DSABNS 2022 Virtual

13th International Conference
Dynamical Systems Applied to Biology
and Natural Sciences (DSABNS)

BOOK OF ABSTRACTS

Basque Center for Applied Mathematics
BCAM, Bilbao, Spain

ISBN: 978-989-98750-9-8

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The Virtual 13th Conference on Dynamical Systems Applied to Biology and Natural Sciences, DSABNS 2022, was hosted by the Mathematical and Theoretical Biology Group (MTB) at the Basque Center for Applied Mathematics in Bilbao, Basque Country, Spain, on February 8-11, 2022.

The International Conference "Dynamical Systems Applied to Biology and Natural Sciences – DSABNS" is a well established International Scientific event that has been organized since 2010, every year during the month of February. The program includes Public Lectures providing a public forum for academic researchers from diverse disciplinary backgrounds; Plenary talks which are scheduled to open and to close the scientific program (morning and afternoon); Invited talks are meant to provide context for a parallel session which is filled with Contributed Talks given by the conference participants. The conference programme covers research topics in scientific areas such as population dynamics, eco-epidemiology, epidemiology of infectious diseases, molecular and antigenic evolution and methodological topics in the natural sciences and mathematics. Poster Sessions are also part of the conference which also counts with this Book of Abstracts with ISBN.

Local Organizers: Maíra Aguiar, BCAM; Vizda Anam, BCAM; Nicole Cusimano, BCAM; Carlo Estadilla, BCAM; Bruno Guerrero, BCAM; Damián Knopoff, BCAM; Mauricio Rincón Bonilla, BCAM; Akhil Kumar Srivastav, BCAM; Vanessa Steindorf, BCAM; Nico Stollenwerk, BCAM.

International Organizers: Carlos Braumann, UE; Bob Kooi, VU; Paula Patrício, UNL; Andrea Pugliese, UniTN; Lucia Russo, UniNa; Costantinos Siettos, UniNa; Ezio Venturino, UT.

NB. BCAM: Basque Center for Applied Mathematics, Basque Country, Spain; UniTN: Università degli Studi di Trento, Italy; UE: Universidade de Évora, Portugal; UNL: Universidade Nova de Lisboa, Portugal; VU: Vrije Universiteit Amsterdam, The Netherlands; UT: Università degli Studi di Torino, Italy; UniNa: Università degli Studi di Napoli Federico II, Italy.

Sponsors: The organizers are grateful for the sponsorship and support of the Basque Center for Applied Mathematics (BCAM) and its Mathematical and Theoretical Biology working group who have hosted the Conference. This event was supported by the Basque Government through the BERC 2018-2021 program and by Spanish Ministry of Sciences, Innovation and Universities: BCAM Severo Ochoa accreditation SEV-2017-0718, by the European Society for Mathematical and Theoretical Biology (ESMTB) and the by the Consiglio Nazionale delle Ricerche, Italy (CNR-STEMS). We thank the Basque Foundation for Science (Ikerbasque).

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DYNAMICAL SYSTEMS APPLIED TO BIOLOGY AND NATURAL SCIENCES

DSABNS 2022

February 8-11, 2022

Virtual

PUBLIC LECTURES

Roberto Natalini - Istituto per le Applicazioni del Calcolo "M. Picone"
Consiglio Nazionale delle Ricerche, Italy

Horacio Rotstein - Federated Department of Biological Sciences and the Institute for Brain and Neuroscience Research
New Jersey Institute of Technology, Rutgers University, USA

PLENARY SPEAKERS

Maíra Aguiar - BCAM, Spain
Malay Banerjee - Indian Institute of Technology Kanpur, India
Nicola Bellomo - Universidad de Granada, Spain
Konstantin Blyuss - University of Sussex, UK
Carlos Braumann - Universidade de Évora, Portugal
Aaaron King - University of Michigan, USA
Yuliya Kyrychko - University of Sussex, UK
Giovanni Marini - Edmund Mach Foundation of San Michele all'Adige, Italy
Carla Pinto - Polytechnic Institute of Porto, Portugal
Ganna Rozhnova - University Medical Center Utrecht, The Netherlands
Lucia Russo - Consiglio Nazionale delle Ricerche, CNR-STEMS, Italy
Costas Siettos - Università degli Studi di Napoli Federico II, Italy
Nico Stollenwerk - BCAM, Spain
Ezio Venturino - Università degli studi di Torino, Italy

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Mauricio Rincón Bonilla - BCAM, Spain
Akhil Kumar Srivastav - BCAM, Spain
Vanessa Steindorf - BCAM, Spain
Nico Stollenwerk - BCAM, Spain

Registration is free but required.

Abstract submission deadline: December 3, 2021

More info at:
https://sites.google.com/view/dsabns2022/home

Contact: dsabns2022@bcamath.org

INTERNATIONAL ORGANIZING COMMITTEE

Carlos Braumann - Évora University, Portugal
Bob Kooi - Vrije University Amsterdam, The Netherlands
Paula Patrício - Universidade Nova de Lisboa, Portugal
Andrea Pugliese - Università degli Studi di Trento, Italy
Lucia Russo - Consiglio Nazionale delle Ricerche, CNR-STEMS, Italy
Costantinos Siettos - Università degli Studi di Napoli Federico II, Italy
Ezio Venturino - Università degli Studi di Torino, Italy

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Costas Siettos - Università degli Studi di Napoli Federico II, Italy
Nico Stollenwerk - BCAM, Spain
Ezio Venturino - Università degli studi di Torino, Italy

INVITED SPEAKERS

Thomas Götz - Universität Koblenz-Landau, Germany
Damián Knopoff - BCAM, Spain
Bob W. Kooi - Vrije Universiteit Amsterdam, The Netherlands
Dae-Jin Lee - BCAM, Spain
Andrey Morozov - University of Leicester, UK
Gianni Pagnini - BCAM, Spain
Paula Patrício - Faculdade de Ciências e Tecnologia UNL, Portugal
Jean-Christophe Poggiale - Aix-Marseille University, France
Andrea Pugliese - Università degli studi di Trento, Italy
Mauricio Rincón Bonilla - BCAM, Spain
Josep Sardanyes - Centre de Recerca Matemàtica, Spain
Urszula Skwara - Maria Curie-Sklodowska University in Lublin, Poland
Edy Soewono - Institut Teknologi Bandung, Indonesia
Max Souza - Universidade Federal Fluminense, Brazil
Abdessamad Tridane - Al Ain, UAE
Maria Enrica Virgillito - Scuola Superiore Sant'Anna, Italy

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SCIENTIFIC PROGRAM

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Central European Time (CET)	8-Feb	9-Feb	10-Feb	Central European Time (CET)	11-Feb
12:45	Opening				
13:00-13.40	Webinar: LECTURE 1 (Public)	Webinar: LECTURE 5 (Public)	Webinar: LECTURE 9 (Plenary)	13:00-13.40	Webinar: LECTURE 13 (Plenary)
13:45-14:25	Webinar: LECTURE 2 (Plenary)	Webinar: LECTURE 6 (Plenary)	Webinar: LECTURE 10 (Plenary)	13:45-14:25	Webinar: LECTURE 14 (Plenary)
14:25-14:40	coffee break	coffee break	coffee break	14:25-14:40	coffee break
14:40-15:10	5 zoom parallel sessions: INVITED Talks 1-5	5 zoom parallel sessions: INVITED Talks 6-10	5 zoom parallel sessions: INVITED Talks 11-15	14:40-15:10	5 zoom parallel sessions: INVITED Talks 16-20
15:10-16:30	5 zoom parallel sessions: CONTRIBUTED Talks (4CT)	5 zoom parallel sessions: CONTRIBUTED Talks (4CT)	5 zoom parallel sessions: CONTRIBUTED Talks (4CT)	15:10-16:30	5 zoom parallel sessions: CONTRIBUTED Talks (4CT)
16:30-16:40	coffee break	coffee break	coffee break	16:30-16:40	coffee break
16:40-17:20	Webinar: LECTURE 3 (Plenary)	Webinar: LECTURE 7 (Plenary)	Webinar: LECTURE 11 (Plenary)	16:40-17:20	Webinar: LECTURE 15 (Plenary)
17:30-18:50	4 zoom parallel sessions: CONTRIBUTED Talks (4CT)	5 zoom parallel sessions: POSTER Presentations (11P)	4 zoom parallel sessions: CONTRIBUTED Talks (4CT)	17:25-18:05	Webinar: LECTURE 16 (Plenary)
19:00-19:40	Webinar: LECTURE 4 (Plenary)	Webinar: LECTURE 8 (Plenary)	Webinar: LECTURE 12 (Plenary)	18:15-19:15	Round Table
19:40	CLOSING	CLOSING	CLOSING	19:15-19:30	PRIZES & CLOSING

13th Conference DSABNS | Feb. 8- 11, 2022 – Main schedule

Important note: all Public Lectures, Plenary Lectures and the Round Table will be hosted on Zoom license 1

Tuesday - February 8, 2022

12:45 – 13:00	**OPENING**		
	Chair: Maíra Aguiar		
13:00 – 13:40	**Public lecture**	Horacio G. Rotstein	*Resonance-based mechanisms of generation of oscillations in networks of non-oscillatory neurons*
13:45 – 14:25	**Plenary lecture**	Yuliya Kyrychko	*Dynamics of coupled Kuramoto oscillators with distributed delays*
14:25 – 14:40	COFFEE BREAK		
14:40 – 15:10	**Invited talks**		
	Zoom license 1 Chair: Horacio Rotstein	Andrea Pugliese	*Effect of sharp changes in contact rates in epidemic models*
	Zoom license 2 Chair: Carlos Braumann	Damián Knopoff	*Spatial epidemiological models of infectious disease transmission*
	Zoom license 3 Chair: Mimmo Ianelli	Andrew Morozov	*Mathematical modelling of control of pathogenic bacteria by a phage with a temperature-dependent life cycle*
	Zoom license 4 Chair: Ezio Venturino	Jean-Christophe Poggiale	*Population growth in a patchy environment*
	Zoom license 5 Chair: Paula Patrício	Alberto d'Onofrio	*Role of chemotaxis in tumor angiogenesis*
15:10 – 16:30	**Parallel session A – Contributed Talks**		
16:30 – 16:40	COFFEE BREAK		
	Chair: Alberto d'Onofrio		
16:40 – 17:20	**Plenary lecture**	Malay Banerjee	*Effect of slow-fast time scale and nonlocal interaction on spatiotemporal pattern formation*
17:30 – 18:50	**Parallel session B – Contributed Talks**		
	Chair: Jean-Christophe Poggiale		
19:00 – 19:40	**Plenary lecture**	Carlos Braumann	*Harvesting profit optimization on random environments: the effects of Allee effects*
19:40	**CLOSING**		

Parallel session A – The LAST speaker will Chair the first 3 contributed talks and the FIRST speaker will Chair the last contributed talk

Parallel session A – Zoom license 1 - Epidemiology			
15:10 – 15:30	Contributed talk 1	Martina Amongero	*Analysing the COVID-19 pandemic in Italy with the SIPRO model*
15:30 – 15:50	Contributed talk 2	Vijay Pal Bajiya	*Mathematical modeling and optimal control strategies for COVID-19 in India: impact of face mask and quarantine*
15:50 – 16:10	Contributed talk 3	Cynthia De Oliveira Lage Ferreira	*Challenges in COVID-19 dynamics and diagnostic in the State of Rio de Janeiro*
16:10 – 16:30	Contributed talk 4	Stefanella Boatto	*COVID-19 epidemics strategies: mobility, testing and vaccination shot dynamics*

Parallel session A – Zoom license 2 - Ecology			
15:10 – 15:30	Contributed talk 5	Purnedu Mishra	*The role of indirect predator-taxis in an extended Schoener's intraguild predator-prey model*
15:30 – 15:50	Contributed talk 6	Nayana Mukherjee	*Effect of hunting cooperation on spatio-temporal pattern formation in prey-predator models*
15:50 – 16:10	Contributed talk 7	Amar Sha	*Pattern formation in a community model with predation fear and density dependent death rate of predator*
16:10 – 16:30	Contributed talk 8	Pranali Roy Chowdhury	*Temporal and spatio-temporal dynamics in a slow-fast predator-prey system*

Parallel session A – Zoom license 3 – Miscellaneous			
15:10 – 15:30	Contributed talk 9	Tahmineh Azizi	*Using fractal geometry to quantify the complexity of nature*
15:30 – 15:50	Contributed talk 10	Juan Calvo	*Model calibration for aggregation-sedimentation dynamics*
15:50 – 16:10	Contributed talk 11	Mauro Garavello	*Well posedness and control in balance laws models inspired by biology*
16:10 – 16:30	Contributed talk 12	Alejandro Rojas	*Mathematical modeling of irrigation scheduling influence in crop growth*

Parallel session A – Zoom license 4 - Cell Dynamics			
15:10 – 15:30	Contributed talk 13	Emine Atici Endes	*Analysing the impact of cellular adhesion on keratinocyte growth factor activity in epidermal wound*
15:30 – 15:50	Contributed talk 14	Giulia Belluccini	*A multi-stage model of cell proliferation and death: tracking cell divisions with Erlang distributions*
15:50 – 16:10	Contributed talk 15	Silvia Berra	*Mathematical model for computing colorectal cancer cell's steady state for drug repositioning and dosage*
16:10 – 16:30	Contributed talk 16	Salvador Chulián	*Dynamics of feedback signalling in B lymphocytes development*

Parallel session A – Zoom license 5 - Methodology in Biomathematics			
15:10 – 15:30	Contributed talk 17	Hristo Inouzhe Valdes	*The time-varying reproduction number of SARS-COV-2 in SIR and SEIR-like models: a Hamiltonian Monte Carlo approach*
15:30 – 15:50	Contributed talk 18	Sandeep Kumar	*Discovering Lagrangian coherent structures in the human abdomen*
15:50 – 16:10	Contributed talk 19	Thi Minh Thao Le	*Quasi-neutral dynamics in a coinfection system with n strains and asymmetries along multiple traits*
16:10 – 16:30	Contributed talk 20	M. Angeles Martínez Carballo	*Dynamical analysis of early after depolarization pattern in the 27D cardiac Sato model*

Parallel session B – The LAST speaker will Chair the first 3 contributed talks and the FIRST speaker will Chair the last contributed talk

Parallel session B – Zoom license 1 - Ecology			
17:30 – 17:50	Contributed talk 21	Masoom Bhargava	*Mathematical modelling to control wild mosquito with sterile release*
17:50 – 18:10	Contributed talk 22	Arnab Chattopadhyay	*Impact of environmental toxicity and infectious disease on consumer population*
18:10 – 18:30	Contributed talk 23	Isabel Coelho	*Seasonally dependent competitive Kolmogorov systems: extinction or coexistence?*
18:30 – 18:50	Contributed talk 24	Bradly Deeley	*Analytical and computational study of the spread of invasive species in the presence of a road in the spatial domain*

Parallel session B – Zoom license 2 – Epidemiology			
17:30 – 17:50	Contributed talk 25	CANCELED	*CANCELED*
17:50 – 18:10	Contributed talk 26	Tanuja Das	*Mathematical modeling of impact of screening and saturated treatment on spread of COVID-19: a case study of India*
18:10 – 18:30	Contributed talk 27	Carlo Delfin Estadilla	*Impact of vaccine supplies and delays on optimal control of the COVID-19 pandemic: mapping interventions for the Philippines*
18:30 – 18:50	Contributed talk 28	CANCELED	*CANCELED*

Parallel session B – Zoom license 3 – Epidemiology			
17:30 – 17:50	Contributed talk 29	CANCELED	*CANCELED*
17:50 – 18:10	Contributed talk 30	Vizda Anam	*Modeling dengue immune responses mediated by antibodies: insights on the biological parameters to describe dengue infections*
18:10 – 18:30	Contributed talk 31	Naba Kumar Goswami	*The role of media on the dynamics of Zika outbreak: A modeling approach*
18:30 – 18:50	Contributed talk 32	Carlo Giambiagi Ferrari	*Coupling a SIS model with opinion dynamics*

Parallel session B – Zoom license 4 - Neuroscience			
17:30 – 17:50	Contributed talk 33	Ximena Fernandez	*Topological methods for real time detection of epileptic seizures in electroencephalographic recordings*
17:50 – 18:10	Contributed talk 34	Maria Leite	*Boolean framework for studying the control of breathing*
18:10 – 18:30	Contributed talk 35	Frank Llovera	*Periodic and chaotic dynamics in a map-based neuron model*
18:30 – 18:50	Contributed talk 36	CANCELED	*CANCELED*

13th Conference DSABNS | Feb. 8- 11, 2022 – Main schedule

Important note: all Public Lectures, Plenary Lectures and the Round Table will be hosted on Zoom license 1

Wednesday - February 9, 2022

12:45 – 13:00	**OPENING**		
	Chair: Lucia Russo		
13:00 – 13:40	**Public lecture**	Roberto Natalini	*A mathematical path towards the understanding of cells' aggregations and morfogenesis*
13:45 – 14:25	**Plenary lecture**	Konstantin Blyuss	*Dynamics of a predator-prey model with ratio dependence and Holling type III functional response*
14:25 – 14:40	COFFEE BREAK		
14:40 – 15:10	**Invited talks**		
	Zoom license 1 Chair: Maíra Aguiar	Bob W. Kooi	*Analysis of an epidemic model with one recurrent infection*
	Zoom license 2 Chair: Damián Knopoff	Mimmo Iannelli	*Dynamics of multi-phasic epidemics: COVID-19 immunization in Italy as case study*
	Zoom license 3 Chair: Ezio Venturino	Josep Sardanyes	*Long transients in ecology arising from local and global bifurcations*
	Zoom license 4 Chair: Carlos Braumann	Gianni Pagnini	*Should I stay or should I go? Zero-size jumps in random walks for Lévy flights*
	Zoom license 5 Chair: Max Souza	Thomas Götz	*Coupling and optimizing hybrid models (CANCELED)*
15:10 – 16:30	**Parallel session A – Contributed Talks**		
16:30 – 16:40	COFFEE BREAK		
	Chair: Carla Pinto		
16:40 – 17:20	**Plenary lecture**	Lucia Russo	*Agent based models for urban mobility: emergent behaviour and bifurcation analysis*
17:30 – 18:50	**Parallel session B: Poster Sessions**		
	Chair: Aaron King		
19:00 – 19:40	**Plenary lecture**	Maíra Aguiar	*On the origin of complex dynamics in multi-strain dengue models*
19:40	**CLOSING**		

Parallel session A – The LAST speaker will Chair the first 3 contributed talks and the FIRST speaker will Chair the last contributed talk

Parallel session A – Zoom license 1 - Epidemiology			
15:10 – 15:30	Contributed talk 37	Sarita Bugalia	*Mathematical modeling of intervention and low medical resource availability with delays: applications to COVID-19 outbreaks in Spain and Italy*
15:30 – 15:50	Contributed talk 38	Augusto Cabrera-Becerril	*An agent-based model for COVID-19 in urban context*
15:50 – 16:10	Contributed talk 39	Mahmoud Ibrahim	*Mathematical modelling of COVID-19 transmission between humans and minks*
16:10 – 16:30	Contributed talk 40	Amira Kebir	*Optimal control of vaccination for age-structured SEIR model of COVID-19*

Parallel session A – Zoom license 2 - Ecology			
15:10 – 15:30	Contributed talk 41	Anupam Priyadarshi	*Existence of highly patchy phytoplankton distributions requires modeling with at least one higher level predators*
15:30 – 15:50	Contributed talk 42	Buddhadev Ranjit	*Dynamics of plankton in coral-reef ecosystem in presence of disease*
15:50 – 16:10	Contributed talk 43	Luca Rossini	*A general ODE-based model to describe the physiological age structure of ectotherms: description and application to Drosophila suzukii*
16:10 – 16:30	Contributed talk 44	Parimita Roy	*Deciphering the role of zooplankton in supporting frog population*

Parallel session A – Zoom license 3 - Methodology in Biomathematics			
15:10 – 15:30	Contributed talk 45	Thomas Woolley	*Bespoke Turing systems*
15:30 – 15:50	Contributed talk 46	Pierre Marie Ngougoue Ngougoue	*Can selection on histidine-rich protein 2/3 gene deletions be detected by traces of selection?*
15:50 – 16:10	Contributed talk 47	Schehrazad Selmane	*A Phlebotomus papatasi sand fly life cycle model*
16:10 – 16:30	Contributed talk 48	Lea Sta	*Control of IL-7 responsiveness through IL-7R subunits balance in effector T cells and analytic expressions for amplitude and EC50*

Parallel session A – Zoom license 4 - Cell Dynamics			
15:10 – 15:30	Contributed talk 49	Hammed Olawale Fatoyinbo	*Effects of ion channels conductance on spontaneous electrical activity in smooth muscles*
15:30 – 15:50	Contributed talk 50	Flavia Feliciangeli	*Why are cell populations maintained via multiple intermediate compartments?*
15:50 – 16:10	Contributed talk 51	Nóra Juhász	*A hybrid PDE-ABM model for viral dynamics with application to SARS-CoV-2 and influenza*
16:10 – 16:30	Contributed talk 52	Daniel Luque	*Stochastic modelling of TCR-epitope recognition in the context of cross-reactivity*

Parallel session A – Zoom license 5 - Epidemiology			
15:10 – 15:30	Contributed talk 53	Biplab Maity	*Quantification of optimal resource allocation towards controlling epidemics*
15:30 – 15:50	Contributed talk 54	Erida Gjini	*Disentangling how multiple traits drive 2 strain frequencies in SIS dynamics with coinfection*
15:50 – 16:10	Contributed talk 55	Vanessa Steindorf	*Modelling secondary infections with cross-protection and disease enhancement factor*
16:10 – 16:30	Contributed talk 56	Peter Rashkov	*Optimal control applied to a model for vector-borne disease*

Parallel session B – Zoom license 1 – Ecology & Miscellaneous			
17:30 – 18:50 Poster presentation lasts 6 minutes: 4 min presentation followed by 2 min questions. Chair: Maíra Aguiar	Poster 1	Marta Biancardi	*An evolutionary game on compliant and non- compliant firms in groundwater exploitation*
	Poster 2	Cyrine Chenaoui	*An agent-based model for assessing the effects of climate variations on ticks population dynamics*
	Poster 3	Yann Ligeiro	*Alpha male effect on group population dynamics*
	Poster 4	CANCELED	*CANCELED*
	Poster 5	Blai Vidiella Rocamora	*Transients and ghosts in collapsing ecosystems*
	Poster 6	Vojtech Kumpost	*Intrinsic noise facilitates population synchronization of uncoupled cellular oscillators to external signals*
	Poster 7	Suzielli Mendonça	*Application of generalized logarithm and exponential functions in multi-fractal detrended fluctuation analysis (MFDFA) of nonstationary time series for medical signal analysis*
	Poster 8	CANCELED	*CANCELED*
	Poster 9	Sinchan Ghosh	*Investigating the roles of helpers in cooperative breeding migratory bird population dynamics*
	Poster 10	Alessandro Triacca	*Predicting soil loss and runoff in vineyards using a gradient boosting framework*

Parallel session B – Zoom license 2 - Methodology in Biomathematics			
17:30 – 18:50 Poster presentation lasts 6 minutes: 4 min presentation followed by 2 min questions. Chair: Damián Knopoff and Rubén Blasto	Poster 11	Eucharia Nwachukwu	*Analytical method of human systemic and global circulation*
	Poster 12	Patrícia Antunes	*Bi-additive models and flood flows*
	Poster 13	Ruddy Urbina	*Breath figures on surfaces with different density of nucleation sites*
	Poster 14	Walid Ben Aribi	*A delayed stochastic SEIRDS epidemic model with temporary immunity*
	Poster 15	Glaucia Bressan	*A fuzzy system to describe the effects of vaccination on COVID-19 dynamics in Brazil*
	Poster 16	Inmaculada Lopez	*Monitoring of single-species population systems*
	Poster 17	Edgar Lourenço	*The role of the innate and adaptive immune system in a mathematical model for non-small cell lung cancer*
	Poster 18	Karmand Khdr Ahmad	*Mathematical model of hearing loss caused by noise hazard*
	Poster 19	Andreas Sofianos	*Analytical and numerical bifurcation analysis of TLR4 signaling dynamics: from oscillations to homoclinic explosions*
	Poster 20	CANCELED	*CANCELED*

Parallel session B – Zoom license 3 – Epidemiology			
17:30 – 18:50 Poster presentation lasts 6 minutes: 4 min presentation followed by 2 min questions. Chair: Nicole Cusimano and Vizda Anam	Poster 21	Shraddha Ramdas Bandekar	*Modeling and analysis of COVID-19 in India with treatment function through different phases of lockdown and unlock*
	Poster 22	Naffeti Bechir	*Comparative study of the basic reproductive number and the transmission rates between the African country for the first three waves : Impact of the economic, demographic and climatic factors*
	Poster 23	Adriano Henrique Danhoni Neves	*Modeling COVID-19 dynamics: a comparison between time-dependent parameters*
	Poster 24	Attila Dénes	*Global dynamics of a compartmental model to assess the effect of transmigration from deceased*
	Poster 25	Dhafer Ferchichi	*Agent based modeling for West Nile virus spreading*
	Poster 26	Letícia Ferreira Godoi	*Optimization applied to transmission of hospital infection*
	Poster 27	Silvia García	*A mathematical model for HIV/AIDS*
	Poster 28	Egberanmwen Barry Iyare	*Global stability of the DFE for a vector transmitted and transfusion transmitted malaria disease*
	Poster 29	Marina Lima	*Mathematical modeling and simulations of COVID-19 in Brazil*
	Poster 30	CANCELED	*CANCELED*

Parallel session B – Zoom license 4 - Miscellaneous			
17:30 – 18:50 Poster presentation lasts 6 minutes: 4 min presentation followed by 2 min questions. Chair: Bruno Guerrero and Carlo Estadilla	Poster 31	Vitaly Sakovich	*Method of evaluation of straine to estimate the dysfunction of heart chambers of patients with ASD*
	Poster 32	Alexandr Sychev	*Microbial population growth indication by coupled electro-optical and chemical dynamic system*
	Poster 33	Miruna-Stefana Sorea	*Disguised toric dynamical systems*
	Poster 34	CANCELED	*CANCELED*
	Poster 35	Yazid Bensid	*Mathematical analysis for a time-delayed Alzheimer disease model*
	Poster 36	Julieta Ruiz	*Temporal dynamics of spike trains in the raphe nuclei during urethane sub-states in rats*
	Poster 37	Baeckkyoung Sung	*Mathematical modelling of the fish neuroendocrine dynamics*
	Poster 38	Viktoriya Fedotovskaya	*Triplet distribution of common mitochondrial and chloroplast genes of plants*
	Poster 39	Maria Eliza Antunes	*Mathematical model of tumor growth under the influence of the immune system and estrogen with chemotherapy treatment*
	Poster 40	CANCELED	*CANCELED*

Parallel session B – Zoom license 5 – Epidemiology and Evolution			
17:30 – 18:50 Poster presentation lasts 6 minutes: 4 min presentation followed by 2 min questions. Chair: Vanessa Steindorf and Akhil Srivastav	Poster 41	Amer Salman	*The dynamics of the SIRS pde epidemic model*
	Poster 42	Amit Sharma	*Modeling and analysis of delay induced impulsive vaccination to control the spread of COVID-19*
	Poster 43	Ian Teixeira	*In or out: how lockdown affects the SARS-COV-2 progression in Portugal*
	Poster 44	Tamás Tekeli	*Comparing COVID-19 testing and other non-pharmaeutical interventions*
	Poster 45	Naima Aissa	*Analysis of a nonlinear parabolic problem*
	Poster 46	Hanaa Zitane	*Optimal control and stabilization of bilinear dynamical systems on time scales*
	Poster 47	Raphael Garcia	*Three species competition: a study about the complexity of different solutions from the numerical point of view*
	Poster 48	Bruno Buffa	*A kinetic model coupling crowd movement with infectious disease contagion*
	Poster 49	CANCELED	*CANCELED*
	Poster 50	CANCELED	*CANCELED*

13th Conference DSABNS | Feb. 8- 11, 2022 – Main schedule

Important note: all Public Lectures, Plenary Lectures and the Round Table will be hosted on Zoom license 1

Thursday – February 10, 2022

12:45 – 13:00	**OPENING**		
	Chair: Konstantin Blyuss		
13:00 – 13:40	**Plenary lecture**	Nicola Bellomo	*Pandemic of mutating virus, a multi-scale "active particles" systems approach*
13:45 – 14:25	**Plenary lecture**	Costas Siettos	*Numerical solution of partial differential equations and stiff problems of ODEs with physics informed random projection networks and extreme learning machines*
14:25 – 14:40	COFFEE BREAK		
14:40 – 15:10	**Invited talks**		
	Zoom license 1 Chair: Yuliya Kyrychko	Mirjam Kretzschmar	*Epidemic cycles and transition to health-positive opinion in a coupled health-opinion and disease transmission model*
	Zoom license 2 Chair: Nicola Bellomo	Maria Enrica Virgillito	*A multiscale network-based model of contagion dynamics: heterogeneity, spatial distancing and vaccination*
	Zoom license 3 Chair: Mimmo Iannelli	Abdessamad Tridane	*Modeling Heroin epidemic and impact of awareness programme*
	Zoom license 4 Chair: Gianni Pagnini	Mauricio Rincón Bonilla	*Atomistic simulation of polymer/ceramic composite solid-state electrolytes: the case of PEO (LiTFSI):LLZO*
	Zoom license 5 Chair: Edy Soewono	Urszula Skwara	*Mathematical modelling of COVID-19*
15:10 – 16:30	**Parallel session A – Contributed Talks**		
16:30 – 16:40	COFFEE BREAK		
	Chair: Malay Banerjee		
16:40 – 17:20	**Plenary lecture**	Ezio Venturino	*A model for the olive tree pest Prays oleae (Bernard)*
17:30 – 18:50	**Parallel session B – Contributed Talks**		
	Chair: Costas Siettos		
19:00 – 19:40	**Plenary lecture**	Carla Pinto	*Patterns of T1D onset on HIV infected patients after immune reconstitution*
19:40	**CLOSING**		

Parallel session A – The LAST speaker will Chair the first 3 contributed talks and the FIRST speaker will Chair the last contributed talk

Parallel session A – Zoom license 1 - Immunology & Neurosciences			
15:10 – 15:30	Contributed talk 57	Clemence Reda	*Gene-network oriented drug discovery: automated inference of boolean networks for drug target prediction*
15:30 – 15:50	Contributed talk 58	Benjamin Ambrosio	*Qualitative analysis of some reaction-diffusion systems in neuroscience context*
15:50 – 16:10	Contributed talk 59	Farzad Fatehi	*Comparative analysis of different therapeutic strategies against HBV infection using an age-structured model*
16:10 – 16:30	Contributed talk 60	Alberto José Ferrari	*A segmentary interpolation numerical method applied to a fractional order model for the treatment of HIV infection*

Parallel session A – Zoom license 2 – Eco-Epidemiology			
15:10 – 15:30	Contributed talk 61	Kaushik Kayal	*Allee effects driven by predation of an eco-epidemiological model*
15:30 – 15:50	Contributed talk 62	Ilenia Luiso	*A novel model for soil organic carbon change: sensitivity analysis of SOC change index to changes of temperature, net primary production and land use*
15:50 – 16:10	Contributed talk 63	Nuno Brites	*Optimal sustainable harvesting policies in random environments: estimation of moments and density of first passage times*
16:10 – 16:30	Contributed talk 64	Oumaima Laraj	*Mathematical model of anaerobic digestion with leachate recirculation*

Parallel session A – Zoom license 3 - Methodology in Biomathematics			
15:10 – 15:30	Contributed talk 65	Richmond Opoku-Sarkodie	*The dynamics of an SIRWS system with asymmetric partition of immunity period*
15:30 – 15:50	Contributed talk 66	Woldegebriel Assefa Woldegerima	*Parameter and state estimation based on observer construction method for a cholera model with threshold immunology*
15:50 – 16:10	Contributed talk 67	CANCELED	*CANCELED*
16:10 – 16:30	Contributed talk 68	Jason Whyte	*Structural identifiability analysis for switching system structures: towards a toolkit for changing times*

Parallel session A – Zoom license 4 - Cell Dynamics			
15:10 – 15:30	Contributed talk 69	Ana del Rosario Niño-López	*Evolution of B lymphocytes leukemia and its treatment*
15:30 – 15:50	Contributed talk 70	Lorand Gabriel Parajdi	*On the controllability of a system modeling cell dynamics related to leukemia*
15:50 – 16:10	Contributed talk 71	Ana Portillo	*Mathematical modeling of human follicle aging*
16:10 – 16:30	Contributed talk 72	Renata Retkute	*Cell planting using glass beads: experimental and modeling study*

Parallel session A – Zoom license 5 – Epidemiology			
15:10 – 15:30	Contributed talk 73	Ram Singh	*Mathematical modeling and analysis of COVID-19 and dengue co-infection*
15:30 – 15:50	Contributed talk 74	Simon Syga	*Inferring the effect of interventions on COVID-19 transmission networks*
15:50 – 16:10	Contributed talk 75	Akhil Kumar Srivastav	*Initial phase of COVID-19 epidemic in the Basque country: a modeling study*
16:10 – 16:30	Contributed talk 76	Subhendu Paul	*Estimation of COVID-19 recovery and decease periods in Canada*

Parallel session B – The LAST speaker will Chair the first 3 contributed talks and the FIRST speaker will Chair the last contributed talk

Parallel session B – Zoom license 1 - Epidemiology			
17:30 – 17:50	Contributed talk 77	Rinaldo M. Colombo	*Macroscopic modeling of vaccination campaigns*
17:50 – 18:10	Contributed talk 78	Alina Glaubitz	*A tale of two vaccine efficacies: Simpson's paradox and bifurcations*
18:10 – 18:30	Contributed talk 79	Mariajesus Lopez-Herrero	*Setting a warning vaccination level on a stochastic model with infection reintroduction when vaccine is partially effective*
18:30 – 18:50	Contributed talk 80	Fernando Saldaña	*How unequal vaccine distribution promotes the evolution of vaccine escape: insights from a simple model*

Parallel session B – Zoom license 2 - Immunology			
17:30 – 17:50	Contributed talk 81	Teddy Lazebnik	*Mathematical model for the BCG-based treatment of type 1 diabetes*
17:50 – 18:10	Contributed talk 82	Macauley Locke	*Quantification of type I interferon inhibition by viral proteins: Ebola virus as a case study*
18:10 – 18:30	Contributed talk 83	Rakesh Pandey	*Modeling the role of cytokines in the pathogenesis of psoriasis*
18:30 – 18:50	Contributed talk 84	Bevelynn Williams	*A stochastic intracellular model of anthrax infection with germination heterogeneity*

Parallel session B – Zoom license 3 – Epidemiology			
17:30 – 17:50	Contributed talk 85	Paul Georgescu	*Off-label use of epidemic modelling: assessing peer influence on student performance and dropout*
17:50 – 18:10	Contributed talk 86	Alice Castelhano	*SEIRS reaction-diffusion model analysis and application*
18:10 – 18:30	Contributed talk 87	Nicole Cusimano	*Human mobility and multi-patch models of infectious disease dynamics*
18:30 – 18:50	Contributed talk 88	Andrew Bate	*Modeling viral exposure in transport settings*

Parallel session B – Zoom license 4 - Methodology in Biomathematics			
17:30 – 17:50	Contributed talk 89	Ismail El Hakki	*The influence of a demand function on the global dynamics of the fish stocks*
17:50 – 18:10	Contributed talk 90	Rafael González-Albaladejo	*Flock formation constrained by boundary conditions or by a confining potential*
18:10 – 18:30	Contributed talk 91	Bruno Valdemar Guerrero Borges	*Quantifying anomalous diffusion and nonergodicity in the unclogging dynamics from experimental findings*
18:30 – 18:50	Contributed talk 92	Gabriele Grifo'	*Eckhaus instability of stationary patterns in hyperbolic vegetation models on large finite domains*

13th Conference DSABNS | Feb. 8- 11, 2022 – Main schedule

Important note: all Public Lectures, Plenary Lectures and the Round Table will be hosted on Zoom license 1

Friday - February 11, 2022

12:45 – 13:00	**OPENING**		
	Chair: Andrea Pugliese		
13:00 – 13:40	**Plenary lecture**	Giovanni Marini	*Environmental drivers of West Nile virus in Europe: A modelling approach*
13:45 – 14:25	**Plenary lecture**	Nico Stollenwerk	*The interplay between sub-critical fluctuations and import in understanding COVID -19 dynamics and recent vaccination impact*
14:25 – 14:40	COFFEE BREAK		
14:40 – 15:10	**Invited talks**		
	Zoom license 1 Chair: Nico Stollenwerk	Edy Soewono	*Generating operator for COVID-19 transmission*
	Zoom license 2 Chair: Damián Knopoff	Paula Patrício	*Modelling COVID-19 in Portugal*
	Zoom license 3 Chair: Thomas Götz	Dae-Jin Lee	*Recent statistical challenges in human growth modelling*
	Zoom license 4 Chair: Zoi Paschalidi	Stefanie Hollborn	*German Weather Service Session: The EnVar plus LETKF data assimilation system at the German Meteorological Service (DWD)*
	Zoom license 5 Chair: Bob W. Kooi	Max Souza	*Insecticide resistance: insights of mathematical and computational modellling*
15:10 – 16:30	**Parallel session A – Contributed Talks**		
16:30 – 16:40	COFFEE BREAK		
	Chair: Maíra Aguiar		
16:40 – 17:20	**Plenary lecture**	Ganna Rozhnova	*The dynamics of SARS-COV-2 during the vaccination rollout and in the post-pandemic period*
17:25 – 18:05	**Plenary lecture**	Aaron King	*Markov genealogy processes for exact phylodynamic inference*
18:15 – 19:15	**ROUND TABLE (Chairs: Andrea Pugliese and Costas Siettos)**		
19:15 – 19:30	**PRIZES and CLOSING**		

Parallel session A – The LAST speaker will Chair the first 3 contributed talks and the FIRST speaker will Chair the last contributed talk

Parallel session A – Zoom license 1 - Epidemiology			
15:10 – 15:30	Contributed talk 93	Miller Cerón	*Generalized SEI model with nonlinear incidence rate and asymptomatic infection transmission*
15:30 – 15:50	Contributed talk 94	Abdennasser Chekroun	*Differential-difference Kermack–McKendrick epidemic model with age-structured protection phase*
15:50 – 16:10	Contributed talk 95	Marcel Fang	*Modelling, analysis, observability and identifiability of epidemic dynamics with reinfections*
16:10 – 16:30	Contributed talk 96	Sara Sottile	*Global stability of SAIRS epidemic models*

Parallel session A – Zoom license 2 - Ecology			
15:10 – 15:30	Contributed talk 97	Carlos Martínez	*Regime shifts through selective fishing in a seasonally forced plankton-fish model*
15:30 – 15:50	Contributed talk 98	Aytul Gokce	*Investigating the role of memory in a population system with Holling type III functional response*
15:50 – 16:10	Contributed talk 99	José Moreira	*Impacts of climate change in temperate and subtropical fish species*
16:10 – 16:30	Contributed talk 100	Ananth V S	*Achieving pest management using feedback control for an additional food provided prey-predator system with type III functional response*

Parallel session A – Zoom license 3 - Methodology in Biomathematics			
15:10 – 15:30	Contributed talk 101	Ibtissam Benamara	*Impact of cooperative behavior on the stability of a delayed predator-prey model with Holling functional response*
15:30 – 15:50	Contributed talk 102	Angela Martiradonna	*Positive and conservative GECO methods for chemical and epidemiological models*
15:50 – 16:10	Contributed talk 103	Sangita Swapnasrita	*Sex-specific differences in diabetic kidney*
16:10 – 16:30	Contributed talk 104	Vijay Kumar Shukla	*Study of matrix projective synchronization of chaotic and hyperchaotic systems*

Parallel session A – Zoom license 4 - Special Session "New techniques on Dynamical Systems for weather prediction - Data Assimilation"			
15:10 – 15:30	German Weather Service Session	Nora Schenk	*A 4D-localized particle filter method for regional data assimilation at DWD*
15:30 – 15:50	German Weather Service Session	Walter Acevedo	*Dealing with model re-initialization spin-up: Ultra-rapid data assimilation for real time weather forecast*
15:50 – 16:10	German Weather Service Session	Maria Reinhardt	*Assimilation of intelligent cloud observation*
16:10 – 16:30	German Weather Service Session	Zoi Paschalidi	*Making crowdsourced observations usable for weather prediction: bias correction of citizen weather stations*

Parallel session A – Zoom license 5 – Evolution & Cancer			
15:10 – 15:30	Contributed talk 105	Suman Chakraborty	*Why insects may prefer to feed on toxic plants?*
15:30 – 15:50	Contributed talk 106	Aniruddha Deka	*Can human vaccinating behaviour influence mutant invasion altering pathogen competition? A game-theoretic analysis*
15:50 – 16:10	Contributed talk 107	Jerónimo Fotinós	*Modeling differentiation therapy for cancer-stem-cell-driven tumors*
16:10 – 16:30	Contributed talk 108	Álvaro Martínez Rubio	*A mathematical description of bone marrow dynamics of car T-cell therapy*

DSABNS 2022 Virtual

13th International Conference
Dynamical Systems Applied to Biology
and Natural Sciences (DSABNS)

PUBLIC LECTURES

Basque Center for Applied Mathematics
BCAM, Bilbao, Spain

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A MATHEMATICAL PATH TOWARDS THE UNDERSTANDING OF CELLS' AGGREGATIONS AND MORFOGENESIS

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In this lecture I will sketch some mathematical ideas used to deal with cells aggregations and more generally with morphogenesis. Starting from the seminal book "On growth and form" by D'Arcy Thompson, mathematicians have tried to understand the laws governing the organization of biological aggregates. From simple bacteria to our bodies, it is difficult to master all the phenomena underlying the formation of a full organism. The modern theories start with the thorough ideas proposed by Alan Turing in the Fifties, and nowadays we are trying to deal with more sophisticated models involving stem cells and the generation and regeneration of tissues. In recent years, the development of the highly multidisciplinary Organs-on-Chip microfluidic technologies has provided valuable in vitro platform tools to study the behavior of cells in different contexts. The coupling with live-cell imaging may enable the extraction of single cell tracking profiles, which can be processed with advanced mathematical tools. In this talk I will review some recent results I obtained with my group in dealing with these new settings. In particular, we considered the recruitment of immune cells to a tumor, which is a key parameter in cancer prognosis and response to therapy. We proposed two different models, one more macroscopic, based on variations of the Keller-Segel approach, and a second hybrid one, more microscopic, based on the coupling of individual based models for cells and continuous models for chemicals. The main challenge of this research was in using real data to give a quantitative calibration to these models, to improve their performances and robustness.

