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LOTKA-VOLTERRA POPULATION MODELS

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INTRODUCTION

In almost every field of science, and nowhere more than in biology, a tension has existed constantly between the experimentalists and the theorists; the tension is particularly strong in biology because there the theorists have not produced the kinds of advances that have come from the theoretical physicists and chemists. Among the biological theorists, the sub-class of mathematical modellers has often suffered the most from the onslaughts of their "more practical" brethren. To some extent, this tension has been the result of misunderstandings on the part of both groups. The experimentalists have often been almost innocent of the mathematical techniques needed for model-building. The modellers, often recruited from either physics or mathematics, have plunged directly into some of the most difficult biological problems with an impressive array of mathematical skills and an equally impressive innocence of biological principles. The reading required for this paper has led me to believe that the ex-mathematicians in particular display an almost cavalier disregard for the biological literature. When a faulty citation in one paper, involving an inversion in the order of authors, is repeated in a series of papers by at least three other authors, I am led to wonder whether the later authors bothered to look up the original paper, much less read it. A result of this lack of care has been the rediscovery of the wheel at regular intervals.

Certainly some of the difficulties between the two groups stem from basic misunderstandings, on both sides, of the nature and function of mathematical models. Models are too often considered simply as predictors, and any inability to predict accurately is accepted as prima facie evidence of the uselessness of the technique. Actually, only those engineering models designed to fit a particular set of circumstances are even moderately successful as predictors. The more general models of theoretical biology are used to deduce the form of possible solutions, rather than to predict future states of the system being modelled.

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If one were to erect a spectrum of model types, the end members would be the descriptive and the analytical models. The descriptive model is essentially a method for expressing data in a condensed form, emphasizing the regularities to be found. Starting with an accumulation of data, a line or curve of best fit is found. In its purest form, the terms in the descriptive model are all fitting constants; no mechanisms or actual physical quantities are implied. There is no unique descriptive model for a given system. Usually one can construct any number of equally valid descriptive models for the same collection of data. The great virtues of this type of model are its simplicity and its closeness of fit.

The analytical model, on the other hand, is constructed from consideration of the mechanisms involved in the system. It is generally based primarily on logic, with a minimum of actual data. It is almost always complex, but all of the variables involved correspond to actual physical or biological quantities or rates. This kind of model allows us to follow the effects of changes in the system on each of the parts, and leads to a better understanding of the workings of the system. Where the descriptive model permits, at best, limited prediction in stable circumstances, the analytical model at its best permits the prediction of the behavior of the system under unstable or greatly changed conditions. Since the aim of many of these models is prediction under changed, or changing, circumstances, it is understandable that the analytical model should be the aim of the theoretical ecologist. The great danger of these models is their abstraction from the natural condition, since the models are so complex that some simplifying assumptions must always be incorporated to permit either analytical solution or numerical approximation.

Most biological models are neither purely descriptive nor purely analytical; they are attempts to construct analytical models, with descriptive terms inserted to make the model fit the available data. The danger in such an in-between model is that the terms that are really fitting constants will be given names and will begin to assume biological characteristics never intended by their creators. I discuss several cases of this sort below.

Model builders make certain explicit assumptions in order to reduce the complexity of their models to a level permitting solution or approximation. They are not always aware, however, of the assumptions implicit in the mathematical form they choose for their models. In many cases, the kinds of solutions possible are severely restricted by these implicit assumptions; we should not be surprised by the answers we get when we ask such limited questions.

In this paper, I do not attempt to review the entire literature on the Lotka-Volterra models; this literature has grown almost as much as has the pollution literature in the past few years. Much of this literature is reviewed in a recent book edited by May (136). Instead, I indicate the major directions of investigation in the field, and mention papers I have found particularly illuminating or stimulating.

THE GROWTH OF A SINGLE SPECIES POPULATION

The foundation of all deterministic models of interactions between species is the model for the growth of a single species in an unvarying environment. Most of these

models are direct descendants of the Malthusian model, which proposes an exponential increase in population size with time, as

$$dx/dt = rx, \qquad 1.$$

where x = population number and r = a constant rate of increase. The explicit assumption made in this equation is that the rate of increase remains the same no matter what the population size. While this appears a priori to be a biologically unreasonable assumption, the exponential mode of population increase is apparently common in laboratory populations whenever they are not limited by lack of nutrients or space.

If we accept r simply as a constant, chosen to fit the equation to the straight-line portion of population growth data, we cannot get into too much trouble. However, if we try to assign biological significance to this constant, we can quickly find ourselves making unsupportable assumptions. One of the usual assumptions is that the constant r can be decomposed into the difference between two terms, as $(b - d)$, where b is the birthrate, d is the death rate, and both are constant with respect to population number. The constant r then becomes the "intrinsic rate of natural increase," which is expected somehow to be a property of the species.

A little reflection on the mechanisms of population growth will suggest that this rate is dependent upon the genetics of the population and the quality of the environment. The rate cannot, therefore, be specific to the species, or even to the particular population; it is specific to the experiment, and to nothing else. We might insist that for a given species there is some value of r that cannot be exceeded, that results when the genetic strain yielding the most offspring is raised in the environment most suited to it. This would be a Platonic ideal of r, since we could never know when we had achieved it. Even when the birthrate was at its physiological limit, there would always be the possibility that in some more salubrious environment the death rate might be decreased.

Such a definition for the intrinsic rate of natural increase is operationally difficult. Since even in experimental populations raised under laboratory conditions this rate will seldom (if ever) be achieved, we must rewrite the original growth equation in the form

$$dx/dt = prx, \qquad 2.$$

where p is the proportion of the maximum rate of increase actually achieved in the particular experiment. This approach makes r an estimated value, based on our judgment of the maximum birth and minimum death rates possible, and thus turns p into our "fitting" constant; for a single fitting constant we have substituted two estimates (of b and d) and a new fitting constant, with little gain in understanding.

In any case, unless we are writing a Sunday supplement article on human population growth we cannot accept a growth equation as simple as equation 1. Every species studied so far has shown some sort of control over its ultimate population size, if only that of starvation of the weaker members as food becomes scarce. Density-dependent population control was the subject of intensive debate in earlier

years. The negative viewpoint was held primarily by field scientists who worked with insect populations on which the influence of the weather was greater than any possible influence of increased density. Similar findings were reported for some northern birds and mammals (154, 155), and Dodson (46) suggested that in natural populations of *Daphnia rosea,* the summer decrease can be explained entirely by predation. The skeptical position was considerably strengthened by the difficulty in determining by mathematical analysis whether a particular population showed any density dependent effects (18, 48, 128, 180, 235). In our later discussion of the effects of time lags on population models we will see theoretical reasons why the efforts to elucidate density dependence by regression techniques should indeed fail.

