Mechanistic transmission models and microbial control: Douglas-fir tussock moth and its baculovirus

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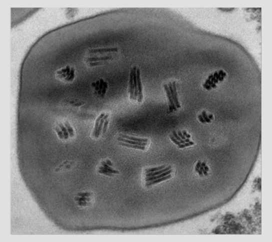
Background

- Douglas-fir tussock moth (DFTM) larvae:
  - Defoliate Douglas-fir and True fir across western North America
  - Reduce timber yields
- Controlled with a baculovirus with very narrow host range:
  - Tussock moth biocontrol-1 (TMB-1) spray



Problem:

- Virus-induced mortality in control plots often the same as in sprayed plots, despite very low initial virus levels in controls

Hypotheses:

1. Epidemiological theory: small number of infected hosts can lead to severe epizootic if hosts are dense enough in control plots.
2. Natural virus is more infectious than spray virus

Goal:

- Apply advanced mathematical epidemiology to the practical problem of biological pest control.

Approach

1. Test hypotheses by combining data and models
   - Model 1: spray and control plots only differ in initial host and pathogen densities
   - Model 2: Control plots have more infectious, wild-type virus than spray plots
   - Field and lab experiments to estimate model parameters and construct prior likelihoods for model fitting
   - Field experiments + epidemic data to choose best models.

2. Longer-term goal: use models to predict when it is more cost-effective to NOT spray virus.
   - Add tree growth
   - Impose different spray plans
   - Estimate costs

SEIR Model And Fitting Algorithm

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

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

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

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

- S: Susceptible hosts
- E: Exposed hosts
- P: Virus
- $\bar{\nu}$: Average transmission rate
- C^2: CV of transmission rate
- ϵ_t: Stochastic term
- m: Num. of exposed classes
- δ: 1 / kill rate
- μ: Decay rate

Fitting Models to Field-Collected Epizootic Data

Model Fitting Components:
1. Integrate the system of ODEs
2. Average likelihoods across realizations
3. Markov Chain Monte Carlo (MCMC)

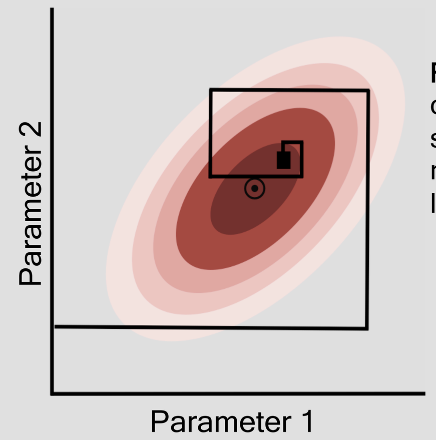


Figure 1. Schematic of 2-dimensional line search. Darker colors represent higher-likelihood posterior.

Line-search / MCMC:

Step 1: Line Search
- Large number of line searches (highly parallel)
- Most line searches get stuck, so keep top 10%
- Use top 10% in principal component analysis (PCA), to generate automated proposals for MCMC

Step 2: MCMC
- Propose parameter sets in PCA space
- Back-transform to original coordinate system
- Resulting proposals are uncorrelated, improving mixing

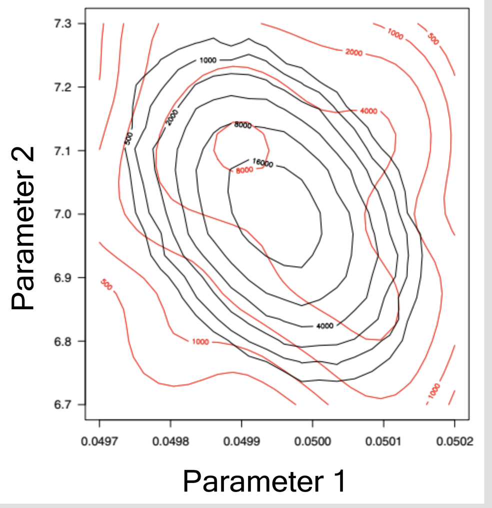


Figure 2. Estimation of posterior with line-search. *Black contours* show true posterior, while *red contours* show estimated posterior generated from line-search routine. (Adapted from Kennedy et al. 2014.)

