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, chance random events, and spatial heterogeneity to mention a few, were ig-

nored. 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).

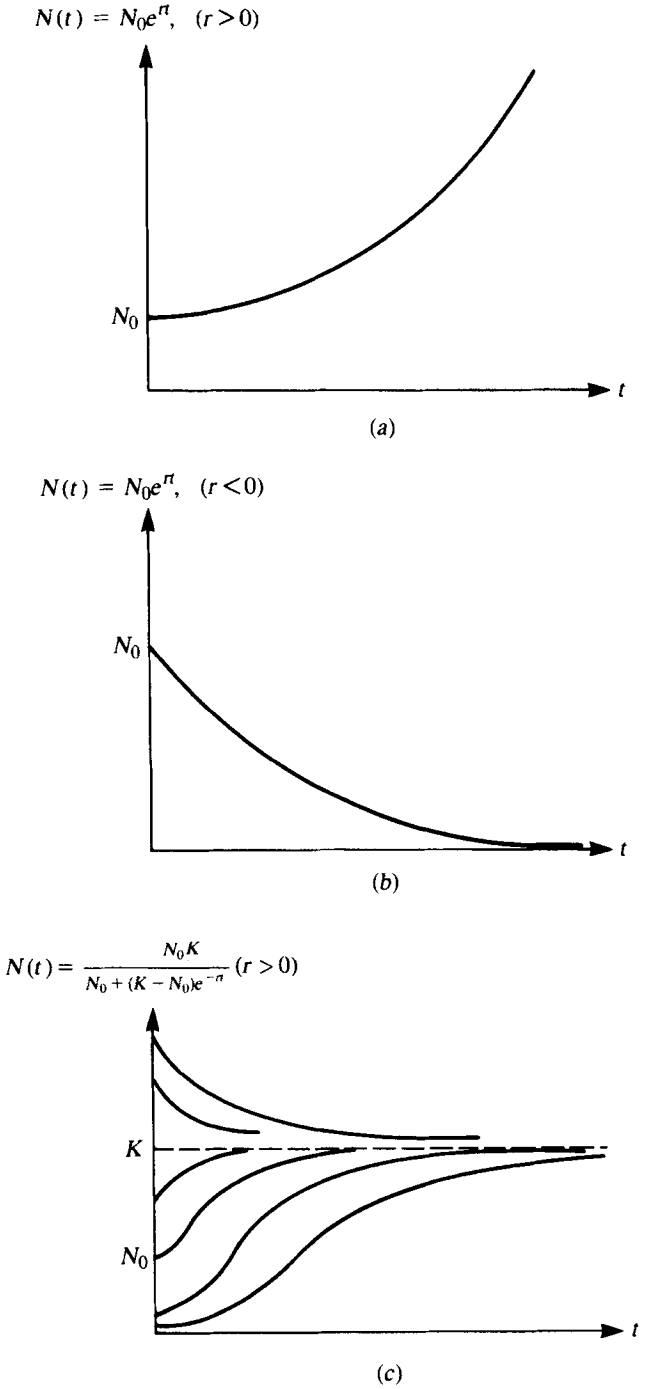


Figure 6.1 *Changes in population size* N(t) *predicted by two models for single-species growth: Exponential growth with (a)* r > 0, *(b)* r < 0, *and (c) logistic growth. See equations (1a) and (2a).*

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

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

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

$$g(N) = a_1 + a_2N + a_3N^2.$$

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.

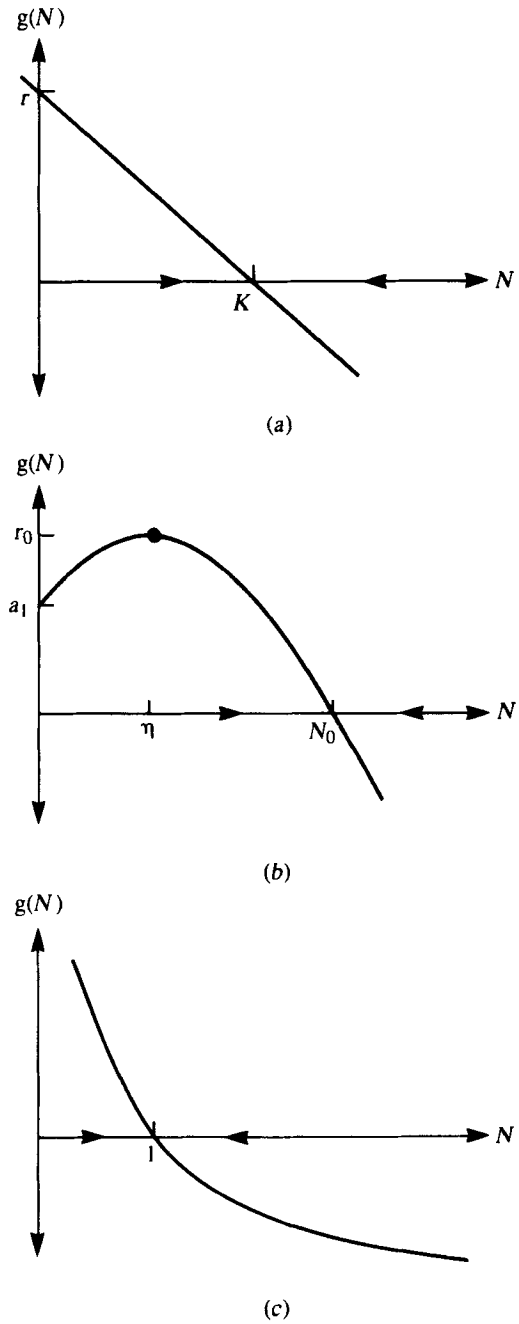


Figure 6.2 *A comparison between three types of density-dependent intrinsic growth rates* g(N). *(a) Logistic growth decreases linearly with density (or population size). (b) In the Allee effect the rate of reproduction is maximal at intermediate densities. (c) The Gompertz law shows a negative logarithmic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

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 greater mathematical sophistication. For example, in Chapter 13 we will see that partial differential equations provide 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).

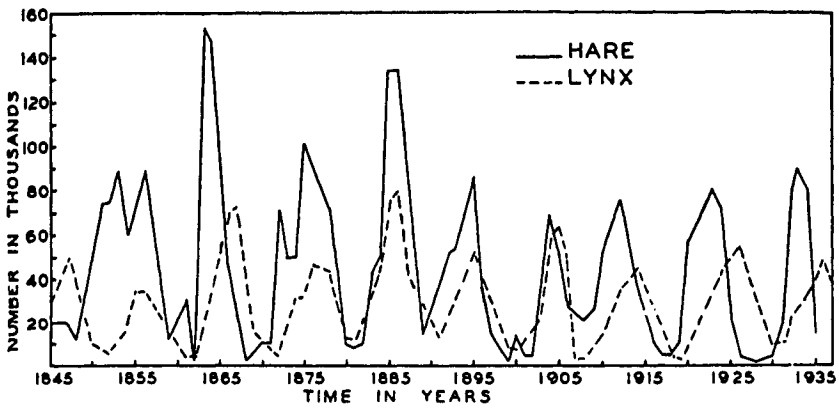


Figure 6.3 *Records dating back to the 1840s kept by the Hudson Bay Company. Their trade in pelts of the snowshoe hare and its predator the lynx reveals that the relative abundance of the two species undergoes dramatic cycles. The period of these cycles is roughly 10 years. [From E. P. Odum (1953), fig.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

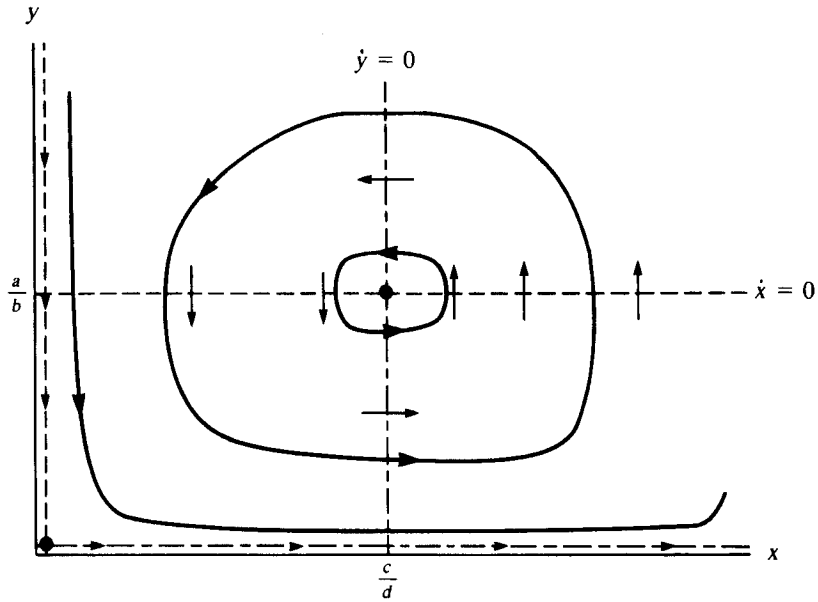
$$\frac{dx}{dt} = \frac{ax\,(K - x)}{K} - bxy, \tag{8a}$$

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

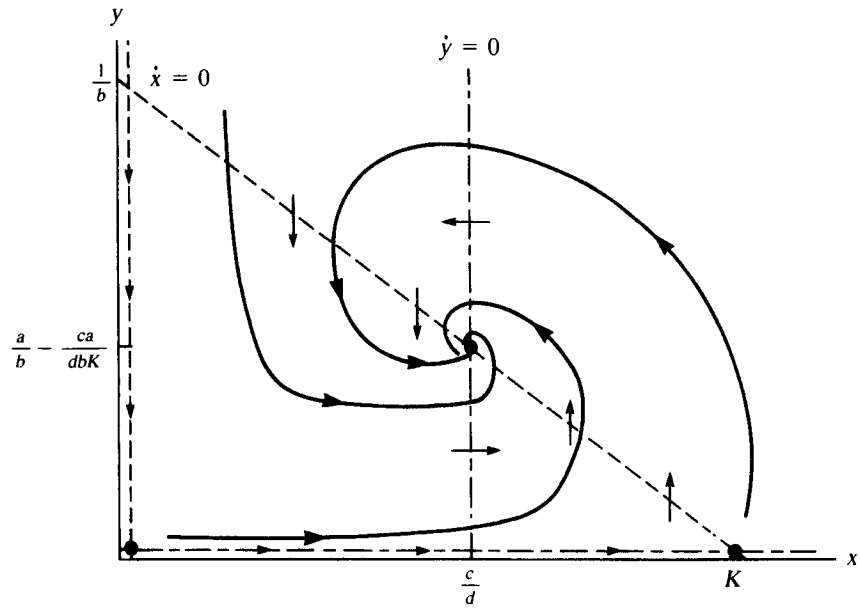
This leads to steady-state values

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

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.



(a)



(b)

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

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

(The condition $1 > c/dK$ must be satisfied so that the steady-state predator level y is positive.) Now $\text{Tr}\,\mathbf{J} = -ac/dK$ 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.

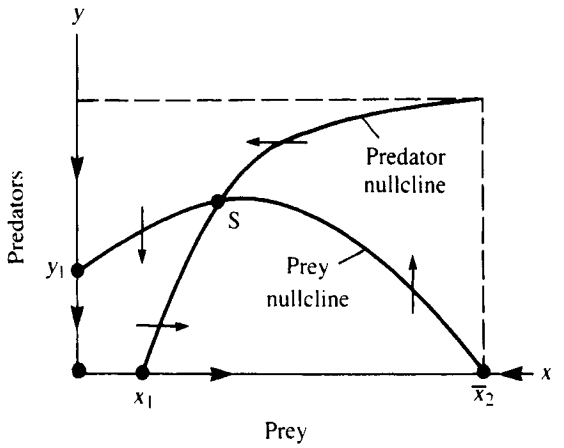


Figure 6.5 *With a set of conditions given by Kolmogorov (1936), the phase plane for a predator-prey system has a nullcline geometry that gives rise to stable (limit cycle) oscillations. See Chapter 8.*

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:

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

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

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

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

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 $(\kappa_2/\beta_{21}, 0)$.

methods given in Chapter 5. (For practice, it is advisable to attempt this independently before continuing to the procedure in the box.)

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:

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

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

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

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

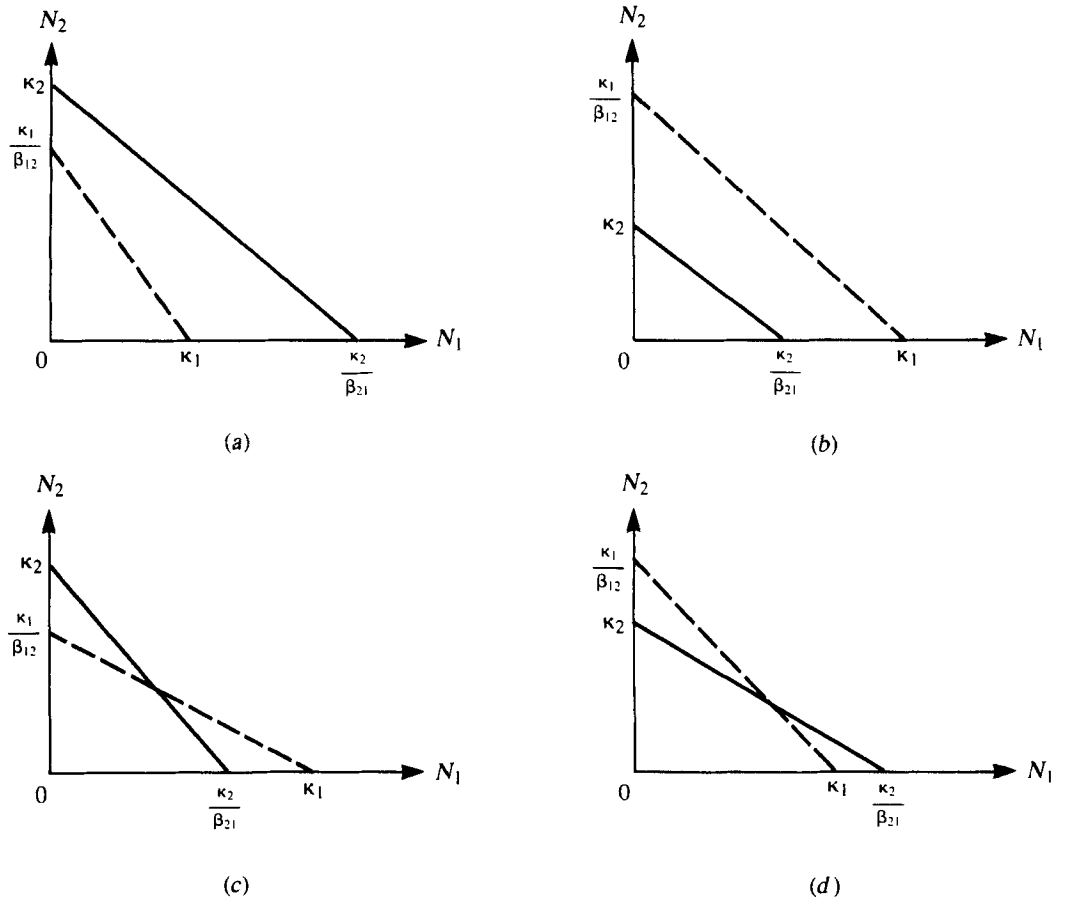


***Figure 6.6** Four possible cases corresponding to four choices in the relative positions of nullclines of equations (9): (a) $\kappa_2/\beta_{21} > \kappa_1$ and $\kappa_2 > \kappa_1/\beta_{12}$; (b) $\kappa_1 > \kappa_2/\beta_{21}$ and $\kappa_1/\beta_{12} > \kappa_2$; (c) $\kappa_1 > \kappa_2/\beta_{21}$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

