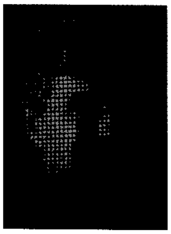
On the Principles of Population Dynamics and Theoretical Models



Berryman

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I HAVE FOLLOWED WITH CONSIDERABLE INTEREST the debate that my Forum article (Berryman 1991a) has generated (Onstad 1991, Berryman 1991b, Logan 1994, Hess 1996). However, my major point seems to have been lost in the shuffle. I did not intend to emphasize the idea that small (beautiful?) models are better than large (ugly?) models, but, rather, that all models should rest on a firm theoretical foundation, and that, when we build population models, the appropriate foundation is the theory of population dynamics. I was protesting the tendency for entomologists to build population models in an ad hoc manner around the life cycle of the insect(s) of interest without any reference to this theory. True, theoretical models often are small, but so are purely statistical models like regression equations. What is more important is that the underlying theory acts as a framework that holds the model together and gives it elegance and comprehension—elegance referring to the refinement and polish of a model (as a model of insecticide resistance would appear crude and unpolished if it ignored the theory of evolution), and comprehension referring to the understanding that the model imparts (as a model of insecticide resistance should impart an understanding of the evolution of resistance).

I was in the process of writing an article to further explain this point, when I was confronted by Price's (1996) claim that a "new paradigm is emerging" in insect population dynamics theory, a new paradigm that supposedly will replace the old one that I was writing about. Because Price does not describe the "old paradigm," and because a description is necessary if the reader is to make an informed decision on whether the "new" should replace the "old," I have changed the slant of my original article to describe, in as straightforward a manner as possible, the modern theory of population dynamics (old paradigm) and to try to show how it is more logical and general than the "new paradigm" proposed by Price (1996). In line with my original intent, I also will try to show that models built around the modern theory have broad predictive and explanatory powers.

Contemporary Theory of Population Dynamics

As most entomologists (including this one) are of a practical bent, I will begin my review of the modern theory of population dynamics by reference to a specific practical problem. Assume we are in the winter of 1985–1986 preparing for an expected outbreak of the Douglas-fir tussock moth, *Orgyia pseudotsugata* (McDunnough), in northern Idaho. Pheromone trap data show that the tussock moth population has been growing steadily for the last 5 years (these are actual data and pest managers were, in fact, faced with this problem):

Year	Mean number of males per trap
1981	1.2
1982	3.1
1983	4.7
1984	12.5
1985	45.3

Obviously, forest managers are interested in knowing what the population trend will be over the next year or two, for then plans can be made to deal with the problem. In other words, we would like to be able to forecast the population expected in 1986 and 1987. To do this, we need a model (or a wild guess!).

The first thing one could do is to take a purely empirical approach and fit a simple linear model to the data,

$$N_t = A + Bt, \qquad (1)$$

with N_t the number of males caught per trap in year t. This model, with parameters $A = -15.92$, $B = 9.76$, provides a reasonably good fit to the data ($r^2 = 0.71$) but contains no population theory or biological insights whatsoever (although it does contain considerable statistical theory!). It is what I would call a purely *empirical* or *statistical* model. Notice that it is also a small (but ugly?) model. Of course, the model predicts a continued increase in the tussock moth population—43 males per trap in 1986 and 52 in 1987—whereas the actual captures were 29 and 2, respectively. In other words, the linear model predicts continued growth whereas, in reality, the population crashed before it exceeded the economic damage level!

Over 200 years ago, Malthus (1789) wrote the first theoretical treatise on population dynamics. His theory was based on a fundamental insight—that populations of living organisms have the potential to grow geometrically—what has been called the "Malthusian Law," or what I like to call the *first principle of population dynamics*. Knowing this principle, most entomologists would transform the pheromone trap data to logarithms (ideally natural logs) before regression analysis, to give the model

$$\ln N_t = A + Bt. \qquad (2)$$

With $A = -0.757$ and $B = 0.866$, this model fits the data extremely well ($r^2 = 0.97$), but its predictions—84 moths in 1986 and 1,222 in 1987—are way out of line. In fact, the purely empirical model gave better results. The failure of the geometric model does not mean that the first principle of population dynamics is incorrect, for all populations have this intrinsic potential, but, rather, that is not sufficient to describe tussock moth dynamics. Geometric growth is only a part, not the whole, of the theory of population dynamics.

