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Author(s): Greg Dwyer and Joseph S. Elkinton

Source: *Journal of Animal Ecology*, Vol. 62, No. 1 (Jan., 1993), pp. 1-11

Published by: [British Ecological Society](#)

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Using simple models to predict virus epizootics in gypsy moth populations

GREG DWYER and JOSEPH S. ELKINTON

Department of Entomology, University of Massachusetts, Amherst, MA 01003, USA

Summary

1. Biologists have made little use of recent advances in the mathematical theory of the dynamics of insect pathogens, because of difficulties with parameter estimation and misgivings about the simplicity of the models in question.
2. We use an existing simple model for the dynamics of insect pathogens, slightly modified both to provide greater accuracy and to allow for more straightforward parameter estimation.
3. Focusing on the nuclear polyhedrosis virus (NPV) of gypsy moth (*Lymantria dispar* (L.)), we estimated each of the model parameters independently, estimating three of the four model parameters from the literature.
4. To estimate the rate of transmission, we present an experimental protocol which involves fitting a reduced version of the model to data from a small-scale transmission experiment.
5. Without circularity or curve-fitting, we tested the model with literature data giving initial densities and weekly NPV mortality for epizootics in eight gypsy moth populations on 4–9 ha plots in Massachusetts, USA.
6. The model predictions are reasonably accurate for five of the eight populations, suggesting that gypsy moth NPV dynamics within a season are driven by a small number of biological processes.
7. The three populations for which the model did poorly began the season with low host densities yet gave rise to more severe epizootics than predicted by the model. This indicates that standard assumptions about disease transmission may not hold for gypsy moth NPV dynamics at low densities; specifically, we suspect that density-related changes in larval behaviour result in higher NPV transmission at low density.
8. These results suggest that simple models of the dynamics of animal pathogens can be used to make quantitative predictions about disease dynamics in the field.

Key-words: *Lymantria dispar*, NPV, mathematical model, host–pathogen dynamics, disease transmission.

Journal of Animal Ecology (1993) **62**, 1–11

Introduction

The mathematical theory of infectious diseases of animals has made important advances in the past 13 years, beginning with Anderson and May's seminal papers (1978, 1979; Levin & Pimentel 1981; Murray, Stanley & Brown 1986; Brown 1984; Andreasen 1989; Hochberg & Holt 1990). Surprisingly, however, this theory has been little used; although field studies of animal diseases are increasingly common (Gill & Mock 1985; Dobson & Hudson 1986; Myers 1990; Dwyer 1991a), field studies related to theory are extremely rare. This under-utilization is due primarily to problems of model simplicity and para-

meter estimation. First, continued research on pathogens has revealed a variety of factors that may affect disease dynamics; since few of these factors are included in simple mathematical models, biologists have been mistrustful of the models (Onstad *et al.* 1990; Onstad & Carruthers 1990). Second, because the models in question are quite simple, their parameters can only rarely be interpreted in terms of easily measurable biological quantities (Anderson & May 1980). This has made it difficult for biologists to estimate model parameters, so that quantitative application of the models has been difficult for non-specialists.

Insect virology provides a clear example of these

problems. Anderson & May (1980, 1981) presented one of the first simple mathematical models for the dynamics of insect pathogens. Their model is

$$\frac{dS}{dt} = r(S + I) - \nu PS \quad \text{eqn 1}$$

$$\frac{dI}{dt} = \nu PS - (\alpha + b)I \quad \text{eqn 2}$$

$$\frac{dP}{dt} = \lambda I - [\mu + \nu(S + I)]P \quad \text{eqn 3}$$

where S is the density of susceptible hosts, I is the density of infected hosts, P is the density of pathogen particles, r is the reproductive rate of the host, ν is the transmission coefficient, α is the rate of disease induced mortality, b is the rate of non-disease mortality, λ is the rate of production of pathogen particles by infected hosts, μ is the decay rate of the virus, and t is time.

This model includes only a small number of processes: host density-independent reproduction and death, host disease-induced death, pathogen production by infected hosts, the breakdown of the pathogen in the environment, consumption of the pathogen by the host, and disease transmission. The model thus ignores much of what biologists have discovered can influence the course of a virus disease in an individual host. For example, host stage can affect the susceptibility (Watanabe 1987; Hochberg 1991) and infectiousness (Thompson & Scott 1979; Kaupp 1983; Teakle & Byrne 1989; Dwyer 1991a) of individual hosts. Chemical constituents of foliage consumed by hosts also can affect the susceptibility of individual hosts (Keating & Yendol 1987; Keating, McCarthy & Yendol 1990). Temperature can affect the time between host infection and death (Watanabe 1987).

Moreover, the difficulty of interpreting the model parameters makes parameter estimation difficult. The transmission parameter ν is especially problematic. Since, for insect viruses, transmission occurs when the host accidentally consumes the pathogen on contaminated foliage, transmission must be affected by, among other things, host behaviour. Although it may be possible to translate measurements of host behaviour into a value for ν , it is not obvious how one would go about doing so. Similarly, since susceptibility affects transmission, the transmission constant ν must be affected by host stage, host-plant foliage chemicals, and so forth, yet it is unclear how these factors could be translated into a value of ν .