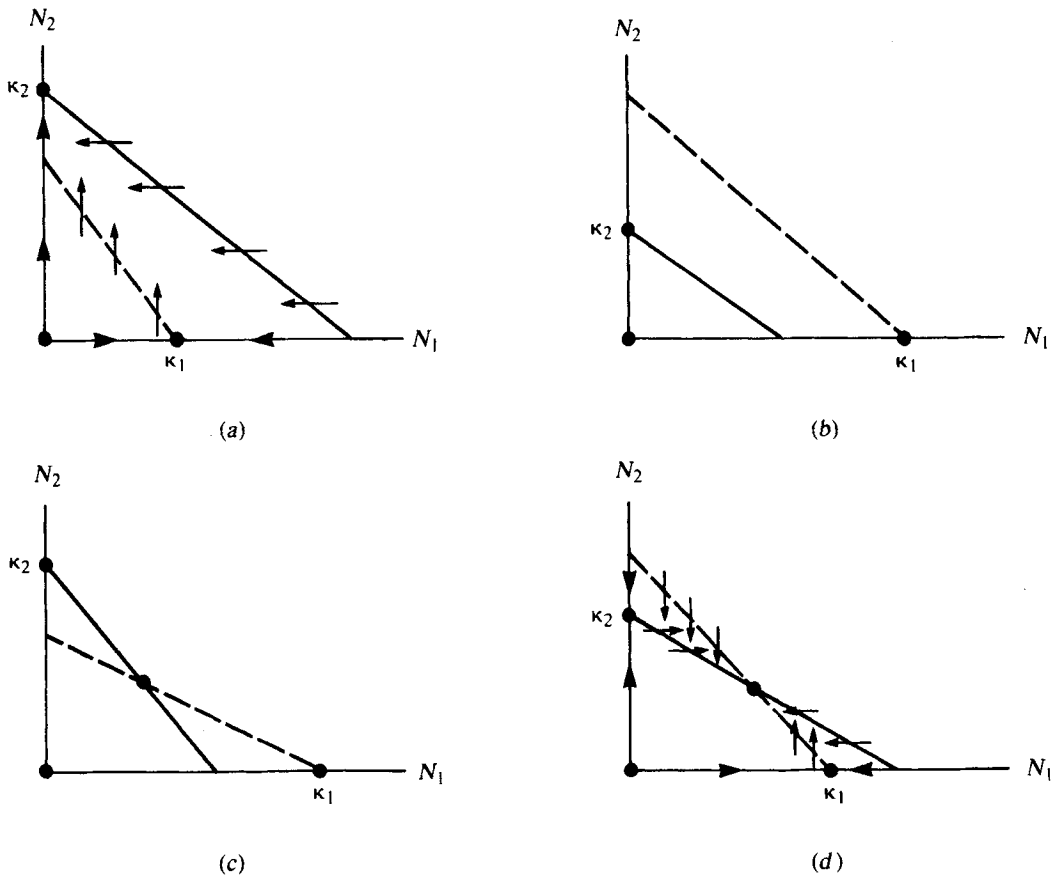


Figure 6.7 *Steady states of equation (9) shown as heavy dots at the intersections of the* N_1 *nullclines (dashed lines) and the* N_2 *nullclines (solid lines). (a–d) correspond to cases 1–4 shown in Figure 6.6 and described in text.*

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.

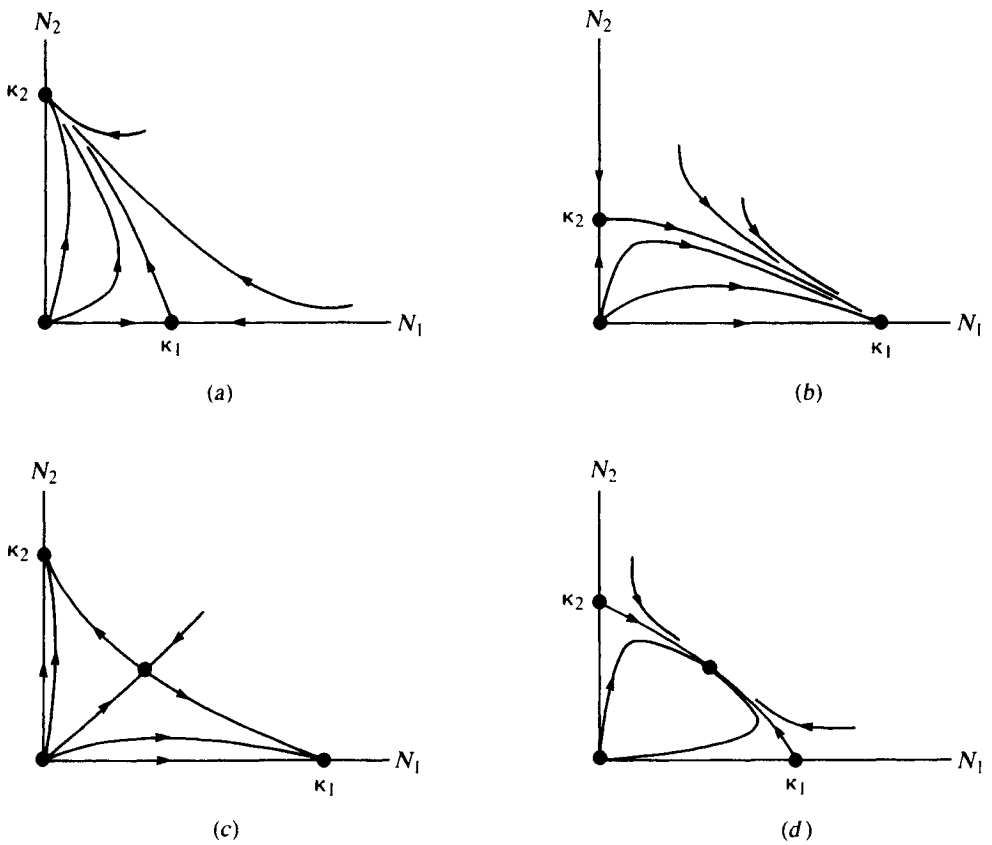


***Figure 6.8** The final phase-plane behavior of solutions to equations (9a,b). (a–d) correspond to cases 1–4 in Figure 6.6. See text for details.*

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 $\beta_{12} < 1$.

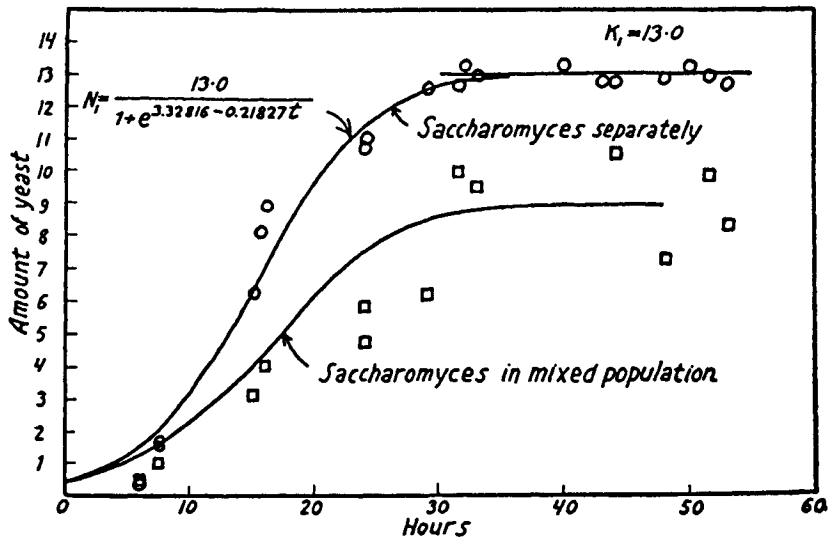
From this, observe that in cases 1, 2, and 3, one or both species are aggressive in competing with their adversary (that is, at least one β is large). In case 4, for which coexistence is obtained, β_{21} and β_{12} are both small, indicating that competition is less intense.

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.)

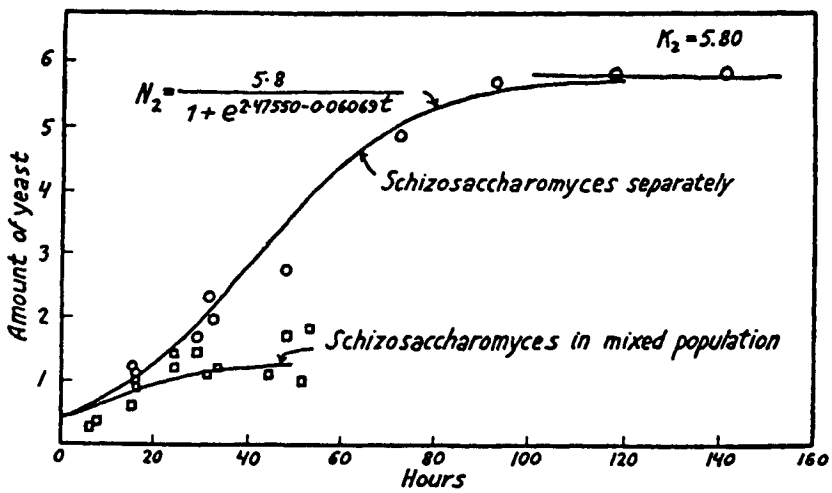
Recent research directions in population biology have focused on questions raised by this principle. Because ecosystems frequently consist of many competitors that appear to vie for common resources, the predictions of this simple model have reshaped some preconceptions about coexistence and species 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.



(*a*)



(*b*)

Figure 6.9 *Growth of (a)* Saccharomyces cervisiae *and (b)* Schizosaccharomyces kephir *in original experiments by Gause (1932). The organisms were grown separately (open circles) as well as in a mixed culture containing both (open rectangles). From Gause, G.F. (1932), Experimental studies on the struggle for existence. I. Mixed population of two species of yeast.* J. Exp. Biol., 9, *Figures 2 and 3.*

Gause: Empirical Tests of the Species-Competition Model

In his book, *The Struggle for Existence,* Gause (1934) describes a series of laboratory experiments in which two yeast species, *Saccharomyces cerevisiae* and *Schizosaccharomyces kephir* were grown separately and then paired in a mixed population (Figure

6.9). Using results shown in Figure 6.9 (*a* and *b*), he was able to estimate the following values for parameters in equation (9):

$$r_1 = 0.21827, \quad K_1 = 13.0, \quad \beta_{12} = 3.15,$$
$$r_2 = 0.06069, \quad K_2 = 5.8, \quad \beta_{21} = 0.439.$$

See problems 33 and 34 for some details and analysis, and Gause for a very readable summary of these and other experiments.

6.4 MULTIPLE-SPECIES COMMUNITIES AND THE ROUTH-HURWITZ CRITERIA

Now that we have spent some time mastering the techniques of linear stability theory, it seems discouraging to realize that the elegance and simplicity of phase-plane methods apply only to two-species systems. In this section we briefly touch on methods for gaining insight into models for k species interacting in a community, where $k > 2$.

The models we have seen thus far take the form

$$\frac{dN_1}{dt} = f(N_1, N_2), \tag{10a}$$

$$\frac{dN_2}{dt} = g(N_1, N_2). \tag{10b}$$

More generally a system comprised of k species with populations $N_1, N_2, \ldots, N_k$ would be governed by k equations:

$$\begin{aligned} \frac{dN_1}{dt} &= f_1(N_1, N_2, \ldots, N_k), \\ \frac{dN_2}{dt} &= f_2(N_1, N_2, \ldots, N_k), \\ &\vdots \\ \frac{dN_k}{dt} &= f_k(N_1, N_2, \ldots, N_k). \end{aligned} \tag{11}$$

Since it is cumbersome to carry this longhand version, one often sees the shorthand notation

$$\frac{dN_i}{dt} = f_i(N_1, N_2, \ldots, N_k) \qquad (i = 1, 2, \ldots, k), \tag{12}$$

or, better still, the vector notation

$$\frac{d\mathbf{N}}{dt} = \mathbf{F}(\mathbf{N}), \tag{13}$$

for $\mathbf{N} = (N_1, N_2, \ldots, N_k)$, $\mathbf{F} = (f_1, f_2, \ldots, f_k)$, where each of the functions f_1, $f_2, \ldots, f_k$ may depend on all or some of the species populations $N_1, N_2, \ldots, N_k$.

We shall now suppose that it is possible to solve the equation (or set of equations)

$$\mathbf{F(N)} = 0, \tag{14}$$

so as to identify one (or possibly several) steady-state points, $\bar{\mathbf{N}} = (N_1, N_2, \ldots, N_k)$, satisfying $\mathbf{F}(\bar{\mathbf{N}}) = 0$. The next step, as a diligent reader might have guessed, would be to determine stability properties of this steady solution. While the idea is essentially identical to previous linear stability analysis, a slightly greater sophistication may be necessary to extract an answer. Let us see why this is true.

In linearizing equation (13) we find, as before, the Jacobian of $\mathbf{F(N)}$. This is often symbolized

$$\mathbf{J} = \frac{\partial \mathbf{F}}{\partial \mathbf{N}}(\bar{\mathbf{N}}). \tag{15}$$

Recall that this really means

$$\mathbf{J} = \begin{pmatrix} \frac{\partial f_1}{\partial N_1} & \frac{\partial f_2}{\partial N_2} & \cdots & \frac{\partial f_k}{\partial N_k} \\ \vdots & & & \\ \frac{\partial f_k}{\partial N_1} & \frac{\partial f_k}{\partial N_2} & \cdots & \frac{\partial f_k}{\partial N_k} \end{pmatrix}_{\bar{\mathbf{N}}} \tag{16}$$

so that $\mathbf{J}$ is now a $k \times k$ matrix. Population biologists frequently refer to $\mathbf{J}$ as the *community matrix* (see Levins, 1968). Eigenvalues λ of this matrix now satisfy

$$\det(\mathbf{J} - \lambda \mathbf{I}) = 0. \tag{17}$$

Thinking of what this means, you should arrive at the conclusion that λ must satisfy a characteristic equation of the form

$$\lambda^k + a_1\lambda^{k-1} + a_2\lambda^{k-2} + \cdots + a_k = 0. \tag{18}$$

If you find this baffling, you may wish to verify this with a 3×3 matrix, that is, by evaluating

$$\det\begin{pmatrix} a - \lambda & b & c \\ d & e - \lambda & f \\ g & h & i - \lambda \end{pmatrix}.$$

(The result is a cubic polynomial.) In general, the characteristic equation is a *polynomial* whose degree k is equal to the number of species interacting. Although for $k = 2$ the quadratic characteristic equation is easily solved, for $k > 2$ this is no longer true.

