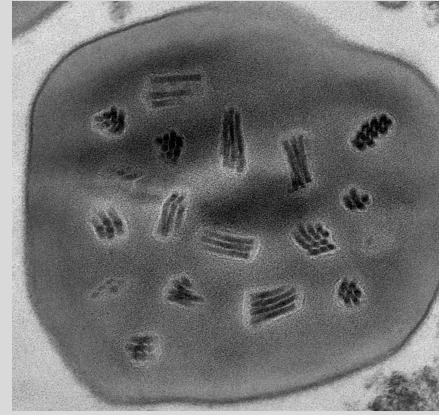
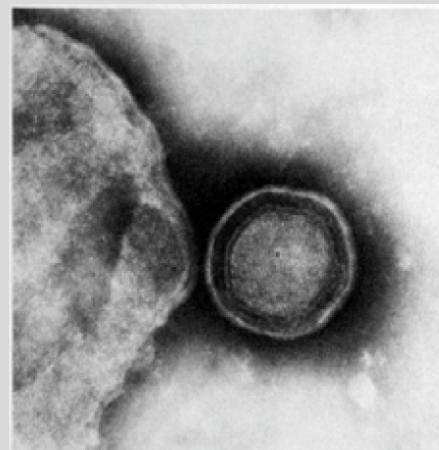


Integrating experiments, field data, and models to understand infectious disease ecology

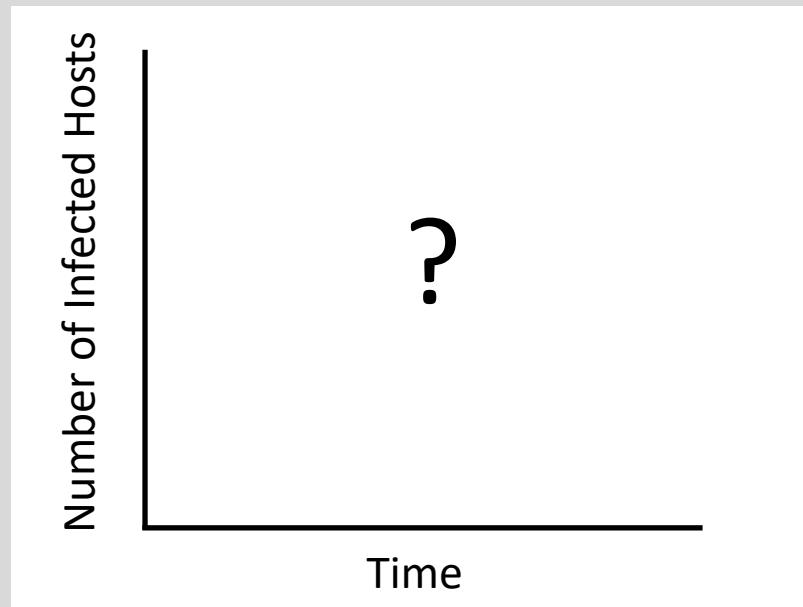


Joe Mihaljevic
Asst. Professor, SICCS
Room 213
INF501 - September 7, 2018

Models of disease transmission

Basic question:

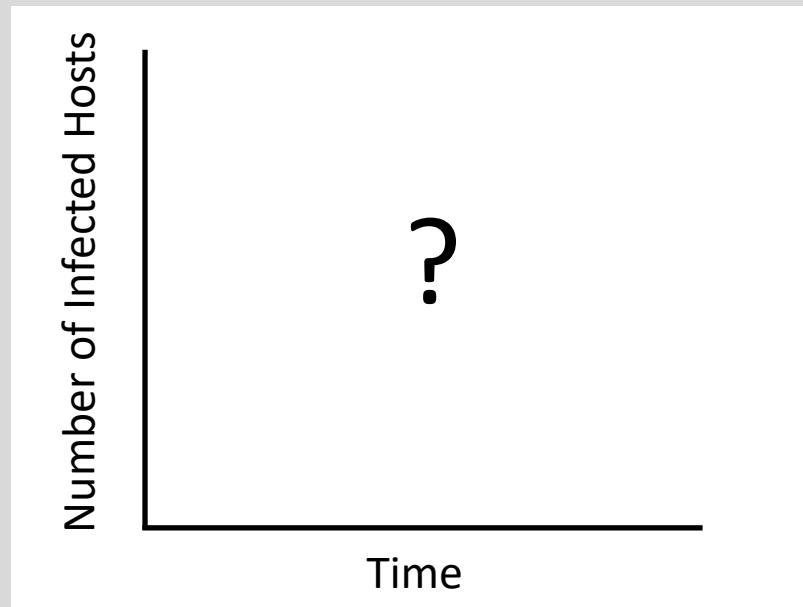
How does the number of infected hosts change over time in a population?



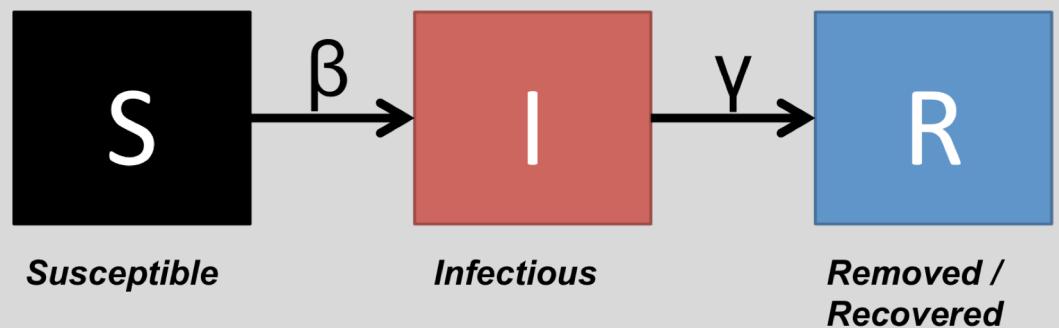
Models of disease transmission

Basic questions:

- Can we accurately represent these dynamics mathematically?
- If so, can we predict future disease spread?
- If so, can we use the models to develop disease control strategies?



The SIR Model

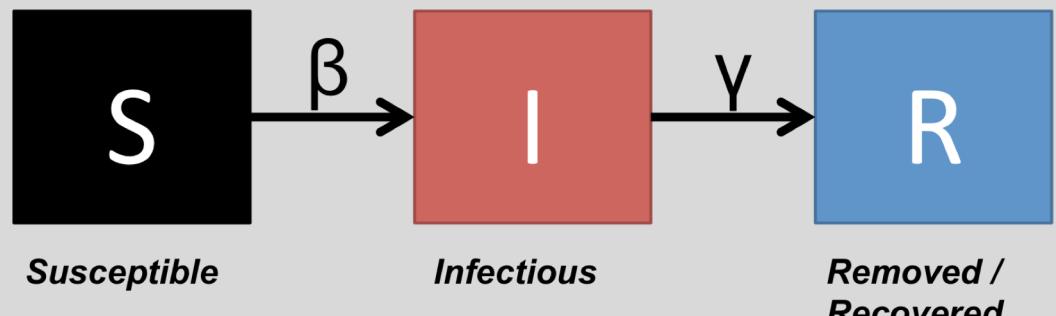


The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$



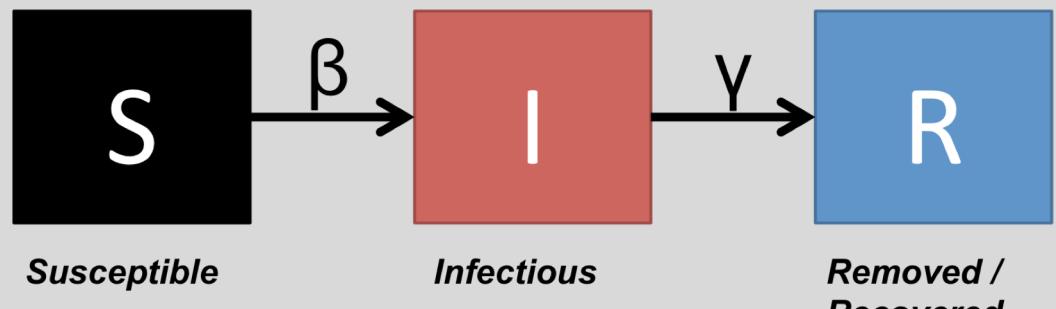
The SIR Model

$$\frac{dS}{dt} = -\boxed{\beta SI}$$

Transmission rate

$$\frac{dI}{dt} = \beta SI - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

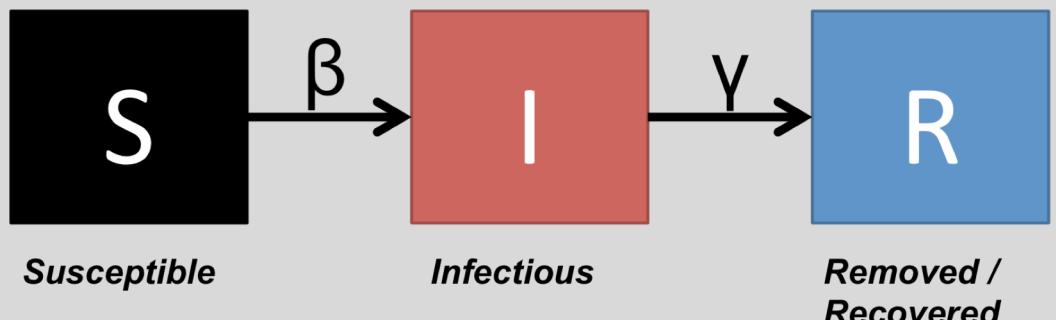


The SIR Model

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \boxed{\gamma I}$$

Removal rate

$$\frac{dR}{dt} = \gamma I$$



The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

The average time that a host is infectious:

$$\frac{dI}{dt} = \beta SI - \gamma I$$

$$1/\gamma$$

Example:

- Influenza : ~2.5 days

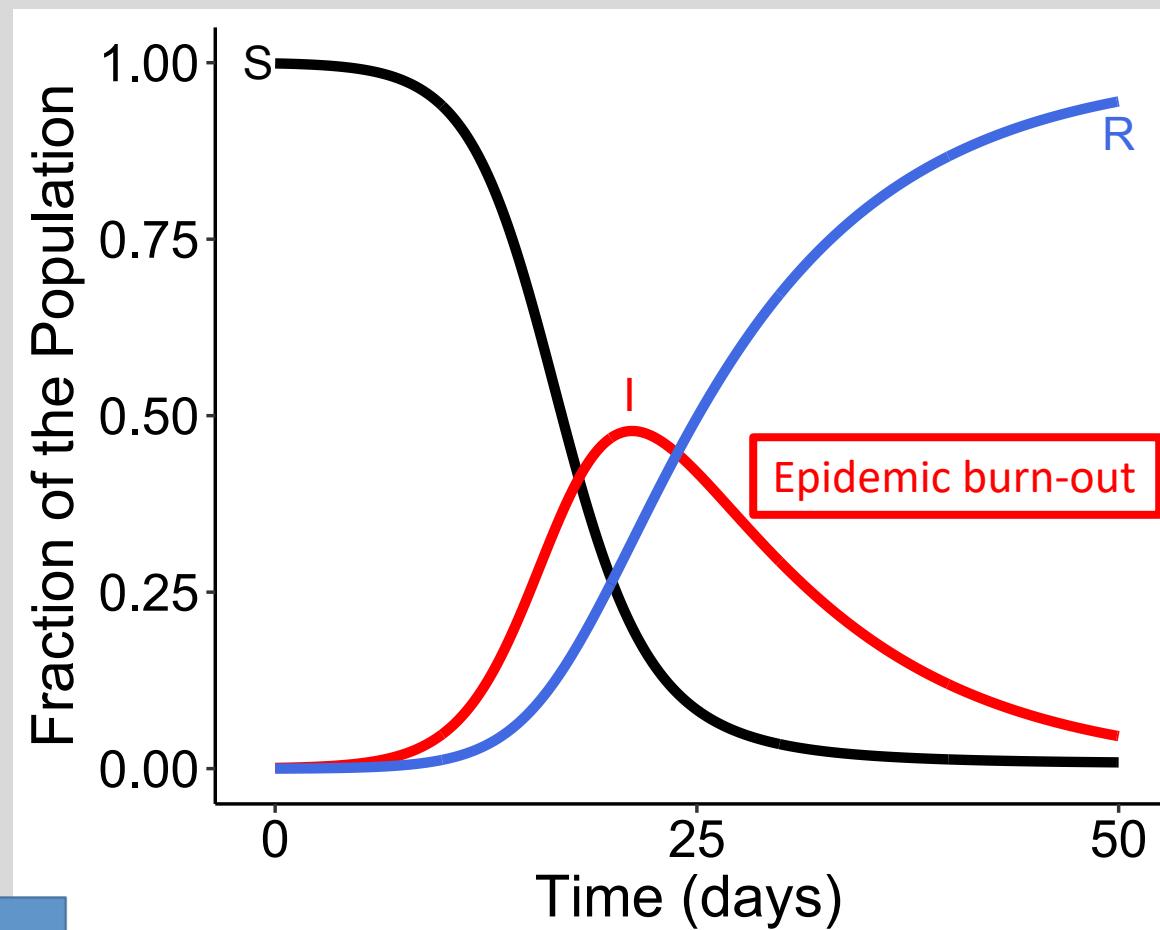
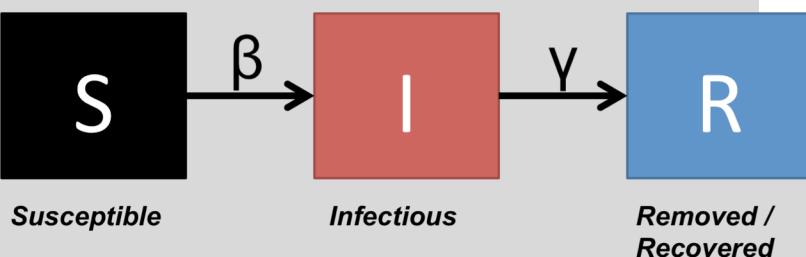
$$\frac{dR}{dt} = \gamma I$$

The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

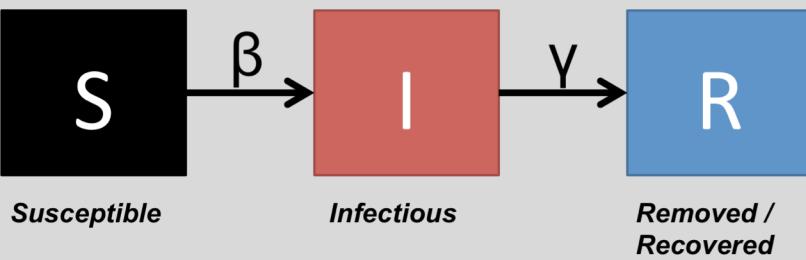
$$\frac{dI}{dt} = \beta SI - \gamma I$$

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The SIR Model

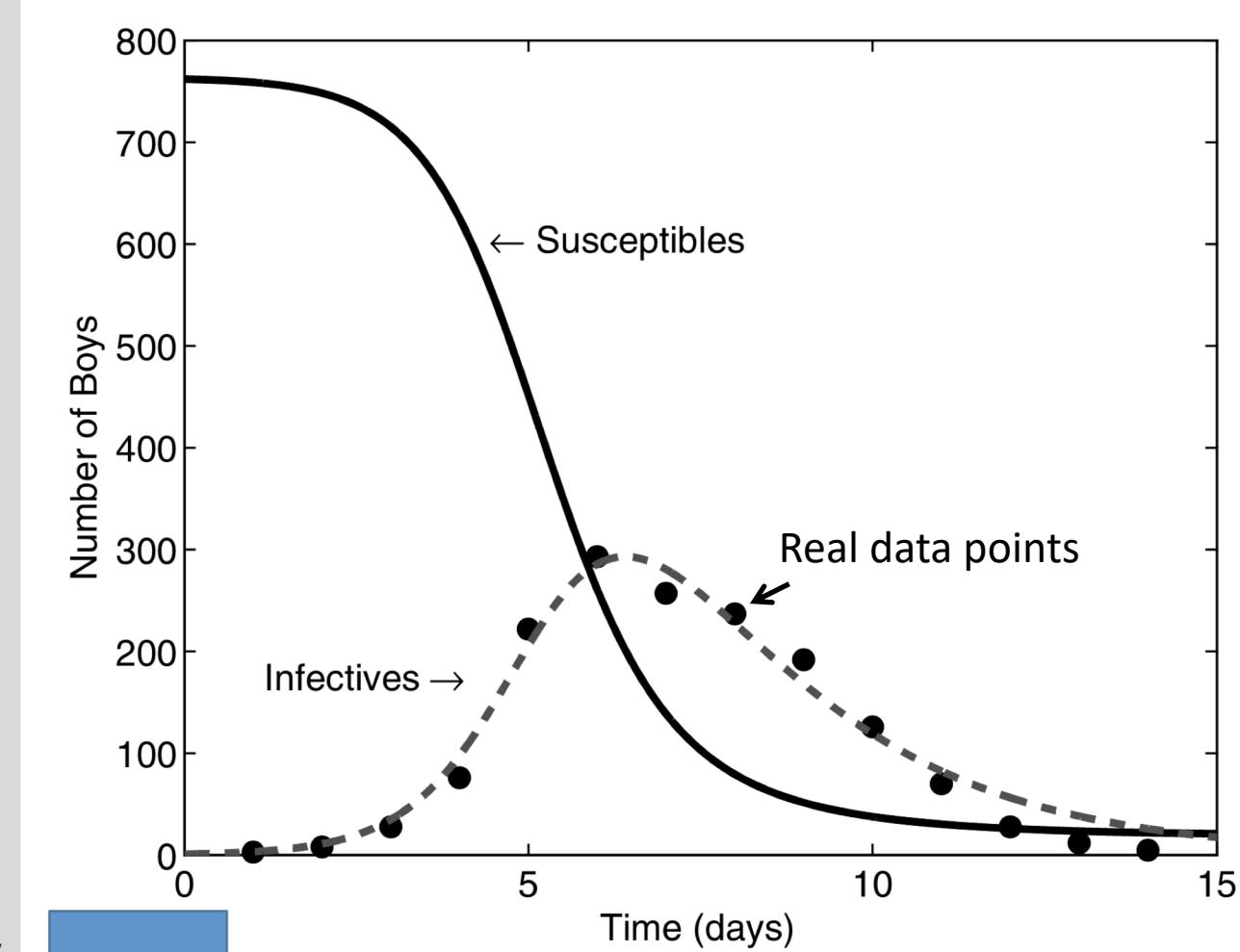
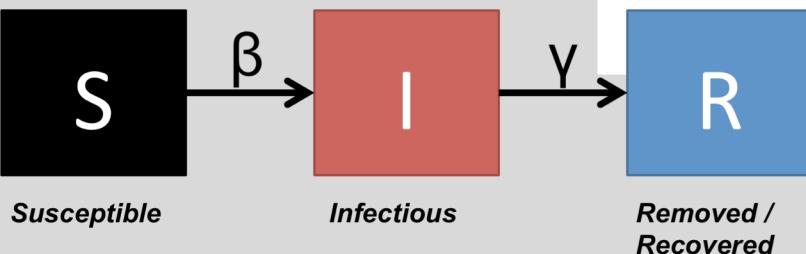
Does it work?



The SIR Model

Does it work?

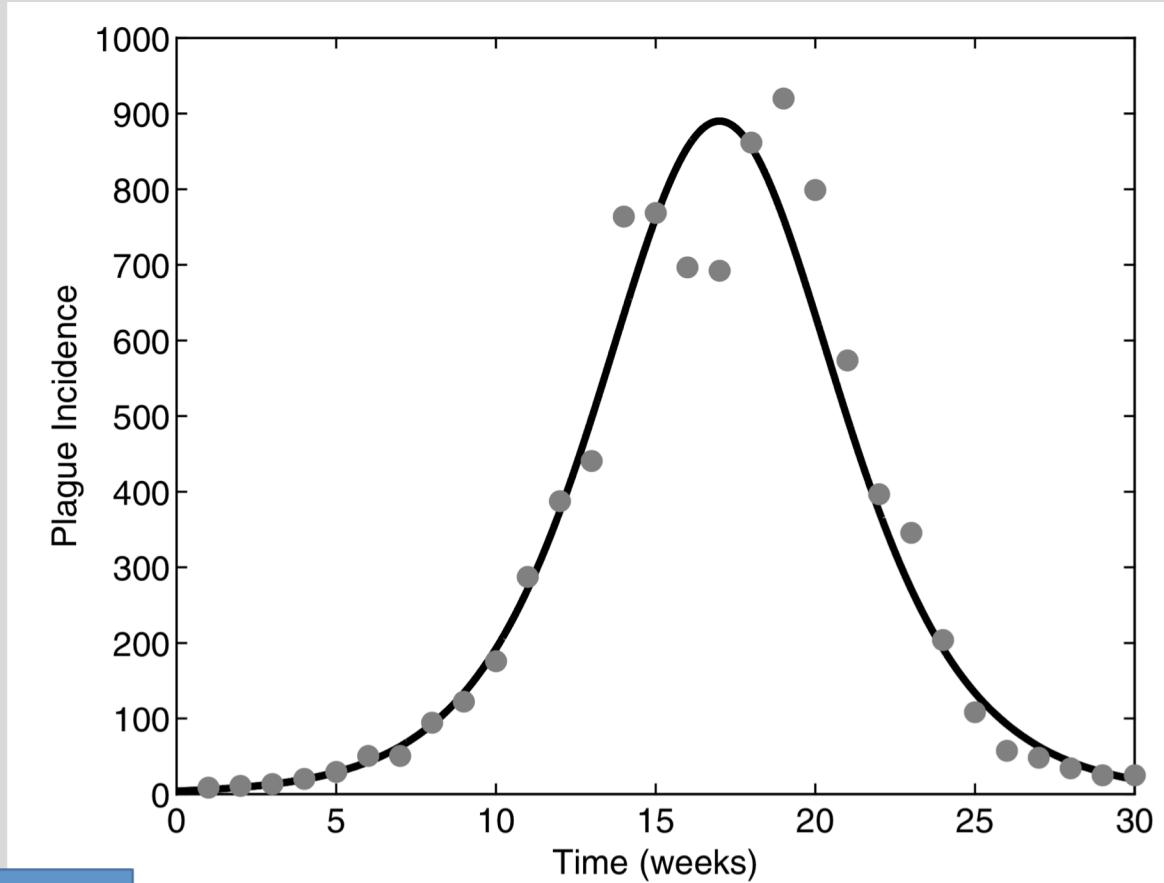
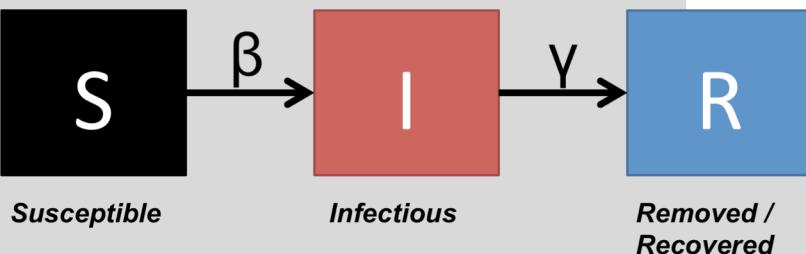
Boys with influenza
English boarding school
1978



The SIR Model

Does it work?

Plague in Bombay
1905-1906



Keeling & Rohani 2008

Mathematical Generalities

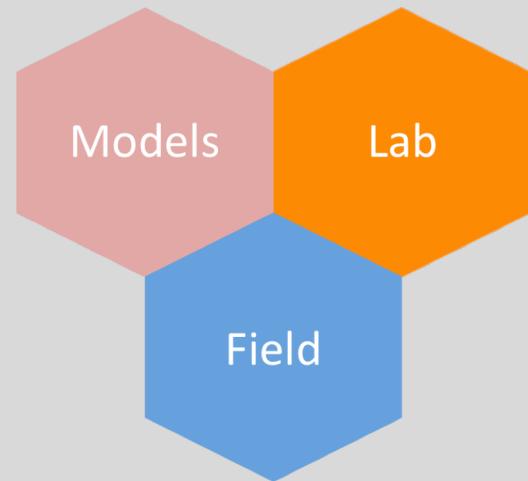
Pathogen Reproductive Ratio:

$$R_0 = \frac{\beta}{\gamma} = \frac{\text{Growth of Infection}}{\text{Loss of Infection}}$$

If $R_0 < 1$, the pathogen cannot invade

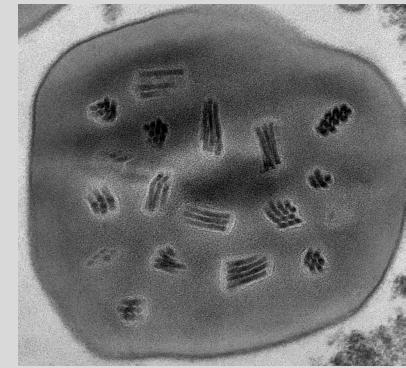
How can we use models to test hypotheses?

