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Modelling the epizootiology of gypsy moth nuclear polyhedrosis virus

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Abstract

Qualitative understanding of the dynamics of epizootics of the nuclear polyhedrosis virus of gypsy moth has become complete enough to justify attempts to quantitatively predict the timing and intensity of epizootics within a season. In earlier work (Dwyer and Elkinton, 1993), we compared the predictions of a simple differential equation model derived from Anderson and May (1981) to time series of virus mortality in each of eight gypsy moth populations (Woods and Elkinton, 1987). The model's predictions were very accurate for high density populations, but seriously under-estimated virus mortality in low density populations. Here we compare the predictions of the simple model to those of the gypsy moth life system model (GMLSM, Sheehan, 1985; Colbert and Racin, 1991), a highly complex computer simulation of gypsy moth population dynamics and forest stand growth that incorporates much of the existing knowledge of the many factors influencing gypsy moth populations. In particular we looked at two different versions of the GMLSM that incorporate two different models of virus transmission. One was identical to that of the simple model (Anderson and May, 1981; Dwyer and Elkinton, 1993), in which the rate of transmission was a constant (the transmission coefficient) times the product of the densities of healthy larvae and the densities infectious virus particles on foliage. The other approach, developed by Valentine and Podgwaite (1982) was a detailed model of production and consumption of infective particles on foliage. The model took account of age-related changes in the amount of foliage consumed and the variation in susceptibility of larvae to virus (LD_{50}). The Anderson and May version of the GMLSM performed about as well as the simple differential equation model, but only for values of the transmission coefficient about 250 times higher that those we had determined experimentally (Dwyer and Elkinton, 1993). The Valentine and Podgwaite (1982) version of the GMLSM gave a much better fit, but only for values of LD_{50} that were 100 times higher than those determined experimentally. Future research will focus on efforts to refine our understanding of virus transmission in order to explain and reduce the discrepancies between model predictions and observed mortality from virus in fieldpopulations.

Keywords: Host-pathogen models; Epidemiology; *Lymantria dispar*; NPV

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1. Introduction

The past few decades have seen the development of a great diversity of models of host-pathogen interactions. These models range from simple differential equations with very few variables to highly complex computer simulations with hundreds of variables. In fact, few models occupy the middle ground in this range of complexity; most are either very simple or very complex. We have recently constructed a simple differential equation model for the dynamics of the nuclear polyhedrosis virus (NPV) of gypsy moth, *Lymantria dispar* (Dwyer and Elkinton, 1993). Here, we compare the predictions of the simple model to those of the gypsy moth life system model (GMLSM), a complex computer simulation that attempts to simulate all aspects of gypsy moth population dynamics (Sheehan, 1985; Colbert and Racin, 1991). We examined two methods of simulating virus transmission in this model. One was identical to that of the simple model (Anderson and May, 1981). The other was a more complex method based on increase in the amount of foliage consumed and decreases in susceptibility of larvae to virus (increasing LD_{50}) as larvae grow. We compare the ability of each version of the model to predict, after the fact, time series of NPV prevalence in eight populations in the field (Woods and Elkinton, 1987).

2. Gypsy moth and nuclear polyhedrosis virus

The gypsy moth is a major defoliator of hardwood forest trees in many parts of the world. In North America it was introduced near Boston, Massachusetts from Europe in 1868 and has been gradually spreading west and south ever since. At irregular intervals gypsy moth populations erupt into outbreaks and cause widespread defoliation. Such high density populations of gypsy moth are often decimated by an epizootic of gypsy moth nuclear polyhedrosis virus (NPV).

The gypsy moth is univoltine and overwinters in the egg stage. Larvae emerge from eggs coincident with bud-break in the spring and mature through five or six larval instars over a period of about 8 weeks. Adults emerge in mid-summer after a brief pupal stage. Females oviposit eggs in a single egg mass, typically on the trunks of trees. Females are flightless; dispersal depends on the ballooning behavior of first instars. Mortality from NPV typically occurs only during the larval stage (Murray et al., 1991).

