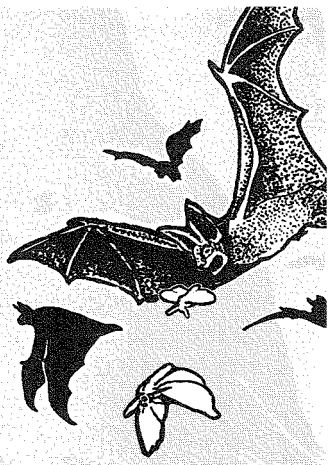
CHAPTER 6

Predation



Model Presentation and Predictions

Competitive interactions in nature are often indirect and subtle, and may be mediated through populations of resources. In contrast, predation is a direct and conspicuous ecological interaction. The image of a wolf pack bringing down a moose, or a spider eating a fly evokes Tennyson's description of "nature red in tooth and claw." Seed predators, such as finches and harvester ants, are less dramatic in their feeding, but may be equally effective at controlling plant populations. Other animals do not consume their prey entirely. Parasites require that their hosts survive long enough for the parasite to reproduce, and many herbivores graze on plants without killing them. In all of these interactions, we can recognize a population of "predators" that benefits from feeding, and a population of "victims" that suffers. In this chapter, we will develop some simple models to give us insight into the dynamics of predation. As in our analysis of competition, the predation equations were first derived independently by Alfred J. Lotka and Vito Volterra. Volterra's interest in the subject stemmed from his daughter's fiancé, a fisheries biologist who was trying to understand fluctuations in the catch of predaceous fish (Kingsland 1985).

MODELING PREY POPULATION GROWTH

We will use the symbol P to denote the predator population and the symbol V to denote the victim or prey population. The growth of the victim population will be some function, f, of the numbers of both victims and predators:

$$\frac{dV}{dt} = f(V,P) \qquad \text{Expression 6.1}$$

Suppose that the predators are the only force limiting the growth of the victim population. In other words, if the predators are absent, the victim population increases exponentially:

$$\frac{dV}{dt} = rV \qquad \text{Expression 6.2}$$

with r representing the intrinsic rate of increase (see Chapter 1). This potential for increase of the victim population is offset by losses that occur when predators are present:

$$\frac{dV}{dt} = rV - \alpha VP \qquad \text{Equation 6.1}$$

The term after the minus sign says that losses to predation are proportional to the *product* of predator and victim numbers. This is equivalent to a chemical

reaction in which the reaction rates are proportional to the concentrations of molecules. If predators and victims move randomly through the environment, then their encounter rate is proportional to the product of their abundances. Note that we have now started recycling symbols: α is *not* the competition coefficient from Chapter 5! Instead, here α measures **capture efficiency**, the effect of a predator on the per capita growth rate $\left(\frac{1}{V}\frac{dV}{dt}\right)$ of the victim population.* The units of α are [victims/(victim • time • predator)]. The larger α is, the more the per capita growth rate of the victim population is depressed by the addition of a single predator. A filter-feeding baleen whale would have a large α, because a single whale can consume millions of plankton. In contrast, a web-building spider might have a fairly low α if the addition of a single web does not greatly depress prey populations. The product αV is the **functional response** of the predator—the rate of victim capture by a predator as a function of victim abundance (Solomon 1949). Later in this chapter, we will derive some more complicated expressions for the functional response, but for now we will represent it is as a simple product of victim abundance (V) and capture efficiency (α). Before we explore the solutions to the equation for victim growth, we will develop an analogous equation to describe the growth of the predator population.

MODELING PREDATOR POPULATION GROWTH

The growth of the predator population is affected by the numbers of both predators and victims:

$$\frac{dP}{dt} = g(P,V) \qquad \text{Expression 6.3}$$

We use the symbol g for this function to distinguish it from the function f that is used for the victim population in Expression 6.1.

The predator we are modeling is an extreme specialist. It will feed only on the victim population and has no alternative source of prey. Consequently, if the victim population is absent, the predator population declines exponentially:

$$\frac{dP}{dt} = -qP \qquad \text{Expression 6.4}$$

where q is the per capita **death rate**. (This is equivalent to the death rate d from the exponential growth model described in Chapter 1; we have changed symbols here to avoid confusion.)

*This same capture efficiency appeared as the interaction coefficient δ in Equation 5.8, where it represented losses to predation in a pair of competitors engaging in intraguild predation.

Positive growth occurs only when the victim population is present:

$$\frac{dP}{dt} = \beta VP - qP \qquad \text{Equation 6.2}$$

Here βVP indicates random encounters of predators and victims. β is a measure of **conversion efficiency***—the ability of predators to convert each new victim into additional per capita growth rate for the predator population $\left(\frac{1}{P}\frac{dP}{dt}\right)$. Its units are [predators/(predator • time • victim)]. We expect β to be high when a single prey item is particularly valuable, such as a moose that is captured by wolves. In contrast, β will be low when a single prey item does not contribute much to growth of the predator population; think of a single seed consumed by a granivorous bird. βV reflects the **numerical response** of the predator population—the per capita growth rate of the predator population as a function of victim abundance.

EQUILIBRIUM SOLUTIONS

To find the equilibrium for the victim and predator populations, we set each equation equal to zero and solve for population size. Beginning with Equation 6.1:

$$0 = rV - \alpha VP \qquad \text{Expression 6.5}$$

$$rV = \alpha VP \qquad \text{Expression 6.6}$$

$$r = \alpha P \qquad \text{Expression 6.7}$$

$$\hat{P} = \frac{r}{\alpha} \qquad \text{Equation 6.3}$$

Although we tried to solve for the victim equilibrium, the solution is in terms of P, the predator population! The important result is that a specific number of predators ($\hat{P}$) will maintain the victim population at zero growth. This predator level is determined by the ratio of the growth rate of the victims (r) to the capture efficiency of the predators (α). The faster the growth rate of the

*Again, this conversion efficiency appeared as the interaction coefficient γ in Equation 5.7 of Chapter 5, where it represented gains from predation in a pair of competitors engaging in intraguild predation.

victim population, the more predators are needed to keep the victim population in check. Conversely, the higher the capture efficiency, the fewer predators needed for control.

Solving the equilibrium for the predators (Equation 6.2) yields an expression in terms of the victim population size:

$$0 = \beta VP - qP \qquad \text{Expression 6.8}$$

$$\beta VP = qP \qquad \text{Expression 6.9}$$

$$\beta V = q \qquad \text{Expression 6.10}$$

$$\hat{V} = \frac{q}{\beta} \qquad \text{Equation 6.4}$$

Thus, the predator population is controlled by a fixed number of victims ($\hat{V}$). The greater the death rate of the predators (q), the more victims needed to keep the predator population from declining. Conversely, the greater the conversion efficiency of predators (β), the fewer victims needed to maintain the predators at equilibrium. Because Equations 6.3 and 6.4 give the conditions for zero growth, they represent the victim and predator isoclines, respectively.

GRAPHICAL SOLUTIONS TO THE LOTKA–VOLTERRA PREDATION MODEL

As in our analysis of the competition model (Chapter 5), we can plot the isoclines for each species in state space to evaluate the joint equilibrium. Plotting the victim population on the x axis yields a horizontal victim isocline, representing the number of predators needed to hold the victim population in check. If the predator population is less than this number, the victim population can increase in size, represented by horizontal arrows pointing to the right. Conversely, if the predator population is above the victim isocline, the victim population declines, represented by horizontal arrows pointing to the left (Figure 6.1).

Similar reasoning applies to the analysis of the predator isocline. This isocline is a vertical line, representing a critical size of the victim population. To the left of the isocline, there are not enough victims to support the predator population. In this region of the state-space graph, the predator population declines, represented by downward-pointing vertical arrows. To the right of the isocline, there is an excess supply of victims, and the predator population increases, represented by upward-pointing vertical arrows (Figure 6.2).

