ANSI/HUMBIO 179:
Notes on Pathogen Persistence in Multi-Species Communities

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1 A Graphical Approach

Bob Holt and colleagues present a very important heuristic framework for thinking about the persistence of pathogens in multi-species communities.(1)

All of the following figures depict zero-growth isoclines for the pathogen. They plot the mix of species at which $\mathcal{R}_0 = 1$. The interior of the space inscribed by the isocline represents the space where the pathogen can not be maintained in the community. This is a central technique in theoretical ecology. It is also widely used in microeconomics, where such curves are typically called "indifference curves."

The basic theory for this work is given in Tilman's classic volume.(2) All but the last of the isoclines were anticipated by Tilman in his work on competition and the structure of ecological communities.

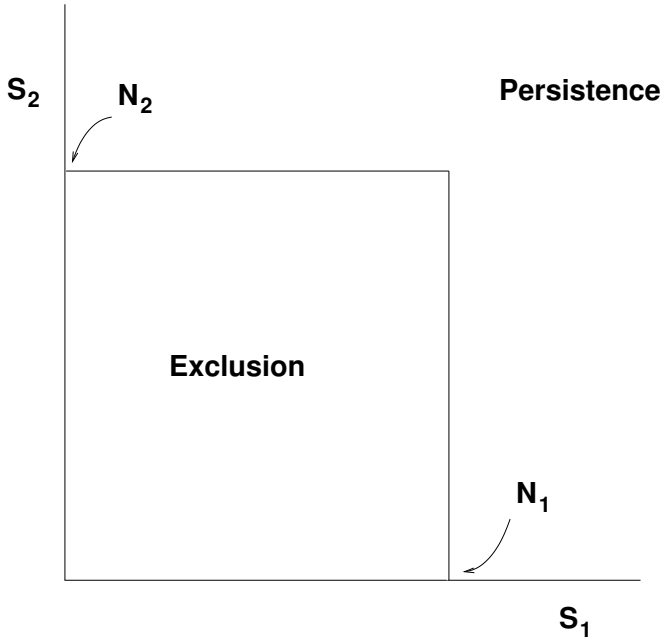

Figure 1: Noninteracting hosts

The basic model – the "noninteractive" case (fig. 1) – is one in which pathogen persistence is predicated on a critical threshold of either species 1 or species 2. The pathogen goes extinct only if *both* species are under their critical densities (N_1 and N_2).

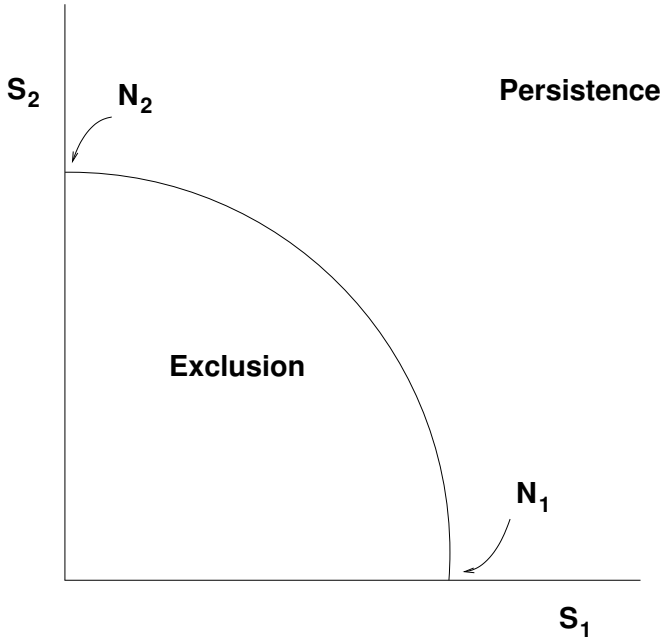

Figure 2: Weakly interacting hosts

In the "weakly interacting" case (fig. 2), a mix of species makes pathogen persistence more likely than a monoculture of either species. That is, a small number of S_1 can substitute, albeit inefficiently, for S_2 being below threshold. (and vice-versa)

