6 Applications of Continuous Models to Population Dynamics

Each organic being is striving to increase in a geometrical ratio . . . each at some period of its life, during some season of the year, during each generation or at intervals has to struggle for life and to suffer great destruction. . . . The vigorous, the healthy, and the happy survive and multiply.

Charles R. Darwin. (1860). *On the Origin of Species by Means of Natural Selection*, D. Appleton and Company, New York, chap. 3.

The growth and decline of populations in nature and the struggle of species to predominate over one another has been a subject of interest dating back through the ages. Applications of simple mathematical concepts to such phenomena were noted centuries ago. Among the founders of mathematical population models were Malthus (1798), Verhulst (1838), Pearl and Reed (1908), and then Lotka and Volterra, whose works were published primarily in the 1920s and 1930s.

The work of Lotka and Volterra, who arrived independently at several models including those for predator-prey interactions and two-species competition, had a profound effect on the field now known as *population biology*. They were among the first to study the phenomena of interacting species by making a number of simplifying assumptions that led to nontrivial but tractable mathematical problems. Since their pioneering work, many other notable contributions were made. Among these is the work of Kermack and McKendrick (1927), who addressed the problem of outbreaks of epidemics in a population.

Today, students of ecology and population biology are commonly taught such classical models as part of their regular biology curriculum. Critics of these historical models often argue that certain biological features, such as environmental effects, are ignored. However, the importance of these models stems not from realism or the accuracy of their predictions but rather from the simple and fundamental principles that they set forth; the propensity of predator-prey systems to oscillate, the tendency of competing species to exclude one another, the threshold dependence of epidemics on population size are examples.

While appreciation of the Lotka-Volterra models in the biological community is mixed, it is nevertheless interesting to note that in subtle yet important ways they have helped to shape certain research directions in current biology. As demonstrated by the Nicholson-Bailey model of Chapter 3, a model does not have to be accurate to serve as a helpful diagnostic tool. We shall later discuss more specific ways in which the unrealistic predictions of simple models have led to new empirical as well as theoretical progress.

The classic population biology models serve several purposes in this text. Aside from being interesting in their own right, models of two interacting species or of epidemics in a fixed population are ideal illustrations of the techniques and concepts outlined in Chapters 4 and 5. The models also demonstrate how the predictions of a model change when slight alterations are made in the equations or in values of the critical quantities that appear in them. Finally, the fact that these models are fairly simple allows us to assess critically the various assumptions and their consequences.

As in previous discussions, we set the stage by a brief discussion of models for single-species populations. (Section 4.1 introduced this topic; here we somewhat broaden the context.) In Sections 6.2 and 6.3 the Lotka-Volterra predator-prey and species competition models are described and then analyzed. The story of Volterra's initiation to this biological area is well known. This Italian mathematician became interested in the area of population biology through conversations with a colleague, U. d'Ancona, who had observed a puzzling biological trend. During World War I, commercial fishing in the Adriatic Sea fell to rather low levels. It was anticipated that this would cause a rise in the availability of fish for harvest. Instead, the population of commercially valuable fish declined on average while the number of sharks, which are their predators, increased. The two populations were also perceived to fluctuate.

Volterra suggested a somewhat naive model to describe the predator-prey interactions in the fish populations and was thereby able to explain the trends d'Ancona had observed. As we shall see, the model's basic prediction is that predators tend to overrespond to increases in the population of their prey. This can give rise to oscillations in the populations of both species.

Because natural communities are composed of numerous interacting species no two of which can be entirely isolated from the rest, theoretical tools for dealing with larger systems are often required. The Routh-Hurwitz criteria and the methods of qualitative stability are thus briefly outlined in Sections 6.4 and 6.6. For rapid coverage of this chapter, these sections may be omitted without loss of continuity. In Sections 6.6 and 6.7 we study models for the spread of an epidemic in a population and then explore certain consequences of the policy of vaccinating against disease-causing agents.

Since the scope of this material is vast, a thorough documentation of sources is

impossible. There are numerous recent reviews (for example, May, 1973). An excellent companion to this chapter is Van der Vaart (1983), which contains historical, biological, and mathematical details on certain topics and which uses an instructive and guided approach. [See also Braun (1979, 1983).] All of these sources have been used repeatedly in putting together the material for this chapter.

6.1 MODELS FOR SINGLE-SPECIES POPULATIONS

Two examples of ODEs modeling continuous single-species populations have already been encountered and analyzed in Section 4.1. To summarize, these are

1. *Exponential growth (Malthus, 1798):*

$$\frac{dN}{dt} = rN, \tag{1a}$$

$$\text{Solution:} \quad N(t) = N_0 e^{rt}. \tag{1b}$$

2. *Logistic growth (Verhulst, 1838):*

$$\frac{dN}{dt} = r\left(1 - \frac{N}{K}\right)N. \tag{2a}$$

$$\text{Solution:} \quad N(t) = \frac{N_0 K}{N_0 + (K - N_0)e^{-rt}}. \tag{2b}$$

$N_0 = N(0)$ = the initial population. (See Figure 6.1.)

To place both of the above into a somewhat broader context we proceed from a more general assumption, namely that for an isolated population (no migration) the rate of growth depends on population density. Therefore

$$\frac{dN}{dt} = f(N). \tag{3}$$

This approach is based on an instructive summary by Lamberson and Biles (1981), which should be consulted for further details.

Observe that for equations (1a) and (2a) the function f is the polynomial

$$f(N) = a_0 + a_1 N + a_2 N^2$$

where $a_0 = 0$; for equation (1a) $a_1 = r$ and $a_2 = 0$; for equation (2a) $a_1 = r$ and $a_2 = -r/K$. More generally, it is possible to write an infinite power (Taylor) series for f if it is sufficiently smooth:

$$f(N) = \sum_{n=0}^{\infty} a_n N^n = a_0 + a_1 N + a_2 N^2 + a_3 N^3 + \cdots.$$

Thus any growth function may be written as a (possibly infinite) polynomial (see Lamberson and Biles, 1981).

