

8

Predator–Prey Interactions

Several long-term data sets have been collected of predator and prey in natural systems with the interaction between lynx, *Lynx canadensis*, and its prey, snowshoe hare, *Lepus americanus* (MacLulick, 1937), providing some of the best long-term data, as illustrated in Figure 8.1. We would like to explain several features of this interaction which are typical of predator–prey relationships. The predator and prey both appear to persist over a long period of time. The coexistence involves cycles: both predator and prey numbers appear to oscillate. Finally, the numbers of predators appear to increase in response to the numbers of prey: the peaks in predator abundance appear to follow the peaks in prey abundance (this is clearest for the last several peaks in the figure).

We can reason verbally that if a predator is too efficient it will drive the prey to extinction, and that if the predator is not efficient enough the predator will go extinct. Is there something special about the predator–prey relationships we observe in nature that allows both species to persist? We can also ask if the predator can regulate, or limit the growth of, the prey population. Finally, we ask what are the dynamics of coexisting predator and prey?

The presence of oscillations is typical of simple predator–prey interactions, but not typical of competition between two species.

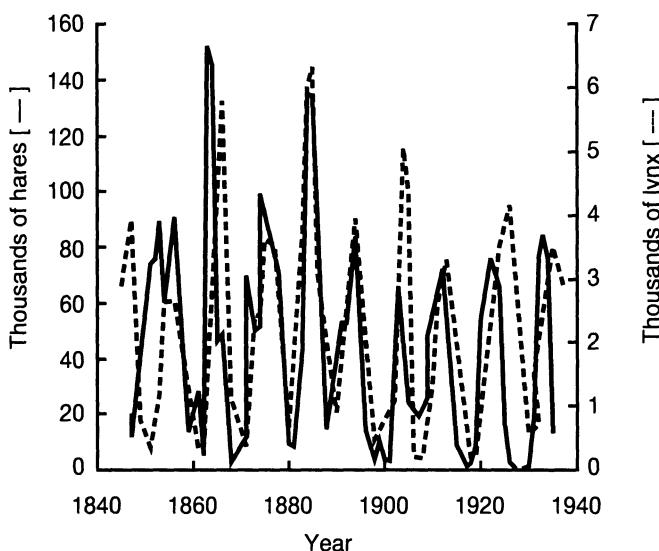


FIGURE 8.1. Dynamics of the predator–prey interaction between lynx (predator) and snowshoe hare (prey) in Canada (data from MacLulich 1937).

8.1 Lotka–Volterra models

We turn to models, just as in our study of competition. We begin with the simplest version of the Lotka–Volterra model, with the following assumptions:

- In the absence of the predator, the prey grows exponentially.
- In the absence of the prey, the predator dies off exponentially.
- The ‘per predator rate’ at which prey are killed is a linear function of the number of prey, as illustrated in Figure 8.2.
- Each prey death contributes identically to the growth of the predator population.

We now formulate a model according to the assumptions we have just made. Let the number of prey be denoted by H (standing for herbivores) and the number of predators be denoted by P . Our first assumption implies that if $P = 0$ then

$$\frac{dH}{dt} = rH, \quad (8.1)$$

Ask yourself about the reasonableness of these assumptions.

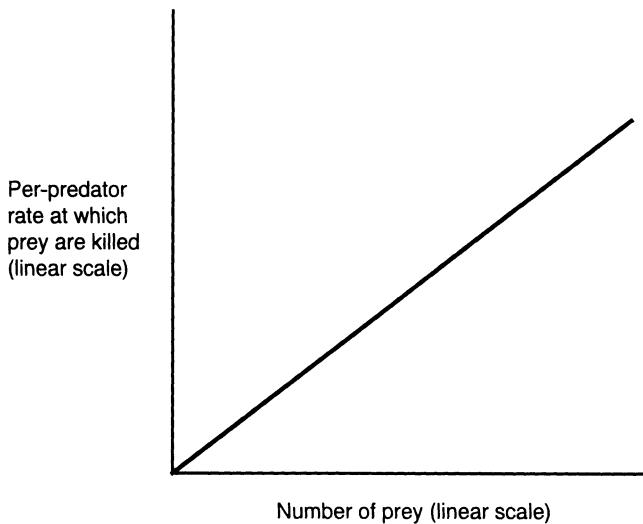


FIGURE 8.2. Predation rate, or functional response, in Lotka–Volterra model.

where r is the intrinsic rate of increase of the prey in the absence of the predator. Our second assumption implies that if $H = 0$ then

$$\frac{dP}{dt} = -kP, \quad (8.2)$$

where k is the rate of decline of the predator in the absence of the prey. Thus $1/k$ is the mean lifetime of the predator. Our third assumption says that the death rate of prey from predation is proportional to the product of prey and predator numbers, bHP , where b is a constant. This says that the full equation for the dynamics of the prey is

$$\frac{dH}{dt} = rH - bHP. \quad (8.3)$$

Our fourth assumption says that the contribution of predation to the growth rate of the predator population is given by cHP , where c is a constant. Thus, the full equation for the dynamics of the predator is given by

$$\frac{dP}{dt} = cHP - kP. \quad (8.4)$$

To see that $1/k$ is the mean lifetime of the predator compute, $\int_0^\infty e^{-kt} dt = 1/k$.