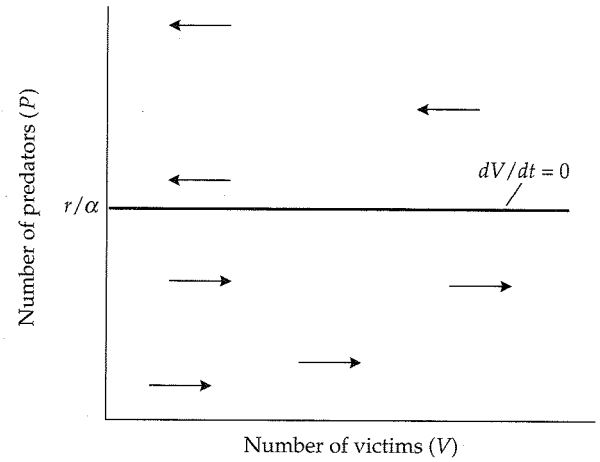


Figure 6.1 The victim isocline in state space. The Lotka–Volterra predation model predicts a critical number of predators (r/α) that controls the victim population. If there are fewer predators than this, the victim population increases (right-pointing arrows). If there are more predators, the victim population decreases (left-pointing arrows). The victim population has zero growth when $P = r/\alpha$.

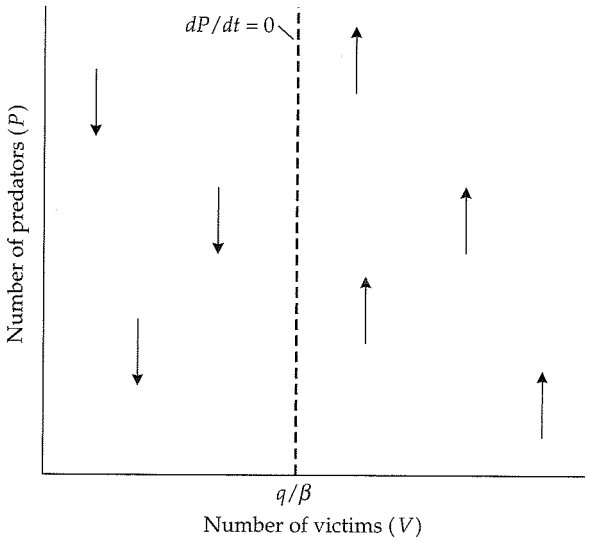


Figure 6.2 The predator isocline in state space. The Lotka–Volterra predation model predicts a critical number of victims (q/β) that controls the predator population. If there are fewer victims than this, the predator population decreases (downward-pointing arrows). If there are more victims, the predator population increases (upward-pointing arrows). The predator population has zero growth when $V = q/\beta$.

In our analysis of competition models, there were four ways that the pair of isoclines could be placed in the state-space graph. For the predation model, there is only one possible pattern: the isoclines cross at 90° angles (Figure 6.3). However, we will see that the dynamics are more complex than in the competition model.

The predator and victim isoclines divide the state space into four regions. Beginning in the upper right-hand corner, we are in a region where both predator and victim are abundant. Because we are to the right of the predator isocline, prey are abundant enough for the predator to increase. However, we are above the horizontal victim isocline. Consequently, there are too many predators, and the victim population declines. The vector of net movement points towards the upper left-hand quadrant. As the victim abundance continues to decline, we cross the vertical isocline into the upper left-hand region of state space.

Now the victim population has declined to the point where the predator population can no longer increase. Both predator and victim populations decrease, and the vector moves into the lower left-hand quadrant. In this region, the predator population continues to decline, but the victim population starts to increase again. The net movement is down and to the right, taking the trajectory into the fourth quadrant. Here, the victim population con-

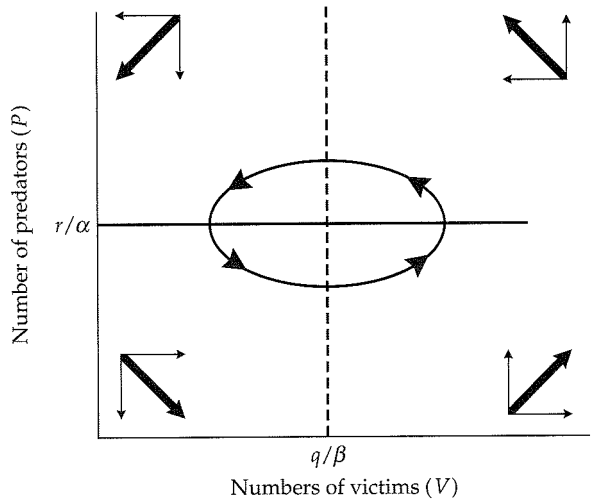


Figure 6.3 The dynamics of predator and victim populations in the Lotka–Volterra model. The vectors indicate the trajectories of the populations in the different regions of the state space. The populations trace a counterclockwise path that approximates an ellipse.

tinues to grow, but it has now become large enough for the predators to also increase. The system again moves back to the starting point, the upper right-hand quadrant.

Thus, the predator and victim populations trace an approximate ellipse in state space. Unless the predator and victim populations are precisely at the intersection of the isoclines, their trajectories will continue to move in this counterclockwise ellipse.

How does this ellipse translate into growth curves for the predator and victim populations? Both populations cycle periodically, increasing and decreasing smoothly from minimum to maximum. The ellipse indicates that the peak of the predator population occurs when the victim population is at its midpoint, and vice versa. In other words, the peaks of the predator and victim populations are displaced by one-quarter of a cycle (Figure 6.4).

What would happen if the predator and victim populations had a different starting point in the state space? This would correspond to different initial abundances of predator and victim, and a new ellipse would be traced. Both populations would again exhibit cycles, although with a different amplitude. The closer the ellipse is to the isocline intersection, the smaller the amplitude of the predator and victim cycles. Thus, the Lotka–Volterra cycles are neutrally stable—the amplitudes are determined solely by the initial conditions.

There are only two exceptions to population cycling: (1) if the victim and predator populations are precisely at the isocline intersection, they will not

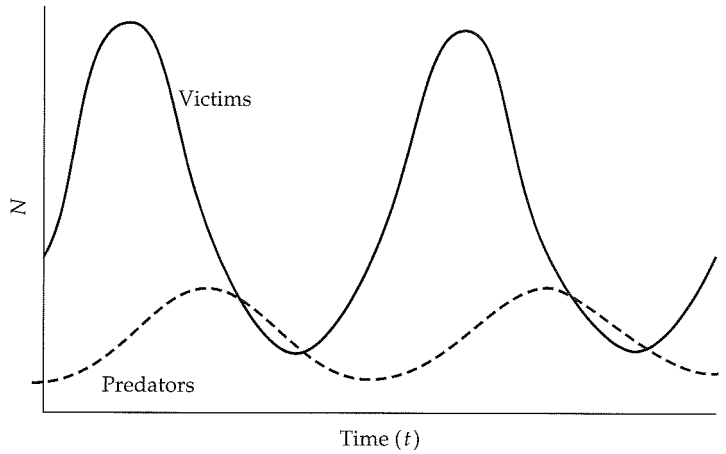


Figure 6.4 Cycles of predators and victims in the Lotka–Volterra model. Each population cycles with an amplitude that is determined by the starting population sizes and a period of approximately $2\pi/\sqrt{rq}$. The predator and victim populations are displaced by one-quarter of a cycle, so that the predator population peaks when the victim population has declined to half its maximum, and vice versa.

change, although if they are displaced any distance from this point, they will begin cycling; or (2) if the starting point of the ellipse is too extreme, it will hit one of the axes of the state-space graph. In this case, the amplitude of the cycle is so large that either predator or the victim population will crash. Although the amplitude of the cycle is determined by the initial population sizes, the period of the cycle (C) is approximately

$$C \approx \frac{2\pi}{\sqrt{rq}} \qquad \text{Equation 6.5}$$

Thus, the greater the prey growth rate (r) and/or the predator death rate (q), the faster the populations cycle between high and low values. The essential feature of the Lotka–Volterra predation model is that the predator and victim populations cycle because they reciprocally control one another's growth.