- Build models based on hypotheses:
 - Which **processes** are important for a system?
- Test the models:
 - Parameterize with experiments (lab, field)
 - Fit the model to observational data (lab, field)
- Use the best models to make forecasts



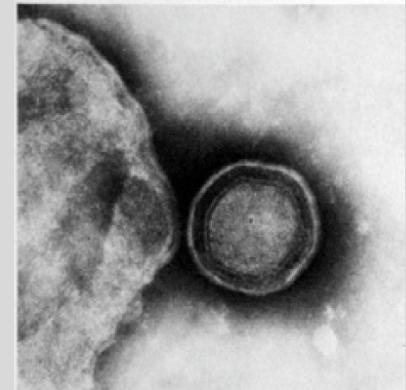
Part I:

Douglas-fir tussock moth
nuclear polyhedrosis viruses, NPV
(stochastic DEs / nonlinear fitting)
(implications for biocontrol)



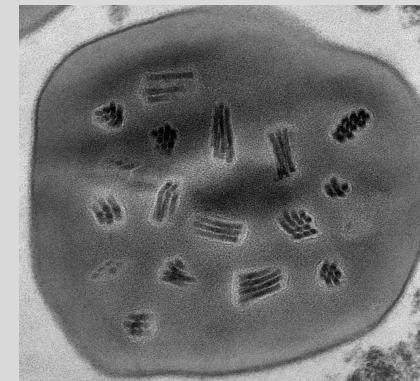
Part II:

Amphibian ranaviruses
(using historical data)
(effects of temperature on epizootics)

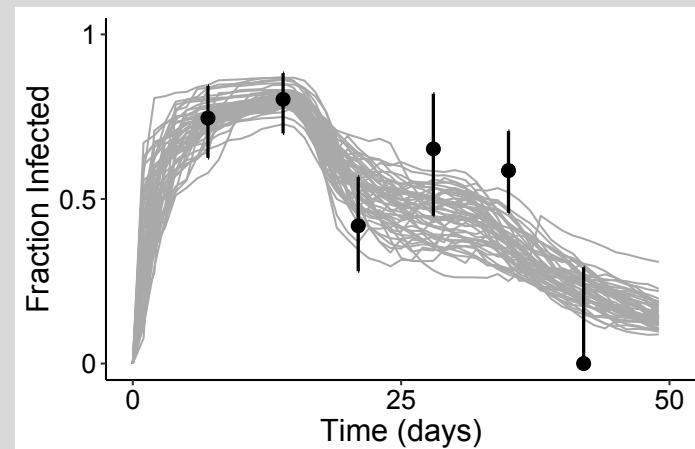
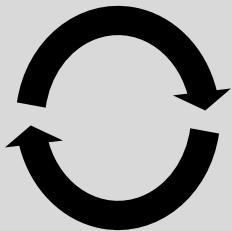


Part I:

Douglas-fir tussock moth
nuclear polyhedrosis virus
(NPV)



$$\frac{dS}{dt} = -\nu SP$$



Collaborators:

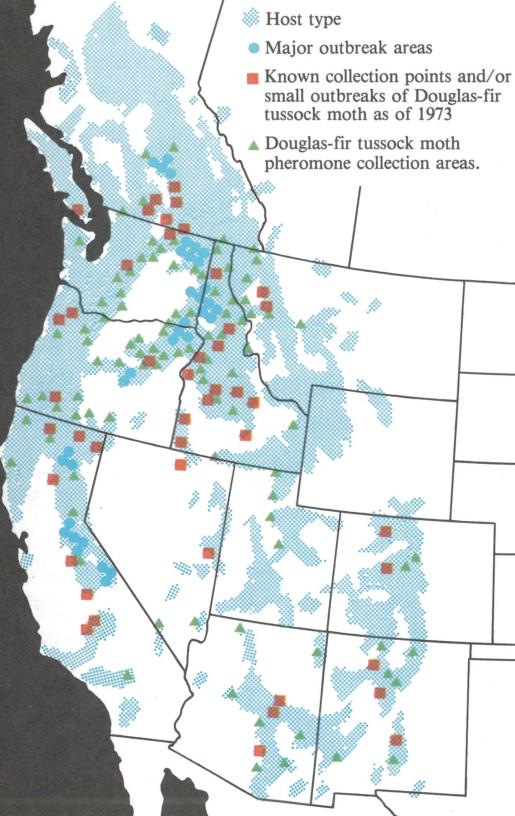
Karl Polivka (USFS)

Connie Mehmel (USFS)

Chentong Li (Univ. Chicago)

Vanja Dukic (Univ. Colorado)

Greg Dwyer (Univ. Chicago)

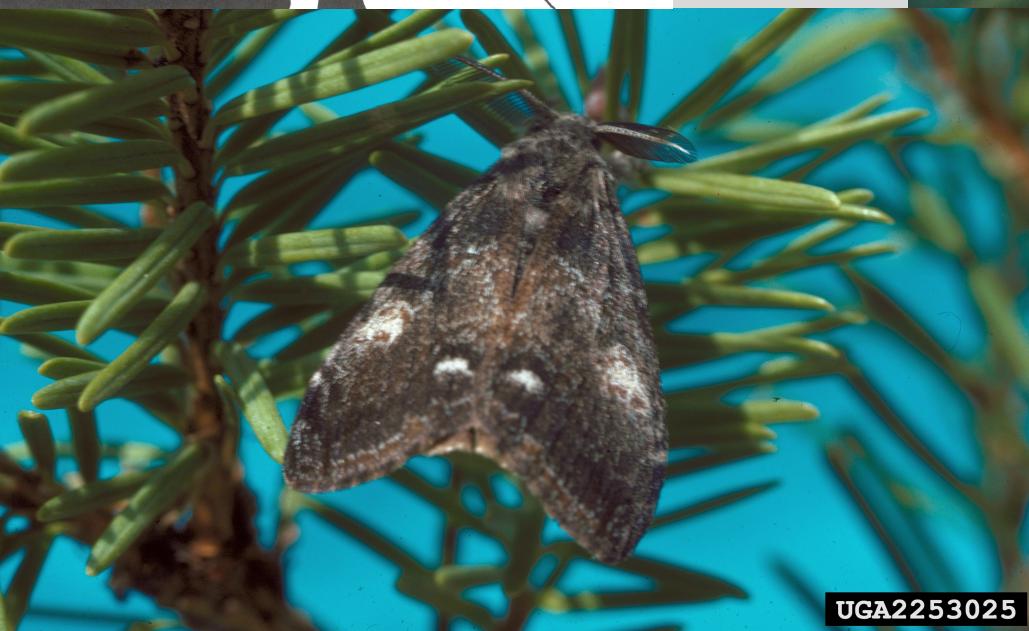


Douglas-fir tussock moth

Orgyia pseudotsugata



UGA2254043



UGA2253025



Dewey, USFS



Massive outbreaks

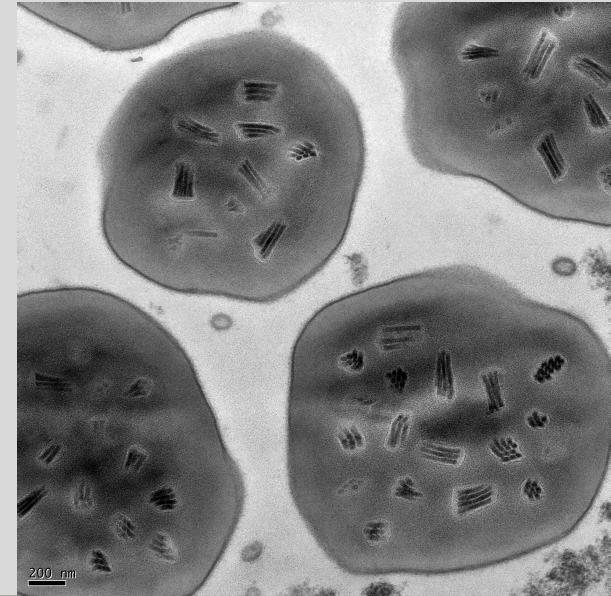
- Idaho:
 - 447,000 acres (1944 - 1947)
 - 225,000 acres (1961 - 1965)
 - 110,000 acres (1971 - 1974)
- Oregon:
 - 438,000 acres (1971 - 1974)
- Washington:
 - 251,000 acres (1971 - 1974)

OpNPV

Orgyia pseudotsugata nuclear polyhedrosis virus



Baculoviridae
Nucleopolyhedrovirus



TM Biocontrol-1

TMB-1: first virus registered in the US for use against a forest insect.

Operational use:

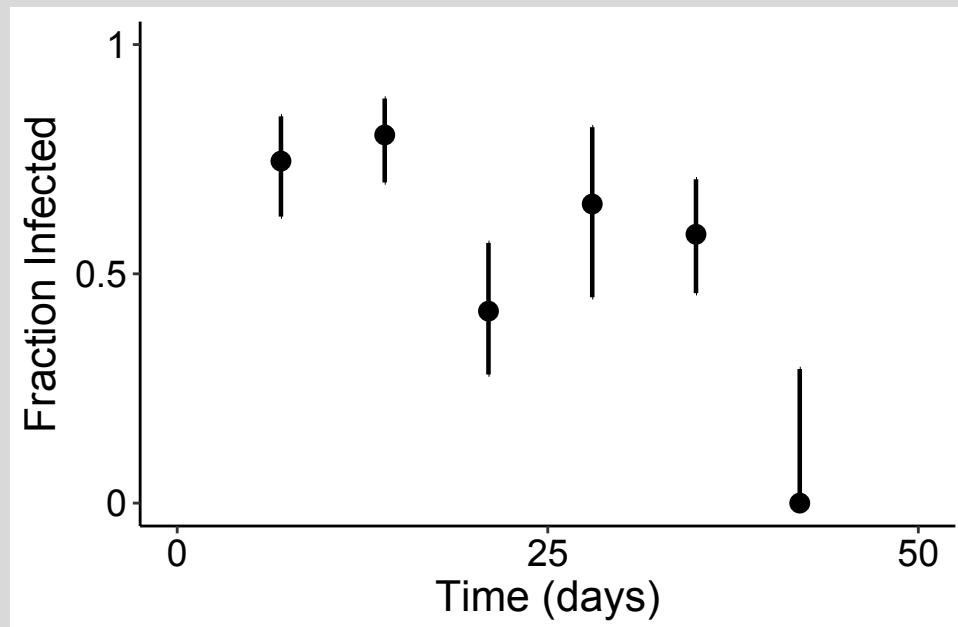
- 2000 (Oregon)
- 2001 (Washington)
- 2010 (Washington)



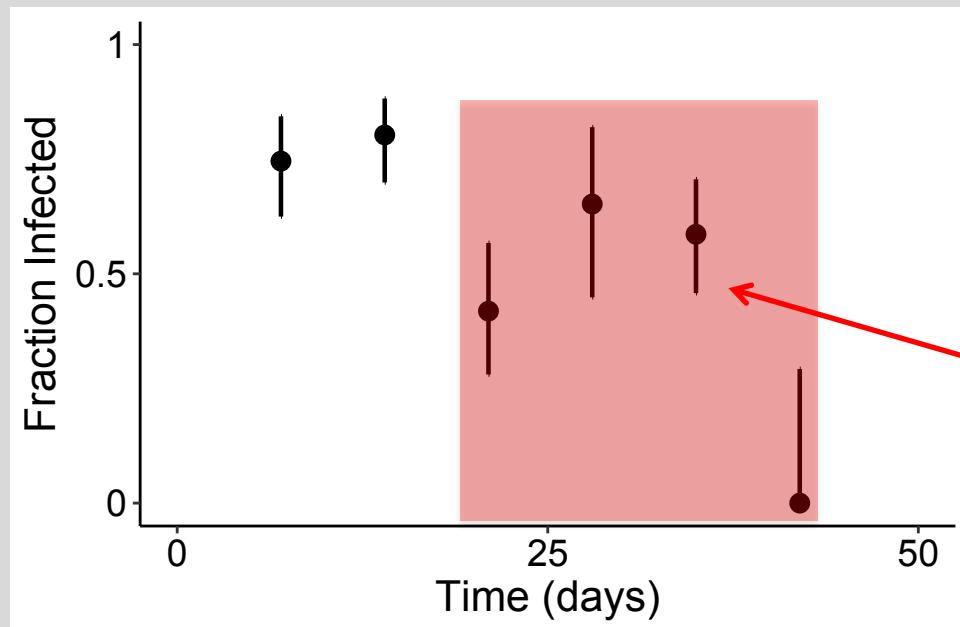
~ 70,000 acres sprayed

> \$5 million spent

The Spray Works

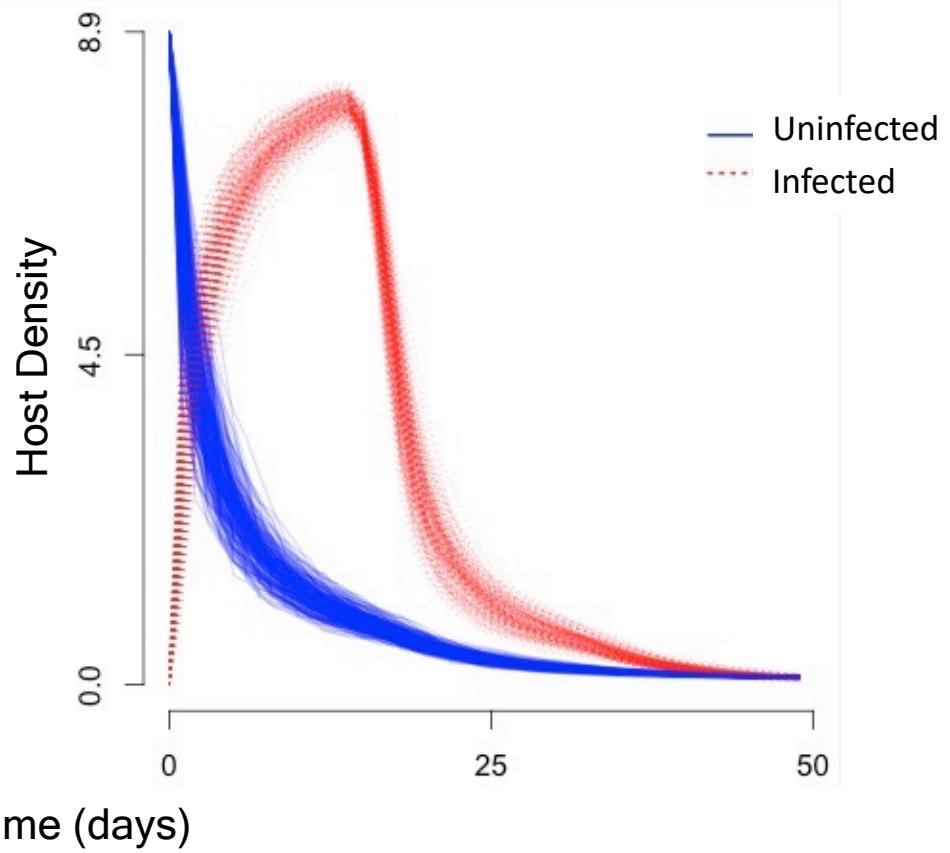
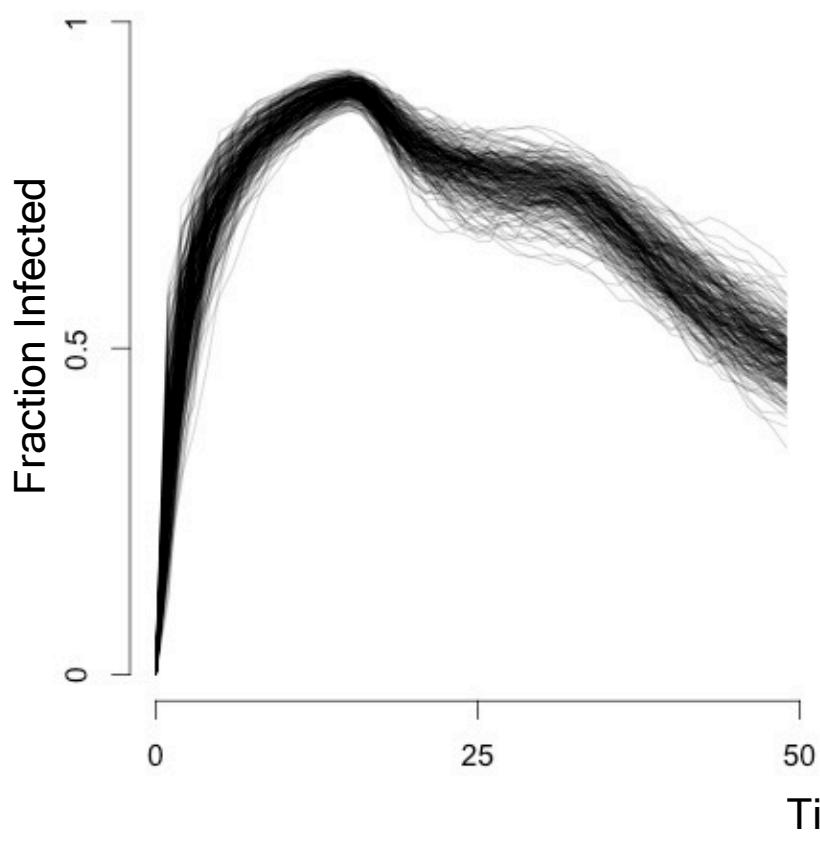


The Spray Works

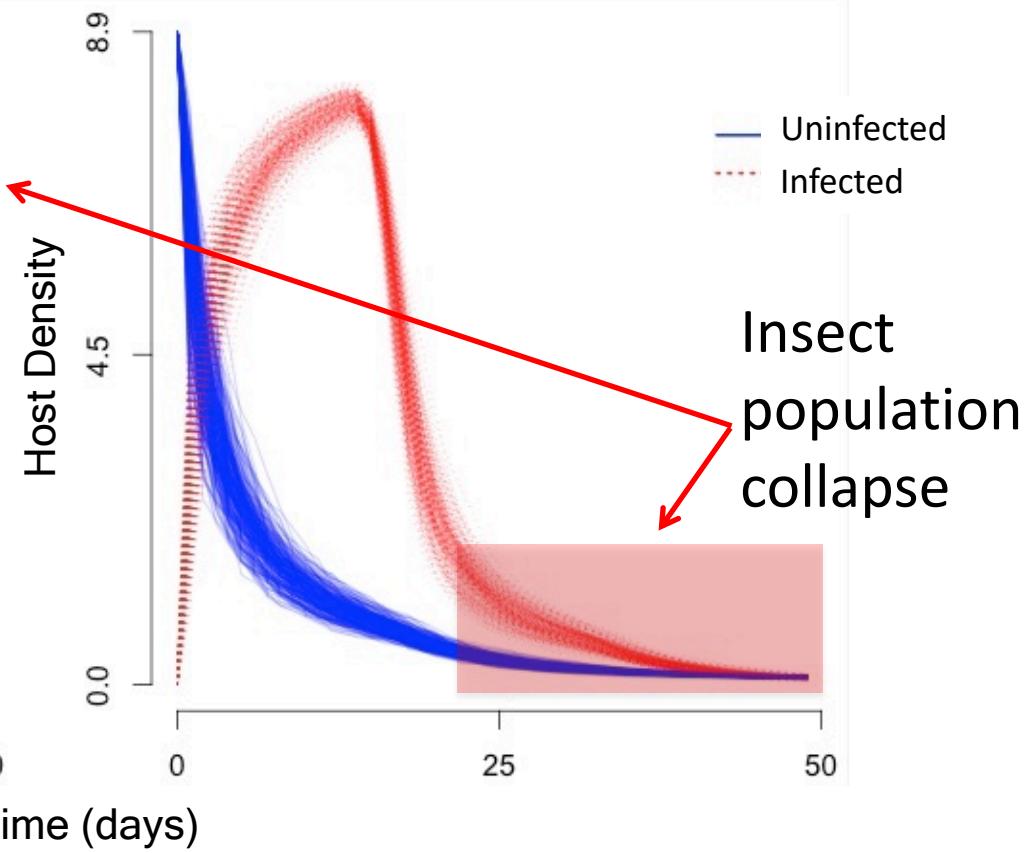
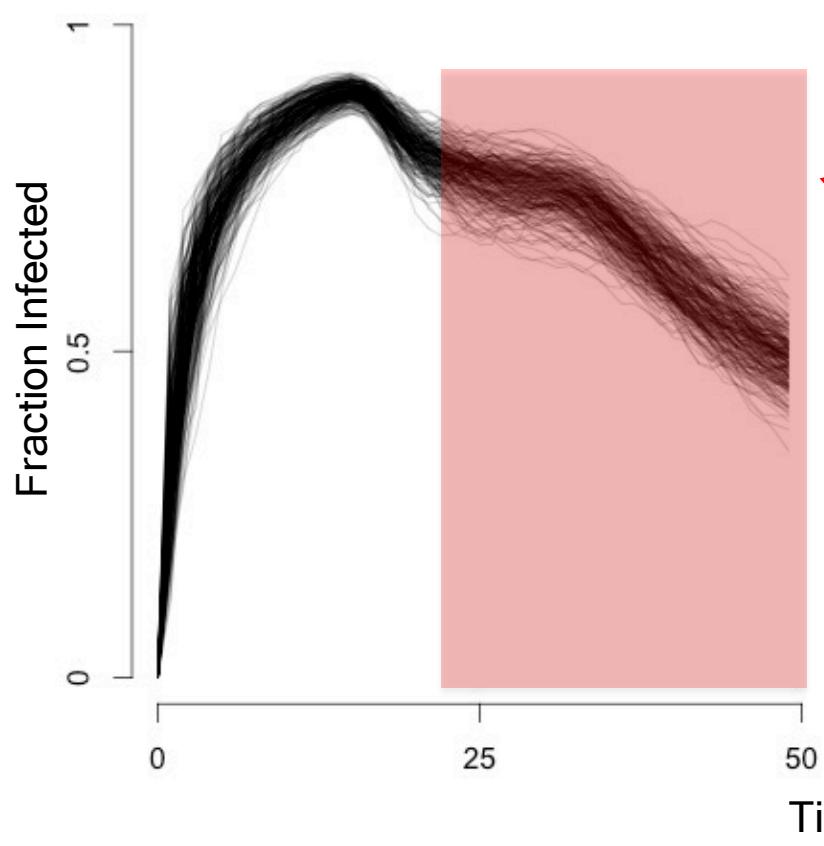


Insect
population
collapse

The Spray Works

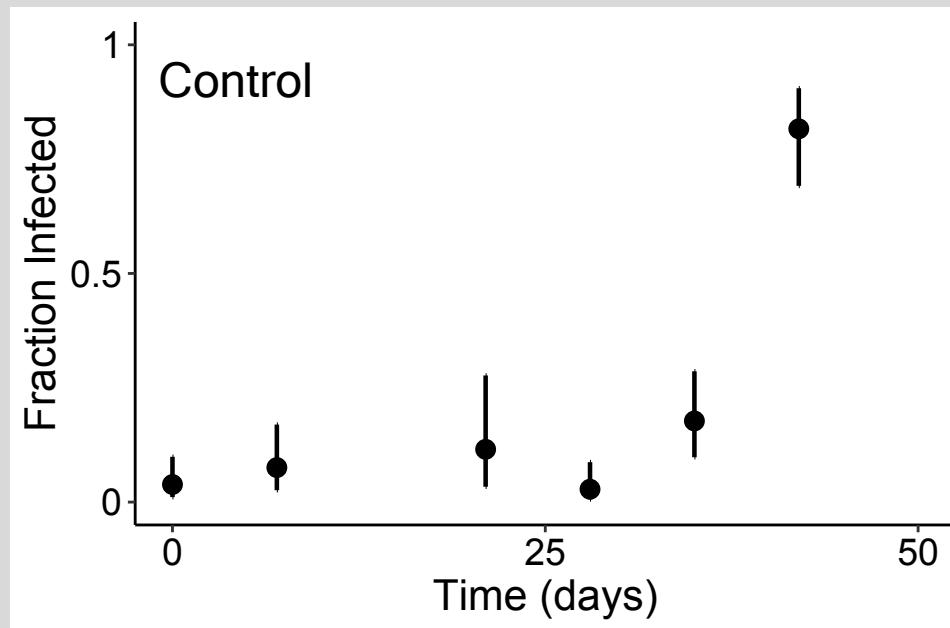


The Spray Works



Unforeseen Problem

- Mortality in the unsprayed (control) plots same as spray plots.



Questions

- Why was mortality in unsprayed plots the same as sprayed plots, *even though control plots had an initially undetectable level of virus infection?*

Applied Questions

- Are there ecological differences between sprayed and naturally occurring variants?

OR

- Do initial conditions (insect density and virus density) and non-linear infection dynamics account for unexpected results?

Basic Questions

- How important is host heterogeneity in susceptibility for explaining natural epizootics?
- How well do our experiments represent dynamics that occur in the field?

