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PREDATOR-PREY THEORY AND VARIABILITY

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INTRODUCTION

Field ecologists are often impressed by the variability of ecological processes (26). In spite of this, mathematical models for predator–prey interactions have been mostly deterministic. Stochastic components are included simply by modifying a deterministic model—often with considerable arbitrariness. There has been a tendency to regard stochasticity either as noise obscuring a deterministic signal or simply as a destabilizing influence. However, variability (stochasticity) plays a fundamental role in predator-prey population processes; it helps to explain observable population phenomena. In this review we explore ways in which deterministic models are inadequate and suggest some methods of modelling variability.

Variability can be classified according to its origin into five categories. These different kinds of variability affect population dynamics in different ways. Especially interesting is the effect of variability in the presence of population subdivision—i.e. when the populations are not homogeneously interacting because movements are not unlimited. In this situation it can be shown that within-individual variation, or demographic stochasticity as it is usually called, does not have a vanishingly small effect as the population gets large. This is contrary to current opinion (41, 57) based on models of populations that interact homogeneously.

The idea of a structural model is introduced below. Such models make qualitative assumptions and are designed to allow different models to be compared within a single organizing framework. Structural models also yield qualitative predictions depending only on their qualitative assumptions and are therefore especially useful for testing ideas in ecology. Current modelling procedures place too great an emphasis on specific models. More general approaches are needed, which depend less on particular models.

Stochastic systems require stochastic stability concepts. The idea of stochastic boundedness is an outgrowth of the concept of tightness of a class of probability measures. As a stability concept, stochastic boundedness seems more applicable to

real systems than do the usual deterministic stability notions. In particular, stability of this kind can be directly estimated from observed population fluctuations.

Although this review deals mainly with population models, it is not suggested that variability is unimportant at the behavior level. To the contrary, variability is fundamental at the behavior level because individual predators and prey are variable; they experience, and must respond to, highly variable phenomena (65). Simple stochastic behavior models are sometimes used to justify the mechanism of deterministic models; for example, the Nicholson-Bailey host-parasite model (63) depends on a random encounter submodel.

Five Kinds of Variability in Predator-Prey Systems

Perhaps the most obvious feature of animals is variation *between individuals*—i.e. differences in phenotypes; but the variability present in classical stochastic models of predator-prey systems (7, 8, 10, 11, 12, 45, 47, 48, 67) is of an essentially different nature: In these models, individuals in the same population are treated as identical in all aspects of the phenotype that matter. Variability, or randomness, enters as a *within-individual* process having the same probability distribution for every individual regardless of its phenotype. This within-individual variation may be compared with the toss of a coin. The outcome (heads or tails) is unpredictable not because this coin differs from other coins unpredictably, nor because the environment of the toss is unpredictable, but as a result of unpredictable factors inseparable from the particular toss of the coin. Within-individual variation is usually given the label "demographic stochasticity." To continue the coin analogy, between-individual variation arises in a population of biased coins where the degree of bias varies from coin to coin.

Environmental variation has both spatial and temporal aspects. Unpredictable temporal changes in the environment are usually called *random environment* (17, 25, 41, 53, 68). The environment may also vary spatially since, for example, not all places make equally good homes for animals. This is a kind of phenotypic variation between locality that we shall call *between-patch* variation. Here a patch may be delineated by genuine environmental discontinuity or may simply correspond to an observer's convenient subdivision of what is essentially a continuum.

Individuals in a population spread over a large area do not interact homogeneously with all individuals in the population, nor do they interact homogeneously with all individuals in any other population on the area. For example, the risk to any prey individual of predation by a given predator individual, in the time interval $(t, t+1]$, will depend on how close the individuals are at time t. Because of this, local variation in population size is important. *Within-patch* variation refers to the randomness present in population numbers for a given locality or "patch," and it arises from the combination of the random component of migration (to and from the patch), within- and between-individual variation, and that part of temporal environmental variation (random environment) that is local to a patch. The latter kind of environmental variation is discussed by Crowley (24).

We shall take a probabilistic view of variability, although in some cases variation may be described deterministically. The latter point of view can be considered within

the probabilistic framework by conditioning on the event that a particular pattern of variation is observed.

Of the five kinds of random variation in our classification two kinds, within-individual variation and random environment, have received the bulk of modelling attention. Moreover, within-individual variation has been discounted by deterministic modellers (57) because of the belief that it is unimportant in large populations. We shall see that this is not so; within-individual variation contributes to within-patch variation and therefore has a direct effect on local dynamics.

Stochastic Population Models

Population models usually consist of a specification of the way in which the state of a system develops from past states. The state of a system at any particular time t is represented by variables $\mathbf{Z}(t) = [Z_1(t), Z_2(t), \ldots]$. For example $Z_1(t)$ and $Z_2(t)$ may be respectively prey and predator population sizes, and there may be variables representing age structure, environmental conditions and so on.

A discrete time deterministic model can take the form

$$\mathbf{Z}(t+1) = \mathbf{g}(\mathbf{Z}(t)). \qquad 1.$$

In this model the state of the system at time $t+1$ is *determined* by, or is a function of, the state of the system at time t. More generally the state of the system at time $t+1$ can depend on both $\mathbf{Z}(t)$ and $\mathbf{Z}(t-1)$ or perhaps even the entire history $\mathbf{Z}(t), \mathbf{Z}(t-1), \ldots$. The model then takes the form

$$\mathbf{Z}(t+1) = \mathbf{g}(\mathbf{Z}(t), \mathbf{Z}(t-1), \ldots) \qquad 2.$$

Further generalizations make $\mathbf{g}$ time-dependent.

Continuous time deterministic modelling replaces the difference equations 1 and 2 by differential equations (56). Both discrete-time and continuous-time deterministic predator-prey models have been thoroughly reviewed recently (37, 58, 61).

In a stochastic model the state of the system at $t+1$ is not determined exactly by previous states; instead, a probability distribution is given. In essence for every set G of possible values for the state of the system, the model specifies the conditional probability that $\mathbf{Z}(t+1)$ belongs to G, given $\mathbf{Z}(t), \mathbf{Z}(t-1), \ldots$. The mathematical notation for this probability is

$$\mathrm{P}(\mathbf{Z}(t+1) \in G | \mathbf{Z}(t), \mathbf{Z}(t-1), \ldots) \qquad 3.$$

or

$$\mathrm{P}(\mathbf{Z}(t+1) \in G | \mathrm{H}_t),$$

where H_t is simply an abbreviation for the history $\mathbf{Z}(t), \mathbf{Z}(t-1), \ldots$.

As an example we present the following simple stochastic predator-prey model, which is called Model I for later reference. Model I is based on within-individual variation alone but this fact is irrelevant to our present discussion.

Take $Z_1(t) = X(t)$ = prey population size at time t, and $Z_2(t) = Y(t)$ = predator population size at time t. Given the history, H_t, the number of prey dying in

the interval $(t, t+1]$ is a binomial random variable with parameters $\{X(t), 1-\exp[-dY(t)]\}$; that is, the probability that x prey die is

$$\binom{X(t)}{x}\{1-\exp[-dY(t)]\}^{x}\{\exp[-dY(t)]\}^{X(t)-x} \qquad 4.$$

The number of prey born is Poisson with mean $bX(t)$. Predators die according to a binominal distribution with parameters $(Y(t), 1-e^{-\delta})$ and are born according to a Poisson distribution with mean $\beta X(t)Y(t)$. For the present we shall assume that, given $\mathbf{H}_t$, prey birth, prey death, predator birth, and predator death are independent. It is very important to note that we are conditioning on (are given) $\mathbf{H}_t$, for these processes could hardly be independent otherwise. Assuming population size changes occur only through birth and death we have enough information to calculate the probability

$$\mathrm{P}(\mathbf{Z}(t) \in G|\mathbf{H}_t) \qquad 3.$$

for any set G. For instance if G is the set $\{X(t+1)=0\}$, then expression 3 represents the conditional probability that the prey are extinct at time $t+1$ given the history up to time t. According to our model this probability equals

$$(1-\exp\{-dY(t)\})^{X(t)}e^{-bX(t)}.$$

Model I is a Markov process since the conditional probabilities, expression 3, are functions of the state at time t alone (9, 70).