Model Assumptions

The Lotka–Volterra predation model carries with it the standard assumptions of no immigration, no age or genetic structure, and no time lags. In addition, the model makes the following assumptions about predators, victims, and the environment:

- ✓ ***Growth of the victim population is limited only by predation.*** Equation 6.1 shows that the victim population grows exponentially in the absence of the predator.
- ✓ ***The predator is a specialist that can persist only if the victim population is present.*** Equation 6.2 shows that the predator population will starve in the absence of the victim.
- ✓ ***Individual predators can consume an infinite number of victims.*** Because the horizontal victim isocline ($dV/dt = 0$) implies a constant number of predators, each predator must be able to increase its consumption as the victim population increases in size. An infinite capacity for consuming prey also implies that there is no interference or cooperation among predators.
- ✓ ***Predator and victim encounter one another randomly in an homogenous environment.*** The interaction terms (αVP and βVP) imply that predators and victims move randomly through the environment, and that victims do not have spatial or temporal refuges for avoiding predators.

Model Variations

The unique prediction of the Lotka–Volterra predation model is cycles of predator and victim populations. However, these cycles are very sensitive to the restrictive assumptions and linear isoclines of the model. In the following sections, we will incorporate more realistic assumptions about predators and victims that bend the isoclines and produce other dynamics. We will not solve the equations for these more complex models, although we will analyze their behavior with state-space graphs.

INCORPORATING A VICTIM CARRYING CAPACITY

The victim isocline tells us how many predators are needed to hold the victim population in check. Notice that as we move to the right in the state-space graph (Figure 6.1), the same number of predators will control the victim population. This is not realistic. We expect that as the victim population becomes more crowded, it will start to be limited by other resources that have nothing to do with predators. We can modify the victim isocline to incorporate a victim carrying capacity by including another term with a new constant c:

$$\frac{dV}{dt} = rV - \alpha VP - cV^2 \qquad \text{Equation 6.6}$$

Now the growth of the victim population is decreased by the presence of predators (αVP) and by its own abundance (cV^2). When graphed in the state space, this new isocline is a straight line with a negative slope, in contrast to the horizontal victim isocline of the simple Lotka–Volterra model. The new isocline crosses the x axis at r/c, which is the maximum population size achieved by the victims when no predators are present. In the absence of predators, Equation 6.6 is equivalent to a model of logistic population growth with a carrying capacity $K = r/c$ (Equation 2.1).

How does the interaction of predator and victim change when the victim population is limited by its own carrying capacity? Figure 6.5 shows that the trajectory for the predator and victim populations spirals inwards to the equilibrium intersection. This is a stable equilibrium point, and the equilibrium abundance for the victim population is lower when the predators are present than when they are absent. The presence of a victim carrying capacity stabilizes the predator–prey interaction. This makes intuitive sense—if the victims are limited by factors other than their predators, then there would be less of a tendency for the two populations to cycle.

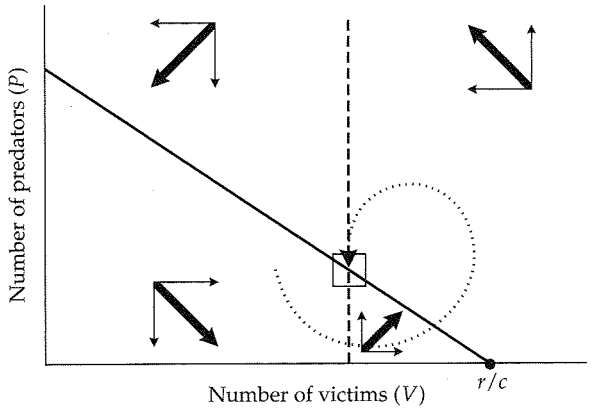


Figure 6.5 The effect of a victim carrying capacity on the victim isocline. The victim isocline slopes downward with a carrying capacity incorporated. The intersection with the vertical predator isocline forms a stable equilibrium point.

MODIFYING THE FUNCTIONAL RESPONSE

One of the most unrealistic assumptions of the Lotka–Volterra predation model is that individual predators can always increase their prey consumption as the victim population increases. This type of foraging is illustrated in a graph of the functional response (Figure 6.6), which plots the rate of prey

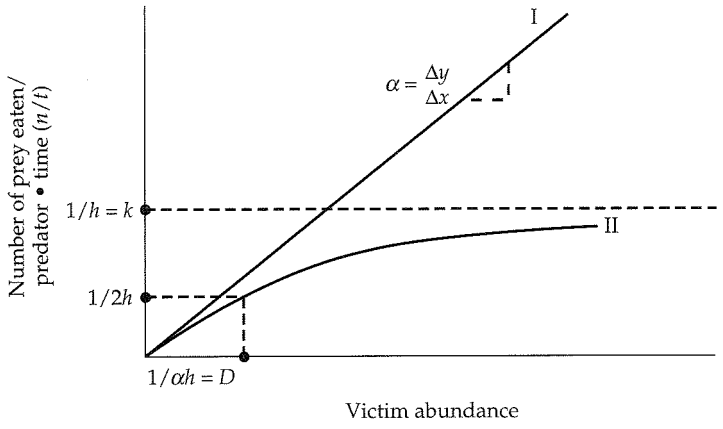


Figure 6.6 The functional response of predators is the feeding rate per predator as a function of prey abundance. The shape of these curves depends on the capture efficiency (α), the maximum predator feeding rate (k), and the victim abundance for which the predator feeding rate is half of the maximum (D).

captured per individual predator (n/t) as a function of prey abundance (V). The Lotka–Volterra model assumes a **Type I functional response**, in which the predator consumes more as prey abundance increases (Holling 1959). The slope of this curve is α, the capture efficiency.

A Type I functional response is unrealistic for two reasons. First, predators will eventually become satiated (stuffed) and stop feeding. Second, even in the absence of satiation, predators are limited by the **handling time** (h) needed to catch and consume each prey item. Consequently, there is a limit to the rate at which individual predators can process prey.

We can construct a more realistic **Type II functional response** by modeling the components that contribute to **feeding rate** (n/t), the rate at which individual predators capture prey (Royama 1971). The total amount of time that a predator spends feeding (t) is the time spent searching for the prey (t_s), plus the time spent "handling" or consuming the prey (t_h):

$$t = t_s + t_h \qquad \text{Expression 6.11}$$

If we let n equal the number of prey items captured in time t and h equal the handling time per prey item, the total handling time is:

$$t_h = hn \qquad \text{Expression 6.12}$$

Similarly, we can derive an expression for the search time. The total number of prey captured by a predator (n) is simply the product of the victim abundance (V), the capture efficiency (α), and the total search time (t_s):

$$n = V\alpha t_s \qquad \text{Expression 6.13}$$

We can rearrange this to give us an expression for the search time:

$$t_s = \frac{n}{\alpha V} \qquad \text{Expression 6.14}$$

Substituting Expressions 6.12 and 6.14 into 6.11, we have:

$$t = \frac{n}{\alpha V} + hn \qquad \text{Expression 6.15}$$

Multiplying the second term by $(\alpha V/\alpha V)$ gives:

$$t = \frac{n}{\alpha V} + \frac{\alpha V h n}{\alpha V} \qquad \text{Expression 6.16}$$

$$t = n\left(\frac{1 + \alpha V h}{\alpha V}\right) \qquad \text{Expression 6.17}$$

Finally, this can be rearranged to give us an expression for the feeding rate (n/t):

$$n/t = \frac{\alpha V}{1+\alpha V h} \qquad \text{Equation 6.7}$$

Equation 6.7 describes the feeding rate per predator as a function of the capture efficiency, the victim abundance, and the handling time. Note that if the victim abundance is very low, the term αVh in the denominator is small, so the feeding rate is close to αV, as in the simple Lotka–Volterra model. But as the victim abundance increases, the feeding rate approaches a saturation value of $1/h$. This value represents the maximum feeding rate that the predator can achieve because of the constraints of handling time. Equation 6.7 is sometimes referred to as the "disc equation" because it fits data from an experiment in which human subjects were blindfolded and required to find and pick up small discs of sandpaper scattered on a flat surface.