Emerging Approach

Integrate experiments, field data, and models



Parameterize w/
Experiments
(Priors)

$$\frac{dS}{dt} = -\bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dE_1}{dt} = \bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1$$

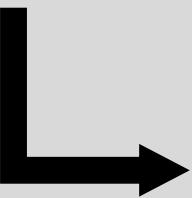
$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i \quad (i = 2, \dots, m)$$

$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

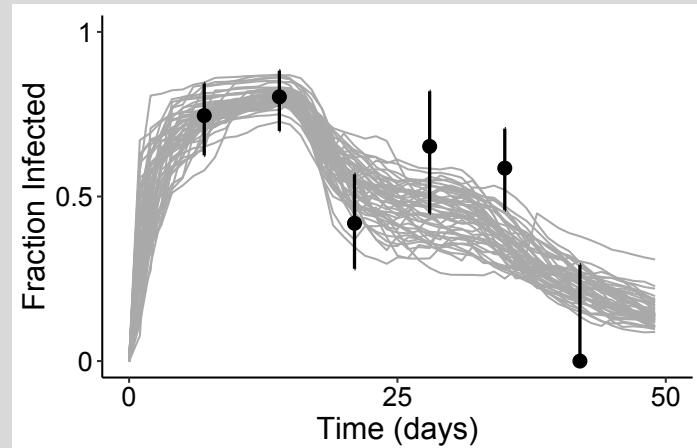
$$\frac{dR}{dt} = \gamma I$$

Build competing models:

- Host heterogeneity in susceptibility
- 1 or 2 distinct transmission rates

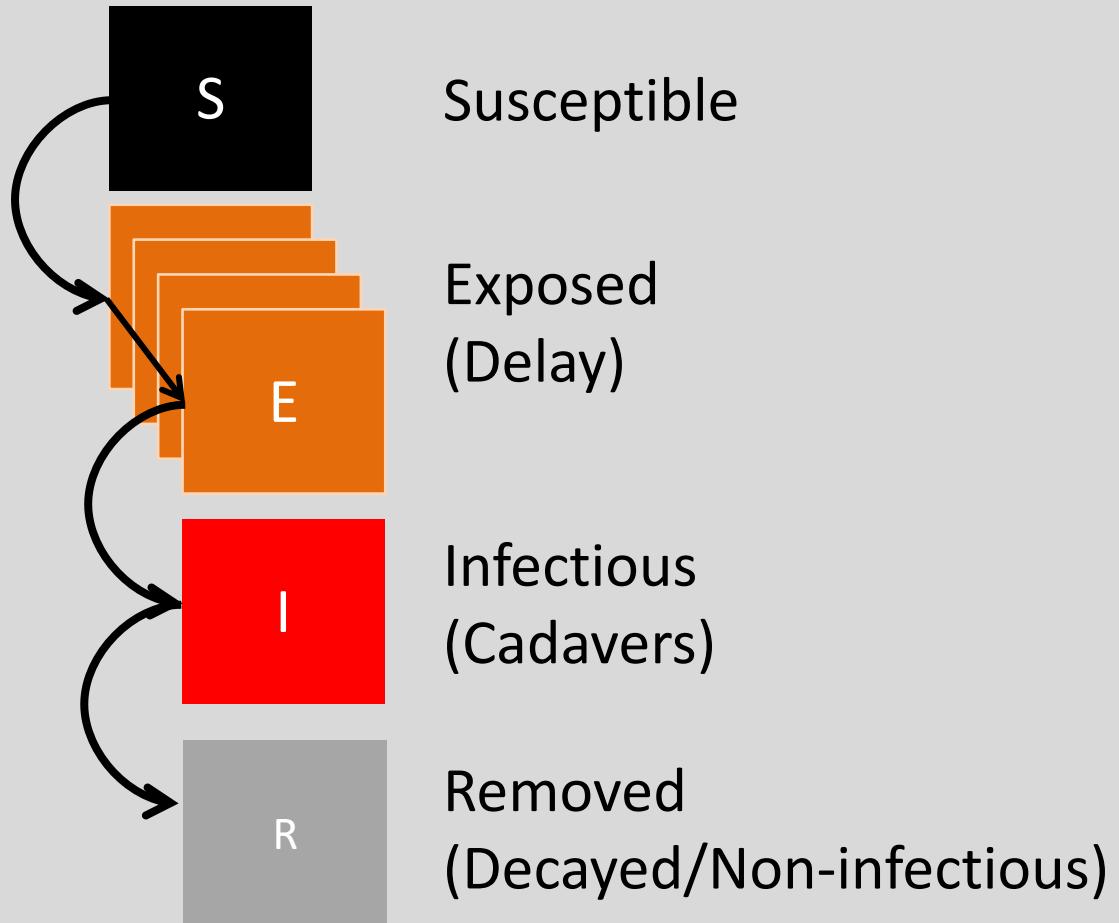


Fit models to field data
(Generate Posteriors)



Prevalence in sprayed (treated) and
non-treated populations

SEIR Model



SEIR Model

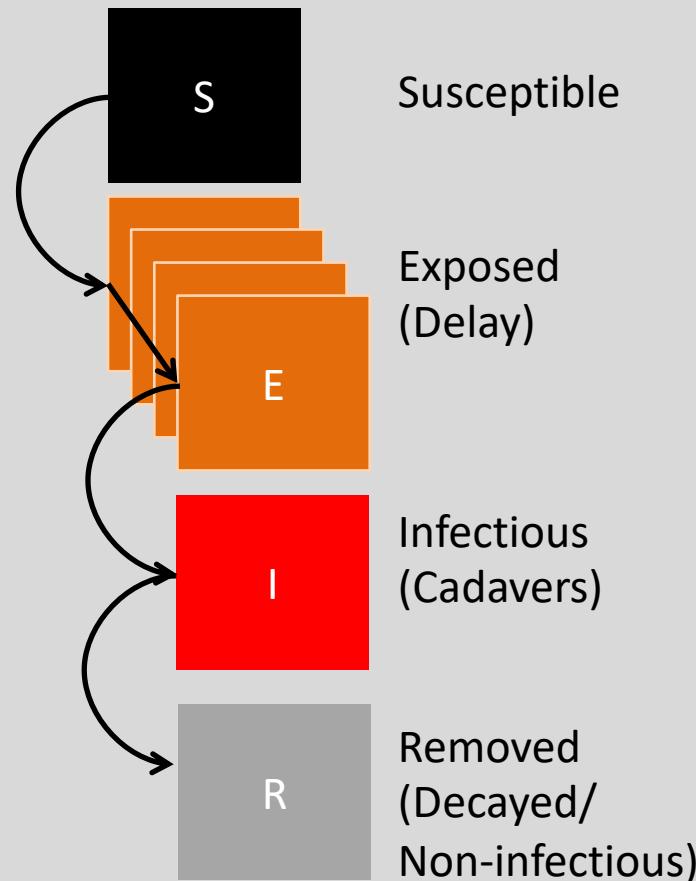
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$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$



SEIR Model

Average transmission rate

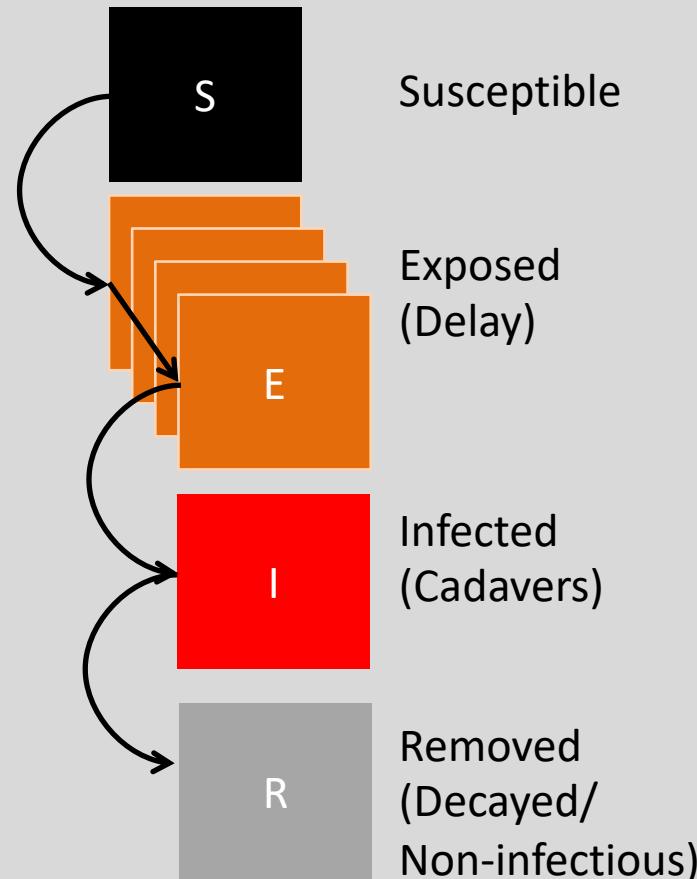
$$\frac{dS}{dt} = -\bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dE_1}{dt} = \bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1$$

$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i \quad (i = 2, \dots, m)$$

$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$



SEIR Model

Daily stochastic variation in transmission

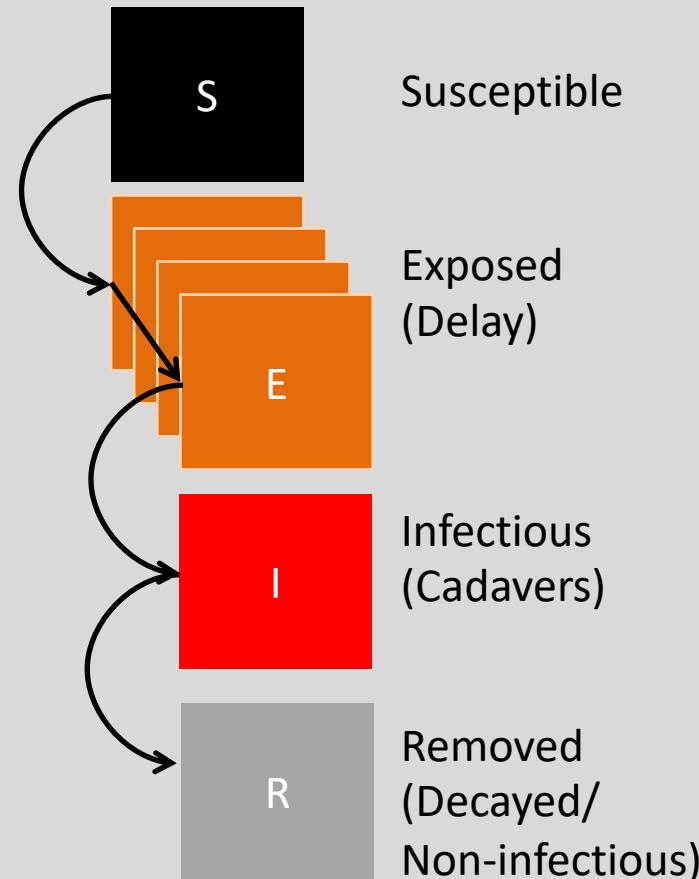
$$\frac{dS}{dt} = -\bar{v}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dE_1}{dt} = \bar{v}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1$$

$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i \quad (i = 2, \dots, m)$$

$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$



SEIR Model

Heterogeneity in host susceptibility

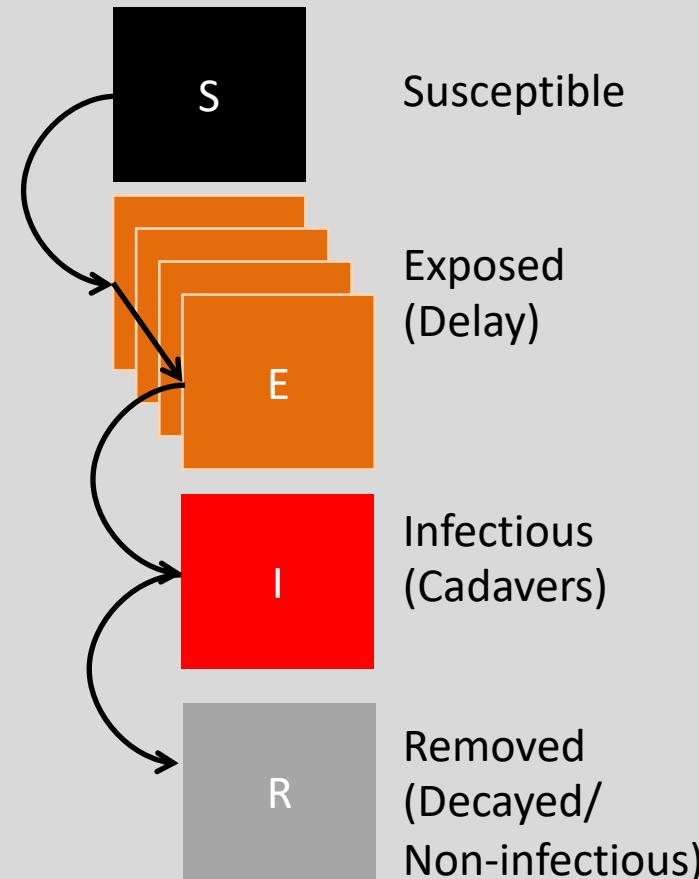
$$\frac{dS}{dt} = -\bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

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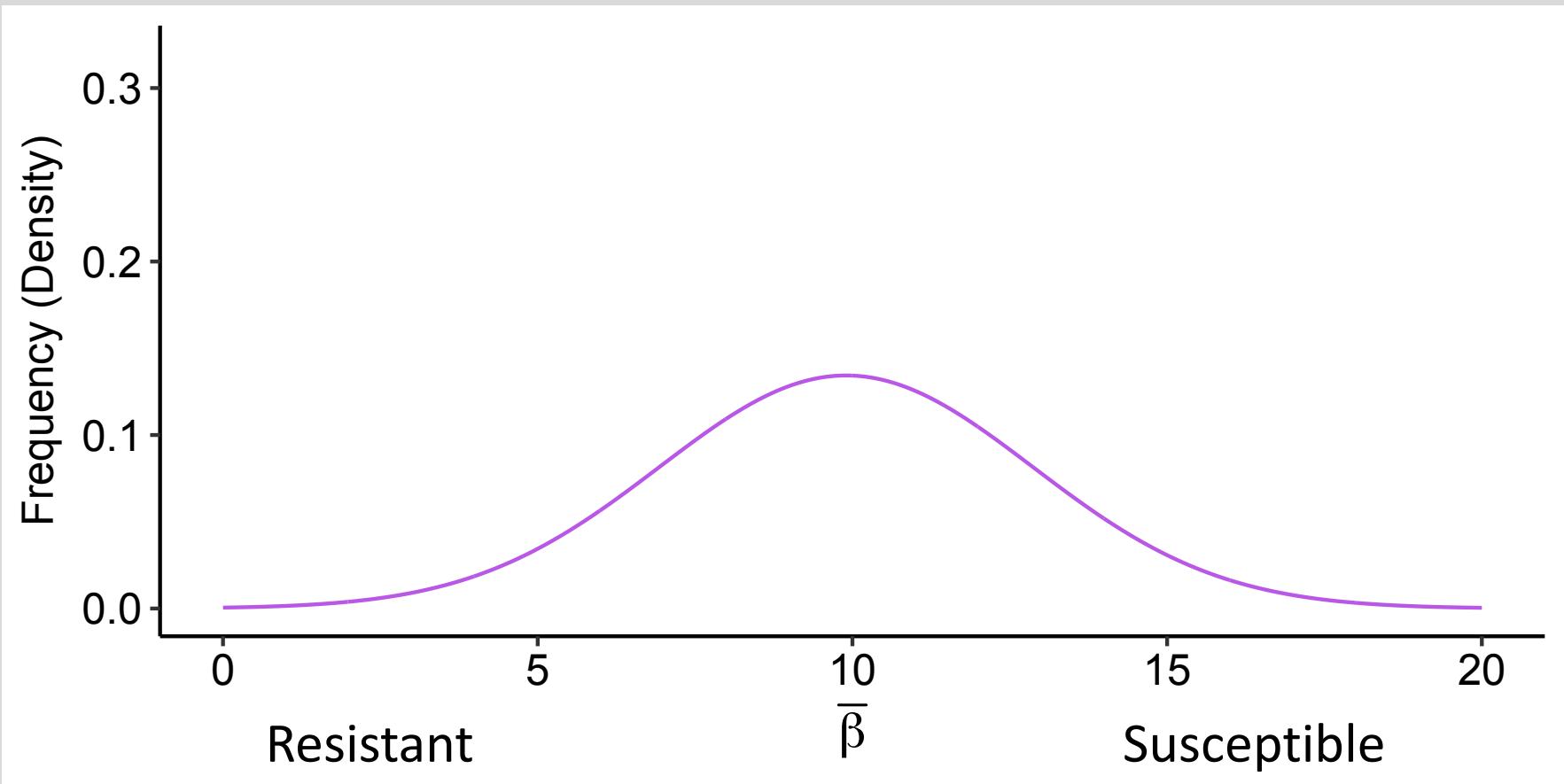
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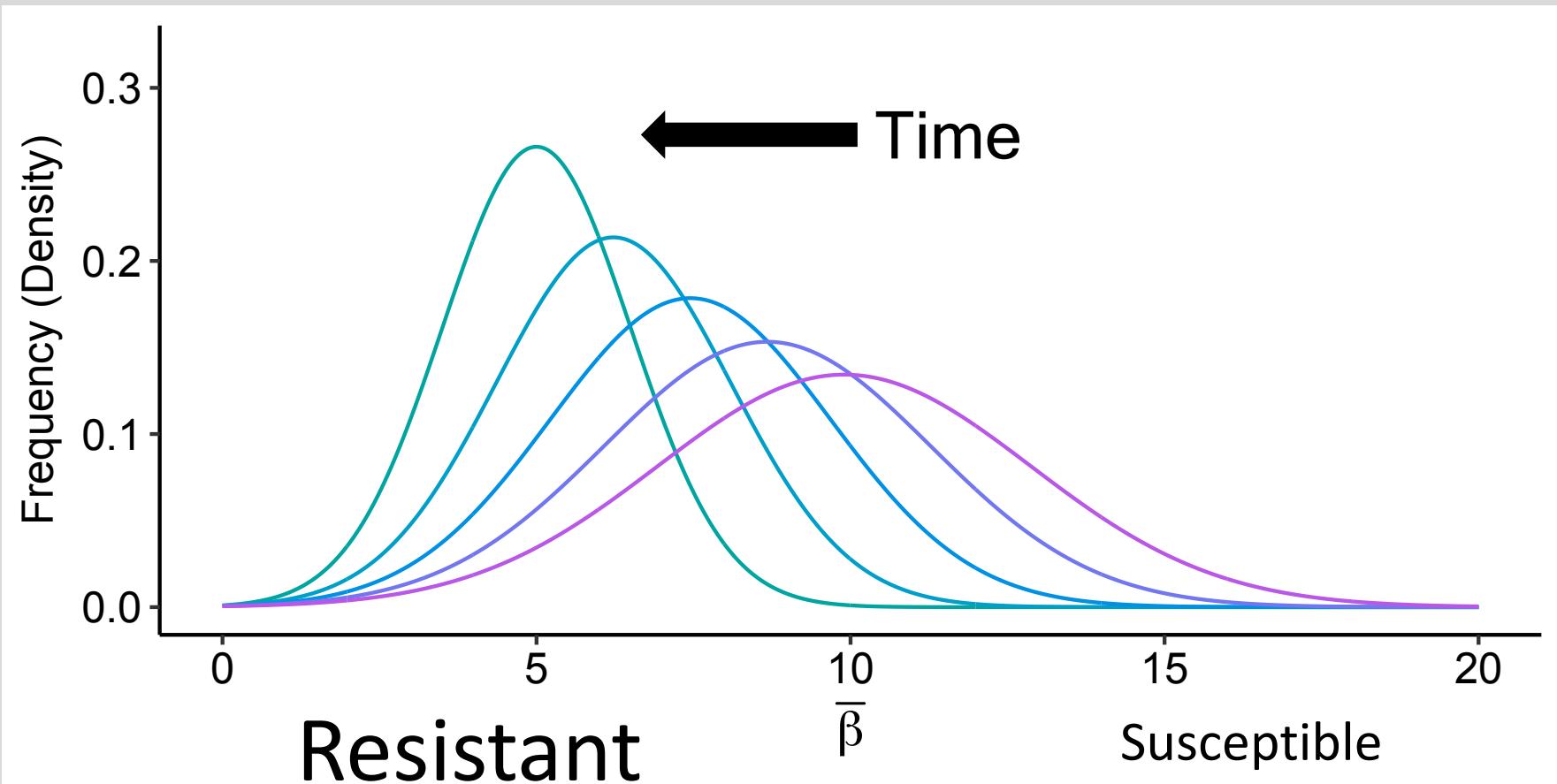


Heterogeneity in Susceptibility



Heterogeneity in Susceptibility

As the epizootic progresses, the more resistant individuals remain



SEIR Model

$$\frac{dS}{dt} = -\bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dE_1}{dt} = \bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1$$

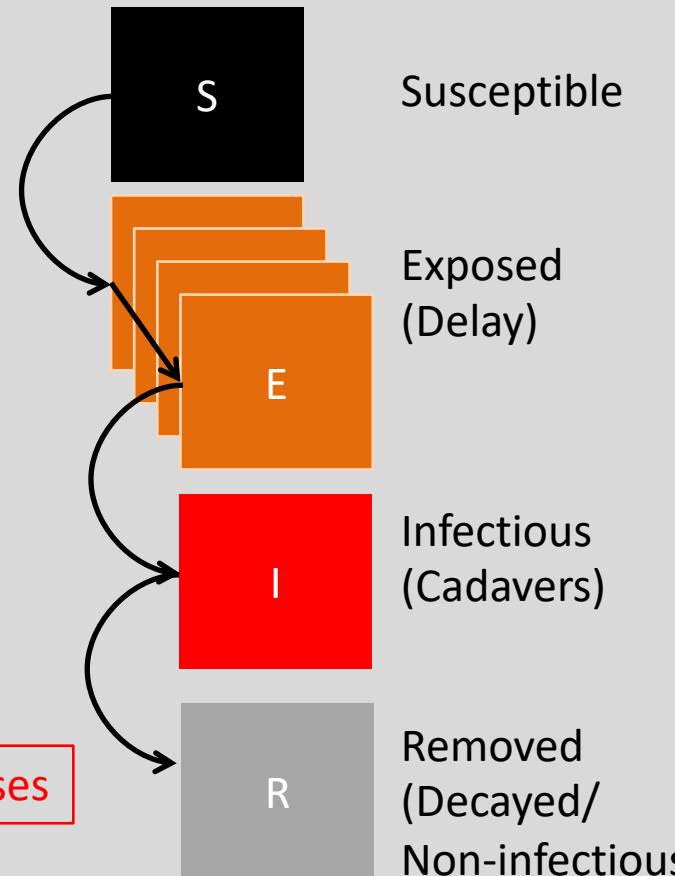
$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i \quad (i = 2, \dots, m)$$

$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

Kill rate

exposed classes



Defines Gamma-distributed speed-of-kill

SEIR Model

$$\frac{dS}{dt} = -\bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

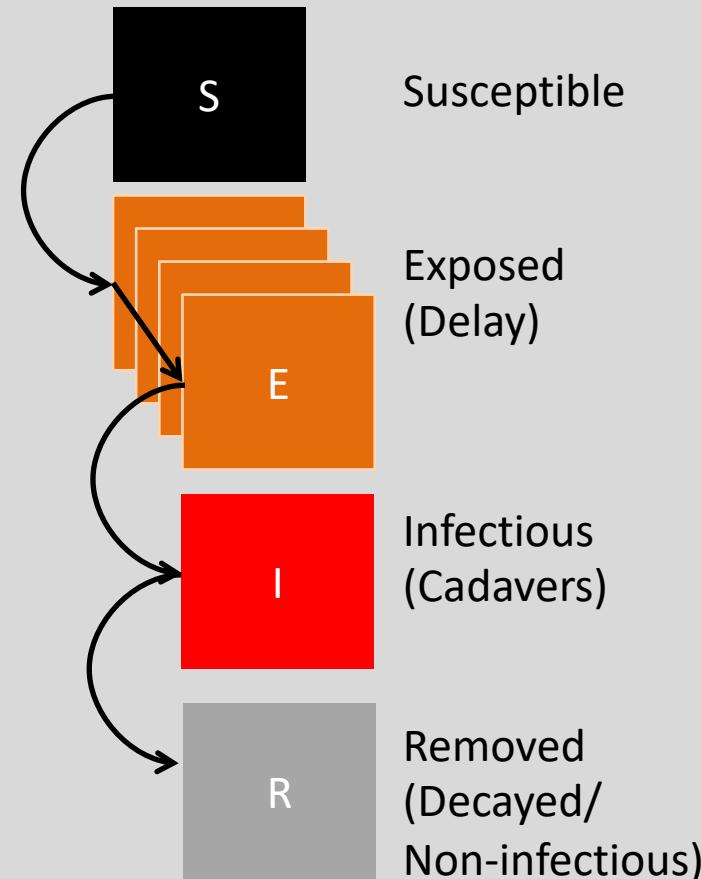
$$\frac{dE_1}{dt} = \bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1$$