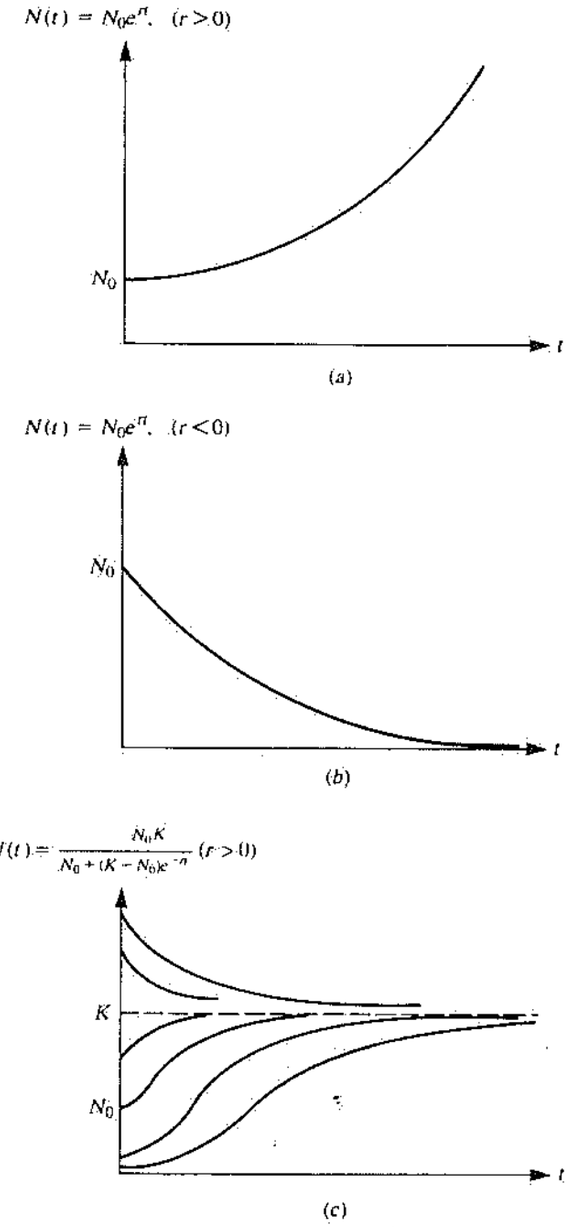

Figure 6.1 Changes in population size N(t) predicted by two models for single-species growth: Exponential growth with (a) r > 0, (b) r < 0, and (c) logistic growth. See equations (1a) and (2a).

About (3) we require that $f(0) = 0$ to dismiss the possibility of *spontaneous generation*, the production of living organisms from inanimate matter. (See also Hutchinson, 1978 for this *Axiom of Parenthood:* every organism must have parents.) In any growth law this is equivalent to

$$\left.\frac{dN}{dt}\right|_{N=0} = f(0) = 0,$$

so that we may assume that

$$\begin{aligned} a_0 &= 0, \\ \frac{dN}{dt} &= a_1 N + a_2 N^2 + a_3 N^3 + \cdots \\ &= N(a_1 + a_2 N + a_3 N^2 + \cdots), \\ &= N g(N). \end{aligned} \tag{4}$$

The polynomial $g(N)$ is called the *intrinsic growth rate* of the population.

Now we examine more closely several specific growth models, including those given in equations (1a) and (2a).

Malthus Model

This can be viewed as the simplest form of equation (4) in which the coefficients of $g(N)$ are $a_1 = r$ and $a_2 = a_3 = \cdots = 0$. As noted before, this model predicts exponential growth if $r > 0$ and exponential decline if $r < 0$.

Logistic Growth

To correct the prediction that a population can grow indefinitely at an exponential rate, consider a nonconstant intrinsic growth rate $g(N)$. The logistic growth model is perhaps the simplest extension of equation (1a). It can be explained by any of the following comments.

Formal mathematical justification

Equation (2a) makes use of more terms in the (possibly infinite) series for $f(N)$ and is thus more faithful to the true population growth rate.

Density-dependent growth rate

Equation (2a) takes the form of equation (4), where

$$g(N) = r\left(1 - \frac{N}{K}\right).$$

It is essentially the simplest rule in which the intrinsic growth rate g depends on the population density (in a *linear decreasing* relationship). It thus accounts for a decreasing per capita growth rate as population size increases.

Carrying capacity

From equation (2a) we observe that

$$\frac{dN}{dt} = 0 \qquad (N = K).$$

Thus $N = K$ is a steady state of the logistic equation. It is easy to establish that this steady state is stable; note in particular that

$$\frac{dN}{dt} > 0 \qquad (N < K),$$

$$\frac{dN}{dt} < 0 \qquad (N > K).$$

The constant K can represent the carrying capacity of the environment for the species. See also Section 4.1 for a derivation of (2a) based on nutrient consumption.

Intraspecific competition

The fact that individuals compete for food, habitat, and other limited resources means that such an increase in the net population mortality may be observed under crowded conditions. Such effects are most pronounced when there are frequent *encounters* between individuals. Equation (2a) can be written in the form

$$\frac{dN}{dt} = rN - \frac{r}{K}N^2.$$

The second term thus depicts a mortality proportional to the rate of paired encounters.

The solution of equation (2a) given by (2b) can be obtained in a relatively straightforward calculation (see problem 5 of Chapter 4). Aside from Gause's work on yeast cultures (Section 4.1), such models have been applied to a variety of populations including humans (Pearl and Reed, 1920), microorganisms (Slobodkin, 1954), and other species. See Lamberson and Biles (1981) for examples and references.

Allee Effect

A further direct extension of equations (1) and (2) is an assumption of the form

$$g(N) = a_1 + a_2 N + a_3 N^2.$$

Provided $a_2 > 0$, and $a_3 < 0$, one obtains the *Allee effect*, which represents a population that has a maximal intrinsic growth rate at intermediate density. This effect may stem from the difficulty of finding mates at very low densities.

Figure 6.2 is an example of a density-dependent form of $g(N)$ that depicts the

Allee effect. Its general character can be summarized by the inequalities

$$g'(N) > 0 \qquad (N < \eta),$$
$$g'(N) < 0 \qquad (N > \eta),$$

where η is the density for optimal reproduction.