We can simplify Equation 6.7 somewhat by letting $k = 1/h$, the **maximum feeding rate**. We can also define the constant D as $1/\alpha h$. This value turns out to be the **half-saturation constant**, which is the abundance of prey at which the feeding rate is half-maximal. If we first multiply the numerator and denominator of Equation 6.7 by $1/\alpha h$, we have:

$$n/t = \frac{\frac{\alpha V}{\alpha h}}{\frac{1}{\alpha h} + \frac{\alpha V h}{\alpha h}} \qquad \text{Expression 6.18}$$

Substituting in the two new constants k and D yields:

$$n/t = \frac{kV}{D+V} \qquad \text{Equation 6.8}$$

This Type II functional response increases to a maximum and constant rate of prey consumption per predator (k). The half-saturation constant (D) controls the rate of increase to this maximum. This equation is identical to the Michaelis–Menten equation of enzyme kinetics (Real 1977).

Finally, a **Type III functional response** can be described by:

$$n/t = \frac{kV^2}{D^2+V^2} \qquad \text{Equation 6.9}$$

For a Type III functional response, the feeding rate also reaches an asymptote at k, but the curve has a sigmoid shape, similar to the logistic curve (see Chapter 2). Consequently, the feeding rate is accelerated at low prey density, but decreases at high prey density as the asymptote is reached (Figure 6.7).

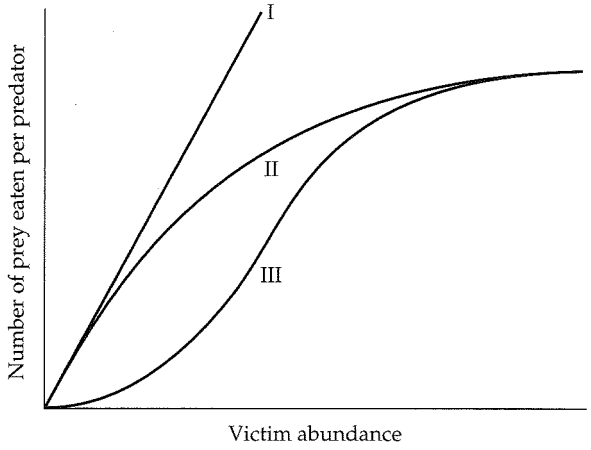


Figure 6.7 Type I, Type II, and Type III functional responses.

This functional response can occur if predators switch to prey items that become more common, if they develop a search image that increases capture efficiency as victim abundance increases, or if there are fixed and variable costs to foraging (Holling 1959, Mitchell and Brown 1990).

The functional response has important consequences for the ability of predators to control victim populations. Figure 6.8 shows the proportion of the prey population that is consumed by an individual predator as victim abundance increases. For the Type I response of the simple Lotka–Volterra model, this proportion remains a constant, because each predator increases its

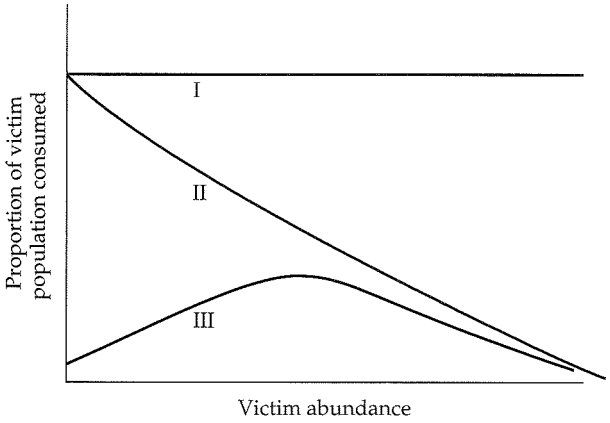


Figure 6.8 The proportion of the victim population consumed by an individual predator as a function of victim abundance.

individual feeding as victim abundance increases. For the Type II response, the proportion decreases steadily because each predator can only process prey at a maximum rate k. The Type III response shows an initial increase because of the accelerated feeding rate, but this quickly decreases and converges on the Type II curve. These curves show that, at high victim abundance, predators with a Type II or Type III response may not be able to effectively control victim populations. Control is possible with the Type III response, but only at relatively low victim abundance. In contrast, the Type I functional response ensures effective control over all levels of victim abundance.

Incorporating a Type II or Type III functional response into the equation for the victim growth rate gives:

$$\frac{dV}{dt} = rV - \left(\frac{kV}{V+D}\right)P \qquad \text{Equation 6.10}$$

$$\frac{dV}{dt} = rV - \left(\frac{kV^2}{V^2+D^2}\right)P \qquad \text{Equation 6.11}$$

Figure 6.9 shows that the isoclines for these growth equations increase in the state space, with an upward swing at low victim abundance for the Type III

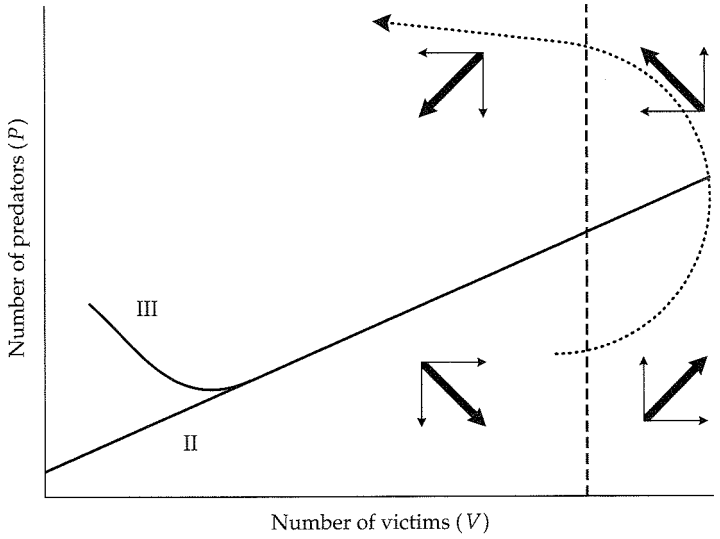


Figure 6.9 Victim isoclines incorporating a Type II or a Type III functional response. The intersection of an increasing victim isocline with a vertical predator isocline generates an unstable equilibrium point.

functional response. Because each predator is limited by a maximum consumption rate, more predators are required to hold large victim populations at zero growth. When these increasing victim isoclines intersect a vertical predator isocline, the equilibrium is unstable, and the predator and victim will not coexist.