$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i \quad (i = 2, \dots, m)$$

$$\frac{dI}{dt} = m\delta E_m - \boxed{\gamma I}$$

$$\frac{dR}{dt} = \gamma I$$

Decay rate



Lab and Field Experiments

$$\frac{dS}{dt} = -\bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dE_1}{dt} = \bar{\nu}e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1$$

$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i$$

$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

Transmission

Transmission parameters (Field Experiment)

- Build miniature epizootics in the field,
measure epizootic size

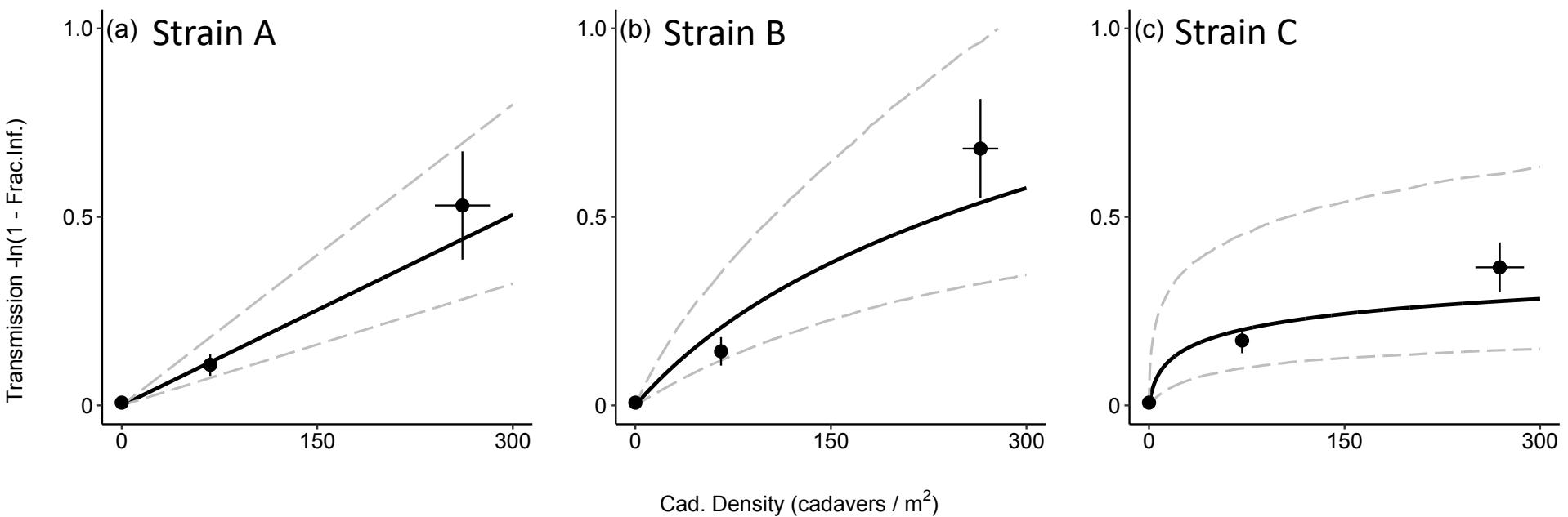
$$\frac{dS}{dt} = -\boxed{-\nu} e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2}$$



Transmission

Transmission parameters (Field Experiment)

- Build miniature epizootics in the field,
measure epizootic size



Speed-of-kill

Speed-of-kill (Lab Experiment)

- Laboratory exposures, measure time to death

$$\frac{dE_1}{dt} = \bar{v} e^{\varepsilon_t} SI \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1$$

$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i$$

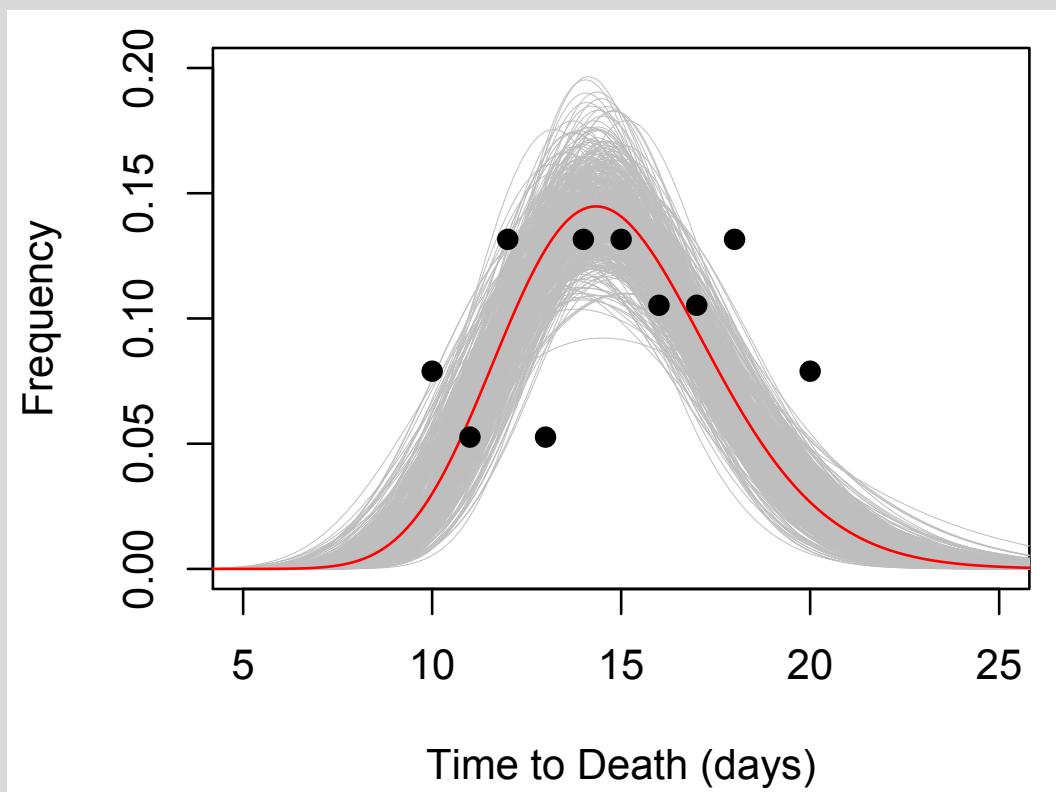


Speed-of-kill

Speed-of-kill (Lab Experiment)

- Laboratory exposures, measure time to death

Gamma-distributed time-to-death



Model Fitting

Model Fitting Components:

1. Integrating the system of DEs
2. Calculating the likelihood
3. Markov Chain Monte Carlo (MCMC)

Bayesian Framework, with and without experimentally-derived priors

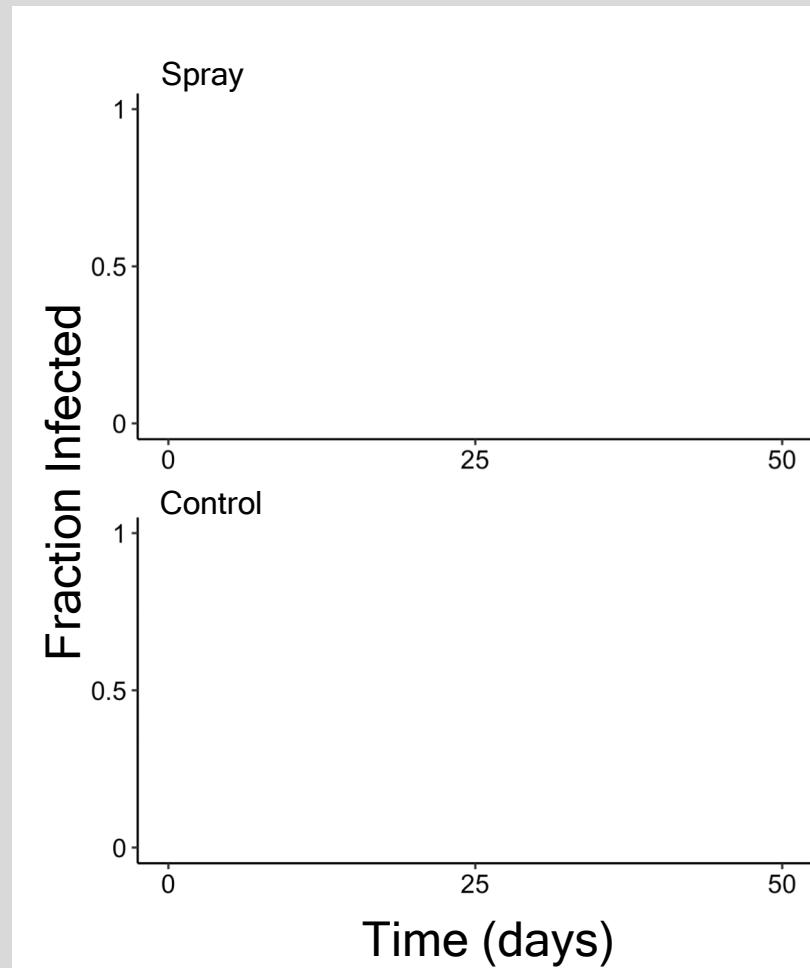
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$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i \quad (i = 2, \dots, m)$$

$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$



Questions

- Are there ecological differences between sprayed and naturally occurring variants?

OR

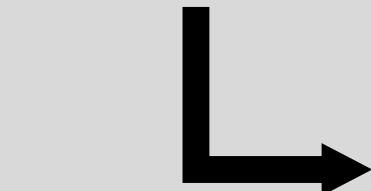
- Do initial conditions (insect density and virus density) and non-linear infection dynamics account for unexpected results?

Basic Questions

- How important is host heterogeneity in susceptibility for explaining natural epizootics?
- How well do our experiments represent dynamics that occur in the field?

Emerging Approach

Integrate experiments, field data, and models



Parameterize w/
Experiments
(Priors)

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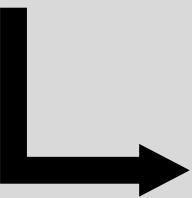
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$$\frac{dI}{dt} = m\delta E_m - \gamma I$$

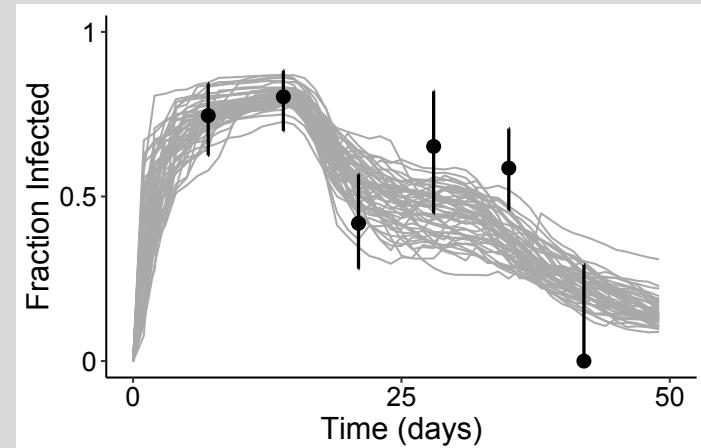
$$\frac{dR}{dt} = \gamma I$$

Build competing models:

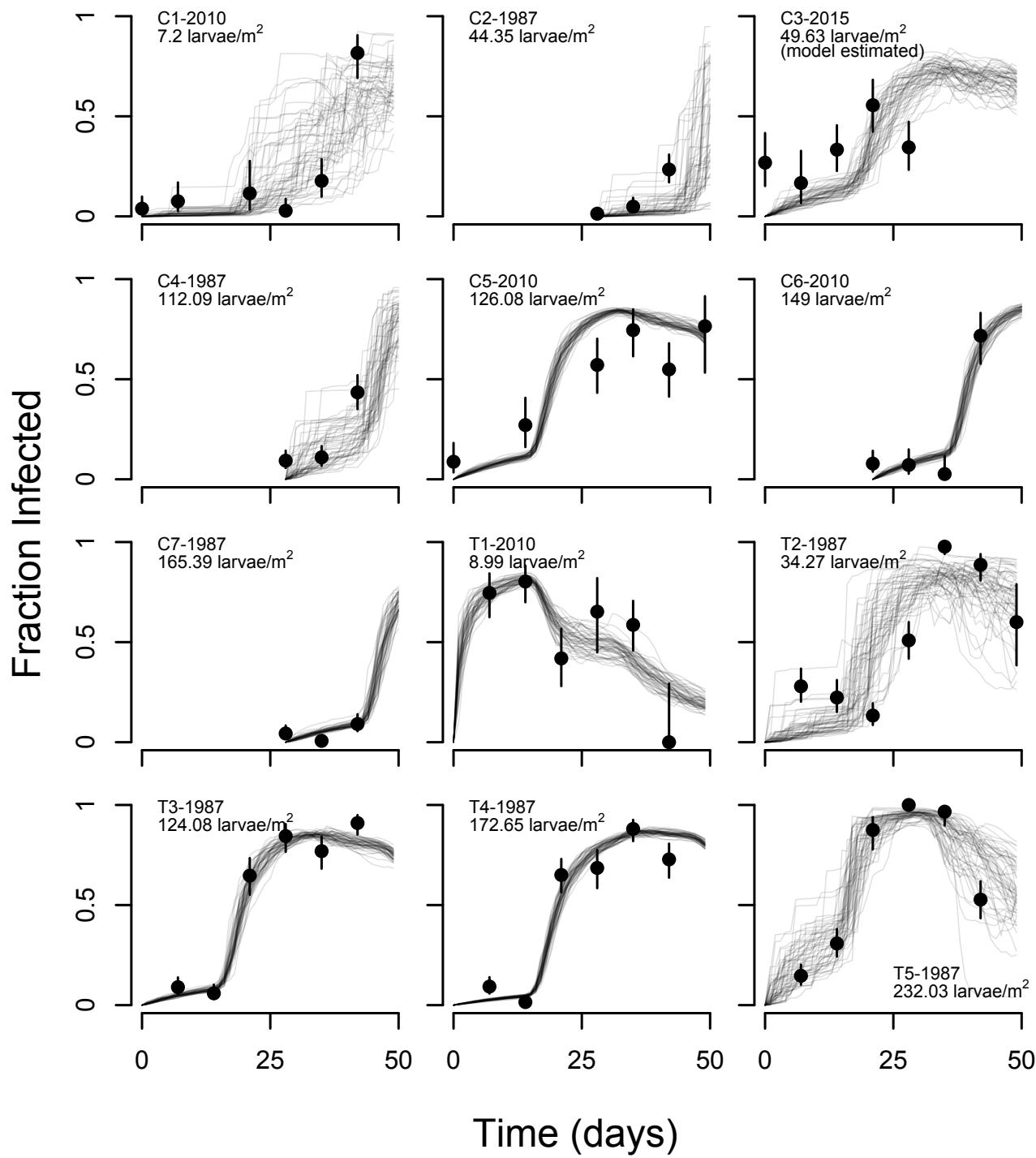
- Host heterogeneity in susceptibility
- 1 or 2 distinct transmission rates



Fit models to field data
(Generate Posteriors)



Prevalence in sprayed (treated) and
non-treated populations

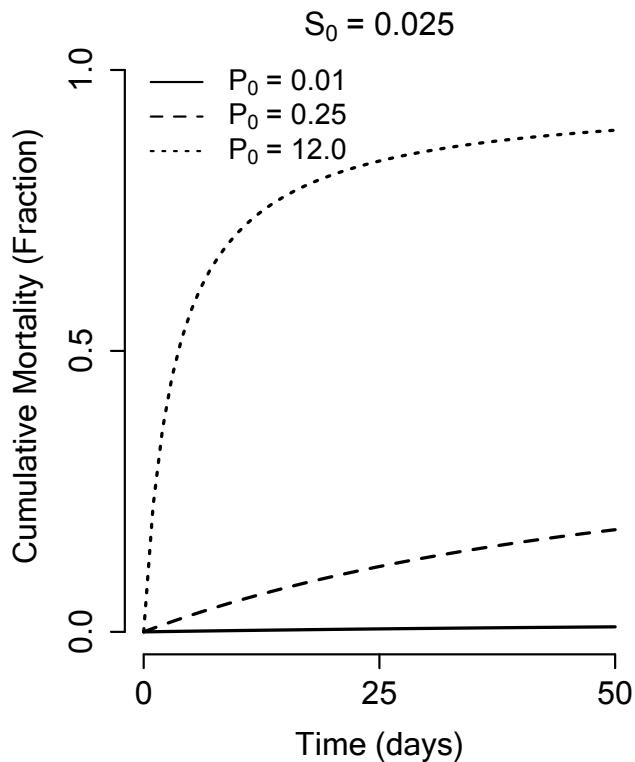


$$r^2=0.76$$

Formal Model Comparison (WAIC)

- Host heterogeneity in susceptibility is necessary for explaining epizootics.
- Variability in host density and virus density can predict epizootics without needing to invoke differences in virus transmission rates.
- We can accurately estimate kill rate and heterogeneity parameters, but our experiments might not adequately capture transmission rate.

Implications for spray programs



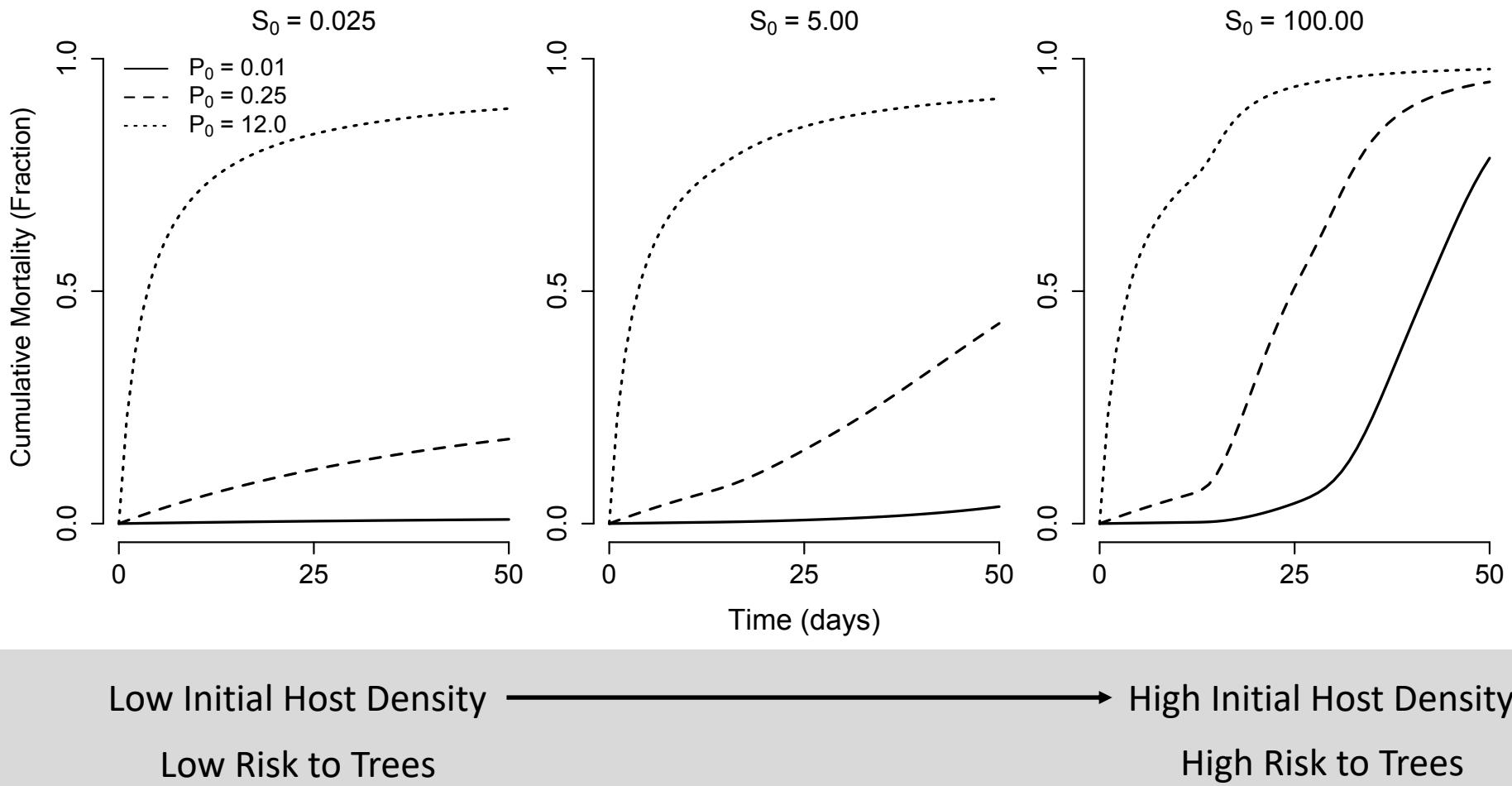
Low Initial Host Density

Low Risk to Trees

High Initial Host Density

High Risk to Trees

Viral augmentation is most beneficial at intermediate host densities



Future Directions: CS Capstone

SEIR Model Predictions

The model structure:

$$\frac{dS}{dt} = -\bar{\nu} e^{E_1} SP \left[\frac{S(t)}{S(0)} \right]^{C^2},$$

$$\frac{dE_1}{dt} = \bar{\nu} e^{E_1} SP \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta E_1,$$

$$\frac{dE_i}{dt} = m\delta E_{i-1} - m\delta E_i \quad (i = 2, \dots, m),$$

$$\frac{dP}{dt} = m\delta E_m - \mu P.$$

To create the plot:

1. Assign the parameters. You can choose whether to use a slider or to manually enter the values yourself.
2. Click the **Run Simulation** button.

How would you like to input parameters?