A deterministic model is said to be the analog of a stochastic model if the deterministic model agrees with the conditional mean of the stochastic model (9). For example, the expression

$$\mathrm{E}[\mathbf{Z}(t+1)|\mathbf{H}_t] \qquad 5.$$

represents the conditional mean of $\mathbf{Z}(t+1)$ given the history, $\mathbf{H}_t$, and in Model I

$$\begin{aligned} \mathrm{E}[X(t+1)|\mathbf{H}_t] &= X(t)(b+e^{-dY(t)}) \\ \mathrm{E}[Y(t+1)|\mathbf{H}_t] &= Y(t)(e^{-\delta}+\beta X(t)) \end{aligned} \qquad 6.$$

and the analogous deterministic model is

$$\begin{aligned} X(t+1) &= X(t)(b+e^{-dY(t)}) \\ Y(t+1) &= Y(t)(e^{-\delta}+\beta X(t)). \end{aligned} \qquad 7.$$

The conditional mean is quite different from the mean. The conditional mean expresses future mean trends as a function of past history. Past history is stochastic, i.e. it has random variability. The mean averages over this random variability in the past history. Equations 7 are typical of a predator-prey model in that they are nonlinear; it is a direct consequence of this nonlinearity that the means fail to satisfy equations 7. By direct calculation

$$\begin{aligned} \mathrm{E}X(t+1) &= \mathrm{E}X(t)(b+e^{-d\mathrm{E}Y(t)}) + \mathrm{E}(e^{-dY(t)}-e^{-d\mathrm{E}Y(t)})\mathrm{E}X(t) + \mathrm{C}(e^{-dY(t)}, X(t)) \\ \mathrm{E}Y(t+1) &= \mathrm{E}Y(t)(e^{-\delta}+\beta\mathrm{E}X(t)) + \beta\mathrm{C}(X(t), Y(t)), \end{aligned} \qquad 8.$$

where $C(U, V)$ is the covariance between the random variables U and V and EU is the mean of U. Because $X(t)$ and $Y(t)$ are random variables, $E\exp\{-dY(t)\}$ and $\exp\{-dEY(t)\}$ will differ; in general the covariances will be nonzero, so that the result of the deterministic model will differ from the mean of the stochastic model.

This illustrates a major difference between deterministic and stochastic models. If the analogous deterministic model is nonlinear, the mean of the stochastic model will almost always differ from the result of the deterministic model (12, 29). The magnitude of the difference between the mean of the stochastic model and the analogous deterministic model depends on a number of factors but the main one is the degree of nonlinearity of the conditional mean equation over the likely range of values for the past history of the process. For example, consider the Markov case where

$$E[\mathbf{Z}(t+1)|\mathbf{H}_t] = \mathbf{g}(\mathbf{Z}(t)).$$

If the probability distribution of $\mathbf{Z}(t)$ is concentrated on a set G, on which $\mathbf{g}$ is *almost linear,* then $E\mathbf{g}(\mathbf{Z}(t))$ will be approximately the same as $\mathbf{g}(E\mathbf{Z}(t))$ and the deterministic model will agree approximately with the mean of the stochastic model. Since all differentiable functions are approximately linear in a small neighborhood of a point, it can be expected that the mean of the stochastic model will agree approximately with the deterministic model when the variance of $\mathbf{Z}(t)$ is small. This is not a general theorem, however, because in extreme circumstances the behavior of $\mathbf{g}$ on a set with very small probability can be overriding.

A stochastic model contains much more information about the state variables $\mathbf{Z}(t)$ besides the mean $E\mathbf{Z}(t)$. The mean is simply one measure of location of a random variable; by looking beyond the mean, interesting phenomena can often be discovered. For example, O'Waugh (66) considers a stochastic exponential growth model for a single-species population that incorporates only within-individual variation. The analogous deterministic model is the standard exponential growth model. This is a linear model, and so the deterministic model agrees exactly with the mean of the stochastic model. However, the actual realized population sizes in the stochastic model may show an Allee effect—i.e. apparent depressed population growth at low population numbers. Neither the mean of the process nor the deterministic model (the two are the same) indicate this possibility.

Sometimes the mean can actually be quite misleading. This is so in models of exponential population growth in a random environment (52). In these models the actual realized population sizes may approach zero while the mean approaches infinity. This happens because the probability distributions for population size are highly skewed and become more so as time progresses.

The mean is useful and can be a good indicator of actual population density in the following situation (which I discuss at length in the section below on Within-Patch Variability). Suppose that $\mathbf{Z}_i(t)$, which equals $(X_i(t), Y_i(t))$, is the vector giving predator and prey population sizes on a patch. Average population density for k patches is

$$k^{-1}\sum_{i=1}^{k}\mathbf{Z}_i(t).$$

Under conditions to be discussed below, this average population density is near the theoretical mean value, $\mathbf{E}\mathbf{Z}_i(t)$, for any one patch, provided k is large.

A final general feature of stochastic models is their variety. For example, in a deterministic model it generally does not matter how the rate of increase of a population is divided into births and deaths, but in a stochastic model this is not so. In a broad class of stochastic population models (7, 47, 48) the variance of the process depends directly on the separate mean magnitudes of the births and deaths making up the overall mean rate of increase. The same mean rate of increase is compatible with a range of values for the probability of extinction, for example, and also with varying degrees of disagreement between the mean of the stochastic model and its analogous deterministic model. Thus the deterministic model hides the variety of different possibilities for actual population behavior compatible with the same mean rate of increase.

WITHIN-INDIVIDUAL VARIATION

The earliest stochastic models to be applied in the predator-prey context considered only within-individual variation (7, 8, 47, 48). Stochastic predator-prey models based on within-individual variation are generally formulated as continuous-time Markov processes. Because they are generalizations of the so-called birth-death processes (44), population sizes are whole numbers, although some models (47, 48, 67) use discrete-time continuous random-variable approximations. Demographic stochasticity is the name given to the random element of population size fluctuation in models based on within-individual variation.

Consider the prey death process in Model I. This process can be derived by assuming that each prey individual has probability $1-\exp\{-dY(t)\}$ of dying in the time interval $(t, t+1]$ and that the deaths of different prey are (conditionally) independent. No consideration is taken of characteristics of an individual that affect its chance of dying; death is treated as an intrinsically within-individual phenomenon. The other processes in the model have similar within-individual variation bases.

Although the within-individual variation basis of a continuous-time Markov process is rarely pointed out, it is readily apparent when the process is expressed as a limit of discrete-time processes such as Model I. Kurtz's (45) stochastic Lotka-Volterra model is exactly a limit of Model I, while Bartlett's model (7) is a minor modification of this limit. To obtain the limit, modify Model I so that it applies to changes over time intervals of length h rather than of length 1, and put $b = b^*h$, $d = d^*h$, $\beta = \beta^*h$, $\delta = \delta^*h$. Now letting $h \to 0$ a continuous-time process is obtained. In this limiting process

$$\begin{aligned} \mathbf{E}[X(t+h)-X(t)|\mathbf{H}_t] &\approx hX(t)(b^*-d^*Y(t)) \\ \mathbf{E}[Y(t+h)-Y(t)|\mathbf{H}_t] &\approx hY(t)(-\delta^*+\beta^*X(t)) \end{aligned} \qquad 9.$$

when h is small. Thus it is a stochastic version of the Lotka-Volterra model (7, 8, 31, 45). As one would expect, Model I also satisfies expressions 9 for small h.

In Model I the probability of death, for example, of an individual prey during $(t, t+1]$ is simply a function of the predator population size, $Y(t)$. This is equivalent to assuming that every predator in the population has the same chance of causing the death of the prey individual. It does not take account of local population densities and how they change through time. An alternative assumption could be that local density is always proportional to some fixed distribution of density—i.e. that changes in density at different localities are always proportional. Neither assumption seems very realistic but the first may be approximately true for small populations on a small area. We shall call the assumption that conditional probabilities for birth and death depend simply on total population sizes or densities, as opposed to local population sizes or densities, the *homogeneous interaction assumption.* All existing stochastic predator-prey models based on within-individual variation make the homogeneous interaction assumption. Later we shall see the important implications of this assumption.

Stochastic predator-prey models have generally been developed as analogs to already existing deterministic models. While this is understandable, it has led to somewhat arbitrary addition of stochastic features without much attention to the particular kinds of stochasticity relevant in the predator-prey context. One omission can be illustrated by a defect in Model I.

In a predator-prey system, death of prey contributes directly to the well-being of the predator population and presumably to its growth; but in Model I, growth of predators and death of prey in the interval $(t,t+1]$ are independent, given $\mathbf{H}_t$. In this situation growth of predators can only depend on the expectation of what they might get to eat on the basis of prey abundance at time t. To overcome this objection assume that, given x prey die in $(t,t+1]$, predators are born in this same time interval according to a Poisson process with mean $\gamma x = x\beta/d$. This is equivalent to assuming that predators give birth to offspring independently according to Poisson distributions with means proportional to the individual predator's food intake. The actual predator birth process now consists of a Poisson process generalized by a binomial distribution. Although the new mean of the predator birth process is different from its previous value, the more significant change is that prey death and predator birth in $(t,t+1]$ have correlation

$$\{\gamma^{-1}\exp[dY(t)]+1\}^{-1/2} \qquad 10.$$

as opposed to a previous correlation of zero.

Clearly this model is only appropriate if the time unit in this discrete-time process is long enough for conversion of prey to predator to occur. When the time scale is altered to yield a continuous-time process, the correlation (expression 10) becomes $(\gamma^{-1}+1)^{-1/2}$, and the unrealistic situation arises in which the death of a single prey instantaneously gives rise to a Poisson distribution of predator offspring. This is a general feature of continuous-time Markov models of the predator-prey situation—if predator birth is at all correlated with prey death, then instantaneous conversion of whole prey to whole predators occurs. In most formulations only a single individual dies or is born at a time and so a single prey is converted into a single predator!