The contribution of predation to the growth of the predator population is directly proportional to the loss of prey from predation that enters into the prey dynamics.

8.2 Dynamics of the simple Lotka–Volterra model

Does the model explain the dynamic behavior of a predator–prey system we have seen in Figure 8.1? The only way to answer this question is to determine the dynamics of the model. The first step is to find the equilibria and the stability of the equilibria.

Graphical analysis and linearization

Our first analysis of the model is graphical, as illustrated in Figure 8.3. The determination of the isoclines, and the direction of change of numbers of predators and prey, proceeds exactly as in the competition models analyzed in the previous chapter. Therefore, we do not go through the steps of the analysis, but simply present the results in the figure.

From the graphical analysis, we see that a new feature emerges that was not present in the competition models. There is a tendency for solutions to oscillate. However, we are unable to conclude whether the equilibrium point is stable from the graphical analysis. Although we have indicated a cycle in Figure 8.3, we do not yet know whether solutions spiral toward the equilibrium, truly cycle, or spiral out. We therefore proceed with a stability analysis of equilibrium points, as described in Chapter 6.

Our first step is to find the equilibria of the model. We will let $F(H, P)$ be the growth rate of the prey population and let $G(H, P)$ be the growth rate of the predator population. We find the equilibria of the model by simultaneously solving the pair of equations $F = 0$, $G = 0$. From the equation $F = 0$, we find that

$$rH - bHP = 0, \quad (8.5)$$

or

$$H(r - bP) = 0. \quad (8.6)$$

We conclude that either

$$H = 0 \quad (8.7)$$

These are just the equations of the isoclines.

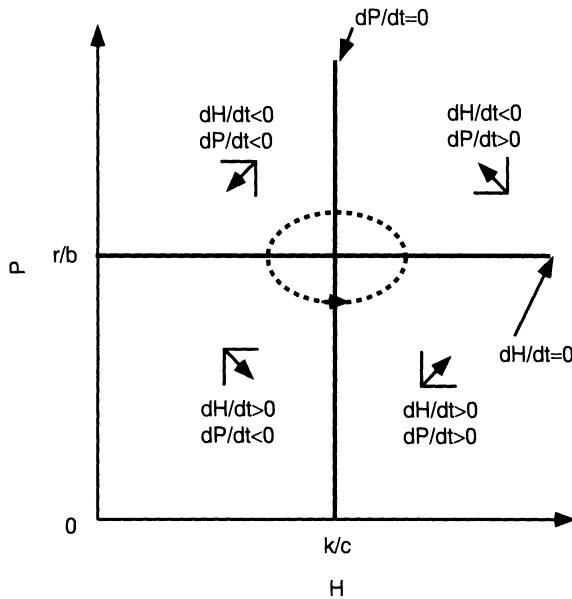


FIGURE 8.3. Phase plane for Lotka–Volterra predator–prey model with no density dependence. The first step in drawing this phase plane is to draw the two isoclines, given by equations (8.8) and (8.12). The next step is to draw the arrows indicating the direction of population change in each of the four sectors created by the isoclines. From this phase plane we are unable to determine the stability of the equilibrium, in contrast to the competition case where stability could be determined from the phase plane. The oscillation indicated on the phase plane can be found by numerical (computer) methods or more sophisticated mathematical methods.

or

$$P = \frac{r}{b}. \quad (8.8)$$

In a similar fashion, we use the equation $G = 0$ to find

$$cHP - kP = 0, \quad (8.9)$$

or

$$P(cH - k) = 0. \quad (8.10)$$

We conclude that either

$$P = 0 \quad (8.11)$$

It is typical of predator–prey models that the equation for the predator dynamics completely determines the equilibrium level of the prey. We will see later that this corresponds to our biological assumption that the per predator predation rate is independent of the number of predators: the predators act independently.

or

$$H = \frac{k}{c}. \quad (8.12)$$

Translate this statement about stability of equilibria with species absent into biological terms. For example, if both species are absent, and we introduce a few of the prey species, what happens?

We use the symbol J for the matrix, because this linearization matrix is known mathematically as a Jacobian matrix. This is the community matrix we discussed in Chapter 6.