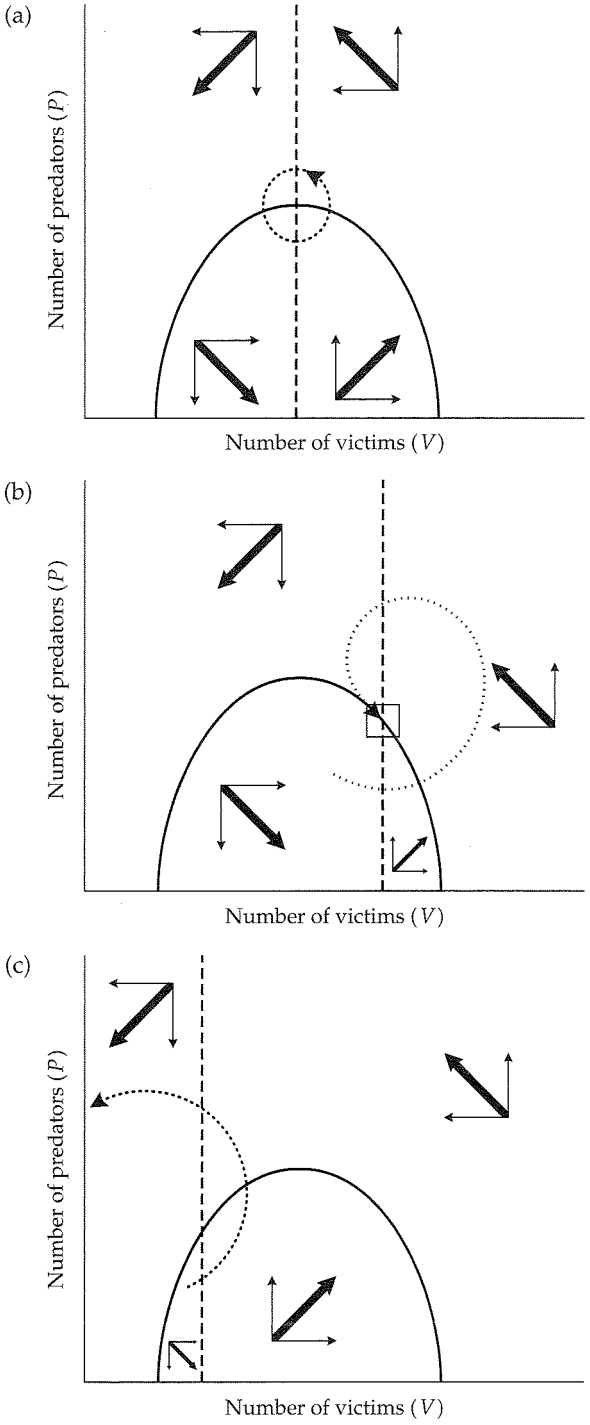
THE PARADOX OF ENRICHMENT

The victim isocline may also increase because of an Allee effect (see Chapter 2) for the victim population. If larger victim populations are more effective at reproducing, obtaining food, or defending themselves from predators, more predators would be needed to control the prey population. Because of a victim carrying capacity, predator functional response, Allee effects, and a variety of other reasons, the victim isocline may have a hump in the middle (Rosenzweig and MacArthur 1963), turning downward at both low and high prey densities.

How does this more realistic victim isocline affect predator–prey dynamics? The answer depends on precisely where the vertical predator isocline intersects the victim isocline. If the intersection is at the peak of the victim isocline, the predator and victim populations will cycle as in the simple Lotka–Volterra model (Figure 6.10a). However, if the predator isocline crosses to the right of the hump, the predator and victim populations converge on a stable equilibrium point, without population cycles (Figure 6.10b). In this case, the predator is relatively inefficient. Thus, from Equation 6.4, the predator population has a relatively high death rate (q) and/or a low conversion efficiency (β). In contrast, if the predator is relatively efficient (low q and/or high β), the isoclines intersect to the left of the hump. In this case, the equilibrium is unstable. The predator population will overexploit the victim population, drive it to extinction, and then starve (Figure 6.10c).

This instability due to a relatively efficient predator has been termed the **paradox of enrichment** (Rosenzweig 1971). The paradox may explain why some artificially enriched agricultural systems are vulnerable to pest outbreaks. Suppose the "victim" population is a crop plant that coexists in a stable equilibrium with a "predator" population of an herbivorous insect. If the productivity of the crop plant is increased with fertilizers, the victim isocline

Figure 6.10 (a) Predator–prey cycles with a humped prey isocline. As in the Lotka–Volterra model, the predator and victim populations cycle as long as the predator and victim isoclines are perpendicular where they intersect. (b) If the predator is relatively inefficient, the predator isocline intersects to the right of the peak of the victim isocline. In this case, predator and victim coexist in a stable equilibrium. (c) If the predator is relatively efficient, the predator isocline intersects to the left of the peak of the victim isocline. In this case, the predator overexploits the prey population, drives it to extinction, and starves.


(a)
Number of predators (P)
Number of victims (V)
(b)
Number of predators (P)
Number of victims (V)
(c)
Number of predators (P)
Number of victims (V)

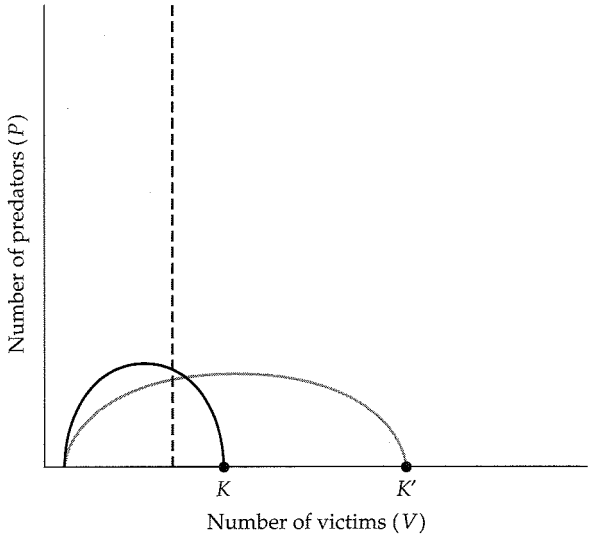


Figure 6.11 The paradox of enrichment. If the victim population has its carrying capacity enhanced from K to K', the system moves from a stable equilibrium to overexploitation by the predator.

may shift to the right to a new, higher carrying capacity (Figure 6.11). If the predator isocline remains stationary, the dynamics may shift from a stable equilibrium to an unstable outbreak of the "pest." This paradox depends on the unrealistic assumption of a strictly vertical predator isocline. More realistic predator isoclines, described later in this chapter, may enhance stability of predator and prey over a wider range of victim abundances (Berryman 1992).

INCORPORATING OTHER FACTORS IN THE VICTIM ISOCLINE

The victim isocline may also turn upward at low victim abundance, generating different population dynamics. There are at least three reasons for an upturn of the victim isocline. First, the isocline will turn up if there is a fixed number of victim refuges that are secure from predators. For example, fish that live in rock crevices and songbirds that establish territories in areas protected by cover have spatial refuges from predation. In this case, no matter how large the predator population gets, the victim population can always persist at low abundance in the refuges. Second, the victim isocline may turn upwards if there is a constant number of victim immigrants that arrive each generation. With immigration, the victim population always has the potential to increase at low numbers. Finally, the isocline may turn upward at low victim abundance because of a Type III functional response, as explained earlier.