This is a feature of Bartlett's model (7); in every other model considered in this review, prey death and predator birth are independent.

This is not intended as a major criticism of work in the field; after all, a model cannot include every feature of reality. It simply indicates that modelling of within-individual variation has not attempted to include the kind of stochasticity inherent in predator-prey systems. When stochastic models are considered more for their own sake, rather than as analogs of deterministic models, more pertinent features of stochasticity can be expected to be included.

Large Populations and the Homogeneous Interaction Assumption

Stochastic models based on within-individual variation do give different results from their deterministic analogs. These differences are most striking when populations are small (see next subsection) or do not interact homogeneously (see the section below on Within-Patch Variability). Where populations are both large and *homogeneously interacting* the differences between the results of the stochastic and deterministic models can be very small. To see how this occurs we need a meaningful procedure for scaling the parameters in a model so that the population can become large. Kurtz (45) has given one such method, which can be explained with reference to Model I.

Let $X(t)$ and $Y(t)$ be prey and predator population sizes on an area of size A (the "size of the environment"). Replacing d by d/A and β by β/A, in Model I, the chances of birth and death for any individual become dependent on average population density over the area rather than absolute population size. Increasing the area allows the absolute population size to increase.

Define $X_A(t) = X(t)/A$, $Y_A(t) = Y(t)/A$ (these are the average population densities); then

$$\begin{aligned} \mathrm{E}[X_A(t+1)|\mathrm{H}_t] &= X_A(t)(b+\mathrm{e}^{-dY_A(t)}) \\ \mathrm{E}[Y_A(t+1)|\mathrm{H}_t] &= Y_A(t)(\mathrm{e}^{-\delta}+\beta X_A(t)), \end{aligned} \qquad 11.$$

and the conditional variances are

$$\begin{aligned} \mathrm{V}(X_A(t+1)|\mathrm{H}_t) &= A^{-1}X_A(t)\ \{b+\exp[-dY_A(t)](1-\exp[-dY_A(t)])\} \\ \mathrm{V}(Y_A(t+1)|\mathrm{H}_t) &= A^{-1}Y_A(t)\{\mathrm{e}^{-\delta}(\mathrm{e}^{-\delta}-1)+\beta X_A(t)\}. \end{aligned} \qquad 12.$$

The conditional variances approach 0 as A gets large, provided $X_A(t)$ and $Y_A(t)$ do not approach ∞, which means that $(X_A(t), Y_A(t))$ very nearly satisfies the deterministic difference equation 7, for large A. This statement can be made precise using results in [(14), section 3] and making use of the fact that the conditional means and variances are continuous functions of $X_A(t)$ and $Y_A(t)$. It can be shown that if the initial densities behave nicely, that is $(X_A(0), Y_A(0))\to(X_\infty(0), Y_\infty(0))$, then the whole process converges as A gets large, $(X_A(t), Y_A(t))\to(X_\infty(t), Y_\infty(t))$, and the limiting process, $(X_\infty(t), Y_\infty(t))$, satisfies the deterministic difference equation exactly (here "$\to$" means "converges in distribution to"). All of this means that as A gets large the stochastic model behaves very much like its deterministic analog.

This sort of result will be true for a very wide class of models in which the stochastic element is entirely within-individual variation. Since population size at time $t+1$ is a sum of independent and identically distributed processes of birth and death for individuals alive at t, the law of large numbers is applicable. Continuous-time processes have this structure in an infinitesimal sense and corresponding results hold which show that the analogous deterministic model (a differential equation model) gives a good description of population dynamics in large populations (45).

In the continuous-time case there is also a central limit theorem (46), which says that the difference $(\mathbf{Z}_A(t)-\mathbf{Z}_\infty(t))$ is of the order $A^{-1/2}$; specifically, $A^{1/2}(\mathbf{Z}_A(t)-\mathbf{Z}_\infty(t))$ converges to a diffusion process, as $A \to \infty$, with probability distribution known in terms of a differential equation for the characteristic function. As well as giving the rate of approach of the stochastic process to the deterministic solution, this result gives an asymptotic method of studying the behavior of the stochastic deviations from the deterministic path when A is large.

The diffusion approximation has been applied to the situation in which $\mathbf{Z}_A(0)$ is close to an equilibrium point $\mathbf{Z}_o$, of the deterministic equations (6, 60). It is found that the approximating diffusion process is of the Ornstein-Uhlenbeck kind, with mean $\exp\{\mathbf{M}t\}\,(\mathbf{Z}_A(0)-\mathbf{Z}_o)A^{1/2}$, where $\mathbf{M}$ is the community matrix of deterministic modelling. Then, if the deterministic equilibrium is locally stable the stochastic process can be expected to fluctuate about this deterministic equilibrium. Eventually the process will leave any neighborhood of the equilibrium, but in the deterministic stable case the waiting time can be large if A is large (6).

As with most asymptotic results there are no clear formulae for how large A must be for these diffusion approximations to be applicable. Some information is available from simulation studies but considerable caution is necessary when attempting to extrapolate to other models or even parameter values. Even more caution is necessary when relating these results to nature, for crucial assumptions, such as homogeneous interaction, may be violated. Leslie & Gower (48) and Poole (67) simulated predator-prey models approximating those for which the asymptotic diffusion process has been demonstrated. Leslie & Gower found good agreement with the asymptotic predictions in a situation where the deterministic equilibria were of the order of 100. However Poole, using a two-prey-species/one-predator-species model, found one prey-species population behaving in a nonstationary manner at variance with asymptotic predictions. The deterministic equilibrium of this prey species was near 40 while the other species had equilibrial populations of more than 100.

The diffusion approximation does not seem to have been applied to predator-prey systems during transient phases of population development—i.e. for populations away from equilibrium. However Becker (12) has exact results applicable to transient phases of population development in a number of predator-prey population models. These results emphasize that as t increases, the deterministic model may provide progressively poorer descriptions of population dynamics. Becker's first model always allows arbitrarily large—even infinite—deviation between the mean of the stochastic model and the deterministic analog. This happens for large but finite values of t when variability is dominant, and neither the mean nor the deterministic model provides a very useful description of population dynamics. The

simplicity of Becker's models, which enables their exact solution, restricts their application mainly to transient phases of population development.

It cannot be emphasized too strongly that the conclusions of this subsection concerning the behavior of stochastic models when populations are large depend crucially on the homogeneous interaction assumption. Therefore their relevance to real systems is in doubt.

Extinction and Small Populations

The phenomenon of extinction reveals striking differences between deterministic models and stochastic models based on within-individual variation. First of all, the stochastic model may predict extinction of a population over a short time span, while the analogous deterministic model predicts stability (48, 67). This is most likely when the deterministic equilibrium values are small. In some cases the stochastic model may leave to chance just which of several populations will become extinct in the short term. There is no way the analogous deterministic model can do this.

A deterministic model may correctly predict that a species goes extinct but it never handles the extinction process very well. This is partly because waiting times for extinction can be extremely variable, thus necessitating stochastic description (5, 6, 48, 67). Additionally, extinction never really occurs in most deterministic models; it is only achieved in the limit as $t \to \infty$. To see this, consider the usual formulation (56) of difference equations for predator-prey models:

$$\begin{aligned} X(t+1) &= X(t)f(X(t),\ Y(t)) \\ Y(t+1) &= Y(t)g(X(t), Y(t)), \end{aligned} \qquad 13.$$

where f and g are strictly positive functions. Clearly, extinction in finite time is impossible. When one redefines extinction as some small positive population level, as Gilpin does (30), then extinction in finite time does occur; but this modification is inconvenient in analytical models embedded in more complex models; it destroys the analytical structure, especially if differential equations are used. Moreover, we are still left with the question of whether extinction at some fixed nonrandom time makes much sense. For example, as May [(57), p. 94] points out, a limit cycle that comes near an axis should be regarded as unstable because in a real population chance processes will cause extinction eventually. However, on many approaches to 0 the population may increase and escape extinction just by chance. It follows that the waiting time for extinction is essentially stochastic. Models that rely in any way on the timing of extinction events will be in error if they are not stochastic.

The majority of Markov process population models based on within-individual variation predict certain eventual extinction of at least one population. Some authors have found this disturbing (15). It has led others [as Reddingius observed (70)] to believe that Markov processes are inappropriate for modelling population dynamics. However, the prediction that extinction must eventually occur does not contradict what we see in the real world; the waiting time for extinction is often so long that we may never expect to observe it (6); in effect, extinction within any observable time

span may not occur. A similar situation is accepted in statistical mechanics, where systems apparently moving irreversibly to equilibrium actually visit nonequilibrium states infinitely often [(16), page 149]. Moreover, this property of certain eventual extinction is a feature of a broad class of models where population sizes are whole numbers. Since if the probability of eventual extinction of the prey (predator) given H_t is greater than some strictly positive function $f(\mathbf{Z}(t))$ of $\mathbf{Z}(t)$ alone, then either the prey (predator) becomes extinct or the sum of prey and predator population sizes approaches ∞ as $t\to\infty$, with probability 1. This result is a trivial generalization of results given by Breiman [(16), Chapter 5, problems 9 and 10]. If population sizes are not allowed to become arbitrarily large it seems that, in the absence of evolution, eventual extinction is a fact of life. It is certainly not a reason for rejecting Markov process models.