Our contention is that the problem of parameter estimation has kept many biologists from seeing the usefulness of simple mathematical models of insect pathogens. Moreover, theoreticians themselves have focused on long-term predictions (Anderson & May 1981). Since long-term data sets are rare, this focus has led to a lack of comparisons between

theoretical predictions and the long-term behaviour of real systems that has further impeded progress. In the present paper we attempt to address these problems by: (i) slightly modifying Anderson & May's (1981) model to make interpretation of the parameters more straightforward; (ii) estimating each of the parameters for the nuclear polyhedrosis virus (NPV) of gypsy moth (*Lymantria dispar*), presenting along the way a protocol for estimating the transmission parameter ν ; (iii) using the model to predict disease epizootics within a season; (iv) comparing the model predictions to replicated time series of gypsy moth NPV mortality within a season. Since our model is only slightly different from equations 1–3, we have thus intentionally omitted many biological details; by doing so, we can assess whether these details are needed, or learn when they become important. Our model thus acts as a kind of null hypothesis, rejection of which may be accompanied by the development of new hypotheses.

Methods

NATURAL HISTORY OF THE HOST-PATHOGEN SYSTEM: GYPSY MOTH AND ITS NUCLEAR POLYHEDROSIS VIRUS

Gypsy moth is an economically important defoliator of deciduous forests in north-eastern North America, and it is spreading west and south from its point of introduction near Boston, Massachusetts, USA (Elkinton & Liebhold 1990). In Massachusetts, where our test data were collected, larvae hatch in early May from eggs laid the previous August. Larvae go through five instars in males, and six in females. In North America, females are flightless, so that the chief mode of dispersal is ballooning in first instars (Mason & McManus 1981). For reasons that are not yet understood, gypsy moth populations sometimes rise to high densities. These outbreak populations typically crash as a result of epizootics of the NPV. NPVs get their name from the polyhedral protein coat that encapsulates the viral capsids, with about 20–50 capsids per polyhedral inclusion body (Wood & Granados 1991). Inclusion bodies are capable of surviving for long periods outside the host (Podgwaite *et al.* 1979), but break down under the UV rays in sunlight (Benz 1987).

Within a season, the initial input of NPV polyhedra into a population is due to eggs that are laid on contaminated substrates (typically bark: Murray & Elkinton 1989, 1990). Larvae that hatch out of these eggs become infected, and die of the virus in the late first or early second instar, about 2 weeks after hatch (Woods & Elkinton 1987). The polyhedra produced by these infected larvae then lead to subsequent rounds of transmission among fourth and later instar larvae, which can result in very high levels of mortality due to the virus (Woods &

Elkinton 1987). An initially low level of NPV infection among early instar larvae can thus lead to high levels of infection in fourth and later instars, which is typically when most larvae become infected (Elkinton *et al.* 1990).

The model

Since epizootics essentially occur within a single gypsy moth cohort (Woods & Elkinton 1987), and since gypsy moth is univoltine, the model that we use describes the within-season dynamics of the NPV, without including host reproduction.

$$\frac{dS}{dt} = -vPS \quad \text{eqn 4}$$

$$\frac{dI}{dt} = vPS - vP(t - \tau)S(t - \tau) \quad \text{eqn 5}$$

$$\frac{dP}{dt} = \Lambda vP(t - \tau)S(t - \tau) - \mu P \quad \text{eqn 6}$$

where S is the density of susceptible hosts, I is the density of infected hosts, P is the density of the pathogen in the environment, v is the transmission constant, τ is the time between infection and death of the host, Λ is the number of pathogen particles produced by an infected larva, μ is the decay rate of the pathogen, and t is time. This model captures the bare essentials of the interaction between insects and their pathogens: an important characteristic of many insect pathogens is that the infectious stage of the pathogen is capable of surviving for long periods outside of the host (Evans & Entwistle 1987). In the present paper, we test the model only with data from an NPV, although the same model could be used for other pathogens with infectious stages that can survive outside of the host, such as fungi (Carruthers & Soper 1987). For NPVs, larvae become infected (at the rate vPS) by consuming inclusion bodies on contaminated foliage (adults cannot become infected; Evans & Entwistle 1987). Approximately 2 weeks (τ) later, the infected larvae die ($vP(t - \tau)S(t - \tau)$), releasing infectious particles into the environment ($\Lambda vP(t - \tau)S(t - \tau)$) to decay (μP) or infect more hosts (vPS).

Our model differs in three respects from the original version (Anderson & May 1981; for other variants, see Brown 1984; Hochberg 1989; Dwyer 1991b; Hochberg & Waage 1991). First of all, Anderson & May (1981) assume that the mortality rate of infected hosts is constant, so that the *distribution* of incubation times is exponential. Observations on individual larvae have demonstrated that there is a substantial time lag between infection and death (Evans & Entwistle 1987), and observations on epizootics have suggested that this time lag can be important for disease dynamics within a season (Dwyer 1991a; Woods & Elkinton 1987). Accordingly, we assume instead that there is an incubation time between infection and death. Because actual

incubation times of nuclear polyhedrosis viruses typically have a low variance (Dwyer 1991a) we assume that the incubation time is constant. Although similar time delays are observed in other diseases, including human diseases (Anderson 1979), they are often ignored (but see for example Andraesen 1989; Aron & May 1982).