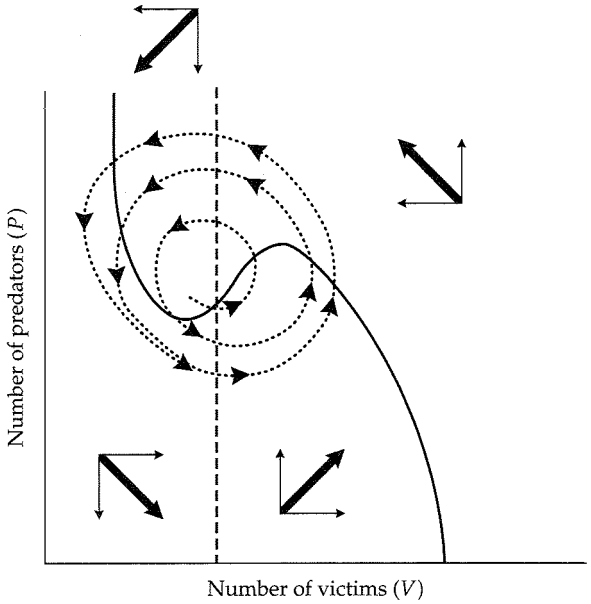
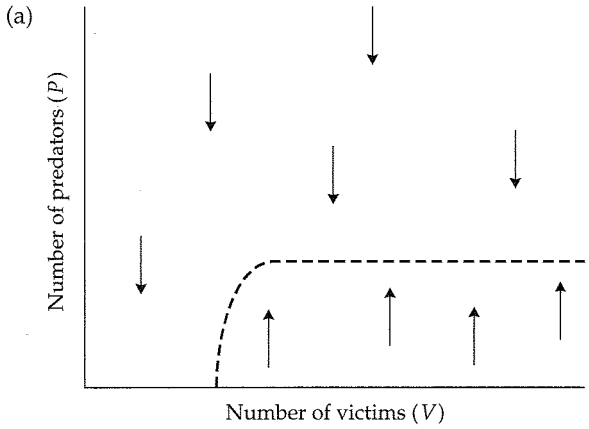


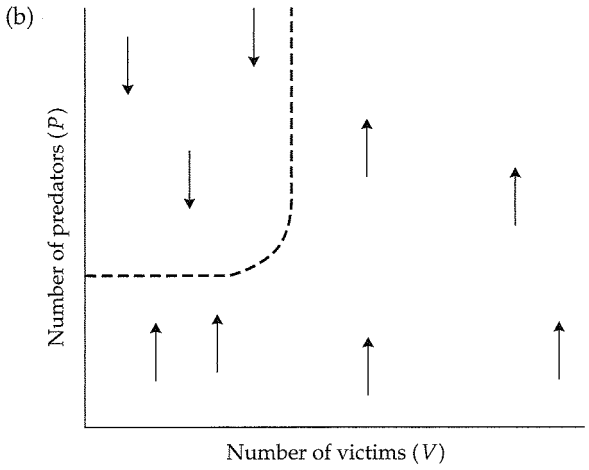
Figure 6.12 Cycling of predator and victim populations because of victim refuges. If there are spatial refuges from predation, the victim isocline becomes vertical at low victim abundance. In this case, the efficient predator cannot overexploit its prey, and begins to starve once all the victims outside of the refuges have been consumed. After the predator population declines below a certain point, the victim population begins to increase again, repeating the cycle.

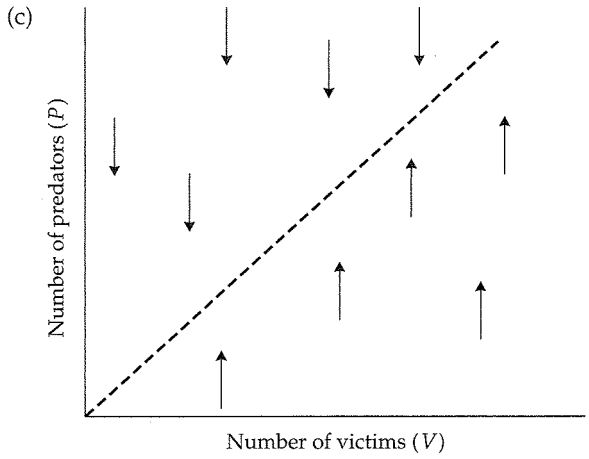
This upward turn of the victim isocline has the potential to stabilize predator–prey interactions. For example, suppose that the predator is relatively efficient, but there is a victim carrying capacity and there are refuges from predation for the victim population (Figure 6.12). In this case, the predators quickly consume all the available victims, as in the destabilized case (Figure 6.10c). But once all the victims outside the shelters are consumed, the predator population begins to starve, and its abundance declines. When the predator population declines below a certain point, the victim population in the refuges starts to increase, and the cycle repeats itself. In contrast to the simple Lotka–Volterra model, these cycles are stable, because no matter what the starting density, the predator population will eventually consume all the victims not in refuges, and the cycle will repeat.

MODIFYING THE PREDATOR ISOCLINE

We can also modify the vertical predator isocline to make it more realistic. These modifications involve changes in the numerical response of Equation


(a)
Number of predators (P)
Number of victims (V)


(b)
Number of predators (P)
Number of victims (V)


(c)
Number of predators (P)
Number of victims (V)

◀ **Figure 6.13** (a) Effects of carrying capacity on the predator isocline. If the predator population is limited by factors other than victim abundance, the predator isocline bends to the right. No matter how large the victim population, the predator population becomes limited when it reaches its own carrying capacity. (b) Effects of the availability of alternative prey on the predator isocline. If the predator is not a specialist on the victim, the predator population may be able to increase even when the victim abundance declines to zero. (c) Effects of victim abundance on the predator isocline. If the size of the victim population acts as a carrying capacity for the predators, the predator isocline increases with increasing victim abundance.

6.2, which will be described qualitatively. For example, the Lotka–Volterra predation model assumes that the predator population can always increase in size if there is an excess of prey available. It is more realistic to suppose that the predator population has its own carrying capacity, so that its growth is limited by other factors. A carrying capacity for the predator bends the predator isocline to the right (Figure 6.13a).

Another unrealistic assumption of the Lotka–Volterra model is that the predator is a specialist on the victim. Suppose instead that the predator has alternative prey sources. Then, when the victim population becomes less abundant, the predator population can continue to increase by feeding on other prey items. This will tip the predator isocline towards the horizontal at low prey abundance (Figure 6.13b). Thus, with alternative prey and a predator carrying capacity, the predator isocline can shift from vertical to horizontal. As we noted earlier, the availability of other prey may shift the victim isocline as well.

As an intermediate case, suppose that the size of the victim population determines the size of the predator population. In other words, the victim population functions as a "carrying capacity" for the predators. In this case, the predator isocline will be a line with a positive slope, intermediate between the vertical isocline of the Lotka–Volterra model and the horizontal isocline of a predator with an independent carrying capacity and alternative prey (Figure 6.13c).

How will these alterations of the predator isocline affect the stability of the model? As a general rule, anything that rotates either the predator or the victim isocline in a *clockwise* direction will tend to stabilize the interaction, whereas anything that rotates the isoclines *counterclockwise* will be destabilizing. These rotations can be compared to the neutral stability of a horizontal victim isocline and a vertical predator isocline in the Lotka–Volterra model (Figure 6.14). For example, giving the victim population a carrying capacity rotates the victim isocline clockwise, leading to a stable equilibrium on the right side of the hump (Figure 6.10b). But adding predator satiation rotates the victim isocline counterclockwise at low abundances, leading to an unstable equilibrium on the left side of the hump (Figure 6.10c). Rotating the

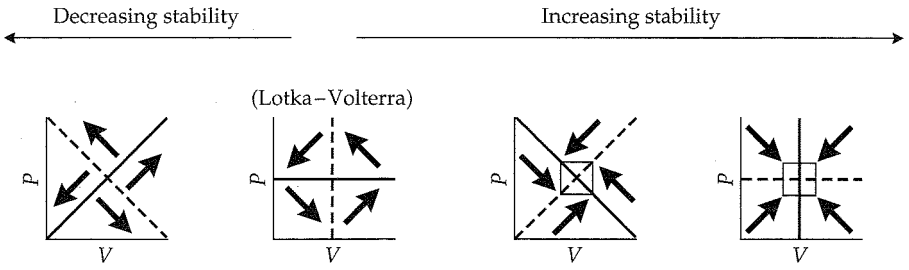


Figure 6.14 Effects of rotating the predator and victim isoclines on the stability of the equilibrium. Relative to the neutral equilibrium of the Lotka–Volterra model, clockwise rotations of the isoclines lead to more stable equilibria; counterclockwise rotations lead to less stable equilibria.

predator isocline also increases the stability of the interaction. Whereas a vertical predator isocline generates population cycles in a neutral equilibrium, an increasing predator isocline generates damped cycles, and a horizontal predator isocline generates a stable equilibrium point (Figure 6.15).