Research by Woods and Elkinton (1987) has elucidated the process of horizontal transmission and epizootic development of NPV in gypsy moth populations, building upon the earlier work of Doane (1970, 1975, 1976). Woods and Elkinton (1987) quantified mortality from NPV and other causes from several high and low density populations of gypsy moth on Cape Cod, Massachusetts over several years. In populations of both high and low density, mortality from NPV followed a bimodal temporal pattern (Fig. 1). There was a first wave of mortality among first to third instars followed by a period of reduced mortality, and then a second wave when the majority of the larvae were in the fifth and sixth instars. The highest rates of mortality from NPV usually occurred during the second wave. The bimodal

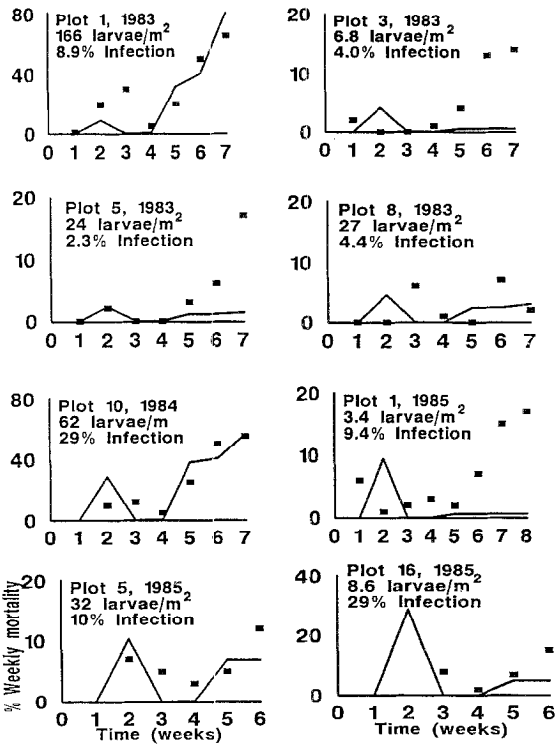

Fig. 1. Mortality from NPV among ca. 100 gypsy moth larvae [■] collected each week from eight different populations on Cape Cod 1983–1985 by Woods and Elkinton (1987) and (solid line) that predicted by the simple differential equation model of Dwyer and Elkinton (1993).

temporal pattern (Fig. 1). There was a first wave of mortality among first to third instars followed by a period of reduced mortality, and then a second wave when the majority of the larvae were in the fifth and sixth instars. The highest rates of mortality from NPV usually occurred during the second wave. The bimodal pattern of mortality was matched by similar patterns both of NPV contamination of foliage, as demonstrated with a bioassay procedure, and by the patterns of mortality among gypsy moth larvae reared in the laboratory from NPV contaminated eggs. Among larvae reared in groups of 10, there was a pronounced second wave of mortality among late instars. Among larvae reared individually, however, there was no NPV mortality among late instars. These results support the hypothesis that early instar mortality contaminates the foliage with NPV and provides the source of viral inoculum for late instar infections (Doane, 1970, 1975, 1976).

Other studies have investigated the process by which NPV is transmitted between generations of gypsy moth. It appears that several mechanisms may exist. First, Shapiro and Robertson (1987) presented evidence for vertical or maternal transmission of NPV in gypsy moth. They fed NPV to gypsy moth larvae and observed virus particles, or polyhedral inclusion bodies (PIBs), in the host tissues of the

Doane (1969) reported that nearly all of the mortality of larvae reared from field collected egg masses could be eliminated by surface disinfection, suggesting that, if vertical transmission occurs at all, it is due to surface contamination of the eggs. Murray and Elkinton (1989, 1990) confirmed that gypsy moth egg masses acquire the NPV primarily from the surface on which the eggs are deposited rather than from the female parent. The bulk of the existing evidence thus suggests that vertical transmission, if it exists, occurs largely by way of surface contamination (transovum transmission) as opposed to within the egg (transovarial transmission). Other studies have shown that gypsy moth neonates can obtain lethal doses of NPV by walking over contaminated bark or other substrates (Weseloh and Andreadis, 1986; Woods et al., 1989).

This brief literature review demonstrates that we now have a fairly extensive understanding about how NPV is transmitted in gypsy moth populations, and have identified some of the most important factors that influence levels of NPV mortality. Our next step is to see whether our qualitative understanding of the dynamics of gypsy moth NPV can be translated into quantitative predictions of levels of NPV mortality. Our thesis is that mathematical modelling is the most obvious tool for testing the adequacy of our knowledge. Specifically, by comparing model predictions with existing data from the field, like that from Woods and Elkinton (1987), we can test our understanding of NPV dynamics. Here we compare the predictions of two such models.