RESONANCE-BASED MECHANISMS OF GENERATION OF OSCILLATIONS IN NETWORKS OF NON- OSCILLATORY NEURONS

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Several neuron types have been shown to exhibit (subthreshold) membrane potential resonance (MPR), defined as the occurrence of a peak in their voltage amplitude response to oscillatory input currents at a preferred (resonant) frequency. MPR has been investigated both experimentally and theoretically. However, whether MPR is simply an epiphenomenon or it plays a functional role for the generation of neuronal network oscillations, and how the latent time scales present in individual, non-oscillatory cells affect the properties of the oscillatory networks in which they are embedded are open questions. We address these issues by investigating a minimal network model consisting of (i) a non-oscillatory linear resonator (band-pass filter) with 2D dynamics, (ii) a passive cell (low-pass filter) with 1D linear dynamics, and (iii) nonlinear graded synaptic connections (excitatory or inhibitory) with instantaneous dynamics. We demonstrate that (i) the network oscillations crucially depend on the presence of MPR in the resonator, (ii) they are amplified by the network connectivity, (iii) they develop relaxation oscillations for high enough levels of mutual inhibition/excitation, and (iv) the network frequency monotonically depends on the resonator's resonant frequency. We explain these phenomena using a reduced adapted version of the classical phase-plane analysis that helps uncovering the type of effective network nonlinearities that contribute to the generation of network oscillations. Our results have direct implications for network models of firing rate type and other biological oscillatory networks (e.g, biochemical, genetic).

DSABNS 2022 Virtual

13th INTERNATIONAL CONFERENCE
DYNAMICAL SYSTEMS APPLIED TO BIOLOGY
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PLENARY TALKS

BASQUE CENTER FOR APPLIED MATHEMATICS
BCAM, BILBAO, SPAIN

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ON THE ORIGIN OF COMPLEX DYNAMICS IN MULTI-STRAIN DENGUE MODELS

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Dengue fever epidemiological dynamics shows large fluctuations in disease incidence, and several mathematical models describing the transmission of dengue viruses have been proposed to explain the irregular behavior of dengue epidemics. Multi-strain dengue models are often modeled with SIR-type models where the SIR classes are labeled for the hosts that have seen the individual strains. The extended models show complex dynamics and qualitatively a very good result when comparing empirical data and model simulations. However, modeling insights for epidemiological scenarios characterized by chaotic dynamics, such as for dengue fever epidemiology, have been largely unexplored. The problem is mathematically difficult and to make the urgently needed progress in our understanding of such dynamics, concepts from various fields of mathematics as well the availability of good data for model evaluation are needed.

In this talk, I will present a set of models motivated by dengue fever epidemiology and compare different dynamical behaviors originated when increasing complexity into the model framework.

Acknowledgements

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EFFECT OF SLOW-FAST TIME SCALE AND NONLOCAL INTERACTION ON SPATIOTEMPORAL PATTERN FORMATION

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Spatiotemporal pattern formation in interacting population models is an interesting field of study as it can capture the stationary as well as dynamic patchy distribution of population within their habitat. Introduction of nonlocal interaction in the spatiotemporal model can produce stationary pattern by a spatiotemporal model with Rosenzweig-MacArthur reaction kinetics [1]. On the other hand, it is evident that growth of various prey and their predators take place at different rates when measured with respect to a fixed time scale. This fact is incorporated in to the mathematical model by introducing different time-scales into the growth equations. The resulting models (with temporal reaction kinetics only), in general, exhibit two different types of oscillatory behavior, namely, canard oscillation and relaxation oscillation [2]. The main objective of this talk is to describe a spatiotemporal model for interacting population with nonlocal interaction term and slow-fast time scale, and discuss various scenarios of stationary and non-stationary pattern formation.

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PANDEMICS OF MUTATING VIRUS: A MULTI-SCALE "ACTIVE PARTICLES" APPROACH

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The pandemic initiated by the aggressive virus, i.e. the so-called *SARS–CoV–2 Severe Acute Respiratory Syndrome, corona virus n.2*, has generated a tremendous impact on our society affecting health, economy, and social interactions. Researchers have been asked to provide tools to contrast the aforementioned impact. Mathematics has been challenged to develop tools suitable to go far beyond the deterministic approach of population dynamics with the aim of providing a multiscale derivation of models with the ability not only to capture the complexity of evolutionary biological dynamics, but also to provide a predictive vision suitable to support the decision making of crisis managers.

The aforementioned ambitious objective requires that modeling should should include, in addition to the biological dynamics, also the heterogeneous complexity features of the society.

This Lecture is based on the approach developed in [2] within a multiscale framework accounting for the interaction of different spatial scales, from the small scale of the virus itself and cells, to the large scale of individuals and further up to the collective behavior of populations. The mathematical framework is that of the mathematical theory of active particles [1].

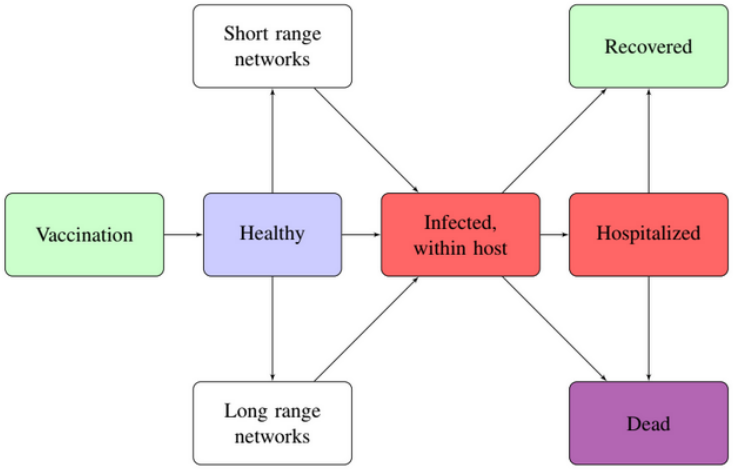

Figure 1: Flow chart representing the dynamics of the system

The first part is devoted to understanding the complexity features of the system and to the design of a modeling rationale. The modeling approach is subsequently developed by focusing both on the contagion dynamics and on how the virus propagates into infected individuals. i.e. on the in-host dynamics, see [5]. Models include post-Darwinist mutations and selection up to the onset of variants, as well as the application of vaccination programs. Models refers to the general framework of a systems approach of a dynamics over a globally connected work.

The main research perspective chases the objective of developing a mathematical theory of the aforementioned complex dynamics suitable to lead to a novel generation of models, as well as to analytic and computational problems generated by the application of models to real world phenomena.

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[1] Bellomo, N., Bellouquid, A., Gibelli, L., Outada, N. (2017). *A Quest Towards a Mathematical Theory of Living Systems*. Birkhäuser-Springer, New York.

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[5] Rapid Assistance in Modelling the Pandemic: RAMP (coordinated by the Royal Society), within host dynamics (coordinated by Mark Chaplain). `https://epcced.github.io/ramp/`

DYNAMICS OF A PREDATOR-PREY MODEL WITH RATIO DEPENDENCE AND HOLLING TYPE III FUNCTIONAL RESPONSE

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In this talk I will discuss the dynamics of a predator-prey model that includes maturation delay in predators, ratio dependence, and Holling type III functional response [1]. Depending on the values of parameters, the model can exhibit an extinction steady state, a prey-only equilibrium, a coexistence steady state, or sustained periodic oscillations around this state. I will show how using a system size expansion of the delayed chemical master equation, we can explore the role of demographic stochasticity, and in particular, analyse the structure of stochastic oscillations around the deterministically stable coexistence state. This is achieved by studying the dependence of variance and coherence of stochastic oscillations on system parameters. Numerical simulations of stochastic model are performed to illustrate stochastic amplification, where individual stochastic realisations can exhibit sustained oscillations in the case, where deterministically the system approaches a stable steady state. I will also discuss complex dynamics of the model in the neighbourhood of extinction steady state in the absence of delay.

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HARVESTING PROFIT OPTIMIZATION ON RANDOM ENVIRONMENTS: THE EFFECTS OF ALLEE EFFECTS

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The growth dynamics of a harvested population in a randomly varying environment can be described by the stochastic differential equation $dX(t) = X(t)$ $(f(X(t))dt - qE(t)dt + \sigma dW(t))$, where $f(X)$ is the *per capita* natural growth rate, $q > 0$ is the catchability coefficient, $E(t) \geq 0$ is the harvesting effort, $\sigma > 0$ measures the strength of environmental fluctuations, and $W(t)$ is a standard Wiener process. The main goal of this work is to assess and compare the influence of Allee effects on profit optimization considering several harvesting policies. For application purposes, the population growth models considered are the logistic model $f(X) = r\left(1 - \frac{X}{K}\right)$ (with $r > 0$ and $K > 0$) and a logistic-like model with weak Allee effects using, to facilitate comparisons, the reparameterization $f(X) = r\left(1 - \frac{X}{K}\right)\left(\frac{X-A}{K-A}\right)$ (with $A \in\ ]-K, 0[$).

In previous work [2, 3], for the logistic model and using data from [8] on the Pacific halibut (*Hippoglossus hippoglossus*), we have shown that the harvesting policy with variable effort is inapplicable, whereas the optimal harvesting policy [1, 2, 3] with constant effort $E(t) \equiv E$ is easily applicable and leads to population

sustainability, although with a slightly lower profit. So, we have also considered [4, 5] stepwise policies, which are applicable but share some of the problems of the optimal variable effort policy, and penalized profit optimal policies (with an artificial running energy cost on the effort), which eliminate some of the disadvantages but are still inapplicable.

We now apply these policies to the logistic-like model with Allee effects [6, 7] to study the influence of Allee effects and check whether or not they should be taken into account when designing harvesting policies.

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MARKOV GENEALOGY PROCESSES FOR EXACT PHYLODYNAMIC INFERENCE

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We describe an approach to phylodynamics that unifies and extends existing likelihood-based methods for extracting information from virus genealogies to parameterize pathogen transmission models. While existing methods rely on approximations that are often violated in practice, our approach yields exact expressions for the likelihood. Specifically, we introduce a class of genealogy-valued Markov processes, induced by population-scale transmission models and show how these lead to a nonlinear filtering equation. The theory is mathematically rigorous and leads directly to computationally efficient inference algorithms.

DYNAMICS OF COUPLED KURAMOTO OSCILLATORS WITH DISTRIBUTED DELAYS

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In this talk I will discuss the effects of two different types of delay-distributed coupling in the system of two mutually coupled Kuramoto oscillators, one where the delay distribution is considered inside the coupling function, and another where the distribution enters outside the coupling function. In both cases, the existence and stability of phase-locked solutions will be analysed for uniform and gamma distribution kernels. The results indicate that while having distribution inside the coupling function only changes parameter regions where phase-locked solutions exist, when the distribution is taken outside the coupling function, it affects both the existence, as well as stability properties of in- and anti-phase states. For both distribution types, various branches of phase-locked solutions are computed, and regions of their stability are identified for uniform, weak and strong gamma distributions [1].

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ENVIRONMENTAL DRIVERS OF WEST NILE VIRUS IN EUROPE: A MODELLING APPROACH

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West Nile Virus (WNV) is one of the most recent emerging mosquito-borne pathogens in Europe. While its main enzootic cycle occurs between mosquitoes and birds, humans might act as incidental hosts. About 1% of the human infections can develop severe neurological diseases, which eventually might be fatal [1]. WNV is now endemic in many European countries, causing hundreds of human cases every year with a high spatial and temporal heterogeneity.

WNV transmission was much greater in 2018 than in previous seasons in Europe. By firstly focusing on northern Italy, we analyzed detailed entomological and epidemiological data collected in 2013–2018 by calibrating temperature-driven mathematical models through a Bayesian approach to simulate mosquito population dynamics and WNV infection rates in the avian and vector populations, which eventually allowed us to assess human transmission risk. Our results suggest that exceptionally high spring temperatures have likely helped to amplify virus transmission at the beginning of the 2018 season [2].

Subsequently, to test this hypothesis at a broader spatial scale, we collated data on the number of human infections recorded in Europe between 2011 and 2019. We then applied generalized linear models to quantify the relationship between human cases and spring temperature, considering both average conditions (over years 2003-2010) and deviations from the average for subsequent years (2011-2019). We found a significant positive association both spatial (average conditions) and temporal (deviations). The former indicates that WNV circulation is higher in usually warmer regions while the latter implies a predictive value of spring conditions over the coming season [3].

Our findings highlight that weather anomalies at the beginning of the mosquito breeding season might act as an early warning signal for public health authori-

ties, enabling them to strengthen in advance ongoing surveillance and prevention strategies.

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PATTERNS OF T1D ONSET ON HIV INFECTED PATIENTS AFTER IMMUNE RECONSTITUTION

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HIV-infected patients may develop autoimmune diabetes (T1D), though in rare cases. The underlying mechanism isn't yet completely understood, nevertheless there are insights, from clinical trials, suggesting that the sudden increase in CD4+ T cells and the macrophages' immune function may trigger the onset of T1D. Here, we propose a reasonable model for the development of T1D in HIV recovering patients.

THE DYNAMICS OF SARS-COV-2 DURING THE VACCINATION ROLLOUT AND IN THE POST-PANDEMIC PERIOD

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Mass vaccination has played a large role reducing pathogen circulation and the burden of disease, but the endgame for SARS-CoV-2 control will not be over soon. It is not clear when and which control measures can be relaxed during the rollout of vaccination programmes. Also, few modeling studies address the trajectory of SARS-CoV-2 on longer time scales and transitioning into an endemic

phase. In the first part of the talk, I will present results from our recent publication [1] on relaxation scenarios using an age-structured transmission model that has been fitted to age-specific seroprevalence data, hospital admissions, and vaccination coverage for Portugal based on the methodology we developed in [2]. Our analyses suggest that the pressing need to restart socioeconomic activities would lead to new pandemic waves, and that substantial control efforts prove necessary throughout 2021. Additional waves could be prevented altogether if measures were relaxed in a step-wise manner throughout 2021. In the second part of the talk, I will provide an extension of the model in [1] that takes into account seasonality in transmission and waning of sterilizing immunity. I will further discuss the projected dynamics of SARS-CoV-2 and implications for the planning and management of COVID-19 measures and vaccination schedules in the post-pandemic period.

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AGENT BASED MODELS FOR URBAN MOBILITY: EMERGENT BEHAVIOUR AND BIFURCATION ANALYSIS

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Traffic flow dynamics is an example of very rich emergent nonlinear behaviour which may arise in complex systems as consequence of the interactions, between the vehicles/agents and the complexity of the street network. Except of the traffic jams and traveling waves, many other complex nonlinear phenomena has been observed, both experimentally and numerically, like stop and go waves and other spatio-temporal sustained oscillations and chaos. An important step towards the understanding of this complexity in large scale urban network is to study the emergent dynamics of traffic flow at networks intersections. Here, we propose and analyze the dynamics of an agent-based model, based on the so-called social force model, for simulating mobility of autonomous vehicles at intersections. The proposed model is able to replicate phenomena of collective behaviour that have been observed in real life situations and can be used as a realistic data generator for optimization purposes. In particular, we show that the emergent traffic flow dynamics exhibit multistabilty for a wide range of the density: except for the state of the free flow in both directions, other two steady states of stop and go-oscillating spatiotemporal patterns are found. Moreover, a methodology, based on the Equation-Free multiscale framework [1], is presented to analyse the coarse-grained dynamics which is able to reconstruct systematically bifurcation diagrams and/or macroscopic fundamental diagrams. The methodology is first applied to well know example of emergent behavior like the ring road [2] and then applied in more complicated situations which include also intersections.

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NUMERICAL SOLUTION OF PARTIAL DIFFERENTIAL EQUATIONS AND STIFF PROBLEMS OF ODEs WITH PHYSICS INFORMED RANDOM PROJECTION NETWORKS AND EXTREME LEARNING MACHINES

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Over the last few years, physics-informed machine learning have been used to solve both direct and inverse problems of time-depended non-linear PDEs and ODEs that model high-dimensional multiscale systems [5]. Here, we address a physics-informed machine learning framework based on Random Projections Networks and Extreme Learning Machines to numerically solve nonlinear PDEs and stiff problems of ODEs and perform numerical bifurcation analysis [1, 3, 4]. The performance of the proposed numerical method is assessed via several benchmark problems, namely four systems of stiff ODEs (Prothero-Robinson, van-der Pol, Rober and HIRES) and, three problems of nonlinear PDEs (1D viscous Burgers, 1D and 2D Bratu, 1D Allen Cahn). The efficiency of the proposed framework in terms of both numerical accuracy and computational cost is compared with classical numerical methods including solvers for stiff ODES, Finite Differences and Finite Elements as well as other proposed machine learning methods including Deep-Learning Networks and Gaussian Processes [2, 6, 7].

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THE INTERPLAY BETWEEN SUB-CRITICAL FLUCTUATIONS AND IMPORT: UNDERSTANDING COVID-19 DYNAMICS AND RECENT VACCINATION IMPACT

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From previous analyses of COVID-19 dynamics in the Basque Country on the initial phases of exponential growth and first control measures in spring 2020 [1, 2, 3] we extended the analysis of the ongoing epidemiological data into summer and autumn 2020, observing and modelling the dynamics which by then was under control of community spreading, hence not in another exponential growth phase, but prone to isolated outbreaks due to external import via relaxation of travel restrictions. In such scenarios one would expect on general grounds large fluctuations close to the critical threshold between community control and exponential explosion of cases [6], which could finally be modelled for the present case of COVID-19 and its data in the Basque Country [4]. The dynamics in autumn 2020 until close to the end of the year could be well described by the sub-critical fluctuations and import.

Based on this analysis we then developed models to describe and analyze the impact of the vaccination campaigns in the Basque Country, starting in January 2021, with various vaccines applied at the same time. One has to take into account that the different vaccines have different efficacies not only after full vaccination scheme, but especially between single dose and second does schemes. Here the question of protection against severe disease and against any infection [5] turned out to be very important in the overall outcome of the vaccination campaigns [7]. Especially, new variants of the virus challenge the vaccine efficacy, still reported to be high against severe disease, but with significantly lowering protection against mild and asymptomatic infection, well describable in SHAR and SHARUCD modelling frameworks, as set up as early as spring 2020 [1, 3].

Some first considerations will be made on such waning of vaccination immunity and its interplay with seasonal variation of infectivity in respiratory diseases.

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A MODEL FOR THE OLIVE TREE PEST *Prays oleae* (BERNARD)

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The olive moth, *Prays oleae* (Bernard) (Lepidoptera: Praydidae) is the main olive pest in Trás-os-Montes, the second Portuguese producing olive region located in the northeaster of Portugal. It is parasitized by the specialist parasitoid *Ageniaspis fuscicollis* (Dalman) (Hymenoptera: Encyrtidae), as well as by the facultative hyperparasitoid *Elasmus flabellatus* (Fonscolombe) (Hymenoptera: Eulophidae).

A mathematical model, which considers spiders as important predatory actors in agroecosystems, is constructed and analyzed. Relationships among the different system equilibria are assessed, through transcritical bifurcations. It allows also to investigate possible population abundance changes under climatic variations.

The results show the role of *A. fuscicollis* as the main natural control agent. In certain conditions its action is suitably supported by both *E. flabellatus* and spiders in curbing the olive tree pests.

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MODELLING AND SIMULATION OF POLYMER/CERAMIC COMPOSITE ELECTROLYTE MATERIALS USING ATOMISTIC SIMULATIONS

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The development of inexpensive, efficient and scalable all-solid-state batteries, in which solid-state electrolytes (SSEs) are used as alternatives to traditional, flammable liquid electrolytes is crucial to enabling safer and more-energy dense technologies than today's Li-ion batteries. Composite materials comprising a conductive, flexible polymer matrix embedding ceramic filler particles have emerged as a promising option to provide the combination of conductivity, mechanical and chemical stability required of SSEs. Moreover, recent efforts have been made to substitute synthetic, fosil fuel - based polymers by biobased polymers such as bacterial cellulose or lignin that are functionally equivalent to synthetic polymers, yet are biocompatible and capable of biodegradation [3].

The electrochemical activity of composite SSEs is highly dependent on the interfacial Li-ion dynamics, whose molecular underpinnings remain elusive. We overcome this shortfall by investigating a polymer-ceramic electrolyte, integrat-

ing polyethylene oxide (PEO) plus lithium bis(trifluoromethanesulfonyl) imide (LiTFSI) and Li-ion conductive $Li_7La_3Zr_2O_{12}$ (LLZO) garnet fillers (Figure 1). Nonetheless, the methodological principles exposed here are applicable to other polymer/composite arrangements, including bio-based polymers.

By combining Molecular Dynamics and an enhanced generalized hybrid Monte Carlo technique, we are able to gain unprecedented insights into the Li-ion dynamics at polymer/composite interface. First, we find that large portions of the polymer chains are detached from the LLZO particle surface, with the presence of only few anchoring points which are associated to the coexistence of strong hydrogen bonds and weak van der Waals interactions. Increasing the temperature leads to the exclusive prevalence of hydrogen bonding and, therefore, tail-like adsorption modes. Second, our simulations automatically produce the interfacial Li-ion distribution assumed in previously reported, continuous space-charge models of the PEO(Li-salt):LLZO composite electrolyte systems[2], including the presence of a Li-ion free Stern layer on the polymer side. Third, we determine that the LLZO particles do not contribute to macroscopic Li-ion transport, but act effectively as a Li-ion adsorbents. Finally, we also observe that the presence of the garnet surface severely diminishes ionic diffusivity in PEO even at large distances from the interface. Our analysis indicates that this is originated by a decrease in polymer segmental mobility due to the interaction with the garnet, even over distances beyond the average end-to-end length of the PEO chains [1].

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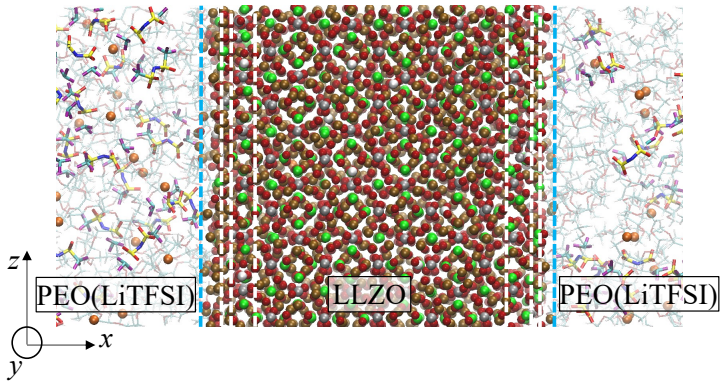

Figure 1: The simulation box for the LLZO:PEO(LiTFSI) system. The garnet was cleaved to the center of the simulation box, generating two interfaces perpendicular to the *x* axis. The boundaries are periodic in all directions. Brown, magenta, bright green, blue and white spheres within the garnet correspond to Li^{+}, O^{2-}, Zr^{4+}, La^{3+} and Ga^{3+} ions, respectively. In the *p*-phase, only the -C- (cyan) and -O- (red) bonds are shown in the polymer chains, while bonds with hydrogen are excluded for clarity. LiTFSI is shown as yellow, red, pale green and orange spheres, corresponding to S, O, F and Li.

ROLE OF CHEMOTAXIS IN TUMOR ANGIOGENESIS

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**Alberto Gandolfi passed away in August 2019.
This talk is dedicated to him.
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This talk is based on our recent paper [1], which focus on the propagation of a tumor from the stage of a small a–vascular sphere in a host tissue and the progressive onset of a tumor neo–vasculature stimulated by a pro-angiogenic factor secreted by hypoxic cells. The way new vessels are formed involves cell sprouting from pre–existing vessels and following a trail via a chemotactic mechanism (CM).
Namely, we propose a new a detailed general family of models of the CM, based on a statistical mechanics approach. The key hypothesis is that the CM is composed by two components:

1. the well–known bias induced by the angiogenic factor gradient;
2. the presence of stochastic changes of the velocity direction, thus giving rise

to a diffusive component.

Then, some further assumptions and simplifications are applied specific to the problem in study.
Numerical simulations show the onset of a traveling wave eventually replacing the host tissue with a fully vascularized tumor. The results of simulations agree with experimental measures of the vasculature density in tumors, even in the case of particularly hypoxic tumors.

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THE EnVAR PLUS LETKF DATA ASSIMILATION SYSTEM AT THE GERMAN METEOROLOGICAL SERVICE (DWD)

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Weather happens at extremely varying time and space scales, from seasonal weather conditions ranging over weeks and continents to rapidly changing high impact weather such as thunderstorms and strong precipitation. Owing to the ubiquitious and vital influence of weather on personal life as well as on society, predicting the weather has been and still is one of the pioneer applications for numerical modelling, data assimilation and high performance computing.

State-of-the-art numerical weather prediction (NWP) systems prevalently run hybrid methods which complement a high resolution deterministic variational method by an ensemble data assimilation system. In our talk, we give a survey on the hybrid EnVAR plus LETKF Data Assimilation System which has been developed and is under constant development at German Meteorological Service (DWD).

For the global forecasts, DWD runs the ICON model on a 6.5km/13km resolution with a 40-member ensemble of 20km/40km resolution. Observations are merged with short term forecasts within a 3-hourly assimilation cycle. The ensemble forecasts provide dynamic estimates of the background error covariances. Based on this data assimilation cycle longer ensemble forecasts (up to 7 days) are run regularly. Moreover, the global system provides boundary conditions for the high resolution (2km) convection resolving model.

DYNAMICS OF MULTI-PHASIC EPIDEMICS: COVID-19 IMMUNIZATION IN ITALY AS CASE STUDY

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Due to the lack of a vaccine, the first epoch of control interventions and mitigation of the COVID pandemic essentially relied on social distancing supplemented by tracing of infective contacts. In Western countries, the arrival of an effective vaccine by end 2020 and the growing (though sub-optimal) vaccine uptakes in the summer period have allowed a honey-moon epoch of gradual lifting of social distancing and the restart of full socio-economic activity. The onset of a fourth pandemic wave during fall 2021 partly feeding on hesitant people but with a possible contribution to transmission by vaccinated individuals in view of the imperfect protection, is further emphasising the multi-phasic nature of this pandemic, even regardless of the continuing threat posed by the appearance of new variants.
In this article, we extend a published modeling framework for an epidemic evolving into a sequence of growth vs control phases, to include vaccination. The infection dynamics is governed by an age-since infection epidemiological equation. The proposed approach allows a unified and consistent treatment of multi-phasic epidemics even when vaccination is considered. The analysis of selected subcases using specific infectiousness and recovery kernels allowing reducibility, allows to draw simple sub-models tobe parsimoniously fitted to data. The framework is ap-

plied to the COVID-19 epidemic in Italy since its very beginning (March 2020) up to the current fourth wave. The model provides an excellent fit to the data by minimal parametrization, confirming the validity of the multi-phasic epidemic concept.

SPATIAL EPIDEMIOLOGICAL MODELS OF INFECTIOUS DISEASE TRANSMISSION

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In this talk, I will provide a mathematical and computational framework to the modeling and simulation of the spatial dynamics of infectious disease contagion, spreading, control and prevention.

Starting from lattice-based models, I will present a stochastic cellular automata disease model based on the SHAR (**S**usceptible-**H**ospitalized-**A**symptomatic-**R**ecovered) compartmentalisation of the population, show some simulation results, and relate them to real-life data for influenza and COVID-19 provided by the Health Department and the Health Services of the Basque Country. By means of stochastic simulations, I will present a numerical study to have an insight into the critical threshold of the SHAR percolating system, as well as some numerical tools to seek infection clusters (groups of connected individuals who have been infected by the same index case), count them and monitor their sizes [1, 4].

Then, some concepts on network theory will be discussed in order to show how networks can be employed as a valuable tool to model spatial spreading. In particular, two models will be discussed: a kinetic model for the spread of a respiratory disease considering heterogeneous populations [2, 3] and a compartmental model accounting for bacterial antibiotic resistance [5], focusing on the role of the

network structure and connectivity on the overall dynamics.

Acknowledgements
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ANALYSIS OF AN EPIDEMIOLOGICAL MODEL WITH ONE RECURRENT INFECTION

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The one-recurrent infection epidemiological pathogen model for a population is studied. This compartmental SIRSIR-model is divided into six classes, three classes for the primary infected (S_P, I_P, R_P) and three for the secondary infected (S_S, I_S, R_S) sub-models, see Fig 1. Both sub-models are extended versions of the classical SIR models. For the primary infection model we have the susceptible class (S_P), those newborn individuals without a previous infection. These individuals get infected by contact with individuals in either the primary (I_P) or secondary infected (I_S) classes. Frequency-dependent transmission described by with rate of infection β. Individuals recovered from this first infection at a rate γ and form the (R_P) class. They are not lifelong immune but due to waning immunity they become susceptible (S_S) again at rate α. Those infected for the second time form the secondary infected class (I_S) with the same contact model as for the primary susceptibles. The recovered individuals form the secondary recovered (R_S) class with lifelong immunity.

The overall force of infection for both primary and secondary infected is such that the secondary is either neutralizing or enhancing. The ratio of secondary and overall force of infection is denoted by ϕ. When $\phi = 1$ there is no difference between infection by both infected classes. The birth rate of susceptible newborn individuals equals the proportional death rate μ. Therefore demographics is neglected: the total number of individuals in the population remains constant over time. We assume β/γ is fixed which is for an SIR-model the basic reproductive ratio R_0.

Numerical phase-plane and bifurcation analyses show that the long-term dynamics is an equilibrium or a limit cycle for the endemic case and an equilibrium for the pathogen-free case. Three types of bifurcations for these equilibria can occur: transcritical, Hopf and tangent (saddle-node) bifurcation, [2, 4]. The tran-

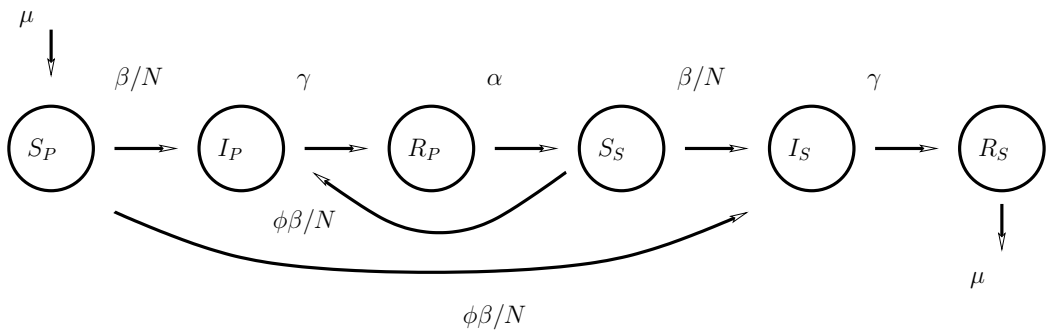

Figure 1: Diagram of compartmental SIRSIR-model with six classes.

scritical bifurcation will be studied in detail. It marks the threshold value of a parameter where the pathogen-free equilibrium becomes unstable and at the same value the endemic equilibrium becomes stable or unstable. In epidemiology the threshold is fixed where the basic reproductive ratio $R_0 = 1$, [1]. Two versions can occur: one is supercritical or non-catastrophic (emerging endemic equilibrium is stable) the other the subcritical or catastrophic (emerging endemic equilibrium is unstable). In [5, 4] the centre manifold theory is used to derive criteria whether it is non-catastrophic or catastrophic. The latter is together with the occurrence of a tangent bifurcation also known as part of a backward bifurcation [3].

A pair of tangent bifurcations emerge from the transcritical bifurcation point when import is introduced of infected individuals, for instance from an separate remote endemic area and replacing the same number of pathogen-free susceptibles. With import the pathogen-free equilibrium does not exist, only equilibria with low number of infected individuals that converges to zero when import diminishes and these equilibria can be stable or unstable.

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EPIDEMIC CYCLES AND TRANSITION TO HEALTH-POSITIVE OPINION IN A COUPLED HEALTH-OPINION AND DISEASE TRANSMISSION MODEL

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Background: Interest in effect of coupling between infection spread and changes in behavior is on the rise and a number of mathematical models were developed in the recent years capturing this relationship. Regardless of the formulation, the analysis of these models' dynamics showed that epidemic cycles and a transition to different health behavior following an outbreak can occur. To design effective health information interventions, it is important to understand which properties of the coupled health-behavior and infection dynamics give rise to these phenomena.

Methods: We investigated the dynamics of a deterministic model that couples epidemic dynamics with health opinion competition (health-positive and health-neutral). Health opinion held determines susceptibility of individuals to becoming infected. We investigated how the basic reproduction number and the rate of switching to the health-positive opinion influences prevalence and incidence of infectious cases, the appearance and characteristics of epidemic cycles, and the possibility of disease extinction.

Results: A large basic reproduction number and sensitive reaction of the opinion switching rate to rise in prevalence can cause appearance of epidemic cycles

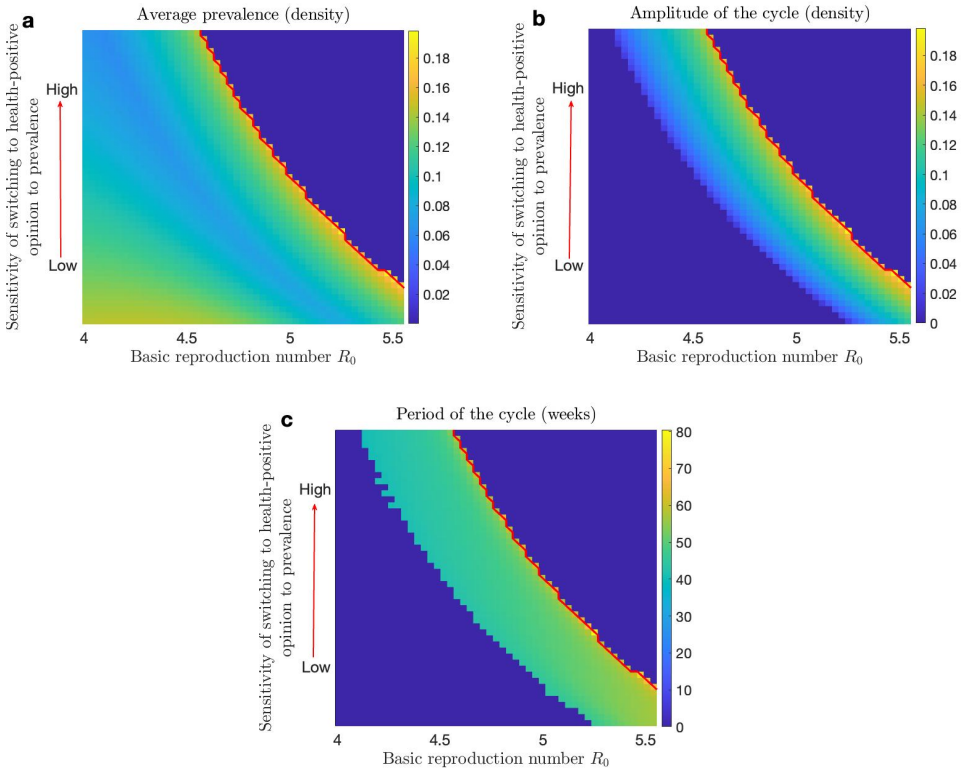

Figure 1: Prevalence, amplitude of cycle and period of cycle as a function of sensitivity of opinion switching to prevalence and basic reproduction number

and switch of the population to a health-positive opinion, potentially eradicating a disease (Figure 1). The region where oscillations appear is adjacent to the region where the population switches to the distribution where everyone holds the health-positive opinion (Figure 1, region to the right of the red line). The faster the societal reaction is, the lower is the prevalence which causes the switch to an entirely health-positive population.

Conclusion: To avoid appearance of epidemic cycles, a swift information campaign spreading the health-positive opinion should be mounted. Success of this measure will benefit the population during the ongoing outbreak and as well as make it better prepared for a next one. Fast spread of infection is another factor that may stimulate the health-positive opinion being universally accepted.

