**MBW:Population Dynamics of Pathogens with Multiple Host Species**

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**Overview**

* Mathematics used: ordinary differential equations (ODEs), systems of ODEs, linear algebra
* Biological system studied: microparasite and host population dynamics

**Executive Summary**

Here we review the paper [*Population Dynamics of Pathogens with Multiple Host Species*](http://www.cs.unm.edu/%7Emelaniem/courses/CAS08_files/DobsonAmnat2004.pdf), by Andrew Dobson (2004). Dobson (2004) extends the standard susceptible-infected-recovered (SIR) epidemic model to the multi-host case, where a generalist pathogen is able to infect and transmit from more than one host species. Building on previous work on epidemics in structured host popoulations in the context of the HIV epidemic (Diekmann et al. 1990), Dobson presents a community-wide pathogen reproductive ratio (community R_{{0}}) that provides a threshold for pathogen invasion analagous to the standard measure of R_{{0}}commonly used in a single species case. He then explores the relationship between host diversity and disease risk - a foundational question in disease ecology, with the potential to provide a utilitarian motivation for biodiversity conservation.

**Background**

Generalist pathogens (that infect multiple host species) are important in human epidemics, livestock disease, and conservation (Jacquez et al. 1988; Deem et al. 2001; Jones et al. 2008). As a result, biologists have sought to understand how host community traits and composition affect pathogen transmission and disease risk. The classical Kermac-McKendrick susceptible-infectious-recovered (SIR) model deals with an epidemic in a single host population:

{\dot  {S}}=-\beta SI

{\dot  {I}}=\beta SI-\gamma I

{\dot  {R}}=\gamma I

The parameters \beta and \gamma are the pathogen transmission and host recovery rates, respectively. Removed individuals are either dead or immune for life; they do not return to being susceptible. Such models have been extensively used to model human, wildlife, and plant epidemics, and are described in detail in [MBW:Population Biology of Infectious Diseases](https://mathbio.colorado.edu/index.php/MBW:Population_Biology_of_Infectious_Diseases). One critical finding for SIR models is a threshold for pathogen invasion in a naive host population, termed R_{{0}}, or the pathogen reproductive ratio:

R_{{0}}={\frac  {\beta N}{\gamma }}.

If R_{{0}}<1, the pathogen cannot invade because hosts recover too quickly and transmit too little for an epidemic to occur. Alternatively, if R_{{0}}>1the pathogen can invade. One intuitive interpretation of R_{{0}}is the expected number of secondary cases arising from one infectious individual in an otherwise naive host population.

**Mathematical Model**

Dobson extends the SIR model to include a set coupled differential equations for each of *N* host species indexed by *i* (*i*=1, *i*=2, *i*=3, ..., *i*=*N*). Further, he assumes that the R class represents recovered (immune for life) individuals only. Removed (dead) individuals are accounted for by deaths in each susceptible, infectious, and recovered class. In addition, Dobson assumes density-dependent population growth and equal reproduction rates across all host classes. The equations are as follows:

{\dot  {S_{i}}}=(b_{i}-\Delta _{i}N_{i})N_{i}-d_{i}S_{i}-S_{i}\sum \beta _{{ij}}I_{j}

{\dot  {I_{i}}}=S_{i}\sum \beta _{{ij}}I_{j}-(d_{i}+\alpha _{i}+\sigma _{i})I_{i}

{\dot  {R_{i}}}=\sigma _{{i}}I_{{i}}-d_{i}R_{i}

Here b_{i}is the birth rate of species *i*, d_{i}is the death rate, \Delta _{i}is the birth rate reduction due to density dependence, \beta _{{ij}}is the pathogen transmission rate from host species *j* to host species *i*, \alpha _{i}is mortaility rate of infected individuals due to infection (in addition to background mortality), and \sigma _{i}is the recovery rate of infected individuals. Interspecific transmission rates are a scaled average of intraspecific transmission rates:

\beta _{{ij}}=c_{{ij}}({\frac  {\beta _{{ii}}+\beta _{{jj}}}{2}}),

where c_{{ij}}, bounded between 0 and 1, determines the strength of interspecific transmission. A simplifying assumption would be to assume that between-host transmission is symmetrical, and that all individuals in the community are well-mixed. As Dobson (2004) points out, however, there are many scenarios in which one would expect asymmetric transmission (as in the case of a reservoir host species) and heterogeneous mixing due to spatial, temporal, and behavioral factors that affect host contact rates.