Although the disease-induced mortality rate in the original model (equations 1–3) can be estimated easily as the inverse of the mean incubation time, our use of the incubation time has the additional advantage that its interpretation is slightly more obvious. It also allows us to use directly the number of pathogen particles produced by an individual host, Λ . This parameter is similarly easier to interpret, even though as Anderson & May (1981) point out, their pathogen production rate $\lambda = \Lambda\alpha$, which thus also can be estimated easily, given a value of α .

A second way in which our model differs from Anderson & May's (1981) model is that we are testing our model with data from a single season in a univoltine host insect, whereas they focused on the long-term dynamics of host and pathogen. We therefore neglect host reproduction, although the model could easily be extended to include multiple cohorts (for an example of how to construct a disease model for a host with non-overlapping generations, see May 1985). Finally, we leave out any loss of pathogen particles due to consumption by the host; our justification is that within a season this loss is slight compared to pathogen production and decay, and that compared with what else we are leaving out, this considerable mathematical complication is a minor consideration.

Predicting NPV mortality in gypsy moth

To compare model predictions to field data, we must first have independent estimates of the model parameters: Λ , τ , μ and v (Table 1). Three of the parameters can be obtained directly from the literature. The number of virus particles produced by an individual larva (Λ) can be estimated directly from infected larvae in the laboratory (Shapiro, Robertson & Bell 1986); since in the field the majority of larvae are infected from polyhedra produced from infected fourth instars (Elkinton *et al.* 1990), we use the value for this instar: about 2×10^9 (Shapiro *et al.* 1986). The incubation time of infected larvae (τ) in the field is about 2 weeks (Woods & Elkinton 1987).

The remaining two parameters, the disease decay rate μ and the transmission constant v , must be estimated at the population level. First, Podgwaite *et al.* (1979) quantified populations of gypsy moth NPV particles in the field at two different times after an epizootic, just after the epizootic and a year later, both in litter and soil. These data can be used to give an estimate of the decay rate of the pathogen in the following way. In the absence of new infections,

the model assumes that, in the absence of virus-caused deaths, the pathogen population will decay exponentially ($\frac{dP}{dt} = -\mu P$), so that

$$P(t) = P(0)e^{-\mu t} \quad \text{eqn 7}$$

where $P(0)$ is the initial pathogen population and $P(t)$ is the population at time t . This equation can be solved for μ to give

$$\mu = -\frac{1}{t} \ln \left[\frac{P(t)}{P(0)} \right] \quad \text{eqn 8}$$

We used an average of the μ values for the soil and the litter, to give $\mu \approx 3 \times 10^{-3} \text{ day}^{-1}$. We recognize that applying values for the soil and litter to what happens on the foliage may result in an unrealistically low value of the pathogen decay rate μ , but we have reason to believe that this value is at least approximately correct (see Results).

Our approach to estimating the transmission constant ν is a modification of a protocol developed by Dwyer (1991a,b). That is, we fit the model to short-term, small-scale data on a single round of NPV transmission in experimental gypsy moth populations in the field. Over a short enough time interval, a week or less, the decay of the pathogen is probably negligible ($<2\%$ loss for $\mu = 3 \times 10^{-3} \text{ day}^{-1}$), and $t < \tau$, so that $\frac{dP}{dt} \approx 0$ and the pathogen population density P is a constant $P(0)$. The model thus becomes

$$\frac{dS}{dt} = -\nu P(0)S \quad \text{eqn 9}$$

$$\frac{dI}{dt} = \nu P(0)S \quad \text{eqn 10}$$

This simpler model can be solved to provide an expression for ν in terms of the initial density of polyhedral inclusion bodies $P(0)$, the number of larvae infected by the end of the experiment $I(7)$, and the initial density of healthy larvae $S(0)$:

$$\nu = \frac{-1}{7P(0)} \ln \left[1 - \frac{I(7)}{S(0)} \right] \quad \text{eqn 11}$$

To fit equation 11 to data, we performed a field experiment in which we placed healthy and infected larvae onto branches of red oak trees (*Quercus rubra* (L.)) (in the Cadwell State Forest, Pelham, Massachusetts) and enclosed them in mesh bags for 1 week. To ensure that the healthy larvae were uninfected, they were reared in the laboratory on artificial diet until the start of the experiment. We infected the initially infected larvae by feeding them a solution of NPV inclusion bodies of sufficient concentration to ensure 100% mortality due to the virus. Each bag contained about 40 oak leaves; calculating leaf area with a computerized digitizer showed that this was equivalent to a leaf area of $\approx 0.05 \text{ m}^2$. Since transmission has been suggested to vary in a complex way with pathogen density

(Valentine & Podgwaite 1982), we used two treatment densities of infected larvae (high = 20, low = 5). Each treatment was replicated four times, with one replicate corresponding to an individual red oak tree, for a total of eight experimental bags. The infected larvae were first instars, while the healthy larvae were a mixture of third and fourth instars. Each bag contained 25 initially healthy larvae. This corresponds to a density of $500 \text{ larvae m}^{-2}$, which is within the range of observed outbreak densities (Campbell 1981). In addition to the eight experimental bags, there was a control bag that contained no infected larvae. No infections appeared in the control bag.

After 1 week, we removed all the initially healthy larvae to individual rearing cups in the laboratory to see how many had become infected as a result of contact with the virus particles released by the initially infected larvae. The results of the experiment are shown in Fig. 1. Transmission was higher at a higher density of infected larvae, resulting in average estimates of ν for the two treatments (see below) that were quite similar, suggesting that transmission does increase linearly with the density of the pathogen (Dwyer 1991).