RECENT STATISTICAL CHALLENGES IN HUMAN GROWTH MODELLING

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Historically, human growth data have often been modelled with graphical images to afford general comparisons among trajectories. J.M. Tanner was the first to identify the visible stages of puberty. Today, these stages are known as the Tanner stages or, more appropriately, sexual maturity ratings [5]. Hauspie et al. (1991) described growth modelling as a technique that allowed an estimation of a smooth curve through observed data, across a part of or the entire growth process, with the goal to capture the essential trends in a data series or to identify certain characteristics, such as the age at maximum velocity at adolescence [1].

Non-linear growth curves are particularly valuable because the defining characteristics of the growth process can be estimated, such as the initial level, the rate of change during the growth spurt, and the asymptotic level. The model proposed by Preece and Baines (PB) is a well-known structural or parametric methods for analyzing anthropometric traits [2]. From a statistical point of view, non-linear models, such as PB, can be extended as mixed-effects models to estimate the population-level effects of growth while also modeling the individual variation between subjects. In recent years, Functional Data Analysis (FDA) has become an active domains in Statistics [3]. The objects under study are real functions which are assumed to be realizations of stochastic processes that can represent curves, surfaces or anything else varying over a continuum.

In this talk, I will present a brief overview of the mathematical and statistical approaches to model human growth curves from linear, non-linear models with different complexity to FDA approaches and distributional regression models for quantiles [4]. Finally, some examples of real applications will be presented.

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MATHEMATICAL MODELLING OF CONTROL OF PATHOGENIC BACTERIA BY A PHAGE WITH A TEMPERATURE-DEPENDENT LIFE CYCLE

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Phages are bacterial viruses, they are the most abundant biological entity on Earth, and they impact all aspects of bacterial evolution and dynamics. However recent empirical studies in microbiology have revealed crucial novel facts regarding phage control of bacteria which have not been addressed in early mathematical/computational models. In particular, this concerns the recently discovered condition-dependent lysogeny, where the type of infection cycle of phage (lytic or lysogenic) is determined by the environmental conditions such as the temperature. Fluctuation of environmental conditions (e.g. daily, monthly, or seasonal) results in a constant switch between lytic or lysogenic infection cycles making bacteria-phage interaction extremely complex. In this talk, I will discuss the recent progress made in mathematical modelling of bacteria-phage interaction of condition-dependent lysogeny. Both homogeneous and spatially heterogeneous systems (soil) will be considered. As an important case study, I will focus on the natural control by the phage of the pathogenic bacteria *Burkholderia pseudomallei* causing melioidosis, which is among the most fatal diseases in Southeast Asia and across the world. I will show that mathematical modelling backed up by experimental data can mimic the observed pattern of variation of risk of melioidosis acquisition across the year. The model also emphasises the role of enrichment of the environment in success of the natural control of bacteria by the phage. The model predicts an eventual spread of zones of endemicity of the disease due to global climate change.

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SHOULD I STAY OR SHOULD I GO? ZERO-SIZE JUMPS IN RANDOM WALKS FOR LÉVY FLIGHTS

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Motivated by the fact that, in the literature dedicated to random walks for anomalous diffusion, it is disregarded if the walker does not move in the majority of the iterations because the most frequent jump-size is zero (i.e., the jump-size distribution is unimodal with mode located in zero) or, in opposition, if the walker always moves because the jumps with zero-size never occur (i.e., the jump-size distribution is bi-modal and equal to zero in zero), we provide an example in which indeed the shape of the jump-distribution plays a role. In particular, we show that the convergence of Markovian continuous-time random walk (CTRW) models for Lévy flights to a density function that solves the fractional diffusion equation is not guaranteed when the jumps follow a bi-modal power-law distribution equal to zero in zero, but, as a matter of fact, the resulting diffusive process converges to a density function that solves a double-order fractional diffusion equation [3]. Within this framework, self-similarity is lost. The consequence of this loss of self-similarity is the emergence of a time-scale for realizing the large-time limit. Such time-scale results to span from zero to infinity accordingly to the power-law displayed by the tails of the walker's density function. Hence, the large-time limit could not be reached in real systems. The significance of this result is two-fold: i) with regard to the probabilistic derivation of the fractional diffusion equation [4] and also ii) with regard to recurrence [1] and to site fidelity in the framework of Lévy-like motion of wild animals [2].

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MODELLING COVID-19 IN PORTUGAL

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First, we present an age-structured SEIR model that uses contact patterns to reflect the physical distance measures implemented in Portugal to control the COVID-19 pandemic. By using these matrices and proper estimates for the parameters in the model, we were able to ascertain the impact of mitigation strategies employed in the past. Results show that the March 2020 lockdown had an impact in the disease transmission, bringing the effective reproduction number ($\mathcal{R}(t)$) below 1 [1].

In a second part, we use a simple model to analyze the impact of vaccination on the control of the pandemic [2]. First, we fit a SEIR type model without vaccination to the Portuguese data on confirmed cases of COVID-19 by the date of symptom onset, from the beginning of the epidemic until the 23rd January of 2021, to estimate changes in the

transmission intensity. The goal here is to see if this very simple model could describe COVID-19 dynamics in Portugal when using different and more noisy data. Then, by including vaccination strategies in the model we develop different scenarios for the fade-out of the non pharmacological intervention (NPIs) measures as vaccine coverage increases in the population according to Portuguese vaccination goals. We also include a feedback function to mimic the implementation and relaxation of NPIs, according to some disease incidence thresholds defined by the Portuguese health authorities. Finally, we go back to the heterogeneous model to evaluate how vaccination and immunity wane affect the transmission. We compare different scenarios for the near future.

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POPULATION GROWTH IN A PATCHY ENVIRONMENT

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Previous works like [2, 3] have studied population growth in patchy environment and provided, under some assumptions, an explicit relationship between the asymptotic size of the total population and the sum of the local capacities. Surprisingly, and based on this relationship, [3] notes that the total realized asymptotic population abundance (TRAPA) in the heterogenous environment may exceed the sum of the local carrying capacities. This result has been discussed in several contexts of environmental management in the literature. More recently, [1] addressed the issue of the perfect-mixing paradox occurring with the logistic growth model (as formulated by Lotka and Gause). The authors suggest that the paradox mentioned in [3] could somehow be the result of bad choice of model formulation and could be logically inconsistent. In this talk, we make a fast review of the debates on this question, including logic arguments and empirical evidences. Then we propose a general result to extend the previous ones obtained in [4] where an explicit relationship between local production and global production in a heterogeneous environment helps to understand why a heterogeneous environment may be more productive under given conditions. In the model proposed here, we follow a mass balanced framework and then avoid the logical problems suggested in [1].

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EFFECT OF SHARP CHANGES IN CONTACT RATES IN EPIDEMIC MODELS

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In the course of the COVID-19 pandemic many countries have experienced sharp changes in contact rates due to lock-downs and similar measures. We examine here how such changes affect disease incidence in some epidemic models. It appears that models based on simpler or more complex models of ordinary differential equations [1, 2, 3, 4] predict that the rate of new infection should also quickly start decreasing, while observations suggest a longer delay. Beyond possible problems in data collection, memory effects have been proposed as an explanation for the delay and shape of the peaks [5]. While memory effects are definitely present and relevant, we surmise that the subdivision of the population into households and other small groups within which transmission continued normally during lockdown can help understanding the dynamics of the epidemics, and the delayed peaks.

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LONG TRANSIENTS IN ECOLOGY ARISING FROM LOCAL AND GLOBAL BIFURCATIONS

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Transients are an important phenomenon in Dynamical Systems Theory with deep implications in Physics, Chemistry, and Biology. Transients are extremely relevant in complex ecosystems. Ecosystems are expected not to be typically close to their stable states (attractors) due to external forces such as climate variations or changes in parameters due to ecological or anthropogenic perturbations. Hence, their study becomes of

paramount importance to understand the properties of such transients, which, in some cases can drive to population collapses. In this lecture, we will discuss transient generator mechanisms in Ecology by means of simple, but representative, mathematical models as well as with stochastic simulations and cellular automata models. These models will consider different ecological processes emphasizing in facilitation, and also including competition, species decay, and habitat destruction. Focusing on transients arising close to bifurcations, we will present recent research on simple trophic chain systems displaying complicated bifurcation patterns [4]. Also, we will discussed delayed transitions tied to saddle-node bifurcations in systems with intrinsic (demographic) noise [3, 2]. Finally, a novel delaying mechanism arising close to a zip bifurcation (involving the destruction of a curve of quasineutral equilibria), displaying interesting scaling laws will be discussed in a deterministic host-parasite system [1].

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MATHEMATICAL MODELLING OF COVID-19

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The periodic outbreaks of infectious diseases are still constitute one of the most serious global health problems. The first case of COVID-19 was reported in China on November 17, 2019. Due to the increasing number of cases of COVID -19, The World Health Organization (WHO) on March 11, 2020, has declared the novel coronavirus (COVID-19) outbreak as a global pandemic [2]. The COVID-19 pandemic has become the most important challenge not only for the health public system, but also for the scientists. One of the most useful tools for describing the dynamics of the epidemic are mathematical models. The approaches to modeling COVID-19 dynamics can be divided into the following groups: spatial epidemiological models [2] and compartmental epidemiological models[1]. We present some result connected to both of them.

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GENERATING OPERATOR FOR COVID-19 TRANSMISSION

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Several countries experienced an unexpected outbreak with new infectious variants in the second wave Covid-19 after authorities in affecting countries implemented a relaxation control of the diseases at the end of the first wave transmission. The second wave Covid-19 global pandemic has become more complicated and unpredictable throughout the world. During the second wave transmission, the reported new cases were dominated by collected data from massive PCR testing following the WHO criteria for the test target. Undetected infections are not measurable, which should be taken care of in estimating the daily reproduction ratio. A generalized SEIR model with recorded and unrecorded EIR compartments and time-dependent infection rates is constructed. This study will exploit total recorded cases (cumulative) from Covid-19 data and define a generating operator acting on the cumulative case function. Further, the unrecorded daily rate of infection is computed from the ratio between estimated IFR and CFR. It is shown that all state dynamics and the effective reproduction ratio are directly produced by implementing the operator to the cumulative function. Simulations are done for selected countries using Covid-19 data from https://www.worldometers.info/coronavirus. This method directly measures daily transmission indicators, which can be effectively used for the day-to-day control of the epidemic.

INSECTICIDE RESISTANCE: INSIGHTS OF MATHEMATICAL AND COMPUTATIONAL MODELLING

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The development of insecticide resistance is becoming a threat to many arboviruses control programs worldwide. While this has been attributed to the indiscriminate use of insecticide, a more theoretical study is apparently not available. Using a combination of in-silico experiments and mathematical analysis [1, 2], we investigate the effects of different policies, while focusing on two particular examples: one used by the Brazilian Ministry of Health (which follows the World Health Organization protocol) and a more permissive one, akin to those employed by various gated communities and private companies. The results show that while the public policy does not lead to resistance fixation, the scenario changes for the permissive application of adulticide. This policy is typically associated with intensive domestic use mainly during epidemic periods, and might lead to the fixation of a resistant population, even when resistance is associated with moderate fitness costs.

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MODELING THE HEROIN EPIDEMIC AND THE IMPACT OF THE AWARENESS PROGRAM

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In this work, we investigate two models of the Heroin epidemic. First, we introduce the global properties of the heroin epidemic model with time distributed delay and nonlinear incidence function. We show that the system has threshold dynamics in terms of $\mathfrak{R}_0$, and we prove, via a Lyapunov function, that for $\mathfrak{R}_0 < 1$ the drug-free equilibrium is globally asymptotically stable. For $\mathfrak{R}_0 > 1$, we give the persistence result of the heroin consumption. We also show the global stability of the endemic equilibrium for $\mathfrak{R}_0 > 1$ using a suitable Lyapunov function. In the second part, we study the effect of the awareness program performed by the media on the spread of heroin dependence using a mathematical model with distributed delay. In addition to the global analysis of this model, we show that the media on reducing the outbreak of heroin abuse. By assuming that the heroin is persistent in the absence of the media effect, we determine the minimal required media coverage and effectiveness to reduce the $\mathfrak{R}_0$ bellow one to guarantee the stop the outbreak of the Heroin addiction in the population.

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A MULTISCALE NETWORK-BASED MODEL OF CONTAGION DYNAMICS; HETEROGENEITY, SPATIAL DISTANCING AND VACCINATION

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Although the use of SIR type models has been widely adopted by policy makers [2, 3, 1] in order to obtain predictions about COVID-19 spreading, a severe respiratory syndrome caused by a new coronavirus (SARS-CoV-2), many limitations have been acknowledged to this simple modelling approach, particularly regarding the role of heterogeneity, which has been shown to significantly affect disease transmission and control. Although the influence of age affecting disease severity and death probability in a population is often assumed, only few modeling frameworks are currently able to include heterogeneity via social networks structures, see e.g., [7, 8, 9].

In this paper, using a multiscale network-based model of contagion dynamics, we explore the role of heterogeneity in shaping and unfolding the overall diffusion process of COVID-19 epidemic. An extensive simulation analysis considering different social network structures is performed to investigate the impact of interactions during the pandemic. Results are compared addressing the effectiveness of social distancing policies. The impact of an immunization strategy is also investigated to understand the effectiveness of the current COVID-19 vaccination policy prioritising the most vulnerable individuals.

The model is able to account for two forms of heterogeneity, namely *between-individual* heterogeneity in virus transmission on the basis of individual attributes influencing the epidemic growth, and *social-structure* heterogeneity, introducing alternative forms of net-

works influencing the contact dynamics as well as different structured nodes within which contacts occur, namely schools, hospitals/nursing homes, workplaces and households. In so doing, we explicitly model the two crucial factors affecting the reproduction number of the epidemic, hence the most sought information by policy-makers during COVID-19 crisis, *contagion*, i.e., between-individual virus transmission, and *contact*, i.e, the social structure interaction of individuals in a population.

Methodologically, we make a bridge between two different approaches to model complex behaviour in living systems. The first approach is the *kinetic theory of active particles* (KTAP thereafter) [5] which allows to model macroscopic states as the result of multi-level interactions occurring at microscopic states, going from the relationship between the virus and the immunological system (within-host dynamics) toward the population dynamics (between-host dynamics). The KTAP approach has been successfully employed to innovatively model the contagion dynamics of the COVID-19 spreading [4, 6]. The second approach is the *complex system analysis of social networks* which has seen in the last decades an increasing number of studies documenting the network properties of social relationships, quite far away from homogeneous distributions, and in general characterised by repeated, structured and clustered contacts [10]. Both approaches share the view of biological and social organizations as complex systems, often evolving, and indeed provide many common interpretations of real world phenomena.

Simulation-based results of the developed model support the role played by network structure in affecting the social distancing policies implemented during the pandemic. In particular, comparing random, scale-free and small-world graphs, we study the dynamics of contacts occurring among the four different types of nodes above mentioned, each of them characterized by different size, immunity of the population and probability of contacts with other nodes. The dynamics of the epidemic is studied with reference to crucial parameters influencing the network structure and connectivity, mainly the wiring probability and the degree (number of contacts).

Inside alternative contact matrices we study the impact of closures of different nodes upon the overall contagion dynamics. As we shall show, social distancing is more effective when targeting the reduction of contacts among and within the most vulnerable nodes, namely hospitals/nursing homes. On the other hand, school closures do not appear to be the most effective policy measure, not affecting significantly the reduction of deaths in the population. Finally, we experiment with a set-up on immunization in order to understand the effectiveness of targeted vaccination policies towards the most vulnerable individuals. According to our model, vaccinating first the most vulnerable segment of the population has an important role to reduce deaths in the population.

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DEALING WITH MODEL RE-INITIALIZATION SPIN-UP: ULTRA-RAPID DATA ASSIMILATION FOR REAL TIME WEATHER FORECAST

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New mobility approaches like autonomous driving require high precision weather solutions, such as real time weather maps and warnings. The different emerging technologies like Internet of Things and 5G mobile networks make available huge mount of high resolved meteorological data from various sources, like automobiles. In the previous four years the German Weather Service collaborated with the German car manufacturer AUDI AG to combine these two facts in the Fleet Weather Maps (Flotten-Wetter-Karte - FloWKar) project. FloWKar explored how environmental data from sensors of vehicles on Germany's roads, respecting data protection regulations, can be used in real time to improve weather forecast, nowcasting and warnings within DWD's products for key customers.

While the operational forecast gives hourly updates, the production of real time weather forecast needs an exceptionally fast data assimilation cycle with an update rate in the order of minutes. These frequencies challenge currently operational forecasting systems as the assimilation of observations at this rate is often prohibitively expensive and typically aggravates the problem of introducing imbalance into the analysis during the assimilation step. Hence, an Ultra-Rapid Data Assimilation (URDA) algorithm has been developed for the regional operational weather model ICON-LAM. The URDA methodology is based on the preemptive forecasting concept, which allows the generation of observationally constrained ensemble forecast while avoiding additional model reinitializations. Here the ensemble transformation matrix, as given for example by an ensemble square root filter, is employed to update a reduced set of the state variables of an existing model forecast. The feasibility of this new method has been successfully tested in both linear and nonlinear control systems, as well as in real-world applications, where the high resolved project observations were employed in a fast assimilation cycle of ICON-LAM.

QUALITATIVE ANALYSIS OF SOME REACTION-DIFFUSION SYSTEMS IN NEUROSCIENCE CONTEXT

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This talk will deal with a mathematical analysis of a non-homogeneous reaction-diffusion (RD) FitzHugh-Nagumo (NhFHN) type model and a related toy model. More precisely, we first consider the following system (NhFHN):

$$\begin{cases} \epsilon u_t &= f(u) - v + du_{xx}, \\ v_t &= u - c(x) \end{cases} \tag{1}$$

with $f(u) = -u^3 + 3u$ on a real segment (α, β) with Neumann Boundary Conditions (NBC). Introducing a space dependency in x in the coefficient c allows to take advantage of both oscillatory and excitability properties of the classical FitzHugh-Nagumo ordinary differential equations (see [7]) in a single RD system. This in turn provides a simple model able to generate a rich dynamical behavior such as bifurcations and wave propagation phenomena, see [2, 3, 4]. Those features are for example of interest for the modeling and understanding of waves of depolarization observed in the brain during various activities [6, 1]. Getting into the rigorous maths brings however various challenges. In order to capture some of the relevant theoretical approaches, we first consider the following toy model:

$$\begin{cases} \dfrac{du}{dt} &= -u^3 + \alpha u - v + du_{xx}, \\ \dfrac{dv}{dt} &= u \end{cases} \tag{2}$$

on the real segment $(0, 1)$ with NBC. For this model, explicit computations can me made and rigorous proofs of qualitative results can be provided. This results include a cascade

of Hopf bifurcations, stability of stationary solutions, stability of periodic solutions, coexistence of stationary and periodic solutions... Inspired by the analysis of equation (2), we will then illustrate how some results can be extended to the NhFHN model (1). The talk is based on the recent work [5].

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ANALYSING THE COVID-19 PANDEMIC IN ITALY WITH THE SIPRO MODEL

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We propose an epidemic compartmental model (SIPRO) that extends the classical SIR, in order to account for the proportion of unobserved infected people that have not been tested. The model is applied to the epidemic curves of the 20 Italian regions using Bayesian methods for mixed-effects models. In particular, we focus on the time-dependent effective reproduction number to reflect the impact of containment measures. Our model allows us to describe, estimate and predict the different phases of the pandemic (before the advent of vaccination) with a single model. We compare the performance of the SIPRO model with those of the simple SIR, and we show that without losing predictive power, we can give a reasonable estimate of the proportion of unobserved invections. We check our results against other works and techniques published in the last months.

MODELING DENGUE IMMUNE RESPONSES MEDIATED BY ANTIBODIES: INSIGHTS ON THE BIOLOGICAL PARAMETERS TO DESCRIBE DENGUE INFECTIONS

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Dengue fever is a viral mosquito-borne disease, a significant global health concern, with more than one third of the world population at risk of acquiring the disease. Caused by 4 antigenically distinct but related viruse serotypes, named DENV-1, DENV-2, DENV-3 and DENV-4, disease severity is associated with the immunological status of the individual, seronegative or seropositive, prior to a natural dengue infection.

Infection by one serotype confers lifelong immunity to that serotype and a short period of temporary cross immunity to other related serotypes. Severe dengue is epidemiologically associated with a secondary infection caused by a heterogeneous serotype via the so-called Antibody-Dependent enhancement (ADE) process, where the pre-existing antibodies act enhancing the new infection.

In this paper, we revisit the within-host modelling framework proposed by Sebayang, A.A et al. in 2021, developed to describe qualitatively the dengue immunological re-

sponses mediated by antibodies. A detailed analysis of three proposed models are presented. Using numerical simulations, we investigate features of viral replication, antibody production and infection clearance over time for three possible scenarios: primary infection, secondary infection caused by a homologous serotype and secondary infection caused by a heterologous serotype. Our models can describe well the information available in empiric immunology literature, giving insights on the immunopathogenesis of severe dengue secondary infection caused by a heterologous serotype.

Our models are under refinement, and the current system studied here is used as a baseline to understand the results of a more complex system. The results presented here are of use for future research directions to evaluate the impact of imperfect vaccines.

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ACHIEVING PEST MANAGEMENT USING FEEDBACK CONTROL FOR AN ADDITIONAL FOOD PROVIDED PREY-PREDATOR SYSTEM WITH TYPE III FUNCTIONAL RESPONSE

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The study of eco-systems where food supplements are provided to the predator in addition to the target prey has gained prominence over the years because additional food has proven to be very effective in controlling invasive or harmful species [1, 2, 4]. Additional food supplements affect the survival, gustatory response, fitness, fecundity and longevity of the natural enemies and this, in turn, helps in controlling pests in the system [1, 7, 8]. Outcomes from the mathematical studies of additional food systems reveal that the provision of additional food to the predators affects the global dynamics of the system, thereby influencing the eventual state of the system. In any mathematical model that describes a prey-predator interaction, one salient feature that characterizes the nature of this interaction is the Functional Response, which is defined in [3] as the rate at which the predator captures prey. Among various functional responses exhibited by species, one response with prey density dependence is the Holling type III functional response, also called the sigmoidal response.

This study aims to drive the system to the desired terminal state leading pest control in finite time. We consider an additional food system with type III response, where additional food is explicitly incorporated into the predator's compartment. We define an error system and associated with that, we formulate and study an optimal control problem based on the Dynamic programming approach, leading to the optimal solution being a Linear Feedback Control using the Algebraic Riccati Equation [5]. We illustrate the theoretical findings with numerical simulations. The results suggest strategies for eco-managers in the inundative bio-control scenario to manage pest without eliminating them from the system.

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ANALYSING THE IMPACT OF CELLULAR ADHESION ON KERATINOCYTE GROWTH FACTOR ACTIVITY IN EPIDERMAL WOUND

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Cells act to respond to many cues, such as chemical stimuli or environmental factors. Cells intrinsically desire to move randomly; however, their passive movement is inevitable due to a cell's interaction with its neighbouring (attractive and repulsive forced), termed adhesion movement.

The tendency of cells to physically adhere to other cells or tissue elements is classified within the non-local process, and non-locality can arise during either diffusional motion of cells or adhesion movement. The latter one has been broadly used in the literature to bring some developed mathematical models based on the importance of cell motility into a more realistic form. This type of model was firstly derived by Armstrong et al. to analyse the cell adhesion ability on the sorting behaviours of cells [1]. Armstrong's proposal has been further developed and applied to various contexts, such as in tissue development and mostly degenerative diseases (cancer); however, its application to tissue repair (wound healing) has remained insufficient. Therefore, we are interested in exploring certain aspects of cell adhesion on epidermal wound healing based on our previous model, which was investigated to analyse the KGF activity on epidermal wound healing [2].

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USING FRACTAL GEOMETRY TO QUANTIFY THE COMPLEXITY OF NATURE

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A "measure" is a mathematical way of identifying a given set with a numerical value, so that, if we divide the set into a countable number of smaller parts, the measure of all smaller sets sum up to the measure of the whole set. According to what we know from elementary Euclidian geometry, line segments and lines have dimension 1, polygons and circles have dimension 2, while pyramids, cubes and spheres have dimension 3.
Definition: [Hausdorff-Besicovitch dimension] For every $E \subset \mathbb{R}^k$, there is exactly one real number s_0, $0 \leq s_0 \leq K$, such that

$$H^t(E) = \begin{cases} 0, & t > s_0 \\ +\infty, & t < s_0 \end{cases}$$

The number

$$s_0 = \sup\{t \geq 0 \,:\, H^t(E) = +\infty\} = \inf\{t \geq 0 \,:\, H^t(E) = 0\}$$

is called Hausdorff-Besicovitch dimension of E and it is denoted by $dim_H E$.
The length of a line segment, the area of a triangle and the volume of a cube are quantities that can be measured easily. One may think that fractals are only the result of mathematical calculations but the most interesting point is that many things in nature have fractals' properties in them. But, how can we measure these complex objects, such as the length of the Cantor set, or the area of the Sierpinski triangle?

Obviously, this is impossible through conventional mathematical tools. In these cases, we employ the so called Hausdorff measure. There are, also, several other ways to "measure" and compare fractal sets. These are, usually, called fractal dimensions and measure the density of the space a fractal set occupies.
Definition: [Fractal dimension] Let A be a bounded set of $\mathbb{R}^k$. Let $N(A, \epsilon)$ be the mini-

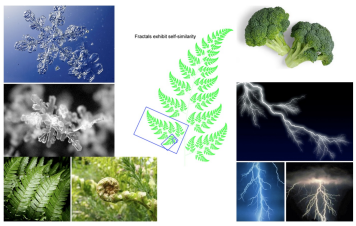

Figure 1: Fractals in nature

mum number of ϵ-radius balls that are needed to cover A. If the limit

$$D(A) = \lim_{\epsilon \to 0} \{ \frac{\ln\left(N(A, \epsilon)\right)}{\ln(\frac{1}{\epsilon})} \}$$

exists, then $D(A) = dim_B A$ is called the Fractal dimension of A.
We will employ these definitions to quantify the complexity of fractals in nature.

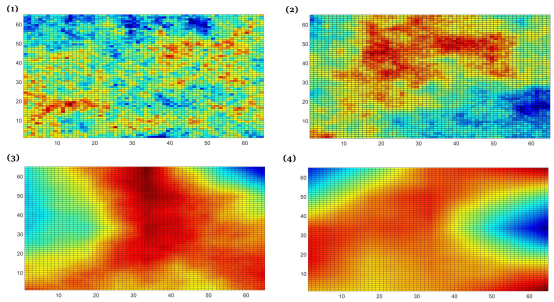

Figure 2: Fractal and random surfaces in $\mathbb{R}^2$

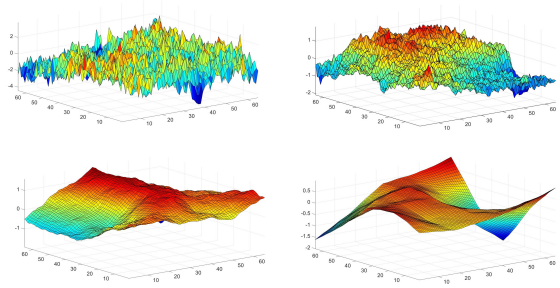

Figure 3: Fractal and random surfaces in $\mathbb{R}^3$

MATHEMATICAL MODELING AND OPTIMAL CONTROL STRATEGIES FOR COVID-19 IN INDIA : IMPACT OF FACE MASK AND QUARANTINE

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In this work, we propose a deterministic mathematical model to understand the transmission dynamics and control strategies of COVID-19 in a population that incorporates the efficacy of face-masks and contact tracing of infected individuals. We discuss the local and global stability of equilibria in terms of the basic reproduction number (R_0). and also show that the system backward bifurcation in case of imperfect quarantine (quarantine with low efficacy). We estimate the model parameter corresponding to the transmission rate and proportion of individuals of the public who cover the mouth by mask in the public palace using the real data of cumulative confirmed cases of COVID-19 for India by the Markov chain Monte Carlo (MCMC) method. We also validate the obtained theoretical results through numerical simulation and perform sensitivity analysis to quantify the key parameters which affect the basic reproduction number and hence control the transmission dynamics of COVID-19. Finally, we extend our proposed model to the optimal control problem to measure the best cost-effective and time-dependent control strategies that can reduce the number of infected in a specified interval of time. In the numerical simulations, we concluded that reducing R_0 below unity is not sufficient enough to eradicate the COVID-19 disease and thus, it is required to increase the quarantine rate and its efficacy by motivating individuals to take precautionary measures. Our finding suggests that quarantine with low efficacy may be harmful in controlling the COVID-19 confirmed cases in India due to the bistable behavior of the system. The efficacy of face masks also plays a significant role to reduce the COVID-19 prevalence in India. We hope that the finding may be helpful to policymakers to make decisions regarding the control strategies.

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MODELLING VIRAL EXPOSURE IN TRANSPORT SETTINGS

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A significant component of COVID-19 transmission occurs through close interactions between infected individuals in enclosed spaces. Few environments result in as many close interactions as public transport settings, where an individual may come into contact with a large number of passengers in a relatively short time period. We use a Quantitative Microbial Risk Assessment (QMRA) approach to model COVID-19 transmission within a subway train carriage and a local bus. We model each passenger's exposure to COVID-19 via small aerosol, close range droplet and fomite pathways. Our model includes stochasticity to account for variability in model parameters (such as transfer efficiencies) and passenger behaviour (such as passenger positioning, boarding and alighting patterns, respiratory behaviour and surface touch frequency). We explore the impact of factors such as mask wearing, loading, ventilation and disease prevalence to provide insight into the key factors which impact COVID-19 risk. We particularly consider at how changes in parameters influence the distribution of exposure risks and therefore have different relative impacts on median risks and outliers for the three modes of transmission included within the model.

A MULTI-STAGE MODEL OF CELL PROLIFERATION AND DEATH: TRACKING CELL DIVISIONS WITH ERLANG DISTRIBUTIONS

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Lymphocyte populations, stimulated *in vitro* or *in vivo*, grow as cells divide. Stochastic models are appropriate because some cells undergo multiple rounds of division, some die, and others of the same type in the same conditions do not divide at all. If individual cells behave independently, each can be imagined as sampling from a probability density of times to division. The most convenient choice of density in mathematical and computational work, the exponential density, overestimates the probability of short division times. We have considered an existing multi-stage model of cell proliferation [4] that proposes an Erlang distribution of times to division, and extended it to incorporate a competing exponential distribution of times to death [1]. The underlying idea is to divide the cell cycle into a given number of stages, and the cell is required to sequentially visit each stage in order to divide. At each stage, each cell may either proceed to the next one or die. Cells can be classified across generations depending on the number of times that they have undergone cell division, and the interest is in estimating the number of cells in each generation over time, which can be then compared to appropriate experimental data. Using Approximate Bayesian Computation based on Sequential Monte Carlo (ABC-SMC) methods [3], we compare our model to published cell counts, obtained after CFSE-labelled OT-I and F5 T cells were transferred to lymphopenic mice [2]. The death rate is assumed to scale linearly with the generation and the number of stages of undivided cells is allowed to

differ from that of cells that have divided at least once. Multiple stages are preferred in posterior distributions, and the mean time to first division is longer than the mean time to subsequent divisions.

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IMPACT OF COOPERATIVE BEHAVIOUR ON THE STABILITY OF A DELAYED PREDATOR-PREY MODEL WITH HOLLING FUNCTIONAL RESPONSE

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In this paper, we propose and analyze a delayed predator-prey model with Holling functional response taking into account cooperation behavior in predators. We investigate the effect of hunting cooperation on both the number and the level of positive steady states. We observe that the level of the positive equilibrium decreases when increasing the hunting cooperation parameter. Then we study the impact of the delay as well as the cooperation in hunting on the dynamics of the system. We prove that the presence of delay in the attack rate induces stability switches around the coexisting equilibrium when predators cooperate. In addition, we consider the discrete delay as a bifurcation parameter and prove that the model undergoes a Hopf-bifurcation at the coexisting equilibrium when the delay crosses some critical values. Numerical simulations are presented to confirm our analytical findings

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MATHEMATICAL MODEL FOR COMPUTING COLORECTAL CANCER CELL'S STEADY STATE FOR DRUG REPOSITIONING AND DOSAGE

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G1-S transition phases of colorectal cells are complex phenomena during which many protein reactions take place. Some of these reactions are particularly critical along this phase since mutations affecting some proteins' function can cause diseases as cancer.
A properly designed chemical reaction network (CRN) is able to describe how these proteins and their compounds interact with each other.

From a mathematical viewpoint, CRNs can be modelled like systems of autonomous ordinary differential equations, whose steady state solutions yield the cell's proteins concentrations [1] [2]. Finding such states is a key step to understand the global effects induced by each mutation and may help to determine which drugs to administer in order to balance mutations' effect and bring cells back to the physiological state.

Steady states of cell's proteins are usually computed by simulating the system's dynamical evolution in time [3]. However, this is a very time-consuming process, especially when the CRN's size increases.

Here we propose a different method, by recasting the steady state computation problem as a root-finding one: to solve the latter, we compute the system's equilibrium points by means of an algorithm that combines the Newton method and the gradient descent approach. The non-negativity constraints on the steady state concentrations are assured by introducing and applying a suitable operator P at the end of every iterative step.

Some simulation results are presented to show the method's performance when working with CRNs modelling both colorectal cells in a physiological state or in the presence

of mutations. By comparing the described approach to the same model but using standard orthogonal projection on the positive convex cone instead of P, we will show how the former reaches a better convergence rate, proving the benefits of choosing P.
This method also outperforms the classic dynamic approach both in terms of speed and accuracy.

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MATHEMATICAL MODELLING TO CONTROL WILD MOSQUITO WITH STERILE RELEASE

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Mosquito-borne diseases like dengue, malaria, Zika, yellow fever, chikungunya, etc. viruses have been a bane for numerous years for the human population. There have been noticeable concerns regarding this in public health. More than one million people die from mosquito-borne diseases every year globally. More than two-thirds of the world population is at risk of mosquito-borne diseases.

With the weight of mosquito-borne diseases increasing, humans adapt various control and protection measures, and the sterile insect technique (SIT) is a reliable weapon in the stash. The method first came into light by an American entomologist, Professor Edward F. Knipling, in the late 1930s. SIT is a scheme of pest control in which a genetic mutation might control the population of insect species. When wild female mosquito mates with a sterile male mosquito, it is ineffective. Keeping sterile-to-wild mosquito ratio in mind, if significant numbers of sterile males are released, so females are mated, the total wild population will reduce in the next generation.

We frame two-dimensional non-linear ODE's to analyze the wild mosquito population with releases of sterile mosquitoes as a linear function. We study the existence and stability for all possible equilibria of the model system. We obtain codimension one saddle node bifurcation, and codimension two cusp bifurcation. We identified when the control is more complex and obtained the threshold density of proportional release of sterile male mosquitoes in several cases to control the wild population.[1, 2, 3, 4, 5].

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COVID-19 EPIDEMICS' STRATEGIES : MOBILITY, TESTING AND VACCINATION SHOT DYNAMICS

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The COVID-19 epidemic has hit the state of Rio de Janeiro and Brazil seriously, leading to the implementation of social distancing, lockdown and testing strategies often as a choice of local administrations. In this study, we investigate some municipalities of the state of Rio de Janeiro as a network of regions (municipalities) and data from Italian regions. The objective is to identify if and when measures adopted by municipal or regional governments have had an effect. Furthermore, we investigate the effects of regional heterogeneity and inter-regional flow on the spread of the epidemic and use theoretical tools to propose and evaluate different interventions at the regional level to reopen the state while avoiding future epidemic outbreaks. Some testing and vaccination strategies are also considered.

Based on work by Della Rossa, F., Salzano, D., Di Meglio, A. et al. [3] for Italy, we created a network model where each node represents a municipality in the metropolitan region of the state of Rio de Janeiro and its edges depict the flow between regions. We use a data-based compartmental model approach (among which a lockdown measure through cell tracking), from which we obtain a set of ordinary differential equations describing the dynamics of seven different compartments for each region (Susceptible, Asymptomatic, Symptomatic , Hospitalized, Quarantined, Dead and Recovered) considering the interregional flows as proposed by Stolerman, LM, Coombs, D. & Boatto, S [4] and also different transmission rates generated by asymptomatic and symptomatic individuals; using data, we perform analyzes to infer flows between compartments. A discrete particle model is also considered [1, 2].

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OPTIMAL SUSTAINABLE HARVESTING POLICIES IN RANDOM ENVIRONMENTS: ESTIMATION OF MOMENTS AND DENSITY OF FIRST PASSAGE TIMES

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In random varying environments, we can describe the evolution of a fished population size using stochastic differential equations. Previously (see [2, 3, 4, 5, 6, 7, 8]), we have compared the profit performance of two harvesting policies, one with variable harvesting effort, called optimal policy, and the other with constant harvesting effort, called optimal sustainable policy. The former is characterized by fast and abrupt variations of the harvesting effort associated with the frequent variations in population size due to the random environmental fluctuations. This type of policy is inapplicable due, for instance, to the logistics of the fisheries being incompatible with abrupt and frequent changes in the harvesting effort. It also poses social problems during the periods of no or low harvesting effort. Furthermore, this type of policy requires the knowledge of the population size at each instant and estimating population size is an inaccurate, lengthy and expensive task. The optimal sustainable policy considers the constant application of the same harvesting effort and leads to population sustainability, as well as to the existence of a stationary probability density for the population size (see [1]). This policy has the advantage of be-

ing easily applicable and there is no need to estimate the population size at every instant. The performance of the two policies was compared in terms of the profit over a finite time horizon. Using data based on a real fished population, we showed that there was only a slight reduction in profit by using the optimal sustainable policy (based on constant effort) instead of the inapplicable optimal policy (based on variable effort).

For the class of models with constant fishing effort, mathematical extinction (population size $X(t)$ converging to zero) occurs with zero probability. However, since we work with ergodic processes, all states in the interior of the state space are attainable with probability one in finite time. In particular, we can consider a threshold $y > 0$ and study how long it takes for the process $X(t)$ to reach y for the first time. This threshold can be a low biological reference point $y < X(0)$, i.e., a minimum biomass value below which the population self-renewable capacity is endangered. It can also be some high biomass level $y > X(0)$ that is important for the fishery, such as a warning level of danger to the survival of another species or to possible deviations from the optimal fishing effort.

Based on general expressions for the mean and standard deviation of first passage times by lower and upper thresholds, we compute such values for the particular cases of the logistic and the logistic-like models with weak Allee effects and for several lower and upper threshold values y. For a fixed threshold value, we also present a way to estimate, by numerical inversion of its Laplace transform, the probability density function of the time to reach the threshold. To check the quality of this estimate, we compare the mean and standard deviation of the first passage time obtained by using this estimated probability density function with the mean and standard deviation obtained directly.

