ARTICLES

Outbreaks and Interacting Factors: Insect Population Explosions Synthesized and Dissected

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Insect outbreaks have attracted a great deal of attention from ecologists, but an understanding of outbreaks has been elusive. We argue that a major reason for this lack of understanding is that most ecologists focus on single factor explanations, while most outbreaks are probably determined by multiple factors. This focus on single factors is not just due to investigator bias, but seems to be inherent in the major approaches used to study outbreaking insects. Theoreticians have focused on fitting mathematical models to time series of densities; we show, however, that this method is not capable of distinguishing among mechanisms. Field biologists typically rely on experiments that test only one factor at a time, probably due to the difficulty of performing experiments on an appropriate scale. We suggest that a way out of this problem may be to closely integrate models and experiments so that moderately complex mathematical hypotheses may be tested in the field without too great expense.

KEY WORDS: population dynamics, mathematical models, insect outbreaks

Insect outbreaks are among the most impressive phenomena in nature, and have inspired vivid prose, beginning with biblical accounts of locust plagues:

> And the locust went up over all the land of Egypt, and rested in all the coasts of Egypt: very grievous were they; before them there were no such locusts as they, neither after them shall be such. For they covered the face of the whole earth, so that the land was darkened; and they did eat every herb of the land, and all the fruit of the trees which the hail had left: and there remained not any green thing in the trees, or in the herbs of the field, through all the land of Egypt.[1]

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The depredations of forest-defoliating insects such as the gypsy moth have engendered equally horrified (albeit less literary) reports:

> My sister cried out one day, "They [the caterpillars] are marching up the street." I went to the front door, and sure enough, the street was black with them, coming across from my neighbor's, and heading straight for our yard. They had stripped her trees, but our trees at that time were only partially eaten.[2]

> For three weeks the destruction wore on, and the incessant munching almost led me to insanity ... All vegetation is being denuded by the gypsy moth. As a result, all life is being threatened, even human.[3]

In addition to horrifying onlookers, outbreaks have captured the attention of numerous ecologists, all trying to explain what leads to these sudden eruptions, and to reduce the associated economic losses.[4,5] Our success in finding explanations, however, has not been impressive. In this article, we briefly review efforts to explain outbreaks in three of the most notorious forest defoliators: the larch budmoth, the western tent caterpillar, and the gypsy moth. By reviewing these three striking cases, we attempt to build some generality about the kinds of approaches that have been taken to understanding violent fluctuations in defoliator population density. The lesson we learn is that investigators have tended to favor single-factor explanations, but in each case the evidence points to a suite of factors, so that predators, pathogens, host-plant quality and weather interact to drive outbreaks.

Our interest is therefore not in which particular mechanism is the correct one for any of these insects, but rather in the contrast between single-factor and multiple-factor explanations. First, we briefly describe the biology of each species. Second, we consider what we can learn about defoliator dynamics by fitting mechanistic models to time series of densities of these forest defoliators. Third, we consider what the

existing field data can tell us about mechanisms driving the dynamics of each insect, as well as what investigators believe are the important mechanisms. Finally, we conclude by arguing that a combination of theory and experiment may allow us to quantitatively test for the importance of multiple-factor explanations of the dynamics of outbreaking insects.

Understanding insect outbreaks is more than a matter of being able to predict when a species will defoliate the forest. By knowing why single species erupt to such high densities that they transform entire landscapes, we will also better understand why most species never outbreak. Although of course all populations fluctuate, only about 3% of forest Lepidoptera[6] are outbreaking, that is, cause significant defoliation of hosts over large areas. Of those species that do outbreak, some have outbreaks at regular intervals, and so are cyclic, although time series that are long enough to statistically demonstrate regular cycles are uncommon.[7] Although not all outbreaking species are demonstrably cyclic by statistical criteria, we focus on cycling species because their comparatively simple dynamics suggest that they are most likely to have single factor explanations. If single-factor explanations are ever correct, then they should be correct for larch budmoths, or western tent caterpillars, or gypsy moths. Also, the best-known mathematical hypotheses to explain outbreaking dynamics all predict long-period cycles.

BASIC BIOLOGY OF OUR OUTBREAK SPECIES

Larch Budmoth

The larch budmoth, *Zeiraphera diniana* Gn., has one generation a year, and overwinters in the form of a diapausing embryo in the egg. Eggs hatch in synchrony with the emergence of larch needles or the production of new needles of the evergreen hosts, cembran pine, scots pine, lodgepole pine, norway spruce, and sitka spruce. The larval stage lasts 40–60 days in the Alps, and the pupal stage lasts 25–36 days. Pupation occurs in a cocoon in the soil. Upon emergence, males and females fly at night if the temperature is above 7°C (with cold nights, flight may occur during the day). Females lay 20–350 eggs underneath lichen on larch branches, or in other sheltered locations.

Western Tent Caterpillar (and Forest Tent Caterpillars)

In what follows, we focus on western tent caterpillar, *Malacosoma californicum* (Dyar), because there are good density time series for this species, but we also consider forest tent caterpillar *M. disstria* Hbn. because of the great similarity in biology and dynamics of the two species.[4,8] Both species overwinter as larvae in egg bands on branches of the host plant, and feed gregariously at least during the early instars. Also, both species oviposit on and feed on several different host plant species,[8] although each has strong oviposition preferences. Pupation occurs in cocoons among leaves, and both males and females fly at night.

Our interest is in the contrast between single-factor and multiple-factor explanations.