Since we know the number of pathogen particles per larva, and since the initially infected larvae died shortly after the beginning of the experiment, we can convert the density of infected larvae that began the experiment into the initial density of pathogen particles, $P(0)$. Since we also know the initial density of healthy hosts $S(0)$ and the final density of infected larvae $I(7)$, we can calculate ν from equation 11. All the infected larvae were first instars, so the value that we use for the number of inclusion bodies per larva is that specific to first instars: $\Lambda = 4 \times 10^8$ (Shapiro *et al.* 1986). We first calculated the transmission constant ν based on pooling the replicates for each treatment (high: $1.13 \times 10^{-12} \text{ m}^2 \text{ day}^{-1}$, low: $1.77 \times 10^{-12} \text{ m}^2 \text{ day}^{-1}$), and then used the mean of the resulting two values ($1.45 \times 10^{-12} \text{ m}^2$

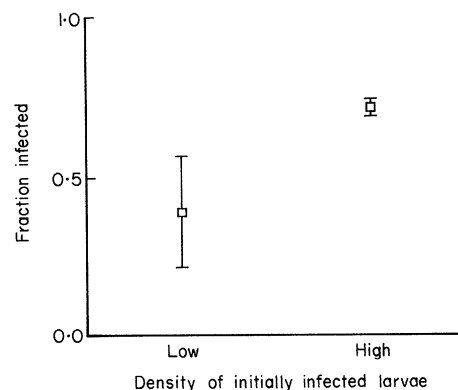


Fig. 1. Results of the experiment used to estimate the transmission constant ν . Fraction infected indicates the fraction of initially healthy larvae that became infected. Error bars indicate 1 SE.

day⁻¹). The values of all the parameters are given in Table 1.

Characteristics of the field plots

Given estimates of each of the parameters, we can use the model to predict the dynamics of NPV epizootics in gypsy moth populations. We compared the model predictions to time series of NPV mortality in gypsy moth larvae collected by Woods & Elkinton (1987) in five plots in 1983, 1984 and 1985, for a total of 8 plot-years. The plots were located in the vicinity of Otis Air National Guard base in Cataumet, Massachusetts, and were composed mostly of black oak (*Quercus velutina* Lam.), white oak (*Quercus alba* L.) and pitch pine (*Pinus rigida* Mill.). The plots ranged in size from 4 to 9 ha, and were a minimum of 100 m apart. 120 larvae were collected per week from plots 3, 5 and 8 in 1983, while 450 larvae per week were collected from all of the other plots in all years.

To use the model to make predictions, we need estimates of the initial densities of infected and healthy larvae for each plot in each year. To measure initial densities in each population, Woods *et al.* (1991) quantified the density of egg masses at the beginning of the season for each plot, and estimated the average number of larvae per egg mass by hatching out egg masses in the laboratory. (For plots 5/1985 and 8/1983, average hatch was not quantified, so we used the average for all of the plots. Also, we did not use Woods & Elkinton's (1987) data for plot 5/1984 because neither average hatch nor initial fraction infected were quantified for this plot.) Since virus epizootics in gypsy moth are initiated by larvae that become infected while hatching from environmentally contaminated eggs (Murray & Elkinton 1989, 1990), Woods *et al.* (1991) were able to estimate the fraction of larvae that hatched out infected in each plot, and thus the initial density of virus polyhedra, by rearing out egg masses.

Woods *et al.*'s data give the density of larvae in terms of ground area (m²), whereas our transmission data make the units of the model foliage area (m²). To convert from ground area to foliage area we used Liebhold *et al.*'s (1989) estimate of the leaf area index for three of the plots: the leaf area index gives the foliage area per unit ground area (median of 1.4 m² of foliage per m² of ground).

Larvae in the field typically hatch out over about

a 7-day period. Since we do not know what the distribution of hatch times was for any of the plots, for simplicity we assume that larvae hatch at time $t=0$, where the first sampling point is at $t=7$ days. Variations on this assumption to spread out hatch have only a slight effect on the model predictions, at least for the parameter values in Table 1. To compare the model to the time series of NPV mortality, we numerically solved the model on a computer (using the method of steps: Hairer, Nørsett & Wanner 1987, pp. 288–289). In some plots, there is epizootic data for more than one season; in the interests of simplicity we treat each plot in each year as an independent unit.

Results

The quality of the fit of the model to the data varies considerably between plots (Fig. 2). For two of the plots (1/1983, 10/1984) the fit is excellent, for three it roughly reproduces the data (5/1985, 8/1983, 16/1985), and for the remaining three it is very poor. In all cases, the model qualitatively reproduces the bimodal temporal pattern of disease incidence: an initial peak due to the death of larvae that hatched out infected, and a second peak representing repeated rounds of transmission. Of course, since the model assumes that the incubation time of infected larvae is constant, the first peak predicted by the model is simply the initial fraction infected.

