blood vessels due to atherosclerotic plaque, remains a primary driver of cardiovascular disease. Traditional computational fluid dynamics (CFD) models provide detailed hemodynamics but are computationally expensive, limiting their utility for long-term disease forecasting. To address this, we propose a highly efficient stochastic modeling framework designed to simulate both the formation and treatment processes of vascular stenosis. Instead of relying on complex, deterministic fluid calculations, our approach utilizes a probabilistic model based on discrete particle movement. The dynamics of these particles are governed by a combination of diffusion processes and an approximated flow field derived from the Stokes equations. This simplified structure successfully captures the essential macroscopic fluid-structure interactions and the microscopic probabilistic fluctuations of plaque growth. By significantly reducing computational loads without losing critical physical behaviors, this methodology offers a flexible and scalable platform for evaluating long-term disease progression and potential therapeutic interventions.

CT1237 — Predicting clogging in complex networks through a minimal continuum model

SPEAKERS Cara Neal (UCL)
AUTHORS Cara Neal (UCL); Duncan Hewitt (University of Cambridge); Philip Pearce (UCL)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Many biological, geophysical and industrial systems rely on the transport of material through fluid flow across complex networks. Suspensions in these systems are prone to clogging, particularly in confined geometries, with potentially severe consequences on system function. However, predictive frameworks that couple suspension rheology with network-scale flow redistribution remain limited. Here we develop a minimal continuum model for dense suspension transport in networks, combining a two-phase description of suspension flow with flux-partitioning rules at junctions.

Using simple network motifs, we show how constrictions impose finite solid-flux limits that redistribute particles between branches, drive upstream accumulation, and increase resistance. We demonstrate how these local mechanisms can give rise to strongly non-local effects, including non-local clogging. We formalise these processes through a clogging algorithm embedded within a global flow solver, enabling prediction of steady particle distributions and clogs in arbitrary networks.

Applying the framework to bio-inspired networks such as the retinal vasculature, we show how junction-level flux constraints combine to produce emergent network-scale heterogeneity via non-local clogging. Our results provide a mechanistic link between suspension physics, network geometry and transport, and offer a simple continuum approach for studying clogging in networks.

CT383 — Quality of information impacts growers' adoption of crop protection methods

SPEAKERS Frédéric Grognard (Université Côte d'Azur, Inria, INRAE, CNRS, MACBES)
AUTHORS Chiara Vanalli (ECEO, Ecole Polytechnique Fédérale de Lausanne); Frédéric Grognard (Université Côte d'Azur, Inria, INRAE, CNRS, MACBES); Frédéric Hamelin (Institut Agro, Université de Rennes INRAE, IGEPP); Ludovic Mailleret (Université Côte d'Azur, INRAE, CNRS, ISA)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Plant diseases have a major impact on crop production worldwide. However, the success of plant disease control methods depends on their adoption by growers ([MHC22]). In the present work, we propose a behavioral epidemiology model, in which a classical SI-model is intertwined with growers' behavior

in terms of roguing (removal of) infected plants or not. This ODE model is built on [MCH26] with variables representing infected and uninfected fields on which roguing is performed or not. The transition of a field from the non-roguing to the roguing pool (and vice-versa) occurs at a rate depending on the perceived payoff difference. Two kinds of perceived payoff differences are compared: *poor information*, in which growers believe roguing can fully control the disease and only know the overall disease prevalence, and *quality information*, in which they assess correctly the effects of roguing and precisely know the prevalences associated with both strategies. Bifurcation analyses show that quality of information is crucial in understanding expected growers' behavior and resulting disease dynamics. The main difference is that, when R_0 is large, poor-information growers keep roguing, while they stop roguing with quality information, realizing that roguing does not improve their revenue enough to compensate for its cost. In the latter case, the transition between universal adoption and no-adoption occurs through an R_0 range where both pure strategies are stable.

References

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CT1374 — Revealing Hidden Organization in High-Dimensional Cancer Proteomic Data: A Geometric and Topological Perspective

SPEAKERS SeyedSamie Alizadeh Darbandi (School of Mathematics and Statistics, The University of Melbourne, Melbourne, Australia)
AUTHORS Robyn Araujo (School of Mathematics and Statistics, The University of Melbourne, Melbourne, Australia); Patricia Menendez Galvan (School of Mathematics and Statistics, The University of Melbourne, Melbourne, Australia); SeyedSamie Alizadeh Darbandi (School of Mathematics and Statistics, The University of Melbourne, Melbourne, Australia)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

High-dimensional molecular profiling has transformed cancer research by enabling detailed characterization of tumor biology and more personalized therapies. Yet patients with highly similar clinical and molecular profiles can still exhibit markedly different treatment responses, suggesting that important biological information remains hidden within these datasets.

This work is motivated by the hypothesis that biologically meaningful information may lie not only in individual molecular measurements, but also in the organization of patient populations. We investigate this hypothesis through complementary geometric and topological perspectives, asking whether patient organization provides biological insight beyond individual molecular features.

To investigate this question, we employ a complementary geometric and topological framework to characterize relationships and connectivity among patients, rather than relying solely on low-dimensional representations or discrete patient groups. Using breast cancer proteomic data from a clinical trial, we show how this perspective provides a richer characterization of molecular heterogeneity by revealing organizational features not readily captured through conventional analyses.

More broadly, this work highlights the value of studying patient organization as a complementary perspective for understanding high-dimensional molecular data and demonstrates how geometric and topological analyses jointly reveal meaningful biological structure.

CT1259 — Sparsest steady-state relations from biochemical network structure

SPEAKERS Ahmad Mokhtar (The University of Melbourne)
AUTHORS Ahmad Mokhtar (The University of Melbourne); Haripriya Sridharan (The University of Melbourne); Robyn Araujo (University of Melbourne)
SCHEDULE Tuesday, 14 July 2026; 17:00–17:20; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

For a biochemical reaction network, the sparsest steady-state relations (that is, steady-state relations involving fewest chemical complexes) reveal the most direct dependencies in the system, and are closely connected to robustness properties. We may ask, what are the sparsest steady-state relations obtainable from all possible linear combinations of the rate equations, and what features of the network structure explain and govern their complexity? It turns out that matroid theory provides a natural answer to this question through two dual perspectives. From an algorithmic side, we characterise all sparsest generators within the linear span of the rate equations through a procedure which — as it requires no assumption on kinetics — applies beyond the mass-action setting. The complementary structural perspective elucidates the 'why'; the complexity of steady-state relations is controlled precisely by the interlinking pattern of network cycles, which may be studied in-depth using graph-theoretic and topological methods (particularly for large and complex networks with unknown kinetics). I will illustrate our analysis on several biological examples, including multisite phosphorylation, the EnvZ-OmpR signalling network, and the bifunctional enzyme system PFK-2/FBPase-2. This talk is based on joint work with R.P. Araujo and A. Mokhtar.

CT1466 — An Information-Based Dynamical Network Model of Multicellularity Across Dimensions

SPEAKERS Sabahat Defne Plattürk (Umea University, Department of Mathematics)
AUTHORS Sabahat Defne Plattürk (Umea University, Department of Mathematics)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The emergence of developmentally regulated life cycles, coordinating cellular growth with multicellular reproduction, was a key step in the evolution of complex multicellularity. Yet how cells use local information to regulate fragmentation in growing multicellular groups, and how these processes depend on spatial structure, remains unclear. Here, we introduce a computational dynamical-network model that tracks how cellular information is generated and propagated during multicellular growth. Starting from a single cell, clusters grow as dynamical networks embedded in spatial lattices representing one-dimensional filaments, two-dimensional hexagonal lattices, and three-dimensional face-centered cubic structures. We store information for each cell, such as cell age, physical stress, and the last reproduction time, which we use to trigger fragmentation events. We find that dimensionality strongly shapes the regulation of multicellular life cycles. In one-dimensional filaments, fragmentation rules can achieve any target size. However, clusters in two- and three-dimensional spaces do not stabilize at the target size but instead maintain the target mean. Our results also reveal characteristic life-cycle patterns that emerge across dimensions. Furthermore, spatial constraints in higher dimensions limit the smallest achievable mean group size, creating new challenges for the evolution of regulated multicellular life cycles.

CT1625 — Bistability between acute and chronic states in a Model of Hepatitis B Virus Dynamics

SPEAKERS Hayriye Gulbudak (University of Louisiana at Lafayette)
AUTHORS Hayriye Gulbudak (University of Louisiana at Lafayette); Jessica Conway (Pennsylvania State University); Nazia Afrin (University of Louisiana at Lafayette); Stanca Cuipe (Virginia Tech)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 01.14 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Understanding the mechanisms responsible for different clinical outcomes following hepatitis B infection requires a systems investigation of dynamical interactions between the virus and the immune system. To help elucidate mechanisms of protection and those responsible from transition from acute to chronic disease, we developed a deterministic mathematical model of hepatitis B infection that accounts for cytotoxic immune responses resulting in infected cell death, non-cytotoxic immune responses resulting in infected cell cure and protective immunity from reinfection, and cell proliferation. We analyzed the model and presented outcomes based on three important disease markers: the basic reproduction number R_0, the infected cells death rate δ (describing the effect of cytotoxic immune responses), and the liver carrying capacity K (describing the liver susceptibility to infection). Using asymptotic and bifurcation analysis techniques, we determined regions where virus is cleared, virus persists, and where clearance-persistence is determined by the size of viral inoculum. These results can guide the development of personalized intervention.

CT1438 — Construction and simulation of a path-valued model of dendrite growth

SPEAKERS Andrew Nugent (University College London)
AUTHORS Andrew Nugent (University College London)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Neurons receive information through their dendrites. During development, dendrites grow, retract, and branch as they search for connections, and the resulting dendritic arbor shapes the structure of the broader neural network. This talk introduces a flexible model describing the full spatial structure of a single neuron through three processes: a lifetime process that determines whether each dendrite is growing or retracting; a growth process describing dendritic extension in space; and a branching process that controls the generation of new branches. Crucially, retraction and branching make it necessary to track entire dendritic paths rather than only their endpoints. While this is handled implicitly in many existing simulations, we present here an explicit construction of a path-valued stochastic process for modelling dendrites. Numerical simulations demonstrate that the model can adapt to a range of developmental environments.

CT334 — Extension of an established dynamic thyroid model for capturing Graves' disease including antibody production

SPEAKERS Thomas Benninger
AUTHORS Markus Reichhartinger (TU Graz); Matthias Müller; Thomas Benninger; Tobias Wolff
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Dynamic mathematical models of the hypothalamic–pituitary–thyroid (HPT) axis provide a framework for studying thyroid physiology. A widely used model originally developed in [Die02; Die+04] has been refined and extended over the past two decades [Ber+18]. This model captures the HPT feedback loop, including TSH, T4, T3, and additional regulatory factors [Die02; Ber+18]. The model was extended to simulate Graves' disease (GD) and the effect of methimazole (MMI) [Wol+25]. In this formulation, the influence of TSH receptor antibodies (TRAb) was not modeled explicitly but was represented by an increased secretory capacity of the thyroid gland. The thyroid secretion rate was fixed at a pathologically elevated level, representing a persistent hyperthyroid state. In GD, however, TRAb constitutes the primary disease-driving factor. In the present work, we extended the widely used model by integrating mechanisms describing antibody-driven stimulation of the thyroid gland. To achieve this, the model was combined with the DigiThy simulation framework [The+22], which has been evaluated against clinical data from patients with GD and explicitly models TRAb dynamics. This enables the simulation of heterogeneous disease courses, including remission and sustained euthyroid states after discontinuation of antithyroid medication. The resulting combined model integrates detailed thyroid hormone regulation with explicit modeling of antibody dynamics and disease progression in GD.

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CT1510 — Mechanistic modelling of adipocyte lipid droplet dynamics

SPEAKERS Thomas Reed (University of Oxford)
AUTHORS Alexander Zagajewski (Novo Nordisk Research Centre Oxford); Helen Byrne (University of Oxford); Ruth Baker (University of Oxford); Thomas Reed (University of Oxford)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 11.34 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Lipid droplet dynamics during adipogenesis play a central role in adipocyte function and metabolic health, yet the mechanisms governing droplet formation and growth remain poorly quantified. We present a mechanistic ordinary differential equation (ODE) model describing the evolution of lipid droplet size distributions measured in time-resolved imaging experiments of differentiating adipocytes. The model captures three key processes –droplet biogenesis, fusion, and lipid accumulation – and predicts

the dynamics of droplet populations across discrete size classes. The model is calibrated to CRISPR knockdown adipogenesis assays from the Novo Nordisk Research Centre Oxford, where lipid droplet sizes are quantified from brightfield imaging over a 14-day differentiation period. Sensitivity analysis shows that biogenesis primarily determines droplet abundance, whereas growth controls total lipid storage, with fusion contributing more weakly. This approach enables gene perturbations to be interpreted in terms of their effects on underlying droplet processes, providing a tractable framework for extracting mechanistic insight from large-scale adipogenesis datasets.

CT274 — Modelling interclonal cooperation in epithelial carcinogenesis using spatial models

SPEAKERS Muhammad Asim Farooq (University of Sydney)
AUTHORS Domenic Germano (University of Melbourne); Muhammad Asim Farooq (University of Sydney); Peter S. Kim (University of Sydney)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

This study uses vertex- and Voronoi-based computational models based on Chaste [Coo+20] to investigate interclonal cooperation within cell populations. We analyze interactions between mutated cell types with distinct hyperproliferative and invasive properties. Modeling the cooperative dynamics among these clones shows how cellular heterogeneity affects tissue growth and invasion, enhancing the understanding of tumor progression and therapeutic target predictions.

References

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CT1234 — Multi-stage volume exclusion models for cell proliferation

SPEAKERS John Carlo Dimaculangan (University of Bath)
AUTHORS Cameron Smith (University of Bath); Chrisitian Yates (University of Bath); John Carlo Dimaculangan (University of Bath)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Cell proliferation and cell movement are fundamentally stochastic processes which lead to variability in the growth and spatial structure of cell populations in many biological settings, such as cell invasion, wound healing, and tumour growth. We develop stochastic, on-lattice agent-based models (ABMs) which incorporate volume exclusion, random movement, and multi-stage representations of the cell cycle. The multi-stage framework enables a more realistic representation of true cell cycle time distributions. We also introduce a novel form of myopic behaviour, where cells sense their local environment when attempting to proliferate. For each ABM, we derive a corresponding continuum partial differential equation (PDE) description under the mean-field approximation. Using numerical simulations, we investigate how different proliferation mechanisms influence population-level dynamics in both the discrete and continuum models. In particular, we consider the biologically relevant contexts of growth-to-confluence assays under uniform initial conditions and travelling wave behaviour associated with cell invasion. We examine how the PDE solutions compare with the average behaviour of the corresponding ABMs over many realisations.

CT1305 — Optimal epidemic mitigation using focused and unfocused interventions

SPEAKERS Martin Bootsma (University Medical Center Utrecht & Utrecht University)
AUTHORS Biplab Maity (Graduate School of Advanced Life Sciences, School of Life Sciences, Hokkaido University, Japan); Martin Bootsma (University Medical Center Utrecht & Utrecht University)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Strategies to mitigate an infectious disease outbreak can be focused or unfocused. Focused interventions (FI) aim to limit transmission from known or likely infectious individuals (e.g., quarantining contacts). Unfocused interventions (UI) affect all individuals irrespective of infection status by lowering the effective contact rate (e.g., lockdowns or universal masking). The costs of FI typically scale with disease prevalence, whereas the costs of UI are largely independent of prevalence but increase rapidly with intervention severity.

We investigate the optimal allocation of resources when both intervention types are available. Using compartmental epidemic models, we analyze optimality for (i) the final epidemic size and (ii) the peak epidemic height, considering cases where isolated infected individuals do or do not contribute to the peak. We consider both a fixed per-period budget and a fixed total budget for the epidemic.

For final size minimization, minimizing the reproduction number is optimal for a fixed per-period budget. With a fixed total budget, interventions should be applied when they yield the largest reduction in infectious output. If FI costs scale linearly with prevalence, timing is irrelevant as long as the budget is used. UI should be applied around the peak. To minimize peak height, FI should start early, while UI should be applied around the peak. Qualitative results are robust to disease characteristics and intervention effectiveness.

CT1236 — Optimizing the resources allocation between biochemical defence and counter-counter defence in pathogenic and trophic interactions.

SPEAKERS Lukas Korn (Friedrich-Schiller-Universität Jena)
AUTHORS Lukas Korn (Friedrich-Schiller-Universität Jena); Stefan Schuster (Friedrich-Schiller-Universität Jena); Suman Chakraborty
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

In many interactions between hosts and pathogens or plants and herbivores, the attacked organism produces a chemical compound to defend itself. The attacker often responds to this by producing a counter defence, e.g. an enzyme that can degrade this defence chemical. Sometimes, the defender responds by producing another chemical, a counter-counter defence, that can inhibit the degrading enzyme. These interactions often attain a steady-state. In this theoretical study, we analysed under which conditions it is favourable to invest resources into the production of a counter-counter defence and, if so, to what extent. A mathematical model based on enzyme kinetics was developed. For this model, we derived the equation for the steady-state concentration of the defence chemical and solved a resource allocation problem with the goal of maximising this concentration. This revealed that only if the inhibitor shows certain properties it is favourable to develop a counter-counter defence. We also found that it depends on the amount of available resources, whether the production of an inhibitor is beneficial. However, it is never optimal to invest more resources into the inhibitor than into the toxin, provided that their costs are approximately equal. The results can be of interest for calculating the optimal mixing ratios in antibiotics and other drugs that are given for a prolonged time.

CT1499 — Quantitative Modelling of Microbial Colony Growth Dynamics

SPEAKERS Attila Csikász-Nagy (Pázmány Péter Catholic University)
AUTHORS Attila Csikász-Nagy (Pázmány Péter Catholic University)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 03.01 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Understanding how spatial structure and environmental variability shape microbial colony growth and inter-strain interactions remains a central challenge in systems biology. I will present an integrated experimental–computational framework for the quantitative analysis of *Saccharomyces cerevisiae* colonies on solid media under both standard and non-standard environmental conditions. Experimentally, we developed a scalable workflow combining automated sample preparation, time-lapse imaging, quantitative image analysis, and fluorescence microscopy to extract high-content measurements of colony expansion and spatial organization. Computationally, we introduced a similarity metric to compare colony growth trajectories and infer competitive dominance in mixed colonies, and validated these predictions against fluorescence-based measurements. W developed and parameterized a quantitative agent-based model to reproduce colony size and cell number across perturbations in humidity, nutrient availability, and inoculation geometry. The combined results reveal complex, environment-dependent interaction networks and demonstrate that initial colony spread has a stronger influence on final colony size and cell number than initial cell number. Together, this work establishes a general modelling and data-analysis framework for linking spatial growth dynamics, environmental heterogeneity, and microbial interactions in structured communities.

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CT258 — Saddle-Node Bifurcation in Macrophage Proliferation Determines Atherosclerotic Plaque Stability

SPEAKERS Emine Atıcı Endes (Ondokuz Mayıs University)
AUTHORS Emine Atıcı Endes (Ondokuz Mayıs University); Neslihan Nesliye Pelen (University of South Florida)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 11.11 - SR
SESSION Contributed Talks
TRACK Cardiovascular Modelling

Abstract

Atherosclerotic plaques are a leading cause of heart attacks and strokes. Macrophage proliferation drives plaque growth, but whether this promotes stability or triggers sudden destabilisation remains unresolved. Using bifurcation analysis of a lipid-structured atherosclerosis model [CWM24], we uncover the nonlinear dynamics governing this transition.

Our main contribution is that the reduced dynamics remain meaningful beyond previously identified validity limits. Combining numerical bifurcation with fast–slow analysis and Fenichel’s theory, we identify a saddle-node bifurcation at infinity. This creates a sharp threshold where proliferation balances emigration. Below this balance, the system stabilises in a biologically reasonable state; above it, macrophage numbers and lipid load grow unboundedly, causing runaway inflammation and plaque instability. Determinant and eigenvalue trends corroborate this bifurcation.

Parameter investigations suggest that increased proliferation or reduced emigration raises macrophage accumulation and necrotic core lipid content. Efferocytosis modulates downstream severity but does not shift the threshold. These findings reconcile conflicting views: proliferation is protective when emigration provides an adequate balance. Co-targeting reduced proliferation and enhanced emigration could maintain plaque stability and reduce cardiovascular risk, which is a prediction warranting experimental investigation.

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CT384 — Transmission Dynamics of Eastern Equine Encephalitis: Global Sensitivity Analysis and SHAP Parameter Importance in an Age-Structured Vector-Host Model

SPEAKERS Christopher Mitchell (University of Texas at San Antonio)
AUTHORS Aurod Ounsinegad (University of Wisconsin-Madison); Christopher Mitchell (University of Texas at San Antonio)
SCHEDULE Tuesday, 14 July 2026; 17:20–17:40; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Eastern equine encephalitis virus (EEEV) is a deadly arboviral pathogen with 30% severe case fatality. EEEV exhibits pronounced 2–3 year cyclical outbreak patterns in the northeastern United States, linked to shifts in mosquito feeding preferences between hatchyear and adult avians. An age-structured vector-host model incorporating differential feeding patterns of Culiseta melanura mosquitoes on European Starlings and American Robins was developed. Sensitivity analysis revealed mosquito biting rate as a dominant driver in transmission, with avian infectivity and exposure playing secondary roles. Pairing machine learning algorithms with SHAP analysis on parameter sets identified parameter hierarchies that govern cyclic transmission. Adult avian mortality was identified as the key parameter underlying cyclic and stable transmission patterns. SHAP further revealed these patterns to be grounded on opposite sides of the same epidemiological mechanism. Stable endemics emerge from demographic stability paired with transmission optimization, while cyclic endemics emerge from demographic instability paired with minimal transmission optimization. Numerical simulations illustrated critical threshold dynamics for exposure towards young, where heightened hatch-year exposure triggers demographic instability responsible for observed 2–3 year cycles, while balanced exposure leads to stable endemics. These mechanisms provide a foundation for targeted surveillance and control interventions.

CT1261 — Autocorrelation-Based Inference of Transcription Dynamics via Infinite-Server Queueing Models

SPEAKERS Jeremy Worsfold (University of New South Wales)
AUTHORS Bailey Hiles-Murison (University of New South Wales); Jeremy Worsfold (University of New South Wales); Richard Morris (University of New South Wales); Scott Berry (University of New South Wales)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Live-cell imaging experiments often track mRNA production using fluorescently tagged transcripts. To infer the statistics of initiation events, standard methods assume that transcription has a fixed duration. However, studies of intron splicing and transcriptional pausing show that stochasticity in elongation and termination are often significant and cannot be ignored. In this talk, we advocate an alternative approach: fitting directly to the autocorrelation function of the fluorescence intensity signal. Building on recent links between transcriptional models and infinite-server queueing systems, we use theoretical autocorrelation expressions to infer parameters in models that incorporate stochasticity in both gene switching and transcription time. This framework provides richer mechanistic insight than approaches requiring fixed transcription durations. We illustrate its advantages using recent live-cell imaging data, fitting autocorrelations under a delay Telegraph model that includes an additional exponential waiting time in the transcription process.

CT1233 — Emergent mechanics from material replacement in biological systems

SPEAKERS Matthew Butler (University of Strathclyde)
AUTHORS Matthew Butler (University of Strathclyde)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Many biological systems contain material components that are repaired and replaced over time. One common example is the extra-cellular matrix, an interconnected network of proteins that provides chemical and mechanical protection and support in many systems, for example in tissue basement membranes and bacterial biofilms. The building blocks in these systems can be supplied by accompanying cells, with stressed, damaged or degraded material extracted, leading to a turnover in the underlying material of the network. Questions remain as to how the bulk mechanical properties of the material is affected by this microscale material replacement. I will present a mathematical model, based on a system of springs, that aims to capture how microscale replacement of elastic material can lead to different observed bulk mechanical behaviours. This model evolves an underlying distribution of material via a population-style model governed by underlying energy barriers, with feedback between the material distribution and the local mechanical deformation. Numerical experiments and mathematical analysis reveal emergent viscoelastic behaviours over sufficiently long times, despite the system behaving purely elastically at any instant. We use this to determine effective bulk parameters from supplied microscale behaviours. Despite the model's simplicity, it has interesting characteristics that are biologically-relevant.

CT1451 — Generating Vascular Network Structures from Real Data

SPEAKERS Rui Travasso (University of Coimbra)
AUTHORS Duarte Leite (University of Coimbra); Ernesto Costa (University of Coimbra); João Simões (University of Coimbra); Rui Travasso (University of Coimbra); Tiago Baptista (University of Coimbra)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Blood vessels are essential components of the mammalian circulatory system, responsible for delivering nutrients and oxygen to tissue cells. Despite recent advances in understanding the biological mechanisms underlying vascular growth, researchers have not yet tackled the generation of synthetic vascular structures using deep learning models that are trained with existing datasets.

This work addresses these challenges by introducing a novel computational approach to generate three-dimensional vascular networks [Sim+25]. By using an encoder-decoder architecture, we treat these

networks as complex spatial graphs in a three-dimensional space, learning sequential moves to create new nodes and edges that mimic real vascular structures. Without replicating the real-truth network, our method can generate new vascular networks with structural characteristics identical to those of existing real data, such as branching pattern, vessel lengths, network density, and degree distribution. Moreover, the generated networks are capable to provide sufficient oxygen perfusion to the tissue. The parameters of the model can be adjusted to regulate and fine-tune the morphology of the generated networks.

The achieved results not only demonstrate the potential of our approach in accurately modelling vascular structure but also open new avenues for exploring data-driven models in tissue engineering and to complement imaging data collected in clinical and research contexts.

References

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CT293 — Interpretable measures for sensitivity and identifiability in models of infectious disease

SPEAKERS Tarek Alrefae (University of Oxford)
AUTHORS Tarek Alrefae (University of Oxford)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Although ordinary and partial differential equation (ODE/PDE) models are widely used in infectious disease inference and public health decision-making, parameter sensitivity and identifiability are often assessed with tools that lack mechanistic interpretability and overlook sensitivity to initial conditions or derived epidemiological quantities. We present a framework for generating interpretable measures of sensitivity and identifiability by computing forward sensitivities, elasticities, and singular value decomposition (SVD) diagnostics of sensitivity structure. Using a susceptible-exposed-infected-recovered model as an example, we augment the system with sensitivity equations using the Jacobian matrix, incorporate an observation mapping from the full (unknown) state space to observed data, and treat initial conditions as parameters. We evaluate sensitivities of both model states and derived latent quantities. Sensitivities and elasticities identify distinct periods of parameter importance, while SVD diagnostics reveal shifting dominant parameter combinations and when inference is stronger or weaker during an outbreak under different observation models. We extend the framework to a PDE vector-borne disease model and a serocatalytic model, demonstrating scalability and the impact of observation design on identifiability. This workflow supports model calibration, experimental design, and early diagnosis of practical identifiability issues.

CT1303 — Mapping bovine trypanosomosis under diagnostic uncertainty: spatial prevalence estimates in Nigeria and Ethiopia

SPEAKERS Alex Kaye (School of Life Sciences, University of Warwick, Coventry, UK)
AUTHORS Alex Kaye (School of Life Sciences, University of Warwick, Coventry, UK); Antoine Barreaux (ICIPE, Nairobi, Kenya); Giuliano Cecchi (Food and Agriculture Organization of the United Nations (FAO), Animal Production and Health Division, Rome, Italy); Hailemariam Kefyalew (Animal Health Institute (AHI), Addis Ababa, Ethiopia); Harriet Wangu (ICIPE, Nairobi, Kenya); Jill de Gier (Food and Agriculture Organization of the United Nations (FAO), Animal Production and Health Division, Rome, Italy); Joachim Ajakaiye (Federal Ministry of Innovation, Science and Technology (FMIST), Abuja, Nigeria); Lena Halos (Gates Foundation, Seattle, USA); Massimo Paone (Food and Agriculture Organization of the United Nations (FAO), Animal Production and Health Division, Rome, Italy); Mike Tildesley (School of

Life Sciences, University of Warwick, Coventry, UK); Philipe Solano (INTERTRYP, Univ Montpellier, Cirad, IRD, Montpellier, France); Sophie Thévenon (Cirad, UMR INTERTRYP, F-34398 Montpellier, France); Warren Tennant (Communicable Disease Surveillance Centre (CDSC), Public Health Wales, Cardiff, UK)

SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

African animal trypanosomosis (AAT) imposes a major constraint on livestock productivity throughout sub-Saharan Africa, but its true prevalence is underestimated due to the limited sensitivity of widely used diagnostic methods, namely the haematocrit centrifugation and buffy coat techniques (HCT/BCT). Although polymerase chain reaction (PCR) testing provides improved sensitivity, it remains expensive and is seldom accessible in routine field conditions. We developed a Bayesian hierarchical latent class framework using bovine trypanosomosis data compiled from more than 40 studies across Africa to infer the sensitivity and specificity of HCT/BCT and PCR. These estimates were then used to correct apparent prevalence, followed by geostatistical modelling in two high-burden, data-rich settings: Ethiopia and Nigeria. Two key forms of uncertainty were explicitly characterised: diagnostic uncertainty arising from imperfect tests, and spatial coverage uncertainty due to uneven sampling. Estimated sensitivities were 29.8% for HCT/BCT and 68.9% for PCR, while specificities were above 99% for both. After accounting for diagnostic performance, mean prevalence increased from 0.086 to 0.299 in Nigeria and from 0.092 to 0.382 in Ethiopia, corresponding to approximately 1.93 million and 3.39 million infected cattle within the tsetse belt. Across endemic regions, diagnostic uncertainty was the predominant contributor. Incorporating test uncertainty into spatial modelling provides a more accurate representation of AAT burden, supporting improved surveillance and more effective targeting of control strategies, with broader relevance to other neglected tropical diseases.

This work was developed in the framework of the FAO project ‘Disease intelligence and modelling for progressive control of animal trypanosomosis in Africa’ (DIMCAT), supported by the Gates Foundation (INV-067581).

CT277 — Mathematical modeling of radiotherapy response in murine glioma using MRI-defined tumor habitats

SPEAKERS Ayesha Das (University of Texas Austin)
AUTHORS Ayesha Das (University of Texas Austin); David A Hormuth (University of Texas Austin); Jack Virostko (University of Texas Austin); Patrik Parker (University of Texas Austin); Thomas Yankeelov (University of Texas Austin)

SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Spatiotemporal variations in hypoxia, cellularity, and vascularity affect the response of glioma to radiotherapy (RT). MRI can quantitatively map these features, identify tumor habitats, and calibrate biology-based models to predict the response to different RT protocols. Our long-term goal is to determine optimal RT strategies through predictive modeling.

Twenty-five female Wistar rats bearing C6 gliomas underwent multiparametric MRI every 72 hours for up to seven visits. RT was administered immediately after the first scan. Animals were assigned to the following groups: one 20 Gy dose ($n = 4$), two 10 Gy doses ($n = 6$), four 5 Gy doses ($n = 6$), five 4 Gy doses ($n = 6$), and a control group ($n = 4$). For fractionated regimens, RT followed the corresponding imaging visit.

We employed five different approaches to analyze tumor habitats with ODE based models by varying the number of habitats (up to four) and number of parameters identified in the imaging data. All models

were run on MATLAB (Mathworks, Natick, MA), using a finite difference approximation. The most parsimonious was selected via the Akaike Information Criterion (AIC).

The model with two habitats and two death terms when compared to the experimentally acquired data had the highest overall CCC value of 0.95 and the lowest AIC when averaged over the cohort. Future work will involve combining these approaches within a data assimilation framework to enable predictions of future habitat dynamics given previous tumor states.

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CT1576 — Membrane Lipid Dynamics in Macrophage Chemotaxis

SPEAKERS Joshua Forrest (University of Melbourne)
AUTHORS Joshua Forrest (University of Melbourne); Robyn Araujo (University of Melbourne)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 11.34 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Macrophages play an important role in the body's innate immune system. They digest smaller external threats, promote healing of internal wounds, and recruit other macrophages using chemokines (chemical signals).

These chemokines bind to specialised receptors on the cell membrane that trigger downstream signals. The receptor CXCR3 in macrophages, for example, is activated by the chemokines CXCL9 and CXCL11. These signals activate membrane-bound phospholipids, which recruit actin remodelling proteins to the cell wall. These membrane lipids can therefore act as a "compass" that point towards the chemical signal and guide the direction of cell motility.

The membrane lipids appear to "lock in" the upstream signal by limiting the ability to change course. How quickly does a macrophage identify the direction of chemotaxis? How does a macrophage respond to competing or conflicting chemotaxis signals? What chemokine concentration is required to override an existing signal?

To answer these questions, we developed a mathematical and computational model of macrophage chemotaxis that combines the internal signalling network cascades with a model of spatially diffused chemokines. We use this model to compare chemotaxis performance under different parameter values and

initial conditions. In particular, we measure the relative strength and efficiency of each signal and the persistence of chemotactic movement in the presence of conflicting signals.

CT1362 — New topologies in the unfolding of a high codimension singularity organizing neuronal dynamics

SPEAKERS Marisa Saggio (Aix-Marseille University, Institut de Neurosciences des Systèmes (INS, UMR1106))
AUTHORS Marisa Saggio (Aix-Marseille University, Institut de Neurosciences des Systèmes (INS, UMR1106))
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 01.14 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

High-codimension bifurcations critically contribute to shaping the dynamics of nonlinear models, as their unfoldings establish structured relationships between lower-codimension bifurcations and, ultimately, the attractors they generate. Owing to this unifying and predictive capacity, such bifurcations are attracting growing interest in mathematical biology.

One notable example is the codimension-3 Degenerate Bogdanov-Takens (DBT) singularity, proposed in the literature as an organizing center for neural dynamics, and playing a key role in fast-slow bursting. The DBT itself arises within the unfolding of an even higher-order singularity, the Doubly Degenerate Bogdanov-Takens (DDBT), whose structure remains only partially understood [KKR98; KO16].

We numerically explore the DDBT four-dimensional unfolding using spheres in parameter space, highlighting similarities and differences with existing conjectures. We show that using planes to explore the unfolding allows for additional bifurcation topologies. We reference the literature for examples of how these topologies emerge in neural dynamics.

Even though one key transition remains to be solved to complete the puzzle, the intermediate configurations and additional bifurcation structures reveal a rich repertoire of dynamical behaviors. These results improve our understanding of what models with a DDBT point can do and how these behaviors are related [Sag25].

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CT1235 — Role of phenotypic plasticity in community assembly and coexistence: from a model organism to theory and back

SPEAKERS Ata Kalirad (MPI Biology Tübingen)
AUTHORS Ata Kalirad (MPI Biology Tübingen)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

The role of phenotypic plasticity in community assembly and promoting and/or hindering coexistence is widely acknowledged but remains poorly understood. The nematode *Prisitiounchus pacificus* exhibits a case of environmentally-induced phenotypic plasticity in which an adult worm irreversibly develops either a predatory or a non-predatory mouth form. Additionally, the natural lifecycle of *P. pacificus* and related nematodes involve the assembly of transient communities on the carcass of the Gymnogaster beetles, followed by the dispersal of the nematodes in the environment [Som25].

We take advantage of the nematode communities emerging on decomposing beetles to construct a range of models, in pursuit of an all-encompassing framework to explore community assembly and dynamics when phenotypic plasticity and predator-prey interactions coincide. On the one hand, we have constructed agent-based models to reconstruct how communities of nematodes assemble in the wild [KS24]. To complement this approach, we have developed more abstract models - e.g., the ecological graph theory [KS25] - to investigate general patterns related to the assembly of such communities over ecological and evolutionary timescales. In this contribution, I will discuss the insights and lessons learned and argue that this approach promises a novel natural-history-informed theoretical understanding of some of the most foundational questions in community ecology.

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CT1428 — The exponential distance rule-based network model predicts topology and reveals functionally relevant properties of the Drosophila projectome

SPEAKERS Balazs Pentek (Faculty of Physics, Babeș-Bolyai University, Cluj-Napoca, Romania)
AUTHORS Balazs Pentek (Faculty of Physics, Babeș-Bolyai University, Cluj-Napoca, Romania); Maria Ercsey-Ravasz (1. Physics Department of the Babes-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Studying structural brain networks has witnessed significant advancement in recent decades. Findings revealed a geometric principle, the exponential distance rule (EDR) showing that the number of neurons decreases exponentially with the length of their axons. This neuron-level information was used to build a region-level EDR network model that was able to explain various characteristics of interareal cortical networks in macaques, mice, and rats. The complete connectome of the Drosophila has recently been mapped providing information also about the network of neuropils (projectome). A recent study demonstrated the presence of the EDR in the Drosophila. In our study, we first revisit the EDR itself and precisely measure the characteristic decay rate. Next, we demonstrate that the EDR model effectively accounts for numerous binary and weighted properties of the projectome. Our study illustrates that the EDR model is a suitable null model for analyzing networks of brain regions, as it captures properties of region-level networks in very different species. The importance of the null model lies in its ability to facilitate the identification of functionally significant features not caused by inevitable geometric

constraints, as we illustrate with the pronounced asymmetry of connection weights important for functional hierarchy.

CT243 — Topological Morphological Descriptor Enables Artery–Vein Discrimination in Whole-Mouse Vasculature

SPEAKERS Sofia Farina (ARTORG University of Bern)
AUTHORS Gabrièle Lienhard (ICM Paris Brain Institute); Maxime Boyer (ICM Paris Brain Institute); Nicolas Renier (ICM Paris Brain Institute); Sofia Farina (ARTORG University of Bern); Franca Schmid (ARTORG University of Bern)
SCHEDULE Tuesday, 14 July 2026; 17:40–18:00; 11.11 - SR
SESSION Contributed Talks
TRACK Cardiovascular Modelling

Abstract

Accurate artery–vein labelling is essential for the analysis of whole-brain vasculature and blood flow simulations to understand cerebrovascular organisation and function. However, most datasets lack artery–vein annotations [Tod+20], since labeling is generally possible only at acquisition using specific staining protocols [Kir+20].

Despite reported topological differences between penetrating arteries and veins [Sch+19], whole-brain manual labeling is impractical, motivating automated mathematical approaches. Using graph-based representations of penetrating trees extracted from whole-brain mouse vasculature with acquisition-derived artery–vein labels [Kir+20], we apply computational topology to compute a topological morphological descriptor (TMD) [Kan+18; Bee+23]. TMD captures topological and geometric features from vascular trees and encodes them as persistence barcodes, which are then transformed into persistent images.

Using persistent images to train a convolutional neural network, we achieve classification accuracies exceeding 80%, demonstrating that persistent homology provides discriminative power for vascular labeling. A Random Forest classifier trained on alternative features reaches similar accuracy, showing that artery–vein separation is learnable across different modeling choices. Finally, the framework generalizes to graph-based unlabeled datasets, enabling scalable, mathematically grounded artery–vein classification.

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CT1247 — AI-enabled discovery of mechanistic mediators in Alzheimer’s disease progression

SPEAKERS Ashish Raj (Department of Radiology and Biomedical Imaging, University of California, San Francisco)
AUTHORS Ashish Raj (Department of Radiology and Biomedical Imaging, University of California, San Francisco); Heng Rao (College of Computer Science and Engineering, Northeastern University, China); Minghan Chen (Department of Computer Science, Wake Forest University, USA); Nuutti Barron (Department of Radiology and Biomedical Imaging, University of California, San Francisco)

SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 01.14 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Tauopathies are a class of neurodegenerative diseases, such as Alzheimer's disease, characterized by a progressive accumulation of toxic misfolded tau proteins in the brain [Iqb+10]. Model-based approaches have shown that this progression, or "staging," follows a tau-spread process along the brain's white matter connective network, dubbed the "structural connectome" (SC). The Network Transport Model (NTM) [Bar+26a] was developed to build upon simple diffusion based models of tau spread [RKW12; SMK20] and demonstrate how global patterns of tau spread emerge from microscopic tau dynamics on the SC. However, the NTM suffers from an exceedingly high computational cost and slow simulations, hindering studies relating tau dynamics to global tau progression.

To overcome this challenge we developed Tau-BNO [Bar+26b], a neural operator based surrogate model that learns the behavior of the NTM. The rapid NTM simulations of tau spread allowed by Tau-BNO reveal insights into how microscopic tau dynamics modulate global progression of tau. Cellular tau uptake and release was shown to be a direct modulator of the timeline of disease progression, while directional bias in tau transport regulated disease staging. Tau aggregation was shown to sequester tau available to spread while increasing the global burden of toxic tau aggregates. Tau-BNO made it possible to study the effect of microscopic tau dynamics on global disease patterns, showcasing the value of deep learning in biological discovery.

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CT1294 — Bayesian Inference for Outbreak Final Size Datasets with Underlying Household Structure

SPEAKERS Joseph Brooks (University of Manchester)
AUTHORS Joe Hilton (University of Manchester); Lorenzo Pellis (University of Manchester); Joseph Brooks (University of Manchester); Thomas House (University of Manchester)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Households represent a key unit of interest in infectious disease epidemiology, in both empirical studies and mathematical modelling. The within-household transmission potential of an infectious disease is often summarised in terms of a secondary attack ratio. Despite their widespread use, estimates of secondary attack ratio depend on the distribution of household compositions seen during the study period, making

them poor indicators of transmission potential in new populations and future epidemic phases. Extending estimates of transmission potential to new populations instead requires estimates of person-to-person transmission rates which can be convoluted with data on population structure to parameterise mechanistic transmission models.

This talk will present a new inference method for estimating the transmission intensity by imputing the unreported household structure underlying the epidemic dynamics with MCMC. This method can be run on household epidemiological data reported at varying levels of resolution. The algorithm was able to reliably recover parameters from synthetic data and provide estimates of transmission rates from existing datasets that could not otherwise be used to directly parameterise transmission models. These results also aim to encourage the reporting of higher resolution outputs in future epidemiological field work as the transmission parameters were not identifiable from low resolution datasets, which are commonly reported.

CT1254 — Cellular resource allocation in budding yeast beyond steady state

SPEAKERS Weida Liao (Imperial College London)
AUTHORS Weida Liao (Imperial College London); Yu Huo (University of Edinburgh); Peter Swain (University of Edinburgh); Vahid Shahrezaei (Imperial College London)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Microbes adapt their physiology in order to grow in a wide variety of environments. Cellular growth laws quantify the relationship between the levels of essential resources such as ribosomes in a cell and the growth rate under steady-state conditions. However, in nature, the environment inhabited by a cell typically varies with time. A key question is therefore how an individual cell allocates its resources dynamically, as it exits and enters steady-state growth.

Here we present a mathematical model of cell physiology in arbitrary time-dependent conditions. Adopting a coarse-grained approach, we group molecules into broad categories based on their function. We demonstrate how the time-dependent resource allocation fractions may be inferred from plate-reader data for population growth rate and ribosome fraction. We apply our framework to experimental data from budding yeast, a model eukaryotic organism, grown in a range of carbon concentrations and exposed to different levels of translation-inhibiting drug. Our results reveal the principles of transient resource allocation and the potential regulatory mechanisms governing the behaviour of the cell. Our work provides a general quantitative method to elucidate cellular growth beyond steady state.

CT1600 — Deciphering Spatiotemporal Dynamics of Oocyte Selection during Ovarian Development: A 3D Multiscale Agent-Based Model

SPEAKERS Pawan Kumar (Université Paris-Saclay, Inria, Centre Inria de Saclay, 91120, Palaiseau, France)
AUTHORS Pawan Kumar (Université Paris-Saclay, Inria, Centre Inria de Saclay, 91120, Palaiseau, France); Fabien Crauste (Université Paris Cité, CNRS, MAP5, F-75006 Paris, France); Frédérique Clément (Université Paris-Saclay, Inria, Centre Inria de Saclay, 91120, Palaiseau, France); Chloe Audebert (Sorbonne Université, CNRS, Laboratoire de Biologie Computationnelle, Quantitative et Synthétique, CQSB, Paris, France)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Female mammals are born with a non-renewable pool of oocytes that underlies the ovarian reserve. The size of the oocyte pool results in great part from a selection process occurring during ovarian development within germ cysts, where clusters of germ cells are surrounded by layers of somatic cells. During selection, oocytes increase in size at the expense of non-selected cells, which are committed to death. We present a multiscale model of oocyte selection, coupling spatial cell organisation with stochastic gene-regulatory dynamics. Implemented in Simuscale, a modular multiscale framework, our 3D agent-based model represents anatomically-realistic germ cysts. Cell fate is driven by a minimal gene-regulatory network accounting for germ-germ and germ-somatic cell interactions. We investigate how cyst geometry and spatial organisation influence oocyte selection. Model parameters are calibrated to meet quantitative specifications on the selection rate. We assess the statistical reliability of simulation outcomes by estimating the minimum number of stochastic runs required for stable and reproducible results. The model generates 3D spatiotemporal distributions of germ-cell fate and monitors the stepwise commitment of germ cells into either selected oocytes or dying non-selected cells, giving insight into how a selected subset contributes to establishing the ovarian reserve.

CT1366 — Multi-scale Modelling of Astrocyte Metabolism in Pathological Morphologies

SPEAKERS Ingrid A. Lizcano-Prada (Department of Engineering, University of Luxembourg, 6 avenue de la Fonte, L-4364 Esch-sur Alzette, Luxembourg)
AUTHORS Alexander Skupin (Luxembourg Centre for Systems Biomedicine, University of Luxembourg, 6 avenue du Swing, Belvaux, L-4367, Luxembourg); Ingrid A. Lizcano-Prada (Department of Engineering, University of Luxembourg, 6 avenue de la Fonte, L-4364 Esch-sur Alzette, Luxembourg); Stéphane P.A Bordas (Department of Engineering, University of Luxembourg, 6 avenue de la Fonte, L-4364 Esch-sur Alzette, Luxembourg)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Astrocytes play an important role in the regulation and support of neural functions in the central nervous system. Neurodegenerative diseases (ND), have been linked to the development of pathological astrocytic phenotypes that result in changes in cellular morphology and mitochondrial dysfunction [Ram+21; Sok+24]. Calcium (Ca2+) dysregulation influence key metabolic pathways that can lead to ND [Voo+24]. While recent studies indicate that neuronal loss is triggered by impaired energy metabolism of astrocytes, the underlying mechanisms are still not fully understood. To address this gap, we have developed a mechanistic multi-scale model that describes the dynamical crosstalk between Ca2+ signaling and mitochondrial activity in pathological morphological of astrocytes in ND condition. Spatially resolved astrocyte metabolism is described by a reaction-diffusion system and solved by Finite Element Methods. Our results show a difference in metabolic energy profiles between morphologies of healthy and diseases astrocytes. Healthy cells produce enough ATP to sustain long diffusion of metabolites and variation in lactate concentration may potentially influence the Astrocyte-Neuron Lactate Shuttle. Numerical solutions also corroborated previous studies were variations in chemical reaction distribution in the cell can influence the overall metabolite concentration [Far+23]. This demonstrates how mechanistic modelling can reveal underlying metabolic mechanisms of ND.

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CT1518 — The Race Between Entropy and Maximum Entropy

SPEAKERS Jiří Nedomlel (Charles University)
AUTHORS Jiří Nedomlel (Charles University); Petr Tureček (Charles University)
SCHEDULE Tuesday, 14 July 2026; 18:00–18:20; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Traditional thermodynamic views describe organisms as dissipative systems maintaining low internal entropy by exporting disorder, yet this fails to explain how complexity and information increase under the second law. We propose that growth and evolution arise from regulating the rate of exploration—the import of matter and energy from the environment. When exploration adapts to environmental conditions, maximum entropy expands faster than actual entropy, increasing a system's thermodynamic information upper bound. The proposed model describes an open non-equilibrium system with exploration rate α and dissipation rate β and defines environmental information ε as available structure. The rate of thermodynamic information change follows $\dot{I} = \alpha N\varepsilon - \beta I$. Simulations show that thermodynamic information grows only when exploration is adapted to environmental information (ε). High exploration rates in structured environments increase complexity, while the same in information-poor environments causes informational decay. Evolution thus emerges as adaptive entropy management—life grows by regulating exploration according to its environment.

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CT266 — Accelerating Parameter Estimation in Large Systems Biology Models

SPEAKERS Andrea Polo Rodríguez (MBG-CSIC)
AUTHORS Andrea Polo Rodríguez (MBG-CSIC); David R. Penas (MBG-CSIC); Julio R. Banga (MBG-CSIC)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mechanistic models based on ordinary differential equations typically involve many parameters that must be estimated from limited experimental data. This process is hindered by multimodal objective landscapes, expensive model simulations, sparse and noisy measurements, plus structural and/or practical identifiability issues, all of which make calibration computationally demanding.

Here, we propose a strategy to reduce the computational burden of model calibration by focusing only on the most relevant parameters. Instead of estimating all parameters simultaneously, we define a reduced calibration problem in which non-influential (non-identifiable) parameters are excluded prior to optimization. In contrast to traditional sensitivity-based approaches relying on local analyses, our method uses global sampling and a decision tree–based strategy to identify and discard parameters that cannot be reliably inferred from the available data, thereby simplifying the estimation problem while preserving model fidelity.

We evaluate the methodology on a collection of representative ODE-based systems biology models. The results show that the proposed approach achieves statistical fits comparable to full-scale calibration while substantially reducing computation time. Overall, the framework provides a practical and promising route to accelerate parameter estimation in complex mechanistic models.

CT1390 — Biophysical Modeling of Molecular Glues: Mechanistic Insights for Design

SPEAKERS Seokjoo Chae (Soongsil University)
AUTHORS Jon DeBonis (Rice University); Oleg Igoshin (Rice University); Seokjoo Chae (Soongsil University)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 11.03 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Molecular glues are small molecules that enable targeting of previously “undruggable” proteins by promoting interactions between a protein of interest (POI) and an effector protein, leading either to stabilization or degradation of the target. Despite increasing interest in these systems, the kinetic mechanisms governing their performance remain poorly understood.

We develop a mathematical framework describing the dynamics of molecular glue–mediated ternary complex formation and downstream effects. Model analysis reveals key determinants that shape system behavior and highlights how kinetic parameters and component abundances influence activity. The framework further provides quantitative insight into potential design principles for optimizing molecular glue strategies.

CT17 — Blocking of Bistable Front Propagation: Application to Wolbachia Population Replacement

SPEAKERS Saoussen Latrach (Université Paris Dauphine-PSL)
AUTHORS Hatem Zaag (Université Sorbonne Paris Nord); Nicolas Vauchelet (Université Sorbonne Paris Nord); Saoussen Latrach (Université Paris Dauphine-PSL)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.12 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

To control mosquito-borne diseases such as dengue, for which no effective vaccine is available, several strategies focus on targeting mosquito populations. One such approach involves introducing Wolbachia,

a symbiotic bacterium that blocks pathogen transmission, into the mosquito population. A mathematical model describing the introduction of Wolbachia and the subsequent population replacement has demonstrated that, in a homogeneous environment, spatial propagation occurs.

The objective of this work is to prove the blocking of this propagation in a heterogeneous environment. To achieve this goal, we divide this part into two parts:

The first part focuses on the reduction of a 2×2 reaction-diffusion system to a single closed equation, providing a simplified framework for studying the dynamics of Wolbachia propagation.

The second part investigates the study of wave blocking in a population replacement technique, where we examine scenarios in which environmental heterogeneity, particularly spatial variations in carrying capacity, may prevent the spread of Wolbachia. We identify the conditions that lead to blockage and validate our results using numerical simulations.

CT1255 — Coarse-grained PDEs for polar auxin transport in plants.

SPEAKERS Alexis Farman (UCL)
AUTHORS Alexis Farman (UCL)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Auxin is a hormone that plays key regulatory roles in plant development. Its diverse functions are enabled by its directional (polar) transport through cells and the feedback between this transport and the intracellular localisation of auxin transporters (PINs). These mechanisms are traditionally modelled using discrete compartmental ODEs, where each compartment represents a single cell.

Here we develop coarse-grained continuum models of polar auxin transport that are better suited to larger biological domains and enable analytical progress. By seeking an asymptotic expansion, we derive a single partial differential equation (PDE) that governs long-time auxin dynamics. We use this reduction to characterise the parameter regimes and feedback structures under which the continuum model reproduces qualitative behaviours observed in cell-based discrete models. Specifically, we investigate the emergence of patterned auxin peaks generated by up-the-gradient PIN polarisation and explore canal-like patterns arising from with-the-flux feedback mechanisms. In doing so we highlight the conditional validity of a simple quasi-steady state assumption.

Finally, we use the continuum framework to identify which mechanisms persist under coarse-graining and which are inherently dependent on the cellular scale, providing a robust comparison between cell-level and tissue-level transport dynamics.

CT1365 — Enhancing Patient Privacy and Model Interpretability with Tensor Trains

SPEAKERS Juliette Sinnott (University of Waterloo)
AUTHORS Jose Ramon Pareja Monturiol (Universidad Complutense de Madrid); Juliette Sinnott (University of Waterloo); Roger Melko (Perimeter Institute for Theoretical Physics); Mohammad Kohandel (University of Waterloo, Canada)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.06 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

When applying Machine Learning (ML) to medical problems, privacy and interpretability are of utmost concern. The inner workings of the model need to be well understood to strengthen user trust, and any patient data used in training should be fully anonymized. These two values can work in opposition to each

other, as transparent models like Logistic Regression (LR) can unintentionally leak information about who was included in the training procedure. We propose the application of Tensor Trains (TTs),which were originally designed for use in quantum physics, to remedy this issue [Sho+16]. Any ML model, including Neural Networks (NNs), can be decomposed into TT format; effectively obscuring training information while maintaining predictive performance and interpretability. We demonstrate this on published LR and NN models designed to predict immunotherapy responses [Cha+24]. We show how employing TTs on these models decreases the accuracy of Membership Inference Attacks [PPP25]. Furthermore, we demonstrate how to extract biological insight from these more private models, including computing feature importance, examining the monotonicity of predictions, and even recovering LR coefficients. These insights are not immediately available in most models, suggesting that TTs have significant interpretability and privacy benefits.

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CT337 — Extending Time to First Subsequent Therapy in the Adjuvant Treatment of High-Grade Serous Ovarian Cancer

SPEAKERS Cristina Koprinski (The University of Sydney, School of Mathematics and Statistics)
AUTHORS Cristina Koprinski (The University of Sydney, School of Mathematics and Statistics); Kirstie McLoughlin (The Daffodil Centre, The University of Sydney, A Joint Venture with Cancer Council New South Wales, Sydney, Australia); Melissa Merritt (The Daffodil Centre, The University of Sydney, A Joint Venture with Cancer Council New South Wales, Sydney, Australia); Peter S. Kim (University of Sydney, School of Mathematics and Statistics)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 02.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

High-Grade Serous Ovarian Cancer (HGSOC) is the deadliest gynaecological cancer and the fourth leading cause of cancer deaths in women. Most patients experience treatment failure and recurrence, primarily driven by resistance to chemotherapy, contributing to a 5-year survival rate of 45%. Existing literature lacks immunological models of HGSOC resistance and developing them could provide critical insights into mechanisms of resistance and interactions within the tumour microenvironment. We address this by constructing a mechanistic immunobiological model of HGSOC using ordinary differential equations that couples the synergistic cytotoxicity of paclitaxel and carboplatin to specific phases of the cell cycle. We then optimise chemotherapy regimens to maximise the time to first subsequent therapy (TFST) across three distinct patient profiles with varying resistance dynamics, while maintaining comparable toxicity to the standard regimen. The results indicate that optimised weekly and 21-day cycles can extend TFST and transition the patient's treatment dynamic toward a multiple response state, where acquired resistance is managed through low-dose, high-frequency chemotherapy. This model provides a robust foundation for

optimising personalised treatment strategies, while offering new insight into the immunological dynamics underpinning chemotherapy resistance in HGSOC.

CT382 — From intracellular oscillations to complex behaviour in the unicellular slime mould Physarum polycephalum

SPEAKERS Linnéa Gyllingberg (University of California, Los Angeles)
AUTHORS Abid Haque; Jason Graham (University of Scranton); Linnéa Gyllingberg (University of California, Los Angeles); Simon Garnier (New Jersey Institute of Technology)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 05.12 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Across living systems, oscillations support coordination, information flow, and decision making, from neural rhythms to calcium signaling in single cells. While most studies focus on organisms with nervous systems, growing interest concerns how similar abilities arise in non-neural organisms. The unicellular slime mold Physarum polycephalum is a key example, exhibiting decision-like behaviors including maze solving, network formation, and exploration–exploitation trade-offs. However, existing models describe either large-scale tube adaptation or local mechanochemical oscillations and therefore do not explain how intracellular oscillations generate whole-cell behavior.

We present a mechanistic model linking intracellular oscillations to behavior by coupling a self-sustained calcium oscillator to active pressure, fluid flow, and morphology. In one spatial dimension, reaction–diffusion dynamics drive pressure and tube radius changes, reproducing contraction waves and stimulus-induced symmetry breaking. We extend the model to two spatial dimensions using a phase-field formulation in which calcium-regulated tension drives cell deformation and migration.

The model reproduces key cell-level behaviors including exploration–exploitation trade-offs and efficient transport network formation. Our results show nonlinear feedback between intracellular oscillations and mechanical deformation generates complex behavior in a unicellular organism without neurons or centralized control.

CT1572 — How does cross-resistance for disease strains influence coexistence outcomes?

SPEAKERS Daniel Maes (University of Utah)
AUTHORS Daniel Maes (University of Utah); Fred Adler (University of Utah)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.21 - SZ
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Recovery from acute infectious diseases can establish a host's cross-resistance to other infectious diseases or other strains of the same disease. Unlike many other ecological contexts, diseases such as rhinovirus can offer very high diversity (of strains) in the same local community over long periods of time. In addition to high diversity, rhinovirus has complex patterns of temporary cross-resistance. We have developed a set of mathematical models to analyze how these patterns of cross-resistance can enhance or diminish ecological diversity over time, with a focus on comparing the differing impacts that resistance between strains and resistance from the same strain have on coexistence outcomes. Using a mix of analytical and numerical techniques to determine cross-resistance impacts on single- and multi-strain coexistence outcomes, we show that intra-strain cross-immunity has a strong negative impact on multi-strain coexistence. Inter-strain cross-immunity generates a much smaller, but positive impact on coexistence, but primarily when it impacts strains with higher infectiousness more than those with lower infectiousness. These idiosyncratic

patterns of cross-resistance thus create predictable patterns of coexistence among strains that differ across multiple trait axes and shape the structure of infectious disease communities.

CT368 — How much should we trust non-identifiable models? Reliable model selection in face of parameter non-identifiability

SPEAKERS Elijah Foo (University of Melbourne)
AUTHORS Elijah Foo (University of Melbourne); Torkel Loman (University of Oxford); Alexander Browning (University of Melbourne); Ivo Siekmann; Ruth Baker (University of Oxford); Jennifer Flegg
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mathematical models are invaluable for understanding and predicting how biological systems behave. Constructing such models requires specifying the mechanisms and relationships involved, which in practice are not perfectly known. In the presence of multiple competing models, principled approaches to modeling need to account for model uncertainty. Bayesian model averaging (BMA) provides a framework for testing mechanistic hypotheses and generating predictions under model uncertainty. BMA requires the calculation of model evidence, which may be obtained via deterministic approximations or estimated using Monte Carlo methods. On the other hand, there is a growing recognition that parameter non-identifiability—the inability to distinguish between parameter values given available data—is pervasive in mathematical biology and has important consequences for statistical inference. In this work, we investigate the reliability of evidence computation methods when parameter non-identifiability is present, and find that deterministic approximations can produce misleading model selection results, as their underlying assumptions are violated. We propose an efficient variant of adaptive multiple importance sampling for evidence estimation, and demonstrate its robustness against non-identifiability. We use ecological case studies to show that our methods yield BMA results that are comparable to those obtained by Markov chain Monte Carlo methods at substantially lower computational cost.

CT1584 — Inferring rooted phylogenies from distances

SPEAKERS Klaus Schliep (IMBT, TU Graz)
AUTHORS Klaus Schliep (IMBT, TU Graz)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.02 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Since Zuckerkandl and Pauling proposed the molecular clock in the 1960s it became common to trace mutations in genomes to infer their evolutionary history [ZP65]. UPGMA, also known as average linkage, is a hierarchical clustering algorithm, which was one of the first used to infer ultrametric phylogenetic trees [SM58]. Trees can inferred efficiently in $O(n^2)$ time, where n is the number of tips [Mur85]. This allows to estimate large trees and made the method also popular for clustering. Here we present a tree arrangement algorithm to improve UPGMA trees under the implicitly used minimum evolution criterion. Our algorithm allows to compute all $2(n-1)$ Nearest Neighbor Interchange (NNI) moves in $O(n^2)$ time. We further extend our algorithm to allow the estimation of tip-dated phylogenies assuming a strict molecular clock. An implementation of the algorithms will be part of the R package phangorn [Sch11].

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CT1540 — Joint Transmission-Mutation Modelling Shapes the Long-Term Dynamics of Evolving Pathogens

SPEAKERS Marvin Schulte (Fraunhofer Institute for Industrial Mathematics ITWM; RPTU University of Kaiserslautern-Landau)
AUTHORS Jan Mohring (Fraunhofer Institute for Industrial Mathematics ITWM); Marvin Schulte (Fraunhofer Institute for Industrial Mathematics ITWM; RPTU University of Kaiserslautern-Landau); René Pinnau (RPTU University of Kaiserslautern-Landau)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 10.11 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Emerging variants are a key driver of long-term dynamics in pathogens such as SARS-CoV-2 and influenza, yet most transmission models do not consider the dynamic nature of the mutation process. We introduce a unified framework that couples multi-variant transmission with an explicit mutation process. Transmission is described by a generic compartmental model accounting for multiple variants with partial cross-immunity and seasonality. Variant emergence is represented by an inhomogeneous Poisson process whose intensity is proportional to the population-level infectious fraction. The resulting system is a stochastic hybrid model of variable dimension, as each mutation event extends the state space. Using deterministic approximations and stochastic simulations, we characterize how dynamically arising variants shift the system away from the classical endemic equilibrium and generate sustained multi-wave dynamics. We identify parameter regimes in which an intermediate mean waiting time between variant emergences maximizes the number of infections. Finally, we illustrate how the joint treatment of transmission and mutation affects the evaluation of vaccination strategies and discuss implications for the design of robust control policies against rapidly evolving pathogens.

CT1492 — Mathematical insights into the dynamics of prostate cancer under phytocannabinoid therapy

SPEAKERS Marianna Cerasuolo (University of Sussex)
AUTHORS Alessia Ligresti (ICB-CNR); Marianna Cerasuolo (University of Sussex); Max Jensen (UCL); Roberto Ronca (University of Brescia)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 01.22 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Mathematical models of prostate cancer progression and treatment response often rely on deterministic dynamics, yet in vivo behaviour reflects stochastic variation across scales. Randomness may arise from clonal heterogeneity, phenotype switching, metabolic plasticity, tumour micro-environment structure, and fluctuating host–microbiome interactions. These factors shape treatment response and resistance, supporting models in which noise is treated as a structural component rather than a perturbation.

This talk examines how stochasticity can be incorporated into dynamical systems for prostate cancer under phytocannabinoid-based therapy [Mah+23], aiming to identify which sources of variability best

explain experimental observations. Stochastic and hybrid models [Bur+23] are introduced to represent tumour–microbiome–therapy interactions, informed by experimental data from phytocannabinoid treatments and accounting for various responses under distinct dietary and metabolic contexts.

Numerical simulations allow comparison between intrinsic cellular variability, environmental fluctuations and therapy-related effects. The aim is to assess how different sources of stochasticity influence simulated treatment response and resistance under mono- and combination phytocannabinoid therapies. By isolating and quantifying distinct noise mechanisms, the framework aids interpretation of heterogeneous experimental outcomes and supports data-informed stochastic models of refractory prostate cancer.

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CT10 — Mathematical modeling of cell proliferation in a scaffold with elastic branching channels

SPEAKERS Pejman Sanaei (Georgia State University)
AUTHORS Pejman Sanaei (Georgia State University)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.05 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Tissue engineering scaffolds consist of pores lined with cells through which a nutrient-filled fluid passes. Over time, cells consume the nutrients and proliferate, causing the pores to shrink until they fill with tissue. Existing literature has investigated the effects of nutrient flow rate, nutrient concentration, cell hunger rate, scaffold elasticity, and shear stress on cell proliferation within cylindrically shaped pores. In this work, we model tissue growth in scaffolds with elastic branching structures; which begins as a single pore that repeatedly bifurcates over the depth of the scaffold. Our objectives are the following: (i) develop a model of cell proliferation which includes nutrient flow dynamics and concentration, cell hunger, and scaffold elasticity; (ii) solve the model and then simulate the cell proliferation process; and (iii) optimize the initial configuration of the scaffold channels to maximize cell growth. We find that elasticity enhances overall tissue yields at the cost of time. Inelastic scaffolds yield tissue faster than elastic scaffolds, however their maximum yield is marginally lower. The results of this study are key to adapting the equations governing cell proliferation to more complex geometries, ones which can more accurately represent scaffolds used in experimental tissue engineering.

CT1301 — Mechanistic Modeling of Cell–Cell Contact Networks in Collective Cancer Invasion

SPEAKERS Edwin Weinholtz
AUTHORS Simon Syga (Technische Universität Dresden); Andreas Deutsch (Technische Universität Dresden); Peter Friedl (Radboud University Nijmegen Medical Centre); Stijn den Daas (Radboud University Nijmegen Medical Centre); Edwin Weinholtz
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 02.23 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Invasion into surrounding tissues is a hallmark of cancer [HW11]. In contrast to individually migrating cells, multicellular clusters exhibit enhanced resistance to radiotherapy and DNA damage [Hae+20]. In this work we seek to identify the mechanisms that generate the cell-cell contact patterns associated with collective therapy resistance using quantitative and mechanistic approaches.

We established a graph-based framework to quantify cell-cell contacts from confocal images of migrating cells in 3D collagen matrix stained for nuclei and actin. Cells are represented as nodes and inferred contacts as edges with weights derived from nuclear distance and contact length. Experimental datasets reveal condition-dependent shifts in neighbor number, clustering, and contact strength.

To uncover underlying mechanisms, we developed a Cellular Potts Model implemented in the simulation software Morpheus. The model incorporates cell–cell adhesion, active cellular motility, and interactions with the extracellular matrix. To enable direct comparison, we then analyzed simulation snapshots using the identical pipeline as for the experimental data.

Systematic variation of the model parameters shows that increased adhesion shifts network topology toward higher neighbor numbers and stronger connections, recapitulating experimental signatures. Hence we argue that our contact observables link contact-based phenotypes to specific biophysical drivers of collective resistance.

References

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CT1422 — Mechanistic modelling of cluster formation in metastatic melanoma

SPEAKERS Nathan Schofield (University of Oxford)
AUTHORS Nathan Schofield (University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Melanoma is a type of skin cancer that becomes much more lethal when it spreads or metastasises to other tissues. During tumour initiation, melanoma cells form clusters in the primary tumour that promote metastasis. In the absence of biological tools to visualise cluster formation at primary tumour sites we use in vitro data for two distinct melanoma cell phenotypes, one more proliferative and the other more invasive. Data is available for both monoculture experiments, which give rise to homogeneous clusters, and co-culture experiments, which result in heterogeneous clusters. We develop a suite of mathematical models to generate mechanistic insight into the cluster formation observed in the data. We use a coagulation-fragmentation-proliferation framework to describe cluster growth dynamics both in a deterministic mean-field ODE model and a stochastic agent-based model (ABM) to investigate motility-driven interactions. We quantify phenotypic differences by fitting the ODE models to experimental data using a Bayesian framework for parameter inference and information criteria to perform model selection. To investigate spatial and collision-driven effects that the ODE framework cannot represent, we analyse the ABM through global sensitivity analysis and derive analytical links between its parameters and the effective coagulation rate in the ODE model. These results provide biological insight into how motility, collisions and proliferation jointly drive cluster formation.

CT1297 — Model-informed optimisation of dosing strategies using mechanistic tumour growth modelling

SPEAKERS Esha Joshi (University of Surrey)
AUTHORS Anne Skeldon (University of Surrey); Carina Dunlop (University College London); Esha Joshi (University of Surrey); Gianne Derks (University of Leiden); James Yates (GSK)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Quantitative systems pharmacology (QSP) models are increasingly used to inform dose scheduling in oncology. Standard tumour growth inhibition models typically combine empirical growth laws with drug action functions. Recent work suggests that non-uniform dosing regimens may outperform uniform dosing regimens.

Here we present a systematic evaluation of how modelling choices on growth law and kill function influence optimal dosing strategies. The results reveal that predicted optimal dosing strategies depend strongly on the underlying model assumptions and parameter selection. When the drug action is nonlinear, the preferred dosing regimen depends on the mean drug concentration.

To evaluate these predictions using experimental data, we analysed tumour growth trajectories from patient-derived xenograft (PDX) models used in pre-clinical cytotoxic chemotherapy studies [Gao+15]. This data spans a wide range of tumour growth kinetics, allowing us to estimate parameter regimes representing fast and slow growing tumours. Incorporating these empirically derived growth dynamics into the modelling framework reveals the sensitivity of the optimal dosing strategy to the growth rate of the tumour. Fast-growing tumours favour different dosing regimens than slow growing tumours based on the underlying tumour growth model. These findings highlight the importance of integrating experimental growth data when using mechanistic models to inform dosing strategies.

References

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CT123 — Modeling the impact of host diversity on the evolution of vector feeding preferences and implications for disease control

SPEAKERS Shravani Shetgaonkar (Birla Institute of Technology and Science, Pilani, K K Birla Goa Campus, Zuarinagar, Sancoale, Goa 403726, India)
AUTHORS Anupama Sharma (Birla Institute of Technology and Science, Pilani, K K Birla Goa Campus, Zuarinagar, Sancoale, Goa 403726, India); Shravani Shetgaonkar (Birla Institute of Technology and Science, Pilani, K K Birla Goa Campus, Zuarinagar, Sancoale, Goa 403726, India)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 03.01 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Vector-borne diseases often infect multiple host species, increasing the likelihood of disease persistence due to the presence of multiple reservoirs. Vector biting patterns and feeding preferences can shift in response to selective pressures introduced by disease control interventions, altering the dynamics of transmission. In this talk, we develop a mathematical model that couples host diversity and adaptive vector behavior with

vector-borne disease transmission dynamics, focusing on a system with two distinct hosts and a vector population exhibiting preference for one host. We derive the basic reproduction number, R_0, a threshold which determines the existence of two equilibria in our model, and obtain conditions that can lead to the long-term persistence of the disease. The analysis suggests that shortening the infectious period of the vector's preferred host is an effective control strategy. We also identify a threshold that determines whether shifting vector preference toward a non-preferred host amplifies or reduces the disease burden on the primary preferred host. We show that protective measures for the preferred host can trigger adaptive shifts in vector preferences, reducing disease prevalence in that host. However, this shift *may* lead to an increase in overall host prevalence.

CT1418 — Modeling water-related dynamics in subaerial biofilms interacting with porous stone substrates

SPEAKERS Fabiana Russo (University of Naples Federico II)
AUTHORS Fabiana Russo (University of Naples Federico II)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.46 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Subaerial biofilms (SABs) are microbial communities colonizing air-exposed mineral surfaces, where water availability is crucial for their metabolic activity. Located at the interface between the mineral substrate and the air atmosphere, SABs interact directly with both environments. Although precipitation and dew contribute to SAB hydration, water availability is largely controlled by evaporation and condensation closely linked to the fluctuating temperature and humidity conditions. As a result, variations in external conditions affect the interaction of SABs with the air and the mineral substrate, and thereby its internal organization. A mathematical model describing the water dynamics in the SAB, the ambient air and the underlying stone is proposed. Based on thermodynamic principles, the model captures changes in water content by accounting for the evaporation and condensation through gradients in water chemical potential. It considers the SAB capacity to delay water loss, while enabling the biofilm to draw water from stone pores to compensate for atmospheric evaporation. The key components of the model are validated by using experimental data reported in literature on water transport in limestone samples with and without SAB. Additional numerical simulations are performed under variable environmental conditions featuring daily fluctuations. Several scenarios are explored by considering different permeability conditions of the stone and the presence or absence of the SAB.

CT1239 — Path-Independent Transformation of Antagonistic Interactions under Minimal Evolutionary Conditions

SPEAKERS Hanna Isaksson
AUTHORS Hanna Isaksson; Vaibhav Sharma (Indian Institute of Science); Michael Sieber (Max Planck Institute for Evolutionary Biology); Arne Traulsen (Max Planck Institute for Evolutionary Biology); Samay Pande (Indian Institute of Science)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 11.32 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Species interactions take many forms, including antagonism and mutualism, yet the evolutionary conditions under which interactions change character remain poorly understood. Here we investigate how antagonistic interactions can evolve into mutualistic ones using a coevolutionary modeling framework inspired by experimental evolution, where a species interaction transitioned from predator–prey to cross-feeding. Using adaptive dynamics and invasion fitness analysis, we study trait evolution in both interacting species.

Mutations can occur in different orders, generating alternative evolutionary paths. Within this framework, we identify the minimal conditions under which antagonism can transition to mutualism. We further ask (1) which evolutionary paths are most likely and (2) which paths most often lead to mutualism. Our analysis shows that some evolutionary paths are more common than others, but the order of mutations does not affect the probability that mutualism evolves, conditional on completing the full trajectory. This path independence indicates that interaction type depends only on the final combination of traits. These results suggest that antagonistic interactions can provide evolutionary starting points for diverse mutualisms, including symbioses and cross-feeding systems.

CT1404 — Prey-predator-parasite models with infection-dependent attack rates

SPEAKERS Frank Hilker (Osnabrück University)
AUTHORS Ezio Venturino (University of Turin); Frank Hilker (Osnabrück University)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

We consider an eco-epidemiological model, where an infectious disease circulates within a predator population. If the disease only alters the attack rate of predators (a so-called trait-mediate effect), this reduces the equilibrium predator population size – no matter whether the disease increases or decreases the attack rate. By contrast, the prey population size at equilibrium can increase (for an enhanced attack rate) or decrease (for a reduced attack rate). Furthermore, if predators and prey cannot coexist in the absence of trait-mediated effects, disease-induced increases in the attack rate can enable the survival of predators. Mathematically, this occurs via transcritical bifurcations (for frequency-dependent disease transmission) or saddle-node bifurcations (for density-dependent disease transmission). The results underline the importance of trait-mediated effects and provide fundamental insights from generic eco-epidemiological models.

CT9 — Qualitative Analysis of a Caputo Fractional-Order System Arising from Within-Host Virus Dynamics

SPEAKERS ShyanShiou chen (Department of Mathematics, National Taiwan Normal University)
AUTHORS ShyanShiou chen (Department of Mathematics, National Taiwan Normal University)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 01.15 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

We investigate a class of autonomous fractional-order nonlinear dynamical systems motivated by an in-host HIV infection model incorporating three structural mechanisms: logistic proliferation of healthy target cells, two transmission pathways–virus-to-cell infection and cell-to-cell transmission through virological synapses [Kai10; LZ14; CCT16; CCR19]–and Caputo fractional-order derivatives accounting for memory effects. A threshold parameter, formulated as the basic reproduction number R_0, is derived and shown to govern the global dynamics. Specifically, the infection-free equilibrium is globally asymptotically stable when $R_0 < 1$, whereas uniform persistence is established when $R_0 > 1$. The local stability of the endemic equilibrium is further examined, and parameter regimes associated with a transition from steady-state convergence to oscillatory dynamics are identified. Due to intrinsic properties of autonomous fractional-order systems, exact periodic solutions and classical Hopf bifurcations are not expected. Instead, the observed oscillatory behavior corresponds to Hopf-type instability manifested through long-lasting transient or quasi-periodic dynamics. Although the fractional order does not modify the threshold structure determined by R_0, it significantly influences convergence rates and qualitative asymptotic

behavior, highlighting fundamental dynamical distinctions between integer-order and fractional-order systems.

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CT256 — Resource Limitations Induce Phase Transitions in Optimal Estimation Strategies

SPEAKERS Takehiro Tottori (RIKEN Center for Brain Science)
AUTHORS Takehiro Tottori (RIKEN Center for Brain Science); Tetsuya J. Kobayashi (The University of Tokyo)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 11.01 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

While biological systems estimate and adapt to fluctuating environments by integrating sensory evidence with internal memory, their information processing is constrained by limited resources. Such limitations introduce intrinsic noise and computational costs, which can fundamentally reshape optimal estimation strategies. Indeed, recent experiments indicate that humans rely on simpler memoryless estimation strategies rather than Bayesian memory-based estimation strategies under some conditions [Tav+22]. However, a theoretical framework for characterizing optimal estimation strategies under resource limitations remains lacking. In this presentation, we propose an optimal estimation framework under resource limitations based on optimal control theory [TK25c]. Applying this framework to a minimal model of biological information processing, we find that resource limitations induce phase transitions in optimal estimation strategies, which exhibit discontinuous, nonmonotonic, and scaling behaviors [TK25a; TK25b]. Finally, we show that the resource-induced phase transitions predicted by our theoretical framework qualitatively agree with recent human behavioral experiments. These results provide a theoretical foundation for understanding how resource limitations shape information processing in biological systems.

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CT1396 — Simulating how turnover influences actomyosin contraction and bundle formation

SPEAKERS Alex Tam (Adelaide University)
AUTHORS Alex Tam (Adelaide University); Alex Mogilner (New York University); Dietmar Oelz (The University of Queensland)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 10.01 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Interactions between actin filaments and myosin molecular motors generate the forces required for muscle contraction, cell division, and cell movement. The mechanism of contraction is well understood in muscle cells, where actin and myosin adopt a regular sarcomeric structure. In contrast, understanding contractility in the disordered networks of the cell cortex remains an open problem. Non-muscle actomyosin networks can also contain contractile filament bundles, known as stress fibres.

We use a 2D agent-based model to investigate how 4 simplified models for filament turnover (uniform, biased, branching, and treadmilling) influence actomyosin contraction and bundle formation [TMO26]. Without turnover, there is negative feedback between bundle formation and contractility. Uniform turnover and branching disrupt bundle formation allowing contraction to persist, whereas treadmilling disrupts the trade-off between bundle formation and contractility. Conversely, biased turnover increases bundle formation and reduces contractility. Our results suggest that cells could tune contractility and bundle formation by varying actin turnover pathways.

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CT54 — Targeting metabolic fluxes to prevent drug tolerance in Mycobacterium Tuberculosis under host-derived stress

SPEAKERS Tanishk Patodi (Indian Institute of Science, Bengaluru, India)
AUTHORS Tanishk Patodi (Indian Institute of Science, Bengaluru, India); Garhima Arora (Translational Health Science and Technology Institute, Faridabad, India); Samrat Chatterjee (Translational Health Science and Technology Institute, Faridabad, India); Amit Singh (Indian Institute of Science, Bengaluru, India); Mohit Kumar Jolly (Indian Institute of Science, Bengaluru, India)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 11.02 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Tuberculosis remains a major cause of death among infectious diseases despite the availability of multiple antibiotics against the causative agent Mycobacterium Tuberculosis (Mtb). Heterogeneity in Mtb population upon host infection prevents clearance of Mtb under antibiotic exposure. Mtb is known to differ in its redox environment, and the subpopulation with a reduced (oxidised) cytoplasmic environment is more tolerant (sensitive) to drugs. Handling such heterogeneity necessitates a protracted treatment and increases the chances of relapse. Cues from macrophages are known to influence the emergence of this population heterogeneity; however, the mechanisms underlying this process remain unclear. In this study, we employed a genome-scale metabolic model of Mtb, `iEK1011_2.0`, to identify how individual bacilli reroute their metabolic pathways crucial for Mtb to become drug-tolerant under host-derived stress. We derived context-specific models for phenotype-specific sub-populations (i.e., tolerant and sensitive populations) using RNA-seq data and employed gene knockout strategies to target the metabolic changes responsible for the phenotypic transitions. Our systems-level analysis reveals metabolic changes that

contribute to the spontaneous emergence of phenotypic heterogeneity in Mtb within infected macrophages and serves as a platform to identify novel potent interventional strategies.

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CT376 — The evolutionary rarity of bacterial endosymbiosis

SPEAKERS Eric Libby (Umeå University)
AUTHORS Eric Libby (Umeå University)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 11.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Endosymbioses have significantly changed the course of life on Earth, by giving rise to energy-producing organelles such as mitochondria and chloroplasts. While common in eukaryotes, endosymbiosis is extremely rare in bacteria, with only a handful of known instances. This rarity is shocking given the tremendous diversity and abundance of bacteria. While many factors may contribute to their rarity, we lack quantitative methods for estimating the extent to which these factors constrain the emergence of bacterial endosymbiosis. Here we assess several proposed constraints using a combination of mathematical approaches, including dynamical systems, metabolic models, and allometric scaling relationships. In the absence of direct empirical evidence, these models provide null predictions for the expected frequency of bacterial endosymbioses. We show that the initial formation of endosymbioses is not limited by internal spatial constraints within bacteria, ecological interactions between species, or the coupling of host–endosymbiont metabolisms. However, we identify potential conflicts involving growth-rate synchronization and evolvability that can destabilize early endosymbioses and lead to their extinction. Together, these results demonstrate how mathematical modeling can help identify the dominant constraints on the emergence of bacterial endosymbiosis.

CT195 — Understanding microbiome assembly and stability through a simplified model

SPEAKERS Andreas Eilersen (ETH Zürich)
AUTHORS Andreas Eilersen (ETH Zürich); Kim Sneppen (University of Copenhagen); Sebastian Bonhoeffer (ETH Zürich)
SCHEDULE Wednesday, 15 July 2026; 08:30–08:50; 12.01 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Microbiomes, their composition and stability have been a topic of great interest in recent years as they turn out to influence human health much more than previously thought. However, the mechanisms determining microbiome health remain unclear. As microbiomes are complex, consisting of many species, attacking this problem through modelling is a challenge [Gil+18; Kum+19]. Several unknown population dynamical parameters must be determined for every species, making accurate modelling a daunting task. We attempt to get around this issue by proposing a highly simplified model of microbiome assembly and stability. Instead of determining the full population dynamics using a typical, Lotka-Volterra-based modelling framework, we propose looking at species existence or absence as a binary variable. The interaction

parameters between species we likewise model as binary relationships: bacterial species may produce or require a given nutrient, and they may each be in a commensal or antagonistic relationship with every other species. Pairs of antagonists are entirely unable to coexist. Despite these (over-)simplified assembly and coexistence rules, the model communities exhibit rich dynamics including multistability and mass-extinction events. We derive conditions for the establishment of a healthy, diverse microbiome and for its collapse. It is our hope that this model will present a new and useful approach to understanding how microbial communities assemble, remain stable, or break down.

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CT210 — A Bayesian phylogenetic approach using fluctuating methylation to resolve inter- and intra-crypt stem cell dynamics

SPEAKERS Calum Gabbutt (Imperial College London)
AUTHORS Pablo Bousquets-Muñoz (Institute of Cancer Research); Heather Grant (Institute of Cancer Research); Darryl Shibata (University of Southern California); Trevor Graham (Institute of Cancer Research); Carlo Maley (Arizona State University); Calum Gabbutt (Imperial College London); Diego Mallo (Arizona State University)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 12.01 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Human intestinal epithelium is organised into test-tube-like structures called "crypts" or "glands". These crypts are maintained by a small pool of stem cells that undergo a process of selectively-neutral replacement, leading each crypt to inevitably drift towards monoclonality [Lop+10]. We previously discovered fluctuating CpGs (fCpGs), endogenous CpG sites that undergo stochastic changes in methylation state and therefore record lineage [Gab+22]. We used these fCpGs to infer the intra-crypt stem cell dynamics of individual human crypts.

Here, we built upon our previous work adapting phylogenetics to somatic evolution [Mar+18] to develop PHYFUM: a Bayesian phylogenetic approach to jointly infer the intra-crypt replacement dynamics and the inter-crypt fission lineage relationships using fCpGs. We validated PHYFUM using simulations, confirming that it improves recovery of the correct topology and branch lengths than our previously published discretised binary approach [Gab+25]. In patient data, we consistently observed that the last universal common ancestor (LUCA) in intestinal samples occurred in early life, in contrast to endometrial samples where the LUCA occurred notably later. We implemented different models of crypt fission, and found strong evidence against the hypothesis that crypts fission via budding of a single stem cell.

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CT188 — A mathematical model studying the interplay between malaria dynamics and economic growth

SPEAKERS Ruijun Zhao (Minnesota State University, Mankato)
AUTHORS Calistus Ngonghala (University of Florida); Hope Enright; Olivia Feldman (University of Tennessee at Knoxville); Ruijun Zhao (Minnesota State University, Mankato)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 03.01 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Malaria imposes significant challenges on human health, healthcare systems, and economic growth/productivity in many countries. In this talk, we will propose a model to understand the interplay between malaria dynamics, economic growth, and transient events. The reproduction number (R_0) is calculated. The model exhibits a backward bifurcation. Additionally, there is a parameter regime for which long transients are feasible. The model reveals a reciprocal relationship between malaria and economic factors, where malaria diminishes economic productivity, while higher economic output is associated with reduced malaria prevalence. The study offers insights into malaria control and underscores the significance of optimizing external aid allocation, especially favoring an even distribution strategy, with the most significant reduction observed in an equal monthly distribution strategy compared to longer distribution intervals. Policy recommendations for effective malaria control from the study include prioritizing sustained control measures, optimizing external aid allocation, and reducing mosquito biting.

CT1479 — A multi-phase field computational framework to model endothelial cell rearrangement and blood flow-induced polarization

SPEAKERS Marcos Gouveia (University of A Coruña)
AUTHORS Marcos Gouveia (University of A Coruña); Matilde Palmeira (Center for Physics of the University of Coimbra (CFisUC)); Eduardo Ferreira (University of Coimbra); Tiago Gonçalves Pereira (University of Coimbra); Rui Travasso (Center for Physics of the University of Coimbra)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.05 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Endothelial cells forming blood vessels display complex collective behavior. During vascular network rearrangement and regression, new capillaries are formed while others regress through cellular rearrangements without relying on cell death or proliferation [Fra+15]. Furthermore, endothelial cells in arterioles and

venules are polarized in the opposite direction of blood flow, promoting cell movement [Bar+22]. However, the biophysical mechanisms driving these rearrangements remain poorly understood.

To investigate these mechanisms, we develop a multi-phase field model in which each cell, the extracellular matrix (ECM), and the vessel lumen are individually modeled using a set of order parameters ϕ_i. A free energy functional $F[\phi_i]$ describes the relevant interactions, including cell–cell and cell–ECM adhesion [Mel+23; Non12]. The time evolution of each order parameter is governed by the Allen–Cahn equation and solved using semi-implicit spectral methods.

Simulation results show that blood flow-induced polarization is essential for obtaining vessel structures with realistic endothelial cell shapes and that lumen stability can be maintained over a range of cell–ECM adhesion values. By incorporating Delta–Notch dynamics, the model reproduces stalk–tip cell patterning in growing sprouts.

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CT1573 — A Spatiotemporal Computational Model of Ultrasound-Triggered Nanoparticle Drug Delivery in Solid Tumors

SPEAKERS Farshad Moradi Kashkooli (University of Waterloo)
AUTHORS Farshad Moradi Kashkooli (University of Waterloo)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 02.23 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Focused ultrasound (FUS) combined with thermosensitive liposomes (TSLs) offers a strategy for localized drug delivery in solid tumors. To investigate the mechanisms governing this therapy, we develop a spatiotemporal multiphysics model of ultrasound-triggered nano-drug delivery in tumor tissue. The framework consists of a coupled system of partial differential equations describing acoustic propagation, bioheat transfer, interstitial fluid flow, drug transport, and pharmacodynamic tumor response. Ultrasound propagation is modeled using the Helmholtz equation, while convection–diffusion equations describe drug transport and temperature-dependent release from liposomes. The equations are solved using finite element methods to quantify temperature fields, drug release dynamics, and intracellular drug accumulation. Simulations are used to examine how ultrasound exposure time and liposomal release kinetics influence spatiotemporal drug distribution in tumors. Parametric analysis of three release regimes (ultrafast, fast, slow) and three exposure durations (10, 30, 60 min) shows that a 30 min exposure combined with rapid

drug release maximizes therapeutic response by increasing drug accumulation within tumor tissue while limiting delivery to healthy regions. These results demonstrate how coupled acoustic, thermal, and transport processes regulate ultrasound-mediated drug delivery and highlight the value of multiphysics modeling for optimizing nanomedicine-based cancer therapies.

CT1611 — A Sufficient Condition for Stable Coexistence Under a Generalist Predator

SPEAKERS Lawrence Seminario-Romero (Haverford College)
AUTHORS Richard Rebarber (University of Nebraska-Lincoln); Brigitte Tenhumberg (University of Nebraska-Lincoln); Lawrence Seminario-Romero (Haverford College)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.21 - SZ
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

The functional response describes how a predator's consumption rate varies with prey density. There are three types of Holling functional responses: Type I (linear), Type II (hyperbolic), and Type III (sigmoidal). Although many predator-prey models adopt a Type II response for analytical convenience, this choice is more appropriate for specialist predators. In contrast, generalist predators are better described by a Type III functional response. In this talk, we analyze a predator-prey model incorporating a Type III functional response, and establish a sufficient condition for the stable coexistence of both species.

CT271 — Addressing Calibration Challenges in Generalized Lotka–Volterra Models of Microbial Ecosystems

SPEAKERS Ana Paredes-Vázquez (MBG - CSIC)
AUTHORS Ana Paredes-Vázquez (MBG - CSIC); Eva Balsa-Canto (IIM - CSIC); Julio R. Banga (MBG - CSIC)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Inferring nonlinear interaction structures in complex microbial communities remains a central challenge in systems biology. Generalized Lotka-Volterra (GLV) models are a foundational framework for modeling interacting dynamical systems and are widely used in ecology. However, parameter estimation in GLV models is often hindered by the exploration of numerically unstable regions of parameter space, where certain parameter combinations generate rapidly diverging trajectories. This leads to numerical instability during integration, deteriorating the performance of optimization algorithms, particularly when broad parameter bounds are considered.

We propose a robust parameter estimation strategy that combines biologically informed initial bounds with an iterative re-optimization scheme. By restricting early searches to dynamically stable regions, the method avoids premature exploration of divergent trajectories.The feasible domain is then progressively adapted through successive re-optimization steps, enabling the search to gradually move toward regions providing high-quality fits.

We validate the approach on several case studies of increasing complexity. Notably, our strategy enables reliable calibration of a 12-species GLV model of the human gut microbiome, which was previously very challenging due to blow-up instabilities. We also illustrate the interplay between experimental design, practical identifiability and mechanistic insights from the inferred networks.

CT1266 — Allocation dynamics of stage-specific interventions in structured populations

SPEAKERS Daniel Strömbom (Department of Biology, Lafayette College)
AUTHORS Daniel Strömbom (Department of Biology, Lafayette College)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 11.32 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Many ecological management problems involve allocating limited resources across the life stages of populations in order to influence population growth. Such problems arise in both invasive species control and conservation of threatened species, yet these objectives are often studied within separate modeling frameworks. We consider a general stage-structured formulation for univoltine populations in which management interventions modify stage-specific demographic rates. The resulting growth function provides a common representation of both suppression strategies, which reduce demographic rates, and enhancement strategies, which increase them. Analysis of this framework highlights differences in how interventions applied to different life stages interact within the growth process. These interaction patterns influence how marginal intervention effects change as effort accumulates and therefore affect prioritization and switching behavior when allocating management effort, particularly when interventions exhibit diminishing returns. The framework provides a general basis for studying stage-specific intervention strategies in both population suppression and conservation contexts.

CT109 — Balancing Stability and Complexity in Boolean Models of Biological Systems

SPEAKERS Venkata Sai Narayana Bavisetty (University of California Los Angeles)
AUTHORS Claus Kadelka (Iowa State University); Matthew Wheeler (University of Florida); Reinhard Laubenbacher (University of Florida); Venkata Sai Narayana Bavisetty (University of California Los Angeles)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 11.02 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Boolean networks, first introduced by Kauffman as models for gene regulatory networks, have gained widespread popularity for their ability to capture complex biological behaviors through simple logical rules. A systematic investigation of these biological models suggests that they are incredibly robust. In particular, they are resilient to perturbations and tend to reach the same phenotype despite small disturbance. An explanation of this phenomenon was first given by Kauffman, who showed empirically that a network's connectivity determines the stability of the Boolean network. This was further expanded on by Derrida, who provided a theoretical explanation for the effect of connectivity. This was succeeded by many empirical studies that explored the relationship between a network's structural parameters and its stability.

Building upon this foundation, our work delves into the intrinsic trade-off between phenotypic complexity and network stability. We extend a conjecture proposed by Willadsen, Triesch, and Wiles, proving that entropy—acting as a proxy for complexity—provides a tight asymptotic upper bound for coherence, our measure of stability. By deriving this Pareto frontier between complexity and stability, we show that natural gene regulatory networks are not merely stable—they are highly optimized, consistently residing on or near this theoretical limit.

CT225 — Catch-bond dynamics of cell-cell adhesion in the Cellular Potts model predicts dynamics in cell clustering, sorting and invasion

SPEAKERS Lisanne Rens (TU Delft)

AUTHORS Lisanne Rens (TU Delft)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 05.12 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Cell–cell adhesion is crucial for tissue organization and collective migration. Cells also adhere to the extracellular matrix (ECM) that surrounds them. Both cell-cell [Man+14] and cell-ECM adhesions [Kon+09] can be catch-bonds, which strengthen under increasing force. Although recent mathematical studies [RM20] have explored how models of catch bonds in cell–ECM interactions [NS13] influence cell dynamics, the effects of catch-bond behavior of cell–cell adhesions remains understudied. Here, I extend the Cellular Potts Model [GG92] with catch-bond dynamics for cell–cell and cell–ECM adhesions. I report three key findings:

1) The model predicts the relative adhesion binding rates required for cells to cluster rather than disperse. 2) Decreasing either contractility or adhesion binding rates can reverse engulfment patterns of two cell types. 3) In a specific parameter regime, the model generates short chain-like invasive protrusions into the ECM, indicative of enhanced invasive potential arising purely from catch-bond mechanics.

In summary, incorporating catch–slip bond dynamics into a Cellular Potts Model reveals complex relationships between adhesion, cell forces, and collective cell behaviors. This framework offers mechanistic explanations for phenomena such as unexpected cell-sorting outcomes that cannot be resolved by classical differential adhesion models.

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CT1551 — Co-modelling epidemics and specific TTI behaviours

SPEAKERS Thomas House (University of Manchester)
AUTHORS Thomas House (University of Manchester)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 10.11 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Since its inception as a discipline, infectious disease modelling has also been aware of the importance of and considered the spreading of behaviours relevant to infection. While there have been many epidemiological modelling studies integrating behaviour in general, it is less common to consider specific behaviours. Here, I will present work that models the specific behaviours around TTI – Test, Trace and Isolate. This specificity makes the mechanism by which changes in infection prevalence can influence awareness more apparent, since this happens via case reporting. And it also makes the mechanism by which behaviour change following increased awareness can influence disease transmission more apparent, since this happens via reduction of contacts.