While we are unable in principle to *find* all eigenvalues, we can still obtain information about their magnitudes. Suppose $\lambda_1, \lambda_2, \ldots, \lambda_k$ are all (known) eigenvalues of the linearized system

$$\frac{d\mathbf{N}}{dt} = \mathbf{J} \cdot \mathbf{N}. \tag{19}$$

What must be true about these eigenvalues so that the steady state $\bar{\mathbf{N}}$ would be stable? Recall that they must *all* have negative real parts since close to the steady states each of the species populations can be represented by a sum of exponentials in $\lambda_i t$ as follows:

$$N_i = \bar{N}_i + a_1 e^{\lambda_1 t} + a_2 e^{\lambda_2 t} + \cdots + a_k e^{\lambda_k t}. \tag{20}$$

(This is a direct generalization of Section 5.6.) If one or more eigenvalues have positive real parts, $N_i - \bar{N}_i$ will be an increasing function of t, meaning that N_i will not return to its equilibrium value $\bar{N}_i$. Thus the question of stability of a steady state can be settled if it can be determined whether or not all eigenvalues $\lambda_1, \ldots, \lambda_k$ have negative real parts. (Contrast this with stability conditions for difference equations.) This can be done without actually solving for these eigenvalues by checking certain criteria. Recall that in the two-species case we derived conditions on quantities β and γ (which were, respectively, the trace and the determinant of the Jacobian) that ensured eigenvalues with negative real parts. For $k > 2$ these conditions are known as the *Routh-Hurwitz criteria* and are summarized in the box.

The Routh-Hurwitz Criteria

Given the characteristic equation (18), define k matrices as follows:

$$\mathbf{H}_1 = (a_1), \qquad \mathbf{H}_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix}, \qquad \mathbf{H}_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ a_5 & a_4 & a_3 \end{pmatrix}, \cdots$$

$$\mathbf{H}_j = \begin{pmatrix} a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & \cdots & 0 \\ a_{2j-1} & a_{2j-2} & a_{2j-3} & a_{2j-4} & \cdots & a_j \end{pmatrix} \cdots \mathbf{H}_k = \begin{pmatrix} a_1 & 1 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & \cdots & 0 \\ \vdots & \vdots & \vdots & & \vdots \\ 0 & 0 & \cdots & & a_k \end{pmatrix},$$

where the (l, m) term in the matrix $\mathbf{H}_j$ is

a_{2l-m}	for $0 < 2l - m < k$,
1	for $2l = m$,
0	for $2l < m$ or $2l > k + m$.

Then all eigenvalues have negative real parts; that is, *the steady-state* $\bar{\mathbf{N}}$ *is stable* if and only if the determinants of all Hurwitz matrices are positive:

$$\det \mathbf{H}_j > 0 \qquad (j = 1, 2, \ldots, k).$$

See Pielou (1969, chap. 6) for a treatment of the above and further references.

May (1973) summarizes these stability conditions in the cases $k = 2, \ldots, 5$. Some of these are given in the small box.

Example 1 illustrates how the Routh-Hurwitz criteria might be applied in a situation in which three species interact.

Routh-Hurwitz Criteria for k = 2, 3, 4

$k = 2$: $a_1 > 0$, $a_2 > 0$.

$k = 3$: $a_1 > 0$, $a_3 > 0$; $a_1 a_2 > a_3$.

$k = 4$: $a_1 > 0$, $a_3 > 0$; $a_4 > 0$; $a_1 a_2 a_3 > a_3^2 + a_1^2 a_4$.

Example 1

Suppose x is a predator and y and z are both its prey. z grows logistically in the absence of its predator. x dies out in the absence of prey, and y grows at an exponential rate in the absence of predator. We shall use the Routh-Hurwitz techniques to discover whether these species can coexist in a stable equilibrium.

Step 1

Writing equations for this system we get

$$\frac{dx}{dt} = \alpha(xz) + \beta(xy) - \gamma x, \tag{21a}$$

($\alpha(xz)$: growth from eating z; $\beta(xy)$: from eating y; γx: mortality)

$$\frac{dy}{dt} = \delta y - \epsilon(xy), \tag{21b}$$

(δy: growth when no predator present; $\epsilon(xy)$: predation mortality)

$$\frac{dz}{dt} = \mu z(\nu - z) - \chi(xz). \tag{21c}$$

($\mu z(\nu - z)$: logistic growth; $\chi(xz)$: predation mortality)

Step 2

Solving for steady state values we get

$$\alpha xz + \beta xy - \gamma x = 0 \Rightarrow \alpha\bar{z} + \beta\bar{y} = \gamma, \text{ or } \bar{x} = 0, \tag{22a}$$

$$\delta y - \epsilon(xy) = 0 \Rightarrow \bar{x} = \frac{\delta}{\epsilon}, \text{ or } \bar{y} = 0, \tag{22b}$$

$$\mu z(\nu - z) - \chi xz = 0 \Rightarrow \mu\nu - \mu\bar{z} - \chi\bar{x} = 0. \tag{22c}$$

From the above we arrive at the nontrivial steady state

$$\bar{x} = \frac{\delta}{\epsilon}, \qquad \bar{y} = \gamma - \alpha\bar{z}, \qquad \bar{z} = \nu - \frac{\chi}{\mu}\bar{x}.$$

This equilibrium makes sense biologically whenever $\gamma > \alpha\bar{z}$ and $\nu > \chi/\mu\,\bar{x}$.

Step 3

Calculating the Jacobian of the system, we get

$$\mathbf{J} = \begin{pmatrix} \alpha\bar{z} + \beta\bar{y} - \gamma & \beta\bar{x} & \alpha\bar{x} \\ -\epsilon\bar{y} & \delta - \epsilon\bar{x} & 0 \\ -\chi\bar{z} & 0 & \mu\nu - 2\mu\bar{z} - \chi\bar{x} \end{pmatrix}. \tag{23}$$

Using the conclusions of step 2, we notice that terms on the diagonal of **J** evaluated at steady state lead to particularly simple forms, so that **J** is

$$\mathbf{J} = \begin{pmatrix} 0 & \beta\bar{x} & \alpha\bar{x} \\ -\epsilon\bar{y} & 0 & 0 \\ -\chi\bar{z} & 0 & -\mu\bar{z} \end{pmatrix} \tag{24}$$

Step 4
To find eigenvalues we must set

$$\det(\mathbf{J} - \lambda\mathbf{I}) = 0.$$

Thus we must evaluate

$$\det \begin{pmatrix} 0 - \lambda & \beta\bar{x} & \alpha\bar{x} \\ -\epsilon\bar{y} & 0-\lambda & 0 \\ -\chi\bar{z} & 0 & -\mu\bar{z} - \lambda \end{pmatrix}, \tag{25}$$

and set the result equal to zero to get the characteristic equation. Expanding, we get

$$-\lambda\begin{pmatrix} -\lambda & 0 \\ 0 & -\mu\bar{z} - \lambda \end{pmatrix} - (-\epsilon\bar{y}) \det \begin{pmatrix} \beta\bar{x} & \alpha\bar{x} \\ 0 & -\mu\bar{z} - \lambda \end{pmatrix} + (-\chi\bar{z}) \det \begin{pmatrix} \beta\bar{x} & \alpha\bar{x} \\ -\lambda & 0 \end{pmatrix}$$

$$= (-\lambda)(-\lambda)(-\mu\bar{z} - \lambda) - (-\epsilon\bar{y})(\beta\bar{x})(-\mu\bar{z} - \lambda) + (-\chi\bar{z})(-\alpha\bar{x})(-\lambda)$$

$$= -\lambda^3 - \lambda^2\mu\bar{z} + \lambda(-\epsilon\bar{y}\beta\bar{x} - \chi\bar{z}\alpha\bar{x}) + (-\mu\bar{z}\epsilon\bar{y}\beta\bar{x}) = 0.$$

Cancelling a factor of -1 we obtain

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{26a}$$

where

$$a_1 = \mu\bar{z}, \tag{26b}$$

$$a_2 = \epsilon\beta\bar{x}\bar{y} + \chi\alpha\bar{x}\bar{z}, \tag{26c}$$

$$a_3 = \mu\epsilon\beta\bar{x}\bar{y}\bar{z}. \tag{26d}$$

Step 5
Now we check the three conditions using the Routh-Hurwitz criteria for the case $k = 3$ (three species): The three conditions are

1. $a_1 > 0$,
2. $a_3 > 0$,
3. $a_1a_2 > a_3$.

Condition 1 is true since $a_1 = \mu\bar{z}$ is a positive quantity. Condition 2 is true for the same reason. Looking at condition 3, we note that

$$a_1a_2 = \mu\bar{z}(\epsilon\beta\bar{x}\bar{y} + \chi\alpha\bar{x}\bar{z}).$$

This is clearly bigger than $a_3 = \mu\epsilon\beta\bar{x}\bar{y}\bar{z}$ since the quantity $\chi\alpha\mu\bar{x}\bar{z}^2$ is positive. Thus condition 3 is also satisfied.

We conclude that the steady state is a stable one.

Remark 1

Because calculations of 3×3 (and higher-order) determinants can be particularly cumbersome, it is advisable to express the Jacobian in the simplest possible notation. We do this by leaving entries in terms of $\bar{x}$, $\bar{y}$, and $\bar{z}$ except where further simplification can be made (such as along the diagonal of **J**). In step 5 we then use the fact that the quantities $\bar{x}$, $\bar{y}$, and $\bar{z}$ are positive.

Remark 2

In some situations the *magnitudes* of the steady-state values also enter into the stability conditions. We will see in a later section why this is not the case here in example 1.

6.5 QUALITATIVE STABILITY

The Routh-Hurwitz criteria outlined in Section 6.4 are an exact but cumbersome method for determining stability of a large system. For communities of five or more species, the technique proves so computationally involved that it is of diminishing practical value. Shortcuts, when available, can be quite useful.

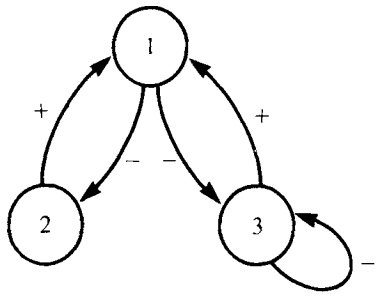
In this section we explore a shortcut method for investigating large systems that needs little if any computation. Because this method is not universally applicable, its importance is viewed as secondary. Furthermore, to understand exactly why the method works requires knowledge of matrices beyond elementary linear algebra. Nevertheless, what makes the technique of *qualitative stability* appealing is that it is easy to explain, easy to test, and thus a refreshing change from intensive computations.

The technique of qualitative stability analysis applies ideally to large complicated systems in which there is no quantitative information about the interrelationship of species or subsystems. Motivation for this method actually came from economics. A paper by the economists Quirk and Ruppert (1965) was followed later by further work and application to ecology by May (1973), Levins (1974), and Jeffries (1974).

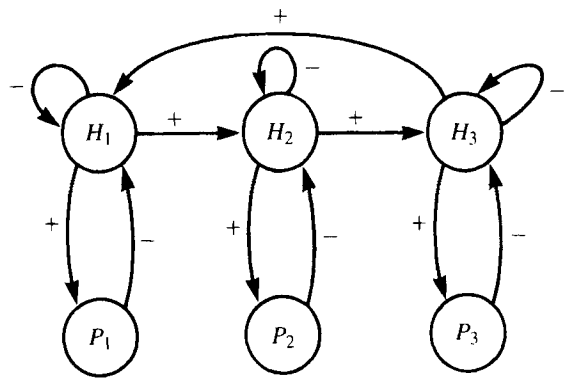
In a complex community composed of many species, numerous interactions take place. The magnitudes of the mutual effects of species on each other are seldom accurately known, but one can establish with greater certainty whether predation, competition, or other influences are present. This means that technically the functions appearing in equations that describe the system [such as equation (11)] are not known. What is known instead is the pattern of signs of partial derivatives of these functions [contained, for example, in the Jacobian of equation (16)]. We encountered a similar problem in the context of a plant-herbivore system (Chapter 3) and of a glucose-insulin model (Chapter 4). Here the problem consists of larger systems in a continuous setting, and even the magnitudes of partial derivatives may not be known.

There are two equivalent ways of representing qualitative information. A more obvious one is to assign the symbols $+$, 0, and $-$ to the (i, j)th entry of a matrix if the species j has respectively a positive influence, no influence, or a negative

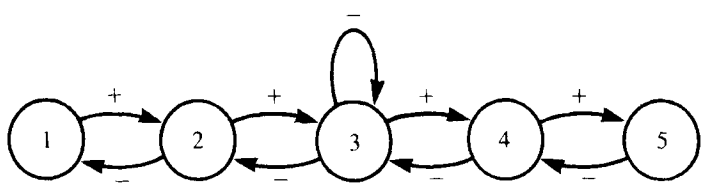
influence on species i. An alternate, visual representation captures the same ideas in a *directed graph* (also called *digraph*) in which nodes represent species and arrows between them represent the mutual interactions, as shown in Figures 6.10 and 6.11. The question is then whether it can be concluded, *from this graph or sign pattern only,* that the system is stable. If so, the system is called *qualitatively stable.*



(a)



(b)



(c)

Figure 6.10 *Signed directed graphs (digraphs) can be used to represent species interactions in a complex ecosystem. The graphs shown here are equivalent to the matrix representation of sign patterns given in the text (a) example 2, (b) example 3, and (c) example 4.*

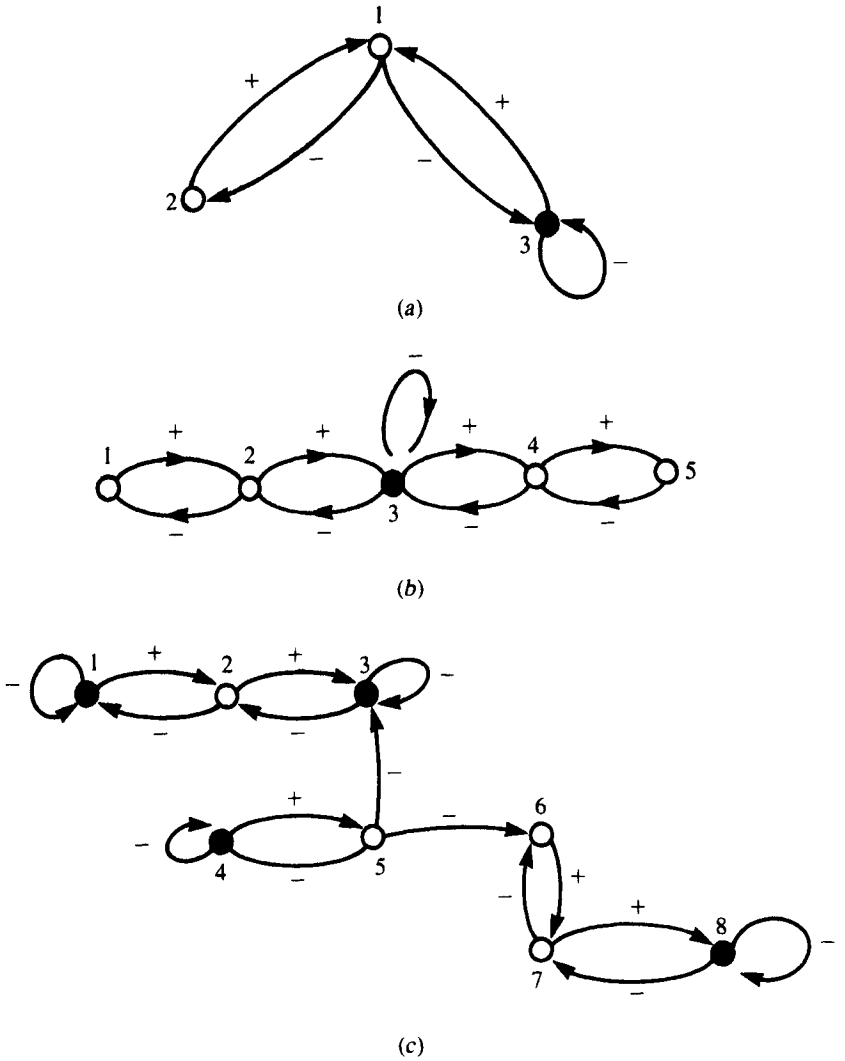


Figure 6.11 *Properties of signed directed graphs can be used to deduce whether the system is* qualitatively stable *(stable regardless of the magnitudes of mutual effects). The Jeffries color test and the Quirk-Ruppert conditions are applied to these graphs to conclude that (a), which corresponds to example 2, and (c) are stable communities, whereas (b), which corresponds to example 4, is not.*

Systems that are qualitatively stable are also stable in the ordinary sense. (The converse is not true.) Systems that are *not* qualitatively stable can still be stable under certain conditions (for example, if the magnitudes of interactions are appropriately balanced.)

