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which the idea of combining models with data serves as a cornerstone for understanding insect epizootics.

In their original work, Anderson and May (1980) showed that insect population models that invoked host–pathogen interactions qualitatively displayed the same dynamics as observational data collected in the field. Following this, more rigorous methods of analyzing observational time series and field data began to take hold. These methods often advocated a likelihood-based approach that simply asked how likely were the data

methods outlined can be applied to many other biological systems. However, since all good mechanistic models need to be motivated by the biology of the system, baculoviruses represent a good place to start, given their importance in driving epizootic dynamics (Cory and Myers, 2003) and the use of mechanistic models in describing these dynamics (e.g., Dwyer et al., 1997; Elderd et al., 2013). To reiterate, while the biology and the associated models throughout draw on baculoviruses as examples, the methodologies discussed have quite a broad use in enhancing our understanding of epizootic dynamics as a whole.

Baculovirus infections begin when a susceptible individual consumes occlusion bodies (OBs), often containing multiple copies of the virus. If enough OBs are consumed, the individual becomes fatally infected. Sublethal or covert infections also occur (Roy et al., 2009), but at relatively low levels (Myers et al., 2000). Covert infections may contribute to the persistence of pathogens at low host densities (Roy et al., 2009) and function in a manner similar to vertical transmission between mothers and their offspring, which also allows pathogens to persist at relatively low host densities (Anderson and May, 1981). However, covert infections likely do not drive the boom-and-bust cycles associated with epizootic dynamics. If a lethal rather than a sublethal infection occurs, the infection process moves through a number of stages before the death of the host, which can release millions of OBs into the environment; transmission resulting from a sublethal infection would be minimal in comparison unless that infection 6neurthoTstlwhicneurthoTstld

degrading due to UV light exposure. The infected individuals then die and, after death, release OBs on to the leaf tissue. Once the first instars have died, healthy third or fourth instars are placed into the mesh bag. These individuals are then allowed to feed for a period of time. Afterwards, the larvae are collected and reared in individual cups until death or pupation (Dwyer et al., 1997; Elderd et al., 2008; Elderd and Reilly, 2014). Infection can be easily diagnosed visually given the drastic manner in which the infection process slowly consumes the larva. Additionally, since the OBs are quite large and can be seen under a light microscope (Elderd, 2013), any potential infections can be readily confirmed. In the simplest approach, one can manipulate the amount of pathogen in the system (the independent variable) and record the fraction of insects surviving (the dependent variable). Thus, experiments that manipulate multiple factors such as temperature and the amount of pathogen in the system (Elderd and Reilly, 2014) can be readily performed. The data produced can then be combined with any suite of models to test the associated hypothesis.

12.3 M D a T a : A S E

The models used to understand short-term epizootic dynamics associated with a single event can be traced back to Kermack and McKendrick (1927), who developed the SIR model to describe epidemic dynamics. Instead of SIR dynamics, baculovirus systems consist of susceptible individuals, infected individuals, and pathogen, since there is little evidence that infected individuals recover. If the simplifying assumption is made that all baculovirus infections are lethal, we need only consider the number of susceptibles and the amount of pathogen in the system, since all infected individuals eventually become pathogen (Dwyer et al., 2000). This assumption is met by the experimental methods described earlier. Mathematically, the equation for the susceptible larvae takes the form of the following differential equation:

$$\frac{}{} = -\beta \qquad (12.1)$$

Here, the change in susceptible larvae over time is simply a product of the disease transmission coefficient β times the number of susceptibles and the amount of virus in the system . The transmission parameter β encompasses the whole of the infection process and can be thought of as the fraction of encounters between the virus and a susceptible larva that leads to an instantaneous infection. As with all models (empirical, mechanistic, or simulation-based), it is important to consider all the assumptions. The main ones for the model of susceptible populations given here are that per capita transmission $\left(\text{i.e., } \frac{1}{}\frac{}{}\right)$ is linear and that all individuals are equally susceptible to becoming infected. Relaxing this assumption, or changing the model structure to better fit the biology of the system and the data, leads to new insights into the transmission process. Equation 12.1, however, serves as a useful starting point.