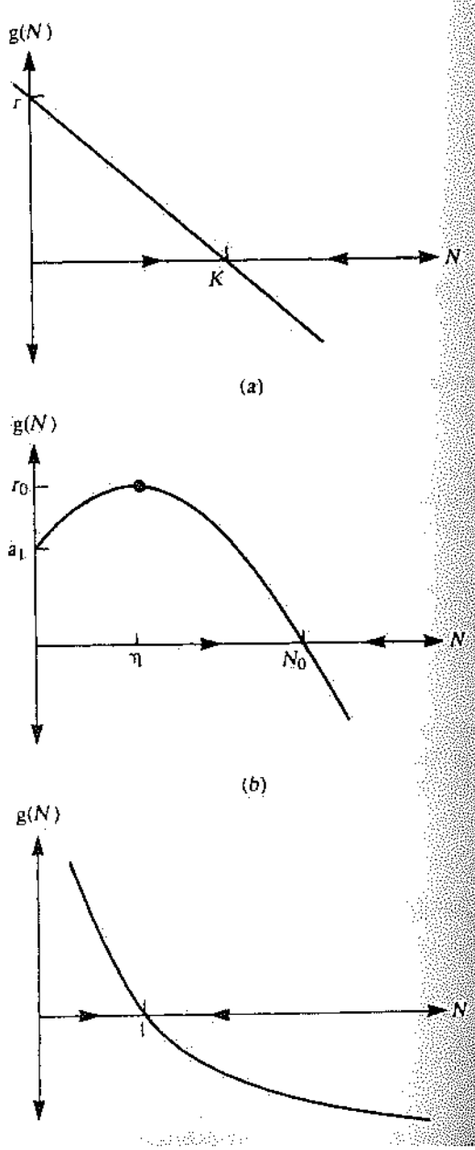

Figure 6.2 A comparison between three types of density-dependent intrinsic growth rates g(N). *(a) Logistic growth decreases linearly with density (or population size). (b) In the Allee effect the rate of reproduction is maximal at intermediate densities. (c) The Gompertz law shows a negative logarithmic*

The simplest example of an Allee effect would be

$$g(N) = r_0 - \alpha(N - \eta)^2, \qquad (\eta < \sqrt{r_0/\alpha}). \tag{5}$$

Notice that this inverted parabola, shown in Figure 6.2*(b)*, intersects the axis at $r_0 - \alpha\eta^2$, has a maximum of r_0 when $N = \eta$, and drops below 0 when

$$N > N_0 = \eta + \sqrt{r_0/\alpha}.$$

Thus for densities above N_0, the population begins to decline. From the curve in Figure 6.2*(a)* we can deduce that $N = N_0$ is a stable equilibrium for the population. (N_0 is an equilibrium point because $g(N_0) = 0$; it is stable because $g'(N_0) < 0$.)

In equation (5) we assumed that $a_1 = r_0 - \alpha\eta^2$, $a_2 = 2\alpha\eta$, and $a_3 = -\alpha$.

Other Assumptions; Gompertz Growth in Tumors

Yet a fourth growth law that frequently appears in models of single-species growth is the *Gompertz law* (introduced in Chapter 4), which is used mainly for depicting the growth of solid tumors. The problems of dealing with a complicated geometry and with the fact that cells in the interior of a tumor may not have ready access to nutrients and oxygen are simplified by assuming that the growth rate declines as the cell mass grows. Three equivalent versions of this growth rate are as follows:

$$\frac{dN}{dt} = \lambda e^{-\alpha t} N, \tag{6a}$$

$$\frac{dN}{dt} = \gamma N, \qquad \frac{d\gamma}{dt} = -\alpha\gamma, \tag{6b}$$

$$\frac{dN}{dt} = -\kappa N \ln N. \tag{6c}$$

See Figure 6.2*(c)*. In (6c) we can identify the intrinsic growth rate as

$$g(N) = -\kappa \ln N.$$

Since $\ln N$ is undefined at $N = 0$, this relation is not valid for very small populations and cannot be considered a direct extension of any of the previous growth laws. It is, however, a popular model in clinical oncology. (See Braun, 1979, sec. 1.8; Newton, 1980; Aroesty et al., 1973.) Biological interpretations for these equations are discussed in problem 7. Considering their relatively simple form, the predictions of any of the Gompertz equations agree remarkably well with the data for tumor growth. (See Aroesty et al., 1973, or Newton, 1980, for examples.)

A valid remark about most of the models for population growth is that they are at best gross simplifications of true events and often are used simply as an expedient fit to the data. To be more realistic one needs a

more powerful way to deal with age-dependent growth, fecundity, or mortality rates. Equations such as (3) or (6) are frequently used by modelers as a convenient first approach to complicated situations and thus are quite useful provided their limitations are not ignored.

6.2 PREDATOR-PREY SYSTEMS AND THE LOTKA-VOLTERRA EQUATIONS

The fact that predator-prey systems have a tendency to oscillate has been observed for well over a century. The Hudson Bay Company, which traded in animal furs in Canada, kept records dating back to 1840. In these records, oscillations in the populations of lynx and its prey the snowshoe hare are remarkably regular (see Figure 6.3).

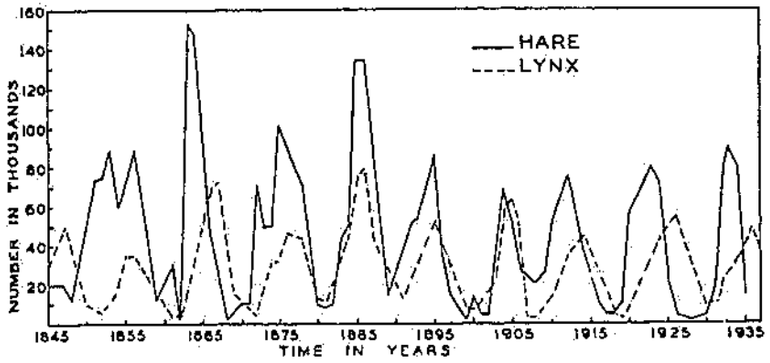

Figure 6.3 Records dating back to the 1840s kept by the Hudson Bay Company. Their trade in pelts of the snowshoe hare and its predator the lynx reveals that the relative abundance of the two species undergoes dramatic cycles. The period of these cycles is roughly 10 years. [From E. P. Odum (1953), fig. 39.]

In this section we explore a model for predator-prey interactions that Volterra proposed to explain oscillations in fish populations in the Mediterranean. To reconstruct his line of reasoning and arrive at the equations independently, let us list some of the simplifying assumptions he made:

1. Prey grow in an unlimited way when predators do not keep them under control.
2. Predators depend on the presence of their prey to survive.
3. The rate of predation depends on the likelihood that a victim is encountered by a predator.
4. The growth rate of the predator population is proportional to food intake (rate of predation).