Malthus, of course, realized that populations cannot grow indefinitely and, therefore, that the geometric law was insufficient. However, it took another 50 years for Verhulst (1838) to develop the first mathematical model of what Malthus called the "struggle for existence," a model that he called the "logistic equation" (note that "struggle for existence" is synonymous with modern concepts of "intraspecific competition," "density dependence," and "density-induced negative feedback" in population dynamics, so that the logistic is the first model for density dependence, a concept formalized and argued over incessantly by entomologists (e.g., Huffaker et al. 1984). I like to call this the *third principle of population dynamics* (we will encounter the second principle later).

Verhulst formulated his model as a differential equation but, in reality, many insects do not reproduce continuously, and, so, a continuous model is not generally appropriate. The fish biologist Ricker (1954) seems to have been the first to propose a *discrete* version of the logistic equation

$$N_t = N_{t-1}e^{A-BN_{t-1}} \qquad (3)$$

Unfortunately, we cannot fit the discrete logistic model to the pheromone trap data because the series of observations is not long enough. This is because the logistic model is an "equilibrium" model in the sense that the population will, in time, attain a constant density or fluctuate around a constant level (sometimes called the "carrying capacity of the environment"). To fit the logistic, therefore, the series of observations must be long enough to exhibit fluctuations at or around equilibrium. Obviously, the pheromone trap data do not conform to this, so we have to look elsewhere. This should not concern us too much because a theory is supposed to be general in which case, models fit to data from one place should be applicable elsewhere, at least in a qualitative sense. We will use 10 years of data collected by Mason (1974 and personal communication) on Aztec Peak in central Arizona, as it was the analysis of these data that first made me aware of the utility of logistic models (Berryman 1978a). Fitting the discrete logistic equation (3) to these data yields the parameters $A = 0.315$, $B = 0.00051$, which neither explains the Arizona data very well ($r^2 = 0.18$) nor predicts the trap captures very accurately (58, 62). The discrete logistic model, therefore, is also insufficient to describe tussock moth dynamics.

Hutchinson (1948) seems to have been the first to realize that circular causal processes can give rise to delayed density dependence and cause density cycles in biological populations (note that circular causality often is caused by trophic relationships between plants and herbivores or prey and predators [e.g., Berryman et al. 1987]). I call this the *fourth principle of population dynamics*. As tussock moth populations are known to exhibit 9–11 year cycles, I decided to test the time-delay hypothesis on the Arizona data (Berryman 1978a). The discrete, *time-delayed* logistic model

$$N_t = N_{t-1}e^{A-BN_{t-d}}, \qquad (4)$$

with d the delay in the density-induced negative feedback, gave a better fit to the Arizona data ($A = 0.451$, $B = 0.000995$, $d = 2$, $r^2 = 0.70$), but its predictions of pheromone trap captures (63, 63) were still not good. The generalization of the logistic model to include time delays still seems insufficient to describe tussock moth dynamics.

In the classical logistic model, density dependent feedback is assumed to respond linearly to changes in population density. However, there is no theoretical or empirical basis for this assumption. On the contrary, most data show a decidedly nonlinear relationship (e.g., Huffaker et al. 1984, Fig. 12.3). Richards (1959) seems to have been the first to propose a *nonlinear* logistic equation, which we write in discrete, time-delayed form

$$N_t = N_{t-1}e^{A-BN_{t-d}^{Q}} \qquad (5)$$

with Q a coefficient of nonlinearity. This model, with parameters $A = 1.543$, $B = 0.289$, $d = 2$, and $Q = 0.32$, fits the Aztec Peak data quite well ($r^2 = 0.81$) and provides us with our first *qualitatively* correct prediction of pheromone trap captures (55, 33), to the effect that the tussock moth population will decline before it exceeds the economic damage level.