It is important to remember that all of the model parameters were estimated independently of the data to which we compare the model. A useful way of assessing the model fit for the independently estimated parameters is therefore to compare it to the model fit for best-fit parameters. We concentrated on fitting the transmission constant v because this is the parameter that we are least confident of, for three reasons: (i) it is estimated at the population level; (ii) there may well be experimental artefacts associated with confining larvae to mesh bags; and (iii) there are many different factors that can affect transmission (Dwyer 1991a). In Fig. 3, for each plot we have calculated the sum of the squared errors (SSE) between the model prediction and the data as a function of the transmission constant v . We have also calculated values of v for *each* of the bags in our transmission experiment. In Fig. 3, we have located on the SSE curve the SSEs for each of these eight estimates (\square), as well

Table 1. Values of parameters from equations 4–6, which were used to produce the simulation output in Fig. 2

Symbol	Parameter	Value
Λ	Number of virus particles per infected cadaver	2×10^9
$*v$	Disease incubation time	14 days
μ	Disease decay rate	0.003 day^{-1}
v	Transmission rate	$1.45 \times 10^{-12} \text{ m}^2 \text{ day}^{-1}$

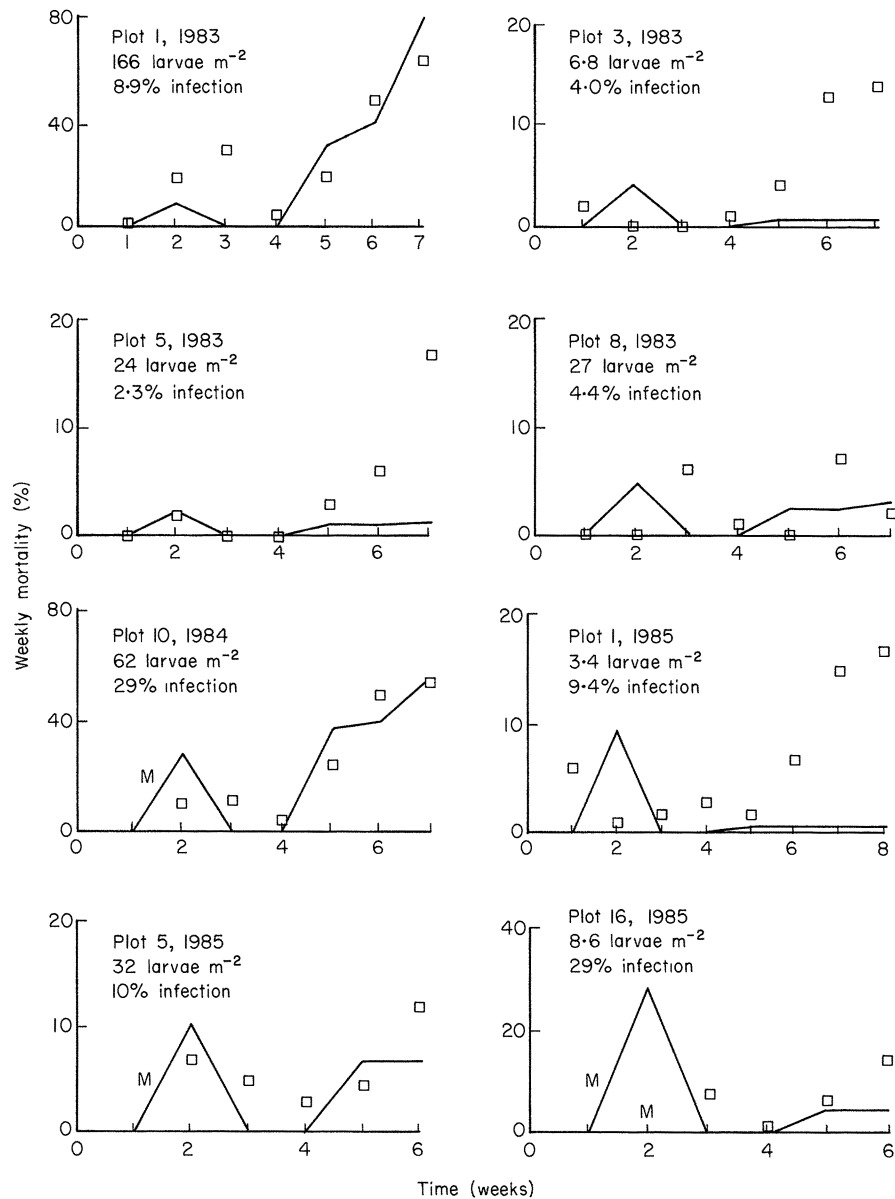


Fig. 2. Comparison of model prediction with time-series of virus mortality in gypsy moth populations: \square , observed weekly percentage mortality; —, model prediction for parameter values given in Table 1. M indicates missing data.

as the value that we used in the simulations in Fig. 2 (■), in order to show how the error in our estimates of v compares to the sensitivity of the model fit to v . This figure confirms the rough impression of the fit of the model; e.g. for plot 1/1983 and plot 10/1984, the bulk of the estimates of v from our experimental data are quite close to the best fitting v (the minimum SSE). Figure 3 also indicates that the model fit is better than it looks for other plots; specifically, although visually the model fit for plot 5/1985 and plot 16/1985 in Fig. 2 is not very good, Fig. 3 shows that, compared to the error in our estimate of v , the model prediction is not far from the minimum SSE. This suggests that the mediocre fit for these two plots could be due in part to small errors in the estimates of either v or the mortality data. For plot 8/1983, however, small changes in v would have little or no effect on the model fit; in fact, visual

inspection of the data suggests that NPV mortality was so low that the time-series of disease incidence was virtually random. Finally, for plot 3/1983, and plot 1/1985, not even the extreme values of v are close to the best-fitting value, while for plot 5/1983 only the largest estimated value of v comes anywhere near the best-fitting value. Since these three plots had the lowest initial densities, this indicates that the model has failed to take into account biological details that become important at low density. In summary, since small changes in v can have a large effect on the fit of the model, it is important to investigate whether the poor fit of the model is a failure in construction of the model or in estimation of the parameters.