Taking the simplest set of equations consistent with these assumptions, Volterra wrote down the following model:

$$\frac{dx}{dt} = ax - bxy, \tag{7a}$$

$$\frac{dy}{dt} = -cy + dxy, \tag{7b}$$

where x and y represent prey and predator populations respectively; the variables can represent, for example, biomass or population densities of the species. To acquaint ourselves with this model we proceed by answering several questions. First let us consider the meaning of parameters a, b, c, and d and of each of the four terms on the RHS of the equations.

The net growth rate a of the prey population when predators are absent is a positive quantity (with dimensions of 1/time) in accordance with assumption 1. The net death rate c of the predators in the absence of prey follows from assumption 2. The term xy approximates the likelihood that an encounter will take place between predators and prey given that both species move about randomly and are uniformly distributed over their habitat.

The form of this encounter rate is derived from the *law of mass action* that, in its original context, states that the rate of molecular collisions of two chemical species in a dilute gas or solution is proportional to the product of the two concentrations (see Chapter 7). We should bear in mind that this simple relationship may be inaccurate in describing the subtle interactions and motion of organisms. An encounter is assumed to decrease the prey population and increase the predator population by contributing to their growth. The ratio b/d is analogous to the efficiency of predation, that is, the efficiency of converting a unit of prey into a unit of predator mass.

Further practice in linear stability techniques given in Chapter 5 can be revealing:

It is clear that two possible steady states of equation (7) exist:

$$(\bar{x}_1, \bar{y}_1) = (0, 0) \quad \text{and} \quad (\bar{x}_2, \bar{y}_2) = \left(\frac{c}{d}, \frac{a}{b}\right).$$

Their stability properties are determined by the methods given in Chapter 5.

The Jacobian of this system is

$$\mathbf{J} = \begin{pmatrix} a - by & -bx \\ dy & dx - c \end{pmatrix}_{(\bar{x},\bar{y})}$$

for steady state 1

$$\mathbf{J} = \begin{pmatrix} a & 0 \\ 0 & -c \end{pmatrix},$$

for steady state 2

$$\mathbf{J} = \begin{pmatrix} 0 & \frac{-bc}{d} \\ \frac{da}{b} & 0 \end{pmatrix},$$

eigenvalues are

$\lambda_1 = a, \qquad \lambda_2 = -c. \qquad \lambda_{1,2} = \pm\sqrt{ca}\,i.$

Thus $(\bar{x}_1, \bar{y}_1)$ is a saddle. Thus $(\bar{x}_2, \bar{y}_2)$ is a center[1].

From the analysis of this model we arrive at a number of somewhat counterintuitive results. First, notice that the steady-state level of prey is independent of its own growth rate or mortality; rather, it depends on parameters associated with the predator ($x_2 = c/d$). A similar result holds for steady-state levels of the predator ($y_2 = a/b$). It is the particular *coupling* of the variables that leads to this effect. To paraphrase, the presence of predator ($y \neq 0$) means that the available prey has to just suffice to make growth rate due to predation, dx, equal predator mortality c for a steady predator population to persist. Similarly, when prey are present ($x \neq 0$), predators can only keep them under control when prey growth rate a and mortality due to predation, by, are equal. This helps us to understand the steady-state equations.

A second result (see problem 10) is that the steady state $(\bar{x}_2, \bar{y}_2)$ is neutrally stable (a center). The eigenvalues of $\mathbf{J}(\bar{x}_2, \bar{y}_2)$ are pure imaginary and the steady state is not a spiral point. See problem 10. Note that the off-diagonal terms, $-bc/d$ and da/b, are of opposite sign (since the influence of each species on the other is opposite) and that the diagonal terms evaluated at $(\bar{x}_2, \bar{y}_2)$ are *zero*. Stability analysis predicts oscillations about the steady state $(\bar{x}_2, \bar{y}_2)$. The factor $\sqrt{(ca)}$ governs the frequency of these oscillations, so that larger prey reproduction or predator mortality (which means a greater turnover rate) result in more rapid cycles. A complete phase-plane diagram of the predator-prey system (7) can be arrived at with minimal further work. See Figure 6.4(*a*).

To gain deeper understanding of the neutral stability of system (7) we will examine a slight variant in which prey populations have the property of self-regulation. Assuming logistic prey growth, equations (7a, b) become

$$\frac{dx}{dt} = \frac{ax\,(K - x)}{K} - bxy, \tag{8a}$$

$$\frac{dy}{dt} = -cy + dxy. \tag{8b}$$

This leads to steady-state values

$$(\bar{x}_2, \bar{y}_2) = \left(\frac{c}{d}, \frac{a}{b} - \frac{ca}{dbK}\right),$$

1. To be more accurate we must include the possibility that this steady state could be a spiral point since the system is nonlinear. (See Section 5.10 for comments.) In problem 10 we will demonstrate that this option can be dismissed for the predator-prey equations.