Here, I will present work with several different collaborations on this problem, including: (i) explicit analysis of a dynamical systems model for coupled epidemic and TTI behaviour spreading [Rya+25]; (ii) network analysis methods for understanding the impact of mixing as a control parameter and associated health-economic trade-offs [TOH25]; and (iii) data-driven non-parametric methods that enable learning from time series of teasting [Lyt+23].

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CT237 — Deciphering Neutrophil Complexity

SPEAKERS Gabriel Calvo (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain)
AUTHORS Gabriel Calvo (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 01.15 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Neutrophils are the most abundant circulating leukocytes and first responders to infection and tissue injury. Once viewed as short-lived and homogeneous, they are now recognised as a dynamically reconfigurable immune compartment with connected phenotypic and functional states in homeostasis and disease [Ng+25]. In this talk, I will present various modelling and high-dimensional data-driven approaches that we have developed at the Mathematical Oncology Laboratory in close collaboration with other biomedical institutions [Cra+22; Maa+23; Ng+24; Cer+26], to decode neutrophil behaviour across sterile inflammation, infection and cancer. Data-driven state-space 'behavioural landscapes' from intravital microscopy reveal stereotyped migratory states and pathogenic vascular-associated programmes that can be pharmacologically tuned [Cra+22]. Age-structured PDE models linked to fate-mapping quantify tissue-dependent neutrophil lifetimes and predict prolonged persistence within tumours [Maa+23]. Single-cell trajectories support deterministic reprogramming of tumour-infiltrating neutrophils towards pro-angiogenic, immunosuppressive niches [Ng+24]. Finally, the transcriptomic compartment architecture (NeuMap) integrates multi-tissue single-cell atlases with dynamical modelling to map perturbation-specific trajectories and nominate regulators of effector programmes [Cer+26]. Together, these approaches provide mathematical tools to study neutrophil plasticity and a framework to rationally target neutrophil states in infection and disease.

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CT321 — Derivation and Analysis of a Low-Dimensional Hopf Oscillator Model for Macroscale Brain Dynamics

SPEAKERS Alexander Edthofer (TU Wien)
AUTHORS Alexander Edthofer (TU Wien); Andreas Körner (TU Wien)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 11.01 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Tracking changes in functional connectivity (FC) between brain regions is essential for understanding transitions between states of consciousness such as wakefulness, sleep, and anesthesia. Whole-brain models provide a framework to investigate these transitions and have predominantly been developed using functional magnetic resonance imaging (fMRI), see [PD24]. However, fMRI is costly and of limited clinical applicability, motivating the development of mathematically rigorous models based on electroencephalography (EEG), which is routinely available during surgical procedures and shows correlation with the fMRI signal, as shown in [Ful+19]. Using amplitude envelope correlation as a time-domain measure compatible with fMRI-derived FC, we identify EEG-based functional clusters based on [Roj+18] as done in our previous work [EK25]. Analysis of simultaneous EEG-fMRI sleep recordings reveals a three-cluster structure, yielding a low-dimensional stochastic differential equation model. Based on this reduction, we determine the parameters of a Hopf whole-brain model derived from the fMRI-based formulation, as motivated by [Lóp+21], and investigate its dynamical properties. In particular, we analyze bifurcation structures and transitions between dynamical regimes associated with altered states of consciousness.

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CT1380 — Divergent mechanics of inner ear morphogenesis are coupled to developmental tempo over vertebrate evolution

SPEAKERS Shuhei Horiguchi (Kanazawa University)
AUTHORS Satoru Okuda (Kanazawa University); Shuhei Horiguchi (Kanazawa University); Shunya Kuroda (Kanazawa University)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 10.01 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

The vertebrate inner ear is a conserved sensory organ for balance. Its morphogenesis follows a two-phase program: (i) expansion of the otic vesicle—a closed epithelial monolayer enclosing a fluid-filled lumen—and (ii) a topological transition from a closed vesicle to the toroidal structure of the semicircular canals. Although this two-phase process is broadly conserved, previous studies [Mos+19; Mun+21] have reported divergence in molecular mechanisms and tissue mechanics between zebrafish and mouse during phase (ii). This raises two questions: how do distinct mechanisms yield a common morphology, and how did such divergence evolve? To address these questions, we focus on phase (i), the otic vesicle expansion phase in zebrafish and mouse, combining pharmacological perturbations with mathematical modeling. We find that zebrafish vesicles expand primarily through fluid influx into the lumen, whereas mouse vesicles expand through coupled fluid influx and mechanosensitive tissue growth. In zebrafish, limited tissue growth reflects restricted cell growth rather than reduced proliferation. Extending our analysis to 12 vertebrate species sampled broadly across the phylogeny, we identify two distinct vesicle-expansion modes and corresponding cell-growth programs, exemplified by zebrafish and mouse. We propose that divergence in these morphogenetic processes reflects the adaptive evolution of developmental duration, accompanied by shifts in cell-growth programs.

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CT1585 — Ecological and stochastic determinants of the growth and persistence of the oral pathogen Porphyromonas gingivalis

SPEAKERS Haralampos Hatzikirou (Khalifa University)
AUTHORS Momen Hussein (Department of Biomedical Engineering and Biotechnology, Khalifa University, Abu Dhabi, UAE); Arnab Barua (Department of Mathematics, Khalifa University); Mohammad Qasaimeh (Division of Engineering, New York University Abu Dhabi, Abu Dhabi, UAE); Matthew Smardz (UB Microbiome Center, University at Buffalo, Buffalo, NY, USA); Patricia I. Diaz (UB Microbiome Center, University at Buffalo, Buffalo, NY, USA); Haralampos Hatzikirou (Khalifa University)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.02 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Population density greatly affects how microbes survive and grow in an environment. *Porphyromonas gingivalis* (Pg), a key pathogen in periodontitis disease, shows a type of growth that depends on population density. This growth suggests that there is a minimum density of Pg needed for it to survive. Interestingly, Pg is often found at low density inside the gum environment but still it survives. To explore this paradox, we combine growth experiments with mathematical models. An Alle effect model reveals a quorum threshold, below which Pg populations decline. Conditioned medium from the early colonizer *Veillonella parvula* (Vp) lowers this threshold, showing that these two species help each other in the early colonization stages. Adding stochastic dynamics and Fokker–Planck analysis shows that environmental factors can allow Pg to survive even below the expected threshold. Furthermore, Co-culture experiments show consistent results where small density of Pg are either saved or go extinct while Vp reaches its growth limits. A two-species game theory framework illustrates that these results explains both facilitation and competition.

References

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CT370 — Exploring Cell-Free Systems Through Robust Sensitivity Analysis

SPEAKERS Emily Hunter (National Physical Laboratory)
AUTHORS Chris Barnes (National Physical Laboratory and University College London); Emily Hunter (National Physical Laboratory); Liam Wright (National Physical Laboratory); Spencer Thomas (National Physical Laboratory)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Recent advances in cell free transcription-translation (TXTL) systems have renewed interest in quantitative cell-free gene expression modelling. Ordinary Differential Equation (ODE) models can describe TXTL dynamics, enabling users to explore a design space difficult to probe experimentally. A common modelling limitation is the unknown effect of variability in inputs with respect to the desired output. To address this lack of trust in the modelling, robust sensitivity analysis was applied; the results of which are shown here. A mechanistic model of an E. coli-derived cell-free gene expression system is developed, extending prior formulations [MN19] to retain the complete reaction network while avoiding quasi-steady state assumptions for fast reversible reactions. The system is described by ODEs derived from the extended stoichiometric and kinetic structure of the reaction network. To assess parameter influence, Sobol sensitivity analysis was performed to produce first- and total-order Sobol indices with respective confidence intervals. These

indices identified which inputs would benefit from precise measurement to more efficiently determine key behaviours. One notable result was that model behaviour was highly sensitive to degradation and to the reversible kinetics of the promoter-sigma factor and promoter-RNA polymerase complexes. This work introduces a customisable framework for statistical analysis of ODE systems, enabling user-defined equations and parameter ranges.

References

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CT98 — Human papillomavirus driving cervical cancer: a mathematical model with persistent infection, cancer progression, and spontaneous remission.

SPEAKERS Rena Hayashi (Graduate School of Systems Life Sciences, Kyushu University)
AUTHORS Akane Hara (University of Toyama); Rena Hayashi (Graduate School of Systems Life Sciences, Kyushu University); Yoh Iwasa (Kyushu University)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Human papillomavirus (HPV) infection is the causal agent of cervical cancer. While most HPV infections are transient and cleared by immune responses, a small fraction establish persistent infection, enabling cancer development through a multistage process from precancerous lesions to invasive disease. A distinctive feature of cervical cancer is the high rate of spontaneous remission at multiple stages. We develop a stochastic mathematical model to analyze cervical cancer incidence and its age distribution, incorporating infection dynamics, immune control, progression, spontaneous remission, and age-dependent exposure risk. Model parameters are estimated by fitting age-specific incidence data using a Markov Chain Monte Carlo approach. Our results show that remission rates substantially exceed progression rates, explaining why most persistent infections and precancerous lesions do not progress to malignancy. The characteristic age distribution of cervical cancer, peaking in the late 30s to early 40s, is primarily driven by age-dependent HPV persistence. Small changes in persistence probability can therefore produce large differences in long-term cancer risk, highlighting the importance of targeting persistent infection in prevention strategies.

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CT1505 — Mathematical modeling of macrophage-precancerous epithelial cell interactions in the oral cavity

SPEAKERS Lia Sela (LJLL, Sorbonne Université)
AUTHORS Jean Clairambault (LJLL, Sorbonne Université); Jean-Philippe Foy (CRSA, Sorbonne Université); Emmanuel Trélat (LJLL, Sorbonne Université); Lia Sela (LJLL, Sorbonne Université)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 01.22 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Oral squamous cell carcinoma (OSCC) represents the vast majority of oral cancers and is associated with high mortality. Patients with Oral Potentially Malignant Disorders (OPMD) present a risk of developing OSCC, and identifying OPMD with a high risk of malignant transformation remains a critical clinical issue.

Macrophages constitute a major immune population infiltrating the tumor microenvironment during carcinogenesis. Previous studies have reported conflicting results on the impact of M2 macrophages enrichment in the risk of OPMD malignant transformation. Bouaoud, Foy et al. [Bou+21] found a positive association between M2 macrophages enrichment and oral cancer-free survival in OPMD.

In this work, we perform immune deconvolution and gene set enrichment analyses using OPMD gene expression data. Then we build a system of two phenotypically-structured partial differential equations describing two cell populations : epithelial cells and macrophages. Numerical results with various parameter configurations, together with data-fitted model outputs, provide biological insights with the perspective of gaining a better understanding of the drivers of OPMD malignant transformation.

References

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CT348 — Mathematical Models for Non-Monotone Dose Responses

SPEAKERS Peter Rashkov (Institute of mathematics and informatics - Sofia, Bulgaria)
AUTHORS Lukasz Skalniak (Faculty of Chemistry, Jagiellonian University, Krakow, Poland); Peter Rashkov (Institute of mathematics and informatics - Sofia, Bulgaria)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 02.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

A non-monotone dose response is a pharmacokinetic response characterised by stimulation at low dose and inhibition at high dose of the therapeutic compound. This paradoxical phenomenon is observed in many contexts from kinase to immune checkpoint inhibitors in cancer treatment. Two examples of models with this property are presented. The first is a kinetic model for the action of a kinase inhibitor in the MAPK signalling pathway and demonstrates that the causes could be purely biochemical. This model provides insight on why this non-monotone response resulting in a simple regulatory mechanism could be masked in biochemical assays. The second example concerns effector T cell reactivation *in vitro* under the action of therapeutic compounds targeting the PD-1/PD-L1 interaction. TCR-mediated activation diminishes in the presence of PD-1/PD-L1 interaction provided by co-cultured artificial antigen-presenting cells. Upon PD-L1 blockade with tested compounds at different dose concentrations, cellular activation is restored. The resulting dose response curve is non-monotone due to the existence of two separate and antagonizing effects: first, specific activation of T cells and second, unspecific toxicity that overlap for a certain range of the tested compound concentration. Parameters of the model are estimated from experimental data and used to provide the EC50 concentration and the maximal safe drug concentration corresponding to the maximum T cell activation.

CT1382 — Memory T Cells as Predictors of Progression-Free Survival in High-Grade Serous Ovarian Cancer: A Mathematical Modelling Approach

SPEAKERS Iman Al Buwaiqi (School of Mathematics and Statistics, University of Sydney, Australia)
AUTHORS Iman Al Buwaiqi (School of Mathematics and Statistics, University of Sydney, Australia); Kirstie McLoughlin (The Daffodil Centre, University of Sydney, Australia); Melissa Merritt (The Daffodil Centre, University of Sydney, Australia); Peter Kim (School of Mathematics and Statistics, University of Sydney, Australia)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.06 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

High-grade serous ovarian cancer (HGSC) almost always relapses after first-line treatment, yet how long patients remain progression-free varies enormously. One plausible explanation is that the immune state at the end of chemotherapy — in particular, how many memory T cells survive treatment — shapes the pace of tumour regrowth. Whether this differs between patients with and without homologous recombination deficiency (HRD), a DNA repair defect that increases sensitivity to platinum-based chemotherapy, remains open. We address this using an ODE model of tumour-immune dynamics in HGSC, built around two cancer cell populations: immune-recognisable cells that drive T cell responses, and immune-invisible cells that accumulate through irreversible escape mutations. A sensitivity analysis shows that T cell proliferative capacity — not per-cell killing rate — determines whether immune control is maintained. Adding platinum-based chemotherapy clears much of the tumour but leaves memory T cells relatively intact: through antigen-driven division, slower natural turnover, and lower chemotherapy sensitivity, memory cells outnumber effector T cells by over 600-fold at end of treatment. We then ask whether this memory reservoir delays relapse, and whether the effect differs in HRD-positive patients — providing a mechanistic rationale for memory T cell levels as a prognostic marker in HGSC.

CT110 — Metabolic games in an autotroph-heterotroph consortium: eco-evolutionary analysis of cooperative, private and cheater exoenzyme strategies

SPEAKERS Tanvir Hassan
AUTHORS Anna Matuszyńska; Tanvir Hassan
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.12 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Synthetic microbial consortia offer a controlled setting to study how ecological and evolutionary forces shape community structure. Here we present a mathematical framework for a cross-kingdom autotroph–heterotroph system in which a sucrose-secreting cyanobacterium feeds a heterotrophic community. We model three heterotrophic metabolic strategies- public metabolizing, private metabolizing and cheating and analyse how their metabolic interactions shape coexistence and competition. The framework combines generalized Lotka-Volterra dynamics, Michaelis-Menten kinetics and evolutionary game theory to represent nutrient exchange and frequency-dependent fitness. Laboratory-inspired eco-evolutionary simulations allow strategies to switch between generations, mimicking mutation or phenotypic plasticity. These dynamics reveal parameter regimes and thresholds separating collapse, dominance and coexistence, and show how growth advantages and switching rates drive shifts in community composition and robustness of this cross-kingdom consortium. Together, these results provide design rules for constructing stable synthetic communities under ecological and evolutionary constraints. To make the model accessible and reusable, we deploy it in mxlweb, a browser-based interface for ODE models, where users can vary parameters and initial conditions, run simulations and explore “what-if” scenarios without installing software, enabling rapid exploration and sharing of model-based insights.

CT1257 — On the Emergence of Traveling Wave Patterns in Resource-Mediated Tissue Competition

SPEAKERS Natalia Briñas-Pascual (Universidad Carlos III de Madrid)
AUTHORS Natalia Briñas-Pascual (Universidad Carlos III de Madrid)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

The understanding of tissue dynamics is critical across various biological contexts, including morphogenesis, regeneration, and tumor proliferation. While traditional models often focus on volume exclusion, this work investigates the emergence of spatial patterns driven by competition for a shared resource, such as oxygen [GG96]. We present a family of coarse-grained evolution models that incorporate phenotypic traits and cellular heterogeneity via an effective proliferation rate derived from cell-cycle variations. To facilitate theoretical analysis, we introduce a quasi-stationary approximation for the resource dynamics, which we show numerically to be highly accurate across a wide range of macroscopic parameters. In the case of a single population, the dynamics resemble those of the FKPP equation [KPP37; HR75]. For competitive systems involving two cellular lineages [VV94], we find that the fitter population, characterized by a higher proliferation rate for a given resource level, progressively takes over the spatial domain as an invading pattern, while the less fit population retreats. Our results indicate that collective invasion speed is primarily governed by the proliferation rate of the fittest population, suggesting a "winner-takes-all" dynamic in the long-term tissue organizationand providing a tool to study dominant phenotypic traits in heterogeneous tissues without necessitating underlying genetic variability.

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CT1445 — Parameter Identifiability Under Limited Experimental Data in Age-Structured Models of the Cell Cycle

SPEAKERS Ruby Nixson (University of Oxford)
AUTHORS Helen Byrne (University of Oxford); Joe Pitt-Francis (University of Oxford); Philip K. Maini; Ruby Nixson (University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

The mitotic cell cycle governs DNA replication and cell division. Efficacy of radiotherapy depends on cell-cycle position and so accurate mathematical models of the cell cycle are essential for understanding and predicting treatment response. Mathematical modellers often face a lack of available, sufficiently resolved data for parametrising models. We consider how the ability to collate summary data across the literature affects identifiability of parameters for a cell cycle model.

Initially synchronised cell populations desynchronise over successive cycles, converging to balanced exponential growth (BEG), characterised by exponential population growth and steady, time-independent phase proportions. These proportions can be obtained from flow cytometry data. The increasing use of the Fluorescent Ubiquitination-based Cell Cycle Indicator (FUCCI) provides higher-resolution information on phase dynamics, such as minimum phase durations and variability.

We present an age-structured PDE model in which cell-cycle phase progression follows a delayed gamma distribution. We derive analytical expressions for BEG phase proportions and other observable quantities, and use them to assess how data availability influences parameter identifiability. When parameters are not uniquely identifiable, we determine identifiable parameter groupings, thereby determining the minimum amount of data that must be available for successfully fitting structured population models of the cell cycle.

CT381 — Plant tolerance is explained by resource-based plant-nematode interactions

SPEAKERS Joseph Penlap (Université Côte d'Azur, Inria, INRAE, CNRS, MACBES, France)
AUTHORS Frédéric Grognard (Université Côte d'Azur, Inria, INRAE, CNRS, MACBES, France); Joseph Penlap (Université Côte d'Azur, Inria, INRAE, CNRS, MACBES, France); Suzanne Touzeau (Université Côte d'Azur, INRAE, ISA, France); Valentina Baldazzi (Université Côte d'Azur, INRAE, ISA, France)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 11.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Plant-parasitic nematodes cause major economic losses worldwide by infecting crops, diverting resources and impairing plant growth. While resistance aims to reduce parasite burden, tolerance allows plants to maintain yield despite infection, offering a complementary and potentially more sustainable strategy. Understanding how tolerance arises requires mechanistic insights into host-parasite interactions and their constraints.

We developed a mathematical model describing the coupled dynamics of plant growth, internal resource allocation and nematode population processes. The model incorporates key biological mechanisms, including resource-dependent plant development, nematode infection and reproduction, and feedbacks between resource limitation and parasitism. We analytically derived the basic reproduction number and identified equilibrium states: extinction, healthy plant and plant-nematode coexistence. We showed that coexistence strongly depends on plant resources, highlighting conditions under which tolerance can emerge. In addition, we numerically explored how variations in plant and nematode parameters affect tolerance over a cropping season. Our study highlighted the dual role of resource production: higher production increases plant growth but also promotes nematode proliferation. A compromise therefore emerges between promoting productive plants and limiting yield losses under infestation, shaped by nematode virulence and plant quantitative resistance.

CT1409 — The evolution of an evolvability-enhancing condition-dependent mutation rate

SPEAKERS Jana M. Riederer (University of Groningen)
AUTHORS Jana M. Riederer (University of Groningen); Timo J.B. van Eldijk; Franz J. Weissing; G. Sander van Doorn
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Mutation provides the raw material for evolution. Mutation rates thus tune evolvability, the ability to undergo adaptive evolution: if mutation rates are too low, evolution is impeded; if mutation rates are too high, adaptive traits cannot be maintained. Empirical studies have demonstrated that mutation rates

may change with individual condition, for instance in the case of stress-induced mutagenesis in bacteria, which is implicated in the evolution of antibiotic resistance. However, it is not clear if a stress-induced increase in mutation rate is an adaptive response which enhances evolvability by increasing the influx of mutations in times of stress, or if it is a (potentially mal-adaptive) byproduct of stress itself. Moreover, while empirical evidence of condition-dependent (i.e., plastic) mutation rates is accumulating, theoretical models studying their evolution are lacking. Here, we employ an individual-based simulation approach to examine the evolution of condition-dependent mutation rates in a changing environment. We show that condition-dependent mutation rates readily evolve, providing "well-timed" variation specifically when organisms are poorly adapted. Such plastic mutation rates facilitate better adaptation to changing environments, and their evolution provides an example of evolvability itself evolving.

CT1427 — Understanding quorum-sensing bacteria one model at a time

SPEAKERS Silas Poloni (Roskilde University)
AUTHORS Johnny Ottesen (Roskilde University); Morten Andersen (Roskilde University); Silas Poloni (Roskilde University)
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 15.46 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Quorum-sensing is a biochemical signaling mechanism used by different bacterial taxa to communicate with their co-specifics. This communication allows bacteria to jointly respond to environmental conditions by collectively expressing particular traits once a certain population density is achieved. Its complex structure and multiple bureaucratic layers of gene activation make it difficult to precisely assess how these responses are modulated. Furthermore, the impact of such responses on population dynamics are even more difficult to infer, because biochemical signals and population densities are intrinsically coupled. One such example is that of P. aeruginosa growing on casein media. In order to grow and reproduce, P. aeruginosa must break casein into amino-acids by collectively expressing enzymes such as elastase. In the hopes of understanding how quorum sensing mediated expression of elastase is triggered, as well as its impacts to population dynamics, I estimate parameters for increasingly complex models and matching experiments, one at a time.

CT1460 — What's in a modelling formalism? On the use of graphical models in systems biology

SPEAKERS Leo Diaz (The University of Melbourne)
AUTHORS Leo Diaz (The University of Melbourne); Michael Stumpf; Sean Vittadello
SCHEDULE Wednesday, 15 July 2026; 08:50–09:10; 11.03 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

An essential step of any modelling effort is to decide which parts of a system should be represented. This decision is implicitly influenced by the experimental questions we ask and hypotheses we make, which in turn determine which aspects of a system are relevant to include in a model. Less explored however is how the choice of modelling formalism itself influences what gets to be represented. Here I focus on reaction networks and Petri nets as two modelling formalisms commonly used across systems biology. Both are based on the framework of graphs and are used to model ecological and biochemical systems, sometimes interchangeably. While they are both able to represent the same processes, they do so differently. A precise comparison of how each formalism is defined at the mathematical level further reveals that reaction networks are in fact a class of the more general Petri nets, contrary to their usually claimed equivalence. Concretely, these differences illustrate different strategies in how to represent system properties, which

are built into the formalism definition. The higher generality of Petri nets therefore provides a practical case of how formalisms can limit how accurately a system can be represented. Overall this work suggests ways to define more precise and general formalisms, which I will demonstrate using the abstraction of hypergraphs.

CT1252 — A Hybrid Model of Tumor Evolution: The Impact of Oxygen on Glioblastoma Dynamics

SPEAKERS Alexandra Whiteside (Technische Universität Dresden)
AUTHORS Alexandra Whiteside (Technische Universität Dresden); Andreas Deutsch (Technische Universität Dresden); Simon Syga (Technische Universität Dresden)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 01.22 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Glioblastoma is an aggressive brain tumor characterized by infiltrative growth, frequent recurrence after treatment, and intratumoral and microenvironmental heterogeneity. A key driver of this heterogeneity is the tumor microenvironment, particularly oxygen gradients that arise as tumor expansion outpaces vascular development. These gradients can induce phenotypic plasticity in tumor cells, including the "go-or-grow" switch between migratory and proliferative phenotypes. Experimental studies link hypoxia to a more migratory phenotype, but the regulatory response to hypoxia and its implications are poorly understood. To explore how oxygen-dependent phenotypic switching influences glioblastoma growth and evolution, we develop a hybrid discrete-continuous mathematical model. The model extends a previous cellular automaton model by adding an oxygen-dependent phenotypic switch function and coupling partial differential equations governing oxygen dynamics. By exploring the parameter space governing the oxygen-dependent switch function, we investigate whether nontrivial regulatory regimes optimize tumor growth and promote spatial heterogeneity. Incorporating oxygen dynamics also enables a detailed description of radiotherapy, which is more effective in normoxic regions, allowing analysis of how oxygen-dependent phenotypic plasticity influences post-treatment tumor recurrence.

CT367 — A Spatially and Phenotypically Structured Model of Radiotherapy under Oxygen Heterogeneity

SPEAKERS Francesco Albanese (Politecnico di Torino)
AUTHORS Francesco Albanese (Politecnico di Torino); Giulia Chiari (University of Oxford / BCAM (Basque Center for Applied Mathematics)); Marcello Edoardo Delitala (Politecnico di Torino)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 02.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Tumor hypoxia drives radiotherapy failure, as reduced oxygenation lowers radiosensitivity and promotes resistant phenotypes. We propose a phenotype-structured partial differential equation model to study fractionation by varying dose–interval pairs under oxygen-limited conditions, extending [CFD23]. The framework couples tumor cell density and oxygen dynamics through a continuous trait encoding resistance to hypoxia and radiation.

Radiotherapy is described by a trait-dependent linear–quadratic model in which radiosensitivity is modulated by an oxygen enhancement ratio depending on the spatial oxygen field and evolving phenotypic distribution. Schedules are constrained by a normal-tissue biologically effective dose and evaluated by time to progression.

Exploration of the dose–interval space shows that oxygenation reshapes protocol ranking. Near normoxia, schedules yield comparable outcomes, whereas hypoxia induces distinct regimes. Severe hypoxia disrupts the classical dose–interval trade-off and may favor either hyperfractionated or hypofractionated strategies. Moreover, spatial heterogeneity in oxygen distribution, even at fixed total supply, alters protocol ranking relative to the spatially homogeneous case.

These results support spatially and phenotypically structured models for schedule selection in hypoxic tumors.

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CT395 — Applying Koopman operator theory to explore the dynamics of bioreactor models

SPEAKERS Cameron Jakub (University of Guelph)
AUTHORS Cameron Jakub (University of Guelph)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 11.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

A challenge in modelling biological phenomena is that the resulting models often involve many interacting variables that exhibit complex nonlinear dynamics, making them difficult to study using dynamical systems theory. The well-established theory behind the Koopman operator helps to address this challenge by representing complex nonlinear models as simpler, linear models evolving in an infinite-dimensional function space, where one can take advantage of the linearity to study the model in a new context. Many numerical methods have been developed that are able to uncover finite-dimensional approximations of the Koopman operator using observed or simulated data from dynamical systems. The eigenfunctions of the (approximated) Koopman operator can then be used to estimate information about the original system, including stable equilibria and their respective basins of attraction. In this presentation, we explore the utility of Koopman operator theory in a mathematical biology setting by applying these methods to recover the dynamics of well-studied chemostat models. We also present preliminary results in applying these methods to recover the dynamics of a more complex model of a gut bioreactor.

CT111 — Asthma-mediated control of optic glioma growth via T cell-microglia interactions: A mathematical model

SPEAKERS Donggu Lee (Konkuk University)
AUTHORS Donggu Lee (Konkuk University); Sean Lawler (Brown University); Yangjin Kim (Konkuk University)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Optic glioma, a slow-growing tumor, is associated with Neurofibromatosis type 1 (NF1) mutations and increased midkine (MDK) production. A connection between asthma and optic glioma has previously been observed, but the mechanisms are unclear. To elucidate the role of asthma in the regulation of glioma formation, we investigated the role of T cells and the subsequent pathways in the regulation of microglia, a key player in the glioma tumor microenvironment (TME). While asthma is often linked to

chronic inflammation, our mathematical analysis and experimental evidence suggest that inflammation can play a key role in suppressing the proliferation of optic glioma cells via immune reprogramming of T cells and the delicate control of signaling networks in microglia. Our mathematical model unveils the complex interactions between tumor and immune cells in optic glioma. Our results indicate that asthma-induced T cell reprogramming inhibits tumor growth by promoting the release of decorin and a subsequent suppression of CCR8 and the intercellular binding kinetics in microglia, followed by blocking of CCL5 production in TME via suppression of NFκB. We developed anti-cancer strategies by leveraging this asthma-induced immune regulation.

CT295 — Identifiability-aware Neural ODEs for parsimonious learning of dynamical systems

SPEAKERS Núria Campo-Manzanares (IIM-CSIC)
AUTHORS Núria Campo-Manzanares (IIM-CSIC); Eva Balsa-Canto (IIM-CSIC)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Hybrid dynamical models are increasingly used to describe complex biological systems. Neural differential equations, such as PINNs, UDEs and Neural ODEs, combine mechanistic structure with data-driven components to model complex dynamical systems. However, this flexibility often weakens practical identifiability, leading to overfitting, redundant neural components, poorly constrained parameters and limited extrapolation.

We introduce identifiability-aware Neural ODEs (iNODEs), a framework that uses practical identifiability as a guiding principle for hybrid model formulation and neural architecture selection. Neural components are embedded explicitly within the differential equations as parametric multilayer perceptrons, enabling closed-form sensitivities and systematic assessment of parameter uncertainty and redundancy. Data availability and identifiability metrics guide an automated architecture search that selects parsimonious models and retains only neural components supported by the available data.

We evaluate the framework on benchmark nonlinear systems such as Lotka-Volterra and SIR models, including cases with latent parameters and partial observability. iNODEs yield more stable parameter estimates, reduced uncertainty and improved generalization compared with standard approaches. Identifiability-guided design thus provides a robust route toward reliable, interpretable and parsimonious neural differential equation models.

CT1436 — Inference of Spatially Varying Tumor Hydraulic Conductivity and Its Impact on Drug Transport

SPEAKERS Mohsen Rezaeian (Department of Applied Mathematics, University of Waterloo, Canada)
AUTHORS Mohmmad Kohandel (Department of Applied Mathematics, University of Waterloo, Canada); Mohsen Rezaeian (Department of Applied Mathematics, University of Waterloo, Canada)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Hydraulic conductivity (K) is a key transport property governing interstitial fluid movement through the porous tumor microenvironment. Despite its importance, measurement of hydraulic conductivity relies on invasive and technically demanding experiments, mostly performed on non-human tumor tissues. Experimental studies also show substantial intra- and inter-tumoral heterogeneity in K, complicating measurement and highlighting the need for patient-specific characterization in drug delivery models. In

this work, we develop a mathematical framework to infer spatially varying K from drug concentration profiles. We first formulate a continuum model describing interstitial fluid flow and drug transport in an isolated tumor nodule representative of peritoneal carcinomatosis treated with intraperitoneal chemotherapy. Finite element simulations show that heterogeneity in hydraulic conductivity substantially alters interstitial fluid pressure and drug distribution. We then derive an explicit analytical inversion formula to reconstruct spatially varying hydraulic conductivity from steady-state concentration profiles. To improve robustness under noisy experimental data, we also implement a parametric inversion approach. The proposed framework provides a theoretical basis for the non-invasive estimation of K from particle distributions and suggests a pathway toward integrating imaging data with mathematical models for quantitative characterization of the tumor microenvironment.

CT1610 — Invasion, replacement and coexistence in ecological models

SPEAKERS Cameron Browne (University of Louisiana at Lafayette)
AUTHORS Cameron Browne (University of Louisiana at Lafayette)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.02 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

How invasions of ecosystems by novel species or variants unfold and their ripple effects on the community are central questions in ecology and evolution. In this work, we explore how an invader can replace a resident species in different ecological networks and/or models. Through the lens of modern coexistence theory and invasion rates, we analyze feasibility and stability of invaded multi-species equilibria, along with persistence of associated species. As invader growth rate increases, there is either generically a gradual transition from invasion to replacement or non-generically an abrupt bifurcation where invasion and replacement occur simultaneously. We find an analytical means for predicting which species are replaced in different scenarios, particularly in the non-generic case which includes specific cases of Lotka-Volterra, consumer-resource and predator-prey networks. In these examples, we also study how the presence and functional form of trade-offs can lead to distinct convergent ecosystem structures.

CT180 — Is (Microbial) Ecology Fair?

SPEAKERS Teemu Kuosmanen (University of Helsinki)
AUTHORS Juhani Rantanen (University of Turku); Dovydas Kičiatovas (University of Helsinki); Sanna Pausio (University of Turku); Ville-Petri Friman (University of Helsinki); Teppo Hiltunen (University of Turku); Ville Mustonen (University of Helsinki); Teemu Kuosmanen (University of Helsinki)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.12 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Understanding and predicting how communities assemble is a paramount challenge in ecology. Here we address these questions normatively by comparing the observed species abundance distribution to a game-theoretically fair distribution based on each species' Shapley value. By analyzing in total 56 distinct community outcomes, we assess how fairly biomass is distributed in microbial communities displaying both competitive and cooperative interactions in different growth conditions. We find examples of fair communities that closely follow their Shapley value across all environments as well as counterexamples where the true abundances deviate from the species' objective contribution to community biomass. Next, we develop a fair assembly rule based on the recursive definition of Shapley value that can predict also unfairly assembled community compositions. Our results give unique empirical insights into the distributive function of ecological dynamics and lay down the theoretical foundations of what might become a normative community assembly theory.

CT1278 — Metapopulation Dynamics with May–Leonard Competitive Lotka–Volterra systems

SPEAKERS Anju Susan Anish (Ghent University)
AUTHORS Anju Susan Anish (Ghent University); Bernard De Baets (Ghent University); Shodhan Rao (Ghent University)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 11.32 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

A metapopulation model describes local populations of interacting species distributed across discrete habitat patches connected through migration. In this work, the local dynamics within each patch are modeled by May–Leonard Competitive Lotka–Volterra (MLCLV) systems consisting of three species, whose pairwise competitive interactions are encoded by a circulant interaction matrix. Such systems model cyclic competition among three species, and the interaction coefficients describing the mutual inhibitory effects in pairwise competition determine the dynamics of these systems. Depending on the interaction coefficients, MLCLV systems may exhibit stable convergence to the coexistence equilibrium, stable periodic limit cycles, convergence to boundary equilibria, or heteroclinic cycles. We consider m patches, where each patch is inhabited by the same three species, and the local dynamics in each patch are governed by an MLCLV system exhibiting a stable periodic limit cycle. The objective of this work is to ensure asymptotic convergence of the trajectories to the coexistence equilibrium in each patch. We propose a scheme to design a metapopulation network that achieves this control objective across all patches via inter-patch species migration. Using Lyapunov methods coupled with LaSalle's invariance principle, we prove that the resulting metapopulation model admits a unique coexistence equilibrium that is globally asymptotically stable.

CT187 — Modelling and inference of wildfire spread dynamics in UK moorland environment.

SPEAKERS Axa-Maria Laaperi (Newcastle University)
AUTHORS Andrew Baggaley (Lancaster University); Axa-Maria Laaperi (Newcastle University); Laura Wadkin (Newcastle University); Nick Parker (Newcastle University)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.21 - SZ
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Wildfires disrupt ecosystems, with much evidence to show that climate change is exacerbating vulnerability in regions poorly adapted to such disturbances. These events are driven by complex, multi-scale interactions where small perturbations in environmental factors can trigger large-scale shifts, complicating prediction efforts. We propose a coupled convection-reaction-diffusion system for modelling wildfire spread dynamics. This system integrates spatial and temporal variability to identify thresholds for spread and identify impacts of abrupt environmental changes on burnt areas and rates of propagation. We incorporate environmental, meteorological, and historical fire data from the Global Wildfire Information System and project partner, the Department for Environment, Food and Rural Affairs (UK), with a specific focus on UK moorland fires. In applying Bayesian inference techniques and Monte Carlo methods for parameter estimation and uncertainty quantification, we aim to produce robust model validation against unseen data. Recent wildfire events around the globe highlight the need for insights into environmental vulnerability, property loss, and infrastructure risk. By enabling near-real-time simulations, this work aims to provide a computational tool for emergency response, long-term management strategies, and assessments of climate change-induced outlier weather patterns influencing fire behaviour.

CT208 — Modelling multiscale cell competitions and cooperations that determine longevity of homoeostasis

SPEAKERS Ijas Ahamed N (Indian Institute of Science)
AUTHORS Ijas Ahamed N (Indian Institute of Science); Mohit Kumar Jolly (Indian Institute of Science); Ramray Bhat (Indian Institute of Science)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.05 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Cell competition is a key mechanism by which tissues regulate cellular fitness and maintain homeostasis. In aging tissues, senescent cells show irreversibly arrested proliferation and yet persist and compete with proliferative non-senescent cells for space. How immune surveillance shapes this competition and regulates health and lifespan of such tissues remains poorly understood especially because of the multiscale nature of interactions. To address this, we developed a multiscale modelling framework to study cell competition between senescent and non-senescent epithelial cells under immune-mediated clearance. At the population level, we formulated a system of ordinary differential equations (ODEs) describing competitive interactions among proliferative cells, senescent cells, and immune effectors, incorporating growth, senescence induction, immune recruitment, and senolysis. Analysis reveals regimes of competitive exclusion, coexistence, and senescent dominance driven by nonlinear feedbacks. To capture the spatial aspects of such competitions, we constructed an agent-based Cellular Potts Model (CPM), in which cells compete locally for space and interact with immune cells. The CPM reveals spatial clustering of senescent cells and stochastic immune clearance absent in mean-field models. By comparing the ODE and CPM models, we aim to shed light on the role of spatial structure, crowding, and cell-niche adhesion, and softness, in immune-regulated competitions during tissue aging

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CT386 — Physiologically Informed Modeling of Aminoglycoside Exposure During Pediatric Maturation

SPEAKERS Felix J. Meigel (Renal Research Institute, LLC)
AUTHORS Felix J. Meigel (Renal Research Institute, LLC); Ana Catalina Alvarez-Elías (Fresenius Medical Care North America); Rasha Hussein (Fresenius Medical Care North America); Doris H. Fuertinger (Renal Research Institute, LLC)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Aminoglycoside antibiotics are widely used in pediatric care due to their strong activity against Gram-negative bacteria. Dosing remains challenging due to large inter-individual variability in exposure and risks of oto- and nephrotoxicity. As aminoglycosides are predominantly eliminated by renal filtration, developmental changes in body size and kidney function affect exposure. To quantify the impact of maturation on exposure variability, we built a physiology-informed two-compartment pharmacokinetic model integrating fragmented evidence from pediatric clinical studies. Model parameters, including their variability, were estimated from cross-study allometric regression. The model was applied to age-homogeneous virtual cohorts (N = 1000 per age) constructed from WHO growth data and age-stratified kidney function percentiles. Simulated target attainment reproduces patterns observed in clinical pediatric cohorts. Age stratification in the virtual cohorts shows that trough attainment increases by >65% between 1-month-old and 2-year-old children under the same weight-based dosing, indicating that mixed-age cohorts conflate maturational changes. Variance decomposition shows that trough variability is maturation-dominated, with kidney function explaining up to 73% of variance in infants and children. Peak variability is insensitive to kidney and body maturation (3–10%) and mostly driven by pharmacokinetic parameter variability.

CT1388 — Qualitative study of a tumor-immune interaction model for hepatocellular carcinoma under radiotherapy

SPEAKERS Cristina Vilar Medina (Universitat de València)
AUTHORS Antonio Baeza (Universitat de València); Cristina Vilar Medina (Universitat de València); Maria Carmen Martí (Universitat de València)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.06 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Tumor progression depends not only on the intrinsic characteristics of the cancerous cells but also on the bidirectional complex interactions within the tumor microenvironment. Among these interactions, lymphocytes, immune cells, play an important role [A S21].

In this work, we will generalize the mathematical model developed by Sung et al. in [Sun+20] for hepatocellular carcinoma to provide a more general framework applicable to a broad range of cancer types. This model describes the stimulating effect of malignant cells on the immune system as well as the immunosuppressive effects of radiotherapy. We will study the dynamics of the model, detailing the equilibrium points of the system and the conditions needed for them to be feasible. We will also present numerical simulations to validate the theoretical results and illustrate the behavior of the system under different radiotherapy fractionation schedules.

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CT1332 — Subclones, Drivers and Microenvironment in a new Genetic Model of Tumour Growth

SPEAKERS Vivien Holmes (University of Manchester)
AUTHORS Chay Paterson (University of Manchester); Vivien Holmes (University of Manchester)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 02.23 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Histologically homogenous tumours often hide distinct genetic subpopulations called subclones. These often come about due to mutations and structural variations of particular driver genes that seem to lead to a rapid acceleration of growth, but do not become fully clonal, perhaps not sufficiently fit to outcompete the rest of the population. The recent expansion of spatial transcriptomics has allowed for further study of these clonal dynamics.

We have developed a new model of the tumour with an invasive front that picks up driver mutations as it progresses through the surrounding tissue, generalising earlier geometric models. We can describe the process of invasion and growth using a simple variational principle, while the model itself is complex enough to describe cells growing at speeds that vary with their microenvironment and genetics. Using spatial transcriptomic data, we can also validate the model, analyse driver events, and quantify the relative speed of different driver events, allowing for insight into the in vitro behaviour of these drivers.

CT1394 — The role of space in the macroecological patterns of microbial abundances

SPEAKERS Adrián Gutiérrez Arroyo (Universidad Carlos III de Madrid)
AUTHORS Adrián Gutiérrez Arroyo (Universidad Carlos III de Madrid); Aniello Lampo (Departamento de Matemáticas, Universidad Carlos III de Madrid); José Antonio Cuesta Ruiz (Universidad Carlos III de Madrid)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.46 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Microbial communities display striking macroecological regularities in their temporal abundance fluctuations across very different biomes [Gri20]. In particular, previous studies reported that species abundances follow a gamma-like distribution, often interpreted as evidence of exogenous stochastic drivers such as environmental noise. In this work, we show [GLC26] that these patterns can also emerge from endogenous ecological dynamics. We study generalized Lotka–Volterra models with many interacting species, first including constant migration and then extending the framework to a spatial metacommunity with dispersal among patches. Our results show that deterministic species interactions alone can generate persistent, irregular fluctuations and reproduce the observed distribution of pairwise abundance correlations, a key empirical feature that stochastic models fail to capture. However, neither the open local model nor single patches of the metacommunity recover the empirical gamma abundance fluctuation distribution. Remarkably, this pattern appears only after aggregating abundances across spatial patches. This suggests that the gamma law may arise from spatial structure and regional-scale averaging rather than from a specific microscopic biological mechanism. Our findings highlight the central role of space in microbial ecology and suggest that macroecological patterns may reflect emergent effects of spatial aggregation as much as local community dynamics.

References

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CT1417 — Total biomass and dispersal: effects of local dynamics and network topology

SPEAKERS Juan Segura (EADA Business School)
AUTHORS Carolin Grumbach (Osnabrück University); Daniel Franco (National University of Distance Education); Femke Reurik (Osnabrück University); Frank Hilker (Osnabrück University); Juan Perán (National University of Distance Education); Juan Segura (EADA Business School); Marcos Marvá (University of Alicante)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Understanding how dispersal affects the total biomass of fragmented populations is a central problem in ecology. For the simplest metapopulation network, consisting of two patches, the continuous-time case with logistic local growth was completely described in [GL22]. In contrast, the analogous discrete-time problem has remained largely unresolved, with previous work describing only the low-dispersal regime.

For two-patch metapopulations, we first present a complete characterization of the biomass response to dispersal in a discrete-time Beverton-Holt model. We then show how the picture changes when local dynamics are modeled by other population maps, such as the Ricker map: in contrast with the Beverton-Holt case, qualitatively new response scenarios can arise, highlighting the role of the shape of local recruitment functions. Finally, we turn to multi-patch metapopulations and discuss how both patch number and network topology affect biomass responses. Exhaustive numerical analyses show that moving from two to three patches can substantially increase the range of possible scenarios, both in continuous and discrete time, while theoretical results show that adding connectivity does not always increase biomass at low dispersal rates, refuting previous claims in the literature.

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CT1491 — Venue access policies and intervention timing under vaccine acceptance heterogeneity

SPEAKERS Maikel Bosschaert (Julius Center for Health Sciences and Primary Care)
AUTHORS Maikel Bosschaert (Julius Center for Health Sciences and Primary Care); Rioshar Yarveisy (Delft University of Technology); Pieter van Gelder (Delft University of Technology); Martin Bootsma (Julius Center for Health Sciences and Primary Care)
SCHEDULE Wednesday, 15 July 2026; 09:10–09:30; 03.01 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

We develop a next-generation matrix (NGM) framework for evaluating venue access policies that restrict or test attendees based on vaccination status. As both vaccination coverage and contact structures are age-dependent, contact structures at venues will change due to access policies [Boo+24]. To isolate the effect of selectively excluding unvaccinated individuals from that of reduced attendance, we introduce an attendance reduction benchmark and show that the relative benefit of selective exclusion depends on vaccination coverage, venue structure, and behavioral responses.

Static NGM analysis based on reproduction numbers cannot address peak magnitude or cumulative burden. We therefore embed the NGM transmission structure in a dynamic age-structured SIR model with vaccination rollout and compare infections across policies and enforcement levels. Finally, we extend both frameworks to distinguish vaccine-hesitant and vaccine-accepting unvaccinated individuals with assortative mixing. At the static level, segregation can change the

relative ranking of policies. At the dynamic level, the standard collapsed model systematically underestimates post-release risk, predicting lockdown release weeks too early and leading to ICU occupancy substantially exceeding capacity thresholds. Although illustrated with COVID-19-inspired parameters, the methodology applies broadly to intervention timing under vaccination heterogeneity.

References

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AW-2024 — Impacts of genetic and phenotypic heterogeneity on tumor evolution

SPEAKERS Simon Syga (Technische Universität Dresden)
AUTHORS Simon Syga (Technische Universität Dresden)
SCHEDULE Wednesday, 15 July 2026; 09:40–10:10
SESSION Award Session

Abstract

This is the keynote presentation for the 2024 Reinhart Heinrich Doctoral Thesis Award.

Cancer is a complex ecosystem where genetic and phenotypic diversity drive tumor evolution, progression, and treatment resistance. Understanding how microscopic cellular processes, such as mutation, proliferation, and migration, shape macroscopic tumor dynamics remains a challenge. In this talk, I present a mathematical framework to study tumor heterogeneity [SND26]. By linking discrete stochastic models to population genetics and evolutionary game theory, I explore the role of the distribution of fitness effects (DFE) and the evolution of phenotypic plasticity in tumor growth. The findings highlight how spatial dynamics influence cancer evolution and how tumor heterogeneity can predict treatment outcomes [Syg+24]. This work provides new insights into cancer progression and potential strategies for therapeutic intervention.

References

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AW-2025 — Modelling and quantifying interactions in collective cell behaviour

SPEAKERS Carles Falco (University of Oxford)
AUTHORS Carles Falco (University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 10:10–10:40
SESSION Award Session

Abstract

This is the keynote presentation for the 2025 Reinhart Heinrich Doctoral Thesis Award.

Collective cell behaviours such as cell invasion and pattern formation emerge from interactions between individual cells, yet understanding how these local mechanisms scale to tissue-level dynamics remains a central challenge. In this talk, I will focus on two aspects of my thesis addressing how cell–cell interactions regulate proliferation and motility. First, I will discuss a continuum modelling and Bayesian inference framework for epithelial tissue expansion, showing how crowding constraints delay cell-cycle progression and generate spatial patterns of proliferation in growing tissues. Second, I will describe how adhesive interactions between cells can drive self-organisation, using a local continuum model derived from nonlocal adhesion principles to capture classical sorting behaviours such as segregation and engulfment.

CT1642 — A structured systems dissection of immune response dynamics in Drosophila

SPEAKERS Manmath Panigrahy (Imperial College London)
AUTHORS Ashima Wadhawan (Imperial College London); J Krishnan (Imperial College London); Manmath Panigrahy (Imperial College London); Marc S. Dionne (IMPERIAL COLLEGE LONDON)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 03.01 - SR
SESSION Contributed Talks
TRACK Miscellaneous

Abstract

The innate immune system represents the first line of defence against pathogens. In organisms such as Drosophila it represents the only line of defence. Understanding the organization and working of this innate immune system and the way it counteracts a variety of pathogens can provide a basis for a deeper understanding of the immune system function and associated design principles.[BSC14; Ryc+25] We develop a systems framework aimed at a structured system dissection of the innate immune response in Drosophila and its response to pathogens. As part of this, we dissect (i)complexities associated with the IMD pathway including the presence of both positive and negative feedback loops (ii)complexities associated with the pathogen including the presence of multiple sub-populations of pathogens as well as the impact of dead pathogens (iii)the interaction of the immune system network and pathogen populations. Our model analysis reveals the role of different feedback regulatory pathways in the IMD pathways and the role of macrophages on one hand, and the impact of resistant subpopulations of pathogens and dead pathogens on the other. This provides a range of insights into the organization and functioning of the innate immune network from a systems perspective, a basis for investigating the response of the innate immune response to a variety of pathogens, and a basis for understanding the impact of rewiring immune system networks in synthetic biology.

References

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CT1502 — A three process approach for insomnia-like awakenings

SPEAKERS Joe Rowland Adams (Aston University)
AUTHORS Joe Rowland Adams (Aston University); Maia Angelova (Aston University)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Insomnia is one of the most prevalent sleep disorders, associated with increased risk of conditions ranging from diabetes to heart disease [Bro+23]. Yet, much about its physiological causes and processes is still far from understood.

Mathematical modelling has had an important role to play in understanding the process of sleep itself. Some of the most important of these contributions have been the two-process model, composing sleep as interacting circadian and homeostatic drives [Bor82], the flip-flop model, inspired by neurological processes to model a 'natural' switch between the sleeping and waking state [SCS01], and physiology-inspired network models, incorporating REM and non-REM sleep states through physically realistic networks [FPR08].

In this talk our new three-process model for insomnia-like awakenings will be presented, introducing a new wakefulness drive to the two-process model of sleep. This will include sensitivity analysis of the original model and an exploration of its dynamical regimes, and the new insights these provide into the mechanisms by which insomnia can arise. These results will be further contextualised with dynamical analysis of key existing sleep models, demonstrating the need for this new approach to incorporate insomnia.

Finally, a new framework for mathematically representing insomnia based on this model will be discussed, including its implications for further modelling developments to understand more insomnia features.

References

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CT270 — Agent-Based Model for Superficial Spreading Melanoma Reveals the Role of Cancer Hallmarks in Shaping Intratumor Heterogeneity and Tumor Morphology

SPEAKERS Khola Jamshad (University of Michigan)
AUTHORS Khola Jamshad (University of Michigan); Trachette Jackson (University of Michigan)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Advances in multiregion sequencing have revealed extensive intratumor heterogeneity (ITH) - the presence of genetically distinct subclones within a single tumor. ITH profoundly influences tumor behavior, including well-accepted hallmarks of cancer such as sustained proliferation, resistance to apoptosis, and immune evasion, and the emergence of therapeutic resistance. In this work, we introduce a hallmark-integrated branching evolution process agent-based model (BEP-HI) to study the emergence of ITH in superficial spreading melanoma (SSM) under coupled genetic, immune, and spatial selection pressures. We identify three distinct evolutionary ITH modes and demonstrate a mechanistic decoupling between tumor growth kinetics and heterogeneity. We further show that immune recruitment exerts the strongest influence on ITH, while motility of melanoma cells shapes complex morphologies in different ITH modes as observed in clinical SSM tumors. This work provides a biologically grounded computational framework for exploring how hallmark interactions shape ITH evolution, tumor behavior, and morphology.

CT49 — Bonsai: Tree representations for distortion-free visualization and exploratory analysis of single-cell omics data

SPEAKERS Daan de Groot (IMBA, Vienna BioCenter, Austria)
AUTHORS Daan de Groot (IMBA, Vienna BioCenter, Austria); Erik van Nimwegen (Biozentrum, University of Basel, Switzerland); Mikhail Pachkov (Biozentrum, University of Basel, Switzerland); Sarah Morillo Leonardo (Biozentrum, University of Basel, Switzerland)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.34 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Single-cell omics methods promise to revolutionize our understanding of gene regulatory processes, offering genome-wide measurements at single-cell resolution. However, there are three central challenges: they are high-dimensional, noisy, and provide only snapshots. Together, these challenges obstruct the extraction of accurate time-resolved gene expression dynamics. To still explore these datasets, the field has relied on embeddings like t-SNE or UMAP, even though such methods distort the intrinsic structure of the data.

To address this, we developed a broadly applicable Bayesian method called *Bonsai*, which reconstructs the most likely tree that relates any set of high-dimensional objects while rigorously accounting for heterogeneous measurement noise. We find that Bonsai exploits a “blessing of dimensionality”: representing cell-to-cell relations with a tree structure becomes virtually perfect at high dimensionality.

Although *Bonsai* can be used to explore data from a broad range of fields, it is particularly natural for studying differentiation processes, where all cells are related through a cell-division tree. We therefore applied *Bonsai* to a blood cell dataset where it recapitulated known differentiation trajectories based on only one snapshot of scRNA-seq data. Moreover, Bonsai finds convincing evidence that NK cells can derive from both myeloid and lymphoid ancestors in vivo, a novel and biologically significant result.

CT1204 — Closed-Form Steady-State Analysis of Multi-Stage Continuous Bioprocess Cascades

SPEAKERS Andrea Graf (University of Vienna)
AUTHORS Andrea Graf (University of Vienna); Jürgen Zanghellini (Department of Analytical Chemistry, University of Vienna)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Model-based design advances bioprocess development [Gra+25a; Gra+25b], yet multi-stage continuous microbial processes remain empirically designed due to poor system-level understanding. Here, we introduce a systems-level, physiology-based framework for analyzing multi-stage continuous bioprocesses, enabling early-stage screening with minimal experimental effort.

Starting from mass balance equations under substrate-limited conditions, we derive closed-form steady-state expressions for biomass, substrate, and product concentrations in reactor cascades. This analytical description enables interrogation of trade-offs between dilution rates, inter-stage coupling, and physiological constraints. We show that optimal cascade performance cannot be achieved by individual optimization, revealing a limitation of stage-wise design intuition.

From this analysis, we derive rules for globally optimal operation of multi-stage continuous processes. Optimal performance requires maximizing substrate feed to the production stage, minimizing downstream dilution, and selecting maximal upstream biomass concentrations.

The framework is implemented as an interactive web tool for design space exploration [GZ26]. It can assess design modifications, like residence time redistribution and feed adjustments. By combining steady-state analysis with an accessible tool, the approach links mathematical modeling with practical process engineering.

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CT1555 — Competing mechanisms for anisotropic lesion formation in cardiac PFA modelling

SPEAKERS Luca Gerardo-Giorda
AUTHORS Argyrios Petras (Johann Radon Institute for Computational and Applied Mathematics); Gerard Amoros Figueras (IR Sant Pau); Zoraida Moreno Weidmann (Hospital de la Santa Creu i Sant Pau); Tomas Garcia Sanchez; David Vilades Medel (Hospital de la Santa Creu i Sant Pau); Aurel Neic (Numericor GmbH); Ed Vigmond (IHU LIRYC Bordeaux); Antoni Ivorra (Universidad Pompeu Fabra); Jose M. Guerra (Hospital de la Santa Creu i Sant Pau); Luca Gerardo-Giorda
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 11.11 - SR
SESSION Contributed Talks
TRACK Cardiovascular Modelling

Abstract

Pulsed field ablation (PFA) is a novel cardiac ablation technology based on irreversible electroporation that has revolutionized the arrhythmia treatment. Despite its wide use, computational models are still unable to accurately capture the experimentally observed range of width-to-depth anisotropy ratio of the resulting lesions. This work enhances the porcine open-chest computational model we introduced in [Pet+25] by incorporating ventricular cardiac fibre orientations, as well as two competing terms that alter the tissue electrical conductivity in an anisotropic manner: an electroporation term that depends on the cardiomyocyte orientation, and a gap junction closure term that acts in the fibre direction. Lesion size was estimated using a lethal electric field threshold (LTE), and the resulting simulated lesions were compared with experimental measurements reported in [Pet+25]. The presence of the two competing mechanisms allows for a more accurate matching of the simulated lesion shape with the experimentally observed ones. A sensitivity analysis for the model parameters highlights the gap junction closure term as the second most important parameter (after the electric field lethal threshold) affecting the lesion

shape. The proposed model enables then accurate simulations and lesion predictions that align with the experimentally observed results.

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CT331 — Counterfactual evaluation of elementary and secondary school policies in the COVID-19 pandemic

SPEAKERS Benedetta Canfora (Faculdade de Ciências da Universidade de Lisboa)
AUTHORS Otilia Boldea (Tilburg School of Economics and Managemet, Tilburg University); Ana Nunes (Faculdade de Ciências da Universidade de Lisboa); Ganna Rozhnova (University Medical Center Utrecht); Benedetta Canfora (Faculdade de Ciências da Universidade de Lisboa); Rey Audie Escosio (Faculdade de Ciências da Universidade de Lisboa)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Understanding the role of schools in SARS-CoV-2 transmission is essential for designing effective public health policies. We developed a stochastic age-stratified transmission model, fitted to epidemiological and contact survey data, to evaluate how alternative school closure and reopening strategies could have shaped the COVID-19 pandemic in the Netherlands from early 2020 to late 2021. Using counterfactual simulations, we quantified the impact of elementary and secondary school interventions on infections and hospitalizations. We also applied time-varying elasticity analysis to quantify how age groups and school levels contributed to transmission over time. School closures reduced transmission and healthcare burden, but their effectiveness was strongly context-dependent and varied across pandemic phases. The relative contribution of age groups to transmission also changed over time: adults dominated early in the pandemic, adolescents played a larger role during late 2020 and early 2021, while children became increasingly important in late 2021. Consequently, secondary schools had a larger impact on hospitalizations during earlier phases, whereas elementary schools became more influential later, partly due to lower infection- and vaccine-induced immunity among younger children. These findings highlight the importance of accounting for age-specific transmission dynamics when evaluating school policies and support more targeted and adaptive strategies during future pandemics.

CT1288 — Deterministic discrete-particle modeling of ribosome stalling beyond mean-field approximations

SPEAKERS Katrin Schröder
AUTHORS Falk Herwig (UViC); Katrin Schröder
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.33 - SR
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Ribosome profiling provides codon-resolution snapshots of translation, but quantitative interpretation requires accurate forward models of ribosome traffic. We revisit mRNA translation stalling by comparing the widely used mean-field Ribosome Flow Model (RFM) with a deterministic discrete-particle TASEP

formulation. We found that mean-field factorization can produce artifacts, including exaggerated upstream jams near low-density/high-density transitions, artificial backfilling at clusters of slow codons, and shifted phase boundaries. To address this, we develop a deterministic credit-based hopping and drop-off framework that preserves strict exclusion at codon resolution while reproducing ensemble-average TASEP behavior without Monte Carlo sampling. The model includes ribosome-footprint constraints and site-specific drop-off, and is computationally efficient for large parameter scans in inference workflows. We classify profiles into four regimes: low density (LD), high density (HD), local high density (LHD), and local density enhancement (LDE), each linked to interpretable rate ratios. Finally, we use measurement-aware forward mappings to generate ribosome-profiling-like observations from predicted densities under finite positional resolution and count noise.

CT1413 — Household-level patterns of vaccine uptake during the COVID-19 pandemic in the UK

SPEAKERS Joe Hilton (University of Manchester)
AUTHORS Joe Hilton (University of Manchester); Thomas House (The University Manchester)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Household-level vaccine uptake patterns are known to have serious impacts on infectious disease dynamics, with clusters of susceptibility in low-uptake households driving global transmission and potentially leading to inequitable burdens of disease [Gea+15]. Anticipating these uptake patterns could improve the design of vaccine rollout campaigns, but they remain poorly understood. The ONS COVID-19 Infection Survey (ONS-CIS) recorded regular COVID-19 test results from 2020-2023 for over 200,000 participating UK households along with vaccination dates and sociodemographic factors, offering an unparalleled source of data on individual- and household-level uptake behaviour. In this initial modelling analysis of vaccination data from ONS-CIS, we aim to quantify household-level clustering of uptake within the survey cohort. We compare the observed uptake patterns with two theoretical extremes: uniform random vaccination and a worst-case scenario in which entire households are either vaccinated or unvaccinated. To project the epidemiological impact of these patterns we calculate weekly estimates of the household reproductive ratio, a key invasion parameter for household epidemic models, over the course of the rollout period. We find that while the UK's vaccine rollout was intermediate between the two extreme scenarios in terms of uptake, the likely effect on overall spread of infection appears to have been closer to the suboptimal by-household uptake scenario.

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CT1570 — Machine Learning and Optimization Approaches for Improving Artificial Insemination Service Delivery in Philippine Water Buffalo Systems

SPEAKERS Renier Mendoza
AUTHORS Edoardo Fadda; Rachelle Anne Guanga; Renier Mendoza
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Artificial insemination (AI) is widely used to improve the genetic quality and productivity of water buffalo in the Philippines through the nationwide program of the Department of Agriculture–Philippine Carabao Center (DA-PCC). Despite ongoing efforts, calf production remains low and inconsistent across regions. This study analyzes five years of AI service data comprising 278,978 records from DA-PCC regional centers to identify factors associated with successful calf production. Several machine learning models were developed and evaluated to predict calf production outcomes, with Extreme Gradient Boosting achieving the best performance across standard evaluation metrics. The results show that the assigned service center and the distance between technicians and buffalo farms are among the most important predictors, with longer travel distances associated with lower calf production rates. Motivated by these findings, a mathematical optimization framework is proposed to improve the delivery of AI services. The resulting AI Technician Routing Problem integrates farmer–technician assignment, route planning, and semen inventory considerations into a unified model formulated as a mixed-integer linear program. The framework provides a quantitative approach for improving the efficiency and reliability of AI service delivery in water buffalo production systems.

CT285 — PerTexP: scenario-based exploration of pertussis dynamics under maternal and infant vaccination

SPEAKERS Emanuela Penitente (Department of Mathematics and Applications, University of Naples Federico II, Naples, Italy.)
AUTHORS Anna Autoriello (Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Pavia, Italy); Sabrina Averga (Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Pavia, Italy); Bruno Buonomo (Department of Mathematics and Applications, University of Naples Federico II, Naples, Italy.); Rossella Della Marca (Department of Mathematics and Applications, University of Naples Federico II, Naples, Italy.); Alfredo Guarino (Pediatric Infectious Disease Unit, Department of Maternal and Child Health, University Hospital Federico II, Naples, Italy); Cristina Moracas (Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Pavia, Italy); Emanuela Penitente (Department of Mathematics and Applications, University of Naples Federico II, Naples, Italy.); Marco Poeta (Pediatric Infectious Disease Unit, Department of Maternal and Child Health, University Hospital Federico II, Naples, Italy)
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Despite the widespread availability of effective vaccines, pertussis remains a significant public health concern in many high-income countries, particularly because of its severe impact on infants. In recent decades, several regions have experienced a resurgence of pertussis, with recurrent outbreaks and an overall increase in incidence [Yeu+17]. These patterns highlight the need for modelling tools that are both mathematically rigorous and accessible to health practitioners, supporting the exploration of intervention scenarios and evidence-based planning. To this end, we present PerTexP (Pertussis Time Exploration), an interactive MATLAB tool for scenario-based analysis of pertussis transmission under different vaccination strategies [Aut+26]. PerTexP is based on a discrete-time, stage-structured compartmental model with two age classes, infants and non-infants, and explicitly incorporates maternal immunisation, infant vaccination, and booster doses in older individuals. Through a graphical user interface, the tool allows users to compare vaccination scenarios and assess their impact on pertussis transmission. Applied to the 2024 Italian pertussis outbreak [Poe+24], PerTexP suggests an increasing burden over a five-year horizon and shows that increasing maternal coverage yields a larger reduction in cumulative incidence than an equivalent increase in booster uptake, while elimination requires strengthening both interventions.

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CT1461 — Spatial heterogeneity accelerates phase-to-trigger transitions of mitotic waves

SPEAKERS Daniel Ruiz-Reynés (IFISC, Institute for Cross-Disciplinary Physics and Complex Systems)
AUTHORS Owen Puls; Daniel Ruiz-Reynés (IFISC, Institute for Cross-Disciplinary Physics and Complex Systems); Franco Tavella; Minjun Jin; Yeonghoon Kim; Lendert Gelens; Qiong Yang
SCHEDULE Wednesday, 15 July 2026; 11:10–11:30; 11.34 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Periodic oscillations in Cyclin B–Cdk1 activity function as the core timing mechanism that controls the onset of cell division. These oscillations emerge from a complex regulatory network of interacting proteins, creating a self-sustained biochemical oscillator that regulates mitotic progression. In spatially extended systems, such oscillatory behavior can propagate as waves that synchronize mitotic entry across space. This mechanism supports rapid and coordinated divisions in large embryos, such as those of Drosophila and Xenopus, which can reach millimeter-scale dimensions. Nevertheless, the mechanisms by which these waves coordinate mitosis across both space and time remain incompletely understood. Here, we present experimental results using Xenopus laevis egg extracts to reconstitute mitotic waves in vitro. We observe a transition from phase waves to trigger waves, which is explained through mathematical modeling that underscores the roles of transient dynamics and spatial heterogeneity. To further probe how these collective dynamics emerge, we introduce nuclei and apply boundary perturbations to examine how wave-mediated coupling promotes entrainment and spatial coordination of mitosis. Together, these findings clarify how phase and trigger waves contribute to the spatial organization of mitotic progression.

CT1681 — A Conservative Network Framework for Coupled Hemodynamics and Physiological Transport

SPEAKERS Lester Caudill (University of Richmond)
AUTHORS Lester Caudill (University of Richmond)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 03.01 - SR
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

We present a discrete-time compartmental model for human circulation that couples pulsatile blood flow with physiological transport processes. The cardiovascular system is represented as a conservative network of interconnected compartments corresponding to major organs and vascular regions, including the heart, lungs, brain, kidneys, liver, gastrointestinal system, systemic tissue beds, and venous reservoir. Blood volume is treated as the primary dynamical state variable, allowing natural enforcement of global conservation laws while deriving pressures from compartment compliance relationships. Flows between compartments are driven by pressure differences and vascular resistances, with directional flow enforced through valve-like constraints. Cardiac pumping is modeled through periodically varying ventricular compliance, producing pulsatile circulation within a computationally efficient reduced-order framework. A primary motivation for the model is extension to transported substances such as oxygen, sodium, glucose, metabolic waste, hormones, and pharmaceuticals. Because transport follows dynamically computed blood

flow, the framework naturally supports coupling among circulation, metabolism, filtration, and organ-level exchange. Future directions include autoregulation, biochemical transport, and applications to collateral organ dysfunction and sepsis.

CT1416 — A Hybrid Cell-Based Model of Lipid-Immune Interactions in Atherosclerosis.

SPEAKERS Thomas Quinlan (University of Oxford)
AUTHORS Thomas Quinlan (University of Oxford); Helen Byrne (University of Oxford); Ruth Baker (University of Oxford); Claudia Monaco (University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 11.11 - SR
SESSION Contributed Talks
TRACK Cardiovascular Modelling

Abstract

Atherosclerosis is a disease driven by cholesterol accumulation within the arterial wall and involving coupled interactions between lipid transport and inflammation. Despite being a leading cause of cardiovascular disease, the spatial and stochastic mechanisms underlying plaque formation remain incompletely understood. To address this, we developed a hybrid continuum-discrete model of atherosclerotic plaque formation using the cell-based modelling framework Chaste. Our model incorporates feedback between lipid accumulation, cell death, and inflammatory chemokine production. We treat macrophages as discrete agents whose behaviour depends on the spatio-temporal distribution of lipoproteins, lipids, and the chemokine CCL2, whose dynamics are determined by a system of reaction-diffusion equations. The model is suitable for exploration of how cell-level processes influence local lipid and immune cell accumulation in early plaque development. Ongoing work focuses on exploring the model dynamics across parameter regimes.

CT1598 — Breaking free from 2D: Extending the cellular Potts model to curved deforming surfaces to understand vascular remodelling

SPEAKERS Jessica Crawshaw (Queensland university of Technology)
AUTHORS Jennifer Flegg (University of Melbourne); Jessica Crawshaw (Queensland university of Technology); James Osborne (University of Melbourne)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The Cellular Potts Model (CPM) is a cell-based model that provides a stochastic, energy minimisation based approach for modelling collective cell behaviour and tissue organisation. Currently, the CPM is defined over a fixed 2D lattice, restricting applicability to tissues that evolve on static planar domains. However, many epithelial and endothelial cell populations reside on curved and deformable surfaces where cellular dynamics and the evolving surface geometry are intrinsically coupled.

In this talk, we will present an extension of the classical CPM in which cells evolve over a deformable elastic surface embedded in 3D in the context of vascular regression. The surface of the capillary walls are represented by triangulated surface meshes and evolve according to a finite element model of hyperelastic deformation, with the coupled cellular dynamics determined through the classic CPM Hamiltonian. Through this coupling, cellular forces influence surface mechanics and vice versa. This framework is implemented within the open-source Chaste infrastructure.

We will first describe and validate our novel deformable CPM model by reproducing behaviour consistent with the classical CPM across static planar and curved surfaces. We then examine how endothelial cells

integrate competing mechanical cues arising from haemodynamic forcing and intercellular adhesion, and how these interactions contribute to the reorganisation and optimisation of vascular networks.

CT1256 — Capturing complex organoid dynamics using image analysis, modeling and simulation

SPEAKERS Franziska Matthäus (Frankfurt Institute for Advanced Studies, Goethe University Frankfurt)
AUTHORS Franziska Matthäus (Frankfurt Institute for Advanced Studies, Goethe University Frankfurt)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.33 - SR
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Organoids are experimental model systems of organ development and function. They have recently gained considerable attention, as they provide insight into processes regulating development and regeneration in different tissues, and allow testing of treatment strategies for various pathogenic conditions. Pancreatic and liver-derived organoids in 3D culture display diverse dynamic behaviors. They grow into spherical epithelial monolayers, interact with the surrounding medium, secrete into an internal lumen, and can exhibit volume oscillations, fusion, drift, and rotational motion. To investigate which cell-level mechanisms, and their interactions, drive these behaviors, we analyzed 3D+t light sheet microscopy data. We employed organoid-level and nucleus segmentation, cell tracking, and 3D particle image velocimetry, to quantify size, morphology, volume oscillations, cell division dynamics, angular velocity, and drift. Using a mathematical approach, we derived a scaling law linking volume oscillations to cell division. We further developed an agent-based model incorporating mechanical cell-cell interactions, division, migration, mechanotransduction, polarity, and lumen pressure. The model reproduces the observed behaviors and enables systematic exploration of how complex organoid dynamics emerge from the interplay of fundamental cell-level mechanisms.

CT1503 — Conditions on Interaction Networks for Achieving Specific Reinforcement under Global Dopamine Signals

SPEAKERS Yonatan Savir (Technion - Israel Institute of Technology)
AUTHORS Or Ben Yaakov (Technion - israel institute of technology); Yonatan Savir (Technion - Israel Institute of Technology)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 11.34 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Reward-based learning relies on a system's ability to distinguish between environmental signals across space or time. However, biological reinforcement systems do not always operate under such idealized conditions. In particular, dopamine reward signals are often broadly distributed rather than spatially localized, while environmental stimuli frequently overlap in time in realistic settings. This raises the question of how learning can remain robust to interference from competing signals [SDM97; Sch24].

In this work, we investigate how robust learning can emerge when reinforcement signals lack temporal or spatial specificity. Building on a theoretical framework for global homeostasis in networks [Ste+11; SMS17], we derive conditions on the network structure and feedback topology that guarantee the stability of dopamine-mediated reinforcement signals in the presence of interfering inputs. We show that robustness is achieved only when dopamine secretion is downregulated by the overall level of global stimulation. Under this topology, global and non-specific regulation gives rise to selective responses by suppressing interference between competing inputs. This result does not rely on parameter tuning or detailed biological

assumptions and reveals a nonlinear feedback principle applicable to a broad class of systems that must achieve specificity using control mechanisms that are not signal-specific.

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CT104 — Enhancing epidemiological parameter inference using quantitative diagnostic data

SPEAKERS Punya Alahakoon (Pandemic Sciences Institute, University of Oxford)
AUTHORS James Hay (Pandemic Sciences Institute, University of Oxford); Punya Alahakoon (Pandemic Sciences Institute, University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.34 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Most infectious disease datasets are dichotomous, typically indicating the presence or absence of infection. However, many contain quantitative measurements, such as cycle threshold (Ct) values from PCR tests or antibody titers from serological assays. Despite this, epidemiological models often rely on dichotomised data to infer key immunological and transmission parameters. This simplification discards valuable information embedded in the original quantitative measurements [Coh83].

In this work, I will first quantify the information loss incurred by dichotomising quantitative data using metrics such as the Kullback–Leibler (KL) divergence. I will then present a framework for incorporating quantitative measurements, specifically Ct values and antibody titers, into epidemiological models to capture population-level dynamics better. I will apply this framework to COVID-19 data in the United Kingdom and explore different testing settings and data availability scenarios. Finally, I will discuss the practical considerations and methodological advantages of using quantitative data over dichotomised alternatives, highlighting implications for inference, model accuracy, and public health decision-making [Ala+25].

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CT1531 — Fluid Transport through the Boundary of the Human Brain

SPEAKERS Emil Eriksson
AUTHORS Eric Libby; Sofia Behndig; Katrine Riklund; Jan Malm; Anders Eklund; Anders Wåhlin; Emil Eriksson
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Fluid transport through brain tissue is important for removing harmful metabolic waste. Experiments on rodents indicate the presence of an active waste efflux system that transports harmful solutes by diffusion and advection. In humans, the existence of this system is still debated. The effective diffusivity within human brain tissue has been estimated using contrast-enhanced magnetic resonance imaging (MRI) data and inverse physics. Quantifying advection has also been attempted, although achieving reliable estimates has been more challenging. Recent modeling results, validated on rodent data, highlight the importance of determining the barrier properties at the brain boundary and its effect on the waste efflux system. Here, we develop a data-driven method to estimate the effective diffusivity of fluid through the boundary of the human brain. We fit a one-dimensional diffusion model to contrast-enhanced MRI data from 980 samples, collected from 14 participants over 22 hours. This is achieved by mapping the gyrified three-dimensional geometry of each sample to a one-dimensional space identified by tissue depth. Using these results, we estimate a non-dimensional boundary parameter that recently has been identified to be essential for waste clearance but has not previously been estimated for humans.

CT238 — Modelling Immune Dynamics in Locally Advanced MSI-H/dMMR Colorectal Cancer with Neoadjuvant Pembrolizumab Treatment: From Differential Equations to an Agent-Based Framework

SPEAKERS Georgio Hawi (University of Sydney)
AUTHORS Georgio Hawi (University of Sydney); Peter P. Lee (Beckman Research Institute, City of Hope); Peter S. Kim (University of Sydney)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Colorectal cancer (CRC) is the world's third most common malignancy, and patients with the microsatellite instability–high/mismatch repair–deficient (MSI-H/dMMR) phenotype respond poorly to conventional chemotherapy yet show striking responsiveness to immune checkpoint inhibitors such as pembrolizumab. Mathematical models based on ordinary differential equations (ODEs) provide a powerful framework for analysing the immunobiology underpinning disease dynamics. In contrast, agent-based models (ABMs) explicitly represent individual entities and their interactions; inherent randomness can naturally give rise to heterogeneity—features that deterministic, mean-field ODEs cannot capture. However, ABMs typically encode many interactions and processes, making simulations computationally expensive and parameter calibration to experimental data difficult.

In this talk, we present a minimal ODE model of pembrolizumab therapy in locally advanced MSI-H/dMMR CRC (laMCRC) to reveal the core drivers of the tumour–immune response and the key immune interactions involved. We also outline a practical workflow for converting ODE models to ABMs and

apply it to our minimal model, enabling accurate and efficient parameter estimation for the ABM. Finally, we compare ABM trajectories with those from the minimal ODE model, verifying consistency and faithful reproduction of the dynamics, thereby laying a foundation for future ABMs of laMCRC and other cancers.

CT1420 — Seasonal Reaction-Diffusion Model of Vector-Borne Diseases: Existence of periodic dynamics

SPEAKERS Michela Sabbatino (University of Trento, Italy)
AUTHORS Andrea Pugliese (University of Trento, Italy); Cinzia Soresina (University of Trento, Italy); Michela Sabbatino (University of Trento, Italy)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Vector-borne diseases are infections transmitted between two interacting biological populations: a host, such as birds, and a vector, typically mosquitoes. The dynamics of these types of diseases can be linked to seasonal fluctuations, which in turn influence the spatial spread of the infection. To this end, following Lewis et al. [LRD06], we couple an SIS model for the host population with an SI model for the vector population. Spatial terms are included, leading to a system of reaction–diffusion equations that describe random movement in space. To incorporate both epidemic spatial spread and seasonal dynamics, we assume that no transmission occurs during the winter season, while epidemiological parameters remain constant during the summer season. Consequently, the inter-annual dynamics are governed by the survival rates of the infected populations. First, we consider the non-spatial model and demonstrate, following Hethcote [HT85], the existence of a unique periodic dynamics, providing a clear threshold for disease persistence. Extending the analysis to the spatial model, and following the framework established by Lui [Lui89a; Lui89b] and Li et al. [LWL05], we derive that the epidemic spreads from year to year with an asymptotic propagation speed c^*. Finally, we illustrate the results through numerical simulations, comparing the speed of propagation c^* obtained from numerical solutions with its analytical estimate.

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CT312 — The effects of vertical transmission on a spatially-structured host-parasite model

SPEAKERS Jack Woodruff (University of Sheffield)
AUTHORS Jack Woodruff (University of Sheffield); Alex Best (University of Sheffield)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Vertical transmission of an infectious disease from parent to offspring is a common transmission route in many systems. Here we explore the role of vertical transmission within a host-parasite system, with a particular focus on how spatial structure effects vertical transmission. We introduce a host-parasite lattice model with a pair approximation that includes both local and global transmission and reproduction. We find that vertical transmission can determine pathogen invasion and reduce the horizontal transmission rate required for invasion. When the majority of transmission and reproduction is local, vertical transmission can destabilise a host population to cause limit cycles. Given the advantages of a pathogen having both horizontal and vertical transmission routes, we extend the model to investigate the likelihood a mutant strain with both transmission modes will outcompete a resident strain with only horizontal transmission. When there is no trade-off the mutant always invades and when there is a trade-off with horizontal transmission, the mutant emerges when the cost to the horizontal transmission rate is not too large. Depending on how the mutant appears within the host population, it may have an initial advantage over the resident strain even if it cannot outcompete in the long-term. Our work demonstrates the potential importance of vertical transmission within host-pathogen dynamics.

CT1196 — The Frustrating Nature of Tissues

SPEAKERS Taylor Woodward (The University of Melbourne)
AUTHORS Taylor Woodward (The University of Melbourne)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Cells are embedded in spatial tissues. This context shapes cell-fate decision, global tissue structures, and, ultimately, the development of multi-cellular systems. One of the pivotal questions is the extent to which coherent tissue structures arise from the interplay between intra- and intercellular signalling and regulatory projects. Here we highlight how spatial context can lead to a phenomenon that is known as “frustration” in spatial statistical physics: what happens at one level can conflict with what happens at another cell. Here we consider frustration as a conflict between the cell intrinsic regulatory processes and the signals that they receive from neighbouring cells. By taking a disciplined approach, guided by recent work in Ising spin glass models, we map out how and when frustration can shape complex tissue processes and lead to rich stable spatial patterns. We conclude by mapping out the biological relevance of these ideas as conceptual and computational frameworks for tissue patterning processes that start explicitly from a cellular rather than continuum points of views.

CT267 — Understanding the Biology Underpinning the Interaction between Arboviruses and Aedes aegypti Mosquitoes Using Statistical and Mathematical Modelling

SPEAKERS Junjie Chen (University of Oxford)
AUTHORS Junjie Chen (University of Oxford); Douglas Brackney (The Connecticut Agricultural Experiment Station); Philip Armstrong (The Connecticut Agricultural Experiment Station); Godfrey Indinda (The Connecticut Agricultural Experiment Station); Teresa Lambe (University of Oxford); Christl Donnelly (University of Oxford); Sumali Bajaj (University of Oxford); Ben Lambert (University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 11:30–11:50; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Despite decades of efforts to control mosquito-borne diseases, the global burden of these diseases remains high due to their complex biological and immunological dynamics. While extensive research has focused on vector-host or between-host transmissions, the within-vector mechanisms remain poorly understood. This gap is critical to fill because mosquitoes have relatively short lifespans, and their midgut acts as the primary bottleneck for viral progression. Following an infectious blood meal, viruses must first infect midgut epithelial cells before reaching the salivary glands and becoming transmissible to the next host. Understanding midgut infection is therefore central to designing more targeted interventions, which could complement traditional vector control strategies and help address the current lack of effective prophylactic drugs and vaccines.

In this study, we establish an iterative cycle between modelling and experiments. We develop a 3D stochastic model of midgut infection dynamics and show that, with parameters informed by experimental assays, the model can reproduce the clustered infection patterns observed in dengue-infected midguts of *Aedes aegypti* mosquitoes. This framework provides a quantitative tool to investigate the pivotal role of the mosquito midgut in shaping infection dynamics, and offers a basis for designing targeted vector control strategies. It can also be extended to study other mosquito-borne viruses.

CT1270 — A Hybrid Agent-Based Framework for Quantifying Mitochondrial Dynamics from Time-Lapse Imaging

SPEAKERS Volkan Ozcoban (The University of Melbourne)
AUTHORS Aidan Quinn (The University of Melbourne); Ashan Wanasinghe (The University of Melbourne); Sanjeev Uthishtran (Monash University); Senthil Arumugam (Monash University); Vijay Rajagopal (The University of Melbourne); Volkan Ozcoban (The University of Melbourne)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.33 - SR
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Mitochondrial networks range from fragmented to hyperfused, spatially organised to meet energetic and signalling demands [CZL23]. Cycles of fission, fusion, biogenesis, mitophagy, and motility (termed mitochondrial dynamics) underpin the capacity for homeostasis and quality control, yet mechanistic frameworks interrogating these emergent properties are scarce [KK14; Dal+17; Hol+24; Fog+25]. We present a hybrid spatial agent-based model of mitochondrial dynamics where nodes evolve via overdamped Langevin dynamics with active transport and dynamic graphs capture remodelling. The model couples motion, collision, and local fission-fusion rules to probe how mechanics and motility shape networks. Population-level distributions, fission-fusion rates, graph metrics, and trajectory statistics facilitate comparison with segmented widefield time-lapse data. The model is being developed to describe key mammalian mitochondrial dynamics insights. Analysis in mammalian cultures yields three insights: (i) cumulative remodelling is driven by accumulation of discrete fission events exceeding fusion, (ii) correlated

fission-fusion rates suggest rapid reciprocal priming of events, and (iii) spatial spread depends on cellular context and transport dynamics. This quantitative framework enables hypothesis testing and parameter inference to explore how mitochondrial dynamics influence cell-level behaviours such as cell polarity, energy supply, and migration.

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CT1604 — A Kuramoto Oscillator Model of Closed-Loop Auditory Stimulation of REM Sleep EEG

SPEAKERS Daniel Knott (School of Mathematics & Physics, Faculty of Engineering and Physical Sciences, University of Surrey, Guildford, UK)
AUTHORS Anne Skeldon (School of Mathematics & Physics, Faculty of Engineering and Physical Sciences, University of Surrey, Guildford, UK; Surrey Sleep Research Centre, School of Biosciences, Faculty of Health and Medical Sciences, University of Surrey, Guildford, UK); Daniel Knott (School of Mathematics & Physics, Faculty of Engineering and Physical Sciences, University of Surrey, Guildford, UK); Derk-Jan Dijk (Surrey Sleep Research Centre, School of Biosciences, Faculty of Health and Medical Sciences, University of Surrey, Guildford, UK; UK Dementia Research Institute, Care Research and Technology Centre at Imperial College, London and the University of Surrey, Guildford, UK); Stefan Klus (School of Mathematical & Computer Sciences, Heriot-Watt University, Edinburgh, UK); Valeria Jaramillo (School of Psychology, Faculty of Health and Medical Sciences, University of Surrey, Guildford, UK; Surrey Sleep Research Centre, School of Biosciences, Faculty of Health and Medical Sciences, University of Surrey, Guildford, UK; UK Dementia Research Institute, Care Research and Technology Centre at Imperial College, London and the University of Surrey, Guildford, UK)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Closed-loop auditory stimulation (CLAS) is a promising non-invasive technique for modulating brain oscillations by delivering auditory stimuli phase-locked to ongoing neural activity. Recent work applying CLAS to theta (4.5 – 7.5 Hz) and alpha (7.5 – 12.5 Hz) oscillations during REM sleep has demonstrated phase-dependent changes in EEG power and frequency [Jar+24]. It has been hypothesized that these effects arise from phase-resetting.

We developed a mathematical model to test the extent to which a phase-reset mechanism can predict the observed changes. Using a Kuramoto oscillator model [Mir+25] with all-to-all coupling, we tracked the

collective network activity of oscillators with natural frequencies in the theta and alpha bands separately, allowing band-specific phase-locked stimulation and analyses.

Baseline parameters were fitted to unperturbed REM activity, with stimulation parameters determined from single-pulse evoked responses using phase-response curves. The system was simulated with phase-locked stimulation, including realistic phase-locking error, to predict phase-dependent frequency and power changes. We compared predictions against the data, analysed parameter sensitivity, and assessed how phase-locking accuracy affects outcomes.

Our work provides a quantitative framework for understanding how stimulation timing influences CLAS effects and for guiding future protocol design.

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CT1646 — Biphasic responses in cell signalling systems

SPEAKERS J Krishnan (Imperial College London)
AUTHORS J Krishnan (Imperial College London); Vaidhiswaran Ramesh (Imperial College London)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 11.34 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

There are many instances in cell biology, and in cell signalling in particular where non-monotonic dose responses are encountered, and these have important biological and physiological implications. In this presentation we dissect biphasic responses at the basic biochemical level, at the network level and in concrete cell signalling systems. At the basic biochemical level we explore a variety of systems which are extensions of covalent modification cycles (eg cascades, substrate modification sequences), with different degrees of commonality of enzymes and/or substrates. We uncover when these system are capable of enzyme-biphasic (biphasic responses to change in total enzyme amount) and substrate biphasic (biphasic responses to change in substrate amount. We determine when biphasic responses will and will not be possible (in the latter case independent of parameters). We then explore the effect of biphasic effects in regulation in networks. Finally we also the presence of enzyme biphasic and substrate biphasic responses in concrete exemplar signalling systems. Overall through a combination of analytical and computational work, we uncover a diversity of sources of biphasic responses, how they combine and how they impact systems behaviour.

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CT1249 — Building Spatial Resolution into Ebola virus VP40 Assembly Models: An ODE-to-ABM Transition

SPEAKERS Trevor Shoaf (Purdue University)
AUTHORS Elsje Pienaar (Purdue University); Robert Stahelin (Purdue University); Trevor Shoaf (Purdue University)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

The Ebola virus matrix protein VP40 oligomerizes at the plasma membrane inner leaflet to drive virus-like particle (VLP) formation in mammalian cells, a process dependent on the membrane lipid phosphatidylserine (PS). We previously developed an ODE model of VP40 oligomerization that provided evidence for a nucleation-elongation mechanism and explicitly incorporated the VP40–PS relationship that influences budding. However, the ODE framework cannot resolve experimentally-observed spatial effects of PS membrane heterogeneity. To address this, we developed an agent-based model (ABM) of VP40 dynamics in a VP40 transfected cell and calibrated the ABM to experimental data from HEK293 cells. Comparing calibrated ODE and agent based models revealed notable differences: the ODE required a monomer production rate of 16 nM/s, while the ABM reproduced equivalent VLP and VP40 dynamics at <1 nM/s. Experimentally these values are hard to measure, but with this analysis we can start to see that these parameter estimates appear to be model-type specific. We use the ABM to investigate how PS spatial organization influences VLP production — specifically, how the fraction of membrane surface area enriched in PS and the spatial arrangement of high-PS domains (small versus large clusters) modulate budding efficiency. From this work, we will be able to test if membrane targeted therapies that affect PS heterogeneity are viable options for reducing VLP production.

CT190 — Efficient Parameter Estimation in Agent-Based Models of Collective Cell Invasion via Gaussian Process Surrogates and Bayesian Optimization

SPEAKERS Ashlee Ford Versypt (University at Buffalo, The State University of New York)
AUTHORS Aneesh Krishna (University at Buffalo, The State University of New York); Khansa Khanam Umar Sultan (University at Buffalo, The State University of New York); Temitope Benson (University at Buffalo, The State University of New York); Ashlee Ford Versypt (University at Buffalo, The State University of New York)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.34 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cell migration and invasion are key processes underlying cancer metastasis. These behaviors are driven by cell–cell adhesion, chemotaxis, and matrix remodeling. CompuCell3D is a platform for agent-based modeling of cell migration; however, a limitation is that it does not natively support systematic parameter estimation for the stochastic simulations. Traditional Monte Carlo sampling-based approaches to parameter tuning are time-consuming and computational demanding. This work focuses on parameter estimation for agent-based modeling of collective cell invasion for two phenotypes: a network (invasive) phenotype and a spheroid (non-invasive) phenotype emerging from growth of tumor spheroids simulated using CompuCell3D. We adopt a data-centric approach based on Gaussian process surrogate models and Bayesian optimization, leveraging simulation outputs alongside experimental data for invasion dynamics and circularity of cell clusters. This framework efficiently operates with limited simulation data and sequentially proposes new parameter sets to evaluate, prioritizing those expected to provide maximal information gain. Through sequential rounds of parameter selection, agent-based simulations, and comparison with experimental data, the calibrated model reproduces experimental invasion dynamics and circularity. This framework provides an efficient approach for producing realistic invasion dynamics while reducing the need for extensive trial-and-error simulations.

CT1607 — Fast, Scalable, and Interpretable Persistent Homology for Biology

SPEAKERS Joshua Bull (University of Oxford)
AUTHORS Joshua Bull (University of Oxford)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Many biological datasets can be represented as point clouds, such as cell centres in spatial proteomics or transcriptomics imaging. These can be analysed using diverse specialised spatial metrics [Bul+25]. One important property of a point cloud is the presence of topological features - connected components, loops, or voids. Topological Data Analysis (TDA), and in particular Persistent Homology (PH), provides a framework to quantify these structures.

PH is powerful, but limited by high computational cost: standard algorithms scale at $O(n^6)$ or worse with the number of points, restricting practical usage to thousands of points – far from the millions typical in modern imaging. A second drawback is the difficulty of identifying "representatives" that allow users to interpret PH by identifying boundaries of topological features.

In this talk, we introduce a new computational approach for PH that dramatically reduces complexity, scaling better than $O(n^2)$ and enabling analysis of millions of points without subsampling. It also produces clear geometric representatives, allowing PH-based segmentation of complex biological structures. Finally, we demonstrate powerful applications of our method for huge datasets in both 2D (prostate cancer) and 3D (developing mouse embryo).

References

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CT1403 — Individual behavior can challenge the management of virulence

SPEAKERS Clément MONAURY (L'Institut Agro Rennes-Angers)
AUTHORS Clément MONAURY (L'Institut Agro Rennes-Angers); Frederic Hamelin (L'Institut Agro Rennes-Angers); Ludovic Mailleret (INRAE); Virginie Ravigné (CIRAD)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Virulence management, which studies the evolution of pathogen virulence (i.e., pathogen-induced mortality), shows that when vaccination reduces susceptible host density, selection may favor lower virulence. However, most studies assume fixed host behavior: a constant fraction of the population vaccinates throughout an epidemic. In reality, vaccination behavior is dynamic and shaped by social, epidemiological, and individual factors, producing temporal changes in vaccine uptake and altering the pathogen's ecological environment and evolutionary trajectory. To investigate the effects of such behavioral dynamics on virulence evolution, we develop a system of ordinary differential equations describing epidemic dynamics with newborn vaccination. Virulence evolution follows a quantitative genetics–inspired framework, while vaccination decisions follow imitation dynamics reflecting social influence. We compare the model under different vaccination scenarios. Results show that behavioral feedbacks can generate recurrent outbreaks and evolutionary cycles of virulence, producing repeated mortality peaks. Strikingly, instantaneous death rates with vaccination dynamics can transiently exceed those without vaccination. Nevertheless, cumulative mortality remains lower with vaccination. Overall, our results highlight the interplay between

epidemiology, evolution, and behavior in social-epidemiological systems. This highlights the need to include behavior in evolutionary epidemiology.

CT1683 — Integrating physics-informed machine learning and mathematical modelling for cardiac digital twinning

SPEAKERS Federica Caforio (University of Graz)
AUTHORS Christoph Augustin (Medical University of Graz); Elias Karabelas (NumeriCor); Federica Caforio (University of Graz); Francesco Regazzoni (Politecnico di Milano); Gernot Plank (Medical University of Graz); Gundolf Haase (University of Graz); Matthias Höfler (University of Graz); Stefano Pagani (Politecnico di Milano)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 11.11 - SR
SESSION Contributed Talks
TRACK Cardiovascular Modelling

Abstract

The research community is rapidly advancing in the development of cardiac biophysical models for clinical applications, due to their predictive capabilities. Nevertheless, the computational expense associated with high-resolution multi-physics models and their personalisation poses challenges for their translation into clinical settings.

This work introduces a novel methodology integrating physics-informed neural networks (PINNs) [Caf+24; HÃf+26] with 3D, time-dependent nonlinear biomechanical models of cardiac tissue to reconstruct displacement fields and estimate heterogeneous patient-specific biophysical properties. The proposed learning algorithm utilises information from a limited dataset of displacement and, in certain cases, strain data that can be routinely acquired in clinical settings, and it incorporates residual-based attention, Fourier features, and tailored regularisation strategies, enabling robust reconstruction of parameter fields under noisy conditions and at high spatial resolution. A Pareto front analysis is also conducted to assess the influence of loss weight selection on parameter estimation.

Several benchmarks are presented to highlight the accuracy and robustness of the proposed method. Notably, the study demonstrates the capability of PINNs to detect the presence, location, and severity of scar tissue, thus potentially greatly enhancing diagnostics and treatment plans for cardiac diseases.

References

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CT299 — Linking Temporal Structure to Epidemic Dynamics in Contact Networks from Epigames

SPEAKERS Estelle McCool (University of Oxford/ HDRUK)
AUTHORS Andrés Colubri (Broad Institute); Estelle McCool (University of Oxford/ HDRUK); Jasmina Panovska-Griffiths (Pandemic Sciences Institute, University of Oxford, Oxford, UK); Jess Enright (University of Glasgow)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

The spread of infectious diseases is fundamentally shaped by who contacts whom, when, and for how long. Contact networks capture the population-level structure of these interactions, yet explicitly recorded networks are rarely available, forcing outbreak models to rely on simplifying assumptions. Understanding how interpersonal contact patterns vary across contexts could meaningfully improve our ability to model disease outbreaks. Experimental epidemic games ("epigames") offer a controlled approach to this problem, using a gamified, Bluetooth-enabled smartphone app to simulate outbreaks among participants in real time.