3. Methods

In Dwyer and Elkinton (1993), we presented the following model for the dynamics of insect pathogens within a season.

$$\frac{dS}{dt} = -\nu P S \tag{1}$$

$$\frac{dI}{dt} = \nu P S - \nu P(t-\tau) S(t-\tau) \tag{2}$$

$$\frac{dP}{dt} = \Lambda \nu (t-\tau) S(t-\tau) - \mu P \tag{3}$$

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, ν 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.

As we discussed in Dwyer and Elkinton (1993), our opinion is that biologists have historically avoided this type of model for two main reasons. The first is that the model includes only the bare minimum of what biologists believe can affect the dynamics of NPVs. For example, horizontal disease transmission in the model is a function only of density; in reality, a variety of factors have been shown to influence horizontal disease transmission, including host stage (Thompson and Scott, 1979; Kaupp, 1983; Watanabe, 1987; Teakle and Byrne, 1989; Dwyer, 1991; Hochberg,

1991), and chemical constituents of foliage consumed by the insects (Keating and Yendol, 1987; Keating et al., 1988). Having been trained to think that more detailed biological knowledge leads to better understanding, many biologists have been drawn instead to complex computer simulations that include a wealth of biological detail (Sheehan, 1985; Onstad and Carruthers, 1990).

A second problem has been the difficulty of estimating parameters that cannot be derived easily from observations of individual host insects. Eqs. 1–3 are essentially Anderson and May's (1980, 1981) model with the addition of a fixed time-delay between infection and death. The introduction of a time delay allows us to easily estimate the disease incubation time (τ) and the number of pathogen particles per infected host (Λ). Although the transmission parameter ν remains problematic, in Dwyer and Elkinton (1993) we presented a protocol for estimating this parameter. The protocol essentially consists of fitting the model to data from small-scale transmission experiments. The experiments involve rearing infected and uninfected larvae in mesh bags on red oak foliage, and recording the fraction of the initially uninfected larvae that become infected.

The Gypsy Moth Life System model (GMLSM) was originally developed by McNamee et al. (unpublished) and Sheehan (1985) and modified into its current form by Colbert and others (Colbert and Racin, 1991). The model simulates the growth and development of gypsy moth in a forest stand and includes the action of the many predators, parasitoids and disease agents that attack gypsy moth at varying times in its life cycle, as well as daily fluctuations in weather. The forest stand component of the model simulates the growth of trees and the impact of gypsy moth defoliation on each of the user-specified tree species in the forest stand. The basic time step of the model can be set by the user for a fixed number of days or degree days. The model summarizes mortality at the end of each season and can be used to investigate the dynamics of gypsy moth and forest growth over many successive years. To compare the model to Eqs. 1–3, here we focus on the within-season dynamics of NPV.

The pathogen portion of the life system model was modified from a model developed by Valentine (Valentine and Podgwaite, 1982). The pathogen submodel simulates the movement of PIBs between various strata within the stand (the overstory, the understory and the litter). Some of the virus inclusion bodies produced by larvae that die on foliage are washed onto the other stand strata. The PIBs that wash onto the bole contaminate the egg masses and determine the level of infection among neonates the following year. The variable incubation time of virus within hosts is simulated with a Weibull function.

Within-generation transmission in the Valentine model is determined by estimating the probability of a larva consuming a lethal dose at each iteration of the model. The probability is estimated from the density of PIBs, the amount of foliage consumed, a clumping factor and known values for LD_{50} for each instar as determined in laboratory experiments (Podgwaite, unpublished). These experiments involved feeding known doses of virus on leaf discs to larvae of different instars.

We developed a second version of the pathogen submodel for the GMLSM, that was intermediate between our simple model and the original Valentine submodel. In

this version transmission rates were calculated as in our simple model ($\nu \times$ density of uninfected larvae $\times$ density of PIBs) based on (Anderson and May, 1981). To estimate the discrete-time transmission coefficient for this version of the GMLSM, we followed Dwyer and Elkinton (1993) by fitting a simplified version of the model to the experimental data on larvae reared in bags on foliage Dwyer and Elkinton (1993). The resulting value of ν (1.89×10^{-12} m^2/day) is close to (1.45×10^{-12} m^2/day) for the continuous-time simple model.