Each is attacked by a nuclear polyhedrosis virus, a bacterial disease and a variety of parasitoids.[8] Finally, invertebrate predators are important among early instar larvae, but all larval instars are relatively unpalatable for vertebrates. Birds readily attack the cocoons.[9]

Gypsy Moth

The gypsy moth, *Lymantria dispar* L., is also univoltine and like the other species considered here, overwinters in the egg stage. Larvae emerge in early spring and sample the host leaves; if leaves are unavailable or unpalatable, the larvae will attempt to disperse by ballooning on silk strands. Although the larvae can generally feed on a very wide range of trees, oaks and aspens are the preferred hosts. Pupation occurs in July in concealed locations on tree trunks or in the litter. Adult females in North America and Europe are flightless, and so tend to lay egg masses near the site of pupation. Males fly during the day. The larvae are attacked by several species of parasitoids as well as invertebrate predators, but are not very palatable to most vertebrates. Pupae are palatable, however, and are consumed by a variety of small rodents. At high densities, a nuclear polyhedrosis virus is very important. Over the last ten years, a fungal pathogen has become important in gypsy moth population dynamics in North America; however, because here we are interested in historical patterns of gypsy moth population dynamics, we concentrate on other sources of mortality.

THEORETICAL APPROACHES TO UNDERSTANDING OUTBREAK DYNAMICS: COMPARING MODELS TO TIME SERIES

Mathematical models have been a popular approach for understanding the dynamics of outbreaking defoliators, both because defoliators provide such interesting dynamics, and because of the practical difficulties of field experiments. Early models were aimed at demonstrating that populations could fluctuate simply because of biotic interactions (for example, because of predator-prey interactions), even without any underlying environmental variation driving the populations of predator or prey.[10] More recently, nonlinear dynamicists have used models as statistical tools for identifying cycles and chaos in the face of environmental perturbations or "noise."[7,11–13] Although this work has greatly improved our ability to analyze population dynamic data, it generally uses phenomenological models that incorporate a kind of generalized delayed density-dependence, and this lack of mechanism has been criticized by some field biologists.[14]

A more biologically satisfying approach has been the exploration of models that specify the mechanism driving the insect's dynamics. For such models, one can ask whether a particular mechanism could at least in theory produce outbreak dynamics, because in some cases models show that certain

hypothesized mechanisms can be eliminated as explanations because they cannot produce outbreak patterns.[15] This style of theory stands apart not only from phenomenological modelling,[11,12] but is also distinct from intricate simulations that are imbued with enormous complexity and realism.[16] Although all of these approaches to modelling have merit, for identifying mechanisms that drive outbreaks, we advocate simple models that have clear-cut mechanisms.

Our first line of analysis uses three models, with each model corresponding to a particular hypothesis about what causes pest outbreaks. Following this modelling analysis, we will examine what field data have to say about the dynamics of each species. The three models that we consider are essentially off-the-shelf models of interspecific interactions. In chronological order, the first model is of host-parasitoid interactions:[17,18]

$$N_{t+1} = \lambda N_t e^{-QP_t^{1-m}} \quad (1)$$

$$P_{t+1} = wN_t(1-e^{-QP_t^{1-m}}). \quad (2)$$

Here N represents host insects, in our case a forest defoliator, P represents parasitoids, λ is the reproductive rate of the host, Q and m describe the degree to which parasitoids interfere with each other while foraging for hosts, w is the number of parasitoids produced from each parasitoid-killed host, and t is the generation. Host-parasitoid models similar to equations (1)–(2) are perhaps most famous for having been the subject of extensive debates about the importance of density-dependence and stable equilibria in biological control.[19,20] Nevertheless, for the right parameter values, equations (1)–(2) show long-period cycles (as do closely related host-parasitoid models with small modifications in functional form), and so one of the first applications of this model was to explain the dynamics of forest defoliators.[21]

The second model considers how maternal effects can determine defoliator dynamics:[22]

$$N_{t+1} = N_t R \frac{x_t}{k + x_t} \quad (3)$$

$$x_{t+1} = x_t^{1-\beta} M \frac{S/N_{t+1}}{p + S/N_{t+1}} \quad (4)$$

$$x_{t+1} \geq x_{\min} \quad (5)$$

where N is the density of the insect and x is its "quality," so that quality represents some physiological feature of the organism that affects its fecundity (high quality leads to high fecundity, and low quality to low fecundity). S represents the total amount of resource available in the environment, R is fecundity at maximum quality, M is the maximum rate of increase of quality, and k and p control the rates at which the rates of increase in fecundity and quality approach their asymptotes. Finally, β allows the change in quality to be nonlinear, and $x_{\min}$ is the minimum quality. The model thus allows the conditions experienced by previous generations to affect the fecundity of the present generation. Fecundity thus increases as the quality of the previous generation increases, reaching an asymptote at R, while quality declines as the density in the current generation increases. This mimics effects reported for some defoliators, in that fecundity can be depressed for more than one generation after a peak in population density, apparently due to the lingering effects of starvation.[23] The model allows for other effects of quality, however, in that fecundity in the model includes the effects of low quality on survival, as described by Wellington[24] in western tent caterpillar.

As with the host-parasitoid model, equations (3)–(5) show long-period cycles for the right parameter values. Ginzburg and Taneyhill[22] therefore used the model to argue that maternal effects may drive outbreaks in forest defoliators.

The third model represents host-pathogen interactions:[25]

$$N_{t+1} = \lambda N_t(1-I(N_t, P_t)) \quad (6)$$

$$P_{t+1} = \frac{\alpha}{\alpha + \mu} S_t I(N_t, P_t) \quad (7)$$

$$1 - I = \left(1 + \bar{\nu} C^2 \left(\frac{1}{\xi + \mu} S_t I + \frac{1}{\delta} P_t\right)\right)^{-1/C^2}. \quad (8)$$

Here N is the density of the host insect, P is the density of infectious pathogen particles outside of any insects, and $I(N_t,P_t)$ is an implicit function that describes the fraction of hosts that survive a yearly epidemic, on the assumption that all infected hosts die of the disease. This model represents the biology of many pathogens of forest defoliators, in that infectious particles from the previous year's epidemic survive to cause an epidemic in the current generation,[25] and only larvae can become infected. Also, particles are unlikely to survive more than two generations (allowing for longer-term survival is trivial[25] and it may sometimes occur, but in the interests of simplicity here we allow only for survival over one generation). Accordingly, α is the rate at which particles are removed to a refuge, μ is the rate at which this year's particles break down, δ is the rate at which last year's particles break down, and $\bar{\nu}$ is the rate at which the disease is transmitted horizontally. Surviving hosts reproduce with fecundity λ, and variability in susceptibility to the disease is represented by C, the coefficient of variation of the distribution of susceptibility in the host population. ξ is the rate at which virus produced during the epidemic is removed to a refuge in which it can survive the winter.

