FROM SOIL TO GUT:

THE ROLE OF URBAN AND FOREST SOIL IN BANK VOLE GUT MICROBIOTA

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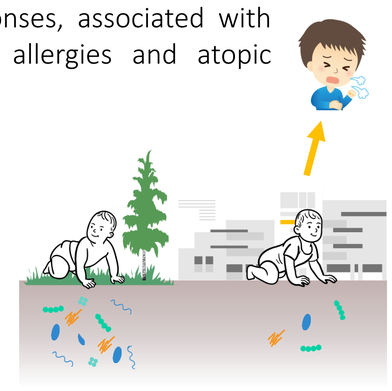
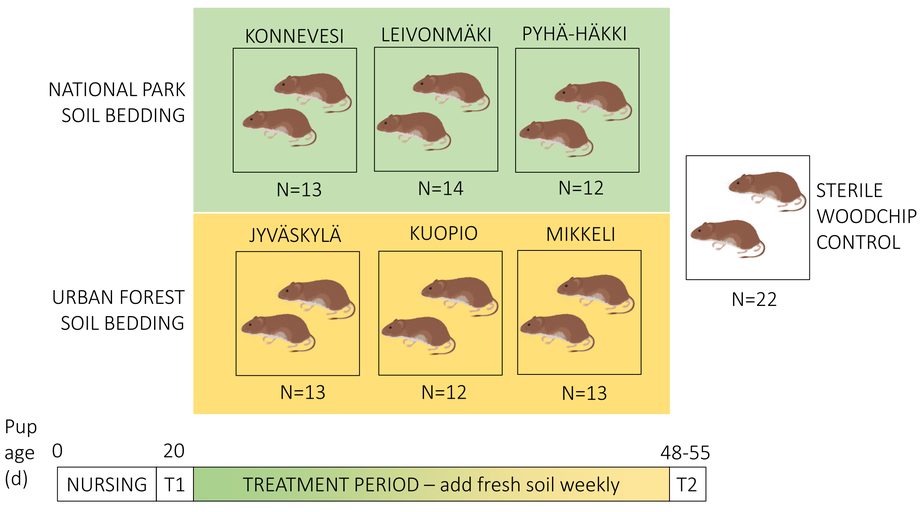
BACKGROUND – THE BIODIVERSITY HYPOTHESIS

- Soils harbor diverse microbiota and are major sources of microbes associated with other organisms, including humans.
- Low diversity in environmental microbiota is associated with dysfunction of type-2 inflammation responses, associated with 'civilizational diseases' such as asthma, allergies and atopic dermatitis in humans and laboratory mice.
- Human land use negatively impacts biodiversity.
- AIM: to determine whether soil from forests with different levels of human use affect gut community assembly in wild rodents.



STUDY DESIGN

- Soil collected from three national parks + three urban forests in central Finland
- Young bank voles exposed to soil material in individually ventilated cages
- Sterile food pellets & water supplied *ad libitum*
- Faecal pellets collected before (**T1**) and after (**T2**) soil treatment
- Bacterial 16S rRNA V3-V4 and fungal ITS2 region sequenced on Illumina Miseq platform
- Statistical analyses in 3 treatment groups: **control**, national park soil (**NP**) and urban forest soil (**UR**)



CONCLUSIONS

- Faecal bacterial communities were relatively stable, which implies host selection and/or barrier effect of bacteria after initial colonization following birth and during nursing.
- In contrast, fungal communities appeared transient, and their dynamics reflect the experimental setup: rare taxa enter the gut from soil impacting community membership, while composition is dominated by common diet effect.
- These results contrast to similar experiments utilizing inbred laboratory mice, which show significantly larger effect sizes than these results for bacteria. Community assembly in lab models is more stochastic, but their gut health is poorer than in wild counterparts, possibly affecting immune system development later in life.
- Results highlight that more research on immune system development in wild models would be insightful.

RESULTS

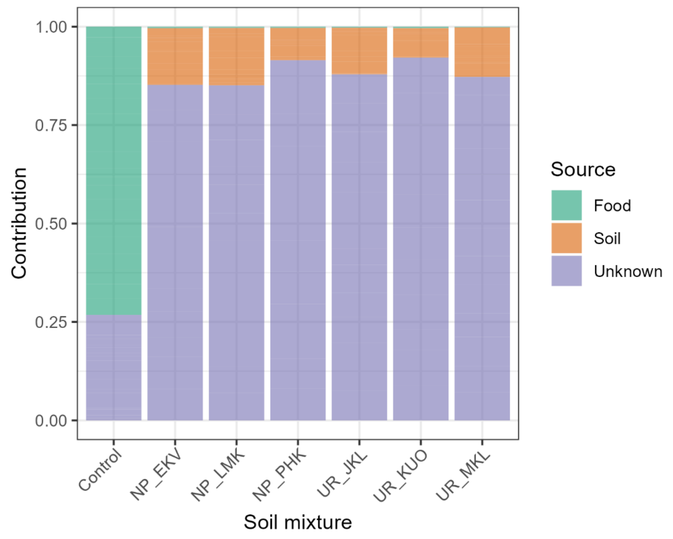


Fig. 1: Source tracking (FEAST)

- Soils (sources) contributed to bank vole gut mycobiota communities (sinks) where present; in control samples (no soil), the highest contribution originated from food pellets.
- Bacterial contributions (not shown) were fully attributed to source other than food and soil, suggesting a higher barrier to colonization through the GI tract after weaning and that the bacterial community was largely established before start of treatment.