In order to compare the predictions of both versions of the pathogen submodel in the GMLSM to our simple differential equation model, we simplified the GMLSM to focus on mortality from virus within a season. We eliminated the effect of variables that we believe are of secondary importance. PIBs produced from larvae that die of the virus remained on the substrate upon which the larvae died whereas in the original GMLSM some fraction of these PIBs are washed off the foliage onto the boles of trees and the forest floor. In each daily interval, the fraction of PIBs that decayed was set to a constant equal to 0.003 (Podgwaite et al., 1979) as in the simple model (Dwyer and Elkinton, 1993). We set all other sources of mortality of gypsy moths from parasitoids, predators and starvation to zero. The initial infection rate was a fixed value (equal to what we observed for the particular plot and year we were simulating), instead of being calculated from the density of PIBs produced the previous year. The effect of PIB clumping in the Valentine submodel was eliminated by setting the clumping parameter (k) to an asymptotically high value of 1000, which implied that the virus was evenly distributed over the foliage surface. We recognize that PIBs may indeed be clumped in natural populations, but we have no data on PIB clumping nor any obvious way of estimating k, even if we had the data. We eliminated a variable in the pathogen submodel that placed an arbitrary upper limit on the fraction of the population becoming infected in any time step. No data exist to support such an upper limit (below 1.0). We ran simulations with a 1 day basic time step for one year for stands comprised entirely of red oak, *Quercus rubra*, using weather data for 1983, the first year of the study of Woods and Elkinton (1987).

The principal differences between the simple model and both versions of the GMLSM were as follows. The density of PIBs in Dwyer and Elkinton (1993) is calculated as PIBs per m^2 of leaf area based on the leaf area index, which was calculated for these sites by Liebhold et al. (1988) and which we assumed would remain constant through the season. In contrast, the GMLSM calculates leaf area by means of a complicated algorithm that takes account of on-going defoliation. Furthermore, the life system model embodies complicated rules of larval behavior, that determine what fraction of larvae die from NPV on foliage compared to other substrates. This fraction varies with population density. In addition, both larval growth and NPV development in the life system model are temperature dependent processes that are driven by weather conditions. Incubation time for the virus inside a larva increases with larval age, as indicated by studies of Shields (1984) instead of the fixed incubation time of two weeks used in Dwyer and Elkinton (1993). Finally the simulated eggs in the GMLSM hatch over a period of several days producing a distribution of age classes or cohorts that persist through the subsequent instars. In contrast, all individuals in the simple model hatch simultaneously. As a result all

individuals initially infected with NPV die in the same week in the simple model, whereas such deaths are distributed over two or more weeks in the GMLSM.

4. Results and discussion

To test these models, we compared their predictions to data on the proportion of larvae dying each week from NPV during the larval stage from 8 populations on Cape Cod, Mass., as reported in Woods and Elkinton (1987). The initial rate of infection for these populations was determined by measuring the fraction of larvae reared from egg masses that die from NPV within a few days of hatch (Woods et al., 1991). The density of larvae was estimated from the number of egg masses per ha times the number of larvae emerging per mass. We have previously reported (Dwyer and Elkinton, 1993) the fit of the simple model to these data as illustrated in (Fig. 1). For two of the plots the fit is excellent, for three others the model roughly reproduces the data and for the remaining three plots the model vastly underestimates the mortality during late instars. These three plots had the lowest densities of gypsy moths among the eight plots that we fit, suggesting that our model may leave out an important component of NPV epizootiology at low density.

In short, in Dwyer and Elkinton (1993), we presented a model of insect host-pathogen dynamics that includes only four parameters (not including initial conditions). Two of the model parameters can be estimated easily from observations on individual insects, and, as we show in the paper, the other two can be estimated from small-scale population-level experiments. Given the simplicity of the model, the fit between the model predictions and the field data is excellent. We thus have attempted to make parameter estimation easier, and to show that simple models can accurately reproduce the dynamics of disease in field populations. Our attempt, however, has been only partially successful; the model does poorly at low densities. Ultimately, we hope to improve the fit between the model and the data by incorporating aspects of gypsy moth biology that change between high and low density, *and* that affect NPV transmission, while maintaining the basic 'parameter-sparseness' of the model (van den Bosch et al., 1990).