By integrating equation 12.1 and using experimental data, estimates of the transmission rate β can be easily calculated. In an experiment, the amount of virus or the number of cadavers in the system at the beginning of the experiment (0) is known, as

is the initial number of susceptibles in the experimental treatment (0). Here, 0 refers to the start of the experiment. After conducting the experiment until time , the number of susceptible individuals (i.e., the number of individuals that pupate rather than die from an infection) is also known, $( )$. These data can be easily plugged into the integral of equation 12.1, which is integrated from time 0 to

linear model held true (equation 12.1). A simple solution to this problem would be to raise the number of susceptibles or the amount of virus by a power (Hochberg, 1991), which would result in a nonlinear model that could better fit the data. This phenomenological model then takes the form:

$$\frac{}{} = -\beta \quad , \tag{12.3}$$

Here, and are the nonlinear effects on transmission of susceptible and infected population densities, respectively (Hochberg, 1991). However, while this power model will fit the nonlinear data better, the biological mechanism or mechanisms driving the nonlinear fit remain unknown. In this instance, what exactly does or mean from a biological standpoint?

A potential mechanism that may drive the nonlinearity in infection rates goes back to one of the main of assumptions of the linear model: that all individuals have the same transmission rate β. In Dwyer et al. (1997), the authors assumed that individuals differ in their susceptibility to virus. Essentially, some individuals are more susceptible than average and others are less susceptible than average. Thus, there was not a single transmission rate, but a mean transmission rate with some variability about the mean rate. Therefore, the transmission rate became a distribution rather than a single point estimate. The modified equation accounting for differences in susceptibility (i.e., heterogeneity in the transmission rate) thus becomes:

$$\frac{}{} = -\bar{\beta}\left[\frac{( )}{(0)}\right]^{C^2} \quad . \tag{12.4}$$

Here, $\bar{\beta}$ is the mean transmission rate. The transmission rate is scaled by the ratio of the number of susceptibles currently in the population ( ) divided by the number of susceptibles at the start of the epizootic (0). The ratio is raised to the square of the coefficient of variation associated with the transmission rate. Integrating equation 12.4 results in:

$$-\ln\left[\frac{( )}{(0)}\right] = \frac{1}{2}\ln\left(1+\bar{\beta}^{2} \quad (0) \quad\right). \tag{12.5}$$

Here, is once again the time that the experiment ran. For equation 12.5, instead of estimating just β from the data, two parameters need to be estimated, $\bar{\beta}$ and . For any single level of heterogeneity, at low pathogen levels, highly susceptible individuals become infected and transmission rises quickly (Fig. 12.1, solid lines). However, as pathogen levels increase, transmission tapers off, since only highly resistant individuals remain in the population. As the heterogeneity in the population increases, the coefficient of variation increases, which results in fewer individuals becoming infected at the end of the epizootic as pathogen levels increase (Fig. 12.1). If, instead, decreases and goes to zero (i.e., little variability in), the dynamics become similar to the linear equation (equation 12.2). While equation 12.5 was developed with epizootics in mind, it borrows from work by Anderson and May (1991) on HIV spread and how varying contact rates influence HIV transmission. Thus, equation 12.4 represents

another example of the give and take between epizootiological and epidemiological research.

Once a model is developed, it is important to test it. Dwyer et al. (1997) exemplified this approach by showing the stepwise process of confronting models with data. In a series of experiments on the invasive gypsy moth ( ) and its species-specific baculovirus, Lymantria dispar multinucleopolyhedrovirus (LdMNPV), the authors tested whether the linear (equation 12.1) or the nonlinear (equation 12.4) model explained the data better, using a series of experimental epizootics. However, it should be noted that baculoviruses do not represent the only pathogen in the system. , a fungal pathogen, also infects gypsy moth larva (Hajek, 1999), but infection rates can be either density-independent (Liebhold et al., 2013) or density-dependent (Hajek et al., 2015) according to the weather conditions (Hajek and van Nouhuys, 2016). LdMNPV, unlike , is always strongly density-depend

models to the data remained a problem until information theory began to gain a foothold in the wildlife literature (Anderson et al., 2000) and was highlighted in two influential books (Hilborn and Mangel, 1997; Burnham and Anderson, 2002).