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MATHEMATICAL MODELING OF INTERVENTION AND LOW MEDICAL RESOURCE AVAILABILITY WITH DELAYS: APPLICATIONS TO COVID-19 OUTBREAKS IN SPAIN AND ITALY

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Infectious diseases have been one of the major causes of human mortality, and mathematical models have been playing significant roles in understanding the spread mechanism and controlling contagious diseases. In this paper, we propose a delayed SEIR epidemic model with intervention strategies and recovery under the low availability of resources. Non-delayed and delayed models both possess two equilibria: the disease-free equilibrium and the endemic equilibrium. When the basic reproduction number $R_0 = 1$, the non-delayed system undergoes a transcritical bifurcation. For the delayed system, we incorporate two important time delays: τ_1 represents the latent period of the intervention strategies, and τ_2 represents the period for curing the infected individuals. Time delays change the system dynamics via Hopf-bifurcation and oscillations. The direction and stability of delay induced Hopf-bifurcation are established using normal form theory and center manifold theorem. Furthermore, we rigorously prove that local Hopf bifurcation implies global Hopf bifurcation. Stability switching curves and crossing directions are analyzed on the two delay parameter plane, which allows both delays varying simultaneously. Numerical results demonstrate that by increasing the intervention strength, the infection level decays; by increasing the limitation of treatment, the infection level increases. Our quantitative observations can be useful for exploring the relative importance of intervention and medical resources. As a timing application, we parameterize the

model for COVID-19 in Spain and Italy. With strict intervention policies, the infection numbers would have been greatly reduced in the early phase of COVID-19 in Spain and Italy. We also show that reducing the time delays in intervention and recovery would have decreased the total number of cases in the early phase of COVID-19 in Spain and Italy. Our work highlights the necessity to consider the time delays in intervention and recovery in an epidemic model.

DIRECT SIMULATION OF THE COVID19 EPIDEMIC

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We introduce an agent-based model to simulate the epidemiological dynamics of COVID-19. Most computational models proposed to study this epidemic do no take into account human mobility. We present a direct simulation model where mobility plays a key role and propose as well four quarantine strategies. The results show that the no-quarantine strategy does lead to a high peak of contagions with no rebound. Quarantined strategies, for their part, show a re-emergence of the epidemic with smaller and softer peaks.

MODEL CALIBRATION FOR AGGREGATION-SEDIMENTATION DYNAMICS

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The understanding of the carbon cycle is an extremely relevant scientific challenge with important implications. Of particular interest is the fact that sedimentation of large amounts of CO_2 to the bottom of lakes and oceans can have an impact in the mitigation of global warming. This sedimentation process involves the formation of particulate organic carbon aggregates, among which the heavier ones gradually sink to the bottom. There are quantitative models (both ODE and PDE models) in Aquatic Sciences that try to capture this aggregation-sedimentation dynamics; however, our knowledge about how does aggregation work in this context is presently quite limited. Model calibration (particularly in its non-parametric version) can be used as a means to determine the nature of aggregation interactions indirectly. In this talk we will review some of the existing techniques and show some preliminary results on specific applications. The set of questions we address here are also relevant for similar mathematical models in the study of protein polymerization and neurodegenerative diseases (e.g. works by Nowak et al. [2] and works by Doumic et al. [1]).

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SEIRS REACTION-DIFFUSION MODEL ANALYSIS AND APPLICATION

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This work seeks to understand the effect of the inclusion of spacial heterogeneity in a SEIRS transmission model by considering reaction-diffusion SEIRS model, following the work in [1]. The disease free equilibrium (DFE) is established and its asymptotic profile determined depending on the basic reproduction number, R0. The basic reproduction number is computed by the next generation method developed in [3]. A numerical method based on finite difference schemes are considered to approximate the solution of the reaction-diffusion system defined on a circular domain in the real plane [2].

Lastly, the model is parametrized according to studies for COVID-19 transmission dynamics in Portugal, for the period prior to vaccination. We compare the model results with non spatial models and we conclude that spatial heterogeneity can explain fluctuations in the notification curve, independently of changes in transmission intensity, due to the existence of local epidemics occurring at different time points.

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WHY INSECTS MAY PREFER TO FEED ON TOXIC PLANTS?

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Plants of the Brassicaceae have a two-component chemical defense system consisting of defense glucosinolates (GLSs) and their corresponding glucohydrolases (myrosinases), which are usually stored in separate compartments in plant leaves. The stored GLSs are not toxic but hydrolyzed by myrosinases upon herbivory to produce isothiocyanates (ITCs), which have been found to be toxic to generalist feeding insect herbivores [2]. Although ITCs cause adverse effects (low growth rate, high mortality, low reproductive output) to generalist feeders, surprisingly some of them (e.g. *Spodoptera exigua, S. littoralis, Mamestra brassicae, Trichoplusia ni and Helicoverpa armigera*) [1] are still regular pests of Brassicaceae. So, an important question is why some generalists actually prefer to oviposit on the plants of Brassicaceae instead of plants that do not make toxic ITCs? The answer may lie in escape from competition with other herbivores that occurs on less toxic host plants. We have investigated the conditions under which it may be beneficial for generalist feeding herbivores to shift to toxic host plants. Using ordinary differential equation models, we have discovered specific circumstances under which generalist insect herbivores may perform better on Brassicaceae plants that produce toxic ITCs.

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IMPACT OF ENVIRONMENTAL TOXICITY AND INFECTIOUS DISEASE ON CONSUMER POPULATION

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The study of infectious disease has been of interest to ecologist since long. Only in recent years, host-resource dynamics has gained a lot of attention in this context. Nature of the underlying consumer (host)-resource model can largely influence the initiation of epidemic and the long term disease dynamics[1]. However, evidences suggest ecological traits related to such inherent systems may be often modulated by environmental toxins[2, 3]. This, in addition to toxin mediated alteration of epidemiological traits, has significant impact on disease progression in ecosystems which is quite less studied. In order to address this, we consider a mathematical model of resource-consumer system with disease in the consumer, where multiple traits are affected by environmental toxin. Long term dynamics show that the level of environmental toxin determine the persistence of disease in a community. Furthermore, the model exhibits bistability between different ecosystem states and for some specific toxicity range, it demonstrates abrupt transition from disease free coexistence states to disease induced extinction of consumer. Overall the results from this study help us gain fundamental insights into disease propagation in natural ecosystems in the face of present anthropogenic changes.

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DIFFERENTIAL-DIFFERENCE KERMACK-MCKENDRICK EPIDEMIC MODEL WITH AGE-STRUCTURED PROTECTION PHASE

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We consider a general class of Kermack-McKendrick SIR epidemic models with an age-structured protection phase with limited duration, for example due to vaccination or drugs with temporary immunity. The characteristics method reduces the model to a coupled system of a differential equation and a continuous difference equation with a time-delay. We investigated the local and the global asymptotic stability of the two steady states: disease-free and endemic. We prove that the global stability is completely determined by the basic reproduction number. After this studies, we considered the diffusive case. We reduced the model by using the method of characteristics, to a reaction-diffusion system coupled with a delayed difference equation. We study the existence and non-existence of non-trivial traveling wave solutions. We get almost complete information on the threshold and the minimal wave speed that describes the transition between the existence and non-existence of non-trivial traveling waves that indicate whether the epidemic can spread or not.

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DYNAMICS OF FEEDBACK SIGNALLING IN B LYMPHOCYTES DEVELOPMENT

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Haematopoiesis is the process of generation of blood cells. Lymphopoiesis generates lymphocytes, the cells in charge of the adaptive immune response. Disruptions of this process are associated with diseases like leukaemia, which is especially incident in children. The characteristics of self-regulation of this process make them suitable for a mathematical study, as it has been previously done for leukemia in general [2, 3].

In our work [1] we develop mathematical models of lymphopoiesis using currently available data. We do this by drawing inspiration from existing structured models of cell lineage development and integrating them with paediatric bone marrow data, with special focus on regulatory mechanisms. A formal analysis of the models is carried out, giving steady states and their stability conditions. We use this analysis to obtain biologically relevant regions of the parameter space and to understand the dynamical behaviour of B-cell renovation. Finally, we use numerical simulations to obtain further insight into the influence of proliferation and maturation rates on the reconstitution of the cells in the B line.

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SEASONALLY DEPENDENT COMPETITIVE KOLMOGOROV SYSTEMS: EXTINCTION OR COEXISTENCE?

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We consider a periodic Kolmogorov system describing two species nonlinear competition. Under assumptions that generalise Gopalsamy conditions, we discuss coexistence or extinction of one or both species, and describe the domain of attraction of nontrivial periodic solutions on the axes.

Our results may be applied to models with nonlinear competition, which have been studied in the biological literature, such as models of microbial growth or of phytoplankton competition under the effect of toxins.

Finally, we consider a two species amensalism model, a biological interaction in which one species may harm the other, but the reverse is not true.

Based on joint work [1, 2] with Carlota Rebelo and Elisa Sovrano.

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MACROSCOPIC MODELING OF VACCINATION CAMPAIGNS

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We present a modeling framework where vaccination strategies can be simulated, tested and compared evaluating their effects against the propagation of a virus.

Different vaccines can be dosed at prescribed ages or at prescribed times to prescribed portions of the susceptible population. The quality of the immunization provided by each vaccine, in particular its duration, explicitly enters the model. The level of immunization provided by recovering from the disease can fade with time, possibly disappearing after a given period. Differences due to individuals' ages are accounted for through the introduction of either a continuous age structure or a discrete set of age classes.

The solutions to the resulting equations share several qualitative features with the present day pandemic. In particular, for instance, they display epidemic waves, whose persistence depends on vaccines' efficiency and on vaccination strategy, and justify to some extent the term *herd immunity*.

Analytically, this framework leads to mixed ODE-PDE systems that are non linear, contain non–local terms and where different time scales coexist. They can be seen as compartmental models, where some compartments have a sort of internal dynamics. Seeking optimal vaccination strategies leads to a variety of new control problems, but, in some cases, already well posedness appears as being still an open question.

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HUMAN MOBILITY AND MULTI-PATCH MODELS OF INFECTIOUS DISEASE DYNAMICS

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Human mobility plays an important role in driving infectious disease dynamics between connected communities [2]. Spatially-extended epidemiological models offer a suitable modelling framework for the study of these effects on the considered populations [1, 3]. Highly detailed mobility data is however often hard to obtain and hence care ought to be taken in the definition of model parameters.

In this talk, I will present two simplified approaches to account for mobility effects in a multi-patch modelling framework, highlighting the respective strengths and weaknesses. Some theoretical considerations will be made for the particular case of two interacting patches, while numerical simulations will be used in the more general multi-patch case. Finally, I will discuss the application of the proposed ideas to modelling the dynamics of infectious disease transmission within the Basque Country and present the main challenges we expect to encounter in doing so.

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MATHEMATICAL MODELING OF IMPACT OF SCREENING AND SATURATED TREATMENT ON SPREAD OF COVID-19: A CASE STUDY IN INDIA

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Prediction of actual infective cases for COVID-19 disease is an improbable task as individual without showing symptoms spread the infection. This characteristic is causing hurdle in controlling the disease among other factors. In this work, we modify the model in [1] and introduce two important factors - screening of the asymptomatic cases and provision of treatment to infected individuals. The screening has two components: one - a baseline constant screening and another- infective density induced additional screening (IDIAS). The IDIAS is additional screening which is based on number of infective cases reported. The model is analyzed in both analytically and numerically. The screening rate and quarantine rate in two high infection states of India are estimated using data - fitting technique. We also provide the effect of various control factors on the spread of disease. It is observed that the availability of treatment and screening of asymptomatic people play major role in reducing the disease burden.

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ANALYTICAL AND COMPUTATIONAL STUDY OF THE SPREAD OF INVASIVE SPECIES IN THE PRESENCE OF A ROAD IN THE SPATIAL DOMAIN

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Invasive plant species pose a significant threat to biodiversity, and their management is often resource-intensive and expensive. Evidence suggests that roads can have an important effect on the spread of invasive plant species [2], although little is known about the underlying mechanisms at play. We have developed a mathematical integro-difference equation based model that incorporates a road in the spatial domain. Integro-difference equations have previously been applied in a number of areas, from modelling populations of plants [1], to the spread of tree disease by beetle [3].

Our model allows for taking into account a different propagation speed of invasive plants over the road. Analysing both logistic and Allee growth functions, analytical and computational findings on how roads can impact the spread of invasive species lead to three distinct types of behaviour in front of the road. We show that roads can act as barriers to invasion, lead to a formation of a beachhead in front of the road, or act as corridors allowing the invasive species to invade the domain in front of the road. We then argue a small change in conditions of the environment favouring the invasive species can change the case for the road, allowing the invasive species to invade the domain in front of the road where it previously could not.

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CAN HUMAN VACCINATING BEHAVIOUR INFLUENCE MUTANT INVASION ALTERING PATHOGEN COMPETITION? A GAME-THEORETIC ANALYSIS

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Competition between multiple strains may lead to exclusion of some pathogens, or may result in the invasion of a novel strain. Previous studies have not emphasized much on the influence of human vaccination choice on pathogen competition or strain invasion. Here we couple imitation dynamics analysis and SIR model of two strains, to explore invasion and persistence of a mutant in the population despite having a lower fitness rate than the resident one. We demonstrate that higher perceived strain severity and lower perceived vaccine efficacy are necessary conditions for the persistence of a mutant strain. This result is important in discussing public health implications since proper risk communication in public about the perceived severity of the disease is an important task to reduce the chance of mutant invasion. which is a crucial component for policymakers for strategic decision-making.

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THE INFLUENCE OF A DEMAND FUNCTION ON THE GLOBAL DYNAMICS OF THE FISH STOCKS

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Mathematical models in the field of fishing make it possible to predict the qualitative evolution of the fishery, in particular major trends such as the collapse of the stock or its maintenance, the variation in fishing effort, or even the surge or stabilization of the market price.
We will analyze and study the importance of using a linear demand function on the maintenance of the fish stock and on its global dynamics, by exploiting the results obtained by the work of P. Auger, R. Mchich and N. Raissi in [4]. So we are going to generalize these last results by a non-linear demand function which increases rapidly at the start (with respect to the demand) before decreasing afterwards.

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IMPACT OF VACCINE SUPPLIES AND DELAYS ON OPTIMAL CONTROL OF THE COVID-19 PANDEMIC: MAPPING INTERVENTIONS FOR THE PHILIPPINES

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Around the world, controlling the COVID-19 pandemic requires national coordination of multiple intervention strategies. As vaccinations are globally introduced into the repertoire of available interventions, it is important to consider how changes in the local supply of vaccines, including delays in administration, may be addressed through existing policy levers. This study aims to identify the optimal level of interventions for COVID-19 from 2021 to 2022 in the Philippines, which as a developing country is particularly vulnerable to shifting assumptions around vaccine availability.

Embedding our work within the local policy landscape, we apply optimal control theory to the compartmental model of COVID-19 used by the Philippine government's pandemic surveillance platform and introduce four controls: (a) precautionary measures like community quarantines, (b) detection of asymptomatic cases, (c) detection of symptomatic cases, and (d) vaccinations. Optimality conditions are identified using Pontryagin's minimum principle.

Simulation results indicate that early and effective implementation of both precautionary measures and symptomatic case detection is vital for averting the most infections at an efficient cost, resulting in $> 99\%$ reduction of infections compared to the no-control scenario. Expanding vaccine administration capacity to $440\,000$ full immunizations daily will reduce the overall cost of optimal strategy by 25%, while allowing for a faster relaxation of more resource-intensive interventions. Furthermore, delays in vaccine administration require compensatory increases in the remaining policy levers to maintain a minimal number of infections. For example, delaying the vaccines by 180 days (6 months) will result in an 18% increase in the cost of the optimal strategy. We emphasize three key takeaways of (a) sustaining efficient case detection, isolation, and treatment strategies; (b) expanding not only vaccine supply but also the capacity to administer them, and; (c) timeliness and consistency in adopting policy measures.

Acknowledgements

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MODELLING, ANALYSIS, OBSERVABILITY AND IDENTIFIABILITY OF EPIDEMIC DYNAMICS WITH REINFECTIONS

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We consider the following general SEIRS model describing the dynamics of an infectious disease including latency, waning immunity and infection-induced mortality :

$$\dot{S} = bN - \beta S\frac{I}{N} + \omega R - \mu S, \tag{3a}$$

$$\dot{E} = \beta S\frac{I}{N} - (\sigma + \mu)E, \tag{3b}$$

$$\dot{I} = \sigma E - (\gamma + \mu + \nu)I, \tag{3c}$$

$$\dot{R} = \gamma I - (\omega + \mu)R, \tag{3d}$$

from which we derive an infinite system of differential equations that provides an image of the same infection process, but counting also the reinfections :

$$\dot{S}_i = \omega R_{i-1} - \beta S_i\frac{I}{N} - \mu S_i, \quad i \geq 1, \tag{4a}$$

$$\dot{E}_i = \beta S_i\frac{I}{N} - (\sigma + \mu)E_i, \quad i \geq 1, \tag{4b}$$

$$\dot{I}_i = \sigma E_i - (\gamma + \mu + \nu)I_i, \quad i \geq 1, \tag{4c}$$

$$\dot{R}_i = \gamma I_i - (\omega + \mu)R_i, \quad i \geq 1, \tag{4d}$$

This system with infinite differential equation is similar to Becker-Döring equations [1, 2]. Very few studies in epidemiology included compartmental models which count reinfections and in our knowledge, only Katriel [3] proposed such a model to study. Existence

and uniqueness of the corresponding Cauchy problem is established in a suitable space of sequence valued functions, and the asymptotic behavior of the solutions is characterized, according to the value of the basic reproduction number. This allows to determine several mean numbers of reinfections related to the population at endemic equilibrium. In the second part of our studies, we derive an identifiability result for a SIS system counting primo-infection. System identifiability is only a recent and marginal topic in mathematical epidemiology, with the first study only dating back to 2003 [4]. We show in fact how using joint measurement of the number of primo-infected individual and of the number of infected gives rise to observability and identifiability, in the simpler case of an SIS model for which none of these two measures is sufficient to ensure on its own the same properties.

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COMPARATIVE ANALYSIS OF DIFFERENT THERAPEUTIC STRATEGIES AGAINST HBV INFECTION USING AN AGE-STRUCTURED MODEL

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In this talk I will present an age-structured model for the immune response to an HBV infection, which integrates contributions from both cell-mediated and humoral immunity [2]. This approach is based on the particle release profiles that we observed in our intracellular model [1]. The model has been validated using published patient data recorded during an acute infection, and has been adapted to the scenarios of chronic infection, clearance of infection, and flare-ups via variation of the immune response parameters. The impact of immune response exhaustion on the viral dynamics is analysed showing that it plays a crucial role in the control of the infection. I will also present a comparison of different treatment options in the context of this model which reveals that drugs targeting aspects of the viral life cycle are more effective than exhaustion therapy, a form of therapy mitigating immune response exhaustion. Our results suggest that antiviral treatment is best started when viral load is declining rather than in a flare-up. The model also suggests that a fast antibody production rate always leads to viral clearance, highlighting the promise of antibody therapies currently in clinical trials.

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EFFECTS OF CONDUCTANCE OF ION CHANNELS ON SPONTANEOUS ELECTRICAL ACTIVITY IN SMOOTH MUSCLES

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Spontaneous electrical activity is common in muscle cells. It may arise as a result of interaction between ion fluxes through the voltage-gated ion channels in the cell membrane [1]. The purpose of this study is to address the significant role of maximal ion conductance on spontaneous electrical signals (action potentials) propagating along the vessel wall of an artery. In this talk, I will discuss the influence of ion conductance on the behaviour of an isolated smooth muscle cell using numerical bifurcation analysis. We perform numerical continuation of the solutions by tuning physiological parameters including ion conductance to identify bifurcations underlying different excitability and show the threshold for transitions between the types of excitabilities in a two-parameter plane. Additionally, we consider a spatially extended model to investigate the dynamics of electrically coupled smooth muscle cells. Numerical simulations reveal various spatiotemporal pattern of the model for different values of the parameters.

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WHY ARE CELL POPULATIONS MAINTAINED VIA MULTIPLE INTERMEDIATE COMPARTMENTS?

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Populations of cells are efficiently maintained in our body through mechanisms that still need to be completely unraveled. Novel experimental techniques allow us to start tracking individual cells and their progeny. We think about "product cells" as mature and fully differentiated cells, and "progenitor cells" as cells able to "self-renew" and generate product cells. A commonly observed pattern in experiments is that a small population of progenitor cells maintains, via a sequence of intermediate states, a larger population of product cells.

Here, we model cells sharing the same attributes (or phenotype) as belonging to the same mathematical state (or compartment). Our compartmental model consists of a sequence of C compartments, containing cells which can either die, divide or change phenotype (entering the next compartment).
This model represents the dynamics of the progeny of a single progenitor cell as a stochas-

tic process. Moreover, we identify cells by their generation: the progenitor cell is said to be in generation zero and daughter cells of a cell in generation n are in generation $n + 1$. Since we are interested in the ultimate fate of the system, we proceed as in the theory of discrete-time branching processes, by defining relationships between random variables making use of probability generating functions.

If there is only one intermediate compartment, a large ratio of product cells to progenitors can only be achieved at the cost of the product cell population being dominated by large families of cells descended from individual progenitors, and large average number of divisions separating product cells from progenitors. These undesirable features can be avoided if there are multiple intermediate compartments. A sequence of compartments is, in fact, an efficient way to maintain a product cell population from a progenitor population, avoiding excessive clonality and minimising the number of rounds of division en route.

TOPOLOGICAL METHODS FOR REAL TIME DETECTION OF EPILETIC SEIZURES IN ELECTROENCEPHALOGRAPHIC RECORDINGS

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We propose a real-time computational method to track and detect epileptic seizures from raw neurophysiological recordings. Our mechanism is based on the topological analysis of the time series sliding window embedding [3] derived from the simultaneously recorded channels.

We extract topological features from the signals via the computation of the persistent homology [2] of time-varying point-clouds obtained from the sequential embedding of the time series in a high dimensional Euclidean space. The evolution of the signals is codified in a path of persistence diagrams whose approximate first derivative [1] captures the different states of the brain activity. Concretely, the values of the first derivative are in correspondence with the changes in the global neural activity, where high values indicate the start and end points of an epileptic seizure. The same procedure applied to a discriminative sliding window embedding for each channel (using Takens delay embedding's theory [4]), may capture the information of the specific channel(s) where the seizure starts and/or

generalizes (see Figure 1).

This approach provides a scalable computational procedure to track the simultaneous evolution of raw neurophysiological recordings in real time, that is robust to noise due to the stability property of persistent homology. We apply our methods in different types of signals including scalp and intracerebral electroencephalography (EEG) and magnetoencephalography (MEG), in patients during interictal and ictal states, showing high accuracy in a range of clinical situations.

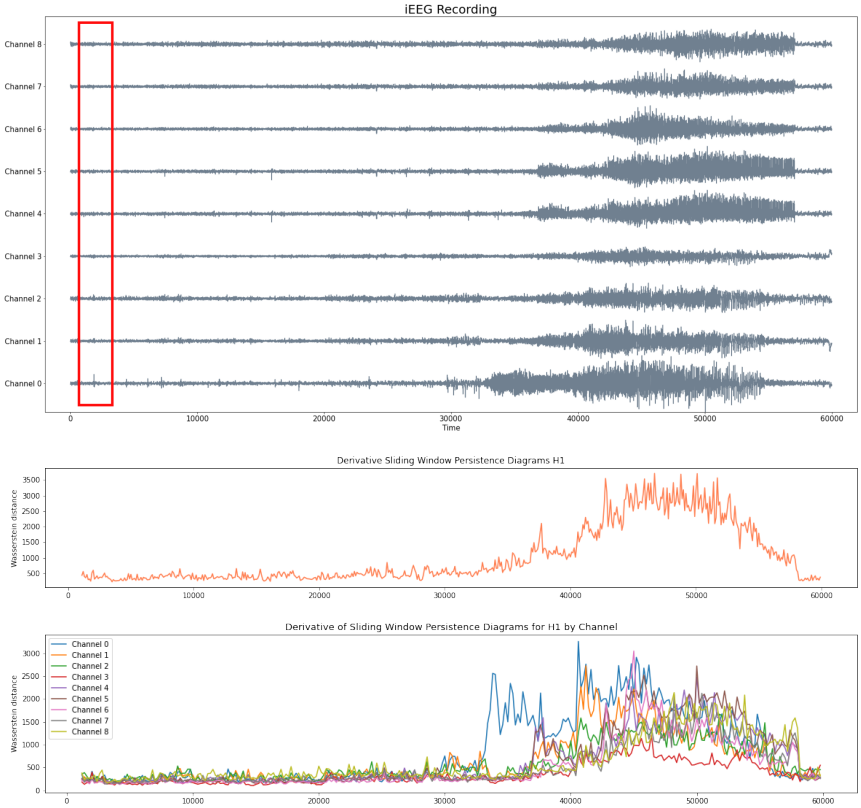

Figure 1: **The topological analysis of a raw iEEG recording of an epileptic seizure. Top.** Recording of neural activity by an iEEG with 9 channels (sampling rate = 200 HZ). The epileptic seizure starts in a few channels and it generalizes by the end of the recording (where the patient losses his consciousness). We compute the sliding window embedding of the signals for a window of size 1000 (in red), for both the joint set of signals and every individual channel. **Bottom.** The approximate of the first derivative of the path of time-evolving persistent diagrams associated to the sliding window embeddings for both the joint set of signals and every individual channel.

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A SEGMENTARY INTERPOLATION NUMERICAL METHOD APPLIED TO A FRACTIONAL ORDER MODEL FOR THE TREATMENT OF HIV INFECTION

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In the work "Estudio comparativo de algunos esquemas numéricos para un modelo de orden fraccionario del tratamiento de la infección por VIH" by Ferrari et al. (2018) [1], four numerical schemes were used to analyze the behavior of the proposed model: Explicit Fractional Euler Method (EFEM), Implicit Fractional Euler Method (IFEM), Fractional Weighted Derivative Method (FWDM) and Adams Method (AM). The convergence of these schemes was studied as well as the sensitivity with respect to the variation of the parameters η (efficiency of the drug) and α (fractional derivative order).

The importance of this fractional order model for the treatment of infection by the human immunodeficiency virus (HIV) [2, 3] is that it evaluates, among other magnitudes, the density of healthy and infected cells of the immune system called CD4+ T cells as well as the viral load. These data are very necessary for the infected subject given the effects of antiretroviral treatment. Furthermore, through the collection of medical records of people living with HIV, the optimal fractional derivative order was determined for the model and compared with the classic one.

In this work a new numerical method [4] is used for this model, which consists in simultaneously considering the approximation by means of a quadratic segmentary interpolation together with an approximation of the Caputo fractional derivative [5]. The objective of the current work is to compare this numerical method with the previously

mentioned numerical methods in order to improve the numerical solution obtained if that is possible.

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CHALLENGES IN COVID-19 DYNAMICS AND DIAGNOSTIC IN THE STATE OF RIO DE JANEIRO

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The State of Rio de Janeiro represents a very heterogeneous set of different socio-

economic, health and mobility contexts between municipalities, within each municipality and with respect Brazilian and foreign tourists. We are also in the presence of different types of tourism. In this talk, using the data bases of e-SUS NOTIFICA and SIVEP-Gripe [5], through the analysis of time series of notifications, onset of symptoms, deaths, corresponding ages and Topological Data Analysis [4], we intend to highlight some aspects of the dynamics of COVID-19 in a part of the state of Rio de Janeiro, in this pandemic times [1].

Furthermore, we investigate the temporal evolution of symptoms of Covid-19 patients registered in the SiVEP-Gripe database of Brazil [6]. Motivated by empirical observation, we analyze the data to spotlight temporal variations in symptoms of hospitalized infected and contextualize them with respect to the emergence of variants. In addition, by modeling using Complex Networks [3], when focusing on the two groups of COVID-19 deceased and recovered individuals we observe a time variation between co-existing symptoms in each group [2].

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MODELING DIFFERENTIATION THERAPY FOR CANCER-STEM-CELL-DRIVEN TUMORS

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Many solid tumors have been found to be driven by chemo- and radiotherapy-resistant cancer stem cells (CSCs). A possible therapeutic avenue in these cases consists of using a differentiating agent (DA) to force the differentiation of the CSCs and then applying conventional therapeutic courses to eliminate the differentiated cancer cells (DCCs). To describe the effects of a DA that reprograms cancer stem cells into DCCs, we adapt a differential equation model developed to investigate tumorspheres, which are assumed to be formed by two jointly evolving cancer cell subpopulations, CSCs and DCCs. We analyze the mathematical properties of the model, finding the equilibria and their stability. We also present numerical solutions and phase diagrams to describe the system evolution and the therapy effects, characterizing the DA strength by a parameter a_{dif}. To obtain realistic predictions, we choose the other model parameters to be those determined previously from fits to various experimental datasets ([1, 2, 3]).

In the case of a tumor that exhibits intraspecific competition and interspecific cooperation between the subpopulations ([1]), low a_{dif} values lead to their stable coexistence, with the final tumor size decreasing with a_{dif}, but there is a critical value above which the end state does not contain any stem cells. For growth on a hard substrate ([2]), the CSCs cooperate, and the tumor grows without limits for small doses of the DA (low a_{dif}). The main effect of the therapy is to delay growth, but if exceeds a critical threshold, the tumor reaches a state formed solely by DCCs. We also studied the consequences of modifying the therapy starting time. In summary, our model shows how the effects of a differentiation therapy depend critically not only on the dosage and timing of the drug application but also on the tumor nature and its environment.

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WELL POSEDNESS AND CONTROL IN BALANCE LAWS MODELS INSPIRED BY BIOLOGY

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We consider a class of hyperbolic balance laws, known as renewal equations [8]. These are differential equations, governing the evolution in time of macroscopic densities, which depend on *space* variables representing some traits of individuals, such as age or size structures. Typically, such equations contain nonlocal terms, used for the description of phenomena like natality and interactions between different individuals. Various realistic applications can be considered. Some examples are: cell growth [8], cancer dynamics [1], biological resources' models [2], epidemic models for the spreading of a disease, like the COVID-19 [6, 5].

These applications require to consider population balance laws both on graphs or on multidimensional spaces. For such models we present some analytic results: existence of a unique solution, continuous dependence with respect to initial conditions, and stability estimates.

Inspired by realistic situations, we propose various control problems, which aim, for example, to minimize the total number of deaths in an epidemic situation [4] or to reduce the mass of cancer cells in a tissue [5]. In specific cases we show the regularity of the input-output map and we prove that optimal controls exist [2, 3].

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OFF-LABEL USE OF EPIDEMIC MODELLING: ASSESSING PEER INFLUENCE ON STUDENT PERFORMANCE AND DROPOUT

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We assess the relevance of social and cognitive factors such as self-efficacy, locus of control and exposure to negative social and peer influence in relation to undergraduate student dropout. To this purpose, upon considering the nature of mechanisms of social influence, we build and analyze a compartmental model of population dynamics involving a system of non-linear ODEs, loosely based upon epidemiological templates and considerations, which describes the academic performance of the student population [1, 2]. We determine and examine threshold values, to be understood as reproduction numbers, that govern the stability of the equilibria and can be viewed as target values to be reached in order to alleviate undergraduate students dropout. A backward bifurcation is observed to occur, analytically and numerically, provided that certain conditions are satisfied.

A sensitivity analysis is then performed to find how the threshold values respond to changes in the parameters, a procedure for estimating these parameters being also proposed. Concrete values are then computed using survey data from a Ghanaian university. The impact of parameter variation upon the dynamics of the system, particularly on certain population sizes and on threshold values, is also numerically illustrated. Our findings are then interpreted from a social cognitive perspective, realistic policy changes being proposed along with appropriate teaching and coaching strategies.

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COUPLING A SIS MODEL WITH OPINION DYNAMICS

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In this work we study an epidemiological model that is influenced by social phenomena. In particular, the coupling of two dynamics is studied, a SIS model for the transmission of a disease, with an opinion model where individuals change their prevention measures as a result of the interaction between them and the evolution of the epidemic, see [1].

We take two approaches. First, an agent-based model, a discrete approach. Each agent has their own opinion that influences their contagion rate. We derive mean-field equations for the proportion of infected and the mean opinion of the population. We solve for the equilibria and we do a stability analysis.

Then we generalize it with a Boltzmann-type kinetic equation. We define two measure functions f^S and f^I that represent the distribution of the opinion of the susceptible and the infected populations. We describe the evolution of both with a system of Boltzmann type equations. We follow the approach made in [2] to prove existence and uniqueness of solutions in a space of positive measures using fixed point theorems, and that the solutions found effectively model the disease dynamic. Then, the passage to the limit is analyzed when the time is rescaled, and a system of Fokker-Planck differential equations is obtained.

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DISENTANGLING HOW MULTIPLE TRAITS DRIVE 2 STRAIN FREQUENCIES IN SIS DYNAMICS WITH COINFECTION

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We study a 2-strain SIS compartmental model with co-infection/co-colonization, incorporating multiple fitness dimensions under the same framework: variation in transmissibility, duration of carriage, pairwise susceptibilities to coinfection, coinfection duration, and transmission priority effects from mixed coinfection. Taking advantage of a singular perturbation approach, under the assumption of strain similarity, we expose how strain dynamics on a slow timescale are explicitly governed by a replicator equation which encapsulates all traits and their interplay. This allows us to predict explicitly not only the final epidemiological outcome of a given 2-player competition, but moreover, their entire frequency dynamics as a direct function of their relative variation and of strain-transcending global parameters. Based on mutual invasion fitnesses, we analyze and report rigorous results on transition phenomena in the 2-strain system, strongly mediated via coinfection prevalence. We show that coinfection is not always a promoter of coexistence; instead, its effect to favour or prevent polymorphism is non-monotonic and depends on the type and level of phenotypic differentiation between strains. This framework offers a deeper analytical understanding of 2-strain competitive games in coinfection, with theoretical and practical applications in epidemiology, ecology and evolution [1].

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A TALE OF TWO VACCINE EFFICACIES: SIMPSON'S PARADOX AND BIFURCATIONS

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Vaccine hesitancy has become of great concern mitigating the spread of COVID-19 in the US. It has led to vast differences in vaccine compliance between different groups [2]. So, it is of great interest to understand dynamics in populations that consist of interacting groups with varying vaccination rates. In this work, we explore a two-group disease spread model. We find Simpson's paradox. While the overall size of the infection decreases, an increased vaccination rate can lead to a relative increase in infections in both the vaccinated and unvaccinated populations. Moreover, similar to the one group model [1], we find an interesting backward bifurcation in the equilibria of infection. In particular, even when the one group model has a transcritical bifurcation, increased heterogeneity in the two group model induces a backward bifurcation. Hence, decreasing the basic reproductive ratio below the threshold $R_0 < 1$ might not be sufficient to converge to a disease-free equilibrium and eradicate the disease.

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INVESTIGATING THE ROLE OF MEMORY IN A POPULATION SYSTEM WITH HOLLING TYPE III FUNCTIONAL RESPONSE

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It may be reasonable to assume that the growth rate of prey may be instantaneously influenced by the predator density in the present time. However, the growth rate of predator depends on prey density in both present and past [1, 3, 5]. Therefore, incorporating a memory term may have an essential role on the dynamics of species in ecology. In this talk, fading memory is considered to understand populations, where prey species are under the influence of Allee effect and predator species are interacted with intraspecific competition within the population. The existence conditions for the steady states is presented and their stability analyses are performed. Single parameter numerical continuation is also performed for some of the key system parameters [4, 2].

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GENERALIZED SEI MODEL WITH NONLINEAR INCIDENCE RATE AND ASYMPTOMATIC INFECTION TRANSMISSION

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A generalized epidemiological SEI model with a general nonlinear incidence rate and death rate functions is proposed. Furthermore, we consider that those who spread the disease are the infected and the exposed. Applying the direct Lyapunov method, we prove that the endemic equilibrium is globally asymptotically stable when the basic reproduction number $\mathcal{R}_0$ is greater than unity and the disease free equilibrium is globally asymptotically stable when $\mathcal{R}_0$ is lower than unity. We conclude that in order to obtain global stability and uniqueness of the equilibrium points, monoticity is necessary but not differentiability in the functions present in the model.

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FLOCK FORMATION CONSTRAINED BY BOUNDARY CONDITIONS OR BY A CONFINING POTENTIAL

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The Vicsek model describes formation of flocks, fish schools or insect swarms in a very basic setting, [2]. Each particle calculates the average direction of particles inside its region of influence and aligns itself with it except for an alignment noise. Position is updated each time assuming a constant particle speed. Confinement of the particles in a region due to periodic boundary conditions is an essential albeit unrealistic feature of the model. In this case, it is possible to derive a discrete-time kinetic equation [1] and analyze the transition between disordered particles and ordered flocks by bifurcation theory. If the periodic boundary conditions are replaced by a more reasonable confinement potential, a variety of spatio-temporal patterns appear including periodic and chaotic dynamics modified by noise. Some relevant features of these patterns agree with experimental observations.

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THE ROLE OF MEDIA ON THE DYNAMICS OF ZIKA OUTBREAK: A MODELING APPROACH

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A non-linear mathematical model has been proposed and analyzed for the role of media on the spread of Zika virus disease during the epidemic. Here, we investigated the epidemiological feasible equilibrium points and computed basic reproduction number (R_0) using the next-generation matrix method. The infected mosquito biting rate and the rate of human to human sexual transmission are the main parameters of the basic reproduction number. The stability of different equilibria of the model is studied and backward bifurcation is discussed, which suggests that merely reducing $R_0 < 1$ is not enough to make disease-free equilibrium globally stable. We present the sensitivity analysis based on the parameters involved in the basic reproduction number and identify some of the key parameters which can be regulated to control the transmission dynamics of the Zika virus. Furthermore, this model is extended to the optimal control model and is analyzed by using the Pontryagin's Maximum Principal and solved numerically. It has been observed that the optimal control model gives better result as compacted to the model without optimal control model as it reduces the number of infectives significantly in a desired interval of time.

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ECKHAUS INSTABILITY OF STATIONARY PATTERNS IN HYPERBOLIC VEGETATION MODELS ON LARGE FINITE DOMAINS

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Vegetation patterns are quite common in many arid and semi-arid areas, nevertheless their physical replication encounters several difficulties due to geographical remoteness and very long timescales. This happens, for instance, in the case of vegetation patterns observed in arid regions, whose occurrence may be interpreted as a warning signal toward desertification. In such contexts, mathematical models become the only tool useful to predict the occurrence of different ecologically-relevant dynamical regimes and to gain some information on pattern stability.

We discuss the phenomenon of Eckhaus instability [4, 6] of stationary patterns arising in the *hyperbolic* extension of the modified Klausmeier model [5, 7, 1] defined on *large* finite domains, in both supercritical and subcritical regime. As known, this conceptual model is used to describe the generation of stationary vegetation stripes over flat arid environments. The hyperbolic generalization here considered accounts for the presence of inertial effects [2] as well as for the occurrence of long transient regimes [3].

Adopting multiple-scale weakly-nonlinear analysis, we deduce the cubic and cubic-quintic real Ginzburg-Landau equations ruling the evolution of pattern amplitude close to criticality. Starting from these envelope equations, we provide the explicit expressions of the most relevant dynamical features characterizing primary and secondary quantized branches of any order: stationary amplitude, existence and stability thresholds and linear growth rate. Particular emphasis is given on the subcritical regime, where cubic and cubic-quintic Ginzburg-Landau equations predict qualitatively different dynamical pictures.

Our analysis also allows to elucidate the role played by inertia during the transient regime, where an unstable patterned state evolves towards a more favorable stable configuration through sequences of phase- slips. In particular, we inspect the functional dependence of time and location at which wavelength adjustment takes place as well as the possibility to control these quantities, independently of each other.