With the Anderson and May version of the GMLSM and a value of $\nu = 1.89 \times 10^{-12}$ m^2/day, calculated from our experiments in bags on foliage, we found that negligible late-instar mortality occurred for the initial conditions of Woods and Elkinton's (1987) plots. Next we tried several different values of ν. We found that when $\nu = 5.0 \times 10^{-10}$ m^2/day, (approximately 250 times higher than that estimated from Dwyer and Elkinton's 1993 data), the resulting epizootics predicted (Fig. 2) were almost identical to those predicted by the simple model (Fig. 1). The simulations accurately predicted the results for high density populations, but greatly underestimated the mortality observed in low density populations.

Next we ran the GMLSM with the Valentine submodel using an $LD_{50} = 1.0 \times 10^5$ PIBs per gram dry foliage biomass (Podgwaite, unpublished). The simulation predicted mortality from NPV that was far higher at all densities than that observed in Woods and Elkinton (1987). However, when the LD_{50} was arbitrarily increased to 1.0×10^7 PIBs per gram foliage dry weight, we obtained an excellent fit to the field

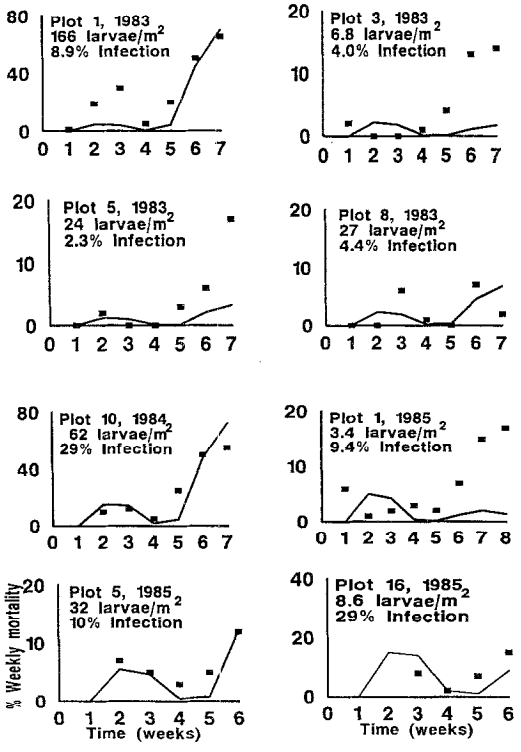

Fig. 2. Mortality from NPV gypsy moth larvae [■] collected weekly from eight populations on Cape Cod 1983–1985 by Woods and Elkinton (1987) and (solid line) that predicted by the gypsy moth life system model (GMLSM) with the Anderson and May version of the pathogen submodel and a transmission coefficient, $v = 5.0 \times 10^{-10}$ m^2/day.

data (Fig. 3). In contrast, for any one set of parameter values, the simpler versions of the model never fit all of the plots equally well. This suggests that the Valentine version of the model captures some important aspect of virus transmission. In the Anderson and May model the rate of virus transmission is proportional to the product of density of healthy larvae and the density of PIBs, as indicated in the first term of Eq. 2. In the Valentine model this rate of change is proportional to $(C/LC_{50})^b/[1 + (C/LC_{50})^b]$ where C is the concentration of PIBs, LC_{50} is the concentration required to kill 50% of the larvae that encounter and ingest a patch of PIBs and b is the slope of the logistic dose response curve (set to 0.77 in the GMLSM) and is a measure of the inherent variation in susceptibility of gypsy moth to NPV. If we compare plots 1 and 3 in 1983 (Fig. 1) the concentration of PIBs (initial density of larvae times the fraction infected) after the first wave of mortality is about 50 times higher in plot 1 than plot three, as is the simulated second wave with the Anderson and May model (Figs. 1 and 2). In the field data, the second wave of mortality is only 4–5 times higher in plot 1 than plot 3 in that year. In the Valentine model if the concentration (C/LC_{50}) is increased by 50 fold from