This model was inspired by Anderson and May's[26] earlier continuous-time model of host-pathogen interactions, with the realistic difference that hosts reproduce only once per year rather than continuously. Also, many of the pathogens in question can only infect larvae, so that there can only be one epidemic per year. Equation (8) for the fraction of hosts recovering from an epidemic is therefore derived from a model for a single epidemic.[25] Because the model shows long-period cycles for a broad range of parameter values, like Anderson and May,[26] Dwyer et al.[25] use this model to argue that pathogens are likely to be the force driving outbreaks in many forest defoliators.

Each of the three models for outbreak dynamics is therefore characterized by cycles with a long period. Before considering the ability of each model to quantitatively explain the dynamics of our three defoliators, we first consider descriptions of time series of their densities as provided by time series analysis, to see if the models agree with the data qualitatively. First, the budmoth data (from ref. 27) show what is perhaps the most convincing evidence for cycles for any insect, with significant negative

autocorrelations at 4 and 12 years, and significant positive autocorrelations at 8 and 16 years, together suggesting a cycle with a period of eight years.[11] Autocorrelation of the much shorter western tent caterpillar time series (data from Myers, pers. comm.) likewise shows a significant negative autocorrelation at four years, and a positive autocorrelation at nine years, suggesting a cycle with a period of about 8–9 years. Unfortunately, however, even the best time series of gypsy moth densities in North America is very short,[28] and perhaps as a result does not show significant autocorrelations. Like the other time series, however, the gypsy moth data show a fluctuating, exponentially damped autocorrelation function, which is at least suggestive of cycles. More importantly, time series of defoliation in New England have shown that at least some populations undergo cycles.[29] These descriptive analyses therefore suggest that each insect shows long-period cycles in at least some part of its range.

Each of the three models for outbreak dynamics is therefore characterized by cycles with a long period.

This rough qualitative agreement between the data and the models encouraged us to compare the models to the data more quantitatively. To do this, we fit each model to each data set by minimizing the sum of the squared errors between the model and the data on a log scale (first computing 10,000 iterations of each model, to eliminate transients). In so doing, we are assuming that the only errors in the fit of the model to the data are due to observation error, rather than to biological processes that occur in nature that are not included in the model (so-called "process errors.")[30] Although undoubtedly there are biological processes affecting each insect that are missing from the models, in fitting the models to the data part of what we are attempting is to determine whether each model provides a sufficient explanation of the data. Our intention is therefore to test whether we can ignore missing processes and still achieve a good fit to the data, and we therefore consider only observation errors. A final detail is that, to minimize the sum of squares, we used the downhill simplex algorithm, with multiple restarts.[31]

Figures 1, 2, and 3 show the fit of each model to each data set. All the models fit the budmoth data best, which is not surprising given how little noise there appears to be in the budmoth data, and given the length of the time series. The models fit the gypsy moth data the worst, but the major reason for this poor fit is the poor quality of the density estimates in the two troughs.[28] Although there are also minor variations in the fit of particular models to particular data sets, the overall picture is that the fit of the models to any particular data set is indistinguishable. In other words, the data do not allow us to distinguish among models.