There are four equilibria for the model, corresponding to any of the possible pairs of predator and prey equilibrium levels we have just found. From the graphical analysis, we can conclude that all three equilibria at which one or both species are absent are unstable. We thus concentrate our stability analysis on the nontrivial equilibrium

$$(H, P) = \left(\frac{k}{c}, \frac{r}{b} \right). \quad (8.13)$$

Our first step in determining the stability of this equilibrium is to approximate the dynamics near the equilibrium by linearizing, as outlined in Box 6.1. We find that the linearization of this model near the equilibrium is given by

$$J = \left(\begin{array}{cc} \frac{\partial F}{\partial H} & \frac{\partial F}{\partial P} \\ \frac{\partial G}{\partial H} & \frac{\partial G}{\partial P} \end{array} \right) \Big|_{(H,P)=\left(\frac{k}{c}, \frac{r}{b}\right)} \quad (8.14)$$

$$= \left(\begin{array}{cc} r - bP & -bH \\ cP & cH - k \end{array} \right) \Big|_{(H,P)=\left(\frac{k}{c}, \frac{r}{b}\right)} \quad (8.15)$$

$$= \left(\begin{array}{cc} r - b\frac{r}{b} & -b\frac{k}{c} \\ c\frac{r}{b} & c\frac{k}{c} - k \end{array} \right) \quad (8.16)$$

$$= \left(\begin{array}{cc} 0 & -b\frac{k}{c} \\ c\frac{r}{b} & 0 \end{array} \right) \quad (8.17)$$

Complex eigenvalues and oscillations

We will now find the eigenvalues of the matrix J to determine the behavior of the model near equilibrium. Mathematically this is analogous to the procedure we used in analyzing age-dependent growth. Thus we set the determinant of $J - \lambda I$ to be zero:

$$0 = \begin{vmatrix} -\lambda & -b\frac{k}{c} \\ c\frac{r}{b} & -\lambda \end{vmatrix} = \lambda^2 + rk \quad (8.18)$$

The solution to this equation requires the use of complex numbers (see Box 8.1). We conclude that

$$\lambda = \pm i\sqrt{rk}. \quad (8.19)$$

Thus in this model the equilibrium point is neither locally asymptotically stable (solutions do not get closer) nor unstable, as determined by the linear approximation. A more sophisticated analysis of this model shows that in fact all the solutions are closed curves, as illustrated in Figure 8.3. Thus we conclude that we must make this model more realistic before we can begin to draw biological conclusions about stability. However, the information about the period of the oscillations can prove useful.

Before we leave our study of this model, we will indicate how we could have determined some of the qualitative information about the dynamics from an examination of the phase plane. As we noted earlier, the entries in the linearization of a predator–prey model take the form

$$\begin{pmatrix} ? & - \\ + & 0 \end{pmatrix}. \quad (8.20)$$

Remind yourself again why the entries take the signs indicated. The ‘?’ in the upper left arises because the effect of the prey species on itself can be affected by the predation term, as we will see.

We will explain how to read off the signs of both the entries filled in and the entries not filled in from the phase plane. Start with the one in the upper right, which is $\partial F/\partial P$, evaluated at equilibrium. This means that we need to look at the effect of changing P on F at equilibrium, while holding H constant. In the phase plane diagram, if we look at what happens, F changes from positive to negative, as indicated in Figure 8.4. Follow the bold arrow and it goes from a region of phase space where F is positive to one where F is negative. Therefore, the partial derivative $\partial F/\partial P$, evaluated at equilibrium, is negative.

In a similar fashion, in the same figure, we note that along the bold arrow G does not change its value: it is always zero. We thus conclude that, at equilibrium, $\partial G/\partial P = 0$. Similarly, we can determine that, at equilibrium, $\partial F/\partial H = 0$ and $\partial G/\partial H > 0$.

Thus, we have concluded that the signs of the entries in the linearization about the equilibrium are

$$\begin{pmatrix} 0 & - \\ + & 0 \end{pmatrix}. \quad (8.21)$$

Do this yourself graphically – which way should you draw a bold arrow? How should you label the head and tail of the arrow?

As the determinant is zero, at least one of the eigenvalues must have zero real part.

Box 8.1. Facts about complex numbers and complex eigenvalues.

Complex numbers arise naturally in the determination of eigenvalues for the linearization of predator–prey models, or in systems with age structure and more than two age classes. We collect a number of facts and implications here. We denote the square root of -1 by \imath :

$$\imath = \sqrt{-1}.$$

This is called an *imaginary* number, and a *complex* number is just the sum of an imaginary number and a real number. When an eigenvalue of the linearization of a continuous time model involves \imath , the eigenvalue is representing a growth rate of a perturbation away from equilibrium. Thus the dynamics near equilibrium are given by an eigenvector multiplied by a term of the form $e^{\lambda t}$, where λ is complex. Let us write

$$\lambda = u + \imath v.$$

Then

$$e^{\lambda t} = e^{ut+vt} = e^{ut} e^{vt}. \quad (\text{a})$$

We understand the exponential e^{ut} on the right-hand side of (a), but to understand the exponential e^{vt} we need to use the important formulas

$$\begin{aligned} e^{\imath\theta} &= \cos(\theta) + \imath \sin(\theta) \\ e^{-\imath\theta} &= \cos(\theta) - \imath \sin(\theta). \end{aligned}$$

Thus, complex eigenvalues correspond to oscillatory behavior. Note that if λ is a complex eigenvalue, λ is always part of a complex pair, $u \pm \imath v$. Consequently, there are two growth rates of deviations away from equilibrium:

$$e^{\lambda t} = e^{ut \pm vt} = e^{ut} [\cos(vt) \pm \imath \sin(vt)]. \quad (\text{b})$$

Because the oscillatory term shows no long-term change in magnitude, the equilibrium is stable if $u < 0$ and unstable if $u > 0$. (Here u is the real part of the eigenvalue.) The

Box 8.1 (cont.)

period of the oscillatory behavior is given by $2\pi/\nu$. To get real values for the growth of the perturbations, we can add the two possible solutions given on the right of (b) and divide by 2 to get $e^{ut}\cos(\nu t)$ or take the difference and divide by $2i$ to get $e^{ut}\sin(\nu t)$. In each case, the other trigonometric term cancels.