Results

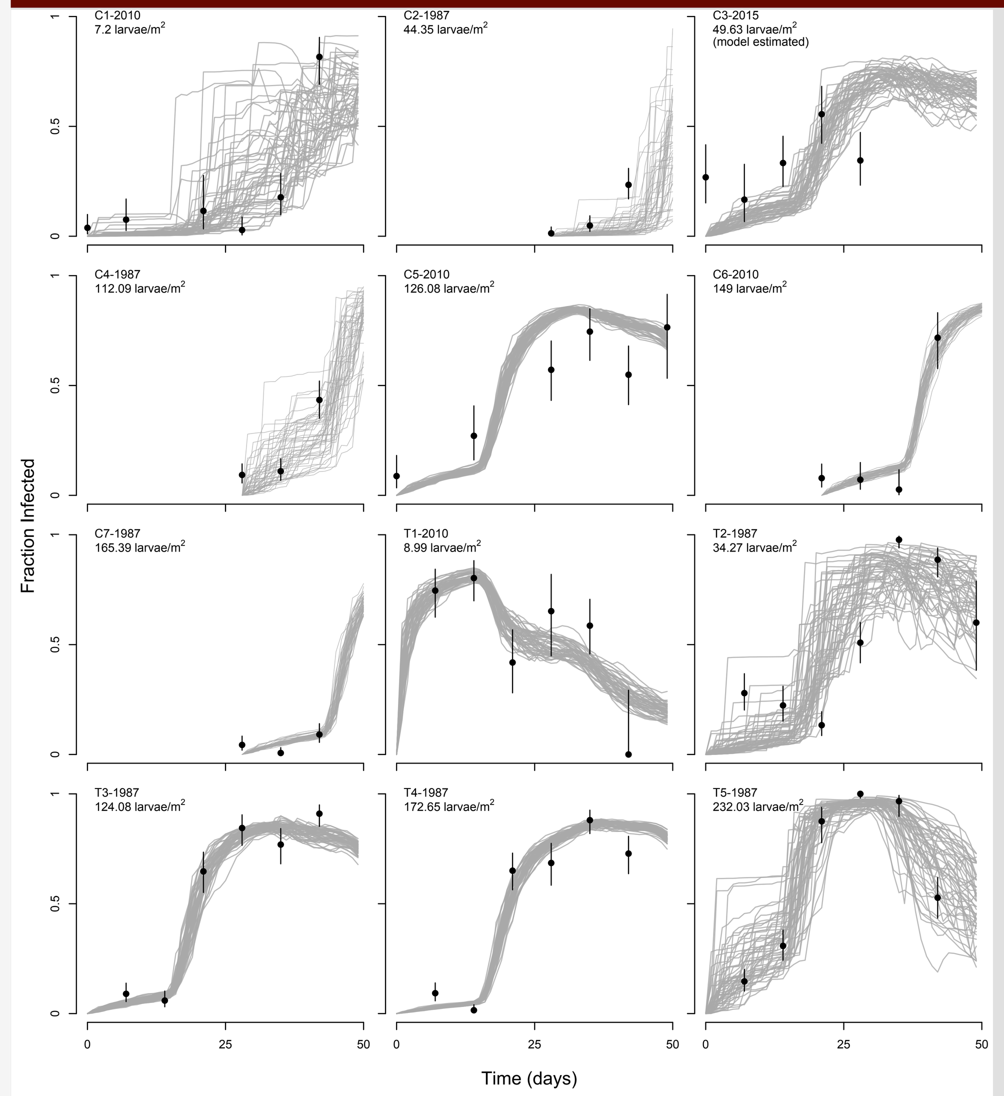


Figure 3. Stochastic simulations from the best fitting model. Data on fraction of infected insects (with 95% CI) are from past spray programs and natural epizootics. C=Control plot, T=Treatment plot.

1. The most parsimonious conclusion is that sprayed virus does not have a different transmission rate than naturally occurring virus.

Model Type	Informative Priors	pWAIC	WAIC	ΔWAIC
1 $\bar{\nu}$	δ	6.54	407.12	0
	No Inform.	7.10	408.80	1.68
	K, ratio, δ	7.06	409.28	2.16
	K	8.71	411.28	4.16
	K, ratio, δ, $\bar{\nu}$	7.61	411.61	4.49
	ratio	8.41	413.78	6.67
	$\bar{\nu}$	8.14	415.00	7.88
2 $\bar{\nu}$, Spray & Control	K, ratio, δ	9.14	411.31	4.19
	No Inform.	8.67	411.41	4.29
	$\bar{\nu}$, TMB-1	9.52	415.79	8.67

Table 1. Selecting between competing models. Smallest WAIC indicates best model. ΔWAIC values of greater than 4 identify models that should be rejected.

2. Experimental estimates of parameters are similar to estimates derived from fitting to whole-forest epizootic data, which is impressive given the difference in spatial scale.

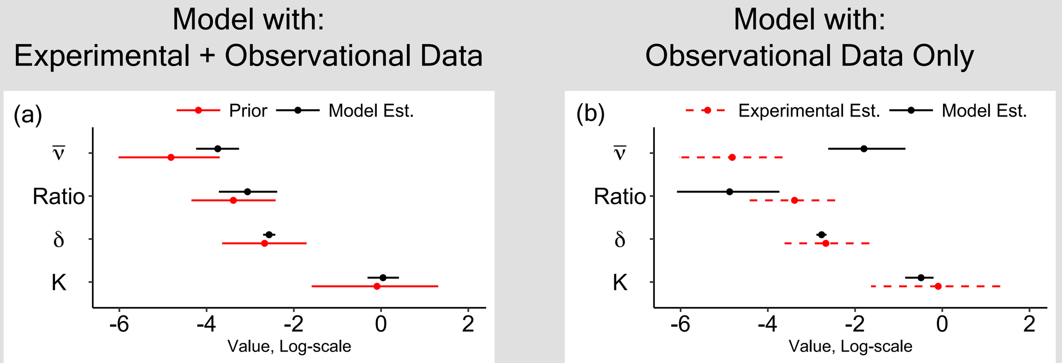


Figure 4. (a) Experimentally-derived prior likelihoods compared to model (marginal) posterior estimates. (b) In this case, all priors are uninformative, so we compare experimentally-derived estimates to model (marginal) posterior estimates.