When populations are small, as they often are locally, extinction in the short term is predicted by stochastic models. This is not unreasonable, since local extinction commonly occurs in nature (2). Leslie & Gower (48) and Poole (67) present detailed analyses of factors affecting extinction in small populations. As indicated above, stochastic models are essential for this kind of study.

For small local populations the assumption of homogeneous interaction may not be unreasonable; the Markov process models used to model within-individual variation may be appropriate to this case. For small populations these models demonstrate that variability is dominant and neither the mean nor the deterministic model give very useful descriptions of population dynamics (12). However, the mean can be a useful description of average population density for a large number of local populations. Thus it is important to recognize that a small population for at least one species, some of the time, can lead to appreciable differences between the mean of a stochastic model and its deterministic analog. These differences can be extreme for transient phases of population development (11, 12). This suggests that in situations where local populations are dominated by such phases—e.g. are increasing or going extinct most of the time—large differences will appear between deterministic and stochastic models for collections of local populations (see the section below on Within-Patch Variability).

VARIATION BETWEEN INDIVIDUALS

Model I is derived by assuming, for example, that each prey individual has the probability $1-\exp\{-dY(t)\}$ of dying by predation in the interval $(t,t+1]$. If individuals vary in their susceptibility to predation, then an appropriate assumption is that d varies from individual to individual according to some probability distribution. In this situation the probability that a randomly chosen prey dies in $(t,t+1]$ is $1-\phi(Y(t))$ where ϕ is the Laplace transform of the random variable d. For example when d is a gamma random variable the probability of death takes the form $1-[1+\alpha Y(t)]^{-\lambda}$ where λ and α are parameters of the gamma distribution. The difference between this functional form and the previous form depends on the variance, $\alpha^2\lambda$, of d.

Bailey, Nicholson & Williams (4) modified the Nicholson-Bailey host-parasite model in this way. Their model is basically deterministic, but their host death rate agrees with the prey death rate of Model I. The functional form $1-\exp\{-dY(t)\}$ is derived on behavioral postulates (63), but the equality of the ds for each host is a separate postulate that is relaxed in the later treatment. With $\alpha=1$ and $\lambda<1$, d has a J-shaped distribution, and the new model is stable, whereas the original model is unstable.

So far in our discussion we have not taken account of the fact that an individual's survival of one period alters its chance of surviving the next period, since the d's vary from individual to individual. If f is the probability density of d for an animal alive at time t, then the appropriate density, if it survives to $t+1$, is

$$f(d)e^{-dY(t)}/\phi(Y(t)). \qquad 14.$$

For an animal born at a time s and still alive at time t the density for d that applies for the period $(t,t+1]$ is

$$f(d)\exp\{-d\sum_{u=s}^{t-1}Y(u)\}/\phi(\sum_{u=s}^{t-1}Y(u)). \qquad 15.$$

Considerable complications have appeared, for this probability density depends on both the age of the individual and past predator population sizes. In principle this model can be handled by replacing $X(t)$ with the vector $\mathbf{X}(t)=(X_0(t), X_1(t), \ldots)$ where $X_i(t)$ is the number of prey of age i. The model is no longer a Markov process because

$$\mathbf{P}([\mathbf{X}(t),\ Y(t)]\in G|\mathbf{H}_t) \qquad 16.$$

depends on the history of the predator population back to the birth of the oldest prey. Thus variation between individuals can involve age structure in population dynamics and it can also introduce non-Markovian properties. Additionally, between-individual variation will have a genetic component that may lead to changes in the function f from generation to generation as a result of selection.

Such complications can be avoided in the short term (e.g. before the next prey reproductive period) and so the short-term effects of between-individual variation are not difficult to study. Also, variation between predators (e.g. in their voracity) need not introduce non-Markovian phenomena or involve genetics, yet it can still lead to population dynamics different from those predicted by models that ignore between-individual variation (12).

Another possibility we have not considered is that individual animals change through time. The value of d, for instance, is a function of an individual's age. This leads to a direct involvement of age structure. For a deterministic discussion see (3), where further references can be found.

At the behavior level it is very easy to demonstrate significant effects of between-individual variation. We give an example from selective predation theory. Manly et al (55) proposed the following formula for the probability that a given predator captures an animal of species 1 as its next prey: $\lambda X_1(\lambda X_1+X_2)^{-1}$. X_1 and X_2 are the

numbers of prey species of types 1 and 2, and λ is the preference for type 1, which is assumed constant for a given predator. This formula can be derived from a simple stochastic model (18). However, preference can vary between predators to a considerable extent (61); it is best to think of λ as a random variable. When there are many predators present the ratio of type 1 to type 2 consumed after each predator has fed is close to

$$(X_1/X_2)\ [\mathrm{E}\lambda(\lambda X_1+X_2)^{-1}][\mathrm{E}(\lambda X_1+X_2)^{-1}]^{-1} \qquad 17.$$

In this situation Murdoch and Oaten's C (61) is given by

$$C(X_1/X_2) = [\mathrm{E}\lambda(\lambda X_1+X_2)^{-1}][\mathrm{E}(\lambda X_1+X_2)^{-1}]^{-1}. \qquad 18.$$

Here the means (or expectations) are taken with respect to the probability distribution of λ. When C is an increasing function, switching occurs (61). On the other hand, if C is decreasing, the representation of a given prey species in the diet, compared with its relative abundance, increases as it becomes rarer. This may be called "anti-switching." Evaluating C we find $C(0)=\mathrm{E}\lambda, C(\infty) = (\mathrm{E}\lambda^{-1})^{-1}$ If there is any between-individual variation at all, $\mathrm{E}\lambda > (\mathrm{E}\lambda^{-1})^{-1}$ (the arithmetic mean is greater than the harmonic mean). So the overall population appears to be anti-switching even though individuals do not change their preference. This result is capable of substantial generalization. In particular, it can be shown (20) that anti-switching is uniform over the entire range of values of X_1/X_2.

RANDOM ENVIRONMENT

Suppose, for example, that the birth rate of the prey increases when more favorable weather results in an increased abundance of food. Model I should then be modified so that the parameter b varies from year to year. The simplest situation is when b_t, the value of b for year t, is a random variable independent of previous years. In this case $\mathbf{Z}(t) = (X(t), Y(t))$ remains a Markov process, but instead of the distribution of prey births being Poisson it is a mixture of Poisson distributions with

$$\mathrm{P}(x \text{ prey births for year } t|\mathrm{H}_t) = \int_0^\infty \frac{e^{-bX(t)}(bX(t))^x}{x!} f(b)db \qquad 19.$$

where f is the density function for b. The most noticeable effect is that the variance is now greater than the mean rather than equal to the mean as in the Poisson process, but this variance also behaves rather differently, as we shall see.

Applying Kurtz's space scaling (as in the section above on Within-Individual Variation) the average density process $\mathbf{Z}_A(t)$ converges to a process $\mathbf{Z}_\infty(t)$, but this limiting process does not satisfy the deterministic equation 7. Instead we have

$$\begin{aligned} X_\infty(t+1) &= X_\infty(t)(b_t+e^{dY_\infty(t)}) \\ Y_\infty(t+1) &= Y_\infty(t)(e^{-\delta}+\beta X_\infty(t)), \end{aligned} \qquad 20.$$

which is a random difference equation. The randomness due to within-individual variation has vanished, but the random environment part, b_t, persists undiminished.

Within-individual variation is modelled as conditionally independent and so it tends to cancel out when averaged over a large number of individuals. On the other hand, random environment is modelled totally dependently because all individuals in the population, living in a common environment, are subject to the same environmental fluctuations. This results in the variance of the number of births per unit area converging to $(X_\infty(t))^2 V(b_t)$, compared with 0 before. Thus a random environment can lead to random fluctuation in average population density over a large area, whereas within-individual variation may not.

The above analysis essentially assumes that environmental fluctuations in different parts of the range of the populations are perfectly correlated. The conclusions remain true if this correlation does not decrease too rapidly to 0 with distance. Crowley (24) points out that some effects of the environment may not remain correlated over large distances. However, this seems unlikely to be the case for weather when considered over a reasonable time span. Even so, some random-environment models assume independence between different places (71–73).

These results do not indicate that within-individual variation is far less important than environmental variation. They simply indicate that within-individual variation is much less likely to produce random fluctuation in average density over a large area. The mechanistic role of within-individual variation persists, as we shall see, when we consider the more realistic model of spatial interaction in the next section. In geometrically growing populations (41) the effect of environmental fluctuations becomes extremely severe when the population gets large. Fluctuations due to within-individual variation are effectively swamped if the population can continue increasing forever. However, within-individual variation may cause early extinction of the population, and, without it, extinction in finite time is impossible. Furthermore, when populations are not allowed to grow indefinitely, the effect of within-individual variation can still be felt (54).