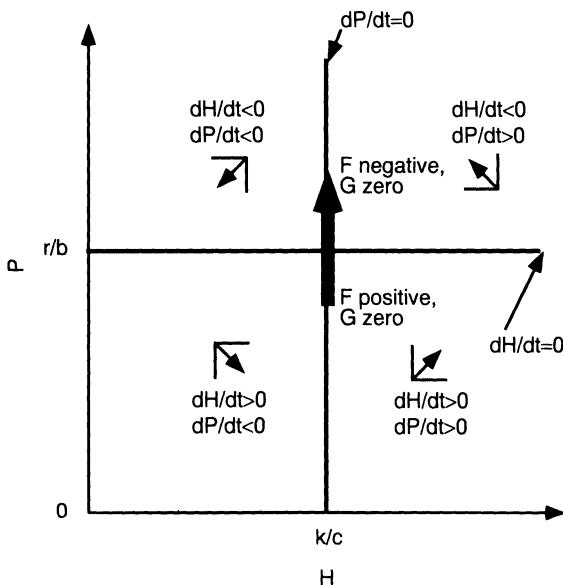


FIGURE 8.4. Determining graphically the linearization at equilibrium of the Lotka–Volterra predator–prey model without density dependence. The bold arrow indicates the meaning in the phase plane of a partial derivative with respect to P . By comparing the sign of F and G at the tail and head of the bold arrow, we can find the sign of $\partial F / \partial P$ and $\partial G / \partial P$. Because F changes from positive to negative, $\partial F / \partial P < 0$. Because G does not change, $\partial G / \partial P = 0$.

Behavior of the Lotka–Volterra model far from equilibrium

This analysis has shown that we cannot determine the stability of the equilibrium using the linearization technique: the model produces an equilibrium that lies right on the border between stability and instability. More sophisticated analyses of this model, or numerical solutions, demonstrate that this inability to determine

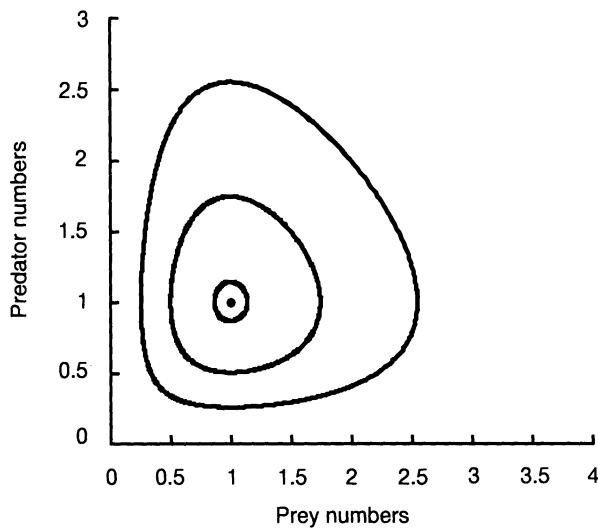


FIGURE 8.5. Numerical solutions of the Lotka–Volterra predator–prey model with no density dependence, displayed in the phase plane. Equations (8.3) and (8.4) are solved numerically with $r = b = c = k = 1$. The closed curves represent sustained oscillations. However, as there is an infinite family of closed curves corresponding to different initial conditions, this model is biologically unrealistic because the outcome will be changed by small changes in the model.

stability actually reflects the fact that the solutions to the full model are always cycles, with *the amplitude of the cycle determined solely by initial conditions* (see Figure 8.5). If the system starts with a large-amplitude cycle, it continues to cycle with large amplitude. If the system starts with a small-amplitude cycle, it continues to cycle with small amplitude. There is no tendency for the amplitude of the cycles to change at all. We have duplicated one feature of the observed dynamics, the presence of oscillations. However, we have no way to explain oscillations of any particular size.

8.3 Role of density dependence in the prey

We argue that the model we have just developed is a poor one to use to describe ecological systems because small changes in the model might lead to large changes in the dynamics of the model. Because the model simply oscillates with whatever amplitude is determined by the initial conditions, a small change in

Because the ‘pure’ Lotka–Volterra model is at the border between stability and instability, we can add various modifications and see if they lead to stability or instability of the equilibrium, and then classify the modification as stabilizing or destabilizing.

our assumptions might lead to either oscillations growing without bound, or to an approach to equilibrium. Because essentially all models in ecology are rather crude, such sensitive behavior of the model outcome to changes in the underlying model is clearly both undesirable and unrealistic. We thus begin to look at modifications of the basic model, which will serve both to remove this curious and unrealistic behavior and to justify the argument made in this paragraph.