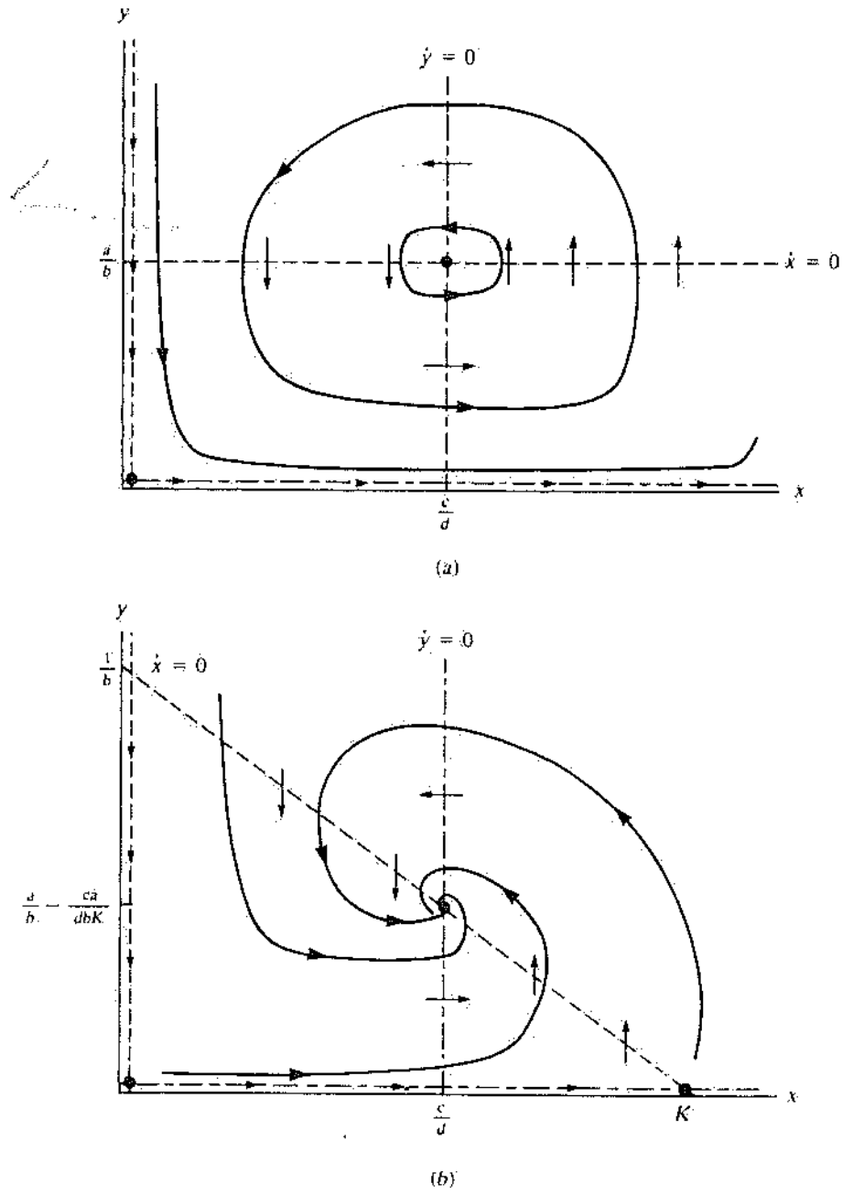

Figure 6.4 (a) The Lotka-Volterra equations (7a,b) predict neutral stability at the steady state (c/d, b/a). (b) When the prey grow logistically [as in equation (8a)], the steady state becomes a stable spiral with somewhat depressed predator population levels.

and the Jacobian is then

$$\mathbf{J} = \begin{pmatrix} \frac{-ac}{dK} & \frac{-bc}{d} \\ d\left(\frac{a}{b} - \frac{ca}{dbK}\right) & 0 \end{pmatrix}.$$

(The condition $1 > c/dK$ must be satisfied so that the steady-state predator level y is positive.) Now $\operatorname{Tr} \mathbf{J} = -ac/dK$ is always negative, and $\det \mathbf{J} = bc\bar{y}_2$ is positive, so that the steady state is always stable. In other words, its neutral stability has been lost. In problem 14 you are asked to investigate whether oscillations accompany the return to steady state after a perturbation.

The lesson to be learned from this example is that a relatively minor change in equations (7a, b) has a major influence on the predictions. In particular, this means that neutral stability, and thus also the oscillations that accompany a neutrally stable steady state, tend to be somewhat ephemeral. This is a serious criticism of the realism of the Lotka-Volterra model.

Taking a somewhat more philosophical approach, we could argue that the Lotka-Volterra model serves a useful purpose precisely because it is so delicately balanced between stability and instability. We could use this model together with minor variants to test out a set of assumptions and so identify stabilizing and destabilizing influences. Following are some of the frequently suggested alterations. It is a relatively easy task to understand what effects such changes have on the stability of the equilibrium. More theoretical results on stable cycles due to Kolmogorov (1936) and others (briefly mentioned below) are recommended for further independent exploration and will be discussed in detail in Chapter 8.

For Further Study

Stable cycles in predator-prey systems

The main objection to the Lotka-Volterra model is that its cycles are only neutrally stable. What additional features are necessary to yield stable oscillations? As we shall see in Chapter 8, stable oscillations (usually called limit cycles) are closed trajectories that attract nearby flow in the phase plane. Kolmogorov (1936) investigated conditions on the general predator-prey system

$$\frac{dx}{dt} = xf(x, y),$$

$$\frac{dy}{dt} = yg(x, y),$$

that would lead to such solutions. The functions f and g are assumed to satisfy several relations consistent with the nature of predator-prey systems:

$$\partial f/\partial x < 0 \text{ (for large } x), \qquad \partial g/\partial x > 0,$$
$$\partial f/\partial y < 0, \qquad \partial g/\partial y < 0.$$

An interpretation of these is left as an exercise. Additional conditions (for example, Coleman, 1978; May 1973) are equivalent to the nullcline geometry shown in Figure 6.5 (Rosenzweig, 1969). It can be proved that when the steady state S is unstable, any trajectory winding out of its vicinity approaches a stable limit cycle that is trapped somewhere inside the rectangular region. See Chapter 8 for further details.

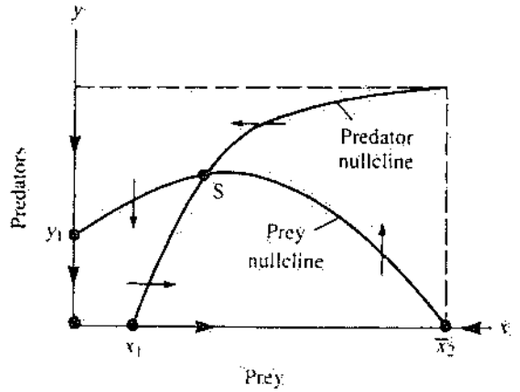

Figure 6.5 With a set of conditions given by Kolmogorov (1936), the phase plane for a predator-prey system has a nullcline geometry that gives rise to stable (limit cycle) oscillations. See Chapter 8.

Other modifications of Volterra's equations

Other assumptions which have been made over the years to modify Volterra's equations are listed below. Details about their effect can be found in May (1973) and in the references as follows.

1. *Density dependence:* More realistic prey growth-rate assumptions in which a is replaced by a density-dependent function f:

$$f(x) = r\left(1 - \frac{x}{K}\right) \qquad \text{Pielou (1969, pp. 19–21).}$$

$$f(x) = r\left[\left(\frac{K}{x}\right)^{g} - 1\right] \qquad (1 \geq g > 0) \qquad \text{Rosenzweig (1971).}$$

$$f(x) = r\left(\frac{K}{x} - 1\right) \qquad \text{Schoener (1973).}$$

2. *Attack rate:* More realistic rates of predation where the term bxy is replaced by a term in which the attack capacity of predators is a limited one. Terms replacing bxy in equation (7a) are:

$$ky(1 - e^{-\alpha x}) \qquad \text{Ivlev (1961).}$$

$$\frac{kxy}{x + D} \qquad \text{Holling (1965).}$$

$$kyx^{g} \qquad (1 \geq g > 0) \qquad \text{Rosenzweig (1971).}$$

$$\frac{kyx^2}{x^2 + D^2} \qquad \text{Takahashi (1964).}$$

6.3 POPULATIONS IN COMPETITION

When two or more species live in proximity and share the same basic requirements, they usually compete for resources, habitat, or territory. Sometimes only the strongest prevails, driving the weaker competitor to extinction. (This is the *principle of competitive exclusion*, a longstanding concept in population biology.) One species wins because its members are more efficient at finding or exploiting resources, which leads to an increase in population. Indirectly this means that a population of competitors finds less of the same resources and cannot grow at its maximal capacity.