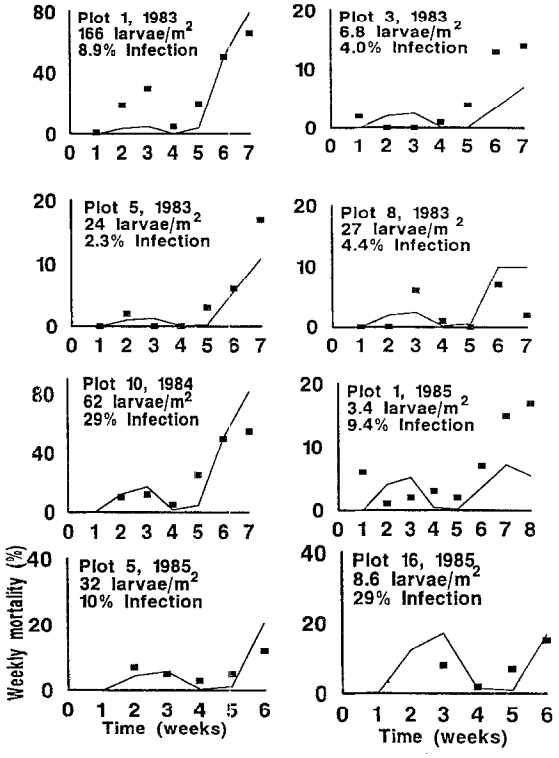

Fig. 3. Mortality from NPV among gypsy moth larvae [■] collected weekly from eight populations on Cape Cod 1983–1985 by Woods and Elkinton (1987) and (solid line) that predicted by the GMLSM with the Valentine (Valentine and Podgwaite, 1982) version of the pathogen submodel and an LD_{50} of 1×10^7 PIBs per gram foliage dry weight.

0.1 to 5.0, $(C/LC_{50})^b/[1 + (C/LC_{50})^b]$ increases by a factor of 5.3. when $b = 0.77$. Incorporation of this term thus reduces the difference in virus mortality between plots of high and low density compared to the corresponding Anderson and May model. Although this functional form is intended as a mechanism for introducing variation in host resistance into the model, we are not clear that this interpretation is correct (C. Godfray, pers. commun., 1993). A broader interpretation is that the Valentine model introduces a term that makes transmission rates a non-linear function of the density of larvae and PIBs. While several previous researchers have explored the theoretical consequences of such non-linearities, Fig. 3 provides the first empirical justification for such a model. The biological cause of this non-linearity remains to be determined.

Whether these effects account for all of the differences between the models we do not know. The complexity of the GMLSM is such that other factors in the model, that we do not now understand, may be influencing these results. In the future we plan to check this conclusion by constructing a simple model similar to Eqs. 1–3, but in which transmission is calculated by a method analogous to the Valentine submodel.

It is interesting to compare the results of our simulation models with the logistic regression models fitted to the same data by Woods et al. (1991). These authors showed that gypsy moth density predicted about 40% of the variance in late instar mortality of gypsy moths and that the addition of estimates of initial percent NPV infection added little to the predictive power of the model. Our models provide a mechanistic explanation of these findings, albeit an explanation already suggested by Woods et al. (1991). This explanation is that several of the plots in this study were in populations that had declined to low density from outbreak conditions the previous year due to an epizootic of NPV. The egg masses in these populations were heavily contaminated with NPV and a large fraction of larvae died in early instars, yet densities were low so that the epizootic was not sustained among later instars. Such a phenomenon was noted in the earlier studies of gypsy moth NPV by Doane (1976) and is a well known property of Anderson and May (1980, 1981) models.

A potentially important source of error in our simple differential equation model is in our estimation of Λ, the number of PIBs per infected cadaver. In estimating the transmission coefficient we used $\Lambda = 4 \times 10^8$ for first instars (Shapiro et al., 1986). Other data suggest (Podgwaite, unpublished), that Λ is closer to 4×10^6, a value consistent with that calculated by the GMLSM which assumes that cadavers produce 1×10^7 PIBs per gram dry weight (Shapiro et al., 1986). Using this adjusted value for Λ increases the estimated value of ν for the simple model to 1.45×10^{-10} m^2/day, which is close to the value that gives a good fit in the GMLSM.

The problem of parameter estimation is vastly greater for the GMLSM, with its hundreds of parameters, than for Eqs. 1–3. Although ostensibly each parameter has been estimated, verifying the estimate and determining the effect of each parameter on model outcome are daunting tasks. In fact, for many parameter values, no data exist and supposedly 'reasonable' values have been selected. Nevertheless, the life system model allows for more straightforward incorporation of biological details, and clearly Eqs. 1–3 do not accurately reproduce the dynamics of gypsy moth NPV epizootics in the field.