Following Quirk and Ruppert (1965), May (1973) outlined five conditions for

Example 2

Here we study the sign pattern of the community described in equations (21a, b, c) of Section 6.4. From Jacobian (24) of the system we obtain the qualitative matrix

$$\mathbf{Q} = \operatorname{sign} \mathbf{J} = \begin{bmatrix} 0 & + & + \\ - & 0 & 0 \\ - & 0 & - \end{bmatrix}.$$

This means that close to equilibrium, the community can also be represented by the graph in Figure 6.10. Reading entries in $\mathbf{Q}$ from left to right, top to bottom:

Species 1 gets positive feedback from species 2 and 3.
Species 2 gets negative feedback from species 1.
Species 3 gets negative feedback from species 1 and from itself.

Example 3 (Levins, 1977)

In a closed community, three predators or parasitoides, labeled P_1, P_2, and P_3, attack three different stages in the life cycle of a host, H_1, H_2, and H_3. The presence of hosts is a positive influence for their predators but predators have a negative influence on their prey. Figure 6.10*(b)* and the following matrix summarize the interactions:

$$\begin{array}{c} \\ P_1 \\ P_2 \\ P_3 \\ H_1 \\ H_2 \\ H_3 \end{array} \begin{array}{c} \begin{array}{cccccc} P_1 & P_2 & P_3 & H_1 & H_2 & H_3 \end{array} \\ \begin{bmatrix} 0 & 0 & 0 & + & 0 & 0 \\ 0 & 0 & 0 & 0 & + & 0 \\ 0 & 0 & 0 & 0 & 0 & + \\ - & 0 & 0 & - & 0 & + \\ 0 & - & 0 & + & - & 0 \\ 0 & 0 & - & 0 & + & - \end{bmatrix} \end{array}.$$

Note that H_1, H_2, and H_3 each exert negative feedback on themselves.

Example 4 (Jeffries, 1974)

In a five-species ecosystem, species 2 preys on species 1, species 3 on species 2, and so on in a food chain up to species 5. Species 3 is also self-regulating. A qualitative matrix for this community is

$$\mathbf{Q} = \begin{bmatrix} 0 & - & 0 & 0 & 0 \\ + & 0 & - & 0 & 0 \\ 0 & + & - & - & 0 \\ 0 & 0 & + & 0 & - \\ 0 & 0 & 0 & + & 0 \end{bmatrix}.$$

See Figure 6.10(*c*).

qualitative stability. Suppose a_{ij} is the ijth element of the matrix of signs $\mathbf{Q}$. Then it is necessary for all of the following conditions to hold:

1. $a_{ii} \leq 0$ for all i.
2. $a_{ii} < 0$ for at least one i.
3. $a_{ij}a_{ji} \leq 0$ for all $i \neq j$.
4. $a_{ij}a_{jk} \cdots a_{qr}a_{ri} = 0$ for any sequences of three or more distinct indices $i, j, k, \ldots, q, r$.
5. $\det \mathbf{Q} \neq 0$.

These conditions can be interpreted in the following way:

1. No species exerts positive feedback on itself.
2. At least one species is self-regulating.
3. The members of any given pair of interacting species must have opposite effects on each other.
4. There are no closed chains of interactions among three or more species.
5. There is no species that is unaffected by interactions with itself or with other species.

For mathematical proof of these five necessary conditions, consult Quirk and Ruppert (1965). May (1973) and Pielou (1969) comment on the biological significance, particularly of conditions 3 and 4. The conditions can be tested by looking at graphs representing the communities. One must check that these graphs have all the following properties:

1. No + loops on any single species (that is, no positive feedback).
2. At least one − loop on some species in the graph.
3. No pair of like arrows connecting a pair of species.
4. No cycles connecting three or more species.
5. No node devoid of input arrows.

These five conditions are equivalent to the original algebraic statement.

Example 5

For examples 2 to 4 we check off the five conditions given earlier:

Condition Number	*Example 2*	*Example 3*	*Example 4*
1	√	√	√
2	√	√	√
3	√	√	√
4	√	No	√
5	√	√	√

It was shown by Jeffries (1974) that these five conditions alone cannot distinguish between *neutral stability* (as in the Lotka-Volterra cycles) and *asymptotic stability,* wherein the steady state is a stable node or spiral. (In other words, the conditions are *necessary but not sufficient* to guarantee that the species will coexist in a constant steady state). In example 4 Jeffries notes that pure imaginary eigenvalues can occur, so that even though the five conditions are met, the system will oscillate. To weed out such marginal cases, Jeffries devised an auxiliary set of conditions, which he called the "color test," that *replaces* condition 2. Before describing the color test, it is necessary to define the following:

A *predation link* is a pair of species connected by one + line and one − line.
A *predation community* is a subgraph consisting of all interconnected predation links.

If one defines a species not connected to any other by a predation link as a *trivial* predation community, then it is possible to decompose any graph into a set of distinct predation communities. The systems shown in Figure 6.10 have predation communities as follows: (*a*) {2, 1, 3}; (*b*) $\{H_1, P_1\}$, $\{H_2, P_2\}$, $\{H_3, P_3\}$; and (*c*) {1, 2, 3, 4, 5}. In Figure 6.11(*c*) there are three predation communities: {1, 2, 3}, {4, 5}, and {6, 7, 8}.

The following color scheme constitutes the test to be made. A predation community is said to *fail the color test* if it is not possible to color each node in the subgraph black or white in such a way that

1. Each self-regulating node is black.
2. There is at least one white point.
3. Each white point is connected by a predation link to at least one other white point.
4. Each black point connected by a predation link to one white node is also connected by a predation link to one other white node.

Jeffries (1974) proved that for asymptotic stability, a community must satisfy the original Quirk-Ruppert conditions 1, 3, 4, and 5, and in addition must have only predation communities that fail the color test.

Example 5 (continued)
Examples 2 and 4 satisfy the original conditions. In Figure 6.11 the color test is applied to their communities. We see that example 2 consists of a single predation community that *fails* part 4 of the color test. Example 4 *satisfies* the test. A final example shown in Figure 6.11(*c*) has three predation communities, and each one fails the test. We conclude that Figure 6.11(*a*) and (*c*) represent systems that have the property of asymptotic stability; that is, these ecosystems consist of species that coexist at a stable fixed steady state without sustained oscillations.

A proof and discussion of the revised conditions is to be found in Jeffries (1974). For other applications and properties of graphs, you are encouraged to peruse Roberts (1976).

6.6 THE POPULATION BIOLOGY OF INFECTIOUS DISEASES

Infectious diseases can be classified into two broad categories: those caused by viruses and bacteria are *microparasitic* diseases, and those due to worms (more commonly found in third-world countries) are *macroparasitic*. Other than the relative sizes of the infecting agents, the main distinction is that microparasites reproduce within their host and are transmitted directly from one host to another. Most macroparasites, on the other hand, have somewhat more complicated life cycles, often with a secondary host or carrier implicated. (Examples of these include malaria and schistosomiasis; see Anderson, 1982 for a review.)

This section briefly summarizes some of the classical models for microparasitic infections. The mathematical techniques required for analyzing the models parallel the techniques applied in Sections 6.2 and 6.3. However, as a general remark, it should be said that the *flavor* of the models differs somewhat from the species-interactions models introduced in this chapter.

With no *a priori* knowledge, suppose we are asked to model the process of infection of a viral disease such as measles or smallpox. In keeping with the style of population models for predation or competition, it would be tempting to start by defining variables for population densities of the host x and infecting agent y. Here is how such a model might proceed:

Primitive Model for a Viral Infection

This model is for illustrative purposes only. Let

x = population of human hosts,

y = viral population.

The assumptions are that

1. There is a constant human birth rate α.
2. Viral infection causes an increased mortality due to disease, so $g(y) > 0$.
3. Reproduction of viral particles depends on human presence.
4. In the absence of human hosts, virus particles "die" or become nonviable at rate γ.

The equations then read:

$$\frac{dx}{dt} = [\alpha - g(y)]x,$$

$$\frac{dy}{dt} = \beta xy - \gamma y.$$

The approach leads us to a modified Lotka-Volterra predation model. This view, to put it simply, is that viruses y are predatory organisms searching for human prey x to consume. The conclusions given in Section 6.2 follow with minor modification.

The philosophical view of disease as a process of predation is an unfortunate and somewhat misleading analogy on several counts. First, no one can reasonably suppose it possible to measure or even estimate total viral population, which may range over several orders of magnitude in individual hosts. Second, a knowledge of this number is at best uninteresting and trivial since it is the distribution of viruses over hosts that determines what percentage of people will actually suffer from the disease. To put it another way, some hosts will harbor the infecting agent while others will not. Finally, in the "primitive" model an underlying hidden assumption is that viruses roam freely in the environment, randomly encountering new hosts.This is rarely true of microparasitic diseases. Rather, diseases are spread by contact or close proximity between infected and healthy individuals. How the disease is spread in the population is an interesting question. This crucial point is omitted and is thus a serious criticism of the model.

A new approach is necessary. At the very least it seems sensible to make a distinction between sick individuals who harbor the disease and those who are as yet healthy. This forms the basis of all microparasitic epidemiological models, which, as we see presently, virtually omit the population of parasites from direct consideration.

Instead, the host population is subdivided into distinct classes according to the health of its members. A typical subdivision consists of susceptibles S, infectives I, and a third, removed class R of individuals who can no longer contract the disease because they have recovered with immunity, have been placed in isolation, or have died. If the disease confers a *temporary immunity* on its victims, individuals can also move from the third class to the first.

Time scales of epidemics can vary greatly from weeks to years. *Vital dynamics* of a population (the normal rates of birth and mortalities in the absence of disease) can have a large influence on the course of an outbreak. Whether or not immunity is conferred on individuals can also have an important impact. Many models using the general approach with variations on the assumptions have been studied. An excellent summary of several is given by Hethcote (1976) and Anderson and May (1979), although different terminology is unfortunately used in each source.

Some of the earliest classic work on the theory of epidemics is due to Kermack and McKendrick (1927). One of the special cases they studied is shown in Figure 6.12(a). The diagram summarizes transition rates between the three classes with the parameter β, the rate of transmission of the disease, and the rate of removal ν. It is assumed that each compartment consists of identically healthy or sick individuals and that no births or deaths occur in the population. (In more current terminology, the situation shown in Figure 6.12(a) would be called an *SIR model without vital dynamics* because the transitions are from class S to I and then to R; see for example, Hethcote, 1976.)

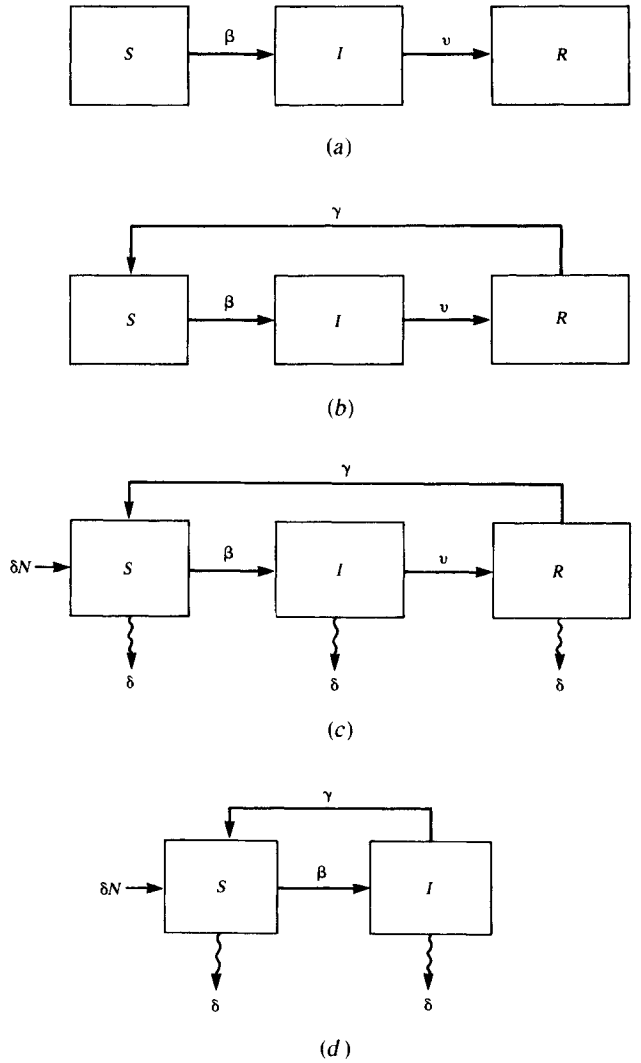


Figure 6.12 *A number of epidemic models that have been studied. The total population* N *is subdivided into susceptible* (S), *infective* (I) *and removed* (R) *classes. Transitions between compartments depict the course of transmission, recovery, and loss of immunity with rate constants* β, ν, *and* γ. *A population with vital dynamics is assumed to be producing new susceptibles at rate* δ *which is identical to the mortality rate. (a)* SIR *model; (b, c)* SIRS *models; and (d)* SIS *model.*

Figure 6.12(a) and those following it are somewhat reminiscent of models we have already studied for the physical flows between well-mixed compartments (for example, the chemostat). A subtle distinction must be made though, since the passage of individuals from the susceptible to the infective class generally occurs as a result of close proximity or contact between healthy and infective individuals. Thus the rate of exchange between S and I has a special character summarized by the following assumption:

> ***Assumption***
> The rate of transmission of a microparasitic disease is proportional to the rate of encounter of susceptible and infective individuals modelled by the product (βSI).

The equations due to Kermack and MacKendrick for the disease shown in Figure 6.12(*a*) are thus

$$\frac{dS}{dt} = -\beta IS, \tag{27a}$$

$$\frac{dI}{dt} = \beta IS - \nu I, \tag{27b}$$

$$\frac{dR}{dt} = \nu I. \tag{27c}$$

It is easily verified that the total population $N = S + I + R$ does not change. Though these equations are nonlinear, Kermack and MacKendrick derived an approximate expression for the rate of removal dR/dt (in their paper called dz/dt) as a function of time. The result is a rather messy expression involving hyperbolic secants; when plotted with the appropriate values given to the parameters it compares rather well with data for death by plague in Bombay during an epidemic in 1906 (see Figure 6.13).