From the point of view of a deterministic modeller, random environment is a destabilizing influence (57), but it can also have mechanistic effects—e.g. it can produce quasi-cyclic population oscillations (17, 35, 64).

The usual approach to modelling a random environment is to assume that the environment is independent from one time interval to the next, as we have done. For continuous-time processes the environment is independent at every instant; in the usual case where the probability distribution does not depend on time (a stationary process), the environment process is called white noise (33). In a study of stochastic logistic models Roughgarden (75) found a greater variety of possible behavior when the environment process is correlated. Therefore alternative approaches are needed.

The analytic advantage of using white noise is that the resulting population process is a Markov process. However, it is possible to remain within the Markov family of models if the state variables $\mathbf{Z}(t)$ include the environment. For example, if $\mathbf{Z}(t) = (X(t), Y(t), b_t)$ in Model I, and b_t is a Markov process, then it follows that $\mathbf{Z}(t)$ is a Markov process. The environment can be arbitrarily highly correlated from one time to the next and the theory of Markov processes can still be used in the

analysis. This idea is applicable quite generally but has not been exploited in predator-prey models. The major disadvantage is the increase in dimension of the process. Other approaches have been suggested by Goh (32) and Cumberland & Rhodes (25).

Generally random environment models have required linearization to make them tractable. Nisbet et al (64) have examined, for a few specific examples, the error involved in linearization. Ludwig (54) has demonstrated an asymptotic method that obviates linearization.

WITHIN-PATCH VARIABILITY

Within-individual variation, between-individual variation, that part of temporal environmental variability which is specific to a patch, and movements into and from a patch, combine to form within-patch variability.

We shall discuss a simple model (Model II) showing the effect of within-patch variability; it is, however, capable of substantial generalization. The major feature of this model is that the individuals in the populations do not interact homogeneously; local randomness thus has a direct effect on population dynamics. In particular the model demonstrates that the effect of within-individual variation does not disappear as the size of the environment increases.

The total range of the populations is made up of k patches. The population totals $(X(t), Y(t))$ are divided up into subpopulations $(X_1(t), Y_1(t)), \ldots, (X_k(t), Y_k(t))$ on each of the k patches. At the beginning of each time interval migration occurs. At other times growth occurs, and during this time the populations are effectively isolated so that the chances of survival and reproduction for any individual are only affected by other individuals living in the same patch.

In Model II, $k^{-1}\mathbf{Z}(t)$, which equals $(k^{-1}X(t), k^{-1}Y(t))$, gives the average population densities. The size of the environment is increased by simply increasing k; the nature of the patches remains unchanged. As $k \to \infty, k^{-1}\mathbf{Z}(t)$ converges to a deterministic process $\mathbf{Z}^*(t) = (X^*(t), Y^*(t))$ in the same way that $\mathbf{Z}_A(t)$ of Model I converges to the deterministic process $\mathbf{Z}_\infty(t)$. So with this new model the average density process also becomes very close to a deterministic process as the size of the environment in which the populations live is increased. However, there is a very important difference between the limiting average density process $\mathbf{Z}^*(t)$ of Model II, and the corresponding process, $\mathbf{Z}_\infty(t)$, from Model I. Whereas $\mathbf{Z}_\infty(t)$ is the deterministic analog of the stochastic model, $\mathbf{Z}^*(t)$ is not. Replacement of any stochastic submodel of Model II by the deterministic analog leads to an average density process different from $\mathbf{Z}^*(t)$.

There are two basic reasons for these differences. First of all, in predator-prey systems the dynamics are nonlinear. Second, the probability that a particular individual dies in the time interval $(t, t+1]$ is a function of $\mathbf{Z}_j(t)$ alone, when the animal is in patch j for this time interval. So we can write this probability as $p(\mathbf{Z}_j(t))$. Increasing the number of patches does not change this, and the variance of $\mathbf{Z}_j(t)$ does not approach 0. On the other hand, the corresponding probability in Model I is a function of the average population density over the whole area. In both models

the average population density becomes deterministic as the area increases. In Model I this probability is dependent on an approximately deterministic process for large A. In Model II this probability is dependent on a stochastic process that does not approach a deterministic process as $k\to\infty$. The same kind of statement also holds for birth probabilities. For Model I conditional means become nonrandom as $A\to\infty$, but for Model II conditional means remain random variables as $k\to\infty$.

The magnitude of these differences depends on the degree of nonlinearity of the conditional means and on the magnitude of the variance of the $\mathbf{Z}_j(t)$. Factors contributing to this variance do not change greatly as $k\to\infty$ because they are generated mostly within a patch that does not change as $k\to\infty$. A greater effect will occur with the migration process because it is the only process directly dependent on the number of patches. As $k\to\infty$, no large changes occur in the relative importance of the four recognizable components of within-patch variability mentioned at the beginning. As anticipated, the contribution of within-individual variation persists as $k\to\infty$. For a proper understanding of what happens we must examine the mathematics. The nature of this article precludes giving full details, but these will appear elsewhere (21).

In Model II, $\mathbf{H}_t$ denotes the history of the populations on each of the k patches. For every interval $(t,t+1]$, $t+$ denotes the time before which migration occurs and after which growth occurs. Migration is of the simplest possible kind: Different animals migrate independently. An individual prey (predator) occupying patch i has probability $m/(k-1)$ ($\mu/(k-1)$) of migrating to patch $j\neq i$. Given $\mathbf{H}_{t+}$, the $\mathbf{Z}_j(t+1)$ are independent and the conditional distribution of $\mathbf{Z}_j(t+1)$, given $\mathbf{H}_{t+}$, is simply a weakly continuous function of $\mathbf{Z}_j(t+)$. We use the notation

$$\mathrm{P}(\mathbf{Z}_j(t+1)\in G|\mathbf{H}_{t+}) = \gamma(\mathbf{Z}_j(t+),G). \qquad 21.$$

γ may, for example, simply specify the same process of birth and death that occurs for the whole area in Model I, scaled down to patch size, of course. We have specified the dynamics as a Markov process but this is not essential.

It is assumed that $\mathbf{Z}_1(0),\ldots,\mathbf{Z}_k(0)$ are symmetrically distributed, as can be arranged by random labelling of the patches, and that the variance of the $\mathbf{Z}_j(t)$ remains bounded as $k\to\infty$, as it will be in most cases. If the observed or empirical distribution of the $\mathbf{Z}_j(0)$ approaches a theoretical probability distribution, in the sense of weak convergence in distribution, then the methods of P. Chesson [(19), section 3.6] apply, and it can be shown that

1. $\mathbf{Z}_j(t)$ has a limiting distribution π_t independent of j
2. $k^{-1}\mathbf{Z}(t)$ converges in distribution to the process $\mathbf{Z}^*(t) = \mathrm{E}\mathbf{Z}_j(t) = \int \mathbf{z}\, d\pi_t(\mathbf{z})$.

It is important to remember that $\mathbf{Z}_j(t)$ is stochastic in the limit while $\mathbf{Z}^*(t)$ is deterministic.

The limiting distribution of the process $\mathbf{Z}_j(t)$ is easy to describe. Given $\mathbf{H}_t$, emigration of prey is binomial with parameters $(X_j(t),m)$ and immigration is Poisson with mean $mX^*(t)$. For predators, μ replaces m etc. We shall use the notation $\chi(\mathbf{Z}^*(t),\mathbf{Z}_j(t), G)$ to denote $\mathrm{P}(\mathbf{Z}_j(t+\ )\in G|\mathbf{H}_t)$ which is specified by these migration

processes. The internal patch dynamics are the same for all k and therefore in the limit we still have $P(\mathbf{Z}_j(t+1)\in G|\mathbf{H}_{t+})=\gamma(\mathbf{Z}_j(t+),G)$. π_t is now defined by the iterative formulae

$$\begin{aligned}\pi_{t+1}(G) &= \int\gamma(\mathbf{z}_1,G)\,\chi(\mathbf{Z}^*(t),\mathbf{z}_0,d\mathbf{z}_1)d\pi_t(\mathbf{z}_0),\\ \mathbf{Z}^*(t+1) &= \int\mathbf{z}d\pi_{t+1}(\mathbf{z}).\end{aligned} \qquad 22.$$

This model has at least three distinct stochastic parts that can be made deterministic, namely the movement processes, the internal patch dynamics, and the variation in the initial value $\mathbf{Z}_j(0)$. Deterministic analogs are found from the appropriate conditional expectations.