We analysed five epigame networks from diverse settings (conferences and campuses, 2020–2025, in the UK, US and China), characterising their structure using structural, spectral, temporal, and epidemic metrics. We demonstrated that epigames capture realistic, high-resolution contact networks across social contexts, validating their use as a tool for studying real-world transmission dynamics.

Building on this, we investigated how the temporal properties of these networks drive their epidemic properties. We explored how features such as contact timing, contact persistence or temporal reachability, shape outbreak dynamics; linking temporal network structure directly to epidemic outcomes. Running epigames across diverse settings, ages, and demographics will be essential for ensuring that the resulting epidemic models are both accurate and equitable.

CT260 — Non-local models for spatio-temporal macrophage-tumour co-invasion: role of competition for resources

SPEAKERS Johan Marguet
AUTHORS Johan Marguet; Raluca Eftimie (University of Franche-Comte)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Resource competition within the tumour microenvironment is a key driver of cancer progression. In this talk, we present a non-local mathematical model to investigate how interactions between M1/M2 macrophages and tumour cells influence tissue invasion.

We demonstrate that the existence and stability of tumour-free versus tumour-present steady states are strictly governed by these competition mechanisms. Furthermore, we show that the minimum invasion speed (a proxy for tumour aggressiveness) is explicitly linked to resource competition coefficients.

We conclude by presenting the simulated spatial distribution of macrophages across different tumour regions. Our results highlight the critical need for more spatio-temporal biological data to further refine our understanding of the dual role played by the immune system in cancer spreading.

CT1269 — Stability Analysis of a Multistage Compartmental Model of Infection

SPEAKERS Jacobs Somnic (Ghent University Global Campus (GUGC))
AUTHORS Jacobs Somnic (Ghent University Global Campus (GUGC)); Manvel Gasparyan (Ghent University Global Campus (GUGC)); Shodhan Rao (Ghent University Global Campus (GUGC))
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

This work explores the dynamics of a multistage SIRS model. Using the Lyapunov method as our primary tool, we begin by revisiting the stability analysis of the disease-free and endemic equilibria in a single-stage SIRS model. We then extend the techniques employed in this analysis to enable a similar stability analysis of multistage infection models. In particular, we propose a systematic approach to construct Lyapunov functions for multistage models using symbolic computation in MATLAB. We find that the structure of the constructed Lyapunov functions is closely related to the subsystems or pathways through which the disease progresses. The complete stability analysis for two- and three-stage infection models is presented, and we show that the equilibria are globally asymptotically stable when several parameter constraints are satisfied. Some of these mathematical constraints also provide meaningful epidemiological interpretations.

CT1618 — The Spatial Regime Conversion Method: An Adaptive Hybrid Framework for Multi-scale Reaction–Diffusion Systems

SPEAKERS Charlie Cameron (University of Bath)
AUTHORS Charlie Cameron (University of Bath)
SCHEDULE Wednesday, 15 July 2026; 11:50–12:10; 03.01 - SR
SESSION Contributed Talks
TRACK Miscellaneous

Abstract

Biological reaction–diffusion systems are inherently multiscale, with particle copy numbers varying substantially across the spatial domain. In regions of low concentration, discrete stochastic methods capture noise-driven fluctuations that may critically influence dynamics. In regions of high concentration, continuum PDE models provide an efficient description. Simulating such systems entirely with stochastic methods becomes prohibitively costly for large systems, motivating hybrid approaches. Existing hybrid frameworks partition the domain into stochastic and deterministic regions separated by a fixed spatial interface. This performs well when the interface is geometrically simple and known in advance, but fails when it is irregular, moving, or a priori unknown — as arises in Turing pattern formation. We present the Spatial Regime Conversion Method (SRCM), a hybrid framework that removes the need for an explicit interface entirely. Mass converts dynamically between discrete and continuous representations according to local concentration, automatically confining stochastic computation to regions where it is required. The method adapts as concentration profiles evolve, faithfully capturing stochastic-driven noise where it is dynamically significant while remaining efficient to run. We demonstrate the accuracy and efficiency of the SRCM on benchmark problems and apply it to pattern-forming systems in which stochastic-driven noise influences qualitative behaviour.

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CT250 — Data-driven dynamical analysis of an age-structured model: A graph-theoretic approach

SPEAKERS Anuraj Singh (ABV-Indian Institute of Information Technology and Management Gwalior)
AUTHORS Anuraj Singh (ABV-Indian Institute of Information Technology and Management Gwalior); Preeti Deolia (IIT Bombay)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

In this work, an age-stratified SEIR epidemiological model incorporating a saturated treatment function and heterogeneous contact rates is developed to study infectious disease transmission dynamics among various age groups. The expression for the basic reproduction number R_0 and conditions for the global stability of the system have been derived by a graph-theoretic (GT) approach. Digraph reduction creates a GT version of the Gauss elimination method for computing the R_0. The global dynamics results have been formed by constructing the Lyapunov function using a GT approach. The numerical simulations are demonstrated by extracting the daily reported COVID-19 cases for the second wave in Italy. The age-dependent contact matrix for the Republic of Italy (data sourced from the POLYMOD study) is computed via paper–diary methodology (PDM) grounded on a population-prospective survey in European countries. Our numerical findings imply that (i) for the age group (20–49) years and (50–69) years, the number of infected persons is quite double as compared with the exposed cases; (ii) approximately 50% of positive cases lies in (20–69) years age group; (iii) for the (00–19) years age group, only half of the exposed individuals got infected; and (iv) a consistent graph is detected for the age group of (70–99) years in both cases; it shows that almost all the exposed got infected.

CT1617 — Evaluation of Tissue Growth Models for Proliferating Cell Colonies

SPEAKERS Zuzanna Szymańska (ICM, University of Warsaw)
AUTHORS Jose Antonio Carrillo (Mathematical Institute, University of Oxford); Piotr Gwiazda (Institute of Mathematics, Polish Academy of Sciences); Maciej Marchwiany (ICM, University of Warsaw); Błażej Miasojedow (Faculty of Mathematics, Informatics and Mechanics, University of Warsaw); Zuzanna Szymańska (ICM, University of Warsaw)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Understanding and accurately describing cell proliferation is a crucial step toward realistic modelling of cancer invasion and progression. We propose a framework for parameter estimation and validation of tissue growth models describing the dynamics of proliferating cell colonies [Gwi+25]. By confronting model simulations with experimental data, we assess the ability of these models to reproduce key features of cancer cell proliferation. Particular attention is given to identifying the range of applicability of the models. We also highlight the importance of a rigorous and systematic approach to parameter estimation [Dęb+25]. Overall, our findings contribute to evaluating whether tissue growth models can provide a reliable foundation for more comprehensive descriptions of cancer progression and for the development of effective therapeutic strategies.

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CT1654 — Mathematical modeling of signal transduction and gene expression in cancer: from information processing to patient specific models

SPEAKERS Jana Wolf
AUTHORS Jana Wolf
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 11.34 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Cellular signaling networks generate dynamic responses that regulate how cells respond to external stimuli, perturbations or drugs. These responses can result in changes of cell fate, for examples, transitions from apoptosis to survival or from quiescence to proliferation. Mathematical modeling combined with targeted experimentation have successfully elucidated the underlying mechanisms and functional consequences for individual pathways, e.g. the role of feedbacks or cross-talk. The models and insights are now being used to create large-scale mechanism-informed models that can integrate various data sets and drive the development of disease- and patient-specific models. Here I introduce an approach to model aggressive B cell lymphoma based on detailed network model capturing key cellular processes and the mapping of perturbations data of a cohort of around 300 lymphoma patients. The resulting personalized patient models capture patient heterogeneity and are used to analyze how the genetic alterations together shape cancer cell states. A systematic study of individual and combinatorial alterations identifies known and so-far-unknown cooperation effects and elucidates a strong context dependency of individual network alterations.

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CT265 — Mathematical modelling reveals that liver architecture impacts tumour size distribution in mouse models of colorectal liver metastases.

SPEAKERS Xiaoyuan Liu (Cancer research UK Scotland Institute)
AUTHORS Sarah Williams (University of Glasgow); Xiao Fu (Cancer research UK Scotland Institute); Xiaoyuan Liu (Cancer research UK Scotland Institute)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Traditional mathematical models for tumour growth are built upon simple assumptions regarding tumour-intrinsic population dynamics, including logistic growth, diffusion volumetric scaling. With progresses in experimental cancer research combined with advanced phenotyping, insights have been gained into how microenvironmental factors shape tumour growth. This motivated the development of more complex mathematical models that account for the microenvironment and tissue architecture. Although these models capture a wider range of biological phenomena in a cancer-type-agnostic fashion, few were calibrated against temporally resolved experimental data for colorectal-liver metastases. Here, we present a two-dimensional PDE model that accounts for spatial biases in proliferation and metastatic seeding position to investigate how liver architecture impacts tumour growth dynamics. By calibrating the PDE model against experimental data, we found that spatially nonuniform proliferation was required to recapitulate the tumour size distribution. By simulating the PDE model across a range of the spatial bias parameters, we predicted how altering liver zonation impacts tumour size distributions, which was

validated in perturbation experiments. Our work reveals how liver architecture may shape tumour growth dynamics. In our ongoing work, were leveraging spatial transcriptomics to investigate molecular and cellular mechanisms underpinning tumour growth.

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CT1231 — Nonlinear Stability Analysis and Pattern Formation in Reaction-Diffusion-ODE Systems

SPEAKERS Finn Münnich (Heidelberg University)
AUTHORS Anna Marciniak-Czochra (Heidelberg University); Chris Kowall (Heidelberg University); Finn Münnich (Heidelberg University); Szymon Cygan (Heidelberg University); Théo André (Heidelberg University)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.33 - SR
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Reaction–diffusion–ODE systems arise in models of biological pattern formation, for example in symmetry breaking during regeneration in Hydra. These systems couple diffusive and non-diffusive nonlinear processes. In contrast to classical reaction–diffusion systems, such models may exhibit patterns with singularities, including jump discontinuities, which complicate the analysis of nonlinear stability. In this work, we develop a general framework for the stability analysis of steady states by establishing conditions for nonlinear stability and instability of bounded stationary solutions in reaction–diffusion–ODE systems. As an application, we derive conditions for diffusion-driven instability (DDI), a key mechanism underlying the emergence of Turing patterns in these systems. Finally, we illustrate the results with numerical simulations of example models.

CT1655 — Probing bacterial fitness in virtual chemostats

SPEAKERS Kaan Öcal (University of Melbourne)
AUTHORS Kaan Öcal (University of Melbourne); Lars Maas (University of Melbourne); Michael Stumpf (University of Melbourne)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

The growth rate of a bacterial population is a fundamental quantity in microbiology, but we lack the tools to predict it for realistic mathematical models. Based on an emerging connection between branching

processes and statistical physics, I present a new method to compute growth rates numerically in virtual "chemostats". These provide accurate estimates of growth rates and evolutionary fitness in quadratic time, bypassing the exponential complexity typically associated with growing populations. The underlying principle can be applied to design lineage-tracking experiments that measure bacterial fitness in vitro. I apply these results to two computational models that illustrate how bacteria optimally navigate their environment (chemotaxis) and investigate the role of noise in bacterial responses to antibiotics.

CT1495 — The Matrameru code of biomolecules – Enumerating unbranched fatty acids and amino acids

SPEAKERS Stefan Schuster (University of Jena)
AUTHORS Stefan Schuster (University of Jena)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 03.01 - SR
SESSION Contributed Talks
TRACK Miscellaneous

Abstract

In metabolomics, a fundamental question is how the potential number of molecular homologues (congeners) increases with their size. Here, we show that the number of unbranched fatty acids and unbranched aliphatic amino acids grows according to the famous Fibonacci numbers when cis/trans isomerism is neglected and adjacent double bonds are excluded [SFS17; FVS17]. This number series had been known in ancient India from Sanskrit prosody as Matrameru numbers. There is a striking mathematical correspondence between the verse structures of poems and bond structures in biomolecules. Moreover, we show that, under consideration of cis/trans isomerism, hydroxy and/or oxo groups, or triple bonds, diversity can be described by generalized Fibonacci (Matrameru) numbers, for example, Pell numbers [SFS17]. Finally, an enumeration procedure is presented for all possible cyclic secondary α-amino and imino acids (proline and its homologues), depending on ring size. A recursion formula is derived that results in a modified Lucas series [SM23]. Our results should be of interest for mass spectrometry, combinatorial chemistry, synthetic biology, patent applications, and the theory of evolution.

References

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CT1530 — Towards digital twins for sleep-wake timing

SPEAKERS Anne Skeldon (University of Surrey)
AUTHORS Anne Skeldon (University of Surrey); Derk-Jan Dijk (University of Surrey)
SCHEDULE Wednesday, 15 July 2026; 12:10–12:30; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Bright light soon after waking or blue-blocking glasses in the evening are standard therapies for sleep timing problems. These approaches are grounded in circadian principles which have established that the effect of light depends on the time of the biological clock with light in the biological morning speeding up

the biological clock, and light in the biological morning slowing down the biological clock. Challenges for application of these principles are that the time of the biological clock is not usually known, there are substantial differences between individuals and poor adherence.

Using established sleep-wake-light regulation principles, we have developed hierarchical coupled oscillator models with feedback that take light data as input, fit to individual sleep timing and duration data and predict circadian phase in healthy unemployed controls, people living with schizophrenia and healthy older adults. This quantitative approach highlights that sleep timing problems may be a consequence of aberrant self-selected light exposure patterns but also that individual differences in physiology matter.

We present a digital twinning approach in which light and sleep timing data are combined with mathematical models in close to real time, personalized to take account of individual differences in physiology. Behavioural nudges for appropriate light consumption could enable individuals to better align sleep and circadian rhythms to lifestyles.

CT230 — A computational study of physicochemical interactions in DCIS microinvasions

SPEAKERS Ana Forero Pinto (Moffitt Cancer Center/USF)
AUTHORS Ana Forero Pinto (Moffitt Cancer Center/USF); Emma Downey (Stony Brook University); Katarzyna Rejniak (Moffitt Cancer Center/USF); Mehdi Damaghi (Stony Brook University)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Microinvasions in ductal carcinoma in situ (DCIS) of breast cancer arise when malignant cells breach the basement membrane (BM) and invade the stroma. Since this is an early step in metastasis, identifying drivers of microinvasion may improve risk prediction and support treatment de-escalation. Mechanical tumor–stroma interactions, including extracellular matrix (ECM) collagen stiffening and deposition, are key contributors to the emergence of microinvasions, as is tumor acidity. We developed silicoDCIS, an off-lattice agent-based model of a DCIS cross-section that incorporates tumor growth, ECM fibril bundle properties, and acidic microenvironmental effects to study conditions leading to microinvasions. The model simulates tumor cell division, migration, and lactate secretion, and captures interactions between epithelial cells, BM, myoepithelium, fibroblasts, and collagen. The silicoDCIS model was parameterized to match the time points of DCIS development in HER2+ mouse experiments. We performed studies assessing mechanical contributions of DCIS components and the stroma to the emergence of microinvasion and how the proliferation-driven invasions can be prevented. We also integrated histology images to simulate spatial mechanical and metabolic interactions under acid stress to test whether acidosis-driven fibroblast reprogramming promotes stromal activation, ECM remodeling, and DCIS progression. This model can be used to generate new hypotheses of microinvasion development.

CT1429 — A Mathematical Model of Two-Stage Nanomedicine-Based Intraperitoneal Chemotherapy for Peritoneal Carcinomatosis

SPEAKERS Mohsen Rezaeian (Department of Applied Mathematics, University of Waterloo, Canada)
AUTHORS Wim Ceelen (Department of Human Structure and Repair, Ghent University, Ghent, Belgium); Biran Ingalls (Department of Applied Mathematics, University of Waterloo, Canada); Mohsen Rezaeian (Department of Applied Mathematics, University of Waterloo, Canada)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.34 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Intraperitoneal (IP) chemotherapy is a promising treatment for peritoneal carcinomatosis because it provides high local drug exposure to tumours within the abdominal cavity. However, its efficacy is limited by tumour microenvironmental barriers and rapid drug clearance from the peritoneal cavity. Nanosized drug carriers have attracted increasing interest because evidence suggests that they prolong peritoneal retention; however, their larger size also hinders tumour penetration. This trade-off creates a size dilemma in the design of nanocarriers. In addition, factors such as drug release rate and binding affinity may also contribute to improved therapeutic outcomes. Here, we present a mathematical model for a two-stage nanocarrier-based IP delivery system. Partial differential equations describe fluid flow and mass transport within the tumour and link nanoparticle retention, release kinetics, and drug binding affinity to the underlying transport mechanisms. Our results show that the two-stage system enhances tumour drug penetration and accumulation, consistent with experimental observations. The model also demonstrates a trade-off between retention time and tumour penetration across different nanoparticle sizes. In addition, it reveals that tuning the release rate and binding affinity can further improve overall treatment efficacy. This framework provides design insight for nanocarriers in IP chemotherapy and identifies when nanomedicine may improve treatment outcomes.

CT249 — A Robust Numerical Framework for Multi-strain Epidemic

SPEAKERS Fadilah Ilahi (The University of Manchester)
AUTHORS Fadilah Ilahi (The University of Manchester); Ian Hall (The University of Manchester); Thomas House (The University of Manchester)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Epidemiological models are often expressed through systems of differential equations of varying levels of complexity. In multi-strain models, complexity arises from the interaction of different serotypes as a result of cross-reactivity with the host's immune system. This study proposes a novel framework for representing multi-strain disease transmission models, with some mapping of sets and integers. By mapping infection histories into indicator matrices, it eliminates explicit iteration over strain combinations while preserving key biological processes, including sequential infection and partial cross-immunity. Benchmark comparisons of numerical solutions of the proposed matrix formulation and a direct set-indexed implementation demonstrate substantial performance gains. Across increasing strain dimensions, the matrix formulation consistently reduced runtime, with speed-up exceeding two orders of magnitude for systems with 11 strains. We will present an application of this approach to the case of COVID-19 in Indonesia, focusing on the replacement of the Delta variant by Omicron. The matrix formulation approach represents a robust and powerful method to automatically generate large multi-strain epidemic models that can be solved in a computationally efficient manner.

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CT1384 — Anticipating epidemic waves in non-stationary disease dynamics

SPEAKERS Joshua Looker (University of Warwick)
AUTHORS Joshua Looker (University of Warwick); Kat Rock (University of Warwick); Louise Dyson (University of Warwick)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

In the wake of the SARS-CoV-2 pandemic, there has been increased interest in mathematical tools that can anticipate changes in infectious disease dynamics from surveillance data. Early warning signals based on critical slowing down have been widely proposed as indicators of tipping points such as disease elimination or resurgence. However, many epidemiological systems are non-stationary and may exhibit transient increases in fluctuations even in the absence of changes in the basic reproduction number.

In this work, we show how critical-like signals can arise from the geometry of stochastic dynamics when the underlying deterministic system is non-normal. We demonstrate that increases in fluctuation variance and recovery time can occur without a change in asymptotic stability. Using a general stochastic framework for fluctuations around mean-field dynamics, we derive conditions under which such behaviour emerges.

As an illustrative example, we analyse the susceptible–infectious–recovered (SIR) model and show how these signals can precede epidemic waves. Our results suggest that critical-like signals may provide useful indicators of impending infection waves in many infectious disease models.

CT1318 — Clean seed use in seasonal agroecosystems: How growers behave shapes plant epidemic dynamics

SPEAKERS Ludovic Mailleret (Université Côte d'Azur, INRAE, ISA, France)
AUTHORS Arindam Mandal (Université Côte d'Azur, INRAE, ISA, France); Frédéric Grognard (Université Côte d'Azur, Inria, INRAE, MACBES, France); Frédéric Hamelin (IGEPP, INRAE, Institut Agro, Université de Rennes, Rennes, France); Ludovic Mailleret (Université Côte d'Azur, INRAE, ISA, France)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

In seasonal agroecosystems, diseases persist through both horizontal transmission during the growing season and vertical transmission via farmer-saved seeds. To break this cycle, growers can purchase and plant disease-free seeds, so called clean seeds. Their choice is then between getting such seeds or reusing cost-free, potentially infected farmer seeds. We investigate the combined dynamics of plant epidemics and grower behavior using a semi-discrete model that couples a metapopulation epidemic framework with game-theoretic imitation dynamics. We show that the equilibrium proportion of growers adopting clean seeds depends critically on the seed-sharing structure, with regimes ranging from polarized bang-bang to mixed strategies as functions of clean seed cost and disease characteristics. When clean seed users do not contribute to the farmer seed pool, small increases in seed price can trigger catastrophic shifts toward widespread infection. Conversely, when seed-sharing creates a common pool, clean seeds provide community-wide disease dilution, initiating a free-riding dilemma: growers may avoid costs while benefiting from the reduced prevalence maintained by others. Finally, we consider not-so-clean seeds, showing that free-riding disappears because of spill-over of the disease they carry to the farmer seed users. When appropriately priced, not-so-clean seeds help stabilize prevalence at manageable levels by balancing the payoff between costly control and epidemic risk.

CT1656 — Mean first passage time as a framework for immune cell search and encounter

SPEAKERS Fatemeh Saghafifar (PhD, UBC)
AUTHORS Fatemeh Saghafifar (PhD, UBC)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 03.01 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Mean first passage time is a fundamental quantity for characterizing search and encounter processes, since it measures the expected time required for a moving agent to first reach a target. In the context of immune cell dynamics, this provides a natural way to connect cell motility to functional outcomes such as target finding and cytotoxic efficiency. For natural killer cells and other immune cells, experimental trajectory data suggest that motion is not purely diffusive, but often exhibits persistence and directional bias, making classical diffusion-based first passage results insufficient for mechanistic interpretation.

This work develops a mathematical framework for studying mean first passage times in persistent random walk models motivated by immune cell migration. The goal is to understand how features such as directional persistence, environmental bias, and spatial geometry shape encounter statistics and influence search efficiency. By linking microscopic movement rules to macroscopic first passage behaviour, the framework aims to provide interpretable quantities that can help distinguish different search strategies and clarify which motility features are most relevant for effective immune surveillance. More broadly, the work highlights mean first passage time as a useful bridge between cell trajectory data, stochastic transport models, and biologically meaningful measures of function.

CT1493 — Mechanistic Bayesian Mixture Modeling of Infection Establishment and Proliferation

SPEAKERS Bruce Rogers (Duke University)
AUTHORS Bruce Rogers (Duke University); Janice McCarthy (Duke University); Linh Do (Duke University)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 11.34 - SR
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Infectious disease models are common at both the cellular scale to describe invasion probabilities and the tissue scale to describe pathogen replication dynamics once an infection has been established. We develop Bayesian methods that link the infection establishment with mechanistic target cell ODE models of viral replication. First, given longitudinal measurements of hosts' viral load, we estimate ODE parameters using Approximate Bayesian Computation. Next, we develop a statistical model where infection status (positive or negative) is modeled as a latent variable, and observations of the viral load are described assuming a censored lognormal distribution. Then, the statistical model is fit using a Hybrid Expectation-Maximization algorithm. We apply the model to influenza human-subject challenge studies where i) the time of infection is known and ii) multiple baseline immune system measures are collected and discover protective thresholds for immunity against infection.

CT1552 — Modeling the spatio-temporal effects of broad-beam and mini-beam irradiation on a cell population

SPEAKERS Mathilde Badoual (CNRS IN2P3/Université Paris Cité)
AUTHORS Basile Grammaticos (CNRS/IN2P3); Delphine Crépin (CNRS IN2P3); Marianne Billoir (CNRS IN2P3/Université Paris Cité); Mathilde Badoual (CNRS IN2P3/Université Paris Cité); Olivier Seksek (CNRS/IN2P3); Stéphane Plaszczynski (CNRS IN2P3)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.33 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

More than 50% of cancer patients undergo radiotherapy. Nowadays, treatment plannings are based on the linear-quadratic model, that predicts the survival fraction of cancer cells but does not include any temporal component. Moreover, it is usually not valide for large doses (that are used in new radiotherapy modalities such as mini-beam). It is therefore of the utmost importance to develop models that can describe the

temporal response to irradiation for a large range of doses. In this work we combine time-lapse fluorescence microscopy experiments (to follow the cell density of F98 glioma cells under varying radiation doses and initial cell densities) and mathematical modeling, in order to characterize the temporal response of a cancerous cell population to single-dose radiation. The model (based on ordinary differential equations) reproduces very well all the experimental data, with only three free parameters (and four others that are fixed). It allows us to have access and to follow the evolution of different cell populations after irradiation, in particular, the senescent and repaired cell populations. Therefore, surviving fractions could also be estimated. Our model allows us to analyze and quantify an inhibition effect (or cohort effect) of the dead and senescent cell populations on the regrowth of the repaired one [Bil+25]. With the same model, we are also able to model mini-beam experiments with a spatially-structured irradiation.

References

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CT1500 — Modelling mass asymptomatic testing strategies for early containment of infectious disease outbreaks in prisons

SPEAKERS Lorenzo Pellis (The University of Manchester)
AUTHORS Joseph Brooks (The University of Manchester); Francesca Scarabel (University of Leeds); Ian Hall (The University of Manchester); Jingsi Xu (The University of Manchester); Pauline Bakker (National Institute for Public Health and the Environment, The Netherlands); James Adamson (Public Health Wales); Rupert Bailie; Rachel Campbell (UK Health Security Agency); Nicola Dennis (UK Health Security Agency); Lianne Straus (UK Health Security Agency); Steve Willner (UK Health Security Agency); Jonathan Van der Veen (UK Department of Justice); Chantal Edge (UK Health Security Agency); Tom Fowler (Public Health Wales); Lorenzo Pellis (The University of Manchester)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Prisons present a unique environment for the spread of infectious diseases such as influenza, tuberculosis, and SARS-CoV-2, as prison residents are generally confined in close proximity of each other, with regulated movements and interaction with staff often physical in nature. Although logistically difficult to manage, non-pharmaceutical interventions have been used during the COVID-19 pandemic. Inspired by this, I will investigate a potential intervention that has not been used before, namely asymptomatic testing and isolation of the entire prison population ("pulse testing") early on, to interrupt an outbreak before it becomes large. I will compare it with the more common approach of isolating cases based on symptoms, for a range of pathogen and implementation scenarios. Symptom-based isolation is relatively easy to implement even with minimal public health support, but requires substantial isolation facilities and its effectiveness quickly degrades with a growing fraction of asymptomatic cases. Instead, pulse testing can be effective even if most cases are asymptomatic and requires isolating substantially fewer individuals. However, it presents unique implementation challenges, so to make it effective logistic support would need to be in place to allow prisons to declare an outbreak as soon as possible, initiate multiple rounds of testing immediately and secure high levels of adherence, as any delay or partial implementation would quickly render the policy ineffective.

CT1643 — Personalizing the Patient Model for Depth-of-Hypnosis Control

SPEAKERS Gorazd Karer (University of Ljubljana, Faculty of Electrical Engineering)
AUTHORS Gorazd Karer (University of Ljubljana, Faculty of Electrical Engineering)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.21 - SZ

SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

One of the anesthesiologist's key tasks during target controlled infusion (TCI) in total intravenous anesthesia (TIVA), where agents such as propofol are administered intravenously, is assessing the depth of hypnosis (DoH). As DoH cannot be measured directly, clinicians rely on indirect indicators such as the Bispectral Index (BIS), derived from EEG signals. However, pharmacokinetic/pharmacodynamic (PK/PD) models implemented in perfusors are population-based and often fail to capture interpatient variability, leading to inaccurate DoH estimation.

To address this limitation, we propose a modelling framework that augments a population-based PK/PD model with a patient-specific residual dynamic component. This component is formulated as a low-order autoregressive model with exogenous input and captures discrepancies between predicted and measured BIS values using real-time clinical data. Model parameters are identified via recursive least-squares with exponential forgetting, enabling online adaptation during surgery while considering the clinically validated population-based model.

Validation with clinical data demonstrates improved BIS prediction accuracy and reduced error, reflecting individual sensitivity to anesthetics. The proposed framework enables personalized, data-driven modelling suitable for closed-loop DoH control, supporting more precise drug delivery, reduced clinician workload, and enhanced patient safety.

CT222 — Quantitative modeling of cytochrome P450 gene expression regulated by pregnane X nuclear receptor

SPEAKERS Veronika Bernhauerová (Charles University)
AUTHORS Bechara Saade; Ellen Tanaka Kahiya; Jurjen Duintjer Tebbens; Lucie Smutná; Lukáš Lochman; Petr Pávek; Tomáš Smutný; Veronika Bernhauerová (Charles University)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

The pregnane X receptor (PXR) is a ligand-activated transcription factor that is highly concentrated in the liver. It is considered a crucial sensor in the xenobiotic response. PXR regulates the transcription of genes involved in drug metabolism, including cytochrome P450 (CYP) enzymes such as CYP3A4, CYP2C9, and CYP2B6. For example, CYP3A4 metabolizes approximately half of all clinically used drugs. Recently, three-dimensional (3D) primary human hepatocyte (PHH) spheroids have emerged as an *in vitro* model that resembles *in vivo* hepatocytes in terms of phenotype and function. This model can be used to study the long-term regulation of drug-handling genes by PXR [Smu+22]. We proposed a mathematical model that describes PXR activation and the expression dynamics of select PXR-regulated target genes [Loc+25]. We calibrated this model using long-term target gene messenger RNA (mRNA) expression data from a state-of-the-art *in vitro* system of 3D PHH spheroids. We examined the impact of two PXR activators — a model PXR ligand (rifampicin) and its primary metabolite (25-desacetyl rifampicin) — on CYP mRNA expression [Kah+26]. Finally, we discuss the experimental validation of the theoretical results.

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CT1475 — Quantitative parameter free methods for toxicokinetics

SPEAKERS Inzish Sajid (University of Reading, United Kingdom)
AUTHORS Inzish Sajid (University of Reading, United Kingdom); Marcus Tindall (University of Reading); Courtney Thompson (Syngenta, Jealott's Hill International Research Center); Joseph Leedale (Syngenta, Jealott's Hill International Research Center); Peter Sweby (University of Reading); Steven Webb (Syngenta, Jealott's Hill International Research Center); Zuowei Wang (University of Reading)
SCHEDULE Thursday, 16 July 2026; 10:40–11:00; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Pharmacology and toxicology studies often use compartmental ordinary differential equation (ODE) mathematical models to understand how a chemical compound distributes and exerts its effects throughout the body. Such models require experimental data to inform parameterisation. However, such data is often costly and not always available and thus we need to identify new ways to parameterise such ODE models under data-limited conditions - the aim of this work is to address this challenge. In this talk, I will explain a novel methodology that can be effectively used to parameterise biologically based ODE models. It involves a stepwise approach, including: generating stochastic Petri net (SPN) qualitative data; scaling the qualitative SPN data into quantitative synthetic data; and using this synthetic data to obtain model parameters and their associated distributions. I will demonstrate this methodology against case examples to illustrate its feasibility and reliability, including a model for the regulation of thyroid hormone levels in the brain. I will also describe troubleshooting methods to improve accuracy of this approach, including: varying the number of initial Petri Net tokens; deploying a scaling approach; and how to flex according to the nature of the experimental data provided. Through this exploration, I will illustrate how you can enhance the robustness and precision of this methodology when applied to more complex systems.

CT1603 — A spatiotemportal model for prostate cancer

SPEAKERS Alethea Barbaro (TU Delft)
AUTHORS Alethea Barbaro (TU Delft); Daniel Balague Guardia (TU Delft); Kateřina Staňková (Delft University of Technology); Rosalie Althuis (World Vision Nederland)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 03.01 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Cancer affects a countless number of lives across the world each day, and mathematical oncology offers a tool to simulate cancer and potentially improve its treatment. One of the current challenges is the development of resistant populations in response to treatment. In this talk, we present an agent-based model for treatment-resistant prostate cancer, with the aim of gaining insights into the dynamics among different populations of tumor cells in response to varying testosterone levels and different treatment strategies. We present a spatial agent-based model on a discrete lattice, simulating the interactions between testosterone-dependent and testosterone-independent tumor cells. We identify a possible phase

transition in the testosterone level in the bloodstream, which could influence which tumor cells dominates the grid. We derive a continuum limit of the discrete model, leading to partial differential equations that describe the tumor's spatial behavior, allowing us to gain deeper insights into the dynamics of the model. The flexibility of our model allows for its application to other hormonal cancers, and our findings support the promising potential of hormonal manipulation in controlling tumor growth and composition, especially extinction therapy. The mathematical analysis together with simulations provide unique insight into tumor dynamics.

CT229 — Cell Trajectory Inference based on Schrödinger Problem and a Mechanistic Model of Stochastic Gene Expression

SPEAKERS Clémence Fournié
AUTHORS Aymeric Baradat (Univ Lyon, Université Lyon 1, CNRS UMR5208, Institut Camille Jordan, 43 Blvd du 11 Novembre 1918, F-69622 Villeurbanne, France); Clémence Fournié; Elias Ventre (INRIA Centre d'Université Côte d'Azur); Fabien Crauste (Université Paris Cité, CNRS, MAP5, F-75006 Paris, France); Olivier Gandrillon (Univ Lyon, ENS de Lyon, Univ Claude Bernard, CNRS UMR 5239, INSERM U1210, Laboratory of Biology and Modelling of the Cell, 46 allée d'Italie Site Jacques Monod, F-69007 Lyon, France, Inria, Lyon, France); Ulysse Herbach (Université de Lorraine, CNRS, Inria, IECL, F-54000 Nancy, France)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Cellular differentiation is the biological process that leads a cell to opt for a particular cellular identity. Recently, single-cell RNA-sequencing has enabled the simultaneous measurement of gene expression levels at specific times for a large number of individual cells and a large number of genes. Repeating such measurements at different time points gives then access to the temporal variation of a distribution on a gene expression space. The whole temporal trajectory of distributions thus characterizes the differentiation process at population level. The optimal transport theory that has been used so far to infer cellular differentiation trajectories from time-stamped single-cell RNA-seq data involves solving the so-called Schrödinger problem in its most common version. This implies assuming that cells move, in the gene expression space, by diffusion. Yet, real gene dynamics are much more complex. In the present work, we assume that mRNA dynamics are characterized by brief and important production of RNA, with long periods of inactivity in between, and consider the so-called Bursty model of gene dynamics. We use this model to define a reference process for the Schrödinger problem. By comparing the solutions of the Schrödinger problems with a Diffusive and a Bursty reference process, under different conditions, we show that the Bursty model provides a better approximation of the underlying gene dynamics than the standard Diffusive process when inferring cell trajectories.

CT1389 — Forecasting SFTS risk in the Republic of Korea under population aging and climate change using machine learning

SPEAKERS Yongin Choi (Research Institute of Applied Statistics, Sungkyunkwan University, Republic of Korea)
AUTHORS Yongin Choi (Research Institute of Applied Statistics, Sungkyunkwan University, Republic of Korea); Giphil Cho (Department of Electronic and AI System Engineering, Kangwon National University, Republic of Korea); Min Seok Kim (National Institute for Mathematical Sciences, Republic of Korea); Hyojung Lee (Department of Statistics, Kyungpook National University, Republic of Korea)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Severe fever with thrombocytopenia syndrome (SFTS) is a tick-borne viral disease primarily reported in East Asia. In the Republic of Korea, the disease burden is concentrated among older adults, who experience

higher incidence and case fatality rates. Meanwhile, Korea is undergoing demographic aging alongside environmental changes associated with climate change. These shifts raise important questions about future SFTS risk. In this study, we developed long-term predictive models to evaluate how population aging and climate change may influence future SFTS incidence. Using nationwide epidemiological surveillance data with meteorological, demographic, and behavioral information, we predicted both tick density and SFTS incidence among individuals aged 60 years and older using machine learning models, including linear regression, XGBoost, and deep neural networks. The best-performing model was then used to generate long-term predictions of SFTS incidence under future demographic projections and climate change scenarios. Model predictions suggest that SFTS incidence among older adults in Korea will increase in the coming decades, with larger increases under high-emission scenarios. The results indicate that demographic aging and climate change may act together to amplify future SFTS risk in elderly populations. These findings emphasize the need to consider both demographic and environmental factors when developing strategies for vector-borne disease preparedness and control.

CT115 — Mathematical Modeling of Biofilm Eradication Using Optimal Control of Antibiotic Treatment

SPEAKERS Rehan Akber (Lahore University of Management Sciences (LUMS))
AUTHORS Adnan Khan (Lahore University of Management Sciences (LUMS), Pakistan); Rehan Akber (Lahore University of Management Sciences (LUMS))
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

We propose and analyze a model for antibiotic resistance transfer in a bacterial biofilm and examine antibiotic dosing strategies that are effective in bacterial elimination. In particular, we consider a 1-D model of a biofilm with susceptible, persistor and resistant bacteria. Resistance can be transferred to the susceptible bacteria via horizontal gene transfer (HGT), specifically via conjugation. We analyze some basic properties of the model, determine the conditions for existence of disinfection and coexistence states, including boundary equilibria and their stability. Numerical simulations are performed to explore different modeling scenarios and support our theoretical findings. Different antibiotic dosing strategies are then studied, starting with a continuous dosing; here we note that high doses of antibiotic are needed for bacterial elimination. We then consider periodic dosing, and again observe that insufficient levels of antibiotic per dose may lead to treatment failure. Finally, using an extended version of Pontryagin's maximum principle we determine efficient antibiotic dosing protocols, which ensure bacterial elimination while keeping the total dosing low; we note that this involves a tapered dosing which reinforces results presented in other clinical and modeling studies. We study the optimal dosing for different parameter values and note that the optimal dosing schedule is qualitatively robust.

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CT1539 — Modelling community and healthcare worker transmission using a two-population SEIS Framework informed by ONS survey

SPEAKERS jingsi xu (University of Manchester)
AUTHORS Daniela De Angelis (University of Cambridge); Ian Hall (University of Manchester); Paul Birrell (UKHSA); Sarah Walker (University of Oxford); Thomas House (University of Manchester); jingsi xu (University of Manchester)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Throughout the SARS-CoV-2 pandemic, healthcare workers (HCWs) played a critical role in the response and faced elevated risk of infection. This work investigates the transmission and interaction between the general community and HCWs, aiming to understand how COVID-19 transmission flows between these two populations.

The COVID-19 Infection Survey run by the Office of National Statistics, (ONS-CIS), provides prevalence data in the general community and HCWs in the UK. The ONS-CIS ran from 26 April 2020 to 13 March 2023 and included 14,001 HCWs and 509,127 participants from the general community. We use a generalised additive model (GAM) to estimate prevalence trends from ONS-CIS data, and incorporate these estimates into the computation of time-varying force of infection experienced by each subpopulation, as well as estimating effective and control reproduction numbers. We then explore scenarios to investigate the impact of major policies and events such as national lockdowns, and vaccine rollout in the UK, and evaluate other potential interventions.

Our results show that HCW infection risk was strongly coupled to community transmission. Targeted protection of HCWs alone was therefore unlikely to be sufficient in the absence of broader community control. These findings highlight the need for integrated strategies combining HCW-focused protection, vaccination, and timely community-level interventions to maintain a functioning healthcare system.

CT231 — Optimization of melanoma treatment with cocktails of CAR-T cells of different affinities

SPEAKERS Emilie Favier (Moffitt Cancer Center/University of South Florida)
AUTHORS Emilie Favier (Moffitt Cancer Center/University of South Florida); Jose A. Guevara-Patiño (Moffitt Cancer Center); Katarzyna A. Rejniak (Moffitt Cancer Center/University of South Florida); Lourdes B. Plaza-Rojas (Moffitt Cancer Center)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

The efficacy of CAR-T cell treatment is heavily impacted by low levels of oxygen (hypoxia) in solid tumors. However, CAR-T cells with different affinities show distinct behavior and functionality in hypoxic areas. Thus, we could optimize the composition of CAR-T cells cocktails of different affinities to maximize their efficacy in the heterogeneous microenvironment of melanoma. To do so, we developed a pipeline to digitize tissue images, simulate oxygen distribution, and create a tissue hypoxia map. We then built a hybrid agent-based model that describes the dynamics of cancer cells, vessels, and CAR-T cells within the tissue, including CAR-T cell division, cytotoxicity (depending on the scFv affinity), and exhaustion. This was combined with kinetics of tissue oxygenation, chemokine secretion patterns, and their microenvironmental distribution. Using these outcomes as a computational domain for our model, we studied how different chemokine secretion patterns impact the travelled distance by CAR-T cells, their killing efficacy, and exhaustion. This suggests that the creation of chemokine gradients can improve CAR-T cell efficacy. Finally, because CARs of different affinities have different properties when exposed to lack of oxygen, we identified the optimal mixtures of CARs of specific affinities that together better target tumor cells in

hypoxic areas, resulting in better anti-tumor control in melanoma. This model can generate experimentally testable hypotheses.

CT1538 — Pathogen non-planktonic phases within the urinary tract impact early infection and resistance evolution

SPEAKERS Amanda de Azevedo-Lopes (Max Planck Institute for Evolutionary Biology)
AUTHORS Michael Raatz (Max Planck Institute for Evolutionary Biology); Amanda de Azevedo-Lopes (Max Planck Institute for Evolutionary Biology); Karolina Drabik (Dioscuri Centre for Physics and Chemistry of Bacteria); Arne Traulsen (Max Planck Institute for Evolutionary Biology); Bartłomiej Wacław (Dioscuri Centre for Physics and Chemistry of Bacteria, University of Edinburgh)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 11.34 - SR
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Treatment of urinary tract infections (UTIs) and the prevention of their recurrence is a pressing global health problem. In a UTI, pathogenic bacteria not only reside in the bladder lumen but also attach to and invade the bladder tissue. Planktonic, attached, and intracellular bacteria face different selection pressures from physiological processes such as micturition, immune response, and antibiotic treatment. We use a mathematical model of the initial phase of infection to unravel the effects of these different selective pressures on the ecological and evolutionary dynamics of UTIs. We explicitly model free-floating pathogenic bacteria in the bladder lumen, bacteria attached to the bladder wall, and bacteria that have invaded the epithelial cells of the bladder. We find that the presence of non-planktonic bacteria substantially increases the risk of infection establishment and affects evolutionary trajectories leading to resistance during antibiotic treatment. We also show that competitive inoculation with a fast-growing non-pathogenic strain can reduce the pathogen load and increase the efficacy of an antibiotic, but only if the antibiotic is used in moderation. Our study shows that including different compartments is essential to create more realistic models of UTIs, which may help guide new treatment strategies.

CT1494 — ROSA: A Geometric Spectral Lift for Detecting Pre-Reorganisation Signals in Noisy Spatial Graphs

SPEAKERS Ben Cardoen (University of Birmingham)
AUTHORS Ben Cardoen (University of Birmingham); Fabian Spill (University of Birmingham)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Spatial biological networks derived from microscopy are commonly represented as embedded graphs whose topology reflects underlying organisation. Subtle precursor events often precede visible structural reorganisation: for example, branch migration preceding mitochondrial fission, local neurite remodelling in dendritic arbors, or early junction displacement in microvascular networks. Detecting these weak structural signals is challenging because vertex localisation uncertainty and geometric noise dilute graph-distance comparisons as network size increases. We introduce ROSA, a geometric spectral lift that transforms a base graph distance into an integrated distance along a filtration that removes edges according to spectral sensitivity. By accumulating metric variation along this path, ROSA amplifies localised structural edits that would otherwise be obscured by noise or scale. We prove that ROSA defines a metric under mild conditions and derive an anti-dilution inequality showing that, for localised perturbations on growing graphs, ROSA retains strictly stronger signal than the base distance. Symmetric geometries provide explicit regimes where amplification cannot occur. Experiments on spatial graph models and imaging-derived networks—including mitochondrial and vascular networks—demonstrate improved recovery of

pre-reorganisation signals under heavy vertex noise when ROSA is applied to spectral and diffusion-based graph distances.

CT1326 — Spatially explicit individual-based model for schistosome transmission: A bipartite network approach

SPEAKERS Melissa A. Iacovidou (University of Oxford)
AUTHORS Betty Nabatte (Uganda Ministry of Health); Narcis B. Kabatereine (Uganda Ministry of Health); Goylette F. Chami (University of Oxford); Melissa A. Iacovidou (University of Oxford); Max M. Lang (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Schistosomiasis is driven by complex transmission dynamics influenced by human behaviour and environmental factors. While existing individual-based models (IBMs) have advanced our understanding of the disease, they often lack a spatial component, limiting their ability to inform geographically targeted interventions. In this work, we present a spatially explicit IBM to better simulate the transmission dynamics of *Schistosoma mansoni* in our study area in rural Uganda. Our approach integrates geospatial data on household and water site locations to construct a bipartite network. This network forms the foundation for our model, explicitly linking humans to water sites. *Biomphalaria* snail dynamics are also explicitly modelled at the site level. By doing so, our model can simulate how factors like human mobility and the spatial arrangement of water sites and households affect the spread of infection. This model is a powerful tool for evaluating the efficacy of different control strategies. We use it to explore how targeted interventions, such as snail control at high-risk water sites or removing access to high-risk water sites altogether, compare to untargeted strategies. Ultimately, this work aims to demonstrate the importance of spatial modelling in identifying water sites of higher risk and optimising resource allocation for more effective control programmes.

CT1623 — Spectral EEG differences using Eleveld and Schnider propofol PK/PD models

SPEAKERS Duygu Aydin (TU Munich)
AUTHORS Antonio Farnia (Treviso Regional Hospital); Duygu Aydin (TU Munich); Federico Linassi (Treviso Regional Hospital, University of Padova); Gerhard Schneider (TU Munich); Leonardo Spano (University of Padova); Malin Frärks (TU Munich); Matthias Kreuzer (TU Munich); Michele Carron (University of Padova); Paolo Burelli (Treviso Regional Hospital); Paolo Zanatta (Treviso Regional Hospital)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.21 - SZ
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Background: Several PK/PD models are used for propofol delivery via target-controlled infusion (TCI). The Schnider PK model [Sch+98] was developed in younger healthy volunteers. The Eleveld PK/PD model [Ele+18] is a newer general-purpose model for children, elderly, and obese patients. The models differ in effect-site equilibrium rates, with Schnider assuming higher values ($k_{e0} = 0.456$ vs 0.146/min).

Methods: We compared effects of these models on brain oscillatory activity as measured by frontal electroencephalogram (EEG) during anesthesia induction. We calculated EEG power spectra over time (=from first propofol administration to incision, time normalized to 100%) using Schnider (n=36) and Eleveld (n=36) models. Differences were statistically tested using 10k-bootstrapped AUROC values with 95% confidence intervals as a measure of effect size [HS11].

Results: Although age, BMI, and effect-site concentration at loss of responsiveness did not differ significantly between groups, the Eleveld group showed significant decreases in beta and low-gamma frequency bands. The decrease was pronounced in the first half of induction with accompanying increases in alpha frequency band.

Conclusion: Anesthesia induction with Schnider and Eleveld PK/PD models may lead to different dynamics at the effect site as measured by the EEG, although future investigation should consider continuous CeP values.

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CT1557 — The Impact of Programmed Cell Death on Cell Population Dynamics: A Stochastic and Moment Dynamics Approach

SPEAKERS Peter Boldog (WIGNER Research Centre for Physics)
AUTHORS Márk Veres (Bolyai Institute, University of Szeged); Peter Boldog (WIGNER Research Centre for Physics)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.33 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Classical mean-field models of cell population dynamics, including cancer progression, often rely on the "well-mixed" assumption and neglect the role of cell death. In this work, we demonstrate how these simplifications lead to a significant overestimation of growth rates and saturation levels, with critical implications for tumor therapy design.

First, we present a stochastic modeling framework comparing mean-field approximations with Agent-Based Models (ABM) to investigate the "Ghost Effect" —a phenomenon where apoptotic cells do not vanish instantly but act as static barriers. These structures preserve spatial "memories" of former clusters, persistently inhibiting the motility and proliferation of living cells. Our results show that spatial correlations and volume exclusion consistently predict a lower carrying capacity than classical models.

Then, to bridge the gap between computationally expensive simulations and overly simplistic ODEs, we use a Moment Dynamics approach. By tracking higher-order moments, we derive a system of coupled ODEs that captures crowding effects and correlation-induced inhibition at a fraction of the computational cost.

Finally, we discuss applications in clinical oncology, highlighting how realistic modeling can help determine minimal effective chemotherapy doses by accounting for self-crowding.

CT254 — Travelling waves in a mathematical model of fireblight during bloom season

SPEAKERS Hermann Eberl (University of Guelph)
AUTHORS Hermann Eberl (University of Guelph)
SCHEDULE Thursday, 16 July 2026; 11:00–11:20; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Fireblight is a bacterial disease of apple and pear trees that can wipe out an entire orchard in a single growing season. We formulate a mathematical model for the spread of this disease during bloom. The resulting model consists of a system of two semilinear PDEs for the pathogen, coupled to three ODEs describing the infection dynamics. Numerical simulations suggest the existence of travelling waves that we then set out to prove. We will discuss how the travelling waves analysis gives insight into disease propagation that can be useful for disease control, management and prevention. Our analytical results, are suboptimal in the sense that the necessary conditions for TW that we find are not also necessary.

CT1395 — Assessing Birth Month Dependent Effectiveness of Monoclonal Antibodies against Infant RSV Hospitalization: A Modelling Study.

SPEAKERS Beryl Musundi (Martin Luther University Halle-Wittenberg, Germany)
AUTHORS Julian Patzner (Martin Luther University Halle-Wittenberg, Germany); Aleksandr Bryzgalov (Martin Luther University Halle-Wittenberg, Germany); Tyll Krueger (Wroclaw University of Science and Technology, Poland); Harry Zaunstöck (Martin Luther University Halle-Wittenberg, Germany); Rafael Mikolajcyzk (Martin Luther University Halle-Wittenberg, Germany); on behalf of ADAPTI-M; Beryl Musundi (Martin Luther University Halle-Wittenberg, Germany)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Respiratory syncytial virus (RSV) is a leading cause of hospitalization among infants. An infant's birth month relative to the RSV season is an important determinant of hospitalization risk, with infants born shortly before peak seasonal activity often experiencing the highest burden. Although existing epidemiological studies largely describe these patterns, mechanistic evaluation of how birth timing interacts with transmission dynamics and emerging immunization strategies remains limited. Recent epidemiological observations suggest shifts in RSV seasonality, following the COVID-19 pandemic, with early seasonal onset in several European countries. Concurrently, Germany has introduced extended half-life monoclonal antibodies to protect newborns against severe RSV, though their effectiveness may depend on birth timing and epidemic variability. We use the German Epidemic Microsimulation System (GEMS), an agent-based modelling framework, to simulate RSV transmission among infants while explicitly representing birth month cohorts, age and contact dependent susceptibility, waning immunity, and seasonal transmission dynamics. We simulate exposure trajectories and evaluate weekly RSV hospitalizations under varying monoclonal antibody coverage and epidemic timing scenarios. We quantify how birth month and seasonal dynamics influence the effectiveness and timing of monoclonal antibody interventions and provide mechanistic insight into observed seasonal risk patterns.

CT1334 — Estimating fitness effects of mutations that evolve in a Long-Term Evolution Experiment of HIV-1

SPEAKERS Jenny Dunstan (Institute of Integrative Biology, ETH Zurich, Zurich, Switzerland)

AUTHORS Ali Movasati (Division of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, Zurich, Switzerland, Institute of Medical Virology, University of Zurich, Zurich, Switzerland); Christine Leemann (Division of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, Zurich, Switzerland, Institute of Medical Virology, University of Zurich, Zurich, Switzerland); Christoph Liechti (Division of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, Zurich, Switzerland, Institute of Medical Virology, University of Zurich, Zurich, Switzerland); Jenny Dunstan (Institute of Integrative Biology, ETH Zurich, Zurich, Switzerland); Karin Metzner (Division of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, Zurich, Switzerland, Institute of Medical Virology, University of Zurich, Zurich, Switzerland); Kathrin Neumann (Division of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, Zurich, Switzerland, Institute of Medical Virology, University of Zurich, Zurich, Switzerland); Roland R Regoes (Division of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, Zurich, Switzerland, Institute of Medical Virology, University of Zurich, Zurich, Switzerland); Rongfeng Chen (Division of Infectious Diseases and Hospital Epidemiology, University Hospital Zurich, Zurich, Switzerland, Institute of Medical Virology, University of Zurich, Zurich, Switzerland); Sneha Sundar (Institute of Integrative Biology, ETH Zurich, Zurich, Switzerland)

SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Quantitative estimates of fitness, particularly of individual genetic mutations, are crucial to predict the evolutionary dynamics of biological systems. Estimating fitness is challenging due to linkage disequilibrium of mutations on the same genome as well as clonal interference between competing genotypes. We estimate the fitness effects of individual mutations that are observed in a long-term HIV-1 evolution experiment, in which we evolved four evolutionary lines of HIV-1 for around 1500 generations, or almost 5 years, and sequenced the population using a high-throughput method every 10 transfers. We used MPL to infer mutational fitness effects, which takes into account genetic linkage and clonal interference. We compared the fitness effects to the results of competition assays against the ancestor virus population with which we determined the viral population fitness every 100 transfers. Combining the mutational fitness estimates to predict population fitness over time, we find good agreement with experimental results. In two of the lines, the agreement diminishes when we do not account for linkage. We analysed the mutational fitness effects distributions and found that mutations in non-coding regions have higher fitness effects, as do mutations that evolve independently in multiple evolution lines. Overall, we show that mutational fitness effects can be inferred from high-throughput sequencing data, allowing for the prediction of population fitness over many generations.

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CT263 — Evaluating the Public Health Programs during the 2010 Haiti Cholera Outbreak: Disease Dynamics and Epidemic Response

SPEAKERS Vijay Pal Bajiya (SRM Institute of Science and Technology, Kattankulathur, TN, India)
AUTHORS R.K. Upadhyay (Indian Institute of Technology (ISM), Dhanbad-826004, India); Vijay Pal Bajiya (SRM Institute of Science and Technology, Kattankulathur, TN, India)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Cholera remains a global threat, as highlighted by the 2010 Haiti outbreak, where poor sanitation led to severe exposure. With limited vaccines and treatments, control efforts rely on awareness campaigns and sanitation to promote preventive behavior. We developed a cholera transmission model incorporating public awareness and behavioral responses. Stability was analyzed using the basic reproduction number, and the model was calibrated to Haiti outbreak data. Sensitivity analysis assessed how awareness-driven behavior influences disease dynamics. Results show that weakening precautions by 38.5% while doubling transmission increases peak infections by 135%. Strengthening precautions by the same amount under baseline transmission reduces the peak to 68.5%, demonstrating the power of behavior change. Doubling sanitation lowers the peak to just 4.66%, confirming hygiene as a key measure. A 50% drop in antibiotic use, combined with reduced precautions, raises peak infections by nearly 20%. Even under baseline transmission, a 37% increase in precautions cuts cumulative cases by about 27%.

Findings indicate that ignoring behavioral and sanitation interventions can worsen outbreaks, while combining awareness campaigns with vaccination significantly reduces infections and disease burden. This work underscores the critical role of human behavior and timely information in cholera control.

CT1498 — Learning dynamical systems with biochemically informed neural ordinary differential equations

SPEAKERS Lucas Böttcher (Frankfurt School of Finance & Management)
AUTHORS Lucas Böttcher (Frankfurt School of Finance & Management); Luis L. Fonseca (University of Florida); Reinhard Laubenbacher (University of Florida)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Ordinary differential equation models of biological systems are often formulated as stoichiometric systems in which the dynamics arise from a collection of interacting processes. A central challenge is that the functional form of each process is rarely known a priori and may be difficult to infer from data. We propose biochemically informed neural ordinary differential equations (BINODEs), a neural-ODE framework that retains the stoichiometric structure of mechanistic models while representing individual process rates by neural network processes (NNPs). In BINODEs, each process is modeled by a feedforward network, and the resulting process outputs are mapped to state derivatives through a linear layer analogous to a stoichiometric matrix. This architecture allows biological side information, such as process-specific inputs, sign constraints, and monotonicity assumptions, to be built directly into the model. We characterize the

approximation properties of NNPs for several standard biochemical rate laws and show that the proposed framework accurately learns both trajectories and process-level structure in Monod, Lotka–Volterra, pharmacokinetic, and ultradian endocrine models. These results suggest that BINODEs offer a useful compromise between mechanistic interpretability and data-driven flexibility for modeling partially known biological dynamical systems.

CT236 — Modeling the reversible transition of glioblastoma cells to resistant-senescent states via transport equations

SPEAKERS José Antonio Romero Rosales (Universidad de Castilla-La Mancha - Campus Ciudad Real)
AUTHORS Carmen Ortega-Sabater (National Center Oncological Research (Madrid)); Gabriel Fernández-Calvo (Universidad de Castilla-La Mancha - Campus Ciudad Real); Giorgio Seano (Institut Curie (Paris)); José Antonio Romero Rosales (Universidad de Castilla-La Mancha - Campus Ciudad Real); Juan Jiménez-Sánchez (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla-La Mancha (UCLM)); Juliette Reveilles (Institut Curie (Paris))
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Understanding glioblastoma resistance and recurrence remains a primary challenge in oncology. PDE models remain critical tools in the study of their underlying biology [Álv+19]. In this study, we model a glioblastoma cell population, analyzing the distribution of resistance levels across the population under various treatment levels. Specifically, we investigate the ”convection factor,” which represents the epigenetic capacity of cells to transition toward higher resistance states in response to different stress types. A core challenge involves the use of a non-differentiable profile, which significantly complicates the calculation; we successfully derive an analytical solution for the governing system of equations. Our improved model extends beyond resistance accumulation to analyze the reversibility of these effects; specifically, the biological mechanisms that drive cells from resistant-senescent states back to more vulnerable phenotypes. By parameterizing our model and fitting it to experimental data [Pic+24], we quantify the significance of various resistance-inducing factors. Crucially, this framework allows us to estimate the time scales required for phenotypic reversion, providing a robust basis for optimizing radiation therapy schedules. Ultimately, our approach enables the exploration of diverse therapeutic strategies, offering precise insights into how we may manipulate cell states to maximize vulnerability and improve clinical efficacy.

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CT330 — Modelling tissue damage in destructive lung disease

SPEAKERS Alice Peyraut (Centre for Mathematical Medicine and Biology, School of Mathematical Sciences, University of Nottingham, Nottingham)
AUTHORS Alice Peyraut (Centre for Mathematical Medicine and Biology, School of Mathematical Sciences, University of Nottingham, Nottingham); Reuben O'Dea (Centre for Mathematical Medicine and Biology, School of Mathematical Sciences, University of Nottingham, Nottingham); Bindi Brook (Centre for Mathematical Medicine and Biology, School of Mathematical Sciences, University of Nottingham, Nottingham)

SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Chronic Obstructive Pulmonary Disease (COPD) is a highly prevalent lung disease characterized by alveolar wall damage, leading to progressive loss of lung function [RG09]. Lymphangioleimyostasis (LAM), a rare respiratory disease, is also characterised by lung tissue destruction, but also by the growth of abnormal smooth muscle-like cells [Don+17]. The interactions between injury and abnormal repair leading to damage are not well understood. In both diseases, tissue destruction is driven by protease secretion due to complex interactions between immune and structural cells and proteins, during injury and abnormal repair processes. This impacts collagen and elastin density in lung tissue, resulting in alveolar wall thinning and eventual destruction. To investigate these mechanisms, we model evolution of tissue constituents and integrity using a system of coupled ordinary differential equations, coupled to the mechanical strain/tension experienced by springs within a 2D elastic spring network [Yua+22] representing the alveolar structure in parenchymal tissue [Cer+18]. As tissue integrity decreases, any increase in strains exceeding a physiological threshold leads to springs breaking (i.e., tissue damage). Our model, coupled with clinical images and spatial omics data, will allow us to investigate tissue destruction in LAM and COPD and provide time-dependent network-level measures, from which potential biomarkers could be derived.

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CT1581 — Quantifying biological heterogeneity in nano-engineered particle–cell interaction experiments

SPEAKERS Ryan Murphy (Adelaide University)
AUTHORS James Osborne (University of Melbourne); Matt Faria (University of Melbourne); Ryan Murphy (Adelaide University); Stuart Johnston (University of Melbourne)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.33 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Nano-engineered particles are a promising tool for medical diagnostics, biomedical imaging and targeted drug delivery. Fundamental to the assessment of particle performance are in vitro particle–cell interaction experiments. These experiments can be summarized with key parameters that facilitate objective comparisons across various cell and particle pairs, such as the particle–cell association rate. Previous studies often focus on point estimates of such parameters and neglect heterogeneity in routine measurements. In this study, we develop an ordinary differential equation-based mechanistic mathematical model that incorporates and exploits the heterogeneity in routine measurements. Connecting this model to data using approximate Bayesian computation parameter inference and prediction tools, we reveal the significant role

of heterogeneity in parameters that characterize particle–cell interactions. We then generate predictions for key quantities, such as the time evolution of the number of particles per cell. Finally, by systematically exploring how the choice of experimental time points influences estimates of key quantities, we identify optimal experimental time points that maximize the information that is gained from particle–cell interaction experiments.

CT1542 — Simulating glioblastoma microenvironment: How hypoxia and vascular occlusion shape immune dynamics

SPEAKERS Raquel Arroyo-Vázquez (TME Lab, Engineering Research Institute of Aragon (I3A), University of Zaragoza, Spain)
AUTHORS Heber L. Rocha (Department of Intelligent Systems Engineering, Indiana University, Bloomington, IN, USA); Randy Heiland (Department of Intelligent Systems Engineering, Indiana University, Bloomington, IN, USA); Manuel Doblare (University of Zaragoza); Paul Macklin (Department of Intelligent Systems Engineering, Indiana University, Bloomington, IN, USA); José Antonio Sanz-Herrera (Higher Technical School of Engineering. University of Sevilla, Spain); Raquel Arroyo-Vázquez (TME Lab, Engineering Research Institute of Aragon (I3A), University of Zaragoza, Spain); Jacobo Ayensa-Jiménez (TME Lab, Engineering Research Institute of Aragon (I3A), University of Zaragoza, Spain)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 03.01 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Glioblastoma is characterized by spatial heterogeneity and severe hypoxia, which shape the immune response and contribute to poor therapeutic outcomes [BRO26]. In this work, we reproduce a multiscale tumor–immune model [Gon+17] and develop an agent-based model with PhysiCell [Gha+18] that captures key biological features driving glioblastoma progression.

The model incorporates two cancer cell phenotypes, three T cell subtypes, and vascular points; together with oxygen, the cytokine IL-2 and the chemokine CXCL12 diffusion. Agents migrate, proliferate, and interact locally, while vascular points act as oxygen sources and T cell entry points. Furthermore, the model incorporates vascular occlusion resulting from increased pressure as the tumor grows, which halts oxygen secretion and immune recruitment.

Preliminary results indicate that the initial density and spatial distribution of vascular points are determinants of tumor progression and lymphocyte efficacy. We observed that specific vascular configurations enhance T cell infiltration, leading to a measurable reduction of tumor growth. These findings suggest that vascular distribution could serve as a predictive marker for treatment response. This work provides a platform for exploring immune–tumor dynamics in glioblastoma and sets the stage for future work assessing immunotherapeutic strategies.

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CT300 — Vitamin C as a nitrosation inhibitor: a modelling study across dietary patterns and water quality

SPEAKERS Gordon R. McNicol (University of Waterloo)
AUTHORS Nandita B. Basu (University of Waterloo); Anita T. Layton (University of Waterloo); Gordon R. McNicol (University of Waterloo)
SCHEDULE Thursday, 16 July 2026; 11:20–11:40; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Rising dietary and drinking-water intake of nitrate (NO_3^-) and nitrite (NO_2^-) poses a significant public health concern. After ingestion, NO_3^- enters the enterosalivary circulation, where oral bacteria reduce it to NO_2^-. When swallowed, NO_2^- enters the acidic gastric environment, where it can react to form N-nitroso compounds (NOCs), many of which are suspected carcinogens. However, epidemiological evidence for this link remains mixed, likely due to the protective effects of antioxidants such as vitamin C. To better understand and quantify these complex interactions, we develop a dynamic, compartmental quantitative systems pharmacology model of human NO_3^- and NO_2^- metabolism and gastric chemistry. The framework tracks NO_3^- and NO_2^- fluxes across the stomach, intestine, plasma, and saliva, incorporates postprandial changes in gastric volume and pH, and includes mechanistic nitrosation pathways with vitamin C inhibition. Using this framework, we evaluate NOC formation under different dietary and water-quality contexts, demonstrating the protective effect of dietary vitamin C and examining the role of supplementation. Simulations suggest supplementation is most effective shortly after meals. These findings provide a mechanistic basis for understanding how diet, drinking-water quality, and vitamin C supplementation interact to shape endogenous NOC formation, with implications for nutritional guidelines and risk reduction.

CT1335 — A temperature-dependant spacial model to optimise sterile male releases for Drosophila suzukii

SPEAKERS Jules Guilberteau (INRAE (Institut Sophia Agrobiotech), CNRS, Université Côte d'Azur)
AUTHORS Jules Guilberteau (INRAE (Institut Sophia Agrobiotech), CNRS, Université Côte d'Azur); Louise van Oudenhove (INRAE (Institut Sophia Agrobiotech), CNRS, Université Côte d'Azur); Ludovic Mailleret (INRAE (Institut Sophia Agrobiotech), Inria, CNRS, Université Côte d'Azur); Suzanne Touzeau (INRAE (Institut Sophia Agrobiotech), Inria, CNRS, Université Côte d'Azur)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Drosophila suzukii is a fruit fly species native to South-East Asia that has become a major invasive pest in Europe and North America. Thanks to its serrated ovipositor, which allows it to lay eggs in healthy fruit, it causes significant damage to several soft fruit crops such as strawberries, blueberries, and cherries. As pesticide use alone is environmentally costly and increasingly inefficient, it is necessary to consider alternative control strategies. In this context, the sterile insect technique – the release of sterilised males that compete with wild males for mating – constitutes a promising and ecologically sustainable method that can be used alone or combined with other pest-management tools.

In this talk, focusing on cherry orchards, we present a stage-structured population dynamics model based on the physiological characteristics of *D. suzukii*. Development time, fecundity and adult survival are modelled as functions of temperature in order to explicitly account for seasonality. The model also accounts for the spatial configuration of cherry orchards and wild resource patches, and population movement between them.

This model is used to explore optimal release strategies: given a certain number of males released during the year, is it better to target spring or fall populations? What is the optimal frequency of releases? Where should sterile males be released when there are multiple orchards and wild plots?

CT1684 — Decoding Clonal Dynamics in MDS Through Adapted Mathematical Models of CHIP

SPEAKERS Cameron Kerr (University of Edinburgh)
AUTHORS Cameron Kerr (University of Edinburgh)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 03.01 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Clonal Haematopoiesis of Indeterminate Potential (CHIP) is characterised by expansion of mutated Haematopoietic Stem and Progenitor Cells (HSPCs), resulting in overrepresentation of their progeny in peripheral blood. CHIP occurs in 15–20% of individuals over 60 years of age and is linked to haematologic malignancy, age-related disease, and increased all-cause mortality. Mathematical modelling has been successfully used to infer mutant fitness from longitudinal variant allele frequency (VAF) data, but extending these approaches to Myelodysplastic Syndromes (MDS), a form of blood cancer, requires relaxing assumptions. First, we investigate how loss of heterozygosity (LoH) alters inferred clonal dynamics in expanding mutant populations and assess whether minimal models are sufficient for reliable fitness inference. We develop a methodology to estimate het/homozygous mutant cell proportions when these are not known from data. Secondly, we develop clonal growth models incorporating mutant-dependent growth, competition, and alternative carrying-capacity and a variable bias for mutants to displace healthy cells show when commonly used exponential growth models fail for fitness inference. Our results demonstrate that modelling assumptions substantially alter inferred clonal dynamics and predicted VAF trajectories, highlighting how clonal expansion may transform during disease progression and how modelling choices impact interpretation of longitudinal sequencing data in CHIP and MDS.

CT1596 — Extended Pseudo-spectral Physics-inferred Neural Networks for phase field models

SPEAKERS Callum Marsh (University of Oxford)
AUTHORS Andreas Münch (University of Oxford); Callum Marsh (University of Oxford); Radek Erban (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Phase-field models provide a fundamental continuum framework for describing phase separation and pattern formation in many physical and biological systems. Their predictive capability depends critically on constitutive quantities such as the bulk free-energy density and interfacial thickness parameter, which are often unknown and must be inferred from limited observations. In this work, we introduce an extended pseudo-spectral physics-informed neural network (ESPINN) framework for the inverse identification of phase-field models from transient snapshot data. The proposed method simultaneously reconstructs the bulk chemical potential and unknown gradient coefficients directly from dynamically evolving structures.

Numerical experiments show that ESPINN accurately recovers both the functional form of the free energy and the interfacial thickness parameter. Remarkably, substantial constitutive information can be extracted even from a single snapshot pair, while additional snapshots improve robustness and reduce variance across training runs. The framework remains stable in the presence of noise, with reconstruction accuracy improving as more observations are incorporated. These results highlight ESPINN as a data-efficient

and physically consistent approach for learning constitutive structure in continuum models of phase separation.

CT1513 — Persistent homology analysis of human populations reveals topological signatures of admixture

SPEAKERS Victoria May Mendoza (Institute of Mathematics, University of the Philippines Diliman)
AUTHORS Al Bien Christian Aculan (Institute of Mathematics, University of the Philippines Diliman, Quezon City, Philippines); Maria Vivien Visaya (Department of Mathematics and Applied Mathematics, University of Johannesburg, South Africa; National Institute for Theoretical and Computational Sciences (NITheCS), Stellenbosch University, South Africa); Rachelle Sambayan (Institute of Mathematics, University of the Philippines Diliman, Quezon City, Philippines); Ricardo del Rosario (Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, Massachusetts, United States of America); Victoria May Mendoza (Institute of Mathematics, University of the Philippines Diliman)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Genetic data sampled from human populations are high-dimensional and complex. Many tools have been developed to analyze such data; however, these methods often require specifying a pre-determined number of ancestral populations, may not scale efficiently to large datasets, or rely on model assumptions about processes such as migration and population bottlenecks. Here, we investigate whether the topological properties of populations, analyzed using Topological Data Analysis (TDA), can reveal features that help characterize population admixture. We used persistent homology, a tool in TDA, to detect topological signatures of admixture in 26 human populations comprising 3,202 individuals from whole-genome sequencing data from the 1000 Genomes Project. We found that the topological structures of populations, visualized as barcodes, can differentiate admixed populations from non-admixed ones. Clustering based on the Wasserstein distance between barcodes groups admixed populations together, indicating that our method can detect common topological signatures of admixture. We also found that the topological signatures of the Iberian populations in Spain are more similar to those of admixed populations than to other European populations.

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CT212 — Stochastic-Deterministic Mathematical Models of Neuroblastoma Spatial Structure

SPEAKERS Perla Mallouk (Université Paris Cité, CNRS, MAP5, F-75006 Paris, France)

AUTHORS Perla Mallouk (Université Paris Cité, CNRS, MAP5, F-75006 Paris, France); Fabien Crauste (Université Paris Cité, CNRS, MAP5, F-75006 Paris, France)
SCHEDULE Thursday, 16 July 2026; 11:40–12:00; 15.33 - SR
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Neuroblastomas are solid tumors and represent the most common extracranial tumors in children. The analysis of tumoroid data (artificial organoids capable of reproducing neuroblastoma growth) has revealed a distinctive spatial organization: cancer stem cells tend to cluster at the center of the tumor. A multiscale agent-based neuroblastoma tumoroid model was developed to simulate neuroblastoma growth, and the data provided by this model represent a unique opportunity to investigate the genetic causes of the spatial structures of neuroblastoma tumors. Our goal is to develop a mathematical model of neuroblastoma growth based on these data to better understand its spatial distribution driven by stochastic gene expression and non-local gene interactions, and ultimately to propose more targeted treatments. We combined a deterministic model of tumor growth with a Piecewise Deterministic Markov Process, which accounts for stochastic gene expression. We conducted a mathematical analysis of this model to prove its well-posedness using semigroup theory and stochastic process theory, and we performed numerical simulations to observe whether the expected spatial distribution emerges. The model reproduces a single central stem-cell cluster in 1D. Existence of multiple clusters, as highlighted by experimental and computational results, still represent a mathematical challenge. I will discuss the current state of my work and notably how to better capture this specific spatial organization.

CT1401 — A dynamical mechanism for intratumoral heterogeneity in breast cancer

SPEAKERS Francisco Lopes (Universidade Federal do Rio de Janeiro)
AUTHORS Alexandre Lima (Universidade Federal do Rio de Janeiro, Duque de Caxias, Brazil); Bruno Pires (Universidade Estadual do Rio de Janeiro (UERJ), Rio de Janeiro, Brazil); Eliana Abdelhay (Instituto Nacional de Câncer, Rio de Janeiro, Brazil); Francisco Lopes (Universidade Federal do Rio de Janeiro); Renata Binato (Instituto Nacional de Câncer, Rio de Janeiro, Brazil)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.06 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Intratumoral heterogeneity in breast cancer is a major challenge for prognosis and treatment, yet the dynamical mechanisms underlying subtype transitions remain poorly understood. Using a gene regulatory network model calibrated with experimental data, we show that stochastic variability in NF-κB can induce irreversible switching from HER2+ to TNBC states, providing a dynamical mechanism for intratumoral heterogeneity [LPL25]. The model reproduces HER2+ and TNBC as distinct stable states within a common regulatory framework, consistent with in vitro expression patterns and further supported by patient-derived bulk and single-cell data. Because these transitions occur asynchronously across cells, the model predicts the coexistence of HER2+ and TNBC subtypes within the same tumor, linking molecular stochasticity to tissue-level heterogeneity. These results provide a mechanistic framework for understanding how subtype heterogeneity may emerge during tumor progression and may help inform strategies for classification and therapeutic intervention.

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CT1506 — A mathematical framework for replication stress in cancer

SPEAKERS Francisco Berkemeier (Departments of Pathology and Genetics, University of Cambridge)
AUTHORS Francisco Berkemeier (Departments of Pathology and Genetics, University of Cambridge); Michael Boemo (Departments of Pathology and Genetics, University of Cambridge); Pauline Pfuderer (Department of Pathology, University of Cambridge); Peter Cook (Sir William Dunn School of Pathology, University of Oxford)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 01.22 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

DNA replication duplicates the genome before cell division, and its faithful completion is essential for genome stability. Failures in this process generate replication stress, a hallmark of cancer, but also a therapeutic opportunity, since drugs that further perturb replication can push tumour cells beyond tolerable limits [Jon+25]. Understanding these effects remains difficult, because experiments are costly and available data often provide only partial views of replication dynamics across the genome. Here we present a mathematical framework for genome wide DNA replication that links mechanistic modelling, inference, and therapeutic applications. The framework is based on an analogy with one dimensional crystal growth, in which stochastic origin firing and replication fork speed shape the spatiotemporal progression of replication. Within this setting, light cone formalisms and transport equations for fork densities connect replication timing, origin usage, and fork kinetics. This makes it possible to reconstruct initiation landscapes from timing data and to identify genomic regions where replication dynamics break down [BCB25]. In addition, we show that combining mechanistic modelling with genome language models and physics informed machine learning opens the way to predictive modelling of targeted therapies with translational potential for cancer biology [Pfu+26].

References

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CT1448 — A reaction-diffusion model of epithelial polarity dynamics in the endometrium over the menstrual cycle

SPEAKERS Claire Miller (Auckland Bioengineering Institute, University of Auckland)
AUTHORS Claire Miller (Auckland Bioengineering Institute, University of Auckland); Daniel Reid (Auckland Bioengineering Institute, University of Auckland (former position)); Alys Clark (Auckland Bioengineering Institute, University of Auckland)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 10.01 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Cell polarity, specifically apico-basal polarity, is defined by the asymmetry of internal cell proteins and structures between the basolateral region of the cell, the region in contact with the basal membrane and cell neighbours, and the apical region, which faces the lumen. The establishment and robust maintenance of apico-basal polarity in epithelial cells is critical to healthy function of the epithelial layer. However,

uniquely, within the endometrial epithelium (the internal lining of the uterus), polarity is also lost over the course of the menstrual cycle. Dysregulation of this polarity loss is hypothesised to be a possible cause of infertility, as it can prohibit embryo implantation [WZD20].

In this talk, we will describe our reaction-diffusion model of cell polarity proteins and complexes within an epithelial cell. This model builds on a previous model of cell polarity establishment in a C. Elegans zygote [Goe+11]. We will show that the model reproduces spatial profiles of polarity proteins and complexes that are characteristic of the establishment, maintenance, and loss phases of the endometrial cell polarity cycle. These profiles emerge when we simulate the observed changes in cell polarity protein levels over the secretory phase of the menstrual cycle from the literature [WSE18]. Using this model, we can hypothesise potential dysfunctions that could lead to dysregulation of polarity loss, and consequently issues with implantation.

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CT397 — A Systematic Framework for Inferring Stochastic Dynamics from Data

SPEAKERS Katarina Bodova (Comenius University)
AUTHORS Katarina Bodova (Comenius University); Richard Kollar (Comenius University)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Recent advances in experimental biology provide time-resolved datasets that capture key features of complex dynamical systems. Recovering the underlying interactions from such data is a nontrivial inverse problem, especially when one must disentangle deterministic dynamics, intrinsic fluctuations, and measurement noise from limited observations.

We build on the inference framework introduced by Brückner et al. [BRB20], which is designed to operate in the moderate-data regime. Our main contribution is to replace the heuristic choices in the original derivation by a controlled expansion of the inference error. This leads to more accurate approximations, identifies the relevant small parameters, and makes the associated validity conditions explicit. The result is an efficient and broadly applicable framework for inferring stochastic dynamics from limited, sparsely sampled data.

References

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CT196 — Application of Radial Basis Function Physics-Informed Neural Networks in Fractional Pharmacokinetics Modeling

SPEAKERS Reza Mokhtari (Department of Mathematical Sciences, Isfahan University of Technology, Isfahan 8415683111, Iran / Department of Mathematics and Scientific Computing, University of Graz, Austria)

AUTHORS Maryam Mohammadi (Department of Mathematical Sciences, Isfahan University of Technology, Isfahan 8415683111, Iran); Reza Mokhtari (Department of Mathematical Sciences, Isfahan University of Technology, Isfahan 8415683111, Iran / Department of Mathematics and Scientific Computing, University of Graz, Austria)

SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.21 - SZ

SESSION Contributed Talks

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Fractional-order compartmental models have been increasingly adopted in pharmacokinetics to capture anomalous drug diffusion, memory effects, and non-exponential elimination patterns that classical integer-order models cannot adequately describe. However, the numerical treatment of these models remains challenging because of nonlocal operators, memory dependence, and mass-balance inconsistencies.

In this work, we build upon the Radial Basis Function-Enhanced Fractional Physics-Informed Neural Networks (RBF-fPINN) framework to investigate fractional pharmacokinetics compartmental models. We consider three classes of fractional pharmacokinetics models formulated from a classical two-compartment system: (i) commensurable fractional models with a uniform fractional order, (ii) non-commensurable models with distinct fractional orders across compartments, and (iii) implicit non-commensurable models that fractionalize transport processes while preserving mass balance. The RBF-fPINN methodology is adapted to each model class, enabling a unified comparison of accuracy and computational efficiency.

Numerical experiments demonstrate that our recently proposed method accurately captures the slow, non-exponential drug dynamics and outperforms classical numerical schemes, particularly for implicit non-commensurable models. Furthermore, the framework enables simultaneous state estimation and parameter identification, making it very useful for data-driven pharmacokinetics applications.

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CT1273 — Cancer-Associated Fibroblast Heterogeneity Predicts Cancer Progression and Treatment Response

SPEAKERS Junho Lee (Korea Institute of Science and Technology)

AUTHORS Eunjung Kim (Korea Institute of Science and Technology); Junho Lee (Korea Institute of Science and Technology)

SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 02.21 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Cancer-associated fibroblasts (CAFs) are key components of the tumor microenvironment (TME) and exhibit highly heterogeneous phenotypes that can either promote or suppress tumor growth. While clinical evidence increasingly links CAF composition to patient survival and drug response, a quantitative framework that predicts treatment outcomes from CAF phenotypic profiles has been lacking.

Here we present an ordinary differential equation (ODE) model that captures the interactions among cancer cells, effector and regulatory T cells, and four functionally distinct CAF phenotypes — anti-immune, pro-immune, anti-cancer, and pro-cancer. CAF fractions enter as direct modulators of PD-1/PD-L1 binding kinetics, PI3K/AKT signaling, and Treg recruitment. Simulations demonstrate that CAF composition profoundly shapes cancer growth and immunotherapy outcomes: the same PD-L1, Treg, or PI3K inhibitor can succeed in one CAF regime and fail in another. A model-guided treatment map identifies CAF profiles for which monotherapy is as effective as combination therapy, profiles requiring combination, and a minority resistant even to triple therapy. Kaplan–Meier simulations across 500 virtual patients show order-of-magnitude survival differences arising purely from CAF composition [LK25].

Building on this framework, our ongoing work extends the model into a hybrid agent-based / PDE / ODE system that additionally represents the spatial arrangement of CAFs and the PD-1/PD-L1 dynamics of individual T cells. Preliminary results suggest that spatial layout is a distinct, complementary axis of TME heterogeneity, offering a path toward CAF-informed, spatially resolved precision oncology.

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CT255 — Dissecting GLUT4 Trafficking: Structural Motifs, Insulin Responsiveness and Retention

SPEAKERS Adelle Coster (School of Mathematics and Statistics, University of New South Wales)
AUTHORS Adelle Coster (School of Mathematics and Statistics, University of New South Wales); Cynthia Mastick (Department of Biology, University of Nevada, Reno)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 11.11 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In mammalian fat cells, insulin regulates glucose uptake by controlling the trafficking of the glucose transporter GLUT4 between intracellular compartments and the plasma membrane. Under basal conditions, GLUT4 is largely sequestered in slowly cycling pools, but insulin stimulates its mobilisation, exocytosis, and cycling, with rapid re-sequestration following insulin withdrawal.

Structural motifs of GLUT4, including the LL and FQQI sequences, govern GLUT4 targeting to insulin responsive compartments; mutations such as LLAA or AQQI are thought to disrupt sorting and produce impaired insulin responsiveness.

To quantify these dynamics, we model GLUT4 trafficking in 3T3-L1 adipocytes using parsimonious compartmental models in which insulin modulates the exocytosis rate constant and the distribution of

GLUT4 between cycling and non-cycling pools. Model optimisation of steady state and transition data supports a dose dependent expansion of the cycling pool at submaximal insulin levels and indicates that insulin also regulates a second kinetic rate constant. The model is then extended to include perturbations to the trafficking kinetics due to mutations of GLUT4 motifs.

Different approaches to parameter inference simultaneously constrained by multiple experimental data sets are discussed and the effects of structural motifs and dynamic regulation of GLUT4 trafficking under varying insulin concentrations and mutations is explored.

CT1282 — Dynamics of a modified piece-wise linear Toivonen-Fromhage model

SPEAKERS Ryusuke Kon (University of Miyazaki)
AUTHORS Ryusuke Kon (University of Miyazaki)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 11.32 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Toivonen and Fromhage (2018) proposed a competition model between size-based and age-based populations to study the evolution of periodicity in periodical cicadas. They demonstrated that the effect of predator satiation triggers the replacement of a size-based population by an age-based one, even when the former possesses a higher reproductive number. This suggests that the perfect periodicity observed in periodical cicadas could have evolved from proto-periodicity. In this talk, we modify the model of Toivonen and Fromhage (2018) to derive a piece-wise linear model. This serves as a generalization of the periodical cicada model proposed by May (1979), which was itself a modification of the Hoppensteadt and Keller (1976) model. Using this modified framework, we show that the proto-periodicity in the size-based population observed by Toivonen and Fromhage (2018) is realized by a specific periodic orbit, which we term an "essentially single-class orbit". We further establish the stability conditions for this orbit, demonstrating that an essentially single-class orbit is more likely to be stabilized than an equilibrium solution if reproduction occurs in later age-classes. Finally, we mathematically prove that a size-based population is invaded and eventually eradicated by an age-based population. These results mathematically complement the results by Toivonen and Fromhage (2018).

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CT1534 — First Explore, Then Settle: A Theoretical Analysis of Evolvability as a Driver of Adaptation

SPEAKERS Juan Jiménez-Sánchez (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla-La Mancha (UCLM))
AUTHORS Carmen Ortega-Sabater (CNIO); Juan Jiménez-Sánchez (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla-La Mancha (UCLM)); Philip Maini (Mathematical Institute, Oxford University); Tommaso Lorenzi (Politecnico di Torino); Víctor Pérez-García (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla-La Mancha (UCLM))

SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 11.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Evolvability—the capacity of a population to generate heritable phenotypic variation—plays a central role in adaptation, yet its evolutionary dynamics are not fully understood. In this work, we investigate how evolvability, treated as a continuous phenotypic trait, shapes the adaptation of asexual populations. To this end, we develop ad hoc discrete and continuum mathematical models describing the evolutionary dynamics of phenotype structured cell populations, characterised by their fitness and evolvability. Through analysis and simulation of these models, we study how proliferative potential and evolvability are selected over short and long time scales, and under various conditions, including asymmetries in the distribution of fitness effects and increasingly steep phenotypic landscapes. Our results reveal a robust two step adaptive trajectory: highly evolvable individuals initially accelerate exploration of the phenotypic landscape, enabling rapid access to fitter proliferative states; once high proliferative potential is reached, selection favours reduced evolvability, promoting stabilisation. We further show that strong selection gradients, high evolvability costs, and asymmetric distributions of fitness effects can slow adaptation or even drive extinction when populations start from highly evolvable, low fitness states. These findings highlight evolvability as a dynamic trait whose short term benefits may conflict with long term persistence [Jim+26].

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CT1550 — Gardner volumes and self-organization in a minimal model of complex ecosystems

SPEAKERS Frederik Thomsen (Delft University of Technology)
AUTHORS Frederik Thomsen (Delft University of Technology); Johan Dubbeldam (Delft University of Technology); Rudolf Hanel (Complexity Science Hub Vienna, Medical University Vienna)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.02 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

We study self-organization in a minimally nonlinear model of large random ecosystems. Populations evolve over time according to a piecewise linear system of ordinary differential equations subject to a non-negativity constraint resulting in discrete time extinction and revival events. The dynamics are generated by a random elliptic community matrix with tunable correlation strength. We show that, independent of the correlation strength, solutions of the system are confined to subsets of the phase space that can be cast as time-varying Gardner volumes from the theory of learning in neural networks. These volumes decrease with the diversity (i.e. the fraction of extant species) and become exponentially small in the long-time limit. Using standard results from random matrix theory, the changing diversity is then linked to a sequence of contractions and expansions in the spectrum of the community matrix over time, resulting in a sequence of May-type stability problems determining whether the total population evolves toward complete extinction or unbounded growth. In the case of unbounded growth, we show the model allows for a particularly simple nonlinear extension in which the solutions instead evolve towards a new attractor.

CT233 — Generation time for diseases with variable infectiousness before and after symptom onset: beyond geometric waiting times.

SPEAKERS Jordi Ripoll (University of Girona, Spain)
AUTHORS Jordi Ripoll (University of Girona, Spain)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 03.01 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

We study the random times between successive cases in a transmission chain, of infectious diseases with asymptomatic carriers. We derive the probability distribution of this generation time (in days) from a discrete-time epidemic model with variable infectiousness both along elapsed times and across phases. The introduced non-Markovian model is a recursive system featuring random waiting times at each of the three infected stages: latent, asymptomatic, and symptomatic. By rearranging the terms of the basic reproduction number R_0, which represents the expected number of secondary cases produced by an asymptomatic primary case who may eventually develop symptoms, we get to the generation-time probabilities. The expected generation time is a convex combination of the expected generation times before and after the onset of symptoms. Additionally, our analysis reveals that the n-th moment of the generation time is related to the weighted forward recurrence time at each phase and the moments up to n-th order of the latent period and the incubation period. These weights are the infectiousness along the elapsed times. Finally, we illustrate several data-driven epidemic scenarios, assuming that infectiousness varies only across phases, each displaying high variability in its respective generation time.

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CT1343 — Identifying alternative explanations for population growth rate behaviour between ancestor and mutant strains of Chlamydomonas reinhardtii in stressful environmental conditions using multiple phenotype models

SPEAKERS Adam Hillman (University of Strathclyde)
AUTHORS Adam Hillman (University of Strathclyde); Gabriela Gomes (University of Strathclyde); John Mackenzie (University of Strathclyde)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Differences in population growth rates between ancestors and mutants in increasingly stressful environments are often attributed to one group of individuals (ancestor or mutant) being more vulnerable to the stressful environment than the other. However, modelling the behaviour of these groups with the assumption of there being more than one phenotype in a particular group (in this case the mutants) can provide the explanation that it is the level of variation in the group’s population that drives the differences in behaviour. To

demonstrate this phenomena, population growth rate data of the micro-algae Chlamydomonas reinhardtii in salt environments from Kraemer et al. [Kra+16] was modelled using both the conventional single-phenotype model and the alternative multi-phenotype models, where the inference of the different parameters provide competitive fits and differing explanations as to the difference in behaviour between the ancestor and mutant populations under stressful environments.

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CT1462 — Inferring CD8^{+} T Cell Differentiation Trajectories in Cancer through Data-Driven Distribution Modelling

SPEAKERS Nissrin Alachkar
AUTHORS Kevin Thurley; Michael Hölzel; Nissrin Alachkar
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

$CD8^+$ T cells play a central role in anti-tumour immunity by eliminating cancer cells. However, sustained exposure to tumour antigens can drive them into a differentiation pathway culminating in a dysfunctional exhausted state, limiting immune control of cancer. This process emerges from collective inter- and intracellular interactions within complex immune signalling networks. Revealing the differentiation trajectories and the mechanisms governing exhaustion therefore requires quantitative frameworks that capture the dynamical processes underlying immune cell fate decisions. First, we develop a mathematical framework to analyse $CD8^+$ T cell decision circuits in cancer. We analyse network motifs governing $CD8^+$ T cell population dynamics, capturing proliferation, differentiation, and cytokine-mediated feedback. State transitions are described using response-time modelling, where waiting-time distributions follow gamma distribution, accounting for multi-step intracellular processes while maintaining analytical tractability. Complementary to this approach, we analyse high-dimensional flow cytometry data from a murine melanoma adoptive T cell therapy experiment. Using unsupervised high-dimensional clustering, we identify phenotypic $CD8^+$ T cell subsets across organs and time points. Based on these subsets, we systematically generate candidate differentiation network topologies and fit them to the data. Model selection identifies the topology that best explains the observed dynamics.

CT393 — Inferring Tissue Evolution Dynamics from DNA Methylation Profiles

SPEAKERS Catherine Engert (University of Oxford)
AUTHORS Catherine Engert (University of Oxford); Helen Byrne (University of Oxford); Ruth Baker (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 05.12 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Genetic mutations can lead to the formation of genetically distinct subpopulations of cells, called clones, which can vary in size over time. Due to the difficulty of obtaining meaningful longitudinal genetic data, one of the main challenges lies in the complexity of inferring the dynamics that govern the emergence and evolution of clones from snapshot data. In this context, DNA methylation can provide useful information: DNA methylation marks can oscillate back and forth between methylated and unmethylated states, leading

to substantial differences in intercellular methylation patterns that can be used to identify clonal cell populations and infer their history [Sch+25; Hay+23; FCW26; Lar+19; UK20].

We propose stochastic models of DNA methylation fluctuation and inheritance to study how methylation patterns evolve in clonal cell populations, considering mono- versus polyclonal evolution and competition dynamics. By developing simple and identifiable models, we reconstruct tissue evolution and identify which biological parameters drive methylation patterns indicative of clonal cell populations and how these differ from the methylation patterns that arise from well-mixed tissues. Finally, we compare the identifiability of discrete agent-based models, coarse-grained models, and continuum models to determine in which cases simple models accurately capture clonal tissue dynamics and in which cases more complex models need to, and can, be learned from data.

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CT1397 — Investigating the Relationship Between Angiogenesis and Tumour Carrying Capacity with Physics Informed Neural Networks

SPEAKERS Morghan van Walsum (University of Waterloo, Canada)
AUTHORS Mohammad Kohandel (University of Waterloo, Canada); Morghan van Walsum (University of Waterloo, Canada)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 02.23 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Angiogenesis is a hallmark of cancer wherein cancerous cells generate new vasculature to ensure that all cells continue to receive adequate nutrients as the tumour grows. Mathematical models exist for dynamics between tumour cells and vasculature, and a primary assumption in many of these models is that tumour carrying capacity is related to vasculature density [Hah+99]. Although this is known to be true in a biological context [LRD20], the precise relationship between vasculature density and tumour carrying capacity is not well defined, which makes the mathematical parameterization of this relationship difficult and subject to uncertainty. Here we employ Physics Informed Neural Networks (PINNs) to infer this critical parameter. PINNs incorporate domain-specific physical constraints to enhance the biological feasibility and predictive accuracy of mathematical models by integrating the models with empirical biological data [RPK19]. Inverse PINNs can be used to uncover unknown model parameters from sparse data. We employ an inverse PINN approach for parameter discovery, using clinical time series data, to quantify the relationship between vasculature density and solid tumour carrying capacity. We verify the predictive accuracy of our resulting model, and use in-silico data to explore implications for anti-angiogenic treatment protocols.

References

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CT1483 — Limiting resistance evolution through drug combinations

SPEAKERS Christin Nyhoegen (ETH Zurich, Theoretical Biology)
AUTHORS Christin Nyhoegen (ETH Zurich, Theoretical Biology); Hildegard Uecker (Max Planck Institute for Evolutionary Biology); Sebastian Bonhoeffer (ETH Zürich)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.46 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Combination therapy can be an effective strategy to limit the evolution and spread of antibiotic resistance, a major challenge for patient treatment worldwide. The administration of multiple drugs during treatment increases the genetic barrier to resistance, as (in the absence of cross-resistance) the bacteria would need to evolve resistance against each drug separately. Yet the success of this strategy ultimately depends on how well the combination controls the dynamics within the bacterial population. How should we thus combine antibiotics to limit the evolution and spread of resistance? We developed a stochastic pharmacodynamic model to determine the probability of population extinction for a given treatment. We compared the success of combination treatments differing in the number of drugs (including monotherapy), drug ratios, or types of drugs (e.g. mode of action or pharmacodynamic characteristics). Our results show that combination therapy is almost always better in limiting the evolution of resistance than monotherapy, but exceptions exist for drugs with steep dose–response curves. We further find combinations of drugs with specific modes of action that are particularly beneficial, and that the mode of action, as well as the pharmacodynamic properties, affect the choice of drug ratio. Our mathematical analysis helps to understand where the benefits of a specific strategy arise from and how to optimize treatment settings for a potential clinical use.

CT128 — Mathematical models for the treatment of leukemias with CAR-T cells alone and in combination with chemotherapy.