A more instructive approach is to treat the problem by qualitative methods. Now we shall carry out this procedure on a slightly more general case, allowing for a loss of immunity that causes recovered individuals to become susceptible again [Figured 6.12(*b*)]. It will be assumed that this takes place at a rate proportional to the population in class R, with proportionality constant γ. Thus the equations become

$$\frac{dS}{dt} = -\beta SI + \gamma R, \tag{28a}$$

$$\frac{dI}{dt} = \beta SI - \nu I, \tag{28b}$$

$$\frac{dR}{dt} = \nu I - \gamma R. \tag{28c}$$

This model is called an *SIRS* model since removed individuals can return to class S ($\gamma = 0$ is the special case studied by Kermack and McKendrick). It is readily shown that these equations have two steady states:

$$\bar{S}_1 = N, \qquad \bar{I}_1 = 0, \qquad \bar{R}_1 = 0; \tag{29a}$$

$$\bar{S}_2 = \frac{\nu}{\beta}, \qquad \bar{I}_2 = \gamma \frac{N - \bar{S}_2}{\nu + \gamma}, \qquad \bar{R}_2 = \frac{\nu \bar{I}_2}{\gamma}. \tag{29b}$$

In (29a) the whole population is healthy (but susceptible) and disease is eradicated. In (29b) the community consists of some constant proportions of each type provided $(\bar{S}_2, \bar{I}_2, \bar{R}_2)$ are all positive quantities. For $\bar{I}_2$ to be positive, N must be larger than $\bar{S}_2$. Since $\bar{S}_2 = \nu/\beta$, this leads to the following conclusion:

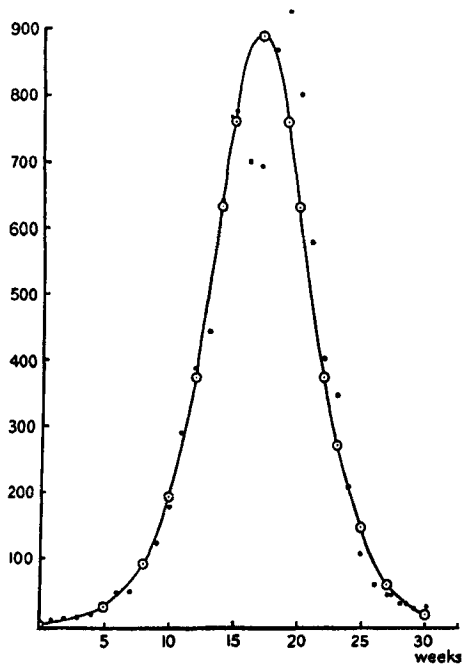
The disease will be established in the population provided the total population N exceeds the level ν/β, that is,

$$\frac{N\beta}{\nu} > 1.$$

This important *threshold effect* was discovered by Kermack and McKendrick; the population must be "large enough" for a disease to become endemic.

Also for the rate at which cases are removed by death or recovery which is the form in which many statistics are given

$$\frac{dz}{dt} = \frac{l^3}{2x_0c^2}\sqrt{-q}\,\operatorname{sech}^2\left(\frac{\sqrt{-q}}{2}lt - \phi\right). \qquad \text{(thirty-one)}$$



The accompanying chart is based upon figures of deaths from plague in the island of Bombay over the period December 17, 1905, to July 21, 1906. The ordinate represents the number of deaths per week, and the abscissa denotes the time in weeks. As at least 80 to 90 per cent. of the cases reported terminate fatally, the ordinate may be taken as approximately representing ds/dt as a function of t. The calculated curve is drawn from the formula

$$\frac{ds}{dt} = 890\,\operatorname{sech}^2(0{\cdot}2t - 3{\cdot}4).$$

Figure 6.13 *On a page from their original article, Kermack and McKendrick compare predictions of the model given by equations (27a,b,c) with data for the rate of removal by death. Note:* dz/dt *is equivalent to* dR/dt *in equations (27).[Kermack, W. O., and McKendrick, A. G. (1927). A contribution to mathematical theory of epidemics.* Roy Stat. Soc. J., 115, *714.]*

The ratio of parameters β/ν has a rather meaningful interpretation. Since removal rate from the infective class is ν (in units of 1/time), the average period of infectivity is $1/\nu$. Thus β/ν is the fraction of the population that comes into contact with an infective individual during the period of infectiousness. The quantity $R_0 = N\beta/\nu$ has been called the *infectious contact number*, σ (Hethcote, 1976) and the *intrinsic reproductive rate of the disease* (May, 1983). R_0 represents the average number of secondary infections caused by introducing a single infected individual into a host population of N susceptibles. (In papers by May and Anderson, the threshold result is usually written $R_0 > 1$.)

In further analyzing the model we can take into account the particularly convenient fact that the total population

$$N = S + I + R$$

does not change (see problem 25 for verification). This means that one variable, say R, can always be eliminated so that the model can be given in terms of two equations in two unknowns. In the following analysis this fact is exploited in applying phase-plane methods to the problem.

Qualitative Analysis of a* SIRS *Model: Epidemic with Temporary Immunity and No Vital Dynamics

Since the total population is constant, we eliminate R from equations (28) by substituting

$$R = N - S - I. \tag{30}$$

The equations for S and I are then

$$\frac{dS}{dt} = -\beta SI + \gamma(N - S - I) \overset{\text{def}}{\equiv} F(S, I), \tag{31a}$$

$$\frac{dI}{dt} = \beta SI - \nu I \equiv G(S, I). \tag{31b}$$

Nullclines

$$I' = 0 \quad \text{for} \quad I = 0 \quad \text{and} \quad S = \nu/\beta,$$
$$S' = 0 \quad \text{for} \quad \beta SI = \gamma(N - S - I).$$

After rearranging,

$$I = \frac{\gamma(N - S)}{(\beta S + \gamma)}.$$

This curve intersects the axes at $(N, 0)$ and $(0, N)$.

Steady states

$$(\bar{S}_1, \bar{I}_1) = (N, 0); (\bar{S}_2, \bar{I}_2) = \left(\frac{\nu}{\beta}, \frac{N - (\nu/\beta)}{\nu + \gamma}\right).$$

Jacobian

$$\mathbf{J} = \begin{pmatrix} F_S & F_I \\ G_S & G_I \end{pmatrix}_{ss} = \begin{pmatrix} -(\beta\bar{I} + \gamma) & -(\beta\bar{S} + \gamma) \\ \beta\bar{I} & \beta\bar{S} - \nu \end{pmatrix}.$$

Stability
For $(\bar{S}_2, \bar{I}_2)$,

$\text{Tr } \mathbf{J} = -(\beta\bar{I}_2 + \gamma)$ is always negative,

$\det \mathbf{J} = \beta\bar{I}_2(\nu + \gamma)$ is always positive.

Thus this steady state is always stable when it exists, namely when the threshold condition is satisfied. It is evident from Figures 6.14 and 6.15(*b*) and from further analysis that the approach to this steady state can be oscillatory.

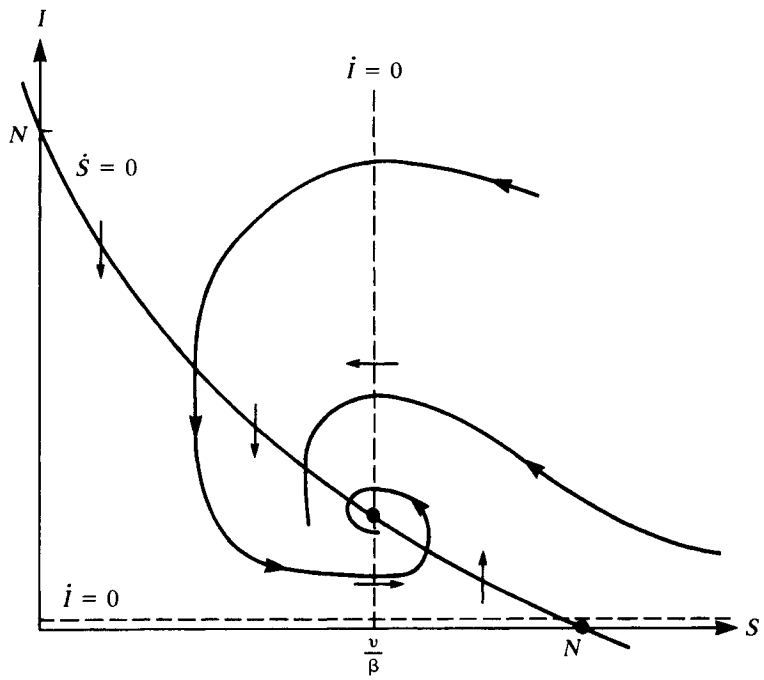
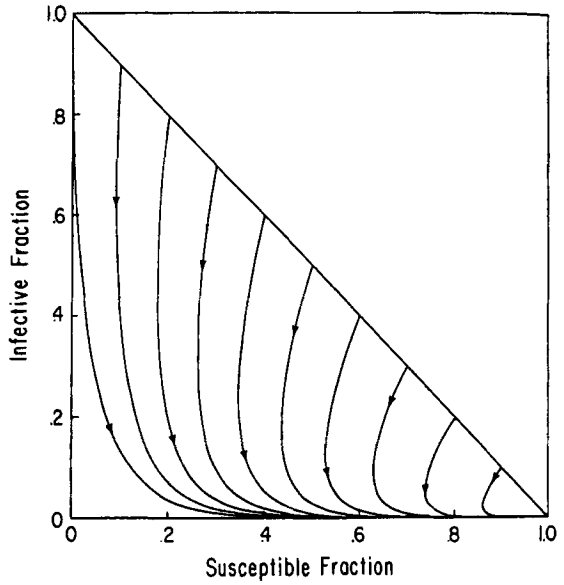
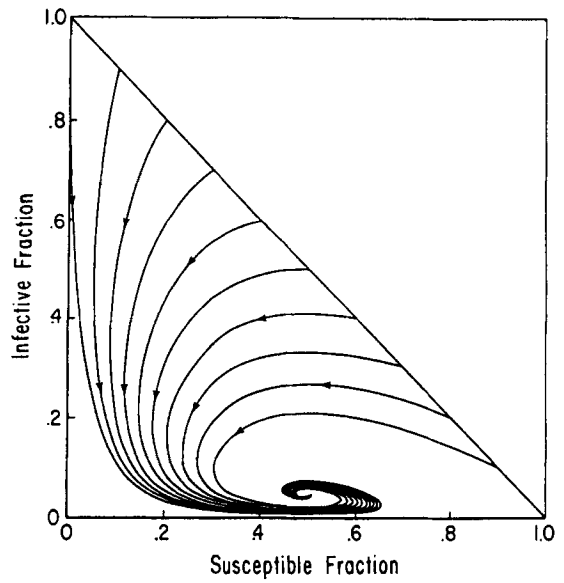


Figure 6.14 *Nullclines, steady states, and several trajectories for the* SIRS *model given by equations (31a,b), which are equivalent to (28a,b,c).*

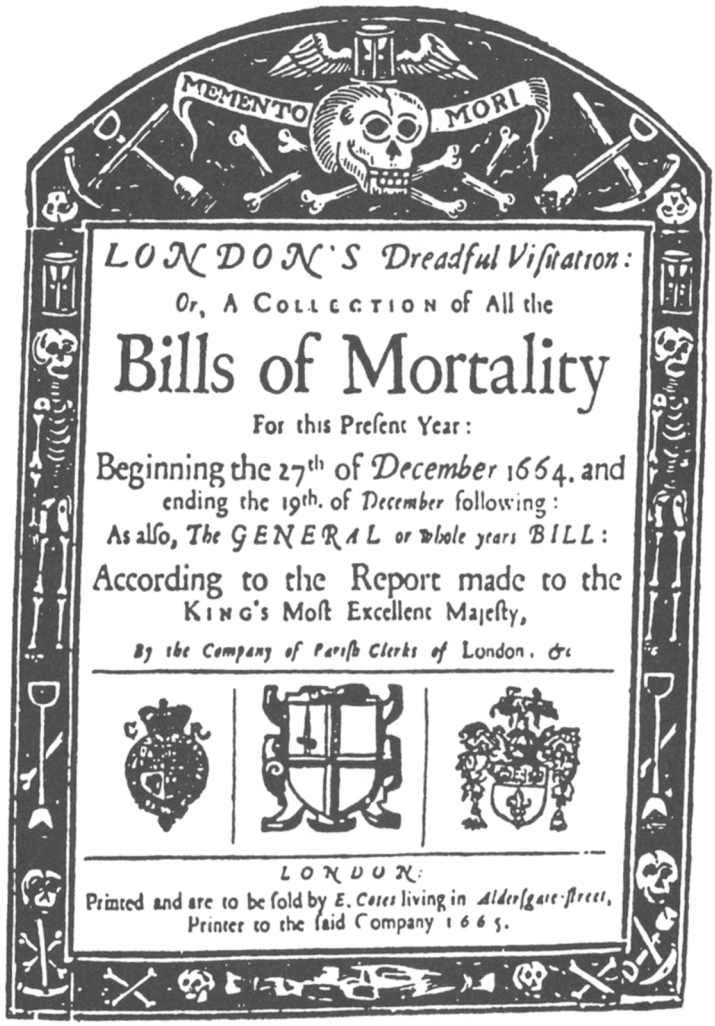


(*a*)



(*b*)

Figure 6.15 *Epidemic models are characterized by the magnitude of an infectious contact number* σ. *(a) When* $\sigma < 1$, *the infective class will disappear. (b) When* $\sigma > 1$, *there is some stable steady state in which both susceptibles and infectives are present. Shown here is an* SIRS *model with vital dynamics.) [Reprinted by permission of the publisher from Hethcote, H. W. (1976). Qualitative analyses of communicable disease models.* Math. Biosci., 28, *344 and 345. Copyright 1976 by Elsevier Science Publishing Co., Inc.]*



Mortality from a variety of afflictions, only some of which were caused by disease, were systematically recorded as early as the 1600s in the Bills of Mortality published in London. Reproduced here is the title page of the London Bills of Mortality for 1665, the year of the great plague. The people of the city followed with anxiety the rise and fall in the number of deaths from the plague, hoping always to see the sharp decline which they knew from past experience indicated that the epidemic was nearing its end. When the decline came the refugees, mostly from the nobility and wealthy merchants, returned to the city, and then for a time the mortality rose again as the disease attacked these new arrivals. The plague of 1665 started in June; its peak came in September and its decline in October. The secondary rise occurred in November and cases of the disease were reported as late as March of the following year. *[From H. W. Haggard (1957),* Devils, Drugs, Doctors, *Harper & Row, New York.]*

The Diseases, and Casualties this year being 1632.