However, with increased examination of the mechanisms by which birth and death rates are affected by population size, the influence of population density on these rates has largely been accepted. Almost every conceivable method for altering either birth or death rates has been found. Perhaps because of the well-recognized fluctuations in population size in some small mammals, much of the early laboratory and field work was done with mice, lemmings, voles, and rats. Christian's early work (25, 26) on fluctuations in adrenal weight with population size focused attention on the physiological basis for density-dependent regulation in these small mammals (10, 89, 120, 122, 205). Other workers have chosen to look particularly at behavioral expressions of physiological change, such as increased aggressiveness with increasing population density, in such diverse organisms as various species of mice (81, 107, 191, 210, 226), the red grouse (234), and the cockroach (52).

Changes in both fetal (93, 118) and infant mortality (53, 73, 85, 145, 146, 148, 204) have been demonstrated. Differences in behavior attributed to crowding of the mother during pregnancy have been seen in mice (100); interruptions in the breeding cycle, including early cessation of breeding (43, 101) and delay of sexual maturity (99) have been reported. In insects, competition for egg-laying sites seems to be a common means of density-dependent control (7, 33, 227), a mechanism also reported in green sea turtles (22). Changes in the growth rate of green hydra (207), in the length of adult life in the azuki bean weevil (215), in its mortality rate (140), and in egg cannibalism in *Tribolium* (141), all connected with increased population density, also affect the rate of increase. Two particularly interesting mechanisms of control are the suppression of growth in tadpoles by soluble materials added to the water by larger tadpoles (176), and the lengthening of the mean sterile period between births in primitive human populations by an increase in the lactation period (189). The regulation of numbers in vole populations has been reported, without examination of possible mechanisms, from a variety of environments (28, 29, 117, 233). Tanner (200) examined 111 populations, including 71 species, and found definite density effects in 47 species. He concluded that most animal species regulate their numbers by some density-dependent mechanism, rather than by predation. An excellent semipopular account of the effects of crowding on populations has been written by McBride (139).

Density-dependent population control can be incorporated into a growth equation by the addition of a term modifying the rate of growth as the population

increases. The damping term introduced in this fashion usually functions as some power of the population number; the term is normally given as a function of x^2, as

$$dx/dt = rx\ [(K - x)/K] \qquad 3.$$

or

$$dx/dt = rx\ [1 - (x/K)]. \qquad 4.$$

In these equivalent representations, it is assumed that there is some number of organisms that the environment can support, expressed as K, the equilibrium number or the carrying capacity. These equations are variant expressions of the logistic equation. Through the years the damping term, $[(K - x)/K]$, has come to be called the "biotic resistance;" along with the name has come the implication that it should in some way be measurable as something other than a fitting function. If we wish to make no assumptions concerning equilibrium levels, the growth equation can be written as

$$dx/dt = rx - ax^2, \qquad 5.$$

where a is a fitting constant.

The logistic equation fits many population growth experiments about as well as any biological data are ever fitted; for this reason, as well as for its simplicity, it has generally been accepted as the standard model for single-species population growth. Only two constants must be determined, the "intrinsic rate of natural increase" or "biotic potential," and the "biotic resistance" or carrying capacity. While neither of these quantities is really amenable to exact mathematical definition, both represent tendencies easily envisioned by the average ecologist. Two extremes of evolutionary strategy may be recognized: (*a*) an evolution toward the fastest possible growth rate: the r strategy; and (*b*) evolution toward the most efficient use of the environment, resulting in the largest carrying capacity, the K strategy. It is generally considered that the pure r strategy is followed by opportunistic species, species that can expand rapidly into favorable environments, or into marginal environments in particularly favorable years. A pure K strategy is followed by species using more stable environments, and possibly by those with a lower carrying capacity. The trade-offs involved in r and K selection strategies were discussed by MacArthur & Wilson (125), and by a large number of other workers since then.

In order for the logistic equation to be used as a model for population growth, certain assumptions, not always stated, must be accepted. The use of a differential equation implies that both birth and death may be treated as continuous processes, with no seasonality involved. One can argue that the model may be applied as long as the sampling interval is long enough so that the birth and death rates can be treated as averages over the span between samples. However, if the population being studied is one that exhibits large fluctuations during the year (28), sampling on a given date or through a single short period may give a false picture of the normal behavior of the population (Figure 1). The model is thus most applicable to those

organisms whose life span is either very short or very long compared to the time span of normal environmental change; it is useful for amoebae and elephants, but not for voles and mice, except during periods of active population increase.

The rate of increase, *r*, is calculated from the straight-line portion of the growth curve, that region in which a stable age distribution is thought to exist. The age distribution will be altered as density-dependent restraints are applied to the population; the way in which this distribution changes will depend upon the mechanisms by which the control is exerted. Obviously, a control based upon increased infant mortality will lead to a different age structure than one based upon decreased length of adult life or decreased length of fertile life. The way in which a population recovers from density-induced constraints will depend upon the age distribution at the time of the release from constraint. There is no way of modelling such differences in a simple deterministic equation. Models do exist in which age structure can be incorporated directly; these involve quite different mathematical approaches, such as network models (115), matrix algebra models (14, 212), or stochastic models (86, 149, 172, 236, 238). While these approaches are interesting, they usually do not lend themselves to the study of multispecies interactions because of the complexity of the mathematics. It is possible to incorporate age structure into deterministic models of the Lotka-Volterra type, but the increase in complexity is considerable.

A number of attempts have been made to generalize the logistic equation, and to examine its mathematical properties (219, 228, 242). Relatively simple variations on the model have been proposed, such as the incorporation of a monotonically increas-

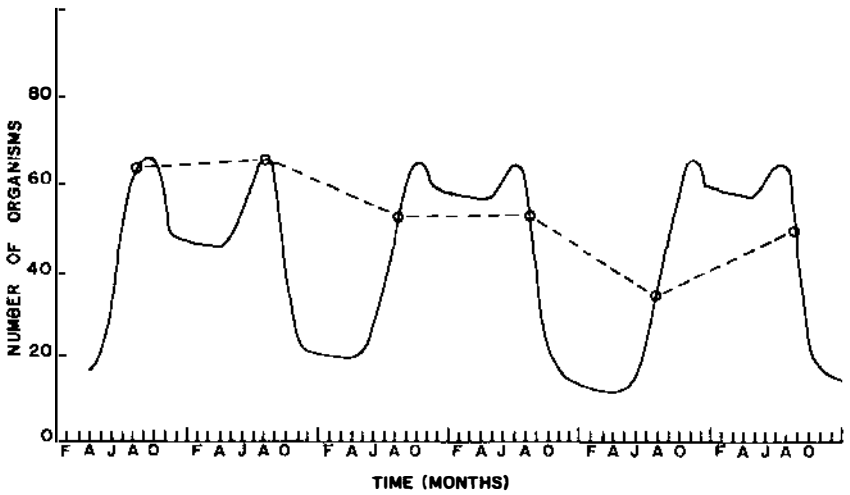

Figure 1 Growth of a population with a summer maximum and a reduced winter kill. Solid line is the actual number, and the dotted line is the number estimated from an annual sampling period of one week. A theoretical extension of work by Clarke (28).

ing value of K (211), random variation in growth rates (116), variations in both r and K (121), variations in the form of the survival curve (183), variations in r and K as functions of population size (113, 184, 198), and the use of a two-stage growth equation, separating mortality and fecundity (208).