It thus appears that the existing time series data for these three defolia-

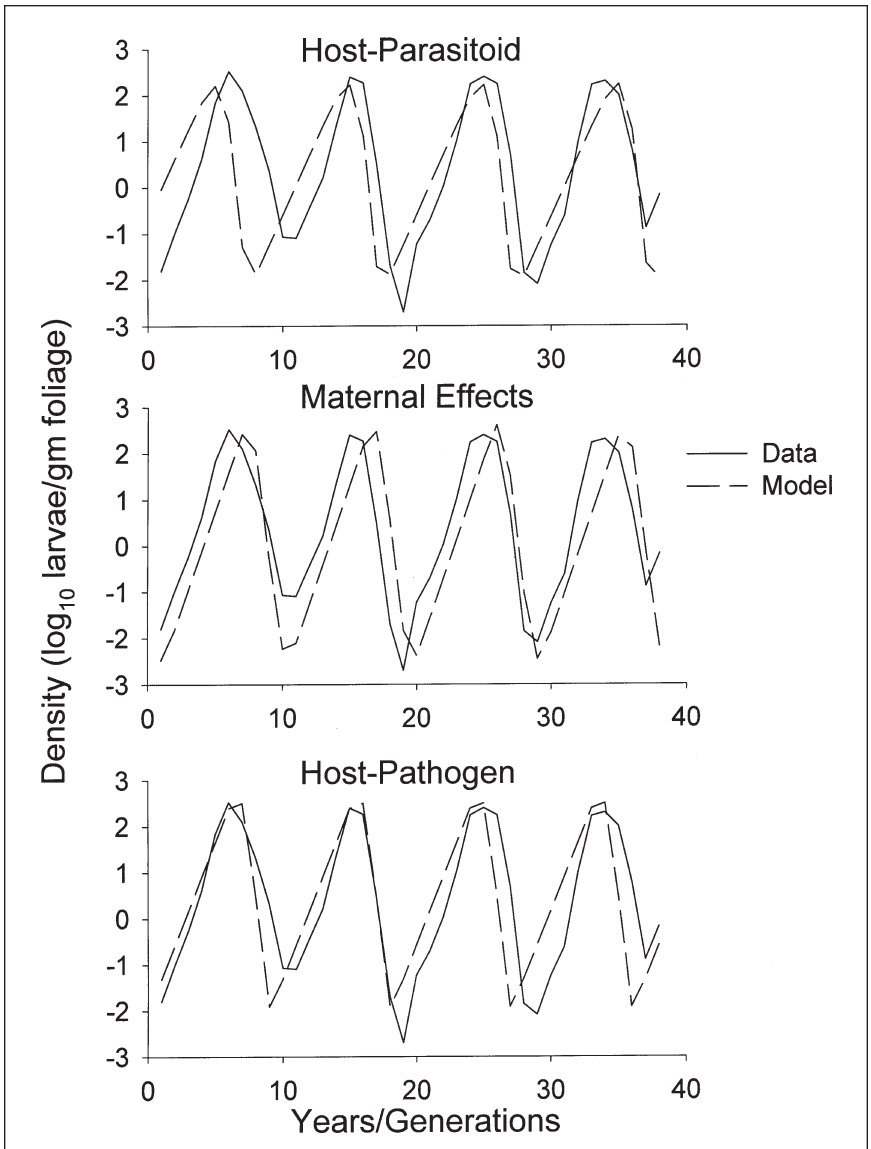

Figure 1. The fit of three models to the larch budmoth density time series. "Host-Parasitoid model" indicates equations (1)–(2), "Maternal effects model" indicates equations (3)–(5), "Host-Pathogen model" indicates equations (6)–(8).

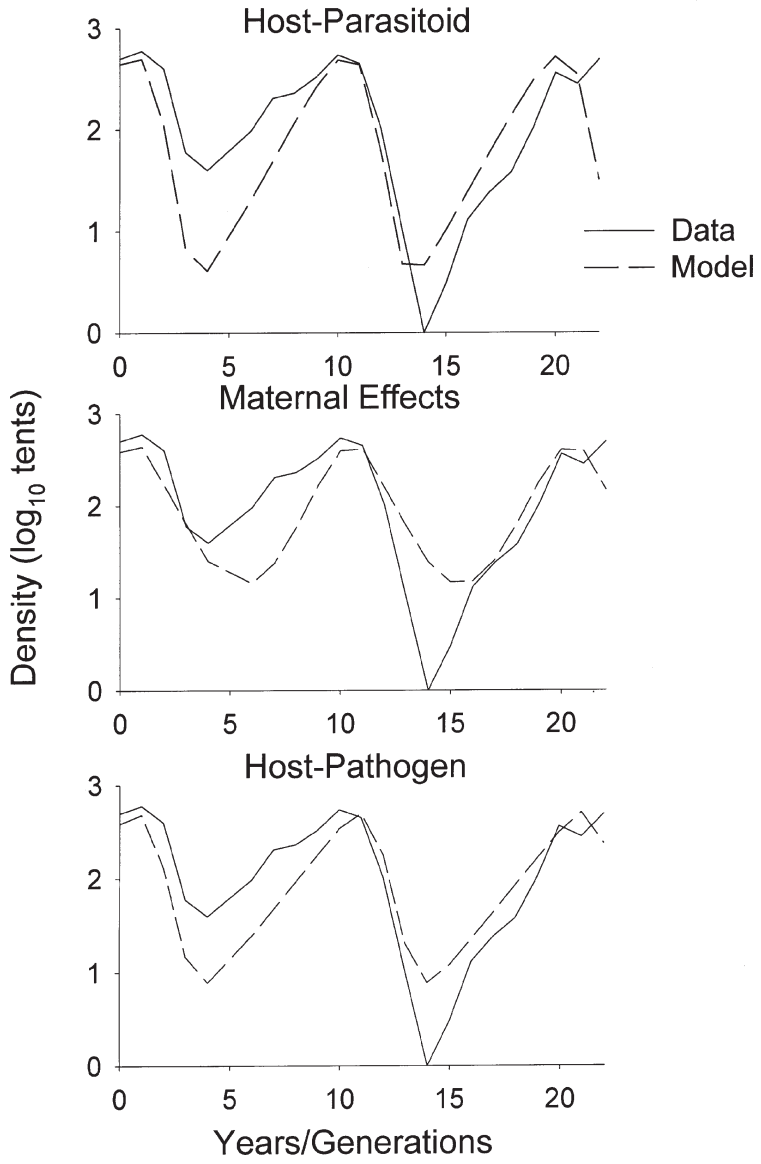

Figure 2. The fit of three models to western tent caterpillar density time series. The model equations are as in Figure 1.

tors do not allow us to determine the mechanism or mechanisms driving their dynamics. In other words, although the mechanisms embodied by the models are biologically very different, mathematically they are too similar to be distinguished by the data. It is worth pointing out, however, that other seemingly similar models do not fit nearly as well. For example, May[32] presented a model that is very similar to equations (6)–(8), except that he assumed that pathogen survival from the end of one epidemic to the beginning of the next is extremely low. The resulting model shows only high-frequency oscillations (i.e., alternating peaks and troughs) that clearly bear no resemblance to forest-defoliator dynamics. More quantitatively, Ginzburg and Taneyhill[22] compared the fit of the maternal effects model, equations (3)–(5), to the delayed logistic model,

$$N_{t+1} = N_t e^{r(1-N_{t-1}/K)}. \qquad (9)$$

Although this model can show long-period cycles, it generally fits data from various defoliators much more poorly than did the maternal effects model. Ginzburg and Taneyhill[22] argued that the difference in the fit of the two models occurs because, in the maternal effects model, densities affect subsequent growth rates, while in the delayed logistic model densities instead affect subsequent densities. They point out that this property is one that the maternal effects model shares with host-parasitoid models; indeed, Lauwerier and Metz[33] showed that a broad class of host-parasitoid models, including both equations (1)–(2) and a model that is closely related to Ginzburg and Taneyhill's maternal-effects model, have very similar dynamics.