Manual

Initial Larval Densities (m^{-2})

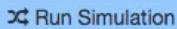
20

Initial Virus Density (4th instars m^{-2})

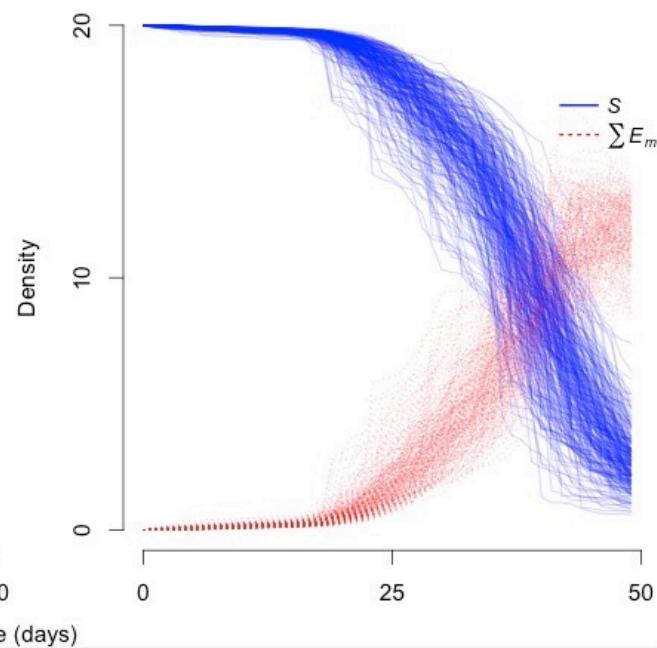
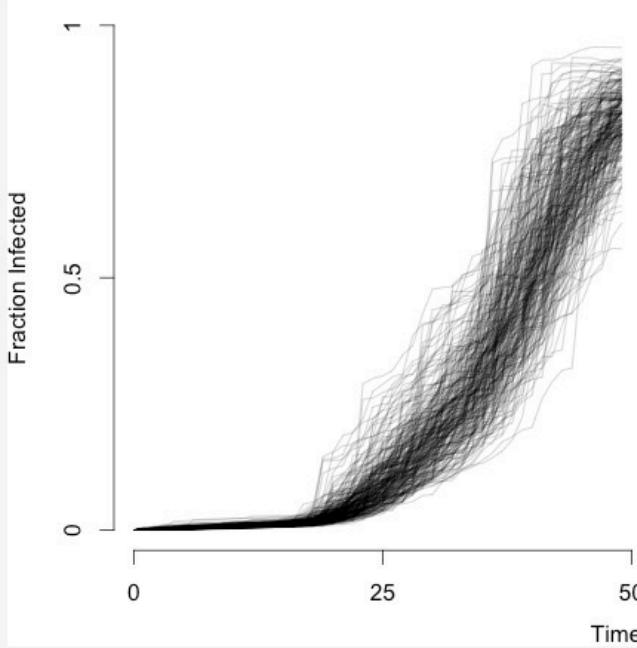
0.005

Stochasticity, σ^2

.9

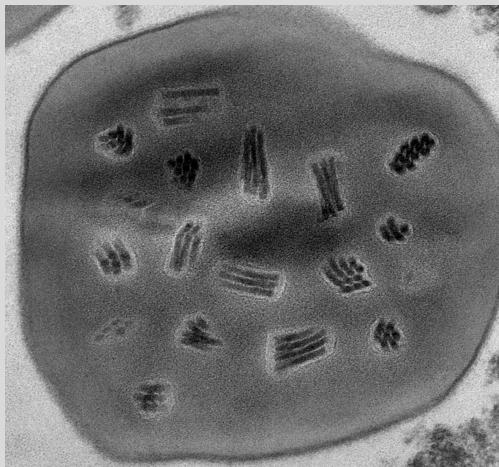
 Run Simulation

Cumulative Mortality (95% CI): 86.5% (74.1, 95)

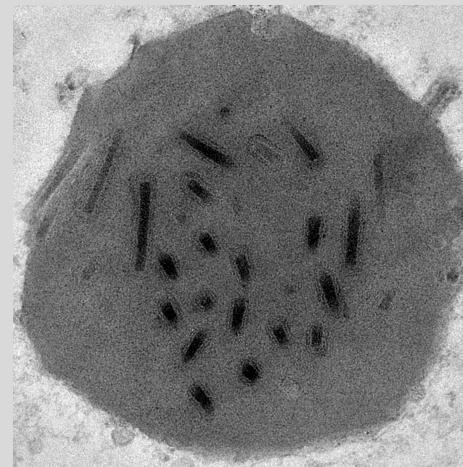


Future Directions

- Explore co-existence of viral polymorphism



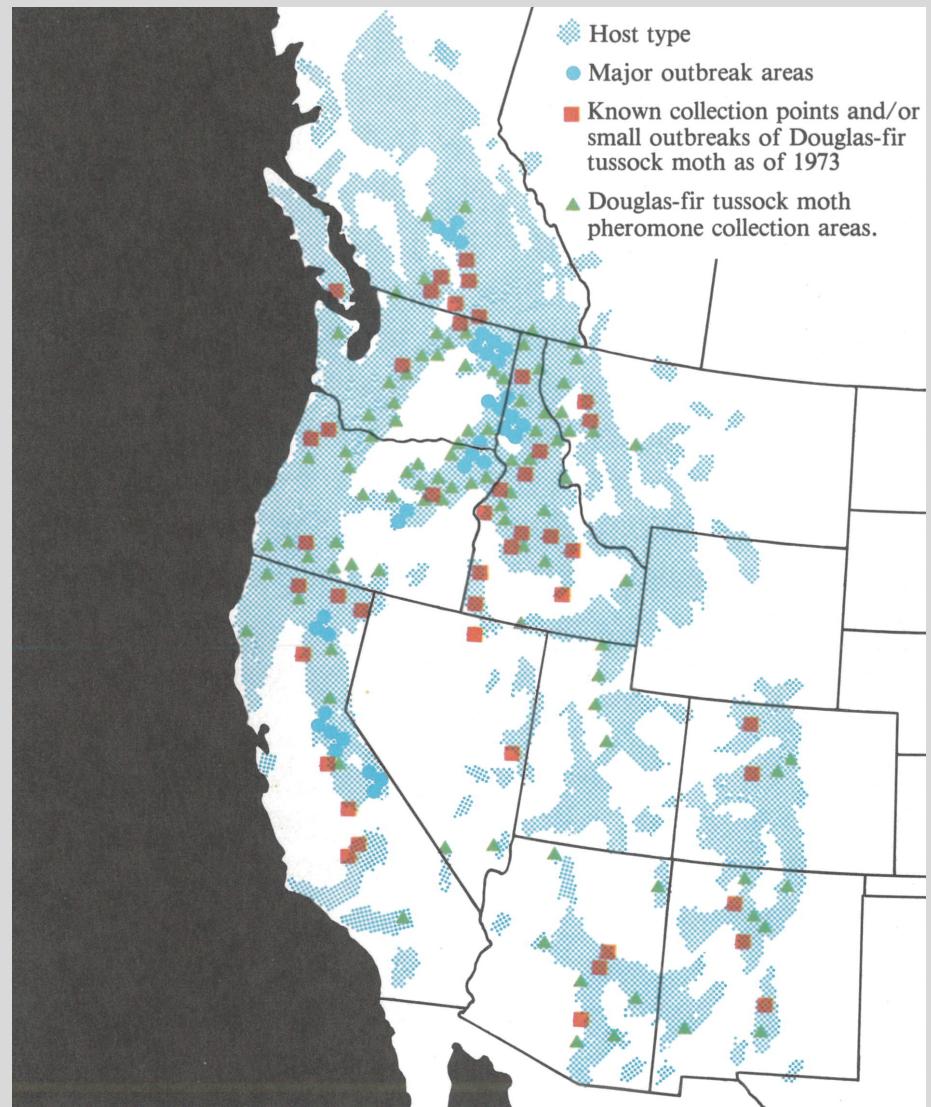
MNPV



SNPV

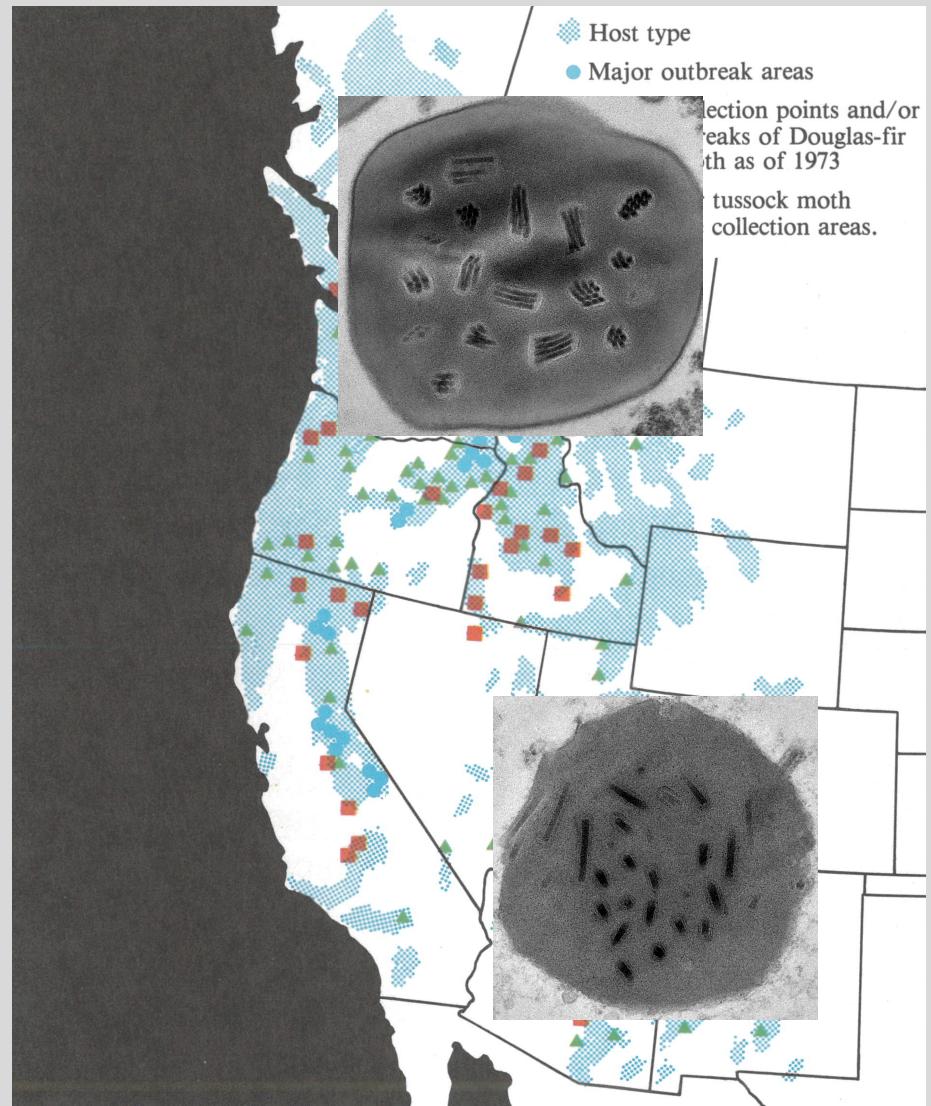
Viral Polymorphism

Latitudinal cline in coexistence



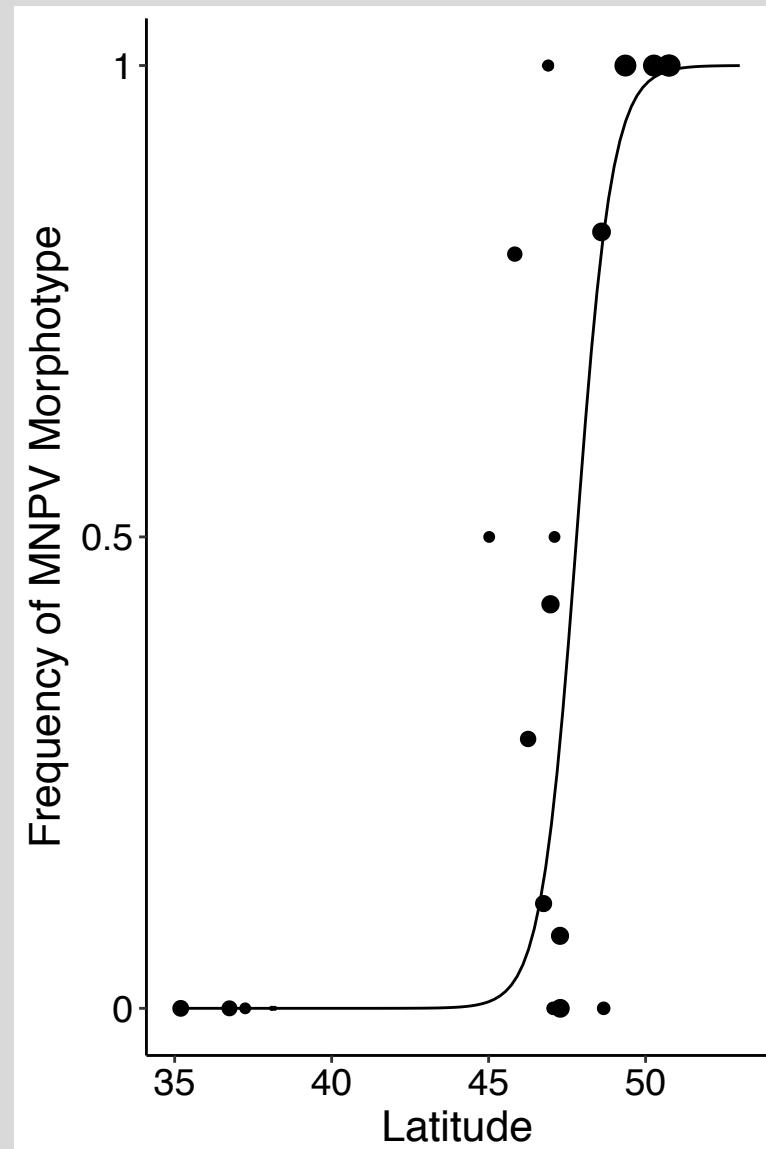
Viral Polymorphism

Latitudinal cline in coexistence



Viral Polymorphism

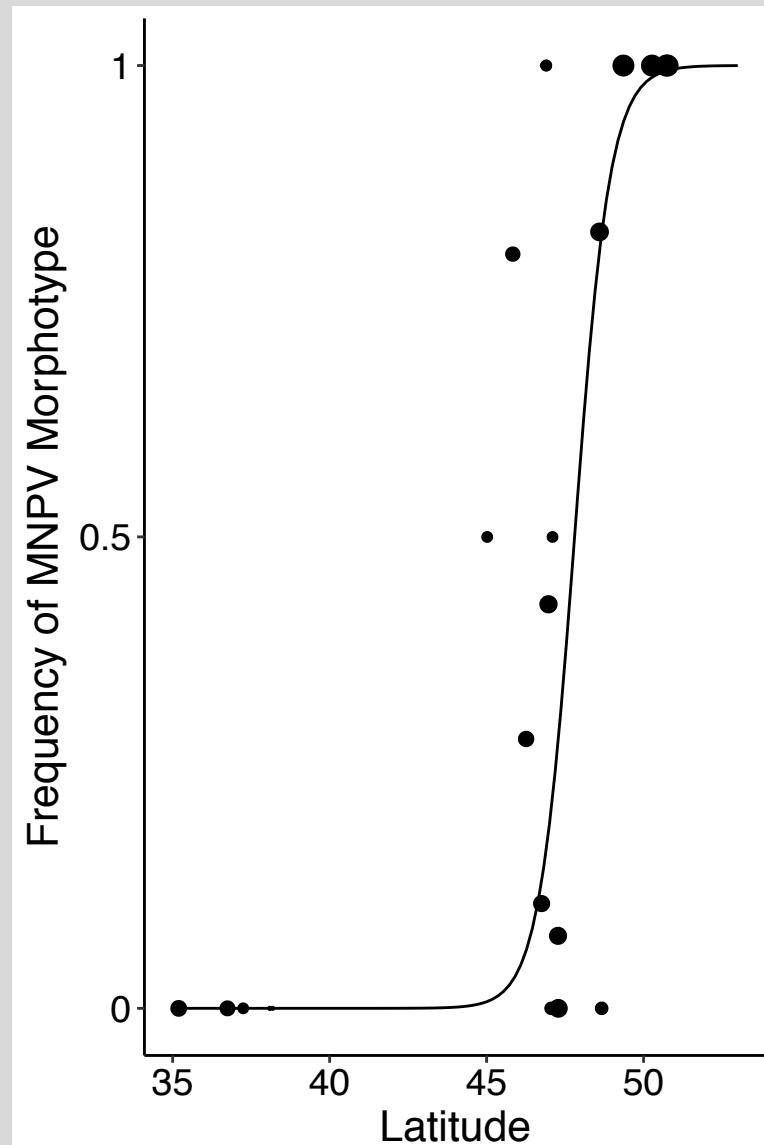
Latitudinal cline in coexistence



Viral Polymorphism

Latitudinal cline in coexistence

- Host tree species
- Environmental tolerances





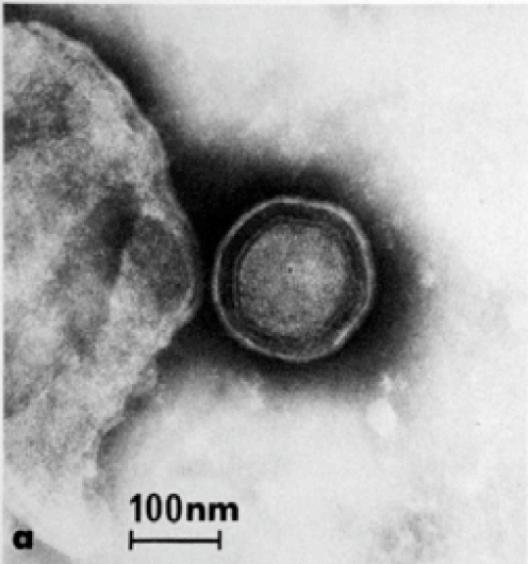
Part II: Amphibian ranaviruses

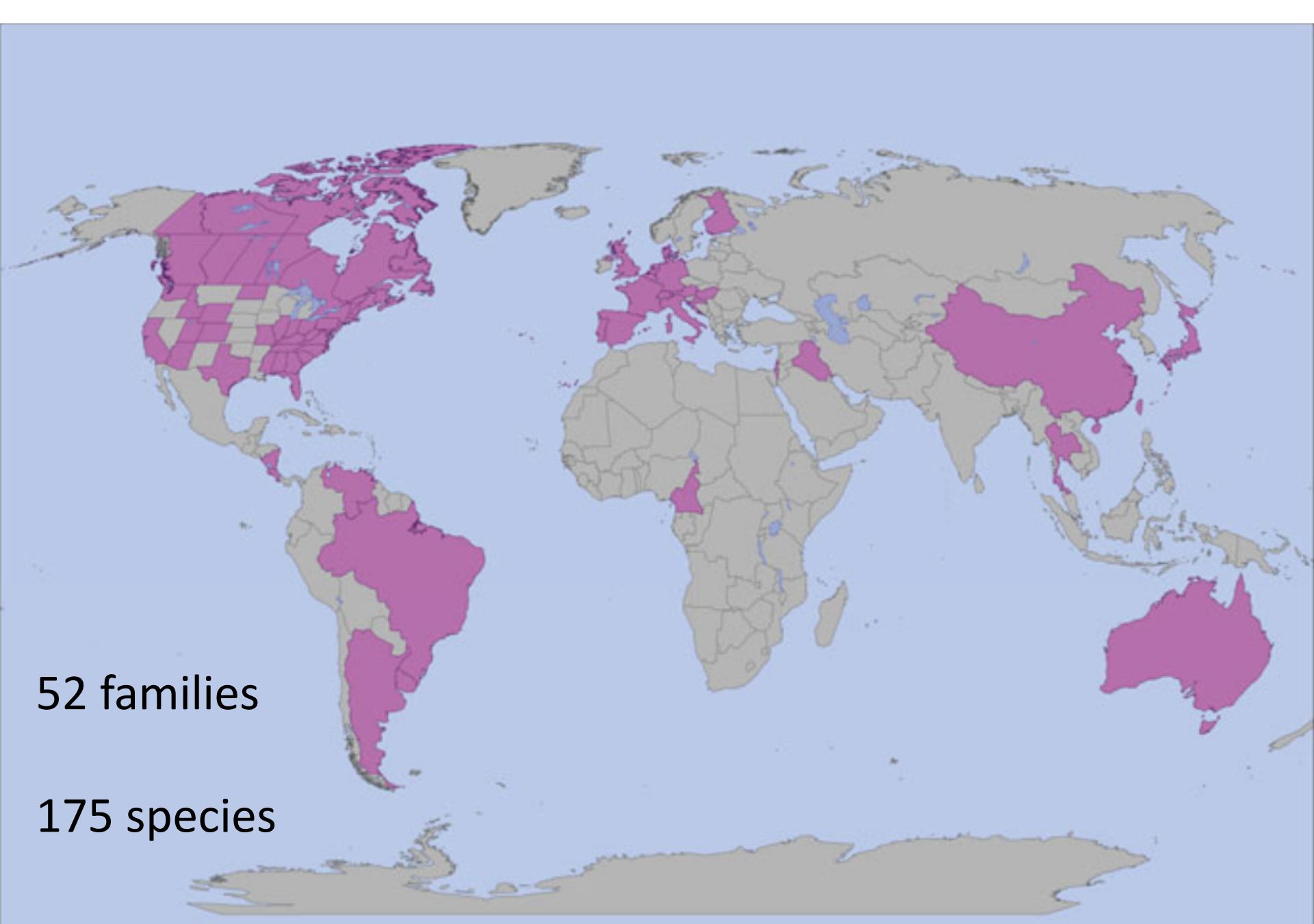
Collaborators:

Jesse Brunner (Univ. Washington)

Amy Greer (Univ. Guelph)

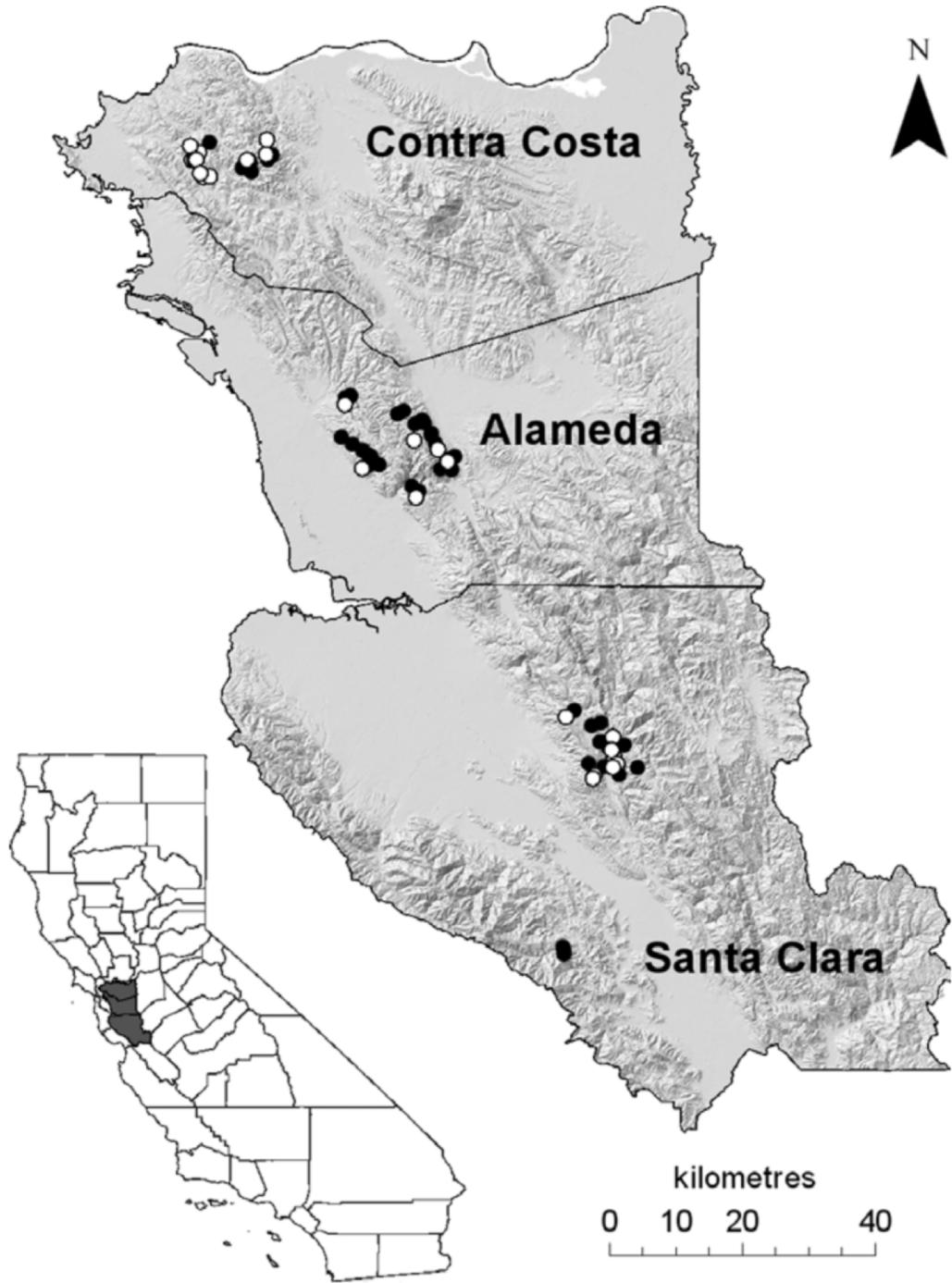
Ranavirus (Family: Iridoviridae)