It would be possible to pursue these lines of research to a still greater complexity; one of the advantages of the relatively simple logistic equation is that its constants can easily be manipulated, with the aid of a modern pocket-sized programmable calculator, to simulate anything from a varying environment to a varying response. However, rather than elaborate what is admittedly too naive a model, let us examine models of the next level of complexity (and possibly reality).

One of the unstated assumptions in the logistic equation is that the damping term, $[1 - (x/K)]$, always acts as the square of the population density. There is no biological justification for this assumption; in fact, it would be unusual if there were only one kind of damping term for all the mechanisms of density dependence. Gilpin, Case, & Ayala (66) proposed a growth equation of the form

$$dx/dt = rx\,[1 - (x/K)^{\theta}], \qquad 6.$$

where θ can be other than one. The authors felt that the values of $\theta > 1$ correspond to growth in species where the controlling mechanism is territoriality, and where no constraint on growth occurs until all of the territories are occupied. Cases where $\theta < 1$ occur when there is some sharing of the scarce commodities, and the pressure of added population is felt early in the population increase.

An increase in the value of θ results in a steepening of the growth curve and a higher value for the inflection point (Figure 2). Theoretically, this change in formulation is very important, since it permits a variety of expressions for the damping factor. Practically speaking, the curves resulting could be fitted within the usual tolerances for biological data by the use of a higher value for r in the simpler form of the equation. The shift in inflection point would probably not be noticed. If r were to be calculated from biological first principles, instead of simple curve fitting, then a variable θ might be useful in fitting theory to experiment, and mechanisms resulting in particular values of θ might be forthcoming. Numerical calculations do point out, however, that two populations with the same birth and mortality rates when not subjected to density-dependent constraints can have different apparent values for r if density dependence is effected by different mechanisms. This again points out that r must be considered as a constant chosen to fit a given data set, and not as a rate determined by relatively simple biological interactions.

We might also make a case for selection for high values of θ as one form of r selection. Higher apparent r values could be obtained for a given value of r, as determined under conditions of low population density, by selecting for density-dependent controls that act as a higher power of the damping fraction (x/K). These controls would take effect later in the growth process and would be more severe. Territoriality would be a good example of a mechanism of this type.

A more important limitation of the logistic equation is its inability to fit any growth curve other than the monotonic approach to equilibrium. It has long been known that some natural populations are subject to periodic fluctuations in size. The

species involved include a wide variety of organisms and types of life history, such as the azuki bean weevil (213), the muskrat (49), *Daphnia* spp. (171), *Tribolium castaneum* (160), the blow fly (150), *Microtus californicus* (106), the cotton rat (76), and the house fly (202), among many others. One of the best-known and most fruitful of ecological symposia concerned itself entirely with the question of cycles in animal populations (83). It is the combination of the inability of the logistic equation to predict population oscillations and the obvious evidence for such oscilla-

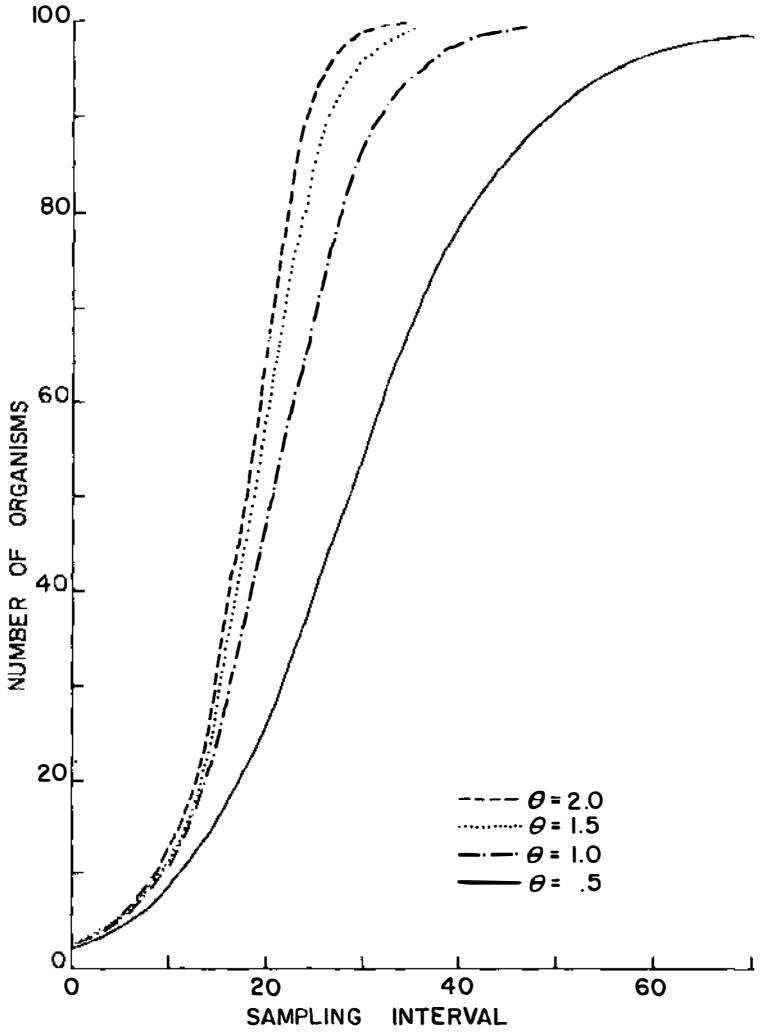

Figure 2 Single species growth curves, r and K held constant, θ varied.

tions in nature that has led to the dominant position in ecological theory of the Lotka-Volterra prey-predator equations; for a period, every cycle found in a natural population was attributed to some form of prey–predator interaction. This viewpoint was undermined considerably by the discovery of similar oscillations in laboratory populations in the absence of predation and in the presence of excess food. It was obvious that the logistic equation was insufficient as a model for these populations.

In biological terms, the logistic equation presents a logical absurdity, since it requires that cause and effect occur simultaneously. It is difficult to imagine a form of density-dependent control that would take effect so abruptly; instantaneous mortality for all members of the population in excess of the equilibrium number might work in this fashion. Most forms of population control take effect some time after the population begins to feel the increase in density. Thus, a time lag should be incorporated into any single-species growth model; in its most general form, a discrete lag is introduced into the damping term of the equation, as

$$dx/dt = rx[1 - (x/K)]_{(t-\tau)}, \qquad 7.$$

where τ is the time lag between cause and effect. The equation in this form has a long and honorable history, having been introduced by Hutchinson (94); the mathematical consequences of the formulation were apparently first worked out on the back of an envelope by Lars Onsager during tea at the Elizabethan Club at Yale. An analytical solution to this equation was later supplied by Cunningham (34). Since that time, a number of essentially similar equations have been proposed (27, 38, 91, 114, 194, 246). In these models differential equations are used. For every differential equation, there is an analogous difference equation; models using the difference-equation structure have also been proposed (132, 135, 138, 179, 192, 225). It is also possible to introduce lags into models using other mathematical forms altogether. Lefkovitch (109) incorporated delayed responses into a matrix algebra model for population growth.