Abortive, and Stilborn ..	445	Grief	11
Affrighted	1	Jaundies	43
Aged	628	Jawfaln	8
Ague	43	Impostume	74
Apoplex, and Meagrom	17	Kil'd by several accidents..	46
Bit with a mad dog.....	1	King's Evil.....	38
Bleeding	3	Lethargie	2
Bloody flux, scowring, and flux	348	Livergrown	87
Brused, Issues, sores, and ulcers, ...	28	Lunatique	5
Burnt, and Scalded.....	5	Made away themselves.....	15
Burst, and Rupture.....	9	Measles	80
Cancer, and Wolf.....	10	Murthered	7
Canker	1	Over-laid, and starved at nurse	7
Childbed	171	Palsie	25
Chrisomes, and Infants.....	2268	Piles.....	1
Cold, and Cough.....	55	Plague.....	8
Colick, Stone, and Strangury	56	Planet	13
Consumption	1797	Pleurisie, and Spleen.....	36
Convulsion	241	Purples, and spotted Feaver	38
Cut of the Stone.....	5	Quinsie	7
Dead in the street, and starved	6	Rising of the Lights.....	98
Dropsie, and Swelling.....	267	Sciatica	1
Drowned	34	Scurvey, and Itch.....	9
Executed, and prest to death	18	Suddenly	62
Falling Sickness.....	7	Surfet	86
Fever	1108	Swine Pox	6
Fistula	13	Teeth	470
Flocks, and small Pox.....	531	Thrush, and Sore mouth...	40
French Pox.....	12	Tympany	13
Gangrene	5	Tissick	34
Gout	4	Vomiting	1
		Worms	27

Christened	Males....4994 Females..4590 In all....9584	Buried	Males....4932 Females..4603 In all....9535	Whereof, of the Plague.8

Increased in the Burials in the 122 Parishes, and at the Pest-house this year..... 993

Decreased of the Plague in the 122 Parishes, and at the Pest-house this year..... 266 [10]

Numerous other cases have been analyzed in detail. Perhaps the best summary is given by Hethcote (1976), in which theoretical results are followed by *biocorollaries* that spell out the biological predictions. His paper was used in drawing up Table 6.1, a composite that describes a number of cases.

One point worth mentioning is the essential difference between models in which the susceptible class is *renewed* (by recovery or loss of immunity) and those in which it is not. [The *SIRS* and *SIS* examples shown in Figure 6.12($b-d$) belong to the former category.] These distinct types behave differently when the normal turnover of births and deaths is superimposed on the dynamics of the disease. In the *SIR* type without births, the continual decrease of the susceptible class results in a decline in the effective reproductive rate of the disease. The epidemic stops for want of infectives, not, as it might seem, for want of susceptibles (Hethcote, 1976). On the other hand, if the susceptible class is replenished by births or recoveries, the subpopulation that participates in the disease is maintained, and the disease can persist.

From Table 6.1 we see that *SIR* models are subdivided into those with and without births and deaths. In other models the chief effect of normal birth and mortality at rates δ is to decrease the infectious contact number σ. This means that a smaller population can sustain an endemic disease. Note that the total population N is taken to be constant in all of these models since the number of deaths from all classes is assumed to exactly balance the births of new susceptibles. Among other things, this permits all such models to be analyzed by methods similar to the method used here since one variable can always be eliminated.

A somewhat different philosophical approach was taken by Anderson and May (1979), who were less interested in the dynamics of the disease itself. By analyzing a model in which a disease-free population grows exponentially, rather than being maintained at a constant level, they demonstrated that epidemics increasing host mortality have the potential to regulate population levels (see problem 30). This adds yet another interesting possiblity to the list of causes of decelerating growth rates in natural populations. Aside from inter- and intraspecies competition and predation, disease-causing agents (much like parasites) can control the population dynamics of their hosts.

The theory of epidemics has numerous ramifications, some of which are mathematical and some practical. In recent years more advanced mathematical models have been studied to determine the effects of delay factors (such as a waiting time in the infectious class), age structure, migration, and spatial distributions. Many of these models require sophisticated mathematical methods of analysis (see, for example, Busenberg and Cooke, 1978). An excellent survey with detailed references is given by Hethcote et al. (1981).

One theoretical question such papers often address is whether models with particular structures lead to stable (limit-cycle) oscillations. This question is of interest since some diseases are associated with periodic outbreaks with very low endemic periods followed by peak epidemic cycles. In some cases the forces driving such cyclic behavior are related to seasonality and to changes in contact rates. (A good example is childhood diseases, which invariably peak during the school year when contact between their potential hosts is greatest.) However, even in the absence of externally imposed periodicity, models similar to *SIRS* can have an inherent ten-

Table 6.1 ***A Summary of Several Epidemic Models***

Type	*Immunity*	*Birth/Death*	*Significant quantity*	*Results*	*Figures*
SIS	None	Rate = δ	$\sigma = \dfrac{\beta}{\gamma + \delta}$	(1) $\sigma > 1$: constant endemic infection (2) $\sigma < 1$: infection disappears	6.9(d)
		Additional disease fatality rate η	σ as above, and $\epsilon = \dfrac{\eta}{\gamma + \delta}$	Disease always eventually disappears leaving some susceptibles.	
SIR	Yes, recovery gives immunity.	None	$\sigma = \dfrac{S_0 \beta}{\gamma}$ (S_0 = initial S)	(1) $\sigma > 1$: infection peaks and then disappears (2) $\sigma < 1$: infection disappears	6.9(a)
		Yes, rate = δ	$\sigma = \dfrac{\beta}{\nu + \delta}$	(1) $\sigma < 1$: susceptibles and infectives approach constant levels (2) $\sigma < 1$: infectives disappear; only S remains	6.9(c) with $\gamma = 0$
(*SIR* with carriers)	Yes	Yes		Disease always remains endemic.	
SIRS	Temporary, lost at rate γ	Rate = δ	$\sigma = \dfrac{\beta}{\nu + \delta}$	(1) $\sigma > 1$: same as *SIR*(1) but higher levels of infectives (2) $\sigma < 1$: same as *SIR*(2)	6.9(c)

dency to give rise to oscillations. This is particularly true of models with long periods of immunity or some other delaying factor. Hethcote notes that a sequence of at least three removed classes will also achieve the result (for example, $SIR_1R_2R_3S$).

The implications of many aspects of applying mathematical theory to natural populations are eloquently described in numerous papers by May and Anderson. Some questions are of a basic scientific nature. For example, the extent to which diseases and hosts have coevolved is a fascinating topic; a second controversial question is whether or not diseases are in fact a predominant factor in controlling natural populations. Other questions have more immediate medical ramifications. Anderson and May suggest that theory has an important place in illuminating the impact of disease on human populations and the ability to eradicate or control disease. Two applications of the theory to vaccination programs are briefly highlighted in the following section.

6.7 FOR FURTHER STUDY: VACCINATION POLICIES

Models for infectious diseases lead to a better understanding of how vaccination programs affect the control or eradication of the disease. Several popular articles by Anderson and May (1982) and a more detailed mathematical version (1983) are thought-provoking and informative. The full theory that takes into account age structure of the population uses a partial differential equation model (which would be understood more fully after covering Chapter 11). However, a number of rather interesting consequences of the theory can be understood with no further preparation.

Eradicating a Disease

Immunization can reduce or eliminate the incidence of infection, even when only part of the population receives the treatment. Those individuals who have been vaccinated will be protected from acquiring infection (this is the obvious direct effect). A secondary effect is that since vaccinated individuals are essentially removed from participating in transmission of the disease, there will be fewer infectious individuals and thus a decreased likelihood that an unvaccinated susceptible will come in contact with the disease. This indirect effect is known as *herd immunity*.

Administering vaccinations to an entire population can be costly. Some vaccines (for example, some measles and whooping cough vaccines) also carry the risk, though rare, of causing various reactions or neurological damage. Thus, if disease eradication can be achieved by partially vaccinating some fraction p of the population, an advantage is gained.

The fraction to be immunized must be such that the remaining population, $(1 - p)N$, will no longer exceed the threshold level necessary to perpetuate the disease. In the terminology of Anderson and May, the reproductive factor R_0 of the infection is to be reduced below 1. Since $R_0 = N\beta/\nu$, for a given disease this factor can be estimated from epidemiological and population records. Table 6.2 lists several common diseases with their corresponding R_0 factors.

Table 6.2 ***Estimates of the intrinsic reproductive rate R_0 for human diseases and the corresponding percentage of the population p that must be protected by immunization to achieve eradication. [Reprinted by permission, American Scientist, journal of Sigma Xi, "Parasitic Infections as Regulator of Animal Populations," by Robert M. May, 71:36–45 (1983).]***

Infection	*Location and Time*	R_0	*Approximate Value of p (%)*
Smallpox	Developing countries, before global campaign	3–5	70–80
Measles	England and Wales, 1956–68;	13	92
	U.S., various places, 1910–30	12–13	92
Whooping cough	England and Wales, 1942–50;	17	94
	Maryland, U.S., 1908–17	13	92
German measles	England and Wales, 1979;	6	83
	West Germany, 1972	7	86
Chicken pox	U.S., various places, 1913–21 and 1943	9–10	90
Diphtheria	U.S., various places, 1910–47	4–6	~80
Scarlet fever	U S., various places, 1910–20	5–7	~80
Mumps	U.S., various places, 1912–16 and 1943	4–7	~80
Poliomyelitis	Holland, 1960: U.S., 1955	6	83

The fraction p to be immunized is then deduced from the following simple calculation:

Intrinsic reproductive rate of disease $= R_0$, Fraction immunized $= p$, Fraction not immunized $= 1 - p$, Population participating in disease $= N(1 - p)$, Effective intrinsic reproduction rate of disease (after immunization) $= R_0' = (1 - p)R_0$.

Thus

$$R_0' < 1 \Rightarrow (1 - p)R_0 < 1 \Rightarrow p > 1 - \frac{1}{R_0}$$

The percentage of the population to be vaccinated thus depends strongly on the infectiousness of the disease. It is noteworthy that smallpox, a disease essentially eradicated by vaccination, has one of the lowest R_0 values and a correspondingly low required vaccination fraction. By contrast, measles and whooping cough require a much higher percentage of immunization and would be harder to eradicate.

Average Age of Acquiring a Disease

Is it always wise to vaccinate at least some people, even if the disease will not be eradicated? When a disease has different impacts on individuals of different ages, vaccination at a young age can have a somewhat surprising deleterious effect. A case in point is German measles (Rubella). Normally a mild short-lasting infection, Rubella can be particularly devastating when contracted by a pregnant woman, as it results in birth defects to the fetus during the first trimester of pregnancy. Vaccinating *against* Rubella raises the average age at which the disease is first acquired (see box). Thus, rather than incurring the disease on average at age 12, it may be more prevalent at ages 20 to 30, precisely the most dangerous period for women of childbearing ages.

How Vaccinations Raise the Age of First Acquiring a Disease

Define $\lambda = \beta I$. Called the *per capita force of infection*, λ has units of 1/time and is the rate of acquiring the disease given a population containing I infectives and a transmission constant β.

Let $A = 1/\lambda$. A is the *average age of first infection*, or average waiting time in the susceptible compartment before acquiring the disease. A has units of time.

Now note that a vaccination program tends to reduce the number of infectives I, thus reducing λ and *raising* A.

For other aspects of the topic of vaccinations, epidemiology, and population dynamics, the many excellent sources quoted in the references are highly recommended.

PROBLEMS*[1]

1. **(a)** Assuming that $N(0) = N_0$, integrate equation (2a) and show that its solution is given by equation (2b). (See problem 5 of Chapter 4 or Braun, 1979; sec. 1.5.)

(b) Show that the solution given by (2b) has the following properties:

(1) $N \to K$ as $t \to \infty$.

(2) The graph is concave up for $N_0 < N < K/2$.

(3) The graph is concave down for $K/2 < N < K$.

(4) If $N_0 > K$, the graph is concave up.

2. Consider the model

$$\frac{dN}{dt} = rN(K - N)(N - M) \qquad \text{where } r > 0 \text{ and } 0 < M < K$$

(a) Express the intrinsic growth rate $g(N)$ as a polynomial in N and find the coefficients a_1, a_2, a_3.

(b) Show that $N = 0$, $N = K$, and $N = M$ are steady states and determine their stability.

(c) Solve the equation and graph the solution.

3. Models that are commonly used in fisheries are

$$\frac{dN}{dt} = Ng(N),$$

where $g(N)$ is given by

$$\text{Ricker model:} \qquad g(N) = re^{-\beta N}.$$

$$\text{Beverton-Holt:} \qquad g(N) = \frac{r}{\alpha + N}.$$

Analyze the behavior of the solutions to these questions. (Assume α, β, $r > 0$).

4. For single-species populations, which of the following density-dependent growth rates would lead to a decelerating rate of growth as the population increases? Which would result in a stable population size?

(a) $g(N) = \dfrac{\beta}{1 + N}$, $\beta > 0$.

(b) $g(N) = \beta - N$, $\beta > 0$.

(c) $g(N) = N - e^{\alpha N}$, $\alpha > 0$.

(d) $g(N) = \log N$.

5. List the assumptions that underlie the logistic equation (2a). You may wish to think about such factors as environmental or individual variability, reproductive ages, and the effects of the spatial distribution of the population. Which assumptions are not generally valid?

*Problems preceded by an asterisk are especially challenging.

1. Several problems were kindly suggested by C. Biles.

6. The factor $g(N) = r(1 - N/K)$ in equation (2a) is a per capita growth rate. Smith (1963) observed that in cultures of the unicellular alga *Daphnia magna* g decreases at a nonlinear rate as N increases. To account for this fact, Smith suggested that the growth rate depends on the rate at which food is utilized:

$$g(N) = r\frac{T - F}{T}$$

where F is the rate of utilization when the population size is N, and T is the maximal rate, when the population has reached a saturated level. He further assumed that

$$F = c_1 N + c_2 \frac{dN}{dt}, \qquad (c_1, c_2 > 0)$$

as long as $dN/dt > 0$.

(a) Explain this assumption for F.

(b) Show that the modified logistic equation is then

$$\frac{dN}{dt} = rN\left[\frac{K - N}{K + (\gamma N)}\right],$$

where $\gamma = rc_2/c_1$ and $K = T/c_1$.

(c) Sketch the expression in square brackets as a function of N.

(d) What would be the qualitative behavior of this population growth? (For a deeper analysis of this problem see Pielou, 1977.)

7. **(a)** Show that equations (6a,b,c) for the Gompertz growth law are equivalent. Find κ in terms of α.

(b) In a tumor the cells without access to nutrients and oxygen stop reproducing and generally die, leaving a *necrotic center*. The Gompertz growth law can be interpreted as a description of this necrosis. Discuss this point and give alternative interpretations of equations (6a,b).

8. Suppose that prey have a refuge from predators into which they can retreat. Assume the refuge can hold a fixed number of prey. How would you model this situation, and what predictions can you make?

9. **(a)** Suppose a one-time fishing expedition reduced the prey population by 10% of its current level. What does the Lotka-Volterra model predict about the subsequent behavior of the system? (*Note:* this prediction is one of the most objectionable features of the model and will be dealt with in a later chapter.)

(b) Now consider the situation in which there is a constant level of fishing in which both prey and predatory fish are caught and removed at rates proportional to their densities, ϕx and ϕy. Compare this to the situation in the absence of fishing, and show what Volterra concluded about d'Ancona's observation. (For one treatment of this problem see Braun, 1979; a more advanced mathematical treatment can be found in Brauer and Soudack, 1979.)

***10.** In Section 6.2 we showed that the steady state $(\bar{x}_2, \bar{y}_2) = (c/d, a/b)$ is associ-

ated with pure imaginary eigenvalues. Since the equations are nonlinear, it is necessary to consider the possibility that the steady state is a spiral point.

(a) Write the system of equations in the form

$$\frac{dy}{dx} = \frac{-cy + dxy}{ax - bxy}.$$

Separate variables and integrate both sides to obtain

$$y^a e^{-by} = Kx^c e^{-dx}$$

(where K is an arbitrary constant).