In the following model, proposed by Lotka and Volterra and later studied empirically by Gause (1934), the competition between two species is depicted without direct reference to the resources they share. Rather, it is assumed that the presence of each population leads to a depression of its competitor's growth rate. We first give the equations and then examine their meanings and predictions systematically. See also Braun (1979, sec. 4.10) and Pielou (1969, sec. 5.2) for further discussion of this model.

The Lotka-Volterra model for species competition is given by the equations

$$\frac{dN_1}{dt} = r_1 N_1 \frac{\kappa_1 - N_1 - \beta_{12} N_2}{\kappa_1}, \tag{9a}$$

$$\frac{dN_2}{dt} = r_2 N_2 \frac{\kappa_2 - N_2 - \beta_{21} N_1}{\kappa_2}, \tag{9b}$$

where N_1 and N_2 are the population densities of species 1 and 2. Again we proceed to understand the equations by addressing several questions:

1. Suppose only species 1 is present. What has been assumed about its growth? What are the meanings of the parameters r_1, κ_1, r_2, and κ_2?
2. What kind of assumption has been made about the effect of competition on the growth rate of each species? What are the parameters β_{12} and β_{21}?

To answer these questions observe the following:

1. In the absence of a competitor ($N_2 = 0$) the first equation reduces to the logistic equation (2a). This means that the population of species 1 will stabilize at the value $N_1 = \kappa_1$ (its carrying capacity), as we have already seen in Section 6.1.
2. The term $\beta_{21} N_2$ in equation (9a) can be thought of as the contribution made by species 2 to a *decline in the growth rate* of species 1. β_{12} is the per capita decline (caused by individuals of species 2 on the population of species 1).

The next step will be to study the behavior of the system of equations. The task will again be divided into a number of steps, including (1) identifying steady states, (2) drawing nullclines, and (3) determining stability properties as necessary in putting together a complete phase-plane representation of equation (9) using the methods given in Chapter 5. (For practice, it is advisable to attempt this independently before continuing to the procedure in the box.)

Nullclines are just all point sets that satisfy one of the following equations:

$$\frac{dN_1}{dt} = 0 \quad \text{or} \quad \frac{dN_2}{dt} = 0.$$

1. From equation (9a) we arrive at the N_1 nullclines:

$$N_1 = 0 \quad \text{and} \quad \kappa_1 - N_1 - \beta_{12} N_2 = 0,$$

2. Whereas equation (9b) leads to the N_2 nullclines:

$$N_2 = 0 \quad \text{and} \quad \kappa_2 - N_2 - \beta_{21} N_1 = 0.$$

To simplify the notation slightly, we shall refer to these lines as L_{1a}, L_{1b}, L_{2a}, and L_{2b} respectively. Notice that L_{1a} and L_{2a} are just the N_2 and N_1 axes respectively, whereas L_{1b} and L_{2b} intersect the axes as follows:

L_{1b} goes through $(0, \kappa_1/\beta_{12})$ and $(\kappa_1, 0)$.
L_{2b} goes through $(0, \kappa_2)$ and $(\kappa_2/\beta_{21}, 0)$.

It follows that the points $(0, 0)$, $(\kappa_1, 0)$, and $(0, \kappa_2)$ are always steady states. These correspond to three distinct situations:

$(0, 0)$ = both species absent,
$(\kappa_1, 0)$ = species 2 absent and species 1 at its carrying capacity κ_1,
$(0, \kappa_2)$ = species 1 absent and species 2 at its carrying capacity.

There is a fourth possible steady-state value that corresponds to coexistence of the two species. (We leave the computation of this steady state as an exercise.)

Proceeding to the second stage, we sketch the nullcline curves on a phase plane. If you have already attempted this independently, you may have hesitated slightly because numerous situations are possible. Figure 6.6 illustrates four distinct possibilities, all of them correct. In order to choose any one of the four cases we must make some assumptions about the relative magnitudes of κ_2 and κ_1/β_{12}, and of κ_1 and κ_2/β_{21}. The cases shown in Figure 6.6 correspond to the following situations:

case 1: $\frac{\kappa_2}{\beta_{21}} > \kappa_1$ and $\kappa_2 > \frac{\kappa_1}{\beta_{12}}$,

case 2: $\kappa_1 > \frac{\kappa_2}{\beta_{21}}$ and $\frac{\kappa_1}{\beta_{12}} > \kappa_2$,

case 3: $\kappa_1 > \frac{\kappa_2}{\beta_{21}}$ and $\kappa_2 > \frac{\kappa_1}{\beta_{12}}$,

case 4: $\frac{\kappa_2}{\beta_{21}} > \kappa_1$ and $\frac{\kappa_1}{\beta_{12}} > \kappa_2$.

