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

In infectious disease ecology, the classical mathematical models have provided valuable insights into infectious disease spread in animal populations, but sometimes these models can't accurately represent these dynamics mathematically. Part of the reason may be that the classical mathematical models have focused on general model predictions and didn't consider about mechanisms of disease transmission which mean detailed knowledge of epidemiological theory is not required to use these models. If the traditional mathematical models don't fit the data very well, we can't use these models to predict future disease spread, develop disease control strategies, and test hypotheses. Hence, we need to build new models based on hypotheses, test the models by using a combination of experiments(lab, field) and observational data(lab, field), and use the best models to make forecasts (see Figure1).

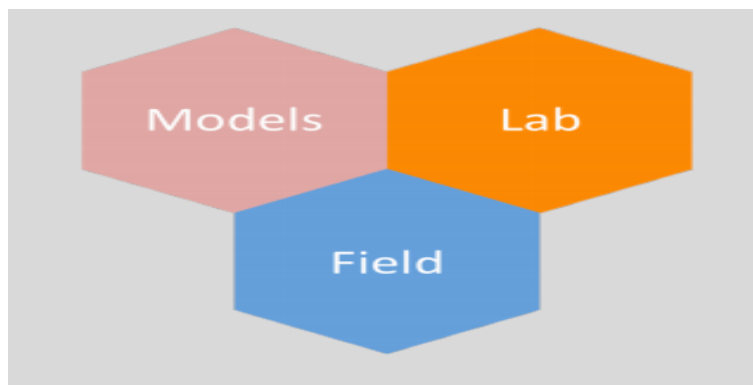


Figure1: Models, Field Experiment, and Lab Experiment

## Part I: Douglas-fir tussock moth nuclear polyhedrosis virus (NPV)

The traditional epidemiological model (SIR Model) can be used to describe natural baculovirus epizootics, which lead to population crashes in Douglas-fir tussock moth (DFTM) populations. However, the spray works in the research show that mortality in the unsprayed (control) plots the same as spray plots even though control plots had an initially undetectable level of virus infection (see Figure2).

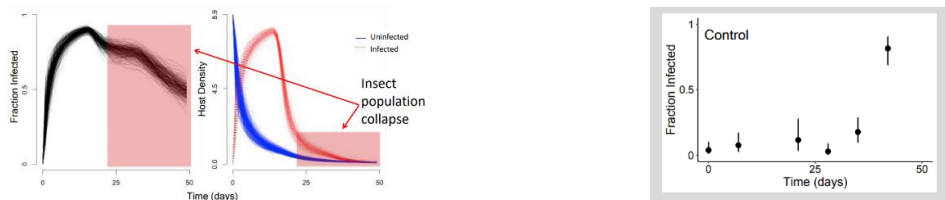


Figure2: The Spray Works

These findings suggest that additional considerations may be warranted in the research. Using a combination of experiments, observational data, and nonlinear-fitting algorithms, we find that when host density is high, even very low initial virus densities can lead to high cumulative infection rates, which means, in some cases, baculovirus sprays may have been applied to populations that would have collapsed from natural epizootics, even without any intervention. The research illustrates that there is a new stage between S stage and I stage and shows that SEIR models can provide valuable guidance for microbial control programs (see Figure3).



Figure3: SEIR Model

## Part II: Amphibian ranaviruses

The research use historical data to parameterize a model of ATV transmission in Arizona tiger salamander populations. However, the classical SIV model doesn't fit data very well. Amphibian ranaviruses grow faster at higher temperatures in cell culture, and viral shedding rate from infected animals also increases (see Figure 4). Hence, it is necessary to analyze an effect of temperature to explain data in this case. Using the model with a temperature dependent shedding rate, we fit well with the data. However, a model without this effect fits equally well, assuming a shallow initial viral concentration in the water. There are insufficient data to support a necessary role of temperature-dependent shedding rate to explain epizootics. So, we need to measure the shedding rate and estimates of initial conditions (host densities and viral concentrations) in the field.

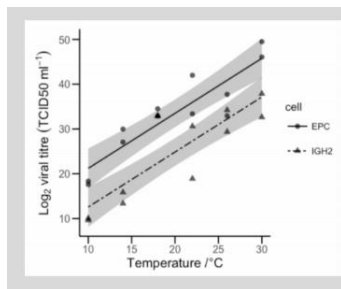


Figure 4: temperature-dependent rate

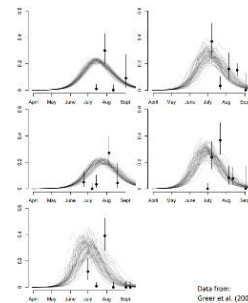


Figure 5: The model with a temperature-dependent

The epidemiological theory point out that host reproduction is essential over the long term is inarguable, but for many pathogens, seasonality prevents transmission for part of the year. Seasonality may avoid a transfer if there are seasons when the vector is not present, or when the pathogen does not co-occur with its host, or when the host is in a life stage that the pathogen cannot infect. In such cases, models of single epizootics (epidemics in animals) may be more useful than models of long-term disease dynamics.