This is similar to the basic SIR model described above, except 1) populations show density dependent growth, 2) removed individuals are no longer lumped with recovered individuals, and 3) individuals can become infected by contacting infectious individuals of multiple species. This latter point can be more plainly seen with a simple example. Here is the model for a two host species case:

{\dot  {S_{1}}}=(b_{1}-\Delta _{1}N_{1})N_{1}-d_{1}S_{1}-S_{1}(\beta _{{11}}I_{1}+\beta _{{12}}I_{2})

{\dot  {I_{1}}}=S_{1}(\beta _{{11}}I_{1}+\beta _{{12}}I_{2})-(d_{1}+\alpha _{1}+\sigma _{1})I_{1}

{\dot  {R_{1}}}=\sigma _{1}I_{1}-d_{1}R_{1}

{\dot  {S_{2}}}=(b_{2}-\Delta _{2}N_{2})N_{2}-d_{2}S_{2}-S_{2}(\beta _{{22}}I_{2}+\beta _{{21}}I_{1})

{\dot  {I_{2}}}=S_{2}(\beta _{{22}}I_{2}+\beta _{{21}}I_{1})-(d_{2}+\alpha _{2}+\sigma _{2})I_{2}

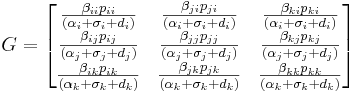
{\dot  {R_{2}}}=\sigma _{2}I_{2}-d_{2}R_{2}

It is assumed that all host populations are at equilibrium prior to the introduction of one infectious individual. In other words, the carrying capacity *K* for each species is defined as:

K_{i}={\frac  {b_{i}-d_{i}}{\Delta _{i}}}.

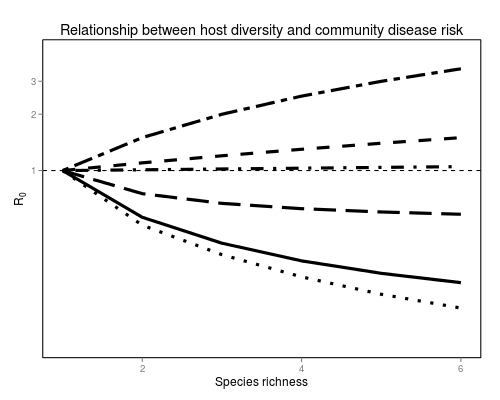
This value is equal to the number of susceptible individuals for each species prior to the epidemic, because it is assumed that the entire host community is naive.

In terms of predicting and preventing epidemics, it is useful to be able to estimate the pathogen reproductive ratio in a host community. Building upon work from Schenzle 1984, Anderson and May 1985, and Diekmann et al. 1990, Dobson (2004) presents an *N*x*N* matrix **G** whose elements represent the product of the transmission rate from species *i* to *j* and the average infectious period of species *i*, which in a 3 species case would be:



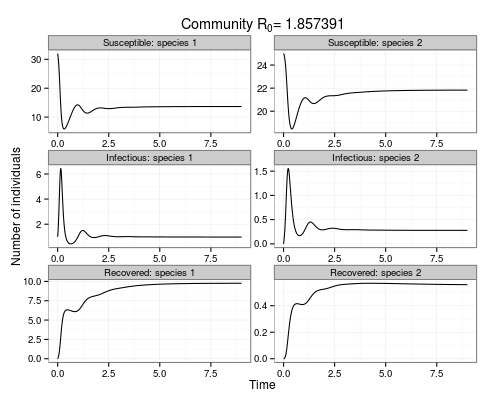
The dominant eigenvalue of *G* provides an estimate of community R_{0}. The p_{{ij}}terms determine the nature of contact rates and transmission dynamics. If transmission is density-dependent, that is, contact rates increase as the density of hosts increases (the case presented in the original system of equations), then p_{{ij}}is the density of species *j*, assuming random mixing in the host community. If host contact rates are fixed and independent of host density, then p_{{ij}}is the density of host species *j* divided by the total density of hosts in the community, again assuming random mixing in the community.