(b) On the nullcline $x = c/d$ observe that

$$y^a e^{-by} = \text{constant}.$$

Graph the function $f(y) = y^a e^{-by}$ and use your graph to demonstrate that $f(y) =$ constant can have at most two solutions for any given constant.

(c) Conclude that the trajectory cannot be a spiral. (*Hint:* Consider how many times it intersects the line $x = c/d$.)

11. Interpret the assumptions 1 to 4 made in the Kolmogorov equations for a predator-prey system.

12. **(a)** At the end of Section 6.2 a number of modifications of the Lotka-Volterra equations are described. Explain these modifications, paying particular attention to their predictions for low and high values of the prey population x.

(b) For each modification you discuss in (a), assume it is the only change made in equations (7a,b) and determine the effect on steady states and their stability properties.

13. Show it is possible, by introducing dimensionless variables, to rewrite the Lotka-Volterra equations as

$$\frac{dv}{dt} = v(1 - e), \qquad \frac{de}{dt} = \alpha e(v - 1),$$

where $v =$ victims and $e =$ exploiters.

14. Determine whether the nontrivial steady state of equations (8a,b) is a stable node or a stable spiral.

15. In this problem we attend to several details that arise in the species competition model [equations (9a,b)].

(a) The four cases shown in Figure 6.6 correspond to four possible sets of inequalities satisfied by the parameters κ_1, κ_2, β_{12}, and β_{21}. Give a biological interpretation of these relations.

(b) Show that a fourth steady state to equations (9a,b) is

$$(\bar{x}, \bar{y}) = \left(\frac{\beta_{21}\kappa_1 - \kappa_2}{\beta_{21}\beta_{12} - 1}, \frac{\beta_{12}\kappa_2 - \kappa_1}{\beta_{21}\beta_{12} - 1}\right),$$

and demonstrate that this steady state has biological relevance only in cases 3 and 4.

(c) The Jacobian of (9a,b) has off-diagonal elements as follows:

$$\mathbf{J} = \begin{pmatrix} ? & \dfrac{-r_1\beta_{12}N_1}{\kappa_1} \\ \dfrac{-r_2\beta_{21}N_2}{\kappa_2} & ? \end{pmatrix}_{(\bar{N}_1, \bar{N}_2)}.$$

Verify this fact, find the remaining entries, and then compute **J** for each of the steady states of (9a,b). (*Note:* one of these involves rather cumbersome expressions.)

(d) Verify the stability properties of steady states to the species competition model [equations (9a,b)] in the four cases discussed in Section 6.3.

(e) Give a biological interpretation of cases 1 through 4 in Figure 6.8.

(1) What is the outcome of competition in case 1? How does this differ from case 3?

(2) In cases 1, 2, and 4 the final outcome does not depend on the initial levels of the competing populations. This is not true in case 3. Give a rough rule of thumb for determining which species wins in case 3.

***16.** In this problem you are asked to generalize the competition model of equations (9a,b) to the case of k species, with particular emphasis on $k = 3$.

(a) Write a set of equations for the populations of species 1, 2, . . . , k that compete pairwise as in equations (9a,b).

(b) For $k = 3$ how many steady states are possible? (*Hint:* in the absence of a third species, each pair behaves according to the original two-species competition model.)

(c) The accompanying figure sketches the case roughly corresponding to case 4. Now suppose that the populations are such that (1) species 2 wins

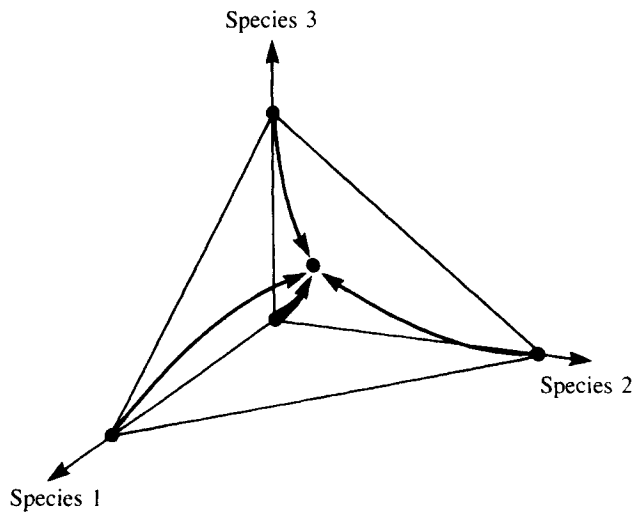


Figure for problem 16(c).

when 3 is absent; (2) species 3 wins when 1 is absent; and (3) species 1 wins when 2 is absent. Sketch the expected dynamics.

17. Species may derive mutual benefit from their association; this type of interaction is known as *mutualism*. May (1976) suggests the following set of equations to describe a possible pair of mutualists:

$$\frac{dN_1}{dt} = rN_1 \frac{1 - N_1}{\kappa_1 + \alpha N_2}, \qquad \frac{dN_2}{dt} = rN_2 \frac{1 - N_2}{\kappa_2 + \beta N_1},$$

where N_i is the population of the ith species, and $\alpha\beta < 1$.

(a) Explain why the equations describe a mutualistic interaction.

(b) Determine the qualitative behavior of this model by phase-plane and linearization methods.

(c) Why is it necessary to assume that $\alpha\beta < 1$?

18. Write down all Routh-Hurwitz matrices $\mathbf{H}_1$, $\mathbf{H}_2$, and $\mathbf{H}_3$ for the case of three species. Show that May's conditions are equivalent to the original Routh-Hurwitz criteria by evaluating the determinants of these matrices.

19. Suppose that in the three-species model discussed in example 1 (Section 6.4), species x and z are competitors. How would the model and its conclusions change?

20. Suppose that in the same example species y is also a prey of species z. How would the model and its conclusions change?

21. In the accompanying directional graph, arrows represent positive and negative effects that each of three species exert on each other when they are at equilibrium. For example, the effect of y on x is negative. Use the Routh-Hurwitz criteria to show that this system must be unstable.

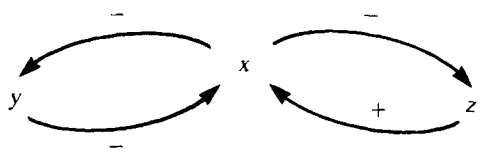


Figure for problem 21.

22. Use the Routh-Hurwitz criteria to investigate stability in problem 29 of Chapter 4.

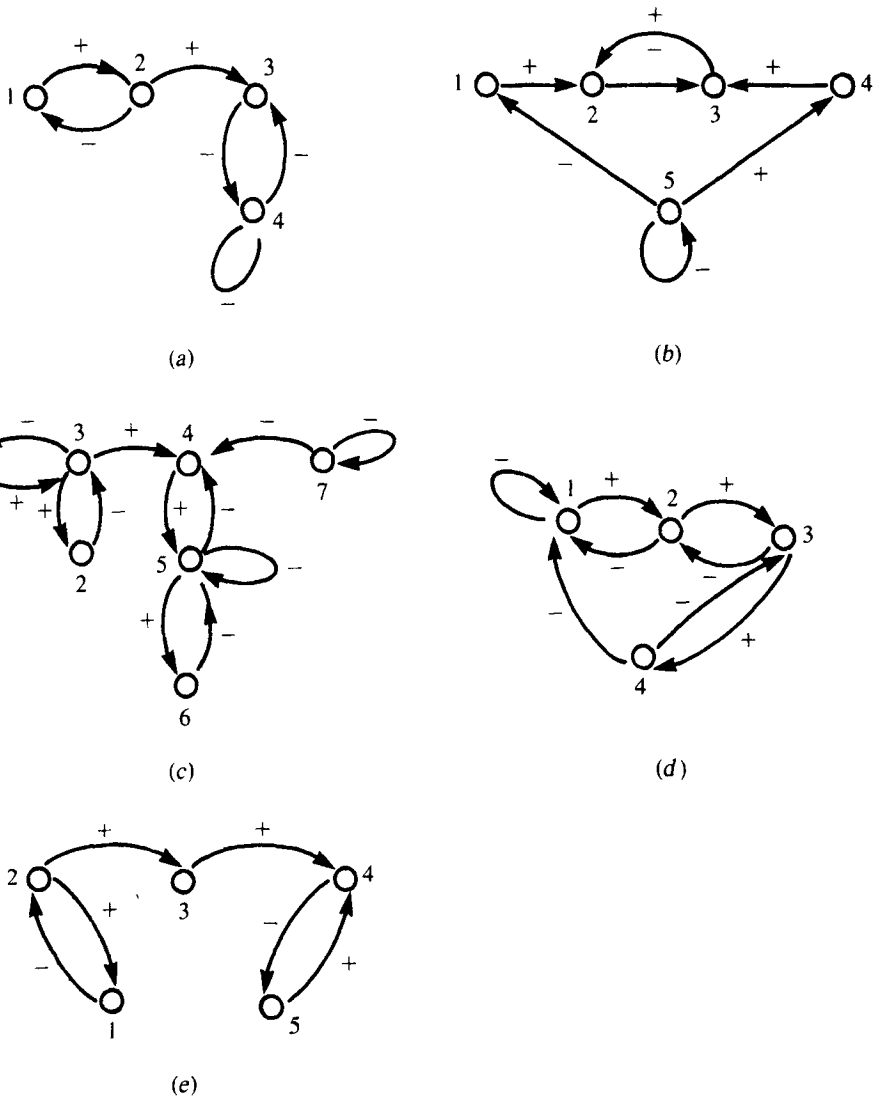
23. Analyze the community structures shown in the accompanying figures in the following way:

(1) Give the patterns of signs in the qualitative matrix $\mathbf{Q}$ that describes the system.

(2) Identify predation communities.

(3) Determine whether or not the system is qualitatively stable. If not, identify which condition(s) it does not satisfy.

(4) Suggest what kind of community might be represented by the graph.



Figures (a–e) for problem 23.

24. Draw directed graphs, identify predation communities, and determine whether the systems depicted in the following qualitative matrices are stable or not.

(a) $\begin{bmatrix} 0 & + & 0 & 0 \\ - & + & 0 & - \\ 0 & 0 & 0 & + \\ 0 & + & - & 0 \end{bmatrix}$ (Levins, 1977: one of two competitors for a common resource is itself preyed on: the other is resistant.)

(b) $\begin{bmatrix} 0 & 0 & 0 & + \\ - & 0 & + & + \\ + & - & - & 0 \\ - & - & 0 & - \end{bmatrix}$ (Levins, 1974: nutrients and organisms in a lake. Two species are algae; the others represent nutrients, one of which is produced by an alga.)

(c) $\begin{bmatrix} - & - & 0 & 0 & + \\ + & - & - & 0 & 0 \\ 0 & + & - & - & 0 \\ 0 & 0 & + & - & - \\ - & 0 & 0 & + & 0 \end{bmatrix}$ Unlikely food chain.

(d) $\begin{bmatrix} 0 & - & 0 & 0 \\ + & - & 0 & 0 \\ + & 0 & - & + \\ 0 & + & - & 0 \end{bmatrix}$ A larval prey and predator whose roles reverse in adulthood.

(e) $\begin{bmatrix} - & 0 & 0 & 0 & 0 \\ + & - & 0 & 0 & 0 \\ + & 0 & - & 0 & 0 \\ 0 & + & 0 & 0 & - \\ 0 & 0 & + & - & 0 \end{bmatrix}$ A species that can exist in two types that compete in adulthood.

25. In this problem you are asked to verify several results quoted in Section 6.6.

(a) Show that $N = S + I + R$ is constant for the *SIRS* model given by equations (28a,b,c).

(b) Verify that the two steady states of (28) are given by (29a,b).

(c) Suppose that all members of a population give birth to susceptibles (at rate δ) and die (at rate δ). How would the equations change?

(d) Find steady states for part (c), and determine what the infectious contact number is in terms of the parameters.

(e) Compare the model with and without the above vital dynamics.

***26.** Analyze an *SIR* model with and without vital dynamics. Verify the results summarized in Table 6.1.

***27.** Show that in an *SIR* model with disease fatality at rate η the disease will always eventually disappear.

***28.** Show that in an *SIR* model with carriers who show no symptoms of the disease, the disease always remains endemic.

29. Capasso and Serio (1978) considered the following model with emigration of susceptibles:

$$\frac{dS}{dt} = -g(I)S - \lambda S,$$

$$\frac{dI}{dt} = g(I)S - \gamma I,$$

$$\frac{dR}{dt} = \lambda S + \gamma I.$$

The function $g(I)$, shown in the accompanying graph, takes into account "psychological" effects. Explain the equations and show that the epidemic will always tend to extinction with respect to both infectives and susceptibles.

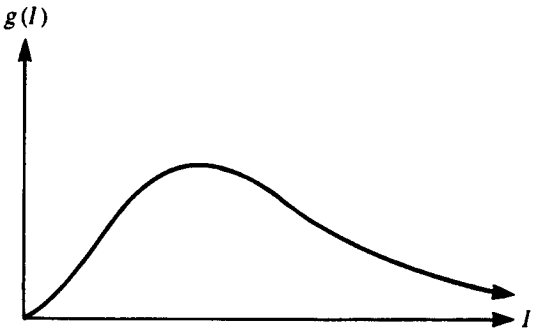


Figure for problem 29.

30. Anderson and May (1979) describe a model in which the natural birth and death rates, a and b, are not necessarily equal so that the disease-free population may grow exponentially:

$$\frac{dN}{dt} = (a - b)N.$$

The disease increases mortality of infected individuals (additional rate of death by infection = α), as shown in the accompanying figure. In their terminology the population consists of the following:

X = susceptible class (= S),
Y = infectious class (= I),
Z = temporarily immune class (= R).

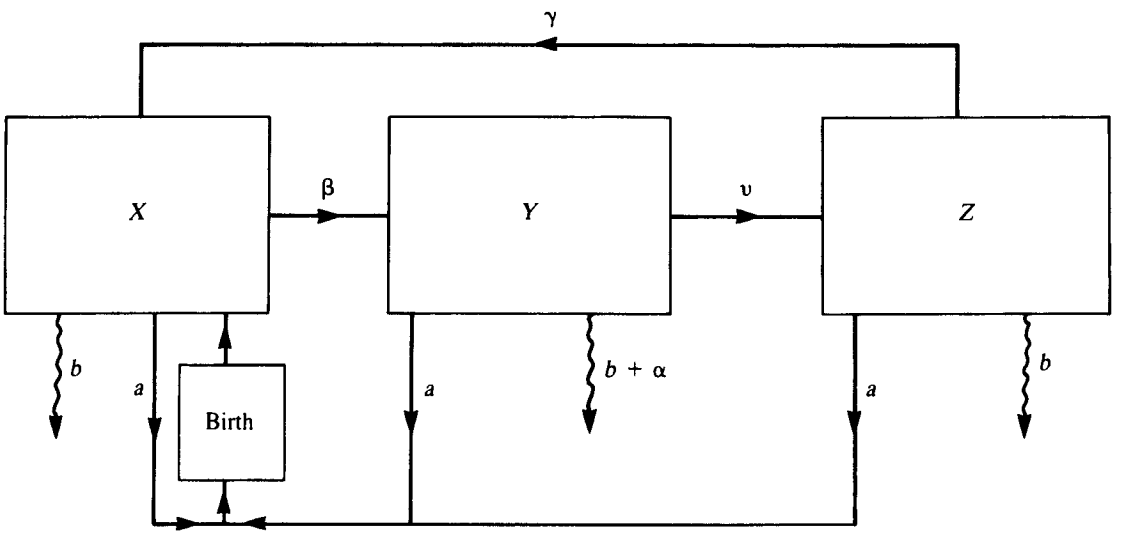


Figure for problem 30.