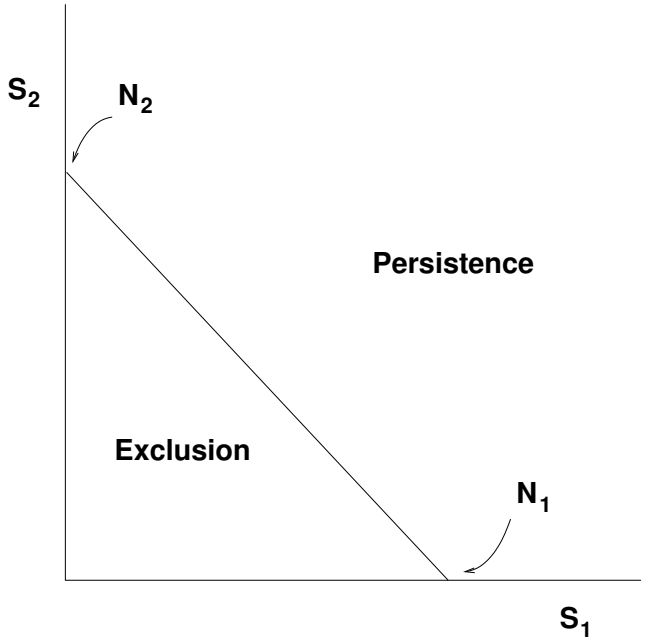

Figure 3: Substitutable hosts

The "substitutable" case (fig. 3) characterizes a community where S_1 and S_2 efficiently substitute for each other. A constant ratio of substitutability (the slope of the line) applies at all host densities. In the special case of perfect substitutability applies when a single S_1 can be substituted for a singe S_2 with regard to maintaining pathogen persistence.

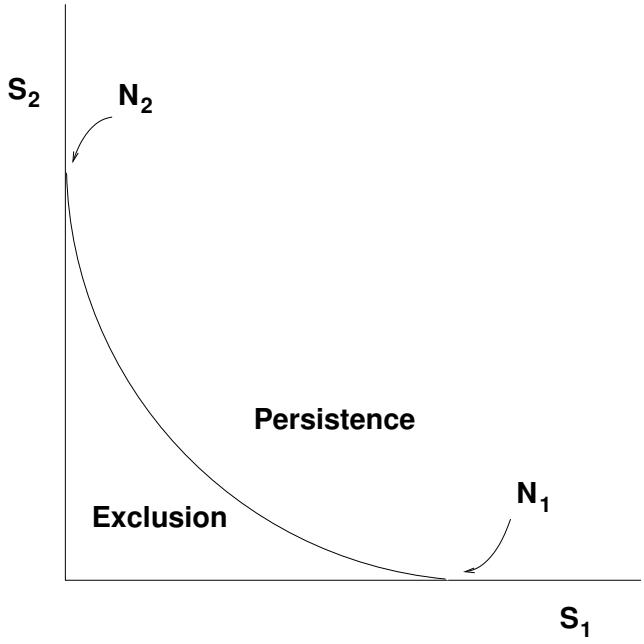

Figure 4: Complementary hosts

It between-host transmission is more efficient than within-host transmission, the isocline bends inward (fig. 4). Thus, in the "complementary" case, pathogen persistence is much more likely in a multi-host community than in single host populations.

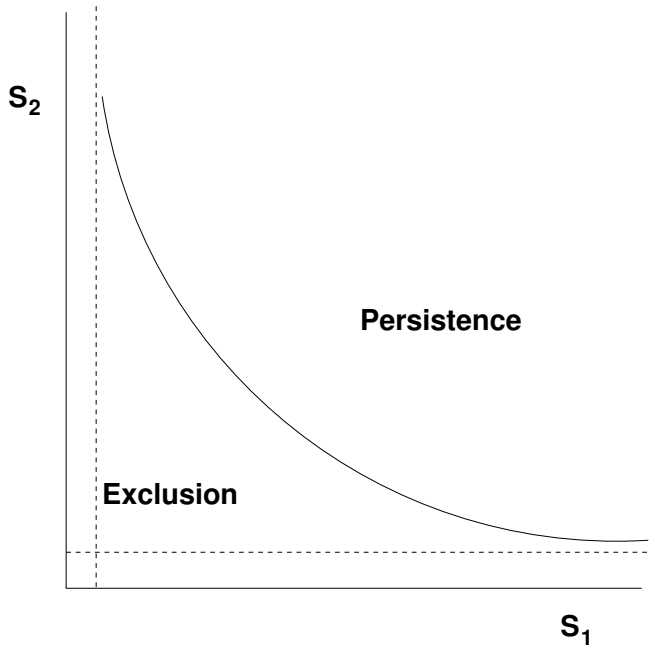

Figure 5: Alternating hosts

For vector-borne pathogens with complex life cycles, passage through an intermediate host is obligate for the perpetuation of the transmission cycle. Frequently, passage through the ultimate host is also obligate. Elimination of either intermediate or ultimate hosts from the community will lead to pathogen extinction. Thus, in the "alternating" case (fig. 5), a critical threshold exists for one or both species. As long as both host species co-exist above their minimum critical densities, the presence of a mix of both hosts makes pathogen persistence more efficient – this is why the isocline bends inward.