52 families

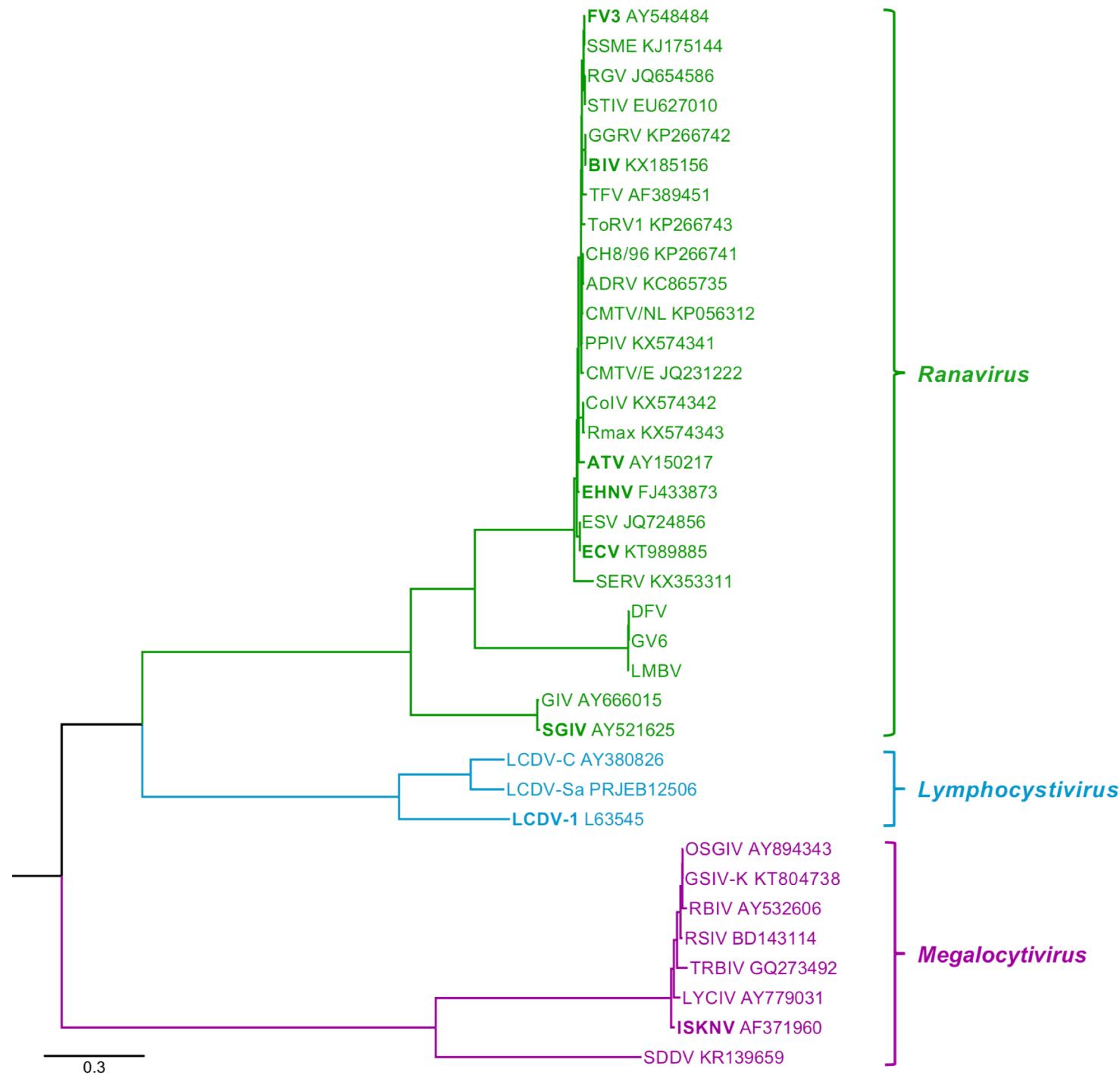
175 species



76 sites sampled

67% sites *Rv* +

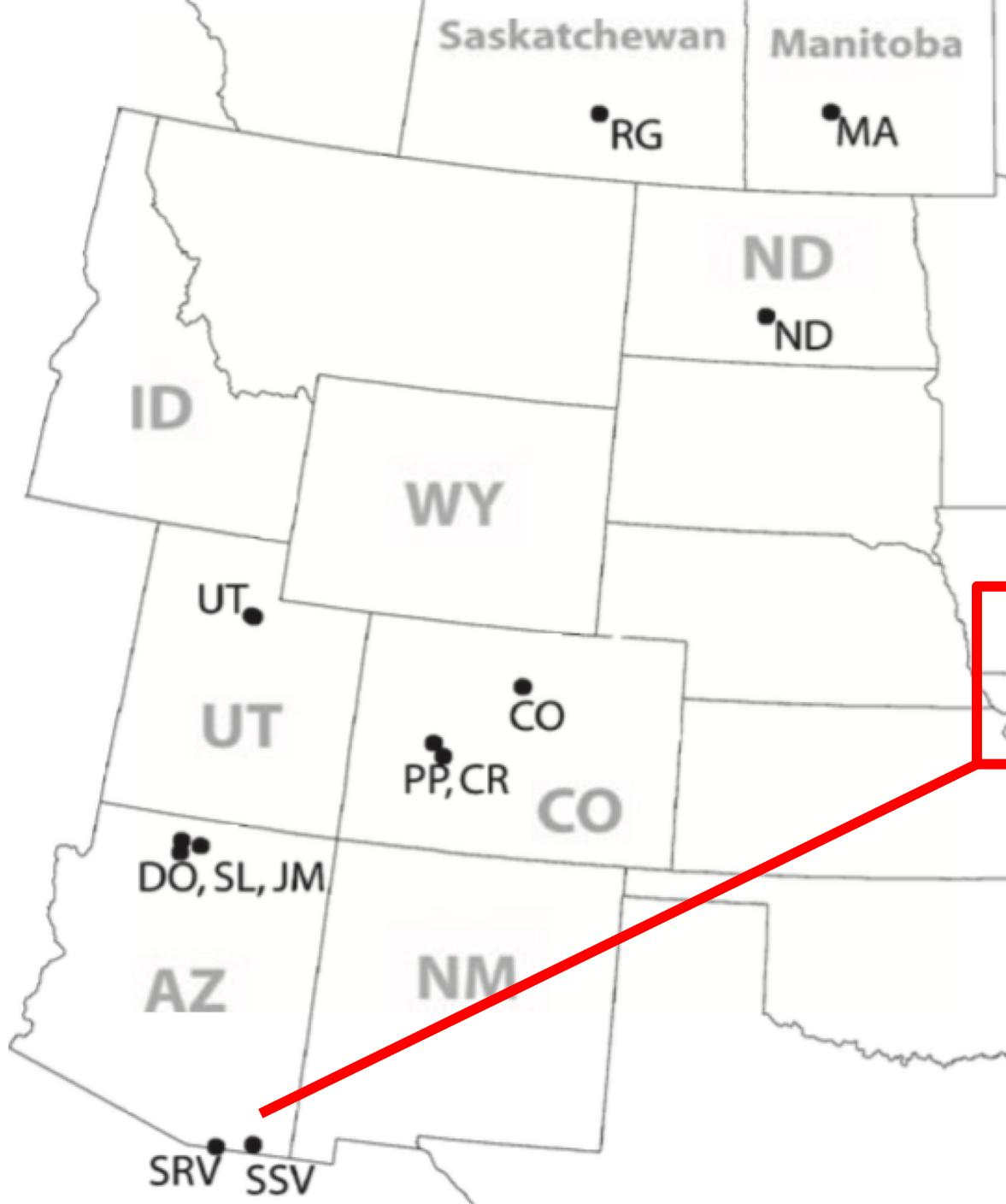
Hoverman, Mihaljevic, et al. 2012
Tornabene et al. 2018





Ambystoma tigrinum virus
(ATV)

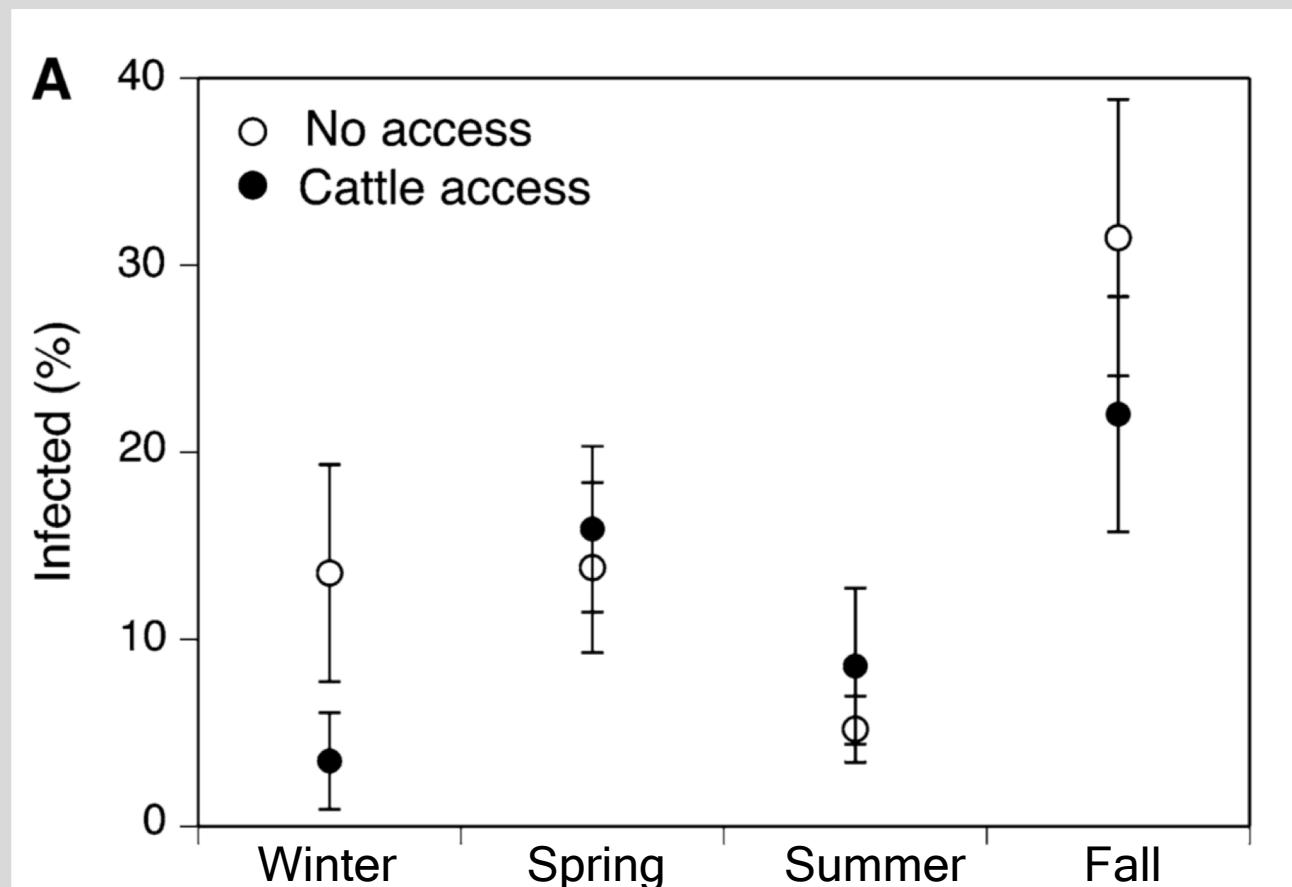
Outbreaks across the west



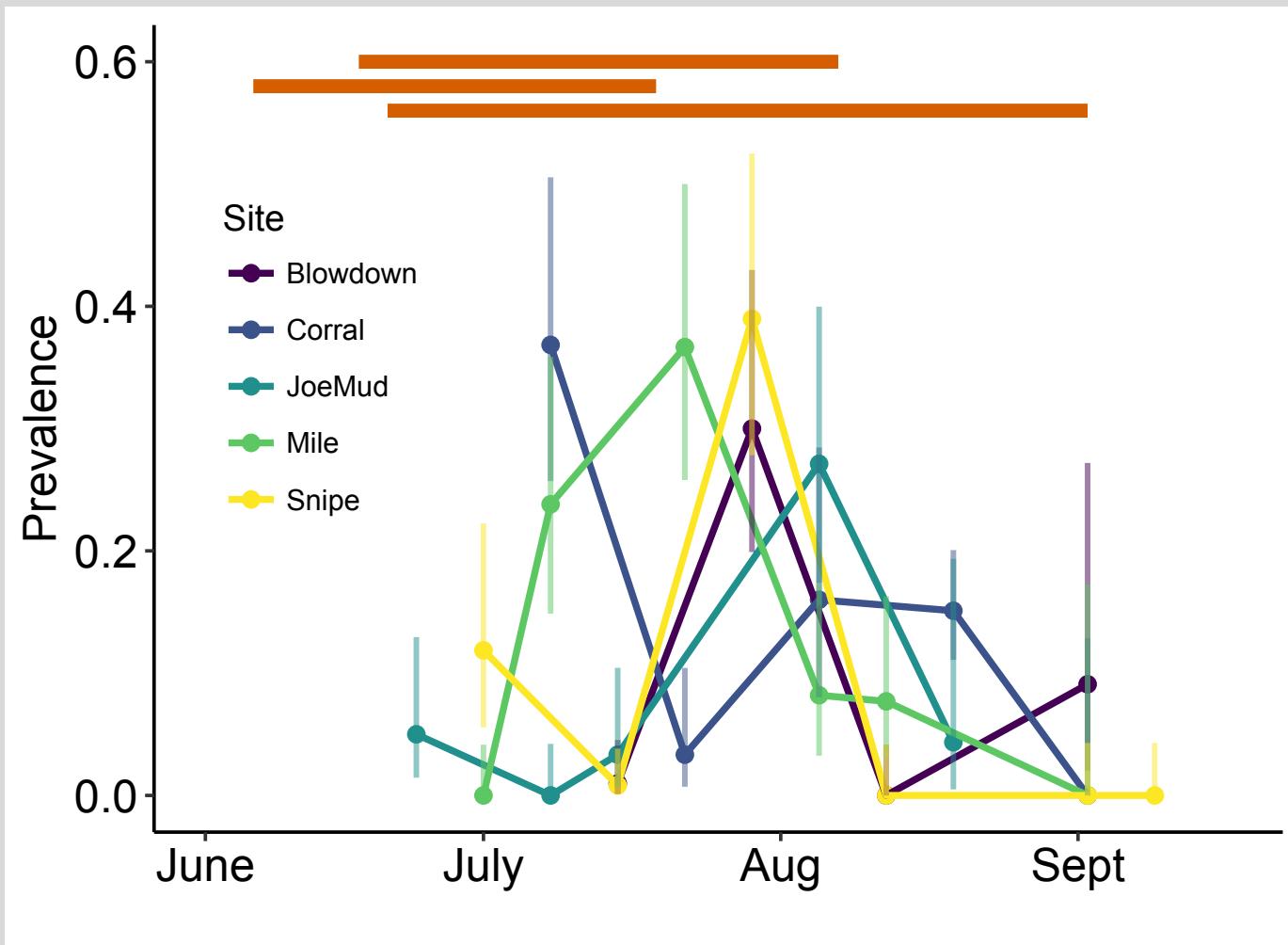
San Rafael Valley –
Spillover from live bait

Epizootics peak later in year

Tennessee

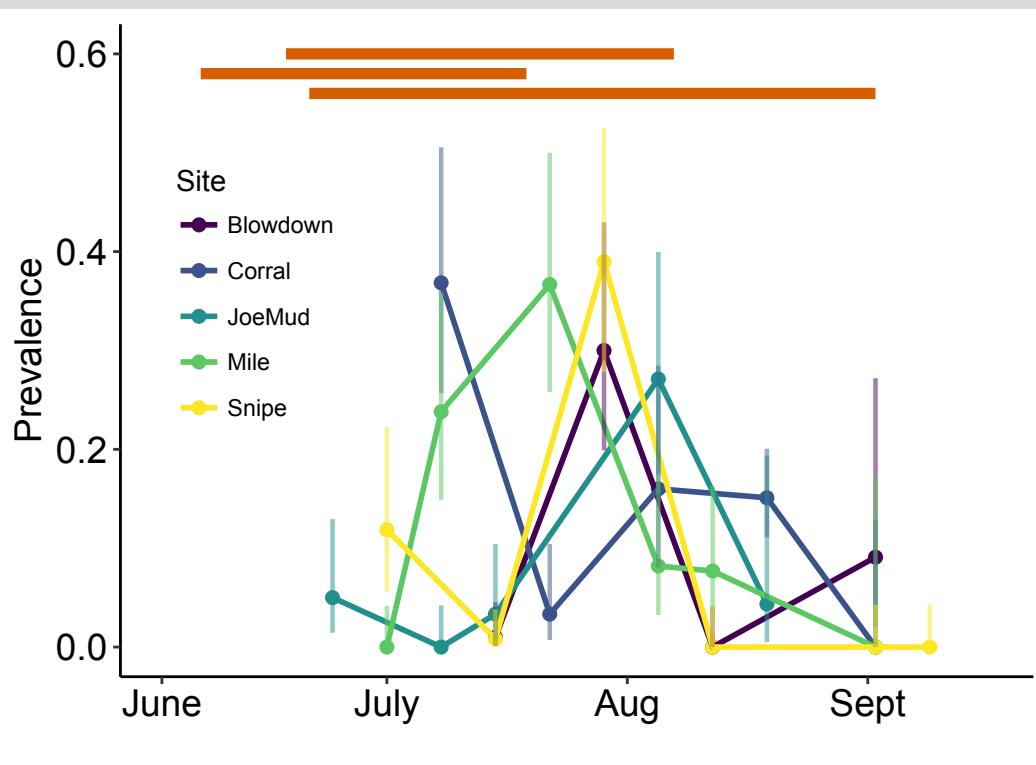


Field epizootics and die-offs



Greer et al. 2009; Berna 1989; Storfer & Brunner (*personal comm.*)

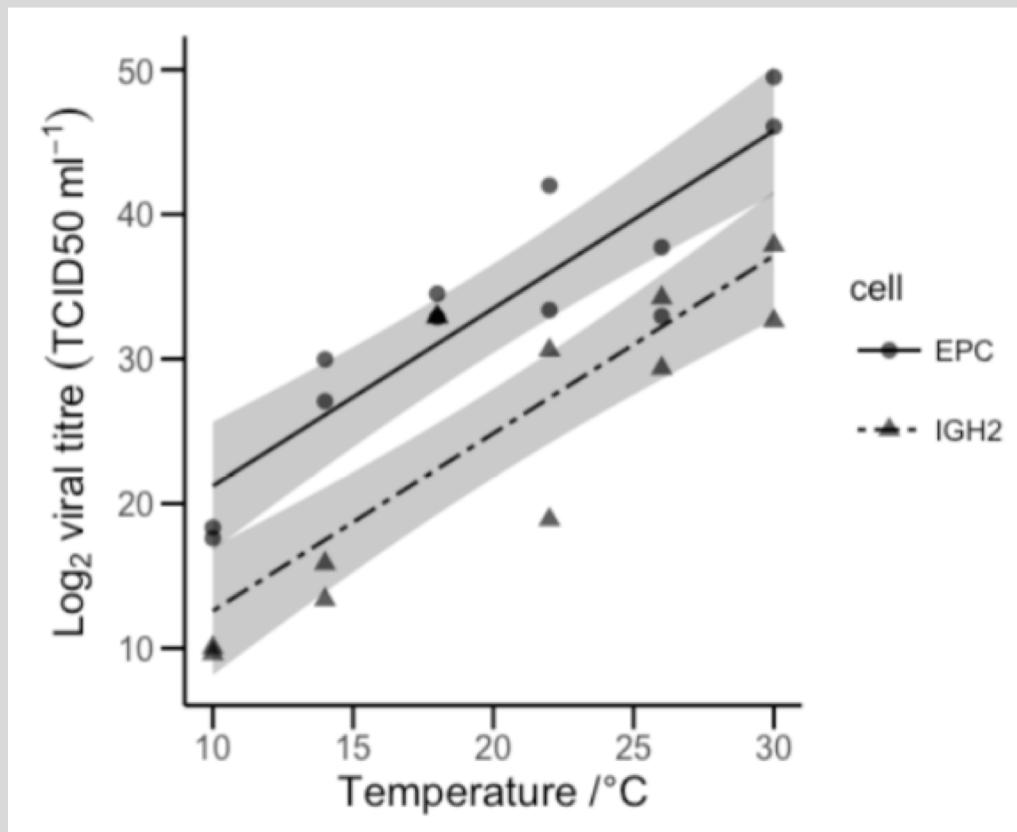
Field epizootics and die-offs



Question:

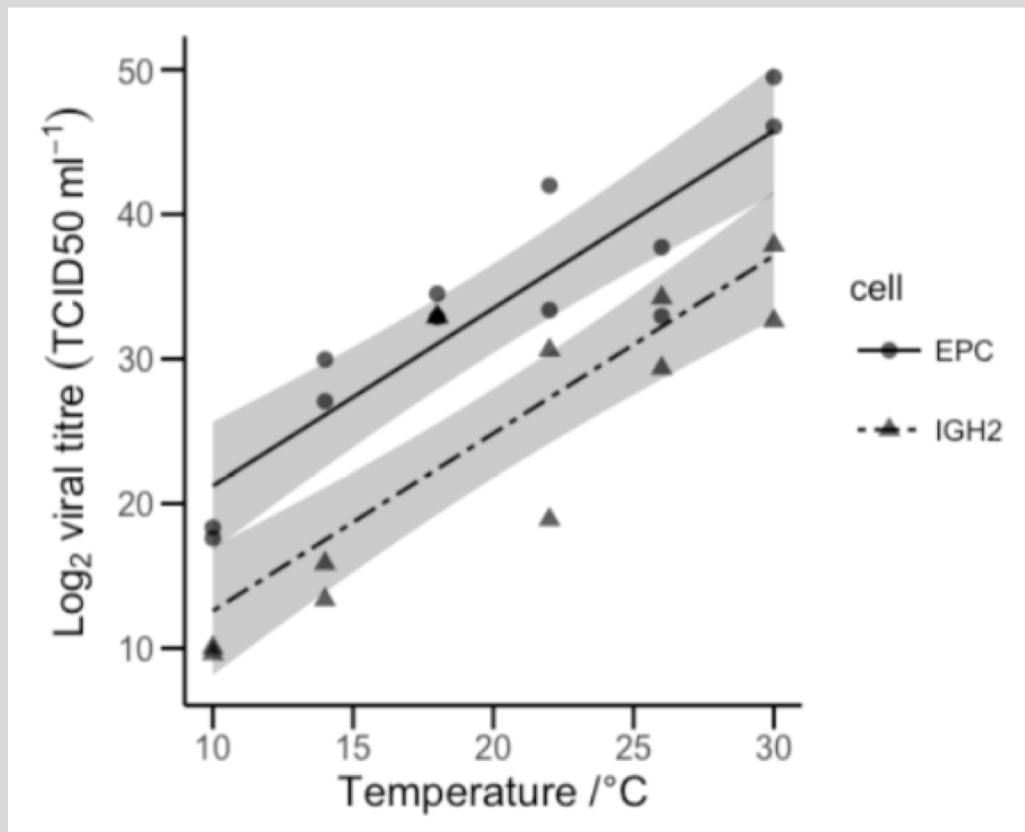
- Why do infections peak in late summer, when virus can be detected in spring?

Hypothesis: Temperature-dependent viral shedding



Ranaviruses grow faster at higher temperatures in cell culture

Hypothesis: Temperature-dependent viral shedding

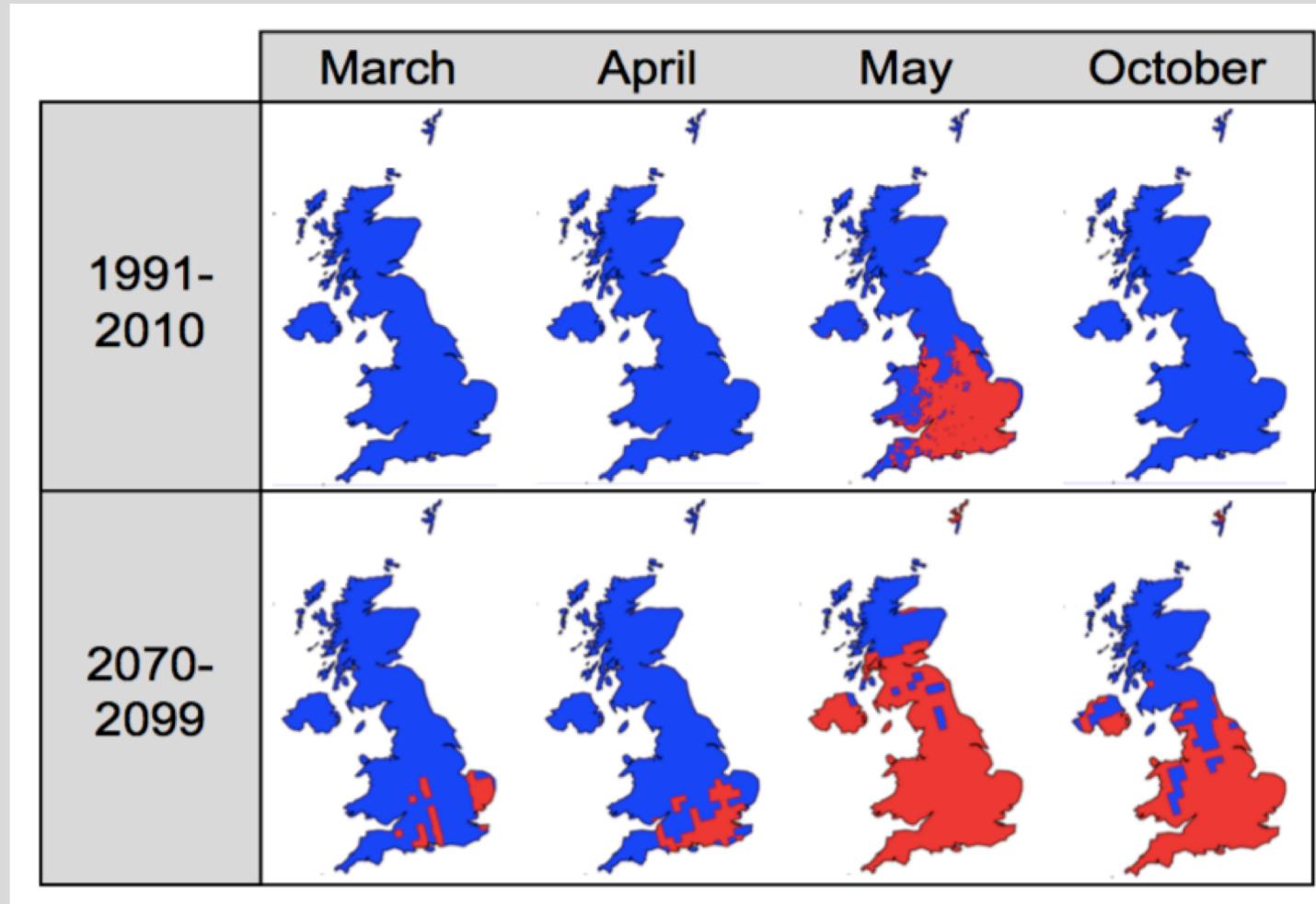


Ranaviruses grow faster at higher temperatures in cell culture

Presumably, viral shedding rate from infected animals also increases

Tenuous Suggestion:

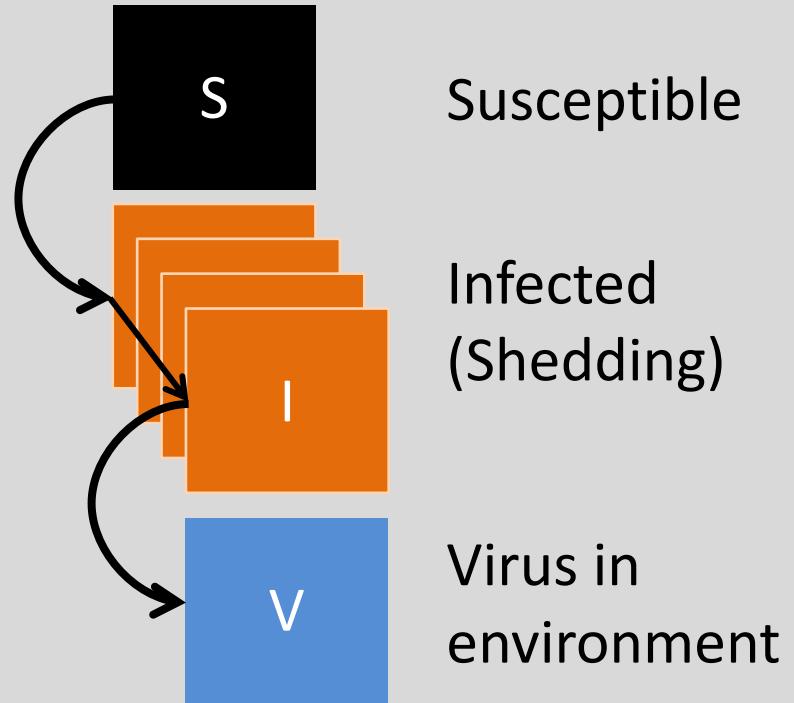
Larger ranaviral epizootics will become more prevalent



Goals

- Use historic data to parameterize a model of ATv transmission in Arizona tiger salamander populations
- Does a temperature-dependent rate of viral shedding affect explain observed epizootics?
- If so, forecast epizootics under warming scenarios

A Model



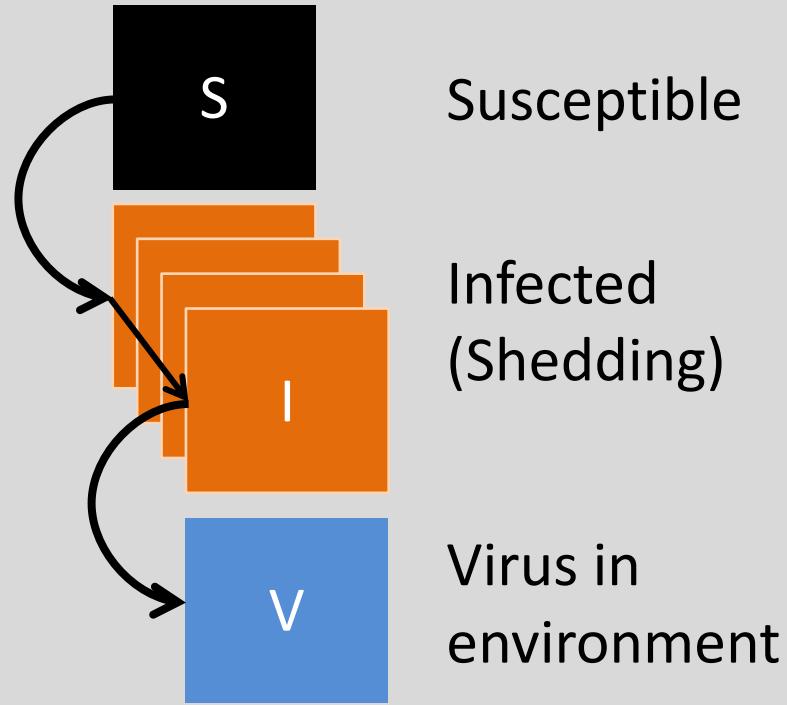
A Model

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \eta V$$



A Model

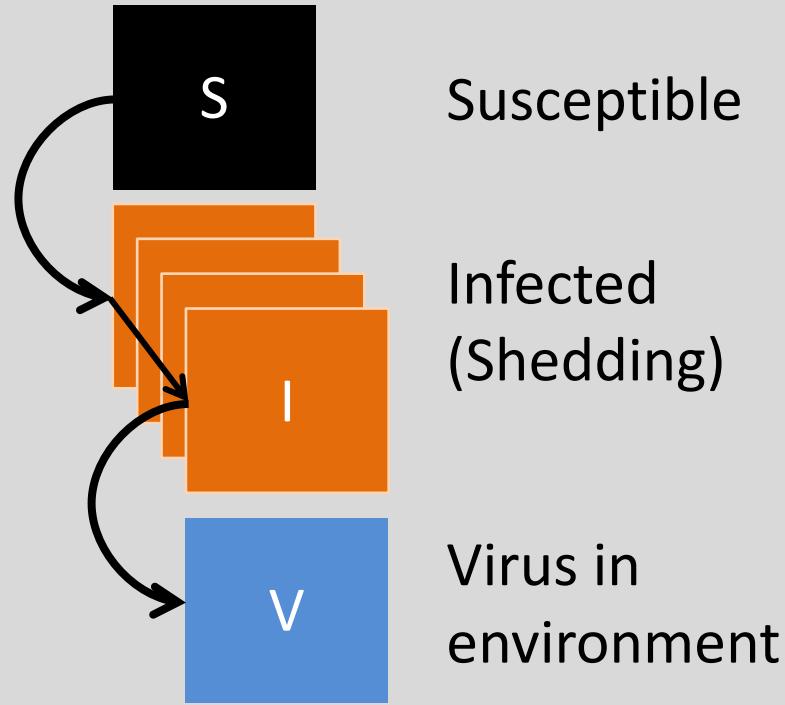
Average transmission, with stochasticity

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \eta V$$



A Model

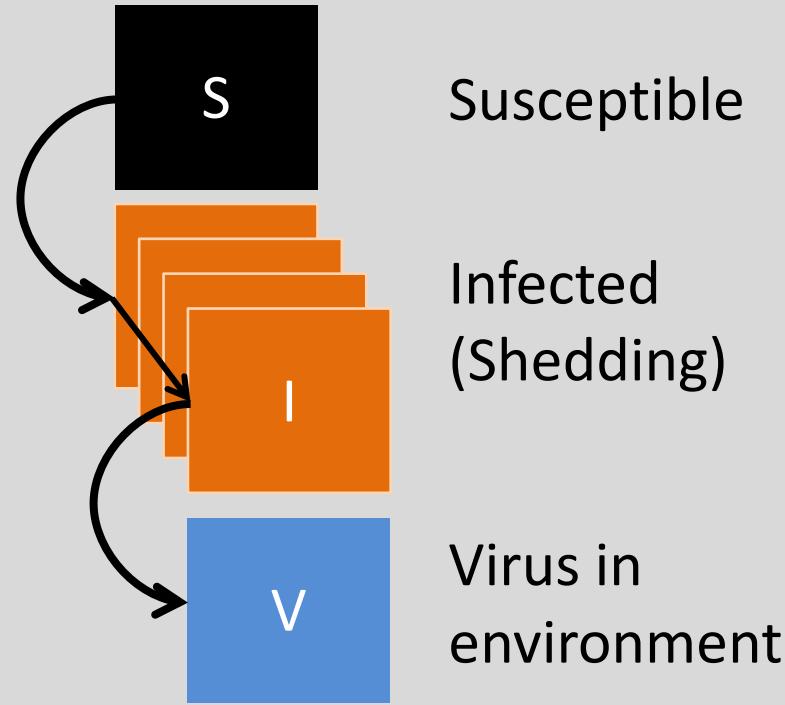
Transmission driven by contact with virus in water

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} \boxed{SV} \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \eta V$$



A Model

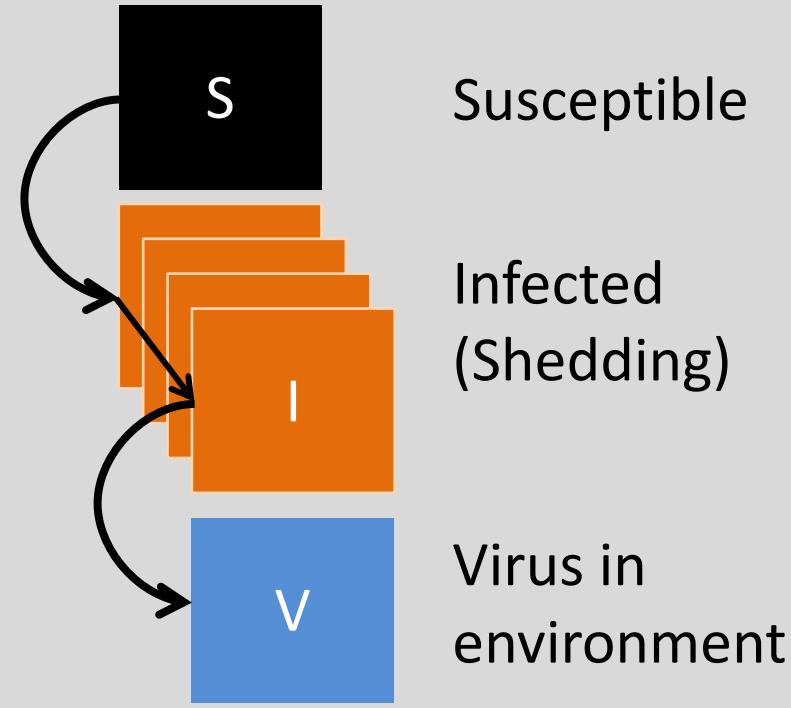
Host heterogeneity in susceptibility

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \eta V$$



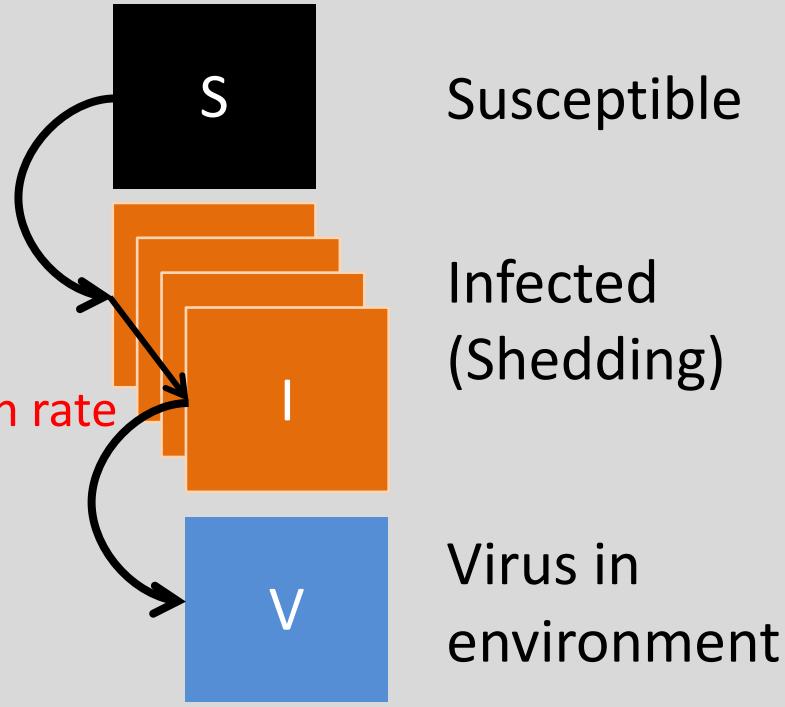
A Model

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \eta V$$



A Model

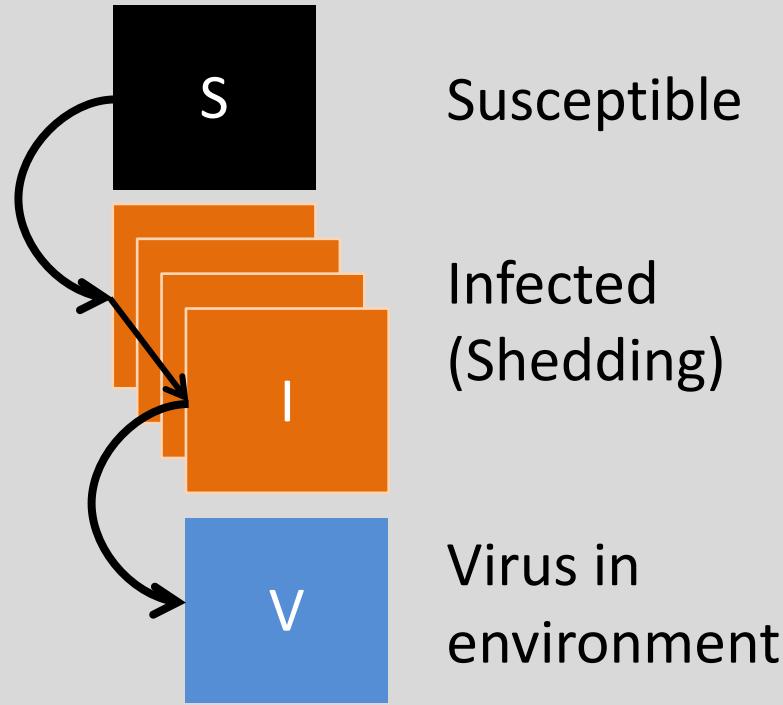
$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \boxed{\eta V}$$

Virus decays in water



A Model

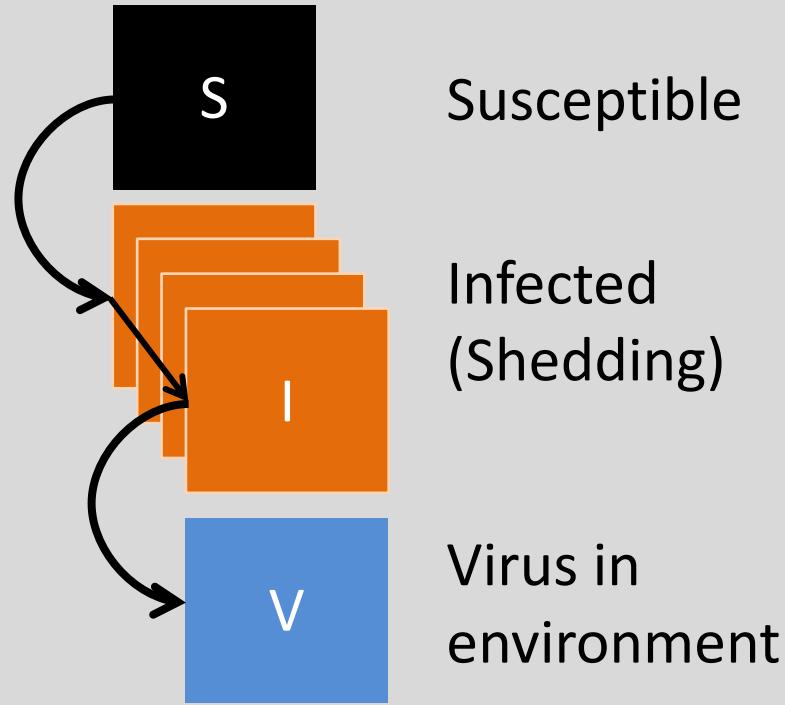
$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi(t) I_i) - \eta V$$

Shedding rate, changes over time



A Model

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

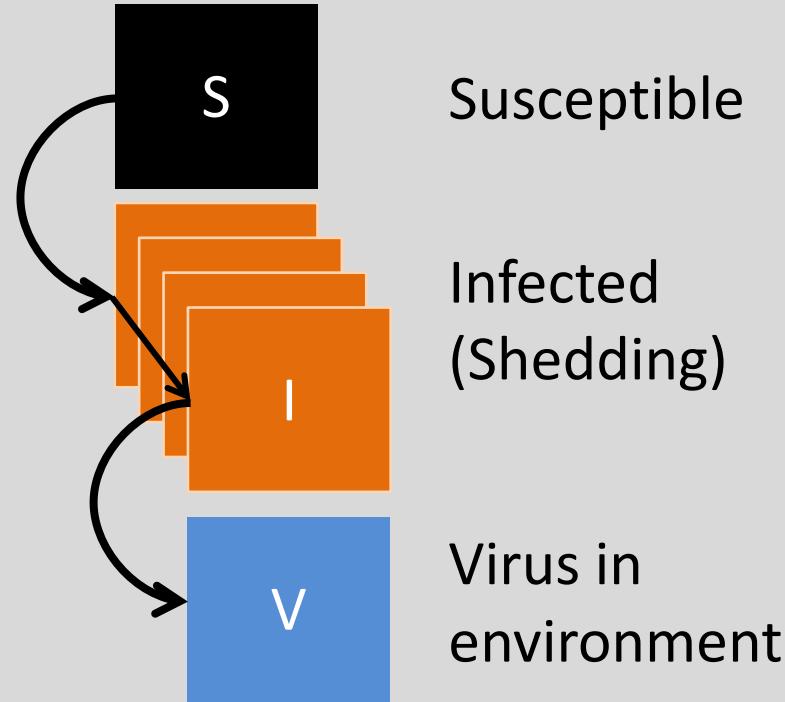
$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \eta V$$

Shedding rate, changes over time,
Function of temperature.

$$\phi_{(t)} = \phi_{(0)} e^{\chi Temp(t)}$$



Parameterization

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \eta V$$

$$\phi_{(t)} = \phi_{(0)} e^{\chi Temp_{(t)}}$$

Transmission

Dose-response experiment to estimate transmission parameters

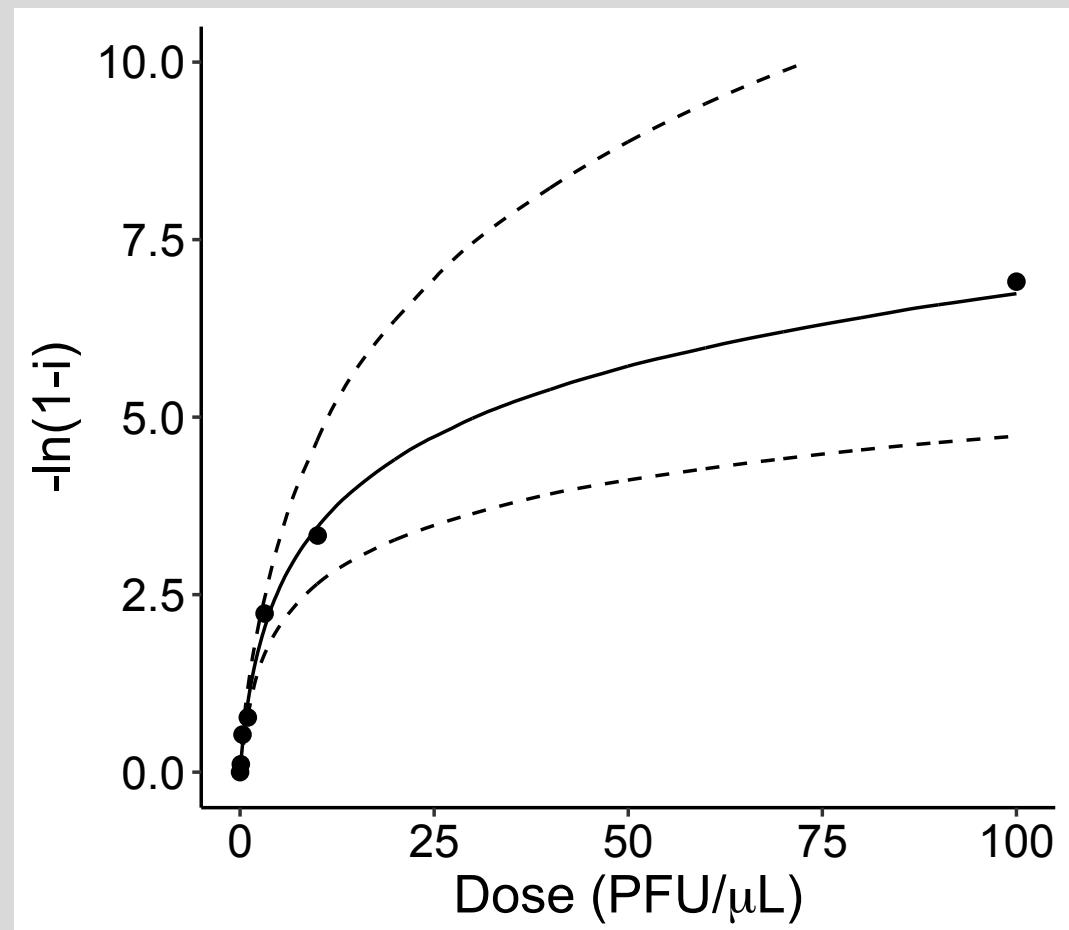
$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S_{(t)}}{S_{(0)}} \right]^{C^2}$$

Transmission

Dose-response experiment to estimate transmission parameters

$$\frac{dS}{dt} = -\bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2}$$

**Significant heterogeneity
($C = 0.82$)**



Speed of kill

Laboratory exposures to measure time to death

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S_{(t)}}{S_{(0)}} \right]^{C^2} - \boxed{m\delta} I_1$$

$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

Speed of kill

Laboratory exposures to measure time to death

$$\frac{dI_1}{dt} = \bar{\beta} e^{\varepsilon_t} SV \left[\frac{S(t)}{S(0)} \right]^{C^2} - m\delta I_1$$

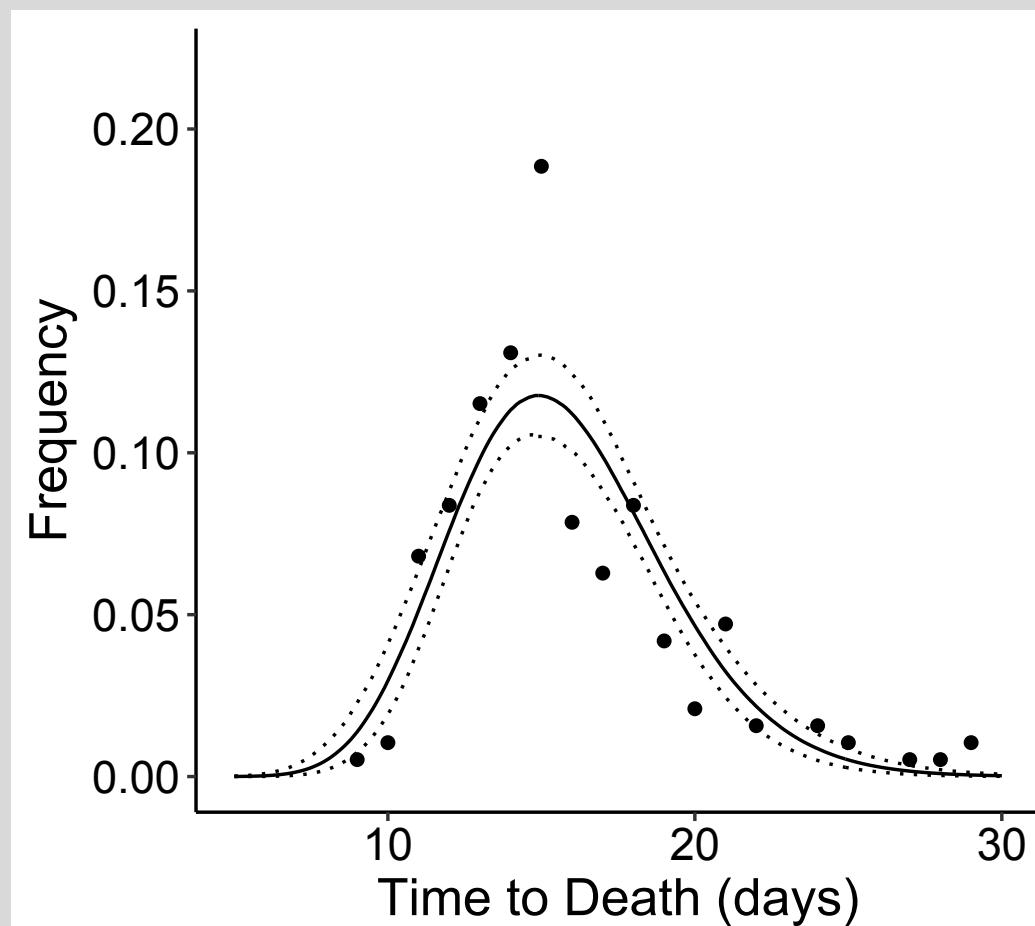
$$\frac{dI_i}{dt} = m\delta I_{(i-1)} - m\delta I_i$$

**Gamma-distributed
speed of kill**

Data from:

Brunner et al. (2009)

Greer et al. (2008)