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QUANTIFYING ANOMALOUS DIFFUSION AND NONERGODICITY IN THE UNCLOGGING DYNAMICS FROM EXPERIMENTAL FINDINGS

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Advances in microscopy and image-processing techniques have made it possible to determine the trajectories followed by individual traced particles with high fidelity, allowing biologists to track the movement of fluorescent-labeled single molecules or submicron tracer particles, to mention a concrete example. It has also shown that there is a myriad of systems in which their stochastic dynamics deviate significantly from the classical Brownian diffusion. In such circumstances, their asymptotic behavior at long times is no longer determined by the linear growth of the mean square displacement with time. This loss of universality entails that the diffusive properties depend on the peculiarities of each system. In fact, this may lead to an ergodicity breaking so that quantities measured as time averages of sufficiently long single trajectories mismatch the results obtained if ensemble averages were taken, which may raise misleading interpretations if the results are not properly analyzed [2].

Here we present a physical system in which its diffusive properties can be quantified from experimental measurements: particle trajectories (obtained with image-processing) and some time variables. In particular, we study the vibration-driven unclogging phenomenon using a silo (filled with rigid spheres) with a narrow outlet (enough to be prone to the formation of arches that clog the outlet) [1]. From observables defined in terms of the measured relative positions of the centers of the arch spheres (disregarding any other information from the granular medium), we analyzed the structural changes that the clogging arch undergoes over time. From this experimental-based information, we have characterized in statistical terms the stability of the vibrated arches, described the

unclogging dynamics, determined some diffusive properties, and measured the ergodicity breaking. Finally, the applicability of a Continuous-Time Random Walk-like subdiffusive model to rationalize the phenomenology of the unclogging process is analyzed.

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MATHEMATICAL MODELLING OF COVID-19 TRANSMISSION BETWEEN HUMANS AND MINKS

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COVID-19 virus is primarily transmitted from human to human, but human-to-animal transmission has also been reported. COVID-19 virus has been detected in minks, dogs, domestic cats, big cats, and raccoons that have been in contact with infected humans. However, animal-to-human transmission has only been documented in minks. The aim of the present research is to build a mathematical model to study the COVID-19 outbreak in Denmark and the Netherlands, taking into account the human-to-human, human-to-mink and mink-to-human transmission of COVID-19. In the proposed new model, we divide the human population into two groups based on their contact with minks: humans in direct contact with minks, and humans in indirect contact with minks. The model takes into account measures to control the spread between the minks, i.e. culling or vaccination of the animals. The reproduction number $\mathcal{R}_0$ can be calculated by determining the next generation matrix. At the same time, numerical simulations are used to investigate the impact of different control measures on the number of infected cases in both humans and minks.

Acknowledgements

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THE TIME-VARYING REPRODUCTION NUMBER OF SARS-COV-2 IN SIR AND SEIR-LIKE MODELS: A HAMILTONIAN MONTE CARLO APPROACH

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Compartmental models have had great success applied to epidemiology. In the recent context of the SARS-COV-2 (COVID 19) pandemic, interest in these models has skyrocketed. It is very attractive to be able to model the dynamics of the spread of a disease in a deterministic fashion through a few parameters and a system of Ordinary Differential Equations (ODEs). However, some of the assumptions required in the popular Susceptible-Infected-Removed (SIR) and Susceptible-Exposed-Infectious-Removed (SEIR) models are very restrictive. Additionally, in the spread of a disease there are many random factors to be considered and data tends to be extremely noisy. Hence, in dealing with SIR and SEIR models a probabilistic approach may be advisable.

In this work we consider dynamic models, i.e., SIR, SEIR and extensions with time-dependent transmission rates in a Bayesian setting. Our aim is to estimate the parameters that govern the dynamics of the spread of the disease. Time-dependent transmission rates allow to capture changes in the dynamics of a disease produced by external factors, for example confinement measures. Bayesian techniques allow us to incorporate a priori knowledge which may be available through medical studies and help us account for some of the randomness in the data. Our approach is data driven and is based on a B-spline basis representation of the time dependent transmission rate and on Hamiltonian Monte Carlo. We provide synthetic and real data examples (on SARS-COV-2 daily incidence) and discuss advantages and limitations of our methods.

A HYBRID PDE–ABM MODEL FOR VIRAL DYNAMICS WITH APPLICATION TO SARS–COV–2 AND INFLUENZA

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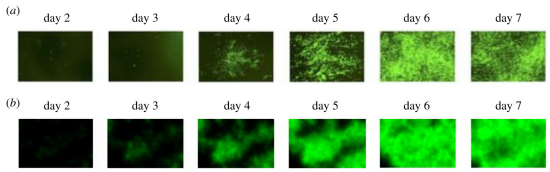
We present a hybrid partial differential equation – agent-based (PDE–ABM) model to describe the spatio-temporal viral dynamics in a cell population [1]. The virus concentration is considered as a continuous variable and virus movement is modelled by diffusion, while changes in the states of cells (i.e. healthy, infected, dead) are represented by a stochastic agent-based model. The two subsystems are intertwined: the probability of an agent getting infected in the ABM depends on the local viral concentration, and the source term of viral production in the PDE is determined by the cells that are infected. By means of a computational tool we developed for this purpose, we study the hybrid system and the generated spatial patterns in detail, moreover, we systematically compare the outputs with a classical ODE system of viral dynamics – according to our results, the ODE model is a good approximation only if the diffusion coefficient is large. Finally, we demonstrate that the model is able to predict SARS–CoV–2 infection dynamics, and replicate the output of in vitro experiments.



(a) Experimental in vitro results of [2] assessing viral spread. (b) Numerical prediction of virus propagation. The picture sequence from our model output shows a striking resemblance to actual experimental results.

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ALLEE EFFECTS DRIVEN BY PREDATION OF AN ECO-EPIDEMIOLOGICAL MODEL

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Allee effect, which corresponds to the positive correlation between population size/density and per-capita growth rate at low population density, drew much attention for research on it. Empirical evidence of Allee effects has been observed in many natural population like birds and mammals, plants, marine invertebrates. Some of the reasons behind this Allee effect attribute to complication in finding mates, reproductive facilitation, predator environment conditioning inbreeding depression, social dysfunction, etc. Allee effect is mainly divided into two broad categories strong Allee effect and weak Allee effect. Strong Allee effect has a threshold limit of population density below which the species becomes extinct. The weak Allee effect, on the other hand takes place when growth rate reduces but remain positive at low population density. It has been observed that disease has been one of the causes of disappearance of the species. The combined impact of Allee effect and disease has substantial biological importance in nature. The major goal of this paper is to investigate the dynamics of Allee effects caused by predation. Kramer in 2010 [2] experimentally demonstrated local extinction of prey due to predator-driven Allee effects in an experimental set up of Daphnia-Chaoborus system. A component Allee effect caused by higher predation rates at low Daphnia density led to positive density dependence in per capita growth rate and accelerated extinction rate at low density. In the present investigation, we have developed a theoretical framework for understanding predator-driven Allee effects as a function of predator functional and aggregative responses using a simple eco-epidemic model. Gascoigne in 2004 [1] showed that predators can create an Allee effect if they have a type I (linear) or type II (saturating) functional response without a type III (sigmoid) aggregative response, or vice versa. In the present work, we consider an eco-epidemic model with disease in prey population, where predators consume both susceptible and infected prey following Holling type II functional response and induce predation driven Allee effect. Understanding the combined impact of Allee effects driven

by predation and disease on population dynamics of predator-prey interactions can help us have a better insights on species abundance as well as the outbreak of disease. That may help to make better policies to regulate the population and disease. We have observed that a very little work has been done on predator driven Allee effects. Thus we propose a general predator-prey model with Allee effects driven by predation and disease in prey to investigate how the interplay of Allee effects and disease in prey affect the population dynamics of both predator and prey.

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OPTIMAL CONTROL OF VACCINATION FOR AGE-STRUCTURED SEIR MODEL OF COVID-19

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In this study, we present an epidemic age structured controlled SEIR model with two compartments: vaccinated and non-vaccinated. The aim of this study is to optimize the vaccination distribution among different age classes under three different vaccination scenarios: 25%, 50% and 75% of the total population. The model is proved to have two disease-free steady states: one that corresponds to the period before the start of the pandemic and one that corresponds to the end of it, achieved by total collective immunity. Also, a thorough study is effected in order to compute the basic reproduction number. As for the optimal control problem, it has been treated numerically to yield the optimal distribution in the three cases. The results of the numerical simulations reveal all three scenarios help in controlling new infections. However, despite their similarities, the 75% strategy exhibits the most rapid results. It is worthy of mentioning that all strategies have no impact on already infected individuals or on the removed compartments' ones, as vaccination is not a treatment and therefore does not interfere with already infected or recovered.

DISCOVERING LAGRANGIAN COHERENT STRUCTURES IN THE HUMAN ABDOMEN

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Laparoscopic surgery involves an operation of the human abdomen, or pelvis where through small incisions in the body, surgical instruments including a camera are passed inside the stomach. The hole thus created, is held open through a tube, i.e., the trocar, and using this the carbon dioxide gas is passed inside the abdomen. In this way, with an inflated abdomen, with more space, the surgeons can perform the operation; however, it can be clearly observed that in this process, a small amount of the gas escapes around the edges of the trocar. This gas can in turn be very dangerous for the surgeons and operating room staff as can contain viral (smoke) particles [2]. In order to address this, first, we need to understand the flow of carbon dioxide as it passes through the human body.

In this talk, we discuss the results from [3] where a computational fluid dynamics (CFD) model is proposed and from the flow dynamics it generates, we describe the movement (transportation, mixing) of smoke particles using techniques based on dynamical systems [1]. The main idea is to work in a Lagrangian framework and describe the transport barriers in the unsteady flow as Lagrangian Coherent Structures. These patterns characterize the major transport pathways for the Lagrangian transport structures as attracting/repelling material lines that are responsible for the accumulation/spread of the material. In our case, using this method, we describe the trajectories that the smoke particles would follow resulting in their accumulation and spread inside the human abdomen.

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MATHEMATICAL MODEL OF ANAEROBIC DIGESTION WITH LEACHATE RECIRCULATION

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Waste management is more relevant than ever, and producing renewable energies while limiting greenhouse gases are part of the environmental challenges of humanity. It is imperative to consider optimized systems for integrated and sustainable waste management, to meet environmental, economic and social needs. This involves, on the one hand, minimizing the landfill rate over the medium and long term, as well as the harmful effects of greenhouse gas emissions, and, on the other hand, optimizing energy yields while respecting the constraints of treatment costs.

In developing countries, household waste is mainly composed of organic matter and its energy recovery is of great interest. Many new technologies have been developed to optimize energy yields, in particular the anaerobic digestion process

Our mathematical model describes the **two-step** anaerobic digestion process (*hydrolysis/acidogenesis and methanogenesis*) with **two types of substrate** and **leachate recirculation** to produce green energy. The dynamic system obtained makes it possible to predict the evolution of the quantities of methane and carbon dioxide over time. It admits an infinity of non-hyperbolic equilibria but presents properties of asymptotic convergence. Thanks to this model, by carrying out simulations, we were able to highlight the influence of the recirculation of leachate and of the initial quantity of organic matter on the production of biogas.

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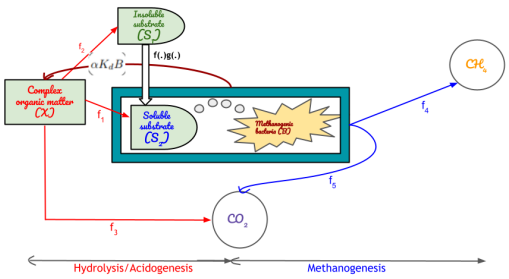

Figure 1: Overall scheme of the anaerobic degradation of organic matter with leachate recirculation.

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MATHEMATICAL MODEL FOR THE BCG-BASED TREATMENT OF TYPE 1 DIABETES

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We introduce a model of immunotherapy treatment, namely the Bacillus Calmette-Guerin (BCG) vaccine, of type 1 diabetes (T1D). The model takes into consideration a clinical interaction network between multiple immune cells and compartments. A set of ordinary differential equations (ODEs) is introduced to capture the connectivity between these variables and clinical presentation of the disease, taking into consideration BCG cells, resting macrophages, activated macrophages, dendritic cells, glucose, autolytic T-cells, and β-cells. Four subsets of the T1D patients and healthy controls that exhibit normal and high-level glucose consumption are evaluated. The results that obtained for mice, suggest that BCG treatment of the T1D patients that follow healthy eating habits normalizes glucose to levels observed in non-diabetic controls. Furthermore, glucose consumption profoundly influences disease progression. The stable equilibrium state with constant glucose levels is not attainable without repeated BCG treatment. This outcome suggests that immunotherapy may modulate molecular and cellular manifestations of the disease but it does not eliminate T1D. Of note, our data indicate that the BCG immunotherapy treatment may benefit healthy controls on a high-glucose diet. One may speculate the preventive BCG treatment to provide long-term health benefits in this specific cohort.

QUASI-NEUTRAL DYNAMICS IN A COINFECTION SYSTEM WITH N STRAINS AND ASYMMETRIES ALONG MULTIPLE TRAITS

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This work includes theoretical and numerical studies of an epidemiological model of multi-strain coinfection. The model is written at the host population level based on a classical susceptible-infected-susceptible system (SIS). The infecting agent is structured into N strains, which differ according to 5 traits: transmissibility, clearance rate of single infections, clearance rate of double infections, probability of transmission of strains, and coinfection rates. The resulting system is a large system ($N^2 + N + 1$ equations) whose complete theoretical study is generally inaccessible. Our work is therefore based on a simplifying assumption of trait similarity - the so-called quasi-neutrality assumption. The system is thus decomposed into two simpler subsystems. The first one is a so-called neutral system - i.e., the value of the traits of all the strains are equal - then the dynamics turn out to be quite simple. Thank the slow-fast dynamics and Tikhonov's theorem, the second one is governed by a "replication equation" system. It describes the frequency dynamics of the strains and contains all the complexity of the interactions between strains induced by the small variations in the trait values. Coefficients of this replicator system, which inherently are fitness numbers of strains, characterize an individual's invasion and resistance to another one.

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BOOLEAN FRAMEWORK FOR STUDYING THE CONTROL OF BREATHING

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Breathing is controlled by a neural network located in the brainstem. This network is essential for supporting a wide range of activities (for example, sleep, exercise and vocalization) as well as heart function. The mechanisms for generating and controlling breathing have been studied for over 30 years but they are still not well understood. Mathematical models that rely on ordinary differential equations have been developed for studying this complex system. These models have many parameters that have been measured directly or estimated from experiments in animals at the neural level. However, experiments at this level are unavailable for humans. This makes scaling of the models from animals to humans unreliable, particularly given the significant difference in respiratory rates across species.

We recently developed a framework for studying neural networks based on Boolean representation [1]. Our framework enabled us to predict the behavior of neural networks based on properties of neurons (e.g. existence of memory, threshold, and self-excitation) without relying on specific parameter values. We used our innovative framework to design a network that mimics many features seen in the respiratory neural network. It provides, for the first time, a good understanding of the way inspiration and expiration times can be controlled selectively at the level of the neural circuitry. Additionally, it provides novel insights and new testable predictions. Importantly, the Boolean neural networks within our framework can be easily scaled to represent breathing rates of different species.

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PERIODIC AND CHAOTIC DYNAMICS IN A MAP-BASED NEURON MODEL

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Map-based neuron models are an important tool in modelling neural dynamics and sometimes can be considered as an alternative to usually computationally costlier models based on continuous or hybrid dynamical systems. However, due to their discrete nature, rigorous mathematical analysis might be challenging. We study a discrete model of neuronal dynamics introduced by Chialvo [Chaos, Solitons & Fractals 5, 1995, 461–479]. In particular, we show that its reduced one-dimensional version can be treated as an independent simple model of neural activity where the input and the fixed value of the recovery variable are parameters. This one-dimensional model still displays very rich and varied dynamics. Using the fact that the map whose iterates define voltage dynamics is S-unimodal, we describe in detail both the periodic behaviour and the occurrence of different notions of chaos, indicating corresponding regions in parameter space. Our study is also complemented by a bifurcation analysis of the mentioned dynamical model.

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QUANTIFICATION OF TYPE I INTERFERON INHIBITION BY VIRAL PROTEINS: EBOLA VIRUS AS A CASE STUDY

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Recent outbreaks, such as the 2014 West Africa Ebola epidemic and the current 2019 SARS-CoV-2 pandemic, have tragically illustrated the need to improve our understanding of the ways viruses subvert immune responses; in particular early innate responses, such as the type I interferon response [1, 6]. The type I interferon family of cytokines is important in the early stages of viral infection and is key to inducing antiviral states within infected cells [4]. There exists ample experimental evidence of different viral proteins

promoting the inhibition of type I interferon secretion in a number of antiviral signalling pathways. We have proposed mathematical models of viral protein antagonism in the type I interferon induction pathway based on current biological evidence [2, 6]. Making use of approximate Bayesian computation, our mathematical models and experimental data sets, we have carried out model selection (to test different molecular hypotheses) and parameter calibration [5]. We provide a case study using experimental data from the EBOV animal model of in vivo infection of rhesus monkeys [3]. With a wish to gain further understanding of early time dynamics of type I interferon production during EBOV infections (or other viral infections, such as SARS-CoV-2), we hope that these models can be extended to other viruses and their mechanisms of innate immune inhibition.

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SETTING A WARNING VACCINATION LEVEL ON A STOCHASTIC MODEL WITH INFECTION REINTRODUCTION WHEN VACCINE IS PARTIALLY EFFECTIVE

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This talk deals with a stochastic Susceptible-Infected-Vaccinated-Susceptible (*SIVS*) model with infection reintroduction.

Health policies depend on vaccine coverage, v_0, that guarantees herd immunity levels in the population. Vaccine failures occur when an organism develops a disease despite of being vaccinated against it. After vaccination, a proportion of healthy individuals unsuccessfully tries to increase antibody levels and, consequently these individuals are not immune to the vaccine preventable disease. When an infectious process is in progress, the initial vaccine coverage drops down and herd immunity will be lost. Our objective is to introduce a threshold for vaccination level and study the so-called *sleeping period*, that is the time until the number of vaccinated descends to this warning vaccination level.

A sensitivity analysis is performed to asses the influence of the model parameters on the variation and robustness of the sleeping period.

A NOVEL MODEL FOR SOIL ORGANIC CARBON CHANGE: SENSITIVITY ANALYSIS OF SOC CHANGE INDEX TO CHANGES OF TEMPERATURE, NET PRIMARY PRODUCTION AND LAND USE

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Soil organic carbon (SOC) is one of the most important indicators of land health. In fact, soil fertility increases with increasing SOC, while reductions in SOC stocks are indicative of soil degradation. Since our main interest is to analyse these variations, we define the *SOC change index* as the difference between the current SOC and the value assumed at an initial reference year, normalized by the initial carbon inputs; then we introduce a novel model for the SOC change index [1], tailored on the RothC carbon model dynamics [2].

In order to evaluate the local response of the model to changes of temperature, Net Primary Production (NPP), and land use soil class (forest, grassland, arable), a *sensitivity analysis*, based on the direct method, is performed. Moreover, we use this novel model to approximate the SOC change index in the Alta Murgia National Park, a protected area in the Italian Apulia region, selected as a test site, and our results indicate positive trends for SOC change in the case of both forest and grassland systems. Whereas, when the arable class is considered without including the input of farm fertilizers, the SOC change index exhibits a negative trend which can be inverted by a suitable organic fertilization program here proposed.

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STOCHASTIC MODELLING OF TCR-EPITOPE RECOGNITION IN THE CONTEXT OF CROSS-REACTIVITY

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The immune system has adapted to fight many different infections, even in the wake of rapidly changing epitope landscapes, such as those seen in influenza infections. Stochastic modelling of TCR-epitope interactions may allow for deeper understanding of the dynamics of the adaptive immune system. To understand these interactions we use TCR-epitope recognition networks, specifically we use bipartite networks since they allow us to naturally separate T cell clonotypes and epitopes, and use the edges to represent the recognition profile of said clonotypes [1]. Using different network construction methods we generate bipartite recognition networks that exhibit different degrees of T cell cross-reactivity. We consider the case where cross-reactivity arises only by chance, and also the case where it is based on the structure of the epitopes being presented. We make use of these networks to simulate the dynamics of an initially naive T cell repertoire when exposed to two different subsequent influenza virus infections.

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QUANTIFICATION OF OPTIMAL RESOURCE ALLOCATION TOWARDS CONTROLLING EPIDEMICS

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Frequent emergence of communicable diseases has been a major concern worldwide. Lack of sufficient resource in order to mitigate the disease-burden makes the situation even more challenging for lower-income countries. Hence, strategy development towards disease eradication and optimal management of the social and economical burden has garnered lot of attention in the recent years. In this context, we investigate what would be the optimal fractions of resources that can be allocated to two major intervention measures, namely, reduction of the intensity of disease transmission and improvement of healthcare infrastructure. Our approach using a simple compartmental disease modelling framework enables study of such trade-off in a resource allocation problem without explicitly considering any economical constraints [1, 2]. Results demonstrate that the effectiveness of each of the intervention strategies have significant impact on the optimal resource and often allocating resources to both the strategies is optimal. We quantify this optimal fraction of resources for both the long-term and outbreak situation that would shrink the disease prevalence and epidemic-peak respectively. We support our numerical simulations with explicit analytical results. Lastly, we also explore the advantages of control-measure implementation at different stages of disease outbreak. Our study provides a fundamental insight into determining the best response strategy in the case of disease outbreak under resource constrained situations.

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REGIME SHIFTS THROUGH SELECTIVE FISHING IN A SEASONALLY FORCED PLANKTON-FISH MODEL

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Aquatic ecosystems exhibit different regimes such as the clear-water and turbid-water regimes [2]. The turbid-water regime is characterized by a yearly increase (bloom) of phytoplankton because of high nutrient loading and represents a paramount environmental problem for lakes worldwide [6]. Reducing the external nutrient loading is the key measure to produce a regime shift from turbid to clear water. However, an improvement in water quality is not always observed. Different methods have been proposed to fight against this resilience [4]. The most frequently used method is the removal of fish or biomanipulation. This strategy is primarily based on the idea of top-down control: the removal of planktivorous fish increases zooplankton grazing rate, thus reducing nuisance phytoplankton [3]. One problem with biomanipulation is its long-term stability, that is, if the effects are only temporary or if they last for a longer period [7]. Another problem is to determine when to fish, how much to fish, and what to fish [5, 8].

In this work, we couple a phytoplankton-zooplankton (PZ) model [9] with an age-structured individual based model of a fish population to evaluate the impact of different fishing strategies. The fish model is described by ordinary differential equations with impulsive effects representing the fish reproduction process [1]. Parameters of a typical freshwater fish are obtained through calibration of long-term weight data of Bleak (*Alburnus Alburnus*) from Rimov reservoir in the Czech Republic. Through bifurcation diagrams, we show the existence of different regime shifts (or attracting sets): one-year cycle with bloom (Figure 1A), one-year cycle without bloom (Figure 1C), two-year cycle with alternating blooms (Figure 1B). Moreover, we show when it is possible to move from one regime to another and how to do it (Figure 1). These results help elucidate the success and long-term stability of biomanipulation.

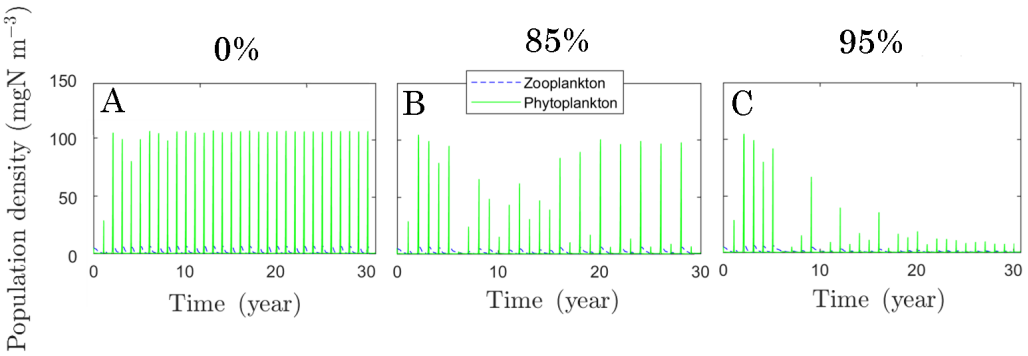

Figure 1: Impact of fish removal on phytoplankton blooms. At the beginning of the fifth year, the following percentage of fish biomass is removed (A) 0%, (B) 85%, and (C) 95%. Each simulation approaches an attracting set.

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DYNAMICAL ANALYSIS OF EARLY AFTERDEPOLARIZATION PATTERNS IN THE 27D CARDIAC SATO MODEL.

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Early Afterdepolarizations (EADs), which are voltage oscillations in cardiac action potential during the repolarization phase, are linked to the appearance of cardiac arrhythmias and other heart conditions. Computational models of cardiac electrical activity have been instrumental in shedding light on various cardiac phenomena, including EADs. Some of these are highly detailed, complex models with tens of state variables and hundreds of parameters, while there are other simpler models with just a few variables and parameters that permit analytical studies, but all of them are fast-slow dynamical systems with multi-timescale phenomena. With the simplest models we can do theoretical studies finding different mechanism as possible explanation for the appearance of EADs ([1] and references therein). If we increase the dimension of the model, we should use other techniques since a theoretical analysis is not possible.

In this talk we show the study of the parametric space of a 27D cardiac cell model proposed by Sato et al. [3]. The mixture of different numerical techniques allows us to study the phase space of one parameter, two parameters and three parameters. In Figure 1 we show one example of a biparametric diagram used in the study. We can observe that the uniparametric evolution varying PCL remains very similar for any fixed value of K_{mNao} lower than $\approx 170\,mM$ (almost double the standard value). Using other bifurcation diagrams and several techniques we have located different phenomena related to the different observed behaviors such as bistability, chaotic attractors and various bifurcations. We will show that in the most relevant parametric region the change, on EADs, is organized by a hysteresis loop that generates two stable orbits, with and without EAD. All these phenomena allow to give a dynamical systems conjecture of a global scheme of

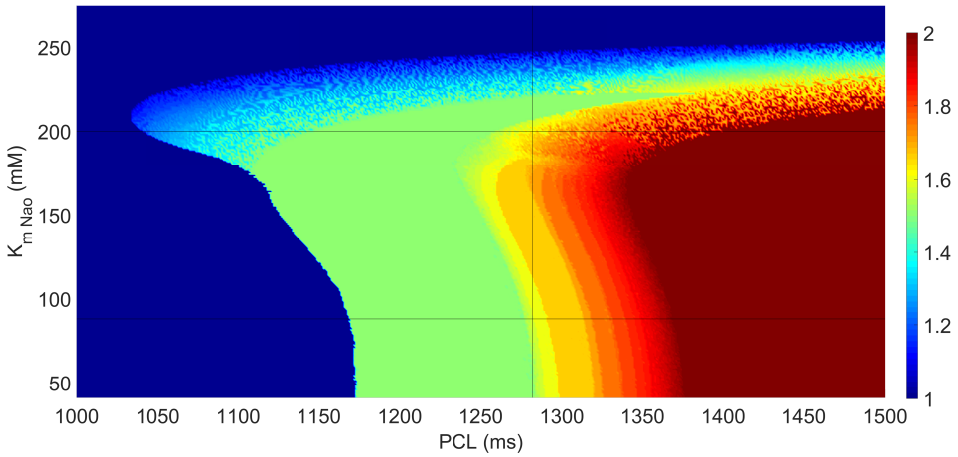

Figure 1: Biparametric bifurcation diagram considering PCL and K_{mNao} as free parameters. Color code corresponds with the ratio between the number of peaks and the number of action potentials.

creation of EADs [2].

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A MATHEMATICAL DESCRIPTION OF BONE MARROW DYNAMICS OF CAR T-CELL THERAPY

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Despite the recent success of CAR-T cell therapy in B-cell malignancies, a fraction of patients still relapse. Relapses are often preceded by recovery of healthy B cells, which suggests loss of the CAR-T cells in bone marrow [2]. This anatomic site, which is where B cells are produced, is harder to access and thus not as monitored as peripheral blood. Understanding the interplay between B cells, leukemic cells and CAR-T in the bone marrow is paramount to ascertain causes for the lack of response. We present a mathematical model describing the interaction between constantly renewing B cells, CAR-T cells and leukemic cells in the bone marrow [1]. We perform numerical simulations to understand the influence of parameters in therapy outcome, and conclude the importance of CAR-T product attributes in determining response.

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POSITIVE AND CONSERVATIVE GECO METHODS FOR CHEMICAL AND EPIDEMIOLOGICAL MODELS

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Many important phenomena in biology, chemistry, epidemiology and ecology are modelled by differential equations with two main qualitative characteristics: positive solutions and conservation of linear invariants, such us the total density. A suitable numerical integrator for this models should therefore be featured by the same two characteristics.

GeCo (Geometric-Conservative) schemes [4] are explicit numerical integrators, up to second order, which preserve both linear invariants and positivity, independently on the choice of the step size. They falls in the general class of nonstandard schemes, where the advancement in time is lead by a non linear function of the temporal step size.

We show the performance of GeCo methods in solving the following models:

- a vibrational kinetics system, where the relaxation of the vibrational levels of diatomic molecules is considered [1]. This problem is an interesting test case from the numerical point of view, because of the large stiffness and the variation of the concentration over many orders of magnitude;
- a SEIR model for Covid-19 pandemic analysed in [3], where the potential of awareness to produce social distancing and self-isolation among susceptibles are exploited, and the Z-control approach is applied to detect what trend must awareness display over time in order to eradicate the disease;
- the SIDARTHE model introduced in [2] for predicting the course of the Covid-19 epidemic in Italy to help the planning of an effective control strategy.

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THE ROLE OF INDIRECT PREDATOR-TAXIS IN AN EXTENDED SCHOENER'S INTRAGUILD PREDATOR-PREY MODEL

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Schoener's model [3] of intraguild-predation counts the competition between the prey and the predator for food resource supplied to a system with a constant rate. Recently researchers [1, 4] claimed that neither self diffusion nor direct taxis can generate patterns in Schoener's predator-prey model. The case of Schoener's model with indirect prey taxis was recently studied in [2] where the formation of patterns in the vicinity of a constant steady state was proved to hold according to Hopf bifurcation mechanism. In this work we extend the model to examine the impact of indirect predator-taxis which demonstrates the prey movement opposite to the gradient of a chemical released by predator. Existence of global-in-time solutions to Schoener's model with indirect-predator taxis is proved for the case of one space dimension. This study reveals that sufficiently large value of taxis sensitivity parameter disturbs the stability of the coexistence steady state giving rise to pattern formation. Numerical simulations illustrate emergence of taxis driven spatio-temporal patterns.

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IMPACTS OF CLIMATE CHANGE IN TEMPERATE AND SUBTROPICAL FISH SPECIES

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Two of the most relevant effects of climate change on marine fish are the effects of ocean acidification and seawater warming. Dynamic Energy Budget (DEB) theory is a universal metabolic theory that allows for the modeling of any species. A mechanistic model based on DEB theory was developed to estimate the effect of seawater warming and ocean acidification on growth and reproduction of three marine fish species: white seabream (*Diplodus sargus*), zebra seabream (*Diplodus cervinus*), and Senegalese sole (*Solea senegalensis*).

Model simulations used a parameter set for each species, estimated by the Add-my-

Pet method using experimental data from two sources and extra data from the literature. An acidification stress factor (s) was added as a modifier of the somatic maintenance costs [$\dot{p}$M] and estimated for each species to quantify the effect of a decrease in pH from 8.0 to 7.4 (white seabream) or 8.0 to 7.7 (zebra seabream and Senegalese sole). The estimation for the acidification stress factor resulted in a change of +36%, -2.%, and +6% on [$\dot{p}$M], for the white seabream, zebra seabream, and Senegalese sole, respectively.

Using the estimated values of the parameters, the model predicts the length and weight of individuals along their usual lifespan and number of eggs produced by a fully developed adult individual within one year, under different climate change scenarios. The acidification effect on white seabream has more influence on size and number of eggs than a temperature change of 3°C, while the acidification effect on zebra seabream and Senegalese sole was much smaller. The number of eggs under the acidification effect decreased 48-53% and 14-34% for the white seabream and Senegalese sole, respectively, and increased 4-5% for the zebra seabream.

EFFECT OF HUNTING COOPERATION ON SPATIO-TEMPORAL PATTERN FORMATION IN PREY-PREDATOR MODELS

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In this talk I will give a detailed idea of a spatio-temporal prey-predator model with hunting cooperation in predator. In available literature, a prey dependent functional response is mostly considered to model the prey-predator interaction. But empirical data shows that functional response can depend on both prey and predator populations. Here we have introduced the cooperative hunting in various functional responses for the predator population and extended the model spatially. Both Turing and non-Turing patterns produced by the diffusion added prey-predator models have been studied in detail. Emphasis is given to the analytical as well as numerical study of the various patterns. The analytical results are verified with extensive numerical simulations.

CAN SELECTION ON HISTIDINE-RICH PROTEIN 2/3 GENE DELETIONS BE DETECTED BY TRACES OF SELECTION?

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One of the cornerstones of controlling P.falciparum malaria is the use of rapid diagnostic tests (RDTs), recommended to confirm infections prior to treat- ment with ACTs as a measure of containment of artemisinin resistance. P. falciparum histidine-rich proteins 2 and 3 (*pfhrp2* and *pfhrp3*) emerged as the most appropriate and popular antigens targeted by RDTs. However, increasing prevalence of parasites with *pfhrp2* and/or *pfhrp3* deletions is severely challenging proper RDT-based diagnostics by yielding false-negative results, potentially leading to improper treatment and an undetected reservoir for malaria transmission. Understanding the evolutionary process underlying the origin and the spread of HRP gene deletions is urgently needed to sustain reliable and cost-efficient diagnostics. The impact of the potential evolutionary mechanisms can be studied by mathematical models tailored to the specifics of malaria transmission, characterised by the presence of genetically distinct parasites haplotypes within infections due to multiple infective contacts (multiplicity of infection, MOI). We introduce a deterministic population-genetic model to study the evolutionary dynamics of HRP2 or HRP3 deletions. The model is tailored to the characteristics of the transmission cycle of P. falciparum. Particularly, the interplay between transmission intensity (MOI) and the spread of deletions is explained in detail. Results:The model shows that selection on HRP deletions originating only from delayed treatment due to false-negative RDT results is extremely weak on emerging haplotypes with deletions, particularly in high transmission areas. The mechanism of selection is more effective in areas of low transmission, or if parasites with HRP deletions already reached appreciable frequencies, either by drift or other mechanisms of selection. The model can be readily adapted to interpret empirical patterns of selection on HRP deletions due to delayed treatment of false-negative-tested patients or reconstruct the underlying evolutionary processes by reverse engineering. It is further employable for study design purposes.

EVOLUTION OF B LYMPHOCYTES LEUKEMIA AND ITS TREATMENT

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In this work, we pay special attention to B-Cell Acute Lymphoblastic Leukemia (ALL), which accounts for 25% of all childhood cancers. Survival of these patients has increased by a considerable amount in recent years, however, around 20% of treatments are unsuccessful. It implies a relapse. For this reason, it is definitely required to come up with new strategies to study and select which patients are at higher risk of relapse [3, 2].

We develop a mathematical models which describes the behavior of the disease [4], from the analysis of works about healthy bone marrow [2, 5], along with considering the evolution of a leukemic clone on the basis of real data. A simulation on first-line therapies [1] is included allowing new work field. Methodology employed combines ordinary differential equations, numerical simulations, data analysis techniques along with statistical tools and data processing with Python.

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THE DYNAMICS OF AN SIRWS SYSTEM WITH ASYMMETRIC PARTITION OF IMMUNITY PERIOD

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The Susceptible-Infectious-Recovered-Waned-Susceptible (SIRWS) system studies the transmission dynamics of infectious disease for which immunity is not lifelong. One example of such a communicable diseases is Pertussis. According to the system, the immunity of recovered individuals wanes over time and consequently individuals transition into the W compartment. In W, individuals have varying levels of immunity with the possibility of immune boosting into R upon re-exposure to the disease causing pathogen. Studies on the SIRWS system often assumes a symmetric partitioning of the immunity period, *i.e* the transition times from R to W and from W to S are the same. A special feature in our investigations borders on the introduction of asymmetric partitioning of the immunity period, as opposed to the common assumption of a symmetric partitioning. Our aim is to study the effects of our proposed new partition of immunity period on the dynamics of the SIRWS system. We obtain analytical results for the disease free and endemic equilibria of the system. We show that a transcritical bifurcation of the forward type occurs for all model parameters when $R_0 = 1$. Using parameters that have been used in literature to model Pertussis , we construct heatmaps to observe regions in the parameter space, where according to the Routh-Hurtwitz stability criterion, the endemic equilibrium is either locally asymptotically stable or unstable. Utilizing the information gained from the heatmaps, we perform numerical bifurcation analysis of equilibria. The system exhibits rich dynamical behavior, one of such is the existence of double endemic bubble. This will have implications on the transmission dynamics of infectious diseases where immunity is temporary.

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MODELING THE ROLE OF CYTOKINES IN THE PATHOGENESIS OF PSORIASIS

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Psoriasis is a common, inflammatory and chronic skin disease whose global prevalence is estimated to be 2-3 % [1]. The most common form of it is chronic plaque psoriasis (Vulgaris) which occurs in 90% of cases. It is clinically characterized by the appearance of well-demarcated inflamed and red coloured plaques covered with silver-white scales and thickened skin. Psoriasis can affect only small isolated areas or the majority of patients' skin. It is believed that the plaques are developed due to the fast proliferation and unusual differentiation of keratinocyte cells that form the outermost layer of the skin. The involvement of immune cells and cytokines secreted from them is well established. Recently, the roles of different immune cells and cytokines along with keratinocytes have been summarized in a form of a complex network [2]. The network depicts indirect cell-cell interactions among cells types assumed via cytokines secreted from them (Figure. 1). However, how the network is involved in the pathogenesis of Psoriasis is still not clear. Our recent findings demonstrate the role of three cytokines: TNFα, IL-17/IL-23 and IL-15 in Psoriasis using a dynamical systems theory approach for the subnetworks of the network mediated by them [3]. These cytokines are chosen based on their known therapeutic potential. Our steady-state analyses for these subnetworks demonstrate that an increase in the levels of TNF$_\alpha$, IL-23/IL-17 or IL-15 could cause Psoriasis. We have observed that the increase in the level of IL-23/IL-17 and IL-15 could lead to psoriasis via a bistable

route, whereas an increase in the level of TNF_{α} would lead to a monotonic and gradual disease progression. In addition, how this insight, bistability, could be applied to develop a novel therapeutic strategy for psoriasis has been shown. The discussed framework could be useful in understanding the pathogenesis of other inflammatory skin diseases.