The incorporation of even the simplest form of time lag into the growth equation results in an increase in the kinds of solutions possible. The lag in the response of the system to an increasing population density allows the population to overshoot the equilibrium level. With relatively short lags, the population growth form is a monotonic approach to equilibrium, much like that of the logistic equation. With higher values, an overshoot appears, which can become a damped oscillation. With still higher values, a limit cycle may result (Figure 3). The point at which the form of the growth curve changes is dependent on the product $r\tau$. When $r\tau < 0.7$, a monotonic approach to equilibrium results. At $0.7 < r\tau < 1.8$, damped oscillations about an equilibrium level are found; at $r\tau > 1.8$, a limit cycle appears (230). With slightly different formulations for the equation, slightly different values may define the shapes of the curves (38), but the principle remains the same.

When lags of this sort are incorporated into the growth equation, it is no longer necessary to postulate prey–predator interactions to explain every oscillating population. Actually, many of the apparent prey–predator oscillations may better be explained as the response of the predator organism to a fluctuating food supply (232).

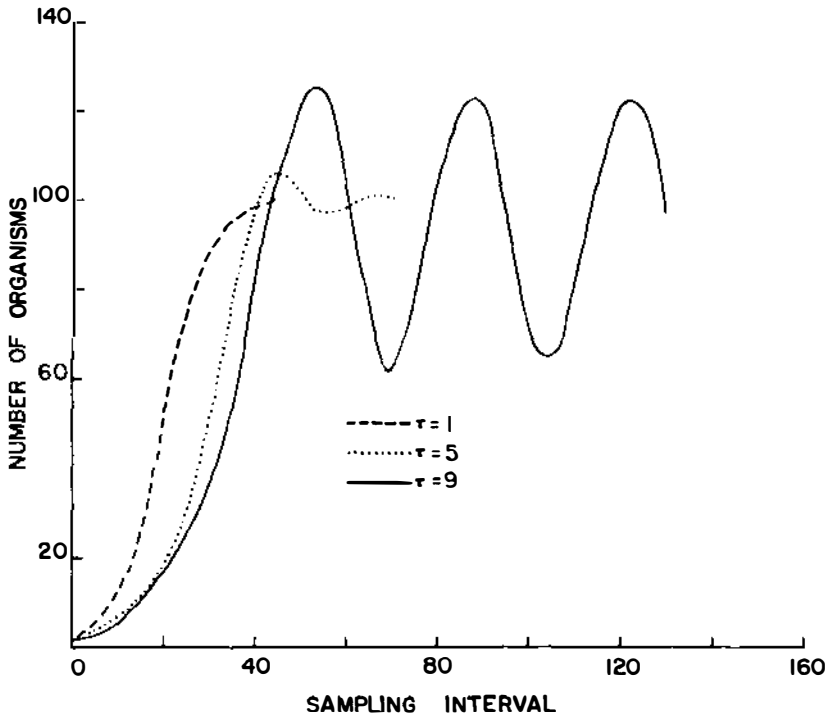

Figure 3 Single species growth curves, r and K held constant, τ varied.

Many complications can be imposed upon this most general time lag equation. The θ formulation can be employed, as in

$$dx/dt = rx[1 - (x/K)^{\theta}]_{(t-\tau)}. \qquad 8.$$

As I have already indicated, the effect of including the θ term is to modify the apparent value of r. There is actually a greater effect than a simple increase or decrease in r; high values of θ lead to limit cycles with very sharp peaks, since the density-dependent controls take effect late in the population cycle. However, this effect is not so marked that the difference between high θ and high r could be distinguished easily in data from experimental populations. For all practical purposes, an increase in θ can be modelled just as well by substituting a higher apparent r.

The lag used in these studies is necessarily a single discrete lag; as such, it is at least theoretically a poor representation of the temporal relationships in real populations. The actual extent of the time lags must depend upon the mechanisms controlling population density and may vary in length with population size. If control is exerted through reduced fertility or reduced breeding, the normal lag for an organism that breeds continuously would be the length of the gestation period. If the

control occurs through increased mortality, then the length of the lag will depend upon where in the life span the increase in mortality appears. The formulation of a realistic lag may be very complex, but the results, as far as they have been developed, appear to be much like those found with discrete lags (24).

Most models of population growth have assumed that the environment remains essentially constant throughout the experiment. Some attempts have been made to consider the effects of variations in the environment. If the environment fluctuates periodically, variations affecting the carrying capacity can force a population, normally growing in the logistic form, into oscillations (152, 232). If the normal growth form of the population is a limit cycle, only those frequencies close to the normal frequency of the population limit cycle will have much effect on the population growth (152).

When the environment fluctuates in a random fashion, it may be difficult to determine the normal growth form. Even a small amount of stochastic variability may render the normal tests for periodicity useless (133, 137, 170). It seems obvious that even a small increase in complexity, particularly in the modelling of the influence of the environment on population growth, can result in a model whose output cannot be distinguished from that of a completely Markovian universe. There seems to be little profit in pursuing the growth model to this extreme.

It is possible to construct a model for population growth that considers actual mechanisms of density-dependent control, rather than the simple damping term $[1 - (x/K)]$. Such a model could have more realistic lags built into the system, and might take the form

$$dx/dt = bx_{(t-\tau_1)} - dx_{(t-\tau_2)}, \qquad 9.$$

where b is the birth rate and is a function of x at time $(t - \tau_1)$, and d is the mortality and is a function of x at time $(t - \tau_2)$. The mechanisms of density-dependent control will then determine the shape of the two functions and the lags involved. However, this form of growth equation is far removed from the Lotka-Volterra equations.

If the normal growth form for a population is a damped oscillation or a limit cycle, the use of autocorrelation functions to determine the extent of density dependence will not be successful. Half of the time the correlation between the population size at time t and that at $t + 1$ will be positive, and half the time it will be negative. Each point on the growth curve should really be designated by a number and a direction, either positive (growing) or negative (decreasing). Some form of nonparametric statistics would then have to be employed, since the relationship of x_t to $x_{(t+1)}$, even if the positives and negatives were grouped separately, will be distinctly nonlinear.

PREY-PREDATOR EQUATIONS

The classical approach to the description of the interaction between two species, one of which feeds upon the other, is the Lotka-Volterra prey-predator model, where

$$\begin{aligned} dx/dt &= rx - \alpha xy \\ dy/dt &= \beta xy - Dy, \end{aligned} \qquad 10.$$

with x = the number of prey organisms, y = the number of predator organisms, α = a proportionality constant linking the prey mortality to the number of prey and predators, β = a proportionality constant linking the increase in predators to the number of prey and predators, and D = a constant of mortality for the predators.