There are many variables in nature that may explain the discrepancy between the predictions of any of these models and the data of Woods and Elkinton (1987). Some of these variables are already embodied in the GMLSM; others have yet to be incorporated. For many variables, we have no good biological research upon which to base parameter values. These factors include PIB clumping, foliage chemistry effects (Keating and Yendol, 1987; Keating et al., 1988) and changes in gypsy moth density caused by mortality factors other than NPV. Further empirical and theoretical work will be required to elucidate the effects of these factors.

With complex simulations like the GMLSM, it is always possible that the observed model behavior is influenced by artifactual effects caused by programming errors or inaccurate biological information. Detecting these errors can be very difficult. However, if the same behavior is observed in a simple model, it increases confidence that the complex model is behaving properly. Furthermore, comparison of the behavior of the simple and complex models helps elucidate which aspects of the complex model are critical to the behavior of the system. Likewise, the complex model can help pinpoint critical aspects of system biology that are needed

for further development in simple models. For example, we never would have realized that something was possibly wrong with our estimate of the number of PIBs produced by early instars (and hence ν), if we had not been working with the GMLSM. Similarly, we might never have thought of the potentially important effects of variation in host susceptibility. In short, we believe our work illustrates the benefits of working simultaneously with complex and simple models.

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References

Anderson, R.M. and May, R.M. (1980) Infectious diseases and population cycles of forest insects. Science, 210: 658–661.

Anderson, R.M. and May, R.M. (1981) The population dynamics of microparasites and their invertebrate hosts. Philos. Trans. R. Soc. London, Ser. B., 291: 451–524.

Colbert, J.J. and Racin, G.E. (1991) Gypsy moth life system model. In: F.S. Zazueta, D.G. Watson and A.B. Botcher (Editors), Proceedings, 1990 Third International Conference on Computers in Agricultural Extension Programs; Lake Buena Vista (Orlando), FL. University of Florida, Gainsville, Fla., pp. 115–120.

Doane, C.C. (1969) Trans-ovum transmission of a nuclear-polyhedrosis virus in the gypsy moth and inducement of virus susceptibility. J. Invert. Pathol., 14: 199–210.

Doane, C.C. (1970) Primary pathogens and their role in the development of an epizootic in the gypsy moth. J. Invert. Pathol., 15: 21–33.

Doane, C.C. (1975) Infectious sources of nuclear polyhedrosis virus persisting in natural habitats of the gypsy moth. Environ. Entomol., 4: 392–394.

Doane, C.C. (1976) Ecology of pathogens of the gypsy moth. In: J.F. Anderson and H.K. Kaya (Editors), Perspectives in Forest Entomology. Academic Press, New York, N.Y., pp. 285–293.

Dwyer, G. (1991) The roles of density, stage and patchiness in the transmission of an insect virus. Ecology, 72: 559–574.

Dwyer, G. and Elkinton, J.S. (1993) Using simple models to predict virus epizootics in gypsy moth populations. J. Anim. Ecol., 62: 1–11.

Hochberg, M.E. (1991) Viruses as costs to gregarious feeding behavior in the Lepidoptera. Oikos, 61: 291–296.

Kaupp, W.J. (1983) Estimation of nuclear polyhedrosis virus produced in field populations of the European sawfly, *Neodiprion sertifer* (Geoff.) (Hymenoptera: Diprionidae). Can. J. Zool., 61: 1857–1861.

Keating, S.T. and Yendol, W.G. (1987) Influence of selected host plants on gypsy moth (Lepidoptera: Lymantriidae) larval mortality caused by a baculovirus. Environ. Entomol., 16: 459–462.

Keating, S.T., Yendol, W.G. and Schultz, J.C. (1988) Relationship between susceptibility of gypsy moth larvae (Lepidoptera: Lymantriidae) to a baculovirus and host plant foliage constituents. Environ. Entomol., 17: 952–958.

Liebhold, A.M., Elkinton, J.S., Miller, D.R. and Wang, S. (1988) Estimating oak leaf area index and gypsy moth, *Lymantria dispar* (Lepidoptera: Lymantriidae), defoliation using canopy photographs. Environ. Entomol., 17: 560–566.