There are thus strong similarities between host-parasitoid models and the maternal-effects model. In turn, the host-pathogen model, equations (6)–(8), is very similar to host-parasitoid models. To see this more clearly, first we use Hassell et al.'s[20] representation of a general host-parasitoid model:

It thus appears that the existing time series data for these three defoliators do not allow us to determine the mechanisms driving their dynamics.

$$N_{t+1} = \lambda N_t F(N_t, P_t) \qquad (10)$$

$$P_{t+1} = wN_t(1-F(N_t, P_t)). \qquad (11)$$

In equations (1)–(2), the per-generation host survival rate $F(N_t, P_t) = e^{-QP_t^{1-m}}$, but Hassell et al. list a wide variety of closely related models with somewhat different expressions for $F(N_t, P_t)$, that have similar dynamics. Comparing equations (10)–(11) with equations (6)–(7), we see that the two would be the same if $I(N_t, P_t) = 1 - F(N_t, P_t)$. In fact, the implicit expression for I, equation (8), is

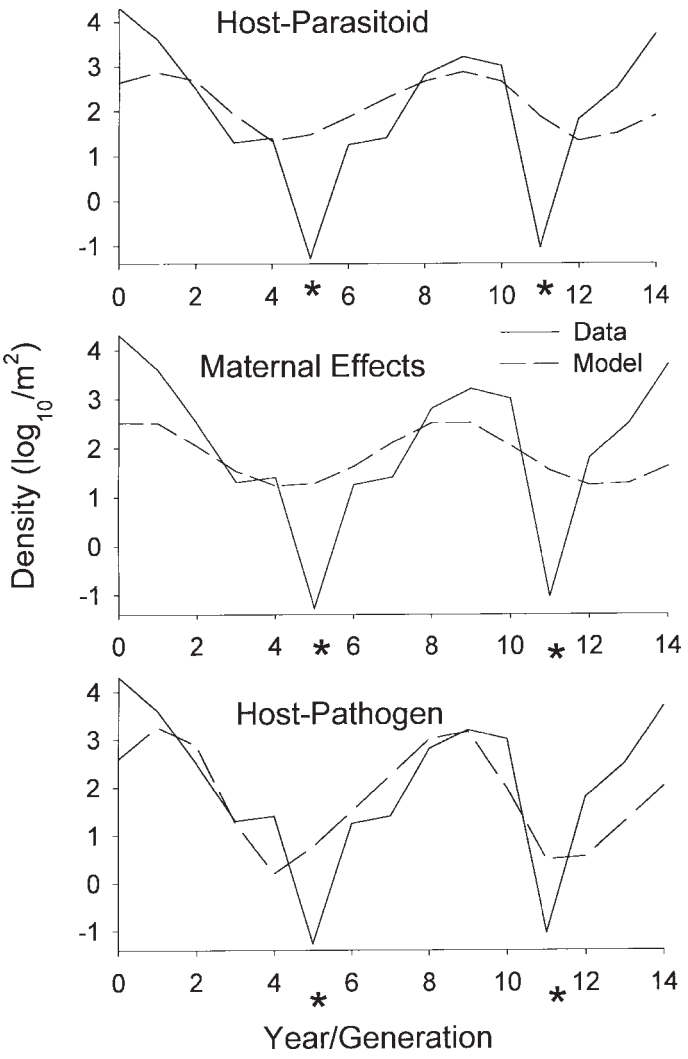

Figure 3. The fit of three models to gypsy moth density time series. The model equations are as in Figure 1. Asterisks (*) indicate densities that are based on very small sample sizes.

simply a host-parasitoid model in which there are many parasitoid generations per host generation, and in which there is heterogeneity for susceptibility in the host. Furthermore, May[34] presented a host-parasitoid model with patches in which parasitoids are distributed independently of host distribution, so that the parasitoid distribution follows a gamma distribution with mean 1 and coefficient of variation C, and the resulting model has $F(N_t,P_t) = (1 + aC^2P_t)^{-1/C^2}$. These two models are thus also very similar. An identical expression results in Godfray and Hassell's[35] host-parasitoid model that allows for host heterogeneity in susceptibility, where instead it is host susceptibility rather than parasitoid spatial distribution that is gamma-distributed.

In short, although the biology underlying our three models is quite different, the models are mathematically very similar. Consequently, not even the larch budmoth data can distinguish among the models, even though for an ecological data set it is very long and not very noisy. It thus appears that fitting models to time series data on outbreaking insects does not give one the ability to determine the details of the mechanism driving the outbreaks. In turn, the conclusions that one can draw from this kind of procedure are not really much different than what has already been concluded from fitting more phenomenological models,[11–13] which is that it seems likely that there is some kind of density-dependence driving outbreaks in many forest-defoliators.

Nevertheless, mechanistic models differ from phenomenological models in that they can be tested with other kinds of data besides time series of densities. Dwyer et al.,[36] for example, used a combination of data from lab experiments, field experiments, and naturally occurring virus epidemics to show that the model underlying equation (8) provides an excellent description of single epidemics in gypsy moth. Similarly, field experiments designed to test for maternal effects in gypsy moth have shown that such effects are negligible, thereby casting much doubt on the applicability of equations (3)–(5) irrespective of how well they fit the time series data. Field experiments by themselves can thus choose among models. More quantitatively, experimental data can be combined with long-term time series data to maximize our ability to distinguish among models. For example, models can be tested by estimating key model parameters using experimental data or short-term observational data, and then comparing the output of the parameterized model to long-term time series data,[25] with the difference that in this case the model

Fitting models to time series data on outbreaking insects does not give one the ability to determine the details of the mechanisms driving the outbreaks.

is making a prediction. Ultimately, however, such a priori approaches may be superseded by more sophisticated Bayesian analyses.[37]

Evidence From Field Experiments

In this section, we review field studies and the hypotheses proposed by field workers on the three systems analysed above. The point here is not to determine which hypothesis is the most strongly supported, but to show that there is evidence for many factors affecting dynamics in all of these systems.

Tent Caterpillars

Population dynamics

Western tent caterpillar outbreaks tend to be of short duration, but forest tent caterpillar outbreaks may last several years at a site. Few good time series of population densities (as opposed to defoliation areas) exist for these species. The best has been gathered by Myers ([38], personal communication) for western tent caterpillar and is now at 22 years. Autocorrelation analysis of this time series indicates a significant periodicity to outbreaks, with a return time of 9 years.