SPEAKERS Aitana Moncho Espí (Carlos III university/CEU San Pablo university)
AUTHORS Aitana Moncho Espí (Carlos III university/CEU San Pablo university); Juan Belmonte Beitia (Castilla-La Mancha university); Pilar Guerrero (Universidad Carlos III de Madrid)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

In this contribution, mathematical models describing the interaction between leukemic cells and CAR-T cells are developed and analyzed from the perspective of mathematical oncology. As a starting point, we consider the system of nonlinear ordinary differential equations proposed by [Pér+21], which describes

the joint dynamics of CAR-T and tumor cells by incorporating proliferation, exhaustion, and fratricide effects. Building on this framework, the model is extended by introducing an additional term representing CAR-T cell reinjection, allowing for the analysis of both continuous constant administration and impulsive reinjection schemes. The analysis focuses on the qualitative behavior of the system, including equilibrium stability, the occurrence of bifurcations, and the dynamic response under different therapeutic regimes. In particular, the study of the continuous reinjection case shows that, for suitable values of the parameter u,the system can stabilize at a state characterized by a low but persistent CAR-T cell population capable of eliminating small tumor reappearances and preventing relapse. For other parameter ranges, the model predicts the coexistence of CAR-T and leukemic cells, describing a scenario of chronic disease control rather than complete eradication, suggesting that continuous dosing may play a key role in the long-term maintenance of tumor control.

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CT181 — Non-ergodicity in ecology and evolution

SPEAKERS Teemu Kuosmanen (University of Helsinki)
AUTHORS Alexandre Minetto (University of Helsinki); Teemu Kuosmanen (University of Helsinki); Ville Mustonen (University of Helsinki)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.12 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Stochasticity plays an important role in all biological systems. The standard way to deal with stochasticity involves averaging over an ensemble of independent realizations. However, such mean statistics need not accurately reflect the typical outcomes in any finite sample unless the system satisfies the property of ergodicity, which guarantees that each trajectory will over time experience the same statistics as the entire ensemble. Here, we argue that, in contrast, non-ergodicity might instead be the rule rather than exception in real biological systems and investigate its implications for eco-evolutionary dynamics through three case-studies. First, we show how demographic stochasticity leads to ergodicity breaking where the asymptotic growth rate carries a signature of the initial condition. This motivates us to define a mutant establishment threshold, which quantifies a critical population size above which the typical mutant population starts to grow. Second, we consider environmental stochasticity and demonstrate that eco-evolutionary feedback can lead to non-ergodic dynamics, which has the important consequence that the fitness of a genotype cannot be simply averaged over the environments. Finally, we show how in a metapopulation structure the evolutionary dynamics within a typical subpopulation can deviate from the ensemble dynamics in the entire metapopulation, which is sufficient to explain the evolution and persistence of cooperation despite a fitness cost.

CT1296 — On the stability of a non-local kinetic model for density-dependent cell migration

SPEAKERS Francesco Maria Mori (University of Parma)
AUTHORS Francesco Maria Mori (University of Parma); Maria Groppi (University of Parma); Martina Conte (Politecnico di Torino)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Cell migration is regulated by the ability of cells to sense their surroundings over distances of several cell diameters and adjust both their direction and speed accordingly. We study the stability of a kinetic model for cell migration [LP20a] that incorporates such non-local sensing mechanisms. In the model, cell density is perceived through sensing kernels that may differ for directional and speed regulation. To investigate their combined effects, we assume that cell–cell adhesion influences cell orientation, while volume-filling effects regulate cell speed. By deriving the dispersion relation, we show that the stability of the homogeneous equilibrium depends on three dimensionless parameters: one measuring the stiffness of the system (related to the turning rate, equilibrium speed, and sensing radius), and two associated with the sensitivity of the speed and directional bias to changes in cell density. Extending previous studies [LP21; LP20b], we derive conditions for instability to both long- and short-wave perturbations in specific scenarios. Finally, numerical simulations are performed to illustrate the system's behaviour and the possible emergence of spatial patterns.

References

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CT240 — OP7, a promising influenza virus-derived interfering defective particle: multiscale modeling, role of the innate immune response and prediction of treatment windows

SPEAKERS Daniel Rüdiger (Max Planck Institute Magdeburg)
AUTHORS Patricia Opitz (Otto-von-Guericke University Magdeburg); Jan Küchler (Max Planck Institute Magdeburg); Sascha Y. Kupke (Max Planck Institute Magdeburg); Udo Reichl (Max Planck Institute Magdeburg, Otto-von-Guericke University Magdeburg); Daniel Rüdiger (Max Planck Institute Magdeburg)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 01.15 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Defective interfering particles (DIPs) are considered promising antiviral agents because they can strongly inhibit viral infections [DE14]. OP7, an influenza virus DIP with numerous point mutations, has demonstrated greater efficacy than other DIPs in animal models [Kup+19; Pel+23]. However, the impact of OP7 on human cells that induce an innate immune response (IIR) has not yet been evaluated. In this study, we examined the coinfection of influenza A virus (IAV) and OP7 in human lung cells *in vitro* to evaluate the impact of the IIR and identify possible application windows for patients. Using experimental and computational methods, we studied coinfection dynamics under different infection conditions. Based on experimental data, we developed a mathematical model that describes intracellular virus replication, the IIR, and infection spread between cells. We found that both low and high doses of OP7 significantly enhanced the IIR and reduced virus titers. The mathematical model closely described virus dynamics for all tested conditions. Model simulations predicted a therapeutic application window of up to 12 hours. Further, the model predicted that the IIR plays an important role at a low multiplicity of infection (MOI) but has a minor impact in high MOI settings. Subsequent experiments confirmed these predictions.

In summary, we developed a mathematical model that allows for a thorough analysis of IAV and OP7 coinfections and supports the development of antiviral applications.

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CT306 — Quantification of Information Flow by Dual Reporter System and Its Application to Bacterial Chemotaxis

SPEAKERS Kento Nakamura (RIKEN)
AUTHORS Kento Nakamura (RIKEN); Hajime Fukuoka (Osaka University); Akihiko Ishijima (Osaka University); Tetsuya J. Kobayashi (University of Tokyo)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mutual information is a theoretically grounded metric for quantifying information flow in cellular signaling pathways. However, its measurement requires characterization of the joint distribution of both input and output variables, which is data-intensive and often impractical, particularly when the input is high-dimensional, such as temporal trajectories of signaling molecules. Here, we present an alternative method that alleviates this requirement using dual reporter systems [Nak+26] — experimental setups where two conditionally independent reporter copies respond to a shared input signal [Elo+02]. By extending the extrinsic–intrinsic noise decomposition [Elo+02], we derive a mutual information estimator that eliminates the need to measure input distribution. We demonstrate our method on the bacterial chemotactic signaling pathway, where multiple flagellar motors within a single cell serve as natural dual reporters [Uch+22]. We validate the biological relevance of the measured information flow by comparing it with theoretical bounds on sensory information from optimal Bayesian filtering [NK21; Mat+21]. As modern single-molecule and single-cell measurement techniques increasingly enable simultaneous observation of multiple output copies, our method opens new directions for quantifying information flow across diverse biological systems.

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CT1274 — Real time response modelling for high pathogenicity avian influenza

SPEAKERS Chris Baker (The University of Melbourne)
AUTHORS Chris Baker (The University of Melbourne)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 10.11 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

The panzootic high pathogenicity avian influenza H5N1 2.3.4.4b (HPAI) has been circulating globally since 2021, causing wild bird and poultry mortality, alongside spillover into cattle and humans. As of early 2026, Australia remains free of HPAI, although it’s arrival is possibly a matter of when, rather than if. The ”HASTE project” has been developing decision tools and forecasting models to support evidence-based decisions in future animal health disease outbreaks, and, in early 2026, HASTE has developed HPAI forecasting models, in collaboration with the national government. Jointly we are testing these forecasting tools, alongside our capacity to deliver timely reporting, by entering the ”HPAI modelling challenge”, a global initiative to promote HPAI forecasting using a simulated challenge dataset. In this presentation I will describe our approach to the challenge and how we have adapted our forecasting tools for HPAI, and I will reflect on lessons for our preparedness for future outbreaks and our ability to provide decision-support modelling in animal health.

CT246 — Selective sweep probabilites in spatially expanding populations

SPEAKERS Alexander Stein (Universite Libre de Bruxelles & VIB-KU Leuven)
AUTHORS Kate Bostock; Maciej Bak; Ramanarayanan Kizhuttil; Alexander Stein (Universite Libre de Bruxelles & VIB-KU Leuven); Robert Noble
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 12.01 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Evolution during range expansions shapes biological systems from microbial communities and tumours to invasive species. A fundamental question is whether, when a beneficial mutation arises during a range expansion, it will evade clonal interference and sweep to fixation. However, most theoretical investigations of range expansions have considered regimes in which selective sweeps are effectively impossible, while studies of selective sweeps have assumed constant population size or ignored spatial structure. Here we use mathematical modelling and analysis to investigate selective sweep probabilities and timings in biologically relevant scenarios, including the case in which mutants can displace a slowly spreading wildtype. Assuming constant expansion speed, we find surprisingly simple approximate and exact expressions for sweep probabilities in one, two and three dimensions, which are independent of mutation rate. Agent-based simulations confirm that our predictions are accurate for the spatial Moran process and remain informative when mutation effects on fitness are random and multiplicative. We further compare and synthesise our results with those obtained for alternative growth laws. Parameterised for human tumours, our model predicts that selective sweeps are rare except during early solid tumour growth, thus providing a general, pan-cancer explanation for findings from recent sequencing studies.

CT1485 — Simulation of Dll-4 and Notch Dynamics in a Blood Vessel with Celular Rearrangement

SPEAKERS Tiago Gonçalves Pereira (University of Coimbra)
AUTHORS Marcos Gouveia (Center for Physics of the University of Coimbra); Rui Travasso (University of Coimbra); Tiago Gonçalves Pereira (University of Coimbra)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.05 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Sprouting angiogenesis is the process by which new blood vessels form through the emergence of multicellular sprouts led by endothelial tip cells and followed by stalk cells. Tip–stalk selection is largely regulated by Delta–Notch signalling, involving Notch receptors and the ligands Delta and Jagged. Delta–Notch interactions can occur within the same cell (cis), leading to mutual degradation of Notch and Delta, or between neighbouring cells (trans), which suppresses Delta production in the receiving cell via lateral inhibition. This feedback can generate contrasting cellular states, characterised by high Delta/low Notch (tip-like) or low Delta/high Notch (stalk-like) profiles. We investigate how lateral inhibition interacts with blood-flow-induced Delta/Notch ligand polarisation [Mac+17] in a rearranging endothelial monolayer. We employ a multi–phase-field model [Car23; Non12] coupled to Delta–Notch–Jagged dynamics [Boa+15], enabling endothelial cell rearrangement together with phenotype regulation. Our simulations indicate that flow-driven ligand polarisation, when coupled to endothelial cell neighbour exchanges, substantially modulates the emergent signalling patterns and can bias tip–stalk outcomes. These results suggest that mechanical cues mediated by flow, together with lateral inhibition, are key determinants of endothelial phenotype at the onset of sprout evolution.

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CT1265 — Simulation-based inference of epidemic and phylodynamic models via neural posterior estimation

SPEAKERS Francesco Pinotti (INRAE)
AUTHORS Francesco Pinotti (INRAE); Julien Theze (INRAE); Xavier Bailly (INRAE); Guillaume Fournié (INRAE)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 15.04 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Robust parameter inference is central to infectious disease modelling. Traditional Bayesian approaches require an analytical likelihood, which is rarely available for complex models. Classical likelihood-free methods circumvent this limitation through simulation but are inefficient and rely on hand-crafted summary statistics. Neural Posterior Estimation (NPE) is a scalable and flexible alternative that uses deep learning to learn the full posterior distribution [PM18]. NPE has shown promise in physics and neuroscience, but is underutilized in infectious disease epidemiology. Here, we evaluate its practical utility for real-world outbreak analysis.

We applied NPE to estimate key epidemiological parameters from two distinct data sources from the 2014 Ebola outbreak in Sierra Leone: (i) time series of reported cases and deaths, and (ii) a phylogeny reconstructed from early viral genome sequences. In both settings, NPE recovered accurate posterior distributions without the need to define any summary statistics.

Our findings demonstrate that NPE is a powerful, general-purpose inference framework for infectious disease modelling. Its flexibility, scalability, and integration within the Bayesian paradigm make it a compelling alternative to traditional inference tools. We advocate for a broader adoption of NPE across epidemiological and phylodynamic applications.

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CT392 — Spectral Parameterization of EEG Dynamics during Emergence from General Anesthesia

SPEAKERS Sandra Widmann (TU Munich)
AUTHORS Sandra Widmann (TU Munich); Alexander Edthofer (TU Wien); Andreas Körner (TU Wien); Gerhard Schneider (TU Munich); Paul García (Columbia University); Matthias Kreuzer (TU Munich)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 11.01 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Background: Neural power spectra contain an aperiodic $\frac{1}{f^{\alpha}}$ background and superimposed oscillatory peaks, but conventional band-power metrics do not separate these contributions. The FOOOF (Fitting Oscillations & One-Over-F) algorithm [Don+20] provides a parametric decomposition of both components, enabling analysis of spectral features associated with distinct physiological processes. Here, we apply this framework to electroencephalographic (EEG) recordings during emergence from general anesthesia.

Methods: We retrospectively analyzed frontal EEG recordings from 306 patients previously classified into six emergence trajectories based on alpha and delta power [Hes+19]. Absolute and relative alpha and delta power were calculated and the FOOOF algorithm was applied to extract the spectral exponent and alpha peak height. All spectral metrics were time-normalized to allow group-level comparisons.

Results: Mapping the emergence trajectories in the alpha-delta plane revealed trajectory-specific patterns, however, they lacked convergence at emergence endpoints. In contrast, FOOOF-derived parameters demonstrated a progressive flattening of the spectral exponent and a decrease in alpha peak height across all trajectories.

Conclusion: While alpha and delta power distinguish emergence trajectories, the spectral exponent consistently tracks neurophysiological transitions during anesthesia emergence, suggesting a trajectory-independent marker of awakening dynamics.

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CT1220 — The evolutionary fitness landscape of extrachromosomal DNA in cancer

SPEAKERS Magnus Haughey (Barts Cancer Institute)
AUTHORS Benjamin Werner (Barts Cancer Institute); Magnus Haughey (Barts Cancer Institute)
SCHEDULE Thursday, 16 July 2026; 14:00-14:20; 02.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Circular, megabase-size, episomal DNA termed extrachromosomal DNA (ecDNA) drive the evolution of about 20% of all tumours, promoting oncogene heterogeneity and therapy resistance [Tur+17; Kim+20; Nat+14]. While recent studies have elucidated its role in driving cell-to-cell phenotypic variation, little remains known about if, or how, the genomic structure of ecDNA itself evolves during tumour evolution. And yet, clinical observations suggest that it does [Lue+23; Noo+25]. But how does this impact tumour evolution? Here, we utilised computational modelling to show that ecDNA-driven tumours evolve upon a fitness landscape spanned by ecDNA abundance and size, and that tumours optimise their ecDNA genomic structure to drive fitter phenotypes. We employ this model to explain the inverse relationship between ecDNA abundance and size observed across a range of human tumours. EcDNA fitness landscapes are tissue- and oncogene-specific, with common oncogene-amplifying ecDNAs, such as *EGFR*-ecDNA in glioblastoma or *MYCN*-ecDNA in neuroblastoma, displaying strong patterns of ecDNA structural optimisation, unlike weaker oncogenic ecDNAs. We show that HeLa cells traverse this fitness landscape *in vitro* by losing superfluous passenger genes on *DHFR*-amplifying ecDNAs in order to develop resistance to methotrexate. Overall, this work demonstrates that structural evolution of ecDNA itself drives tumour evolution and provides a framework for understanding oncogenesis and predicting treatment responses.

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CT186 — Toward single-cell control: noise-robust perfect adaptation in biomolecular systems

SPEAKERS Dongju Lim (KAIST)
AUTHORS Byung-Kwan Cho (KAIST); Dongju Lim (KAIST); Jae Kyoung Kim (KAIST); Jinsu Kim (POSTECH); Jinyeong Kim (KAIST); Kangsan Kim (KAIST); Minjun Kim (POSTECH); Seokhwan Moon (University of Wisconsin-Maddison); Yun Min Song (IBS)
SCHEDULE Thursday, 16 July 2026; 14:00–14:20; 11.02 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Robust perfect adaptation (RPA), whereby a consistent output level is maintained even after a disturbance, is a highly desired feature in biological systems. This property can be achieved at the population average level by combining the well-known antithetic integral feedback (AIF) loop into the target network. However, the AIF controller amplifies the noise of the output level, disrupting the single-cell level regulation of the system output and compromising the conceptual goal of stable output level control. To address this, we introduce a regulation motif, the noise controller, which is inspired by the AIF loop but differs by sensing the output levels through the dimerization of output species. Combining this noise controller with the AIF controller successfully maintained system output noise as well as mean at their original level, even after the perturbation, thereby achieving noise RPA. Furthermore, our noise controller could reduce the output noise to a desired target value, achieving a Fano factor as small as 1, the commonly recognized lower bound of intrinsic noise in biological systems. Notably, our controller remains effective as long as the combined system is ergodic, making it applicable to a broad range of networks. We demonstrate its utility by combining the noise controller with the DNA repair system of Escherichia coli, which reduced the proportion of cells failing to initiate the DNA damage response [Lim+26].

References

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CT1562 — A model of adipose cell population dynamics under variable energy balance

SPEAKERS Aleksandra Tomaszek (LJLL, Sorbonne Université)
AUTHORS Aleksandra Tomaszek (LJLL, Sorbonne Université)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.02 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Adipocytes serve as an energy reservoir for the body thanks to their capacity to store and release lipids by adapting their size. Under energy surplus conditions, adipocytes store excess calories in the form of triglycerides through lipogenesis [LS22]. When existing cells reach storage capacity, new cells are recruited via pre-adipocyte differentiation [HB23]. During periods of energy deficit, adipocytes release the needed energy through lipolysis [LS22]. We propose a model incorporating transitions between two processes: lipogensis and lipolysis, as well as cell recruitment, depending on energy balance. We present a population dynamics model structured by the lipid content of the cell, formalized as an advection-diffusion partial

differential equation, with non-local nonlinearities on the velocity term and on the boundary condition. The non-local term reflects the energy balance at a given moment. It determines whether cells increase or decrease their lipid content, and its value determines the rate of both processes. Further, it governs the recruitment of new cells. We investigate model well-posedness in the general formulation and present its theoretical properties, as developed in [Tom26]. We then numerically explore the model, predicting the evolution of the adipose tissue cell size in response to a given diet. We show that, with appropriate term selection, model predictions qualitatively agree with the measured data in rats [Jac+14].

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CT347 — Control of electrophysiology experiments in open and closed loop

SPEAKERS Melvyn Tyloo (Living Systems Institute)
AUTHORS Melvyn Tyloo (Living Systems Institute)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 11.11 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mathematical modelling is the primary tool to predict and control the evolution of complex natural and engineered systems. However, sometimes many different models might predict similar behavior. This might typically happen when the microscopic rules governing an observed phenomenon are not known, and one then has to rely on effective, empirical models. In this talk, I will introduce an iterative approach leveraging optimal control that aims at selecting the best model to predict the evolution of a reference system. I will illustrate the outcome of the closed-loop algorithm when the reference system is numerically simulated. Then, I will show an implementation of the algorithm on an electrophysiology experiment, where we compared different models of the photocurrent response of cells made sensitive to light by optogenetics. To conclude the talk, I will move to an open loop framework that aims at quantifying the uncertainty when one controls such experiments assuming a mathematical model.

CT16 — Coupled Information-Infection Dynamics Produce Information-Driven Shifts in Epidemic Regimes via Competing Vaccine Narratives

SPEAKERS Asma Azizi (Kennesaw State University)
AUTHORS Asma Azizi (Kennesaw State University); Caner Kazanci (University of Georgia)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 10.01 - HS
SESSION Contributed Talks
TRACK Miscellaneous

Abstract

Vaccine-related communication can help curb infection, yet it can also depress vaccine uptake and sustain transmission even when effective vaccines exist. We examine how vaccine-information dynamics shape epidemic spread and how they can be steered to support control. We propose a coupled information–infection framework that links a standard SVIRS epidemic model to an integrate-and-fire-inspired dissemination model. Vaccine-positive and vaccine-critical informants spread competing narratives that influence hesitant individuals through threshold-based acceptance and persistence of engagement, while infection prevalence feeds back by heightening caution. These evolving attitudes modulate vaccination uptake, and the model captures the joint evolution of information flows and epidemic trajectories. Our analysis shows that information dynamics can shift epidemic regimes and determine long-term outcomes. Notably, strengthening vaccine-positive engagement (by lowering resistance to pro-vaccine messages and sustaining participation) delivers larger and more reliable infection-control benefits than strategies centered on suppressing vaccine-critical voices. The results underscore information governance as a core component of epidemic control and suggest pairing biomedical interventions with approaches that cultivate durable, vaccine-positive engagement to prevent resurgence and support sustained containment.

CT234 — Eco-Epidemic Reaction–Diffusion Model with Fear and Carryover Effects: Stability and Pattern Formation

SPEAKERS Namrata Mani Tripathi (Indian Institute of Technology (Indian School of Mines) Dhanbad, Jharkhand, 826004 India.)
AUTHORS Namrata Mani Tripathi (Indian Institute of Technology (Indian School of Mines) Dhanbad, Jharkhand, 826004 India.)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 03.01 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Eco-epidemic systems offer a fundamental framework for examining the interaction between predator–prey dynamics and infectious disease transmission. Motivated by the European rabbit–Iberian lynx system, we extend the model in [Upa+16] by incorporating a nonlinear predator-induced fear function that captures immediate behavioral responses and memory-driven carryover effects [WZZ16; OCo+14]. The resulting reaction–diffusion model describes susceptible prey, infected prey, and predator populations under zero-flux boundary conditions. In the absence of diffusion, the temporal system exhibits saddle-node, Hopf, and cusp bifurcations with respect to the fear parameter. The analysis reveals contrasting roles of fear and carryover: increasing fear can destabilize the coexistence equilibrium and generate oscillatory outbreaks, whereas stronger carryover effects enhance stabilization and species persistence. Analytical results on local and global stability are supported by simulations. With diffusion, the system undergoes Turing instability, producing spatiotemporal structures including clustered patterns, spirals, and wave-like chaotic dynamics. Lower levels of fear and carryover amplify spatial heterogeneity and disease spread. These findings demonstrate that predator-induced fear and its carryover effects shape ecosystem stability, pattern formation, and epidemiological outcomes, providing insight into fear-mediated eco-epidemic dynamics.

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CT1341 — Exploiting acid-dependent 'public bad' dynamics to develop normalizing adaptive therapies

SPEAKERS Kevin Scibilia (Moffitt Cancer Center)
AUTHORS Alexander Anderson (Moffitt Cancer Center); Kevin Scibilia (Moffitt Cancer Center); Kit Gallagher (Harvard Medical School); Maximilan Strobl (The Institute of Cancer Research and Imperial College London); Mehdi Damaghi (Associate Professor); Nathan Schofield; Philip K. Maini; Rafael Bravo; Robert Gatenby
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 02.23 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

The current cancer treatment paradigm of administering a 'maximum tolerated dose' to patients fails to account for the eco-evolutionary dynamics that drive disease relapse. Adaptive therapy has shown clinically encouraging results in controlling tumors by modeling patient-specific evolutionary dynamics, based on the premise that resistance comes at a cost and that sensitive cells can suppress resistant cell growth through competition. In this study, we have constructed agent-based and ordinary differential equation models that recapitulate in vitro experiments of doxorubicin-sensitive and resistant MCF7 cell lines. We show the cost of resistance depends on the microenvironmental pH that can be manipulated via acid-normalizing agents (such as buffer therapy). We find that drug-resistant cells are more dependent on anaerobic glycolysis and consequently produce more acid, acting as a 'public bad' that inhibits the growth of the drug-sensitive population more than the drug-resistant subpopulation. Model simulations showed the impact of buffer therapy on the cost of resistance and on cell competition dynamics, allowing us to infer optimal treatment schedules of buffer combined with chemotherapy that rescue the drug-sensitive population. Overall, this study showed the importance of treating both the tumor and its microenvironment to develop model-informed evolutionary therapies, extending the traditional modeling paradigm of adaptive therapy beyond pure cell competition.

CT1319 — Genetic Switching Dynamics in a Spatial Branching Model of Cancer Metastasis

SPEAKERS Lena Zuspann
AUTHORS Lena Zuspann; Maria Bruna (University of Oxford); Ruth Baker (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Biologically, metastatic spread refers to the process by which a primary tumour releases cancer cells into the bloodstream that eventually colonise new organs, thereby often acquiring genetic mutations. Metastases cause about 80% of cancer-related deaths [Fra+19], hence, predicting when and where metastases are likely to emerge, and identifying advantageous mutations presents an urgent medical challenge. Mathematically, modelling metastatic spread is exceptionally difficult because it encompasses multiple temporal and spatial scales, is affected by both collective as well as rare events, and is governed by genetic evolution processes. Previous modelling efforts have made significant progress in each of these areas but remain mostly exclusive from one another [SJ25; WH25].

My research aims at bridging this gap by considering a time-continuous spatial branching process tracking both the genetic genealogy of cancer cells and their spatial trajectories during dissemination from a primary tumor to metastatic sites, linking population-level spread with underlying stochastic genetic dynamics [BTZ25]. The genetic component is formulated as a dynamical system describing phenotypic switching from healthy to cancerous states, which is analyzed systematically by considering different approximations of an appropriate reduced model. Finally, extensions to more complex genetic switching networks relevant to metastatic initiation are discussed.

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CT1470 — Geometric analysis of an epidemiological model for Olive Quick Decline Syndrome (OQDS)

SPEAKERS Keren Tapper (University of Edinburgh)
AUTHORS Nikola Popovic (University of Edinburgh); Mariya Ptashnyk (Heriot-Watt University); Keren Tapper (University of Edinburgh)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.46 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Olive Quick Decline Syndrome (OQDS), caused by the bacterium Xylella fastidiosa, is a vector-borne disease affecting olive groves across Europe, severely impacting areas of southern Italy. We consider an ODE model for the olive tree canopies, the vector insects, and the weeds. Under the assumption that the insects reproduce significantly faster than the trees and weeds, the model equations are singularly perturbed and can then be analysed via the dynamical systems-based geometric singular perturbation theory. This allows us to reduce the long term dynamics of the model solutions to the dynamics of a globally attracting slow manifold. We extend the model by introducing a Heaviside cut-off to the spread of the disease cross terms and regularise by applying the desingularisation technique, know as "blow-up".

CT1536 — How group communication changes under competition

SPEAKERS Gayashan Jayavilal (University of Cincinnati)
AUTHORS Gayashan Jayavilal (University of Cincinnati); Hyunjoong Kim (University of Cincinnati)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 11.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Communication plays a fundamental role in collective behavior among social animals. In honeybees, waggle dances recruit nestmates to profitable food sources, allowing colonies to coordinate foraging effort. However, natural environments often contain multiple colonies competing for the same limited resources, raising the question of how recruitment communication adapts under inter-colony competition.

We develop a mathematical model of collective foraging that describes the interaction of competing honeybee colonies exploiting shared resources. The model is formulated as a system of nonlinear differential equations capturing recruitment dynamics, resource exploitation, and communication-driven feedback in

forager allocation. Analytical and numerical approaches are used to investigate equilibrium structure and dynamical regimes arising from variation in communication intensity and resource availability.

Our analysis identifies parameter regions leading to competitive dominance, stable coexistence, or destabilization of recruitment strategies. In particular, nonlinear recruitment feedback generates threshold effects and transitions between qualitatively different dynamical behaviors. These results provide insight into how communication efficiency shapes competitive outcomes among social foragers and offer a mathematical framework for studying collective decision-making under ecological competition.

CT1293 — Identical ancestor points and maximal specieslike clusters

SPEAKERS Samuel Alexander
AUTHORS Samuel Alexander
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 11.32 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

We introduce the so-called identical ancestor point axiom, a formalization (under the simplifying assumption that life does not go extinct) of Hennig's non-splitting criterion for species. This axiom states that for any organism in any species, either the species contains at most finitely many descendants of that organism, or else the species contains at most finitely many non-descendants of that organism. We argue this axiom is plausible for species. We show that, together with a mild convexity axiom, this reduces the subjective nature of species definition. We call connected sets satisfying these two axioms "specieslike clusters." We consider the question of identifying a set of biologically plausible constraints that would guarantee every organism inhabits a maximal specieslike cluster subject to those constraints. We provide one such set consisting of two constraints, and show that no proper subset thereof suffices. Finally, we sketch two new applications of these ideas to biology.

CT1441 — Integrating Mechanistic Modeling and Machine Learning to Predict CAR-T Therapy Outcomes from CD4+/CD8+ Dynamics

SPEAKERS Saranya Varakunan (University of Waterloo)
AUTHORS Melissa Stadt (University of Waterloo); Mohammad Kohandel (University of Waterloo); Saranya Varakunan (University of Waterloo)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Chimeric antigen receptor (CAR) T cell therapy has shown remarkable success in hematological malignancies, yet patient responses remain highly heterogeneous. Clinical evidence suggests that the CD4:CD8 T cell ratio in CAR-T cell treatments influences therapeutic outcomes [Gal+23], but the quantitative roles of these subsets in shaping treatment dynamics are incompletely understood. Building on a tumor-regulated effector-exhaustion CAR-T cell framework [Kir+23], we develop a mechanistic model that explicitly represents CD4+ helper and CD8+ cytotoxic T cell populations and their interactions with tumor burden [Bou+21].

Due to the absence of detailed clinical datasets, we evaluate the model using virtual patient simulations; we show that our model reproduces qualitative clinical trends in CAR-T expansion and treatment response at varying CD4:CD8 ratios, while also revealing substantial inter-patient variability. We then identify key determinants of therapeutic outcome using sensitivity analyses, and assess predictive performance using these determinants (i.e. how measurement errors for important parameters affect model prediction).

We show that a simple feedforward neural network improves the model's predictive decay under noise, highlighting the potential of hybrid mechanistic and machine learning approaches for forecasting CAR-T therapy outcomes.

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CT199 — Interaction-based networks describe chemical pathways within proteins

SPEAKERS Fabian Schuhmann (Niels Bohr Institute, University of Copenhagen)
AUTHORS Fabian Schuhmann (Niels Bohr Institute, University of Copenhagen); Heloisa Bordallo (Niels Bohr Institute, University of Copenhagen); Weria Pezeshkian (Niels Bohr Institute, University of Copenhagen)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.21 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Molecular dynamics simulations have been remarkably effective for observing and analyzing structures and dynamics of proteins, with longer trajectories being computed every day. Still, often, relevant time scales are not observed. Adequately analyzing the generated trajectories can highlight the interesting areas within a protein such as mutation sites or allosteric hotspots, which might foreshadow dynamics untouched by the simulations. We employ a physics-based protein network and propose that such a network can adequately analyze the protein dynamics. The analysis is conducted on simulations of cathepsin G and neutrophil elastase, which are remarkably similar but with different specificities. However, a single mutation in cathepsin G recovers the specificity of neutrophil elastase. The physics-based network built on the interactions between residues instead of the distances can pinpoint the active triad in the proteins studied. Overall, the network seems to capture the structural behavior better than purely distance-based networks.

CT398 — Invariant Image Reparameterisation for Identifiability Analysis of Individual-Based Models

SPEAKERS Oliver Maclaren (The University of Auckland)
AUTHORS Cameron Walker (The University of Auckland); Oliver Maclaren (The University of Auckland); Rochelle Constantine (The University of Auckland)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Advances in data collection and simulation-based inference now enable fitting complex individual-based models (IBMs) to rich, fine-grained observations of interacting systems. However, mere data fitting is not

understanding: these methods do not clarify which model quantities are truly constrained by the data and which are fundamentally underdetermined. This is the problem of identifiability.

Identifiability analysis for complex, heterogeneous IBMs remains a fundamental open problem. Heterogeneous IBMs are particularly challenging due to intractable likelihoods and emergent, context-dependent behaviour. Our recent Invariant Image Reparameterisation (IIR) method, and the related Profile-Wise Analysis (PWA), provide a theoretical foundation for tackling identifiability in heterogeneous IBMs, but require an auxiliary mapping linking data features to mechanistic parameters.

Here, we present work on constructing such auxiliary mappings for IBMs and investigate what IIR can tell us about identifiability in these models.

A challenging real-world example of collective behaviour motivates our work: multi-predator feeding aggregations (MPFAs) in the Hauraki Gulf, New Zealand. We show the kinds of drone-collected data available for this application and present preliminary steps for constructing auxiliary maps from such data.

CT279 — Mathematical Modeling of Tertiary Lymphoid Structure Development and Stage Progression in Chronic Kidney Disease

SPEAKERS Jinghao Chen (Kyoto University)
AUTHORS Takahisa Yoshikawa (Kyoto University); Naoya Toriu (Kyoto University); Steffen Plunder (Kyoto University); Matoko Yanagita (Kyoto University); Sungrim Seirin-Lee (Kyoto University); Jinghao Chen (Kyoto University)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 01.15 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Tertiary lymphoid structures (TLSs) are inducible ectopic lymphoid structures that emerge under chronic inflammatory conditions, including in the kidneys of patients with autoimmune diseases, and are closely associated with poor renal outcomes. Recent studies have classified TLS development into three stages correlated with chronic kidney disease severity. However, the mechanisms governing TLS initiation and stage transitions in the kidney remain unclear. To address this, we developed an interdisciplinary approach combining mathematical modeling with *in vivo* experimental data from a mouse model of kidney injury to capture immune cell interactions during TLS formation. The model tracks the dynamics of injured proximal tubular cells, lymphocytes, inflammatory fibroblasts, follicular dendritic cells, and other key populations. Using this framework, we found that T cell recruitment is mediated by distinct mechanisms across TLS stages: it is initiated by injury-associated signals in early stages and later sustained by components within the developing TLS structure. In addition, *in silico* sensitivity analysis identified stage-specific key regulators that may serve as potential therapeutic targets. Overall, our findings provide new insights into stage-dependent TLS development and offer a theoretical framework to guide future experimental and clinical studies.

CT1216 — Mathematical modelling of 5-aminolevulinic acid in glioblastoma

SPEAKERS Charlotte Baxter (University of Nottingham)
AUTHORS Charlotte Baxter (University of Nottingham)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 02.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Glioblastoma (GBM) is a highly invasive brain tumour with a 5-year survival rate below 5%. The first step of treatment for GBM is surgery with the goal of removing the entire visible tumour. However, even when all the visible tumour is removed, it almost always recurs leading to the cancer's poor prognosis. Margins between tumour tissue and healthy tissue can appear unclear during surgery. To aid in identification of tumour tissue 5-ALA is consumed prior to surgery. This leads tumour cells to accumulate protoporphyrin IX (PpIX) causing tumour tissue to fluoresce under UV light during surgery improving identification and the extent of resection during surgery. 5-ALA is converted to PpIX in cells as part of the heme biosynthesis pathway. However, the biological reason for the accumulation of PpIX in tumour cells and not healthy cells is unclear. To better understand this behaviour, we develop and analyse a mathematical model of a simplified heme biosynthesis pathway consisting of a system of ODEs. A key part of the analysis of this model is parameter sensitivity and identifiability to allow us to consider model fits to data and compare the fits for different cell type. We then compare this model to data in the literature and use RNA sequencing data to understand the relationship between the parameters and gene expression in fluorescent and non-fluorescent GBM tissue. This model can then be combined with a model of tumour growth to predict 5-ALA induced fluorescence in a tumour.

CT135 — Mechanisms of Synergy and Resistance: Modeling Dynamic PD-L1 Regulation in Combination Immunotherapy

SPEAKERS Bruce Pell (Lawrence Technological University)
AUTHORS Aigerim Kalizhanova (Nazarbayev University); Aisha Tursynkozha (Astana IT University); Ardak Kashkynbayev (Nazarbayev University); Bruce Pell (Lawrence Technological University); Denise Dengi (University of California Berkeley); Yang Kuang (Arizona State University)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

The efficacy of Immune Checkpoint Inhibitors (ICIs) is frequently limited by adaptive immune resistance via the PD-1/PD-L1 pathway. While pairing ICIs with potent immunostimulants (such as NHS-muIL12) offers a strategy to overcome this, foundational mathematical models often struggle to capture complex preclinical behaviors, specifically non-monotonic tumor responses. In this talk, we introduce a refined deterministic model that addresses this limitation by incorporating PD-L1 expression as a fully dynamic variable rather than a static parameter. We demonstrate that this model, using a single parameter set, successfully reproduces experimental data across six distinct treatment arms, including monotherapies and combinations. This framework provides a robust mechanistic explanation for dose-dependent treatment failure driven by adaptive PD-L1 upregulation, while also elucidating the drivers of therapeutic synergy. We will present the systematic development of the model and simulation results.

CT1281 — Modeling Administration Time-Of-Day Effects in Clinical Research: A Simulation Study with Application to Cancer Chronotherapy

SPEAKERS Sacha Revillon (Institut Curie, Cancer Systems Pharmacology)
AUTHORS Annabelle Ballesta (Institut Curie, INSERM U1331, Cancer Systems Pharmacology team); Jimmy Mullaert (Institut Curie, Statistical Methods for Precision Medicine); Sacha Revillon (Institut Curie, Cancer Systems Pharmacology)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 02.21 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Administration time-of-day may modulate antitumor treatment efficacy both in preclinical and clinical studies, likely due to the circadian regulation of physiological processes [Fey+25; Lév+24]. However,

appropriate biostatistical methods to analyze such periodic variables in clinical studies are lacking. This project aims to identify robust survival analysis methods integrating treatment timing into chronotherapy studies.

Starting from real-world data [Cat+24], we conducted a simulation study to evaluate statistical approaches (discrete, sinusoidal, and spline-based models) for modeling the effect of administration timing on patient survival. Simulation scenarios examined the combined impact of (i) cohort size, (ii) observation time windows and (iii) potential confounding factors on model performance, assessed through likelihood ratio tests or Wald delta method–based tests for sinusoidal models. First, we observed a limited ability of discrete methods to detect circadian effects. Moreover, we showed that the delta method could falsely detect such effects (inflated type 1 error) and overestimate their amplitude. Next, all methods lacked sufficient power to detect interaction effects with patient characteristics for cohort sizes typically encountered in clinical trials (e.g 100-300 patients), advocating for large trials or meta-analysis. Overall, these results guide the choice of the most appropriate methods to study patient survival's dosing time dependencies.

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CT191 — Modeling circadian–contraceptive interactions: Simulations reveal daytime dosing as optimal for pill efficacy

SPEAKERS Brenda Lyn Gavina (University of the Philippines Diliman)
AUTHORS Brenda Lyn Gavina (University of the Philippines Diliman); Taeyong Lee (Yonsei University); Olive Cawiding (Korea Advanced Institute of Science and Technology); Sunhwa Choi (National Institute for Mathematical Sciences); Sungwook Choi (M Fertility Clinic); Soyoung Kim (National Institute for Mathematical Sciences); Jae Kyoung Kim (Institute for Basic Science)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 11.02 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Oral contraceptive pills remain widely used for both birth control and the management of reproductive disorders. However, side effects such as thrombosis and myocardial infarction continue to raise concerns, even with current formulations. While formulation improvements persist, alignment of drug administration with circadian rhythms – proven to enhance safety and efficacy in other drug classes - remains underexplored in reproductive health. To address this gap, we developed a mathematical model of hormonal contraception incorporating circadian rhythms of key reproductive hormones (LH, FSH, E_2, and P_4). A drug pharmacokinetic component was added to simulate how dosing time affects contraceptive action. Simulations revealed that contraceptive efficacy varies with timing of intake, with daytime dosing consistently providing stronger suppression of ovulatory signals compared to evening dosing. This effect arises from alignment between the drug's concentration peak and the LH production peak. These findings

offer a mechanistic basis for time-of-day–specific contraceptive recommendations, which may enhance both the safety and efficacy of hormonal contraception.

CT1455 — Modeling the Geospatial Dynamics of Lyme Disease in Maryland Under Projected Climate Scenarios

SPEAKERS Salihu Musa (Department of Mathematics, University of Maryland, College Park, MD, 20742, USA)
AUTHORS Salihu Musa (Department of Mathematics, University of Maryland, College Park, MD, 20742, USA)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.04 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Lyme disease, the most prevalent vector-borne disease in the United States, has been expanding across Maryland, with rising temperatures accelerating its spread. Transmission of Borrelia burgdorferi by Ixodes scapularis ticks is highly sensitive to climatic variables. This study develops a climate-driven epidemiological model to investigate the spatiotemporal dynamics of Lyme disease in Maryland. Integrating fine-scale ecological data with temperature-dependent vector-host interactions, we assess the impact of climate change on tick population dynamics, seasonal activity shifts, and transmission intensity under Representative Concentration Pathways (RCP 4.5 and 8.5). Results project substantial expansion of Lyme disease across Maryland counties, driven by increased tick activity and host population growth. We find significant increases in disease incidence, basic reproduction number (R_0), and infected nymphal density, with the most pronounced effects in Central and Western Maryland under RCP 8.5. Spatial analyses reveal a northward shift of high-risk zones. Further analysis demonstrates that environmental clearance and rodent-targeted tick suppression can reduce transmission risk, with R_0 declining significantly when intervention compliance exceeds 50%. These findings emphasize the need for climate-adaptive vector management and provide a framework for evidence-based Lyme disease prevention.

CT1215 — Novel methods for analyzing dynamics of functional brain networks: complex correlation patterns defining a robust functional architecture

SPEAKERS Maria Ercsey-Ravasz (1. Physics Department of the Babes-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania)
AUTHORS Antonio Diaz-Para (Universitat Politecnica de Valencia); Balazs Pentek (1. Physics Department of the Babes-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania); Botond Molnar (Babes-Bolyai Universiy, Mathematics and Computer Science Department); David Moratal (Universitat Politecnica de Valencia); Laura Perez-Cervera (Instituto de Neurociencias, San Juan de Alicante); Levente Varga (Babes-Bolyai Universiy, Mathematics and Computer Science Department); Maria Ercsey-Ravasz (1. Physics Department of the Babes-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania); Mohamed Kotb Selim (Instituto de Neurociencias, San Juan de Alicante); Raul C Muresan (Transylvanian Institute of Neuroscience); Santiago Canals (Instituto de Neurociencias, San Juan de Alicante); Vasile Vlad Moca (Transylvanian Institute of Neuroscience); Wolfgang Sommer (Central Institute of Mental Health Mannheim)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 11.01 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Functional magnetic resonance imaging (fMRI) provides insights into cognitive processes with significant clinical potential. However, delays in brain region communication and dynamic variations are often overlooked in functional network studies. In our [Var+24] paper we demonstrated that networks extracted from fMRI cross-correlation matrices considering time lags between signals show remarkable reliability when focusing on statistical distributions of network properties, instead of analyzing a single averaged functional network. This reveals a robust brain functional connectivity pattern, featuring a sparse backbone of strong

0-lag correlations and weaker links capturing coordination at various time delays. This dynamic yet stable network architecture is consistent across rats, marmosets, and humans, as well as in electroencephalogram (EEG) data, indicating potential universality in brain dynamics. Validation using alcohol use disorder fMRI data uncovers broader shifts in network properties than previously reported, demonstrating the potential of this method for identifying disease biomarkers. We also have freely available software [25] for analyzing datasets: extracting functional networks, examining their properties, comparing different groups, extracting the most important functional regions in case of a comparison, and more.

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CT218 — Opposites attract – the role of electrostatic polarisation in the sensory ecology of arthropods

SPEAKERS Ryan Palmer (University of Bristol, UK)
AUTHORS Beth Harris (University of Leeds, UK); Daniel Robert (University of Bristol, UK); Fraser Woodburn; Isaac Chenchiah (University of Bristol, UK); Liam O'Reilly; Ryan Palmer (University of Bristol, UK); Samuel England (Museum für Naturkunde Berlin); Samuel Harris (University of Bristol, UK)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.05 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Arthropods possess a variety of novel senses to acquire environmental and biotic cues, with recent research reveals the possibility for arthropods to detect and respond to naturally occurring electrical fields. In this talk, I will present a suite experiments and models that show the role that this electrical sense plays in the sensory life and ecology of terrestrial arthropods. Our latest results reveal how the mechanism of charging fundamentally changes the biomechanics of arthropod electrical sensing in spiders, caterpillars and treehoppers, revealing new sensory niches for this modality due to electrostatic polarisation and induction. Hence, we will discuss (i) the biophysical mechanisms that enable this electrosense, (ii) the typical electrical signals experienced in nature, and (iii) the possible effect of electrical fields on other senses and behaviours. Here, I will touch on several of these aspects, giving an overview of our knowledge to date, but also the enticing scientific and mathematical problems that remain.

CT341 — Optimal control of symmetry-breaking in cell fate specification

SPEAKERS Pearson Miller (University of California, San Diego)
AUTHORS Pearson Miller (University of California, San Diego)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 05.12 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

A classic model of a cell fate transition, following on Waddington, treats distinct cell phenotypes as attractors emerging from the nonlinear dynamics of biochemical signaling pathways. Transitions between

phenotypes are achieved by external modulation of the system, which produces bifurcations among the stable states and otherwise drives the system from one attractor to another. This talk presents efforts to apply optimal control theory to derive new, universal results on the response of such systems to upstream morphogen signals. We pose an optimal control problem in which a system near a pitchfork bifurcation is driven to a target state with minimal effort. Optimality conditions are derived using the Pontryagin Maximum Principle and then studied asymptotically. Across different scaling regimes, we assess the applicability of the standard center-manifold reductions to this class of systems, and, under certain soft constraints on control effort, we construct analytical optimal feedback laws that match the lowest-order behavior. These results are validated numerically for both single-cell and spatially extended models.

CT280 — Optimal strategies for utilizing host plant distributions to slow the spread of plant pests

SPEAKERS Adam Lampert (Robert H. Smith Faculty of Agriculture, Food and Environment, The Hebrew University of Jerusalem, Israel)
AUTHORS Adam Lampert (Robert H. Smith Faculty of Agriculture, Food and Environment, The Hebrew University of Jerusalem, Israel); Andrew Liebhold (USDA Forest Service Northern Research Station, Morgantown, WV, USA)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 12.01 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Invasive species are spreading globally, threatening ecosystems, biodiversity, agriculture, and human health. When efforts to prevent the establishment of invasive species fail and eradication is not possible, containment becomes necessary to slow or stop the spread of established invaders. A major question is, therefore, how to allocate treatments across space and time to contain populations cost-effectively. Here, we examine how to optimize strategies for slowing the spread of the spongy moth (Lymantria dispar) in North America by utilizing gaps with lower densities of host plants. Around these gaps, managers can apply both pesticides and mating disruption using synthetic pheromones to disrupt male mate finding. We develop a spatially explicit model of the moth's population dynamics, and we develop a novel algorithm that finds the optimal spatial allocation of the two methods across landscapes with heterogeneous host-plant distributions. Our results show that combining pesticide application and mating disruption around host-plant gaps significantly improves cost efficiency: Optimal treatment allocates mating disruption to low moth-density regions nearer the moth-free area and pesticides to higher moth-density regions nearer the invasion front, with minimal spatial overlap in treatments. Using this rule, containment can be made markedly more cost-effective by prioritizing landscape features that naturally impede spread.

CT1511 — Pharmacokinetic-Based Optimization of Dynamic PET Time Acquisition Protocols for Cancer Theranostics

SPEAKERS Diego Samuel Rodrigues (School of Technology, State University of Campinas)
AUTHORS Diego Samuel Rodrigues (School of Technology, State University of Campinas); Letícia Fernanda Alves (PhD Program in Applied Mathematics, State University of Campinas); Sérgio Querino Brunetto (Biomedical Enginnering Center, State University of Campinas); Ernesto Augusto Bueno da Fonseca Lima (TACC / The Center for Computational Oncology, Oden Institute, University of Texas at Austin); Carmen Silvia Passos Lima (School of Medical Sciences, State University of Campinas); Bárbara Juarez Amorim (Nuclear Medicine Service, Clinical Hospital, State University of Campinas); Celso Dario Ramos (School of Medical Sciences, State University of Campinas)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 01.22 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Dynamic positron emission tomography (dPET) enables the estimation of pharmacokinetic parameters from time-activity curves, providing quantitative information relevant to cancer theranostics [Tak+19]. However, its clinical adoption remains limited, partly due to the lack of optimized acquisition protocols. Here, we optimize dPET acquisition using the theory of design of experiments applied to the human pharmacokinetics of ^{18}F-FDG. Tracer kinetics are modeled with an irreversible two-tissue, three-compartment model [Rod+24] with observables C_b and $(C_f + C_p)$, where C_b, C_f, and C_p denote radiotracer concentrations in blood and target tissue (free and phosphorylated). As in [Liu+22], 97 acquisition points are considered over 75 minutes, with a minimum interval of 5 seconds, while allowing the time points to be freely optimized. Time-moment optimization is performed under D-, A-, and E-optimality criteria using nominal k_1, k_2, and k_3 values for nine organs (grey matter, white matter, cerebellum, thyroid, myocardium, liver, spleen, pancreas, and kidney). Our analysis revealed that these 9 organs cluster into three kinetic classes (slow, intermediate, fast), and our optimal designs consistently show that the empirical protocol from [Liu+22] is not optimal, offering a novel framework for dPET protocol development. The authors thank the funding from São Paulo Research Foundation (FAPESP): #2020/05556-0, #2024/21475-1, #2021/10265-8 (CEPID CancerThera).

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CT182 — Quantifying fitness, selection and survival in population trees with cell death

SPEAKERS Arthur Genthon (Max Planck Institute for the Physics of Complex Systems, Dresden, Germany)
AUTHORS Arthur Genthon (Max Planck Institute for the Physics of Complex Systems, Dresden, Germany); David Lacoste (Gulliver UMR CNRS 7083, ESPCI Paris, Université PSL); Luca Peliti (Santa Marinella Research Institute); Takashi Nozoe (The University of Tokyo)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.12 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Cell lineage statistics is a powerful tool for inferring cellular parameters, such as division rate, death rate, or population growth rate. Yet, in practice such an analysis suffers from a basic problem: how should we treat incomplete lineages that do not survive until the end of the experiment? In this talk, I will introduce a model-independent theoretical framework to address this issue. This framework provides practical definitions of fitness landscape, survivor bias, and selection strength for arbitrary cell traits from cell lineage statistics in the presence of death. Analyses of experimental data where a cell population is exposed to a drug that kills a large fraction of the population reveals that failing to properly account for dead lineages can lead to misleading fitness estimations. For simple trait dynamics, the fitness landscape and the survivor bias can in addition be used for the nonparametric estimation of the division and death rates, using only lineage histories. Finally, this framework provides universal bounds on the population growth rate, and a fluctuation-response relation that quantifies the change in population growth rate due to the variability in death rate.

References

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CT322 — Semi-amortised simulation-based inference for stochastic nonlinear mixed-effects models

SPEAKERS Henrik Häggström (Department of Mathematical Sciences, Chalmers University of Technology and the University of Gothenburg)
AUTHORS Henrik Häggström (Department of Mathematical Sciences, Chalmers University of Technology and the University of Gothenburg); Marija Cvijovic (Department of Mathematical Sciences, Chalmers University of Technology and the University of Gothenburg); Sebastian Persson (Francis Crick Institute); Umberto Picchini (Department of Mathematical Sciences, Chalmers University of Technology and the University of Gothenburg)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The analysis of data from multiple experiments, such as observations of several individuals, is commonly approached using mixed-effects models, which account for variation between individuals through hierarchical representations. This makes mixed-effects models widely applied in fields such as biology, pharmacokinetics, and sociology. In this work [Häg+26], we propose a novel methodology for scalable Bayesian inference in hierarchical mixed-effects models. Our framework first constructs amortized approximations of the likelihood and the posterior distribution, which are then rapidly refined for each individual dataset, to ultimately approximate the parameters posterior across many individuals. The framework is easily trainable, as it uses mixtures of experts but without neural networks, leading to parsimonious yet expressive surrogate models of the likelihood and the posterior. We demonstrate the effectiveness of our methodology using challenging stochastic models, such as mixed-effects stochastic differential equations emerging in systems biology-driven problems. However, the approach is broadly applicable and can accommodate both stochastic and deterministic models. We show that our approach can seamlessly handle inference for many parameters. Additionally, we applied our method to a real-data case study of mRNA transfection. When compared to exact pseudomarginal Bayesian inference, our approach proved to be both fast and competitive in terms of statistical accuracy.

References

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CT1350 — Stability in asymmetric evolutionary games

SPEAKERS Aradhana Narang (BML Munjal University)
AUTHORS A. J. Shaiju (Indian Institute of Technology Madras); Aradhana Narang (BML Munjal University)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Several game-theoretic models such as oligopoly, war of attrition, and international trade, are inherently asymmetric (role dependent). However, in contrast to symmetric games (role independent), relatively limited research has been devoted to the evolutionary analysis of asymmetric games. Systematic attention to these games emerged over the last four to five decades.

For asymmetric games with finite strategy sets, the pioneering evolutionary analysis was initiated in [Sel80], followed by further developments by other authors. In the context of asymmetric games with continuous strategy sets, the work in [MH15] is particularly significant, as it provides an elegant evolutionary framework of such games using replicator dynamics (a type of population dynamics). Subsequently, other researchers also contributed to the evolutionary study of asymmetric continuous games.

In our work (see e.g., [NS19], [NS21], [NS22]), we introduced and generalized several stability concepts for population profiles and sets of profiles in asymmetric continuous games with the associated replicator dynamics. In particular, we established results connecting static stability notions such as strong uninvadability, strong unbeatability, strong immutability, strong immovability, and neighborhood strong superiority with dynamic stability properties like Lyapunov stability, asymptotic stability, weak attraction, and neighborhood attraction.

References

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[NS19] Aradhana Narang and A. J. Shaiju. “Evolutionary stability of polymorphic profiles in asymmetric games”. In: *Dynamic Games and Applications* 9.4 (2019), pp. 1126–1142.

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CT1639 — Understanding breakthrough infections following mpox vaccination: variation in the frequency of clinical characteristics

SPEAKERS Anna Fairweather (PSI, University of Oxford)
AUTHORS Estelle McCool (PSI, University of Oxford); Lucie Abeler-Dörner (PSI, University of Oxford); Christophe Fraser (PSI, University of Oxford); Jasmina Panovska-Griffiths (PSI, University of Oxford); Anna Fairweather (PSI, University of Oxford)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 10.11 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Background: Real-world evidence on the clinical presentation and severity of mpox infections following vaccination with the MVA-BN vaccine remains limited.

Methods: We conducted a scoping review and meta-analysis of 45 studies published between 2022 and 2025 to epidemiologically characterise mpox breakthrough infections following MVA-BN vaccination.

Results: 17 studies provided suitably stratified data for an odds ratio estimation of progression to more severe forms of disease, conditional on first being recognised as a case. Vaccinated cases had significantly lower odds of developing systemic symptoms and becoming hospitalised compared to unvaccinated cases.

Interpretation: Although many breakthrough infections after MVA-BN vaccination have been documented, the vaccine effectiveness (VE) in reducing the rate of infection is unknown. We did not estimate total $\mathrm{VE} = 1 - (1 - \mathrm{VE}_S)(1 - \mathrm{VE}_P)$, with VE_S the VE in reducing the risk of becoming a case and VE_P the VE in reducing the risk of progression to severe outcomes once already diagnosed. The 17 studies contributed to an estimate of VE_P but not to an estimate of VE_S, which could be the primary benefit of the vaccine in protecting against severe mpox disease. Future studies are needed to address this.

CT1340 — When Saboteur Bacteria Undermine Chemotherapy: A Mathematical Model of Cancer Treatment Dynamics

SPEAKERS Anna Geretovszky
AUTHORS Anna Geretovszky; Gergely Röst (University of Szeged)
SCHEDULE Thursday, 16 July 2026; 14:20–14:40; 15.06 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

We construct a mathematical model of cancer dynamics with chemotherapeutic treatment, in the presence of bacteria that are capable of metabolizing the chemotherapeutic drug, hence sabotaging the therapy. We investigate the possibility of complementing the cancer treatment with antibiotic drugs, thus eradicating the bacteria or at least mitigating their negative impact on the prospects of therapy. Our model is a system of nonlinear differential equations, for which we perform a complete analysis, explicitly characterizing the four possible outcomes, depending on whether the cancer cells or the bacteria become extinct or persist. Global stability results are proven by the iterative application of a comparison principle, and a bifurcation diagram is created to show the transitions between scenarios with respect to the controllable parameters. We apply our model to an experiment on mice with colon cancer and the drug Gemcitabine. We also examine the phenomenon from a control theory perspective, where we consider the chemotherapy and the antibiotics as controls and thus seek the optimal combined therapy. The talk is based on the authors' published article [GR25].

References

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CT1454 — A mathematical model of osteocyte network growth

SPEAKERS Jack Fenwick (Queensland University of Technology)
AUTHORS Alexandra Tits (Max Planck Institute of Colloids and Interfaces); Jack Fenwick (Queensland University of Technology); Pascal Buenzli (Queensland University of Technology); Richard Weinkamer (Max Planck Institute of Colloids and Interfaces); Vivien Challis (Max Planck Institute of Colloids and Interfaces)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 10.01 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

The osteocyte network is a spatial cellular network connected by dendritic cell protrusions whose structure is determined by the embedment of bone forming cells (osteoblasts) at the moving bone-forming surface. Network architecture is important for many bone functions, but little is known about how the network forms during bone growth. It has been suggested that osteoblast embedment may be controlled by connections between osteoblasts and the network. In this contribution, we develop a mathematical model

of osteocyte network growth to explore how the regulation of osteoblast embedment by the existing osteocyte network may affect the development of the osteocyte network.

We propose a novel, two dimensional agent-based model of osteocyte network growth. In this model, each osteocyte produces cell protrusions that are connected to other osteocytes in the network and osteoblasts in the bone surface, generating embedment signals. Embedding osteoblasts differentiate into osteocytes, thereby expanding the osteocyte network. The osteocyte networks produced by this model are analysed using a variety of geometric and network properties, such as betweenness centrality.

We find that network architecture depends strongly on the type of embedment signals received by osteoblasts. Inhibitory embedment signals consistently produced qualitatively realistic osteocyte networks. Excitatory embedment signals produce networks with clusters of osteocytes, that are not observed experimentally.

CT1232 — A Nonlinear ODE Model of Butyrate-Tumour-Immune Cell Dynamics in Colorectal Cancer

SPEAKERS Alexandra Lawryshyn (University of Guelph)
AUTHORS Alexandra Lawryshyn (University of Guelph); Hermann Eberl (University of Guelph)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 02.23 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Colorectal cancer progression is strongly influenced by metabolic signals from the gut microbiome, yet the underlying mechanisms remain poorly understood. In particular, the short-chain fatty acid butyrate - produced by fiber-fermenting gut bacteria - has been shown to inhibit cancer cell proliferation while supporting the growth and function of healthy colonocytes and immune cells. We formulate and analyze a system of non-linear ordinary differential equations that describe key metabolic and immunological interactions between butyrate, colorectal cancer cells and host cell populations. This model is then coupled to a comprehensive carbohydrate digestion model developed by [Moo+15]. The parameter space is explored through steady-state and sensitivity analyses. Simulation experiments illustrate the emergence of varying dynamical behaviour driven by butyrate availability.

References

[Moo+15] Arun S Moorthy et al. “A spatially continuous model of carbohydrate digestion and transport processes in the colon”. In: *PlOS One* 10.12 (2015), e0145309.

CT1361 — Agent-based modeling of multiple myeloma in the aged bone microenvironment

SPEAKERS Riley Hall (Moffitt Cancer Center)
AUTHORS Riley Hall (Moffitt Cancer Center)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Multiple myeloma is a hematological malignancy that primarily impacts older individuals. Despite this, preclinical models often use younger animals that do not accurately represent the aged bone microenvironment in which the disease flourishes. Aging in the bone microenvironment is characterized by changes in bone structure, altered remodeling mechanisms, and increased cellular senescence. This work uses a hybrid agent-based model to capture these age-induced transformations in the bone microenvironment

and assess their impact on multiple myeloma progression. Cell types involved in bone remodeling, such as osteoblasts, osteoclasts, and mesenchymal stem cells, are modeled alongside signaling of RANKL and bone-derived factors. The model is parameterized with data from aged tumor-naive and 5TGM1-inoculated C57BL/KaLwRij mice representative of the average age of a myeloma patient. We first modify the simulated bone microenvironment to reflect homeostatic bone remodeling and trabecular bone architecture in an aged disease-free patient. Next, we introduce multiple myeloma cells to assess disease progression in the aged simulated microenvironment. We model the impact of zoledronate, a bisphosphonate used in the standard of care regimen for multiple myeloma, on aged bone remodeling, tumor burden, and the development of environment-mediated drug resistance. This work emphasizes the importance of accounting for age-dependent changes in parameters in computational models.

CT1258 — An agent-based modelling approach to investigate the impact of gender on tuberculosis transmission in Uganda

SPEAKERS James Doran (University of Bath)
AUTHORS Christian Yates; Dennis Mujuni; James Doran (University of Bath); Kit Gallagher; Ruth Bowness (University of Bath)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 10.11 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Tuberculosis (TB) is an airborne disease caused by the pathogen *Mycobacterium tuberculosis*. In 2023, it returned to being the leading cause of death from an infectious agent globally, replacing COVID-19; in the nineteenth century, one in seven of all humans died of tuberculosis. More than 10 million people are diagnosed with TB every year. The majority of cases in adults occur in males (62.5% of all global adult cases in 2023, compared to 37.5% in females). The main reasons for males suffering from a higher burden of global TB cases, compared to females, may be in large part due to population-scale factors, such as employment type, the quantity and type of social contacts they make, and their health-seeking behaviours (e.g. differences in diagnostic and treatment delays between genders). To investigate which population-scale factors are most important in determining this higher TB burden in males, we have developed an age- and gender-stratified, spatially heterogeneous epidemiological agent-based model. We have focused specifically on Kampala, the capital of Uganda, which is a high-burden TB country. We considered counterfactual scenarios to elucidate the impact of gender on the epidemiology of TB. Setting disease progression parameters equal between the genders leads to a reduction in both male-to-female case ratio and total case numbers.

CT304 — Eco-evolutionary dynamics of proliferation: from a community model to tumor growth

SPEAKERS Farnoush Farahpour (Quantitative Oncology and Systems Modeling, University of Duisburg-Essen)
AUTHORS Daniel Hoffmann (University of Duisburg-Essen); Farnoush Farahpour (Quantitative Oncology and Systems Modeling, University of Duisburg-Essen); Lara Schmalenstroer (Bioinformatics and Computational Biophysics, University of Duisburg-Essen); Russell Rockne (Professor & Chair, Department of Computational and Quantitative Medicine Beckman Research Institute, City of Hope.)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 12.01 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Proliferation rate is a central life-history trait linking ecological interactions and evolutionary changes. This talk reviews eco-evolutionary models in which reproduction rate explicitly evolves, generating feedback between ecological structure and trait dynamics that shapes community composition and diversity. A generic interaction and agent-based model with a life-history trade-off is presented to

demonstrate how evolving reproduction can reproduce canonical macro-evolutionary patterns, including disruptive selection and long-term coexistence without imposed niche separation, offering a dynamical perspective on competitive exclusion through eco-evolutionary self-organization. These principles are then transferred to a phenotype-structured PDE model of tumor growth in which proliferation evolves under ecological constraints and treatment pressure. Embedding trait dynamics directly into population structure generalizes classical growth models and enables systematic comparison of treatment modalities in terms of their impact on proliferation heterogeneity. The framework reproduces some well-known characteristics of clinical endpoints of cytotoxic therapies and elucidates how therapeutic pressure on proliferation reshapes tumor structure and influences long-term tumor dynamics. Overall, this trait-based eco-evolutionary perspective across biological systems clarifies how selection on proliferation affects diversity, coexistence, and responses to perturbations.

CT206 — Explaining the dynamical consequences of cis binding in a mathematical model of Notch signaling.

SPEAKERS Specioza Nambooze (African Institute for mathematical Sciences Ghana)
AUTHORS Annah Naturinda (African Institute for Mathematical Sciences Ghana); Nick Monk (African Institute for Mathematical Sciences Ghana); Specioza Nambooze (African Institute for mathematical Sciences Ghana)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.21 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Inter-cellular signaling through the Notch receptor plays a central role in a wide range of developmental and physiological processes. In many contexts, signalling-dependent feedback can drive cellular decisions contributing to the regulation of proliferation and differentiation and spatial pattern formation.

In addition to mediating inter-cellular signaling by trans-binding, Notch ligands can also bind Notch on the same cell (cis-binding), sequestering the ligand and receptor in an inactive complex. It has been proposed that this inactive cis-binding can enhance both the speed and regularity of spatial pattern formation by Notch signaling. We have developed a mathematical model to explore the effects of cis-binding on the dynamics of Notch signaling processes. In particular, we focus on the oscillatory transients that have previously been observed in simplified models of Notch signaling. Using linear stability analysis and numerical solutions, we show that these are also present in binding models and that cis-binding can reduce both the amplitude and duration of oscillatory transients. Our results suggest an additional role of cis-binding in regulating the balance between oscillatory and patterning dynamics in Notch signalling, allowing for the control of the tempo and outcome of inter-cellular Notch signalling.

CT153 — From clustering to dispersal: collective invasion driven by adhesion and motility

SPEAKERS Josue Manik Nava (National Autonomous University of Mexico)
AUTHORS Josue Manik Nava (National Autonomous University of Mexico)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Metastatic cells are often assumed to undergo epithelial–mesenchymal transition (EMT). However, recent studies suggest that, in several cases, malignant cells retain adhesive contacts. In addition, highly invasive cells have been observed to migrate superdiffusively. Here, the interplay between cell–cell adhesion and high motility is investigated using a simple cellular automaton model. The model shows that increasing motility can induce a slow and smooth transition from clustering to dispersal without the loss of adhesive interactions, indicating that adhesion and migration need not be mutually exclusive. Moreover,

highly adhesive and motile populations tend to form transient clusters that spontaneously assemble and disassemble.

References

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CT1546 — Global well-posedness and stability analysis of a degenerate system for a prey-predator and natural enemy interaction

SPEAKERS Duc-Anh Nguyen (University of Graz)
AUTHORS Bao Tang (University of Graz); Duc-Anh Nguyen (University of Graz)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 11.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

In this study, we consider a degenerate reaction-diffusion system that models the interactions among the prey, the predator, and the predator's natural enemy. A typical scenario of our model arises from the plant-insect-natural enemy interaction in agriculture, in which the prey is a plant species that lacks diffusion; meanwhile, the natural enemy not only eradicates the predator but also supports the growth of the prey. Firstly, the well-posedness properties of the solution, including global existence and the uniform boundedness in time, are proved. Next, we present the stability analysis of the spatially homogeneous equilibria, in which we identify a sufficient condition under which Turing instability does not occur and prove the nonlinear stability of these equilibria.

CT352 — In vivo spatial dynamics of actomyosin oscillations

SPEAKERS Jochen Kursawe (University of St Andrews)
AUTHORS Jochen Kursawe (University of St Andrews)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 05.12 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

The apicomedial actomyosin network is crucial for generating mechanical forces in cells. Pulsatile, oscillatory behaviour of this contractile network occurs in multiple organisms and contexts, for example during mouse embryo compaction or xenopus neurulation. However, the emergence and control of such pulsed contractions are not fully understood. Here, we study pulsed contractions of larval epithelial cells during development of the abdomen in fruit flies. Specifically, we combine *in vivo* 4D microscopy and simulations of an active elastomer to investigate subcellular spatial patterns of actomyosin dynamics. Our model quantitatively matches *in vivo* observations. We find that cell shape, cell polarity, and organisation of the cell's actomyosin network codetermine spatiotemporal network dynamics both *in vivo* and in simulations. Furthermore, the model correctly predicts changes to LEC contractile activity under genetic perturbation of the actomyosin network. Our results highlight that cell geometry and polarity are important contributors to *in vivo* actomyosin dynamics. Moreover, our findings support the notion that spatiotemporal oscillatory behaviour of the actomyosin network is an emergent property, rather than driven by upstream signalling.

References

[Mac+26] Euan D. Mackay et al. “An active matter model captures spatial dynamics of actomyosin oscillations in larval epithelial cells during *Drosophila* morphogenesis”. In: *Proceedings of the National Academy of Sciences* 123.3 (2026), e2503955123. DOI: 10.1073/pnas.2503955123. eprint: https://www.pnas.org/doi/pdf/10.1073/pnas.2503955123. URL: https://www.pnas.org/doi/abs/10.1073/pnas.2503955123.

CT1352 — Intraspecific trait variation and niche choice in predator-prey dynamics

SPEAKERS Anastasiia Enne (Bielefeld University)
AUTHORS Anastasiia Enne (Bielefeld University); Meike Wittmann (Bielefeld University)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Intraspecific trait variation (ITV) can be important for population performance in a variable and changing environment because individuals with different traits have different fitness consequences in different environments. Furthermore, individuals can potentially improve their fitness by interacting with their environment, for example, by performing niche choice and moving to a different patch with more favorable conditions. This becomes important when modelling predator-prey dynamics. For example, to come up with the best strategy for pest control (when movement of predators is affected by pest prey availability), or establishing protected areas (when predators migrating out of the protected area may be beneficial for commercial harvesting industries). In this talk, we show a model incorporating ITV and niche choice in a patchy and changing environment to investigate how they affect predator-prey dynamics. We use individual-based model and an analytical approximation by ODEs to quantify the effects of ITV and niche choice on the population persistence of both predator and prey. This can provide insight into the interplay between ITV and niche choice, sometimes ignored by existing models, and lead to a better understanding of how these mechanisms can buffer populations against environmental change.

CT1321 — Lightning Laplace methods for floral-arthropod electrostatics

SPEAKERS Samuel Harris (University of Bristol)
AUTHORS Robb McDonald (University College London); Ryan Palmer (University of Bristol, UK); Samuel Harris (University of Bristol)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Insects have acquired a variety of sensory receptors through coevolution with plants, which transmit floral cues such as scent, colour and shape. Recently discovered among this suite of arthropod senses is electroreception – the ability to detect electric fields. This raises the question of whether plants also convey biologically relevant information electrically to nearby pollinators, and how they interact electrically with each other and the wider environment. To investigate this, the electric field is modelled numerically using a two-dimensional approximation. A variant of recently developed lightning Laplace methods known as the AAA-least squares algorithm computes a rational approximation of the harmonic electric potential with high accuracy and speed, even on a standard laptop. This numerical approach is extended in two non-trivial ways: first to two-domain problems to solve the electric field inside and outside a single flower; and second to general multi-domain problems representing an entire flower meadow. These new extensions are presented in detail, followed by some biologically motivated analysis of floral morphology, pollen load and various plant-arthropod configurations. Comparison with results produced in COMSOL shows that the two-dimensional results give a good proxy of the overall three-dimensional behaviour.

References

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CT1458 — Mesoscale community organization governs epidemic onset and spread in metapopulations

SPEAKERS Malbor Asllani (Florida State University)
AUTHORS Haoyang Qian (Florida State University); Malbor Asllani (Florida State University)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.04 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Understanding how internal community structure shapes the course of epidemics remains a fundamental challenge in modeling real-world populations [KMS17]. Standard metapopulation models often assume uniform mixing within communities, overlooking how internal heterogeneity affects global outcomes [Dav+20]. Here, we develop a general framework for epidemic spreading in hierarchically structured metapopulations, where individuals interact locally within dense communities and move across a broader network. We show that transmission dynamics are governed by the mesoscale organization of these communities: highly connected groups accelerate and amplify outbreaks, while less connected ones dampen spread. Through a combination of mean-field theory [QA25], spectral analysis, and stability methods, we reveal a direct link between internal connectivity and the emergence of uneven, spatially structured epidemic patterns. We further validate these predictions using real-world data, where social contact networks capture the local scale of transmission while spatial transport networks govern global connectivity, confirming the robustness of our framework across scales. These results demonstrate how community structure fundamentally governs the shape of epidemics in complex, networked populations, offering new insights into vulnerability, containment, and epidemic control.

References

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CT1357 — Modeling of glioblastoma recurrence based on local microenvironment characteristics

SPEAKERS Hermann Frieboes (University of Louisville)
AUTHORS Jackson Huse (University of Louisville); Dylan Goodin (University of Louisville); Hermann Frieboes (University of Louisville)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 02.21 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Efforts to suppress glioblastoma (GBM) recurrence include targeting peritumoral regions by increasing extent of tumor resection and volume/dose of radiotherapy. Due to their broad application, these

methods include risks of radiation toxicity and excess healthy tissue excision. Techniques to better predict where GBM recur could circumvent these limitations. Recent evidence indicates that recurrence location depends on unique metabolic programs of residual peritumoral GBM cells along with selective tumor microenvironment (TME) factors including biophysical characteristics of the migration zone and interactions with immune cell populations. This study implements a continuum-scale model to simulate GBM recurrence post-resection. The model represents tumor growth and simulation of resection with the goal to predict likely location(s) of recurrence based on local TME factors and metabolic characteristics. The model simulates GBM interactions with the TME, including vascularization and immune species. We recently analyzed patient tumor core (contrast enhancing) and peritumoral (T2/FLAIR hyperintense) samples via metabolomics, finding key metabolic signature differences between these tumor regions. The model results show that GBM recurrence is biased towards locations of higher vascularization and disrupted TME, modulated by dysregulated metabolism in peritumoral tissue. This work could facilitate spatially targeted approaches that prevent recurrence and improve outcomes.

CT294 — Optimising booster vaccination against SARS-CoV-2 variants: when should a variant-adapted vaccine be developed?

SPEAKERS Abbie Evans (University of Oxford)
AUTHORS Abbie Evans (University of Oxford); Robin Thompson (University of Oxford); William Hart (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 03.01 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Novel variant emergence during viral outbreaks, as in the COVID-19 pandemic, undermines vaccine effectiveness. When a variant emerges, policy advisors must decide whether to recommend continuing booster vaccination with the existing vaccine or delaying until a variant-adapted vaccine is available. We present a mathematical model for evaluating these strategies by quantifying severe outcomes (deaths or Years of Life Lost; YLL), accounting for time-varying immunity. We compare two primary strategies: (1) boosting with the existing vaccine and (2) delayed boosting with a variant-adapted vaccine (after a development lag). If the updated vaccine is available within a threshold time, delaying vaccination leads to fewer deaths (or YLL). This threshold depends on characteristics including variant transmissibility, with more transmissible variants yielding shorter thresholds. We further explore mixed strategies involving vaccinating some individuals with the existing vaccine and others with the variant-adapted vaccine (when available). In a scenario motivated by the emergence of the SARS-CoV-2 Omicron variant, delaying all booster vaccination is optimal for short development times, whereas for longer development times vaccinating older individuals with the existing vaccine and younger individuals with the variant-adapted vaccine is preferable. Our modelling framework facilitates evidence-based decisions on vaccine updating and deployment during variant-driven outbreaks.

CT1240 — PINNs for structured population dynamics inference: Application to oocyte dynamics in fish ovaries

SPEAKERS Louis Fostier (INRIA Paris)
AUTHORS Frédérique Clément (Université Paris-Saclay, Inria, Centre Inria de Saclay, 91120, Palaiseau, France); Louis Fostier (INRIA Paris); Manon Lesage (INRAE, Fish Physiology and Genomics Institute, UR1037, LPGP, Rennes, France); Romain Yvinec (INRAE, CNRS, Université de Tours, PRC, 37380, Nouzilly, France); Violette Thermes (INRAE, Fish Physiology and Genomics Institute, UR1037, LPGP, Rennes, France)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Population dynamics are often observed as distributions of individuals with respect to structuring variables such as size or age. The associated inverse problem consists of inferring the underlying vital rates (recruitment/birth, growth, and death), which are typically complex, nonparametric functions due to limited prior knowledge. Physics-Informed Neural Networks (PINNs) provide a natural framework for such problems by combining neural networks as universal function approximators with mechanistic constraints encoded in differential equations. We present a novel application of PINNs to inverse problems for size-structured partial differential equation models with nonlocal interaction terms. Our goal is to describe early oocyte dynamics in juvenile fish ovaries, a key process determining lifelong reproductive capacity and regulated by hormonal feedback. We develop a model describing the dynamics of precursor germ cells and growing oocytes structured by size and coupled through a hormone-mediated feedback regulating precursor cell renewal. Using repeated cross-sectional measurements of oocyte size distributions, we apply the PINN framework to infer nonparametrically the size-dependent oocyte growth rate, the time-dependent recruitment of new oocytes, and the regulated renewal rate of precursor cells. The calibrated model reproduces experimental oocyte size distributions and provides in silico access to key biologically meaningful quantities that are not directly observable.

CT185 — Predicting the fate of mutation rate modifiers that shift the mutation bias

SPEAKERS Diego Tenoch Morales Lopez (University of Western Ontario)
AUTHORS Diego Tenoch Morales Lopez (University of Western Ontario); Lindi Wahl (University of Western Ontario); Mete Yuksel (University of Toronto); Puneeth Deraje (University of Toronto)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.12 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

The rate of de novo mutations varies across species and is predicted to be dependent on parameters such as population size, the average generation time, and how far the population is from the fitness peak. Mutations that modify the mutation rate itself often emerge in bacterial evolution experiments, either increasing or decreasing the mutation rate of ancestral lineages. Previous theoretical approaches have delineated the conditions necessary for these modifiers of mutation rate to spread in the population, either by hitchhiking with beneficial mutations or by reducing the load of deleterious mutations. One implicit assumption in previous work is that the fractions of beneficial and deleterious mutations are identical in the modifier and ancestral lineages (which differ only in how often new mutations arise). Recent empirical work, however, has highlighted the fact that modifications of the mutation rate are often accompanied by changes in mutation bias, such that different types of mutations occur at different rates. This can dramatically alter the availability of beneficial and deleterious mutations for the modifier lineage. In this talk, I will present a stochastic model and computer simulations which explore the effect of mutations that modify both the mutation rate and the mutation bias, demonstrating how these two effects simultaneously play a role in the evolution of microbes.

CT310 — Regimes and mechanisms of inflammation described by reaction-diffusion systems

SPEAKERS Wissam El Hajj (University of Rennes)
AUTHORS Nader El Khatib (Lebanese American University); Vitaly Volpert (University Claude Bernard Lyon 1); Wissam El Hajj (University of Rennes)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 01.15 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Inflammation is a physiological process aimed at protecting the organism from various external stimuli. It plays an important role in numerous diseases including viral infection, atherosclerosis, cancer, and neurodegenerative diseases. Although each type of inflammatory disease has its own characteristic stimuli, the inflammatory response mechanism is generic.

In this work, we present generic mathematical models of inflammation based on reaction–diffusion equations describing the spatiotemporal dynamics of healthy cells, inflamed cells, immune cells, and both pro- and anti-inflammatory mediators. The objective is to develop generalized models capable of capturing the main phases of the inflammatory process, initiation, progression, and resolution, using systems of reaction–diffusion and integro-differential equations, with and without delay.

Our analysis shows that inflammation can propagate within tissue as a reaction–diffusion wave, and we determine the corresponding propagation speed. Finally, numerical simulations are performed to investigate how anti-inflammatory mechanisms influence the global dynamics of the system and the resolution of inflammation.

CT1406 — Spatial remodeling of the tumor-immune microenvironment by neoadjuvant chemoradiotherapy in NSCLC

SPEAKERS Zuzanna Nowicka-Kaszkowiak (Medical University of Łódź)

AUTHORS Julia Sołek-Siejka (Department of Pathology, Chair of Oncology, Medical University of Łódź, Łódź, Poland); Bartłomiej Tomasik (Department of Oncology and Radiotherapy, Faculty of Medicine, Medical University of Gdańsk, Gdańsk, Poland; Centre for Experimental Cardiooncology, Medical University of Gdańsk, Poland); Maria Wołyniak (Department of Biostatistics and Translational Medicine, Medical University of Łódź, Łódź, Poland); Robert Dziedzic (Department of Thoracic Surgery, Faculty of Medicine, Medical University of Gdańsk, Gdańsk, Poland); Witold Rzyman (Department of Thoracic Surgery, Faculty of Medicine, Medical University of Gdańsk, Gdańsk, Poland); Michał Bieńkowski (Department of Pathomorphology, Medical University of Gdańsk, Gdańsk, Poland); Bartosz Sobocki (Department of Oncology and Radiotherapy, Faculty of Medicine, Medical University of Gdańsk, Gdańsk, Poland); Patrick Doyle (Department of Radiation Oncology, Brigham and Women's Hospital, Boston, MA, USA); Aleksandra Ellert-Miklaszewska (Laboratory of Molecular Neurobiology, Nencki Institute of Experimental Biology, Polish Academy of Sciences, Warsaw, Poland); Bożena Kamińska (Laboratory of Molecular Neurobiology, Nencki Institute of Experimental Biology, Polish Academy of Sciences, Warsaw, Poland); Marcin Braun (Department of Pathology, Chair of Oncology, Medical University of Łódź, Łódź, Poland); David Kozono (Department of Radiation Oncology, Brigham and Women's Hospital, Boston, MA, USA); Wojciech Fendler (Department of Biostatistics and Translational Medicine, Medical University of Łódź, Łódź, Poland; Department of Radiation Oncology, Brigham and Women's Hospital, Boston, MA, USA); Zuzanna Nowicka-Kaszkowiak (Medical University of Łódź)

SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.06 - HS

SESSION Contributed Talks

TRACK Mathematical Oncology

Abstract

The combination of chemoradiotherapy (CRT) and immune checkpoint inhibition (ICI) in unresectable stage III NSCLC yields markedly divergent outcomes depending on treatment sequencing: consolidation ICI following CRT confers significant survival benefits [Spi+22], whereas concurrent administration fails to improve survival [Bra+25]. This paradox implicates radiation-induced remodeling of the tumor-immune microenvironment as a key, yet poorly understood, determinant of ICI response.

We applied Opal multiplex immunofluorescence (AKOYA Biosciences) to resected NSCLC following neoadjuvant chemotherapy (CHT, $n = 16$), CRT ($n = 22$), or primary resection ($n = 10$), with matched pre-treatment biopsies in a subset. A six-plex panel (CD8, CD4, FOXP3, CD20, CD68, PanCK) was quantified in QuPath to profile the immune composition across tissue compartments and classify the tumor ecosystem by KNN into inflamed, excluded, and cold phenotypes.

Tumors resected after CRT showed selective depletion of CD20+ B cells at the invasive margin compared to CHT and primary resection ($p = 0.01$). FOXP3+ regulatory T cell density also differed significantly across treatment groups ($p = 0.02$), with highest density observed after CHT, while CD8+ effector infiltration

was preserved following CRT and CHT. These findings suggest cytotoxic treatment preferentially remodels humoral and immunoregulatory compartments at the tumor-stroma interface, revealing spatial immune barriers relevant to subsequent immunotherapy response.

References

[Bra+25] Jeffrey D. Bradley et al. "Simultaneous Durvalumab and Platinum-Based Chemoradiotherapy in Unresectable Stage III Non–Small Cell Lung Cancer: The Phase III PACIFIC-2 Study". en. In: *Journal of Clinical Oncology* 43.33 (Nov. 2025), pp. 3610–3621. ISSN: 0732-183X, 1527-7755. DOI: 10.1200/JCO-25-00036. URL: https://ascopubs.org/doi/10.1200/JCO-25-00036 (visited on 05/10/2026).

[Spi+22] David R. Spigel et al. "Five-Year Survival Outcomes From the PACIFIC Trial: Durvalumab After Chemoradiotherapy in Stage III Non–Small-Cell Lung Cancer". en. In: *Journal of Clinical Oncology* 40.12 (Apr. 2022), pp. 1301–1311. ISSN: 0732-183X, 1527-7755. DOI: 10.1200/JCO.21.01308. URL: https://ascopubs.org/doi/10.1200/JCO.21.01308 (visited on 05/10/2026).

CT1227 — The trade-off between fractal morphology and function of Drosophila neurons

SPEAKERS Ildikó-Beáta Márton (1. Faculty of Physics, Babeș-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania)

AUTHORS Ildikó-Beáta Márton (1. Faculty of Physics, Babeș-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania); Balázs Péntek (1. Faculty of Physics, Babeș-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania); Mária Ercsey-Ravasz (1. Faculty of Physics, Babeș-Bolyai University; 2. Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania)

SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 11.01 - HS

SESSION Contributed Talks

TRACK Neuroscience and Neural Systems

Abstract

Studying the correlation between structure and function in brain networks has long been a central and compelling topic in neuroscience. The FlyWire project achieved a major milestone by completing a fully digitized brain map of Drosophila melanogaster [Dor+24]. In this study, we analyzed a total of 125,330 three-dimensional neuron skeletons from intrinsic, afferent, and efferent types belonging to different functional superclasses and classes. This study focuses on comparing different morphological and topological properties of axons and dendrites, such as cable length, fractal dimension, number of nodes, maximum width, and tree height. Our results show that axons tend to have simpler structures than dendrites, however, in some functional classes (typically some afferent and efferent ones), the situation appears to be reversed. Examining the correlation between fractal dimension and other properties reveals that neurons from the same superclasses or classes form clusters in state space, indicating similar morphological characteristics. As the hierarchy descends, some of these clusters become denser and more clearly separated. These results suggest that the brain specializes in fractal dimension and other topological properties for different functional cell types.

References

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CT344 — Threshold-Driven Collapse and Recovery in a Simplified Wasp-Waist Ecosystem Model

SPEAKERS Sam Subbey (Institute of Marine Research)
AUTHORS Sam Subbey (Institute of Marine Research)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 15.02 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

We study the dynamics of a three-trophic level food web model representing a wasp-waist marine ecosystem, where a single or multiple intermediate consumers mediate energy flow between primary producers and apex predators. Using a system of nonlinear ordinary differential equations with ratio-dependent functional responses, we investigate conditions under which the consumer populations collapse or persist. By analyzing the system near a producer-only boundary equi- librium, we identify critical thresholds for the initial predator-to-prey biomass ratio that delineate collapse, escape, and intermediate (”race condition”) regimes. Our analysis employs quasi-stationarity arguments, asymptotic expansions, and differential inequalities to rigorously characterize the system’s trajectories and basins of attraction. The results provide a mechanistic understanding of transient collapse phenomena and offer mathematically tractable early-warning indicators for instability in structured ecological systems. We propose that the approach can be extended to analyze output from more complex ecosystem models, enabling reduced-form interpretations of resilience and early warning indicators in empirical or high-dimensional simulations of wasp-waist systems

CT1512 — TopROI: Topological partition and analysis of colorectal cancer tissue architecture

SPEAKERS Sergio Serrano de Haro Iváñez (University of Oxford)
AUTHORS Sergio Serrano de Haro Iváñez (University of Oxford); Joshua W. Moore (University of Oxford); Lucile Grzesiak (University of Oxford); Eoghan J. Mullholand-Illingworth (University of Oxford); Simon J. Leedham (University of Oxford); Heather Harrington (Max Planck Institute of Molecular Cell Biology and Genetics, TU Dresden, University of Oxford); Helen Byrne (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 01.22 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

A hallmark of colorectal cancer is the progressive deterioration of the colonic tissue architecture. Intestinal crypts – tubular epithelial glands essential for homeostasis – gradually lose their characteristic morphology and function due to uncontrolled cell proliferation and tissue invasion. Quantifying this disruption in biopsied samples is critical for both patient diagnosis and prognosis.

Spatial biology offers a mathematical and computational framework for analysing tissue organisation. In this work, we combine techniques from topological data analysis and network science to quantify structural changes associated with colorectal cancer progression. Using cell point clouds extracted from immunohistochemistry imaging, we construct cell networks that encode topological features of tissue structure. We use these networks to partition large, imaged samples into smaller, biologically meaningful regions of interest that preserve local architectural information. Within these segmented regions, we apply methods from persistent homology to quantify multiscale structural properties of tissue. Our results reveal topological signatures that correlate with tumour progression and describe a continuum of architectural changes from healthy to cancerous samples.

CT324 — Using Approximate Bayesian Computation for Model Selection in a Dual-Layer Cell Migration Model

SPEAKERS Tracy Stepien (University of Florida)
AUTHORS Holley Lynch (Stetson University); Lance Davidson (University of Pittsburgh); Sanandan Ojha (University of Florida); Tracy Stepien (University of Florida)
SCHEDULE Thursday, 16 July 2026; 14:40–15:00; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

During gastrulation, an early stage of embryonic development, cell migration is crucial for the formation of germ layers that eventually develop into tissues and organs. We develop a continuum mechanical model of dual layer cell migration during gastrulation that includes intercalation of cells between the mesenchymal and epithelial cell layers. Approximate Bayesian computation with sequential Monte Carlo (ABC-SMC) and sensitivity analysis are used to obtain parameter estimates and to compare between various model formulations. A goal of this work is to determine the functional form of intercalation and cell layer growth that best reflects the experimental data from spreading aquatic frog *Xenopus laevis* tissue.

CT1359 — A phylodynamic model for within-host HCV infection integrating genomic sequences and time series biomarkers

SPEAKERS Wei-Ting Chen (The Ohio State University - Columbus, OH)
AUTHORS Christiaan van Dorp (Columbia University); Ethan Romero-Severson (Los Alamos National Laboratory); Qianying Lin (The Ohio State University); Wei-Ting Chen (The Ohio State University - Columbus, OH)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 11.34 - SR
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Hepatitis C virus (HCV) is a blood-borne RNA virus that remains the major cause of liver-related morbidity worldwide. Following acute infection, outcomes vary drastically across individuals: some patients spontaneously clear the virus, while others develop chronic infection progressing to severe liver disease. This variation is known to be driven by the heterogeneities in host immunity, an interplay among innate immunity, antibodies, and T-cell response. This talk aims to characterize and quantify the impact of different immune response mechanisms, providing insights into future vaccine design.

In this project, we develop a phylodynamic framework to study within-host HCV infection in a cohort of injection drug users (IDU). Our approach integrates viral genetic sequences, reconstructed phylogenetic relationships, and longitudinal biomarker measurements within a unified viral dynamics model. This approach enables joint inference on infection dynamics and viral evolution within the host, allowing genetic diversification to be interpreted alongside observed biomarker trajectories.

The primary objective of this work is to estimate key features of viral dynamics, including viral infection dynamics on cells and viral clearance.Eventually, we seek to determine whether the combined analysis of genetic and longitudinal clinical data can identify signatures associated with distinct infection outcomes.

CT1317 — Branching Process Models of Sub-Critical FMD Transmission: Inference, Control, and Surveillance Design in Karnataka, India

SPEAKERS Glen Guyver-Fletcher (University of Warwick, UK)
AUTHORS Glen Guyver-Fletcher (University of Warwick, UK); Matt Ferrari (Huck Institutes of the Life Sciences, The Pennsylvania State University); Mike Tildesley (School of Life Sciences, University of Warwick, Coventry, UK)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 11.32 - SR
SESSION Contributed Talks

TRACK Mathematical Epidemiology

Abstract

Effective control of livestock diseases often produces a sub-critical phase – outbreaks occur but die out before becoming epidemic – in which standard epidemic models are poorly suited to characterising transmission dynamics. Foot-and-Mouth Disease (FMD) in India offers a clear example: national control programs have been in place and intensifying since 2012, yet the disease persists across multiple states, including Karnataka. Branching processes are a natural framework for this disease regime, capturing the stochastic extinction dynamics that define sub-critical spread.

We fit a branching process model to FMD outbreak data from Karnataka, using the offspring distribution to characterise how transmission intensity and individual-level variation have changed under successive control policies. Our best-fit model implies substantially smaller outbreak sizes and reduced variance in transmission compared to the pre-control period, consistent with a population-level reduction in R below 1. We further apply this model to derive the minimally-sufficient surveillance effort required to detect transient outbreaks before fade-out, with implications for establishing eradication and designing post-control monitoring programs.

CT21 — Can Cheating Go Viral? A Classroom-Based Social Contagion Model of AI Misuse

SPEAKERS Donna Dyer (The University of the West Indies, St. Augustine Campus, Trinidad and Tobago, West Indies)
AUTHORS Donna Dyer (The University of the West Indies, St. Augustine Campus, Trinidad and Tobago, West Indies); Joanna Sooknanan (The University of the West Indies, St. Augustine Campus, Trinidad and Tobago, West Indies)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 03.01 - SR
SESSION Contributed Talks
TRACK Education and Outreach

Abstract

The rapid expansion of remote and technology-mediated assessment during and after the COVID-19 period exposed serious challenges to academic integrity, particularly in contexts where students were left to self-regulate with limited institutional oversight. These challenges have been amplified by increasingly easy access to generative AI tools, through which answers to problem sets and even full project reports can now be produced by simply uploading a photograph of an assignment to a large language model. In many university settings, AI-assisted cheating has spread rapidly through peer networks in ways that resemble social contagion [SC17].

Inspired by this trend, we present a differential-equation model that treats AI-assisted cheating as a social contagion driven by peer influence and moderated by institutional intervention. The model provides an accessible yet mathematically rich example for undergraduate teaching, allowing students to apply equilibrium analysis, local stability classification, and numerical simulation using a reduced planar system. A worked MATLAB example illustrates how analytical results are reinforced through phase-plane and time-series plots. By enabling students to explore academic integrity through quantitative modelling rather than moral instruction, this contribution offers a powerful and transferable approach for teaching mathematical epidemiology, differential equations, and applied modelling.

References

[SC17] Joanna Sooknanan and Donna M. G. Comissiong. “When behaviour turns contagious: the use of deterministic epidemiological models in modeling social contagion phenomena”. In: *International Journal of Dynamics and Control* 5 (2017), pp. 1046–1050. DOI: 10.1007/s40435-016-0271-9.

CT297 — Discrepancies between discrete and continuum models: insights from oncolytic virotherapy

SPEAKERS David Morselli (University College London)
AUTHORS David Morselli (University College London); Federico Frascoli (Swinburne University of Technology); Marcello Edoardo Delitala (Politecnico di Torino)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Oncolytic virotherapy uses engineered viruses to selectively infect and destroy tumour cells. Despite some clinical successes, the spatial dynamics governing viral spread in solid tumours remain poorly understood. The integration of agent-based models, continuum models and hybrid approaches has been particularly valuable for capturing the multi-scale and stochastic nature of this biological system. In this talk, we compare agent-based and continuum models to assess when the macroscopic model captures the essential behaviour of the underlying discrete system. In a baseline setting, both models predict travelling waves and show good qualitative agreement. However, oscillatory dynamics may emerge in the discrete model, sometimes leading to eradication of the infection and, in some cases, complete tumour clearance not captured by the continuum formulation [MFD25]. While Poissonian tumour control probability is often used to estimate extinction events, this approach may fail when a strong immune response is present [Mor+24]. Moreover, when viral diffusion and tumour cell motility are spatially constrained, the continuum model predicts infection failure, whereas the discrete system may still sustain viral spread [MDF23; Mor+24]. These findings illustrate how the transition from microscopic to macroscopic descriptions may introduce non-trivial discrepancies, emphasizing the need for caution when using continuum approximations to interpret multi-scale biological dynamics.

References

[MDF23] David Morselli, Marcello Edoardo Delitala, and Federico Frascoli. “Agent-based and continuum models for spatial dynamics of infection by oncolytic viruses”. In: *Bulletin of Mathematical Biology* 85.10 (2023). DOI: 10.1007/s11538-023-01192-x.