The model produces the prey–predator cycles familiar to all ecologists, with growth in the predator populations trailing that in the prey. As I have already mentioned, almost every population that showed periodic fluctuations was once considered to be part of such a prey–predator interaction. In field populations, such interactions could be postulated, but seldom demonstrated conclusively. Perhaps the most famous examples have been found in the fluctuations of arctic animals: the snowshoe hare–lynx (50), and the brown lemming–grass (206) interactions. Neither of these cycles has yet been demonstrated conclusively to be due to a prey–predator interaction, in spite of many years of work. It is unlikely that any amount of statistical analysis or collection of data in the field will settle this question; long-term field experiments are clearly necessary. Somewhat better data and analyses are available from insect populations (88, 218), in part because of the shorter life spans of the organisms involved.

Examples of prey–predator interactions that fit the model more closely are available from laboratory populations. These experiments have included such pairs of species as the bacterium *Klebsiella aerogenes* and its protozoan predator *Tetrahymena pyriformis* (220); the classical prey–predator pair, *Paramecium aurelia* and *Didinium nasutum* (181); *Paramecium* and the rotifer *Asplanchna* (129); *Hydra* and its parasite, *Hydramoeba hydroxena* (195); two studies using the house fly, *Musca domestica,* and parasites, *Mormoniella mestica* (41) and *Nasonia vitripennis* (169); two species of mites (175); the host-parasite pair *Trialeurodes vaporariorum* and *Encarsia formosa* (21); and the azuki bean weevil, *Callosobruchus chinensis,* and its parasite, *Heteropilus prosopidis* (216). Oscillations were found in most of these populations. In most cases, the oscillations were of the type predicted by the Lotka-Volterra equations. Pimentel (169) and Utida (216) found damped oscillations rather than limit cycles. These forms cannot result from Lotka-Volterra prey–predator interactions. These authors attributed the damping to genetic selection. DeBach & Smith (41), using a somewhat artificial experimental plan, predicted expanding oscillations leading to the extinction of either prey or parasite.

The classical Lotka-Volterra formulation has been carried much further; the effect of minor perturbations has been considered (13, 182), the effect of varying age structures has been studied (12), and the general behavior of such systems of equations has been studied (20, 47, 54, 57, 72, 87, 111, 119, 244). An attempt has been made to use the Lotka-Volterra model to select the point at which to interfere with a prey–predator interaction in order best to control the predator population (70). Stochastic versions of the model have been constructed (77, 237). A graphical approach to predation theory has also developed (177, 178, 197, 223); to some extent, this is outside the frame of reference of this paper.

Several investigators have analyzed the behavior of the standard prey-predator equations, sometimes with purely theoretical populations, and sometimes with numbers derived from experimental populations. In many cases, some form of density

dependence had to be included in the growth term of the prey population in order to find stable solutions (51, 60, 75, 124, 167, 199, 201, 222). One mathematical statement of this incorporation of density dependence might be

$$\begin{aligned} dx/dt &= rx[1 - (x/K)] - axy \\ dy/dt &= \beta xy - Dy. \end{aligned} \qquad 11.$$

There are a number of ways in which density dependence can be introduced in these models: simple density dependence, without specification of the mechanisms involved (51, 124, 201); limitation of prey birthrates (199); territoriality (60); and migration and local extinction (222). Models have also been constructed with density-dependent limitation on the predator population (75, 167). It is evident that in many models the rate of growth of the prey population, if unmodified by density effects, cannot be controlled by predation as long as the interaction between the two populations is kept strictly linear. In other models, strict linearity results in the extinction of the prey during periods of high population of the predators.

This restriction on interactions becomes even more marked when we consider models in which temporal relationships are maintained. As in the single-species growth curve, effect must follow cause by some perceptible lag. While an extremely complex model can be constructed, with lags incorporated into every term, such a model is unusable except on the largest computers. Much can be learned by the examination of even so simple a system as

$$\begin{aligned} dx/dt &= rx - axy \\ dy/dt &= \beta xy_{(t-\tau)} - Dy. \end{aligned} \qquad 12.$$

This pair of equations was investigated by Wangersky & Cunningham (231). The equations in this form have no stable solutions. In ecological terms, this means that in a system so described, with a lag between the reaction of the predator population to changes in the prey population, the growth of the prey cannot be regulated simply by any linear interaction between prey and predator. This situation has also been discussed by Luckinbill (123). When density-dependent regulation of the prey growth is incorporated into the model, as

$$\begin{aligned} dx/dt &= rx[1 - (x/K)] - axy \\ dy/dt &= \beta xy_{(t-\tau)} - Dy, \end{aligned} \qquad 13.$$

the solutions fall into three classes and one special case. The special case, where $\tau = 0$, is the familiar Lotka-Volterra prey–predator cycle. For low values of τ, both prey and predator populations approach equilibrium monotonically. For higher values, a damped oscillation of both populations about their equilibrium values can be found. Still higher values result in limit cycles of the Lotka-Volterra variety. Thus, instead of a single response, the limit cycle, a whole array of responses can be found; the form of the system response is determined by both the size of the lag between the change in prey population and the response of the predator, and by the strength of the coupling between the populations. If the growth of the prey popula-

tion is rapid compared to that of the predator, and α, the interaction factor, is small, the predator may have little effect on the growth of the prey. In such cases, the growth curve of the predator may be a simple response to that of the prey population.

Interactions between prey and predator populations can be other than linear. Arguments can be made for almost any interaction form one would care to use. In one admittedly highly artificial experiment on predation by DeBach & Smith (41), linear interaction terms were used, and the resulting population forms were expanding oscillations of the type postulated by Nicholson & Bailey (151), leading to extinction of one or both of the populations. The interaction could be fitted more closely by a hyperbolic tangent function. When such a function was inserted, the two populations quickly settled into limit cycles, with an exceptional fit to the experimental data points (232).

It is apparent that there is not just one possible model for the prey–predator interaction with time lag, but at least three: one in which there is no density-dependent control on the growth of the prey population, but the interaction terms are nonlinear; one with linear interaction terms and density dependence; and one with nonlinear interaction terms and density dependence. One could also consider the case in which both prey and predator are subject to density-dependent control. The model to be preferred must depend upon the biology of the system.

Models of predation and parasitism incorporating time lags have become more numerous in recent years (6, 15, 17, 36, 37, 104, 126, 127, 144, 190). Many of these papers are more rigorous in their mathematics than the Wangersky & Cunningham papers (230, 231, 232), but suggest little that is new in their conclusions. Arditi, Abillon & Da Sylva (6) consider the effects of a lag in the predator's death term, and suggest that such a lag can stabilize an otherwise unstable model. Murray (144) includes a term for diffusion of a population, which permits travelling wave solutions. Brauer (17) introduces the possible use of harvesting to stabilize the populations. Most of the modellers who have worked with time lags have concluded that there is little to be gained from the study of systems more complex than the one-lag models described in this paper.