For both western tent caterpillar and forest tent caterpillar, outbreaks in widely separated areas tend to decline synchronously despite variation in the year they were initiated and the amount of defoliation.[4,39,40] Western tent caterpillar populations show a characteristic pattern of increasing fecundity during the increase phase, with the highest fecundity at the peak, and several years of low fecundity after the peak.[38]

An interesting aspect of outbreaks of forest tent caterpillar is that they tend to last longer in areas having more forest edge in Ontario.[41] While several potential mechanisms could explain this pattern, reduced transmission of virus at forest edges has been demonstrated.[42] Sunlight breaks down the virus particles at a rapid rate. Three important parasitoids also show higher attack rates in continuous than fragmented forests.[43] Experimental colonies of larvae survived better at forest edges mostly because of reduced mortality from NPV, but also because of reduced mortality from parasitoids as a group.[44] These field data and experiments suggest that virus is very important to forest tent caterpillar dynamics, with an additional contribution from parasitoids.

Very long outbreaks of forest tent caterpillar have been recorded on water tupelo in Louisiana, in some places. This is presumably because parasitoid puparia drown (they normally pupate in soil, while tent caterpillars pupate on branches).[45] Some Alabama populations have 2–4 year cycles that are repeated continually, apparently driven by larval starvation.[46] Virus incidence has not been investigated in these areas, but again the field work suggests an important role of parasitoids in the dynamics and mean levels of abundance.

Myers and her co-workers strongly implicate the importance of virus in western tent caterpillar population dynamics.

Proposed hypotheses

The first hypothesis proposed for western tent caterpillars was a maternal effects hypothesis, combined with weather.[47,48] Maternal provisioning of eggs resulted in different activity levels of larvae, and more active larvae make a bigger, differently shaped tent. Wellington[48] recorded changes in tent shapes with density. Low densities have high proportions of very active colonies, but the moths are also more active and tend to disperse away, according to this hypothesis, and less active colonies accumulate in the area of increasing density. The less active colonies were thought to be more susceptible to natural enemies and poor weather. In times of good weather, both types of colonies survive well, but in poor weather inactive colonies are decimated. Wellington thought the maternal effects resulted in poor fecundity for several generations after the decline, keeping population densities low. However, the first decline in the population occurred when the proportion of active colonies was still high,[4] suggesting that the decrease in insect quality follows the decline instead of initiating it. As well, a problem with this hypothesis and with others that rely on weather, is the lack of periodicity in weather conditions at the same 8–10 year time period as the insect outbreaks.[49]

Tent caterpillars are affected by several viruses, and bacterial diseases (*Clostridium brevifaciens* and *C. malacosomae*). Relatively little is known about the inter-colony and inter-generation transmission of *Clostridium*, and its abundance at different population densities. Spores are extremely infectious in lab trials[8] and can contaminate labs so that it is impossible to raise larvae in the same year. It could be important to dynamics, but it has not been studied.

Much more is known about the NPVs of tent caterpillars. Some outbreaks have been terminated by virus, but others were not.[8] Myers,[4,39,38] noting the pattern of increased fecundity up until the population peak and low fecundity for several years after the peak, suggested that virus and its sublethal effects on fecundity drive the cycles of western tent caterpillars. Sublethal effects of virus[50] could reduce fecundity for several generations after the outbreak, without causing noticeable mortality from virus. Selection for virus resistant genotypes that have lower fecundity could produce the same effect,[39] but in fact there is no relationship between fecundity and resistance to virus.[50,51] The experiments and observations of Myers and her co-workers strongly implicate the importance of virus in western tent caterpillar population dynamics.

The results of experimental introductions[39] also refute two alternative hypotheses. The fact that populations with different histories decline at the same time, also suggests that the maternal effects hypothesis cannot explain outbreaks of western tent caterpillar and forest tent caterpillar.[39] As well, the syn-

chronous declines refute the induced foliage defense hypothesis, because the declines did not depend on the defoliation history of the trees. Similarly, many forest tent caterpillar populations decline before they cause much defoliation of hosts, and synchronously with populations in other regions with full-fledged outbreaks.[40] Additional evidence that there are no defoliation-induced defenses in this system comes from the experiments of Myers and Williams[52] although Adams (1989, cited in 8) found defoliation-induced defenses in red alder affected the growth of western tent caterpillar larvae, at least in the short term.

Parasitoids and predators contribute high mortality to declining populations of both western tent caterpillar and forest tent caterpillar. Larval and pupal parasitism rates increase over the course of forest tent caterpillar outbreaks, and pupal parasitoids in particular could potentially have driven the cycle.[9] Experimental populations of forest tent caterpillar had spatially density-dependent responses by two larval parasitoids, but surprisingly little mortality from the most important pupal parasitoid of outbreaks, and were decimated by bird predation.[53] While parasitoids and predators contribute to declines, Myers notes that they cannot account for the pattern of fecundity which is consistently observed in different populations of western tent caterpillar and possibly also forest tent caterpillar.[39]

In the 40s, 50s and 60s, weather-related hypotheses were favoured forest tent caterpillar dynamics. In particular, freezing weather in spring that affected newly-hatched larvae directly or by killing leaves of the host plant, was thought to be the most important factor limiting population, especially when followed by prolonged periods of cool temperatures and overcast skies later in the season (for review see 8, 40, 54). Fitzgerald[8] reviewing this literature, concluded that weather was the most frequently cited cause of the termination of tent caterpillar outbreaks in northern regions. The most recent analysis shows there is no consistent relationship between weather and increases or declines in forest tent caterpillar in different regions of Ontario.[40] However, defoliation was less severe in areas with low overwintering temperatures, which may have suffered more egg mortality.[40]

Nonetheless, there are many difficulties with this type of analysis of population change.[54] Foremost among them is the large number of climate variables that could be correlated to the occurrence of outbreaks. As well, the duration of the release period is important. That is, how many years before the outbreak do favourable conditions occur? A long window increases your chances of finding a correlation to some weather variable. Finally, these correlative methods are not easily tested by experimental manipulations, and thus the hypotheses are difficult to refute.