One of the simplest modifications possible to the basic Lotka–Volterra model is to change our assumption that the prey grows exponentially in the absence of the predator to say that the prey grows logistically in the absence of the predator. In this case, we find that the equations for our model become

$$\frac{dH}{dt} = rH \left[1 - \frac{H}{K} \right] - bHP \quad (8.22)$$

and

$$\frac{dP}{dt} = cHP - kP. \quad (8.23)$$

Adding density dependence does not change the equation for the predator population.

A phase plane analysis of this model is displayed in Figure 8.6. The first step in drawing the phase plane is to find the isoclines. The prey isocline is found by solving

$$0 = rH \left[1 - \frac{H}{K} \right] - bHP, \quad (8.24)$$

yielding

$$H = 0, \quad (8.25)$$

or

$$0 = r \left[1 - \frac{H}{K} \right] - bP. \quad (8.26)$$

As in the competition case, it is easy to draw this line on the phase plane by noticing that it passes through the P axis at $P = r/b$ and through the H axis at $H = K$. The predator isocline is unchanged from the case without density dependence.

Once again, we determine the signs of the entries in the linearization about the nontrivial equilibrium by following along the bold arrows in Figure 8.7.

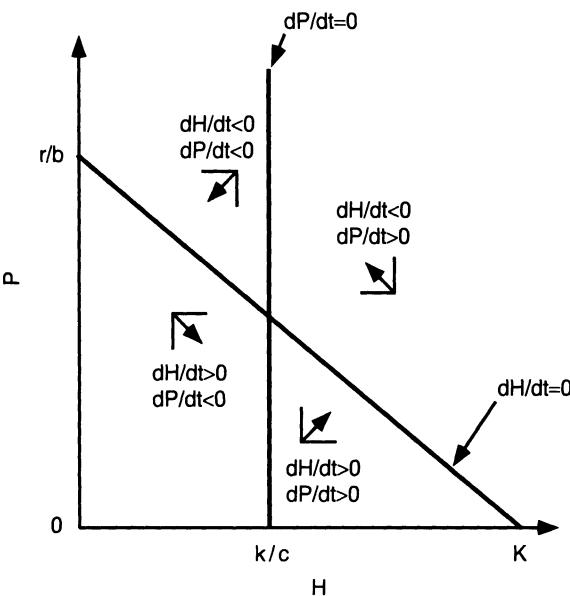


FIGURE 8.6. Phase plane for Lotka–Volterra predator–prey model with density dependence in the prey.

Work out for yourself that the signs of the entries in the linearization matrix now have the form

$$\begin{pmatrix} - & - \\ + & 0 \end{pmatrix}. \quad (8.27)$$

The trace is the sum of a negative number and zero, which is negative. The determinant is given by a number that is $(-)(0) - (-)(+)$, which is positive. Thus by the criterion for stability outlined in Box 6.2, we conclude that the equilibrium is stabilized by the addition of density dependence in the prey. However, the equilibrium is still approached in an oscillatory fashion, as illustrated in Figure 8.8. Also, although the equilibrium is stable, the numbers of predators can still reach very low levels before the system reaches equilibrium.

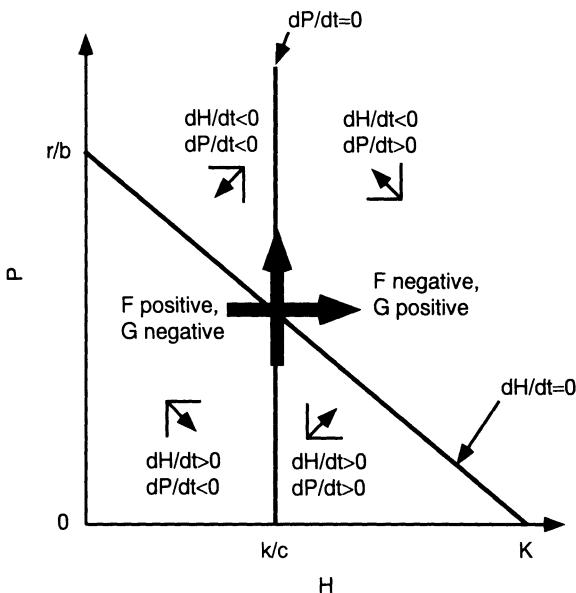


FIGURE 8.7. Determining graphically the linearization at equilibrium of the Lotka–Volterra predator–prey model with density dependence. The horizontal and vertical bold arrows indicate in the phase plane the meaning of the partial derivatives with respect to H and P , respectively. By comparing the sign of F and G at the tail and head of the horizontal bold arrow, we find that $\partial F/\partial H < 0$ and $\partial G/\partial H > 0$. Using the vertical bold arrow, we can find $\partial F/\partial P$ and $\partial G/\partial P$ as we did in the case without density dependence.