To illustrate more clearly how changes in the transmission constant v affect the model fit, in Fig. 4 we have compared the model prediction to the data

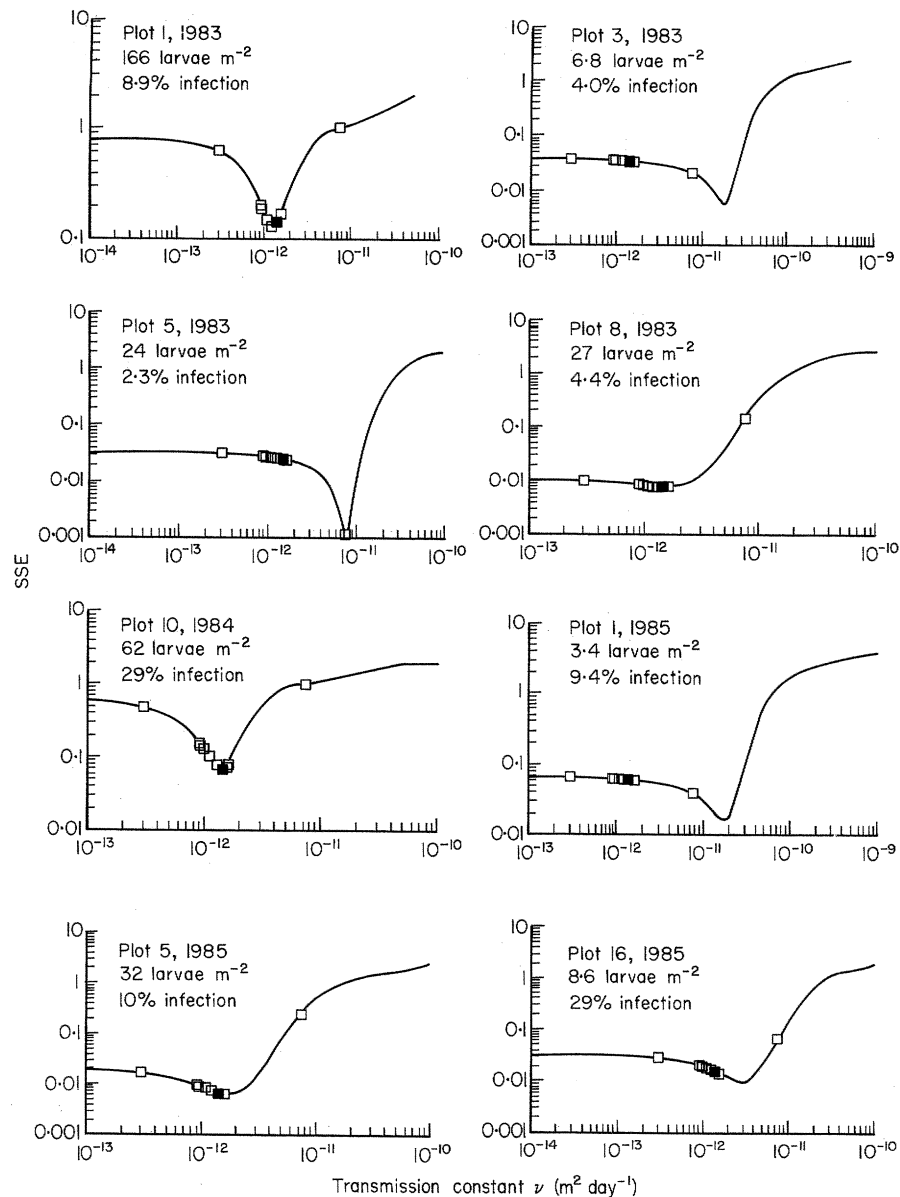


Fig. 3. Comparison of the fit of the model to the data for different values of the transmission constant v : —, sum of the squared errors (SSE) between the model and the data; \square , SSE for values of v estimated from each replicate of the experimental data in Fig. 1; \blacksquare , SSE for the estimate of v that we used in the model predictions in Fig. 2. Since in any given graph both the squares and the line plot the same variable, the squares only show the location of the SSE for our estimates of v . In other words, all the squares *must* be on the line.

for the best-fit value of v for each plot. The fit for the three poorly fitting plots (3/1983, 5/1983, 1/1985) improves dramatically as v is increased; significantly, the best values for these three plots are very similar. The improvement in the fit is noticeable but slight for plots 5/1985 and 16/1985, and negligible for the remaining plots.

