

Dispersal and spatial heterogeneity promote pathogen coexistence

Sophia Horigan¹, Jiawei Liu^{1,2}, Will Koval¹, Greg Dwyer¹

¹Department of Ecology and Evolution, University of Chicago, ²Purdue University

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.

Virus-killed
spongy moth



Fungus-killed
spongy moth

FIELD WORK

We created whole-tree artificial epizootics to test infection dynamics under four treatments: **virus-only**, **fungus-only**, **fungus+virus**, and a **control**.



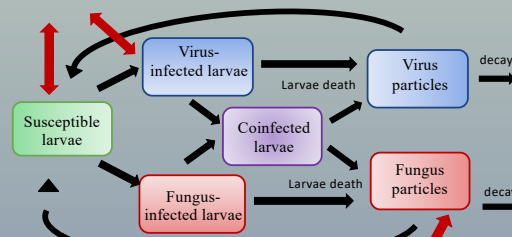
	virus-only	fungus-only	fungus+virus	control
uninfected insects released	X	X	X	X
virus-infected insects released	X		X	
fungus soil transplant/watering		X	X	

3 replicates

MODELING

Motivated by our field data, we built a metapopulation SEIR model of spongy moth-fungus-virus dynamics that allows for dispersal and pathogen heterogeneity across subpopulations.

Larval and virus dispersal: daily movement during first 7 days when larvae "balloon" on silk – some are virus-infected.

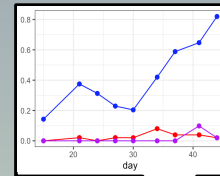


Fungal dispersal: infectious "conidia" are transported on the wind throughout the epizootic.

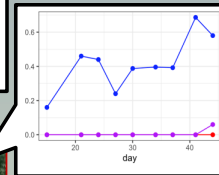
Infection data from the field experiments and natural populations demonstrated

1) high spatial heterogeneity in fungus infection, and 2) low to intermediate fungus and larval dispersal. Both appear to promote coexistence.

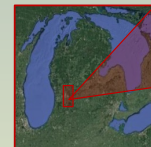
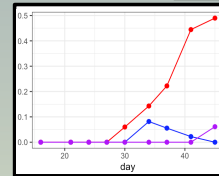
The one natural epizootic dominated by the **fungus** was 10 km from the two epizootics dominated by the **virus**.



Two natural epizootics dominated by the **virus** were close together.



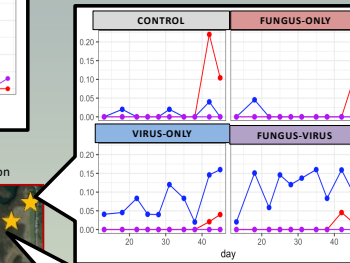
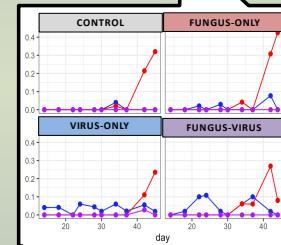
Coinfections were extremely rare in natural and experimental populations, despite coexistence.



Michigan, United States

Fungus infections in non-fungus experimental plots could come from either or both of:

1. infectious conidia dispersing on the wind,
2. overwintering resting spores in the soil



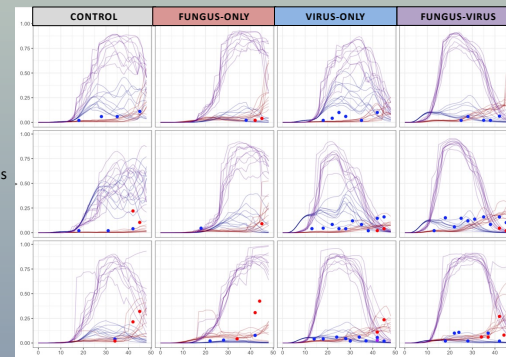
Virus infection rates were roughly similar between sub-pops, suggesting high dispersal of virus-infected larvae, but **fungus** infection rates varied greatly between sub-pops: Differences in spore dispersal on wind? Differences in overwintering spores?

Virus infections in non-virus experimental plots suggest that virus infected larvae dispersed hundreds of meters, but not kilometers.

We are currently fitting the model to the field data to
1) determine the relative importance of fungus/larval dispersal, and
2) disentangle dispersal from initial host and pathogen densities.

lines
stochastic
model
simulations

points
data



metapop 1

metapop 2

metapop 3

So far, the model fit is not great...

Our theories as to why:

- 1) incorrect models of dispersal
- 2) coinfection too frequent in model
- 3) too restrictive parameter bounds

What are your suggestions?
Send me an email!



Our work is supported by NSF OPUS award DEB-2043796 from the US National Science Foundation.

Acknowledgements: Caroline Chael, Dr. Alison Hunter, Katie Dixon, Dr. Cara Brook, Alex Koch, Kellogg Biological Station