8.4 Classic laboratory experiments on predation

How realistic is this conclusion that we obtain stability in predator–prey systems? We will first look at this question in the context of laboratory experiments, and then focus on examples from the field after refining our theory.

Gause

Inspired by the work of Lotka and Volterra on models describing the interaction between predator and prey, Gause (1934) undertook a series of experiments in a microcosm to test the predictions of the theory. In the microcosm, bacteria were supplied as food for *Paramecium caudatum*, which in turn was consumed by *Didinium nasutum*. *Didinium* is a voracious predator, consum-

By working with microorganisms, Gause was able to carry out his experiments over a relatively short time and in a relatively small space.

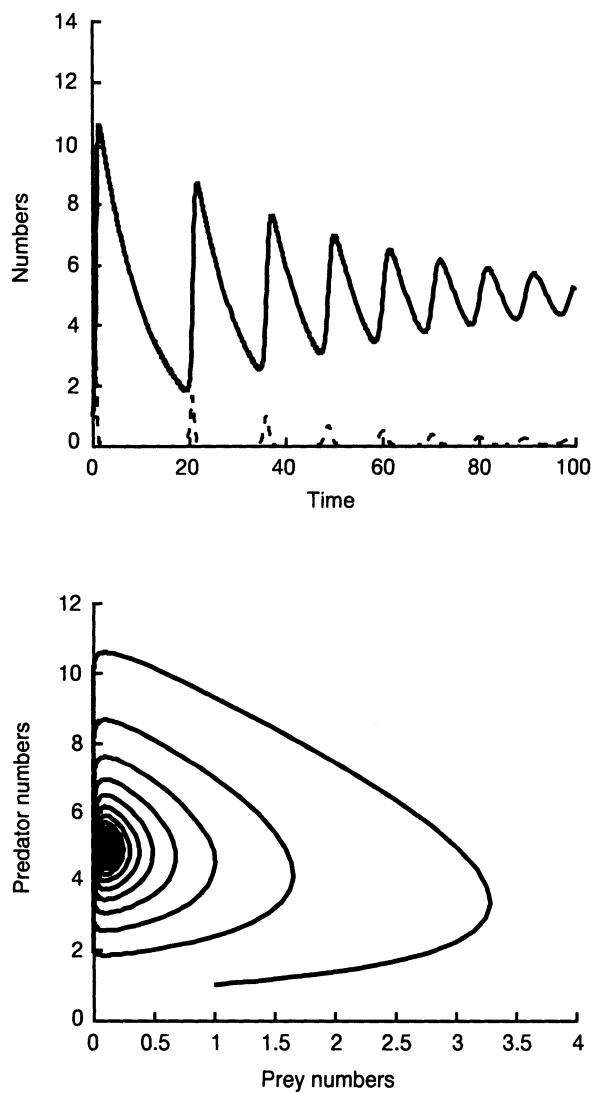


FIGURE 8.8. Dynamics against time (top) and in the phase plane (bottom) for the Lotka–Volterra predator–prey model with density dependence in the prey. In the plot of numbers against time, the prey is the solid line; the predator is the dashed line. Note how close solutions come to the axes before approaching the equilibrium.

ing at least one fresh *Paramecium* every 3 hours. In this system, Gause concentrated on the interaction between *Paramecium* and *Didinium*, with the bacteria introduced as part of the substrate.

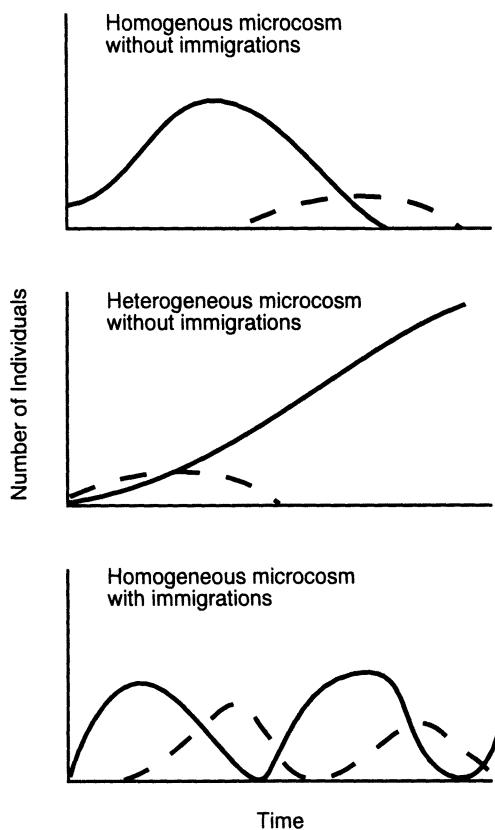


FIGURE 8.9. Schematic diagram of dynamics against time for *Didinium nasutum*, predator, shown in dashed lines, and *Paramecium caudatum*, prey, shown in solid lines, under three different experimental protocols (redrawn from Gause 1935).

In the first experiments performed by Gause, he simply placed the prey, and then the predator, in the experimental microcosm. The outcome was always the same – the predator invariably consumed all the prey, and then the predator also went extinct (see top panel of Figure 8.9). This was not the outcome predicted by the theory, where coexistence was the expected outcome. However, at least the inherent tendency to oscillate did show up in this experiment.