[MFD25] David Morselli, Federico Frascoli, and Marcello Edoardo Delitala. “The role of viral dynamics and infectivity in models of oncolytic virotherapy for tumours with different motility”. Accepted in Bulletin of Mathematical Biology. 2025. DOI: 10.48550/arXiv.2509.24522.

[Mor+24] David Morselli et al. “A hybrid discrete-continuum modelling approach for the interactions of the immune system with oncolytic viral infections”. Preprint. 2024. DOI: 10.48550/arXiv.2404.06459.

CT1616 — How inertia affects autotoxicity-mediated vegetation pattern dynamics

SPEAKERS Giancarlo Consolo (University of Messina, Italy)
AUTHORS Annalisa Iuorio (University of Naples “Parthenope”, Italy); Carmela Currò (University of Messina, Italy); Gabriele Grifò (Istituto Nazionale di Alta Matematica “F. Severi”); Giovanna Valenti (University of Messina, Italy); Frits Veerman (University of Leiden, The Netherlands); Giancarlo Consolo (University of Messina, Italy)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

In this talk we discuss the influence of inertial effects on the formation and evolution of vegetation patterns on sloped arid terrains in different dynamical regimes. Analyses are carried out in a hyperbolic extension

of the one-dimensional Klausmeier model [Kla99], where autotoxicity effects are also considered. As the system moves away from the wave bifurcation threshold, two classes of solutions, corresponding to the emergence of qualitatively different coherent structures, arise: small-amplitude periodic migrating bands near onset and large-amplitude travelling pulses in far-from-equilibrium conditions [Con+26].

For the first class of solutions, we address both linear and multiple scale weakly nonlinear analyses [Con+24]. Our results reveal that inertia acts as a destabilising mechanism, reduces the pattern migration speed and may even reverse the dynamical regime, from supercritical to subcritical, thus leading to hysteresis.

For the second class of solutions, we investigate the key qualitative features of travelling vegetation pulses via Geometric Singular Perturbation Theory [GIV25]. In far-from-equilibrium conditions, inertia is shown to increase pulse speed while preserving the intrinsic multiscale structure of the solution, in full agreement with the numerical findings.

Overall, we show that inertia does not exclusively act as a time lag, but it constitutes an additional degree of freedom which should be considered in shaping vegetation dynamics under environmental stress.

References

[Con+24] Giancarlo Consolo et al. "Stationary and Oscillatory patterned solutions in three-compartment reaction–diffusion systems: Theory and application to dryland ecology". en. In: *Chaos, Solitons & Fractals* 186 (Sept. 2024), p. 115287. ISSN: 09600779. DOI: 10.1016/j.chaos.2024.115287. URL: https://linkinghub.elsevier.com/retrieve/pii/S0960077924008397 (visited on 05/11/2026).

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CT1280 — Kron Reduction in Species-Reaction Graphs of Mass-Action Chemical Reaction Networks

SPEAKERS Manvel Gasparyan (Ghent University)
AUTHORS Manvel Gasparyan (Ghent University); Ovidiu Radulescu (University of Montpellier); Shodhan Rao (Ghent University Global Campus (GUGC))
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

We propose a novel approach for model reduction in kinetic models of mass-action chemical reaction networks (CRN), based on the Kron reduction of the species-reaction graph associated with the network. This type of reduction comes from the theory of linear electrical networks. Although CRNs are not linear, they are endowed with a linear structure defined by the graph Laplacian, describing how the information propagates in the network. We show how to define a graph Laplacian for the species-reaction graph, by

assigning appropriate weights to the edges of this graph. We use the Laplacian to rewrite the chemical kinetics of the CRN as a hybrid differential-algebraic system. We show that the Kron reduction preserves the hybrid structure and transforms the Laplacian into the Schur complement of the original one. The entire procedure is automated through a userfriendly Python package. We validate the applicability of this reduction technique using a real-world example of a CRN and demonstrate its effectiveness in practical scenarios.

CT345 — Mapping the spread of artemisinin resistance in Africa

SPEAKERS Lucy Harrison (University of Oxford)
AUTHORS Lucy Harrison (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.21 - SZ
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Artemisinin-based combination therapies (ACTs) are the most widely used treatment for Plasmodium falciparum malaria. Kelch 13 mutations associated with artemisinin partial resistance (ART-R) have emerged in Sub-Saharan Africa (SSA) and are now reported in an increasing number of countries. ACT treatment failure rates are at risk of unprecedented increase. To summarise existing surveillance data and guide future surveillance, we produce modelled estimates of the spatiotemporal distributions of Kelch 13 and partner drug marker prevalence in SSA. We develop and validate spatiotemporal Gaussian Process models, fitted within a Bayesian framework. We estimate the prevalence of Kelch 13 mutations that are validated or candidate markers of ART-R and the prevalence of four further mutations associated with selection by pressure from ACT partner drugs.Our models reflect all existing clusters of ART-R-associated Kelch 13 mutations. We estimate the prevalence of these Kelch 13 mutations to be greater than 10% in 23% of the area of endemic malaria transmission in SSA in 2026. We also estimate that 5.8% of malaria cases in 2026 will be affected by a validated or a candidate ART-R marker. To monitor the changing distribution of antimalarial resistance markers within the constraints of the current global health funding climate it is critical that validated, statistical frameworks are incorporated into decision-making workflows to make the best use of molecular surveillance data.

CT1577 — Modeling PSA dynamics to predict resistance during intermittent androgen deprivation therapy

SPEAKERS Minhye Kim (Korea Institute of Science and Technology)
AUTHORS Eunjung Kim (Korea Institute of Science and Technology); Minhye Kim (Korea Institute of Science and Technology)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Prostate cancer is commonly treated with androgen deprivation therapy, yet resistance frequently emerges during long-term treatment. Intermittent androgen deprivation therapy (IADT), in which treatment is periodically suspended and reintroduced, has been proposed to reduce treatment burden while potentially delaying resistance. Determining when resistance is likely to arise remains a central challenge for optimizing treatment schedules in individual patients, given the variability in prostate-specific antigen (PSA) dynamics across patients.

Here we develop a mathematical model describing the interaction between treatment-sensitive and resistant tumor cell populations and their relationship to PSA dynamics during IADT. To enable patient-specific inference from clinical data, we incorporate Bayesian inference to estimate PSA dynamics from longitudinal measurements and update tumor composition estimates.

Using longitudinal PSA measurements from patients undergoing IADT [Bru+06], we examine how early PSA trajectories reflect tumor dynamics and assess the probability of resistance during subsequent treatment cycles. Our results suggest that patient-specific PSA patterns contain signals about tumor composition and the likelihood of resistance under treatment cycling. The proposed framework provides a quantitative approach for interpreting PSA dynamics under intermittent therapy and provides a basis for predicting resistance in patient-specific strategies.

References

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CT1560 — Modelling the active coordination of cell adhesion and contractility in cells and tissues

SPEAKERS Carina Dunlop (University College London)
AUTHORS Carina Dunlop (University College London); Josephine Solowiej-Wedderburn (Umeå University)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.34 - SR
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Experimental investigation consistently reveals that mechanical signals direct and coordinate cell behaviours across tissue types, with cell adhesion and contractility playing key roles. In individual cells, it is observed that focal adhesions and cytoskeletal contractility are both mechanosensitive, changing in response to environmental stiffness. Within tissues, however, adhesion and contractility adaption to the microenvironment is made more complex by the introduction of cell-cell attachments introducing confounding forces at cell junctions. Here, we use a continuum theoretical model for cell contractility coupled actively to the microenvironment to enable a whole systems approach. In collaboration with Dr N. Tapon, Francis Crick Institute and Dr J.R. Davis, Manchester University we apply the framework to tissue layers, micropatterned to control adhesion. We show that the model correctly predicts mechanical contractility in the layer and indeed identifies specific regions of peak mechanical activity. Furthermore, we show that increasing gel stiffness promotes more uniform contractility activity in the cells within adhered layers.

References

[Dav+24] John Robert Davis et al. *Emergence of active contractile patterns alters monolayer force generation and transmission in response to focal adhesion distribution.* en. Apr. 2024. DOI: 10.1101/2024.04.10.588783. URL: http://biorxiv.org/lookup/doi/10.1101/2024.04.10.588783 (visited on 05/11/2026).

CT1195 — Perception of infection risk drives backward bifurcation during outbreaks

SPEAKERS Alexis Erich Almocera (University of the Philippines Mindanao)
AUTHORS Alexis Erich Almocera (University of the Philippines Mindanao); Esteban Hernandez-Vargas (University of Idaho,); Tinashe Gashirai (University of Idaho)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

The interplay between the perceived risk of infection and the host's protective behavior in response to an emerging infection is complex and difficult to abstract. We present a human behavior model here, based on the assumption that the human host exhibits positive adaptive behavior when the disease incidence reaches a certain threshold. Furthermore, we assume that when incidence is low, the adverse effects of negative messaging and risky human behavior outweigh the positive influence of risk perception, thereby amplifying the disease burden. Our mathematical analysis shows that the recruitment rate of susceptible individuals and the incidence that triggers protective behavior determine whether the disease persists or dies out. Moreover, changes in the transmission rate due to risk perception-modulated host behavior may result in a backward bifurcation. This complicates disease control, as the basic reproduction number does not predict epidemic occurrence. This study highlights the importance of understanding the role of complacency in engaging the human adaptive response and risk perception in combating disease spread. It also illustrates that control strategies that reduce the recruitment of susceptible individuals and the infectious period of diseased individuals regulate the size of long-term disease incidence.

CT1356 — Transmission dynamics of Mpox: Implications for Global Control from the 2024 Outbreak

SPEAKERS Rehan Akber (Lahore University of Management Sciences (LUMS))
AUTHORS Adnan Khan (Lahore University of Management Sciences); Mohsin Ali (National Institute of Technology, Lahore, Pakistan); Rehan Akber (Lahore University of Management Sciences (LUMS)); Salman Safdar (University Of Maryland, College Park)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 11.33 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

The outbreak of Mpox in the Democratic Republic of Congo (DRC) in 2024 spread internationally, prompting the WHO to declare a public health emergency and underscoring the need to understand the disease's transmission dynamics and potential control measures. We develop and analyze two different models for Mpox transmission. The first deals with the outbreak of Mpox in a single human population group, incorporating transmission between humans and animals. Using this model, we derive an expression for the basic reproductive number (R_0) for the Democratic Republic of Congo (DRC) and the Rest of the World (ROW) using surveillance data. The second model extends the single human population to a two-patch framework, coupling DRC with ROW and examines the role of inter-patch mobility through a residence-time framework under a range of mobility scenarios. We estimate the meta-population reproduction number R_{0T}. We note that while mobility has a modest effect on R_{0T}, the disease burden varies substantially with changing mobility. Across a wide range of mobility values, the infected population in DRC increases with mobility, while that in the ROW decreases with mobility; this observation has important policy implications for travel restrictions during the outbreak.

CT1200 — When does it pay to eat your friend?

SPEAKERS Josephine Solowiej-Wedderburn (Umea University)
AUTHORS Josephine Solowiej-Wedderburn (Umea University)
SCHEDULE Thursday, 16 July 2026; 17:00–17:20; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

A distinguishing feature of eukaryotic cells is their ability to consume other cells. By contrast, prokaryotes are much more likely to engage in competitive and cooperative interactions. However, in the evolutionary transition from prokaryotes to eukaryotes, predation must have emerged from communities with primarily

competitive or cooperative interactions. There are clear benefits to developing the ability to eat a competitor, but can it ever be beneficial to eat a cooperator?

We use a dynamical systems framework and invasion analysis to identify the ecological conditions under which predatory mutants can invade a system of cooperating microbes. We identify a critical threshold that determines whether an invading predator will coexist with the cooperative community or replace members. Repeatedly invading with different predators, we find that the system saturates when there are three coexisting species, though this community does not have a single optimal composition. However, if we incorporate additional costs, we find there is a single evolutionarily stable community of two cooperators and a pure predator. Ultimately, we show that it is possible for phagocytosis to evolve from a cooperative community and, moreover, coexist with the original cooperating species.

CT1320 — A Kinetic Model of Yeast Fermentation Incorporating Nitrogen Metabolism

SPEAKERS Diego Troitiño-Jordedo (IIM-CSIC)
AUTHORS João Soares-Monteiro; Laura Pérez-Través (IATA-CSIC); Romain Minebois (IATA-CSIC); Amparo Querol (IATA-CSIC); Eva Balsa-Canto (IIM-CSIC); Diego Troitiño-Jordedo (IIM-CSIC); Oana-T Chis (IIM-CSIC)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Yeasts play a key role in fermentation processes widely used in industrial biotechnology. During fermentation, yeasts produce ethanol and a wide range of metabolites, including aroma compounds. Among the factors controlling the process, nitrogen availability strongly influences yeast growth, metabolic activity, and metabolite production. Understanding how nitrogen metabolism affects fermentation dynamics is therefore essential for improving process control and performance. In this study, we present a kinetic mathematical model for yeast batch fermentation that captures the uptake dynamics of sugars and organic (e.g. amino acids) and inorganic (e.g. ammonium) nitrogen sources, while accounting for supplementation strategies. The model describes how nitrogen availability and specific nitrogen sources influence yeast metabolism, including how nitrogen is allocated between biomass growth and maintenance, and metabolite production such as aroma compounds. It also accounts for regulatory mechanisms underlying phase transitions in yeast growth. The model was applied to several yeast strains to analyse the impact of nitrogen and carbon sources on fermentation dynamics. The results reveal how different nitrogen sources influence fermentation behaviour and metabolite production across strains. These findings illustrate the potential of the proposed modelling framework to support the analysis and optimisation of fermentation processes.

CT387 — Designing a feedback-oriented outreach activity: insights from a trial and error approach

SPEAKERS Christin Nyhoegen (ETH Zurich, Theoretical Biology)
AUTHORS Christin Nyhoegen (ETH Zurich, Theoretical Biology)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 03.01 - SR
SESSION Contributed Talks
TRACK Education and Outreach

Abstract

Scientific outreach provides many benefits for researchers, as it helps to acquire and establish (or strengthen) essential skills for an academic career. Yet many barriers prevent interested researchers from participating in outreach programs, such as the lack of time or the lack in experience and information on designing an outreach activity. During my graduate research, I participated in an outreach program targeted at school students. I developed an interactive outreach activity on antibiotic resistance evolution and mathematical

models for treatment optimization. Lacking experience, I followed a recommended framework for outreach design. During this process, I encountered multiple challenges, which I addressed through a time-intense trial and error approach in multiple iterations of the program. The key improvement was to incorporates the students feedback before the actual visit. With this case report I want to share my experience and provide helpful insights into designing an outreach activity, which I hope contributes to lowering the barrier to participate in valuable (and fun!) outreach activities.

CT1634 — Dynamics of Consumer–Resource Systems

SPEAKERS Rui Li (Shenzhen Technology University/University of Oxford)
AUTHORS Rui Li (Shenzhen Technology University/University of Oxford)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Consumer–resource models provide a framework for describing interacting populations mediated by shared resources and have been widely used to study the dynamics of complex biological systems. In this work, we examine consumer–resource systems from a dynamical perspective. Starting from simple model settings, we extend the analysis to a triangular region and investigate how structural constraints shape system behavior. Alongside the theoretical analysis, we discuss how these models connect to concrete biological contexts, including ecological and microbial systems. This combination of analytical results and applications provides a unified perspective on consumer–resource dynamics and offers insight into the mechanisms underlying complex biological processes.

CT1602 — Effects of Cell Orientation on Formation of Ordered Arrangement of Photoreceptor Cells in the Zebrafish Retina: A Lattice Model Study

SPEAKERS Keiichi Yamamoto (Institute for Life and Medical Sciences, Kyoto University; Graduate School of Science, Kyoto University)
AUTHORS Keiichi Yamamoto (Institute for Life and Medical Sciences, Kyoto University; Graduate School of Science, Kyoto University); Takashi Okada (Institute for Life and Medical Sciences, Kyoto University; Interdisciplinary Theoretical and Mathematical Sciences Program (iTHEMs), Riken); Yuji Sakai (Yokohama City University); Atsushi Mochizuki (Institute for Life and Medical Sciences, Kyoto University)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.34 - SR
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

In vertebrate retinas, multiple types of cone photoreceptors, each sensitive to different wavelengths, exhibit various two-dimensional arrangements. In zebrafish, each of four cone types (blue, red, green, and UV) appears periodically, forming a nearly square lattice, called “cone mosaic”. Such regularity seems to realize uniform color resolution across the retina.

Regular mosaic patterns are considered to emerge from irregular arrangements through cell rearrangements. A previous mathematical model postulated that cells move on a two-dimensional lattice ([TMI03]). In this model, cell movement was affected by cell adhesion between contacting cells, whose strength depended on the combination of contacting cell types. In addition, a red-green cone pair was assumed to contact strongly to form a “double cone”. Contact of other cells with the double cone was restricted orthogonally: either the red or the green cone only (single-side) or both at once (double-side). Thus, the red-green pair aligned along one of the four directions ($\leftarrow$,$\downarrow$,$\uparrow$,$\rightarrow$).

We introduced four additional oblique directions in the double cone orientations. Our model allows larger freedom in the dynamics, revealing that optimal parameters of the adhesion strength for mosaic formation

only partially overlap with the previous ones. Moreover, mosaics can be formed even when single-side adhesion of the double cone is weaker than double-side adhesion, unlike the previous result favoring stronger single-side adhesion.

References

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CT1554 — From Classrooms to Communities: Modeling the Impact of School-Based Interventions for Future Pandemic Response

SPEAKERS Zicheng Fang (Wageningen University & Research)

AUTHORS Alexandra Teslya (University Medical Center Utrecht, The Netherlands); Zicheng Fang (Wageningen University & Research); Büşra Atamer Balkan (Wageningen University & Research); Clarize De Korne (UMC Utrecht); Matthijs De Winter (National Institute of Public Health and the Environment (RIVM)); Lucie Vermeulen (National Institute of Public Health and the Environment (RIVM)); Quirine ten Bosch (Wageningen University & Research); Patricia Bruijning-Verhagen (UMC Utrecht); Ganna Rozhnova (University Medical Center Utrecht, The Netherlands)

SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.21 - SZ

SESSION Contributed Talks

TRACK Mathematical Epidemiology

Abstract

Introduction Schools are a critical setting during respiratory pandemics. While their high-contact environments can amplify community transmission, they remain essential for children's learning and wellbeing. The RE-PASS project develops a multi-scale modeling framework to support school-based interventions that balance infection control with minimizing educational disruption.

Methods The framework captures transmission across three interconnected scales: 1) within-classroom dynamics reflecting daily interactions of students and teachers; 2) within-school transmission mediated by shared staff and cross-group contacts; and 3) a school–community network linking households, schools, and the community. Epidemiological states of individual agents are tracked daily, including time-dependent virus shedding. The framework is demonstrated using SARS-CoV-2 data from the early outbreak in the Netherlands before vaccines were available. The synthetic school–household network is based on Dutch data and incorporates socio-economic stratification.

Results The model simulates outbreaks of respiratory pathogens and evaluates school-based interventions at multiple levels. In addition to epidemiological outcomes, it quantifies educational outcomes, including in-person learning days lost and projected learning loss.

Conclusion This framework supports policy-relevant evaluation of school-based interventions by quantifying both health and educational impacts during future respiratory pandemics.

CT1322 — Impact of infant monoclonal antibody protection on RSV dynamics: a stage-structured modelling study

SPEAKERS Anna Autoriello (PhD National Programme in One Health approaches to infectious disease and life science research, Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Department of Mathematics and Applications, University of Naples Federico II, Italy)

AUTHORS Alfredo Guarino (Pediatric Infectious Disease Unit, Department of Maternal and Child Health, University Hospital Federico II, Department of Translational Medical Science, University of Naples Federico II, Italy); Anna Autoriello (PhD National Programme in One

Health approaches to infectious disease and life science research, Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Department of Mathematics and Applications, University of Naples Federico II, Italy); Bruno Buonomo (Department of Mathematics and Applications, University of Naples Federico II, Italy); Cristina Moracas (PhD National Programme in One Health approaches to infectious disease and life science research, Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Pediatric Infectious Disease Unit, Department of Maternal and Child Health, University Hospital Federico II, Italy); Emanuela Penitente (Department of Mathematics and Applications, University of Naples Federico II, Italy); Marco Poeta (Pediatric Infectious Disease Unit, Department of Maternal and Child Health, University Hospital Federico II, Department of Translational Medical Science, University of Naples Federico II, Italy); Rossella Della Marca (Department of Mathematics and Applications, University of Naples Federico II, Italy); Sabrina Averga (PhD National Programme in One Health approaches to infectious disease and life science research, Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Department of Mathematics and Applications, University of Naples Federico II, Italy)

SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Respiratory syncytial virus (RSV) is one of the leading causes of hospitalisation for bronchiolitis and other lower respiratory tract infections in infants, representing a major cause of pressure on paediatric healthcare systems [Man+25]. Recent post-pandemic seasons, for instance in Italy, have highlighted how changes in respiratory virus circulation and heterogeneous prevention policies can complicate planning based on surveillance alone. In this context, there is a clear need for mathematical tools that complement clinical and public health evidence by translating intervention assumptions into quantitative, population-level expectations and by enabling transparent comparisons across alternative implementation scenarios [Lan22]. To this end, we develop a stage-structured compartmental model with three age classes to investigate RSV transmission, compare different prevention strategies and support public health decision-making aimed at reducing disease burden. The model explicitly incorporates infant immunoprophylaxis with long-acting monoclonal antibodies, consistent with current preventive practices, and includes vaccination of older adults. Scenario-based comparisons suggest that infant prophylaxis may reduce RSV infection incidence and affect the seasonal pattern of outbreaks. The proposed framework provides an analytical tool to inform coordinated RSV prevention policies in Italy.

References

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CT1360 — Inferring the rules of TCR–pMHC binding using synthetic co-evolution

SPEAKERS Antonio Matas Gil (UCL)
AUTHORS Antonio Matas Gil (UCL)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 11.34 - SR
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Predicting T cell receptor (TCR) recognition of peptide–MHC (pMHC) complexes remains a major challenge in immunology. Existing computational approaches are limited by the lack of dense, locally

informative binding data. Synthetic co-evolution via yeast display has recently emerged as a powerful technique to probe protein–protein interactions at scale. Applying these methods to TCR–peptide interactions enables datasets where binding affinity across millions of TCR–peptide combinations can be inferred from rounds of in vitro library-on-library selection. I present a computational pipeline to tackle this inference problem. First, I show how maximum entropy (Potts) models learn patterns of sequence restriction and co-evolutionary couplings, accurately predicting selection dynamics on held-out data. Second, I show how these models disentangle selection pressures from binding affinity versus confounding factors such as differential expression efficiency. Finally, I address what local library scans reveal about the global binding landscape: I simulate how varying complexity of global models shapes the reduced model, then invert this relation to bound the complexity of rules governing TCR–pMHC recognition. Our work provides a foundation for inferring binding interactions at scale from library-on-library experiments, offering a path towards the throughput needed to guide next-generation models of immune recognition.

CT1208 — Mathematical modeling and optimal control analysis of impact of information-induced vaccination and saturated treatment on disease prevalence

SPEAKERS Anuj Kumar (Thapar Institute of Engineering & Technology, Patiala, India)
AUTHORS Anuj Kumar (Thapar Institute of Engineering & Technology, Patiala, India)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

This study investigates the impact of information-induced vaccination, behavioural responses, and saturated treatment on infectious diseases via a delay mathematical model. Also, the dynamics of information are quantified via a separate rate equation, which influences healthy individuals to adopt controls. Stability and bifurcation analysis are carried out to understand qualitative changes in the disease pattern and dynamics under delay and non-delay cases. The existence of multiple stability switches, Hopf, and Backward bifurcations, along with Bogdanov-Takens and generalized Hopf are investigated. Importantly, due to delayed information-induced behavioural response, the emergence of torus and double frequency oscillations is found numerically via the presence of Neimark-Sacker (NS) and double Hopf (HH) bifurcations. Our study infers that the model system gives rise to rich and complex dynamics. Further, considering information-induced vaccination and treatment as controls, an optimal control problem is proposed that minimizes costs incurred due to the disease burden and applied controls. A comparative cost-effective study is conducted by choosing various control strategies. We observe that the comprehensive use of control interventions reduces the severity of the disease burden and also minimizes the economic burden. Our findings suggest that the delay in execution of controls may alter the effect of optimal controls and the cost-effectiveness of control strategies.

CT307 — Personalizing Cancer Therapy by Modeling the Evolutionary Dynamics of the Tumor Immune Microenvironment

SPEAKERS Qingpeng Zhang (The University of Hong Kong)
AUTHORS Qingpeng Zhang (The University of Hong Kong)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

In this talk, we present our research on using mathematical modeling and machine learning to characterize the evolutionary dynamics of the tumor immune microenvironment (TIME) for personalized cancer therapy. We developed various ODE-based TIME models, integrating genomic and transcriptomic data.

These models, enhanced by deep reinforcement learning (DRL), optimize therapy regimens, including intermittent androgen deprivation therapy (IADT) for prostate cancer and personalized schedules for immune checkpoint inhibitors (ICIs) and chemotherapy. Our findings demonstrate the potential of these approaches to improve patient outcomes by tailoring treatments to individual tumor dynamics.

References

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CT1367 — Phylodynamic inference of the contributions of different contact types to infectious disease transmission

SPEAKERS Don Klinkenberg (Netherlands Institute for Public Health and the Environment (RIVM), Bilthoven, The Netherlands)
AUTHORS Bastiaan Van der Roest (Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht, The Netherlands); Don Klinkenberg (Netherlands Institute for Public Health and the Environment (RIVM), Bilthoven, The Netherlands); Egil Fischer (Department of Population Health Sciences, Faculty of Veterinary Medicine, Utrecht University, Utrecht, The Netherlands); Martin Bootsma (University Medical Center Utrecht & Utrecht University); Mirjam Kretzschmar (Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht, The Netherlands)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 11.33 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Understanding which types of contact drive pathogen transmission is critical for outbreak control. Traditionally, high-risk contacts are inferred with contact tracing or network data. Including genetic data may better elucidate the contribution of different types of contact to transmission. Vice versa, contact data may improve transmission inference (who infected whom) of outbreaks.

We formulated a transmission likelihood for who infected whom, from data about different contact types observed between cases of an outbreak, based on [Cam+19]. We implemented this in the phylodynamic model `phybreak`, to analyse outbreaks using pathogen sequences and sampling times. The resulting model estimates the fractions of transmission events attributable to the types of contact. We evaluated performance with simulations across various scenarios of contact frequencies, transmission risks by contact type, and correlations between contact types.

Results show that sparse contact types with high transmission risk can be accurately identified. Performance declines when most transmission occurs via common contact types, or when contact types are positively correlated. Use of contact data improves inference of who infected whom, particularly when genetic data alone lack resolution. When applying the method to the 2020 SARS-CoV-2 outbreak in Dutch mink farms, shared personnel was identified as a high-risk contact, with a limited role of veterinary service providers and feed suppliers.

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CT1206 — Stochastic foraging and resource accumulation with queueing theory

SPEAKERS José Giral-Barajas (Imperial College London)
AUTHORS José Giral-Barajas (Imperial College London); Paul Bressloff (Department of Mathematics, Imperial College London)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Foraging is a ubiquitous strategy employed by diverse species and organisms for collecting, accumulating, and consuming vital resources. Examples of this can be observed in various populations, from the intracellular environment where vesicles explore the cytoplasm, carrying resources to the lysosome for consumption, to a bumblebee hive where foragers explore the exterior in search of nectar and water. In both cases, resource search has a random component induced by the chaotic and turbulent environment, and the populations have a finite capacity to consume resources. In this talk, we will present a model that combines queuing processes and first-passage processes, motivated by foraging systems, to track the accumulation of resources as a product of the interplay between random delivery and consumption. The main objective is to shed light on the viability of the foraging strategy, assuming stochastic search and consumption. Finally, we will introduce novel results relating the spatial configuration of a 1D stochastic search process to the convergence to a steady-state number of resources in the queuing system.

CT1566 — Towards a thermodynamic consistent framework for reversible-irreversible hysteresis in epigenetic landscapes

SPEAKERS Jacobo Ayensa-Jiménez (Universidad Politécnica de Madrid)
AUTHORS Jacobo Ayensa-Jiménez (Universidad Politécnica de Madrid); Ignacio Romero (Universidad Politécnica de Madrid)
SCHEDULE Thursday, 16 July 2026; 17:20–17:40; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Epigenetic transitions in the tumor microenvironment exhibit history-dependent memory effects and abrupt phenotypic shifts that classical smooth dynamical systems fail to capture from a physical perspective [EH02]. We present a novel mathematical framework grounded in the theory of rate-independent systems to describe the evolution of epigenetic marks—specifically DNA methylation—under hypoxic stress.

Using the Mielke-Theil energetic formulation [MR15], we model the epigenetic state as an internal variable governed by a double-well energy function coupled with Michaelis-Menten hypoxia kinetics and a 1-homogeneous dissipation potential, which axiomatically enforces the energy-dissipation balance and the Clausius-Duhem inequality, driving biological irreversibility.

By deriving the Kuhn-Tucker differential inclusions governing the system evolution, we demonstrate that the model captures key experimental phenomena: epigenetic catastrophes such as Epithelial-Mesenchymal Transitions [Yoo+11], reversible-irreversible damage under hypoxia and long-term adaptation [Thi+16] as well as the imprint of epigenetic scars [KNM25]. In addition, it allows for quantitative predictions of cellular state stability based on the thermodynamic potentials.

This work provides a rigorous physical basis for computational epigenetics and lays the groundwork for a systematic analysis of the energetic barriers and irreversibility of tumor progression.

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CT1377 — A Stochastic Germinal Center Model in Antigenic Space for Cross-Variant Antibody Responses to SARS-CoV-2 Vaccination

SPEAKERS Rodolfo G. Blanco-Rodriguez (University of Idaho)
AUTHORS Esteban Hernandez-Vargas (University of Idaho,); Janine Kimpel (Medizinische Universität Innsbruck); Marta Bermejo Jambrina (Medizinische Universität Innsbruck); Rodolfo G. Blanco-Rodriguez (University of Idaho)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 11.34 - SR
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Understanding how germinal center (GC) dynamics generate antibody responses against emerging viral variants is important for vaccine modeling. Here we develop a stochastic GC model formulated in an antigenic space framework to study antibody responses to SARS-CoV-2 vaccination. Naïve B cells and antigens are represented as coordinates in antigenic space, and affinity is determined through a bit-matching score that drives B cell selection and differentiation into plasma and memory cells. Plasma cells (PCs) are classified as aligned or non-aligned with the target variant, allowing the model to capture both variant-specific and cross-reactive antibody production.

The model parameters were calibrated using experimental data (50% neutralizing titer) across multiple variants from different vaccination regimens. The simulations reproduced the qualitative antibody dynamics observed in the data. The model suggests that early antibody production is primarily driven by PCs targeting the vaccine-matched variant, whereas broader cross-variant responses emerge later and are sustained by longer-lived PC populations generated during germinal center maturation and booster-induced reactions. Memory B cells contribute to improved affinity following booster vaccination. This framework provides a mechanistic approach to linking GC selection dynamics with antigenic distance, which could help to interpret scenarios involving vaccination and breakthrough infection with emerging variants.

CT1595 — A topological framework for the analysis of complex cellular interactions

SPEAKERS MARIA JOSE JIMENEZ (UNIVERSIDAD DE SEVILLA)
AUTHORS Abhinav Natarajan (University of Oxford); Thomas Chaplin (University of Oxford); Eoghan J. Mulholland-Illingworth (Centre for Human Genetics, Univrsity of Oxford); Joshua A. Bull (University of Oxford); Simon J. Leedham (Centre for Human Genetics, University of Oxford); Helen Byrne (University of Oxford); MARIA JOSE JIMENEZ (UNIVERSIDAD DE SEVILLA); Heather Harrington (Max Planck Institute of Molecular Cell Biology and Genetics, TU Dresden, University of Oxford)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Spatial relationships in multi-species data can shape biological system behaviors, from cancer progression in pathology to coral reef resilience in ecology. We propose a topological data analysis framework to quantify higher-order spatial relationships in multi-species point cloud data [Nat+26]. Our approach is applicable to the analysis of interactions between three or even more species, capturing both (i) the presence of topological features in the data—such as connected components, loops and voids—and (ii) the combinatorial arrangement of these features across multiple subsets of species. The method leverages recent developments in chromatic topological data analysis [Cul+26], introducing a new construction called the *k-chromatic gluing map*, and providing interpretable descriptors. We validate our framework on two datasets: (1) synthetic data from an agent-based model of the tumor micro-environment [BB23], where our results are consistent with existing analysis by Stolz et al. [Sto+24]; and (2) a colorectal cancer dataset, where the method recovers biologically reasonable patterns, including the importance of periostin–macrophage spatial interactions (consistent with existing literature [Zho+15]), as well as potentially important three-way interactions among macrophages, neutrophils, periostin, and SMA (interactions that are not directly captured by pairwise analysis).

References

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CT1405 — Community-Engaged Learning in an Interdisciplinary Math-Biology Course

SPEAKERS Brynja Kohler (Utah State University)
AUTHORS Bertha Neequaye; Brynja Kohler (Utah State University); Noelle Beckman (Utah State University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 03.01 - SR
SESSION Contributed Talks
TRACK Education and Outreach

Abstract

This talk describes a junior-level interdisciplinary Math/Biology course that integrates mathematical modeling with community-engaged learning. Students explore complex social and environmental systems through project-based learning, applying modeling approaches from ecology, public health, conservation, and sociology. The course emphasizes modeling as a tool for understanding and addressing real-world issues and as a disposition for approaching complex problems. For the final project, students collaborate with local partners—such as a municipal landfill, a regional zoo, and conservation nonprofits—to propose data-informed solutions to problems. Examples from Spring 2023 and Spring 2025 illustrate how students engage with stakeholders, develop teamwork and communication skills, and connect mathematical modeling to civic and scientific contexts. The course builds on institutional support for community-engaged learning and draws on research in team science and project-based learning.

CT1635 — Effects of Fitness Gradient

SPEAKERS Jakub Svoboda (Dartmouth College)
AUTHORS Hossein Nemati; Josef Tkadlec; Kamran Kaveh; Krishnendu Chatterjee; Jakub Svoboda (Dartmouth College)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Evolutionary biology studies populations of reproducing individuals and how their composition changes over time. An important question is to determine the fixation probability of a single advantageous mutant that attempts to invade a homogeneous population of N residents. Many real populations experience gradients of chemicals or nutrients that cause mutations to be beneficial in some spatial regions and possibly harmful in others. We study the fixation probability of a mutant placed on a simple one-dimensional spatial structure that experiences such a gradient. The mutant's fitness varies linearly from $1 - s$ to $1 + s$, whereas the resident fitness is constant and equal to 1.

The existing literature suggests that such heterogeneity in mutant's fitness should lead to a decrease in its fixation probability. However, in this work, we find that small, non-negligible gradients ($s < 1/\sqrt{N}$) substantially increase the fixation probability, while too large gradients ($s > (\log N)/\sqrt{N}$) substantially decrease it. Moreover, we quantify the strength of this phenomenon analytically and we precisely delimit the range of the gradients for which it occurs. Computer simulations closely match those findings. Our results indicate that subjecting a simple population structure to natural environmental conditions produces strong counterintuitive effects.

CT1214 — Far-from-equilibrium patterns in a cross-diffusion vegetation-autotoxicity model

SPEAKERS Frits Veerman (Leiden University)
AUTHORS Annalisa Iuorio (Parthenope University of Naples); Cinzia Soresina (University of Trento, Italy); Frits Veerman (Leiden University)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Many mathematical models describing vegetation patterns are based on biomass–water interactions, due to the impact of this limited resource in arid and semi-arid environments. However, in recent years, a novel biological factor called autotoxicity has proved to play a key role in vegetation spatiotemporal dynamics, particularly by inhibiting biomass growth and increasing its natural mortality rate. Recent work [GIS25] has shown that it is possible to produce stable close-to-equilibrium patterns using biomass-autotoxicity coupling alone, without water as a model component, using a cross-diffusion model for biomass and

toxicity dynamics as the fast-reaction limit of a three-species system involving dichotomy and different time scales.

We study the emergence of multiscale, far-from-equilbrium patterns in the biomass-autotoxicity cross-diffusion model using geometric singular perturbation theory (GSPT). We prove that, when the cross-diffusion term is sufficiently strong, periodic multi-scale patterns can be explicitly constructed, and confirm our theoretical results with numerical simulations. In addition, we show that for weaker cross-diffusion, the family of far-from-equilibrium patterns naturally contains the Turing patterns that were observed in [GIS25]. Our research combines the novel application of GSPT to cross-diffusion systems with the fundamental ecological insight that multiscale vegetation patterns can be produced by biomass-autotoxicity interaction only.

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CT1624 — How little is enough? Can low-complexity parameters discriminate wakefulness and anesthetized states?

SPEAKERS Sandra Widmann (TU München)
AUTHORS Andreas Körner (TU Wien); David Obert (TUM); Gerhard Schneider (TU Munich); Matthias Kreuzer (TU Munich); Sandra Widmann (TU München); Stefanie Pilge (TUM); Zoi-Lemonia Papanagiotou (TU München)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.34 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Background: Monitoring anesthesia levels via EEG is essential to ensure adequate unconsciousness while minimizing complications such as intraoperative awareness or postoperative delirium. Established metrics like SEF95 and SpEn are robust but computationally demanding due to frequency domain transformation. This study evaluates whether low-complexity time domain EEG parameters can provide comparable performance to spectral measures in distinguishing awake and anaesthetized states [Pur+15].

Methods: EEG data from 76 patients under general anesthesia were retrospectively analyzed using frontal recordings [Geo+20]. Power spectral density was estimated via Welch's method to derive SpEn and SEF95. The spectral exponent (SpEx), was obtained using the FOOOF algorithm, separating oscillatory from aperiodic (1/f) activity [Wid+25]. Spectral parameters were compared to four low-complexity time-domain metrics (Benford-1-Index, Coastline, Entropy of Difference (EoD), and Zero-Crossing Rate (ZCR)) using Friedman tests with post-hoc comparisons.

Conclusion: All parameters significantly distinguished ECBaseline from BeforeCut. SpEx increased with deeper anesthesia [Col+19], while SpEn and SEF95 decreased. SpEx achieved the best performance. Benford-1-Index and EoD performed very well, while Coastline was weakest. ZCR performed similarly to SEF and SpEx, highlighting its potential as a reliable and efficient metric for real-time anesthesia monitoring, especially in resource-limited or time-critical settings.

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CT1219 — Improving Parameter Identifiability in Household-Transmission Models Using Genomic Data

SPEAKERS Thomas House (The University of Manchester)
AUTHORS Golsa Sayyar (The University of Manchester); Joe Hilton (The University of Manchester); Thomas House (The University of Manchester)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Household transmission models are widely used in infectious disease epidemiology, yet transmission ordering is typically ignored because it does not affect final outbreak size. Consequently, many distinct transmission histories produce identical epidemiological outcomes, making it difficult to distinguish internal from external transmission and limiting parameter identifiability.

Rather than introducing time-dependent transmission rates, we retain the standard time-homogeneous continuous-time Markov chain formulation and instead refine the household state representation. The model state space is expanded to include transmission graphs describing infection direction and order, while pathogen genomic data are used to restrict the transmission histories compatible with observations.

Simulation studies show that incorporating genetic information substantially sharpens the likelihood surface compared with models based on epidemiological data alone. In particular, genomic data reduce the dependence between internal and external transmission parameters, removing the characteristic ridge associated with non-identifiability and yielding more concentrated posterior distributions. These results demonstrate that graph-resolved household models enable improved transmission inference without sacrificing analytical or computational tractability.

CT1323 — Linking aggrephagy and mTOR signaling in amyotrophic lateral sclerosis: a minimal ODE model

SPEAKERS Alex De Nardi (Fondazione The Microsoft Research – University of Trento Centre for Computational and Systems Biology (COSBI); Department of Mathematics, University of Trento)
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Trento); Mario Lauria (Fondazione The Microsoft Research – University of Trento Centre for Computational and Systems Biology (COSBI); Department of Mathematics, University of Trento)

SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.27 - SR
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

ALS is a neurodegenerative disease driven by proteotoxic stress. Many ALS-associated mutations affect aggrephagy, a selective autophagy pathway that clears protein aggregates [LPC13]. The mTOR pathway is a bulk autophagy regulator and a potential therapeutic target. We identified, through literature analysis, a link between p62, an aggrephagy receptor and an ALS-associated protein, and mTOR signaling, providing a mechanistic explanation for mTOR-dependent autophagy activation under proteotoxic stress [Dur+11]. This interaction was modeled using a parsimonious ODE system with four equations and thirteen parameters, describing the dynamics of p62 mRNA and protein, toxic aggregate mass, and aggregate-bound p62, with mTOR activity represented as a state-dependent variable. A biologically plausible parameterization was proposed, and the system's qualitative behavior was explored as a function of the proteotoxic stress parameter S. The model reproduces distinct dynamical regimes, such as a low-stress steady state with negligible autophagy and a high-stress regime with persistent p62-bound aggregates (consistent with ALS pathology). Moreover, two Hopf bifurcations guarantee the existence of a periodic solution at intermediate stress values. Such periodic behavior, previously reported in bulk autophagy, can be protective towards apoptosis [Hol+20]. Experimental validation is needed to assess oscillatory behavior in aggrephagy.

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CT313 — Mathematical modelling of combination treatment of polycythaemia vera explains observed synergy of drugs

SPEAKERS Tobias Idor Boklund (Roskilde University)
AUTHORS Tobias Idor Boklund (Roskilde University); Gurvan Hermange (CentraleSupélec, Université Paris-Saclay); Johanne Gudmand-Høyer (Roskilde University, Denmark); Anders Lindholm Sørensen (Zealand University Hospital); Morten Kranker Larsen (Zealand University Hospital); Trine Alma Knudsen (Zealand University Hospital); Christina Schjellerup Eickhardt-Dalbøge (Zealand University Hospital); Vibe Skov (Zealand University Hospital); Lasse Kjær (Zealand University Hospital); Christina Ellervik (University of Copenhagen, Boston Children's Hospital, Harvard Medical School, Zealand University Hospital); Hans C. Hasselbalch (Zealand University Hospital); Morten Andersen (Roskilde University); Johnny T. Ottesen (Roskilde University); Thomas Stiehl (RWTH Aachen University)

SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Polycythaemia Vera (PV) is a haematological malignancy characterised by an overproduction of blood cells. Recent clinical studies have shown that combination therapy with interferon-α-2a (IFN-α) and

ruxolitinib (RUX) yields substantial reductions in *JAK2* V617F variant allele frequency (VAF), a measure of disease burden. Here, we study the mechanisms behind the observed reductions in VAF using mathematical modelling and Bayesian inference. We consider a multi-compartmental ODE model which accounts for the self-renewal, differentiation, and death of mature and immature blood cells in presence and absence of treatment. The cell kinetics depend on non-linear regulatory feedbacks. We combine our previously developed models of single-drug treatment with IFN-α and RUX in order to obtain a model for the combination treatment. Using prior distributions based on the single-drug studies, we fit the model to data from 22 patients and infer the effects of combination treatment. The combination model accurately captures the data, demonstrating that the knowledge of single-drug treatment effects is sufficient for explaining combination treatment data. The combination model suggests an explanation for the observed efficacy of the combination treatment: IFN-α forces malignant stem cells to differentiate, depleting the malignant stem cell pool that drives the disease. RUX targets malignant progenitor and precursor cells which quickly reduces the mutated cell burden.

CT1372 — Modeling the Heterogeneous Efficacy of Broad-Spectrum Medical Countermeasures: A Hybrid Approach from Outbreak Containment to Disease Burden Mitigation

SPEAKERS Youngsuk Ko (University of California, Berkeley)
AUTHORS Youngsuk Ko (University of California, Berkeley); Jacob Stapley (University of California, Berkeley); Geetha Jeyapragasan (University of California, Berkeley); Elena Chan (University of California, Berkeley); Charles Whittaker (University of California, Berkeley)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 11.33 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Pandemics are expected to emerge more frequently due to population growth and climate change. Alongside traditional vaccines, broad-spectrum medical countermeasures offer potential to control diverse, unknown pathogens. We developed a hybrid mathematical framework to evaluate these interventions, combining an individual-based network branching process for early stochastic outbreaks with a deterministic SIR-type compartmental model for large-scale transmission. The model incorporates key pathogen traits (basic reproductive number, incubation, symptom ratio, and severity) and simulates antivirals with multi-faceted mechanisms affecting transmission, susceptibility, and mortality. Applying this to profiles like Influenza H1N1 and SARS-CoV-2, we found that optimal distribution strategies are highly sensitive to pathogen kinetics and the epidemic phase. For pathogens with high transmissibility and short generation times, early ring-administration often fails to contain the outbreak. However, during widespread transmission, shifting focus to high-risk groups substantially reduces hospitalization and severity. Our results demonstrate that a "one-size-fits-all" approach to pandemic preparedness is inadequate. Effective stockpiling and deployment require dynamically tailored strategies that balance early containment with late-stage mitigation, rigorously accounting for pathogen-specific kinetics and the evolving scale of transmission.

CT1556 — The Effectiveness of Contact Tracing on Clustered Networks: An Event-Driven Network Simulation Study

SPEAKERS Martin Bootsma (Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht); Oluwapelumi Stephen Awolude (Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht)
AUTHORS Janneke Heijne (GGD Amsterdam); Jizzo Bosdriesz (GGD Amsterdam); Martin Bootsma (Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht); Oluwapelumi Stephen Awolude (Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht); Pieter Trapman (Bernoulli Institute, Faculty of Science and Engineering, University of Groningen)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 15.21 - SZ
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Contact tracing (CT) is a targeted intervention that can reduce transmission during infectious disease outbreaks without requiring population-wide restrictions. Most mathematical models evaluate CT using transmission-tree frameworks that assume independent infection events and don't explicitly account for the underlying contact network. These approaches ignore that traced susceptible contacts may temporarily quarantine and that, in clustered networks, CT may identify cases infected by individuals other than the index case.

We develop a stochastic event-driven epidemic simulation model that represents transmission and interventions directly on contact networks. Infection, detection, isolation, testing, and CT events are scheduled in continuous time and processed through a priority queue, allowing transmission and interventions to interact dynamically on the network. The framework allows flexible generation-time and detection-time distributions without restrictive Markovian assumptions.

Using this model, we compare the effectiveness of isolation, one-step CT, and multi-step CT across unclustered and clustered network structures. Our simulations show that network clustering can enhance the effectiveness of CT at moderate transmission levels by enabling quarantining of traced contacts to interrupt transmission within overlapping neighborhoods. Tracing both susceptible and infected contacts also yields a small but consistent improvement over tracing infected contacts alone.

CT1339 — The elasticity of SARS-CoV-2 transmission across the demographic landscape

SPEAKERS Rey Audie Escosio (Faculty of Sciences, University of Lisbon)
AUTHORS Ana Nunes (Faculdade de Ciências da Universidade de Lisboa); Benedetta Canfora (Faculdade de Ciências da Universidade de Lisboa); Ganna Rozhnova (University Medical Center Utrecht); Otilia Boldea (Tilburg School of Economics and Managemet, Tilburg University); Rey Audie Escosio (Faculty of Sciences, University of Lisbon)
SCHEDULE Thursday, 16 July 2026; 17:40–18:00; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

Age-structured heterogeneity in contact behavior, susceptibility, and severity makes epidemic dynamics difficult to interpret from aggregate measures alone. The effective reproduction number $R_e(t)$ collapses this heterogeneity into a single quantity, concealing which subgroups drive transmission and how interventions reshape that balance over time.

We extend elasticity analysis, a method rooted in ecology and population dynamics, to $R_e(t)$. Our framework decomposes $R_e(t)$ into time-varying contributions from each age group, tracking how these shift as epidemic conditions and interventions evolve.

We apply this to age-structured SARS-CoV-2 transmission models fitted to epidemiological data via Bayesian inference, evaluating model outputs across multiple epidemic periods and counterfactual school-based scenarios. In Portugal, adults dominated transmission throughout 2020, yet counterfactual simulations reveal that children and adolescents would have played a decisive role had schools remained open. In the Netherlands, adult dominance was confined to the first lockdown, giving way to adolescents in late 2020 and children in 2021. Across counterfactuals, adolescents consistently exerted stronger early influence than children.

These findings underscore that the dynamical role of any subgroup is not intrinsic but contingent on the surrounding intervention landscape, a contingency that elasticity decomposition of $R_e(t)$ makes explicit and quantifiable.

CT1640 — A reference panel assisted geometric structure non-negative matrix factorization for the deconvolution of bulk tissue RNA-seq data

SPEAKERS Shaoyu Li (University of North Carolina at Charlotte)
AUTHORS Nilüfer Ertekin-Taner (Mayo Clinic); Suxian Zhou (University of North Carolina at Charlotte); Su Xu (University of North Carolina at Charlotte); Xue Wang (Mayo Clinic); Duan Chen (University of North Carolina at Charlotte); Shaoyu Li (University of North Carolina at Charlotte)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.34 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Deconvolution of bulk RNA-seq data for simultaneous estimation of cell-type-specific gene expression profiles (GEPs) and relative cell abundances can be formulated as a constrained matrix factorization problem, but the widely used nonnegative matrix factorization (NMF) framework is mathematically ill-posed, as multiple factorizations may explain the observed bulk expression matrix equally well without further assumptions. Principled approaches for enforcing identifiability and improving estimation robustness remain underdeveloped.

We introduce GSNMF+, an augmented geometric-structure-guided NMF framework that imposes theory-motivated identifiability and solvability constraints on both factor matrices. The method leverages geometric structure associated with marker genes in bulk expression space to stabilize recovery of latent GEPs, and augments the factorization with artificially generated pseudo-bulk samples to strengthen the linear dependence between cellular composition and marker-gene expression. This augmentation improves robustness to instability in the underlying inverse problem. In addition, GSNMF+ includes a component annotation module to facilitate biological interpretation of inferred components. Evaluations on simulated data and realistic Plasmodium bulk RNA-seq datasets show that GSNMF+ accurately recovers latent stage compositions and exhibits greater stability and reliability than existing deconvolution methods across a range of settings.

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CT1597 — Deep reinforcement learning driven evolutionary therapy design for non-small cell lung cancer

SPEAKERS Laura Jansen-Storbacka (Delft University of Technology)
AUTHORS Alethea Barbaro (Delft University of Technology); Kateřina Staňková (Delft University of Technology); Laura Jansen-Storbacka (Delft University of Technology); Sepinoud Azimi (Delft University of Technology)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 01.14 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Non-small cell lung cancer (NSCLC) treated with tyrosine-kinase inhibitors (TKIs) initially responds well but rapidly develops resistance. This happens because in heterogeneous tumour microenvironments, continuous standard-of-care treatment rapidly eliminates drug sensitive cells, enabling resistant populations to dominate and leading to treatment failure. Evolutionary therapies (ET), guided by mathematical models, aim to delay treatment failure and prolong survival by maintaining a sufficient sensitive population to suppress resistance, and have shown promise in recent prostate cancer trials. Clinical implementation of ET in NSCLC is challenging due to its fast growth and limited biomarkers. Recent work suggests that deep reinforcement learning (DRL) can generate more robust, patient specific treatment schedules. We present a DRL framework tailored to NSCLC treated with TKIs. Using virtual patients based on clinical data, our learned treatment schedules predict improved survival across all patients compared with both standard-of-care protocols and conventional ET strategies. We then extend the DRL reward structures to incorporate patient specific quality of life preferences, enabling evaluation of treatment policies through quality-adjusted survival and supporting more clinically meaningful decision making.

CT1226 — Evaluating infectious disease forecasts in a cost-loss situation

SPEAKERS Philip Gerlee (Chalmers University of Technology & University of Gothenburg)
AUTHORS Anna Saxne Jöud (Lund University); Henrik Thorén (Lund University); Philip Gerlee (Chalmers University of Technology & University of Gothenburg); Torbjörn Lundh (Chalmers University of Technology & University of Gothenburg)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.46 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

In order for epidemiological forecasts to be useful for decision-makers the forecasts need to be properly validated and evaluated [Cra+22]. Although several metrics fore evaluation have been proposed and used none of them account for the potential costs and losses that the decision-maker faces. We have adapted a decision-theoretic framework to an epidemiological context which assigns a Value Score (VS) to each model by comparing the expected expense of the decision-maker when acting on the model forecast to the expected expense obtained from acting on historical event probabilities [Wil01]. The VS depends on the cost-loss ratio and a positive VS implies added value for the decision-maker whereas a negative VS means that historical event probabilities outperform the model forecasts. We apply this framework to a subset of model forecasts of influenza peak intensity from the FluSight Challenge and show that most models exhibit a positive VS for some range of cost-loss ratios [Rei+19]. However, there is no clear relationship between the VS and the original ranking of the model forecasts obtained using a modified log score. This is in part explained by the fact that the VS is sensitive to over- vs. underprediction, which is not the case for standard evaluation metrics. We believe that this type of context-sensitive evaluation will lead to improved utilisation of epidemiological forecasts by decision-makers.

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CT1645 — Forcing-Driven Nonlinear Dynamics of Satellite-Informed Agro–Forestry Socio-Ecological Systems

SPEAKERS Gerard Olivar-Tost (Universidad Católica del Maule)
AUTHORS Deissy Milena Sotelo-Castelblanco (Universidad Nacional de Colombia); Gerard Olivar-Tost (Universidad Católica del Maule)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 11.11 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Agro–forestry socio-ecological systems in high-latitude regions are strongly influenced by climate variability, snow dynamics, and land-use change, leading to nonlinear behaviors and regime shifts [al01]. We develop a satellite-informed dynamical systems framework to model these systems under environmental forcing.

The objective is to construct a non-autonomous system of differential equations capturing threshold mechanisms and transitions between regimes. Satellite-derived variables (Sentinel data), including vegetation productivity, pasture–forest distribution, and carrying capacities, are incorporated as parameters and time-dependent forcing terms.

The model exhibits nonlinear dynamics driven by the interaction between internal feedbacks and external forcing. We analyze equilibrium structures under quasi-stationary conditions, local stability, and bifurcation mechanisms [Kuz04] associated with sustainability and collapse regimes. Composite parameters linking grazing pressure, forest cover, and climate forcing define critical thresholds.

A stochastic extension with multiplicative noise reveals shifts in stability and variability around equilibria. Numerical simulations illustrate sensitivity to forcing and the emergence of nontrivial trajectories.

This framework contributes to nonlinear and hybrid dynamical systems by providing a data-informed approach to modeling biologically relevant systems under non-autonomous and stochastic dynamics.

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CT318 — Investigating the impact of infusion time-of-day on the antitumour efficacy of immune checkpoint inhibitors

SPEAKERS Marie Haghebaert (Inserm U1331, Institut Curie, Mines-Paris, PSL Univ., France)
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SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 01.18 - SZ
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Accumulated evidence suggests that the antitumour efficacy of immune checkpoint inhibitors (ICI) may depend on infusion time-of-day. Although circadian regulation of many immune processes has been documented [Lév+24], the mechanisms of such dosing time dependency remain unclear. To investigate this, we developed models of ICI pharmacokinetic (PK) and pharmacodynamic (PD) accounting for key circadian rhythms.

First, simulations of an existing PK model [Baj+17] - extended to incorporate parameter circadian variability - showed no dosing time differences in either central or peripheral exposure. Sobol sensitivity analyses indicated that this behaviour was mainly driven by ICI slow clearance. Similar results were obtained when modelling patient-reported clearance modifications over weekly cycles.

Inspired by [Jaf+19], we developed models of ICI PD stepwise, starting with a tumour-free model of T-cell trafficking and extending it to include tumour-driven T-cell activation, antigen-presenting cell dynamics, and cancer-mediated inhibition of cytotoxic T cells reversed by ICI. Parameters were calibrated using clinical data or preclinical extrapolation [Wan+23; For+24]. Models achieved a good fit to data. Sensitivity analyses are ongoing to identify parameters driving infusion time-of-day dependencies.

This work provides a mechanistic framework to investigate ICI chronoPK-PD. An ongoing prospective study will generate circadian immunological data to refine and validate these models.

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CT1559 — Mathematical Modeling of Tuberculosis Transmission in Comorbid Populations: A Six-Compartmental Stability Analysis

SPEAKERS Abigael Dangate (LAMFA, University of Picardie Jules Verne)
AUTHORS Abigael Dangate (LAMFA, University of Picardie Jules Verne); Mohamed Guedda (LAMFA, University of Picardie Jules Verne); Nabil Bedjaoui (LAMFA, University of Picardie Jules Verne)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.21 - SZ
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

This study presents a six-compartment deterministic model for tuberculosis TB) transmission dynamics in populations stratified by comorbidity status. TB caused 1.3 million deaths globally in 2023, while diabetes(a major TB comorbidity), affects 537 million adults and contributes to $15-20\%$ of TB cases worldwide [Bi+25; Kum+24]. The syndemic is most severe in high-burden nations: India, Indonesia, China and the Philippines account for nearly half of the 10.8 million annual TB cases, with diabetes prevalence among TB patients exceeding 15% globally reaching $> 45\%$ in parts of the Western Pacific [Kum+24]. The model includes elevated TB susceptibility in comorbid individuals ($\alpha_c \geq \alpha$), accelerated progression to active disease ($\delta_c \geq \delta$), increased mortality ($\nu_c \geq \nu$), reduced recovery ($\gamma_c \leq \gamma$), and

comorbidity acquisition θ. Cross-transmission between subpopulations occurs via weighted infectious pressure $T = \beta I + \beta_c I_c$. The Jacobian matrix is derived to enable equilibrium stability analysis; the basic reproduction number $\mathcal{R}_0$ is computed, and critical parameters driving TB persistence are identified. Numerical simulations show that rising comorbidity prevalence substantially increase endemic TB burden, risking elimination goals.

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CT1330 — Mechanistic modeling of extracellular vesicle–mediated immune evasion in Candida albicans

SPEAKERS Anastasia Solomatina (Leibniz Institute for Natural Product Research and Infection Biology, Hans Knöll Institute, Jena, Germany)

AUTHORS Yann Bachelot (Department of Applied Systems Biology, Leibniz Institute for Natural Product Research and Infection Biology - Hans Knöll Institute (Leibniz-HKI), Jena, Germany; Faculty of Biological Sciences, Friedrich Schiller University, Jena, Germany); Kerstin Hünniger-Ast (University of Würzburg, Institute for Hygiene and Microbiology, Würzburg, Germany; Fungal Septomics, Leibniz Institute for Natural Product Research and Infection Biology - Hans Knöll Institute, Jena, Germany); Natalie Nieuwenhuizen (University of Würzburg, Institute for Hygiene and Microbiology, Würzburg, Germany); Jennifer Patitz (University of Würzburg, Institute for Hygiene and Microbiology, Würzburg, Germany); Oliver Kurzai (University of Würzburg, Institute for Hygiene and Microbiology, Würzburg, Germany; Fungal Septomics, Leibniz Institute for Natural Product Research and Infection Biology - Hans Knöll Institute, Jena, Germany); Anastasia Solomatina (Leibniz Institute for Natural Product Research and Infection Biology, Hans Knöll Institute, Jena, Germany); Marc Thilo Figge (Department of Applied Systems Biology, Leibniz Institute for Natural Product Research and Infection Biology - Hans Knöll Institute (Leibniz-HKI), Jena, Germany; Institute of Microbiology, Faculty of Biological Sciences, Friedrich Schiller University, Jena, Germany)

SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.27 - SR

SESSION Contributed Talks

TRACK Systems Biology and Biochemical Networks

Abstract

Candida albicans can cause life-threatening invasive infections, yet in an *ex vivo* human whole-blood infection model a substantial fraction of fungal cells remains extracellular despite the presence of phagocytes. Previous work suggested that fungal cells acquire host-derived surface markers via binding of neutrophil-derived extracellular vesicles (EV), reducing their uptake by immune cells.

To investigate the emergence of this immune-evasive phenotype, we extended a previously developed state-based virtual infection model of host–pathogen interactions [Hün+14]. Specifically, we formulated two mechanistic models representing alternative hypotheses for the relationship between EV-decoration and immune evasion, where the immune evasion either (i) depends on the EV-decoration or (ii) occurs independently of it.

The decoration-dependent model overestimated the amount of free extracellular pathogens. In contrast, the decoration-independent mechanism best reproduced the data and predicted that 40% of fungal cells first become EV-decorated and subsequently immune-evasive, whereas 60% follow the reverse order. The predicted pathway distribution strongly depends on the time scale of decoration: if it occurs within minutes, nearly all fungal cells become EV-decorated prior to immune evasion, effectively reducing the system to the decoration-independent mechanism. The model predicts targeted follow-up experiments to discriminate between proposed mechanisms.

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CT1516 — Population dynamics of gene drives with density-dependent fitness costs

SPEAKERS Adam Lampert (The Hebrew University of Jerusalem)
AUTHORS Sviatoslav R. Rybnikov; Gili Greenbaum; Adam Lampert (The Hebrew University of Jerusalem)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Gene drives are engineered genetic constructs with biased inheritance that can spread in a population despite deleterious fitness effects, and are a promising tool for suppressing malaria mosquitoes, invasive rodents and other pest species. A key safety requirement for field deployment is the predictability of gene drive dynamics in natural populations. Most existing gene drive models assume a constant fitness cost of the drive allele, whereas under natural settings the fitness cost will likely vary as the gene drive alters the population density, in response to the changing environmental conditions and intraspecific competition conditions [DLG20; Kim+23]. Here, we develop a mathematical model in which the fitness cost depends on population density. We show that introducing such eco-evolutionary feedback considerably expands the range of outcomes that can be attained. For example, gene drives that are expected to spread or disappear without density dependence may instead exhibit threshold-dependent spread, stable polymorphism with the wild-type allele, or sustained oscillations. Threshold-dependent drives can generate even richer dynamics, including non-trivial bistability between fixation and polymorphism or between fixation and oscillations. Our results demonstrate that complex regimes may arise even in a single panmictic population, in contrast to previous views that such oscillatory dynamics require interacting populations [HG23] or explicit population structure [Cha+21].

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CT1378 — The evolution of latency in tuberculosis

SPEAKERS Alexander Beams (Simon Fraser University)
AUTHORS Alexander Beams (Simon Fraser University)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 11.33 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

The observation that infectious agents (such as viruses and bacteria) limit their ability to persist if they kill their hosts too rapidly underpins a large body of work related to the evolution of virulence. Theory stemming from this explains many phenomena, such as the sustainment of deadly infectious diseases in large, dense populations. However, it is not clear whether the existing theory explains one of the most salient features of tuberculosis (TB) epidemiology, which is its capacity to cause latent (symptom-free and non-infectious) disease for decades before transitioning to a symptomatic and infectious stage. TB’s epidemiology today is likely shaped by its evolutionary history with humans spanning over ten thousand years. During much of this time, host populations were smaller and more dispersed than today, and local eradication of disease would have posed a challenge for the pathogen. With a view to the impact of demographic stochasticity in small, weakly interconnected populations, we examine how latency might have helped the disease survive to the present day, where it continues to plague humanity.

CT1349 — Time Until Disease Detection in a Stochastic Epidemic Model with Imperfect Vaccination

SPEAKERS Diana Taipe Hidalgo (Complutense University of Madrid)
AUTHORS Diana Taipe Hidalgo (Complutense University of Madrid); María Jesús Lopez-Herrero (Complutense University of Madrid); Verdiana Mustaro (Università degli Study di Salerno)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 11.32 - SR
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

We study the detection of a contagious disease using a stochastic SVIR (susceptible–vaccinated–infected–removed) epidemic model. In this framework, vaccination is introduced as a preventive measure. However, the administered vaccine may fail to protect some individuals, and vaccine-induced immunity may wane over time.

The epidemic dynamics of this model are described through a continuous-time Markov chain, which allows us to analyze the time required for disease detection, the total number of infections that occur before confirmation, and the number of infections arising within the vaccinated population due to vaccine failure. By exploiting the transition structure of the underlying stochastic process, we derive recursive schemes for computing the moments and probabilities of these quantities.

The theoretical results are illustrated in the setting of outbreaks of foot-and-mouth disease in cattle. We consider several scenarios to show how imperfect vaccination and detection delays influence epidemic dynamics during the early stages of an outbreak.

CT1481 — What happens when an epidemiologist gets refractory giardiasis? Within-host modelling of duodenal infection dynamics and oral treatment

SPEAKERS David Haw (University of Liverpool)
AUTHORS David Haw (University of Liverpool)
SCHEDULE Thursday, 16 July 2026; 18:00–18:20; 11.34 - SR
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Giardiasis is usually treated successfully with short oral courses of nitroimidazoles or related agents, yet refractory infection remains clinically important and mechanistically under-modelled. We present a human within-host model for Giardia duodenalis motivated by a prolonged refractory infection in the presenting author, an epidemiologist. The model links trophozoite and cyst dynamics in the duodenum to oral treatment regimens, incorporating uncertain parasite growth, encystation, host clearance, and optional microbiome-mediated suppression. A central feature is explicit representation of duodenal drug exposure, rather than relying only on plasma pharmacokinetics, since orally administered drugs physically reach the proximal small intestine before systemic absorption. Parameters are informed by drug-label pharmacokinetics and published treatment-effectiveness estimates for metronidazole, tinidazole, albendazole, quinacrine, and combination regimens, with uncertainty propagated throughout calibration. The calibrated model is then used to examine whether prolonged or combination treatments are expected to reduce infection duration and treatment-failure risk, and to compare plausible mechanisms underlying acute versus chronic infection. More broadly, the study asks how much clinically observed refractory giardiasis can be explained by local exposure, within-host persistence, and uncertainty in treatment effect. Spoiler alert: the epidemiologist is cured.

CT1306 — A distance-space framework for inferring selection from time-resolved trait data

SPEAKERS Farnoush Farahpour (Quantitative Oncology and Systems Modeling, University of Duisburg-Essen)
AUTHORS Sina Roshandel (Quantitative Oncology and Systems Modeling, University of Duisburg-Essen); Kamran Kaveh (Therapy Modeling and Design Center, University of Minnesota); Farnoush Farahpour (Quantitative Oncology and Systems Modeling, University of Duisburg-Essen)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 11.32 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Neutral birth–death–mutation dynamics in finite populations can produce patterns in low-dimensional summaries of trait-distance space (e.g., mean pairwise distance) often interpreted as signatures of selection, making inference of selection from snapshot data fundamentally ambiguous. We introduce a distance-space framework for finite-length, one-parent Moran populations that separates neutral dynamics from selective bias directly at the level of trait distances.

Our key observable is a parent-bias signal defined as the difference between two distance repertoires: distances between reproducing parents (with higher fitness) and individuals randomly selected for death, and distances obtained by uniformly sampling birth–death pairs from the population. The resulting ODE for pairwise-distance dynamics decomposes into an exact neutral baseline and a measurable parent-bias forcing term. The baseline is determined by mutation transition probabilities at birth, while the forcing term is obtained by applying the same mutation kernel to the parent-bias signal.

By using forcing inputs obtained from agent-based simulations with recorded parental histories, the ODE reproduces both selection-driven convergence and mutation-driven relaxation of pairwise distances. A first-moment summary of the parent-bias signal provides a quantitative readout of selection timing and strength. The framework applies whenever parent–offspring relationships (or equivalent parental assignments) are available.

CT221 — A hybrid response surface–ODE framework for multiscale predictions of microalgae growth dynamics

SPEAKERS Mélanie Pietri (Université Paris-Saclay, ENS Paris Saclay, CNRS, Satie & Université Paris-Saclay, CNRS, ENS Paris Saclay, LMF)
AUTHORS Thomas Rodet (Université Paris-Saclay, ENS Paris Saclay, CNRS, Satie); Bruno Le Pioufle (Université Paris-Saclay, ENS Paris-Saclay, CNRS, LuMIn); Thomas Nowak (Université Paris-Saclay, CNRS, ENS Paris Saclay, LMF & Institut Universitaire de France); Matthias Függer (Université Paris-Saclay, CNRS, ENS Paris Saclay, LMF); Sakina Bensalem (Université Paris-Saclay, ENS Paris Saclay, CNRS, Satie); Mélanie Pietri (Université Paris-Saclay, ENS Paris Saclay, CNRS, Satie & Université Paris-Saclay, CNRS, ENS Paris Saclay, LMF)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.21 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Accurately predicting microbial growth across varying environmental conditions and cultivation scales remains a central challenge in mathematical biology. ODE models offer mechanistic interpretability but typically rely on fixed kinetic parameters, restricting their validity to narrow operating ranges. Data-driven approaches such as Response Surface Methodology (RSM) [BL21] accommodate parameter variability yet either disregard temporal dynamics or risk generating biologically implausible trajectories.

We propose a hybrid framework embedding bounded, biologically interpretable RSM surfaces into ODE systems, letting kinetic parameters vary continuously with environmental factors. We validate the framework on microalgae cultures grown in microplates (200 µL) and flasks (50 mL), spanning 85 combinations of light intensity and nutrient availability for a total of over 1,300 growth curves. The model captures nonlinear light–nutrient interactions, predicts growth trajectories under untested conditions, and consistently outperforms classical mechanistic benchmarks [Kam+22; MMB20].

We further show that microplate- and flask-derived parameter surfaces are related through morphological transformations, suggesting that the functional structure of growth responses is conserved across a 250-fold volume increase. This opens perspectives for high-throughput screening strategies [Wol+24] where small-scale experiments inform larger-scale bioprocess design.

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CT1218 — A Novel Immunological Therapeutic Strategy Based on Morphology-Defined Endotypes of Skin Disease

SPEAKERS Ying Xie (Kyoto University)
AUTHORS Ryo Saito (Hiroshima University); Daiki Matsubara (Hiroshima University); Shunsuke Takahagi (JA Hiroshima General Hospital); Michihiro Hide (Hiroshima University); Takahiro Hiraga (Kyoto University); Ying Xie (Kyoto University); Sungrim Seirin-Lee (Kyoto University)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 01.15 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Chronic spontaneous urticaria (CSU) is an immune-mediated skin disease characterized by red, itchy eruptions of various shapes, known as wheals. Second-generation H1 antihistamines are a mainstay of treatment, but treatment responses vary widely among patients, with approximately 30% remaining symptomatic despite conventional therapy. On the other hand, the results of our previous studies suggest that CSU can be classified into five distinct types of wheal morphology, which corresponds well to clinical observations, and new, more effective targeted therapies could be developed based on these endotypes. To address this, we studied the effectiveness of antihistamines across these endotypes. We first evaluated the severity of CSU and efficacy of antihistamines *in silico* using three key measures: wheal area, itching severity, and wheal expansion dynamics. Our results showed that even though we assumed the same intensity of H1 histamine receptor suppression, the overall effectiveness to reduce the symptom varied depending on the endotypes. Furthermore, we found a key pathological network associated with antihistamine efficacy, which enables the proposal of new targeted immune drugs for the treatment of CSU. Our study demonstrates that mathematical and morphological endotyping of CSU can uncover hidden therapeutic determinants and pave the way for next-generation targeting critical components in the mechanism of individual patients.

CT1279 — Existence of periodic traveling waves in neural fields with direction preference

SPEAKERS Maren Bråthen Kristoffersen (Norwegian University of Life Sciences)
AUTHORS Maren Bråthen Kristoffersen (Norwegian University of Life Sciences); Pierre Roux (Centrale Lyon); Susanne Solem (Norwegian University of Life Sciences)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 11.01 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Since their introduction, neural fields have been a popular topic of research. However, most existing studies fix specific parameter choices or make physical approximations. The aim of this presentation is to provide a general and mathematically rigorous framework for traveling wave solutions in neural fields with direction preference, modeled by an asymmetric connectivity kernel, on a periodic domain (torus). The existence of traveling waves is proven by bifurcation theory. In addition, numerical bifurcation analyses will be presented to enhance understanding of the traveling waves beyond the theoretical results. Lastly, if time permits a discussion of the stability of these traveling waves will be included.

CT326 — Exploring reaction-diffusion equations: Modelling wildfires and resistance development

SPEAKERS Luca Madita Nieding (TU Braunschweig)
AUTHORS Luca Madita Nieding (TU Braunschweig)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 12.01 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Reaction-diffusion equations exhibit a wide range of spatial and temporal behaviours and are often used to describe propagating phenomena in biological systems. In this talk, we present two such models. One describes the spread of wildfires, the other the development of resistance in agriculture. We compare how the reaction terms relate to the dynamics of the system, considering similarities and differences. Moreover, we expand the model family by exploring the influence of spatial barriers on the dynamics numerically.

CT1478 — From Checkerboards to Synchrony: How Mobility Alters Coupled Oscillator Dynamics

SPEAKERS Tilmann Glimm (Western Washington University)
AUTHORS Tilmann Glimm (Western Washington University)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 10.01 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Synchronization of coupled oscillators is a common phenomenon in biological systems. A closely related phenomenon, anti-synchronization, occurs when neighboring elements adopt opposite phases, as seen in checkerboard-like patterns in cell populations. Such patterns have been observed in the auditory sensory epithelium.

We investigate systems of coupled Kuramoto-type oscillators with identical periods and nearest-neighbor interactions. Such models have been well studied on static networks, but the effect of spatial rearrangements of the oscillators has received much less attention. We add attraction and repulsion to these models (following either the “like attracts like” rule or the “opposites attract” rule) and show how this affects the

ability to achieve global synchrony or anti-synchrony in this and related models. One surprising finding is that the "opposites attract" rule tends to enhance the likelihood of reaching global synchrony.

CT197 — Heavy-tailed dormancy and the role of cancer cell death in metastatic reactivation: a data-driven mathematical modelling study

SPEAKERS Nikolaos Sfakianakis (University of St Andrews)
AUTHORS Dimitrios Katsaounis (RWTH Aachen University); Mark Chaplain (University of St Andrews); Nikolaos Sfakianakis (University of St Andrews)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Metastatic relapse can occur years after diagnosis, highlighting the need to better understand the mechanisms behind cancer cell dormancy and reactivation.

Here we present a mathematical model, [SKC25] that integrates cancer-cell dormancy, reactivation, proliferation and death, tested/calibrated with experimental murine metastasis datasets. The model couples population dynamics with statistical representations of dormancy times, enabling for a systematic comparison of various probability distribution functions.

To this end we study two complementary experimental settings: a) lung metastasis including post-extravasation cancer cell death, [Cam+00] and b) liver metastasis without cell death, [Nau+99]. When cancer cell death is present, heavy-tailed dormancy distributions better fit to the experimental tumour progression data, and predict prolonged dormancy periods consistent with delayed metastatic relapse. In contrast, when cell death is absent, heavy- and light-tailed distributions perform comparably, indicating that heavy-tails emerge as a compensatory mechanism in the presence of cell attrition. These results demonstrate the significance of selecting biologically relevant metastatic progression models.

We conclude that the proposed mathematical model is able to provide a qualitative and quantitative basis for investigating dormancy-driven metastasis and offers a new direction into integrating experimental data with mathematical models for metastasis insights.

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CT327 — HORIZON: Hierarchical Optimization for Residual Inference with Zero-drift Ornstein-Uhlenbeck Noise

SPEAKERS Yuhong Liu (Bonn Center for Mathematical Life Sciences, University of Bonn)

AUTHORS Dilan Pathirana (Bonn Center for Mathematical Life Sciences, University of Bonn); Domagoj Doresic (Bonn Center for Mathematical Life Sciences, University of Bonn); Jan Hasenauer (Bonn Center for Mathematical Life Sciences, University of Bonn); Yuhong Liu (Bonn Center for Mathematical Life Sciences, University of Bonn)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Parameter estimation for ordinary differential equation (ODE) models of biological systems commonly assumes independent and identically distributed (IID) measurement noise. However, many experimental techniques, such as fluorescence measurements or western blots, yield data proportional to or shifted from true species concentrations, requiring unknown scaling and offset parameters. In addition, residuals from biological time series often exhibit temporal autocorrelation, violating the IID assumption and potentially biasing inference. Here, we present HORIZON, a hierarchical optimization framework that jointly accounts for relative measurement scales and temporally correlated noise by integrating scaling and offset parameters with an Ornstein–Uhlenbeck (OU) process noise model. Within the hierarchical formulation, these observable parameters can be analytically eliminated, reducing the optimization to only mechanistic parameters. We derive closed-form solutions for all observable parameters and provide analytical gradients for efficient parameter estimation. Using profile likelihood analysis, we quantify differences in parameter uncertainty between correctly and incorrectly specified noise models. Furthermore, we show how experimental sampling design influences OU parameter identifiability, and propose a diagnostic workflow to detect autocorrelation and model misspecification directly from data.

CT1299 — Information degradation across data transformations affect causal inference

SPEAKERS Henrik Høiberg Jessen (Umeå University)
AUTHORS Henrik Høiberg Jessen (Umeå University); Per Stenberg (Umeå University); Åke Brännström (Umeå University)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Inferring causal relationships from ecological time series enables the study of ecosystem dynamics without direct observation. Methods such as Unified Information-Theoretic Causality (UIC) make this possible but assume independent abundance measurements. In many ecological applications, particularly eDNA studies, data are compositional and contain only relative abundances. The common remedy is log-ratio transformation to restore Euclidean structure, but this introduces shared noise across taxa that may obscure causal signals.We evaluated UIC's ability to recover known causal relationships from simulated ecological communities under varying process and observational noise. Time series were generated using generalized Lotka–Volterra dynamics for systems with 3, 10, or 30 interacting species. Each dataset was analyzed as original abundances, closed compositions, and CLR-transformed compositions. As expected, causal inference performed best on original abundance data. However, analyses using compositional data consistently outperformed CLR-transformed data. This advantage increased with community size, with compositional data even approaching the performance of raw abundance data at high dimensionality. These findings indicate that CLR transformation can degrade rather than improve causal signal detection in compositional ecological data, suggesting that untransformed compositional data may be preferable for causal inference.

CT1477 — Machine Learning–Based Classification of Microsatellite Instability in Colorectal Carcinoma Using PCR Fragment Length Analysis

SPEAKERS Isabel Gernand (Heidelberg Institute for Theoretical Studies (HITS) and Engineering Mathematics and Computing Lab, IWR, Heidelberg University)
AUTHORS Isabel Gernand (Heidelberg Institute for Theoretical Studies (HITS) and Engineering Mathematics and Computing Lab, IWR, Heidelberg University); Maria Kessler (Heidelberg University Hospital, Department of Applied Tumor Biology); Matthias Kloor (Heidelberg University Hospital, Department of Applied Tumor Biology); Vincent Heuveline (Heidelberg Institute for Theoretical Studies (HITS) and Engineering Mathematics and Computing Lab, IWR, Heidelberg University)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Microsatellite instability (MSI) describes the accumulation of length alterations in microsatellite loci caused by deficiencies in the DNA mismatch repair system. In clinical diagnostics, MSI status is commonly assessed using polymerase chain reaction (PCR) assays targeting a panel of microsatellite markers. This testing plays an important role in the molecular characterization of colorectal cancer and in identifying patients who may benefit from specific therapeutic strategies. The electropherogram signals represent fragment length distributions whose interpretation is typically based on predefined rules comparing tumor and reference samples. However, signal variability and borderline cases can make this evaluation challenging. We investigate the use of machine learning models for MSI classification based on PCR fragment analysis data, exploring embedding and feature engineering strategies to distinguish MSI-negative and MSI-positive cases by capturing characteristic patterns in the fragment profiles. In addition, the model is used to assess the number and contribution of biomarkers required for reliable prediction, indicating that accurate classification can be achieved with a reduced biomarker set. We further analyze the adaptability of the approach to other tumor types, such as endometrial carcinoma. Our results indicate that machine learning methods can reliably infer MSI status and may support automated interpretation and decision assistance in diagnostic workflows.

CT1490 — Mathematical Modelling and Numerical Simulations for Lipolysis on Lipid Droplets

SPEAKERS Reymart Salcedo Lagunero
AUTHORS Klemens Fellner; Reymart Salcedo Lagunero
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 11.03 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Lipolysis, also called lipid hydrolysis, is a life-essential biochemical process that releases fatty acids and glycerols stored in the form of water-insoluble triglycerides within lipid droplets of specialised cells known as adipocytes. The enzyme adipose triglyceride lipase (ATGL) is believed to be the rate-limiting and key regulator of lipolysis. However, recent studies show that besides lipolysis, it also catalyses in parallel a transacylation reaction of two diglycerides, the first downstream product of lipolysis of triglycerides, to produce one triglyceride and one monoglyceride molecule.

All previous mathematical modelling approaches that describe the lipolytic and transacylase activities of ATGL have used the Michaelis-Menten and mass-action-law as reaction rates, which can only be considered as approximative models with unknown accuracy as ATGL catalyses both lipolysis and transacylation on the same active site.

In this talk, we therefore derive kinetic models that describe both the lipolytic and transacylase activities of ATGL in a single system, which we base on the hypothesised molecular mechanisms of lipolysis and transacylation. We present two chemical reaction networks and derive quasi-steady-state-approximative systems. In addition, we present a first PDE modelling approach which considers that the enzymes act on or near the surface and thus, lipolysis and transacylation are localised. Finally, we provide simulations as well as parameter estimations.

CT1411 — Mathematical Modelling and Optimisation of Combined Oncolytic Virotherapy and Anti-PD-L1 Immunotherapy

SPEAKERS Hongfei Qiao (The University of Sydney)
AUTHORS Hongfei Qiao (The University of Sydney); Peter Kim (The University of Sydney)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.06 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Immune checkpoint inhibitors (ICIs) restore antitumour immunity by blocking checkpoint interactions such as PD-1/PD-L1, while oncolytic viruses (OVs) selectively infect and lyse tumour cells, releasing viral progeny, tumour associated antigens, and danger signals that further stimulate immune responses. Motivated by these complementary mechanisms, we study the combination of virotherapy and immunotherapy using an ordinary differential equation (ODE) model for oncolytic adenovirus carrying IL-12 and GM-CSF together with anti-PD-L1 treatment. We fit model parameters to a series of experimental data sets corresponding to different treatments and use the model to show that the combining oncolytic virotherapy with anti-PD-L1 achieves stronger and more durable tumour control than either therapy alone. We then investigate how treatment efficacy depends on the injection protocol. In particular, we study the effects of varying injection timing, frequency, and dose allocation. Our results indicate that treatment schedules can be adjusted to meet different clinical objectives and improve therapeutic outcomes. These findings provide a quantitative basis for the design and optimisation of combination cancer immunotherapy protocols.

CT1291 — Mechanistic mathematical modeling of primary myelofibrosis pathophysiology and progression

SPEAKERS Qiujie Wang (RWTH Aachen University)
AUTHORS Qiujie Wang (RWTH Aachen University); Steffen Koschmieder (Department of Hematology, Oncology, Hemostaseology, and Stem Cell Transplantation, Faculty of Medicine, RWTH Aachen University, and Center for Integrated Oncology Aachen Bonn Cologne Düsseldorf (CIO-ABCD), Aachen, Germany); Thomas Stiehl (Institute for Computational Biomedicine & Disease Modeling, RWTH Aachen University Hospital, Aachen, Germany and Center for Computational Life Sciences (CCLS) RWTH Aachen University, Aachen, Germany and Department of Science and Environment Roskilde University, Roskilde, Denmark)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 02.21 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Primary myelofibrosis (PMF) is a malignant clonal disease of the hematopoietic system. It is characterized by an excess of fiber production in the bone marrow and eventually leads to an impaired blood cell formation and an increased risk of leukemic transformation. Although driver mutations in the genes for JAK2, CALR, and MPL have been identified in most patients, the pathophysiologic mechanisms remain incompletely understood, and curative treatment options are limited. We developed a mechanistic non-linear ordinary differential equation model to investigate the dynamic interactions between healthy and malignant hematopoietic cells during PMF progression. The model incorporates competition for niche space, fibrosis-driven niche deterioration, differentiation dynamics and feedback regulations. The model successfully reproduces the dynamics of mature blood cells across different disease stages. In particular, leukocyte and platelet counts increase during the early disease phase and subsequently decline, consistently with clinical observations. Simulations further indicate that the clonal expansion and competition may start decades before clinical manifestation. Our model offers mechanistic insights into how niche deterioration and stem cell competition shape disease dynamics and contribute to inter-individual heterogeneity of the clinical course. The model can be used to identify potential therapeutic strategies aimed at preserving niche function and restoring hematopoiesis.

CT253 — Mind the Gaps: How the Spiral of Silence Drives Partisan Misperception in the United States Political System

SPEAKERS Daniel Levy (Princeton University)
AUTHORS Yphtach Lelkes (University of Pennsylvania); Naomi Leonard (Princeton University); Corina Tarnita (Princeton University); Daniel Levy (Princeton University)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.02 - HS
SESSION Contributed Talks
TRACK Miscellaneous

Abstract

Recent studies in political science have revealed a counterintuitive phenomenon: American partisans systemically underestimate the heterogeneity of opinions within each party. Yet, they correctly perceive each party's mean opinion. Since these studies were published, political scientists have speculated about the causes and effects of this phenomenon, but their results have remained vague due to insufficient temporal data for empirical analysis. We develop an individual-based model showing that such systemic misperception can emerge from well-understood local behavioral and interaction biases. We take the model to its continuum limit and, within a subset of the parameter space, we analytically show the emergence of partisan misperception and its dependence on system properties. We then extend the model to a second slower timescale via an opinion dynamics framework, where we demonstrate a mechanistic link between partisan misperception and political polarization. We hope to eventually be able to inform the development of tools that can mitigate polarization and radicalization, strategies that have the potential to be particularly effective because they leverage nonlinear feedback mechanisms already documented in the United States political system.

CT204 — Molecular noise modulates transitions in the cell-fate differentiation landscape

SPEAKERS Yujing Liu (University of Melbourne)
AUTHORS Adriana Zanca (The University of Melbourne); Lucy Ham (The University of Melbourne); Michael Stumpf (University of Melbourne); Yujing Liu (University of Melbourne)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 11.02 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Waddington's epigenetic landscape has become one of biology's cornerstone metaphors, widely used both conceptually and computationally. Cell types are often associated with the stable qualitatively stationary points or valleys of this landscape. In previous work, we showed that the molecular noise dominating sub-cellular dynamics can distort and profoundly reshape this landscape. In non-equilibrium systems, an equally profound question arises: to what extent does noise alter the transition paths between valleys in such dynamic landscapes. We tackle this question using a set of illustrative exemplars, and show that noise gives rise to paths that differ substantially from the canonical least-action paths calculated under deterministic dynamics. We dissect the dynamics of these exemplars and determine the reactive density and transition currents, which show us, respectively, where and how transitions occur for different realizations of stochastic dynamics. Our analysis unambiguously demonstrates that reaction paths for stochastic dynamics diverge non-trivially from their deterministic least-action paths or simple barrier crossing models.

CT1225 — Pattern Selection in mechanochemical model of pattern formation

SPEAKERS Szymon Cygan (Heidelberg University)
AUTHORS Szymon Cygan (Heidelberg University); Finn Münnich (Heidelberg University); Anna Marciniak-Czochra (Heidelberg University); Dietmer Oelz (University of Queensland)

SCHEDULE Friday, 17 July 2026; 09:30–09:50; 05.12 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

The freshwater polyp Hydra is a classical model organism for studying body axis formation due to its remarkable regenerative capacity. In regenerating tissue spheroids, mechanical cues arising from tissue deformation interact with biochemical signaling pathways and influence pattern formation.

In this talk I will propose a mechanochemical model for body axis formation in regenerating Hydra tissue spheroids that couples morphogen dynamics with tissue mechanics through a positive feedback loop: tissue stretching enhances morphogen production, while morphogen concentration modulates tissue elasticity. This coupling provides a mechanochemical realization of the local activation–long-range inhibition principle.

Within a reduced one-dimensional framework, I will perform a rigorous analytical study of pattern formation. I will prove the existence of stable non-constant steady states and show that all multimodal steady states are linearly unstable, implying that only unimodal patterns persist as stable solutions.

Bifurcation analysis reveals both subcritical and supercritical pitchfork bifurcations, with fold bifurcations generating bistable regimes. The results demonstrate that mechanochemical feedback alone can robustly generate single-peaked patterns without requiring a second diffusible inhibitor, providing a minimal and mechanically grounded mechanism for axis selection in regenerating tissues.

CT1368 — Phenotypical Characterization and Regional Morphometric Variation of the Invasive Indian Crow (Corvus splendens) in the Sultanate of Oman

SPEAKERS Qais Abdullah SULIAMAN ALRawahi (Asharqiyah university)
AUTHORS Adamu Abubakar (Asharqiyah university); Qais Abdullah SULIAMAN ALRawahi (Asharqiyah university)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.33 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

The Indian House Crow (Corvus splendens) is an invasive species of growing concern in Oman due to its rapid expansion and ecological impact. This study provides a detailed phenotypic and morphometric assessment of 367 adult crows collected from four governorates: Muscat, Dhofar, North Al Batinah, and South Al Sharqiyah. Plumage coloration was highly conserved, with primary, secondary, tail, alula, beak, and back feathers uniformly black. As noted in the manuscript, "the color of the primary, secondary, and tail feathers… was predominantly black (100%), with only one exception in the eye ring (99.7% black, 0.3% blue)." Quantitative measurements revealed significant sexual size dimorphism, with males exhibiting greater body weight, wingspan, tarsus length, and overall body dimensions ($P<0.001$). Regional variation was also evident, with Dhofar birds being the largest. No significant associations were found between sex or region and qualitative color traits. These findings provide essential baseline data for future genetic and management strategies.

References

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CT1486 — Replicator Dynamics as a Framework for Invasion in Microbial Communities

SPEAKERS Tomas Freire (IST)
AUTHORS Erida Gjini; Marina Garcia-Romero; Sten Madec; Tomas Freire (IST)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.46 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Determining whether a new species can invade an established microbial community is a central problem in ecology and evolution. We develop a replicator-dynamics framework that links invasion outcomes to microbiome composition through frequency-dependent interactions between species [MG20; GM23].

In this formulation, the initial growth of an invader has two components: interactions between the invader and resident species, and the system's intrinsic resistance to invasion generated by interactions between species already present. Both contributions arise from pairwise interaction terms and together determine whether invasion succeeds.

We illustrate this framework in two microbiome systems. First, we analyze experiments where antibiotic-resistant and wild-type *E. coli* strains co-invade mouse gut microbiota [FMG25]. Using this approach, we infer how community composition modulates the selection coefficient between the two invaders.

Second, we apply the framework to vaginal microbiome data from a large cohort by mapping Nugent scores (an indicator of vaginal health) to invader growth rates, allowing us to quantify community susceptibility to pathogenic invasion [FGG26]. The resulting model predicts vaginal health states with accuracy comparable to machine-learning approaches while providing a mechanistic ecological interpretation.

More broadly, this work illustrates how replicator dynamics can be used to study invasion processes in complex microbial communities.

References

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CT1434 — Self-Organization in Stochastic Multiscale Systems with Conflict Resolution Dynamics

SPEAKERS Richard Kollar (Comenius University Bratislava)
AUTHORS Katarina Bodova (Comenius University); Richard Kollar (Comenius University Bratislava)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

We investigate self-organization in stochastic dynamical systems using rigorous and numerical multiscale analysis. At the mesoscale, the model describes conflict resolution between two types of individuals in interconnected domains. Conflicts occur as a Poisson process governed by the mass-action law. Each conflict in a given domain triggers the transfer of one of the interacting individuals to a neighboring domain. This general mechanism arises in a variety of biological settings.

Our main questions are whether the system self-organizes in finite time and how the self-organization time scales with population and system size. We also examine microscopic variants of individual-level interactions underlying the mesoscopic model. We show that, while some microscopic variants give rise to absorbing Markov chains, others unexpectedly do not. Nevertheless, at the macroscale, all variants reduce to an absorbing Markov chain model, which guarantees self-organization in finite time. We derive upper bounds on the self-organization time and identify a non-Markovian microscopic variant that promotes crowd formation, thereby prolonging self-organization. Overall, our study provides a framework for applying absorbing Markov chain theory to self-organization, especially in systems driven by conflict resolution mechanisms.

CT1324 — Soil Biodiversity and Ecological Niches Under Climate Change: Modelling Insights from the BIOservicES Project

SPEAKERS Rajneesh Kumar (University of Sussex, United Kingdom)
AUTHORS Marianna Cerasuolo (University of Sussex); Rajneesh Kumar (University of Sussex, United Kingdom)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Soil organisms regulate essential ecological processes such as nutrient cycling and pest suppression [BV14], yet much about them is still unknown. At the global scale, soil degradation causes substantial economic losses each year [Sut+16]. Understanding how soil biota responds to land-use and climate change is therefore vital for ecosystem services and long-term soil sustainability.

Despite their importance, the habitats, environmental needs, and climate sensitivity of soil organisms remain poorly understood. This knowledge gap limits our ability to evaluate soil sustainability innovations, assign economic value to soil biodiversity services, and support evidence-based policy. This project addresses these challenges by linking soil biodiversity to ecosystem functioning in Europe.

We analyse European soil biodiversity patterns using ecological niche analysis, combining mathematical modelling, multivariate statistics, and species-distribution modelling with environmental gradients, land-use data, and microbial taxa records. Machine learning reveals environmental structure across land-use types and key gradients shaping soil communities.

Our analyses show clear environmental differences across European land-use categories and ecological strategies from specialists to generalists. This niche-based work forms the basis for a hybrid species-distribution model to predict how soil biodiversity may shift under future climate conditions.

References

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[Sut+16] Paul C Sutton et al. “The ecological economics of land degradation: Impacts on ecosystem service values”. In: *Ecological Economics* 129 (2016), pp. 182–192.

CT1571 — Standing Wave Formation in In-Vitro Ultrasound Cell Culture Systems: Multiphysics Modeling and Experimental Investigation

SPEAKERS Farshad Moradi Kashkooli (University of Waterloo)
AUTHORS Farshad Moradi Kashkooli (University of Waterloo)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 15.05 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Acoustic boundary conditions strongly influence pressure fields and biological responses in in-vitro ultrasound experiments, yet their mechanistic role remains poorly quantified. This study investigates the effects of standing waves on acoustic pressure and cell viability. A multiphysics finite-element model integrating pressure acoustics, solid mechanics, and electrostatics was developed to simulate wave propagation and represent the piezoelectric transducer as both transmitter and receiver. A 1 MHz transducer delivered pulsed ultrasound into a water-filled chamber with cells suspended in RPMI medium. The upper boundary was configured with either air or an acoustic absorber to compare reflective and absorptive interfaces. Simulated pressure fields and pulse-echo responses agreed with experimental measurements. The air interface produced strong reflections and standing waves with minimal heating, whereas the absorber reduced reflections but increased medium temperature. Simulations showed constructive and destructive pulse interference depending on pulse duration and duty cycle, producing localized pressure amplification. Standing waves impaired cell viability more than thermal effects at comparable acoustic pressure. Cells exposed to 1.8 W and 3.2 W showed similar viability reductions without an absorber, while pronounced apoptosis occurred at 6.15 W. These results highlight the importance of controlling reflections for mechanistically interpretable LIPUS studies.

CT1613 — Total progeny analysis for infectious clusters within epidemics, with applications to COVID-19 in Albania.

