

The role of host dispersal in multiple-pathogen coexistence

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Background

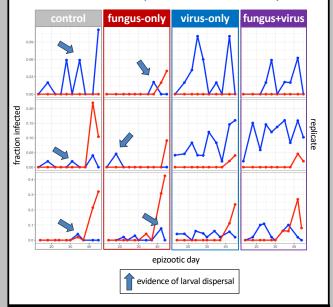
How do pathogens coexist? Lots of theory, not much data. Our approach: combine experimental and observational field data with mechanistic models.

The spongy moth-fungus-virus system is an experimentally tractable system that allows us to test multi-pathogen theory.

Spongy moth: invasive, outbreaking caterpillar. Outbreaks terminated by epizootics (epidemics in animals) of two fatal, species-specific pathogens: a virus and a fungus. Simple theory predicts no coexistence, but virus & the fungus coexist in nature.

Data

We created 3 replicate blocks of whole-tree artificial epizootics to test infection dynamics under four treatments. Across all replicate blocks, we detected evidence of dispersal that altered infection dynamics.



This unexpected result was interesting to us, particularly because the spongy moth, which invaded the US from the east coast, has been slowly migrating westward since, threating hardwood forests across North America. If we can build a model that describes larval movement and use it to estimate key dispersal parameters, we can use those dispersal estimates to predict future disease dynamics in this invasive system.

Methods

To explain these data, we built a multi-pathogen metapopulation SEIR-type model allowing for larval dispersal between treatments.

 $\frac{dS_{r,i}}{dt} \ = \ -\bar{\nu_v} \left[\frac{S_{r,i}}{S(0)} \right]^{C^2} P_{r,i} S_{r,i} - (\nu_{R,r} R + \nu_{C,r} C_{r,i}) S_{r,i} + \sum_{i=1,i \neq j}^j S_{r,i}^* m e^{-ad_{ij}} - \sum_{i=1,i \neq j}^j S_{r,i}^* m e^{-ad_{ij}} \right]^{C^2} P_{r,i} S_{r,i} P_{r,i} P_$

$$\frac{dE_{f,1,\tau,i}}{dt} = (\nu_{R,\tau}R + \nu_{C,\tau}C_{\tau,i})(S_{\tau,i} + \zeta \sum_{i=1}^{n_1} E_{vf,k,\tau,i}) - m\delta_f E_{f,1,\tau,i}$$

fungus exposed class n

$$\frac{dE_{f,n,\tau,i}}{dt} = m\delta_f E_{f,n-1,\tau,i} - m\delta_f E_{f,k,\tau,i}$$

 $\frac{dE_{vf,1,\tau,i}}{dt} \ = \ \bar{\nu_v}P_{\tau,i}S_{\tau,i} \left[\frac{S_{\tau,i}}{S(0)}\right]^{C^2} - \zeta(\nu_{R,\tau}R + \nu_{C,\tau}C_{\tau,i})E_{vf,1,\tau,i} - n\delta_vE_{vf,1,\tau,i} + \sum_{i=1}^{j} {}_{t,i}E_{vf,1,\tau,i}me^{-ad_{ij}} - \sum_{i=1}^{j} {}_{t,i}E_{vf,1,\tau,i}m$