To make movement deterministic, treat $\mathbf{Z}_j(t+)$ as though it equals $E[\mathbf{Z}_j(t+)|\mathbf{H}_t]$, namely $\mathbf{Z}_j(t)+\mathbf{M}(\mathbf{Z}^*(t)-\mathbf{Z}_j(t))$ where $\mathbf{M}$ is the diagonal matrix with diagonal (m,μ). Internal dynamics become deterministic by equating $\mathbf{Z}_j(t+1)$ with $E[\mathbf{Z}_j(t+1)|\mathbf{H}_{t+}]=\mathbf{h}_0(\mathbf{Z}_j(t+))$ where $\mathbf{h}_0$ is a function that can be specified in any particular case. Variation in the initial value is eliminated by putting $\mathbf{Z}_j(0)=\mathbf{Z}^*(0)$. Doing all this simultaneously we get the deterministic model (Model III):

$$\mathbf{Z}_j(t+1)=\mathbf{h}_0(\mathbf{Z}_j(t+))\text{ and }\mathbf{Z}_j(t+)=\mathbf{Z}_j(t). \qquad 23.$$

In this model, $\mathbf{Z}_j(t)$ is equivalent to $\mathbf{Z}^*(t)$. If the internal dynamics are described by Model I then this deterministic model agrees exactly with the deterministic limit of Model I. To see the deviation from the limit of Model II note that, in Model II

$$\mathbf{Z}^*(t+1)=E\mathbf{h}_0(\mathbf{Z}_j(t+)) \qquad 24.$$

and

$$\mathbf{Z}^*(t+)=\mathbf{Z}^*(t)=E\mathbf{Z}_j(t+)=E\mathbf{Z}_j(t).$$

Because of nonlinearity, $E\mathbf{h}_0(\mathbf{Z}_j(t+))\neq\mathbf{h}_0(E\mathbf{Z}_j(t+))$, in general, and so Model III and the limit of Model II disagree. It is instructive to examine just what the deviation actually is in the case where internal patch dynamics are described by Model I. Here

$$\mathbf{h}_0(\mathbf{Z})=\begin{bmatrix}X(b+e^{-dY})\\ Y(e^{-\delta}+\beta X)\end{bmatrix} \qquad 25.$$

and the deviation is:

$$E\mathbf{h}_0(\mathbf{Z}_j(t+))-\mathbf{h}_0(\mathbf{Z}^*(t))=\begin{bmatrix}E[e^{-dY_j(t+)}-e^{-dY^*(t)}]X^*(t)+C(e^{-dY_j(t+)},X_j(t+))\\ \beta C(X_j(t+),Y_j(t+))\end{bmatrix}. \qquad 26.$$

This may be compared with equation 8 for Model I, where the deviation between the stochastic mean and the deterministic model is similar. The difference here is that the deviation terms do not vanish as the size of the environment increases.

Although Model II has not yet received detailed analysis, a number of simple predator-prey models have been published (38, 39, 59, 79) that are similar to Model II: They are based on patchy environment and contain at least some within-patch variation and nonlinearity in internal dynamics or migration. These authors chose

unstable deterministic internal dynamic processes, which, because they are deterministic, correspond exactly to Model III. Thus removing all within-patch variation in these models—i.e. taking away randomness in movements and variation in initial values—leads to unstable processes. However, all of these models allow coexistence of predator and prey for wide ranges of parameters, thus demonstrating the essential role of within-patch variation.

Hastings (38) also discusses, but dismisses as unrealistic, a modification of his model in which internal dynamics are stochastic. The latter model is actually more stable than the model with deterministic internal dynamics. The stochasticity in the internal dynamics is introduced by replacing a constant waiting time for extinction by an exponentially distributed one. Since such waiting times can be highly variable and can have skewed distributions, the stochastic alternative need not be unrealistic (48, 67). Moreover, Keilson (42, 43) shows that, in broad situations, Markov processes have asymptotically exponentially distributed exit times. See also Barbour (6). Thus the modification of Hastings' model provides a further demonstration of the effects of within-patch variation.

Another area of deterministic predator-prey modelling concerns production or elimination of spatial or temporal patterns through movements, with growth and interaction dependent on local population densities (1, 22, 27, 36, 49, 50, 62, 74, 76, 77). Although initial population densities may vary, these models are deterministic in both movements and local dynamics. A comparable model can be obtained from Model II by replacing movements and within-patch dynamics by their deterministic analogs but retaining the variation in the initial values. We shall call this Model IV. Our models contain less-realistic movement processes than the models cited above; in the latter, animals migrate to nearby places, while in ours the migrating individuals are scattered at random in space. However, this is not an essential feature of our models and does not affect our conclusions.

Since we are interested in changes in spatial patterns through time, we shall compare the development of $\mathbf{Z}_j(t)$ from its initial value, to the stochastic counterpart $\mathrm{E}[\mathbf{Z}_j(t)|\mathrm{H}_0]$, which represents the conditional mean population sizes—given, or as a function of, the initial conditions. As before, within-patch variation, together with nonlinearities in dynamics, produces differences between the stochastic and deterministic models. For deterministic models to be applicable, within-patch variation must be small. This means that local random environmental variation must be small and that populations must be locally dense and must interact homogeneously.

To show how these differences appear, Model IV is written as

$$\begin{aligned} \mathbf{Z}_j(t+) &= \mathbf{Z}_j(t)+\mathbf{M}(\mathbf{Z}^*(t)-\mathbf{Z}_j(t)) \\ \mathbf{Z}_j(t+1) &= \mathbf{h}_0(\mathbf{Z}_j(t+)). \end{aligned} \qquad 27.$$

$\mathbf{Z}^*(t)$ is still defined by formula 22, with appropriate deterministic substitutions for the dynamics. In Model II,

$$\begin{aligned} \mathrm{E}[\mathbf{Z}_j(t+)|\mathrm{H}_0] &= \mathrm{E}[\mathbf{Z}_j(t)|\mathrm{H}_0]+\mathbf{M}(\mathbf{Z}^*(t)-\mathrm{E}[\mathbf{Z}_j(t)|\mathrm{H}_0]) \\ \mathrm{E}[\mathbf{Z}_i(t+1)|\mathrm{H}_0] &= \mathrm{E}[\mathbf{h}_0(\mathbf{Z}_i(t+))|\mathrm{H}_0]. \end{aligned} \qquad 28.$$

Thus we see that the variation in $\mathbf{Z}_j(t+)$, given H_0, and the nonlinearity in $\mathbf{h}_0$ results in deviation between the stochastic and deterministic models. If movements are nonlinear as in (34), a second source of deviation is produced.

BETWEEN-PATCH VARIABILITY

Not all places make equally good homes for animals. Parameter values for internal dynamic processes and migration may differ from place to place. Looking at this from the point of view of random environment one might say that the probability distribution for the state of the environment is space-dependent. Prey refuges provide an extreme case of this *between-patch variability,* which can have significant ecological effects (61). In addition, Comins & Blatt (23) have demonstrated stabilizing effects of space-dependence of the migration process in a predator-prey model; Gurney & Nisbet (34) show how local exponential growth can be stabilized by between-patch variation and nonlinear migration.

Since between-patch variability is assumed to be fixed for all time, it does not matter greatly whether its origin is regarded as random or deterministic. However, when it is treated as random it is easily incorporated in Model II of the previous section. When this is done, patches vary in type with f_j representing the type of patch j. As $k\to\infty$, $\mathbf{Z}_j(t)$ has a limiting distribution and $k^{-1}\mathbf{Z}(t)$ converges to the deterministic process $\mathbf{Z}^*(t) = \mathbf{E}\mathbf{Z}_j(t)$. The most easily observable difference is that the limiting $\mathbf{Z}_j(t)$ are no longer Markov processes but mixtures of Markov processes. Thus between-patch variability can produce non-Markov behavior. However, the enlarged process $(f_j,\mathbf{Z}_j(t))$ is a Markov process. The determination of general effects of between-patch variability awaits more detailed analysis of this model.

MODELLING AND STABILITY

Structural Models

The classical Lotka-Volterra predator-prey model and Model I are examples of specific models. They are specific because the precise form of the interaction between predator and prey is specified in the model. The only things not specified are a few parameters—e.g. b, d, β, and δ in Model I. Specific models rapidly become difficult to analyze and interpret as more features are included to increase their realism (51). Conclusions based on specific models are specific conclusions—i.e. they are valid for the precise form of predator-prey interaction specified in the model. Such models are incapable of furnishing the material for a general predator-prey theory. It could be argued that concurrence of a large number of specific models can lead to general theory; however it is possible to take a more general approach.

Model II is a general model of the kind we shall call a structural model. In Model II the precise form of the interaction between predator and prey is left unspecified. If patch dynamics are specified to follow Model I then Model II becomes a specific model. However, it is not necessary to make Model II into a specific model before worthwhile conclusions are available. For example, in the section above on Within-

Patch Variability, Model II is used in its general form to draw conclusions about differences between stochastic and deterministic models. Its use there is a means of exploring relationships between models falling within a general framework. But the conclusions apply more generally than just to models, for they indicate that field and laboratory studies should consider variability and should not restrict attention to mean tendencies. Structural models have also been used in this exploratory manner in the study of animal movements (19).

Can a structural model be more than exploratory; can it explain or predict real-world phenomena? Model I does yield predictions when qualitative assumptions are made about the predator-prey relationship. For example, using Model I it is possible to determine the way the correlation between local predator and prey density is affected by such factors as predator dispersal rate and the existence of alternative prey for the predator. When only qualitative assumptions are made, the model retains its structural character and so remains general. Model I needs further study to see what other predictions are available. A worthwhile investigation would be to seek general conditions under which predator and prey densities are stable in the sense to be discussed below ("stochastic boundedness"). To make Model I really useful we must modify it to include (*a*) more general movement processes, and (*b*) dependence between the random environment processes for different patches.