SPEAKERS Liam Critcher (University of Manchester)
AUTHORS Christopher Overton (University of Liverpool); Liam Critcher (University of Manchester); Lorenzo Pellis (The University of Manchester); Thomas House (University of Manchester)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 10.11 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

We consider the problem of inferring R_0 for an epidemic where contact tracing data are available, from which we observe the size of several infectious clusters within a large population. By fitting the observed cluster sizes to the total progeny of a branching process, we use the Dwass formula for total progeny to estimate both R_0 and the index of dispersion of the offspring distribution. We extend this model to allow for the first generation of the branching process to have a different distribution to subsequent generations, mirroring the scenario in which traced contacts change their behaviour. We also explore right-censored cluster sizes and under ascertainment of cases, both of which can have appreciable impact

on the resulting estimate of R_0. We provide results obtained by applying the method to FFX COVID-19 data from Albania which, in addition to synthetic data, are used to assess goodness of fit.

CT1465 — Treatment Scheduling Modulates Vascular Normalization, Interstitial Fluid Pressure, and Drug Delivery in Solid Tumors

SPEAKERS Defne Yilmaz (Dr.)
AUTHORS Defne Yilmaz (Dr.); Farshad Moradi Kashkooli (University of Waterloo)
SCHEDULE Friday, 17 July 2026; 09:30–09:50; 03.01 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Elevated interstitial fluid pressure (IFP) and abnormal tumor vasculature limit the delivery of therapeutic agents to solid tumors. Although anti-angiogenic therapy can transiently normalize tumor vasculature and improve transport, the impact of treatment scheduling on IFP dynamics and drug delivery remains poorly understood. In this study, we develop a spatiotemporal multiphysics model that explicitly couples vascular normalization with interstitial transport to quantify how treatment scheduling modulates tumor transport barriers. The framework enables systematic comparison of intermittent, metronomic, and hybrid dosing strategies. It is formulated as a system of coupled nonlinear partial differential equations describing tumor growth, angiogenesis, interstitial fluid flow, and drug transport. Simulation results reveal that treatment scheduling strongly regulates tumor IFP and drug distribution, even when the cumulative drug dose is fixed. All regimens induce vascular normalization and reduce average tumor IFP by 51–57%, consistent with reported in vivo observations, with the largest reduction under metronomic therapy. When combined with intermittent chemotherapy, this strategy produces the highest intratumoral drug accumulation (AUC=1.15) and the most uniform spatial distribution. These results highlight the strong dependence of drug transport on dosing frequency and administration timing, and provide a quantitative framework for optimizing combination therapy schedules.

CT1437 — A mathematical model for a therapeutic approach for atherosclerosis

SPEAKERS Nader El Khatib (Lebanese American University)
AUTHORS Ghada Abi Younes (Lebanese American University); Nader El Khatib (Lebanese American University); Wissam El Hajj (Université de Rennes)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.02 - HS
SESSION Contributed Talks
TRACK Miscellaneous

Abstract

We consider a previously developed qualitative model for the inflammatory response in atherosclerosis and extend it to account for time-dependent anti-oxidant and anti-inflammatory therapies. The model consists of a five-dimensional system of ordinary differential equations tracking the concentrations of LDL, free radicals, oxidized LDL, macrophages and cytokines. Drug actions are incorporated through time-decaying efficacy factors that reduce the oxidation rate of LDL and the cytokine-mediated amplification of inflammation. We characterize the existence and uniqueness of an inflammatory equilibrium, and derive explicit conditions for its local asymptotic stability in terms of biologically interpretable parameters. We then investigate numerically several therapeutic scenarios, including no therapy, mono-therapies and a combination therapy acting simultaneously on oxidative stress and cytokine amplification. Then we perform numerical simulations for the system. Time courses of oxidized LDL, macrophages and cytokines, as well as numerical bifurcation diagrams of the steady-state cytokine level with respect to drug intensities and to the cytokine production rate, are presented and interpreted. Our results suggest that the combination therapy yields a pronounced synergistic reduction of both oxidized LDL and inflammatory

burden, and can move the system from a high-inflammatory regime to a low-inflammatory one in agreement with the qualitative stability analysis.

References

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CT1262 — Can we hear the shape of a neural network?"

SPEAKERS Giovanni Naldi (Università degli studi di Milano, Department of Environmental Science and Policy, DESIRE Center)
AUTHORS Giacomo Aletti (Università degli studi di Milano, Department of Environmental Science and Policy, DESIRE Center); Giovanni Naldi (Università degli studi di Milano, Department of Environmental Science and Policy, DESIRE Center); Shyam Diwakar (Amrita University, Amrita Mind Brain Center); Thierry Ralph Nieus (Università degli studi di Milano, Department of Environmental Science and Policy, DESIRE Center)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 11.01 - HS
SESSION Contributed Talks
TRACK Neuroscience and Neural Systems

Abstract

Understanding how structural synaptic connectivity shapes neuronal network function remains a fundamental challenge in neuroscience. While optical imaging now allows the simultaneous recording of thousands of neurons, most studies rely on calcium signals, which lack the temporal resolution to capture fast spiking and synaptic dynamics [Pal+17]. Recent advances in Genetically Encoded Voltage Indicators (GEVIs) offer a promising alternative for recording these events across large populations. In this work, we present a methodology to infer network connectivity directly from voltage traces [Nie+22; ABN22]. Our approach first identifies putative synaptic events and then employs Multi-Class Logistic Regression with L_1 regularization (MCLRL) to map these events to spiking activity while enforcing network sparseness. Benchmarked against in silico simulations, the MCLRL framework demonstrated high precision, as evidenced by the Matthews Correlation Coefficient (MCC). Notably, the method accurately reconstructed connectivity even when sampling only subgroups of neurons and proved remarkably robust to noise (MCC > 0.95 with 95% noise). Finally, we propose a procedure to optimize the regularization term, facilitating the application of MCLRL to experimental data. This research was conducted in collaboration with G. Aletti, T. Nieus, and S. Diwakar (Amita University).

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CT1575 — Connecting Vertex Based and Centroid Based Epithelial Monolayer Models

SPEAKERS Patrick Grant (University of Melbourne)
AUTHORS James Osborne (University of Melbourne); Matt Faria (University of Melbourne); Patrick Grant (University of Melbourne); Ryan Murphy (Adelaide University); Stuart Johnston

SCHEDULE Friday, 17 July 2026; 09:50–10:10; 03.01 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

In this research, we are investigating the relationship between a two-dimensional, quadrilateral, vertex model based on the work of (Brown et al, 2025) and a one-dimensional cell centroid model. The aim being to derive a continuum limit PDE approximation for the behaviour of epithelial monolayers. The movement of the vertices in the two-dimensional model is determined by the minimisation of the cell energy where each cell wants to maintain a target area and perimeter, determined from the cell age. Several mechanisms are introduced to allow stability with realistic levels of cell proliferation. In order to calculate motion in the centroid model forces are computed by first reconstructing a two-dimensional cell structure (from the cell centroids) and the forces calculated on each reconstructed vertex using a vertex dynamics model. These forces are then acted on the cell centroids which allow the monolayer to be represented as a single chain of centroids aiding calculation of a continuum representation. We compare the behaviour of these two models and investigate the parameters that can be used to connect them.

References

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CT207 — Coupled plasticity axes give rise to coordinated cellular behaviour and differentially aggressive attractor states in breast cancer

SPEAKERS Ritesh Kumar Meena (IISc)
AUTHORS Ritesh Kumar Meena (IISc); Vicky Fan (Texas A&M); Soundharya Ramu (IISc); Jason T. George (Texas A&M); Mohit Kumar Jolly (IISc)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 11.02 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Phenotypic plasticity underlies cancer progression and metastasis [Jeh+22], yet how distinct regulatory axes interact to shape tumour progression and patient outcomes remains unresolved. We investigated major axes of plasticity in ER-positive breast cancer, including metabolic reprogramming, epithelial-to-mesenchymal plasticity, and drug-resistance using ODE-based gene regulatory network simulations, integrative bulk and single-cell transcriptomic analyses, and patient survival analyses. We show that the network exhibits multistability, organising into two mutually inhibiting modules of regulatory programs that define distinct attractor states. One module (favouring high glycolysis, mesenchymal/hybrid and tamoxifen-resistant phenotype) was found to be associated with aggressive disease progression and worse survival. On the other hand, the opposing module (favouring high oxidative phosphorylation, epithelial and tamoxifen-sensitive phenotype) correlated with better outcomes. Further, perturbations along one axis induced coordinated responses along other axes, indicating strong inter-axis coupling [Mee+25]. Our findings establish phenotypic plasticity in cancer as an emergent property of coupled dynamical systems, suggesting that targeting regulatory interactions between axes can constrain transitions toward aggressive states [Ker+26].

References

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CT1287 — Dynamical systems analysis of a model coupling hematopoiesis, hematological malignancy and inflammation.

SPEAKERS Morten Andersen (Roskilde University)
AUTHORS Morten Andersen (Roskilde University); Tobias Idor Boklund (Roskilde University); Thomas Stiehl (RWTH Aachen University); Johnny T. Ottesen (Roskilde University)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 02.21 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Myeloproliferative neoplasms (MPN) are a group of hematological malignancies characterized by an overproduction of myeloid blood cells. A recent mathematical model ([Bok+24]) coupling hematopoiesis, inflammation, MPN progression and treatment inhibiting inflammation is here analyzed from a dynamical systems perspective. The model includes time varying populations of stem cells, progenitor cells, mature blood cells and their mutated counterparts, cellular debris, and inflammatory feedback resulting in an eight dimensional, nonlinear ode-model. A compact, positively invariant subset of the phase space is found, guaranteeing trajectories exist for all forward time (no blow up, even in the case of malignant cells). The problem of identifying steady states is greatly simplified, by derivation of a low order polynomial (at most 4th order) in a single variable, providing analytical upper bounds on healthy, malignant and coexistence steady states and ensure a fast algorithm to describe all steady states for a given set of parameter values. Critical parameter values related to treatment of various type acting on stem cell, progenitor cell and inflammatory feedback is then varied, resulting in more than 10^6 cases to be analyzed. 13 different flow patterns are observed and categorized, and the effects of perturbing several parameters at a time are summarized in bifurcation diagrams providing a theoretical test bed for potential treatment, including combination of several drugs.

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CT328 — Efficient Integration of Binary Qualitative Data into ODE Model Estimation

SPEAKERS Domagoj Doresic (Bonn Center for Mathematical Life Sciences, University of Bonn)
AUTHORS Daniel Weindl (Bonn Center for Mathematical Life Sciences, University of Bonn); Domagoj Doresic (Bonn Center for Mathematical Life Sciences, University of Bonn); Jan Hasenauer (Bonn Center for Mathematical Life Sciences, University of Bonn)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 01.18 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Parameter estimation for ODE models of biological systems is commonly based on continuous or semi-quantitative measurements. Many relevant observations are however inherently binary — such as gene essentiality classifications from CRISPR knockout screens. Treating such data as continuous introduces statistical misspecification, biasing parameter estimates and undermining uncertainty quantification.

We propose a hierarchical gradient-based framework for integrating binary observables into parameter estimation of mechanistic ODE models. The estimation problem is decomposed into an outer optimisation over mechanistic parameters and an inner optimisation over observable-level parameters. Via a KKT-based envelope argument, we show that exact outer gradients are obtained without tracking the dependence of inner on outer parameters — a result that holds even under box constraints. The fully likelihood-based formulation enables standard uncertainty quantification of mechanistic parameters.

We demonstrate the method on joint inference from drug-response viability measurements and binary gene-essentiality data across 233 cancer cell lines using a pan-cancer ERK-RAS-AKT signalling model, and investigate the effect of appropriate binary likelihood specification on parameter identifiability and predictive performance. The method is implemented in pyPESTO.

CT1472 — Extended Kalman Filter for Real-Time Estimation of Hidden Bone Marrow Dynamics from Peripheral Blood Measurements

SPEAKERS Haralampos Hatzikirou (Khalifa University)
AUTHORS Haralampos Hatzikirou (Khalifa University); Symeon Savvopoulos (Khalifa University); YUSUF JAMILU UMAR (Khalifa University)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.11 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Background: A fundamental challenge in clinical hematology is the inability to monitor bone marrow dynamics in real time without invasive procedures.

Objective: We present an Extended Kalman Filter (EKF) framework that reconstructs unobservable bone marrow cell populations (stem, progenitor, differentiated) from noisy measurements of peripheral blood inflammation.

Methods: The hematopoietic system is modeled as a five state nonlinear dynamical system with inflammatory feedback. We establish observability through Lie derivative analysis, proving all internal states can be reconstructed from peripheral inflammation when feedback pathways are active ($g_3 \neq 0$ or $g_2 \neq 0$). The EKF uses analytically derived Jacobians and is tested under varying process noise (0.1 to 3%), measurement noise (5 to 30%), and initial uncertainty (5 to 50%) for healthy and pathological (MDS/AML) conditions.

Results: The EKF demonstrated robust convergence across all scenarios. Higher process noise introduced oscillatory errors but maintained tracking accuracy, while increased measurement noise produced smoother estimates with slower convergence. The filter showed remarkable insensitivity to initial errors, converging reliably even with 50% error. Observability analysis revealed active inflammatory feedback ($g_3 \neq 0$) is essential for reconstructing marrow inflammation from peripheral measurements.

Conclusion: The EKF provides accurate estimation of hidden hematopoietic states from peripheral blood measurements.

CT1245 — Filtered Schemes for a Continuum Model of DNA Transcription

SPEAKERS Lisa Davis (Montana State University)
AUTHORS Faranak Courtney-Pahlevani (Penn State - Abington); Kevin Courtney (Penn State); Lisa Davis (Montana State University)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.27 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The focus of the presentation is the development, numerical simulation and parameter analysis of a model of the transcription of ribosomal RNA in highly transcribed genes. The well-known nonlinear Lighthill-Whitham-Richards (LWR) equation is used to model the transcription process. The numerical treatment for model simulations includes introducing a low complexity time accurate method by adding a simple linear time filter to both explicit and implicit finite difference schemes. Time filters were first introduced in 1966 and subsequently analyzed and expanded by many scholars. The work presented here is motivated by a time filter proposed by Guzel and Layton in 2018. The new filtered methods require a minimal modification of the code for the original unfiltered schemes, avoiding extra computational expense. Convergence results demonstrate that the unfiltered semi-implicit scheme outperforms the fully explicit scheme, and combining the filtering scheme with each of the fully explicit and semi-implicit schemes further increases computational accuracy. Finally biologically relevant transcription data can be extracted from the model simulations. One can calculate transcription time results for individual RNAPs transcribing the DNA strand, and average transcription time results over a specified period of time can be calculated. Simulation results are shown for two benchmark regimes of the LWR model, a shock wave example and a rarefaction wave case.

CT1456 — Habitat fragmentation promotes spatial scale separation under resource competition

SPEAKERS Malbor Asllani (Florida State University)
AUTHORS James Austin Orgeron; Malbor Asllani (Florida State University)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.46 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Habitat fragmentation, often driven by human activities, alters ecological landscapes by disrupting connectivity and reshaping species interactions. In such fragmented environments, habitats can be modeled as networks, where individuals disperse across interconnected patches. We consider an intraspecific competition model, where individuals compete for space while dispersing according to a nonlinear random walk [Asl+18], capturing the heterogeneity of the network. The interplay between asymmetric competition, dispersal dynamics, and spatial heterogeneity leads to nonuniform species distribution: individuals with stronger competitive traits accumulate in central (hub) habitat patches [BA99], while those with weaker traits are displaced toward the periphery. We provide analytical insights into this mechanism, supported by numerical simulations, demonstrating how competition and spatial structure jointly influence species segregation. In the large-network limit, this effect becomes extreme, with dominant individuals disappearing from peripheral patches and subordinate ones from central regions, establishing spatial segregation. This pattern may act as a potential precursor to both speciation and diversity, as physical separation can reinforce divergence within the population over time and potentially support coexistence at the landscape scale.

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CT1544 — Hexagons of Health: Robust Liver Detoxification Emerges from Lobular Geometry

SPEAKERS Daniel Sanchez-Taltavull (University of Bern)
AUTHORS Daniel Sanchez-Taltavull (University of Bern); Pilar Guerrero (Universidad Carlos III de Madrid); Rubén Perez-Carrasco (Imperial College London)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 05.12 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

The liver contributes to maintaining metabolic homeostasis by clearing toxins from the bloodstream. Blood enters through portal veins, flows through the tissue where hepatocytes remove toxins, and exits through central veins. This process occurs within a highly organized microarchitecture in which portal and central veins form repeating functional units called lobules. In cross-section, lobules display a polygonal arrangement with portal veins at the vertices and a central vein at the center. Although lobules are commonly depicted as hexagonal, the precise functional advantage of this geometry remains unclear.

We investigate how lobule geometry influences detoxification using a reaction-diffusion framework in which portal veins act as sources of toxin-rich blood and the central vein acts as a sink. Toxins spread through the tissue while hepatocytes remove toxins locally. Within this framework we explore several measures of detoxification efficiency across different spatial organizations of the veins.

To capture more realistic conditions, we develop a spatial CompuCell3D model in which hepatocytes lose their detoxification capacity when exposed to damage. Under these nonlinear dynamics, hexagonal lobule organization proves more robust than other tessellations in minimizing toxin levels reaching the central veins, revealing a potential functional advantage of the hexagonal architecture of liver lobules.

CT1327 — Inferring Cell-Cell Interaction Dynamics from Cell Trajectory Data Using Deep Attention Networks, and the Mechanisms Underlying T Cell Behavioural Heterogeneity in the Response to Cancer

SPEAKERS James Boyle (University of Oxford)
AUTHORS James Boyle (University of Oxford); Veronika Pfannenstill (University of Oxford); Jacky K L Ko (University of Oxford); Marco Fritzsche (University of Oxford); Helen Byrne (University of Oxford); Ruth Baker (University of Oxford)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cell movement is an important part of many biological processes, such as collective cell migration and the immune response to cancer. In many systems interactions between nearby cells are a key driver of cell movement, yet it can be difficult to infer and express the rules governing cell-cell interactions in a manner that is biologically interpretable. Here we present a model, based on the theory of deep attention networks, that learns how cell-cell interactions affect cell movement from cell trajectory data. We develop a suite of tools that leverage the structure of this model to present the learned interaction dynamics in an interpretable manner. Our work extends previous applications of deep attention networks to cell

movement, moving beyond only inferring how strongly cells interact in monocultures, to also inferring the effect of these interactions on cell movement in both monotype and multi-type cell movement systems.

We use our model to understand how cytotoxic T cells and tumour cells interact in an in vitro co-culture, and use these results to build an agent-based model of the T cell response to cancer. Recent experimental studies have observed significant variability in the killing capabilities of cytotoxic T cells – i.e. some T cells kill many more cancer cells than others. We use our model to investigate what mechanisms might give rise to this heterogeneity, and ask whether it can be explained by a model in which all T cells follow the same behavioural rules.

CT338 — Interferon-mediated antiviral protection limits Influenza spread: A spatial agent-based model

SPEAKERS Rodolfo Blanco-Rodriguez (University of Idaho)
AUTHORS Esteban Hernandez-Vargas (University of Idaho); Rodolfo Blanco-Rodriguez (University of Idaho); Tanya A. Miura (University of Idaho)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 01.15 - HS
SESSION Contributed Talks
TRACK Immunobiology & Infection

Abstract

Predicting disease progression hinges on understanding how innate immune responses regulate viral spread at the tissue level. Type I interferon (IFN) induces local antiviral protection in epithelial cells, yet the combined effects of virion diffusion, IFN transport, and cellular heterogeneity remain difficult to capture in spatial models. We present a spatial agent-based model of influenza A virus infection in mouse lung epithelium that couples virion propagation with IFN transport. Cells transition between susceptible, refractory, infected, and IFN-producing infected states according to probabilistic rules governed by local viral load and IFN concentration. Viral replication and IFN secretion occur at the single-cell level, while extracellular virions and IFN diffuse through a heterogeneous tissue microenvironment characterized by irregular cell spacing and local density variations. Simulations reveal a competition between virion spread and IFN-mediated antiviral protection. IFN diffusion can halt infection by forming barriers of refractory cells when IFN diffusion exceeds virion diffusion. However, containment is probabilistic: under identical conditions some simulations produce effective barriers while others allow continued virion spread. The likelihood of containment depends strongly on the ratio of IFN to virion diffusion. This framework provides a stochastic spatial platform to study how innate immune responses regulate viral spread in heterogeneous tissues.

CT1304 — Macaque social network structure beyond rank assortativity

SPEAKERS Diogo Pires (Center for Social Data Science, University of Copenhagen)
AUTHORS Lauren J Brent (University of Exeter); Sune Lehmann (DTU Compute, Technical University of Denmark and Center for Social Data Science, University of Copenhagen); Josefine Bohr Brask (Center for Social Data Science and Behavioural Ecology Group, University of Copenhagen); Diogo Pires (Center for Social Data Science, University of Copenhagen)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 11.32 - SR
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Social structure influences ecological dynamics, fitness, and evolution in animal populations. Network science provides tools to link individual interactions to emergent social organisation, which are increasingly used in behavioural ecology [KLJ09; BEC21]. In animal societies, this approach explains how individual and relational characteristics influence the formation, stability, and evolution of social relationships [Bra+25; BSW25]. The aim of this study is to examine how dominance rank, kinship, and demography

influence social structure in rhesus macaques, focusing on the unusual social connections between the highest-and lowest-ranking individuals. We analyse the social networks of seven groups of free ranging rhesus macaques from Cayo Santiago, PR [Tes+24]. Social networks in this species are strongly assortative by dominance rank. However, we have found that systematic deviations emerge across groups and years: lowest-ranking individuals frequently connect with the highest-ranking ones and are often detected within their social communities. These associations suggest the presence of latent or historical dynamics not explained by the statistically inferred effects of rank assortativity, kinship, or demographic structure. We hypothesise that such ties reflect strategic behaviour by low-ranking individuals seeking to associate with high-ranking individuals without directly challenging the dominance hierarchy. These may ultimately contribute to the long-term success of low-ranking matrilines.

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CT1430 — Macroscopic Models for Hard Anisotropic Particles

SPEAKERS Carmela Moschella (University of Oxford)
AUTHORS Carmela Moschella (University of Oxford); Maria Bruna (University of Oxford)
SCHEDULE Friday, 17 July 2026; 09:50-10:10; 15.34 - SR
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Volume-exclusion interactions play a crucial role in the self-organisation observed in many biological systems. In particular, they are fundamental to explain the spontaneous emergence of nematic order in populations of anisotropic particles, as observed, for example, in dense suspensions of myxobacteria [BI15]. More generally, they also play an important role in cell migration, where particle shape and crowding strongly affect the behaviour of the migrating population [DB15]. In this talk, we consider a stochastic model of hard-core anisotropic particles in two dimensions, represented as non-overlapping rectangular particles undergoing Brownian motion with drift. Starting from this particle model, we use matched asymptotic expansions in the dilute regime to derive a kinetic equation for the one-particle probability density in position and orientation space. The resulting kinetic model explicitly shows how excluded-volume effects are encoded in the interaction kernel, which depends on particle aspect ratio. In the limit of vanishing width, we recover the corresponding description for non-overlapping needles [BCS23]. Finally, we discuss possible extensions to ellipsoidal or capsule-shaped particles, as well as to anisotropic particles in three dimensions.

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CT1325 — Mathematical modelling of actin polymerisation in biological condensates

SPEAKERS Fynn Wolf (University of Bergen, Norway)
AUTHORS Fynn Wolf (University of Bergen, Norway); Susanna Röblitz (University of Bergen, Norway); Thomas Böddeker (Charité – Universitätsmedizin Berlin, Germany)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 11.33 - SR
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Biological condensates are membraneless organelles within the cell or the nucleus which perform an array of different tasks and typically consist of DNA/RNA and protein. Actin is a protein that exists in most eukaryotic cells and transitions between monomeric and filamentous states. In its filamentous state, actin forms networks, which perform vital tasks inside the cell. Recent research has shown interactions between biological condensates and cytoskeletal filaments, such as actin. The focus of these works was on morphological changes of condensates [Gra+23], dynamics of condensates with cytoskeletal structures [Nad+10] and polymerisation kinetics inside condensates [Bar+25; Sun+25]. However, a full mathematical description of the cooperativity between condensates and actin polymerization is still missing. In this work we use a stochastic simulation algorithm to capture the polymerization kinetics of actin in a multicompartment system of condensates and dilute phase containing monomeric and filamentous actin. We show how condensates regulate the uptake of monomeric actin and how the enclosed environment of the condensate affects the polymerisation of actin. We make predictions about the number and length of polymerized actin filaments inside the condensate. This study, together with recent experimental findings, will help us understand how biological condensates are involved in the creation and maintenance of actin networks inside the cell.

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CT1415 — Multiple observations in blood cancer dynamics of AML, CML, and MPNs are comprehensively explained by the unifying Multiple Clone Cancitis model.

SPEAKERS Johnny Ottesen (Roskilde University)
AUTHORS Johnny Ottesen (Roskilde University); Katrine Bangsgaard (Novonordisk); Morten Andersen (Roskilde University)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.06 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

We present the Multiple Clone Cancitis (MCC) model describing the blood cancers AML, CML, and MPNs despite its differences [Ban+20]. The MCC model is based on biological mechanisms, basic cell descriptors such as proliferation rates, and apoptosis rates connecting stem cell dynamics with mature blood cells and immune mediated feedback including an explicitly cytotoxic T-cell mediated death of malignant stem cells. The unifying and flexible MCC model approach allows for arbitrary numbers of mutated stem cell clones with perturbed properties. The stem cell niche mediate suppression of the wild type clones due to resources restrictions etc. and the microenvironment may cause the cytotoxic T-cell to have different death rates in killing malignant stem cells and stem cell like progenitors.

The unifying MCC model comprehensively explains:

(1) Patient data, (2) Frequently observed oscillations in cell count of time scale 2–3 months for hydroxyurea treated CML patients, (3) Less frequently observed oscillations in cell count of time scale 1 month for hydroxyurea treated MPN patients, (4) Consequences of CHIP leading to good, fair, poor, or vital responders to treatment, (5) Elimination, equilibrium, and escape phases related to immunoediting, (6) Developed of immune resistance to treatment, (7) The evolution of clones over the life span of a human.

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CT1541 — Phase-Field Model of Red Blood Cell Morphology in Sickle-Cell Anaemia

SPEAKERS João Maria Loureiro de Oliveira Costa Santos (University of Coimbra)
AUTHORS Aurora Hernández-Machado (University of Barcelona); Giuseppe Negro (The University of Edinburgh); João Maria Loureiro de Oliveira Costa Santos (University of Coimbra); Rui Travasso (University of Coimbra); Tyler Shendruk (The University of Edinburgh)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.05 - HS
SESSION Contributed Talks
TRACK Multiscale and Multiphysics Modelling

Abstract

Sickle cell anaemia is a genetic and molecular disease that affects the structure and dynamics of red blood cells (RBCs), leading to a variety of complications in blood circulation. A single amino-acid mutation in the haemoglobin molecule leads to the polymerization of long, rigid fibres that deform the cell from the inside, affecting its morphology and the blood’s rheological properties [Eat20]. The scope of this work is to develop a computational model with a phase-field approach for the RBC membrane deformation time evolution in sickle cell anaemia that considers its Canham-Helfrish bending rigidity [LHP19; Lop+23]. Haemoglobin fibres in the cytoplasm are modelled as a nematic liquid crystal and introduce a phase-field model to simulate how these fibres interact with the cell membrane. With this model we explore systematically the stationary morphology of the RBC as a function of the fibre anchoring

angle and characterize the range of angles and nematic liquid crystal parameters [Neg+25; RY21] that lead to the sickle shape. Moreover, analysing the dynamics of RBCs acquiring the sickle morphology can give us important insights on how the RBCs are deformed as they transverse the narrower capillaries in the organism.

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CT323 — Population modelling under limited data: using expert knowledge in model calibration

SPEAKERS Sarah Vollert (Umea University)
AUTHORS Sarah Vollert (Umea University)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 12.01 - HS
SESSION Contributed Talks
TRACK Population Dynamics, Ecology & Evolution

Abstract

Many modelled biological systems are data-limited, due to the costs, time constraints, invasiveness or untrustworthiness of data collection. However, ecologists and biologists often possess valuable knowledge about how systems should behave, derived from theory, experiments, or expert understanding. We develop a new statistical framework for incorporating expert knowledge directly into mechanistic models via calibration. We calibrate population models with expert-elicited long-term population sizes, theoretical expectations of a stable coexisting equilibrium, and observed population responses to perturbations. Our results reveal that combining data with expert knowledge in calibration leads to enhanced predictions that better align with the underlying system, insightful inferences from the additional information, and an improved capacity to inform complex decisions.

CT1355 — Robust regimens predicted using a virtual murine cohort of bladder cancer with experimental validation

SPEAKERS Hannah Anderson (Moffitt Cancer Center)
AUTHORS Hannah Anderson (Moffitt Cancer Center); Jamie Blauvelt (Moffitt Cancer Center); Catherine Liu (Moffitt Cancer Center); Shari Pilon-Thomas (Moffitt Cancer Center); Katarzyna Rejniak (Moffitt Cancer Center)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 02.01 - HS
SESSION Contributed Talks
TRACK Mathematical Oncology

Abstract

Mice orthotopically implanted with bladder cancer showed improved responses on average to intravesical (inV) T cell therapy (OT1) when preconditioned with inV gemcitabine; however, responses varied among individual mice. We developed a virtual cohort for this combination therapy to expand of the murine population while capturing response variability.

Using parameters identifiable with ultrasound data, we fit the model to the 55 experimental mice to create 55 subject-specific calibrations. Then, we performed a treatment sweep of possible regimens on each calibration, aiming to minimize a cost function that balances the final tumor size with stable tumor growth over the treatment period. 13 regimens were optimal for at least one subject-specific calibration representing an experimental mouse. These 13 were tested on the virtual cohort and evaluated for robustness by determining which regimen produced the smallest final tumor size and the longest overall survival for the most virtual mice. Results led to the experimental testing of 3 regimens, which showed improved response compared to the original regimen. An additional digital twin cohort was included to stratify mice into the 3 regimens in real time based on their initial response from ultrasounds.

Better murine administration of these therapies can improve response in clinical trials for non-muscle-invasive bladder cancer, where patient-derived tumor-infiltrating lymphocytes would be used instead of murine OT1 cells.

CT1609 — Some measures for epidemic decision-making under limited resources in a stochastic SVIR model

SPEAKERS María Gamboa Pérez (Complutense University of Madrid)
AUTHORS María Gamboa Pérez (Complutense University of Madrid)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 10.11 - HS
SESSION Contributed Talks
TRACK Mathematical Epidemiology

Abstract

This contribution studies the dynamics of an infectious disease outbreak in a closed homogeneously mixed population using a stochastic SVIR model with a pre-epidemic vaccination program and imperfect vaccine protection [GLL24]. The model is formulated as an absorbing continuous-time Markov chain with susceptible, vaccinated, infected, and recovered compartments. We focus on two key epidemiological quantities: the peak number of infections and the final epidemic size, defined as the total number of infections among both susceptible and vaccinated individuals [BR15]. Recursive schemes are derived to compute the corresponding probability mass functions, enabling the quantification of outbreak risk and variability through relevant summary measures. This approach is particularly well-suited for populations of moderate size, such as hospitals or nursing homes, where stochastic effects play a crucial role. Numerical results illustrate how imperfect vaccination shapes epidemic intensity and overall impact. Finally, some applications are presented to support decision-making in epidemic management under resource constraints [GLT23] and to inform the assessment of vaccine effectiveness [OBD+85]. This work is based on a manuscript currently under review.

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CT1497 — Spectral Fragility in Biological Spatial Networks under Geometric Vertex Noise

SPEAKERS Ben Cardoen (University of Birmingham)
AUTHORS Ben Cardoen (University of Birmingham); Fabian Spill (University of Birmingham)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 10.01 - HS
SESSION Contributed Talks
TRACK Cellular and Developmental Biology

Abstract

Spatial networks reconstructed from microscopy such as mitochondrial, neuronal, and vascular networks are frequently modelled as embedded graphs whose edge weights depend on inter-vertex distance. Uncertainty in vertex localisation arising from segmentation, registration, or temporal sampling introduces geometric perturbations that propagate nonlinearly into graph Laplacians. These perturbations induce structured spectral noise that is not captured by independent edge-noise models. We study the spectral effects of tempered heavy-tailed vertex displacement in spatial graphs. We derive bounds showing that spectral fragility is governed by small geometric configurations: extremal witness motifs saturate Weyl-type eigenvalue inequalities and provide conservative envelopes on eigenvalue perturbations. This allows global sensitivity to be approximated through vertex-disjoint motif tilings. To assess when spectral comparisons remain meaningful under geometric noise, we introduce stochastic spectral separation indices (S3I) that quantify whether observed spectral differences exceed noise-induced variability. Experiments on spatial graph models and imaging-derived networks reveal geometry-dependent noise floors across organisational regimes ranging from sparse tubular mitochondrial networks to dense reticulated structures. These results identify when spectral differences reflect genuine biological reorganisation rather than uncertainty in vertex localisation.

CT242 — Structural identifiability of stochastic processes: from single-particle trajectories to total particle density data

SPEAKERS Arianna Ceccarelli (University of Oxford)
AUTHORS Alexander Browning (University of Oxford); Arianna Ceccarelli (University of Oxford); Ruth Baker (University of Oxford)
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 15.21 - SZ
SESSION Contributed Talks
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The increasing availability of experimental data has motivated interest in calibrating stochastic models, raising fundamental questions about parameter identifiability. Structural identifiability analysis determines whether parameters can be uniquely inferred from idealised, noise-free data; however, existing methods are not suited to partially-observed stochastic processes with hidden internal states.

We develop a framework to analyse structural identifiability in stochastic models with unobserved internal dynamics and investigate how identifiability depends on the type of data available, namely single-particle trajectories or total particle densities. For density measurements, we derive a partial differential equation describing the population-level dynamics and apply the differential algebra approach. We show that the initial condition plays a crucial role and cannot be fully characterised using the differential algebra techniques alone. To address this, we introduce a method to determine identifiable parameter combinations arising from the initial condition.

We apply the framework to a model in which individuals alternate between constant-velocity motion and pauses governed by a hidden state, and we show that parameters are structurally identifiable from trajectory data but only locally identifiable from density data.

CT1488 — Structural Identifiability of Stoichiometric Matrix in Mass-Action Biochemical Reaction Networks

SPEAKERS Shivam Yadav (Ghent University, Ghent University Global Campus)
AUTHORS Manvel Gasparyan (Ghent University Global Campus); Shivam Yadav (Ghent University, Ghent University Global Campus); Shodhan Rao (Ghent University Global Campus (GUGC))
SCHEDULE Friday, 17 July 2026; 09:50–10:10; 11.03 - HS
SESSION Contributed Talks
TRACK Systems Biology and Biochemical Networks

Abstract

Structural identifiability in chemical reaction networks is often studied for kinetic parameters, but much less is known about identifiability of the stoichiometric structure itself. We study this question for non-autocatalytic mass-action biochemical reaction networks with fixed rate constants. We ask when the stoichiometric matrix can be uniquely recovered (identifiable) from the induced dynamics. Our results show that non-identifiability is not arbitrary. It is tied to specific structural features of the network. When several reactions share a common reactant complex, the stoichiometric structure across those reactions need not be uniquely recoverable from the dynamics, even under idealised observations. Yet this failure is highly structured. It is confined to the shared-reactant part of the network and shaped by precise relations among the associated rate constants. This leads to a clean separation between identifiable and non-identifiable regimes, and gives a sharper understanding of what mass-action dynamics can and cannot reveal about biochemical network structure.

Chapter 5

Poster Presentations

PP226 — How do bee's smell? The multi-physics of honeybee olfaction

SPEAKERS Ryan Palmer (University of Bristol, UK)

AUTHORS Alexander Borg (Rothamsted Research); Beth Harris (University of Leeds, UK); Daniel Robert (University of Bristol, UK); David Withall (Rothamsted Research); Frank Smith (UCL); Fraser Woodburn (University of Bristol); Jozsef Vuts (Rothamsted Research); Liam O'Reilly (University of Bristol); Robert Harniman (University of Bristol); Ryan Palmer (University of Bristol, UK)

SCHEDULE Tuesday, 14 July 2026; 18:30–18:50; ReSoWi Building

SESSION Poster Presentations

BOARD F1

TRACK Multiscale and Multiphysics Modelling

Abstract

The sense of olfaction (smell) in honeybees occurs through sensory receptors along their antennae. We study one type of sensor called a placode, which densely covers each antenna in a regular formation. Sitting close the antennae's surface, each placode is covered by hundreds of innervated pores that capture olfactory particles. We seek to understand how the morphology and configuration of placodes along the antenna affect the flow of air and how this fluid-structure interaction impacts a bee's ability to smell.

The precise role of the placode's morphology in olfaction is unknown. Two candidate shapes have been identified, which we shall examine. Each is of distinct morphology presenting as either a pit (with an initial sharp ring and smooth inner) or a mound (with a small divot on top).

We model the fluid-structure interaction of the airflow and placodes considering their morphology and configuration to assess their role in volatile capture. Due to the depth and relative length of the placodes, the so-called condensed flow equations apply. Initially, we consider two and three-dimensional configurations for a single placode to investigate the local influences of its morphology on the fluid flow. We later extend this scenario to that of a three-dimensional periodic case, whereby the streamwise and crosswise interactions of many placodes is considered. Finally, we compare these results to FEM models and assess the potential role of electrostatics in this olfactory process.

PP136 — ABM–Deep Gaussian Process Virtual Patient Population Reveal Interferon-Dependent Efficacy of Sirolimus in SARS-CoV-2 Infection

SPEAKERS Henrique de Assis Lopes Ribeiro (Univeristy of Florida)
AUTHORS Sandra Tsiorintsoa (Univeristy of Florida); Reinhard Laubenbacher (Univeristy of Florida); Borna Mehrad (Univeristy of Florida); Henrique de Assis Lopes Ribeiro (Univeristy of Florida)
SCHEDULE Tuesday, 14 July 2026; 19:30–19:50; ReSoWi Building
SESSION Poster Presentations
BOARD F2
TRACK Immunobiology & Infection

Abstract

Early SARS-CoV-2 infection is governed by nonlinear and often antagonistic interactions among immune cells, cytokines, and intracellular signaling pathways. We developed a mechanistic agent-based model (ABM) of early lung infection integrating pneumocytes, macrophages, natural killer (NK) cells, type-I interferon (IFN) signaling, and the mTORC1 inhibitor sirolimus. To enable large-scale analysis, we trained a Deep Gaussian Process (DGP) surrogate on ABM simulations and generated a virtual patient population spanning physiologically plausible parameter ranges. Using Morris and functional ANOVA sensitivity analyses, we identified regime-dependent drivers of infection outcomes at 48 hours post-infection. When IFN-mediated viral inhibition spans a wide range, viral replication kinetics dominate system behavior, and sirolimus strongly reduces viral load and inflammation despite immunosuppressive effects. In contrast, when IFN inhibition is highly effective, IFN secretion rate becomes the primary determinant of viral load, and sirolimus has diminished impact. The model further predicts context-dependent roles for macrophage polarization, reconciling conflicting experimental findings. These results highlight how immune–viral feedback structure determines therapeutic efficacy and underscores the value of surrogate-assisted ABM sensitivity analysis for interpreting heterogeneous treatment responses.

PP1632 — A combined mathematical and experimental approach to understanding tristetraprolin regulation in inflammation

SPEAKERS Teri Parsonage (Department of Inflammation and Ageing, University of Birmingham, UK)
AUTHORS Andy Clark (University of Birmingham); John King (University of Nottingham); John O'Neil (University of Birmingham); Sara Jabbari (University of Birmingham); Teri Parsonage (Department of Inflammation and Ageing, University of Birmingham, UK)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B45
TRACK Systems Biology and Biochemical Networks

Abstract

Tristetraprolin (TTP) is a messenger RNA (mRNA)-binding protein that targets pro-inflammatory mRNAs and recruits protein complexes that promote their degradation, making it a key regulator of inflammation resolution. TTP phosphorylation and activity are regulated by the mitogen-activated protein kinase (MAPK) p38 pathway, a cascade of phosphorylation events that also promotes the expression of pro-inflammatory mRNAs. This pathway is complex, involving multiple protein interactions and feedback loops, and is therefore difficult to understand intuitively.

Biological evidence highlights the therapeutic potential of TTP: in a mouse knock-in model (TTPaa), in which TTP is constitutively active, mice are highly resistant to inflammation. However, clinical trials targeting this pathway have been unsuccessful. This disconnect between compelling biological evidence and poor clinical translation indicates gaps in mechanistic understanding that must be addressed to harness the potential of TTP.

To address this, mathematical modelling and experimental biology are combined in an iterative framework. A system of ordinary differential equations is used to generate experimentally testable predictions that

highlight discrepancies between theoretical and biological results, thereby deepening understanding of the pathway. In this way, the model informs experimental design, and experimental results refine the model to help identify new therapeutic strategies for chronic inflammatory disease.

PP1414 — A Dynamical Model of Pandemic Dynamics with Healthcare Capacity Constraints

SPEAKERS Carmen Martí Raga (Universitat de València)
AUTHORS Antonio Baeza (Universitat de València); Carmen Martí Raga (Universitat de València); Pep Mulet Mestre (Universitat de València)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B20
TRACK Mathematical Epidemiology

Abstract

Epidemics place strong pressure on healthcare systems, particularly on hospital and intensive care unit (ICU) capacity. In this work, we present an extended compartmental SAIR model to study epidemic dynamics and their interaction with healthcare resources. The model explicitly distinguishes between hospitalized and ICU patients, providing a detailed representation of hospital dynamics. It is designed to analyze how hospital occupancy influences epidemic progression and to investigate the impact of healthcare system saturation on mortality rates.

We present the model formulation, including the functions describing hospital and ICU bed occupancy, and analyze the equilibrium points and the basic reproduction number. Numerical simulations are provided to support the theoretical results and illustrate the model dynamics.

PP1289 — A Mathematical Approach for Enhancing Tumor Drug Delivery via Collagen Normalization

SPEAKERS Onur Kerem Özmen (Özyeğin University)
AUTHORS Onur Kerem Özmen (Özyeğin University); Elif Basak Sisman (Özyeğin University); Defne Yilmaz (Dr.)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F37
TRACK Mathematical Oncology

Abstract

The tumor microenvironment (TME) presents significant physical barriers, such as elevated interstitial fluid pressure (IFP) arising from disorganized and leaky vasculature together with a dense extracellular matrix (ECM). These barriers limit the effectiveness of systemic therapies by restricting drug penetration into the tumor core [NMJ20]. We develop a coupled mathematical model to investigate how ECM remodeling influences drug delivery within solid tumors. The model is formulated as a system of nonlinear partial differential equations describing the spatiotemporal interactions among tumor growth, vasculature, IFP, and drug concentration [Yon+17]. To capture the structural role of the ECM, the model explicitly incorporates key constituents, including collagen, hyaluronic acid, and elastin, which together regulate interstitial hydraulic resistance and fluid pressure.

We focus on collagen normalization as a therapeutic priming strategy by modeling collagenase-mediated degradation of collagen fibers prior to chemotherapy administration. Simulations demonstrate that partial collagen degradation substantially enhances drug penetration throughout the tumor domain, resulting in improved therapeutic exposure compared to chemotherapy alone. Furthermore, the model enables systematic investigation of treatment schedules, providing a predictive computational framework for optimizing combination strategies involving ECM-targeted therapies and cytotoxic agents.

References

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PP1545 — A Mathematical Framework for Per-Read and Per-Sequence Error Characterization in Single-Molecule Sequencing

SPEAKERS Pranjal Srivastava (University of Michigan)
AUTHORS Brian Athey (University of Michigan); Gregory Farnum (University of Michigan); Haoran Li (University of Michigan); Mingze Sun (University of Michigan); Pranjal Srivastava (University of Michigan)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B35
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

We present a mathematical framework for quantifying basecalling error at multiple scales in single-molecule (nanopore) sequencing, from individual bases to whole-sequence classification. We define hierarchical Phred-like quality scores — per-base, per-read, and per-sequence — and prove via Jensen's inequality that averaging in the Phred domain systematically overestimates accuracy relative to the Phred transform of the mean error. This concavity-driven bias has direct consequences for quality reporting. We address it with an alignment-based correctness score incorporating predicted basecaller confidence and empirical accuracy into a single Phred-scale summary. We then lift the analysis to sequences by constructing a confusion matrix indexed by true targets and basecaller assignments. Row-normalization produces a stochastic matrix of pairwise misclassification probabilities; its Frobenius distance from the identity yields a Phred-like scalar of classifier fidelity. Clopper–Pearson confidence intervals from the multinomial row structure, with minimum read-count bounds, ensure reliable estimation of rare confusions. A bridge between scales is provided by per-base reliability zones: under conditional independence, the product of position-specific error rates at discriminating sites predicts which off-diagonal entries dominate, enabling anticipation of sequence-level misclassification from basewise profiles

PP1648 — A mathematical framework for quantifying T cell expansion in acute and chronic regulatory contexts

SPEAKERS Joanna Schnorr (Institute for Experimental Oncology, University Hospital Bonn)
AUTHORS Joanna Schnorr (Institute for Experimental Oncology, University Hospital Bonn); Kevin Thurley (Institute for Experimental Oncology, University Hospital Bonn)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B46
TRACK Immunobiology & Infection

Abstract

A fast and pathogen-specific supply of effector T cells is essential in the mammalian immune response to acute and chronic infections, as well as cancer. While the basic principles of T cell activation and differentiation are well established, the mechanisms that control the balance between proliferation and differentiation remain incompletely understood. Motivated by the observation that simple T cell differentiation motifs where proliferation competes against differentiation fail to explain clonal expansion,

we developed a mathematical framework to quantify desirable expansion-related properties. Specifically, we defined measures for the potential of controlled expansion, the efficiency of the expansion, and the chronicity under persistent antigen exposure. Within this framework, we systematically analyzed different regulation mechanisms of the proliferation, involving cytokine- and antigen-mediated feedback, under acute and chronic conditions. Subsequently, we compared the best-performing motifs to a published T helper cell differentiation motif in the context of LCMV infection in mice [BT23] and to a tumor-immune interaction model [REG12]. In both cases, we fitted the dynamics of the derived motifs to the dynamics in the published models and then compared proliferative potential and chronicity. Our framework provides a foundation for exploring how circuit-level mechanisms influence T cell population dynamics in acute and chronic scenarios.

References

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PP311 — A mathematical framework to accurately reconstruct cell lineage from single cell transcriptomics on barcoded cells: application for therapeutics optimization

SPEAKERS Bence Hajdu (Institut Curie)
AUTHORS Annabelle Ballesta (Institut Curie, INSERM U1331, Cancer Systems Pharmacology team); Anne-Sophie Giacobbi (Institut Curie); Bence Hajdu (Institut Curie)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F13
TRACK Mathematical Oncology

Abstract

Introduction: Quantifying tumor plasticity is essential for understanding resistance in glioblastoma (GBM). A mathematical pipeline is presented for the joint analysis of time-resolved single-cell (sc) transcriptomics and lineage barcoding to characterize cancer cell states and quantify their dynamics (proliferation, death, state transitions) in control or temozolimide-treated conditions. This model is used to design interventions targeting specific cell states to maximize drug efficacy. Methods: Cell states are identified through clustering and enrichment analysis of known signatures . An ordinary differential equation (ODE)-based model is utilized to infer cell state dynamics by integrating both sc barcoding and transcriptomics. Existing work [Lar+21] was extended through the incorporation of exponential growth, improved parameter optimization via the CMA-ES algorithm, and relaxation of sparsity constraints. Results: In GBM lines, lineages are reconstructed across three time points into a hierarchical tree that chieved a close fit to data. Estimating parameters in control or temozolomide-treated conditions revealed key resistance mechanisms . The pipeline was benchmarked against the original model. Strategies to maximize efficacy were designed bt predicting optimal interventions on cell state death or transition rates. Identifying corresponding molecular targets remains the next challenge to allow for clinical translation.

References

[Lar+21] Ida Larsson et al. "Modeling glioblastoma heterogeneity as a dynamic network of cell states". In: *Molecular systems biology* 17.9 (2021).

PP1369 — A Mechanistic ODE Framework for Hormetic Dose–Response: Formulation, Identifiability, and the Limits of Static Data

SPEAKERS Aini Fitriyah (University of Birmingham)
AUTHORS Aini Fitriyah (University of Birmingham); Fabian Spill (University of Birmingham); Ben Cardoen (University of Birmingham); Mabel Lizzy Rajendran (University of Birmingham)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B10
TRACK Systems Biology and Biochemical Networks

Abstract

Hormesis, where low doses stimulate and high doses inhibit, is well characterised empirically, but its mechanistic basis, especially the role of exposure history, remains poorly understood. Existing statistical models describe hormetic curves effectively but impose the biphasic structure through functional form rather than biological principles. To the best of our knowledge, an explicit, interpretable memory state variable has not been integrated into a general mechanistic ODE framework for hormesis. Therefore, we propose a Minimal Mechanistic Model combining dose-dependent stimulation, saturation-type inhibition, and homeostatic recovery, with analytical conditions derived to guarantee a stable hormetic maximum. This is extended to a Memory-Augmented Model, where a leaky-integrator state variable accumulates exposure history and modulates the response through a saturating feedforward term. Both models are validated against cadmium–fecundity data [God18], achieving goodness of fit comparable to empirical models. A central result is an identifiability finding: under steady-state conditions, the memory variable collapses algebraically to a rescaled dose, rendering the models observationally equivalent. We prove analytically that static endpoint data are structurally insufficient to identify memory effects. This formal result motivates an experimental prescription: time-course measurements and repeated-exposure protocols constitute the minimal data requirement for memory identification.

References

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PP1260 — A non-autonomous continuous mathematical model for the Alzheimer biomarker cascade

SPEAKERS Antonio Baeza (Department of Mathematics, University of Valencia, Spain)
AUTHORS Antonio Baeza (Department of Mathematics, University of Valencia, Spain); Cristina Vilar (Department of Mathematics, University of Valencia, Spain); M. Carmen Martí (Department of Mathematics, University of Valencia, Spain); María Albarrán (University of Valencia, Spain)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F31
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

This work presents a mathematical study of the progression of Alzheimer's disease through a dynamical model based on ordinary differential equations. A detailed analysis is carried out on a biomarker cascade model which describes the sequential interaction between beta-amyloid accumulation, tau hyperphosphorylation, neurodegeneration, and cognitive decline, in the line of the models introduced in [HLP22; Pet+19]. A new causal model is proposed, introducing a time-dependent growth rate for amyloid accumulation, providing greater flexibility to represent genetic factors, therapeutic interventions, and cognitive reserve. A theoretical analysis is developed, including analytical properties, equilibrium points, and stability of the

resulting non-autonomous system. Simulations show the model's ability to fit diverse clinical patterns and highlight the usefulness of the model as a predictive tool and its potential for future personalized clinical applications based on real patient data.

References

[HLP22] Wenrui Hao, Suzanne Lenhart, and Jeffrey R. Petrella. "Optimal anti-amyloid-beta therapy for Alzheimer's disease via a personalized mathematical model". In: *PLOS Computational Biology* 18.9 (Sept. 2022). Ed. by Adrianne Jenner, e1010481. ISSN: 1553-7358. DOI: 10.1371/journal.pcbi.1010481. URL: http://dx.doi.org/10.1371/journal.pcbi.1010481.

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PP1228 — A parallel asymmetric particle Gaussian mixture filter for state-space estimation of highly nonlinear oscillators

SPEAKERS San Kim (KAIST)
AUTHORS Daewook Kim (KAIST); Daniel Forger (University of Michigan, Ann Arbor); San Kim (KAIST)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F28
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Understanding biological oscillators often requires reconstructing internal states from measurement time series. This becomes difficult when dynamics contain slow–fast manifolds that produce strongly nonlinear trajectories. Under such conditions, common state estimation methods face fundamental limitations. In particular, the Kalman–Bucy filter assumes system dynamics can be locally approximated by a single Gaussian distribution, which fails in strongly nonlinear regimes. Particle filtering can capture such dynamics but often incurs high computational cost due to Monte Carlo sampling. To address these limitations, we introduce an asymmetric particle Gaussian mixture filter (AP-GMF) for nonlinear oscillatory systems. The posterior distribution is represented by Gaussian particles whose structure adapts dynamically. Particles are split based on local nonlinearity and combined using the Kullback–Leibler divergence, enabling efficient approximation. Compared with the asymmetric particle population density method, AP-GMF achieves comparable or higher accuracy with fewer particles. We evaluate AP-GMF on the van der Pol oscillator, a general nonlinear oscillator, across multiple parameter regimes. The method consistently outperforms existing filters, including the level-set Kalman filter and the continuous–discrete cubature Kalman filter. We also provide a parallel implementation enabling accurate state estimation at practical computational cost.

PP1379 — A Scalable Network-Based Parameterization of Positive Steady States for Dynamical Analysis of Biochemical Systems

SPEAKERS Exequiel Jun Villejo (Institute of Mathematics, University of the Philippines Diliman)
AUTHORS Aurelio V de los Reyes (Institute of Mathematics, University of the Philippines Diliman); Bryan Hernandez (University of the Philippines Diliman); Exequiel Jun Villejo (Institute of Mathematics, University of the Philippines Diliman)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B13

TRACK Systems Biology and Biochemical Networks

Abstract

Understanding how the structure of biochemical reaction networks determines their long-term dynamical behavior remains one of the core problems in systems biology. Analysis of these models becomes increasingly challenging as network size and complexity grow. In this work, we present an enhanced framework for parameterizing the positive steady states of biochemical systems using the structural properties of the associated reaction network. The approach integrates network decomposition with a graph-theoretic network translation method based on elementary flux modes, enabling for an efficient derivation of positive steady states while significantly reducing computational overhead and addressing the various issues encountered in existing approaches. By benchmarking across a diverse set of biochemical models, we demonstrate the improved scalability of the resulting procedure. Finally, we apply the method to illustrate how steady state parameterizations can be used to investigate important biological properties of multistationarity and absolute concentration robustness in the EnvZ-OmpR signaling pathway and a large-scale CRISPRi toggle switch model. These results highlight how the newly developed framework enables a scalable analysis of larger, more complex biochemical systems and provides deeper insights into the long-term behavior of such models.

PP1407 — A Water Transfer Experiment Linking Physical Systems and Mathematical Models

SPEAKERS Brynja Kohler (Utah State University)
AUTHORS Brynja Kohler (Utah State University); Miles Wind (University of Texas Dallas)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B18
TRACK Education and Outreach

Abstract

This poster presents a hands-on laboratory activity that introduces students to modeling water transfer processes across physical systems. Using simple experimental setups, students collect data on flow and transport, develop mathematical models to describe observed behavior, and test model predictions against measurements. The lesson connects concepts from biology, environmental science, and differential equations while emphasizing data analysis, parameter estimation, and model validation. Adaptable for undergraduate courses and secondary classrooms, the activity provides an accessible entry point to interdisciplinary modeling grounded in real-world environmental phenomena.

PP1364 — Advance Prediction of Immunotherapy Outcomes Using Deep Learning

SPEAKERS Juliette Sinnott (University of Waterloo)
AUTHORS Ali Haghighatgooasiabar (University of Waterloo); Chris Bauch (University of Waterloo); Juliette Sinnott (University of Waterloo); Mohammad Kohandel (University of Waterloo, Canada)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B9
TRACK Mathematical Oncology

Abstract

Immunotherapy has varied results and how to predict whether a patient will respond is an open question. It is crucial to closely monitor a patient's response once immunotherapy begins, and to potentially change to an alternate treatment if needed. Using a mathematical model, Creemers et al. argued that a patient's tumor growth rate and immune cell killing rate are the primary parameters affecting response [Cre+21]. These can be difficult to estimate as they vary stochastically over time. We tackle this by training a deep

learning model that takes in noisy time series data of a patient's tumor growth during treatment, and predicts whether or not they will recover during the next five years. We use a CNN-LSTM architecture designed to detect time series transitions far in advance[Bur+21]. This model uses CNN layers to extract important features and then uses LSTM layers to detect patterns over time. Heterogeneous patients are generated using a mathematical model with noisy, realistic parameters. The deep learning model is trained on these synthetic patients, and then a reserved test dataset is used to investigate how far in advance the model can accurately predict outcomes. We propose that frequent tumor measurements of an immunotherapy patient can be fed to the model to help assess whether or not to continue treatment, thus improving the patient's quality of life and chances of survival.

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PP1591 — An agent-based model to investigate the dynamics of HIF, TGF-α, and EGFR signalling under hypoxic conditions in cancer spheroids

SPEAKERS Vaishnudebi Dutta (University of Bristol)
AUTHORS Vaishnudebi Dutta (University of Bristol); Lucia Marucci (School of Engineering Mathematics and Technology); David Barton (School of Engineering Mathematics and Technology, University of Bristol)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B39
TRACK Mathematical Oncology

Abstract

Cancer spheroids capture the hallmark intratumoral heterogeneity of solid tumours, including phenotypic diversity and spatially distinct gene expression patterns, making them invaluable tools for studying therapeutic resistance. Differential receptor expression across tumour subpopulations remains a major challenge in cancer therapy, yet most computational models treat receptor proteins as a uniform, population-wide property, overlooking the functional consequences of mixed receptor populations within a single tumour. Here, we present a multi-scale agent-based model of a two-dimensional cross-section of a cancer spheroid, implemented in the Chaste framework, that resolves heterozygous Epidermal Growth Factor Receptor (EGFR) expression at the single-cell level. By coupling hypoxia-inducible factor (HIF)-driven transforming growth factor-α (TGF-α) autocrine and paracrine signalling, we show that the ratio of wild-type to mutated EGFR governs emergent spheroid phenotypes - proliferation in normoxic zones, quiescence under hypoxia, and necrosis under anoxia. We envision our modelling framework will provide a foundation for designing therapeutic strategies by accounting for the heterozygous receptor landscapes observed in clinical tumour samples.

PP1453 — An investigation of The Role of Light in Sleep-Wake Dynamics based on Mathematical Model of Sleep pressure and Circadian Rhythm

SPEAKERS Seunghun Lee (KAIST, Department of Mathematical Science)

AUTHORS Seunghun Lee (KAIST, Department of Mathematical Science); Yun Min Song (Biomedical Mathematics Group, Pioneer Research Center for Mathematical and Compuational Sciences, Institute for Basic Science, Daejeon, Republic of Korea); Jae Kyoung Kim (Department of Mathematical Sciences, KAIST, Daejeon, 34141, Republic of Korea)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B25
TRACK Cellular and Developmental Biology

Abstract

Human sleep and daytime sleepiness are fundamentally governed by the intricate interplay of two physiological processes: homeostatic sleep pressure and the circadian rhythm. Homeostatic sleep pressure represents the progressive accumulation of sleep propensity that builds continuously during periods of wakefulness and systematically dissipates during sleep. On the other side, circadian rhythm is an endogenous, self-sustaining oscillation of approximately 24 hours driven by the biological master clock. This biological pacemaker dictates the optimal timing for sleep and wakefulness and is primarily synchronized, by environmental light exposure.

While an extensive body of empirical literature suggests that robust daytime bright light exposure is positively associated with enhanced nighttime sleep architecture, increased sleep efficiency, and reduced fragmentation, the precise mechanistic pathways driving this correlation remain incompletely understood. In this study, we use a mathematical model to investigate how light exposure influences the interaction between sleep pressure and the circadian rhythm in the regulation of sleep

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PP1594 — Analysis of the Role of Network-Plasticity-Related Proteins in Molecular Networks

SPEAKERS Mark Kerestely (Department of Molecular Biology, Semmelweis University, Budapest, Hungary.)
AUTHORS Mark Kerestely (Department of Molecular Biology, Semmelweis University, Budapest, Hungary.); Istvan Narozsny (Department of Molecular Biology, Semmelweis University, Budapest, Hungary); Levente Szarka (Department of Molecular Biology, Semmelweis University, Budapest, Hungary); Daniel Veres (Turbine Simulated Cell Technologies, Budapest, Hungary); Peter Csermely (Department of Molecular Biology, Semmelweis University, Budapest, Hungary); David Keresztes (Department of Molecular Biology, Semmelweis University, Budapest, Hungary)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B40
TRACK Systems Biology and Biochemical Networks

Abstract

Cellular plasticity – the ability of cells to adapt to environmental changes by producing diverse phenotypes – plays a vital role in the reprogramming of cells across a broad spectrum of diseases, including cancer, diabetes, and neurodegeneration. It is also a key component of embryonic development and tissue remodelling. Targeting this plasticity is a promising therapeutic approach, as demonstrated by sequential and differentiation therapies, drug holidays, and alternating treatments [Ker+26]. An effective way to model complex cellular processes like plasticity is through network representations, such as protein–protein interaction (PPI) networks, signalling networks, and gene regulatory networks, whose network plasticity reflects cellular plasticity. We aim to identify proteins that modulate network plasticity and elucidate their roles within the molecular networks of diseased cells. From the literature, we have gathered multiple regulators of network plasticity and cross-referenced these with existing drug targets. Our analysis indicates that intrinsically disordered proteins, a source of molecular-level plasticity, are enriched among regulators

of network plasticity. The results of the network analysis suggest that direct signalling interactions involving network plasticity regulators are generally transient or causational, such as phosphorylation and transcriptional activation.

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PP1410 — Applying applied maths: mathematical biology in health economics

SPEAKERS Joe Hilton (University of Manchester)
AUTHORS Joe Hilton (University of Manchester)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B19
TRACK Mathematical Epidemiology

Abstract

Health economic analyses are a key stage in implementing new technologies and policies in health care, supporting healthcare providers in making effective use of limited resources. While health economists and mathematical biologists both take quantitative approaches to problems in human health, health economic models typically do not take advantage of the full range of methods accessible to mathematical biologists. This leaves health economic modelling as a site where mathematical biologists can make important contributions, with health economists representing a large potential user base for mathematical models. In this talk I will introduce the basics of health economic modelling with an emphasis on the role mathematical biologists can take as part of an interdisciplinary health economics team, and the challenges that can arise due to different conceptions of modelling within the two fields. I will also suggest ways for mathematical biologists to make their research more accessible to health economists and thus more likely to be applied in health economic analyses, where we often see modular combinations of multiple models from distinct sources. Throughout the talk I will reflect on my own experiences as a mathematical biologist working within a health economics department and contributing to health economic analyses, with particular focus on an ongoing health economic study of HPV self-sampling in cervical cancer prevention.

PP1620 — Assessing the Safety of Pulsed Field Ablation in the presence of Implantable Medical Devices

SPEAKERS Bakhtawar bibi (University of Ljubljana, Faculty of Electrical Engineering)
AUTHORS Damijan Miklavcic; Lars M.Mattison; Daniel C.Sigg; Bakhtawar bibi (University of Ljubljana, Faculty of Electrical Engineering); Bor Kos
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B42
TRACK Multiscale and Multiphysics Modelling

Abstract

Pulsed field ablation (PFA) is an ablation technology for treatment of cardiac arrhythmias, which relies on electroporation. Although PFA has a nonthermal mechanism, some heating is expected due to the high power of the pulses. The presence of metallic cardiac implants may alter the electric field around the

ablation catheter which may lead to increased heating. The aim was to investigate the PFA interaction with metallic devices: atrial appendage occlusion device, mechanical valve, stent, and ICD lead. A FEM model was developed in COMSOL including a tissue block, a bipolar or monopolar catheter, and different device geometries. Distance between catheter and device was varied from 1–20 mm. A 200 ms duration waveform: 2000 V, and 0.02 duty factor for monopolar delivery; 1800 V with a duty factor of 0.01 for bipolar delivery was used. The mean volume of ablation with Electric field >550 V/cm was 1.8 cm^3 for monopolar and 1.6 cm3 for bipolar. At close distances to the implant the average relative change in ablation volume was -0.63 %, and -7.7 % for monopolar and bipolar delivery, respectively. Maximum tissue temperatures exceeded 100 °C when the catheter was positioned within 2 mm of the implant surface; at larger separations the temperature increase was substantially lower. At distances over 5 mm the thermal effect of the implants was negligible. Metallic implants increased the total current, with a peak increase of 30%, depending on implant geometry and catheter orientation.

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PP1392 — Bacterial Attachment to Medical Implants: How can Mathematics Help us Understand and Prevent Biofilm Formation?

SPEAKERS Otilia Casuneanu (University College London)
AUTHORS Edwina Yeo (UCL); Otilia Casuneanu (University College London); Philip Pearce; Shervanthi Homer-Vanniasinkam
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B16
TRACK Multiscale and Multiphysics Modelling

Abstract

Biofilms are complex, intricate colonies of bacteria that often attach to medical implants, causing complications and failure of medical procedures, including the need of implant removal, sepsis, and eventually loss of life. According to the National Biofilms Innovation Centre, biofilm contamination also represents an economical burden, leading to over £45 billion in financial losses yearly in the UK alone. Due to the wide variety of processes involved in biofilm formation, as well as difficulty in observing these processes in a clinical setting, there is still a lot unknown about the initial attachment of bacteria on implant surfaces, with several hypotheses arising in the literature. We describe the various factors that are involved and need consideration in the mathematical models, as well as highlight possible mathematical methods that can lead to a better understanding of the biological context.

PP1626 — Benchmarking temporal causal discovery for fully and partially observed biochemical kinetic models

SPEAKERS Holly Chambers (Imperial College London)
AUTHORS Barbara Bravi (Imperial College London); Hervé Isambert (Institut Curie); Holly Chambers (Imperial College London); Vahid Shahrezaei (Imperial College London)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B44
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Systems of intracellular biochemical reactions are highly complex, usually involving parts that cannot be directly measured. Representing these systems as networks, with nodes for biochemical species and edges, their reactions help to quantitatively characterize their function and the effects of dysregulation. Causal discovery methods can uncover interactions within these networks from observational data, detecting hidden effects from partial observations. We benchmark state-of-the-art temporal causal discovery methods on time series data from simulations of biochemical kinetics models. Our results demonstrate good performance on toy models for this task, particularly when data is sampled in a way that is consistent with the timescales of the system. By omitting data, we consider the problem of reconstructing these networks in the presence of latent confounders and unobserved species participating in reactions. Causal discovery indicates time-uncorrelated confounders with bidirected edges and unobserved species through time-delayed edges, locating hidden effects, and estimating their typical timescales. Finally, we extend these benchmarks to the reconstruction of a model of the epidermal growth factor receptor signalling network, a well-studied system frequently dysregulated in cancer. Altogether, our work showcases the feasibility and usefulness of causal discovery methods as part of the data-driven mathematical modelling pipeline for systems of biochemical reactions.

PP1622 — Characterizing and Modeling Age and Drug Related EEG Waveform Asymmetry during Anesthesia

SPEAKERS Marie Steil (TU Wien)
AUTHORS Andreas Körner (TU Wien); Emilia Kawecka (TU München (TUM)); Marie Steil (TU Wien); Matthias Kreuzer (TU Munich)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B43
TRACK Neuroscience and Neural Systems

Abstract

Current commercial EEG anesthesia monitoring systems put older patients at risk of overdose [Ni+19; Obe+21]. Such monitors rely on frequency-domain EEG analysis, which assumes quasi-periodic stationary signals [Coh14], but omitting phase data causes information loss. As EEG waveforms are often asymmetric

[CV17], an overlooked feature in the context of anesthesia may be the asymmetry of EEG oscillatory activity in the time-domain.

Waveform asymmetry measures were assessed across frequency bands in two independent datasets with respect to age and anesthetic compound. Additionally, the Hilbert envelope of EEG bands was extracted and characterized using the same measures, zero-crossing variability, and sinusoidal parametric model fitting.

Sharpness and steepness asymmetry decreased with patient age in a frequency-dependent manner, strongest in the delta band. Sevoflurane and desflurane showed stronger concentration-dependent effects than propofol. Envelope analysis revealed consistent sharpness asymmetry across all frequency bands. High zero-crossing variability and limited parametric model fit suggest that simple sinusoidal models may not fully capture the envelope.

Building on these findings, we aim to investigate whether asymmetric waveforms can be described by computational models to develop more physiologically accurate EEG representations.

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PP349 — Comparing systems of ODEs to structured models for modeling heterogeneity of neural stem cells

SPEAKERS Carolin Lindow (Heidelberg University)
AUTHORS Anna Marciniak-Czochra; Carolin Lindow (Heidelberg University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F17
TRACK Cellular and Developmental Biology

Abstract

Adult Neurogenesis is the generation of neurons from neural stem cells after the juvenile development is completed. It is essential for the upkeep of cognitive functions and for reaction to injuries. To protect the cells from depletion and mutations, the cells cycle between dividing and non-dividing (quiescent) states. It has been seen experimentally that the quiescent cells are heterogeneous and exist on a continuum of quiescent depths. An established framework to model cell populations is by a compartmental model using ODEs. Here, heterogeneity of the cells can be included by dividing the cells into many compartments that can have different evolution and interaction dynamics. Investigating the limit where heterogeneity becomes continuous, a structured model based on quiescence depth arises through parameter rescaling. Even though the limit of the ODE system solves the structured model, the densities of the ODEs embedded in the state space of the structured model differ from the solutions of the structured model. Similar effects have been seen before. We explore this effect and the reasons for the limited comparability between the models. This will allow us to understand in which cases it is a suitable approximation to use established methods for compartmental models, e.g. for efficient parameter estimation and numerical simulations to learn about the structured models.

PP59 — Considerations on Model Complexity with respect to Parameter Sensitivity and Identifiability: A case study of cell invasion

SPEAKERS Veronika Hofmann (Technische Universität München)
AUTHORS Veronika Hofmann (Technische Universität München); Sebastian Doctor (Technische Universität München); Christina Kuttler (TU Munich)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F3
TRACK Cellular and Developmental Biology

Abstract

Cell invasion is a process in which cells degrade surrounding tissue and start populating the newly created space. It occurs in healthy and ill cells, during wound-healing but also during cancer. There are many mathematical models of different modalities and complexity levels that aim to describe and quantify this phenomenon. In this work, we compare the outputs of two partial differential equation (PDE) models [Cro+23; Col+21] and a hybrid model based on [van+14] using a naïve data fit, and the relationships of their parameters with a variance-based sensitivity analysis to see how different parameter interactions can or cannot produce similar results. We observe rather superficial parameter relationships in the PDE models, and more deeply intertwined relations in the hybrid model. Despite these differences on a deeper level, we find that the PDE models are suitable to describe the behavior of the hybrid model when restricted to the same, one-dimensional domain. We conclude with a parameter identifiability analysis with the simplest model to understand whether it is reduced enough to allow for a confident parameter estimation. We find that only half of the parameters are practically identifiable, and justify this discovery with the findings from the sensitivity analysis.

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PP1553 — Contrastive Self-Supervised Learning for Decoding Physical Parameters in Stochastic Gene Expression Dynamics

SPEAKERS Xi (Ian) Yang (University of Edinburgh)
AUTHORS Xi (Ian) Yang (University of Edinburgh)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B37
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Cells encode environmental information through the nuclear localisation dynamics of transcription factors (TFs) - stochastic time-series governed by physical parameters: mean expression (μ), coefficient of variation

(CV), and autocorrelation time (T_{ac}). Labelling these is costly, motivating a foundational self-supervised model generalisable across TF localisations and biological contexts [Zha+22; Yue+22].

Benchmarks reveal no single approach generalises: raw SVMs achieve 86% on full-length trajectories by exploiting transient bursts, but collapse to chance (49%) on truncated series. Catch22 [Lub+19] - 22 interpretable time-series features - scores 67% on truncated but only 61% on full-length data. Dataset design governs apparent performance.

To learn dataset-agnostic representations, we train a SimCLR-style [Che+20] contrastive Transformer [Vas+17] on Gillespie-simulated trajectories via InfoNCE loss. The Transformer acts as a feature extractor whose embeddings feed a downstream SVM used for classification.

We uncover a fundamental paradox: normalisation is essential for stable contrastive training, yet destroys absolute scale. Consequently, μ - trivially recoverable by a raw SVM - collapses to chance (50%) across all SSL variants, while CV (87%) and T_{ac} (63%) are well-encoded. This exposes a hard incompatibility between SSL stability and scale preservation in stochastic biological time-series.

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PP1504 — Decoding Nonequilibrium Dynamics in Biological Systems with Horizontal Visibility Graphs

SPEAKERS Lucía Benito Barca (Universidad Francisco de Vitoria)

AUTHORS Mary Luz Mouronte López (Universidad Francisco de Vitoria); Francisco Monroy (Department of Physical Chemistry, Complutense University of Madrid, Ciudad Universitaria, 28040 Madrid, Spain.); Lucía Benito Barca (Universidad Francisco de Vitoria); Diego Herraez Aguilar (Universidad Francisco de Vitoria)

SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations

BOARD B30

TRACK Cutting Edge Research Areas

Abstract

Fluctuations in red blood cell (RBC) membranes and chromatin domains encode key information about cellular mechanical properties and metabolic activity, which are often linked to physiological states and pathological alterations [Di +18]. Quantitative analysis of these fluctuations provides access to the underlying dynamics that characterize living systems as stochastic systems operating far from equilibrium. Here we transform experimental time series into complex networks using the *Horizontal Visibility Graph* (HVG) method [Luq+09] and exploit their topological properties as quantitative descriptors of active dynamics. We show that, without requiring additional preprocessing beyond the time-series-to-network conversion, graph-based metrics discriminate metabolic states across different cellular groups. Furthermore, the analysis reveals the presence of the precision–efficiency uncertainty principle in biological systems [Gne+18], whereby reductions in dynamical uncertainty occur at the expense of system efficiency. Together,

these results indicate that the HVG framework provides a sensitive and robust approach to characterize active biological dynamics and to detect physiological alterations from fluctuation patterns.

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PP1425 — Development of a computational microscopy pipeline for the inference of the subcellular and population-scale mechanome of red blood cells

SPEAKERS Jorge Barcenilla González (Computational Biophysics and Biological Data Analysis, Institute for Biosanitary Research, Faculty of Experimental Sciences. Francisco de Vitoria University (UFV))

AUTHORS Diego Herráez (Computational Biophysics and Biological Data Analysis, Institute for Biosanitary Research, Faculty of Experimental Sciences, Francisco de Vitoria University (UFV), Pozuelo de Alarcón, 28223 Madrid, Spain.); Francisco Monroy (Department of Physical Chemistry, Complutense University of Madrid, Ciudad Universitaria, 28040 Madrid, Spain.); Jorge Barcenilla González (Computational Biophysics and Biological Data Analysis, Institute for Biosanitary Research, Faculty of Experimental Sciences. Francisco de Vitoria University (UFV))

SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations

BOARD B22

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Abstract This work presents an automated pipeline designed for the inference of the subcellular and population-level mechanome in red blood cells (RBCs). The system utilizes high-resolution spatial (65 nm/px) and medium-resolution temporal (30 Hz) video microscopy to capture the dynamics of cell membrane thermal fluctuations (*flickering*).

From a mathematical and data-driven perspective, the computational framework integrates deep learning models (YOLO/ResNet) for cell localization. Membrane quantification is performed by searching for the maximum intensity gradient within the polar-transformed bounding box of the selected cell, achieving subpixel contour tracking. This architecture allows for modeling membrane fluctuations as stochastic processes, from which fundamental biophysical properties constituting the mechanome—such as viscoelasticity, mechanical power, and entropy production—are derived.

The pipeline demonstrates real-time performance levels, having processed a total population of approximately 5,000 individual cells. The method was tested by evaluating the effect of quercetin on membrane stiffness, yielding results consistent with established literature and demonstrating high sensitivity for detecting molecular modulations and subcellular mechanical heterogeneities. This advancement provides a robust statistical and computational framework for large-scale analysis in cellular mechanobiology.

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PP1209 — Development of a Generalized Real-Time Early Detection Model based on Bootstrap Clustering

SPEAKERS Yeonsu Lee (Department of Statistics, Kyungpook National University)
AUTHORS Hyojung Lee (Department of Statistics, Kyungpook National University); Yeonsu Lee (Department of Statistics, Kyungpook National University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F26
TRACK Mathematical Epidemiology

Abstract

Following the COVID-19 pandemic, seasonal infectious disease trend shifts have highlighted the need for advanced early warning systems. Conventional methods, such as change point detection and hockey-stick regression, are widely used but are designed for retrospective analysis. Building on these approaches, we aim to develop a generalized real-time early detection model based on bootstrap clustering for various seasonal infectious diseases. This study utilizes influenza-like illness (ILI), respiratory syncytial virus (RSV), norovirus, and hand-foot-and-mouth disease (HFMD) from the Korea Disease Control and Prevention Agency (KDCA). Bootstrap clustering traditionally evaluates stability via the Jaccard coefficient [Yu+19]. In this study, we extend this approach beyond stability assessment to develop a distribution-based early detection framework. Furthermore, we conducted real-time detection simulations by sequentially incorporating weekly data. This approach supports a three-level warning system consisting of Caution, Alert, and Severe stages using distribution-based early detection. This framework provides a generalized early detection model that can be applied to various seasonal infectious diseases. Unlike conventional statistical methods, the proposed approach enables real-time early detection. By introducing a three-level warning system, this approach has the potential to enhance timely preparedness and response for a wide range of seasonal infectious diseases.

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PP120 — DNA Strand Displacement Sequential Release Circuit for Scheduled Therapeutic Interventions

SPEAKERS Alivia Behnke (Washington State University)
AUTHORS Alivia Behnke (Washington State University); Dominic Scalise (Washington State University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F4
TRACK Systems Biology and Biochemical Networks

Abstract

HIV remains a major global health challenge because antiretroviral therapy (ART) suppresses active virus but cannot eliminate the latent reservoir of infected CD4+ T cells, which can persist for years and reignite infection if treatment stops. New strategies are therefore needed to address viral latency. This theory project explores the use of DNA strand displacement (DSD) circuits to autonomously control the timing and sequence of drug delivery for chronic infections such as HIV. Using simulations, we couple a programmable ten-stage molecular release circuit to a mechanistic HIV disease model that tracks uninfected, infected, and latently infected CD4+ T cells, free virus, and drug concentrations. The model incorporates both ART and latency-reversing agents (LRAs). We integrate a sequential DSD circuit that acts as a molecular controller, releasing payloads that activate latent virus, deliver ART, or pause treatment based on therapeutic effectiveness. In simulation, this system can implement a "shock and kill" strategy by activating latent reservoirs, evaluating multiple drug options, and continuing the most effective treatment until the reservoir is reduced, after which the circuit remains dormant but responsive. These results suggest that programmable DSD timing circuits could enable autonomous, patient-specific therapies for HIV and other diseases requiring long-term, staged interventions.

PP1373 — Doubly Structured Models of Tumour-Immune Cell Reactions

SPEAKERS Fenora David (UCL)
AUTHORS Fenora David (UCL); Helen M. Byrne (University of Oxford); Benjamin J Walker (UCL)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B12
TRACK Mathematical Oncology

Abstract

Tumour progression is shaped by a dynamic interaction between tumour cells and the immune system. One specific immunosuppressive mechanism to study tumour evasion causes cytotoxic T-cells, a major driving force of the immune system, to differentiate to an 'exhaustive' state where they have reduced immune effectiveness due to increased tumour interactions. While this is incorporated in existing mathematical models, we now extend this framework by introducing a doubly structured tumour-immune model in which tumour cells are additionally structured by accumulated damage due to immune attacks. Initially considering binary structure variables helps us build intuition, before then examining larger compartment numbers towards the continuum limit.

PP1442 — Dynamical System Model Discovery from Sparse Data with GFlowNets and PINNs

SPEAKERS Saranya Varakunan (University of Waterloo)
AUTHORS Mohammad Kohandel (University of Waterloo); Saranya Varakunan (University of Waterloo)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B24
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Discovering differential equations governing dynamical systems is a fundamental challenge in mathematical biology, where mechanistic models are used to study complex processes such as gene regulation, cell fate decisions, and tumor dynamics. This becomes particularly difficult when experimental data is sparse. Generative Flow Networks (GFlowNets) are a probabilistic framework for generating diverse candidate solutions using a reward function [Ben+21], and recent work has shown that GFlowNets can be applied with symbolic regression for mechanistic model discovery [LMM23]. However, biological data are often sparse and noisy, making the derivative estimates required for this approach unreliable.

We present a method that combines GFlowNets with physics-informed neural networks (PINNs) to discover differential models from sparse time-series data. The GFlowNet sequentially generates candidate symbolic expressions for a differential equation; each expression is then fit using a PINN, which learns trajectories consistent with both sparse/noisy observations and the proposed differential equation. The resulting PINN loss defines the reward used to train the GFlowNet. We demonstrate this approach on synthetic biological data from gene regulatory networks, and evaluate whether the method can recover governing equations under sparse data conditions.

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PP1264 — Early Detection of Malaria Outbreaks Using Incubation Period Distributions and Machine Learning

SPEAKERS Jeonghwa Seo (Department of Statistics, Kyungpook National University, Daegu, 41566, Korea)
AUTHORS Hiroshi Nishiura (Graduate School of Medicine, Kyoto University, Yoshidakonoecho, Sakyo-ku, Kyoto, 606-8501, Japan); Hyojung Lee (Department of Statistics, Kyungpook National University, Daegu, 41566, Korea); Jeonghwa Seo (Department of Statistics, Kyungpook National University, Daegu, 41566, Korea)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F32
TRACK Mathematical Epidemiology

Abstract

Malaria remains a major global health concern. In the Republic of Korea, Plasmodium vivax is the dominant parasite and is characterized by both short- and long-incubation periods. Climate change has altered the mosquito habitats and expanded outbreak areas. However, the current malaria warning system in Korea is activated only when Plasmodium parasites are detected in captured mosquitoes. This study aims to improve the early detection of malaria outbreaks. We utilized malaria case data from the Korea Disease Control Prevention and Control Agency (KDCA) and meteorological data from the Korea Meteorological Administration (KMA). First, we estimated infections at the time of infection using a back-calculation approach based on incubation period distributions. Second, we compared random forest, XGBoost, and LSTM models using meteorological variables from 2012 to 2020 and selected the best-performing model to predict infections. Finally, we identified outbreak periods using change point detection methods. The XGBoost model showed the best performance in predicting infections. The outbreak onset estimated from long-incubation cases was detected earlier than the time when cases started to rise sharply. These findings suggest that change points derived from estimated long-incubation cases can serve as an effective tool for the early detection of P. vivax outbreaks. Overall, this approach may serve as a useful basis for determining the timing of preventive interventions.

PP1224 — Emergent patterns in systems coupling diffusive and non-diffusive components

SPEAKERS Finn Münnich (Heidelberg University)
AUTHORS Anna Marciniak-Czochra (Heidelberg University); Finn Münnich (Heidelberg University); Szymon Cygan (Heidelberg University); Théo André (Heidelberg University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F27
TRACK Cellular and Developmental Biology

Abstract

Reaction-diffusion-ODE systems have emerged as powerful models for pattern formation in developmental biology, capturing the interplay between diffusive and non-diffusive nonlinear processes. These systems exhibit a rich variety of spatial structures, including classical Turing patterns and far-from-equilibrium patterns. Previous studies focused on diffusion-driven instability (DDI) generated by instability of the purely non-diffusive subsystem, which destabilizes classical Turing patterns. In contrast, we show that DDI can also arise from subsystems coupling non-diffusive and slowly diffusing components, giving rise to dynamics involving three distinct spatial scales. Further, we characterize conditions for the emergence of stable Turing patterns via bifurcation theory in a three-equation system and prove the existence of far-from-equilibrium patterns in general reaction-diffusion-ODE frameworks. As illustrative examples, we apply our results to specific models, demonstrating the broad applicability of the approach.

PP1344 — Evolution of the cell transcriptome during differentiation

SPEAKERS Julien Blohm
AUTHORS Julien Blohm
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B6
TRACK Systems Biology and Biochemical Networks

Abstract

Cells have several possible fates (diseases, differentiation, death). A way to describe those is via ‘cell trajectories’, in which the transcriptome of a cell evolves from a state A to a state B in a defined manner. Understanding transcriptome evolution is critical for identifying or improving treatments for various diseases. Here, we chose to study a particular cellular fate: differentiation. To understand this mechanism, we aim to predict the real-time evolution of the transcriptome in reprogrammed stem cells as they differentiate into neuronal cells via neural stem cells. We develop a new mathematical approach to identify genes that regulate neuronal differentiation from RNA sequencing data, using a workflow recently developed by our team. It involves three steps. First, the selection of bimodal genes, i.e., those with two expression modes (high or low) within the same population, as differentiation is a switch phenomenon. Then, the cells were clustered based on bimodal gene expression patterns. Finally, applying differential expression analysis to clustered data, we can identify genes that are over- or underexpressed between clusters. These correspond to genes that regulate neuronal differentiation. Starting from a sample consisting of two replicates of 19500 genes and 13000 cells each, we can isolate the 500 most relevant genes for cell differentiation. It will lead to the construction of gene regulatory networks and the development and analysis of deterministic models.

PP1285 — Exploring the Neuroblastoma Microenvironment Using Cellular Automata: Insights into Celyvir Therapy.

SPEAKERS José García Otero (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha)

AUTHORS José García Otero (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha); Juan Belmonte Beitia (Castilla-La Mancha university); Juan Jiménez-Sánchez (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla-La Mancha (UCLM))
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F36
TRACK Mathematical Oncology

Abstract

Neuroblastoma is a significant health concern in children, as it is one of the most common types of cancer among this age group and is associated with poor survival rates. Currently, there are no effective therapies that significantly improve outcomes for these patients. This study explores the efficacy of Celyvir – an advanced therapy comprising mesenchymal stem cells carrying the oncolytic virus against neuroblastoma, by means of an individual-based model. A probabilistic cellular automaton was developed to implement the dynamic interactions between neuroblastoma cells, T lymphocytes, and the treatment Celyvir. The model examines various sizes, shapes, and positions of the tumour within a lattice, along with different infection probabilities associated with the action of Celyvir and various treatment schedules. This analysis identifies the most influential infection probabilities according to the model, and demonstrates that different treatment regimens can effectively eradicate the tumour, in contrast to standard clinical approaches. Additionally, Kaplan–Meier curves have been generated to assess different treatment schedules under specific tumour scenarios, highlighting the importance of precise treatment scheduling to optimise therapeutic outcomes. This study provides insights into the potential of Celyvir in neuroblastoma treatment, emphasising the need to understand tumour dynamics and strategically implement treatment schemes to improve clinical outcomes [OBJ25].

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PP1292 — Extending the Next Generation Matrix method outside of Mathematical Epidemiology

SPEAKERS Andrei-Dan Halanay (Bucharest University)
AUTHORS Andrei-Dan Halanay (Bucharest University); Florin Avram (Université de Pau, 64000 Pau, France); Miruna Beldiman (ISMMA of the Romanian Academy); Rim Adenane (Université Ibn-Tofail)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F38
TRACK Systems Biology and Biochemical Networks

Abstract

We present the main results from our recent work, extending methods from Mathematical Epidemiology (ME) to other systems that can be framed as Chemical Reaction Networks (CRNT). Our main result concerns the stability of equilibria for positive system of differential equations. Positive systems appearing in CRNT have their dynamics determined by a family of forward-invariant faces of some cones. These faces correspond to disease-free or partially disease-free manifolds in ME. We construct an analogue of the Next Generation Matrix and use it to give a stability criterion in terms of its spectral radius. We aim also to present some applications to concrete systems.

PP1338 — Extracting Hidden Cellular Dynamics: A Bayesian Approach to Estimating Time-varying Growth Rates from Noisy Microscopy Data

SPEAKERS Vishvas Ranjan (Centre INRIA de saclay)
AUTHORS Vishvas Ranjan (Centre INRIA de saclay)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B4
TRACK Systems Biology and Biochemical Networks

Abstract

Estimating time-varying cellular growth rates from time-lapse microscopy remains computationally challenging [Ksc+25]. Image segmentation errors propagate into size measurements, and because traditional methods rely on finite-difference approximations, they amplify this noise. This obscures biological fluctuations and forces reliance on moving averages that artificially flatten true dynamics. To address this, we propose a continuous state-space model uncoupling biological growth from observation noise. The unobservable cellular growth rate is modeled as a mean-reverting Ornstein-Uhlenbeck (OU) process. Cell area is modeled as the exponential of the integral of this growth rate, subject to multiplicative segmentation noise. We deployed a Bayesian inference pipeline, combining MCMC parameter estimation with a RTS Kalman Smoother, to retroactively extract the hidden time-varying growth rate from noisy area measurements [Dav+22]. Validation on simulated data demonstrates our Bayesian approach significantly outperforms moving-average techniques. By leveraging the exact cross-covariance between area and growth rate, the model successfully suppresses noise without sacrificing temporal resolution. Currently being tested on raw experimental data, this model will subsequently be extended to jointly estimate cell-internal biochemical processes, specifically plasmid copy numbers, to quantify how internal process time-scales correlate with overall growth rate fluctuations.

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PP1238 — Fluctuations of plasmid copy numbers in a population

SPEAKERS Valentine Brulard
AUTHORS Valentine Brulard
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F30
TRACK Systems Biology and Biochemical Networks

Abstract

Plasmids are extrachromosomal DNA molecules that replicate independently of the chromosome and are widely found in bacteria and other organisms. In nature, plasmids are ubiquitous and can carry various genes that play essential roles in the life of bacteria. In synthetic biology, plasmids are used as the standard tool to equip cells with designed gene circuits. Despite their importance, the theoretical literature on plasmid copy number dynamics remains fragmented, and many models are only described in the supplementary material of experimental studies. This situation often leads experimentalists to rely on models whose mechanistic basis is not fully established. In this poster, I present a new

mathematical modeling framework to connect single-cell models of plasmid replication and segregation with experimentally measured plasmid copy-number distributions in growing populations. The approach couples a structured population model with a Markov jump process.

PP286 — From adaptation to collapse: a model of thermoregulation under prolonged cold stress

SPEAKERS Pavel Morcinek (Department of Mathematics and Statistics, Faculty of Science, Masaryk University)
AUTHORS Filip Zlámal (Department of Physical Activities and Health Sciences, Faculty of Sport Science, Masaryk University); Jan Kučera (Department of Physical Activities and Health Sciences, Faculty of Sport Science, Masaryk University); Julie Bienertová-Vašků (Department of Physical Activities and Health Sciences, Faculty of Sport Science, Masaryk University); Pavel Morcinek (Department of Mathematics and Statistics, Faculty of Science, Masaryk University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F9
TRACK Multiscale and Multiphysics Modelling

Abstract

Mathematical models of human thermoregulation are widely used to study temperature regulation in challenging environments, yet their complexity often limits the systematic analysis of long-term behaviour. Many established models include non-smooth, threshold-based mechanisms and do not explicitly represent the gradual loss of thermoregulatory capacity during prolonged cold exposure. In this talk, we introduce a new smooth thermoregulatory model that incorporates a thermoregulatory fatigue mechanism. The model reproduces behaviour consistent with experimental data and established models while enabling rigorous analysis of steady states and long-term dynamics. Using bifurcation analysis, we reveal a bistability phenomenon that offers a mechanistic explanation for sudden collapse after small perturbations, such as a brief lapse in physical activity, during prolonged cold exposure. Finally, the analysis yields approximate conditions for long-term survival and links the long-term response to a transition from homeostatic regulation to allostatic adaptation, culminating in thermoregulatory collapse.

PP1342 — Genotoxic Effects of Bacteria and Drugs: Investigation of Hidden Interactions with a Differential Equation-Based Approach

SPEAKERS Anna Geretovszky
AUTHORS Anna Geretovszky; Gergely Röst (University of Szeged)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B5
TRACK Mathematical Oncology

Abstract

We investigate the genotoxic effects that bacterial infections and antibiotics exert on human cells. Bacteria may induce mutations either by invading cells or by generating extracellular stress, and drugs can also have mutagenic effects. Moreover, complex interactions between the bacteria and the drug take place in the background. We modify the usual in-host pathogen models for chronic infections to create a new model by including self-replication for the bacteria, as well as a pathogen-loss term. We obtain a system of nonlinear differential equations which describes the interactions between healthy and infected human cells, the external bacteria, and possibly drug treatment. We carried out a detailed analysis of the model. We discovered surprisingly complex dynamical phenomena, including bistability and different types of bifurcations (saddle-node, transcritical, backward). We determined the optimal infection rate from the bacteria's perspective, and noted that for certain parameter combinations, there is evolutionary risk when striving for this value. Finally, we looked into the cumulative genotoxic effect of different therapies

and compared the results of the model with experimental data measuring DNA damage in cells. Our model provides insight and helps to understand how the aforementioned interactions influence the total mutagenic effects on cells.

PP380 — Identifying Key Patient-Specific Parameters of the Schnider PK-PD Model using Population Kinetics

SPEAKERS Christina Huber (TU Wien)
AUTHORS Alexander Edthofer (TU Wien); Andreas Körner (TU Wien); Christina Huber (TU Wien)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F21
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Population pharmacokinetic (PPK) modeling, as presented in [EW04] and [MU12], offers a systematic approach for quantifying inter-individual variability and estimating population parameters in PK-PD models. Within this framework, co-variate effects on the pharmacokinetic parameters of the Schnider PK-PD model [Sch+98], commonly implemented in target controlled infusion (TCI) infusion pumps for anesthesia, are examined. This helps identify key patient-specific parameters to simulate the concentration-time profile of individual patients as accurately as possible since the overall goal is to improve individualized anesthesia. Particular emphasis is placed on the central compartment volume $V1$ and the initial phase of drug administration, as the overarching aim is to prevent an overshoot at the onset of the infusion in the simulation of TCI pumps. Based on the estimated population parameters, an adapted model is developed in which the central volume $V1$ is scaled according to the significant co-variates height and gender, which were selected using a backward co-variate screening procedure. Compared to the original Schnider model, the adapted model demonstrates substantial improvement, particularly in its ability to more accurately represent high concentration levels.

References

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PP1383 — Impact of Influenza Antiviral Treatment on the Risk of Household Transmission and the Serial Interval

SPEAKERS Jihyeon Kim (Kyungpook National University)
AUTHORS Nishiura Hiroshi (Kyoto University); Lee Hyojung (Kyungpook National University); Jihyeon Kim (Kyungpook National University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B14
TRACK Mathematical Epidemiology

Abstract

Influenza is one of the respiratory diseases, and symptoms can be alleviated by treating with antiviral agents such as baloxavir, oseltamivir, laninamivir, and zanamivir. It is known to have high household transmission risk and the risk differs by age and treatment timing [HSH19]. Baloxavir is particularly effective at reducing secondary attack rate (SAR), and serial interval (SI)—the time between symptom onset in primary and secondary case—also varies with treatment timing. However, there is a lack of prior research analyzing the relationship between SI and SAR. This study aims to compare household SAR by individual characteristics and explain how SAR varies based on treatment timing by estimating SI distribution. A household survey was conducted in Japan (October 2018–February 2019), enrolling 915 housholds. Index cases untreated or treated and houehold contacts were stratified by age and treatment timing, and SARs and odds ratios (ORs) were calculated. Assuming the SI distribution of baloxavir-treated cases reflects infectiousness, likelihood-based models were defined to estimate the reduction effect due to baloxavir on SI and SAR by the treatment timing. SAR was highest in the 0–14 age and treatment initiated within 48 hours of symptom onset was effective in reducing risk. Baloxavir (OR=0.62) showed greater preventive effects than oseltamivir, and earlier treatment produced greater shortening of the SI, which corresponded to lower SAR within households.