Although we have concentrated on the transmission constant v , our estimate of the disease decay rate μ is another likely source of inaccuracy. The estimate that we use is based on inclusion body survival in soil and in litter, whereas within a season inclusion bodies must survive on foliage to be transmitted. Indeed, when gypsy moth NPV is used as an insecticide it breaks down rapidly on foliage; such

insecticidal preparations, however, may reduce the survival ability of inclusion bodies by removing them from the host cadaver (Podgwaite *et al.* 1979). In fact, an estimate of the decay rate μ on foliage for the closely related Douglas-fir tussock moth *Orgyia pseudotsugata* is very close to our estimate for gypsy moth (Dwyer 1992). We therefore think that our estimate is not too inaccurate. A final consideration is that, if μ is actually much higher than we think, then our estimate of v is similarly far too low. This is because to estimate v we assumed that $\mu = 0$ in the mesh bags. If μ is actually higher, then the transmission parameter v must be higher as well to account for the level of infection in our transmission experiments.

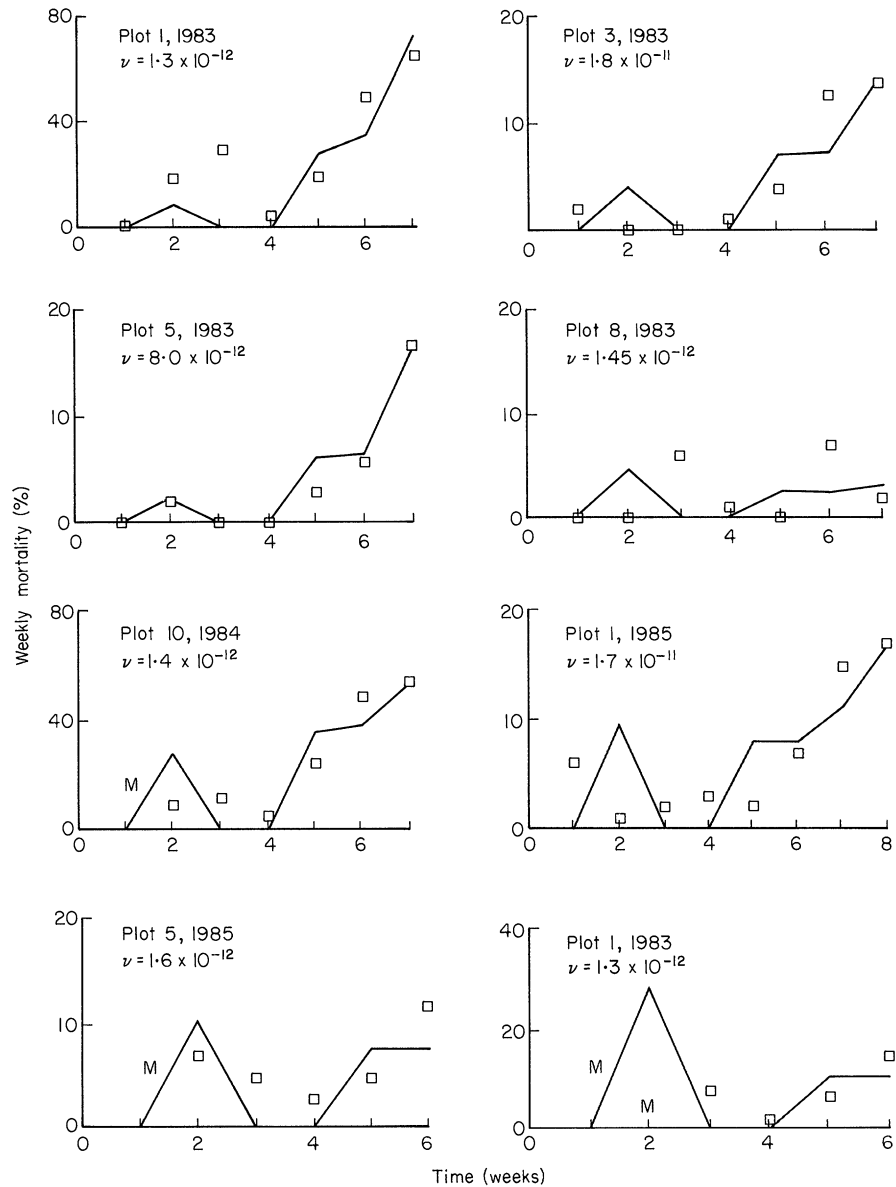


Fig. 4. Comparison of model prediction with time-series of virus mortality for the same plots as in Fig. 2, except that here we use the best-fit value of the transmission constant ν for each plot. The value of ν that we used for each plot is indicated on the appropriate graph. M indicates missing data.

Discussion

Considering how simple the model is, and that *all* of the model parameters were estimated independently of the test data, the model equations 4–6 do an excellent job of predicting the NPV mortality in five of the eight plots. Since all of the model parameters were estimated at a small scale, it is apparently often possible to deduce the large-scale phenomenon of NPV epizootics from small-scale measurements of transmission. In other words, transmission at a small scale may be what drives epizootics of NPV in gypsy moth populations. Moreover, for the three cases for which the model does poorly, a clear pattern emerges: transmission was higher than expected at low density. Clearly, our experimental protocol

did not lead to inflated values of the transmission constant ν .

Our protocol for estimating ν is significant in itself, since in previous tests of the model this parameter has simply been fitted to the data (Anderson & May 1980, 1981). An independent estimate of ν has the additional advantage that it suggests that a different process takes place in gypsy moth NPV epizootics at low density than at high density; specifically, a process that leads to higher transmission at low densities. In contrast, using a single value of ν that minimized the SSE for all the plots combined would lead to a poor fit in every case, and thus to complete rejection of the model.