12.4.1 Akaike Information Criterion

An information-theoretic approach to data analysis became widely used after the publication of Burnham and Anderson (2002). This approach allows a researcher to compare multiple models (i.e., alternative hypotheses) and determine which best fit the data. This is in direct contrast to classical statistics, which focuses on either accepting or rejecting a null hypothesis. The rejection of the null hypothesis simply means that the

to feed. After 2 days, we collected the larvae and reared them until pupation or death.

information about the system. The basis for the approach stems from Bayes' theorem, which states:

$$(\Theta \mid \text{Data}) \propto \pi(\Theta)\mathcal{L}(\text{Data} \mid \Theta) \tag{12.9}$$

where the posterior probability of the model parameters Θ given the data is proportional to ($\propto$), the prior probability of the parameters $\pi(\Theta)$ times the likelihood of the data given the model parameters $\mathcal{L}(\text{Data} \mid \Theta)$. In the past, the implementation of a Bayesian approach was often limited due to the complexity of the computations associated with the analysis. Recently, a proliferation of Bayesian books with ecological perspectives (e.g., Clark, 2007; Kéry, 2010; Hobbs and Hooten, 2015) and the availability of freeware programs (e.g., WinBugs, JAGS, STAN) have made Bayesian approaches much more accessible.

A distinct advantage of Bayesian methods is that they provide a framework for incorporating prior information about a system (e.g., preliminary studies), which is especially valuable when data are sparse. Typically, prior information enters into the classical analysis framework in the discussion when the authors state whether their current findings are similar to or different from those of previous studies (Hille Ris Lambers et al., 2005). In a Bayesian approach, the prior contains quantitative information and becomes a parameter in the analysis ($\pi(\Theta)$ in equation 12.9). If no prior information is available, vague priors can be used, which contain relatively little information. Explicitly stating a prior can be controversial to some, but if individuals are uncomfortable selecting a prior, the easiest way to minimize prior influence is to overwhelm it with data (Hobbs and Hooten, 2015). However, the use of informed priors makes the most of previously hard-won data and represents a powerful approach to developing mechanistic models for understanding epizootic dynamics.

A fundamental difference between a Bayesian approach and more classical approaches stems from the difference in how the parameters are treated. Classic frequentist approaches assume that a parameter's value is fixed and that the exact estimate becomes better resolved as sample size increases (Hobbs and Hooten, 2015). In contrast, Bayesian approaches assume that a parameter is a random variable drawn from a distribution. This is the difference between a single value for quantifying disease transmission rates, which is estimated with increasing precision, and a distribution of uncertainty reflecting the inherent variability of the transmission rate (Ellison, 2004; Hobbs and Hilborn, 2006). A more in-depth examination of Bayesian analysis from a philosophical perspective, as touched upon earlier, can be found elsewhere in the literature (e.g., Dennis, 1996; Ellison, 1996, 2004).

12.5.1 Fitting a Bayesian Model

For the linear model (equation 12.1), and assuming there is no difference in the treatment effects, a simple Bayesian model can be constructed such that:

$$\sim \text{binomial}(\ ,\ ), \tag{12.10}$$

$$\ln(\ ) = -\beta \quad (0) \quad , \tag{12.11}$$

$$\beta \sim \text{lognormal}(0{,}1000). \tag{12.12}$$

The number of survivors or non-infected larvae is distributed ($\sim$) binomially with a probability π given an initial number of healthy larvae n. Here, π is simply the fraction of uninfected larvae ($I(t)/I(0)$). Thus, equation 12.11 is equivalent to equation 12.1. The disease transmission rate β has a prior probability that is log normally distributed with a mean of 0 and a variance of 1000. Thus, the prior is considered vague and contains little information. The resulting posterior for each replicate i becomes:

Posterior

$$(\beta \mid i) \propto \left(i \mid -\beta (t), i \right)$$

visually or can be used to compute a Bayesian p-value (′), which quantifies the fre-

the same across treatments. Note, for the WAIC, that there are no equivalent metrics associated with model comparisons, such as weights used in the AIC (see earlier). However, the use of the WAIC continues to be developed and refined. When applied to fall armyworm virus data, the model results show that the coefficient of variation increases as temperatures increases, which results in an increase in overall transmission at higher cadaver densities (Fig. 12.2). Using the WAIC, the same conclusion can be drawn: that when temperatures rise the coefficient of variation associated with transmission declines and the dynamics become more and more similar to linear transmission dynamics (Elderd and Reilly, 2014). At the end of the day, both the AIC and the WAIC result in the same best model. The advantage of using a Bayesian framework becomes more readily apparent as the models considered become increasingly complicated.

12.6 L -T D a

The focus, so far, has been on single occurrences of a high prevalence of disease in a population (i.e., a single epizootic). Considerable research also focuses on modeling the long-term dynamics of insect populations driven by semiregular epizootic events. As this research has shown via the use of mechanistic models, epizootics drive or help drive the boom-and-bust population cycles often associated with insects, particularly those of economic concern.

As previously mentioned, Anderson and May's (1980) seminal paper combined ideas from two often disparate fields of research: predator–prey dynamics and epidemiology. Most previous efforts in modeling disease outbreaks focused on single epizootic events. These models are best exemplified in the epidemiological literature as the SIR models (Kermack and McKendrick, 1927), in which a main assumption is that the overall popu-

May, 1979). This approach works well with questions focused on near-term consequences, such as, "How many individuals will become infected over the course of an epizootic?" On the other hand, predator–prey models focus on the long-term population dynamics of prey and their predators, which are based on the classic work of Lotka (1932) and Volterra (1926). Anderson and May used ideas from both fields to construct a model showing that larch bud moth ( ) outbreaks could be driven by host–pathogen interactions (Anderson and May, 1980). Surprisingly, prior to their work, ecologists generally ignored the ability of pathogens to control the population dynamics of an insect (Anderson and May, 1981). Interestingly, more recent work on the same larch bud moth system has shown that parasitoids, not pathogens, drive the boom-and-bust cycles (Kendall et al., 1999; Turchin, 2003). When expanding the model to include spatial dynamics, dispersal, along with plant quality, can play an important role (Bjørnstad et al., 2002). The change in the driver of the cycle from the pathogen to the parasitoid exemplifies the importance of continually confronting observational data with mechanistic models and modifying a model as new data and new hypotheses emerge.

12.6.1 Long-Term Dynamics: Confronting Models with Data

For the univoltine gypsy moth, the short-term dynamics associated with epizootics during the larval phase and the long-term dynamics associated with adult reproduction can be considered separately. First, the epizootic occurs (a within-generation process), and then reproduction occurs (a between-generation process). A number of mechanistic models have been developed to describe this within- and between-generation process (e.g., Dwyer et al., 2004; Bjørnstad et al., 2010; Elderd et al., 2013). The general gestalt of these models is summarized nicely by Fuller et al. (2012).

To start off, consider the short-term or within-generation dynamics, which are governed by a series of differential equations that track the entirety of the epizootic process. The equations are:

$$\frac{}{} = -\overline{\beta}\left[\frac{(\,)}{(0)}\right]^{C^2} \quad , \tag{12.15}$$

$$\frac{1}{} = \overline{\beta}\left[\frac{(\,)}{(0)}\right]^{C^2} \quad - \quad \delta_1 , \tag{12.16}$$

$$\frac{}{} = \quad \delta_{-1} - \quad \delta \quad (\ = 2, \ldots, \ ), \tag{12.17}$$

$$\frac{}{} = \quad \delta \quad - \mu \quad . \tag{12.18}$$

Here, the equivalent terms have the same meanings as before (see equation 12.4). A major change from the classic SIR model is reflected in the fact that there is now an

class in the model, some larvae will instantly become pathogen, as exposed individuals continually move at an exponential rate out of the single exposed class (Keeling and Rohani, 2008). By allowing for total stages, the infected stages becomes a sum of exponential distributions, which is a gamma distribution with a mean of $1/\delta$, where δ is the average speed of kill, and a variance of $1/\ \delta$. The number of stages depends upon both the mean and the variance estimates of the speed of kill. For gypsy moth larvae, the best estimates are $1/\delta$ = 12 days and = 20 (Fuller et al., 2012). To reiterate, equations 12.15–12.18 only describe the within-season dynamics of the insect host when it is susceptible and succumbs to the baculovirus.