McNamee, P.J., Bunnel, P., Jones, M.L. and Marmorek, D.R. (1983) Final report of a project to identify and evaluate important research questions for the gypsy moth life systems. Northeastern Forest Experiment Station, RWU-4557, 180 Canfield St., Morgantown, WV 26505-3101, USA

(unpublished report on file).
Murray, K.D. and Elkinton, J.S. (1989) Environmental contamination of egg masses as a major component of transgenerational transmission of gypsy moth nuclear polyhedrosis virus (LdMNPV). J. Invert. Pathol., 53: 324–334.
Murray, K.D. and Elkinton, J.S. (1990) Transmission of nuclear polyhedrosis virus to gypsy moth *Lymantria dispar* (L.) (Lepidoptera: Lymantriidae) eggs via contaminated substrates. Environ. Entomol., 19: 662–665.
Murray, K.D., Shields, K.S., Burand, J.P. and Elkinton, J.S. (1991) The effect of gypsy moth metamorphosis on the development of nuclear polyhedrosis virus infection. J. Invert. Pathol., 57: 352–361.
Onstad, D.W. and Carruthers, R.I. (1990) Epizootiological models of insect diseases. Annu. Rev. Entomol., 35: 399–420.
Podgwaite, J.D., Shields, K.S., Zerillo, R.T. and Bruen, R.B. (1979) Environmental persistence of the nucleopolyhedrosis virus of the gypsy moth, *Lymantria dispar*. Environ. Entomol., 8: 528–536.
Shapiro, M. and Robertson, J.L. (1987) Yield and activity of gypsy moth (Lepidoptera: Lymantriidae) nucleopolyhedrosis virus recovered from survivors of viral challenge. J. Econ. Entomol., 80: 901–905.
Shapiro, M., Robertson, J.R. and Bell, R.A. (1986) Quantitative and qualitative differences in gypsy moth (Lepidoptera: Lymantriidae) nucleopolyhedrosis virus produced in different-aged larvae. J. Econ. Entomol., 79: 1174–1177.
Sheehan, K.A. (1985) Gypsy moths and northeastern forests: modeling a complex biological system. In: P.E. Dress and R.C. Field (Editors), Symposium on Systems Analysis in Forest Resources. Society for American Forestry, Publ. No. SAF 86-03, pp. 444–454.
Shields, K.S. (1984) Transmission and course of infection of the nucleopolyhedrosis virus (Baculovirus) of the gypsy moth, *Lymantria dispar*. Ph.D. Dissertation, University of Connecticut, Storrs, Conn.
Teakle, R.E. and Byrne, V.S. (1989) Nuclear polyhedrosis virus production in *Heliothis armigera* infected at different larval ages. J. Invert. Pathol., 53: 21–24.
Thompson, C.G. and Scott, D.W. (1979) Production and persistence of the nuclear polyhedrosis virus of the Douglas-fir tussock moth, *Orgyia pseudotsugata* (Lepidoptera: Lymantriidae), in the forest ecosystem. J. Invert. Pathol., 33: 57–65.
Valentine, H.T. and Podgwaite, J.D. (1982) Modelling the role of NPV in gypsy moth population dynamics. In: Invertebrate Pathology and Microbial Control. Proceedings of the 3rd International Colloquium on Invertebrate Pathology. University of Sussex, Brighton, pp. 353–356.
van den Bosch, F., Metz, J.A.J. and Diekmann, O. (1990) The velocity of spatial population expansion. J. Math. Biol. 28: 529–566.
Watanabe, H. (1987) The host population. In: J.R. Fuxa and Y. Tanada (Editors), Epizootiology of Insect Diseases. Wiley, New York, N.Y., pp. 71–112.
Weseloh, R.M. and Andreadis, T.G. (1986) Laboratory assessment of forest microhabitat substrates as sources of the gypsy moth nuclear polyhedrosis virus. J. Invert. Pathol., 48: 27–33.
Woods, S.A. and Elkinton, J.S. (1987) Bimodal patterns of mortality from nuclear polyhedrosis virus in gypsy moth (*Lymantria dispar*) populations. J. Invert. Pathol., 50: 151–157.
Woods, S.A., Elkinton, J.S. and Podgwaite, J.D. (1989) Acquisition of nuclear polyhedrosis virus from tree stems by newly emerged gypsy moth (Lepidoptera: Lymantriidae) larvae. Environ. Entomol., 18: 298–301.
Woods, S.A., Elkinton, J.S., Murray, K.D., Liebhold, A.M., Gould, J.R. and Podgwaite, J.D. (1991) Transmission dynamics of a nuclear polyhedrosis virus and predicting mortality in gypsy moth (Lepidoptera: Lymantriidae) populations. J. Econ. Entomol. 84(2): 423–430.