Although there are a variety of possible reasons why the model did poorly at low densities, some

possibilities can be eliminated. For example, the model failure is unlikely to be due to a lack of consideration of defoliation-induced changes in allelopathic chemical constituents of host-tree foliage. At low density one would expect low defoliation and thus low levels of induced foliage chemicals. Since transmission is lower with reduced levels of such chemicals (Keating & Yendol 1987), and the model does poorly because of an underestimate of transmission, this factor is probably not responsible. Similarly, differences in host-tree species between plots were probably unimportant, as the fit of the model for plots 1 and 5 goes from good to bad between different years. Furthermore, the plots differed little in tree species composition. It also seems unlikely that the poor fit at low density is because we ignored stage structure, since changes in transmission with stage would presumably be the same in all populations. Weather is also not likely to be the cause, because the model did well for some plots and badly for other plots in the same year, and as the plots were all in the same area of the state, it is unlikely that weather conditions varied much between plots. Moreover, Woods & Elkinton (1987) showed that there was no correlation between NPV mortality and a variety of weather variables. A possibility that cannot be assessed is that either the host or the pathogen differed genetically among plots or years. This would require, for example, that pathogen infectiousness increased with time in plot 1, or that host resistance increased with time in plot 5. Although it may be possible for such changes to occur after two generations (Myers 1988), there is no evidence for it.

A somewhat more likely possibility is that our assumption that each plot constitutes an isolated population is incorrect. The three plots in question all had low density, so there was probably net immigration into these plots. Moreover, high and low density populations coexisted side-by-side throughout the region during the period 1983–85 (J.S. Elkinton, unpublished observation). It is thus possible that enough larvae ballooned into these three plots to lead to a more severe epizootic than was predicted by the model. Another possible explanation for the model failure in three of the plots has to do with larval behaviour. Specifically, larvae may behave differently at low density, so that as density declines new infections are no longer a linear function of host density. It is known, for example, that at low density a higher proportion of larvae visit daytime resting sites in the litter (Lance, Elkinton & Schwalbe 1987). This behavioural change should lead to increased larval contact rates both with other larvae and with inclusion bodies that survived earlier epizootics, either of which may in turn increase transmission enough to explain the discrepancy between the model and the data. In any case, the model failure at low host densities indicates that fur-

ther research should be directed towards an understanding of disease dynamics at low host density, with a particular eye to changes in transmission with density.

By building and testing the model we have thus accomplished three tasks. First, we have partially confirmed Woods & Elkinton's (1987) hypothesis that the timing and severity of NPV epizootics in gypsy moth are determined by densities of initial healthy and infected hosts, horizontal transmission, and the delay between infection and death. In other words, many of the biological details that we left out of the model may not be necessary for predicting NPV population dynamics. Second, we have generated a new hypothesis, or more accurately a modification of Woods & Elkinton's (1987) hypothesis; i.e. that an as yet unidentified aspect of the biology of gypsy moth and its NPV leads to increased NPV transmission at low densities. In other words, some detail that we ignored in the model becomes important at low densities. Although we have ruled out some of the possible influences on host and pathogen interactions as being unimportant at the population level, this is not to say that such effects are not real, or that they would not be more apparent at the population level with better data. Nevertheless, Woods & Elkinton's (1987) data represent perhaps the most detailed and thoroughly replicated data set for any animal disease. For all practical purposes, then, our model may be nearly sufficient.

Finally, we have presented a protocol for estimating the transmission parameter v . This protocol has the advantage that, at least for plots 1/1983 and 10/1984, it apparently gave the correct value of v . This is distinct from plots 3/1983, 5/1983 and 1/1985, for which a *different* value of v gives an excellent fit of the model to the data (Fig. 4), i.e. although there is certainly something that we did not take into account for those plots, increasing v would alone virtually eliminate the problem.

Our protocol nevertheless does not solve the problem of interpreting v in terms of more fundamental biological parameters, such as food consumption rate, etc. It is important to remember, however, that v can be interpreted as the fraction of encounters between hosts and pathogens that result in infection (Anderson 1979); it is a mistake to think that the transmission term vPS is somehow not mechanistic. Moreover, as Anderson (1979) points out, minor modification of this term allows the incorporation of additional biological detail (see also Liu, Levin & Iwasa 1987); unfortunately, we have not been able to find an existing formulation in which transmission increases with decreasing host density in a biologically meaningful way.

In short, our results suggest that there is a need for further development of insect host–pathogen models to include details of host behaviour that affect transmission. Such a development, of course,

must be tempered by a realization that problems of parameter estimation can render detailed models extremely unwieldy. Indeed, even including only those factors that are believed to affect susceptibility could make parameter estimation nearly impossible.

Acknowledgements

Earlier versions of this paper were greatly improved by the comments of Bill Morris and Peter Kareiva, Michael Hochberg and a second, anonymous, referee. We would like to thank Jeff and Cindy Boettner for assistance with the field work. Our work was supported by National Science Foundation grant BSR-9107380 to Joseph Elkinton and John Burand and by USDA Forest Service Cooperative Agreements Nos. 23-146 and 23-516 to Joseph Elkinton, Greg Dwyer and John Burand.

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Received 25 November 1991; revision received 17 February 1992