Long-term or between-season dynamics of the host population track host reproduction after the epizootic ends. Recall, the epizootic ends either due to the uninfected individuals pupating or due to epizootic burnout (Dwyer et al., 2000; Fuller et al., 2012). At the end of the epizootic, the equations describing the long-term dynamics are:

$$+1 = \lambda \left[1- \left(, \right)\right]\left(1-\frac{}{^2+\ ^2}\right), \tag{12.19}$$

$$+1 = \left(, \right)+\gamma \ . \tag{12.20}$$

Here, and are the densities of the hosts and the cadavers before the epizootic in generation and $\left(, \right)$ is the fraction of the larvae that become infected (equations 12.15–12.18). The net reproductive rate is λ. For outbreaking insects, population densities are kept at low levels during inter-outbreak periods by generalist predators or parasitoids (Dwyer et al., 2004). For gypsy moth populations, this can take the form of a Type III functional response. The fraction surviving predation is represented by the term $1- \ /\left(^2+\ ^2\right)$, where is the maximum predation rate and is the saturation constant. Baculovirus densities depend upon the survival of virus derived from the current generation and the survival of virus γ from previous generations. While it is likely that sublethal or covert infections play only a small role in the long-term dynamics, the preceding model also adequately describes covert infections. It assumes that some fraction of the virus survives from one generation to the next, which could be derived from covert infections. As long as this fraction is density-independent, the model provides an accurate accounting of covert infections (Elderd et al., 2013). Over the course of multiple generations, the modeling consists in stringing together the short-term (e.g., one season for univoltine gypsy moths) epizootic followed by adult reproduction, which sets the stage for the next epizootic.

12.6.2 Time-Series Diagnostics

While fitting models to data using results from short-term experiments draws directly from the standard statistical literature, long-term data sets represent a different problem from an analytical perspective. They are often observational and constitute a classic example of an "inverse problem" (Kendall et al., 1999), such that the data collected may arise due to many different mechanistic processes (e.g., intraspecific density-dependent regulation vs. host–pathogen interactions). How best to decide which mechanisms may

be responsible for the observed data is central to understanding what drives the boom-and-bust cycles associated with long-term epizootic dynamics.

For many of these observational data sets, the data are not directly fitted to the model. For instance, a number of papers exploring gypsy moth long-term dynamics use defoliation data as a proxy for gypsy moth population numbers (e.g., Dwyer et al., 2004; Elderd et al., 2008; Bjørnstad et al., 2010). To compare the model output with the observational data, authors often rely on matching various metrics associated with the time series of the data (e.g., average period between peak outbreaks or defoliation events) with the model output. Directly fitting the model to the data becomes increasingly problematic if the dynamics of the system are chaotic, since the model and the data are sensitive to initial conditions (Dwyer et al., 2004). Thus, instead of directly fitting the data to determine which model drives the observed dynamics, "time-series" probes are advocated (Kendall et al., 1999; Turchin, 2003).

Kendall et al. (1999) were among the first advocates in the ecological literature to push for the use of "time-series" probes by combining time-series statistics with mechanistic population models. Previous to this paper, most time-series analyses consisted of fitting nonmechanistic models that could be considered biologically naïve to observational data. On the other side of the coin were the theoretical population ecologists

a method of choice (e.g., a Bayesian approach), and the results are compared to the known simulated truth (Kéry, 2010).