Although the discrete, nonlinear, time-delayed logistic equation seems to provide a reasonable qualitative description of the dynamics of Douglas-fir tussock moth populations, it certainly is not the only model that could be used for this purpose (see Hassell 1975, Royama 1977, Turchin 1990, Turchin and Taylor 1992 for examples of alternative models). In fact, a two-delay generalization of the Gompertz equation (Royama 1977)

$$N_t = N_{t-1}e^{A-B\ln N_{t-1}-C\ln N_{t-2}} \qquad (6)$$

fits the Arizona data better than the logistic ($A = 2.347$, $B = -0.302$, $C = 0.795$, $r^2 = 0.86$) and also provides a better forecast of pheromone trap captures (42, 19). Hence, although the logistic and Gompertz models are theoretically correct, in that they contain the relevant principles of geometric growth and delayed nonlinear density dependence, and although both give correct qualitative predictions (a population decline before serious defoliation), the Gompertz model provides a better quantitative forecast. The point to remember is that the model is not the theory but merely one of many possible *representations* of the theory. The form of the model, which is often a matter of personal preference, is less important than that it conform to the theory, or has theoretical integrity.

Of course, the fact that we have been able to describe and predict the population dynamics of a particular insect, the Douglas-fir tussock moth, does not mean that we have finished with population dynamics theory. The theory may be sufficient in this particular case but not in others. For example, my students and I have worked for many years on the population dynamics of the mountain pine beetle, *Dendroctonus ponderosae* Hopkins. Unlike the tussock moth, mountain pine beetle outbreaks in a given locality occur at irregular and widely separated intervals (>60 years). Also, unlike the tussock moth, the outbreaks do not arise and subside in restricted localities but often spread over vast areas—what I have termed *eruptive* outbreaks (note that Price [1996] used the term eruptive to describe *any* outbreak whereas the theory of population dynamics clearly distinguishes between gradient and eruptive outbreaks, with the Douglas-fir tussock moth being of the gradient type [Berryman 1987]). The reason for this eruptive phenomenon is that large beetle populations are capable of overwhelming the resistance of relatively healthy trees by pheromone-mediated mass attacks (Raffa and Berryman 1983). In this way, populations that build up in susceptible localities are able to spread into previously resistant stands (Berryman et al. 1984, Raffa and Berryman 1986). A general explanation for this phenomenon requires the notion of intraspecific *cooperation*, or what is sometimes called the "Allee effect" (Allee 1931). This is what I call the *second principle of population dynamics*.

Cooperation between individuals within a population gives rise to density-induced positive feedback, or inverse density dependence, a major cause of instability in population systems (Berryman 1981, 1989). This positive feedback can create unstable thresholds such as extinction thresholds below which the population automatically declines to extinction, and escape thresholds above which the population escapes from its limiting factors. In pine stands infested by mountain pine beetles, there is a beetle population density (escape threshold) above which the population can successfully attack almost any tree in the stand and below which it is restricted to dying or severely stressed trees. A bark beetle eruption will occur only if the population exceeds this escape threshold.

The notion of unstable thresholds and multiple stable states was first raised in monographs by Ricker (1954) on fisheries dynamics and by Morris (1963) on spruce budworm dynamics. Holling (1966) showed how the response of predators that switch to more abundant prey species or aggregate at localities where prey are more abundant can give rise to escape thresholds in the prey population. These ideas, which have been extended and generalized by Takahashi (1964), Southwood and Commins (1976), and me (Berryman 1978b, 1981, 1987), are the most recent contributions to the theory of population dynamics.

So, the modern theory of population dynamics rests on four fundamental principles: geometric growth (GD), inverse density dependence (IDD), rapid density dependence (RDD), and delayed density dependence (DDD). I currently think that these four principles are necessary *and* sufficient to describe the population dynamics of any group of organisms living in a variable environment, just as the principles of variability, heritability, and mutation are necessary and sufficient to describe the evolution of species in a variable environment. As with the theory of evolution, population dynamics theory does not attempt to identify the specific biological mechanisms by which these principles are evoked. In fact, Darwin formulated his theory of evolution without any knowledge of the molecular mechanism that controls variability, heritability, and mutation. Some of the mechanisms that could evoke the principles of population dynamics include competition for resources (RDD), predator switching/aggregation (RDD and IDD), cooperative hunting and defense behaviors (IDD), plant-herbivore and prey-predator interactions (DDD), and physiological or genetic polymorphism (DDD?). Some of these mechanisms may involve adaptive traits, such as territorial or social behavior; others may not, as in "scramble" competition for resources (for detailed discussions, see Berryman 1981, Berryman et al. 1987, Myers 1988, Sinclair 1989).

New Paradigms?