(a) Write equations describing the disease.
(b) Show that the steady-state solution representing the presence of disease is given by

$$\bar{X}_2 = \frac{\alpha + b + \nu}{\beta}, \qquad \bar{Y}_2 = \frac{r}{\alpha} N, \qquad \bar{Z}_2 = \frac{r}{\alpha}\left(\frac{\nu}{b + \gamma}\right) N_2,$$

where

$$N_2 = \frac{\alpha(\alpha + b + \nu)}{\beta(\alpha - r[1 + \nu/(b + \gamma)])}, \qquad \text{and} \qquad r = a - b.$$

(c) The steady state in part (b) makes biological sense whenever

$$\alpha > r\left(1 + \frac{\nu}{b + \gamma}\right).$$

Interpret this inequality.
(*Note:* Anderson and May conclude that disease can be a regulating influence on the population size.)

31. Consider a lake with some fish attractive to fishermen. We wish to model the fish-fishermen interaction.

Fish Assumptions:

i. Fish grow logistically in the absence of fishing.

ii. The presence of fishermen depresses fish growth at a rate jointly proportional to the fish and fishermen populations.

Fishermen Assumptions:

i. Fishermen are attracted to the lake at a rate directly proportional to the amount of fish in the lake.

ii. Fishermen are discouraged from the lake at a rate directly proportional to the number of fishermen already there.

(a) Formulate, analyze, and interpret a mathematical model for this situation.

(b) Suppose the department of fish and game decides to stock the lake with fish at a constant rate. Formulate, analyze, and interpret a mathematical model for the situation with stocking included. What effect does stocking have on the fishery?

32. Beddington and May (1982) have proposed the following model to study the interactions between baleen whales and their main food source, *krill* (a small shrimp-like animal), in the southern ocean:

$$\dot{x} = rx\left(1 - \frac{x}{K}\right) - axy$$

$$\dot{y} = sy\left(1 - \frac{y}{bx}\right)$$

Here the whale carrying capacity is not constant but is a function of the krill population:

$$K_{\text{whales}} = bx$$

Analyze this model by determining the steady states and their stability; include a phase-plane diagram.

33. *Estimating parameters in the logistic equation:* We would like to address the curve-fitting problem of how to find the logistic equation of best fit when given appropriate data. First, rearrange equation (2b) and show that

$$N(t) = \frac{K}{1 + [(K - N_0)/N_0]e^{-rt}}.$$

Then conclude that

$$N(t) = \frac{K}{1 + \exp\{-rt + \ln[(K - N_0)/N_0]\}}.$$

Now rearrange the above to demonstrate that

$$\frac{K - N}{N} = \exp\left(-rt + \ln\frac{K - N_0}{N_0}\right).$$

Thus

$$\ln\frac{K - N}{N} = -rt + \ln\frac{K - N_0}{N_0}.$$

Now the quantity $\ln[(K - N)/N]$ is a linear function of time with slope r. The following data is given in Gause (1969) for the growth in volume of each of the yeasts *Saccharomyces* and *Schizosaccharomyces* growing separately. (See Figure 6.9, top curves in each graph.)

Age (h)	*(1) Saccharomyces*	*(2) Schizosaccharomyces*
6	0.37	—
16	8.87	1.00
24	10.66	—
29	12.50	1.70
40	13.27	—
48	12.87	2.73
53	12.70	—
72	—	4.87
93	—	5.67
141	—	5.83

(a) Use the maximal population levels to estimate K_1 and K_2, the carrying capacities for each of the two species.

(b) Plot $(K_i - N_i)/N_i$ on a log scale and use this to determine r_1 and r_2. Do your values agree with those given by Gause in Figure 6.9?

34. Estimating the species-competition parameters.

(a) Use equations (9a,b) to show that

$$\beta_{12} = \frac{K_1 - (dN_1/dt)K_1 - N_1}{r_1 N_1 N_2},$$

$$\beta_{21} = \frac{K_2 - (dN_2/dt)K_2 - N_2}{r_2 N_1 N_2}.$$

(b) Suggest how such parameters might be estimated given empirical observations of N_1 and N_2 growing in a mixed population. (See Gause for experimental data.)

(c) The values of all parameters determined by Gause are given in Section 6.3, Figure 6.9. Determine whether the two species can coexist in a stable mixed population or if one wins over the other.

35. Populations of lemmings, voles, and other small rodents are known to fluctuate from year to year. Early Scandinavians believed the lemmings to fall down from heaven during stormy weather. Later in history, the legend developed that they migrate periodically into the sea for suicide in order to reduce their numbers. . . . None of these theories, however, was supported by any accurate observations. (H. Dekker, 1975)

An alternate hypothesis was suggested by Dekker to account for rodent population cycles. His theory is based on the idea that the rodents fall into two genotypic classes (Myers and Krebs, 1971) that interact. Type 1 reproduces rapidly, but migrates in response to overcrowding; type 2 is less sensitive to high densities but has a lower reproductive capacity.

The following simple mathematical model was given by Dekker to demonstrate that oscillations could be produced when types 1 and 2 rodents were both present in the population:

$$\frac{dn_1}{dt} = n_1[a_1 - (b_1 - c_1)n_2 - c_1(n_1 + n_2)],$$

$$\frac{dn_2}{dt} = n_2[-a_2 + b_2 n_1],$$

where

n_1 = density per acre of type 1,

n_2 = density per acre of type 2.

The term $b_1 - c_1$ was chosen for convenience in the mathematical calculations rather than for particular biological reasons.

(a) On the basis of the information, give an interpretation of the individual terms in the equations.

(b) Using phase-plane methods, determine the qualitative behavior of solutions to Dekker's equations. If there is more than one case, pay particular attention to the case in which oscillations are present. Give conditions on the parameters a_j, b_j, and c_j for which oscillatory behavior will be seen.

(c) Give a short critique of Dekker's model, indicating whether you would change his assumptions and/or equations. Dekker's article has received a somewhat critical peer review by Nichols et. al. (1979). You may wish to comment on their specific points of contention.

REFERENCES

Single Species Growth

Lamberson, R., and Biles, C. (1981). Polynomial models of biological growth, *UMAP Journal, 2*(2), 9–25.

Malthus, T. R. (1798). An essay on the principle of population, and A summary view of the principle of population. Penguin, Harmondsworth, England.

Pearl, R., and Reed, L. J. (1920). On the rate of growth of the population of the United States since 1790 and its mathematical representation. *Proc. Natl. Acad. Sci. USA, 6,* 275–288.

Slobodkin, L. B. (1954). Population dynamics in *Daphnia obtusa* Kurz. *Ecol. Monogr., 24,* 69–88.

Smith, F. E. (1963). Population dynamics in *Daphnia magna. Ecology, 44,* 651–663.

Verhulst, P. F. (1838). Notice sur la loi que la population suit dans son accroissement. *Correspondance Mathematique et Physique, 10,* 113–121.

Predator-Prey Interactions and Species Competition

Armstrong, R. A., and McGehee, R. (1980). Competitive exclusion. *Am. Nat., 115*(2), 151–170.

Brauer, F., and Soudack, A. C. (1979). Stability regions and transition phenomena for harvested predator-prey systems. *J. Math. Biol., 7,* 319–337.

Braun, M. (1979). *Differential Equations and Their Applications*. 3d ed., Springer-Verlag, New York.

Braun, M. (1983). Chaps. 4, 5, 15, 17 in M. Braun, C. S. Coleman, and D. A. Drew, eds. *Differential Equation Models*. Springer-Verlag, New York.

Coleman, C. S. (1978). Biological cycles and the fivefold way. In M. Braun, C. S. Coleman, and D. A. Drew, eds., *Differential Equation Models,* Springer-Verlag, New York.

Gause, G. F. (1932). Experimental studies on the struggle for existence. I. Mixed population of two species of yeast. *J. Exp. Biol., 9,* 389–402.

Gause, G. F. (1934). *The Struggle for Existence*. Hafner Publishing, New York. (Reprinted 1964, 1969).

Holling, C. S. (1965). The functional response of predators to prey density and its role in mimicry and population regulation. *Mem. Entomol. Soc. Can., 45,* 1–60.

Ivlev, V. S. (1961). *Experimental Ecology of the Feeding of Fishes*. Yale University Press, New Haven, Conn.

Kolmogorov, A. (1936). Sulla Teoria di Volterra della Lotta per l'Esistenza. *G. Ist. Ital. Attuari, 7,* 74–80.

Lotka, A. J. (1925). *Elements of Physical Biology*. Williams & Wilkins, Baltimore.

May, R. (1973). *Stability and Complexity in Model Ecosystems*. Princeton University Press, Princeton, N. J.

May, R. (1976). *Theoretical Ecology, Principles and Applications*. Saunders, Philadelphia.

Odum, E. P. (1953). *Fundamentals in Ecology*. Saunders, Philadelphia.

Pielou, E. C. (1969). *An Introduction to Mathematical Ecology*. Wiley-Interscience, New York. (Reprinted 1977).

Rosenzweig, M. L. (1969). Why the prey curve has a hump. *Am. Nat. 103,* 81–87.

Rosenzweig, M. L. (1971). Paradox of enrichment: Destabilization of exploitation ecosystems in ecological time. *Science, 171,* 385–387.

Roughgarden, J. (1979). *Theory of Population Genetics and Evolutionary Ecology: An Introduction*. Macmillan, New York.

Schoener, T. W. (1973). Population growth regulated by intraspecific competition for energy or time. *Theor. Pop. Biol., 4,* 56–84.

Takahashi, F. (1964). Reproduction curve with two equilibrium points: A consideration on the fluctuation of insect population. *Res. Pop. Ecol., 6,* 28–36.

Van der Vaart, H. R. (1983). Some examples of mathematical models for the dynamics of several-species ecosystems. Chap. 4 in H. Marcus-Roberts and M. Thompson, eds. *Life Science Models*. Springer-Verlag, New York.

Volterra, V. (1926). Variazioni e fluttuazioni del numero d'individui in specie animal conviventi. *Mem. Acad. Lincei., 2,* 31–113. Translated as an appendix to Chapman, R. N. (1931). *Animal Ecology*. McGraw-Hill, New York.

Volterra, V. (1931). *Leçons sur la theorie mathematique de la lutte pour la vie*. Gauthier-Villars, Paris.

Volterra, V. (1937). Principe de biologie mathematique. *Acta Biotheor., 3,* 1–36.

Whitaker, R. H., and Levin, S. A., eds. (1975). *Niche: Theory and Application*. Dowden, Hutchinson & Ross, New York.

Qualitative Stability

Jeffries, C. (1974). Qualitative stability and digraphs in model ecosystems. *Ecology, 55,* 1415–1419.

Levins, R. (1974). The qualitative analysis of partially specified systems. *Ann. N. Y. Acad. Sci., 231,* 123–138.

Levins, R. (1977). Qualitative analysis of complex systems, in D. E. Matthews, ed., *Mathematics and the Life Sciences. Lecture Notes in Biomathematics,* vol. 18, 153–199. Springer-Verlag, New York.

Roberts, F. S. (1976). *Discrete Mathematical Models, with Applications to Social, Biological and Environmental Problems*. Prentice-Hall, Englewood Cliffs, N. J.

Quirk, J., and Ruppert, R. (1965). Qualitative economics and the stability of equilibrium. *Rev. Econ. Stud., 32,* 311–326.

Mathematical Theory of Epidemics

Anderson, R. M., ed. (1982). *Population Dynamics of Infectious Diseases, Theory and Applications*. Chapman & Hall, New York.

Anderson, R. M., and May, R. M. (1979). Population biology of infectious diseases, part I. *Nature, 280,* 361–367; part II, *Nature, 280,* 455–461.

Busenberg, S. N., and Cooke, K. L. (1978). Periodic solutions of delay differential equations arising in some models of epidemics. In *Proceedings of the Applied Nonlinear Analysis Conference, University of Texas*. Academic Press, New York.

Capasso, V., and Serio, G. (1978). A generalization of the Kermack-McKendrick deterministic epidemic model. *Math. Biosci., 42,* 43–61.

Hethcote, H. W.; Stech, H. W.; and van den Driessche, P. (1981). Periodicity and stability in epidemic models: A Survey. In *Differential Equations and Applications in Ecology, Epidemics, and Population Models*. Academic Press, New York, pp. 65–82.

Hethcote, H. W. (1976). Qualitative analyses of communicable disease models. *Math. Biosci., 28,* 335–356.

Kermack, W. O., and McKendrick, A. G. (1927). Contributions to the mathematical theory of epidemics. *Roy. Stat. Soc. J., 115,* 700–721.

May, R. M., (1983). Parasitic infections as regulators of animal populations. *Am. Sci., 71,* 36–45.

Vaccination Programs

Anderson, R. M., and May, R. M. (1982). The logic of vaccination. *New Scientist,* (November 18, 1982 issue) pp. 410–415.

Anderson, R. M., and May, R. M. (1983). Vaccination against rubella and measles: Quantitative investigations of different policies. *J. Hyg. Camb., 90,* 259–325.

May, R. M. (1982). Vaccination programmes and herd immunity. *Nature, 300,* 481–483.

Miscellaneous

Aroesty, J., Lincoln, T., Shapiro, N., and Boccia, G. (1973). Tumor growth and chemotherapy: mathematical methods, computer simulations, and experimental foundations. *Math. Biosci., 17,* 243–300.

Beddington, J. R., and May, R. M. (1982). The harvesting of interacting species in a natural ecosystem. *Sci. Am.,* November 1982, pp. 62–69.

Dekker, H. (1975). A simple mathematical model of rodent population cycles. *J. Math. Biol., 2,* 57–67.

Freedman, H. I. (1980). *Deterministic Mathematical Models in Population Ecology.* Marcel Dekker, New York.

Greenwell, R. (1982). *Whales and Krill: A Mathematical Model.* UMAP module unit 610. COMAP, Lexington, Mass.

Hofbauer, J. (in press). Permanence and persistence of Lotka-Volterra systems.

Hutchinson, G. E. (1978). *Introduction to Population Ecology.* Yale University Press, New Haven, Conn.

Kingsland, S. E. (1985). *Modelling Nature.* University of Chicago Press, Chicago.

Levins, R. (1968). *Evolution in Changing Environments.* Princeton University Press, Princeton.

Luenberger, D. G. (1979). *Introduction to Dynamic Systems.* Wiley, New York, pp. 328–331.

May, R. M., Beddington, J. R., Clark, C. W., Holt, S. J., and Laws, R. M. (1979). Management of multispecies fisheries. *Science, 205,* 267–277.

Myers, J. H., and Krebs, C. J. (1971). Genetic, behavioral, and reproductive attributes of dispersing field voles *Microtus pennsylvanicus* and *Microtus Ochrogaster. Ecol. Monogr., 41,* 53–78.

Newton, C. M. (1980). Biomathematics in oncology: modelling of cellular systems. *Ann. Rev. Biophys. Bioeng., 9,* 541–579.

Nichols, J. D.; Hestbeck, J. B.; and Conley, W. (1979). Mathematical models and population cycles: A critical evaluation of a recent modelling effort. *J. Math. Biol., 8,* 259–263.

Nisbet, R. M., and Gurney, W. S. C. (1982). *Modelling Fluctuating Populations.* Wiley, New York.