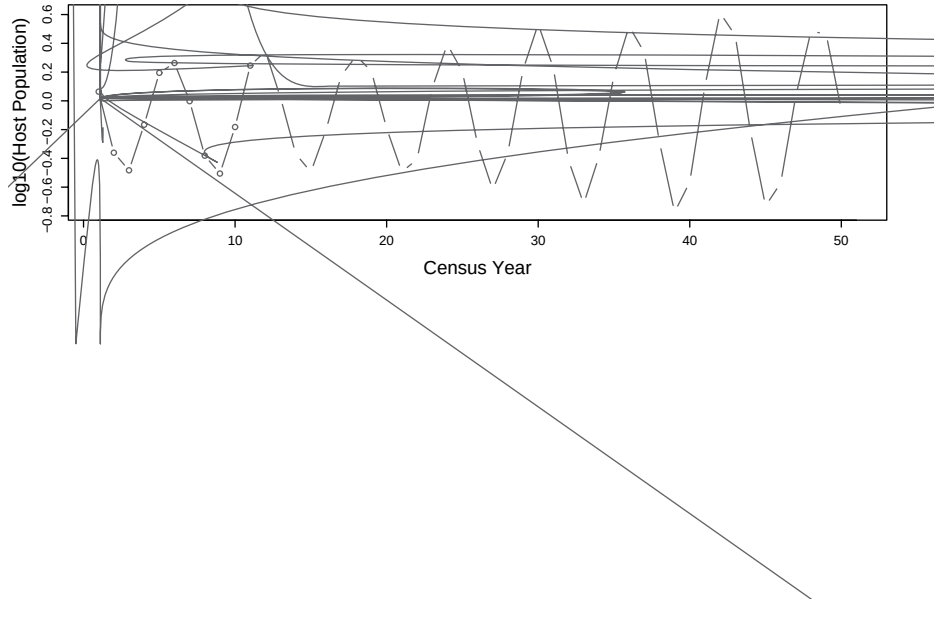
To examine the methods used in Ives et al. (2008) from a Bayesian perspective, data were simulated using the nondimensionalized version of the burnout approximation model in Dwyer et al. (2000). The simulation used three equations to represent the dynamics, as follows:

$$_{+1} = \lambda\varepsilon_{} _ \left[1 - \left(_ , _ \right)\right], \tag{12.21}$$

$$_{+1} = \phi _ \left(_ , _ \right) + \gamma _ , \tag{12.22}$$

$$1 - \left(_ , _ \right) = \left[1 + {}^{2} _ \left(_ , _ \right) + _ \right]^{-1/ {}^{2}}. \tag{12.23}$$

Here, ϕ is the product of pathogen survival and mean susceptibility of newly emerging larvae (Dwyer et al., 2000) and ε is a log normally distributed random variable with a median of 1 and a standard deviation of σ. For simplicity of presentation, γ is set to 0 and there are no generalist predators in the model. To understand the boom-and-bust dynamics of the insect host population given the preceding, there are only three parameters in the nondimensionalized model that matter: λ, , and ϕ. All of the other parameters simply move the population mean up or down, and do not affect the period or amplitude of the population cycles. In terms of the simulated data, the analysis only uses the time series associated with the host population, , as an input. Overall, the Bayesian approach



epizootics in insect populations, they also provide a useful tool for asking questions of an applied nature.

12.8 C

The use of models to understand epizootic dynamics has a long history in the ecological literature. Much of the past debate concerning which methodology is best suited for moving the field forward centered on the historic false dichotomy between empirical and theoretical approaches, while sometimes invoking simulation-based methods. However, the ability to confront models with data has led to new and exciting developments in the field, since models can now be used as hypotheses to drive research questions. While using the preceding techniques and ideas may seem easy to some and daunting to others, they do not necessarily need to be mastered by all. Instead, they represent a framework to begin a conversation about questions that can be answered, how to design empirical studies, and how best to use the data produced. The reason the false dichotomy of empiricism and theory continues to blur stems from more individuals being able to speak in multiple languages. Thus, mastering each technique is not essential, but being able to communicate across the false divide is. As the dialogue advances and individuals speak across their own expertise, the biology of the system becomes better connected to the mechanistic framework, which leads to a better understanding of what drives the epizootic process.

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