COMPETITION MODELS

If the Lotka-Volterra prey–predator cycle is considered one of the founding principles of modern theoretical ecology, then the competition equations are surely another, and of equal importance. In their simplest form, these equations describe the interaction of two species in competition for a common resource. The equations can take the form

$$\begin{aligned} dx/dt &= r_x x\ [(K_x - x - \alpha y)/K_x] \\ dy/dt &= r_y y\ [(K_y - y\ \beta x)/K_y]. \end{aligned} \qquad 14.$$

This form of the equation states that the presence of a species y depresses the rate of growth of a species x in a manner proportional to the numbers of species y

present, and vice versa. The form of the solutions thus depends upon the strength of the interaction terms. If α is very high relative to β, species y will affect the growth of x more than x affects y, and species x will eventually be eliminated from the competition.

The principle of competitive exclusion, the concept that two species in competition for the same resource cannot coexist, has become one of the foundations of theoretical ecology. It has given the concept of ecological niche a sharper focus, since the coexistence of any two species, no matter how similar they might seem, implies that the species are not in true competition, and that differences removing them from direct competition should be present in their life histories. Such differences can always be found; whether they are enough to remove species from direct competition, however, has always been difficult to prove. The consideration of more and more components of the environment leads naturally to the description of the niche of a population as a volume in a hyperspace, each dimension of which is a gradient in one attribute of the habitat (95), and of the evolution of a population as the trajectory of a niche through time, following an optimal pathway (229).

While such speculations and extrapolations have been fruitful, and, in fact, have shaped modern ecology, we must remember that the solutions found for the mathematical statements in no way test or prove the truth of the principle of competitive exclusion. The results are inherent in the equations used; once the equations are written and the constants evaluated, the solutions are determined. If the growth and interaction terms could be evaluated from first principles, some predictive capacity might be claimed for the equations. Often, the values of r can be determined from single species growth experiments, but the interaction terms must almost always be determined from actual experimental interactions. As it is most often used, the principle of competitive exclusion is a tautology. The place of this principle in ecological theory has been disputed for many years (4, 5, 30, 78, 165, 176); perhaps the best discussions have been those of Hardin (78) and Peters (168). Whatever our personal belief on the reality of the phenomenon of competitive exclusion, the concepts derived from it have become so firmly embedded in modern ecological theory that even were the principle to be proven false, it is unlikely that any attempt would be made to rebuild theoretical ecology from scratch.

The evidence for competitive exclusion in natural populations is spotty and often almost anecdotal, based largely on the mutual exclusiveness of closely related species and on speculations about possible mechanisms of separation (19, 23, 42, 82, 84, 98, 188). Occasionally it has been possible to conduct experiments in field situations; thus, Wilbur (240) investigated the nonlinear, nonadditive terms in the competition interaction functions in a field enclosure study of a salamander–tadpole community; Brown (19), Heller (82), and Sheppard (188), examining chipmunk communities, considered aggressiveness as the main mechanism of competition; and Istock (96), determining α and β in experimental populations of water-boatmen, found he could explain seasonal variations in natural populations on the basis of simple competition.

The possible mechanisms of competition have been studied much more closely in experimental populations. As might be expected, the laboratory studies have been

conducted with insects, zooplankton, and other organisms small enough to be grown in quantity in aquaria and small cages. The actual data are thus largely restricted to organisms with short life spans and generation times, and usually to species that reproduce continuously. Experimental organisms are chosen to fit the convenience of the experimenter, and neither the graduate student nor the granting agency can be expected to become involved in the population dynamics of elephants. We are thus continually experimenting with organisms of one type, the short-lived continuous breeders, and extrapolating to all others, including those with definite, restricted breeding periods and long life spans. We are examining ants and commenting on the habits of water buffalo.

The best-studied competition is probably that between the flour beetles *Tribolium castaneum* and *T. confusum* (39, 40, 110, 156–163). Park and his co-workers were able to correlate environmental conditions with success or failure in two-species competitions, and demonstrated a region of indeterminacy of outcome, where changes in population inoculum size could alter the normal course of competition. Later workers have shown (39, 40, 110) that such results may be specific not to the particular species involved, but to the genetic strains used; thus, competition coefficients determined in laboratory experiments can be applied to natural populations only with considerable reservation, especially when experimental populations are started with a small inoculum.

Opposing conclusions have been reached on the utility of competition models in explaining experimental results in *Paramecium* populations. Vandermeer (221) felt that the logistic and simple competition equations could explain his results satisfactorily, while Gill (63) thought the presence of endosymbionts might be responsible for the outcome of competition in his populations. The situation with *Drosophila* seems somewhat simpler, with most workers agreeing that the outcome of competition could be predicted from population growth rate measurements, with competition being limited strictly to the acquisition of food (8, 59, 97, 142, 143, 173). Richmond et al (173), predicting the outcome of three-species competitions by linear superposition of two-species competition coefficients, felt that the simple Lotka-Volterra theory was incomplete.

Utida (214) summed up many years of study of bean weevil population dynamics in a paper on competition between two species in the presence and absence of parasites. Under the conditions of these experiments, competition in the absence of parasitism always resulted in the extinction of one species. Coexistence was possible when the parasite was present in both species. Yoshida (245) found that the order in which the species were put into the arena and the size of the population inoculum were more important than the competition coefficients in determining the outcomes.

Frank demonstrated in laboratory experiments that two cladoceran species, *Daphnia pulicaria* and *Simocephalus vetulus,* could not coexist (55), and that in cultures of two species of *Daphnia* (*D. pulicaria* and *D. magna*) although these species seemed more likely to coexist, *D. magna* was always eliminated under the conditions of the experiment (56). Allan (2), working with *D. parvula* and *Holopedium gibberum,* measured competition coefficients from laboratory experiments. The measured coefficients suggested that under natural conditions the species

should coexist, with *Holopedium* having the larger population, although it is the poorer competitor. In nature, *Daphnia* was rare and declining throughout the study, presumably because of selective predation. We can safely say, from evidence derived from both natural and laboratory populations, that the situation is almost always more complicated than is evident from the models.

There is a considerable literature on somewhat more complex models, largely derived from the Lotka-Volterra competition models. The commonest form of complication is the shift from linear to nonlinear terms (16, 35, 64, 67, 217). These nonlinearities can take the form of nonlinear growth rates, interaction terms, or both. The case of the linear model in a fluctuating environment has also been studied (74).

Schoener (185–187) has been a prolific and interesting theorist in this area of research. He has looked at the differences between linear and nonlinear models, both for models of competition involving interference between species and for those involving exploitation of a common resource (185); he has calculated competition coefficients for species feeding on the same resource (186); and he has proposed a model of some complexity, incorporating mechanisms of competition, as an alternative to the standard Lotka-Volterra models.