$$\begin{array}{ll} & \text{virus exposed class k which is susceptible to fungus} \\ & \frac{dE_{\nu f,k,r,i}}{dt} & = & n\delta_u E_{\nu f,k-1,r} - n\delta_u E_{\nu f,k,r} - \zeta(\nu_{R,r}R + \nu_{C,r}C_r) E_{\nu f,k,r} + \sum_{i=1,i\neq j}^{j} E_{\nu f,k,r,i}me^{-ud_{ij}} - \sum_{i=1,i\neq j}^{j} E_{\nu f,k,r,i}me^{-ud_{ij}} \\ & \frac{dE_{\nu f,k,r,i}}{dt} & = & n\delta_u E_{\nu f,k-1,r} - n\delta_u E_{\nu f,k,r} - \zeta(\nu_{R,r}R + \nu_{C,r}C_r) E_{\nu f,k,r} + \sum_{i=1,i\neq j}^{j} E_{\nu f,k,r,i}me^{-ud_{ij}} - \sum_{i=1,i\neq j}^{j} E_{\nu f,k,r,i}me^{-ud_{ij}} \\ & \frac{dE_{\nu f,k,r,i}}{dt} & = & n\delta_u E_{\nu f,k-1,r} - n\delta_u E_{\nu f,k,r} - \zeta(\nu_{R,r}R + \nu_{C,r}C_r) E_{\nu f,k,r} + \sum_{i=1,i\neq j}^{j} E_{\nu f,k,r,i}me^{-ud_{ij}} \\ & \frac{dE_{\nu f,k,r,i}}{dt} & = & n\delta_u E_{\nu f,k-1,r} - n\delta_u E_{\nu f,k,r} - \zeta(\nu_{R,r}R + \nu_{C,r}C_r) E_{\nu f,k,r} + \sum_{i=1,i\neq j}^{j} E_{\nu f,k,r,i}me^{-ud_{ij}} \\ & \frac{dE_{\nu f,k,r,i}}{dt} & \frac{dE_{\nu f,k,r,$$

$$\frac{dE_{v,1,\tau,i}}{dt} = n\delta_v E_{vf,n_1,\tau} - n\delta_v E_{v,1,\tau}$$

virus exposed class w

 $\frac{dE_{v,w,\tau,i}}{dt} \quad = \quad n\delta_v E_{v,w-1,\tau,i} - n\delta_v E_{v,w,\tau,i} \label{eq:energy}$

infectious fungal conidia

 $\frac{dC_{\tau,i}}{dt} = (1 - X_{R,\tau})m\delta_f E_{f,m,\tau,i} - \mu_{C,\tau}C_{\tau,i}$

infectious viral particles

$$\frac{dP_{\tau,i}}{dt} = n\delta_v E_{v,n,\tau,i} - \mu_v P_{\tau,i}$$

We modeled larval dispersal in an open system using an exponential dispersal kernel. This is based on the following assumptions: **Biological**

- Larvae disperse only during the first week of the epizootic, when they are small enough to balloon.
- Virus-infected and non-infected larvae are equally likely to disperse.
- 3. Larvae are equally likely to disperse in any direction.

Mathematical

1. All larvae disperse according to the full dispersal kernel over radius r and angle θ according to the function:

$$g(r, \theta) = \frac{1}{2\pi} ae^{-ar}$$

2. Angle and distance traveled are independent. Thus, the expected distance traveled is $\frac{1}{2}$

3. Dispersal from the center point of the subpopulation results in traveling a distance less than r, thus remaining in the same subpopulation, with probability p. Thus larvae that leave a subpopulation,

$$p = \frac{1}{2\pi} \int_0^{2\pi} \int_0^{6\pi} a e^{-ar} dr d\theta$$

$$= \frac{1}{2\pi} \int_0^{2\pi} \left(-e^{-ar} \Big|_0^{\hat{r}} \right) d\theta$$

$$= \frac{1}{2\pi} \int_0^{2\pi} \left(1 - e^{-a\hat{r}} \right) d\theta$$

$$p = \frac{1}{2\pi} \int_0^{2\pi} \left(1 - e^{-a\hat{r}} \right) d\theta$$

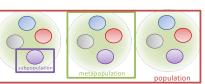
 $1-p=-e^{-ar}$ Shat $=-e^{-ar}$

4. Of those that disperse out of the subpopulation, 1-p, arrival at another subpopulation is moderated by the distance between both subpopulations, d_{ij} , and a term m.

METAPOPULATION

subpopulation

Results



We built competing versions of this model that vary in how they fit the dispersal parameters, a and m.

These models range from fitting a separate a and m for each subpopulation (12), metapopulation (3), and population (1). Identifying which model fits the data best will help us

understand the scale of dispersal that is important for disease dynamics.

Model Fit

We will use our estimates of dispersal in a long-term model to predict future population and disease dynamics of the spongy moth-fungus-virus system.

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