**Project description**

The goal of the project is to explore in silico how microbial interaction rules within hosts--which influence the composition and stability of the microbiome--interact with patterns of contact between hosts--which affect who may acquire microbial taxa from whom. The overarching interest is in how host populations might collectively influence the biogeography of their microbiomes, (and consequences of microbiome variation, especially regulating infectious disease transmission)?

**Background**

Multiple studies have documented instances where microbiomes tend to be more similar in hosts that are connected in some way, for example sharing the same houses, transit systems, or cities. The mechanisms underlying these patterns are not clear. More specifically, these patterns could be explained by several different processes, which may interact: microbiomes could be similar because they share a common rule set for microbial interactions, for example arising from a common environment; microbiomes could also be similar because they are connected by frequent dispersal events - all else equal, sharing microbes by dispersal should make microbiomes more similar in their community structure.

Why do we care how dispersal and intrahost dynamics combine to produce similarities in microbiomes among connected hosts?

Because microbiomes affect health outcomes so want to understand and control them? Transmissibility of sets of microbes that confer resistance or resilience to disease?

Fundamentally: population structures that amplify or attenuate bass-becking?

What does an answer to this question look like (beyond the mushy middle where 'it's both and it depends' and what difference would a more refined understanding make?

Ecology has a well-developed theoretical framework for exploring this issue, which treats host populations as networks of microbiomes--microbial metacommunities. Within-host dynamics are described by a Generalized Lotka-Volterra (GLV) model, and host microbial communities are connected by a microbial dispersal network, which models the rate of microbial transmission between each pair of hosts.

In a GLV model, a community of S taxa has 2S possible equilibria, corresponding to nodes in a binary tree where each taxa can be either present or absent. Not all these equilibria will exist. However, for a given set of community interaction rules A, there are often many equilibria, each consisting of a subset of the global taxa pool of S taxa. A given equilibrium may or may not be invasible by a taxon it does not currently contain, depending on the community interaction rules. So which equilibrium a microbiome ends up at can depend on both the hosts's microbial interaction rules, as well as which microbial taxa a host is exposed to over time.

What is not obvious is the specific characteristics of microbial communities that may amplify or attenuate the impact of dispersal, and the specific characteristics of host contact networks that may promote or suppress the consequences of variation microbiomes community rulesets. Microbiomes provide important ecosystem services to their hosts, so it would be good to understand where variation in microbiome community structure comes from, how it can be predicted or controlled, the role that host population structure may have in determining microbial biogeography, and how that might feedback to affect host populations.

**Hypotheses**

Two non-mutually exclusive hypotheses explain correlations between host contact structure and microbiome biogeography. These correspond to long-standing hypotheses about the fundamental drivers of community ecology (Velland).

Microbial community interaction rules differ among hosts, and hosts in contact tend to have more similar rules, leading to more similar microbiomes.

Hosts in contact swap more microbial taxa, so they have more similar communities. Swapping taxa may increase community overlap, and attenuate ecological drift.

Crucially, the force of each hypothesis depends on the other. The second hypothesis depends on the first, because the impact of microbial dispersal on microbiomes may depend how similar microbial rule sets are among the hosts. The force of the first hypothesis may depend on the second because sharing the same community ruleset does not guarantee that two hosts will have similar communities. In strongly dispersal-limited scenarios, ecological drift will dominate, leading to random differences among communities, even if they share the same interaction rules.

**Questions**

If microbial interaction rules within hosts are universal, does that facilitate microbial spread among hosts?

Theoretical studies of evolutionary dynamics on graphs have shown that it is possible to build 'evolutionary drift amplifiers' - dispersal networks that favor drift over selection (Novak). Might the same logic be used to construct ecological drift amplifiers for metacommunities?

If community rules are universal, do metacommunities drift slower?