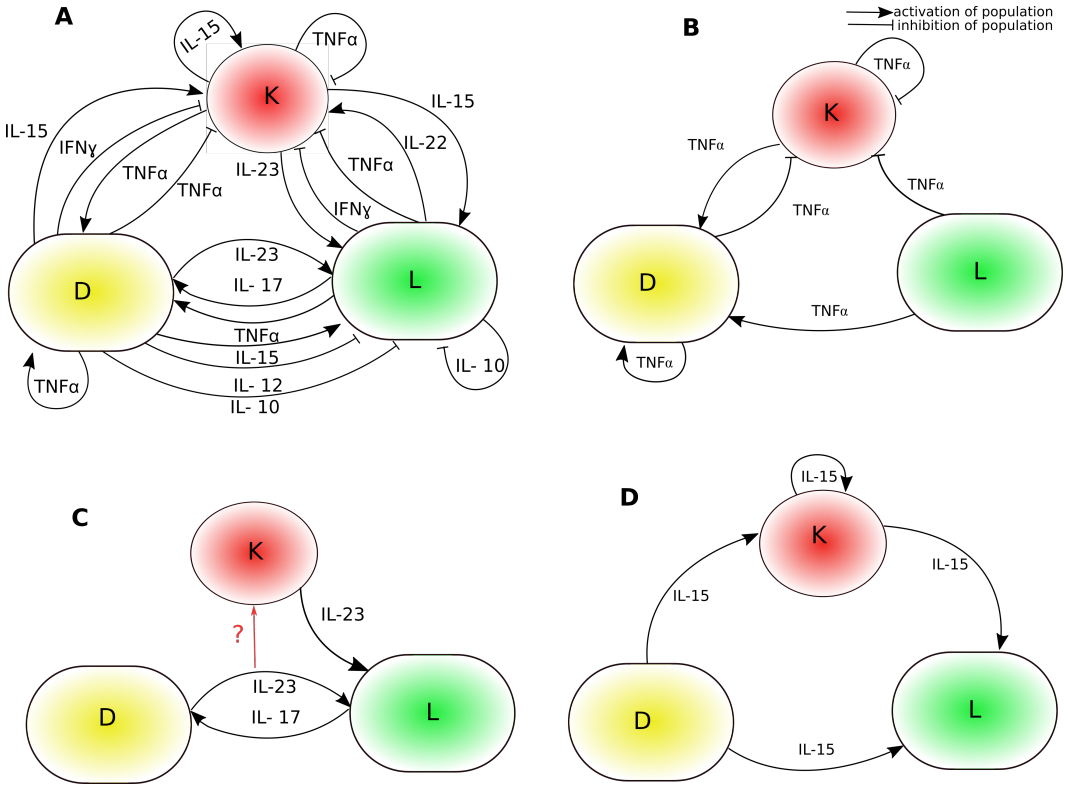

Figure 1: (A) The complex network depicts cytokines mediated interactions among the cell types predominantly implicated in psoriasis. Here, symbols L, D and K denote the population of T-cells, matured dendritic cells and keratinocytes, respectively. An increase in the population is depicted by an arrow and a line with a bar end denote inhibition of that. (B) The subnetwork of the network (panel A) is mediated by only TNF_{α}. (C) Cytokine IL-17 and IL-23 mediated subnetwork of the network. The shown red coloured arrow represents the net effect of the dendritic and T-cell population on keratinocytes. The question mark is to show the lack of clarity about that signal. (D) The subnetwork is mediated by cytokine IL-15 alone.

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ON THE CONTROLLABILITY OF A SYSTEM MODELING CELL DYNAMICS RELATED TO LEUKEMIA

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In this talk, I will present two control problems for a model of cell dynamics related to leukemia. This mathematical model on which the control problems are based, was introduced by Dingli and Michor [1], and analyzed by Parajdi and Precup [4, 3]. The first control problem is in connection with classical chemotherapy, which indicates that the evolution of the disease under treatment should follow a prescribed trajectory assuming that the drug works by increasing the cell death rates of both malignant and normal cells. In the case of the second control problem, as for targeted therapies, the drug is assumed to work by decreasing the multiplication rate of leukemic cells only, and the control objective is that the disease state reaches a desired endpoint. The solvability of the two problems as well as their stability are proved by using a general method of analysis. Some numerical simulations are included to illustrate the theoretical results and prove their applicability. The results can possible be used to design therapeutic scenarios such that an expected

clinical evolution can be achieved. All of this results presented in this talk are original and are contained in the paper of Haplea et al. [2].

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MAKING CROWDSOURCED OBSERVATIONS USABLE FOR WEATHER PREDICTION: BIAS CORRECTION OF CITIZEN WEATHER STATIONS

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The growing network connectivity of high quality environmental sensors widely placed in different locations, from weather stations and auto-mobiles to homes for private use, makes now available meteorological observation streams with measurement rates of the order of minutes. This new unprecedented massive amount of near-surface meteorological measurements from crowd-sourced observation networks offers various new possibilities for improved weather forecast on regional and local scale, as well as for the development of real time weather products. That is why in the German Weather Service (DWD) we study the impact of assimilating observations from the private weather station network NETATMO, within the regional weather model ICON-LAM.

As the crowd-sourced measurements suffer from strong biases and high noise, a bias correction approach is applied, based on the diurnal cycle of temperature and humidity variables. The bias correction scheme takes into account the separate bias calculations of each measuring instrument at each location, the cloud coverage and the model background field.

The preliminary results show that the assimilation of NETATMO observations profits always from the introduced bias correction and it can keep up with the positive effect of assimilating temperature and humidity synoptic observations. The assimilation of the bias corrected crowd-sourced data can boost the dynamical system for the surface forecast, reducing the model's cold bias in the lower atmosphere.

ESTIMATION OF COVID-19 RECOVERY AND DECEASE PERIODS IN CANADA

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We derive a novel model[1] escorted by 830 compartments, based on a set of coupled delay differential equations with extensive delays, in order to estimate the incubation, recovery and decease periods of COVID-19, and more generally any infectious disease. This work is an extension of the earlier approach [2]; the previous model simply allowed for the calculation of the incubation period, while the current model allows for the calculation of all key periods, incubation, recovery and decease. This is possible thanks to some optimization algorithms applied to publicly available database of confirmed corona cases, recovered cases and death toll. In this purpose, we separate i) the total cases into 14 groups corresponding to 14 incubation periods, ii) the recovered cases into 406 groups corresponding to a combination of incubation and recovery periods, and iii) the death toll into 406 groups corresponding to a combination of incubation and decease periods. In this paper, we focus on recovery and decease periods and their correlation with the incubation period. The estimated mean recovery period we obtain is 22.14 days (95% Confidence Interval(CI): 22.00 to 22.27), and the 90th percentile is 28.91 days (95% CI: 28.71 to 29.13), which is in agreement with statistical supported studies. The bimodal gamma distribution reveals that there are two groups of recovered individuals with a short recovery period, mean 21.02 days (95% CI: 20.92 to 21.12), and a long recovery period, mean 38.88 days (95% CI 38.61 to 39.15). Our study shows that the characteristic of the decease period and the recovery period are alike. From the bivariate analysis, we observe a high probability domain for recovered individuals with respect to incubation and recovery periods. A similar domain is obtained for deaths analyzing bivariate distribution of incubation and decease periods.

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MATHEMATICAL MODELLING OF HUMAN FOLLICLE AGING

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Some cases of infertility are associated with short telomeres and low levels of telomerase activity in Granulosa cells (GCs) which are somatic cells, critical for the follicular growth and development. There is a gradual telomere shortening due to the inability of the replication machinery to copy the very ends of chromosomes. However, in addition, other factors as high level of oxidation (free radicals or reactive oxygen species (ROS)), for example due to cumulated stress, inflammation or tobacco smoke, accelerate telomere shortening. In humans, the average telomere length is about 10-15 kb (kilobase) at birth and telomeres shorten at a pace of 70 bp (base pair) per year. However, when cells are exposed to reactive oxygen species, telomere attrition happens at a faster pace, generating a wide variety of telomere size distribution in different length percentiles, which are different to what is expected just by age. In this work, the generational age of a cell is associated with its telomere length (TL), from certain maximum to the minimal TL that allows replication. In order to study the accumulation of aged GCs in human follicles, from preantral to preovulatory size, a mathematical model is proposed, regarding different degrees of accelerated telomere shortening, which reflect the action of reactive oxygen species in addition to the telomere shortening that happens after cell division. Instead of considering that mitosis produces two cells whose generational age increases by one (normal telomere shortening due to replication) as in the previous model [2], we assumed that mitosis produces two cells whose generational age may be any greater than the previous one.

In order to simulate a reduction in telomerase activity induced by high levels of chronic stress, the value of telomerase rate at preantral stage was taken as $r_1 = 0.2$. In Figure 1 was showed the aging rate of preovulatory follicle versus parameter h, the number of times that a cell can be divided before reaching the senescent state, for $r_1 = 0.2$ and several values of the parameters x_i. The figure would show that the higher the oxidation, the higher the aging.

In cases of cells with TL shorter than the mean, with low telomerase activity and accelerated telomere shortening the mathematical model predicts a more aged outcome in

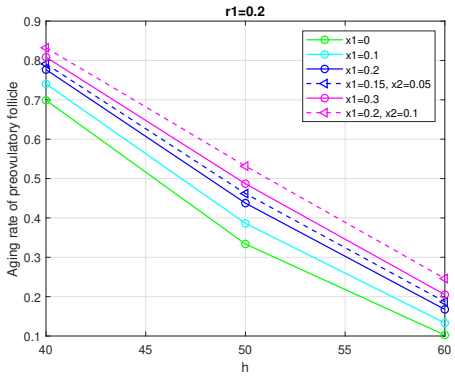

Figure 1: Aging rate of preovulatory follicle (at day 85) versus h for $r_1 = 0.2$ and several values x_i.

preovulatory follicles [1] . In particular, for TL corresponding to the average of women in their forties, the model foretells an high aging rate even with mild oxidation. This may be one of the reasons why fertility declines dramatically in women around 40. The model provides a plausible explanation for what has been observed in oocytes from older women, which have been exposed to ROS for a longer period of time and have bad outcomes after in vitro fertilization.

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EXISTENCE OF HIGHLY PATCHY PHYTOPLANKTON DISTRIBUTIONS REQUIRES MODELING WITH AT LEAST ONE HIGHER LEVEL PREDATORS

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Marine ecosystems are complex and assumed to be multi-trophic networks of biotic and abiotic interactions among physical, biological and chemical components. Yet, nutrient-phytoplankton (NP) interactions are in almost every cases assumed to be representative of higher trophic-level interactions in plankton ecosystems. Here we investigate the degree to which NP interactions capture the overall dynamics of multi-trophic marine ecosystems when accounting for realistic levels of heterogeneity/patchiness. Phytolankton, as the productive base of aquatic food webs account for almost 90% of primary production in marine ecosystems and half of the global primary production [1, 2, 4]. Interactions from the level of individual organisms to marine communities spanning ocean basins to the global scale are mediated by various processes, including predator-prey interactions, nutrient and carbon cycling, biodiversity-productivity relationships within marine ecosystems. These complex planktonic processes remain only partially understood despite extensive efforts by ecologists [4, 3, 5, 6]. Therefore, further theoretical developments are necessary to understand the complex and interesting dynamics of marine ecosystems. Plankton models are typically developed based on the mean-field approach, which considers only first central moments (i.e., spatio-temporal means). Such conventional plankton models may be appropriate for meso- or larger-scales, but inappropriate for the highly intermittent spatial fluctuations of phytoplankton that are ubiquitous at the micro-scale $(< 1m)$. Using Reynold's decomposition, the closure approach accounts for patchiness and temporal fluctuations (higher central moments) of the phytoplankton distributions. We apply closure models of various combinations of Nutrient (N), Phytoplankton (P), and Zooplankton (Z), each with linear, hyperbolic, sigmoidal and quadratic phytoplankton mortality forms to test previously observed hypothesis 'patchiness enhance higher trophic level biomass in plankton models' and the degree of trophic levels interactions as a proxy for higher trophic ecological processes. The inconsistent results from NP

models with different phytoplankton mortality forms reveals that accounting NP interactions alone is insufficient for understanding the impacts of patchiness in particular, and more broadly the general response of plankton ecosystems to climate change. For these purposes, models need to include at minimum one class of predators (Z, herein), as found in previous theoretical and experimental works [2], which did not account for patchiness.

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DYNAMICS OF PLANKTON IN CORAL-REEF ECOSYSTEM IN PRESENCE OF DISEASE

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Coral diseases have been identified as one of the leading causes of collapse of coral reefs worldwide. Coral reefs worldwide are facing severe stress due to the increase in disease outbreaks. Yet the mechanisms of coral disease are poorly understood. To study the role of zooplankton in the transmission of White Band Disease (WBD) in corals, we propose a five-dimensional non-linear (ODE), eco-epidemiological model under the assumption that the transmission of WBD occurs through contagious and non-contagious pathways. The WBD is assumed to be transmitted due to the exposure of corals to zooplankton apart from the direct contact with infected corals and the presence of free-living pathogens in the environment. The WBD is assumed to propagate following nonlinear incidence. It is also assumed that a fraction of the microscopic phytoplankton (zooxanthellae) population has a mutualistic relationship with the corals. We perform equilibrium and stability analysis of the system and found that the system undergoes a Hopf bifurcation when each of the three modes of the WBD transmission rates exceeds some critical threshold and the oscillatory coral population may be driven to extinction when the WBD transmission rates become sufficiently higher. Computer simulations have been carried out to illustrate different analytical results.

OPTIMAL CONTROL APPLIED TO A MODEL FOR VECTOR-BORNE DISEASE

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The effect of introducing mosquito repellents in textiles or household items as a mitigation strategy against a vector-borne disease is studied from the stance of optimal control theory [1]. The basis is a mathematical model described by a controlled nonautonomous system of ordinary differential equations of *Susceptible-Infected-Recovered* type for the human host, and *Susceptible-Infected* for the mosquito vector. The control is constrained by the maximum proportion of the host population employing the repellent-treated products.

Transient dynamic behaviour of controlled trajectories is studied using viability kernels, which represent the largest set of initial states of the dynamical system such that the proportion of infected individuals is sustained below a given ceiling for all future times. Theoretical analysis allows us to distinguish when the Lebesgue measure of the associated viability kernel is positive or null in the phase space of the system. Viability kernels with positive Lebesgue measure are approximated numerically via a variational framework.

Acknowledgements

The inspiration for this work originates within COST Action 16227 *Investigation and mathematical analysis of the effects of avant-garde disease control via mosquito nano-tech-repellents (IMAAC)*. This work is partially supported by the Bulgarian National Science Fund within the National Science Program *Petar Beron i NIE* [contract number KP-06-DB-5].

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GENE-NETWORK ORIENTED DRUG DISCOVERY: AUTOMATED INFERENCE OF BOOLEAN NETWORKS FOR DRUG TARGET PREDICTION

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Gene regulatory networks, and, more specifically, Boolean networks [5, 8], provide a qualitative summary of regulatory interactions at molecular level. Provided the current amount of biological data, being able to integrate all this data to automatize each step of the model inference procedure becomes crucial ; on the one hand, it might help targeting true causal regulatory interactions and replay probable regulatory cascades and mechanisms which had lead to a given observed phenotype. On the other hand, this systematization of the inference allows higher replication, lower time and wet-lab costs.

We propose a fully automated Boolean network inference pipeline. In order to illustrate our method, we will focus on a gene module, named M30, which global gene expression has been shown to be anti-correlated to epileptic phenotypes [3], thus potentially being of interest for therapeutic purposes. First, we describe a fully automated pipeline to identify the network associated with M30, using perturbation experiments of interest from the dataset LINCS L1000 [7], and integrating supplementary biological information to constraint further the selected inference procedure [2, 1]. Then, we use the inferred model, combined with CytoCtrlAnalyzer [9], in order to find master regulator genes ; that is, genes which change in expression affects the whole network. Using this

model, instead of the M30 protein-protein interaction network, helps considerably in restricting the resulting set of genes. This approach allowed the retrieval of SNAP25, a target for antiepileptic drug Brivaracetam [4]; as well as ADAM22 and ADAM23, which are the genetic cause for some types of epilepsies [6], thus emphasizing the relevance of the method.

Our method paves the way for systematic and reproducible gene network-oriented drug target discovery, and can be applied to any disease. This pipeline only requires a set of genes as input, as all data are directly retrieved from public databases.

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ASSIMILATION OF INTELLIGENT CLOUD OBSERVATION

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We present an innovational way of assimilating visible and infrared observations of clouds into the weather forecasting model for regional scale: ICON-D2 (ICOsahedral Nonhydrostatic), which is operated by the German Weather Service (Deutscher Wetterdienst, DWD). For the visible camera photographs, a convolutional neural network is trained to detect clouds in pictures. The result is a greyscale picture, in which each pixel has a value between 0 and 1, describing the probability of the pixel belonging to a cloud. By averaging over a certain section of the picture one gets a value for the cloud cover of that region. To build the forward operator, which maps an ICON model state into the observation space, a three dimensional grid in space from the camera point of view had to be constructed and the ICON model variables were interpolated onto this grid. The pixels of the picture are modelled as rays, originating at the camera location and the maximum interpolated cloud cover (CLC) along each ray is taken as a model equivalent for each pixel. CLC is a diagnostic variable of an ICON model state describing the probability of the cloud coverage within the respective grid box. After superobbing, monitoring experiments have been conducted to compare the observations and model equivalents over time. The results of these experiments look promising with RMSE values below 0.32 and we continued by performing single assimilation steps as well longer experiments.

For assimilating the infrared camera pictures we use a forward operator created by Leonhard Schek at LMU Munich which provides a fast solution for the radiative transfer equations. Monitoring experiments as well as Data Assimilation experiments were conducted and will be presented.

CELL PLATING USING GLASS BEADS: EXPERIMENTAL ANS MODELLING STUDY

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Billiard is a type of mathematical model describing a dynamical system where one or more particles moves in a container and collides with its walls. We devised a model of shifting billiard to explain the observed spatial patterns when cells are spread by shaking of glass beads within a Petri dish [1]. Together with the model for cell plating via the movement of glass beads, we formulated a stochastic spatially-explicit model of cell dispersal in growth media.

Standard suspensions of *Escherichia coli* were spread while varying the number of beads, the shape of movement, and the number of movements to assess their impact on spatial distribution of Colony Forming Units (CFUs). Model parameter inference was performed assuming that the observed number of colonies is Poisson-distributed around simulated number of colonies.

Numerical analysis of the dynamics of this simple yet efficient billiard, indicated a close relationship between plate movement and quality of colony distribution. In conclusion, we recommend using 'L-shape' trajectory with 2-10 beads and 25 loops which should produce good colony separation in terms of the number and spatial spread of colonies.

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MATHEMATICAL MODELING OF IRRIGATION SCHEDULING INFLUENCE IN CROP GROWTH

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Due to the climate crisis derived from global warming, the water resources available for agriculture have been significantly reduced. Mediterranean agricultural systems have been severely affected because of the decrease in rainfall and more frequent and severe droughts. The current and future scenario of water deficit could have a negative effect on the growth and development rates of crops, reflected in the drop in production. For this reason, efficient use of water in agricultural production, particularly in irrigation scheduling can be beneficial in ecological and economic terms. This work focuses on the proposal of a mathematical model based on a system of non-linear differential equations that describes the dynamics between a generic crop and the soil water available for its growth, considering the influence of irrigation. The analysis will focus on the local and global stability of the system without considering irrigation and then the irrigation schedule will be considered as both continuous and impulsive perturbation. This qualitative approach is expected to help us determine some key factors in dynamics and their influence on the growth of the crop. Some simulations to reinforce our analytical findings, as well, the interpretations of these results are shown.

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A GENERAL ODE-BASED MODEL TO DESCRIBE THE PHYSIOLOGICAL AGE STRUCTURE OF ECTOTHERMS: DESCRIPTION AND APPLICATION TO *DROSOPHILA SUZUKII*

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This study introduces a novel general model based on Ordinary Differential Equations (ODEs) which is able to describe the population dynamics of a large class of insect pests. The proposed model is a physiologically-inspired generalization of a number of *ad hoc* models presented through the years in the literature. Its main feature is that it allows the systematic generation of a population model for a species by simply defining its key features, namely the sex ratio and the development, fertility, and mortality rates. The first part of the study provides a detailed description of the model and shows that most ODE-based models existing in literature can be obtained as a special case of the proposed model. The second part of the study shows an application of the model to the spotted wing drosophila *Drosophila suzukii*, which is a highly relevant pest in agriculture. The biological features of this species, i.e., the sex ratio and the various rates, were retrieved from the existing scientific literature. The obtained model was validated using data from a three-year survey conducted in two experimental fields. Results show that the model describes faithfully the experimental populations, although the simulations were performed completely in open-loop and without any adaptation of the parameters extracted from the existing literature.

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DECIPHERING THE ROLE OF ZOOPLANKTON IN SUPPORTING FROG POPULATION

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Chytridiomycosis is the most significant reason for amphibian decline and extinction. It is caused by a fungal pathogen *Batrachochytrium dendrobatidis* (B_d) and affects many species of amphibians. This paper is mainly concerned with designing conservation policies for the frog population (Corroboree, an endangered species) diminishing due to this disease. Our research aims at demonstrating mathematically and supporting the role of zooplankton as a potential biological control for B_d. For this purpose, we designed a stochastic as well as diffusive ecoepidemic model consisting of *Batrachochytrium dendrobatidis*, frog and zooplanktons. We have shown the global existence, non-negativity, and long-term behavior for the designed stochastic model. Existence and stability analysis of the equilibrium points for the corresponding ODE and diffusive model is also done. We have also done bifurcation plotting for the ODE model and found the existence of Hopf-bifurcation. We adopted the Partial Rank Correlation Coefficient (PRCC) to conduct global sensitivity analysis to estimate the most sensitive parameters responsible for disease prevalence and frog mortality. We provided a complete numerical analysis of our deterministic, stochastic, and diffusive models and compared the result. We found that the persistence and extinction of the frog population depend on the environmental stochasticity of zooplanktons. Numerical simulation of corresponding spatially explicit systems brings out complicated spatiotemporal dynamics, typically resulting in the formation of a patchy pattern. It also reveals that B_d tends to decline in the places resided by zooplankton. [1, 2, 3, 4, 5, 6, 7, 8].

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TEMPORAL AND SPATIO-TEMPORAL DYNAMICS IN A SLOW-FAST PREDATOR-PREY SYSTEM

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The ecological interaction among the species of different trophic levels is complex in nature. Over the years, many mathematical models have been studied to replicate the species interaction closely. However, there are still many properties that remain understudied. One of them is the effect of the slow-fast time scale in the spatio-temporal pattern formation by prey-predator models. Incorporating multiple time scales in the mathematical model can give new insight in understanding the population fluctuation in many species [1, 2]. In this talk, I will consider a predator-prey model with slow-fast time scale assuming that the prey population grows much faster than the predator population. Considering that the prey population is affected by weak Allee effect, I will discuss the role of Allee effect and slow-fast time scales in the periodic solutions, namely canard cycle, and relaxation oscillation [3]. In the second part of the talk, I will consider the corresponding slow-fast spatio-temporal model and will discuss the effect of the time scale parameter on the speed of the traveling waves and associated spatio-temporal pattern formation.

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HOW UNEQUAL VACCINE DISTRIBUTION PROMOTES THE EVOLUTION OF VACCINE ESCAPE: INSIGHTS FROM A SIMPLE MODEL

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Since the beginning of 2021, health officials have warned that SARS-CoV-2 vaccines must be uniformly distributed within and among countries if we are to quell the still ongoing COVID-19 pandemic. Yet there has been little critical assessment of the underlying reasons for this warning. Here, we investigate why vaccine equity is necessary using a multi-patch extension of the classical SIR Kermack-McKendrick model. Perhaps counter-intuitively, we find that vaccine escape mutants are less likely to come from highly vaccinated regions where there is strong selection pressure favoring vaccine escape and more likely to come from neighboring unvaccinated regions where there is no selection favoring escape. Unvaccinated geographic regions thus provide evolutionary reservoirs from which new strains can arise and cause new epidemics within neighboring vaccinated regions and beyond [1].

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A 4D-LOCALIZED PARTICLE FILTER METHOD FOR REGIONAL DATA ASSIMILATION AT DWD

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Nonlinear data assimilation methods like particle filters aim to improve the numerical weather prediction in a non-Gaussian setting. The localized adaptive particle filter (LAPF), introduced by R. Potthast, A. Walter and A. Rhodin in [1], overcomes filter collapse in a high-dimensional framework. This particle filter was further developed by Walter et al. in [2] to the local mixture coefficients particle filter (LMCPF) which was tested within the global ICON model. In the LMCPF method the background distribution is approximated by Gaussian mixtures. After a classical resampling step, Bayes' formula is carried out explicitly under the assumption of a Gaussian distributed observation error. Furthermore, the particle uncertainty can be adjusted which affects the strength of the shift of the particles toward the observation. Lastly, Gaussian resampling is employed. All steps are carried out in ensemble space and observation localization is applied in the method.

We explore the potential of the LMCPF in the kilometre-scale ensemble data assimilation (KENDA) system with the limited area mode of the ICON model (ICON-LAM) and compare the particle filter method to the localized ensemble transform Kalman filter (LETKF) which is operationally used at the German Meteorological Service (DWD). Both methods describe four-dimensional data assimilation schemes if the observation operators are applied during the model forward integration at the exact observation times and not only at analysis time. This leads to four-dimensional background error covariance matrices at times and locations of the observations which are employed to derive the analysis ensemble. In addition to a mathematical introduction of the LMCPF method, we present experimental results for the LMCPF in comparison with the LETKF method in KENDA used at DWD for the ICON-LAM model.

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A PHLEBOTOMUS PAPATASI SAND FLY LIFE CYCLE MODEL

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Leishmaniasis is a vector-borne disease caused by protozoa parasite from over 20 Leishmania species. These parasites are transmitted to humans through the bites of infected female phlebotomine sandflies. The present study aimed at estimating the size of phlebotomine sand fly population and to gauge the impact of temperature on the size of this population. A deterministic model describing the dynamics of the phlebotomine sand fly population is presented. The global stability of the sand fly free equilibrium and the endemic equilibrium are proved and the basic offspring number, which represents the capacity of sand fly reproduction, is derived. Sensitivity analyses on basic offspring number and the endemic equilibrium with respect to the model parameters are carried out. The most sensitive parameters are considered, afterwards, temperature-dependent to show the influence of temperature on the population size of phlebotomine sand flies. The point was made that the sand fly population cannot be maintained in an area when the temperature is below 15°C or when it exceeds 32°C, and the optimum temperature for reaching the high *Phlebotomus papatasi* population densities was found to be 28°C. Such information would help policy makers determine the most suitable strategies timely provided temperature estimates are accurate.

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PATTERN FORMATION IN A COMMUNITY MODEL WITH PREDATION FEAR AND DENSITY DEPENDENT DEATH RATE OF PREDATOR

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In this manuscript, the effects of diffusion on the spatial dynamics of a predator–prey model with predation fear and density dependent death rate of predator population is investigated. The spatial distribution of the species population studied over a specific region with the help of reaction-diffusion equations [1]. Consequently, over the last few years, considerable attention was given to understand the spatial spread of species and thus the pattern formation in much more complex domains. In prey predator system, the impact of predation fear in the population dynamics receive significant attention by the researchers recently [2]. So we extend the work of Gao et. al. [2] by incorporating predation fear. The formation of various Turing patterns such as hexagonal spots and stripe patterns are identified. Based on the linear stability analysis, Turing instability conditions are derived analytically and the region of parameters in which Turing instability occurs are identified. The analytical results are validated numerically. Numerical results are shown the sensitivity of fear effect to enrich the pattern dynamics in predator–prey models and provide a deep insight into the dynamics of predator–prey interactions.

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STUDY OF MATRIX PROJECTIVE SYNCHRONIZATION OF CHAOTIC AND HYPERCHAOTIC SYSTEMS

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In this article, matrix projective synchronization of chaotic and hyperchaotic systems has studied. The uncertainties and external disturbances are also used in systems. The sufficient conditions are derived for achieving matrix projective synchronization in both cases. Numerical simulations are introduced to exhibit the adequacy of matrix projective synchronization.

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MATHEMATICAL MODELLING AND ANALYSIS OF COVID-19 AND DENGUE CO-INFECTION

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The four new variants of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have been reported in different nations around the world. These variants affect more people than the current covid-19 pandemic. The hybrid mathematical models of the new strains of covid-19 and dengue are presented. The idea behind this co-infection modelling is that, as per medical reports, both dengue and covid-19 have similar symptoms at the early stages. Our aim is to evaluate and predict the dynamical transmission of both deadly viruses. The qualitative study through stability analysis is discussed at equilibria and the basic reproduction number $\mathcal{R}_0$ is computed. The numerical analysis is carried out by the Adams-Bashforth-Moulton and Newton methods. Sensitivity analysis to assess the effects of various biological parameters and rates of transmission on the dynamics of both viruses is also discussed. We also compared our results with some reported data against infected, recovered, and death cases.

GLOBAL STABILITY OF SAIRS EPIDEMIC MODELS

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We study an SAIRS (Susceptible-Asymptomatic infected-symptomatic Infected-Recovered-Susceptible) epidemic model with vaccination, where the role of asymptomatic and symptomatic infectious individuals is explicitly considered in the transmission patterns of the disease. A peculiar feature of the recent Covid-19 pandemic is that asymptomatic individuals play a significant role in the infection transmission [2, 4, 5]. Indeed, symptomatic cases often remain unidentified and presumably have more contacts than symptomatic cases, allowing the virus to circulate widely in the population. The contribution of the so-called "silent spreaders" is relevant also for other communicable diseases, such as influenza, cholera, and shigella [3, 6, 8, 7]. Although models incorporating asymptomatic individuals already exist in the literature, they have not been analytically investigated as thoroughly as more famous compartmental models. Our main scope is to fill this gap and provide a global stability analysis of the model described by the following system of ODEs

$$\begin{aligned}
\frac{dS(t)}{dt} &= \mu - \Big(\beta_A A(t) + \beta_I I(t)\Big)S(t) - (\mu+\nu)S(t) + \gamma R(t),\\
\frac{dA(t)}{dt} &= \Big(\beta_A A(t) + \beta_I I(t)\Big)S(t) - (\alpha + \delta_A + \mu)A(t),\\
\frac{dI(t)}{dt} &= \alpha A(t) - (\delta_I + \mu)I(t),\\
\frac{dR(t)}{dt} &= \delta_A A(t) + \delta_I I(t) + \nu S(t) - (\gamma+\mu)R(t),
\end{aligned}$$

with initial condition $(S(0), A(0), I(0), R(0))$ belonging to

$$\Gamma = \{(S, A, I, R) \in \mathbb{R}^4_+ | S + A + I + R = 1\}.$$

Here, β_A and β_I are disease transmission rates of the asymptomatic and symptomatic individuals, respectively, α the rate to pass from the asymptomatic to the symptomatic class, δ_A and δ_I recovery rates of the asymptomatic and symptomatic individuals, respectively, ν the proportion of vaccinated susceptible, and γ the immunity-loss rate. We assume, for simplicity, that the rate of births and deaths are the same, equal to μ; we do not distinguish between natural deaths and disease-related deaths. We determine the value of the basic reproduction number $\mathcal{R}_0$ and prove that the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$, a condition under which a unique endemic equilibrium exists. We investigate the global stability of the endemic equilibrium for some variations of the original model under study and answer to an open problem proposed in [1]. Succesively, the work has been extended to a multi-group model.

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INITIAL PHASE OF COVID-19 EPIDEMIC IN THE BASQUE COUNTRY: A MODELING STUDY

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Declared a pandemic by the World Health Organization (WHO) in March 2020 [6], COVID-19 has spread rapidly around the globe. With eventually substantial global underestimation, more than 255 million cases were confirmed by the end of November 2021, counting more than 5.1 million deaths [7].

COVID-19 symptoms can range from mild (or no symptoms) to severe illness, with disease severity and death occurring according to a hierarchy of risks, with age and pre-existing health conditions enhancing risks of disease severity [2].

Applied to the first wave of COVID-19 outbreaks in Basque country, Spain, a deterministic and stochastic mathematical models is proposed and analyzed. The model stratifies the studied population into two groups, older and younger, with different risks for severe disease manifestation [5]. We first analyze the deterministic model. The disease-free equilibrium and the basic reproduction number (R_0) are calculated. We proof that disease-free equilibrium is global asymptotically stable. A sensitivity analysis is performed to identify the key parameters influencing the basic reproduction number, and hence, regulate the transmission dynamics of COVID-19. Epidemiological data for the Basque country population referring to confirmed cases, hospitalization and deceased cases, stratified by age groups is used to calibrated the models. The simulations for the deterministic and stochastic differential equation models were obtained using ODE45 and Euler-Muryama scheme respectively [1, 4, 3].

Although the data fitted well with both approaches, the stochastic model shows a better result, with a smaller RMSE (root mean square error) value. Moreover, numerical simulations have demonstrated that ϵ and ϕ are important parameters influencing the disease spreading in the population.

Acknowledgements

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CONTROL OF IL-7 RESPONSIVENESS THROUGH IL-7R SUBUNITS BALANCE IN EFFECTOR T CELLS AND ANALYTIC EXPRESSIONS FOR AMPLITUDE AND EC50

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Effector T cells rely on Interleukin-7 (IL-7) signaling for their survival. The IL-7 receptor (IL-7R), composed of the common gamma chain and the specific alpha chain, is also associated with the kinase JAK3 which triggers its signaling pathway. Recently, study of cell-to-cell variability and flow cytometry yielded a seemingly paradoxical observation: increased availability of gamma chains reduces the IL-7 response. We describe a mathematical model that provides an explanation for this inhibitory activity through the formation of dummy complexes and shows that a balance between the IL-7R subunits is crucial for optimal signaling. Use of the Groebner basis provides analytical expressions for the maximum IL-7 response (or amplitude) and for the half maximal effective concentration (EC50) of our model. It could also account for the amplitude and EC50 of similar protein receptors. While predicted amplitudes agree with experimental data, measurements of EC50 exhibit more complicated behaviour than we have managed to capture with our IL-7R models.

MODELLING SECONDARY INFECTIONS WITH CROSS-PROTECTION AND DISEASE ENHANCEMENT FACTOR

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Dengue fever is a viral mosquito-borne disease, caused by four antigenically related but distinct serotypes (DENV-1 to DENV-4). While a primary natural dengue infection is often asymptomatic or mild, the clinical response on exposure to a second serotype is complex. Infection by one serotype confers life-long immunity to that serotype and a period of temporary cross-immunity (TCI) protection to other serotypes. However, individuals undergoing a secondary dengue infection with an heterologous serotype have a higher risk of developing the severe form of the disease, due to a process described as antibody-dependent enhancement (ADE), where the pre-existing antibodies to previous dengue infection do not neutralize but rather enhance the new infection [1, 2, 3].

Motivated by dengue fever epidemiology, we study a general two infection SIR-type model to describe the spread of an infectious disease with cross-protection (α) and eventually disease enhancement (ϕ) in subsequent infections. In order to investigate the role of the ADE effect on disease modelling, we fixed the cross-immunity period and vary the ADE as bifurcation parameter. Our model shows a rich dynamical behaviour with bifurcations from fixed point to limit cycles, until completely irregular behaviour. A backward bifurcation was also found in a set of parameters regions. Disease persistence is defined by an important threshold, this is, the disease dyes out if $\mathcal{R}_0 < 1$, and disease persist otherwise .

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SEX- SPECIFIC DIFFERENCES IN DIABETIC KIDNEY

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We have developed computational models of kidney physiology, separate for male and female patients with diabetes [2]. The model predicts luminal and cytosolic solute concentrations of 15 typical solutes found in kidneys along with fluid flow rates and hydrostatic pressure. We increased the plasma glucose concentration from 5mM in a non-diabetic case by 72% and 300% in (i) moderately and (ii) severely diabetic cases respectively. We also increased the surface area of the renal tubules to account for diabetic hypertrophy. The goals of this study are to computationally (i) analyse how kidney function is altered in male and female patients with moderate and severe diabetes [1], and (ii) assess the renal effects, in diabetic women and men, of an anti-hyperglycemic therapy that inhibits the sodium-glucose cotransporter 2 (SGLT2) [3].

Kidney function in diabetic patients without SGLT2 inhibition Without drug intervention, glucose excretion was absent in moderately diabetic (MD) patients, similar to non-diabetic patients (ND) but increased to 0.6 mol/day in severely diabetic (SD) patients (1, left column). In MD, hypertrophy compensates for the increased glucose load. Na+ and Cl- excretion was limited in either cases but urine K+ concentration increased in both sexes and both cases. Osmotic diuresis was also observed in both cases.

Kidney function in diabetic patients with SGLT2 inhibition Under SGLT2 inhibition, the glucose and sodium reabsorption reduces by 80%. The glucose excretion increases up to 0.7 mol/day in MD and up to 2 mol/day in SD (Fig. 1, right column). Severe natriuresis is observed in men with sodium excretion increased by 228% but more limited in women, increasing by 68%. The thick ascending limbs in women with higher activity

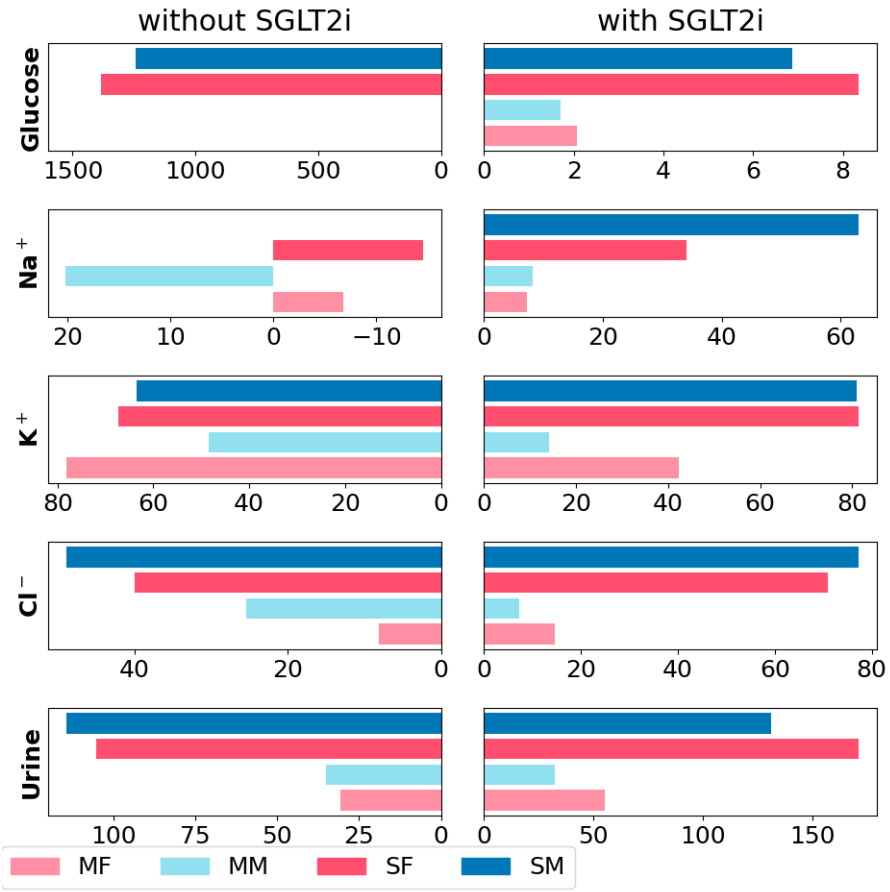

Figure 1: Solute concentrations and urine volume compared to levels in healthy patients. MF/MM: moderately diabetic female/male, SF/SM: severely diabetic, SGLT2i: SGLT2 inhibition.

of NKCC2 transporter is able to compensate for the lower Na+ absorption in the proximal tubules. Similar effects are seen for K+ and Cl- excretion. Extreme diuresis is seen in all cases.