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PP1300 — Improving Parameter Identifiability of Stochastic Reaction Network Systems Using Approximate Bayesian Computation

SPEAKERS Matthew Brown (University of Strathclyde)
AUTHORS Matthew Brown (University of Strathclyde); Lyndsay Kerr (University of Strathclyde); John Mackenzie (University of Strathclyde)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F40
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Approximate Bayesian Computation (ABC) is a common tool to tackle statistical inference problems for systems where the likelihood function is intractable, a feature common in biological settings due to the inherent complexity of the models under investigation. ABC replaces the likelihood with a comparison of experimental and simulated data, finding parameters which minimise any discrepancy. To improve the efficiency of ABC techniques such as Sequential Monte Carlo (SMC) are typically implemented where the tolerance used in the rejection sampling procedure is gradually reduced over multiple iterations. We have found that designing appropriate tolerance schedules is critical not only for efficiency but also reliability of ABC SMC, with commonly used techniques for schedule selection often leading to inferred parameter values associated with local minima in discrepancy space, rather than the global minima. This problem is particularly acute in stochastic systems. We propose a new procedure to overcome this issue underpinned by reliably choosing the value for the first tolerance to be just below any local minima, with subsequent iterations refining inference around the true parameter values. We have also found that inference of stochastic systems with limit-cycle dynamics is particularly challenging. However, we show identifiability is improved by incorporating reaction-event level stochastic statistics into the discrepancy metric used to compare observed and simulated data.

PP1268 — Inferring Incubation-Related Temporal Structure in Korean Malaria Incidence Using Attention-Based Time-Series Models

SPEAKERS Seongyu Yu (Korea University)
AUTHORS Daeil Jang (Innovation center for Industrial Mathematics, National Institute for Mathematical Science, Seongnam,Republic of Korea); Sunwha Choi (Innovation center for Industrial Mathematics, National Institute for Mathematical Science, Seongnam,Republic of Korea); Soyoung Kim (Innovation center for Industrial Mathematics, National Institute for Mathematical Science, Seongnam,Republic of Korea); Seongyu Yu (Korea University); Boseung Choi (Korea University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F33
TRACK Mathematical Epidemiology

Abstract

Background & aims of study With the increasing risk of malaria transmission driven by climate change and continued imported cases, understanding malaria dynamics from surveillance data has become important for disease control and public health preparedness. This study aims to infer incubation-related temporal structures and predict malaria incidence from time series using long short-term memory (LSTM) models with attention mechanisms, with validation using synthetic data from an extended SEIR model.

Methods & results Weekly malaria incidence data from Korea spanning 2013–2024 were used to develop and evaluate the proposed models. The attention mechanism assigns varying importance to past observations, enabling identification of incubation-related temporal patterns. The analysis revealed two dominant peaks corresponding to short- and long-term incubation effects. Additional validation using synthetic epidemic data from an extended SEIR model with short and long incubation compartments showed that the attention-based analysis identified long-incubation components and reproduced shifts in temporal influence patterns under different incubation parameters. The models also showed reasonable predictive performance.

Implications The proposed LSTM–attention framework provides a data-driven approach for linking surveillance time series with latent epidemiological structures such as incubation dynamics and may complement mechanistic epidemic models in mathematical epidemiology.

PP1345 — Inferring Intercellular Interaction Rules for HER2+ Breast Cancer Cell Motility Using Weak Form Inference

SPEAKERS Allison Introne (Department of Mathematics, North Carolina State University)
AUTHORS Allison Introne (Department of Mathematics, North Carolina State University); Melanie Sadecki (Department of Mathematics, North Carolina State University); Joseph Fernandes (Department of Cell Biology, Duke University Medical Center); Hillary Zawada (Department of Surgery, Division of Surgical Sciences, Duke University Medical Center); Bruce Rogers (Department of Surgery, Division of Surgical Sciences, Duke University Medical Center); Joshua Snyder (Department of Cell Biology, Duke University Medical Center; Department of Surgery, Division of Surgical Sciences, Duke University Medical Center; Department of Pharmacology and Cancer Biology, Duke University School of Medicine); Kevin Flores (Department of Mathematics, North Carolina State University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B7
TRACK Mathematical Oncology

Abstract

Metastasis is a major determinant of survival and treatment efficacy in cancer, yet the mechanisms by which the competition and interaction of heterogeneous tumor cell clones leads to metastasis remains poorly understood. Prior experiments comparing fluorescently barcoded models of human HER2+ breast cancer show that wild-type, d16, and p95 isoforms differ in their invasion and motility (speed, persistence,

mean-square displacement) properties. We developed a generative AI-based pipeline that extracts single-cell trajectories from in vitro live-cell timelapse microscopy videos of mixed-isoform cultures of HER2+ breast cancer cells. Then we construct a computational pipeline to infer agent-based model (ABM) rules describing the interactions between different isoforms using the single-cell trajectories. We simulate data from an ABM that recapitulates the motility characteristics of mixed-isoform in vitro HER2+ breast cancer cell populations. We then apply weak least squares to test whether intercellular interactions governing cell movement can be recovered from simulated data, and the degree to which recovery accuracy depends on ABM initial conditions such as initial cell density and the proportion of cells within each isoform subpopulation (wild-type, d16, p95).

PP305 — Inferring the Interaction Between Circadian Rhythms and Heart Rate Dynamics from Wearable Data

SPEAKERS Kangmin Lee (KAIST)
AUTHORS JaeKyoung Kim (KAIST); Kangmin Lee (KAIST); Minjun Kim (KAIST); Taekwan Kim (KAIST)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F12
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Circadian rhythms regulate a wide range of physiological processes, including core body temperature, hormone secretion, and cardiovascular activity. Among these, heart rate exhibits pronounced diurnal variation arising from endogenous circadian regulation. Despite this close relationship, the dynamical interaction between circadian rhythms and heart rate variation remains poorly understood. In this study, we investigate the interaction between circadian regulation and heart rate dynamics using long-term wearable device data. We used a mathematical modeling to infer latent dynamics underlying heart rate and circadian variation. This approach enables us to characterize how circadian regulation modulates heart rate dynamics over daily timescales. Our analysis reveals systematic differences in the inferred interaction patterns across demographic groups and clinical outcomes. These results highlight the importance of studying interactions among multimodal physiological rhythms and demonstrate how wearable data combined with dynamical modeling can provide new insights into the regulation of human physiological systems.

PP365 — Investigating the roles of prior chemotherapy and risk type in bi-specific T-cell engager therapy against B-ALL via mathematical modeling and Bayesian inference

SPEAKERS Yifan Chen
AUTHORS Guranda Chitadze (University Hospital Schleswig-Holstein Kiel, Kiel University, Kiel, Germany); Philipp Altrock (Cancer Modeling & Evolution UKSH Campus Kiel); Yifan Chen
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F19
TRACK Mathematical Oncology

Abstract

Blinatumomab is a bi-specific T-cell engager that links CD3+ T-cells to CD19+ B-cells, leading to T-cell-mediated B-cell lysis. While it has improved outcomes in relapsed/refractory B-cell precursor ALL (r/r B-ALL), treatment response remains highly variable and the underlying immunological mechanisms are not fully understood. T-cell exhaustion and impaired immune fitness may contribute to treatment resistance. CD226 (DNAM-1) is a co-stimulatory receptor possibly implicated in anti-tumor immunity and its downregulation may reflect early transitions toward dysfunctional states. We developed a mechanistic

ordinary differential equation (ODE) model distinguishing CD226+ and CD226- T-cell subpopulations during blinatumomab treatment. We incorporate differences in baseline immune fitness between medium- and high-risk pediatric r/r B-ALL patients, reflecting differences in prior chemotherapy exposure. By integrating longitudinal patient cell-count data with Bayesian inference, we characterize patient-specific tumor-immune dynamics under treatment. We further assess parameter identifiability and sensitivity, and evaluate the clinical relevance of inferred immune fitness dynamics. Overall, our modeling, in close collaboration with clinical colleagues, establishes a productive cycle that integrates mathematical modeling and novel data collection strategies at clinically relevant time points to improve bi-specific T-cell engager therapy for pediatric B-ALL.

PP1469 — Large-Scale Mechanistic Modeling for Patient-Specific Drug Recommendation and Organoid Testing in Colorectal Cancer

SPEAKERS Moritz Richter (Life and Medical Sciences (LIMES) Institute, University of Bonn, Bonn, Germany, Bonn Center for Mathematical Life Sciences, University of Bonn, Bonn, Germany)

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SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations

BOARD B26

TRACK Mathematical Oncology

Abstract

Colorectal cancer (CRC) remains a major cause of cancer morbidity and mortality, increasing mostly in adults younger than 50. Precision therapy and drug repurposing approaches may improve outcomes for patients who are ineligible for or do not respond to standard targeted therapy. We describe a computational framework that constructs individualized mechanistic "digital twin" models from patient transcriptional profiles. Proposed in silico drug combinations are then evaluated in patient-derived organoids (PDOs).

To this end, biopsy tissue from CRC patients is used to derive PDO lines and to generate bulk RNA-seq from both original tumor tissue and the established PDOs. Large-scale mechanistic models [Frö+18] of intracellular signaling important for proliferation and survival form the basis of these in silico models, augmented with semi-mechanistic machine-learning components to represent treatment effects not captured by the mechanistic scaffold. Models are trained on transcriptional and cell-viability data from cell lines in CCLE and GDSC. In a second step, these models are individualized by selectively (de-)activating aberrant gene/protein species and parametrizing components using gene expression data. Model construction, parameter estimation and simulation are performed using AMICI, pyPESTO and PEtab [Frö+21], [Sch+23], [Sch+21].

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PP1484 — Learning Optimal Adaptive Therapies in Space and Time: Dynamic Threshold Therapy for Prostate Cancer

SPEAKERS Yunli Qi (Wolfson Centre for Mathematical Biology, University of Oxford, Oxford, UK and Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, USA)

AUTHORS Alexander Anderson (Moffitt Cancer Center); Kit Gallagher (Harvard Medical School); Maximilan Strobl (The Institute of Cancer Research and Imperial College London); Philip K. Maini; Robert Gatenby; Yunli Qi (Wolfson Centre for Mathematical Biology, University of Oxford, Oxford, UK and Department of Integrated Mathematical Oncology, H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL, USA)

SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations

BOARD B28

TRACK Mathematical Oncology

Abstract

BACKGROUND: Adaptive therapy delays drug resistance by modulating treatment instead of continuously applying the maximum tolerated dose [Zha+17]. While Deep Reinforcement Learning (DRL) can optimize adaptive therapy in non-spatial, well-mixed deterministic tumor models [Gal+24], extending it to spatial models is challenging because tumor dynamics become stochastic and clinically observable data are limited. METHODS: We combined DRL with an existing spatial Agent-Based Model [Str+22] and addressed the challenge of stochastic spatial dynamics by introducing a memory mechanism based on historical tumor responses to treatment and holiday periods, using clinically observable tumor burden. We also developed a transfer-learning framework to stabilize learning in stochastic spatial environments. RESULTS: We found that the memory mechanism can significantly improve time to progression relative to memory-free agents. Besides, we show that decision-relevant information is stored in the memory, allowing the agent to infer latent tumor composition and spatial structure from treatment history. Memory therefore provides a biological interpretation for decision-making by acting as a proxy for hidden tumor state. The learned strategies remained robust under treatment perturbations, and dynamic threshold therapy emerged from memory-informed control. CONCLUSION: Historical response memory provides an interpretable and robust mechanism for controlling tumors under limited access to spatial data.

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PP1439 — Linking Social Signals from Large Language Models to Epidemic Transmission Dynamics through Physics-Informed Neural Networks

SPEAKERS Dasom Lee (Korea University)
AUTHORS Boseung Choi (Korea University); Dasom Lee (Korea University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B23
TRACK Mathematical Epidemiology

Abstract

The Susceptible–Infectious–Recovered (SIR) model has long been a foundational framework for modeling infectious diseases and has played an important role in informing public health policies during the COVID-19 pandemic. However, traditional SIR-based approaches primarily rely on epidemiological case data and often fail to account for behavioral and societal factors that influence disease transmission dynamics.

In this study, we propose a novel framework that integrates large language models (LLMs) with physics-informed neural networks (PINNs) to enhance epidemic modeling. Specifically, LLMs are employed to derive structured social signals from large-scale textual sources, capturing information related to policy interventions, public risk perception, and healthcare system pressure. These signals are incorporated into a physics-informed neural network framework to estimate the time-varying transmission rate governing the SIR dynamics.

By embedding the governing differential equations of the SIR model directly into the neural network training process, the proposed approach ensures consistency with epidemiological dynamics while leveraging both observed case data and LLM-derived social signals. Applying the framework to COVID-19 case data from Seoul, we demonstrate that integrating LLM-based social signals improves the model’s ability to capture temporal variations in transmission dynamics compared with conventional SIR models and standard PINN-based approaches.

PP355 — Linking tumour microenvironment dynamics to PET radiomic signatures: a computational modelling framework

SPEAKERS Sahar Jafari Nivlouei (University of Coimbra)
AUTHORS João Carvalho (University of Coimbra); Rui Travasso (University of Coimbra); Sahar Jafari Nivlouei (University of Coimbra)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F18
TRACK Multiscale and Multiphysics Modelling

Abstract

We present a hybrid multiscale model that establishes a mechanistic link between tumour microenvironment dynamics and FDG-PET radiomic signatures. The key innovation is the integration of multiscale tumour modelling with synthetic PET-like image generation, enabling PET radiomic signatures to be interpreted in terms of underlying biological processes. The model integrates intracellular signalling, cell-based tumour dynamics, and tissue-scale transport to investigate how cellular heterogeneity and microvascular organization shape SUV-based, morphological, and texture-derived features across tumour progression. We compare homogeneous and heterogeneous tumour scenarios and show that distinct microenvironmental conditions give rise to measurable differences in PET radiomic patterns. In vascularized tumours, chemotherapy induces reductions in compactness, mean uptake, and texture contrast, consistent with

decreased tumour burden and altered spatial heterogeneity. Agreement with published vascular sprout dynamics, together with consistency between synthetic and clinical lung PET/CT radiomic ranges, supports the biological plausibility of the framework. These findings highlight the potential of mechanistic modelling to provide biologically grounded interpretation of PET radiomics and improve quantitative assessment of therapy response.

PP389 — Machine Learning for Gene Calling: Neural Network Integration for BBTools' CallGenes

SPEAKERS Brandon Imstepf (University of California, Merced)
AUTHORS Brandon Imstepf (University of California, Merced); Brian Bushnell (Lawrence Berkeley National Lab)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F22
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Gene calling is a critical step in genome annotation, where errors propagate into downstream biological interpretation. We present a hybrid approach that integrates machine learning into BBTools CallGenes by adding a neural advisory score to candidate open reading frames (ORFs) before dynamic-programming selection. Rather than replacing the existing gene-calling framework, the model complements it by improving ORF score discrimination while preserving the speed and structure of the original pipeline. Using curated NCBI-derived training and evaluation datasets, this neural augmentation improves overall calling accuracy, reducing false positives and false negatives relative to baseline CallGenes. These results show that lightweight neural rescoring can deliver measurable accuracy gains in practical, high-throughput prokaryotic gene annotation workflows.

PP1605 — Mathematical model of adipose cell size and number

SPEAKERS Aloïs Dauger (Laboratoire Jacques-Louis Lions)
AUTHORS Aloïs Dauger (Laboratoire Jacques-Louis Lions)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B41
TRACK Cellular and Developmental Biology

Abstract

Obesity is a major global public health issue characterized by an excessive accumulation of lipids that impairs health. White adipose tissue is responsible for this storage through its main cells, adipocytes. The expansion of this tissue relies on two mechanisms: an increase in adipocyte size (hypertrophy) and an increase in adipocyte number (hyperplasia). Although the combined effects of these mechanisms remain insufficiently understood, they play a crucial role in the development of obesity-associated metabolic diseases.

Adipocyte diameter ranges from about 20μm to 200μm, leading to large variations in cell volume. Moreover, adipocyte size distribution is typically bimodal, with two characteristic populations around 30μm and 130μm, with inter-individual variability. In this work, we introduce a system of ordinary differential equations describing adipocyte size dynamics based on lipid fluxes. The model reproduces experimental size distributions while accounting for cell variability.

The number of adipocytes is difficult to measure in vivo, and current estimation methods in organisms such as rats or humans remain approximate. Here, we propose a method to estimate adipocyte number

during weight loss. Assuming that adipocyte number remains constant during this process, the total number of cells can be inferred from variations in adipocyte size.

PP1590 — Mathematical Modelling of Antibiotic Diffusion in Bacterial Biofilms

SPEAKERS Florence WILLIAMS (UCL)
AUTHORS Florence WILLIAMS (UCL); Philip Pearce (UCL)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B38
TRACK Immunobiology & Infection

Abstract

It has been hypothesised that a mechanism for antibiotic tolerance in bacterial biofilms -populations of bacteria embedded in an extracellular matrix - is the failure of the antibiotic to penetrate throughout the biofilm. In *P. aeruginosa* colonies for example, filamentous viral phages produced by bacterial cells have been shown to provide strong protection against antibiotics. Fluorescence and light microscopy imaging reveal reduced uptake of antibiotics by cells associated with phages, suggesting that they act as a barrier to diffusion. Binding is known to affect diffusion in other biological systems, such as antibody penetration in tumours. This poster presents a mathematical model for antibiotic diffusion in biofilms with binding to phages, and extensions to the model that I aim to work on as part of my PhD, in the hope of further elucidating the mechanisms by which phages protect bacteria in biofilms.

PP1400 — Mathematical Models for the Mechanics of Soft Tissues: From Linear Elasticity to Morpho-Visco-Poroelasticity

SPEAKERS Sabia Asghar (HASSELT UNIVERSITY)
AUTHORS Etelvina Javierre (University of Zaragoza, Spain); Fred Vermolen (HASSELT UNIVERSITY); Qiyao (Aliec) Peng (Lancaster University); Sabia Asghar (HASSELT UNIVERSITY)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B17
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Biological tissues are often subjected to forces, and modeling their response is crucial in cases like tumor growth or skin contraction to improve therapies. Linear elasticity is the simplest constitutive law, allowing superposition and fundamental solutions to analyze multiple force points, as illustrated by the immersed interface method [RDB20]. We discuss this principle in terms of convergence using singularity removal [GKN19].

However, real tissues are porous and contain moisture, and microstructures change due to cellular activity. To capture this, we use a morpho-visco-poroelastic framework [Hal08], which accounts for elasticity, porosity, and microstructural evolution. This framework is analyzed for stability around equilibria [AV25]. To address spurious oscillations in numerical solutions, we provide monotonicity conditions and propose a numerical stabilization method.

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PP1527 — MICAL2 expression promotes invasion and metastasis by cell autonomous and non-cell autonomous mechanisms in Pancreatic Cells

SPEAKERS Katherine Simms
AUTHORS Andrew Lowy; Esra Karabay; Katherine Simms; Parag Katira (San Diego State University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B33
TRACK Mathematical Oncology

Abstract

Pancreatic ductal adenocarcinoma (PDAC) is a lethal cancer due to its propensity for early metastasis. MICAL (microtubule-associated monooxygenase) proteins, which are highly expressed within PDAC, directly induce actin depolymerization and indirectly cause cytoskeleton reorganization through transcription factors. Despite this knowledge, the holistic impact of MICAL2 on cytoskeleton states of cells remains unknown. Here, we have taken a multi-scale modeling approach to connect cytoskeletal gene expression programs exhibited by pancreatic cancer cells to biochemical signaling networks and extracellular matrix conditions that regulate the cellular mechanical state. Our model allows us to determine how the expression of MICAL2 impacts pancreatic cancer cell migration across soft and stiff substrates. Our preliminary results using our modeling framework indicate that MICAL2 activity confined to its direct interaction with the cytoskeletal actin network does not significantly influence cell migration. However, activation of SRF transcriptional factor downstream of MICAL2 activity towards nuclear actin significantly adds to the cytoskeletal changes and increased migration of cells in 3D environments.

PP374 — Modeling Adult Neural Stem Cells in Zebrafish With Feedback

SPEAKERS Theo Andre (Heidelberg University)
AUTHORS Theo Andre (Heidelberg University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F20
TRACK Neuroscience and Neural Systems

Abstract

The zebrafish serves as a powerful model of lifelong neurogenesis in vertebrates. Their Neural Stem Cells (NSCs) persist in specialized niches to generate neurons and glial cells, exhibiting high constitutive neurogenic activity and remarkable regenerative capacity [Lab+20; Gra+06].

Key gaps remain in understanding the molecular and cellular regulators of NSCs quiescence, activation, proliferation dynamics, migration, and fate decisions, particularly in domains like the telencephalon. Filling these gaps could inform strategies for enhancing regeneration in less neurogenic species, such as mice or humans, with implications for regenerative medicine and neurodegenerative disease therapies [Elk+25; Kal+19].

In this poster, I will present a new mechanistic modeling framework, based on experimental data, that capture the spatio-temporal regulation of NSCs located in the adult zebrafish pallium.

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PP223 — Modeling tumor dynamics under PROTAC therapy: a mathematical model for resistance analysis

SPEAKERS Cecilia Torboli (University of Udine, Italy)
AUTHORS Cecilia Torboli (University of Udine, Italy); Cinzia Soresina (University of Trento, Italy); Ján Eliaš (Boehringer Ingelheim RCV GmbH & Co KG, Vienna, Austria)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F7
TRACK Mathematical Oncology

Abstract

We present a mathematical framework to describe tumor growth under PROTAC treatment, integrating PK, PD, and tumor growth inhibition. PROTACs are a new class of drugs that promote selective degradation of oncogenic proteins [HR25]. Although preclinical studies show tumor shrinkage, relapse often occurs, suggesting resistance mechanisms [Ott+19]. The model links PROTAC-induced protein degradation to tumor growth inhibition. To account for reduced treatment efficacy, we introduce a cumulative resistance mechanism, allowing a progressive reduction of drug efficacy over repeated dosing [Pan97]. The model combines a two-compartment PK model, a protein turnover module with constant synthesis and drug-stimulated degradation [Vet+24], and classical tumor growth laws (Linear, Exponential, Logistic, Gompertz) [MJD16]. After calibration on *in-vivo* xenograft data, the model accurately

reproduces both initial regression and subsequent regrowth observed experimentally. A key contribution is the mathematical analysis. By treating the remaining protein level as a bifurcation parameter, we identify a critical threshold separating tumor eradication from relapse. Crossing this threshold induces a qualitative change in system stability, marking the transition from regression to tumor outgrowth. This analysis provides a predictive tool to anticipate resistance onset from protein dynamics, supporting adaptive therapeutic strategies aimed at delaying relapse while optimizing drug exposure.

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PP1523 — Modelling the Range Change Dynamics of Host-Macroparasite Systems under Climate Change using Reaction-Diffusion Equations

SPEAKERS Haley Morris (Department of Ecology & Evolutionary Biology, University of Toronto)

AUTHORS Haley Morris (Department of Ecology & Evolutionary Biology, University of Toronto); Maggie Kou (Department of Applied Mathematics and Theoretical Physics, University of Cambridge); Peter Molnar (Department of Ecology & Evolutionary Biology, University of Toronto; Department of Biological Sciences, University of Toronto Scarborough)

SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations

BOARD B32

TRACK Population Dynamics, Ecology & Evolution

Abstract

Macroparasites, such as helminths, mosquitoes, and ticks, can impact host fitness and population dynamics, both directly and through the diseases they transmit. Climate change is expected to lead to macroparasite range shifts as habitats become more suitable towards the poles and less suitable towards the equator. These shifting distributions can have negative consequences on host-macroparasite population persistence and survival; however, mathematical models that can accurately predict such spatiotemporal changes are lacking. Here, we develop trait-based reaction-diffusion equations to model the range change dynamics of environmentally transmitted macroparasites under climate change. Using MATLAB to numerically simulate model solutions over a discrete space-time grid, we focus our analyses on a single-host-single-macroparasite system and track how the macroparasites progress throughout various life stages, die and reproduce; traits which are temperature dependent. We outline in which climate warming scenarios macroparasites and/or their hosts are expected to undergo range contractions vs expansions, as well as which scenarios may lead to increases or decreases in disease burden and extinction risk. Our framework lays the foundation for predicting climate change impacts on the changing spatial distributions of parasites

and parasitic diseases and will help managers develop proactive plans for mitigating the subsequent impacts on human, animal and environmental health.

PP1298 — Molecular dynamics simulations of epilepsy- associated SCN1A variants for guiding pharmacological treatments

SPEAKERS Mistre Bekele (University Of Luxembourg)
AUTHORS Mistre Bekele (University Of Luxembourg)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F39
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Ion channels play an essential role in brain communication networks. Mutations in the SCN1A gene encoding the alpha subunit of the voltage-gated sodium channel NAV1.1, expressed in the central nervous system, are responsible for severe epilepsy and migraines often resistant to treatments.

We used molecular dynamic simulations to investigate consequences of four clinically relevant SCN1A variants (R1636Q, I1498M, R859H, R946C). Structural models were generated using AlphaFold and subsequently subjected to large-scale atomistic simulations using GROMACS to characterize alterations in channel dynamics.

Our simulations revealed that the gain-of-function mutations R1636Q and I1498M exhibited a more buried IFM binding pocket and altered structural dynamics in domain IV. In contrast, the loss-of-function variant R946C displayed pronounced perturbations in the selectivity filter region. The mixed-function variant R859H showed distributed conformational changes across all four channel domains.

We further simulated the effect of the antiepileptic drugs oxcarbazepine (a sodium-channel blocker), and 8DE (a selective Nav1.1). Oxcarbazepine increased the exposure of the IFM binding pocket in both gain-of-function variants, suggesting a previously unreported mechanism that may partially restore inactivation.

Together, these results show how atomistic simulations link genetic variation to channel dynamics and drug responses, supporting variant-specific therapies in channelopathies.

PP391 — Multi-scale modeling of embryonic organoids

SPEAKERS Marielle Péré (Aix Marseille Univ, INSERM, Marseille Medical Genetics, Marseille, France)
AUTHORS Anaïs Baudot (Aix Marseille Univ, INSERM, MMG, Marseille, France); Bastien Chassagnol (Aix Marseille Univ, INSERM, MMG, Marseille, France); Kenza Zeghari (Aix Marseille Univ, INSERM, MMG, Marseille, France); Marielle Péré (Aix Marseille Univ, INSERM, Marseille Medical Genetics, Marseille, France); Youssef boulaimen (Aix Marseille Univ, INSERM, MMG, Marseille, France)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F23
TRACK Multiscale and Multiphysics Modelling

Abstract

Organoids - 3D multicellular structures cultivated *in vitro* from self-aggregating stem cells - revolutionize preclinical research as an alternative to animal experimentation. But the design of *in silico* organoids is lagging behind, preventing modelling predictive control to optimize their characterization and production. The few numerical models developed mostly focus on tumoroids and do not leverage multi-omic data available nowadays. Here, we present a first digital twin of a non-cancerous organoid: a numerical pipeline parametrized using omics data, simulating organoid growth dynamic. We propose a multi-scale

approach combining Boolean Networks to model intra-cellular gene regulatory networks inserted into an Agent-Based Model ([Pon+23]) simulating cell-cell communication and micro-environment interactions. Each cell type identified from single-cell RNA-seq data is associated with its own Boolean model guiding cell agent evolution rules in the Agent-Based Model. The position of each cell and its interaction with the micro-environment regulate growth factors reaching the cell gene regulatory network, modifying the BN. Transition probabilities model cell differentiation trajectories upon multiple divisions. We apply our pipeline to gastruloids ([Arg+24]), organoids mimicking embryonic gastrulation,to identify optimal culture conditions ensuring differentiation toward the cardio-pharyngeal complex, which gives rise to the heart and pharyngeal muscles.

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PP1309 — Multiscale Simulation of Ovarian Follicle Dynamics: Linking PDE and ODE Descriptions

SPEAKERS Edilbert Christhuraj (Anhalt University of Applied Sciences)
AUTHORS Alexander Lange (Anhalt University of Applied Sciences); Claudio Iuliano (Anhalt University of Applied Sciences); Edilbert Christhuraj (Anhalt University of Applied Sciences)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B1
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Follicular phase in the ovarian development involves two key mechanisms: follicle maturation (via cellular growth and mass increase) and competition among follicles. Capturing both remains challenging. Compartmental ODE models (e.g., [HHS14]) describe maturation through discrete stages and reproduce macroscopic dynamics, but neglect follicular competition and cellular maturation. Conversely, models like [FR22] include competition but lack maturity structure. Physiologically structured population models (PSPMs) (e.g., [Mon+16]), formulated as transport PDEs, account for both maturity growth and follicular competition; numerical simulations exist (e.g., [Aym+16]). Yet, they remain computationally intensive and less directly connected to established ODE frameworks.

We introduce a class of transport equations bridging these approaches via perturbative moment closures. These PDEs allow the application of standard numerical procedures for solving them. We present numerical simulations showing how the resulting PDEs reproduce cellular development and follicular competition consistent with the macroscopic ODE models. The structured PDEs preserve the key nonlinear mechanisms governing recruitment, selection, and atresia, providing a computationally efficient framework for multiscale modelling of ovarian follicle development for different physiological and pathological conditions.

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PP1489 — Numerical Eigenvalue Analysis of an ODE Model Representing the Endocrine System

SPEAKERS Clara Horvath (TU Wien); Marie-Sophie Kohlmayer (TU Wien)
AUTHORS Andreas Körner (TU Wien); Clara Horvath (TU Wien); Marie-Sophie Kohlmayer (TU Wien)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B29
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Endocrine regulatory systems play a pivotal role in preserving physiological homeostasis through intricate hormonal feedback mechanisms. Mathematical models formulated as systems of ordinary differential equations (ODEs) provide a quantitative framework for investigating the dynamics of such regulatory processes.

In this study, we examine established ODE models describing endocrine feedback systems. These models encompass those of the hypothalamic–pituitary–thyroid[PMB14] and hypothalamic–pituitary–ovarian[GEA23] axes. In order to characterize the local stability properties of these models, a numerical analysis of the eigenvalues[HNW] of the Jacobian matrices is performed, with the matrices evaluated at physiologically relevant equilibrium states. The resulting eigenvalue spectra offer insight into the stability of equilibrium states, the associated relaxation dynamics, and the presence of stiffness in the governing equations. Furthermore, systematic exploration of parameter variations is undertaken to investigate the influence of physiological changes on the stability structure of the models and the potential induction of qualitative transitions in system behavior, such as shifts from stable regulation to oscillatory dynamics.

The analysis contributes to a more profound quantitative understanding of endocrine regulatory mechanisms and may support future efforts to connect mathematical models with clinical observations of endocrine dysfunction.

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PP288 — One item per disorder: a cross-disorder optimization framework for simultaneous prediction of six psychiatric conditions

SPEAKERS Myna Lim (Graduate School of Data Science, KAIST)
AUTHORS Daeil Jang (Innovation Center for Industrial Mathematics, National Institute for Mathematical Sciences); Jae Kyoung Kim (Department of Mathematical Sciences, KAIST); Jinhyeon Byeon (Department of Psychology, Sungshin University); Myna Lim (Graduate School of Data Science, KAIST); Seockhoon Chung (Department of Psychiatry, Asan Medical Center, University of Ulsan College of Medicine); Sooyeon Suh (Department of Psychology, Sungshin University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F10
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mental disorders are a major contributor to the global burden of disease and often manifest as overlapping symptom profiles rather than isolated diagnoses. In clinical and digital health settings, this requires simultaneous assessment of multiple psychiatric domains, including depression, anxiety, trauma-related symptoms, substance use, and suicidality. However, current screening frameworks rely on separate disorder-specific questionnaires, producing lengthy assessments that increase response burden and limit scalability for large-scale or longitudinal monitoring. From a systems perspective, this raises the question of whether multi-disorder psychiatric risk signals are distributed across many questionnaire items or concentrated within a smaller set of core symptom dimensions. Here, we investigate structural compression in multi-disorder psychiatric assessment by identifying representative symptoms across six commonly used instruments. Our framework compresses 57 questionnaire items into a six-item representation while preserving strong predictive performance across all disorders (AUROC $\approx$ 0.90–0.97). Network analysis further shows that the selected symptoms occupy central positions within the symptom interaction network, suggesting that multi-condition psychiatric risk can be captured through a compact set of symptom dimensions.

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PP1354 — Parameter estimation in infectious disease models from infected case data using metaheuristic-tuned PINN

SPEAKERS Arrianne Crystal Velasco
AUTHORS Arrianne Crystal Velasco; Eunok Jung (Konkuk University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B8
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

One crucial part of modeling infectious disease dynamics is accurate parameter estimation. This study proposes a framework using physics-informed neural networks with metaheuristic hyperparameter tuning

to estimate parameters in infectious disease models. For practical applicability, the method uses only infected case data while enforcing the governing differential equations during training. The results shows that the approach can effectively recover model parameters from limited observations.

PP1370 — Perturbing MECOM self-activation reveals multistable hematopoietic cell fate landscapes with cusp and elliptic umbilic-like catastrophes

SPEAKERS Jonathan Martinez (University of Southern California)
AUTHORS Jonathan Martinez (University of Southern California); Anupam Dey (University of Southern California); Adam MacLean (University of Southern California)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B11
TRACK Cellular and Developmental Biology

Abstract

Hematopoietic stem cells maintain the blood system. In previous work, we discovered that transitions between hematopoietic stem and multipotent progenitor cells can be controlled by mutual inhibition of MECOM and CDK6. High MECOM corresponds to a quiescent stem cell state and high CDK6 to a multipotent progenitor state. The IGF pathway, under the influence of metabolism and diet, promotes CDK6 and inhibits MECOM. To investigate stem cell-to-multipotent transitions, we modeled the MECOM-CDK6 dynamics using coupled differential equations. Bifurcation analysis revealed that the model permits tetrastability, with two stable intermediates, suggesting that early stem cell differentiation proceeds by fine-scaled transitions. Bifurcation analyses to examine the impact of MECOM self-activation on the transition landscape revealed two IGF-dependent tristable regimes with distinct geometries. At high IGF, transitions occurred through a stable intermediate via a cusp geometry. At low IGF, a very different landscape emerged, reminiscent of an elliptic umbilic-like catastrophe. On this landscape, transitions bypassed the intermediate state. Stochastic simulations and minimum action path analysis revealed that while the multipotent state was most stable at high IGF, the stem cell state was highly stable at low IGF, locking cells into a quiescent state. Overall, we show how MECOM self-activation reshapes stem cell fate decisions under the influence of diet and metabolism.

PP268 — Physics-Informed Neural Networks for Inferring Tumor Glucose–Lactate Metabolic Dynamics

SPEAKERS Shadi Vandvajdi (University Of Waterloo.Department of Electrical and Computer Engineering, Department of Applied Mathematics)
AUTHORS Bahare Shokouhian (University Of Waterloo.Department of Electrical and Computer Engineering, Department of Applied Mathematics); Hesam Abouali (University Of Waterloo.Department of Electrical and Computer Engineering); Mahla Poudineh (University Of Waterloo.Department of Electrical and Computer Engineering); Mohammad Kohandel (University Of Waterloo, Department of Applied Mathematics); Shadi Vandvajdi (University Of Waterloo.Department of Electrical and Computer Engineering, Department of Applied Mathematics); Thomas Daubon (University of Bordeaux / CNRS); Yuannong Mao (University Of Waterloo, Department of Applied Mathematics)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F8
TRACK Mathematical Oncology

Abstract

Metabolic reprogramming is a central hallmark of cancer, enabling tumor cells to sustain rapid proliferation and adapt to fluctuating nutrient availability and microenvironmental stress. In particular, the coupled dynamics of glucose consumption and lactate production provide insight into tumor metabolic

phenotypes such as the Warburg effect and metabolic switching between glycolytic and oxidative states. Mathematical models have been widely used to study these processes; however, linking experimental metabolite measurements with mechanistic models of tumor metabolism remains challenging due to limited observability, measurement noise, and uncertainty in model parameters. In this work, we present a hybrid experimental–computational framework for identifying tumor metabolic dynamics by integrating multiplexed microfluidic biosensing with physics-informed neural networks (PINNs). We developed a microfluidic bead-based aptamer sensing platform capable of quantifying glucose and lactate concentrations in cancer cell culture media, enabling time-resolved monitoring of metabolic changes during cell proliferation. Experimental data are integrated with a differential-equation model of tumor glucose–lactate metabolism using PINNs, enabling inference of metabolic parameters and reconstruction of metabolic trajectories. Application to glioblastoma and prostate cancer cell lines reveals distinct metabolic behaviors consistent with glycolytic and hybrid phenotypes.

PP1230 — PK-PD Model-Based Control Design for Anesthesia

SPEAKERS Alexander Edthofer (TU Wien)
AUTHORS Alexander Edthofer (TU Wien); Andreas Körner (TU Wien); Fabrizio Renzaglia (Universitá di Padova); Hannah Zweigart (TU Wien)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F29
TRACK Neuroscience and Neural Systems

Abstract

Propofol is a widely used intravenous anesthetic whose safe administration requires precise dosing to achieve adequate hypnosis while avoiding under- or overdosing. Pharmacokinetic–pharmacodynamic (PK-PD) models are commonly applied in target-controlled infusion systems to guide dosing based on patient characteristics and predicted effect-site concentrations.

This work compares model-based control strategies for propofol infusion using established PK–PD frameworks developed by Marsh, Schnider, and Eleveld [Ele+18]. Model predictions were evaluated against recorded Bispectral Index (BIS) values across datasets. For models including an intrinsic BIS pharmacodynamic component, predicted BIS was directly obtained from the published formulation. For models lacking an explicit BIS relationship, a validated external PD mapping was integrated to ensure comparability [Mar+13].

A sensitivity analysis examined the influence of key parameters on predicted dynamics and robustness. Additionally, open-source EEG-derived indices, Permutation Entropy and Entropy of Difference, were computed from raw EEG and assessed as alternative PD markers of anesthetic depth [Edt+25].

Finally, infusion scenarios targeting predefined hypnotic levels were simulated to analyze differences in predicted drug distribution, dosing behavior, and time to steady state, highlighting how structural model differences affect control performance and decision-support design.

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PP332 — Protein-Ligand Affinity Prediction via Topologically-Aware Graph Neural Network

SPEAKERS Perry Beamer (North Carolina State University)
AUTHORS Jingfeng Wang (North Carolina State University); Perry Beamer (North Carolina State University); Tanner Byer (North Carolina State University); Zixuan Cang (North Carolina State University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F16
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Recent computational methods predict the binding affinity between pairs of proteins and ligands, often applied to evaluate how well a candidate drug might attach to a target protein. In complex-based protein-ligand affinity prediction, binding strength is inferred from the known 3-dimensional structures of protein-ligand complexes. State-of-the-art methods employ neural networks, but certain geometric structures remain difficult to encode as learnable features. To address this challenge, we apply persistent homology cohomology (PCH)[CW20], a mathematical technique that identifies “holes” in data across spatial dimensions. In the protein-ligand complex, PCH identifies 0-dimensional holes, representing clusters of nearby atoms, 1-dimensional holes representing ring-like atomic structures, and 2-dimensional holes representing cavities in the binding pocket. To represent PCH features in a learnable format, we introduce the spatially-aware topological graph. We locate spatial representatives of each n-dimensional PCH feature, and compute the shape-preserving Wasserstein distance between each pair. These distances induce the spatially-aware topological graph, nodes represent n-dimensional holes and edges connect holes sharing a Wasserstein-neighborhood. Embedding nodes and edges with known physiochemical properties, we train a graph neural network on the PDBbind dataset[Wan+04] of known protein-ligand complexes, achieving accuracy comparable to existing high-end methods.

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PP298 — Quantifying Relative Contributions of Driving Factors of ILI Incidence Dynamics across Europe

SPEAKERS Pauline Bakker (National Institute for Public Health and the Environment (RIVM), the Netherlands)
AUTHORS Jacco Wallinga (National Institute for Public Health and the Environment (RIVM), the Netherlands); Fuminari Miura (National Institute for Public Health and the Environment (RIVM), the Netherlands); Pauline Bakker (National Institute for Public Health and the Environment (RIVM), the Netherlands)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F11
TRACK Mathematical Epidemiology

Abstract

Previous studies have identified several drivers of influenza-like illness (ILI) incidence dynamics in temperate regions, yet it remains unclear whether these associations are reproducible across countries or shaped by local context. This study quantifies the relative contributions of climate and behavioral factors to ILI incidence dynamics across multiple European countries, using weekly incidence data from 2014 to 2025 reported to ERVISS, the surveillance database maintained by ECDC. We compute the weekly effective reproduction number (R_e) from incidence data and model them as a function of explanatory variables within a regression framework. These variables include absolute humidity, holiday periods, and the Normalcy Index. The framework separates persistent associations from season-specific baseline components and reporting-related variation, and enables comparison between pre- and post-pandemic periods. We find that absolute humidity shows consistent negative associations with weekly R_e across countries, whereas behavioral factors exhibit greater heterogeneity. This framework provides a way to distinguish between generalizable and context-dependent drivers by comparing cross-regional dynamics, informing interpretation of epidemic trends during both normal and disrupted periods, such as the COVID-19 pandemic.

PP194 — Quantifying serial killing via multiscale Bayesian comparison of models for sequential NK cell responses

SPEAKERS Elephes Sung (Imperial College London)
AUTHORS Elephes Sung (Imperial College London); Rubén Perez-Carrasco (Imperial College London)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F5
TRACK Immunobiology & Infection

Abstract

Natural killer (NK) cells eliminate stressed and transformed cells by integrating signals from activating and inhibitory receptors. Although “serial killing” has been widely discussed, two quantitative gaps remain. First, it is unclear whether apparent serial killers reflect stable, intrinsic cytotoxic capacity differences, stochastic encounter dynamics, or both, making the definition ambiguous: how many kills, or how many productive stimulations, are required before a cell is more than a stochastic outlier? Second, the effect of sequential stimulation is unresolved, with repeated engagements potentially reinforcing commitment or inducing exhaustion.

We combine multiscale modelling with Bayesian inference to link intracellular activation to population-level single-cell readouts under sequential stimulation. Using flow-cytometry CD107a degranulation measurements across four consecutive stimulations, we summarise response histories as a 16-state distribution and compare mechanistic hypotheses, including heterogeneity in cytotoxic capacity, dependence on prior responses, and a highly responsive subpopulation. Bayes factors and posterior uncertainty quantification provide a principled way to adjudicate between hypotheses and to suggest informative follow-up experiments for immune surveillance and immunotherapy.

PP200 — Reconstructing High-Resolution Tau Distributions from Regional tau-PET Data Using Implicit Neural Representations

SPEAKERS Ashish Raj (University of California San Francisco)
AUTHORS Ashish Raj (University of California San Francisco); Anil Kamat (University of California San Francisco); Daren Ma (University of California San Francisco)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F6

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Introduction & Methods: Alzheimer's disease (AD) progression involves the accumulation and spread of tau pathology, measurable via tau PET imaging. Conventional analyses using regional standardized uptake value ratios (SUVRs) obscure fine-grained spatial heterogeneity and early tau propagation. We developed an Implicit Neural Representation (INR) model to reconstruct voxel-level tau distributions from regional measures. Using multimodal neuroimaging data from 61 ADNI participants, AV1451 Tau-PET scans were parcellated into 86 regions via the Desikan–Killiany atlas, and MRI-derived atrophy measures were obtained using FreeSurfer. The INR—a 16-layer fully connected network —took spatial coordinates, regional SUVRs, and z-scored atrophy as inputs to predict voxelwise SUVR. **Results:** The model achieved a mean per-subject correlation of $R = 0.891 \pm 0.111$ and a pooled correlation of $R = 0.959$ ($p < 0.01$), with unbiased, normally distributed residuals. Excluding MRI atrophy reduced performance to $R = 0.831$, underscoring its importance. Region-wise accuracy averaged $R = 0.936$, with highest fidelity in AD-critical regions including the entorhinal cortex ($R = 0.952$), left thalamus ($R = 0.974$), and inferior parietal cortex ($R = 0.958$). **Conclusion**: Our study bridges coarse regional summaries and high-resolution voxel data, offering a scalable, biologically grounded tool for early AD detection, mechanistic modeling, and individualized tau pathology assessment.

PP1649 — Reconstruction of neuromorphic dynamics from a single scalar time series using variational autoencoder and neural network map for cell culture data

SPEAKERS Nataliya Stankevich (HSE University)
AUTHORS Denis Kudryavtsev (HSE University); Pavel Kuptsov (HSE University); Nataliya Stankevich (HSE University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B47
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

One of the most appealing characteristics of neural networks is their ability for data generalization and the extraction of data features that may initially appear obscure. In the context of dynamical systems reconstruction, this aptitude could facilitate advancements in the development of models based on experimental data. We investigate the extent to which neural networks can reproduce the dynamical regimes of a system and nonlinear effects, observed for various values of parameters, when only a single scalar time series of this system is available. Created neural network models are a family of dynamical systems parameterized by a control parameter. This family exhibits behavior consistent with that of the original system. This is demonstrated by the example of a neuromorphic Hodgkin–Huxley system. The reconstruction is carried out in two steps. First, the delay-coordinate embedding vectors are constructed form the original time series and their dimension is reduced with by means of a variational autoencoder to obtain the recovered state-space vectors. Second, pairs of the recovered state-space vectors at consecutive time steps supplied with a constant value playing the role of a control parameter are used to train another neural network to make it operate as a recurrent map. This approach was applied to the experimental time series of transmembrane potential of a cell culture. The work was supported by the project "International academic cooperation" of HSE University.

PP315 — Same Calories, Different Outcomes: the Dynamics of Fasting in Cancer

SPEAKERS Andrés Méndiz Fernández (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla - La Mancha (UCLM))
AUTHORS Andrés Méndiz Fernández (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla - La Mancha (UCLM)); Juan Jiménez-Sánchez (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla - La Mancha (UCLM)); Víctor M. Pérez-García (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla - La Mancha (UCLM))

SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F14
TRACK Mathematical Oncology

Abstract

Intermittent fasting has been proposed as a potential strategy to modulate tumor progression through systemic metabolic regulation[Cli+21]. In this work, we develop a mathematical framework that integrates a model of glucose homeostasis with a tumor growth law, enabling the study of how fasting schedules may influence tumor dynamics. We first introduce a dynamical model of glucose metabolism that includes glucose, insulin, glucagon, and glycogen. The model is specifically designed to capture alternating periods of fasting and feeding through time-dependent glucose inputs. Model parameters are calibrated using data from real patients, ensuring that the simulated glucose dynamics remain physiologically realistic. Then, we generate a cohort of virtual patients from the calibrated model that reflects inter-individual variability while remaining consistent with clinically observed behavior. Each virtual metabolic profile is then coupled to a tumor growth law. We identify the virtual metabolic patient whose glucose dynamics best reproduce the effective growth rate estimated by fitting the growth law to volumetric tumor data, thereby constructing consistent metabolic–tumor patient pairs. This integrated approach enables quantitative comparison of tumor evolution under standard feeding conditions and under various intermittent fasting protocols. Overall, the framework provides a basis for investigating how metabolic variability and dietary interventions may help control tumor growth.

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PP1272 — Self-Organizing Neural Tissue Simulation Platform for Inferring and Validating Developmental Mechanisms

SPEAKERS Jaekyoung Lee
AUTHORS Daewook Kim (KAIST); Jaekyoung Lee
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F34
TRACK Cellular and Developmental Biology

Abstract

Human neurodevelopmental disorders often originate from disruptions during early cortical development. This motivates the use of brain organoids which provide an experimental platform that recapitulates key aspects of human neurogenesis. However, brain organoid exhibit substantial variability in developmental dynamics due to intrinsic cellular heterogeneity. Although mathematical models have been proposed to study organoid growth, few approaches explain how complex developmental dynamics emerge from simple cellular rules. Here, we develop an agent-based model of brain organoid development in which radial glial cells, intermediate progenitors, and neurons interact through minimal rules governing minimal rules governing proliferation, differentiation, migration, cell–cell signaling, and the temporal regulation of division modes. By systematically perturbing these rules in silico, we investigate which mechanisms are necessary to reproduce characteristic organoid growth dynamics. The model further provides a framework to explore how disruptions in progenitor behavior may lead to neurodevelopmental disorders such as ASPM-associated microcephaly.

PP1519 — Sensitivity analysis for biological differential equation models with many parameters

SPEAKERS Dilan Pathirana (Bonn Center for Mathematical Life Sciences, University of Bonn, Germany. Life and Medical Sciences (LIMES) Institute, University of Bonn, Germany.)
AUTHORS Dilan Pathirana (Bonn Center for Mathematical Life Sciences, University of Bonn, Germany. Life and Medical Sciences (LIMES) Institute, University of Bonn, Germany.)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B31
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Mathematical models of biology commonly use differential equation formulations. Certain application areas, such as signal transduction modeling or scientific machine learning, involve models that contain many parameters. Efficient training of these models requires sensitivity analysis that scales well as the number of parameters grows. Hence, adjoint sensitivity analysis (ASA) is typically employed, instead of forward sensitivity analysis (FSA) [Frö+17].

In this work, we derive a new sensitivity analysis method that has similar scaling properties to ASA but, unlike ASA, can be solved in the forward direction. This provides some computational efficiency gains in terms of memory and complexity, especially for the stiff systems that are common when modeling biology. Furthermore, higher-order sensitivities are cheaper to compute with the new method. A drawback is that, when the parameter size is small or the state size is large, then the FSA or ASA methods, respectively, can naïvely outperform the new method.

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PP1276 — Sex-Specific Temporal Dynamics of Human Sleepiness

SPEAKERS Hyunji Jeong (Korea Advanced Institute of Science and Technology)
AUTHORS Hyunji Jeong (Korea Advanced Institute of Science and Technology); Yun Min Song (Biomedical Mathematics Group, Institute for Basic Science); Jae Kyoung Kim (KAIST)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F35
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Sleepiness is commonly described by the two-process model, in which sleep pressure—a homeostatic drive that accumulates during wakefulness—and the circadian rhythm—an endogenous 24-hour physiological cycle—interact to regulate alertness. Although this framework explains the primary fluctuations in alertness, substantial inter-individual variability remains unexplained. In this presentation, we apply mathematical modeling to long-term sleepiness data to investigate temporal dynamics beyond the canonical circadian scale and examine potential sex-related differences in these patterns.

PP1197 — Structural Gradients in Excess Mortality During and After the COVID-19 Pandemic: Economic Development, Preparedness, and Cross-National Differences, 2020–2024

SPEAKERS Daeil Jang (National Institute for Mathematical Sciences)
AUTHORS Sunhwa Choi (National Institute for Mathematical Sciences); Boseung Choi (Korea University); Daeil Jang (National Institute for Mathematical Sciences); Soyoung Kim (National Institute for Mathematical Sciences)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F24
TRACK Mathematical Epidemiology

Abstract

Background Excess mortality captures the comprehensive burden of COVID-19, yet cross-national disparities remain poorly understood. We examined the impact of economic development and national preparedness on mortality outcomes across 59 countries from 2020 to 2024.

Methods Countries were stratified by IMF economic classifications. We evaluated the association between the 2021 Global Health Security (GHS) Index and excess mortality using multivariable regression and explainable machine learning (SHAP).

Findings Emerging market economies experienced significantly higher total and non-COVID excess mortality during 2020–2021. While higher GHS scores correlated with lower mortality in pooled analyses, this association attenuated upon stratification, suggesting an aggregation bias driven by structural heterogeneity. Early detection and the national risk environment were the primary drivers of resilience. Crucially, these factors operated on a functional threshold basis rather than through linear, incremental improvements.

Conclusions Pandemic mortality follows structural capacity gradients rather than transient policy variations. Preparedness indices should be interpreted as markers of broad structural capacity. Strengthening global resilience requires prioritizing foundational surveillance and institutional robustness to achieve functional readiness over uniform indicator improvements.

PP1202 — Systems-level Analysis of CAF Signaling Networks Identifies MASTL-driven Angiogenic Regulation

SPEAKERS Jae-Young Kim (Chungnam National University)
AUTHORS Hyun-Mee Park (korea institute of science and technology); Jae-Young Kim (Chungnam National University); Sung-Jin Kim (Chungnam National University); Yoon-Hee Shin (korea institute of science and technology)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F25
TRACK Systems Biology and Biochemical Networks

Abstract

Cancer-associated fibroblasts (CAFs) play a key role in tumor progression by orchestrating complex signaling interactions within the tumor microenvironment. However, the regulatory architecture of CAF-mediated signaling networks remains poorly understood at the systems level. Microtubule-associated serine/threonine kinase-like (MASTL), a mitotic kinase involved in cell cycle regulation, has been implicated in gastric cancer cell proliferation, but its role in stromal signaling is unknown. Here, we investigated whether MASTL functions as a regulatory node in CAF signaling networks. Conditioned media from gastric cancer cells induced MASTL expression in CAFs, suggesting tumor-driven modulation of stromal kinase activity. Functional assays showed that MASTL silencing impaired CAF migration and tumor-promoting properties. To characterize the underlying mechanisms, we performed quantitative secretome profiling using high-resolution LC–MS/MS. Systems-level proteomic analysis identified MASTL-dependent

changes in secreted factors. Network-based bioinformatic analyses highlighted VEGFA–VEGFR2 signaling as a central pathway. Consistently, CAF secretomes regulated by MASTL enhanced angiogenic activity in endothelial tube formation assays. These findings suggest that MASTL reshapes the CAF secretory signaling network to regulate tumor angiogenesis.

PP1423 — T-cell Motility in Confined Geometry: a Statistical Analysis and Model Comparison

SPEAKERS Lorenzo Huang (Swinburne University of Technology)
AUTHORS Federico Frascoli (Swinburne University of Technology); Lorenzo Huang (Swinburne University of Technology); Marcello Edoardo Delitala (Politecnico di Torino); Sarah Russell (Peter MacCallum Cancer Center)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B21
TRACK Mathematical Oncology

Abstract

As immune cells circulate in the body, different factors may influence their movement, such as other immune cells, target cells, and the topology of the space [JQ20]. This work investigates how confinement geometry influences T-cell motion and compares results with mathematical models in the literature. The analysis is based on experiments provided by the Swinburne team, focusing on T-cells confined in a square well in the absence of external stimuli. A statistical analysis reveals non-Brownian behaviour, with MSD exponents exceeding 1 at short timescales before dropping below 1 at longer timescales. The Time-Average Mean Square Displacement, Distributional Mean Square Displacement, and Normalised Autocorrelation Function of the velocities serve as benchmarks for evaluating models, including the Self-Avoiding Random Walk, the Beauchemin Model [TSB13], and the Generalised Lévy Walk [HBC+12]. The former two fail to reproduce the superdiffusive regime, predicting subdiffusive behaviour throughout. The Generalised Lévy Walk can match both regimes after fine-tuning, and while it lacks a mechanistic justification, it remains a valuable approximation. This work is ongoing, aiming to uncover the underlying dynamics driven by persistence and boundary interactions.

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PP1313 — The Nuclear Mechanome: A Stochastic Dynamical Characterization of Leukemic Cell Nuclei

SPEAKERS Carlos del Pozo Rojas
AUTHORS Carlos del Pozo Rojas; Diego Herraez Aguilar; Francisco Monroy; Manuel Ramirez Orellana
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B3
TRACK Cutting Edge Research Areas

Abstract

We present a computational framework for the mechanical characterization of leukemic cell nuclei integrating videomicroscopy, image-processing algorithms, and stochastic mechanics. The methodology has been developed within the Leukodomics project, aimed at constructing a digital twin for pediatric acute lymphoblastic leukemia.

Nuclear dynamics are quantified through automated tracking of intranuclear domains. Resulting trajectories are converted into time series of domain positions and analyzed using stochastic process theory. Within a generalized Langevin framework, nuclear fluctuations provide quantitative information on mechanical properties. Tracking data are integrated with confocal fluorescence microscopy, enabling biological annotation with chromatin density and differentiation markers (CD19, CD7, CD34).

Trajectory analysis in temporal and spectral domains yields four descriptor groups: spectral properties (power spectra, entropy, relaxation times); viscoelastic parameters inferred from fluctuation dynamics (effective stiffness, viscosity, diffusion); nonequilibrium activity indicators (Kullback–Leibler divergence, volatility, thermodynamic uncertainty, waiting-time variance); and spatial coherence metrics describing mechanical organization within the nucleus.

Together these descriptors define the nuclear mechanome, enabling single-cell characterization and patient-level signatures, and linking nuclear mechanical heterogeneity with functional biological variability.

PP1474 — The role of phenotypic plasticity in evolution of coexistence.

SPEAKERS Vishnu Venugopal (Bielefeld University)
AUTHORS Gaurav Baruah (Bielefeld University); Meike Wittmann (Bielefeld University); Vishnu Venugopal (Bielefeld University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B27
TRACK Population Dynamics, Ecology & Evolution

Abstract

Modern coexistence theory (MCT) has been the mainstay of coexistence research in recent years. While competition creates evolutionary changes in traits that affect coexistence, it can also induce rapid plastic changes in individuals. However, a generalised framework that integrates both plasticity and evolution into MCT is lacking. Here, we incorporated competition-induced plasticity into a generalised eco-evolutionary Lotka-Volterra two-species competition model based on quantitative genetic inheritance. We analysed how abiotic environmental breadth modulates the effect of plasticity on the evolution of coexistence. We find that for a broad environment, adaptive plasticity accelerates evolution to coexistence, whereas maladaptive plasticity hinders it. By contrast, for a narrower environment, adaptive plasticity can hinder coexistence when competition width is comparable to abiotic environmental breadth. We find that in narrower environments, the direction of evolutionary change can oppose the direction of plastic trait responses, ultimately shifting the balance between niche overlap and fitness differences and thus affecting coexistence. Our results show that plasticity can reshape the eco-evolutionary trajectories to coexistence and highlight the importance of integrating short-term plastic responses with long-term evolutionary dynamics.

PP325 — Three-delay model for glucose–insulin–glucagon regulation

SPEAKERS Michelle Ann Abhijit (Aston University, UK)
AUTHORS Ali Jabran (Aston University); Benoit Huard (Northumbra Univeristy); Maia Angelova (Aston University); Michelle Ann Abhijit (Aston University, UK)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F15

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Type-2 Diabetes Mellitus (T2DM) is characterised by dysregulated glucose levels involving complex interactions between insulin and glucagon. While delay differential equation (DDE) models of glucose-insulin dynamics reproduce ultradian oscillations [BHA20], glucagon is often omitted despite its physiological role, and its dynamics are not well understood. In T2DM, α-cell dysfunction leads to impaired glucagon suppression and hyperglycemia [UC12].

To address this gap, we extend the Cohen–Li glucose–insulin–glucagon model [CL21] by introducing a delay τ_3 representing insulin-mediated glucagon suppression, alongside delays for insulin secretion (τ_1) and hepatic glucose production (τ_2). The resulting three-delay DDE system is analysed by deriving the equilibrium and the quasi-polynomial characteristic equation governing linear stability. We use Hopf bifurcation analysis with respect to τ_3 to assess the role of glucagon feedback in oscillatory behaviour.

Our numerical simulations reproduce ultradian oscillations and support the analytical results. The analysis indicates that glucagon feedback modulates oscillatory stability while having a limited role in generating oscillations independently. This model provides a framework for studying delayed hormonal regulation and contributes to the development of digital-twin models for investigating diabetes progression.

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PP1537 — Threshold Dynamics in a Time-Delayed Logistic Model of Cell Populations

SPEAKERS Villő Glavosits (SZTE Bolyai Institute)
AUTHORS Gergely Röst (University of Szeged); Villő Glavosits (SZTE Bolyai Institute)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B34
TRACK Population Dynamics, Ecology & Evolution

Abstract

We extend the delayed logistic cell-population model of Baker and Röst. The generalized equation incorporates distributed delays expressed via both discrete and integral terms, and explicitly features the death rates of dividing and motile cells as parameters.

We first establish well-posedness, along with the nonnegativity and boundedness of biologically relevant solutions. We then derive an explicit threshold parameter that determines the stability of the zero equilibrium and the existence of a positive equilibrium. When the zero equilibrium is stable, no positive equilibrium exists and the cell population goes extinct.

When the zero equilibrium is unstable, there exists exactly one positive equilibrium, which is stable; in this system we prove uniform strong persistence of the population. Our results quantify how the death rates of dividing and motile cells, as well as the delay representing the duration of the division process, shape the system's global dynamics.

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PP1312 — Topological Data Analysis Reflects Bifurcation Structure in a Spatial SIR Agent-Based Model with a Locality Parameter

SPEAKERS Miriam Van de Water (North Carolina State University)
AUTHORS Kevin Flores (North Carolina State University); Miriam Van de Water (North Carolina State University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B2
TRACK Mathematical Epidemiology

Abstract

Agent-based models (ABMs) of disease spread are capable of capturing the complexity of host populations, such as behavioural heterogeneity that influences virus dispersal. However, the complex spatial patterns generated by high-dimensional ABM simulations introduce challenges when applying analytical techniques to interpret ABM outputs across parameter space. Here we present a computational pipeline that combines lattice-based ABMs with topological data analysis (TDA) to characterize epidemic dynamics across parameter space without requiring analytical tractability. We implement a stochastic SIR model on a lattice and introduce a locality parameter to continuously interpolate between fully local and fully global transmission. TDA summaries of ABM simulations are computed across a wide range of locality, infection, and recovery rates. Dimensionality reduction is used to embed TDA summaries in R^3 and visualize topological differences in parameter space via RGB color mapping. K-means clustering on the resulting embedding further identifies distinct groupings corresponding to qualitatively different epidemic outcomes. Under fully global transmission, the bifurcation boundaries delineated by our TDA approach recapitulate the mean-field prediction that epidemics transition from extinction to endemicity at $R_0 = 1$. As the locality parameter increases, bifurcation boundaries derived from TDA diagrams shift to correspond to a lower effective R_0.

PP1549 — User-friendly Modeling and Simulation of Multiscale Biological Processes using Morpheus

SPEAKERS Lutz Brusch (Technische Universität Dresden, Germany)

AUTHORS Jörn Starruß; Diego Jahn; Michael Kücken; Justin Bürger Michaelis; Andreas Deutsch; Lutz Brusch (Technische Universität Dresden, Germany)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B36
TRACK Multiscale and Multiphysics Modelling

Abstract

Collaborative modeling and simulation become increasingly important to study complex disease processes across molecular to organism scales. To support collaborations and construction of digital twins in a modular way, we present Morpheus (https://morpheus.gitlab.io) - an extensible open-source software framework with user-friendly GUI to develop multiscale models in a modular manner and in a standardized language, enabling model exchange, versioning and sharing through a public model repository (https://morpheus.gitlab.io/models) [Sta+14]. A numerical simulation is automatically composed by parsing the MorpheusML model definition and scheduling of solvers in the simulator. Moreover, Morpheus is SBML-compliant, it supports simulations based on experimental data, e.g. segmented cell configurations, and offers a broad set of analysis tools to extract features right during simulation. The associated FitMultiCell toolbox supports parameter estimation for stochastic Morpheus models (https://gitlab.com/fitmulticell/fit) [Ala+23]. We demonstrate the use of Morpheus for liver systems medicine and regenerative processes in tissues and organs.

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PP1386 — Wavelet-Based Machine Learning Prediction of Norovirus Detection Rates in South Korea

SPEAKERS Giphil Cho (Kangwon National University)
AUTHORS Giphil Cho (Kangwon National University)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B15
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Norovirus is a primary agent of acute gastroenteritis in all age groups, with young children under five being particularly vulnerable. Due to the virus’s pronounced seasonal behavior, forecasting its detection rate based on climatic factors is essential. To characterize the periodic relationship between climate variables and norovirus detection rates, wavelet coherence analysis was applied. The phase shift in the one-year cycle was examined to identify temporal changes in seasonal behavior. To further capture long-term nonlinear trends, generalized additive models (GAMs) were used to estimate the underlying trend in the detection rate. Based on the GAM-derived trend, the data were adjusted to reduce long-term variability and emphasize seasonal signals. The model was trained using data from January 2007 to June 2019 and tested using data from June 2019 to December 2020. Weekly detection rates were predicted using four machine learning models, incorporating wavelet-derived features that reflect time-varying seasonal characteristics. The inclusion of wavelet analysis and trend adjustment improved prediction accuracy by approximately 10–15% compared with models using the original climate variables alone.

PP1667 — What Survival Reveals and Misses: Dose-response inference in a preclinical immuno-oncology study

SPEAKERS Teresa Ackerl (University of Graz; Boehringer Ingelheim RCV GmbH & Co KG, Austria)
AUTHORS Teresa Ackerl (University of Graz; Boehringer Ingelheim RCV GmbH & Co KG, Austria)
SCHEDULE Tuesday, 14 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B48
TRACK Mathematical Oncology

Abstract

Survival is commonly used as a primary endpoint in preclinical cancer studies to assess treatment effects across different dose levels. Using a preclinical immuno-oncology study as a case study, we examine the limits of survival-based dose-response inference. Cox and AFT models indicate a statistically significant dose-response effect. However, key dose-response parameters show substantial uncertainty. Using profile-likelihood and Fisher-information methods, we show that this uncertainty stems from having too few doses in the transition region of the dose-response curve, indicating a limitation of the experimental design rather than the model itself.

Motivated by this, we use a pseudo-Bayesian design approach to examine how dose placement could be improved in future studies. To complement the survival-based analysis, we analyze longitudinal tumor trajectories. RECIST-based stratification reveals a bifurcation-like response pattern, with tumors showing either sustained regression or rapid escape growth, which is not apparent from survival analysis alone. Aggregate measures such as tumor growth area under the curve confirm modest overall treatment effects but do not explain this separation.

Overall, while survival analysis effectively detects treatment effects, its ability to characterize dose-response relationships is limited by dose placement and censoring. Moreover, key dynamic response patterns remain hidden without additional trajectory-level analyses.

PP292 — Evaluating and developing models for survival extrapolation

SPEAKERS Maiya Tebäck (Uppsala University)
AUTHORS Maiya Tebäck (Uppsala University)
SCHEDULE Thursday, 16 July 2026; 18:30–18:50; ReSoWi Building
SESSION Poster Presentations
BOARD B47
TRACK Mathematical Oncology

Abstract

Survival analysis is the study of time-to-event data. In a clinical trial, this could be the time to death, or the end of the trial, along with an indicator of whether or not death occurred. Common non- and semi-parametric survival models can only predict survival for at most as long time as a clinical trial is run. Here, we implement survival extrapolation models that extend the survival prediction beyond the trial time window. We train and test the models on a dataset consisting of approximately 10 million individuals from the Swedish general population. From these, we model survival for those who were diagnosed with cancer between 2003 and 2018, and who were at least 30 years old at the time of diagnosis. Follow-up data is available until 2023, and we have access to 10 years lookback for previous diagnoses. This dataset is randomly split into training and testing sets. We investigate how the models perform on different subpopulations.

PP1194 — Hopf Bifurcations Unravel Complex Antibody Dynamics in COVID-19 Patients

SPEAKERS Alexis Erich Almocera (University of the Philippines Mindanao)
AUTHORS Alexis Erich Almocera (University of the Philippines Mindanao); Esteban Hernandez-Vargas (University of Idaho,); Rodolfo Guadalupe Blanco Rodriguez (University of Idaho)
SCHEDULE Thursday, 16 July 2026; 18:50–19:10; ReSoWi Building
SESSION Poster Presentations
BOARD B46
TRACK Immunobiology & Infection

Abstract

The introduction of vaccines during the later phases of the 2019-2022 coronavirus pandemic (COVID-19) emphasized the importance of understanding our immune response and the dynamics of antibody production. We improved a model that previously concentrated on viral replication and T-cells to illustrate the antibody dynamics seen in COVID-19 patients. Our analysis revealed the existence of Hopf bifurcations, where changes in the virus-positive equilibrium point (VPE) stability correspond with limit cycles. These bifurcations illustrated a more complex immune response than suggested by our earlier model. When T-cell immunity is compromised, resulting in the emergence of VPE, moderate antibody levels can facilitate pathways to manage prolonged infections through an unstable VPE. Our findings show that while T-cells can eliminate the infection by achieving a stable virus-free equilibrium, antibody responses are crucial when T-cells become overwhelmed.

PP335 — Developing a digital twin of 3D vascular systems to study haemorrhagic viral diseases

SPEAKERS Jiale Miao (UCL)
AUTHORS Carina Dunlop (UCL); Jiale Miao (UCL); Paola Campagnolo (University of Surrey)
SCHEDULE Thursday, 16 July 2026; 19:10–19:30; ReSoWi Building
SESSION Poster Presentations
BOARD B45
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Disruption of the microvascular barrier is a hallmark of severe haemorrhagic diseases such as Dengue, while the mechanisms leading to acute vascular leakage and hypovolaemic shock remain incompletely understood. We are developing a cell-based digital twin of 3D microvascular organoid culture systems to investigate how endothelial cells and pericytes self-organise and how this organisation is altered in disease. In particular, we focus on the interplay between differential adhesion and cell contractility within vascular organoids. By exploring how changes in pericyte and endothelial mechanical properties affect spheroid architecture, we aim to provide mechanistic explanations for microvascular dysfunction in haemorrhagic disease, including Dengue-associated barrier failure. This digital twin complements experimental organoid studies from Dr Campagnolo's lab (University of Surrey).

PP228 — FEM Simulation of STING Pathway Activation for Glioblastoma Immunotherapy

SPEAKERS Minjong Kim (Konkuk University)
AUTHORS Donggu Lee (Konkuk University); Yangjin Kim (Konkuk University); Minjong Kim (Konkuk University)
SCHEDULE Thursday, 16 July 2026; 19:30–19:50; ReSoWi Building
SESSION Poster Presentations
BOARD B44
TRACK Mathematical Oncology

Abstract

Glioblastoma (GBM) creates an immunosuppressive microenvironment that renders current therapies largely ineffective. The STING (Stimulator of Interferon Genes) pathway has emerged as a potent target to remodel this environment and stimulate local immune responses. This study presents a Finite Element Method (FEM) framework to investigate the therapeutic impact of the STING agonist ADU-S100 on glioma growth. We implemented a coupled reaction-diffusion system on a realistic brain mesh, incorporating tissue heterogeneity by assigning distinct diffusion coefficients to gray and white matter. The model simulates the diffusion of injected ADU-S100 and subsequent intracellular signaling that triggers the production of Type I interferons (IFN-β), leading to tumor cell death. Simulation results demonstrate that while the control (PBS) group shows rapid tumor expansion along white matter tracks, the ADU-S100 treatment group exhibits significant tumor suppression driven by STING-mediated immune activation. Our results are in good agreement with experimental data (Sean Lawler group, Legorreta Cancer Center, Brown University). This work bridges the gap between computational modeling and mechanistic immunotherapy, providing a predictive tool for optimizing treatment strategies in GBM.

PP247 — A forecasting pipeline for triple-negative breast response to neoadjuvant chemotherapy: combining MRI and PK/PD-informed modeling with a mechanical constraint on proliferation.

SPEAKERS Thomas Gallagher Romero (Universidade da Coruña)
AUTHORS Guillermo Lorenzo (Universidade da Coruña); Michael R. A. Abdelmalik (Eindhoven University of Technology); Reshmi Patel (The University of Texas at Austin); Thomas E. Yankeelov (The University of Texas at Austin, US); Thomas Gallagher Romero (Universidade da Coruña)
SCHEDULE Thursday, 16 July 2026; 19:50–20:10; ReSoWi Building
SESSION Poster Presentations
BOARD B43
TRACK Mathematical Oncology

Abstract

We present an image-driven computational model to enable patient-specific forecasting of triple-negative breast cancer (TNBC) response to neoadjuvant chemotherapy (NAC). Our approach integrates longitudinal magnetic resonance imaging (MRI) data with a biologically-based mechanistic model of tumor growth and therapy response.

Building upon our previously published model that couples drug pharmacokinetics/pharmacodynamic with a reaction-diffusion equation for tumor cell density, we introduce a key enhancement: a mechanical constraint on tumor cell proliferation, motivated by recent experimental evidence. The model's reduced parameter set, identified via global sensitivity analysis, is calibrated for each patient using Gauss-Newton iterations informed by early-treatment MRI scans. Forward simulations then generate spatiotemporal forecasts of tumor response for the remainder of the NAC regimen.

Global biomarkers are computed at regular intervals. The pipeline produces volumetric exports, time-series summaries, and calibration histories to support reproducible evaluation of model fidelity and predictive performance in a unified imaging-calibration-to-forecast workflow. This work outlines our unified imaging-to-forecast-to-prediction workflow, which will be validated on a cohort of TNBC patients to assess its ability to forecast tumor dynamics and accurately identify pCR status.

PP1346 — A Time-Dependent Analysis of Protein Homeostasis Stability

SPEAKERS Kaylie Green (Student- Department of Mathematics, Florida State University, Tallahassee, FL, USA)
AUTHORS Sarah R. Fausett (Department of Biology and Marine Biology, University of North Carolina Wilmington, Wilmington, NC, USA); Stephen Kinsey (Department of Biology and Marine Biology, University of North Carolina Wilmington, Wilmington, NC, USA); Bruce Locke (Department of Chemical and Biomedical Engineering, FAMU-FSU College of Engineering, Florida State University, Tallahassee, FL, USA);

Monica K. Hurdal (Department of Mathematics, Florida State University, Tallahassee, FL, USA); Kaylie Green (Student- Department of Mathematics, Florida State University, Tallahassee, FL, USA)

SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B4
TRACK Cellular and Developmental Biology

Abstract

This research investigates the dynamic interplay between energy metabolism and protein homeostasis (proteostasis) under conditions of energetic stress. We present a two state kinetic model that tracks how proteins transition between unfolded and native conformations through synthesis, folding, unfolding, and degradation. This framework captures both transient and steady state behavior of the protein pool, allowing us to identify biologically realistic parameter ranges to establish baseline mechanisms and parameter bounds. We explore different kinetic assumptions, such as linear, nonlinear, and Michaelis–Menten forms to determine how they affect stability, with a key focus on identifying conditions under which the system becomes unstable, as an overload of unfolded proteins is expected to drive proteostatic collapse. We will also discuss how we are extending this preliminary model by introducing an aggregation state to better reflect the proteostasis mechanisms. Comparing the two and three state models will enable us to evaluate whether aggregation acts as a stabilizing or destabilizing force. We first establish proteostasis baselines before adding energetics, revealing whether instability arises internally or from energy limits. Together, these results define theoretical boundaries between resilience and collapse, providing mechanistic insight into stress responses across molecular and cellular scales.

PP1588 — Accurate noise models and their impact on parameter inference for mathematical models of collective cell behaviours

SPEAKERS Marjorie Watts (University of Oxford)
AUTHORS Carles Falco (University of Oxford); Marjorie Watts (University of Oxford); Ruth Baker (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B32
TRACK Cellular and Developmental Biology

Abstract

Advances in cellular and tissue biology have enabled robust tracking of large populations of interacting cells while simultaneously collecting detailed, multiplex molecular measurements. In such data-rich settings, mathematical models provide a powerful framework for integrating observations, capturing cellular interactions, and explaining emergent collective behaviours.

A range of approaches link partial differential equation (PDE) models with experimental data, including data-driven methods such as sparse regression and neural networks that infer governing equations and parameter values directly from data, as well as more traditional approaches that estimate parameters for a prescribed PDE model. The latter typically rely on error models that connect experimental measurements to theoretical predictions.

While considerable effort has been devoted to improving these error models, they are typically based on convenient statistical assumptions, most commonly that residuals are Gaussian and uncorrelated, which are unlikely to reflect underlying biological mechanisms. We propose a mechanistic framework for constructing error models by analysing the fluctuations that naturally arise when deriving PDE descriptions from interacting particle systems. By grounding error models in particle-level interactions, this approach aims to produce more realistic parameter estimates and reveal cell–cell interaction mechanisms that may remain hidden under conventional statistical assumptions.

PP1308 — Adaptive Finite Element method for reconstructing dielectric properties of malign melanoma in 3D

SPEAKERS Georg Kyhn (Department of Mathematical Sciences, University of Gothenburg, and Chalmers University of Technology)
AUTHORS Georg Kyhn (Department of Mathematical Sciences, University of Gothenburg, and Chalmers University of Technology); Larisa Beilina (Department of Mathematical Sciences, University of Gothenburg, and Chalmers University of Technology)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F39
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

This poster concerns the coefficient inverse problem for Maxwell's equations, related to finding the space-dependent dielectric permittivity and effective conductivity functions using backscattered time-dependent electrical field in three dimensions. The aim is to reconstruct the dielectric properties of Malignant Melanoma tissue, both shape and values. The forward problem is solved using a Domain Decomposition method developed in [BL22; BR23; LB24], where both the Finite Element Method and the Finite Difference Method is used. The inverse problem is solved using Lagrangian approach and the Conjugate Gradient Descent method, and numerical examples are performed in a homogeneous and non-homogeneous setting while adaptively refining a finite element mesh using a posteriori error estimates of [BL23].

This method was applied for breast phantoms in [BL22; BL23] and for malignant melanoma detection in a homogeneous setting in [KLB25].

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PP308 — AI-based multiomics profiling reveals complementary omics contributions to personalized prediction of cardiovascular disease

SPEAKERS Qingpeng Zhang (The University of Hong Kong)
AUTHORS Qingpeng Zhang (The University of Hong Kong)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F11
TRACK Cardiovascular Modelling

Abstract

Genomics, metabolomics, and proteomics offer complementary insights into cardiovascular disease (CVD) risk. In this published work, we introduce the CardiOmicScore, a multitask deep learning framework, to learn disease-specific proteomic (ProScore) and metabolomic (MetScore) risk scores for the six most common CVDs by profiling 2920 proteins and 168 metabolites. Experiments demonstrate that ProScore and MetScore are strong sole CVD risk predictors (C-index range: 0.69–0.82 for ProScore and 0.64–0.74 for MetScore), and can significantly enhance risk prediction across CVDs up to 15 years prior to disease onset when combined with clinical data, increasing the C-index by 0.005–0.102. These findings suggest that incorporating multiomics profiling into clinical practice can improve personalized risk assessments at early stages. CardiOmicScore also identifies important CVD-related proteins and metabolites, which represent promising data-driven pathways, calling for further external validation, to develop novel biomarkers and targeted therapies, facilitating precision medicine for primary prevention of CVDs.

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PP1657 — An individual-based model of Glioblastoma cell migration and PDGF-Driven recruitment

SPEAKERS Gerda Suchodolskaite
AUTHORS Gerda Suchodolskaite
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B37
TRACK Mathematical Oncology

Abstract

Glioblastoma is a highly aggressive primary brain tumour characterised by rapid growth and diffuse infiltration into surrounding tissue, making complete surgical removal and effective treatment extremely challenging. An important feature of glioblastoma is the behaviour of tumour cells at the invasive margin, which drives the disease progression, highlighting the need for mathematical models to understand the motility of these cells. The Proliferation-Invasion-Recruitment (PIR) partial differential equation (PDE) model was previously developed to model an experimental system in which virally transduced Platelet-Derived Growth Factor (PDGF) expressing glial progenitor cells produce PDGF, and therefore promote migration and proliferation of both transduced and already present non-transduced cells. Here, we develop an individual-based model for transduced and non-transduced glial cell migration and proliferation coupled to a PDE for PDGF dynamics. We use the model, along with experimental cell-tracking data, to explore the dynamics of chemotaxis, random motility and tissue-dependent migration of both transduced and non-transduced cells in response to the emergent PDGF gradients and grey-white matter tissue distribution. Overall, these models help explain how cell migration, PDGF driven recruitment, and tissue dependent migration may contribute to glioblastoma progression.

PP1517 — An initial stress-based Arruda-Boyce model for soft-solids and its FE implementation to study surface instabilities

SPEAKERS Srikanth Krishnakumar (BML Munjal University, Gurugram, India)
AUTHORS Parag Ravindran (Indian Institute of Technology Madras, Chennai, India); Soumya Mukherjee (Indian Institute of Technology Palakkad, Kerala, India); Srikanth Krishnakumar (BML Munjal University, Gurugram, India)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B24
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Biological processes, e.g., growth, repair, and remodeling generate residual stresses that significantly alter the biomechanical response of biomedical materials. The present work develops the constitutive relations for residually stressed Arruda–Boyce materials through a numerical inverse analysis. The model is validated using Treloar's uniaxial tension data, mapped to initially stressed reference states, and analytical solutions for boundary value problems. The developed model is implemented for finite element analysis through Abaqus using UMAT subroutine. This framework studies the buckling of residually stressed thick cylinders under external pressure. A significant influence of residual stress fields and the chain-stiffening parameter (N) is investigated on the onset of surface instabilities. Based on the distribution of the residual stress fields, circumferential wrinkles develop on the inner or outer surfaces. Increasing the chain-stiffening parameter reduces stability. We further investigate the post-buckling evolution of surface instabilities for smaller and larger values of N. Residual stress appears to interfere and influence the post-buckling evolution of instability modes, both driven by residual stress and external mechanical load. The same method can examine several pattern formation and biological materials under residual stress.

PP1487 — Analysis of the Association Between COVID-19 Prevalence and Wastewater Viral Concentrations Using Survival Dynamical System

SPEAKERS Sookyung An
AUTHORS Boseung Choi (Department of Big Data Science, Korea University Sejong Campus); Sookyung An; Sunhwa Choi (Innovation center for Industrial Mathematics, National Institute for Mathematical Science, Seongnam,Republic of Korea)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B23
TRACK Mathematical Epidemiology

Abstract

Background and objectives The transition to sentinel surveillance for SARS-CoV-2 has made it difficult to accurately determine the true scale of infections, underscoring the need for complementary indicators such as wastewater-based epidemiology (WBE). This study aims to estimate unobserved prevalence by developing a predictive framework that infers actual community-level infection scales directly from WBE data.

Methodology and results A Survival Dynamical System (SDS) model was applied to national weekly SARS-CoV-2 wastewater concentrations and sentinel data in South Korea. The SDS integrates probabilistic distributions of infection and recovery times, enabling inference of continuous epidemic trajectories and estimation of true prevalence even when effective population size is unknown. Modelled prevalence was regressed against wastewater measurements, revealing a robust positive association. A Generalized Additive Model (GAM) with an Autoregressive (AR(1)) error term further captured nonlinear dynamics and time-series autocorrelation, yielding high predictive performance.

Implications These findings demonstrate that wastewater viral concentrations serve as a reliable indicator of true infection prevalence. Integrating WBE with SDS modelling mitigates clinical underreporting,

enabling timely estimation of epidemic intensity and transmission hotspots, and providing a robust complement to traditional public health surveillance.

PP1284 — Antigenic drift and therapy resistance in CAR T-cell treatment: insights from a cellular automata model

SPEAKERS Pablo Sanz Galarreta (Mathematical Oncology Laboratory (MOLAB), Universidad de Castilla-La Mancha (UCLM))
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SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F36
TRACK Mathematical Oncology

Abstract

Chimeric antigen receptor T-cell (CAR T) therapy has demonstrated remarkable efficacy in hematological malignancies. However, resistance remains a major clinical challenge, as it can compromise treatment response and promote relapse. Among the mechanisms that may underlie resistance, loss or downregulation of the target antigen is particularly relevant, since it enables tumor cells to escape immune recognition. In this work, we develop a spatial agent-based model based on cellular automata to investigate the role of tumor phenotypic heterogeneity and antigen-dependent cytotoxicity in the emergence of resistance. The model represents interactions between tumor cells and CAR T-cells on a lattice and incorporates experimental measurements of antigen expression obtained from flow cytometry. Tumor proliferation includes a stochastic phenotypic inheritance mechanism that allows antigen levels to vary across generations. Simulations reproduce sequential co-culture experiments and show that reduced cytotoxicity against low-antigen cells promotes the clonal selection of resistant subpopulations. This process can be interpreted as a therapy-driven drift in antigen expression, analogous to resistance dynamics described in other targeted therapies, and reveals evolutionary dynamics of the antigen under treatment pressure. These findings highlight the importance of accounting for treatment-specific efficacy when designing more effective therapeutic strategies.

PP1263 — Asymptotic problems for the Keller-Segel system with density cut-off

SPEAKERS Mingyue Zhang (Vienna University of Technology)
AUTHORS Mingyue Zhang (Vienna University of Technology)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F32
TRACK Population Dynamics, Ecology & Evolution

Abstract

We consider the asymptotic problems of the Keller-Segel system with the nonlinear diffusion of porous medium type and logistic sensitivity. We classify three possible distinguished limits and reveal the relations between the Keller-Segel system and the limit systems. We prove that our model converges to three various limits as the parameters are chosen adequately: a porous medium equation when the chemotactic sensitivity is small and the chemical diffuses slowly; a hyperbolic Keller-Segel system when the cell diffusion vanishes; a surface-tension-driven free boundary problem when the chemical diffuses slowly and attracts the cells strongly. Unlike previous works, the strong convergence of the density is necessary due to the nonlinearity of the sensitivity. The mathematical methods employed vary for the three different cases, including the energy formulation, the entropy equality, the kinetic formulation, and L^1 compactness.

PP1222 — Benchmarking of the robustness of mechanistic generative models in cellular trajectory and gene regulatory network joint inference

SPEAKERS Yann Maugé (Aix-Marseille University - CRCM)
AUTHORS Elias Ventre (INRIA Centre d'Université Côte d'Azur); Yann Maugé (Aix-Marseille University - CRCM)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F29
TRACK Systems Biology and Biochemical Networks

Abstract

Cellular models based on single-cell RNAseq data have emerged as a common tool for a system-level exploration of cell mechanisms, especially relevant for tasks such as gene regulatory network (GRN) inference or cell trajectory prediction. Some early mechanistic models allow dataset simulation from GRNs, thus using underlying biological knowledge and offering greater interpretability. However, these models cannot be calibrated from scRNAseq data to formulate predictions. In contrast, multiple generative models fitted on temporal snapshots of scRNAseq data can predict cell trajectories, but most until recently were based on non-mechanistic models lacking explicit GRN formulation. Yet since a GRN encodes the regulatory interactions driving gene expression dynamics, GRN structure and cell trajectories are two facets of the same process. Addressing this, new generative models calibrate a GRN from temporal data and use it to constrain trajectory inference, exploiting this coupling so that both tasks reinforce each other. In our recent work, we evaluate this joint modeling performance gain by benchmarking recent GRN-driven generative models (including [Zha24b; Ber+25; Ish+23; Zha24a; Rim+25]) against state-of-the-art methods performing these tasks separately. We evaluate their ability to reconstruct gene networks from simulated and curated datasets, assess the biological coherence of reconstructed trajectories, predict cell states at held-out timepoints, and generalize to unseen perturbations.

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PP309 — Beyond Markovian Assumptions: A History-Dependent Framework for Epidemic Inference

SPEAKERS Sunhwa Choi (National Institute for Mathematical Sciences)
AUTHORS Sunhwa Choi (National Institute for Mathematical Sciences)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F12
TRACK Mathematical Epidemiology

Abstract

Epidemic models commonly assume Markovian disease progression, in which transitions between infection states occur at constant, memoryless rates. However, real infectious diseases are inherently history-dependent: the probability of becoming infectious depends on time since exposure, and latent and infectious periods rarely follow exponential distributions. This mismatch can bias epidemic inference, leading to overestimation of key transmission parameters and mischaracterization of hidden initial conditions.

Here, we present two data-driven, history-dependent frameworks for more accurate epidemic inference. The first incorporates gamma-distributed latent and infectious periods to improve estimation of epidemiological parameters, including the reproduction number, directly from reported case data. The second reconstructs hidden exposure histories to more accurately infer the initial exposed population, even under noisy or rapidly changing outbreak conditions. Together, these approaches move epidemic inference beyond conventional Markovian assumptions, reduce systematic bias in both parameter and initial-condition estimation, and provide a more reliable foundation for epidemic analysis and prediction.

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PP396 — Bifurcation analysis of a distributed-delay Wilson–Cowan model for sleep spindle dynamics

SPEAKERS Anca Stanoev (West University of Timisoara)
AUTHORS Anca Rădulescu (State University of New York at New Paltz); Anca Stanoev (West University of Timisoara); Eva Kaslik (West University of Timisoara)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F23
TRACK Neuroscience and Neural Systems

Abstract

Sleep spindles are hallmark thalamo-cortical oscillations of non-REM sleep implicated in memory processing and emotion regulation. We develop and analyze a Wilson–Cowan mean-field model of a corticothalamo-reticular circuit comprising cortical pyramidal and inhibitory populations, thalamic relay cells, and reticular thalamic populations, to capture spindle-like dynamics at the population level. Particular emphasis is placed on neuroprocessing times within the circuit, modeled through weak Gamma-distributed delayed feedback. We study the system with and without distributed delays to determine how temporal effects interact with coupling architecture to reshape the underlying dynamics. Using stability and bifurcation analysis, we characterize the impact of delayed feedback and connectivity strengths on equilibria, oscillatory regimes, and transitions into and out of spindle-like activity. Our results show that timing acts jointly with connectivity to govern the emergence and modulation of spindle-generating dynamics. The model provides a mathematically tractable framework for investigating mechanisms underlying sleep spindle activity in thalamo-cortical networks.

PP1275 — Bridging Reaction–Diffusion Models and Empirical Data: Reparameterization for Transporter Kinetics

SPEAKERS Se Jun Ahn (Graduate School of Data Science, KAIST, Daejeon, Republic of Korea)
AUTHORS Hyeong Jun Jang (Graduate School of AI for Math, KAIST, Daejeon, Republic of Korea); Jae Kyoung Kim (Department of Mathematical Sciences, KAIST, Daejeon, Republic of Korea); Junghyun Lee (Department of Pharmacology, College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea); Se Jun Ahn (Graduate School of Data Science, KAIST, Daejeon, Republic of Korea); Suein Choi (Department of Pharmacology, College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F35
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Transporter-mediated drug delivery is a crucial factor in pharmacokinetic and pharmacodynamic (PK/PD) studies. Transporter kinetics are typically evaluated using the Michaelis–Menten (MM) model, which estimates the maximum reaction rate($V_{\max}$) and the Michaelis constant(K_M). However, the MM model assumes a well-mixed environment, whereas transporters operate in localized spatial settings, which can lead to estimation inaccuracies. While previous studies addressed this spatial discrepancy, they introduced an additional spatial parameter that is difficult to determine experimentally. To overcome this practical limitation, we reformulated the reaction–diffusion equations in terms of more accessible parameters. Furthermore, we rigorously evaluated the accuracy and practical identifiability of these transformed parameters with real experimental data. By transitioning to practically measurable variables, this study bridges the gap between complex spatial models and empirical laboratory data, offering a robust and experimentally interpretable framework for analyzing transporter kinetics.

PP261 — Catalyst: Fast and flexible modeling of reaction networks

SPEAKERS Torkel Loman (University of Oxford)
AUTHORS Torkel Loman (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F3
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Chemical reaction networks (CRNs) are a commonly used model type in biology and chemistry (with applications in e.g. systems biology, pharmacology, and epidemiology). Here, we demonstrate Catalyst.jl, a flexible and feature-filled Julia library for the creation, simulation, and analysis of CRN models. Models can be created both using the Catalyst DSL (which enables the implementation of CRNs in their native reaction format), or built programmatically. Next, they can be simulated using ODE, SDE, and jump process (e.g. Gillespie simulation) approaches. Internally, models are represented symbolically, enabling e.g. automatic symbolic simplifications and Jacobian constructions to improve simulation performance. Finally, Catalyst's internal CRN model representation enables it to compose with many additional modelling packages. This facilitates features such as steady state computations, bifurcation analysis, identifiability analysis, and parameter fitting.

PP314 — Challenging the standard protocol: Mechanistic mathematical model for ^{177}Lu-PSMA therapy optimization

SPEAKERS Silvia Bordel-Vozmediano (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha)

AUTHORS Silvia Bordel-Vozmediano (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha); Matteo Italia (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain); José García Otero (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain); Gabriel Calvo (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain); Víctor M. Pérez-García (Mathematical Oncology Laboratory (MOLAB), University of Castilla-La Mancha, Spain)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F13
TRACK Mathematical Oncology

Abstract

Radiopharmaceutical therapy with ^{177}Lu–PSMA has emerged as an effective treatment for metastatic prostate cancer, yet current clinical protocols rely on empirically fixed and non-personalized schedules. A mechanistic mathematical model based on ordinary differential equations is introduced to integrate tumor growth dynamics, radiation damage and organs pharmacokinetics. Radiopharmaceutical activity in tumor and organs at risk is described through compartment-specific effective decay rates combining physical and biological clearance, while injected activity is distributed according to uptake-weighted mass fractions. The toxicity is quantified using biologically effective dose thresholds, enabling a quantitative assessment of efficacy–toxicity trade-offs [Ita+25]. Virtual patient cohorts generated through stochastic sampling of biologically grounded parameter ranges allow in silico trials. Model predictions reproduce published dosimetry and survival outcomes from independent clinical studies, showing good agreement in absorbed doses and Kaplan–Meier survival distributions. Exploration of treatment schedules reveals a clear efficacy–toxicity landscape: consolidated regimens with fewer, higher-activity injections increase median overall survival but raise renal toxicity, whereas excessive cycle delays markedly reduce therapeutic efficacy. Notably, a nine-week cycle preserves survival comparable to the standard six-week protocol while significantly reducing toxicity.

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PP1447 — Clonal dynamics deviate from neutral drift in zebrafish spermatogenesis

SPEAKERS Connor Shrader (University of Utah)
AUTHORS Andrea Sposato (University of Utah); Connor Shrader (University of Utah); Drew Shapiro (University of Utah); Frederick Adler (University of Utah); James Gagnon (University of Utah); Jenna Weber (University of Utah); Kimberly Truong (University of Utah)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B17
TRACK Cellular and Developmental Biology

Abstract

An active pool of spermatogonial stem cells (SSCs) maintain male fertility. The effects of aging on the SSC pool remains poorly understood. Here, we used in vivo CRISPR barcoding to label zebrafish SSCs to study how SSC clones contribute to sperm production. We sampled sperm from barcoded zebrafish every month over their entire fertile lifespan. Sequencing of these sperm samples suggests that only a fraction of embryonic germ cells ever participate in sperm production. Moreover, we find that the relative contributions of each clone shifts dramatically over time. To understand the basis of this temporal variation, we developed a mathematical model of stem cell clonal dynamics and serial sperm sampling. Our model demonstrates that most SSC clones show evidence of population drift and that the clonal dynamics we observe do not conform to a neutral model where clones have equal fitness and their dynamics are driven solely by random chance. While past literature argues that individual clones drift neutrally in the testis, our results suggest that clonal interactions across the entire organ are more complex. Overall, our

findings reveal the in vivo consequences of SSC heterogeneity and provide new insight into the mechanisms linking genetic variation, aging, and evolutionary dynamics in the testis stem cell niche.