**Results**

Species demographic and tranmission rates were assigned based on allometric scaling laws following De Leo and Dobson (1996). Furthermore, every species' population was assumed to be equally invasible by a pathogen so that intraspecific R_{{0}}=1. Assuming that communities are non-interactive, so that the addition or loss of a species has no effect on the densities of other species, host diversity is predicted to increase disease risk under density-dependent transmission, and reduce disease risk under frequency-dependent transmission. [](https://mathbio.colorado.edu/index.php/File:MBW_fig1.jpeg)

All lines for which R_{0}>1correspond to density dependent transmission with c_{{ij}}= 0.5, 0.1, and 0.01 in descending order. All lines for which the community pathogen reproductive ratio is below 1 correspond to the case of frequency-dependent transmission with the same values of c_{{ij}}in reverse order from top to bottom. Thus, the way in which hosts contact eachother appears to mediate the effect of host diversity on disease risk. Density-dependent transmission is common for directly transmitted pathogens such as influenza, which frequency-dependent transmission is more common for vector transmitted infections (such as malaria, West Nile Virus, and Avian Influenza) and sexually transmitted infections.

**Extensions**

We have constructed an interactive module to explore the behavior of multi-host epidemics for a two species case, available [here](http://glimmer.rstudio.com/mbjoseph/2spSIR/). The module allows a relaxation of many of the assumptions originally presented in Dobson (2004), including total control over demographic and transmission rates. Some interesting dynamics related to host conservation emerge from an exploration of the module. For instance, host extinction is possible under both density- and frequency-dependent transmission if one species acts as a reservoir host that greatly increases transmission of a virulent pathogen in a focal species. Those interested in the construction of this module can view the source code in [this GitHub repository](https://github.com/mbjoseph/2hostSIR). [](https://mathbio.colorado.edu/index.php/File:Trajectory.png)

**Discussion**

Dobson (2004) predicted opposite effects of host diversity on disease risk under frequency- and density-dependent transmission. One implication of this result is that biodiversity loss may promote the emergence of pathogens that exhibit frequency-dependent transmission, including most vector-transmitted pathogens. On the other hand, we might expect lower epidemic risk for density-dependent transmitted pathogens, such as those that are directly transmitted or transmitted by plumes of infectious particles.

The model presented makes a strong assumption of non-interactivity of species within communities, resulting in a positive relationship between total community density and species richness. This positive relationship drives the increase in disease risk predicted under density-dependent transmission; when community density is fixed and richness is varied, host diversity theoretically should reduce epidemic risk (Rudolf and Antonovics 2005). Thus, transmission mode as well as the degree of species interactivity are likely to affect diversity-disease relationships in an interactive manner.

[](https://mathbio.colorado.edu/index.php/File:Bullfrog.jpg)

Bullfrogs are thought to asymmetrically increase fungal disease risk for other co-habitating amphibians

Dobson (2004) notes that in the future it will be beneficial to incorporate realistic transmission asymmetries (i.e. reservoir dynamics) and stochastic transmission events to understand the dynamics of generalist pathogens in nature. The analysis presented here assumes symmetrical transmission from species *i* to species *j*, but asymmetrical transmission appears common in natural systems.

For example, it is widely thought that bullfrogs increase fungal disease risk resulting from infection by the chytrid fungus *Batrachochytrium dendrobatidis* for other amphibians because they tolerate high infection levels for sustained periods (Shloegel et al. 2010). Dobson (2004) also discusses the potential for spatial structure to affect host contact rates. The current model does not include spatial dynamics, but in a real system, such questions may be relevent to managers attempting to spatially contain an epidemic.

Finally, Dobson (2004) raises the possibility that because climate change is expected to facilitate the invasion of tropical vector-transmitted parasites into less-diverse temperate areas, there may be reason to expect more severe epidemics of diseases such as Lyme Disease and West Nile Virus.

**Conclusions**

The effects of host diversity on disease risk are mediated by transmission dynamics and the relationship between host contact rates and host density. Dobson (2004) helped motivate a wealth of recent theoretical and empirical investigations into diversity-disease relationships (reviewed in Keesing et al. 2010). Further, the model presented in his article provides a general framework for exploring the dynamics of transmission when host demographic rates are known, and a way to anticipate the effects of prospective management options, such as focusing efforts on hosts that account for a high amount of transmission amplification or reduction in a community. Although the net effect of host diversity on disease risk is still debated and empirically unresolved (Salkeld et al. 2013), this mathematical framework has provided a convenient and tractable device for representing the dynamics of generalist pathogens in host communities.

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