In summary, field data and experiments suggest a) virus and its sublethal effects are the most important element in western tent caterpillar dynamics; b) parasitoids contribute to dynamics via numerical responses to tent caterpillar density, but these responses may be altered by forest structure, i.e. edge effects; c) weather is potentially a powerful modifier of dynamics through direct effects on overwintering or early spring mortality.

The outbreak predicted for 1990 failed to materialize.

Larch Budmoth

Population dynamics

The species displays regular cycles in the Alps, particularly at elevations of 1600–2100 m in the Engadine Valley in Switzerland, that can be traced back 500 years using tree rings.[55] At lower elevations, the outbreaks are less frequent and less regular.[56] The reduction in outbreaks at lower levels was attributed to high egg mortality, due to higher temperatures in the overwintering stage.[57] At very high elevations, moth emergence is late enough that there are few oviposition opportunities, so densities are kept low. The subalpine habitat has the highest diversity of natural enemies,[57] so reduced complexity does not explain these outbreaks. Outbreaks have been recorded as well on other host species, including introduced lodgepole pine and sitka spruce at other locations in Europe, but these outbreaks apparently lack periodicity.[57]

The outbreak predicted for 1990 failed to materialize. Initial defoliation in 1989 followed the historical pattern, but defoliation did not occur in 1990 or 1991 as populations declined to very low levels.[58] Unusual weather conditions in all three of these years were proposed to be the proximate causes for the failure of the expected outbreak.[58] Very warm weather in March, but cold weather in April and May were thought to deplete egg resources, stimulate too early hatch, and result in starvation of young larvae.

Proposed hypotheses

For the larch budmoth, nearly all field work has been done by Werner Baltensweiler and his colleagues. Early on, Baltensweiler (1966, cited in 57) proposed a weather-driven model, and confirmed it with multiple regression. However Auer,[59] using key factor analysis, could not support this model, but found that parasitoids and defoliation were the dominant regulating factors. Varley and Gradwell[60] criticized this conclusion, but their model did not incorporate defoliation which has important effects.[57] Next, host-pathogen models were proposed for this system.[26] An epizootic of granulosis virus was associated with the termination of the 1954 outbreak. While the fit of a host pathogen model to the data was excellent, no epizootics were observed in subsequent cycles, and the prevalence of disease was too low even in the 1954 epizootic. Host-parasitoid models have also been discounted, since Berryman[61] analysed Baltensweiler's data on larch budmoth and its parasitoids and found that the two dominant parasitoids only explained 28% of the per capita growth rate of the budmoth.

Later, Baltensweiler favored a host plant quality hypothesis. Food quality decreases after defoliation (or in response to frost kill), with a recovery period of 2 to 7 years.[62] The depressed

survival and fecundity of budmoths after defoliation could drive the cycles.[56] In the growth phase, high survival and fecundity allow 10-fold growth from one generation to another, until they reach the defoliation threshold density and the cycle starts again. This is the hypothesis favored by Baltensweiler and Fischlin,[56] with synchronization of outbreaks caused by dispersal among valleys. Flight activity is increased after defoliation, and females prefer to oviposit on green foliage rather than defoliated trees. Hence populations are redistributed to less defoliated areas.[57]

Later still, Baltensweiler modified this scenario, proposing the "polymorphic fitness hypothesis,"[63] a variation on the Chitty hypothesis involving interaction between genotypes and defoliation. There are different genotypes of the species, corresponding to colour morphs that vary in abundance according to the stage of the population cycle (with the dark morph most abundant during the growth stage). The colour morphs have different preferences for and performances on larch versus cembran pine hosts. They also have slight differences in mating phenology that arise from the synchrony of egg hatch with the budbreak of their respective preferred hosts (2 weeks apart). The darker, larger, more fecund morph associated with outbreaks does not perform as well on suboptimal food, i.e. after defoliation. This morph is homozygous recessive, and maintained in the population by assortative mating which is caused partly by earlier development and partly by pheromones.[63] Baltensweiler [63] hypothesized that the assortative mating breaks down after outbreaks, because of low survival of the dark morphs. Only after 3–4 years of recovery are they able to increase in frequency. This is a complex hypothesis involving both defoliation-induced changes in food quality and the genetic variation in this organism.

In summary, the data suggest a) some external factor, probably weather affecting egg mortality, is important in determining where (and in the case of the 1990 non-outbreak, when) populations of LBM will have regular cycles; b) a complex hypothesis involving both genetic polymorphism and long-term induced resistance probably drives the cycles in the Engadin valley.

Gypsy Moth

Population dynamics

The species has outbreaks in most of the Eurasian range where oaks occur.[64] It also has outbreaks in North America, where it was inadvertently introduced. Much of the work on population dynamics has focussed on the North American populations. European populations have had outbreaks every 8–11 years with some regularity,[3] although no quantitative analysis has been applied to these data. In Massachusetts and Maine, there is little evidence of cyclic recurrence of defoliation, but defoliation has occurred at 8 year intervals in New Hampshire and Vermont.[29]

Since gypsy moth outbreaks are often synchronous over large regions, weather is probably a factor but the mechanism is obscure.

Proposed hypotheses

While some workers believe that gypsy moth in North America are regulated by parasitoids,[61] rates of parasitism are much lower than for European populations, for which there is better evidence of regulation by parasitoids.[3] As well, temporal density dependence of parasitism has not been detected in North American populations.[65] Although experimental populations are decimated by parasitoids, these releases are on a relatively small scale.[66] Area-wide outbreaks would not allow the immigration of parasitoids that suppresses these small experimental releases.