Model simulations suggest that SGLT2 inhibition, which constricts the afferent arteriole to attenuate glomerular hyperfiltration, can limit Na+-glucose transport, consequently raising luminal [Cl-] at the macula densa and finally restore the tubuloglomerular feedback signal. By inducing osmotic diuresis in the proximal tubules, SGLT2 inhibition reduces paracellular transport, eventually leading to diuresis and natriuresis, albeit blunted in women, in part due to their higher distal transport capacity.

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INFERRING THE EFFECT OF INTERVENTIONS ON COVID-19 TRANSMISSION NETWORKS

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Countries around the world implement nonpharmaceutical interventions (NPIs) to mitigate the spread of COVID-19. Design of efficient NPIs requires identification of the structure of the disease transmission network. We here identify the key parameters of the COVID-19 transmission network for time periods before, during, and after the application of strict NPIs for the first wave of COVID-19 infections in Germany combining Bayesian parameter inference [1] with an agent-based epidemiological model [2]. We assume a Watts–Strogatz small-world network [3] which allows to distinguish contacts within clustered cliques and unclustered, random contacts in the population, which have been shown to be crucial in sustaining the epidemic. In contrast to other works, which use coarse-grained network structures from anonymized data, like cell phone data, we consider the contacts of individual agents explicitly. We show that NPIs drastically reduced random contacts in the transmission network, increased network clustering, and resulted in a previously unappreciated transition from an exponential to a constant regime of new cases. In this regime, the disease spreads like a wave with a finite wave speed that depends on the number of contacts in a nonlinear fashion, which we can predict by mean field theory.

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STRUCTURAL IDENTIFIABILITY ANALYSIS FOR SWITCHING SYSTEM STRUCTURES: TOWARDS A TOOLKIT FOR CHANGING TIMES

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A model structure is a collection of related models. A "state-space" structure is often appropriate for modelling biological systems over time. In continuous time, the structure is defined by a differential-algebraic system involving parameter vector $\boldsymbol{\theta}$, relating observables ($\mathbf{y}$) to unobservable "state variables" ($\mathbf{x}$), and possibly inputs ($\mathbf{u}$). Any feasible $\boldsymbol{\theta}$ corresponds to a particular model in the structure.

Given state-space structure S, we may expect obtaining data and estimating parameters to enable estimating $\mathbf{x}$, or predicting $\mathbf{y}$ for novel situations. However, we are not certain to obtain a unique estimate. Further, alternative estimates may lead to quite different predictions. Thus, an inability to distinguish between alternatives makes us unable to confidently make predictions. Fortunately, we may anticipate non-uniqueness of parameter estimates ahead of data collection (and discern a remedy) by first testing S for the property of structural global identifiability (SGI). This yields a lower bound on the number of feasible parameter estimates; we cannot expect fewer when calibrating our model to real (limited, noisy) data.

Typical SGI testing methods are inappropriate for structures describing a system having a sudden change. For example, infectious disease dynamics may change following a coordinated modification to susceptibles' behaviour (e.g., mask-wearing). We can model this situation ([1]) with a structure of uncontrolled linear switching systems (ULSSs),

having the form:

$$\dot{\mathbf{x}}(t,\boldsymbol{\theta}) = \mathbf{A}_{\gamma(t)}(\boldsymbol{\theta}_{\gamma(t)})\mathbf{x}(t;\boldsymbol{\theta})\,, \quad \mathbf{x}(0,\boldsymbol{\theta}) = \mathbf{x_0}(\boldsymbol{\theta}_1)\,,$$
$$\mathbf{y}(t;\boldsymbol{\theta}) = \mathbf{C}_{\gamma(t)}(\boldsymbol{\theta}_{\gamma(t)})\mathbf{x}(t;\boldsymbol{\theta})\,,$$

where deterministic "switching function" $\gamma(\cdot)$ determines which state-space system applies at any time.

Such structures arise in our motivating application: biochemical kinetics studied with a flow-cell optical biosensor experiment of two phases. (Phases are delineated by an abrupt change in experimental conditions, as shown in Figure 1.) We will use an example to demonstrate progress in methods of testing a ULSS structure for SGI.

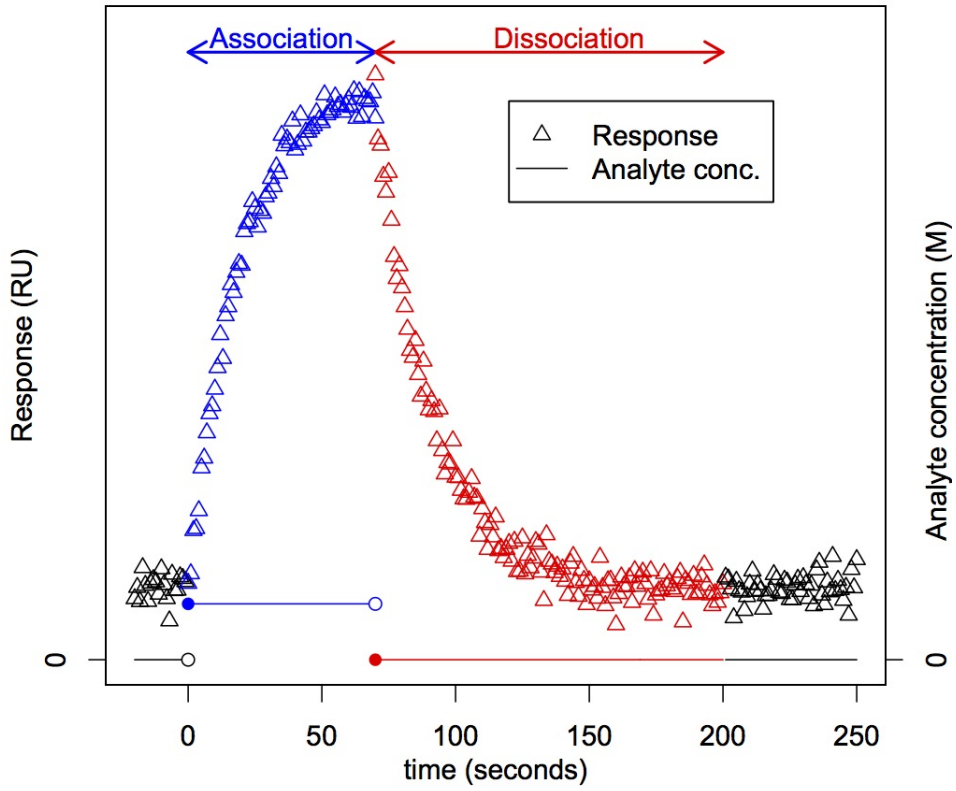

Figure 1: A schematic of Biacore biosensor response from a kinetic experiment showing the association and dissociation phases, and assumed concentration of analyte over time. (From [2], reuse permitted under CC-BY-ND 4.0 International license.)

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A STOCHASTIC INTRACELLULAR MODEL OF ANTHRAX INFECTION WITH SPORE GERMINATION HETEROGENEITY

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During inhalational anthrax infection, *Bacillus anthracis* spores are ingested by phagocytes such as alveolar macrophages and dendritic cells. The spores begin to germinate and then proliferate inside the phagocytes, which may eventually lead to death of the host cell and the release of bacteria into the extracellular environment. Alternatively, some phagocytes may be successful in eliminating the intracellular bacteria and will recover. As a generalisation of modelling work previously developed for *Francisella tularensis* [1], we consider a stochastic, Markov chain model for the intracellular infection dynamics of *B. anthracis* in a single phagocyte, incorporating spore germination and maturation, bacterial proliferation and death, and the possible release of bacteria due to cell rupture [4]. The model accounts for potential heterogeneity in the spore germination rate, with the consideration of two extreme cases for the rate distribution: continuous Gaussian and discrete Bernoulli. Through Bayesian inference, the model is parameterised using *in vitro* measurements of intracellular spore and bacterial counts for the Sterne 34F2 strain of *B.*

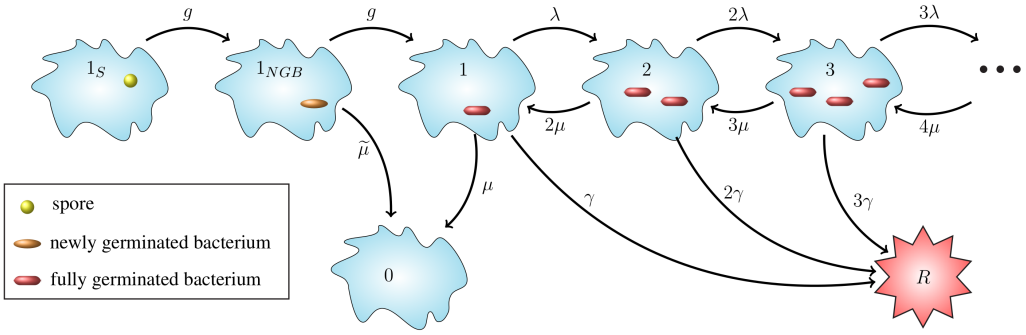

Figure 1: Intracellular infection model. State 1_S represents a phagocytosed spore and state 1_{NGB} a newly germinated bacterium (NGB). The germination rate from spore to NGB, and also the maturation rate from NGB to vegetative bacterium for a given spore is denoted by $g\ hours^{-1}$, which leads to an Erlang(2, g) distribution for the germination-maturation time. The rate g is assumed to vary between spores. The death rate of the newly germinated bacterium is given by $\widetilde{\mu}\ hours^{-1}$. States $i \in \mathbb{N} \cup \{0\}$ represent i intracellular bacteria. State 0 represents recovery and state R the rupture of the cell. 0 and R are absorbing states for the stochastic process. Transitions between states $i \in \mathbb{N}$ represent three types of events: transition to state $i+1$ (division of a bacterium), to state $i-1$ (death of a bacterium), and to state R (rupture of the host cell with release of i bacteria). The per bacterium division, death, and rupture rates are $\lambda > 0$, $\mu > 0$ and $\gamma > 0$, respectively, all with units $(bacteria \cdot hours)^{-1}$, leading to a linear birth-and-death process with catastrophe. The infected cell survives for as long as it does not reach state R.

anthracis [2, 3]. By extending and adapting the methodologies used for *F. tularensis*, we can estimate the rupture size distribution for infected phagocytes, as well as the mean time until phagocyte rupture and bacterial release. Our results support the existence of significant heterogeneity in the germination rate across different spores, with a subset of spores expected to germinate much later than the majority. Furthermore, in agreement with experimental evidence, our results suggest that the majority of spores taken up by macrophages are likely to be eliminated by the host cell, but a few germinated spores may survive phagocytosis and lead to the death of the infected cell. Finally, we discuss how this stochastic modelling approach, together with dose-response data, can allow us to quantify and predict individual infection risk following exposure.

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PARAMETER AND STATE ESTIMATION BASED ON OBSERVER CONSTRUCTION METHOD FOR A CHOLERA MODEL WITH THRESHOLD IMMUNOLOGY

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It is often impossible to measure all states and parameters affecting spread of a disease. In cholera, asymptomatic and cholera pathogen densities are not practically measurable despite playing a big role in its transmission.They are referred to as inaccessible states of the model and can only be manipulated using the measurable states of the given model. Our interest lies in estimating such states and the parameters catalyzing the spread. For this, a mathematical model for cholera dynamics is considered. A method based on observer (from modern control theory) is proposed to estimate the state variables not accessible to measurement and the time dependent parameters from real data. An auxiliary system is used, an observer whose solutions converge exponentially to those of an original system and solely utilizes known inputs and output of the model. The system together with the observer designed is detectable but is not observable. We derive the expressions for time dependent infection rate, induced cholera death rate and symptomatic recovery rate and their estimations done using real data. The observer delivered estimates reflect a close trend already ascertained by other researchers. Numerical simulations are then performed for the validation of estimation results. We have analytically showed and numerically confirmed the exponential convergence to zero of the estimation errors resulting from the observer model hence the high quality of the estimates. Some of the refrences used are include [1, 2, 3, 4, 5, 6, 8]. This presentation is based on our paper [7].

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BESPOKE TURING SYSTEMS

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Reaction-diffusion systems are an intensively studied form of partial differential equation, frequently used to produce spatially heterogeneous patterned states from homogeneous symmetry breaking via the Turing instability. Although there are many prototypical "Turing systems" available, determining their parameters, functional forms, and general appropriateness for a given application is often difficult. Here we consider the reverse problem. Namely, suppose we know the parameter region associated with the reaction kinetics in which patterning is required – we present a constructive framework for identifying systems which will exhibit the Turing instability within this region, while in addition often allowing selection of desired patterning features, such as spots or stripes. In particular, we show how to build a system of two populations governed by polynomial morphogen kinetics such that the: patterning parameter domain (in any spatial dimension), morphogen phases (in any spatial dimension), and even type of resulting pattern (in up to two spatial dimensions) can all be determined. Finally, by employing spatial and temporal heterogeneity, we demonstrate that mixed mode patterns (spots, stripes, and complex prepatterns) are also possible, allowing one to build arbitrarily complicated patterning landscapes. Such a framework can be employed pedagogically, or in a variety of contemporary applications in designing synthetic chemical and biological patterning systems.

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MATHEMATICAL MODEL OF HEARING LOSS CAUSED BY NOISE HAZARD

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Hearing loss can be caused by a variety of factors, including genetics, aging, noise exposure, certain infectious diseases, complications at birth, ear trauma, and some drugs or chemicals. In this study, we propose a new mathematical model of hearing loss caused by exposure to noise using ordinary differential equations. The analysis of the model will be done using mathematical techniques and further examined using numerical simulation to strengthen the validity of the model [1, 2, 3, 4, 5].

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ANALYSIS OF A NONLINEAR PARABOLIC PROBLEM

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We prove existence of global solutions to a nonlinear parabolic system suggested by [1].
The main difficulty is the possibility of blow up for the death term as the activator compex goes to zero.
We write the system in the form of an abstract evolution equation and adapt the ideas of [2] to perform the existence of a global solution in the Sobolev spaces .

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MATHEMATICAL MODEL OF TUMOR GROWTH UNDER THE INFLUENCE OF THE IMMUNE SYSTEM AND ESTROGEN WITH CHEMOTHERAPY TREATMENT

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Breast cancer is the most diagnosed subtype of cancer in the world according to the World Health Organization (WHO) [2]. Based on [1] and [3], we develop a mathematical model for cell competition using normal, cancerous and immune cells and the level of circulating estrogen in the body, in order to assess the impact of the latter on the dynamics of breast cancer under chemotherapy treatment. Firstly, as in [1], we assume an exponential growth of tumor cells and then, we performed a local stability analysis of the proposed model, finding four equilibrium points and establishing conditions for stability to occur. The results of the analysis were compared with those obtained by the study by [1], and it is possible to conclude that the inclusion of chemotherapy treatment prevents estrogen from making the dynamics of the system unstable.

For numerical simulations, based on [2] and [3], we rewrote the model considering an already clinically detectable tumor, with a logistic growth of normal and cancerous cells. Considering a continuous administration of chemotherapy over time, we sought to obtain scenarios similar to those described by the equilibrium points.

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BI-ADDITIVE MODELS AND FLOOD FLOWS

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Bi-additive models given by the sum of a fixed effects term, $X_0\underline{\beta}$, with independent terms $X_i\underline{Z}_i, i = 1, ..., w$. The vectors $\underline{Z}_1, ..., \underline{Z}_w$ will have $c_1, ..., c_w$ independent and identical distributed components, with variances $\sigma_1^2, ..., \sigma_w^2$. Therefore, the covariance matrix of the model will be $\sum_{i=1}^{w} \sigma_i^2 M_i$, with $M_i = X_i X_i^\top$, $i = 1, ..., w$, hence its bi-additive name. The distributions of the components vector $\underline{Z}_1, ..., \underline{Z}_w$ can be of different types making these models more flexible. In this work we will consider that the components follow Gumbel distributions so the models can be used to study flood flows at the confluence of rivers, having then $w = 2$, and two terms, one corresponding to the main river and another to the affluent.

Acknowledgements

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MODELING AND ANALYSIS OF COVID-19 IN INDIA WITH TREATMENT FUNCTION THROUGH DIFFERENT PHASES OF LOCKDOWN AND UNLOCK

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India, unlike several other countries, has witnessed a greater challenge in overcoming the pandemic during the second wave crisis. The strategies on lockdown imposition during the first wave of the pandemic were well implemented due to which the year-long wave did not witness uncontrollable surges in the infections. In this study, we present a detailed work on the disease spread during the first and second wave of COVID-19, by performing numerical simulation in a phase-wise manner as per the lockdown and unlock phases implemented in India. The inclusion of a piecewise treatment function in the framed epidemiological model is a noteworthy aspect of the study since this function takes into consideration the availability of medical equipment, based upon the threshold number of infections. This function framed in this model first grows linearly reaching a peak, then it declines due to shortage of medical resources and finally gets saturated. Analysis on the impact of lockdown is presented for each phase of the pandemic in India. Though the data considered for the study is for a period that marked the beginning of the pandemic, major analysis and predictions are presented based on the second wave data in terms of sensitivity analysis and time series behavior. A comparison of deterministic and stochastic differential equations is presented with simulation results on certain parameter set to examine variation in treatment and recovery. The simulations are performed using MATLAB and R softwares. The work is validated with the real data and model fitting is done applying the Maximum Likelihood method. The study implies that, under accurate lockdown strategies and sufficient medical care, the peak in cases would be attained by 16 May 2021, after which a decline in the cases could be observed.

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COMPARATIVE STUDY OF THE BASIC REPRODUCTIVE NUMBER AND THE TRANSMISSION RATES BETWEEN THE AFRICA COUNTRY FOR THE FIRST THREE WAVES : IMPACT OF THE ECONOMIC, DEMOGRAPHIC AND CLIMATIC FACTORS

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In this work, we use a SIR epidemic model to study and understand the spread of COVID-19 in Africa at the early first three waves. The aim of this study is to make a comparative study on reproduction number R_0 and the contact rate τ between different waves and different African regions. The model showed that, even though the decreases values of some countries, the African continent had a more severe second and second waves of the COVID-19 pandemic than the first. There is no significant regional specificity for the three waves and the values are distributed in a heterogeneous manner even between regions with great geographical proximity. We study the reason for the disparity. We show an insufficient diagnostic capacity linked to the economic factors, a young population reducing the population at risk and a favourable climate (hot and humid) limiting transmission [1, 2, 3].

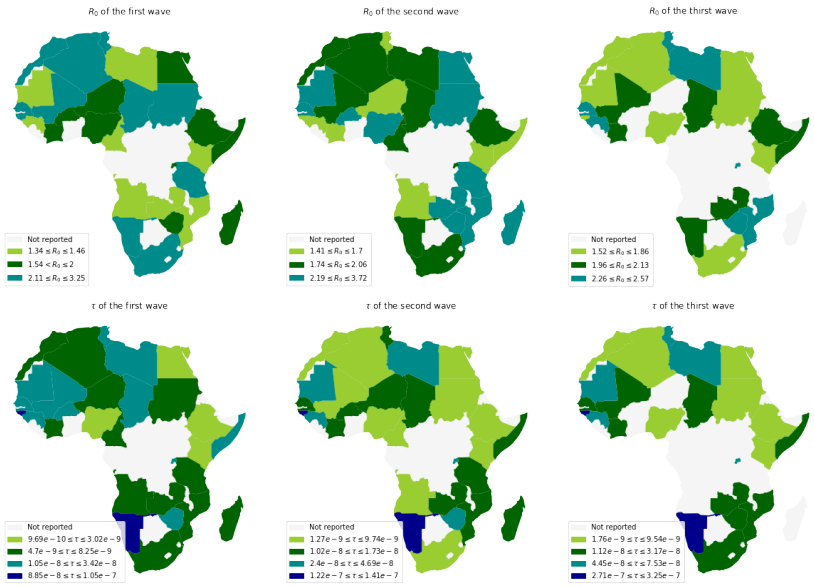

Figure 1: Top: Distribution of the R_0 of 30 country in Africa for the first, second and thirst wave, 6 country from them have only two waves. The high class is represented by the dark cyan color. The second class represent the regions of less dangerous from the first and it is represented by the dark green color. The last one, represented by yellow green color, is the class of the lower values. Bottom: Distribution of the transmission rate τ of 30 country in Africa for the first, second and thirst wave, 6 country from them have only two waves. The high class is represented by the dark blue and the dark cyan colors. The second class represent the regions of less dangerous from the first and it is represented by the dark green color. The last one, represented by yellow green color, is the class of the lower values.

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A DELAYD STOCHASTIC SEIRDS EPIDEMIC MODEL WITH TEMPORARY IMMUNITY

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The main objective of this paper is to study the effects of delay on the dynamics of the Susceptible-Exposed-Infected-Recovered-Death and Susceptible (SEIRDS) model during which we add a stochastic term to handle uncertainty within the estimates of COVID-19 parameters like transmission rate, death rate, and immunity loss rate. On the other hand, we study the effect of loss of immunity on the emergence time of a new wave. Sufficient conditions for extinction are obtained. The results of the numerical simulations reveal that the time between two successive peaks depends mainly on the temporary immunity acquired. Indeed, it'll be very short when the delay of the loss of immunity is lower or adequate to the time of appearance of the primary peak of infection, otherwise, this era becomes very long. Moreover, we show that the simulations of the stochastic model are very consistent with those of the deterministic model when the delay time is large.

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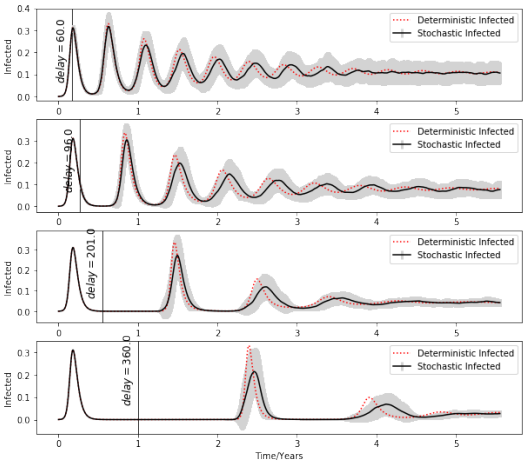

Figure 1: Infected simulation with varies delay, 60 days, 96 days, 201 days, 360 days, presented as the mean of 1000 simulation run, for roughly 6 years. The solution trajectories of the infected show that the disease persists if the delay is extremely short. The light-weight gray color present the error bar of the solution.

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MATHEMATICAL ANALYSIS FOR A TIME-DELAYED ALZHEIMER DISEASE MODEL

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Alzheimer disease (AD) is a neurodegenerative incurable disease of cerebral tissue that causes progressive and irreversible loss of mental functions such as memory. It is the most frequent cause of dementia in humans. AD was first described by german physiologist Alois Alzheimer in 1906.

Although factors that causes AD are still being investigated, recent studies such as [1] and [2] suggest that oligomers which are derived from a protein called (APP) after binding with healthy prions (PrPc) misfold these latter into a pathogenic form (PrPsc) that could be responsible for AD.

In 2014, [3] proposed an in vivo model that takes into account for the first time prions in modeling the evolution of AD. Their model consists of 4 species:

1. $A\beta$ oligomers concentration;
2. Prions PrPc concentration;
3. Concentration of complexes obtained by the binding of an oligomer and a prion;
4. The fourth equation describes the density of the insoluble plaque $A\beta$.

The model that we propose is much simpler and consists of a system of three delayed differential equations. The first equation describes the temporal evolution of concentration of oligomers $A\beta$ denoted by the variable U. In the second equation, concentration of healthy prions is denoted by P. Finally, in the third equation the variable P_c represents the concentration of pathogenic prions. We assume that there are fixed sources of oligomers and healthy prions denoted respectively S_u and S_p. Both oligomers, healthy and pathogenic prions are eliminated by a constant rate called respectively d_u, d_p and d_{pc}. Any oligomer can interact with one or more prions to form a complex with a constant

rate δ_1. Only after a certain time τ, the complex can split into the original oligomer and pathogenic prions with a constant rate δ_2.

Under those assumptions, we obtain the following system:

$$\left\{\begin{array}{lcll} \dot{U}(t) & = & S_u - d_u U(t) - \delta_1 m P(t) U^m(t) + m\delta_2 P(t-\tau) U^m(t-\tau) & \\ \dot{P}(t) & = & S_p - d_p P(t) - \delta_1 P(t) U^m(t) & t \in [\tau, +\infty) \\ \dot{P}_c(t) & = & -d_{pc} P_c(t) + \delta_2 P(t-\tau) U^m(t-\tau) & \end{array}\right. \tag{5}$$

For $t \in [0, \tau)$, the model is described with the same equations without delay, as prions and oligomers are not yet let out from the complexes in the first τ units of time.

Let

$$\left\{\begin{array}{lcl} \delta^* & = & \left(\frac{4S_u}{(m-1)^2 S_p} + 1\right)\delta_1 \\ G(U) & = & \left(\frac{mS_p(\delta_2-\delta_1)}{(d_u U - S_u)} - \delta_1\right) U^m \\ U_1 & = & \frac{S_u}{d_u} + \frac{(m-1)(\delta_2-\delta_1)S_p - \sqrt{\Delta_1}}{2\delta_1 d_u} \text{ with: } \Delta_1 = (\delta_2-\delta_1)S_p\left[(m-1)^2 S_p(\delta_2-\delta_1) - 4S_u\delta_1\right] \\ U_2 & = & \frac{S_u}{d_u} + \frac{(m-1)(\delta_2-\delta_1)S_p + \sqrt{\Delta_1}}{2\delta_1 d_u} \end{array}\right.$$

Suppose $d_p \geq d_u$, results obtained could be summarised in Table 1.

Table 1: Existence and local stability of equilibria depending on the parameter δ_2

δ_2	**number of equilibria**	**local stability** for $\tau = 0$	**local stability** for $\tau > 0$
$0 < \delta_2 \leq \delta_1$	one nontrivial equilibrium E_1	E_1 stable	E_1 stable
$\delta_1 < \delta_2 < \delta^*$	one nontrivial equilibrium E_1	E_1 stable	No results for E_1
$\delta_2 = \delta^*$	one nontrivial equilibrium E_1	E_1 unstable	E_1 unstable
$\delta_2 > \delta^*$	one nontrivial equilibrium E_3 if $d_p < G(U_1)$	E_3 stable	No results for E_3
	two nontrivial equilibria E_1 and E_3 if $d_p = G(U_1)$	E_1 unstable E_3 stable	E_1 unstable No results for E_3
	three nontrivial equilibria E_1 , E_2 and E_3 if $G(U_1) < d_p < G(U_2)$	E_1 stable E_2 unstable E_3 stable	No results for E_1 E_2 unstable No results for E_3
	two nontrivial equilibria E_1 and E_3 if $d_p = G(U_2)$	E_1 stable E_3 unstable	No results for E_1 E_3 unstable
	one nontrivial equilibrium E_1 if $d_p > G(U_2)$	E_1 stable	No results for E_1

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AN EVOLUTIONARY GAME ON COMPLIANT AND NON-COMPLIANT FIRMS IN GROUNDWATER EXPLOITATION

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The paper studies how to counter the illegal exploitation of common groundwater resource in an evolutionary game approach. The access is not free and firms have to pay a royalty depending on the quantity of water pumped. However, some firms could decide to not pay the royalty and face the risk of being sanctioned by the regulator authority. The overall sanction is composed of a fixed amount and of the royalty not payed. From the analysis of the model it emerges that coexistence at the equilibrium between compliant and non-compliant firms is possible and policy instruments are partially able to counter the unauthorized exploitation. In particular, increasing the sanction level reduces the number of non-compliant firms but raises the incidence of illegal pumping. The opposite occurs if the regulatory authority increases the royalty price. To pursue both goals, applying a balance of policies is necessary.

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A FUZZY SYSTEM TO DESCRIBE THE EFFECTS OF VACCINATION ON COVID-19 DYNAMICS IN BRAZIL

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The COVID-19 pandemic has spread widely through the world, since 2019 [3, 2]. Since then, the search for vaccines has become fundamental against the disease and has generated great expectations on population. In Brazil, the first case of COVID-19 was registered in February 2020 and the process of vaccination started in early 2021. In this context, considering the advance of vaccination and the behavior of epidemics, the objective of this work is to present a fuzzy approach [2] to describe the effects of the vaccination process on COVID-19 spread in Brazil. The input variables are the infected population - which is discretized into 6 classes: low, medium-low, medium, medium-high, high and very high, performed using triangular membership functions - and the vaccination rate - which is discretized into 4 classes: no vaccination, low, medium and high vaccination, using trapezoidal membership functions. The output is the level of infection, discretized into 6 classes: low-negative, medium-negative, high-negative, low-positive, medium-positive and high-positive, which is performed using triangular and trapezoidal membership functions. The combination of input classes generates a linguistic rule base composed of 24 rules. Results of the proposed fuzzy system show that the level of infestation tends to decrease as the number of people vaccinated increases, as shown in Figure 1. This fact can be illustrated by the behavior of new daily cases of COVID-19 in Brazil, using numerical data provided by the Ministry of Health [1], from January, 2021 to November, 2021.

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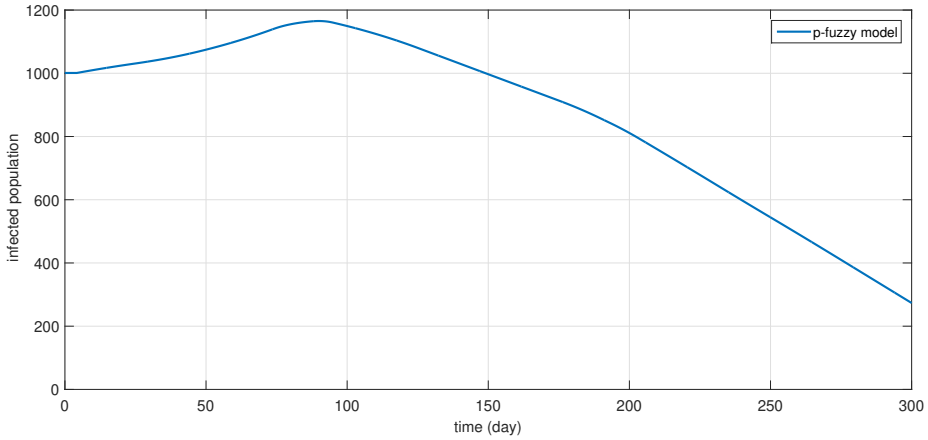

Figure 1: Result of the proposed fuzzy system

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A KINETIC MODEL COUPLING CROWD MOVEMENT WITH INFECTIOUS DISEASE CONTAGION

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We present a multiscale approach for the modeling of disease spreading and crowd movement in bounded environments during a short period of time. The model is based on the kinetic theory of active particles [2] and consider a system of interacting pedestrians, whose microscopic state is identified by their position and velocity direction, as introduced in [1]. The first attempts to couple this model with disease contagion can be found in [3, 4] for the 1D and 2D cases, respectively. In this new setting we add a microscopic variable accounting for the individual state related to an infectious disease: namely, pedestrians can be either susceptible, exposed or infected. We will introduce the mathematical model and show how the transitions modeled at the microscopic level affect the macroscopic dynamics of the system. A qualitative analysis will be performed and some numerical simulations to study the sensitivity to model parameters and to understand how exposure to an infectious agent may promote disease spreading will be discussed.

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AN AGENT-BASED MODEL FOR ASSESSING THE EFFECTS OF CLIMATE VARIATIONS ON TICKS POPULATION DYNAMICS

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Ticks are vectors of multiple and varied pathogens responsible for zoonoses and significant economic losses in livestock. The sheer inevitability of climate change on transmission rates of tick-borne diseases encourages the evaluation of the adaptive capacity of health-threatening vectors. Insights into the interaction between the tick, its hosts, and the environment are required, to better manage vector-borne diseases. Since large-scale empirical studies are impractical, modelling tools may give a mechanism to evaluate vector control strategies and to understand ticks population dynamics. Agent-Based Models are one of the most widely used decision-making models. However, they have been utilized sparingly for tick population modelling (i.e.[1, 3]). Therefore, we developed a spatially implicit agent-based model, to simulate the ecological interaction of the complex "tick-host" and to evaluate the impact of climate change on the dynamics of the vector. The underlying mechanisms of life stages changes of ticks are based on Dynamic Energy Budget (DEB) [4]. By integrating the deterministic elements of DEB theory with the stochasticity of individual-based models (IBM), the coupling of DEB dynamics models

to individual-based models appears to be a promising combination for providing a deeper understanding of the dynamics of vectors. Using the GAMA platform [2], a first simulation figure (1) is built to illustrate the relative importance of the underlying tick population dynamics processes under different climate conditions.

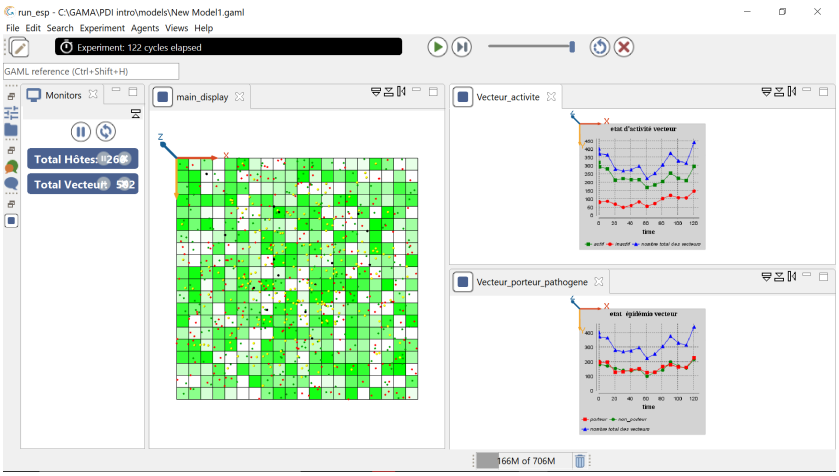

Figure 1: The model simulation

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GLOBAL DYNAMICS OF A COMPARTMENTAL MODEL TO ASSESS THE EFFECT OF TRANSMISSION FROM DECEASED

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During several epidemics, transmission from deceased people significantly contributed to disease spread. Transmission of Ebola during traditional burials was the most well-known example, however, there are several other diseases such as hepatitis, plague or viral haemorrhagic fevers which can potentially be transmitted from disease victims. This is especially true in the case of serious epidemics when health care is overwhelmed and the operative capacity of health sector is diminished, such as it could be seen during the ongoing COVID-19 pandemic.

We present a compartmental model for the spread of a disease with an imperfect vaccine available, also considering transmission from deceased infected. Using the graph theoretical method by Shuai and van den Driessche, we completely describe the global dynamics of the system. We perform numerical simulations to assess the importance of transmission from deceased.

AGENT BASED MODELING FOR WEST NILE VIRUS SPREADING

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The main objective of the current study is to build a conceptual agent-based model following the ODD protocol to track the spread of West Nile virus in birds between regions in Haute-Savoie, France.

A computational Agent Based Model coded on the GAMA platform will follow the developed ODD (Overview, Design concepts, Details) protocol. This study provides new insights into strategies for monitoring and controlling virus spread in the context of climate change.

West Nile Virus (WNV) is a mosquito-borne Flavivirus. It is maintained in the wild through an enzootic cycle involving transmission between birds and mosquitoes (mosquitoes/birds/mosquitoes). Humans and horses are accidental terminal hosts considered a dead end for its replication.

The Agent Based Model being developed is a predictive model that simulates the transmission of WNV by using agents to represent: environment, mosquitoes, avian hosts and humans. The activities and interactions of these various individual agents are simulated within a specified geographic area. A raster map is used to represent the collection of regions agent, which are linked to agent-specific and environmental data, weather conditions, vegetation cover, and other parameters. Habitat values represent the suitability of specific locations (region agents) for foraging, nesting and migration by birds. The simulation also incorporates a data as temperature for region. These meteorological parameters influence habitat quality for the mosquito and bird.

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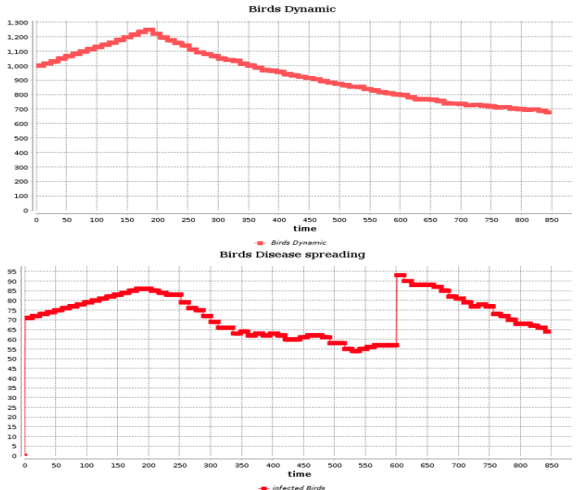

Figure 1: Birds population dynamic

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THREE SPECIES COMPETITION: A STUDY ABOUT THE COMPLEXITY OF DIFFERENTS SOLUTION FROM THE NUMERICAL POINT OF VIEW

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Numerical solutions of differential equation systems are important tools in research on population growth, interactions between species, epidemic propagation, to help in decision-making and control measures. Nonetheless, the choice of the numerical method to be used requires attention, because it can result in spurious solutions that impair the study of real populations. The objective of this work was to investigate the numerical solutions of a prey-predator interaction system between three species, as a function of an α parameter of interaction between two prey and one predator [3]. The complexity of the system alteres as the value of α varies, changing its profile from a stable equilibrium point, going through a limit cycle of different periodicities and reaching a chaotic attractor point, directly impacting the behavior of the numerical solution. In this work, the approximate solutions were obtained via explicit one-step schemes, namely, Euler, Modified Euler, Midpoint and classical Runge-Kutta. Besides, implicit Euler method was also implemented [1, 2]. Multistep schemes were used, namely, Adams-Bashforth method of 2 and 4 points (explicit) and Adams-Moulton method of 2 and 4 points (implicit). In addition, the Runge-Kutta-Felberg 3(4) method of variable step was considered [1, 2]. For all methods we used Octave [2, 4]. Comparisons between the numerical solutions were carried out and we noticed that, as α increased, the numerical solution was altered, transforming the system into a stiff problem. In this situation, an inadequate numerical treatment can lead to solutions that are incompatible with the biological phenomenon. In stiff problem case, the modified Euler, Midpoint and Classic Runge-Kutta methods have not worked well.

We concluded that a higher order method does not always perform better when compared to a lower order method, because the methods de Euler, Adams-Bashforth 2 and Adams-Moulton 2 had better behavior when compared the classic Runge-Kutta method (4th order).

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MODELING HIV/AIDS DYNAMICS

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The acquired immunodeficiency syndrome (AIDS) is a chronic, potentially life-threatening condition caused by the human immunodeficiency virus (HIV). HIV interferes with the body's ability to fight infection and disease. The virus spread primarily by unprotected sex, contaminated blood transfusions, hypodermic needles, and from mother to child during pregnancy, delivery, or breastfeeding.

HIV/AIDS is considered a global pandemic. According to UNAIDS there were approximately 37.7 million people across the globe with HIV in 2020. Thanks to better antiviral treatments, most people with HIV today do not develop AIDS. Untreated, HIV typically turns into AIDS in about 8 to 10 years.