These geometrical rules make intuitive biological sense. The more independent the predator and prey are of one another, the more stable the inter-

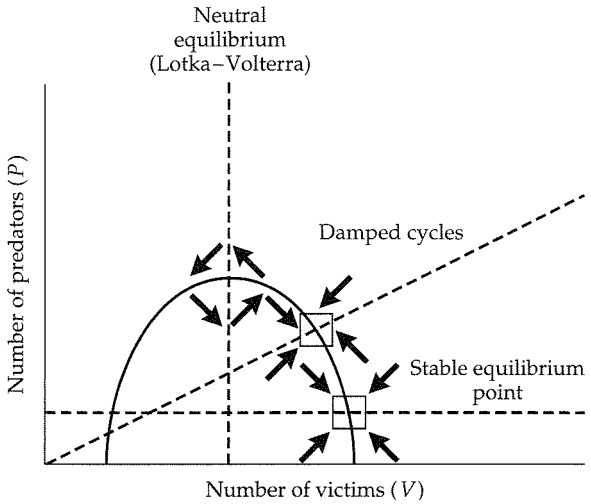


Figure 6.15 Effects of clockwise rotation of the predator isocline. As the predator isocline is rotated, the dynamics change from cycles with a neutral equilibrium, to damped cycles, to a stable equilibrium point. Biologically, the three predator isoclines correspond to a predator that is a complete specialist on the victim, to one whose carrying capacity is proportional to victim abundance, to one whose carrying capacity is independent of victim abundance.

action. For example, suppose the victim isocline is vertical and the predator isocline is horizontal (Figure 6.14). In this case, the carrying capacities of the predator and victim are completely independent of one another, and both species coexist in a very stable equilibrium. Cycles are difficult to generate with simple predator–victim models, and require a special dependence of predator and victim populations upon each other, as in the original Lotka–Volterra model.

Empirical Examples

POPULATION CYCLES OF HARE AND LYNX

The basic prediction of the Lotka–Volterra model is the regular cycling of predator and prey populations. The most famous example of this cycling is the case of the Canada lynx (*Lynx canadensis*) and its principal prey, the snowshoe hare (*Lepus americanus*). The ecologist Charles Elton analyzed fur-trapping records from the Hudson's Bay Company in Canada and found a long-term record of population cycles (Elton and Nicholson 1942). The major source of hare mortality is predation (Smith et al. 1988), and the hare population cycles with a peak abundance approximately every 10 years (Figure 6.16). The lynx population is highly synchronized with the hare and usually peaks one or two years later. These are not the only prey and predator species that cycle in the boreal north. Populations of muskrat, ruffed grouse, and ptarmigan exhibit 9 to 10 year cycles, whereas smaller herbivores such as voles and lemmings cycle with peaks every 4 years. Predators such as foxes, mink, owls, and martens also cycle synchronously with their prey.

What is the explanation for the striking hare–lynx cycle? An early suggestion that the hare cycles were correlated with sunspot activity was dismissed

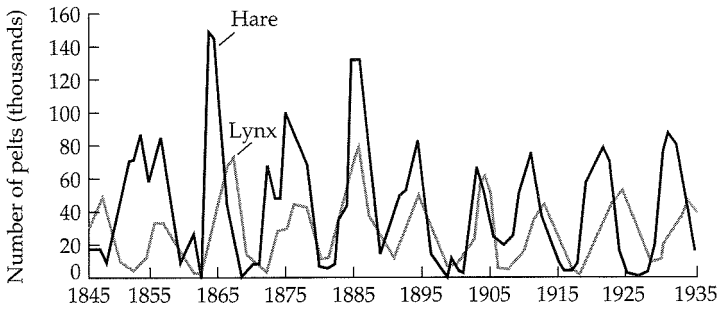


Figure 6.16 One-hundred-year record of population cycles of the snowshoe hare (*Lepus americanus*) and the Canada lynx (*Lynx canadensis*), based on pelt records of the Hudson's Bay Company in Canada.

because sunspot activity peaks every 11 years, whereas the hare cycle is approximately 10 years in length (Moran 1949). For many years, the hare–lynx cycle was the classic textbook example of predator and prey populations that cycled according to the Lotka–Volterra model. More recently, ratio-dependent predator–prey models have been applied to the hare–lynx data (Akçakaya 1992). These models are based on the assumption that the functional response of the predator depends not simply on victim abundance (V), but on the ratio of prey to predator abundance (V/P) (Arditi and Ginzburg 1989).

Unfortunately, two additional pieces of data complicate the story. First, the hare–lynx cycles seem to be broadly synchronized within a year or two over wide areas of North America (Smith 1983). If the predator–prey models were correct, we would expect cycles of different amplitude and period to arise in different local populations. Second, there are places on the coast of British Columbia and on Anticosti Island, Quebec, where there are no lynx, but the hare population cycles nonetheless!

These results suggest that the hare and lynx do not reciprocally influence each other. Instead, the lynx population is probably "tracking" the hare cycle. The hare cycle seems to be caused, in part, by interactions with its food supply. Heavily grazed grasses produce shoots with high levels of toxins that make them less palatable to hares (Keith 1983). This chemical protection persists for two or three years after grazing, further contributing to the hare decline. A single-species logistic model with a time lag (see Chapter 2) would qualitatively describe this sort of cycle. However, as most hares die of predation, not starvation, food quality probably contributes to their susceptibility to predation.

Finally, recent evidence again suggests that sunspots may indeed contribute to the cycles. Sunspot activity is associated with hare browse marks in tree rings and with periods of low snow accumulation (Sinclair et al. 1993). Sunspot activity may serve as a phase-locking mechanism through indirect influences on climate and plant growth. These broad climatic effects could be responsible for the synchrony of hare–lynx cycles over large areas of Canada and Alaska. However, the degree of synchrony among continents is currently being debated (Ranta et al. 1997; Sinclair and Gosline 1997). Whatever the ultimate explanation, it is clear that the hare-lynx cycle is more complex than suggested by the superficial match of the data to the simple predictions of the Lotka–Volterra model.

POPULATION CYCLES OF RED GROUSE

Interactions between hosts and parasites represent a special kind of "predation" in which the life history of the predator is intimately tied to that of its host. Whereas most predators benefit from rapidly killing and consuming their prey, a parasite must keep its host alive at least long enough to success-

fully reproduce and infect a new host. To understand the population dynamics of hosts and parasites, we must therefore model the dynamics of the egg or larval stages, as well as those of the host and the adult parasite (Anderson and May 1978).

A nice example illustrating these complexities is the case of the parasitic nematode *Trichostrongylus tenuis*, which infects red grouse (*Lagopus lagopus scoticus*) on the moors of England and Scotland. Adult worms inhabit the large caeca of red grouse, and their eggs pass out of the host with the feces. If the environment is warm and moist, the eggs hatch and develop into a larval stage. The larval nematode moves to the growing tips of heather plants, where it is consumed by a new host, and the life cycle repeats itself. A single bird may be host to over 10,000 worms. As the intensity of the parasite infection increases, winter mortality, egg mortality, and chick losses all increase (Figure 6.17). Thus, *T. tenuis* has the potential to regulate the population growth of red grouse.

Because red grouse are an important game bird in England and Scotland, there are detailed records on its population dynamics and the prevalence of parasite infection (Hudson et al. 1992). Figure 6.18 shows a 14-year record of host and parasite populations at Gunnerside, North Yorkshire. The red grouse population cycles, with a period of approximately 5 years. Parasite burden (number of worms per host) also cycles, with peaks occurring near the low point of the red grouse cycle.