Mathematics can be very general (69); but the application of this general mathematics to the real world is mostly indirect: It is used to study specific models. General mathematical results are in use in population ecology; for example, Kolmogorov's theorem (17, 57) is a general result used in the study of specific models of the predator-prey interaction. In the section on within-individual variation we have seen a general result on the inevitability of population extinction. In both of these examples the list of assumptions is essentially a structural model, i.e. the assumptions are qualitative. Predator-prey theory will benefit from attempts to formulate structural models whose conclusions will apply directly to field situations. Only in this way can field data actually refute not the irrelevant details of specific model but rather the idea being tested. The real ideas being tested are nearly always qualitative and should not be tied to specific models.

What general conclusions do apply to predator-prey systems? Only by seeking to prove or disprove general results can we begin to find answers. Specific models do have a place in this process, as examples, counterexamples, and guides to intuition.

A predator-prey system can be divided into several levels of organization. The most basic level is behavior. Behavior contributes to the birth, death, and movement processes of individual animals. These in turn lead to local dynamics, which then yield the dynamics of average population density. To consider too many levels at once would lead to highly complicated models. A method more likely of success is to make assumptions at one level—e.g. that of local dynamics—and use these to draw conclusions about the next level—in this case the dynamics of average patch density. Field studies and models for lower levels can suggest appropriate assumptions. In this scheme, within- and between-individual variation and local environmental variation can be studied for their effect on local dynamics but need not be

considered explicitly in studies of average population density for many patches. Their effects should be implicit in the assumptions about local dynamics.

Stability Notions For Stochastic Systems

The usual notions of stability for deterministic models are difficult to relate to real-world population phenomena (15, 40). In the real world, stochastic phenomena, especially of the random environment kind, constantly alter the state of the system. These stochastic effects are not external to the system; they are an inseparable part of it.

The stability notions of Wu (78), and Botkin & Sobel's θ-persistence (15), are essentially deterministic concepts and are not suitable for stochastic systems. Recurrence of any particular set of states (15) is an aspect of a system's stability, but nonrecurrence for a particular set would not necessarily lead us to conclude that the system is unstable.

Ludwig (54) has suggested judging the stability of a model by the expected waiting time to extinction. However, this idea is difficult to relate to the field, where such waiting times will only be available for local populations and will in most cases still be very difficult to determine. For average density over a large area the waiting time for extinction may be enormous and, correspondingly, models for limiting average density, such as the limit as $k\to\infty$ of Model II, may not allow extinction over the total area at all.

One might say that a model for limiting average density is stable if there is no possibility that the populations will decline to zero or increase in an unbounded way. This idea can be formulated mathematically so that it applies to one of several populations in a model. This is done below. However, it is not difficult to extend the definition so that it applies simultaneously for several populations. Let $Z(t)$ be the average density of one population in the model. The model is assumed to be of the limiting average density kind, for example the limit of model II as $k\to\infty$. Z will be *stochastically bounded* if for every positive probability ϵ there are numbers $U_\epsilon<\infty$ and $L_\epsilon>0$, such that for any t, $Z(t)$ is greater than L_ϵ with probability at least $1-\epsilon$ and less than U_ϵ with probability at least $1-\epsilon$. Consider as a simple illustration the case where $\log Z(t)$ is normal with mean $\mu(t)$ and variance $\sigma^2(t)$. Then for $\epsilon = .025$ we find

$$\log L_\epsilon = \min_t \mu(t)-1.96\sigma(t), \text{ and}$$

$$\log U_\epsilon = \max_t \mu(t)+1.96\sigma(t).$$

Clearly the maxima or minima could be $\pm\infty$ in which case Z is not stochastically bounded. Notice that for any given time the probability that $Z(t)$ is between L_ϵ and U_ϵ is at least .95 for $\epsilon = .025$.

The bounds L_ϵ and U_ϵ can be estimated from observational data—e.g. in the lognormal case from the estimates of $\mu(t)$ and $\sigma^2(t)$—and so U_ϵ and L_ϵ have genuine meaning for real-world systems. The magnitudes of L_ϵ and U_ϵ are quantita-

tive measures of the amount of fluctuation in a system. In model systems, especially of the structural kind, the aim should not be to find L_ϵ and U_ϵ explicitly; this will often be difficult or impossible. Success is more likely in proving their existence or nonexistence, which is equivalent to proving or disproving the stochastic boundedness of the model population. In models where U_ϵ and L_ϵ exist, factors affecting their size and difference should be important objects of study. Stochastic boundedness corresponds to the concept of tightness from the theory of convergence of probability measures (13, 14); therefore the latter theory is directly applicable to the study of stochastic boundedness.

In Model II there is no common component of random environment for the whole collection of patches. As a result, limiting average density is deterministic; in this case, stochastic boundedness reduces to the deterministic notion that the density is stable if it always remains within certain fixed bounds. Stochastic boundedness applies in a more interesting way when environment is random. Stochastic logistic processes as studied by Feldman & Roughgarden (28) provide an interesting application. For these processes L_ϵ and U_ϵ can be calculated directly from the distribution of $Z(t)$ given by the model.

Stochastic boundedness can also be considered on a local scale. For an individual patch or collection of patches U_ϵ and L_ϵ are defined as before. However, since local extinction is to be expected, it is not useful to insist that $L_\epsilon > 0$ for every $\epsilon > 0$ before a population is regarded as stable. Instead say that a population is stochastically bounded, in the local sense, if $U_\epsilon < \infty$ for all $\epsilon > 0$ and there is an $\epsilon < 1$ such that $L_\epsilon > 0$. For this definition to be sensible some kind of recurrence condition is also required (15). As a simple illustration consider this concept in relation to Hasting's model (38) with average population densities at equilibrium. For a single patch we find $U_\epsilon = 1$ for all ϵ. $L_\epsilon = 0$ for $\epsilon < p$ (equilibrium density), and $L_\epsilon = 1$ for $\epsilon \geqslant p$. In Hasting's system patches are statistically independent, therefore the binomial distribution can be used to calculate L_ϵ and U_ϵ for the average density of a collection of n patches. Alternatively one can use the normal approximation which, with $\epsilon = 0.25$, leads to

$$U_\epsilon \approx p + 1.96n^{-1/2} [p(1-p)]^{1/2}$$

and

$$L_\epsilon \approx p - 1.96n^{-1/2} [p(1-p)]^{1/2}.$$

In cases where there is statistical dependence between patches, U_ϵ and L_ϵ will also depend on the geometry of the collection of patches. This kind of model analysis again allows direct comparison with the real world because L_ϵ and U_ϵ can be estimated from observational data.

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Literature Cited

1. Allen, J. C. 1975. Mathematical models of species interactions in time and space. *Am. Nat.* 109:319–41
2. Andrewartha, H. G., Birch, L. C. 1954. *The Distribution and Abundance of Animals.* Chicago: Univ. Chicago Press, 782 pp.
3. Auslander, D., Oster, G., Huffaker, C. 1974. Dynamics of interacting populations. *J. Franklin Inst.* 297:345–76
4. Bailey, V. A., Nicholson, A. J., Williams, E. J. 1962. Interaction between hosts and parasites when some host individuals are more difficult to find than others. *J. Theor. Biol.* 3:1–18
5. Barbour, A. D. 1975. The duration of a closed stochastic epidemic. *Biometrika* 62:477–82
6. Barbour, A. D. 1976. Quasi-stationary distributions in Markov population processes. *Adv. Appl. Probab.* 8:296–314
7. Bartlett, M. S. 1957. On theoretical models for competitive and predatory biological systems. *Biometrika* 44: 27–42
8. Bartlett, M. S. 1960. *Stochastic Population Models in Ecology and Epidemiology.* London: Methuen. 90 pp.
9. Bartlett, M. S. 1973. Equations and models of population change. In *The Mathematical Theory of the Dynamics of Biological Populations,* ed. M. S. Bartlett, R. W. Hiorns, pp. 5–21. London: Academic. 347 pp.
10. Bartlett, M. S., Gower, J. C., Leslie, P. H. 1960. A comparison of theoretical and empirical results for some stochastic population Models. *Biometrika* 47:1–11
11. Becker, N. G. 1970. A stochastic model for two interacting populations. *J. Appl. Probab.* 7:544–64
12. Becker, N. G. 1973. Interactions between species: some comparisons between deterministic and stochastic models. *Rocky Mount. J. Math.* 3: 53–68
13. Billingsley, P. 1968. *Convergence of Probability Measures.* New York: Wiley. 253 pp.
14. Billingsley, P. 1971. *Weak Convergence of Measures.* Philadelphia: SIAM.
15. Botkin, D. B., Sobel, M. J. 1975. Stability in time-varying ecosystems. *Am. Nat.* 109:625–46
16. Breiman, L. 1968. *Probability.* Menlo Park, Calif.: Addison-Wesley. 421 pp.
17. Bulmer, M. G. 1976. The theory of predator-prey oscillations. *Theor. Pop. Biol.* 9:137–50
18. Chesson, J. 1978. Measuring preference in selective predation. *Ecology:* In press.
19. Chesson, P. L. 1976. *Models for Animal Movements.* Ph.D. thesis, Univ. of Adelaide, South Australia
20. Chesson, P. L. 1978. Variable predators and anti-switching. In preparation
21. Chesson, P. L. 1978. Patch processes in population ecology. In preparation
22. Chewning, W. 1975. Migratory effects in predator-prey models. *Math. Biosci.* 23:253–62
23. Comins, H. N., Blatt, D. W. E. 1974. Prey-predator models in spatially heterogeneous environments. *J. Theor. Biol.* 48:75–83
24. Crowley, P. H. 1977. Spatially distributed stochasticity and the constancy of ecosystems. *Bull. Math. Biol.* 39: 157–66
25. Cumberland, W. G., Rhodes, C. A. 1977. A multivariate model for growth of populations. *Theor. Pop. Biol.* 11: 127–39
26. den Boer, P. J. 1968. Spreading of risk and stabilisation of animal numbers. *Acta Biotheor.* 18:165–94
27. Dubois, D. M. 1975. A model of patchiness for prey-predator plankton populations. *Ecol. Modelling.* 1:67–80
28. Feldman, M. W., Roughgarden, J. 1975. A population's stationary distribution and chance of extinction in a stochastic environment with remarks on the theory of species packing. *Theor. Pop. Biol.* 7:197–207
29. Getz, W. M. 1976. Stochastic equivalents of linear and Lotka-Volterra systems of equations—a general birth-and-death process formulation. *Math. Biosci.* 29:235–58
30. Gilpin, M. E. 1975. *Group Selection in Predator-Prey Communities.* Princeton, NJ: Princeton Univ. Press. 108 pp.
31. Goel, N. S., Maitra, S. C., Montroll, E. W. 1971. On the Volterra and other nonlinear models of interacting populations. *Rev. Mod. Phys.* 43:231–76
32. Goh, B. S. 1976. Non-vulnerability of ecosystems in unpredictable environments. *Theor. Pop. Biol.* 10:83–95
33. Guess, H. A., Gillespie, J. H. 1977. Diffusion approximations to linear stochastic differential equations with stationary coefficients. *J. Appl. Probab.* 14:58–74