As the COVID-19 pandemic progressed, research on mathematical modeling became imperative and very influential to understand the epidemiological dynamics of disease spreading. With many thousands of mathematical models developed in a record time to assist public health managers during the COVID-19 crises, research on other infectious diseases of public health concern have been postponed.

In this poster we develop a simple SPAD model to describe the dynamics of HIV transmission at population level. The model divides the population into susceptible S, people infected but not ill P, people with AIDS A and deaths D. We present the stationary states and the stability analysis of the system.

Future model refinements include the addition of stochasticity and the available prophylactic measures for disease prevention and control.

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INVESTIGATING THE ROLES OF HELPERS IN COOPERATIVE BREEDING MIGRATORY BIRD POPULATION DYNAMICS

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Cooperative breeding with migratory property is a complex system abundant in many bird species. Young and adult nonbreeding birds help breeders produce more chicks, therefore, potentially altering populations' sustainability. Which of these two helper groups are more influential in determining the population trajectory has long been a research interest of behavioral ecologists. Ghosh [1] is the first to prived a clue to the question through a four-dimensional deterministic model. However, their model assumes a controlled environment and limited resources other than prey. Since many newly developing habitats may experience growing resources and changing environments, the model of [1] is not applicable in all scenarios. This study introduces a new modeling framework to explore the same question under variable environments and growing resources. This new framework is an alternative to the Game theory approach for the cooperative breeding avian system. Like [1], this study also uses Merops philippinus as a testbed species for simulation experiments. The model parameters are obtained by field and literature surveys for simulation experiments. Simulation of the model also helps a behavioral ecologist the

optimum number of helpers and breeders in light of evolution.

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OPTIMIZATION APPLIED TO TRANSMISSION OF HOSPITAL INFECTION

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Hospital infection can be caused by different bacteria, whose spread can occur in several ways, especially in hospital environments. The disease is even more common in Intensive Care Units (ICUs) and a very common species in these scenarios is *Acinetobacter baumannii*, which easily adapts to environmental changes and its capable of developing resistance to several classes of antibiotics [2]. Therefore, due to the limitations of treatment, other control measures must also be used, such as hand hygiene which is a very important measure with high beneficial impact on infection control and prevention. However, on average, 61% of hand hygiene does not follow recommended practice [3, 4]. The mathematical model proposed by [1] is applied to the transmission of this bacterium which causes hospital infection in an ICU setting. The present work proposes an optimization model, based on the mathematical model, to find the minimum measures necessary that minimize the spread of the disease. The optimization model was solved using the metaheuristic Variable Neighborhood Search and the results could be an important tool for the effective control of the disease.

Acknowledgements
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THE ANALYSIS OF A DISEASE-FREE EQUILIBRIUM OF VECTOR-TRANSMITTED AND TRANSFUSION-TRANSMITTED MALARIA DISEASE

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In this paper, we study the transmission dynamics of malaria infection under the administration of treatment, where the disease is transmitted through mosquitoes and transfusion of blood components. The basic reproduction number R_O, is computed using the epidemiological approach. Stability of the disease-free state is investigated. The result shows that the dynamics of the malaria model is determined by the basic reproduction number R_O. Such that, if $R_O < 1$, the disease-free equilibrium is both locally and globally stable and the disease dies out. If $R_O > 1$, the disease-free equilibrium is unstable and the disease persist in the population.

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INTRINSIC NOISE FACILITATES POPULATION SYNCHRONIZATION OF UNCOUPLED CELLULAR OSCILLATORS TO EXTERNAL SIGNALS

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Cellular oscillators, such as NF-κB signaling or the circadian clock, are subject to stochastic fluctuations (noise) due to low molecular concentrations. At the same time, it is necessary for those systems to provide reliable timing for various cellular functions and synchronize to external signals. It has been previously shown that whereas noise distorts the ability of individual cells to synchronize to the external signals, at the population level the synchronization is actually improved by the presence of noise [1]. Here, we investigate, how this effect depends on the noise intensity and the number of cells used to construct the population mean. We have found that increasing noise intensity leads to faster recovery after a jet lag and increases sensitivity to low-amplitude input signals. On the other hand, a sufficient number of cells must be averaged to offset the distorting effects of noise.

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ALPHA MALE EFFECT ON GROUP POPULATION DYNAMICS

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In neotropical primates, dominance hierarchy status [1] represents a dimension of social organization that confers benefits on dominant individuals, such as preferential access to food and mating partners [4, 7], that positively affects the dominant's fitness [6]. The Cebidae family presents a hierarchical multi-male and multi-female affiliative and cooperative organization [2, 3], whose alpha male has priority access to females during the reproductive period [2]. We present here an analysis concerning dominance time and longevity of populations of *Sapajus* sp. (Primates: Cebidae) governed by the reproductive dominance of males. The in silico population analyzes used the modified version [5] of the Penna Model of biological aging [8], where each entity is represented by a double bit string representing its chronological life history (life expectancy) and hereditary. Each position in the string represents an entity's life cycle filled with 0 or 1, where 1 indicates a mutation (M) or inherited disease in its life cycle. The maximum number of previously defined mutations (bits = 1) (T-threshold) that an individual will have throughout his life will determine how many years he will survive. Control of population explosion and stochastic death is represented by the modified Verhuslt logistic equation ($V = 1 - 0.01 * N_t/k$). Ecological and social characteristics of *Sapajus* sp. were added to the algorithm, as well as the status of alpha male with priority access to females for periods of 3, 6, 9, 15 or 25 years. The results indicate that population longevity oscillates around the time of dominance of the alpha in power (Figure 1). The longer reigning time of alpha males

prevents crossing between siblings, keeping descendants with a sequence of strings with a lower number of deleterious mutations, increasing “genetic diversity”, especially for small and isolated populations (K = 40).

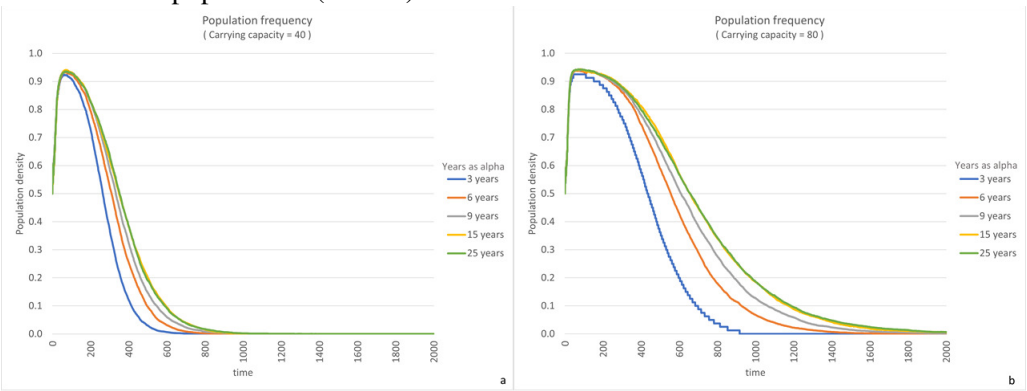

Figure 1: Population behavior of *Sapajus* sp. in different periods of reign. Left one we present the behavior for a small population, with carrying capacity = 40 and N 0 of 50% of K. Right one we present the behavior for a large medium population, with carrying capacity = 80 and N 0 of 50% of K. The lines represent the reign periods of the alpha males.

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MATHEMATICAL MODELLING AND SIMULATIONS OF THE COVID-19 IN BRAZIL

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Since December 2019, all the countries have been facing the devastating pandemic of COVID-19, that has resulted in more than 260 million of cases and more than 5 million of deaths [2]. This disease is caused by a new coronavirus, named SARS-CoV-2, whose symptoms include fever, dry cough and difficulty breathing and the disease has exposed society to very serious problems, such as saturation of health services and the collapse of the funerary system.

Although there are vaccines available against COVID-19, some factors, such as political, economic, cultural issues, among others, have difficult the access of the World population to complete immunisation, especially in the most poor countries, which has made it difficult to control the pandemic. Thus, some countries have faced several waves of cases and this has enabled the emergence of new variants of the virus and, thus, the pandemic, which was initially an outbreak, has already lasted for almost two years and there is no expectation of when it will be possible its termination.

In this work, we develop mathematical models that describe the first and second waves of COVID-19 in Brazil, including the original strain, the two variants with the highest circulation (Gamma and Delta variants) and population vaccination. We started from an initial model and using mathematical modelling techniques we improved it according to the interest problem, whether the new variants or vaccination. For all models, we use as a basis the schemes and parameters proposed by [1] and the data from [2], we performed computer simulations and compared the curve obtained with real data from the active cases, which reinforced the model's efficiency in describing the behaviour of the pandemic and highlighted the importance of population vaccination in controlling and reducing cases.

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MONITORING OF SINGLE-SPECIES POPULATION SYSTEMS

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By a population system we mean a dynamic model describing either a single population where the individuals are classified in some way, or a community of several interacting populations. The monitoring problem, in both cases means that, instead of the population state $x(t)$, we can observe a transform $y(t) = h(x(t))$ of it, and from this observation we want to recover the original state process.

At the beginning of this research line, in [8] the monitoring problem was formalized as an observation problem of systems theory. The system is called observable, if to different state processes $x(t)$ there correspond different observations $y(t)$.

In [8] we considered a diploid population with random mating and n alleles at an autosomal locus, where the genetic state of the population is defined in terms of the allele frequency vector x, and the dynamics is based on Fisher's model of natural selection. It is a real situation when we can observe only the frequency of certain phenotype, and we want to recover the underlying genetic process. We found a sufficient condition for this system to be observable near a polymorphic equilibrium. The mathematical background we developed for this purpose have been also applied in reaction kinetics, see [1]. Later on, these studies were extended to selection-mutation processes in [4]. Furthermore, in [5] applying the observer design methodology of systems theory, we could also numerically estimate the unknown genetic state process.

In addition to the above frequency-dependent models, we also developed the above monitoring approach for some classical density-dependent models, see e.g. [2, 3, 6, 9].

Recently, in submitted paper [7] we also analyzed the monitoring problem for a nonlinear version of the classical Leslie model, observing the densities of certain age classes, we recover the unknown age structure of a population as function of time.

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THE ROLE OF THE INNATE AND ADAPTIVE IMMUNE SYSTEM IN A MATHEMATICAL MODEL FOR NON-SMALL CELL LUNG CANCER

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Lung cancer had the second highest incidence rate and the greatest fatality rate among those who suffered from any type of cancer in 2020, according to data published in [2]. Based on [1] and [3], a proposed ordinary differential equation model was used to observe the temporal evolution of the tumor cells quantities, macrophage cells (of pro and antitumoral phenotypes), and cytotoxic T cells (CD8+) in the tumor microenvironment, in order to mathematically investigate the dynamic of the immune system's innate and adaptive responses to non-small cell lung cancer (NSCLC). The analytical and numerical results show that the apoptotic rate of antitumoral macrophage cells is important for the local stability of the dynamic system derived from the proposed mathematical model, and that cytotoxic T lymphocytes aid in tumor cell reduction.

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APPLICATION OF GENERALIZED LOGARITHN AND EXPONENTIAL FUNCTIONS IN MULTIFRACTAL DETRENDED FLUCTUATION ANALYSIS (MFDFA) OF NONSTATIONARY TIME SERIES FOR MEDICAL SIGNAL ANALYSIS

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Over the last decades, multifractal analysis, via detrended fluctuation analysis (DFA), have been applied extensively in both segmentation and characterization processes, in medical signals. There is a vast range of its applications, which go from electro-cardiogram (ECG), to bone imaging [2]. Regarding ECG signals, the multifractal analysis of ECG data reported are popularly made by using the method of detrended fluctuation analysis, which can quantify the variability in the scalling of the fluctuations in data [5]. The multifractal detrended fluctuation analysis of non-stationary time series (MFDFA) method generalizes DFA method and, by now, is widely used to avoid spurious detection of correlations arising from process trends [6]. It has been already analysed how variations in ECG signals can be detected through multifractal analysis, in which the multifractal analysis of the ECG signals has helped to distinguish healthy and unhealthy cases [1]. Here, our goal is to pursuit more efficient methods to deal with non-stationary time series. Firstly, we rewrite the complicated formulas of the MFDFA algorithms in terms of the Hölder mean. Next, we show that the Hölder mean can be suitably written in terms of the generalized logarithm and exponential functions of the non-extensive statistical mechanics [3, 4]. Thus, we write the MFDFA algorithm in terms of these generalized functions, making it compact and ready for implementation. Finally, we have tested our implementation in ECG signals from the PhysioNet public database to validate the algorithm.

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MODELING COVID-19 DYNAMICS: A COMPARISON BETWEEN TIME-DEPENDENT PARAMETERS

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In the present work, we analyze the COVID-19 pandemic outbreak using an adaptative SIR model approach [3, 4, 5], assuming a homogenous population, to estimate the time-dependent transmission rate and reproduction number of the disease based on official data released from several countries [1, 2] and states from Brazil [7]. The number of infected individuals reported overtime was adjusted according to the cited model for the United States of America, India, and Brazil, presenting less than 10% error. Then, the same treatment was carried out regarding the five southernmost states from Brazil to obtain a regional fit comparing the empirical data and the proposed model. Finally, an analysis was performed through an extension of the adaptative SIR model considering vital dynamics [6] to compare both results concerning the time-dependent parameters, leading to a more accurate description of the reproduction number.

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ANALYTICAL METHOD OF HUMAN SYSTEMIC AND GLOBAL CIRCULATION

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The human circulatory system is one of the admirable rhythms of nature. The heart and the vasculature are constitutive structures. The vasculature consists of arterial and venous appurtenances which are arranged in an idealized network capable of enhancing circulation. The crux of this study is the representation of the cardiovascular system as a network in which electrical constraints apply. As a network, the system is amenable to graph analytic treatment; as edge-nodal parameters ensue, topological constraints apply. In virtue of cardiac auto-rhythmicity, electrical impulses are driven through the vessels to the body cells. As a rule, the vessels must elicit a modicum of resistance. This work weaponized the elements of graph theory and electrical properties of the heart in elucidating the flow mechanism associated with the cardio-vascular system. The voltage drop across the connecting vessels (idealized as wires) was carefully depicted and analyzed by the method of matrices. When the cardiac function is within physiological definition a vascular compartment may be a liability in the event of poor circulation. Therefore the knowledge of vascular resistive capacities, which this work portrayed, is a sine-qua-non to the assessment of flow integrity of the system under consideration. [1, 2, 3, 4, 5].

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TEMPORAL DYNAMICS OF SPIKE TRAINS IN THE RAPHE NUCLEI DURING URETHANE SUB-STATES IN RATS

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Neurons of the dorsal and median raphe nuclei (DRN and MnR, respectively) play a key role in the regulation of the sleep-wake cycle [1]; nevertheless, the dependence of their neuronal dynamic on the state remains uncharacterized. The present study aims to estimate the entropy rate using binary Lempel-Ziv complexity (LZC) [2] for spike trains (LZC ST) and for interspike intervals (LZC ISI bin), as well as to determine the firing rate (FR), coefficient of variation (CV) and Lempel-Ziv complexity for interspike intervals discretized by ordinal patterns (LZC ISI OP), of raphe neurons in two different urethane-induced brain sub-states in rats (n = 64): active or "REM-like" and slow wave or "SWS-like" (SW) [3]. The hypothesis was that the complexity at the neuronal level

would be lower during SW state [4]. For statistical analysis, multilevel linear models were built using rats as a random effect and the state as a fixed effect. The different variables were tested comparing active and SW state groups. As Figure 1 shows, the LZC ST was significantly higher in the active state compared with the SW state ($0.084 \pm 1.2 \times 10^{-3}$ vs $0.080 \pm 1.7 \times 10^{-3}$ mean $\pm$ SD; $n = 91/156$; $p < 0.05$). The FR of the neurons was also significantly higher during the active state (14.30 ± 1.98 vs 9.68 ± 1.98; $p < 0.01$). However, LZC ISI OP, LZC ISI bin and CV, were not significantly different between both groups ($0.69 \pm 3.6 \times 10^{-3}$ vs $0.69 \pm 4.3 \times 10^{-3}$, $p = 0.27$; $1.049 \pm 8.9 \times 10{-}3$ vs $1.041 \pm 11.1 \times 10^{-4}$; $p = 0.48$; 0.53 ± 0.06 vs 0.60 ± 0.05; $p = 0.20$, respectively). We conclude that a lower complexity during SW state is only captured when using LZC ST.

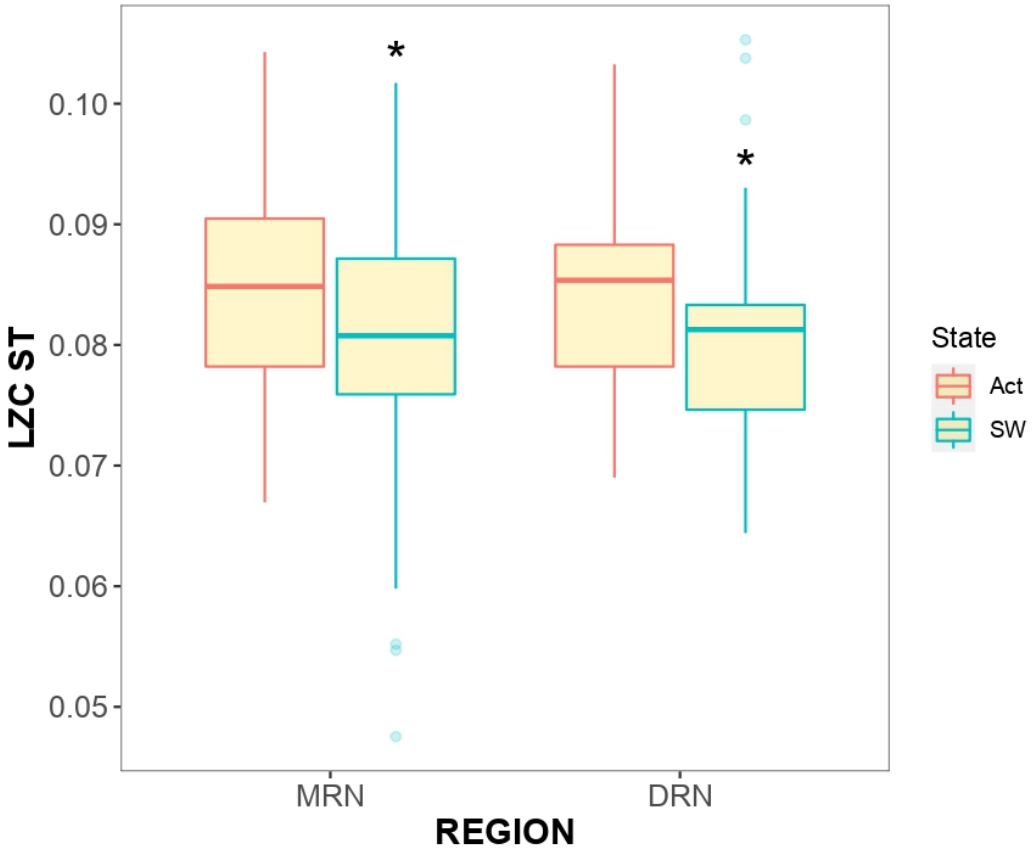

Figure 1: Plot of Lempel-Ziv complexity for spike trains (Dorsal and Median raphe nuclei) by state

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TRIPLET DISTRIBUTION OF COMMON MITOCHONDRIAL AND CHLOROPLAST GENES OF PLANTS

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The interplay of structure of nucleotide sequences, the functions, and taxonomy of their bearers still challenges researchers. We compared common mitochondrial and chloroplast genes of the same species. ATP synthase genes has been studied. In total, 170 (85 mitochondrial and 85 chloroplast) plant genomes were used in this study. Each gene sequence was transformed into triplet frequency dictionary, where the reading frame shift was equal to $t = 1$.

The relation is revealed through the unsupervised clustering via elastic map implementation of the points in 64-dimensional space of the triplet frequencies of the genes. Three types of clustering have been analyzed: for mitochondria genes solely, for chloroplast genes solely, and for the merged set of the genes from the genomes of both organelles. Fig 1 shows the distribution of genes from the genomes of both organelles. Mitochondrial genes are shown with triangle labels: *atp*1 is colored in red, *atp*4 is colored in lime, *atp*6 is colored in blue, *atp*8 is colored in yellow, *atp*9 is colored in dark cyan. Chloroplast genes are shown with circle labels *atp*A is colored in magenta, *atp*B is colored in green, *atp*E is colored in dark orange, *atp*F is colored in dark violet, *atp*H is colored in light pink, and finally, *atp*I is colored in brown.

It was found that the encoded function determines clustering: all the clusters in all

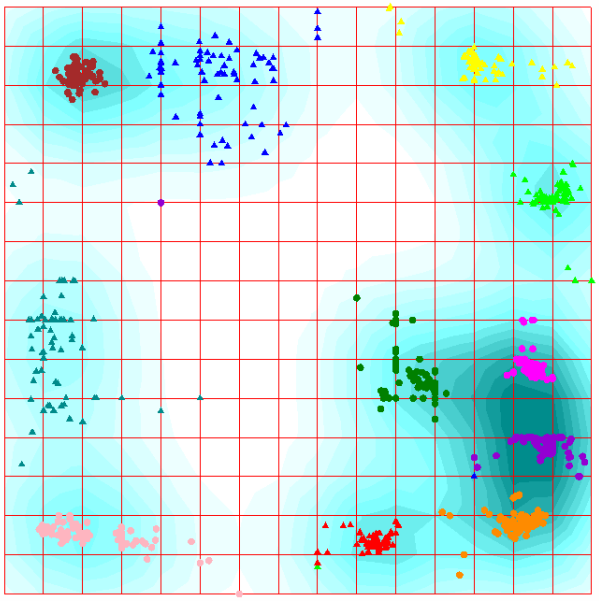

Figure 1: Cluster patterns observed on the set of ATP synthase genes of both organelles.

three versions of clustering patterns clearly exhibit distinct separation of the genes encoding the same subunit into a separate cluster. This behaviour was found for all three types of cluster patterns. Thus, an evidence of the prevalence of function over the taxonomy is shown, for ATP synthase genes family of mitochondrial and chloroplast genomes of plants.

METHOD OF EVALUATION OF STRAINE TO ESTIMATE THE DYSFUNCTION OF HEART CHAMBERS OF PATIENTS WITH ASD

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We investigated the conditionally health persons ($n = 17$) and the patients with atrial septal defect (ASD) ($n = 74$) through medical ultrasound of heart. The healthy group comprised 7 boys and 10 girls aged from 4 months to 16 years old. The ASD patients group comprised 29 boys (39,2 %) and 45 girls (60,8 %) ages from 1 month to 17 years old. It should be stressed that these groups differ statistically in age ($p = 0{,}003$).

We studied the structural features of hearts: linear dimensions, volumes of a heart compartments, mass of ventricle cardiac muscle and left atrium (LA), the thickness of interventricular septum (IVS) and the posterior wall of the left ventricle (LVWS) of the heart posterior wall of LV. Also, we studied the functional indicators including stroke volume (SV), ejection fraction and LV shortening fraction (LVEF and LV FU), fraction of changes in the ventricle (PI S RV), time of blood ejection into the aorta, amplitude and velocity of movement of lateral segments of the mitral (MAPSE, LVs) and tricuspid (TAPSE, RVs) valves, peak velocities and duration of phases of passive (E, t_E) and active filling of the ventricles ($E', t_{E'}$).

Longitudinal deformation assessment of the myocardium of the heart compartments (strain) was carried out with QLAB software package. Doppler analysis of tissue shows

no statistically significant difference between the ventricles (strain was 23.37 ± 5.62 for the left ventricle and 21.50 ± 2.87 in the control group, $p = 0.729$; the strain was $-21.50[-24.00, -20.00]$ for the right ventricle and $-23.00[-27.00, -19.00]$ in the control group, $p = 0.996$).

We plan to study the contractility of the atria, since they are primarily subjected to pathological transformation in ASD. Probably, a variation in the atrial Strain index may be an early predictor of cardiac dysfunction in ASD.

THE DYNAMICS OF THE SIRS PDE EPIDEMIC MODEL

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COVID-19 is a global health problem, which causes severe acute respiratory syndrome (SARS). To examine this problem, mathematical modelling techniques can be used to provide a perspicuous description of SARS-CoV-2 viral infection dynamics. In this study, we re-formulate the Susceptible-Infected-Removed (SIR) system by incorporating a spatial diffusion term to model the effects of local dispersal in shaping the transmission dynamics of COVID-19. Then, the possibility of reinfection is considered in this PDE model [1, 2], and the combined influences of migration process and reinfection force are investigated further. Numerical simulations are implemented and analyzed to study the long-term behavior of the epidemic model with local dispersal [4, 3]. Our results demonstrate that the number of active cases increase because of the joint effects of reinfection problem and migration process of infected individuals.

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MODELING AND ANALYSIS OF DELAY INDUCED IMPULSIVE VACCINATION TO CONTROL THE SPREAD OF COVID-19

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The process of modeling is broadly used in deciding the scheme for the alleviation of the influence of infectious diseases. These days, the modeling of COVID-19 infection is among recent topics. The purpose of this article is to investigate the effect of the vaccine on the spread of COVID-19 infection. We analyze the Susceptible-Exposed-Infected-Vaccinated-Recovered (SEIVR) epidemic mathematical model of COVID-19. Our model includes two important aspects of COVID-19 infection: delayed start and effect of impulsive vaccination. The model has been analyzed theoretically and numerically both. We found that the COVID-19 infection-free periodic solution is globally asymptotically stable. Numerical simulations further show that impulsive vaccination with the vaccine of high efficacy will have the potential to reduce the spread of COVID-19 infection.

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ANALYTICAL AND NUMERICAL BIFURCATION ANALYSIS OF TLR4 SIGNALING DYNAMICS: FROM OSCILLATIONS TO HOMOCLINIC EXPLOSIONS

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We perform bifurcation analysis of a model of three ordinary differential equations that has been addressed to model the oscillations observed in TLR4 signaling [4]. We provide analytical results for the fixed points and their stability with respect to the model parameters and the location of the Andronov-Hopf bifurcation points. We present shortly the general properties and connections between the various Lorenz-like systems under homothetic transformations [2]. We also perform a one-parameter numerical bifurcation analysis to trace the branches of oscillating solutions past a limit point bifurcation of limit cycles. By doing so, we reveal a homoclinic bifurcation and we show that due to this bifurcation a chaotic regime emerges due to a homoclinic explosion at the origin [1, 5]. We provide also an analytical proof of the existence of the homoclinic orbit [3] is also given and we show that the model is similar to Lu, yet having an extra parameter that it is not present at the original system of Lu.

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DISGUISED TORIC DYNAMICAL SYSTEMS

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We study families of polynomial dynamical systems inspired by biochemical reaction networks. We focus on complex balanced mass-action systems, which have also been called toric dynamical systems. These systems are known or conjectured to enjoy very strong dynamical properties, such as existence and uniqueness of positive steady states, local and global stability, persistence, and permanence. In [1], we consider the class of disguised toric dynamical systems, which contains toric dynamical systems, and to which all dynamical properties mentioned above extend naturally. We show that, for some families of reaction networks, this new class is much larger than the class of toric systems. For example, for some networks we may even go from an empty locus of toric systems in parameter space to a positive-measure locus of disguised toric systems. We focus on the characterization of the disguised toric locus by means of (real) algebraic geometry.

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MATHEMATICAL MODELLING OF THE FISH NEUROENDOCRINE DYNAMICS

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The neurohormonal communication networks between the brain (notably the hypothalamus - pituitary complex) and the peripheral glands (such as the gonads) build up the dynamic endocrine signalling pathways of the aquatic lower vertebrates, especially fish [3]. The neuroendocrine controller involved in this circuitry can be effectively reduced to the hypothalamic-pituitary-gonadal (HPG) axis. In the classical HPG axis, the gonadotropin releasing hormone (GtRH) is produced in the hypothalamus, which reaches the pituitary gland to stimulate the gonadotropins, such as the follicle stimulating hormone (FSH) and the luteinising hormone (LH) [1]. These hormones regulate the steroidogenesis in the reproductive system, however, the fully quantitative and mechanistic understanding on the fish HPG dynamics has not yet been achieved.

In this presentation, a completely analytical framework is shown as a model for the signalling pathway that governs the neuroendocrine dynamics of HPG axis in fish. Based on the numerical simulation model reported by Kim et al. [2], a linear system of ordinary differential equations was constructed to represent the metabolic networking structure. We analytically solve the equations to obtain approximate solutions for predicting the time-varying behaviours of the hormone (FSH and LH) biosynthesis in the fish HPG axis, by assuming environmentally relevant situations that affect the GtRH signalling. This theoretical model enables the quantitative data analysis and mechanistic prediction of the reproductive pharmacokinetics of fish in the context of ecotoxicology.

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MICROBIAL POPULATION GROWTH INDICATION BY COUPLED ELECTRO-OPRTICAL AND CHEMICAL DYNAMIC SYSTEMS

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Here we report the results of mathematical modelling aimed at the determination of the microbial population growth via a newly developed portable microbiological analyser[3]. The device functioning is based on the measurement of the optical density (colour-filtered light flux created by SMD 2835 LEDs) in wells of a microbiological plate filled by a suspension of micro-organisms in a solution with broth medium and resazurin redox indicator.

This device has certain advantages over standard spectrophotometers due to its compactness, possibility to operate autonomously (e.g. at filed studies), read and save for further computer-based processing of multiple high-resolution data as the system operates under AT91SAM7S321-AU Atmel microcontroller. At the same time, these simplifications have their dark side in the requirement of developing a more complicated computational counterpart, which should take into account dynamics of the emitted light flux accompanying switching on and relaxation of LEDs, the chemical dynamic system, which describes the kinetic transition between blue resazurin and its products, pink resorufin and colourless dihydroresorufin, and, finally, the population dynamics system. The latter is coupled to the chemical subsystem since the transition from resazurin to resorufin occurs

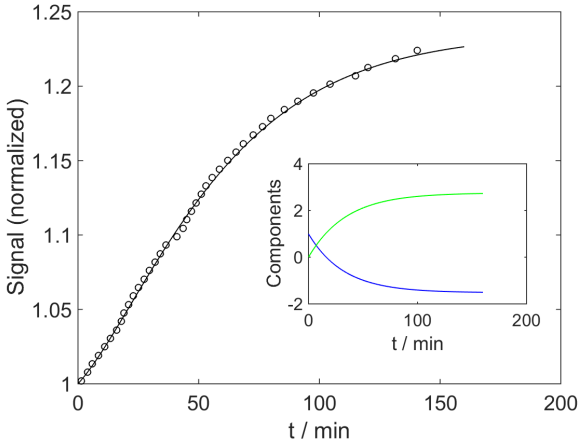

Figure 1: An example of the photometric curve registered during the growth of the culture of baker's yeast *S. cerevisiae* (markers), the solution to its dynamic model (curve) and the decomposition of the latter into components (inset) corresponding to chemicals affecting the indicator solution's colour.

intracellularly and is associated with the vital activity of micro-organisms.

The analysis of the chemical system directly related to the measured rate of colour change was based on the kinetic mechanisms described in the works [1, 2]. It is argued that the registered photometric curve can be represented as a combination of two solutions to the von Bertalanffy equations, see Fig. 1,

$$LI(t) = \sum_{j=1}^{2} \left[(K_j - d_j) \left(1 - e^{-r_j t}\right) + d_j \right],$$

where the first component is coupled with the population growth model and defined the transition from resazurin to resorufin while the second has a purely chemical origin. Coefficients of the equations mentioned above were found by using Loglet Lab 4 software.

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IN OR OUT: HOW LOCKDOWN AFFECTS THE SARS-COV-2 PROGRESSION IN PORTUGAL

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SARS-CoV-2, also known as Covid-19, has been the major player on the most recent pandemic. Even though all countries are being affected by the virus, the way each country faces the situation is different. Portugal has been multiple times complimented on the measures used but also reproached on some situations. Even though we can model it with a simple SEIR situation, it was decided to implement a new variable in the equation, the lockdown, following ideas from Glass [1]. By taking into account the official lockdowns dates in Portugal, we tried to understand how to model the current pandemic in Portugal. Looking into official and mandatory lockdowns we even inferred how willingness was important to recreate the situation. It is possible to understand that the mandatory lockdowns already have enough impact to discard a simple SEIR model, and how it can be helpful to model willingness, which supposedly follows an game theory pattern like the imitation game described by Wang et al [2]. Other classes to the model can be considered to further make the model more accurate.

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COMPARING COVID-9 TESTING AND OTHER NON-PHARMACEUTICAL INTERVENTIONS

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We would like to apply the method of Dénes and Gumel [1] which uses the previous work of Lipsitch, to describe the isolation via testing more precisely. According to the model, a proportion of individuals are going to quarantine, from those who contacted infected individuals. We would like to apply this method to introduce two different methods of testing in an epidemic: mass testing and testing with contact-tracing. With introducing this approach, we could determine that with different capacities and disease prevalences, which testing method is more feasible.

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PREDICTING SOIL LOSS AND RUNOFF IN VINEYARDS USING A GRADIENT BOOSTING FRAMEWORK

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Accelerated soil water erosion in agricultural hilly areas impacts negatively on crop yield, surface water resources and territorial infrastructures [3]. In the context of an increasing anomaly of rainfall patterns and water scarcity due to climate change, reducing soil erosion by sustainable management practices is a priority for Europe [4]. Soil erosion is influenced by several factors, both natural and human-driven. In the last decades, empirical models have been developed and largely used to assess soil erosion, which is the first step in soil conservation. Machine learning techniques offer new possibilities to face the quantification of soil erosion, based on agro-meteorological data available from existing databases and field monitoring.

The objective of this investigation was to estimate single event soil loss and runoff using a machine learning approach. The inference was developed using 20-years data collected on an hydraulically bounded vineyard plot in Piedmont, North Italy. At first, rainfall erosivity for each event was derived from hourly precipitation records, using well calibrated conventional model and a simple feed-forward neural network. Then, the non-linear relationship between hydrological variables (soil loss and runoff) and rainfall characteristics (rainfall amount, rainfall duration, maximum intensity, derived rainfall erosiv-

ity) was tested using a decision-tree-based ensemble machine learning algorithm, namely XGBoost [1]. The ensemble and the training/test set splitting were designed to deal with the difficulties of managing small dataset.

Results are encouraging: the out-of-sample R^2 between the simulated and the observed soil loss values reached 0.33, while the runoff of unseen events was estimated with a mean absolute error of 14 mm. Furthermore, we studied the explicability of the model using the SHAP method [2]. In this way, we identified the most influential features in predicting each dependent variable. Our findings could contribute to the development of machine learning applications in the context of soil erosion related problems.

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BREATH FIGURES ON SURFACES WITH DIFFERENT DENSITY OF NUCLEATION SITES

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Breath Figures (BF) [1] are ubiquitous in Nature and artificial processes. They are patterns that appear upon heterogeneous drop-wise condensation of vapors, usually, on colder surfaces. When condensation takes place on a clean, homogeneous and hydrophobic surface, we can observe four stages: 1) nucleation, 2) diffusive growth of droplets, 3) coalescence dominated regime and 4) re-nucleation and appearance of new families of drops.

In this work, we report experimental results [4] on the condensation of water vapor on coated glass substrates with different density of nucleation sites. We characterize the initial morphology and the time evolution of corresponding topological features. To analyze the results, we use standard techniques in BF, together with topological data analysis. We show that higher densities produce faster dynamics, where early stages can be too short to be detected at the scales used in this study. However, at increased flow rates, the efficiency displayed by each substrate in yield measurements does not directly correspond with the one observed locally. The deviations may be associated with the coating procedure, that allows us to obtain a surface with many nucleation sites (microscopically hydrophilic) being macroscopically hydrophobic [3]. Both properties play an important role in water collection, the former enhances the condensation process while the latter increases the drainage ratio [2].

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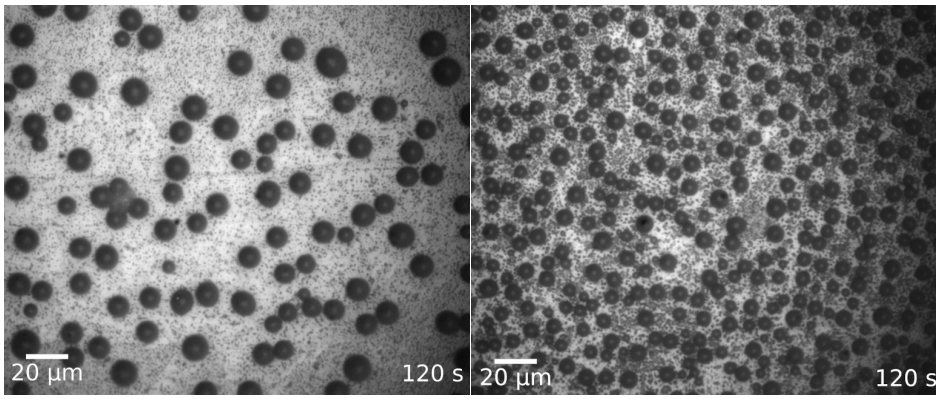

Figure 1: Breath figures, produced under the same experimental conditions, except for the substrate. Left: Substrate with low density of nucleation sites; Right: Substrate with high density of nucleation sites.

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TRANSIENTS AND GHOSTS IN COLLAPSING ECOSYSTEMS

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Current ecosystems are suffering Anthropogenic perturbations which threaten our own existence. Human societies are pushing the biomes towards their limits, even making some of them to reach tipping points. For instance, the rise of global temperature is increasing the aridity in already arid and semiarid ecosystems, thus promoting changes in e.g., the vegetation cover. Recent studies have dentified multiple states at the same aridity levels [1]. Depending on the different drivers (such as wildfires, land use, or grazing) different regimes can be achieved [2]. These transitions between the different regimes (biomes) are not immediate in time since they often involve long transients between them. The time needed to reach the new configuration depends on the type of the tipping points and thus in the underlying bifurcations governing such transitions [5]. In highly cooperative systems (i.e. soil-plant interactions), cooperation typically induces tipping points driven by saddle-node bifurcations [3]. Near these bifurcations, ecosystems can experience extremely long transients remaining in the healthy state but, eventually, they can rapidly collapse. These are the so-called dynamical ghosts [6]. Similar behaviour occur near the zip bifurcation, where a curve of quasinetural equilibria appears[4]. In this poster, we show the different transient behaviours that can be found in simple ecological models. From local (i.e. transcritical, saddle-node, Hopf-Andronov) to global (i.e. heteroclinic and zip) bifurcations. Many ecosystems may be currently beyond their tipping points but seeming healthy because they are living in their ghost state. At the same time, some periodic interventions could maintain ecosystems in such ghost regime (avoiding their collapse) before making a more radical change involving the restoration of the parameters involving ecosystems asymptotic persistence [6].

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OPTIMAL CONTROL AND STABILIZATION OF BILINEAR DYNAMICAL SYSTEMS ON TIME SCALES

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In this work, we study the strong stabilization of bilinear dynamical systems on arbitrary time scales with bounded graininess function. The attempted approach here is based mainly on Lyapunov's techniques. So, we establish sufficient conditions for an explicit control to guarantee the strong stabilisation by minimising a time scale quadratic cost criterion. Moreover, we estimate the convergence rate of the state. An illustrating numerical example is given.

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