Even a relatively simple model of the grouse–nematode interaction requires a minimum of three differential equations: one for the host (H), one for the adult worms (P), and one for the free-living egg and larval stages (W; Dobson and Hudson 1992). The growth of the host population can be modeled as:

$$\frac{dH}{dt} = (b - d - cH)H - (\alpha + \delta)P \qquad \text{Expression 6.19}$$

The first term $[(b - d - cH)H]$ represents the growth of the red grouse population in the absence of the parasite. The constants b and d represent intrinsic birth and death rates, and cH is a density-dependent term. The first part of this equation is really a model of logistic growth, with a carrying capacity of $[(b - d)/c]$. A finite carrying capacity is realistic for the grouse population because the birds are territorial. The second part of the equation $[(\alpha + \delta)P]$ represents the losses due to parasites. α is the reduction in host population growth due to effects of the parasite on the survivorship of grouse, and δ is the reduction due to parasite effects on the reproduction of grouse. We distinguish between these two mechanisms because α and δ appear separately in other equations in the model.

Next, we write an equation for the growth rate of the free-living stages:

$$\frac{dW}{dt} = \lambda P - \gamma W - \beta WH \qquad \text{Expression 6.20}$$

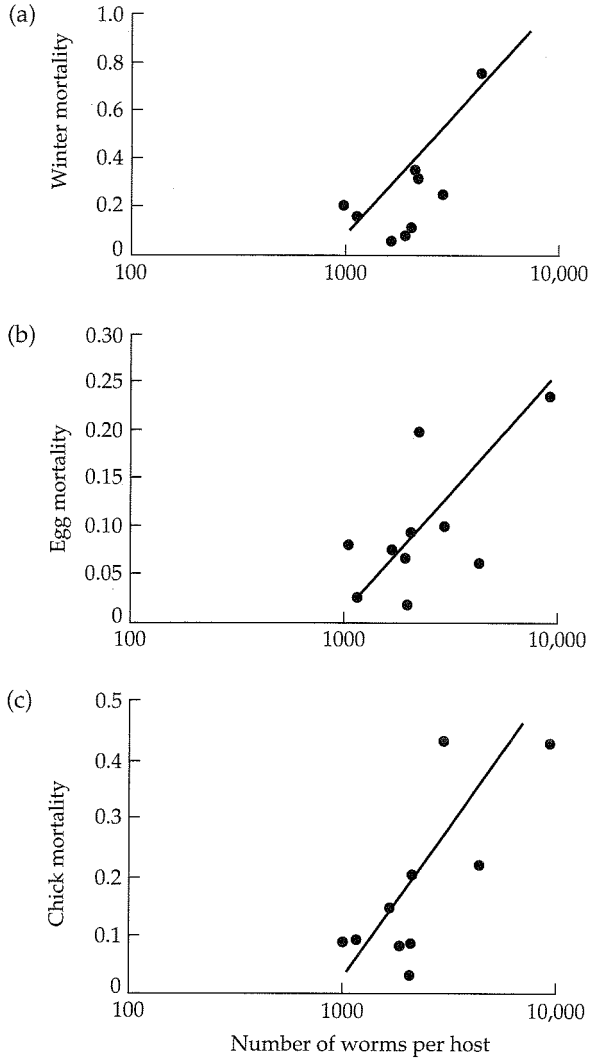


Figure 6.17 Effects of parasite load on (a) the winter mortality, (b) egg mortality, and (c) chick losses of red grouse (*Lagopus lagopus scoticus*). The x axis is the average parasite load (worms per host), and the y axis is the proportional mortality caused by each factor. Because the nematode *Trichostrongylus tenuis* reduces both the survivorship and reproduction of red grouse, it has the potential to regulate host numbers. (From Hudson et al. 1992.)

Here, λ is the per capita fecundity of the parasite in the host, γ is the death rate of the egg and larval stages in the field, and βWH is the rate at which larvae are transmitted to new hosts. Note the similarity of this latter expression to the

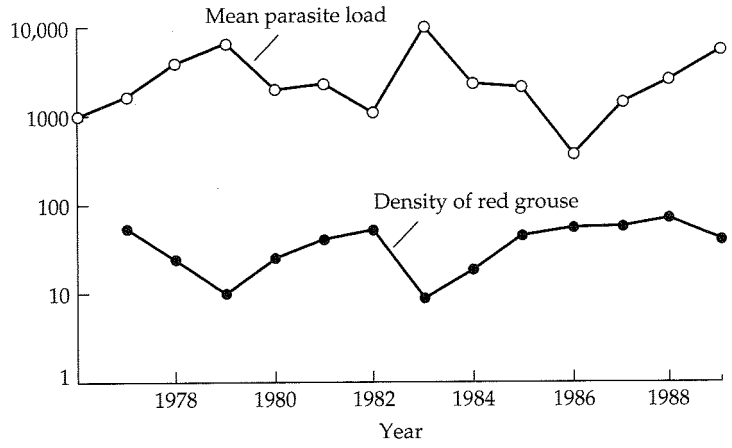


Figure 6.18 Changes in red grouse (*Lagopus lagopus scoticus*) density (breeding hens per square kilometer) and mean parasite load (worms per host) over 14 years at Gunnerside, North Yorkshire. Both the grouse and the nematode populations cycle with a period of approximately 5 years. Note the logarithmic scale on the y axis. (From Dobson et al. 1992; data from Hudson et al. 1992.)

"random encounter" term in the Lotka–Volterra model (Equations 6.1 and 6.2).

Finally, we can describe the dynamics of the adult worm population as:

$$\frac{dP}{dt} = \beta WH - (\mu + d + \alpha)P - \alpha \frac{P^2}{H}\left(\frac{k+1}{k}\right) \quad \text{Expression 6.21}$$

The first term (βWH) represents the increase in the adult worm population from transmission. This is equivalent to the loss component of the egg-larva population. The second term $[(\mu + d + \alpha)P]$ represents decreases in growth of the worm population due to parasite death (μ), intrinsic host mortality (d), and host mortality from parasitism (α). The final term, $[\alpha(P^2/H)][(k + 1)/k]$, represents losses due to the spatial dispersion of the worms among hosts. The constant k describes the distribution of worms among hosts. The smaller k is, the more aggregated the worms are in a few hosts. Aggregation will tend to decrease the growth of the worm population as the few heavily infected hosts die and take their parasites with them! In contrast, if the worms are distributed randomly or evenly among hosts, the growth rate of the parasite population is increased.

With ten different parameters in the model, there are a variety of possible outcomes. If parasite and host fecundity are not high enough, the parasite will go extinct, and the grouse population will rise to its carrying capacity. If the larval life of the parasite is relatively short, the grouse and parasite populations will coexist in a stable equilibrium. But if the larval and egg stages

are fairly long-lived, the model generates stable cycles of host and parasite populations. Cycles in this model arise when $\alpha/\delta > k$. In other words, the ratio of parasite effects on survivorship (α) to parasite effects on reproduction (δ) must exceed the degree of parasite aggregation among hosts (k).

Field data were used to independently estimate the parameters of Expressions 6.19–6.21. The resulting model predicted population cycles with a period of approximately five years, which was observed at Gunnerside (Dobson and Hudson 1992). The model also provides insight into other grouse populations in England and Scotland. Not all grouse and nematode populations cycle, and these noncycling populations are in areas of relatively low rainfall (Hudson et al. 1985). Under these circumstances, the survival of eggs and larvae outside of the host is poor, and the model does not predict cycles.

The interaction of red grouse and its nematode parasite is one of the few well-documented cases of a predator and victim that cause each other's populations to cycle. However, the biology of the system is considerably more complex than that described by the simple Lotka–Volterra model. Models of host–parasite interactions have also been used to predict the dynamics of HIV (the AIDS virus) that infects humans.