The next step was to try and look for processes that would allow predator and prey to both persist. So Gause tried to provide a refuge for the prey, a place where the *Paramecium* could

not be consumed. Once again, the species did not coexist, although the outcome was different (middle panel of Figure 8.9). Here, the predator starved, and those prey protected in the refuge eventually produced exponential growth of the prey population.

Finally, Gause sought a way to produce sustained oscillations and coexistence, which was apparently achieved as illustrated for the case illustrated in the bottom panel of Figure 8.9. However, the only way that this coexistence was achieved was for the experimenter to add 1 *Paramecium* and 1 *Didinium* to the experimental microcosm at regular intervals, thus preventing either species from going extinct. This experiment did demonstrate, however, oscillations in a predator-prey system.

At this point the agreement between theory and experiment is quite weak. We will go back to the theory and determine what destabilizing influences we have left out. Before returning to the theory, we first present the results of another classic experiment that built upon the stabilizing influence that Gause used. If persistence of predator and prey could be achieved by the experimentalist adding predator and prey to a microcosm, could the same effect be achieved by predator and prey themselves moving among different habitats?

Huffaker

Huffaker (1958) performed several experiments with a predator-prey system where the habitat was a series of oranges laid out in trays. The predatory mite *Typhlodromus occidentalis* fed upon the six-spotted mite, *Eotetranychus sexmaculatus*, which fed upon the oranges. We describe in more detail one of these experiments, which demonstrated how persistence could be achieved.

In this experiment, the ‘universe’ consisted of three trays, each containing 40 oranges arrayed in a grid of 4 rows with 10 oranges. Partial barriers were set up between the oranges within a tray, and connections were made between trays. Huffaker then began the experiment by placing 1 female six-spotted mite on each of the oranges. Five days later 27 predators were added to 27 oranges distributed throughout the experimental universe. Af-

Mites crawl from orange to orange.

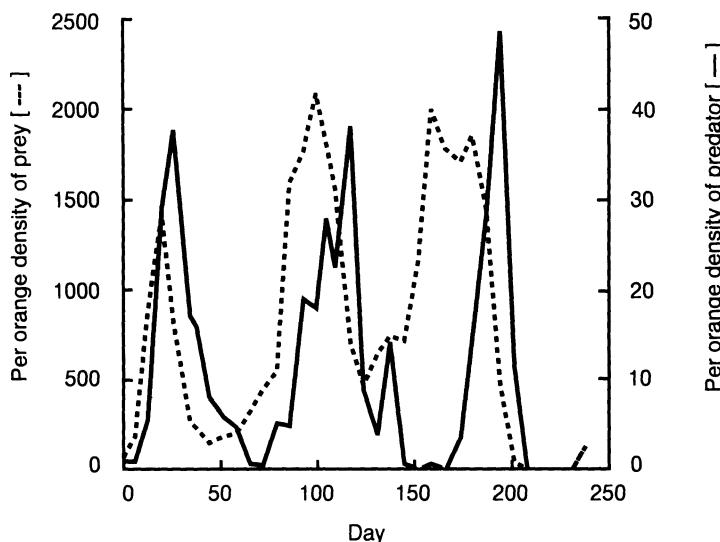


FIGURE 8.10. Oscillations in a laboratory predator–prey system with spatial structure, where the prey, the six-spotted mite *Eotetranychus sexmaculatus*, was eaten by the predator, *Typhlodromus occidentalis*. The experimental universe consisted of 120 oranges (data from Huffaker 1958).

ter this, the numbers of predator and prey were recorded, as well as the locations of ‘occupied’ oranges.

The results for the total number of predator and prey were quite dramatic, with sustained oscillations resulting (Figure 8.10). We thus have experimental evidence that a metapopulation structure can lead to persistence that would be impossible without this kind of spatial structure. Huffaker had previously shown, not surprisingly, that at smaller spatial scales, persistence did not result.

Before presenting further laboratory or field examples, we will return to the theoretical development and try to explain the two classic experiments we have just described.

We have already seen metapopulations in both the context of single species and competing species.

8.5 Functional response

Gause’s (1934) experiments clearly indicated that the Lotka–Volterra model with density dependence in the prey was much more stable than the dynamics of the predator–prey system observed

Think of potentially destabilizing influences.

in the laboratory. Therefore, we conclude that we have left out of the model important destabilizing features of the predator–prey interaction. Here we will analyze the effects of changing another of the assumptions in the Lotka–Volterra predator–prey model that is unrealistic. We recognize that as the number of prey increases the rate of prey capture per predator cannot increase indefinitely. At some point the rate of prey capture per predator must level off, as the number of prey is not the limiting factor. We refer to the predation rate as a function of the number of prey per predator as the *functional response*. If we included just this effect, we would obtain the type II functional response illustrated in Figure 8.11 (Holling, 1959).