3. Stochasticity in transmission rate results in a larger effective force of infection, and therefore larger epizootics.

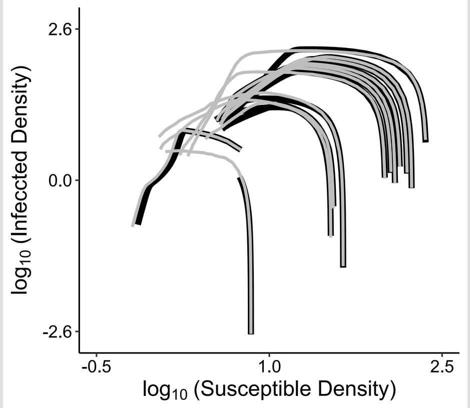


Figure 5. Simulated phase-portrait based on the starting conditions of the field plots (from Fig 3). The black lines represent the deterministic trajectories, while the gray lines represent the median trajectories from 1000 stochastic realizations. Stochasticity leads to larger epizootics.

4. At high host density, even a small amount of virus results in a large epizootic.

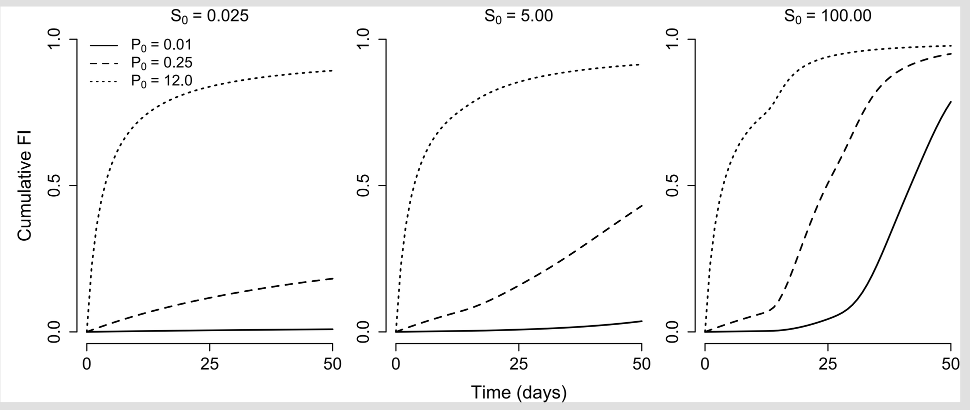
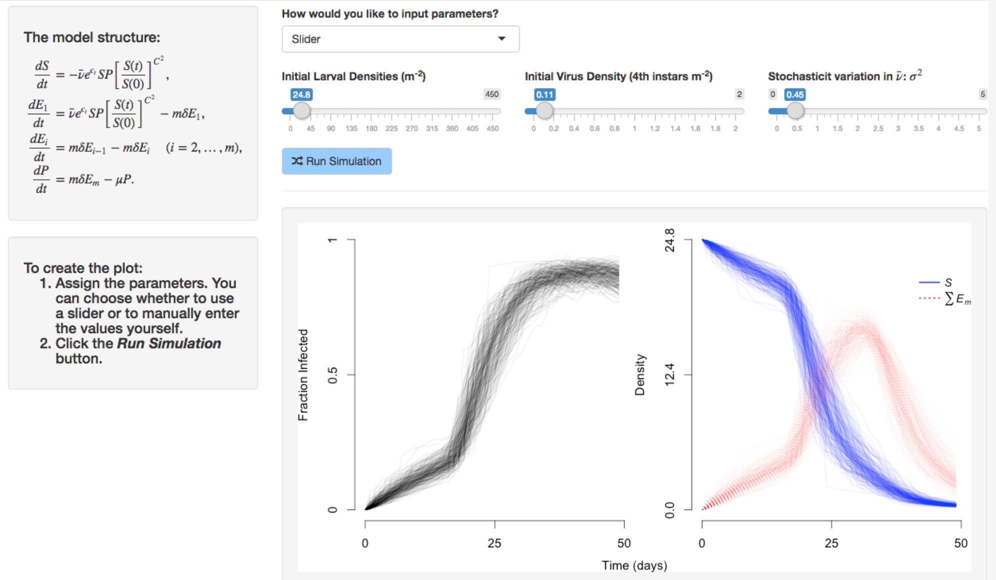


Figure 6. Simulated cumulative fraction infected with variable starting conditions. S_0 is the initial susceptible host density, and P_0 is the initial virus density in the environment.

Conclusions

- Theory is correct: high mortalities in control plots result simply from multiple rounds of transmission
- Management implications of models:
  1. Predicting when natural virus epizootics will occur is straightforward
  2. Control efforts should focus on intermediate insect populations with low initial virus densities

App for Managers



Future Directions

- Spatially-explicit model that includes tree biomass dynamics in response to defoliation, to predict long-term costs and benefits of virus spray for timber production and climate change mitigation