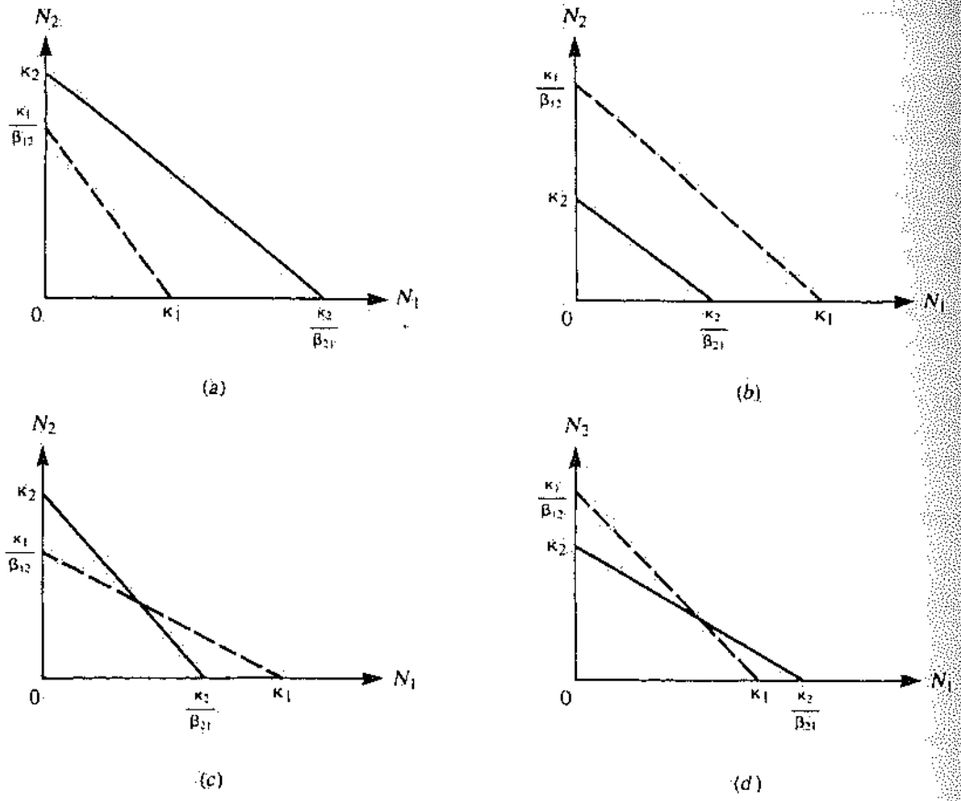

Figure 6.6 Four possible cases corresponding to four choices in the relative positions of nullclines of equations (9): (a) $\kappa_2/\beta_{21} > \kappa_1$ and $\kappa_2 > \kappa_1/\beta_{12}$; (b) $\kappa_1 > \kappa_2/\beta_{21}$ and $\kappa_1/\beta_{12} > \kappa_2$; (c) $\kappa_1 > \kappa_2/\beta_{21}$ and $\kappa_2 > \kappa_1/\beta_{12}$; (d) $\kappa_1/\beta_{12} > \kappa_2$ and $\kappa_2/\beta_{21} > \kappa_1$.

In problem 15 the reader is asked to interpret these inequalities within the biological context of the problem.

Our next step will be to identify the steady states of equations (9a,b) in Figure 6.6($a-d$). By drawing arrows on the nullclines we will also indicate the directions of flow in the N_1N_2 plane for each of the four cases shown. To do this, one can combine geometric reasoning with results of analysis. We recall that steady states are located at the intersections of two nullclines (which must be of opposite types). It helps to remember that L_{1b} (the line $N_1 = 0$) is an N_1 nullcline; it is simply the N_2 axis. Thus the point at which any N_2 nullcline meets the N_2 axis will be a steady state. It is evident that this happens at (0, 0) as well as at (0, κ_2). By similar reasoning we find that (κ_1, 0) is at the intersection of two (opposite type) nullclines. A fourth steady state occurs only when L_{1b} and L_{2b} intersect, as is true in (c) and (d) of Figure 6.6.

To sketch arrows on the N_1 and N_2 nullclines, recall that the directions of flow

on these are parallel to the N_2 and N_1 axes respectively. Arrows have been put in for cases 1 and 4 in Figure 6.7, with case 2 and 3 left as an exercise. Notice that once the flow along the N_1 and N_2 axes is drawn the rest of the picture can be completed by preserving the continuity of flow. (See remarks in Section 5.5.) For a more pedestrian approach, we can use equations (9a,b) to tabulate the directions associated with several points in the plane.

At this stage the problem is practically solved; with the directions of flow determined on the nullclines, we can draw sensible phase-plane pictures in only one distinct way for each case. For example, it should be evident in case 1 that for any starting value of (N_1, N_2) provided $N_2 \neq 0$, the populations eventually converge to the steady state on the N_2 axis. (To see this, notice that there is no other exit from the region bounded by the two slanted lines L_{1b} and L_{2b} in case 1; moreover, all flows pass through this region.) In case 4, any point within the two triangular regions must eventually converge to the steady state at the intersection of L_{1b} and L_{2b}. (What can be said about other regions of the plane in case 4?)

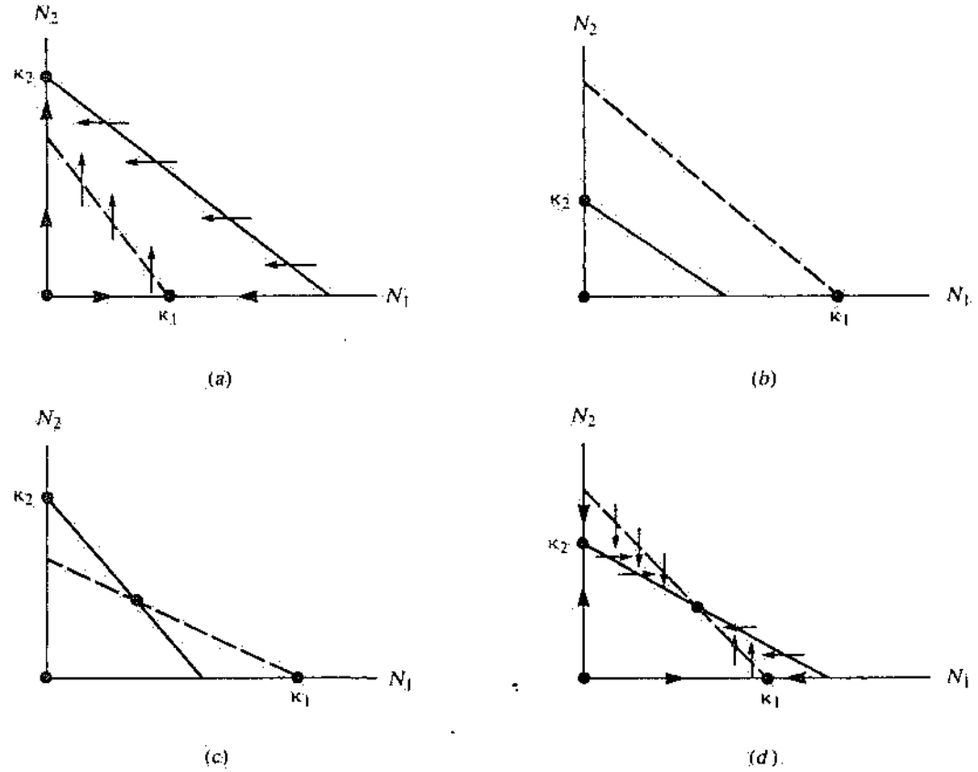

Figure 6.7 Steady states of equation (9) shown as heavy dots at the intersections of the N_1 nullclines (dashed lines) and the N_2 nullclines (solid lines). ($a-d$) correspond to cases 1–4 shown in Figure 6.6 and described in text.