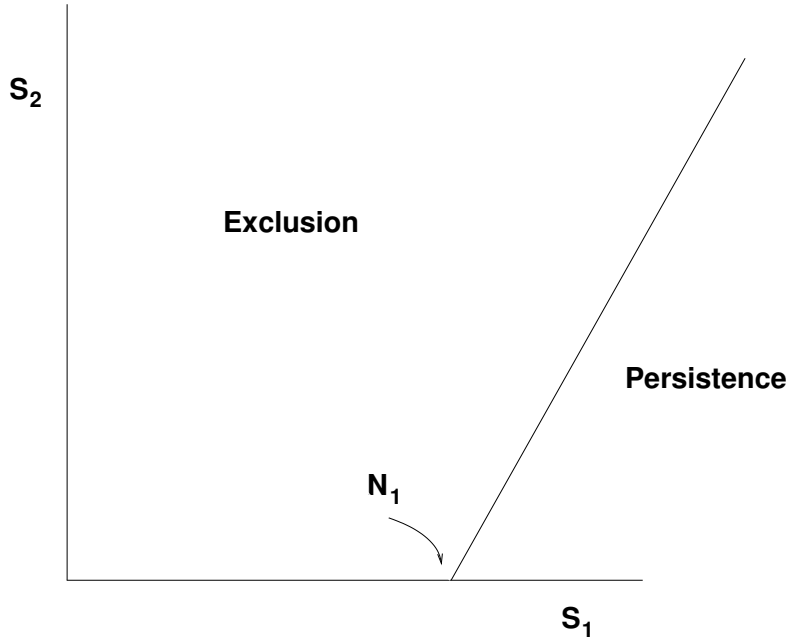

Figure 6: Inhibitory hosts

The "inhibitory" case (fig. 6) is unusual, as the slope of the isocline is *positive*. For the given plot, a critical threshold of S_1 is required for pathogen persistence. The presence of *any* of S_2 means that there must be more S_1. Pathogen persistence will be much less likely in multi-host communities.

Holt et al. note that this particular isocline does not appear in Tilman's original typology for competitive communities. There are some very interesting applications in infectious disease ecology that we will discuss in class. The first is the case of intact mammalian communities in Eastern North American woodlands and their diluting effect on Lyme disease transmission (discussed in the reading by LoGiudice et al.). The second is discussed in the Cohen and Gürtler reading, in which the presence of peri-domestic chickens acts as a sink for Chagas' disease transmission.

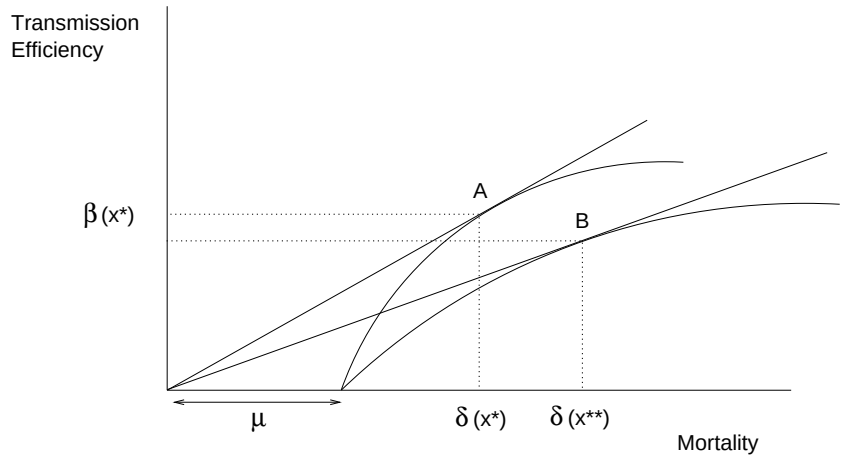

Figure 7: Virulence trade-off for a specialist pathogen where there is a trade-off between transmission probability $\beta(x)$ and the disease-induced mortality $\delta(x)$ as a function of virulence x. Baseline mortality μ is not a function of x.(3)

Multi-host communities change our expectations regarding the pathogen evolution. Specialist pathogens are expected to evolve toward intermediate virulence. One way of thinking about this is to imagine that transmissibility and duration of host infection are both functions of virulence. Highly virulent pathogens make lots of copies of themselves, making transmission more likely, but lots of copies makes the host sick, potentially killing them in a short amount of time (and thus making the duration of infection shorter). We can easily imagine that spillover infection from one host (to which the pathogen is adapted) to another (to which the pathogen may not be adapted) will lead to reduced transmissibility relative to disease-induced mortality (i.e., moving from curve A to curve B in figure 7). In the situation, we expect virulence – and, hence – disease-induced mortality – to increase. For generalist pathogens, hosts which are not essential for pathogen fitness will not exert a sufficient selective force to push the pathogen toward reduced virulence. This is why some zoonoses are so pathogenic. For example, *Echinococcus multilocularis* is a cestatode worm enzootic in Central European foxes. When it spills over into human populations, the case fatality rate can exceed 98%!

References

1. Holt, R.D., A.P. Dobson, M. Begon, R.G. Blowers, E.M. Schauber. 2003. Parasite establishment in host communities. *Ecology Letters* 6: 837-842.

2. Tilman, D. 1982. Resource competition and community structure. Monographs in Population Biology, vol. 17. Princeton: Princeton University Press.

3. van Baalen, M. and M.W. Sabelis. 1995. The dynamics of multiple infection and the evolution of virulence. *American Naturalist* 146: 6: 881-910.