Predation on the late instars was the favored hypothesis of Campbell[67] based on his analysis of life table data from New England. Late larval survival was the highest source of variation in population density at any density, and late larval and pupal survival were density dependent at low densities, though not at high densities. From these observations, the bimodal stability hypothesis was developed. It suggests that there are two stable densities of gypsy moth, with rapid transition between them.[68] Low densities are regulated by predation, and high densities by depletion of resources and virus. The transition from low to high density ranges occurs when the functional and numerical responses of predators are saturated by a sudden increase in GM density. Analysis of long-term data on gypsy moth, vertebrate predators, and acorns (an important winter food resource for the predators) suggest that acorn supplies regulate mouse populations, which in turn can regulate low density gypsy moth populations.[28,69] Collapses of acorn supplies reduce mouse populations, allowing gypsy moth populations to increase. It is also possible that other perturbations could increase gypsy moth density to the point where they satiate the vertebrate predators. Acorn crop production is synchronized at large scales,[70] so this may help to explain the very large scale of gypsy moth outbreaks. Mouse densities do not explain all variation in low density populations of gypsy moth,[69] so weather or timing effects mentioned below, or other predators, parasitoids and disease contribute additional variation.

The induced defense hypothesis can also be proposed for gypsy moth. Defoliation causes the leaves produced in the next year to be lower in quality, reducing gypsy moth fecundity and survival, and increasing development time.[71] However, no long-term studies have examined whether host quality is depressed for several years after defoliation, as required to explain the long intervals between outbreaks. Foliage

quality also affects gypsy moth susceptibility to virus[72] and to parasitism.[73] A hypothesis involving defoliation and changes in susceptibility to virus leading to outbreaks was proposed;[74] however subsequent experiments have failed to show the necessary response to defoliation.[75]

The conditions experienced by the adults can affect the characteristics of offspring, such as growth rate, ultimate size and propensity to disperse. For outbreaks, a time-delayed feedback is required and this could be provided by such parental (usually maternal) effects.[22,76] Elkinton et al.[69] rejected this and other simple, single-factor models of gypsy moth dynamics because they do not allow the low-density fluctuations seen in their data.

Since gypsy moth outbreaks are often synchronous over large regions, weather is probably a factor but the mechanism is obscure. Analysis of weather associated with outbreaks gives a somewhat inconsistent picture. This is probably due to choice of variables to analyse. Most frequently, however, a correlation to warm dry weather, especially in early spring, is cited (for review see 3, 29, 77). Potential mechanisms through which these weather conditions could affect gypsy moth populations are: more rapid development of larvae, decreasing the duration of exposure to natural enemies; moisture stress effects on host trees, increasing the quality of food; and greater establishment success during the critical post-hatch host-seeking period.

Many outbreaking species are, like these three species, spring-feeding. Success in a given year may depend on ability to synchronize with the new leaves of the host plant, which are more nutritious than older leaves. A strong correlation between the amount of time that larvae have to seek hosts in early spring (mostly determined by weather after hatching began) and population size was found for gypsy moth.[78] Variable conditions in early spring could contribute to variability in population sizes. Indeed, experimental releases of gypsy moth eggs at different times in early spring resulted in very different levels of mortality of populations (Hunter and Elkinton, in review). The differences were mostly due to natural enemies, rather than the host plant (host plant effects were expressed as differences in fecundity). In fact the differences in natural enemy mortality were partly due differences in density caused by differential ballooning from the foliage, as well as to direct natural enemy effects (Hunter and Elkinton, in review). Weather effects such as these, or effects on gypsy moth predators and parasitoids more directly may bring about the initiation of outbreaks.

In summary, for gypsy moth, it is clear that the most important factor depends on the density range. At low densities, populations are regulated by vertebrate predation, but can escape this regulation by increasing beyond the density level that satiates the predators. These increases may be caused by acorn crop failures that reduce mouse populations, by massive immigration of larvae, or by weather effects on larval survival. At high densities, virus disease, and perhaps also defoliation-induced defenses become important. Thus gypsy moth dynamics are controlled by multiple factors.

Insect outbreaks may rarely, if ever, be attributable to a single factor.

CONCLUSIONS

A great deal of ecological field work and ecological theory is concerned with understanding particular processes or factors. For example, we have models of competition and hundreds of competitor removal experiments, and we have have models of predation and hundreds of predator removal experiments. Although we have learned a great deal from this single-factor approach, our review of studies of outbreaking insects shows that such an approach is not very effective in studying the dynamics of particular systems. The existing data suggest that cyclic outbreaks of larch budmoth, western tent caterpillar, and gypsy moth are driven by at least several factors, and that weather, natural enemies and host-plant quality play a role in the dynamics of all three species. Insect outbreaks may rarely, if ever, be attributable to a single factor; indeed the one generality about outbreaking insects appears to be that their populations explode because of the coincidence of several factors, and collapse because of several other factors. Nevertheless, this is not a conclusion that is reached by fitting mechanistic models to data, nor is it a popular conclusion among most field biologists that have studied these insects. In fact, most field biologists and most theoreticians have instead favored single hypotheses which they test to the exclusion of all others.

Investigator bias, however, is not the only problem, in that different methods have different strengths and weaknesses. For example, field experiments often take place over such small temporal and spatial scales that they may not be relevant to real outbreaks. Although models in contrast are focused on the relevant (large spatial and long temporal) scales, very different models can fit the same data equally well, so it does not appear to be possible to use time series data to distinguish among models. On the other hand, as we have discussed, experimental estimates of model parameters can rule out some models,[30] but efforts in this direction have again focused largely on single-factor models.[25] What is needed is a combination of theory and experiment that focuses on hypotheses of intermediate complexity, thus filling the gap between the extremely simple and extremely complex models that currently dominate much of population ecology. This would permit models to correspond more closely to the experiments and observations of field workers, and would make it easier for experimenters to test models. The ultimate result will be a deeper understanding of population dynamics, not just of forest defoliating insects, but of living things in general.

ACKNOWLEDGMENTS

Both authors gratefully acknowledge the support of the National Science Foundation, Division of Environmental Biology, Ecological Studies Cluster. Alison Hunter was supported by award number DEB-95-27792. Greg Dwyer was supported by award number DEB-97-07610. Also, both authors have benefited enormously from the advice, influence and general scientific intelligence of Judy Myers and Joe Elkinton. The opinions expressed are those of the authors, although if you are searching for someone to blame you might just as well blame Judy and Joe as well.

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