As a somewhat optional final step, we can confirm the conjectured flow by determining what happens close to steady-state values, using the linearization procedures outlined in Chapter 5 [see problem 15(c)]. By carrying out this analysis it can be shown that the outcome of competition is as follows:

case 1: only $(0, \kappa_2)$ is stable,
case 2: only $(\kappa_1, 0)$ is stable,
case 3: both $(0, \kappa_1)$ and $(0, \kappa_2)$ are stable,
case 4: only the steady state given by the expression in problem 15(b) is stable.

With the combined information above, the qualitative pictures in Figure 6.8 can be confirmed, and the mathematical steps in understanding the model are complete. It is now necessary to make a biological interpretation of the result. Part of this is left as a problem for the reader. A rather clear prediction is that in three out of the four cases, competition will lead to extinction of one species. Only in case 4 does the interaction result in coexistence, and then at population levels *below* the normal carrying capacities.

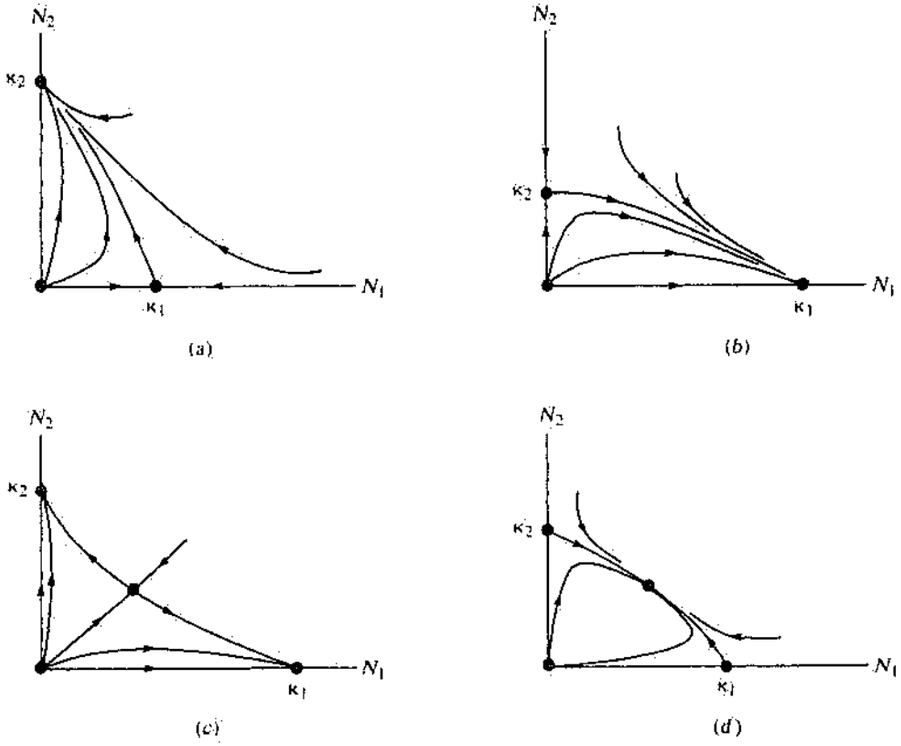

***Figure 6.8** The final phase-plane behavior of solutions to equations (9a,b). (a–d) correspond to cases 1–4 in Figure 6.6. See text for details.*

A small change in the format of the inequalities for cases 1 through 4 will reveal how the *intensity of competition*, which is represented by the β parameters, influences the outcome. To make things more transparent, suppose the carrying capacities are equal ($\kappa_1 = \kappa_2$). Conditions 1 to 4 can be written as follows:

1. $\beta_{21} < 1$ and $\beta_{12} > 1$.
2. $\beta_{21} > 1$ and $\beta_{12} < 1$.
3. $\beta_{21} > 1$ and $\beta_{12} > 1$.
4. $\beta_{21} < 1$ and $\beta_{12} < 1$.

From this, observe that in cases 1, 2, and 3, one or both species are aggressive in competing with their adversary (that is, at least one β is large). In case 4, for which coexistence is obtained, β_{21} and β_{12} are both small, indicating that competition is less intense.

An accepted biological fact is that species very similar in habits, size, and/or feeding preferences tend to compete more strongly for resources when confined to the same habitat (Roughgarden, 1979). For example, species of fish that have similar mouth parts and thus seek the same type of food would overlap in their resource utilization and, thus be more aggressive competitors than those that feed differently. With this observation, a prediction of the model is that similar species in the same habitat will not coexist. (This is a popular version of the principle of competitive exclusion.)

Recent research directions in population biology have focused on questions raised by this principle. Because ecosystems frequently consist of many competitors that appear to vie for common resources, the predictions of this simple model have reshaped some preconceptions about coexistence and species interactions. It has become more challenging to discover the numerous ways competitive exclusion can be foiled.

The model ignores spatial distributions of species and variations in both space and time of the significant quantities as well as many other subtle influences (such as the effects of predation on one of the species). This points to numerous possible effects that could come into play in permitting species to live and share a common habitat. In fact, it is now recognized that species are distributed in a patchy way, rather than uniformly partitioning their habitat so that competition tends to diminish somewhat. A time-sharing arrangement with succession of species or seasonal variability can effect a similar result. Other factors include gradual evolution of differing traits (*character displacement*) to minimize competition, and more complex multispecies interactions in which predation mediates competition. Observations of such special cases are abundant in the current biological literature. Sources for additional readings are Whitaker and Levin (1975) and a forthcoming monograph on theoretical ecology by Simon Levin (Cornell University). Chapter 21 of Roughgarden (1979) also makes for good reading on the competition model and its implications.

There are recent extensions of the competition model to handle n species. Luenberger (1979, sec. 9.5) gives an excellent presentation. A good discussion of the principle of competitive exclusion is given in Armstrong and McGehee (1980). A number of other contributors have included T. G. Hallam, T. C. Gard, R. M. May, H. I. Freedman, P. Waltman, and J. Hofbauer.