Having outlined the contemporary theory of population dynamics, we can now turn our attention to Price's "new paradigm" for population dynamics (see also Price and Hunter 1995). Price (1996) argued that the dynamics of a species population are determined by the genetic properties of the species, what he calls an "adaptive syndrome" and others might call a "life history strategy." More specifically, he proposes that latent (nonoutbreak) dynamics are characteristic of species in which females select the food supply for their offspring, whereas outbreak dynamics are typical of those that do not. Price seeks an evolutionary explanation for population fluctuations and, like others who have attempted this, he is largely unsuccessful. This is because population dynamics are the result of ecological interactions that are only partly dependent on evolutionary strategies (Wallner 1987, Berryman et al. 1995). True, the genetic characteristics of all the species in the community, including host plants, competitors, predators, and parasites determine the potential network of ecological interactions between species, but physical factors like topography and climate may strongly affect the strength and persistence of these interactions. For this reason, outbreaks may occur in one location but not in others. For example, Douglas-fir tussock moth outbreaks are observed in the Blue Mountains of Oregon but not in the Cascades (Mason 1996), whereas the autumnal moth, *Oporinia (Epirrita) autumnata* Borkhausen, outbreaks occur regularly in northern Fennoscandia but not in the Swiss Alps, even though the insects are present at both localities (Berryman 1996). If the "adaptive syndrome" of these insects was the primary cause of population fluctuations, we should observe similar dynamics in all environments where the species exist. In fact, it is the properties of the physical and biotic environment, as well as the genetic properties of a particular species, that determine the population dynamics of that species (e.g., Huffaker et al. 1984). Price's new paradigm is but a part of the old.

Price makes the general prediction, based on his work with galling willow sawflies, that outbreak species will be characterized by females who are not particularly selective about where they lay their eggs, whereas the females of nonoutbreak species will be more selective. However, this prediction does not seem to hold even amongst the sawflies, many of which are fastidious about where they lay their eggs. For example, diprionid sawflies are very particular about the age of pine foliage in which they oviposit, yet several species exhibit dramatic outbreaks (Larsson et al. 1993). Neither does the prediction hold for gall-forming insects, some of which exhibit outbreaks under certain environmental conditions (e.g., Isaev et al. 1988). Hence, the major prediction of the new paradigm generally does not hold.

Conclusions

A theory is a statement of the principles and/or processes that describe and explain an observed phenomenon, as the theory of evolution describes and explains speciation and the diversity of life. The principles on which the theory rests define the general processes that cause the phenomenon, not necessarily the specific mechanisms in any given situation. Hence, 10-year cycles of Douglas-fir tussock moths result from the action of delayed density-induced negative feedback (or DDD), as do cycles of snowshoe hares and lemmings. This principle applies to all dynamic systems and is, therefore, a principle of general systems theory, of which ecological systems are part (Berryman 1989). The specific mechanisms that give rise to the delayed negative feedback, however, may be different in tussock moths, snowshoe hares, and lemmings.

The general theory of population dynamics rests upon the four fundamental principles defined above:

1. The principle of *geometric growth* (Malthus' "law"),
2. The principle of *inverse density dependence* or *density-induced positive feedback* (Allee's ideas on animal aggregation),
3. The principle of *rapid density dependence*, or what I prefer to call *rapid density-induced negative feedback* (Malthus' idea of "struggle for existence"), and
4. The principle of *delayed density dependence* or *delayed density-induced negative feedback* (Hutchinson's notion of circular causal processes).

Although not essential, I would like to add one more to this list:

5. The principle of *limiting factors* or "Liebig's law of the minimum" (Liebig 1840, Blackman 1905).

This principle recognizes that, although populations are embedded in webs of interactions with their biotic and physical environments, only one factor (or a guild) is likely to limit numbers in the vicinity of equilibrium—the so-called limiting factor (Knisley and Juliano 1988, Paine 1992, Morris 1992, Berryman 1993). This principle implies that we often can expect to find key factors dominating the dynamics of insect populations (Morris 1959), and that it is the interaction with this factor that determines which principles are involved in the dynamics of the population.

The first four principles of population dynamics are probably sufficient to describe and explain the *endogenous* (density dependent) dynamics of any population (but not all of them will always be necessary), and the fifth is useful for identifying the specific mechanism(s) involved in these dynamics. Furthermore, if one can identify the principles operating in a specific situation, it may be possible to choose an appropriate mathematical model to describe the dynamics. Finally, if data are available, then it also may be possible to estimate the parameters of the model and, thereby, make quantitative forecasts of population change. In other words, the theory can be put to use.