Fig. 2: Alpha diversity

Bacteria

- Significant increase in ASV richness from T1 to T2 in all treatment groups, potentially due to blooming of very rare taxa (**A**).
- Shannon index remained unchanged in soil treatments, during microbiota assembly, while the same index in control groups increased significantly (**B**).

Fungi

- ASV richness increased in soil treatments from T1 to T2 (**C**), consistent with source tracking (**Fig. 1**).
- Drop in Shannon evenness in control groups was likely an effect of food pellet diet without input of rare taxa from soil (**E**).

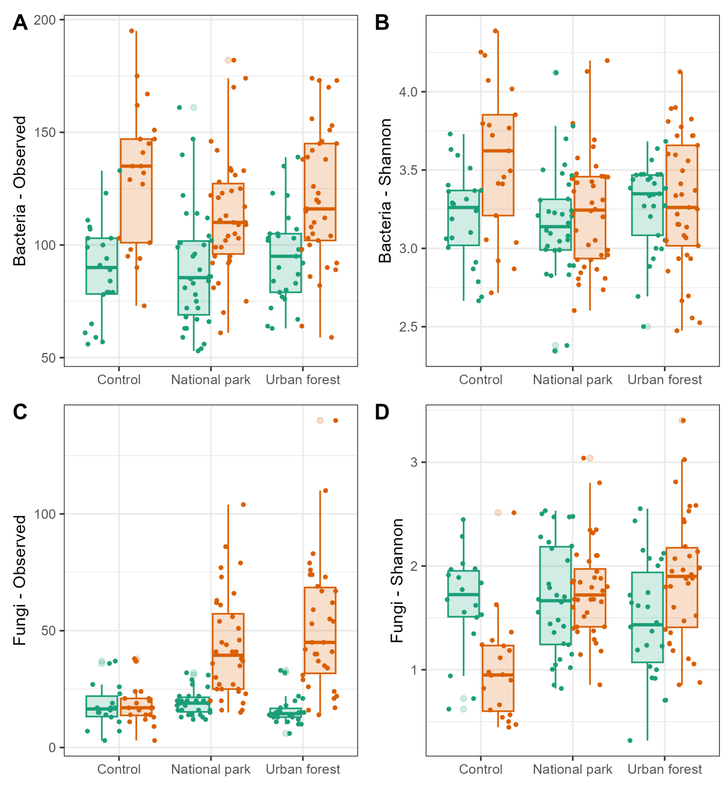


Fig. 3: Beta diversity at T2

Bacteria

- No significant difference in membership of bacterial taxa between treatment groups and control (**A**).
- Differences in community composition between treatment groups driven by change in abundance of phylogenetically distinct taxa (**B**).

Fungi

- Distinct treatment effect on fungal community membership (**C**), less so on abundance (**D**), an effect of common pellet diet and gain of rare taxa from soil.

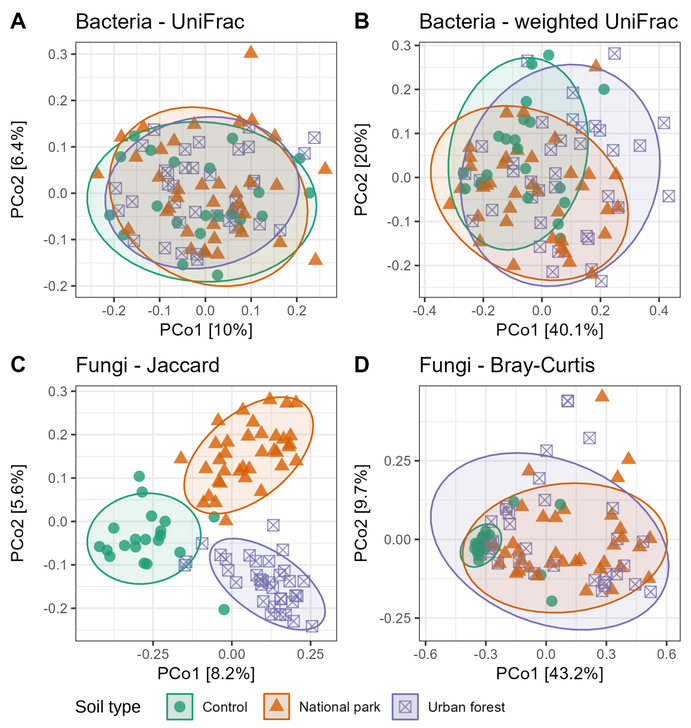
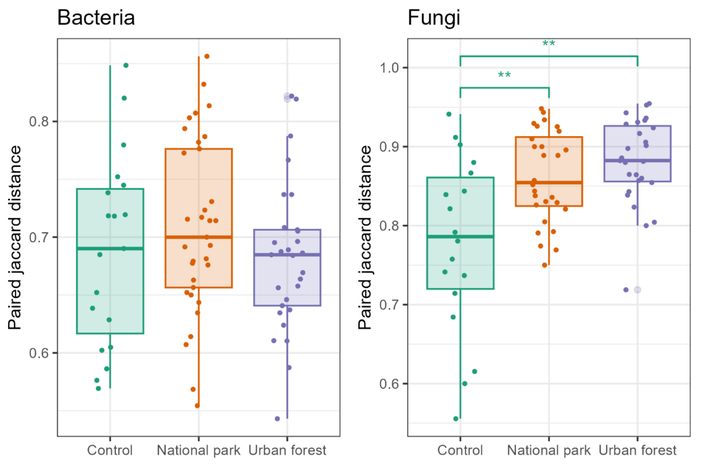


Fig. 4: Paired Jaccard distance analysis

- Change in fungal membership through time is treatment-specific, but this effect was not evident in bacteria.



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