Viral decay

Laboratory experiments measuring infectivity over time

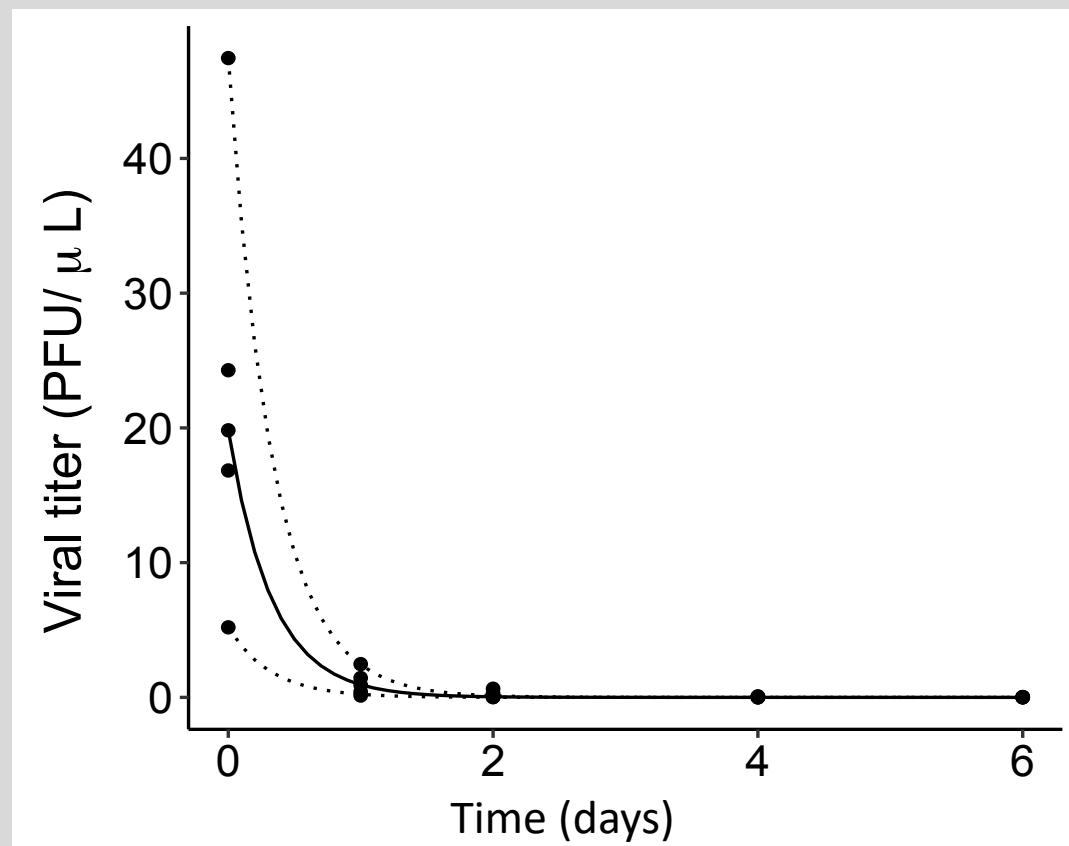
$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \boxed{\eta V}$$

Viral decay

Laboratory experiments measuring infectivity over time

$$\frac{dV}{dt} = \sum_{i=1}^m (\phi_{(t)} I_i) - \boxed{\eta V}$$

Rapid viral decay
in pond water



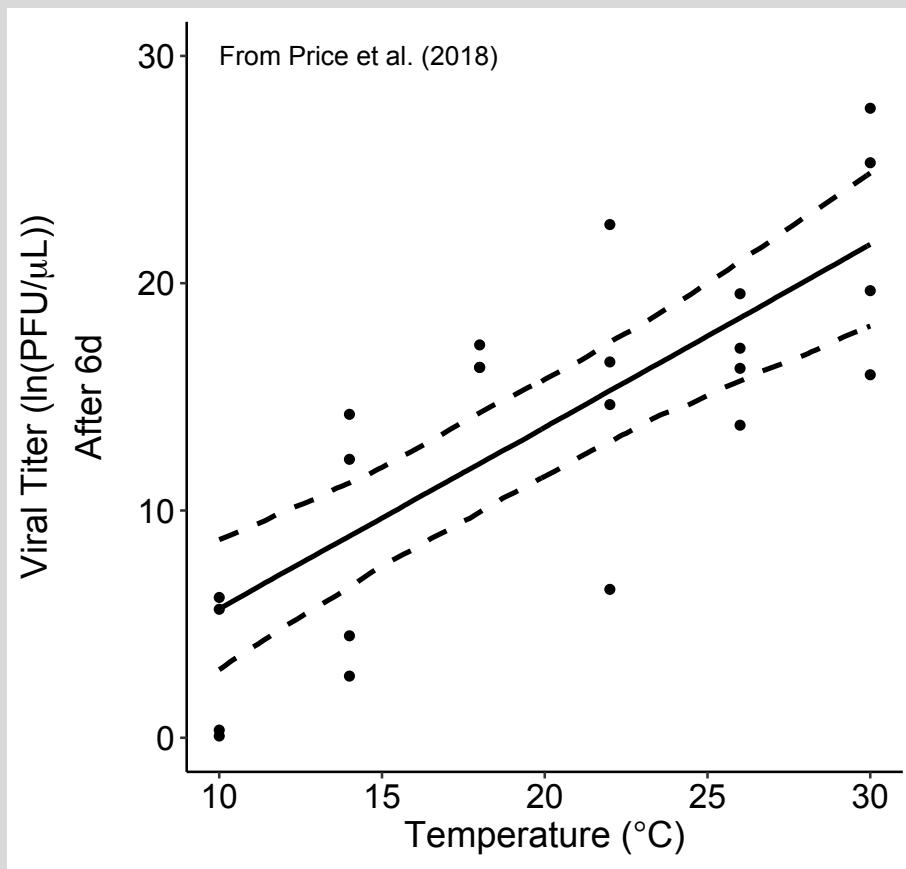
Data from:
Johnson & Brunner (2014)

Shedding rate (from cell culture)

$$\phi_{(t)} = \phi_{(0)} e^{\chi Temp(t)}$$

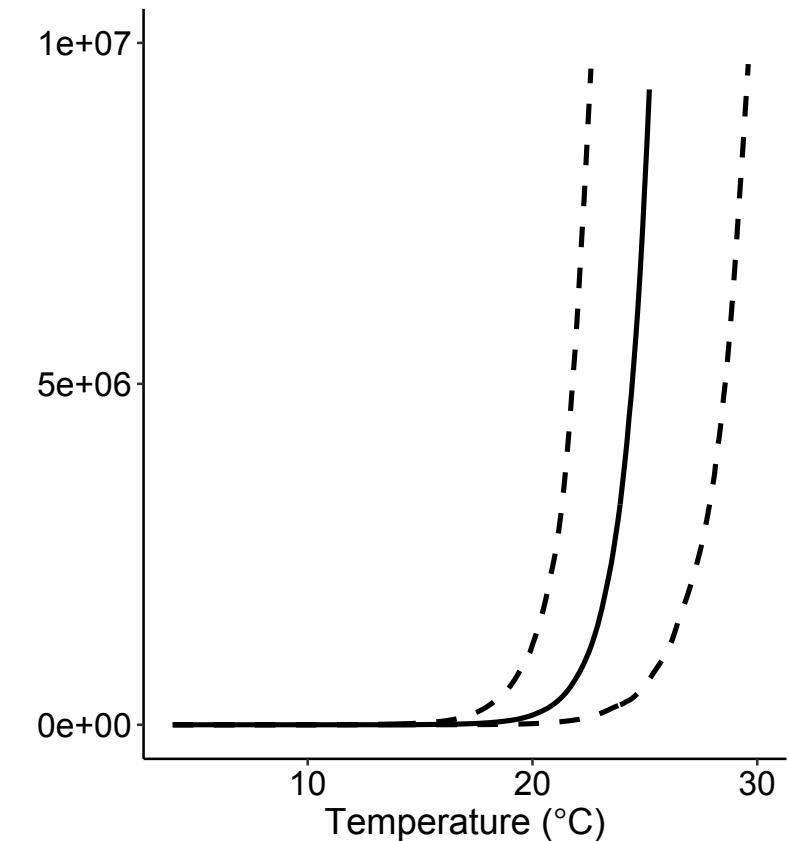
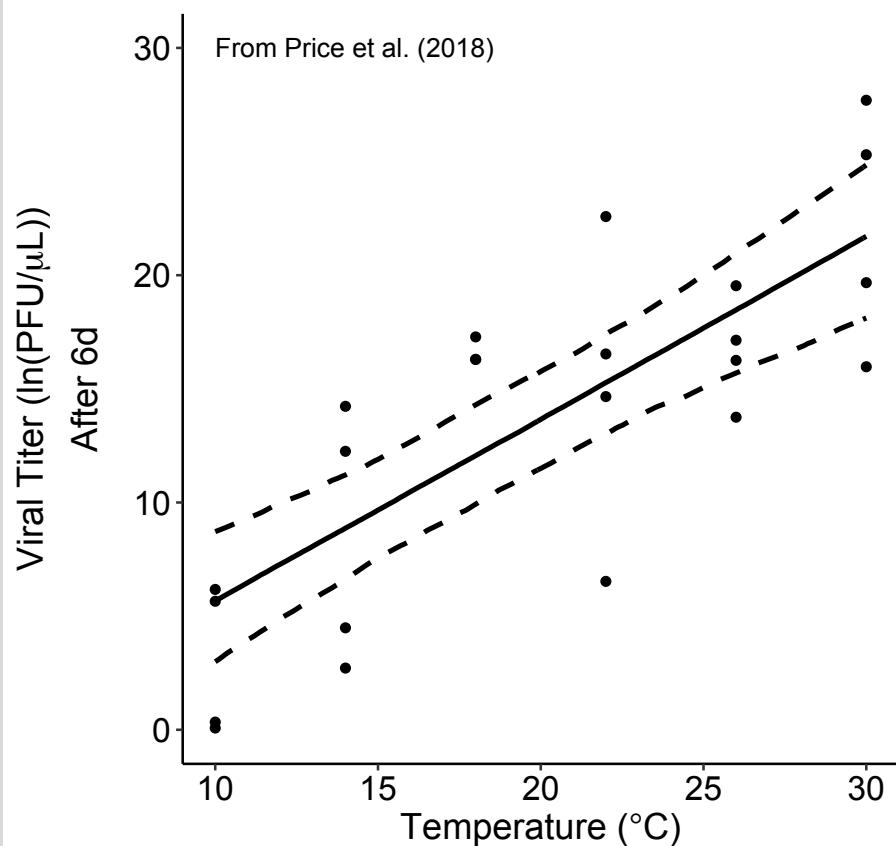
Shedding rate (from cell culture)

$$\phi_{(t)} = \phi_{(0)} e^{\chi Temp(t)}$$



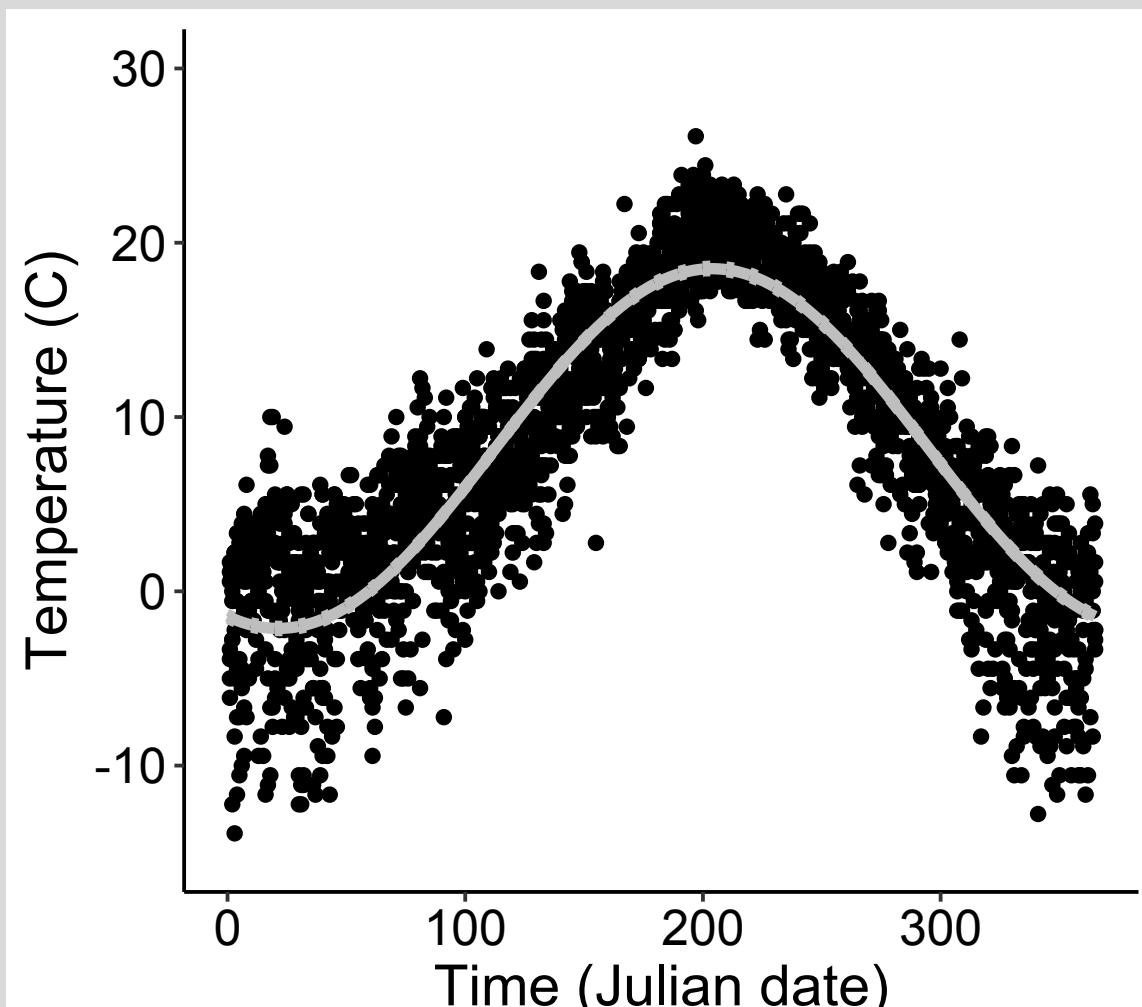
Shedding rate (from cell culture)

$$\phi_{(t)} = \phi_{(0)} e^{\chi Temp(t)}$$



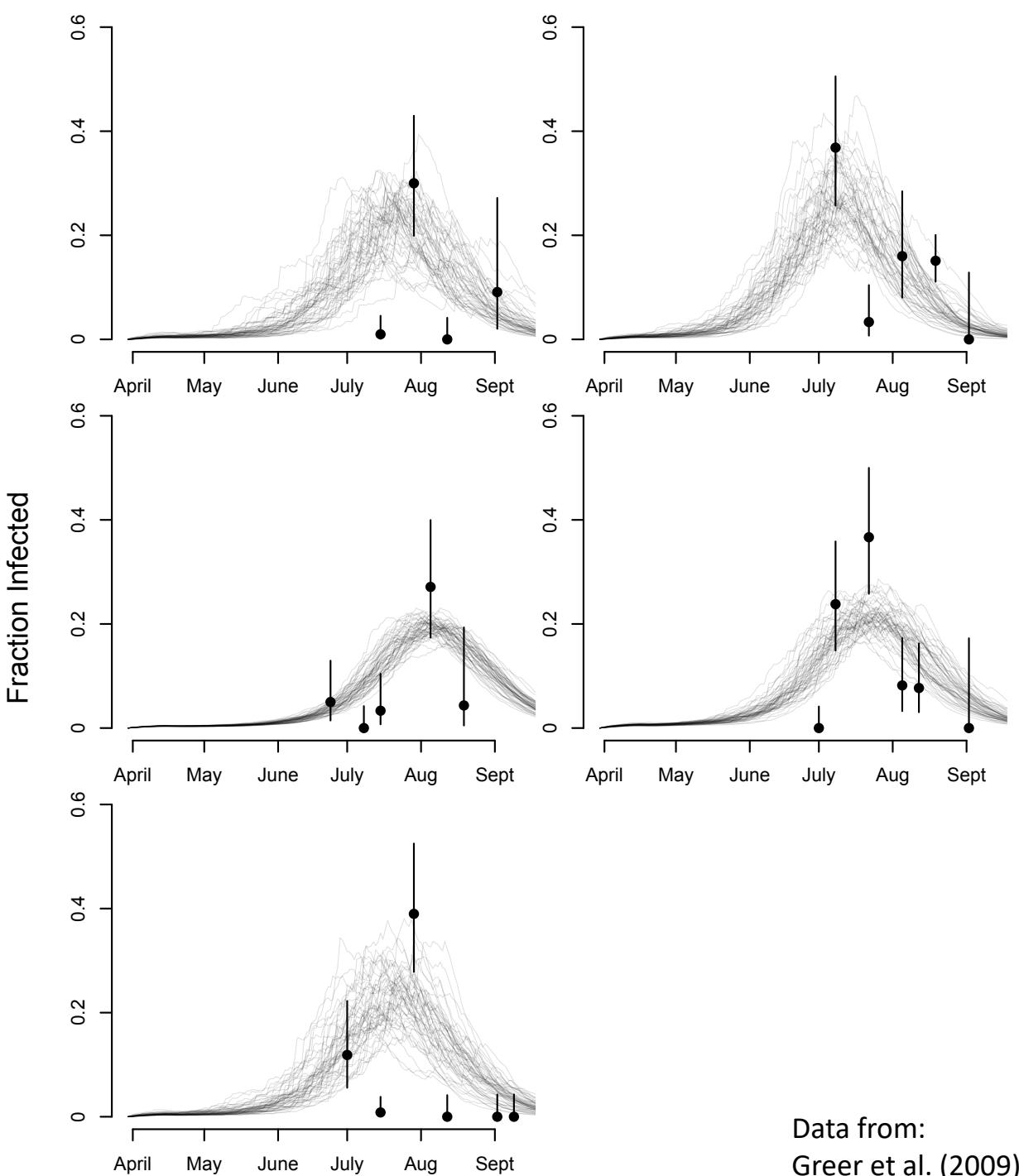
Arizona Temperature

Weather stations near North Rim



Fit models to epizootic data:
Do we need an effect of temperature to explain data?

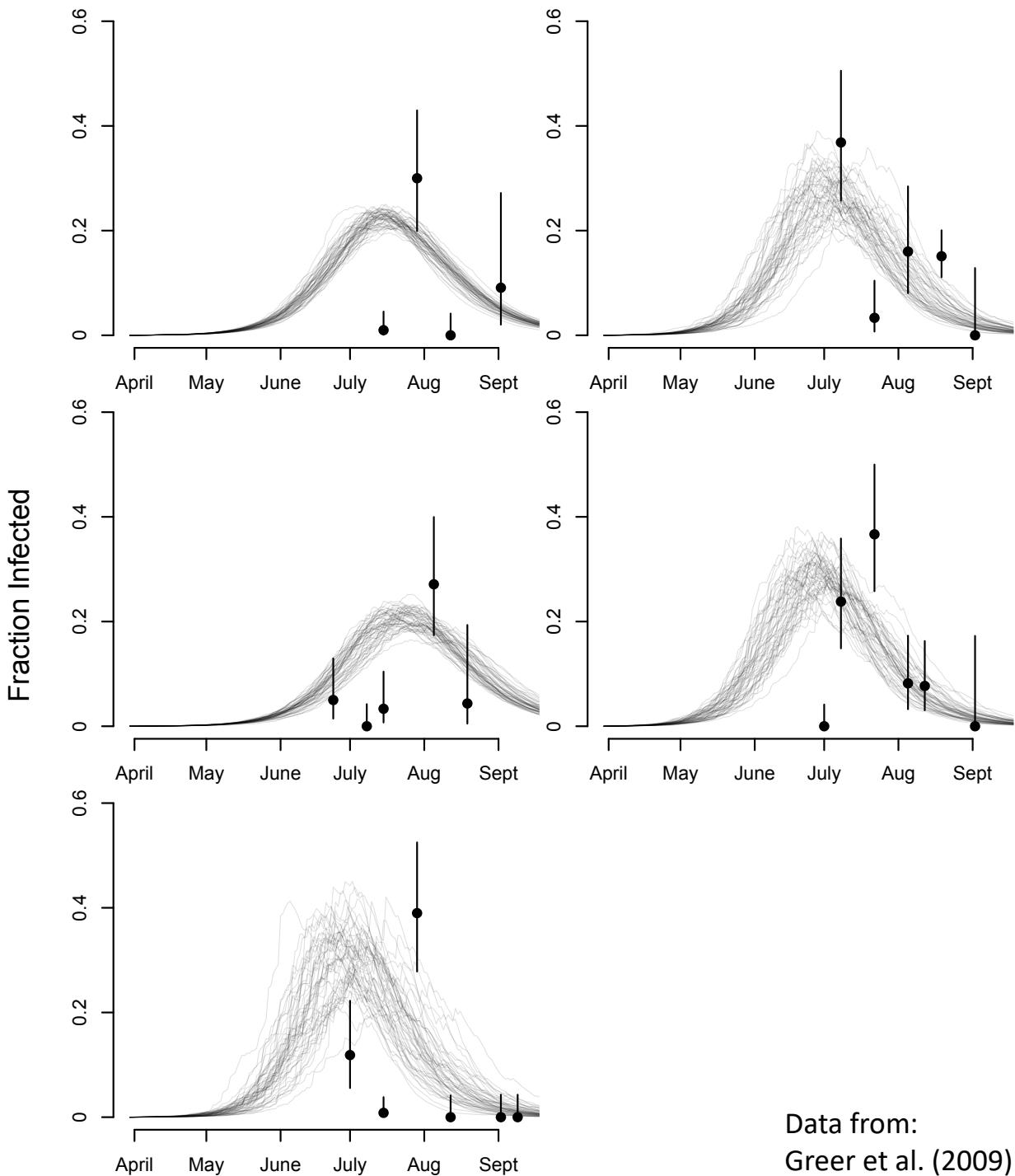
The model with a temperature-dependent shedding rate fits decently to the data.



Data from:
Greer et al. (2009)

The model with a temperature-dependent shedding rate fits well to the data.

However, a model without this effect fits equally well, assuming a very low initial viral concentration in the water.



Data from:
Greer et al. (2009)

Preliminary Conclusions

- There are insufficient data to support a necessary role of temperature-dependent shedding rate to explain epizootics.
- We need:
 1. Experimental measurements of shedding rate
 2. Estimates of initial conditions in the field:
 - a) Host densities
 - b) Viral concentrations

Future Directions: Temperature affects several vital rates

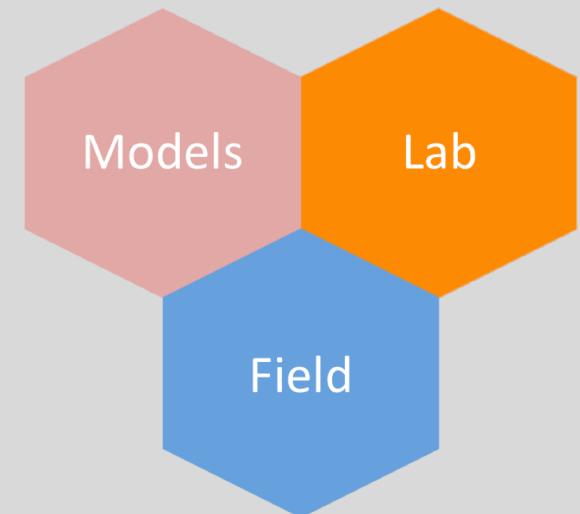
- Increases mortality rate of infected individuals
- Increases viral decay rate
- Causes wetlands to dry (increasing host density)

Future Directions

Understanding the community ecology of disease through space and time

Roles of host and viral community diversity (**Heterogeneities**)

Interaction between host heterogeneities and responses to temperature



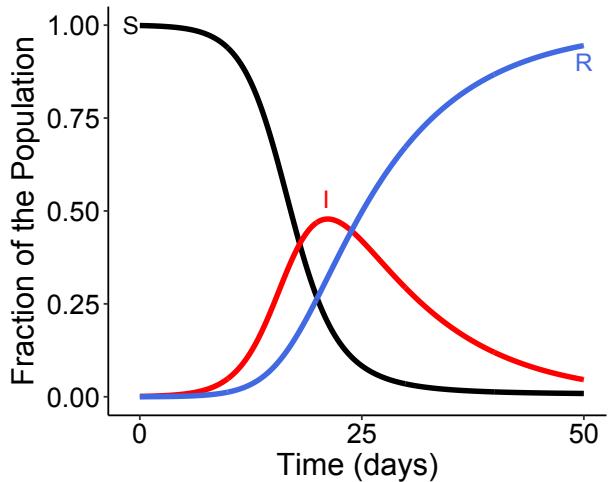


Infectious Disease Ecology & Epidemiology

INF 599-001 Spring 2019

T/Th 2:20p – 3:35p, SICCS 102

Contact: Joseph.Mihaljevic@nau.edu



This course will provide students with a basic understanding of disease modeling. We will derive and simulate fundamental insights about infectious disease dynamics, with topics including: predicting the size of epidemics, understanding cyclical disease outbreaks, and simulating spatially-explicit pathogen spread. Throughout, we will reinforce quantitative insights with biological concepts, computer-based assignments, and relevant readings from the literature.

Thank you!