A number of workers have constructed competition models to fit specific situations or specific experiments. These models often deviate considerably from the original Lotka-Volterra model. For example, Taylor (203) and Niven (153) devised models of *Tribolium* species in competition. De Wit et al (45) proposed a number of models of competition for space in mixed crops, one of the few cases of the application of competition theory to terrestrial plants. Andersen (3) examined the special case of competition between populations all of one age group. This kind of competition would be restricted to organisms without overlapping generations. Stewart & Levin (193) were interested in species whose growth was seasonally restricted, and found that this mode of existence permitted the coexistence of competitors. This is a result that could not normally be predicted from experimental competitions carried out in the unvarying conditions of the laboratory. Many different models were constructed by Ayala, Gilpin & Ehrenfeld (9) in an attempt to fit experimental data from competition between species of *Drosophila*. Several of the models fit the experimental data considerably better than did the simple Lotka-Volterra formulation. Wiegert (239) and Vandermeer (224) used equations that allowed different forms of competition and of population growth. Hubbell (92) used signal-flow graph notation in his models, in place of the usual differential equations, and Riebesell (174) examined the effects of enrichment of the systems, leading to a situation of great density, and finally to instability in a system that was stable at lower densities. One of the most interesting of these variations on a theme is the paper by Wuenscher (243), in which he attempts to examine competition models in the context of overlapping of hyperspace niches.

All of these models, simple and complex, can have only one general form of solution; one or both populations may rise in the early part of the competition, while total populations are low, but one of the two reaches a peak and then declines to extinction. Once one of the populations has started to decline, as long as environ-

mental conditions remain constant there is no possibility of reversal of the direction of competition. Experimental populations, particularly when run in replicate, often display one or several reversals before extinction occurs. Such reversals would certainly be expected if one or both populations normally followed a damped oscillation or limit cycle growth form. If equations incorporating time lags were to be written, the lagged terms would be, as usual, the damping factors, and the equations would take the form

$$\begin{aligned} dx/dt &= r_x x[(K_x - x - \alpha y)/K_x]_{(t-\tau_x)} \\ dy/dt &= r_y y[(K_y - y - \beta x)/K_y]_{(t-\tau_y)}. \end{aligned} \qquad 15.$$

Equations of this form have been studied by Wangersky & Cunningham (232). These equations are variations on the standard Lotka-Volterra equations, and are therefore subject to the same constraints. If the two species are in complete competition, the ultimate result must be the elimination of one of the populations. However, the addition of the lag terms adds to the number of variables that together determine the direction of competition. Given two species in complete competition, the end result of the competition can depend upon the relative growth rates, the interaction coefficients, the relative numbers at the start of the competition, and the sizes of the lags. If we consider that in such a competition a saddle point exists, dividing the probable outcomes, and that the ultimate direction of the competition depends upon the side of the saddle point where the populations finally stay on (Figure 4), then the choice of starting conditions is as important as the choice of the various coefficients in determining the outcome. The incorporation of lags into the equations allows the populations to fluctuate considerably, and also allows reversals of the apparent direction of competition; such a reversal cannot occur with the non-lag formulations.

The effects of lags on competition have not been explored to any great extent. Hassell & Comins (79) have formulated their competition models as discrete time equations, thereby making lags of a fixed length implicit in the structure of the systems. They find oscillatory solutions resembling some of the Wangersky & Cunningham (232) results. Gomatam & MacDonald (71) use a mathematical formulation of much greater complexity, but find much the same results as we found with the simpler formulation; in general, longer lags result in greater instability in the system.

All of the models discussed in this paper assume genetic constancy, at least on the average, in the populations modelled. Thus, the various coefficients are considered as constants over the length of the experiment. Population growth forms that involve oscillation, either as damped oscillations or limit cycles, are efficient mechanisms for selection, since the populations involved undergo booms and crashes, with a relatively small number of survivors at the bottom of a crash supplying the genetic material for the next boom. In the unchanging environment of the laboratory, an initial population containing enough genetic variability should display an increasingly better adaptation to the laboratory environment, and higher K values, with time. Such a series of damped oscillations about progressively higher equilibrium levels was displayed by a prey-parasite pair studied by Utida (216).

Modelling this kind of genetic change, or more correctly the change in distribution of genotypes, might be attempted, but it is far too complex for most purposes. In still unpublished work I have attempted to approximate the effects of selection on the outcome of competition by means of constraints placed upon the competition. Each of the coefficients, r_x, r_y, K_x, K_y, τ_x, τ_y, α and β, was represented not by a single value but by a distribution of values. The choice of starting coefficients was determined with the use of a random-number generator. Once the competition had started, the population was held to its starting coefficients as long as it increased in number. When it began to decrease, it was issued a new batch of coefficients (again chosen from the distributions by means of a random-number generator). The frequency with which new choices were made was a function of population size; the smaller the population, the more frequent the choices. If the population began to increase, it was held to the coefficients associated with the increase. Under these rules, populations were sometimes eliminated very rapidly by a bad choice of starting coefficients. Occasionally, both populations would be maintained, with frequent changes in the direction of competition, for the duration of my computer run. While

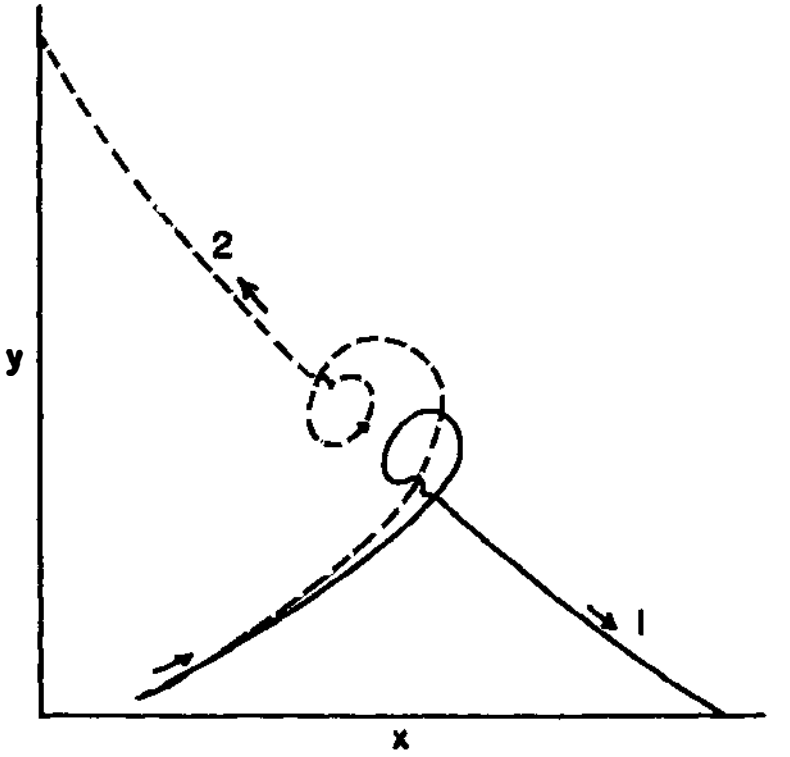

Figure 4 Competition between two species, with the outcome determined by the size of the time delays. Curve 1: $\tau_x = 2$; $\tau_y = 5$. Curve 2: $\tau_x = 2$; $\tau_y = 6$ [After Wangersky and Cunningham (232)].

the ultimate direction of competition could be decided by an especially fortunate choice of starting conditions, often the result was undetermined until late in the competition. In a sense, these models mimic the behavior of the indeterminate competitions between *Tribolium* species discussed by Park (159).