34. Gurney. W. S. C., Nisbet, R. M. 1975. The regulation of inhomogeneous populations. *J. Theor. Biol.* 52:441–57
35. Gurney, W. S. C., Nisbet, R. M. 1976. A simple mechanism for population cycles. *Nature* 263:319–20
36. Hadeler, K. P., van der Heiden, U., Rothe, F. 1974. Non-homogeneous spatial distributions of populations. *J. Math. Biol.* 1:165–76
37. Hassell, M. P. 1978. *The Dynamics of Arthropod Predator-Prey Systems.* Princeton, NJ: Princeton Univ. Press.
38. Hastings, A. 1977. Spatial heterogeneity and the stability of predator-prey systems. *Theor. Pop. Biol.* 12:37–48
39. Hilborn, R. 1975. The effect of spatial heterogeneity on the persistence of predator-prey interactions. *Theor. Pop. Biol.* 8:346–355
40. Holling, C. S. 1973. Resilience and stability of ecological systems. *Ann. Rev. Ecol. Syst.* 4:1–23
41. Keiding, N. 1975. Extinction and exponential growth in random environments. *Theor. Pop. Biol.* 8:49–63
42. Keilson, J. 1964. A review of transient behavior in regular diffusion and birth-death processes. *J. Appl. Probab.* 1: 247–66
43. Keilson, J. 1965. A review of transient behavior in regular diffusion and birth-death processes. Part II. *J. Appl. Probab.* 2:405–28
44. Kendall, D. G. 1949. Stochastic processes and population growth. *J. R. Stat. Soc. B* 11:230–64
45. Kurtz, T. G. 1970. Solutions of ordinary differential equations as limits of pure jump Markov processes. *J. Appl. Probab.* 7:49–58
46. Kurtz, T. G. 1971. Limit theorems for sequences of jump Markov processes approximating ordinary differential processes. *J. Appl. Probab.* 8:344–56
47. Leslie, P. H. 1958. A stochastic model for studying the properties of certain biological systems by numerical methods. *Biometrika* 45:16–31
48. Leslie, P. H., Gower, J. C. 1960. The properties of a stochastic model for the predator-prey type of interaction between two species. *Biometrika* 47: 219–34
49. Levin, S. A. 1976. Population dynamic models in heterogeneous environments. *Ann. Rev. Ecol. Syst.* 7:287–310
50. Levin, S. A., Segel, L. A. 1976. An hypothesis to explain the origin of planktonic patchiness. *Nature* 259:659
51. Levins, R. 1966. The strategy of model building in population biology. *Am. Sci.* 54:421–31
52. Lewontin, R. C., Cohen, D. 1969. On population growth in a randomly varying environment. *Proc. Natl. Acad. Sci. USA* 62:1056–60
53. Ludwig, D. 1975. Persistence of dynamical systems under random perturbations. *Soc. Ind. Appl. Math. Rev.* 17:605–40
54. Ludwig, D. 1976. A singular perturbation problem in the theory of population extinction. *Soc. Ind. Appl. Math.–Am. Math. Soc. Proc.* 10:87–104
55. Manly, B. F. J., Miller, P., Cook, L. M. 1972. Analysis of a selective predation experiment. *Am. Nat.* 106:719–36
56. May, R. M. 1973. On relationships between various types of population models. *Am. Nat.* 107:46–57
57. May, R. M. 1974. *Stability and Complexity in Model Ecosystems.* Princeton, NJ: Princeton Univ. Press. 265 pp. 2nd ed.
58. May, R. M., ed. 1976. *Theoretical Ecology.* Philadelphia, Pa.: Saunders. 317 pp.
59. Maynard Smith, J. 1976. *Models in Ecology.* Cambridge: Cambridge Univ. Press. 145 pp.
60. McNeil, D. R., Schach, S. 1973. Central limit analogues for Markov population processes. *J. R. Stat. Soc. B* 35:1–23
61. Murdoch, W. W., Oaten, A. 1975. Predation and population stability. *Adv. Ecol. Res.* 9:1–131
62. Murray, J. D. 1975. Non-existence of wave solutions for the class of reaction diffusion equations given by the Volterra interacting-population equations with diffusion. *J. Theor. Biol. 52:459–69*
63. Nicholson, A. J., Bailey, V. A. 1935. The balance of animal populations. *Proc. Zool. Soc. London* 3:551–98
64. Nisbet, R. M., Gurney, W. S. C., Pettipher, M. A. 1977. An evaluation of linear models of population fluctuations. *J. Theor. Biol.* 68:143–60
65. Oaten, A. 1977. Optimal foraging in patches: a case for stochasticity. *Theor. Pop. Biol.* 12:263–85
66. Waugh, W. A. O'N. 1972. The apparent 'lag phase' in a stochastic population model in which there is no variation in the conditions of growth. *Biometrics* 28:329–36
67. Poole, R. W. 1974. A discrete time stochastic model of a two prey one predator species interaction. *Theor. Pop. Biol.* 5:208–28

68. Prajneshu. 1976. A stochastic model for two interacting species. *Stoch. Proc. Appl.* 4:271–82
69. Reddingius, J. 1971. Models as research tools. In *Dynamics of Population. Proc. Adv. Study Inst. 'Dynamics of Numbers in Populations,' Oosterbeek, Netherlands, 1970,* pp. 64–76
70. Reddingius, J. 1971. Gambling for existence. *Acta Biotheor.* 20: (Suppl. 1) 1–208
71. Reddingius, J., den Boer, P. J. 1970. Simulation experiments illustrating the stabilisation of animal numbers by spreading of risk. *Oecologia* 5:240–84
72. Roff, D. A. 1974. Spatial heterogeneity and the persistence of populations. *Oecologia* 15:245–58
73. Roff, D. A. 1974. The analysis of a population model demonstrating the importance of dispersal in a heterogeneous environment. *Oecologia* 15:259–75
74. Rosen, G. 1977. On the persistence of ecological systems. *J. Theor. Biol.* 65: 795–99
75. Roughgarden, J. 1975. A simple model for population dynamics in a stochastic environment. *Am. Nat.* 109:713–36
76. Segel, L. A., Jackson, J. L. 1972. Dissipative structure: an explanation and an ecological example. *J. Theor. Biol.* 37:545–59
77. Segel, L. A., Levin, S. A. 1976. Application of non-linear stability theory to the study of the effects of diffusion on predator-prey interactions. *Top. Stat. Mech. Biophys.: A Memorial to Julius L. Jackson, Proc. AIP Conf.* 27:123–52
78. Wu, L. S.-Y. 1977. The stability of ecosystems—a finite-time approach. *J. Theor. Biol.* 66:345–59
79. Zeigler, D. P. Persistence and patchiness of predator-prey systems induced by discrete event population exchange mechanisms. *J. Theor. Biol.* 67:687–713

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