PP1402 — Closed-Loop Control of Anesthesia with Propofol Using Reinforcement Learning on Clinical Data

SPEAKERS Madlen Martinek
AUTHORS Andreas Körner (TU Wien); Daniel Pasterk; Madlen Martinek
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B11
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Advances in computational methods are rapidly transforming modern medicine, particularly in safety-critical domains such as anesthesia. In these settings, clinicians must make complex and time-critical decisions, motivating the development of automated and data-driven support systems. A recent study demonstrated the feasibility of reinforcement learning (RL) based propofol infusion control using a clinical dataset [Yun+23]. This work implements a learning framework based on the methodology proposed in the original study, providing an independent validation of the approach. To explore the generalizability of the method, the framework is subsequently evaluated on an additional clinical dataset, assessing its robustness beyond the original experimental constraints. To provide further insight into the model, patient demographic data and administered drug doses are combined with pharmacokinetic–pharmacodynamic models for bispectral index determination. These data serve as the training input for the RL agent, which learns to control propofol infusion automatically, incorporating the effects of manual remifentanil administration by anesthetist. The framework is applied to an independent clinical dataset for evaluation. The evaluations allow assessment of policy generalization, robustness across patients, and interactions with partially manual drug administration, while the study as a whole contributes to enhancing transparency and reproducibility in automated anesthesia systems.

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PP1203 — Codifying the rules of microbial community physiology for biotechnology designs: fighting natural biochemistry with its own sword

SPEAKERS Pranas Grigaitis (Karlsruhe Institute of Technology)
AUTHORS Pranas Grigaitis (Karlsruhe Institute of Technology)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F25
TRACK Systems Biology and Biochemical Networks

Abstract

We are still actively searching for principles and recipes to design scalable, robust, efficient, and modular microbial bioproduction processes. Engineering natural microbes, however, also means balancing between the interests of the microbe, hardwired in natural biochemistry, and the biotechnologist who aims to

upcycle poorly accessible, abundant feedstocks. Could we unlock alternative feedstocks for synthetic biotechnology by rational engineering of microbial communities? I argue that we should seek inspiration from metabolism(s) from natural environments, and codify their physiology, as we did with "cellular economics" of single microbes in isolation. For this we propose to blend multiple meta-omics data with mechanistic, stoichiometric modeling of metabolic networks. We are continuously developing a library of metabolic "jigsaw parts" by reconstructing metabolic networks from metagenome-assembled genomes of environmental microcosms. In parallel, we use metatranscriptomics data generated from the same samples to identify mutually compatible jigsaw parts, i.e. metabolic networks which are co-expressed in multiple microorganisms forming natural microbial communities. In the long run, we aim to design a "rulebook" of synthetic metabolic networks, based on the insights of our current data-driven approach.

PP1599 — Cooperative motility emerges in crowds of T cells but not neutrophils

SPEAKERS Inge Wortel (Computational Immunology, Data Science, Radboud University, Nijmegen, the Netherlands)
AUTHORS Inge Wortel (Computational Immunology, Data Science, Radboud University, Nijmegen, the Netherlands); Jérémy Postat; Mihaela Mihaylova; Mauricio Merino; Aanya Bhagrath; Maryl Harris; Lin Wouters; Daniel Parisi; Judith Mandl; Johannes Textor
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B33
TRACK Multiscale and Multiphysics Modelling

Abstract

T cells are among the most motile immune cells in the body, and their migration into and within tissues is key to their function. Remarkably, T cells can maintain their motility even in highly crowded environments like the densely packed T cell areas of lymphoid organs, but how they do so remains incompletely understood. In this work[Wor+24], we use microfluidic devices and Cellular Potts Models[NTB15][Wor+21] to study T cells from a crowd dynamics perspective *in vitro* and *in silico*, focusing on a hallmark scenario that has proven instrumental in characterizing other crowded systems such as pedestrians and ants: single-lane traffic. Unexpectedly, T cells in narrow, straight microchannels synchronize their speeds and form stable, motile trains. We show that this behavior can be explained by a preference of T cells to maintain contact with each other after collisions, and an ability of faster T cells to "push" slower ones. We demonstrate that this behavior does not extend to all immune cells; neutrophils in the same settings slow down with increasing cell density. Cooperative motion may benefit T cell motility in difficult and crowded tissue environments, ultimately preventing jams that impair motion in other crowded systems.

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PP385 — Decoding Cis-Regulatory Information Underlying Gene Expression in Forest Trees Using Field Transcriptomes from Natural Environments

SPEAKERS Shuichi Kudo (Kyushu University)
AUTHORS Akiko Satake (Kyushu University); Koji Noshita (Kyushu University); Shuichi Kudo (Kyushu University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F21
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Regulation of gene expression is essential for organisms to survive and reproduce in fluctuating environments. Machine learning has increasingly been used to understand relationships between non-coding sequences and gene expression. However, most studies rely on data from a limited set of model organisms under controlled laboratory conditions, restricting our understanding of gene regulation in natural environments. Here, we applied machine learning-based cis-regulatory motif detection to two species of Fagaceae, a family of dominant forest trees, using field transcriptome data collected across a two-year seasonal cycle in leaf and bud tissues. First, we trained a convolutional neural network (CNN) to predict the presence or absence of gene expression from 2-kb flanking sequences. The model achieved high predictive accuracy (ROC-AUC > 0.8) for most genes, except those related to photosynthesis, suggesting distinct regulatory mechanisms in these pathways. We then extracted DNA motifs based on importance scores and identified motifs corresponding to several known transcription factors, including the BBR/BPC family, a transcriptional repressor in maize and Arabidopsis. These results suggest that binding sites of transcriptional repressors known across a wide range of plants may also contribute to gene expression in forest trees and provide candidates for future experimental validation.

PP1217 — Deconstructing Pathogen Transmission: Cumulative Edge Flux in Empirical Temporal Networks

SPEAKERS Nathan Millen (University of Manchester)
AUTHORS Nathan Millen (University of Manchester)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F28
TRACK Mathematical Epidemiology

Abstract

Traditional analyses of epidemic spreading on networks often rely on spectral properties, steady-state approximations, or aggregate measures of final outbreak size. While these metrics provide essential insights into threshold behaviour, they frequently obscure the granular mechanistic processes by which individual interactions and temporal variations drive population-level dynamics. This work utilizes the Generalized Epidemic Mean-Field (GEMF) framework—an N-intertwined deterministic approximation—to extend the Susceptible-Infected-Removed (SIR) model across structured populations. By mapping the stochastic Markovian process onto a system of 3N nonlinear differential equations, we investigate the spreading process within both stationary and temporal empirical contact networks. We extend the standard GEMF formulation to capture the cumulative edge flux: a time-integrated measure of the probability-weighted infection transmission across every discrete contact in the network. This approach allows for a precise decomposition of the "force of infection," identifying how specific individuals and their localized contact patterns contribute to the overall trajectory of a disease. This granular methodology provides a rigorous framework for exploring the interplay between individual behaviour and population-scale spreading, offering new avenues for the design of targeted, behaviour-aware intervention strategies.

PP1692 — Decoupling density- and adhesion-driven rigidity transitions in embryonic tissues

SPEAKERS Elisa Floris (University of Graz)

AUTHORS Laura Rustarazo-Calvo (European Molecular Biology Laboratory Heidelberg); Cristina Pallares-Cartes (European Molecular Biology Laboratory Heidelberg); Adrián Aguirre-Tamaral (University of Graz); Elisa Floris (University of Graz); Maximilian Hingerl (European Molecular Biology Laboratory Heidelberg); Camilla Autorino (European Molecular Biology Laboratory Heidelberg); Arif Ul Maula Khan (European Molecular Biology Laboratory Heidelberg); Bernat Corominas-Murtra (University of Graz); Nicoletta I. Petridou (European Molecular Biology Laboratory Heidelberg)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B42
TRACK Cellular and Developmental Biology

Abstract

Organism development occurs through sequences of astonishingly precise spatiotemporal changes, along which embryonic tissues switch between fluid- and solid-like states, arising from non-linear interactions among cellular constituents. In this context, tissue fluidization observed *in vivo* in zebrafish embryos was interpreted, within the framework of rigidity percolation, as a rigid-to-floppy transition occurring at a critical point in cell connectivity. The microscopic parameters triggering these changes in cell connectivity, and whether they are interdependent, remain unclear. A density-driven rigidity transition is observed in *wildtype* embryos at a critical cell fraction, ϕ_c, marked by the emergence of a Giant Rigid Cluster (GRC), in which cell movements are hindered. Using genetic manipulations, we uncoupled the tissue material response from the jamming behaviour, generating living tissues displaying solid-like or fluid-like properties, although being unjammed or jammed, respectively. We explain this apparently paradoxical behaviour by analytically deriving a critical point in the relative surface tension, α_c, beyond which a floppy motif of soft, adhesive spheres spontaneously rigidifies. Simulations of arbitrary arrays of cells, whose energy is described by a soap-bubble-like Hamiltonian, show a sharp transition in the GRC size at the predicted α_c; a transition also observed in real embryonic tissues with cell fraction below the critical jamming fraction $\phi < \phi_c$. Embryonic tissues are thus characterized by a phase diagram organized around a double critical point (ϕ_c, α_c): when uncoupled from changes in cell fraction ϕ, the relative surface tension α is the main control parameter of the tissue material properties. Furthermore, at the critical point α_c at which adhesion-driven rigidification takes place, a sudden formation of tricellular contacts is observed, dictating an abrupt tissue reorganization and defining a transition from a non-confluent to a fully confluent regime. Our theoretical and experimental results show how basic physical parameters have far-reaching roles in tissue architecture, properties, and functions.

PP1358 — Dynamical paths to depolarization block and back

SPEAKERS Marisa Saggio (Aix-Marseille University, Institut de Neurosciences des Systèmes (INS, UMR1106))
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SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B6
TRACK Neuroscience and Neural Systems

Abstract

Depolarization block (DB) occurs when strong depolarization prevents neurons from generating action potentials. It is critically involved in several brain disorders, including epilepsy, migraine, and stroke, but also serves physiological roles, for example in odor encoding. Despite the relevance of DB for brain function and dysfunction, relatively few modelling studies directly address its underlying mechanisms.

At the same time, DB states systematically emerge in models of neuronal spiking, bursting, or seizure activity when parameters are varied, suggesting that DB is a robust component of neuronal dynamics.

Here we exploit a rare dataset of whole-cell patch-clamp recordings that reveals a progression of transition patterns from baseline excitability to DB and back. Using dynamical systems theory, we unfold the minimal dynamical structures that enable Type I excitability as in the recorded pyramidal neurons. Coupling this minimal neuron model to slow variables representing collective homeostatic processes naturally reproduces the observed progression. The framework explains DB ubiquity in neuron models and predicts distinct dynamical types of DB with different experimental signatures and responses to perturbations. This frames depolarization block as a nuanced dynamical phenomenon and provides constraints for both experimental and modelling design.

PP1337 — Dynamics and stability of microbial communities under community-level selection

SPEAKERS Sofia Almirante (Umeå University)
AUTHORS Eric Libby (Umeå University); Sofia Almirante (Umeå University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B3
TRACK Population Dynamics, Ecology & Evolution

Abstract

There is growing interest in designing microbial communities that perform collective functions, with potential applications ranging from human health to pollutant degradation and crop production. However, attempts to artificially select communities have reported limited success, with modest improvements in performance and a lack of ecological or evolutionary stability.

Here, we approach this challenge from an organismal perspective. We propose that microbial communities can be engineered to behave more like integrated organisms by imposing a coordinated stress response as a required feature. We develop a theoretical framework to describe microbial populations interacting under selection for community-level traits. Using differential equation models, we analyze the stability and dynamics of multi-species communities subjected to perturbations.

We identify common sources of instability and derive a set of minimal trait requirements for engineering communities that maintain their function. In particular, stress responses at the community level can stabilize functional outputs despite environmental fluctuations or shifts in species abundances. Our results suggest potential design principles for the construction of microbial communities with more stable and reliable collective behavior.

PP291 — Epidemiological model under vaccination regime and non-permanent immunity driven by time-changed Lévy noise

SPEAKERS Nenad Šuvak (School of Applied Mathematics and Informatics, University of Osijek)
AUTHORS Nenad Šuvak (School of Applied Mathematics and Informatics, University of Osijek)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B7
TRACK Mathematical Epidemiology

Abstract

Stochastic version of Susceptible-Infected-Recovered-Vaccinated (SIRV) epidemiological model is observed. The model is constructed from the ordinary differential SIRV model by introducing the additive time-changed Levy noise in the transmission coeffcient. The noise is constructed in terms of a conditional Brownian motion and a doubly stochastic Poisson random field. The structure of these noises can be strongly related to the corresponding time-changed Brownian motion and the time-changed Poisson random measure, when the time-change is independent of the Brownian motion and Poisson field. The existence of a unique global positive solution of this system of stochastic differential equations is proved. The conditions under which the infectious disease in the population of non-constant size extincts and persist are given in terms of the parameters of the model and the time-change processes from the noise.

PP1666 — Estimating the Distribution of Methylation Entropy in Genomic Data

SPEAKERS Monika Kurpas (Department of Systems Biology and Engineering, Silesian University of Technology, Gliwice, Poland)
AUTHORS Emmanuel Asante (Department of Statistics, Rice University, Houston, TX, USA); Monika Kurpas (Department of Systems Biology and Engineering, Silesian University of Technology, Gliwice, Poland); Meng Li (Department of Statistics and Ken Kennedy Institute, Rice University, Houston, TX, USA); Marek Kimmel (Department of Statistics and Ken Kennedy Institute, Rice University, Houston, TX, USA; Department of Systems Biology and Engineering, Silesian University of Technology, Gliwice, Poland); Peter Van Loo (Department of Genetics, Division of Discovery Science and Department of Genomic Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX, USA); Huw Ogilvie (The University of Texas MD Anderson Cancer Center, Houston, TX, USA)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B40
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

DNA methylation is an epigenetic modification in which a methyl group is added to cytosine within CpG dinucleotides. CpG-rich regions are often found in gene promoters and other regulatory elements. Methylation at these loci is a stable, heritable mark that influences transcription, chromatin state, and genome stability [Bir02]. Its dysregulation has been linked to several diseases, including cancer [BJ16]. At the level of a single DNA molecule, each CpG site is either methylated or unmethylated. Mean methylation levels describe the overall tendency of a locus, but they do not distinguish between different stochastic configurations. We use Shannon entropy to quantify methylation heterogeneity and develop a simulation framework to explore the entropy landscape [Asa26]. Probability vectors are sampled from a symmetric Dirichlet prior on the simplex and conditioned on a fixed mean methylation level. The accepted distributions are then used to compute entropy. By simulating approximately 10 million reads per setting and comparing the results with whole-genome nanopore calls from human blood, we found that entropy is lowest at extreme mean values and peaks near 0.5. Increasing the number of CpG sites expands the space of possible methylation patterns and increases entropy, while the Dirichlet concentration parameter controls dispersion. Overall, Shannon entropy is a sensitive measure of epigenetic heterogeneity and simple Dirichlet-based models reproduce features observed in empirical data.

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PP1267 — Fluctuating Trait-Switching Opportunities Generate Bistable Cultural Dynamics in the Absence of Conformist Bias

SPEAKERS Rinto Yoshizaki (Kyushu University)
AUTHORS Motohide Seki (Kyushu University); Rinto Yoshizaki (Kyushu University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F33
TRACK Population Dynamics, Ecology & Evolution

Abstract

In cultural evolution, the frequency of a cultural trait often exhibits a nonlinear relationship with its frequency in the previous time step: a trait held by the majority tends to become increasingly common even in the absence of any advantage over the minority trait. This pattern has been attributed to conformist bias, which has been shown to generate a sigmoidal relationship between past and present frequencies. However, recent studies suggest that other mechanisms, including sampling biases, can produce similar patterns. Here, we identify a novel mechanism: fluctuations in the proportion of individuals allowed to switch traits. Consider the discrete-time dynamics of two cultural traits, A and B. Let p_t denote the frequency of trait-A carriers at time t. At each time step, a fraction k_t^+ of trait-A carriers and a fraction k_t^- of trait-B carriers update their traits based on their observations of the frequencies of the two traits. The values of (k_t^+, k_t^-) change periodically with period n. For $n = 2$, we analytically obtain the following results. Only the trivial equilibria, $p = 0$ and $p = 1$, can be locally stable. The sigmoidal relationship between p_t and p_{t-n} can arise when the changes in p_t at two consecutive steps occur in opposite directions; in this case, an unstable internal equilibrium exists, and $p = 0$ and $p = 1$ are simultaneously locally stable. We confirmed analytically or numerically that these properties also hold for $n \geq 3$.

PP1295 — Fluctuation analysis of neural stem cell dynamics

SPEAKERS Filip Klawe (Heidelberg University)
AUTHORS Filip Klawe (Heidelberg University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F37
TRACK Cellular and Developmental Biology

Abstract

Inter-individual variability in biological systems is often considered experimental noise, yet its temporal structure can contain valuable information about underlying regulatory mechanisms. Here, we present a stochastic framework that uses fluctuations in cell population dynamics to infer regulatory processes that remain hidden when analyzing only mean behavior. Focusing on adult neural stem cells, we develop a stochastic model describing transitions between quiescent and active states and derive a diffusion approximation capturing the dynamics of both mean and variance of observable quantities.

Applying this approach to longitudinal data from wild-type and interferon-receptor knockout mice reveals that different regulatory mechanisms can produce similar average population dynamics while generating distinct fluctuation patterns. By jointly fitting mean and variance trajectories, we identify proliferation-rate regulation as the main determinant of fluctuation amplitude, whereas activation and self-renewal primarily control mean dynamics and long-term population outcomes.

These results demonstrate that stochastic variability is not merely noise but a rich source of mechanistic information that complements traditional analyses based solely on average dynamics.

This is join work with Anna Marciniak-Czochra, Diana-Patricia Danciu and Ren-Yi Wang.

PP1467 — From Flickering to Dynamical States: A Data-Driven Analysis of Nonequilibrium Dynamics in Red Blood Cells

SPEAKERS Irene Alférez Gómez (Universidad Francisco de Vitoria)
AUTHORS Diego Herráez Aguilar (Universidad Francisco de Vitoria); Inmaculada Mora Jiménez (Universidad Rey Juan Carlos); Irene Alférez Gómez (Universidad Francisco de Vitoria)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B19
TRACK Cutting Edge Research Areas

Abstract

Active biological systems often operate far from thermodynamic equilibrium, leading to violations of detailed balance and irreversible stochastic dynamics. Red blood cells provide a model system to study nonequilibrium phenomena through membrane flickering, spontaneous fluctuations driven by thermal noise and active intracellular processes. The statistics of these fluctuations reflect the mechanical and dynamical state of the membrane. The aim of this work is to determine whether the observed dynamics depart from thermodynamic equilibrium. Classical approaches detect nonequilibrium behaviour by estimating entropy production or temporal irreversibility from stochastic trajectories. However, these methods require an explicit discretization of the state space and reliable statistical estimation, which is challenging in noisy experimental data. We propose a data-driven machine-learning framework that learns tokenized stochastic time-series embeddings, transforming continuous trajectories into sequences of discrete latent states (tokens) through vector quantization. This representation provides a data-driven coarse-graining of the dynamics. Token distributions are analysed using a Self-Organizing Map, yielding a topological embedding of the learned dynamical states. The method is validated on Brownian dynamics simulations and applied to erythrocyte membrane fluctuations, enabling identification of dynamical states associated with nonequilibrium behaviour.

PP343 — Global Dynamics of Two-prey-one-predator Models with Direct Competition between the Prey : The Mathematical Parts

SPEAKERS Tinghui Yang (the department of applied mathematics and data science, the college of science, Tamkang University)
AUTHORS MING CHIEH HUANG (the department of applied mathematics and data science,the college of science,TamKang UNIVERSITY); Tinghui Yang (the department of applied mathematics and data science, the college of science, Tamkang University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F17
TRACK Population Dynamics, Ecology & Evolution

Abstract

In this work, we revisit classical three-dimensional Lotka-Volterra two- prey-one-predator models with direct prey competition. Employing a rescaling technique, we reduce the system to a simpler model with eight key parameters. With minimal assumptions, all parameters are grouped into four generic categories with a total of nine subcases, which are further refined into 56 sub-items to explore diverse dynamical behaviors, including the globally asymptotically stability, bistability of boundary equilibria, the existence and stability of the positive equilibrium, the existence of periodic solutions, and the complex dynamics of some solutions. Many results are novel; most findings are shown analytically; some are verified numerically; and some are open.

PP339 — Global Dynamics of Two-prey-one-predator Models with Direct Competition between the Prey:The Biological parts

SPEAKERS MING CHIEH HUANG (the department of applied mathematics and data science,the college of science,TamKang UNIVERSITY)
AUTHORS MING CHIEH HUANG (the department of applied mathematics and data science,the college of science,TamKang UNIVERSITY); Tinghui Yang (the department of applied mathematics and data science, the college of science, Tamkang University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F15
TRACK Population Dynamics, Ecology & Evolution

Abstract

In this work, we revisit classical three-dimensional Lotka-Volterra two prey-one-predator models with direct prey competition. Employing a rescaling technique, we reduce the system to a simpler model with eight key parameters.With minimal assumptions, all parameters are grouped into four generic categories with a total of nine subcases, which are further refined into 56 sub-items to explore diverse dynamical behaviors, including the globally asymptotically stabilities and bistabilities of boundary equilibria, the existence and stability of the positive equilibrium, the existence of periodic solutions, and the complex dynamics of some solutions. Many results are novel, most findings are shown analytically, and verified numerically. Fromabiological perspective, we find three competition interactions among species: the resource, apparent, and trade-off competitions. These explore how the interplay between direct competition for resources, apparent competition mediated by a shared predator, and trade-off competition dictates species survival and ecosystem diversity. By symmetry and without loss of generality, we assume that one prey species (e.g., u1) is superior in its birth-to-consumption ratio, the apparent competition. Biological implications are provided to interpret the origin of the dynamics

PP1426 — Graph-Based Nonequilibrium Analysis of RNA Polymerase Transcription from Optical Tweezers Time Series

SPEAKERS Diego Herraez Aguilar (Universidad Francisco de Vitoria)
AUTHORS Diego Herraez Aguilar (Universidad Francisco de Vitoria); Francisco Monroy (Universidad Complutense de Madrid); Lucía Benito-Barca (Universidad Francisco de Vitoria)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B14
TRACK Cutting Edge Research Areas

Abstract

RNAPolII transcription is a stochastic process where efficiency and fidelity emerge from nonequilibrium dynamics. We present a methodology to quantify the transcriptional efficiency of RNAPII using time series obtained from dual-bead optical tweezers experiments.

Our approach combines stochastic thermodynamics and graph-based time-series analysis. Probability fluxes along the inferred transcriptional states are estimated and used to quantify the breaking of detailed balance associated with transcriptional progression. To characterize the dynamics of RNAPII motion along the DNA template, the experimental TS are mapped into Horizontal Visibility Graphs, allowing the evaluation of the efficiency–precision trade-off in terms of the structural properties of the corresponding graphs.

Special attention is devoted to transcriptional pauses, which appear as dynamical regimes where entropy production increases significantly, suggesting enhanced nonequilibrium dissipation during these events. Finally, we incorporate hydrodynamic coupling between the two beads of the optical tweezers setup as a potential mechanism of energy transfer within the experimental system, and assess its influence on the inferred thermodynamic and graph-theoretic observables.

This framework provides a quantitative link between transcription dynamics, nonequilibrium thermodynamics, and graph-theoretical descriptors of experimental time series.

PP1658 — Integrating spatial statistics and agent-based modelling to study tertiary lymphoid structures in cancer

SPEAKERS Nicholas Lai (University of Oxford)
AUTHORS Helen Byrne (University of Oxford); Joshua Bull (University of Oxford); Nicholas Lai (University of Oxford)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B38
TRACK Mathematical Oncology

Abstract

Tertiary lymphoid structures (TLSs) are organised immune aggregates composed of T- and B-cells that form in tumours and are associated with improved patient outcomes. They develop from initially well-mixed lymphocyte populations into spatially organised structures. The mechanisms governing this process remain poorly understood, and there is no clear quantitative framework to relate TLS spatial organisation to underlying cellular processes.

We combine spatial analysis of experimental data with agent-based modelling to study TLS dynamics. We apply spatial statistics, including cross pair correlation functions and Moran's I, to quantify cell–cell colocalisation and organisation in spatial transcriptomic data. This reveals a maturation axis from well-mixed aggregates to segregated B- and T-cell zones.

Motivated by these observations, we develop an on-lattice agent-based model in which T- and B-cells migrate in response to chemokine signals from stromal cells. Feedback between lymphocytes and stromal cells drives the emergence of spatially segregated TLS organisation.

Finally, we integrate these approaches by applying the same spatial metrics to model simulations, projecting simulations onto the inferred maturation axis. This provides a quantitative framework for linking observed TLS structure to underlying cellular mechanisms.

PP1633 — Inverse modelling of mechanochemically coupled tumour growth under paclitaxel treatment and hypoxic stress

SPEAKERS Maryam Alka (University of Birmingham)
AUTHORS Fabian Spill (University of Birmingham); Maryam Alka (University of Birmingham); Maryam Parvizi (University of Birmingham)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B36
TRACK Multiscale and Multiphysics Modelling

Abstract

Tumour growth and treatment response emerge from coupled interactions between mechanics, hypoxia, and drug transport, yet most phase-field models remain purely forward with fixed parameters. This limits their ability to capture tumour-specific variability. We develop a mechanochemically coupled phase-field model integrating tumour evolution, oxygen dynamics, paclitaxel transport, and quasi-static elasticity. Tumour dynamics are described using an Allen–Cahn formulation to reflect non-conservative mass under therapy, with a modified Cahn–Hilliard model used for comparison. The model incorporates oxygen-limited proliferation, hypoxia-induced death, drug cytotoxicity, and stress-mediated inhibition. Simulations reproduce key spatial features, including hypoxic cores and drug exclusion regions, arising from feedback between stress, oxygen depletion, and transport. To enable predictive modelling, we embed the system within a PDE-constrained inverse framework to recover tumour-specific parameters from imaging data. Ongoing work focuses on adjoint-based optimisation, identifiability analysis, and stress-dependent boundary conditions. This framework provides a pathway toward personalised prediction of tumour response to therapy.

PP1205 — Ladderpath: A Compression-Based Hierarchical Framework for Biological Sequence Analysis

SPEAKERS Yu Liu (Beijing Normal University)
AUTHORS Da Zhou (Xiamen University); Yu Liu (Beijing Normal University); Ziwei Dai (Southern University of Science and Technology)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F26
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Ladderpath is a compression-based framework, grounded in Algorithmic Information Theory, for extracting repeated, nested, and hierarchically organized structure from symbolic sequences. Rather than treating biological sequences as flat strings, it reconstructs reusable building blocks and their dependency relations, yielding interpretable representations together with quantitative measures of complexity, hierarchical reuse, and sequence distance.

This poster presents Ladderpath as a unified mathematical and computational framework for biological sequence analysis. I introduce its core formalism, including ladderons, laddergraphs, and indices of repetition and compressibility, and show how the same representation supports multiple problems in mathematical biology. For example, Ladderpath-derived distances provide an alternative to purely alignment-based similarity measures for phylogenetic and comparative sequence analysis, enabling the study of duplication, modular reuse, and evolutionary innovation; Ladderpath-based tokenization offers a compression-guided alternative to amino-acid-level and BPE-style segmentation in protein and DNA language models, with the potential to capture longer-range regularities and biologically meaningful reusable units.

Overall, the poster argues that compression-based hierarchical decomposition provides an interpretable analytical tool and a mathematical interface connecting sequence complexity, evolution, and learning.

PP1476 — Learning Population Dynamical Models in Systems Biology with Mixed-Effect Gaussian Process ODEs

SPEAKERS Julien Martinelli (Aalto University)
AUTHORS Julien Martinelli (Aalto University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B22
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Ordinary differential equation models are central to systems biology and computational biology because they provide interpretable descriptions of regulatory, signaling and population-level dynamics [Mac+11].

In many biological applications, however, longitudinal data are sparse, noisy, irregularly sampled and heterogeneous across individuals or experimental units. Classical nonlinear mixed-effects ODE models capture such heterogeneity, but rely on a fixed parametric vector field and can therefore be sensitive to model misspecification [Lav14].

We present a Bayesian nonparametric mixed-effect ODE framework in which each subject is described by a shared population vector field together with an individual-specific deviation, both modeled with Gaussian process priors. Building on GP-ODE [Hei+18] and Mixed-Effects GPs [Ler+22], the proposed approach combines state-space trajectory priors with collocation-based ODE constraints, yielding tractable inference without repeated numerical ODE solves during training.

We evaluate the method on synthetic heterogeneous dynamical systems, including classical biological oscillators, and on real post-vaccination antibody kinetics data. The results show improved recovery of shared and subject-specific dynamics, better forecasting and adaptation to new subjects, and reliable uncertainty quantification, supporting the use of mixed-effect GP-ODEs as flexible data-driven dynamical models for heterogeneous biological systems.

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PP1393 — Mantis: A Foundation Model for Mechanistic Disease Forecasting

SPEAKERS Carson Dudley (University of Michigan)
AUTHORS Carson Dudley (University of Michigan)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B9
TRACK Mathematical Epidemiology

Abstract

Infectious disease forecasting in novel outbreaks or low-resource settings is hampered by the need for large disease and covariate datasets, bespoke training, and expert tuning, all of which can hinder rapid generation of forecasts for new settings. To address these challenges, we developed Mantis, a foundation model trained entirely on mechanistic simulations, which enables out-of-the-box forecasting across diseases, regions, and outcomes, even in settings with limited historical data. We evaluated Mantis against 68 forecasting models across 14 diseases with diverse modes of transmission, assessing both point forecast accuracy (mean absolute error) and probabilistic performance (weighted interval score, coverage). Despite using no real-world data during training, Mantis achieved lower mean absolute error than all models in the CDC's COVID-19 Forecast Hub when tested on early pandemic forecasts. Across all other diseases tested, Mantis consistently ranked in the top two models across evaluation metrics. Mantis further generalized to diseases with transmission mechanisms not represented in its training data, demonstrating that it can capture fundamental contagion dynamics rather than memorizing disease-specific patterns. These capabilities illustrate that purely simulation-based foundation models such as Mantis can provide a practical foundation for disease forecasting: general-purpose, accurate, and deployable where traditional models struggle.

PP390 — Mathematical and Mouse Models Identify T Regulatory Cell Influx as A Key Determinant of Acquired Resistance to PD-1 Immunotherapy

SPEAKERS Rachel Sousa (University of California, Irvine)
AUTHORS Rachel Sousa (University of California, Irvine); Shannon Geels (University of California, Irvine); Claire Murat (University of California, Irvine); Alexander Moshensky (University of California, Irvine); Mauro Di Pilato (MD Anderson Cancer Center); S Armando Villalta (University of California, Irvine); John Lowengrub (University of California, Irvine); Francesco Marangoni (University of California, Irvine)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations
BOARD F22
TRACK Mathematical Oncology

Abstract

The immune system can eradicate cancer, but various immunosuppressive mechanisms active within a tumor curb this beneficial response. Unraveling the effects of multimodal interactions between tumor and immune cells and their contributions to tumor control using an experimental approach alone is time- and resource-intensive. To identify key immunological features associated with tumor control and escape, we built a mathematical model of the interactions between CD8+ T cells, Tregs, DCs, and tumor cells. A distinguishing feature of our model is that it captures Treg accrual occurring after checkpoint blockade immunotherapy. After fitting the model to data from an immunogenic melanoma mouse model showing resistance to αPD-1, we generated hundreds of parameter sets, each representing a unique 'virtual mouse' to capture individual variability. Our model indicates that the initial tumor and immune conditions instruct cancer control or progression. Increasing the initial number of CD8+ T cells alone does not always yield better outcomes; instead, the model implies there exist optimal initial immune cell ratios that result in improved tumor control. The model also predicts Treg influx as a key determinant of αPD-1 resistance. All predictions were experimentally validated. Overall, this integrated approach of modeling and experimental validation identified key determinants of resistance to immunotherapy and can be used to guide the development of more effective therapeutic strategies.

PP275 — Mathematical model of excitation and contraction in cardiomyocytes for simulation of in vitro systems for Heart Failure drug development

SPEAKERS Jacob Bendsen (Roskilde University. Novo Nordisk a/s)
AUTHORS Giulia Simoni (Novo Nordisk A/S); Ross Bretherton (Novo Nordisk A/S); Morten Andersen (Roskilde University); Johnny T. Ottesen (Roskilde University); Jacob Bendsen (Roskilde University. Novo Nordisk a/s)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F4
TRACK Cardiovascular Modelling

Abstract

Heart failure with preserved ejection fraction (HFpEF) is growing in prevalence, often attributed to an ageing and obese population. It lacks effective treatments due to poorly understood pathophysiology and absence of translationally relevant animal models [Gao+24]. The aim of this work is to couple an in vitro system of strip-based engineered heart tissue (EHT) with a mathematical model of excitation-contraction coupling. This is used to gain a translational model of heart failure for investigation of potential drug targets. The mathematical model integrates the ODE-based "ORd" [OHa+11] electrophysiology model of dimension 41 with the ODE-based "HMT" [HMK98] contractile force model of dimension 6. The physiological coupling of the models is through calcium binding to troponin, which initiates the muscle contraction. The model is calibrated against twitch force data from EHTs consisting of human stem cell-derived cardiomyocytes and primary fibroblasts. The model reproduces baseline twitch force and captures the sigmoidal dependence of peak force on extracellular calcium concentration (0.2–1.8 mM). Experiments with administration of the well characterized calcium channel blocker verapamil (0.1, 0.5 and 1 µM) produced a dose dependent decrease in twitch force in the EHTs, which was in close agreement with model predictions. Ongoing work couples this model to a cardiac mechanics framework, enabling simulation of in vivo cardiac function and drug effects.

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PP1569 — Mathematical modeling of drug delivery by intra-vaginal rings, with application to trans-species ring designs

SPEAKERS Bhavana Morankar (North Carolina State University)
AUTHORS Bhavana Morankar (North Carolina State University); David Katz (Duke University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B30
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

An alternative to daily or on-demand methods (pills/gels), intravaginal rings (IVRs) provide long-term, topical drug delivery for contraception, HIV prophylaxis, and hormone therapy. Current IVR designs are based on empirical interpretations of in vitro and in vivo experiments in animals and humans. We developed a deterministic mathematical model of IVR-based drug delivery, to enhance rational IVR design. This multi-compartmental model is a set of coupled PDEs and ODEs embodying conservation of mass that predicts drug diffusion across the ring and into target compartments in the female reproductive tract – vaginal fluid, epithelium, stroma, and bloodstream. Model parameters specify designer-controlled IVR (e.g. ring size, drug properties, initial drug load) and host (e.g. anatomy, histology, drug-cell interactions) characteristics. IVR parameter estimation uses a simplified one compartment model, fit to in-vitro release-into-sink data. We created scaling rules to connect ring designs and experimental data across animal and human trials. This equates/scales biologically relevant model outputs by conserving volume- and time-averaged drug concentration in target regions like the stroma . MCMC simulations captured sensitivity of model outputs to varying IVR sizes, drug loads, and biological variations within/across species. This method is robust to target Islatravir (anti-HIV) drug delivery, and is a step toward comprehensive pharmacokinetic modeling and scaling for diverse IVRs.

PP1302 — Mathematical Modelling of Plant Cell Polarity Induced by SOSEKI Proteins

SPEAKERS Annika Brockhaus (Mathematical Institute, Leiden University)
AUTHORS Annika Brockhaus (Mathematical Institute, Leiden University); Joao Diniz Ramos (Mathematical Institute, Leiden University, NL); Roeland Merks (Mathematical Institute and Institute for Biology Leiden, Leiden University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F38
TRACK Multiscale and Multiphysics Modelling

Abstract

We develop mathematical models to study the role of mechanical forces in determining plant cell and tissue polarity. Our focus lies on SOSEKI proteins (SOKs) that distribute anisotropically along the cell membrane. Recent work suggests that SOKs are mechanosensitive [Pie+25], creating a potential connection between mechanics and plant cell polarity essential to plant organ development. The deeply conserved SOKs share a homologous domain with Dishevelled, an animal protein involved in Planar Cell Polarity (PCP) [Pie+25], the polarization of epithelial tissue normal to the apical-basal axis. Despite fundamental differences between the mechanism of PCP and SOKs, mathematical models of PCP (e.g., [Sch+10]) are a useful starting point for our research. Many models of PCP consist of three interacting components: (1) a cell-autonomous tendency to polarize, (2) information exchange between adjacent cells, and (3) a mechanism communicating tissue-wide directional information to individual cells. Although the molecular mechanism will differ, we expect animal PCP mechanisms may share conceptual similarities with plant models. We present first results of our multicellular SOK-model implemented in the vertex-based 2D plant tissue modelling framework VirtualLeaf [Ant+22]. We study how this model explains tissue polarity and present first results of the effect of mechanics.

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PP1223 — Mathematical Modelling of the Tumour Microenvironment and $\gamma\delta$ T cells in Ovarian Carcinoma

SPEAKERS Cedrik Neber (University Hospital Schleswig-Holstein, Kiel)
AUTHORS Cedrik Neber (University Hospital Schleswig-Holstein, Kiel); Philipp Altrock (Cancer Modeling & Evolution UKSH Campus Kiel)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F30
TRACK Mathematical Oncology

Abstract

Ovarian epithelial cancer still claims the highest mortality rates of all gynaecological cancers to date, in part due to its complex tumour immune microenvironment (TIME) [Ven+23]. The lymphocyte $\gamma\delta$ T cell subset has emerged as a promising target in cancer-related disease, such as ovarian carcinoma, due to its immunological plasticity [Sch+24]. Cytotoxic properties of $\gamma\delta$ T cells can be enhanced using bi-specific antibodies, but are subjected to the modulation of other TIME components [Gon+20; Sch+24]. One key modulator is the immuno-suppressant galectin-3, which binds β-galactoside on the cell surface [Gil+19]. In this mathematical modelling approach, we develop a set of ordinary differential equations (ODE) which explores the system dynamics of $\gamma\delta$ T cells and galectin-3 in ovarian cancer. More specifically, we analyse characteristic T cell behaviour including exhaustion, proliferation and tumour cell killing by assessing the functional response models.

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PP1473 — Mechanistic Integration of Longitudinal RNASeq Data to Reveal Novel Biomarkers in Parkinson's Disease

SPEAKERS Cyrille Lorenz-Dittmar (University of Luxembourg)
AUTHORS Cyrille Lorenz-Dittmar (University of Luxembourg); Alexander Skupin (University of Luxembourg)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B21
TRACK Systems Biology and Biochemical Networks

Abstract

Parkinson's disease (PD) is a progressive neurodegenerative disorder whose underlying molecular mechanisms remain poorly understood, hindering development of targeted therapeutics. While 10% of cases are linked to mutations in over 20 genes, the majority are idiopathic, reflecting the disease's complexity. Recent approaches use multi-OMICS characterization of patient-derived neurons obtained through differentiation of induced pluripotent stem cells (iPSC), however, identifying underlying mechanisms of PD remains challenging. We addressed this challenge by a Non-negative Matrix Tri-Factorization (NMTF) framework that mechanistically integrates longitudinal RNASeq data from patient-derived dopaminergic neurons carrying PD-associated mutations in the PINK1 and SNCA genes. Single-cell RNA sequencing, proteomics, and metabolomics data — spanning iPSC differentiation across seven timepoints — are processed via a high-performance computing pipeline and enriched with biological knowledge from the BioGrid database. The NMTF decomposes data into genotype and phenotype embedding spaces, enabling comparison of genotype-phenotype coupling matrices across conditions to identify impaired regulatory pathways. Applying this pipeline to the iPSC derived neurons, reveals both common and mutation-specific disease mechanisms that extend our knowledge about PD development and progression beyond the classical differentially expressed gene analysis.

PP1665 — Message-passing graph neural network models to predict protein-protein interactions

SPEAKERS Dan Mark Orapa
AUTHORS Dan Mark Orapa; Renier Mendoza; Victoria May Mendoza
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B39
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Computational, data-driven predictors of protein-protein interactions have become an active research area in interaction proteomics. Interestingly, high-performing baseline sequence-based models designed to predict interacting protein pairs were recently found to be trained on datasets of high similarity, resulting in data leakage during model training. In this study, we analyze protein-protein interaction networks (PPINs) and its biological and topological implications for computational models. We experiment with structure-aware machine learning models using various message-passing graph neural networks (MP-GNNs) paired with protein language models on varying PPINs—an unprocessed dataset from [PZS10] and a clustered dataset from [BBL24]. We found that MP-GNN models trained on clustered dataset paired with the protein language model ProtTrans perform best. Moreover, we found that model performance evidently changed when trained on an unprocessed dataset and evaluated on an unseen clustered dataset, and models trained on clustered datasets more reliably predict interactions than those trained on unprocessed ones. In essence, findings highlight that the framework can effectively learn interaction patterns, but its predictive power is still constrained by the quality and composition of the training data. Extensively, we discuss what the limitations of MP-GNNs are as computational models to enhance PPINs and, in turn, how they should be used as effective predictive models.

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PP373 — Metabolic Bacterial Game

SPEAKERS Oleksandr Cherednichenko (Umeå University)
AUTHORS Eric Libby (Umeå University); Josephine Solowiej-Wedderburn (Umeå University); Oleksandr Cherednichenko (Umeå University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F19
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Bacteria interact in different ways, from competition to cooperation, depending on resource availability in the environment. Identifying environments that foster these dynamics is both important and challenging, as the outcomes often depend on subtle alignment between the dynamic metabolic needs of species. A common community approach to this challenge has been metabolic modeling. Genome-scale metabolic models can identify interactions between species, but they are computationally expensive when applied to large communities. In fact, metabolic modeling often cannot fully capture the discrete nature of species, as these models typically optimize the total biomass of the system rather than the behavior of individual organisms.

We propose a simpler and more tractable alternative, the Metabolic Bacterial Game (MetaBGame), in which bacteria strategically consume or produce environmental compounds according to their metabolic needs. The framework employs multi-agent reinforcement learning and can operate under perfect or imperfect information, reflecting whether agents fully observe or only infer each other’s metabolic states. Our results demonstrate that in MetaBGame we can scale and learn communities up to thousand agents in 34 hours. On top of that, our simple design allows easily identifying competitive and cooperative

strategies. MetaBGame is implemented in JAX, allowing scaling across different hardwares, including CPUs and GPUs.

PP302 — Modelling biochemically coupled viscoelastic systems: an open-source computational framework

SPEAKERS Gordon McNicol (University of Waterloo)
AUTHORS Gordon McNicol (University of Waterloo); Peter S. Stewart (University of Glasgow)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F9
TRACK Multiscale and Multiphysics Modelling

Abstract

We present an open-source computational framework for the numerical solution of mechanochemical continuum models based on viscoelastic mechanics, with optional coupling to reaction-diffusion-advection dynamics of biochemical species. The framework is implemented in Python and adopts a method-of-lines formulation: spatial derivatives are discretised using finite-difference schemes, transforming the governing partial differential equations into a large system of ordinary differential equations with a structured, possibly state-dependent mass matrix. The resulting semi-discrete system is advanced in time using modern implicit solvers suitable for stiff systems, avoiding bespoke time-stepping or operator splitting whilst preserving full mechanical time dependence. The framework supports heterogeneous material parameters and general boundary conditions in one or more spatial dimensions, and is designed to be modular and reproducible. Core functionality is illustrated through a minimal, runnable implementation of deformation in Kelvin-Voigt, Maxwell, and Standard Linear Solid materials, with mechanical properties coupled to material biochemistry. This is followed by application to a detailed model of cell-substrate interaction. These examples demonstrate that existing open-source solver infrastructure provides a robust and extensible route for studying a broad class of biologically driven viscoelastic systems.

PP1520 — Modelling Disease Progression through Manifold Approximation

SPEAKERS Andreas Körner (TU Wien)
AUTHORS Andreas Körner (TU Wien); Christine Zeh (TU Wien); Daniel Pasterk (TU Wien)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B25
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

The adoption of artificial intelligence in healthcare is increasing rapidly, yet only a small number of applications have reached routine clinical use. A central obstacle is the lack of transparency, as many machine learning models operate as black boxes that produce predictions without interpretable reasoning. This contribution explores an alternative approach based on geometric data representations. Patient trajectories are embedded into a low-dimensional manifold that is learnt directly from the clinical measurements using the UMAP approximation algorithm [MHM20]. This replaces the opaque model parameters with a structure that remains interpretable within the original feature space. The method is designed for time-dependent intensive care unit data and introduces a semi-metric on trajectories inspired by the discrete Fréchet distance [EM94], which measures similarity between a new patient trajectory and a disease-specific manifold representation. This allows both disease membership testing, which evaluates whether a trajectory is consistent with a given condition, and outcome prediction by comparing trajectories to manifolds trained on surviving and non surviving patient groups.

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PP303 — Modelling pattern formation in a spatially discrete plant cell signalling system

SPEAKERS Patience Aba Sakyi (African Institute for Mathematical Sciences, Ghana)
AUTHORS Nicholas Alexander Miles Monk (University of Sheffield); Patience Aba Sakyi (African Institute for Mathematical Sciences, Ghana)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F10
TRACK Cellular and Developmental Biology

Abstract

Biological pattern formation is often based on non-linear interactions of chemicals and their movement, and reaction-diffusion models are a classic theoretical framework for explaining such patterning. A good example is seen in the root of Arabidopsis thaliana, where each cell commits to one of two fates: trichoblast and atrichoblast. Experimental data reveal a puzzling phenomenon in this system: two core factors in the regulatory network are predominantly transcribed in one cell type, but their proteins accumulate in the opposite cell type – a counter-intuitive observation that challenges classical reaction-diffusion models.

In this work, we use the mutual support model by Savage et al. (2008) as a framework to study a spatially discrete reaction-diffusion system in which protein and mRNA distributions are out of phase, and proteins move between neighbouring cells through plasmodesmata rather than by extracellular diffusion. The model captures a distinctive feature of this system: proteins form immobile complexes acting as sinks, thereby influencing intercellular distribution. Unlike classical activator-inhibitor systems, there is no obvious local activation, raising new questions about the regulatory setup that allows patterned cell fate decisions. We investigate how these properties allow the network to generate and maintain stable spatial patterns and consider how factors such as positional cues and directed nonlinear movement might further refine pattern selection.

PP1535 — Modelling scenarios to mitigate the potential spread of smallpox in Great Britain

SPEAKERS Luke Webb
AUTHORS Luke Webb
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B28
TRACK Mathematical Epidemiology

Abstract

Smallpox, caused by the variola viruses, led to high mortality throughout human history. Although eradicated, virus samples are held in laboratories and accidental or deliberate release remains possible. Many countries therefore maintain smallpox vaccine stockpiles as part of preparedness planning.

Historically, vaccination strategies were constrained by the risks associated with first generation vaccines, which carried high rates of severe adverse events. Modern third generation vaccines have an improved safety profile, shifting the balance between vaccine risk and outbreak control.

Modelling studies in the 2000s explored the potential impact of a smallpox outbreak in a contemporary UK population. These analyses concluded that mass-vaccination was suboptimal, due to the associated risks. Additionally, they relied heavily on expert judgement to quantify key elements of the public health response, such as vaccination rollout and contact tracing. Experience from the COVID-19 pandemic has since provided detailed real-world data on the performance and limits of these interventions.

We developed a stochastic metapopulation model for Great Britain, combining the strengths of earlier modelling work with updated population mobility data and empirically informed estimates of response capacity. Simulating outbreaks seeded with between 1 and 200 index cases, we reassess response strategies and the role of vaccination in contemporary smallpox preparedness.

PP316 — Multi-Scale Mathematical Modelling of Prostate Cancer Response to Iron-Supplemented Ferroptosis

SPEAKERS Eisha Arif (University of Sussex)
AUTHORS Eisha Arif (University of Sussex); Guillermo Lorenzo (University of A Coruña); James Van Yperen (University of Sussex); Marianna Cerasuolo (University of Sussex)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F14
TRACK Mathematical Oncology

Abstract

Prostate cancer (PCa) has been widely studied, yet new strategies are needed to mitigate resistance and recurrence. Ferroptosis, an iron-dependent form of regulated cell death, is emerging as a promising therapeutic strategy, yet its mathematical modelling in PCa remains limited [Mac+23]. This is especially relevant for castration-resistant prostate cancer (CRPC), where hormonal control fails and new therapies are needed. In this work, we developed a hybrid multi-scale framework to investigate the response of PCa to iron-supplemented, RSL3-induced ferroptosis. We use an ABM to represent the growth and treatment response dynamics of normal and cancer cells (e.g. proliferation, necrosis, phenotypic switching, and ferroptotic death), PDE reaction-diffusion equations for nutrients and drugs in the tumour microenvironment, and ODEs for the intracellular dynamics of key ferroptosis factors (e.g. iron, lipid hydroperoxides, and GPX4). Simulations of this hybrid ABM-PDE-ODE system calibrated to *in vivo* experiments reasonably capture tumour progression, treatment response, spatial patterns of the cellular species and drug-combination effects. Thus, by coupling intracellular biochemistry with spatial tumour dynamics, our framework enables investigation of how iron availability and GPX4 inhibition govern ferroptotic sensitivity, and how treatment scheduling and dosing can be optimised to maximise tumour elimination and prevent progression to CRPC.

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PP1471 — Multiscale Analysis of Gut Dysbiosis Identifies Time-Adaptive Immune Augmentation as an Inflammatory Flare Suppression Strategy

SPEAKERS Haya Mayoof (Khalifa University)
AUTHORS Dimitris Manias (Khalifa University); Haya Mayoof (Khalifa University); Haralampos Hatzikirou (Khalifa University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B20

TRACK Immunobiology & Infection

Abstract

Chronic inflammatory disorders, such as inflammatory bowel disease (IBD), are characterized by unpredictable transitions between disease remission and debilitating flare-ups driven by gut dysbiosis. Despite extensive research, the mechanistic drivers of these relapse-remission cycles and the optimal strategies to restore stability remain elusive. Here, we introduce a minimal mechanistic ordinary differential equation model coupling bacterial burden and innate immune activity, which we calibrated to longitudinal in vivo infection data and validated against an independent chemically induced colitis dataset. Using steady-state control maps, we delineate distinct disease regimes and demonstrate that increasing immune recruitment capacity can shift flare-prone dynamics into a stable recovery state. To operationalize this finding, we applied Computational Singular Perturbation (CSP) to dissect the multi-scale dynamics of the system, pinpointing a critical ”explosive” time window preceding flare peaks. CSP diagnostics identified immune decay as the dominant driver of oscillation growth, enabling the design of a time-adaptive, threshold-triggered intervention protocol that systematically suppresses recurrent flares. Ultimately, this analysis challenges the strictly immunosuppressive paradigm of current therapies, providing a mathematically grounded framework for stabilizing dysbiosis through targeted immune augmentation.

PP1522 — Non-Periodic Steady State in a Closed Ferroin-Catalyzed Belousov–Zhabotinsky Reaction induced by High Stirring Rates

SPEAKERS Mihnea Hristea
AUTHORS Florian Wodlei (Living Systems Research); Mihnea Hristea; Bao Tang (University of Graz)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B26
TRACK Systems Biology and Biochemical Networks

Abstract

It is intuitive that stirring homogenizes and increases the efficiency of liquid chemical reactions. However, in complex nonlinear systems, stirring can also alter intrinsic dynamics. Such systems may show bistability, excitability, or periodicity. A prominent example is the Belousov-Zhabotinsky reaction, where stirring effects have long been studied [MJ90; SM96; Ruo82]. In a closed ferroin-catalyzed batch reaction, high stirring rates within the periodic regime suppress oscillations [Ruo82]. Experiments show that oscillations reappear once stirring stops or the stirring rate is decreased. For moderate stirring rates, oscillations remain, but amplitudes increase after stirring stops. The effect also depends on reaction volume, geometry, and the phase at which stirring begins. Due to the complex interplay of reaction, diffusion, and convection, theoretical explanation is challenging [Nos+91; KRS10]. We aim to model the effect using diffusion-controlled reactions where the diffusion is affected by the turbulence created by the process of stirring. We conjecture that the stirring rate acts as a bifurcation parameter and present a detailed overview of our experimental findings and discuss a first version of the proposed model.

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PP209 — ON A MODIFIED LESLIE-GOWER MODEL WITH PREY DEFENSE AND PREDATOR CANNIBALISM

SPEAKERS Gloria Botchway (AIMS, Ghana)
AUTHORS Eric Takyi (Ursinus College); Gloria Botchway (AIMS, Ghana)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F1
TRACK Population Dynamics, Ecology & Evolution

Abstract

Recent experiments show that cannibalism in predators and de- fense in prey can both occur concurrently. Motivated by this, we investigate a predator-prey system where cannibalism occurs in predators and defense in prey simultaneously. System analysis show that depending on the prey defense (µ) and predator cannibalism (c) parameters respectively, one can have global stability of the coexistence and prey free states, bi-stability dynamics or up to three interior equilibria. Local stability analysis shows that, the combined effects of c and µ cannot drive both populations to extinction and for certain parameter choices can destabilize the system. After varying the parameters µ and c, the system undergoes standard co-dimension one bifurcations includ- ing Hopf bifurcation, transcritical bifurcation and saddle-node bifurcation. In addition, the system undergoes co-dimension two bifurcations such as cusp bi- furcation and Bogdanov-Takens bifurcation. For the spatially explicit model, we observe that in the absence of both µ and c, the system cannot exhibit Tur- ing instability, whereas the system can produce Turing instability depending on system parameters when present. We support our results with numerical experiments and discuss their ecological implications.

PP282 — Optimal control approach to obesity reduction

SPEAKERS Olga Vasilieva (Universidad Del Valle)
AUTHORS Amr Radwan (Jouf University); Olga Vasilieva (Universidad Del Valle)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F5
TRACK Miscellaneous

Abstract

This study numerically investigates optimal intervention strategies for reducing overweight and obesity using a compartmental dynamical model with three classes: normal-weight, overweight, and obese

individuals. Two time-dependent controls are considered: a preventive strategy promoting healthy lifestyles and a treatment intervention targeting obese individuals. The optimal control problem is solved using Pontryagin's Minimum Principle and implemented numerically through the Forward–Backward Sweeping Method with a fourth-order Runge–Kutta scheme. Simulations are performed over a 10-year horizon, assuming a constant population and initial prevalence levels consistent with reported data. Three intervention scenarios are examined: prevention only, treatment only, and a combined strategy. Results show that preventive measures significantly reduce the overweight population and indirectly lower obesity prevalence, whereas treatment directly reduces obesity but has a weaker impact on overweight individuals. The combined strategy yields the best outcomes, increasing the proportion of normal-weight individuals while reducing both overweight and obesity levels. Optimal control profiles indicate sustained preventive effort and gradually decreasing treatment intensity over time. Overall, the numerical analysis highlights the importance of integrating preventive lifestyle campaigns with treatment programs to achieve effective long-term control of obesity.

PP1574 — Optimizing cell-cycle–targeted drug combinations for high-grade serous ovarian cancer: An integrated approach

SPEAKERS Agata Xella (Moffitt Cancer Center)
AUTHORS Agata Xella (Moffitt Cancer Center); Alexander Anderson (Moffitt Cancer Center); Angie Rivera (Moffitt Cancer Center); Erin George (Moffitt Cancer Center); Lucia Mazzacurati (Moffitt Cancer Center); Zoe Zhou (Moffitt Cancer Center)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B31
TRACK Mathematical Oncology

Abstract

High-grade serous ovarian cancer (HGSOC) is the leading cause of gynecologic cancer mortality. Late diagnosis, widespread disease, and frequent relapse limit the effectiveness of current therapies. p53 deficiency indicates HGSOCs are vulnerable to cell-cycle inhibitors; however, currently available agents have shown less clinical benefit than expected. We hypothesize that maximizing the efficacy of these drugs requires treatment schedules that exploit HGSOC cell-cycle dynamics.

Experimental results showed the CDC7 inhibitor Simurosertib (active in G1 and S) and the PKMYT1 inhibitor Lunresertib (active in G2 and M) have strong synergy, even with sequential administration. Our associated ODE model, incorporating mechanistic knowledge of the cell cycle and drug action, showed that cell-cycle dynamics were key to this synergy. Subsequent time- and cell-cycle-resolved tracking data revealed cell-line-specific differences in cell-cycle distribution, motivating an expansion of the model that better integrated drug effects across phases and cell lines.

The calibrated model predicts schedule-dependent differences in cell death and tumor burden driven by cell-cycle dynamics and pre-existing mutations. These results highlight the potential of quantitative modeling to guide scheduling of cell-cycle inhibitors and support the development of personalized treatment strategies for HGSOC.

PP1371 — Optimizing Dengue Vaccination Strategies in Brazil: The Role of Regional Immune Profiles and Age-Targeted Allocation

SPEAKERS Youngsuk Ko (University of California, Berkeley)
AUTHORS Youngsuk Ko (University of California, Berkeley); Cleber Santos (Yale University); Angkana Huang (University of Cambridge); Derek Cummings (Johns Hopkins University); Albert Ko (Yale University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B7

TRACK Mathematical Epidemiology

Abstract

In 2024, Brazil faced its worst dengue epidemic, with 6 million cases and 5,000 deaths. A shift toward higher mortality among the elderly challenges WHO strategies prioritizing children. As immunity varies across Brazil's geography, a "one-size-fits-all" vaccination approach may not be optimal. We reconstructed state- and age-specific immune profiles across Brazil by fitting an age-structured catalytic model, which assumes a piecewise constant force of infection, to surveillance data (2000–2024). These profiles, accounting for historical force of infection and demographics, were integrated into an SIR-type transmission model. We simulated vaccine strategies to project disease burden and evaluate impact under supply constraints. Our model revealed substantial spatial heterogeneity in immunity. In hyperendemic states like Rio de Janeiro, most elderly have had multiple infections. In southern states like Santa Catarina, many remain at risk. While child-centric strategies reduce DALYs, targeting the elderly in regions with lower historical exposure yields larger mortality reductions. Findings emphasize that dengue vaccination must be tailored to local immunity. In regions where older adults lack cumulative exposure, including them in programs significantly mitigates severe disease and death. This research provides a data-driven framework for sub-national vaccine prioritization.

PP287 — Optimizing enzyme inhibition analysis: precise estimation with a single inhibition concentration

SPEAKERS Hyeong Jun Jang (Graduate School of AI for Math, KAIST, Daejeon, Republic of Korea)

AUTHORS Hwi-yeol Yun (College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea); Hyeong Jun Jang (Graduate School of AI for Math, KAIST, Daejeon, Republic of Korea); Jae Kyoung Kim (Department of Mathematical Sciences, KAIST, Daejeon, Republic of Korea); Jang Su Jeon (College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea); Sang Kyum Kim (College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea); Yun Min Song (Biomedical Mathematics Group, Pioneer Research Center for Mathematical and Compuational Sciences, Institute for Basic Science, Daejeon, Republic of Korea)

SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations

BOARD F6

TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Enzyme inhibition analysis is essential in drug development and food processing, necessitating precise estimation of inhibition constants. Traditionally, these constants are estimated through experiments using multiple substrate and inhibitor concentrations, but inconsistencies across studies highlight a need for a more systematic approach to set experimental designs across all types of enzyme inhibition. Here, we address this by analyzing the error landscape of estimations in various experimental designs [Jan+25]. We find that nearly half of the conventional data is dispensable and even introduces bias. Instead, by incorporating the relationship between IC50 and inhibition constants into the fitting process, we find that using a single inhibitor concentration greater than IC50 suffices for precise estimation. This IC50-based optimal approach, which we name 50-BOA, substantially reduces (>75%) the number of experiments required while ensuring precision and accuracy. Additionally, we provide a user-friendly package that implements the 50-BOA.

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PP379 — Parameter Optimization of a Thalamocortical Network Model for Simulation of EEG under Propofol

SPEAKERS Olga Lazic (TU Wien)
AUTHORS Afra Wohlschläger (TUM, Klinikum rechts der Isar); Alexander Edthofer (TU Wien); Andreas Körner (TU Wien); Juliana Zimmermann (TUM, Klinikum rechts der Isar); Olga Lazic (TU Wien)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F20
TRACK Neuroscience and Neural Systems

Abstract

Propofol anesthesia reshapes electroencephalogram (EEG) dynamics, producing prominent slow-delta and alpha oscillations, see [Pur+15]. Biophysical models provide a mechanistic framework for understanding how these rhythms emerge from neuronal interactions. In this work, we implement the biophysical thalamocortical network model of Soplata et al. [Sop+23], which describes interactions between cortical and thalamic neuron populations underlying these oscillations. Following their approach, we model EEG as the summed activity of cortical pyramidal neurons to generate simulated EEG traces for subsequent power spectral density analysis. While the reproduced model captures anesthesia-related oscillatory dynamics, the simulated spectra reveal differences compared with the spectral features reported in EEG recordings during propofol anesthesia[Pur+13]. In particular, although slow–delta activity emerges as expected when varying parameters associated with anesthetic depth, the increase in alpha-band power typically seen in frontal EEG recordings is not fully reproduced. Instead, the simulated spectra show a shift of spectral power toward higher frequencies above the alpha band, with a peak in the low-beta range. Motivated by this observation, we explore how variations in model parameters influence the resulting spectral structure, aiming to identify parameter regimes that reproduce the alpha-band dominance characteristic of frontal EEG during propofol anesthesia.

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PP1408 — Percolation theory and Voronoi geometry for modeling protein-ligand interactions

SPEAKERS Miranda Lynch (University at Buffalo/Hauptman-Woodward Institute)
AUTHORS Miranda Lynch (University at Buffalo/Hauptman-Woodward Institute)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B12
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

In the field of structure-based drug design, there is enormous interest in determining the binding characteristics and physical orientations of putative therapeutic ligands within the binding sites of the proteins they target. A primary tool to investigate this binding interaction involves characterizing the structure of the protein bound to its ligand, typically using X-ray crystallography and Cryo-EM as structural methods. In this work, we take a modeling approach to the problem, using tools from spatial stochastic processes and stochastic geometry to understand the dynamics and shape configurations underlying this problem. We use percolation theory to model aspects of ligand contact with the protein target, coupled with Voronoi geometry (and its dual, Delaunay tessellation) to capture protein pocket geometry and ligand orientation effects. Percolation theory is a mathematical formalism that deals with how connected clusters occur in random graphs/networks. It is employed for modeling a wide array of problems involving disordered or porous media, such as protein crystalline arrays used in ligand structural studies. Our work addresses new challenges in applied percolation, including comparing diffusional versus invasive percolation, and specific problems arising from definition of boundary conditions for the percolation process.

PP1387 — Physics-Informed Neural Networks for Mathematical Models on Alzheimer's Disease Dynamics

SPEAKERS Samira Breitling (PhD student)
AUTHORS Samira Breitling (PhD student)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B8
TRACK Cutting Edge Research Areas

Abstract

Mathematical models can be used in order to verify medical hypotheses and quantify the mechanisms of the progression of neurological pathologies like Alzheimer's disease. Here we consider a model based on ordinary differential equations incorporating dynamics of toxic proteins like Amyloid β species and tau tangles and describing their spread on a brain graph based on the human connectome, where brain regions are connected by edges representing fiber tracts.[Bia+24] In order to solve the differential equation and compare the results with clinical data, Physics-Informed Neural Networks (PINNs[RPK19]) can be leveraged. The deep-learning framework has shown to be powerful by combining previous physical knowledge with sparse or noisy data[Zha+24], in this case tau concentrations inferred from PET scans of subjects with Alzheimer's disease [Pet+10]. Choosing the best suited hyperparameters and the weights of the individual loss function terms is delicate, but even with a quite simple network architecture, results have been achieved that are comparable with those given by standard numerical methods in a steady-state setup. Future applications and extensions of the approach will involve model parameters estimation by setting specific variables trainable within the PINN. Identifying the most suitable parameter values will allow further assessment of the model's ability to reproduce observed patterns of pathology.

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PP1398 — Quantifying End-of-Outbreak Probability of Ebola via Stochastic Models

SPEAKERS Sumin Park (Kyungpook National University)
AUTHORS Hyojung Lee (Kyungpook National University); Sumin Park (Kyungpook National University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B10
TRACK Mathematical Epidemiology

Abstract

Accurate assessment of the infection scale is essential for determining the end of Ebola virus disease (EVD) outbreaks in West Africa. Previous compartmental modeling studies often assume the total population to be the effective population size or simply preset the initial number of infections. These simplifying assumptions can distort transmission dynamics and reduce prediction accuracy. This study aims to (1) directly estimate the effective population size and pre-surveillance spread period within a stochastic compartment model using surveillance data, and (2) quantify the end-of-outbreak probability by accounting for stochastic uncertainty through stochastic differential equations. Using 2014–2016 West African Ebola data, we jointly estimated transmission parameters, revealing substantial differences between the estimated effective size and total census population—a discrepancy that improved overall model fit. Building on prior analyses, our stochastic differential equation framework confirmed that the current WHO 42-day waiting criterion may be insufficient to ensure a 95% confident level for declaring an end of outbreak [TMJ19]. These findings suggest that the adequacy of the WHO 42-day criterion may vary depending on country-specific dynamics and surveillance capacity, indicating the need for more sophisticated approaches to end-of-outbreak criteria.

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PP340 — Quantifying the Impact of Healthcare Accessibility on Non-COVID Excess Mortality During the COVID-19 Pandemic in South Korea

SPEAKERS Soyoung Kim (National Institute for Mathematical Sciences)
AUTHORS Sunhwa Choi (National Institute for Mathematical Sciences); Boseung Choi (Korea University); Soyoung Kim (National Institute for Mathematical Sciences); Daeil Jang (National Institute for Mathematical Sciences)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations

BOARD F16
TRACK Mathematical Epidemiology

Abstract

Spatial accessibility to healthcare can influence population health outcomes during large-scale public health crises. However, its contribution to excess mortality beyond directly reported COVID-19 deaths remains insufficiently quantified. This study evaluates the relationship between regional healthcare accessibility and excess mortality across South Korea during the COVID-19 pandemic.

Regions were grouped using hierarchical clustering based on average travel time to medical facilities, producing high- and low-accessibility clusters. A counterfactual mortality baseline was estimated using a machine-learning prediction model trained on pre-pandemic mortality data from 2014–2019. Excess mortality during the pandemic period (2020–2022) was calculated as the difference between observed and predicted deaths. The association between accessibility and excess mortality was then assessed using multiple linear regression.

Regions with lower healthcare accessibility showed substantially higher excess mortality, particularly during the Omicron wave when healthcare demand was highest. In high-accessibility regions, excess mortality largely aligned with reported COVID-19 deaths, whereas low-accessibility regions exhibited additional excess mortality not explained by COVID-19 fatalities. These findings highlight how spatial healthcare accessibility may amplify indirect mortality during pandemics.

PP1431 — Quantifying the influence of inflammation on spiral artery dynamics through the expansion of a mechanistic endometrial model

SPEAKERS Alexandria Johnson (North Carolina State University)
AUTHORS Alexandria Johnson (North Carolina State University); Mette Olufsen (NC State)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B15
TRACK Miscellaneous

Abstract

Heavy menstrual bleeding (HMB) is linked to dysregulated inflammatory signaling and abnormal spiral artery function. However, the mechanism connecting endometrial inflammation and vascular response is not well understood. To address this, we are extending an existing mechanistic ordinary differential equation model of the menstrual cycle. Our expanded framework incorporates the inflammatory modulation of spiral artery dynamics, specifically mediated by prostaglandin activity.

Our model investigates the relationship between hormone-regulated prostaglandin production in endometrial tissue and the resulting vascular dynamics, including spiral artery constriction, vasodilation, and breakdown throughout the menstrual cycle. We utilize local and global sensitivity analyses to assess the impact of various inflammatory parameters on vascular stability and predicted blood loss. This allows us to quantify how changes in tissue-level inflammatory signaling ultimately affect outcomes at the vascular scale.

This study establishes a multiscale mechanistic framework for investigating inflammation-driven vascular dysfunction. Key prostaglandin-related parameters that govern spiral artery reactivity were identified through preliminary results, which also pinpointed structurally sensitive model components. The sensitivity analysis highlights areas where greater cellular-scale detail may be necessary. This work provides a crucial foundation for future integration with clinical data

PP1207 — Quantifying Variant-Specific Transmissibility and Forecasting Summer COVID-19 Outbreaks in Korea

SPEAKERS Yoonah Kang (Kyungpook National University)
AUTHORS Yoonah Kang (Kyungpook National University); Hyojung Lee (Department of Statistics, Kyungpook National University, Daegu, Republic of Korea)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F27
TRACK Mathematical Epidemiology

Abstract

COVID-19 epidemics have repeatedly coincided with shifts in dominant variants, and summer hospitalization increases were observed in South Korea in 2024 and 2025. This study aims to quantitatively evaluate and forecast the impact of dominant variant transition on the scale of the 2026 summer epidemic in South Korea.

Hospitalization surveillance and SARS-CoV-2 sequence data from January 1, 2024, to January 19, 2026, were analyzed. Variant-specific relative transmissibility was estimated from temporal changes in variant proportions using multinomial logistic regression [Obe+22] and incorporated into an age-structured SEIHR-VD model for individuals aged < 65 and ≥ 65 years. The transmission rate was decomposed into variant-associated transmissibility and background transmission. Scenario analyses varied the timing of dominance and transmissibility of a new variant.

KP.3 and NB.1.8.1 became dominant in August 2024 and May 2025, with relative reproduction numbers of 1.07 and 1.25, respectively. Despite the higher transmissibility of NB.1.8.1, the weekly hospitalization peak declined from 1,441 to 459. In the 2026 scenarios, transmissibility had a greater effect on epidemic size than the timing of dominance. The largest epidemic was forecast under May dominance with high transmissibility, with a peak of 993 weekly hospitalizations and 7,655 cumulative hospitalizations. Adults aged ≥ 65 years accounted for a large share of hospitalizations in all scenarios.

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PP1253 — Reaction Kinetics of Ryanodine Receptors in a Closed–Open–Inactivated Model

SPEAKERS Anna Hlubinová (Comenius University Bratislava)
AUTHORS Anna Hlubinová (Comenius University Bratislava); Christo Morison (Comenius University Bratislava); Pavol Bokes (Comenius University Bratislava)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F31
TRACK Systems Biology and Biochemical Networks

Abstract

Calcium-induced calcium release plays a significant role in mammalian skeletal and cardiac cell excitation. The ion channels responsible for this process are called ryanodine receptors (RyRs), which can exist in several conformational states, including open, closed and inactivated. Transitions between these states are regulated by concentrations of ligands, particularly calcium and magnesium. Recently, Zahradníková and colleagues proposed a structurally-informed allosteric model for this system, that reproduces the steady-state probabilities of RyR channels being open across a range of ligand concentrations [Zah+25]. In this work, we develop a continuous-time Markov chain model for the Closed–Open–Inactivated (COI)

gating scheme corresponding to this framework, allowing the kinetic behaviour of RyR channels to be studied beyond steady-state thermodynamics. The model enables the calculation of experimentally observable quantities such as open- and closed-time distributions and burst statistics, providing a link between structural models of RyR regulation and single-channel experimental recordings.

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PP1310 — Reaction-diffusion modelling of siderophore-mediated iron uptake and cross-feeding in bacteria communities.

SPEAKERS Tosin Samuel Osikoya (Technical University of Munich)
AUTHORS Tosin Samuel Osikoya (Technical University of Munich)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F40
TRACK Systems Biology and Biochemical Networks

Abstract

The growing prevalence of antibiotic-resistant bacteria poses a major global health challenge. Pseudomonas aeruginosa, identified by the WHO as a critical antibiotic-resistant pathogen, highlights the need for alternative therapies. Because iron is essential for bacterial survival and is often acquired through siderophores, disrupting iron uptake, particularly through interactions with competing microbial species, offers a promising therapeutic strategy. Previous studies on iron chelation dynamics and cross-feeding have shown that siderophores production and cross-feeding interactions provide a growth advantage to bacteria capable of engaging in these processes. This has been demonstrated in experimental studies under well-mixed chemostat conditions. However, these studies neglect spatial heterogeneity, even though bacterial populations in natural environments typically live in spatially structured communities. In this study, we extend an ordinary differential equation model of iron chelation and cross-feeding, we formulate a reaction-diffusion model for two bacteria strains in a two-dimensional space. The resulting nonlinear partial differential equations are solved numerically using a semi-implicit finite difference method. The model properties were analyzed, and our results show that siderophores production and cross-feeding provide a significant growth advantage even in spatially structured environments, suggesting potential strategies to address antibiotic resistance.

PP1435 — Real time modeling of the 2025 foot-and-mouth disease outbreak in the Hungary-Slovakia border region

SPEAKERS Olivér Régaisz (University of Szeged)
AUTHORS Fanni Nedény (University of Szeged); Gergely Röst (University of Szeged); Olivér Régaisz (University of Szeged)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B16
TRACK Mathematical Epidemiology

Abstract

In Spring 2025, a foot-and-mouth disease (FMD) outbreak occurred along the Hungary-Slovakia border region, affecting 11 farms before being contained. While the small number of cases is encouraging from a control perspective, it poses a major statistical challenge: standard methods for inferring transmission

parameters and evaluating control measures are not suitable for such limited data. Nevertheless, timely and quantitative estimates are crucial for guiding contingency planning and resource allocation.

To address this challenge, we developed a simulation-based inference framework built on a farm-level branching-process model. Our approach is to generate a large ensemble of simulated outbreaks under a range of plausible parameters and retain those that closely match the observed epidemic. These "look-alike" epidemics allow us to construct posterior distributions for key epidemiological quantities. Applied to the Hungary-Slovakia data, the posterior concentrates the pre-control farm-to-farm effective reproduction number around 3-4 (median ≈ 3.6) and the post-control effective reproduction number below one (median ≈ 0.8), with a generation-interval median of about 12 days.

Our method is transparent, biologically grounded, and computationally efficient, making it suitable for real-time decision support during small outbreaks. It was used in real time to inform the food safety authority of Hungary responsible for animal health and agriculture production as well.

PP1457 — Real-time detection of aberrant physiological changes from wearable data using Kalman Filtering and Autoencoder

SPEAKERS Yejoon Wang (KAIST)
AUTHORS Daewook Kim (KAIST); Yejoon Wang (KAIST)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B18
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Consumer-grade wearables enables continuous monitoring and early detection of aberrant physiological signals associated with diverse health issues. However, substantial measurement noise and natural physiological variability in wearable data have prevented reliable identification of physiological anomalies, limiting their deployment in real-world and clinical settings. Here, we propose a real-time anomaly detection framework that first estimates latent physiological dynamics using a Kalman filter and subsequently detects anomalies using an autoencoder-based model. By explicitly modeling physiological dynamics, our approach projects out known sources of variation such as circadian rhythms, intrinsic biological fluctuations, and measurement noise, leaving anomalies visible in the residual. We tested our method on real-world wearable body temperature data from cancer patient and showed that it can detect early signals of impending fever events. Applying anomaly detection after physiological filtering outperforms an existing method of directly applying the autoencoder to raw data, including LSTM-autoencoder based models previously used for wearable anomaly detection. This indicates the importance of incorporating physiological structure and filtering into machine learning pipelines.

PP1201 — Reconstructing Influenza Incidence from Sentinel Surveillance Data Using a Latent INGARCH Model

SPEAKERS Changdae Son (Department of Statistics, Kyungpook National University, Daegu, Republic of Korea)
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SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F24
TRACK Mathematical Epidemiology

Abstract

Influenza is a seasonal infectious disease, and real-time forecasting of outbreaks is essential for effective public health responses. In Korea, influenza surveillance relies on two types of data. Influenza-Like Illness (ILI) data collected through sentinel surveillance reflects outbreak trends, whereas confirmed case data obtained through universal surveillance better represent the total number of patients but are not updated in real time. Previous studies applied different likelihood functions to universal and sentinel surveillance periods to estimate population-level incidence. We extend this approach to a statistical time-series model.

We propose a latent INGARCH model in which the latent process represents population-level influenza incidence while observations correspond to counts recorded under different surveillance systems. Because sentinel observations represent only a subset of cases, a scale mismatch arises between the input data and the model output. To address this issue, we use the Poisson splitting property to reconstruct population-level incidence from sentinel observations and use the reconstructed counts as model inputs through an input data scaling procedure.

The proposed framework enables estimation of population-level influenza incidence using sentinel surveillance data while accounting for scale differences between surveillance systems. By addressing this mismatch, the proposed model improves prediction accuracy compared with existing approaches.

PP1424 — Reconstruction of Nosocomial Infectious Disease Outbreaks Using Bayesian Inference

SPEAKERS Oskar Holmstedt (Chalmers University of Technology)
AUTHORS Philip Gerlee (Chalmers University of Technology & University of Gothenburg); Jon Edman Wallér (Gothenburg University); Torbjörn Lundh (Chalmers University of Technology & University of Gothenburg); Oskar Holmstedt (Chalmers University of Technology)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B13
TRACK Mathematical Epidemiology

Abstract

Nosocomial infections, also known as hospital-acquired infections, pose a major public health challenge, particularly due to the increase of antibiotic-resistant pathogens.[Edm+25] Understanding how such infections spread within hospitals is vital for effective infection control. Outbreak reconstruction, to infer the underlying transmission tree based on observed data, is of particular interest.[DDC24] Commonly, Markov Chain Monte Carlo methods are used to sample transmission trees consistent with the data in a Bayesian framework.[Cam+19] In this work, we consider the problem of reconstructing transmission trees for nosocomial outbreaks. We present a stochastic model of hospital-wide outbreaks with heterogeneous infectivity and environmental transmission. Metropolis-Hastings sampling is then used to infer the posterior distribution of the transmission trees, infection times and model parameters of partially observed simulated outbreaks. Compared to previous approaches, the model is designed specifically for nosocomial outbreaks. This allows the model to capture realistic hospital dynamics where infections may arise both through direct patient contact and indirectly via contaminated environments. Our results show that Bayesian inference can provide useful insights into the structure of hospital outbreaks. In practice, improved outbreak reconstruction tools could support the work of infection prevention and control practitioner.

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PP1351 — Risk-aware mechanistic framework for early detection of post-radiotherapy biochemical relapse in prostate cancer

SPEAKERS Miguel Anxo Vicente Pardal (University of A Coruña)
AUTHORS Miguel Anxo Vicente Pardal (University of A Coruña); Chiara Lucrezia Deantoni (IRCCS San Raffaele); Nadia di Muzio (IRCCS San Raffaele); Anirban Chaudhuri (The University of Texas at Austin); Ernesto Lima (The University of Texas at Austin); Guillermo Lorenzo (Universidade da Coruña)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B5
TRACK Mathematical Oncology

Abstract

Patient monitoring after radiotherapy for prostate cancer relies on population-based thresholds of rising prostate-specific antigen (PSA), ignoring patient-specific tumor dynamics and uncertainty. To avoid delays in recurrence detection and treatment, we propose a personalized Bayesian mechanistic framework to forecast post-radiotherapy PSA dynamics. These forecasts enable the definition of risk-aware biomarkers of biochemical relapse, such as surviving tumor cell proliferation rate and time to progression. The mechanistic model is calibrated using longitudinal measurements, yielding posterior distributions of PSA over time and the model-based biomarkers. To quantify biochemical relapse risk, we summarize posterior distributions using α-superquantiles to capture adverse tail behavior and evaluate them using leave-one-out cross-validated ROC analyses and comparison across post-treatment time horizons. The most informative model-based biomarkers achieve high discriminative performance, which is superior to standard PSA-based metrics (e.g., PSA nadir, time to PSA nadir). To further assess clinical relevance, we introduce a days gained metric, representing the time gained by model-based relapse detection compared with standard-of-care PSA-based criteria (e.g., nadir+2 ng/mL). Pareto front analyses reveal trade-offs between classification accuracy (sensitivity/specificity) and early detection, enabling risk-aware selection of biomarker thresholds for clinical decision-making.

PP1533 — Social information under restricted observations

SPEAKERS Andrew Bate (University of Leeds)
AUTHORS Andrew Bate (University of Leeds); Charlie Pilgrim (University of Leeds); Richard Mann (University of Leeds)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B27
TRACK Population Dynamics, Ecology & Evolution

Abstract

Observing the decisions and actions of others within a group (such as running from a predator) provides social information that can inform actions such as whether to follow. We consider a model where all agents simultaneously gather stochastic private information (weighted towards an unknown preference), coming to a decision once sufficiently confident. These observing agents infer the state of the private information from these decisions and use this as social information that they incorporate with their private information; they will follow the observed decision if they are sufficiently confident with this new social information. Unlike previous models, we assume that agents can only observe the decisions of some (but not all) other agents at any one time. Consequently, first decisions are unseen by many, meaning decisions instead spread through a 'network of observation' and that the first decision seen by an agent could be the result of social information acquired by earlier hidden decisions. We will explore how the properties of the network of observation affect how social information and decisions spread, as well as how this affects decision accuracy and consensus within the group.

PP1548 — Spatial and Stochastic Microtubule Dynamics Model in the Neuronal Dendrite

SPEAKERS Meghan Kwon
AUTHORS Meghan Kwon; Veronica Ciocanel (Duke University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B29
TRACK Cellular and Developmental Biology

Abstract

Neurons are specialized cells which transmit signals and move biological material across their axons and dendrites. The fundamental organization of neurons relies on microtubules (MTs) which are elongated protein polymers with a plus and minus end. These MTs form tracks on which cargo can be transported within the cell. It is well known that dendritic MTs are extremely dynamic, reorganize rapidly, and have mixed polarity where plus ends may point in opposing directions. Despite this, the long arrays formed by MTs are highly stable structures. Our group has developed a stochastic and spatial model which tracks individual MTs throughout time in a linear region of a dendrite and predicts the overall behavior. We carried out sensitivity analysis of the model to understand how key parameters such as number of MTs, length between branches, and MT length impacted the establishment and maintenance of biased polarity. We have preliminary results which extend our 1D model to include dendritic branches and thus incorporating the contribution of additional biological mechanisms. These results suggest that MT content depletes in the absence of ample nucleation sites, which is informing our collaborators' experimental work and our recent updates to the 2-dimensional spatial model.

PP1615 — Stationary and oscillatory corrosion patterns in extended Barkley models

SPEAKERS Giancarlo Consolo (Università degli studi di Messina, Italy.)
AUTHORS Guglielmo Inferrera (Università degli studi di Messina, Italy); Giancarlo Consolo (Università degli studi di Messina, Italy.); Cinzia Soresina (Università degli studi di Trento, Italy)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B34
TRACK Miscellaneous

Abstract

We present corrosive-passivating models to describe the initiation of corrosion patterns on metal surfaces, particularly fuel tank bottoms. The classical Barkley model for excitable systems [Bar91] has been also applied in the context of corrosion [Bat+24], modeling the interaction between corrosive and passivating species as a fast activator and a slow inhibitor, respectively. However, the original Barkley model relies on overly simplistic assumptions: the passivating species is considered diffusionless, and its reaction dynamics

are restricted to linear growth and decay terms. To better capture the phenomenon of localized pitting corrosion, we propose some extensions to this model. First, we relax the diffusionless assumption by exploiting the dichotomy between different states of the passivating species occurring at a faster time scale. In the fast-reaction limit, this yields a novel model featuring a cross-diffusion term for the passivating species [Con+25]. Second, the passivating species is assumed to follow a Leslie-Gower dynamics with a Holling-type II functional response. The resulting frameworks allow the emergence of both stationary and oscillatory corrosion patterns, enabling qualitative comparisons with experimental data. Numerical continuation, bifurcation and simulations in 1D and 2D domains complement our theoretical analysis.

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PP1691 — Stem cell competition in the pre-neoplastic intestine under immunosurveillance

SPEAKERS Birgit Tschiatschek (Department of Biology, University of Graz)

AUTHORS Bernat Corominas-Murtra (Department of Biology, University of Graz); Birgit Tschiatschek (Department of Biology, University of Graz); Gundolf Haase (Department of Mathematics and Scientific Computing, University of Graz); Haris Mirza (Department of Pathology, Yale School of Medicine); Miraal Maqsood (Department of Pathology, Yale School of Medicine)

SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building

SESSION Poster Presentations

BOARD B41

TRACK Cellular and Developmental Biology

Abstract

The dynamics and geometry of intestinal crypts have been revealed as a key factor in the regulation of stem cell numbers in homeostasis, defining a framework in which cells compete for colonization of the organ in a neutral way [Lop+10; Azk+22]. A key question is whether these same principles extend to pathological conditions where neutrality is broken by malignant clones. To address this, we build on the Stochastic Conveyor Belt (SCB) theory [Azk+22; Cor+20], whose main parameters are the rate of continuous stem cell divisions, the rate at which stochastic relocations to all directions with neighboring cells occur, and the geometry of the organ.We studied the effect of the breakdown of neutral competition among stem cells by introducing mutant cells with a higher division rate at the start, both experimentally and theoretically. Interestingly, gene expression analysis showed up-regulation of an antigen presentation pathway in those mutant clones, indicating the involvement of the immune system in maintaining neutrality in the stem cell competition. Disruption of antigen presentation machinery or immune cell depletion further accelerated mutant clone fixation, suggesting the immune microenvironment's role in suppressing oncogenic stem cell expansion. Our results show that while specific mutations confer a selective advantage to mutant clones, the majority are stochastically removed. However, an increased immune response is observed in the presence of these mutant clones, which may prevent the fixation of oncogenic clones in the colon. This finding potentially provides a new starting point for future research into immune preventive strategies that support the elimination of such clones.

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PP369 — Structural inference over reaction network spaces

SPEAKERS Elijah Foo (University of Melbourne)
AUTHORS Torkel Loman (University of Oxford); Alexander Browning (University of Melbourne); Ivo Siekmann; Ruth Baker (University of Oxford); Jennifer Flegg; Elijah Foo (University of Melbourne)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F18
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Dynamical systems in biochemistry are complex, and one often does not have comprehensive knowledge about the interactions involved. Chemical reaction network (CRN) inference aims to identify, from observing time-series of species concentrations, the unknown reactions between the species. Most frequentist approaches to CRN inference focus on identifying a single, most likely CRN, without addressing uncertainty about the network structure. On the other hand, Bayesian treatments of CRN inference typically involve trans-dimensional and multimodal posterior distributions, which are computationally challenging to deal with. This poster illustrates how Bayesian CRN inference can be tackled with tempered spike-and-slab distributions, with applications to population models in ecology. Results are benchmarked against approaches that exhaustively consider all networks to evaluate how well our method explores the relevant networks.

PP252 — SymScore: Machine Learning Accuracy Meets Transparency in a Symbolic Regression-Based Clinical Score Generator

SPEAKERS Olive Cawiding (Department of Mathematical Sciences, KAIST, Daejeon, 34141, Republic of Korea)
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SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F2
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Self-report questionnaires are widely used in healthcare to assess disease risk and symptom severity. However, their length can burden respondents and compromise data quality. While machine learning

models have enabled the development of shortened questionnaires with high predictive performance, they often operate as black boxes, limiting transparency and requiring specialized expertise that hinders clinical adoption. To address this, we have developed the Symbolic Regression-Based Clinical Score Generator (SymScore), a framework designed to produce score tables for shortened questionnaires while maintaining accuracy comparable to machine learning approaches. SymScore employs symbolic regression to optimize response grouping and assign predictive weights that capture the relationship between questionnaire responses and disease severity. The resulting score tables provide a transparent and practical tool for clinical use. SymScore achieves performance comparable to high-accuracy machine learning-based instruments, including MCQI-6 (MAE = 9.94, $R^2 = 0.82$) and SLEEPS (AUROC = 0.88–0.94), developed for assessing sleep disorders. Beyond these applications, SymScore has also been applied to questionnaires evaluating sleep-related cognitive dysfunction in patients with cancer. By combining predictive performance with interpretability, SymScore offers a practical pathway for translating advanced computational methods into trustworthy and accessible tools for healthcare professionals.

PP1627 — The Curse of Black Sigatoka: A Backward Bifurcation Perspective

SPEAKERS Bernard Asamoah Afful (Utah State University)
AUTHORS Bernard Asamoah Afful (Utah State University); Luis F. Gordillo (Utah State University)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B35
TRACK Mathematical Epidemiology

Abstract

Black Sigatoka disease is an airborne fungal infection caused by *Pseudocercospora fijiensis* that severely impacts global banana and plantain production. Its persistence and resistance to eradication make it one of the most challenging plant diseases to manage. We develop a deterministic host–pathogen model that captures BSD dynamics through dual transmission pathways and mate limitation in sexual reproduction. The model exhibits a backward bifurcation, where a stable endemic equilibrium coexists with the disease-free equilibrium even when $\mathcal{R}_0 < 1$. This result explains why control strategies based solely on reducing $\mathcal{R}_0$ may fail. We analyze the backward bifurcation regime using normalized forward sensitivity indices, Latin Hypercube Sampling, and Partial Rank Correlation Coefficients. The results show that host recruitment and susceptibility strongly drive endemic infection, while sanitation is a key control mechanism. A stochastic formulation using the Gillespie SSA captures variability in transmission dynamics. Simulations reveal that stochastic effects are significant near low-density thresholds, where extinction may occur despite deterministic persistence. Sobol sensitivity analysis further shows that variability in the endemic state is dominated by a few parameters, particularly κ, but also shaped by nonlinear interactions. These findings highlight the need for integrated control strategies combining sanitation and resistant plants.

PP296 — The role of individual capillaries for the brain's oxygen supply and the consequences of their occlusion

SPEAKERS Gaia Stievano (University of Bern)
AUTHORS Gaia Stievano (University of Bern); Timo Koch (University of Stuttgart); Franca Schmid (ARTORG University of Bern)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F8
TRACK Cardiovascular Modelling

Abstract

Maintaining a stable oxygen supply to the brain is essential to meet its high metabolic demands and sustain cognitive function. Assessing how microvascular changes affect cerebral energy supply is challenging due to complex capillary networks and small-scale vascular alterations. To address this, we developed a computational model of oxygen transport incorporating microvascular perfusion heterogeneity. Our approach employs a mixed-dimensional framework, representing the vascular network as a 1D graph embedded in 3D brain tissue. Unlike conventional models, it does not assume equilibrium between oxygen partial pressure (PO2) in red blood cells (RBCs) and plasma, capturing inter-vessel differences in oxygen unloading dynamics. Coupled non-linear PDEs are solved at steady state within the open-source framework DuMux [Koc+21]. The model computes PO2 in tissue, plasma, and RBCs across 13 tissue volumes (0.064 mm^3) obtained by two-photon microscopy in mice [Bli+13], each with $\sim$ 1100 capillaries. Tissue hypoxia is rare, affecting $\sim$ 15% of cells, mainly distal to descending arterioles or in low-vascular-density areas. Capillary stalling, linked to aging and disease, was assessed via 160 single-capillary occlusion simulations. While domain-averaged PO2 remained stable, local oxygenation patterns changed notably, with 27% of cells showing PO2 changes $>$ 1 mmHg. Strongest effects occurred in capillaries with high baseline flow [Sch+21] or high RBC oxygen saturation ($\sim$ 25% of cases).

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PP1271 — Thermodynamically Consistent Mechanochemical Modelling of Actin Dynamics

SPEAKERS Volkan Ozcoban (The University of Melbourne)
AUTHORS Michael Pan (University of New South Wales Sydney); Vijay Rajagopal (The University of Melbourne); Volkan Ozcoban (The University of Melbourne)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD F34
TRACK Cellular and Developmental Biology

Abstract

Actin assembly drives force generation and membrane remodelling (protrusion, budding, tethering). Many actin kinetic models omit explicit energy accounting with force production [VYO05; Dit+09], limiting reliable coupling to multiscale models of cell remodelling. Prior efforts linking actin kinetics to continuum mechanics often approximate energy flows and omit explicit nucleotide states [MO96; GPR25]. We present a modular, thermodynamically consistent actin model combining nucleation, polymerisation, depolymerisation, ATP/ADP states, and force production. Built using a bond graph approach (thermodynamically consistent ODEs) [GPR25; GC14], it automates energy accounting and provides modular ports for mechanical coupling. The model addresses three gaps: (i) energy-consistent ATP/ADP tracking, (ii) complete reaction sets for spontaneous actin dynamics with force coupling, and (iii) sensitivity-guided parameter estimation. Preliminary results demonstrate conservation of thermodynamic consistency, reproduce canonical behaviours (growth regimes, force-velocity relations, nucleotide turnover),

and show how Gibbs free energies tune force-energy relationships. This thermodynamically consistent scaffold lays a foundation for predictive multiscale studies of ATP consumption during cell migration and protrusion generation.

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PP1331 — Topological Data Analysis for Unsupervised Feature Selection in Large Scale Spatial Omics Data Sets

SPEAKERS James Boyle (University of Oxford)
AUTHORS James Boyle (University of Oxford); Gregory Hamm (AstraZeneca); Robin JG Hartman (AstraZeneca); Magnus Soderberg (AstraZeneca); Eleanor Williams (University of Cambridge); Ian Henry (AstraZeneca); Michael Casey (AstraZeneca)
SCHEDULE Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION Poster Presentations
BOARD B2
TRACK Numerical, Computational, and Data-Driven Methods

Abstract

Spatial transcriptomics studies are becoming increasingly large and commonplace, necessitating the simultaneous analysis of a large number of spatially resolved variables. Correspondingly, a diverse range of methodologies have been proposed to compare the spatial expression structure of genes. Here we apply persistent homology, a method from topological data analysis, to produce a continuous quantification of spatial structure in a given gene's expression, and show how this metric can be used for downstream tasks such as spatially variable gene identification. We explore the unique advantages of topology for this task, deriving biologically meaningful insights into kidney disease and myocardial infarction using public spatial transcriptomics data. We also show how the non-parametric nature of homology enables our methodology to extend naturally to other spatial omics modalities, demonstrating this on a spatial metabolomics sample. Our work showcases the advantages of using a continuous quantification of spatial structure over p-value based approaches to spatially variable gene identification, the potential for developing unified methods for the analysis of different spatial omics modalities, and the utility of persistent homology in big data applications.

PP1316 — Turing Instability in a Reaction-Diffusion Predator-Prey Model with Prey Infection and Intraspecific Predator Competition

SPEAKERS Mark Lois Fortin (University of the Philippines Diliman)
AUTHORS Gervy Marie Angeles (University of the Philippines Diliman); Mark Lois Fortin (University of the Philippines Diliman)

SCHEDULE	Thursday, 16 July 2026; 20:10–20:30; ReSoWi Building
SESSION	Poster Presentations
BOARD	B1
TRACK	Population Dynamics, Ecology & Evolution

Abstract

This study investigates a predator–prey system incorporating a transmissible disease that spreads exclusively within the prey population and intraspecific competition among predators. The dynamics are modeled using a system of reaction–diffusion equations to account for both local interactions and spatial movement. In this system, we focus on the occurrence of diffusion-driven instability, or Turing instability. Using linear stability analysis, we determine parameter regimes under which diffusion destabilizes the stable homogeneous interior equilibrium. This instability can lead to the emergence of spatially heterogeneous structures known as Turing patterns, which may correspond to clustering phenomena observed in ecological systems. Numerical simulations are performed in a two-dimensional spatial domain in order to illustrate these dynamics. The results demonstrate the emergence of complex spatial patterns and emphasize the significant role of diffusion coefficients in shaping spatial heterogeneity. These findings provide insight into the mechanisms underlying spatial pattern formation in predator–prey interactions influenced by prey infection and predator competition.

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