MULTI-SPECIES POPULATION MODELS

Extension of the Lotka-Volterra models to multi-species communities has long been a goal of ecological modellers. Both Lotka and Volterra explored this area of modelling to some extent. As in the other areas of research mentioned in this paper, the multi-species problem has been approached both experimentally and theoretically. The papers by Addiscott (1) and Maly (130) are typical of the experimental approaches. Both are concerned with the interactions of multiple prey species with a single predator species, a topic of considerable interest to the theoreticians. Addiscott (1) found that the presence of predators did not permit greater diversity in the protozoan community in pitcher plants, as would have been predicted by theory. He felt that the departure from theory occurred because the prey organisms were not competing for the same resource, and thus competition was not regulating population numbers. Maly (130), working with a rotifer predator and two prey species, a *Paramecium* and a *Euglena,* also found a contradiction between theory and experiment: The addition of a second prey species did not result in increased stability for the system. He felt that this contradiction occurred because the predator could not exist entirely on *Euglena,* and therefore the two prey species were not completely equivalent. In most cases where experiment and theory are compared, it is found that the experimental situation is much more complicated than the model.

There have been many purely theoretical studies of multi-species interactions. The one-predator–two-prey or two-predator–one-prey systems have been investigated by several workers (31, 32, 105, 164). In general, these systems show an increase in stability over either the simple competition or the simple prey-predator systems. The three-trophic-level systems have also been investigated (58, 69, 80, 241), usually with a finding of increasing stability as more components are added to the system. Gilpin (65) has examined competition among three species, finding stable limit cycles as the population growth forms. De Wit (44) and Garfinkel (61) examined the increase in stability resulting from the introduction of density dependence into the model, with De Wit using competition for space as the limiting condition.

More complex situations have been considered by Garfinkel & Sack (62), who modelled a six-species, three-trophic-level system involving three plants, two herbivores, and one carnivore. Their general conclusions were largely those implied by the mathematical form of their model. Kilmer (103) applied boundary conditions determined by biological constraints to his multi-species model. Very complex mathematical analyses were applied to multi-species systems by Huang & Morowitz (90), Kerner (102), and Trubatch & Franco (209)—techniques I feel to be far more sophisticated than the initial assumptions warrant. Neill (147) and Patten (166) take opposite sides of the argument on linearization of the equations used in their models;

Neill argues that the competition coefficients should all be nonlinear, and demonstrates nonadditivity effects by eliminating one or two species from a four species competition model; Patten argues that since the nonlinear models constructed by the investigators in the International Biological Program were unstable, perhaps evolution aims at the linearization of all systems.

The sensitivity of ecological systems to sudden changes in the environment has been investigated experimentally by Storer & Gaudy (196) and theoretically by Goh (68). Storer & Gaudy grew sewage organisms to equilibrium in a chemostat, then dumped in three times the normal nutrient load. This produced damped oscillations in the population growth rates. Goh attempted to set some stability limitations on complex systems in the face of disturbances, using the Lyapunov number as a criterion of stability.

These models, like most of the simpler models, assume an essentially Markovian universe; only the present state of the system is important, and the history of the system, the route by which the system arrived at that state, can be disregarded in any prediction of future states. Yet if any of the populations involved exhibits either damped oscillations or limit cycles as its population growth form, this assumption is obviously untrue. In order to predict the state of that population in the next time interval it is necessary to know both population size and direction. When the growth form of population is a limit cycle, it is important to know where in that cycle the population is; the distribution of probable futures for a growing population is different from that for one that is crashing. Even with a population that apparently follows a logistic growth form, the necessary delay between cause and effect must be taken into account in prediction. In a system composed of several interacting populations with delays of varying lengths, predictions cannot be made simply by inspection, and intuition can be wrong as often as it is right.

Multi-species models incorporating delays have been constructed by several investigators (11, 108, 112, 134). In general, their results suggest that delays lead to increased instability in the strongly linked systems. Ladde (108), using a Lyapunov-function approach, found that stability could be increased by building density dependence into the several species. Much more work must be done with multi-species models that incorporate delays. We need to determine whether the instabilities seen in the models are common in natural communities; if they are not, we must discover how natural communities differ from the models. Our experiences with natural communities suggest that both stability and instability are to be expected, sometimes in the same community. Some species, particularly those following an *r*-strategy, fluctuate violently, while other, closely related species seem stable. It may be that interaction coefficients between these two types of populations must be low if a stable community is to result. Limits to the degree of interaction might be set by investigations into the properties of such models. It has been suggested (131) that communities evolve from species with many offspring and great dispersal power, following an *r*-strategy, to tight breeding communities with little dispersal and complex structure. I feel some species with high reproductive potential invariably exist to take advantage of the good years and occasional especially favorable circumstances.

CONCLUSIONS

Complex and closely fitted growth curves for populations of a single species can certainly be devised, based on mechanisms of density-dependent control of population size. They may even be useful in predicting the outcome of specific experiments, in a manner impossible with the more general equations. For most purposes, there is little reason to use models more complicated than the time-lag version of the logistic equation; however, the simple logistic, without time lag, is too restricted in the range of populations it will describe. Models incorporating some degree of genetic change should be investigated.

The presence of population oscillations cannot be taken as proof of the existence of prey-predator interactions, since there is both experimental evidence and theoretical justification for damped oscillations and limit cycles in populations without predation. Conversely, the presence of a prey-predator pair does not ensure that any fluctuations are due to the Lotka-Volterra type of prey-predator cycling. Prey–predator interaction models incorporating time lags can produce noncyclical population growth forms when growth rates or delay terms are small. Cyclical growth patterns in the predator may be simply responses to naturally fluctuating food supplies.

Competition models exhibiting most of the features found in laboratory competitions, including a certain degree of indeterminacy of outcome, can be constructed if delays are incorporated into the models. The restriction of these models to two species is probably excessively artificial. Multi-species models incorporating delays would seem to be the most profitable direction for future research. The small amount of work of this type done so far has produced results that are not always intuitively obvious; further work may permit the general outlines of a theory for multi-species interactions to emerge.

The mathematical apparatus brought to bear on the problems of growth and interaction has often been unjustifiably complex, given the unreality of many of the underlying assumptions. The most complex mathematics usually requires that there be no historical effects in the populations modelled, a circumstance that can never occur in nature. It is worth getting complicated in the analysis if we can also get more realistic in our assumptions; if we simply extend the original models beyond their sensible limits, our results may be mathematically sound and biologically irrelevant.

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