Let me try and illustrate how population dynamics theory can be applied to real situations. We can approach the problem from two different directions: In the first, we study the ecology of the organism by observation and experiment in an attempt to discover the major biological mechanism(s) affecting its reproduction and survival. This is the approach taken by Price with his willow sawflies, and by us with the mountain pine beetle. If these investigations are successful, we should be able to identify the major factor(s) limiting population growth, and from this to formulate hypotheses for the principles and mechanism(s) regulating population dynamics. For instance, Price identified the supply of fast growing shoots as the major factor limiting the abundance of stem-galling sawflies on shrubby willows. Because he found no evidence of cooperative interactions or that food supply was affected by sawfly abundance, the second and fourth principles are unlikely to be evoked, and so only the first and third are needed to describe and explain sawfly dynamics. Following this line of reasoning, the most plausible mechanism regulating the dynamics of willow sawfly populations is the Malthusian struggle (competition) between females for oviposition sites and/or larvae for space. This means that equation (5), with the time delay set to $d = 1$ (or some other similar model), would be appropriate for modeling sawfly dynamics. However, as far as I am aware, there are no population data suitable for estimating the parameters of the model, and, so, quantitative predictions are not possible.

The case of the mountain pine beetle is somewhat more complicated. Although our studies led us to the same conclusion as Price, that beetle abundance was determined by the supply of susceptible hosts, we also found that this quantity was affected by beetle density (i.e., large beetle populations not only increased their food supply [second principle], but also decreased it by killing the trees [fourth principle]). The appropriate model could have a low-density stable equilibrium, determined by the supply of stressed or dying trees; an unstable escape threshold, determined by stand resistance; and a high-density cycle due to stand destruction (e.g., see Berryman et al. 1984, Raffa and Berryman 1986). Once again, data for estimating model parameters are unavailable and so quantitative forecasts are not possible.

The second approach used in studying population dynamics is to analyze a relatively long series of observations on the density of a population (a time series) in an attempt to model and understand the causes of the observed fluctuations (e.g., Berryman 1994). Time series analysis is an appropriate approach when populations are censused at regular intervals, say annually, by wildlife or pest management agencies, but where detailed ecological studies are impossible or inconclusive. It is the approach I used with the Douglas-fir tussock moth population in central Arizona and Mason (1996) applied to several other localities with similar results. It led us to conclude that equation (5) or (6), with maximum time delay of $d = 2$, provided the best description of the data. Cyclic dynamics and delayed density dependence implies that the dynamics are dominated by the fourth principle, and that the mechanism involves feedback between the species and some component of its environment. In the case of the tussock moth, the most most plausible hypothesis is that the guild of parasitoids attacking eggs, larvae, and pupae is the limiting factor. However, because the principle of limiting factors only holds strictly close to equilibrium, and because tussock moth populations may spend much of their time far from equilibrium, other factors such as food and viral disease could have a significant impact on the dynamics at certain times. Notice that the time series approach enables us to develop hypotheses for the mechanisms responsible for observed population fluctuations and, at the same time, to parameterize models and make quantitative forecasts, whereas the other approach only allows qualitative predictions.

It is important to realize that the principles of population dynamics are not concerned with the effects of density independent, or *exogenous* factors. However, the effects of these factors can often be inferred from the endogenous model. Hence, willow sawfly numbers are expected to respond to changes in the abundance of fast-growing juvenile shoots, which, itself, is determined by soil moisture, an exogenous factor (Price 1996). Similarly, mountain pine beetle eruptions should follow disturbances that affect tree vigor, whereas the amplitude of Douglas-fir tussock moth population cycles should be sensitive to variations in the physical environment.

Before finishing, I would like to reemphasize my point that models are not *the* theory, but merely particular formal representations of the theory. The choice of mathematical formalism, and whether the model is large or small, is less important than that it conforms to the theory. Any model that does not consider, and incorporate where necessary, the basic principles of population dynamics, will be neither elegant nor comprehensible. Nor should it be trusted by those who depend on its predictions, any more than one would trust a rocketship controlled by those who have no knowledge of the theories of gravity and planetary motion.

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