Another biological effect is that when a particular prey item is rare, it may be ignored by the predator. This leads to a faster than linear rise in the per predator predation rate as a function of prey numbers, when prey numbers are small. This is a type III functional response, which is also illustrated in Figure 8.11.

Effect of functional response on stability

We will determine the consequences of a type II functional response by determining the effect on stability. We write the model in this case as

$$\frac{dH}{dt} = rH \left[1 - \frac{H}{K} \right] - bf(H)P \quad (8.28)$$

and

$$\frac{dP}{dt} = cf(H)P - kP, \quad (8.29)$$

where the function $f(H)$ represents the functional response. Once again the predator isocline is vertical.

We begin by finding the prey isocline, which is given by

$$rH \left[1 - \frac{H}{K} \right] - bf(H)P = 0. \quad (8.30)$$

Solve this for P , obtaining

$$P = \frac{rH(1 - H/K)}{bf(H)}. \quad (8.31)$$

We solve for P because it is easier than solving for H .

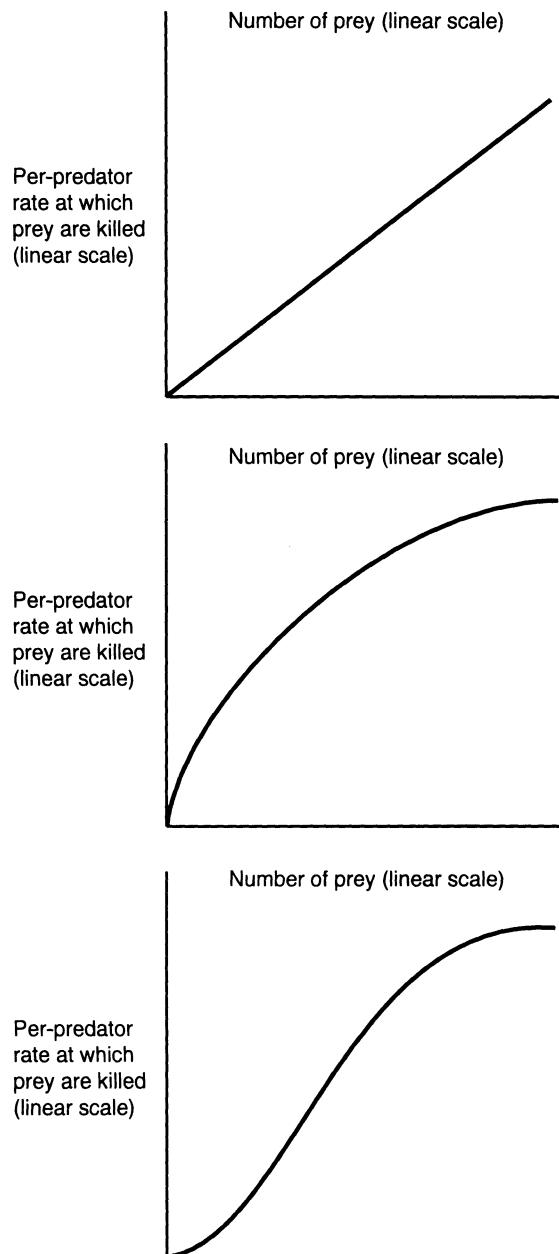


FIGURE 8.11. (Top) Lotka–Volterra or linear functional response. (Middle) Type II functional response. (Bottom) Type III functional response.

We now specialize to a particular form of f that has often been used to model a type II functional response:

$$f(H) = \frac{aH}{1 + dH}, \quad (8.32)$$

If H is large, f is approximately a/d . If H is small, f is approximately aH .

where a and d are positive parameters.

With this choice of f , we can now find the location of the predator isocline. It is given by the solution of the equation

$$0 = \frac{acPH}{1 + dH} - kP, \quad (8.33)$$

which is

$$P = 0, \quad (8.34)$$

or

$$0 = \frac{acH}{1 + dH} - k. \quad (8.35)$$

We can solve (8.35) for H to find the location of the vertical predator isocline

$$H = \frac{k}{ac - kd}. \quad (8.36)$$

This isocline is drawn on Figures 8.12 and 8.13.

We now continue with our computation of the prey isocline for this particular functional response. Substituting from (8.32) into (8.31), we obtain

$$P = \frac{rH(1 - H/K)}{b \left(\frac{aH}{1 + dH} \right)} \quad (8.37)$$

$$= \frac{r(1 - H/K)(1 + dH)}{ab}. \quad (8.38)$$

Equation 8.38 is a parabola because the right-hand side is quadratic in H .

To find the points where the parabola crosses the axis, set the numerator of (8.38) equal to zero and solve for H . This is easy to do because the numerator is already factored.

This is a parabola. As we shall see, the important feature will be the location of the maximum, which is located midway between the points where the parabola crosses the H axis (i.e., where P is zero). Since these points are $H = -1/d$ and $H = K$, the maximum is at $H = (K - 1/d)/2$.

We graph two different possibilities in Figures 8.12 and 8.13, depending on the relative location of the maximum (or hump) in the prey isocline and the predator isocline. In the first case,

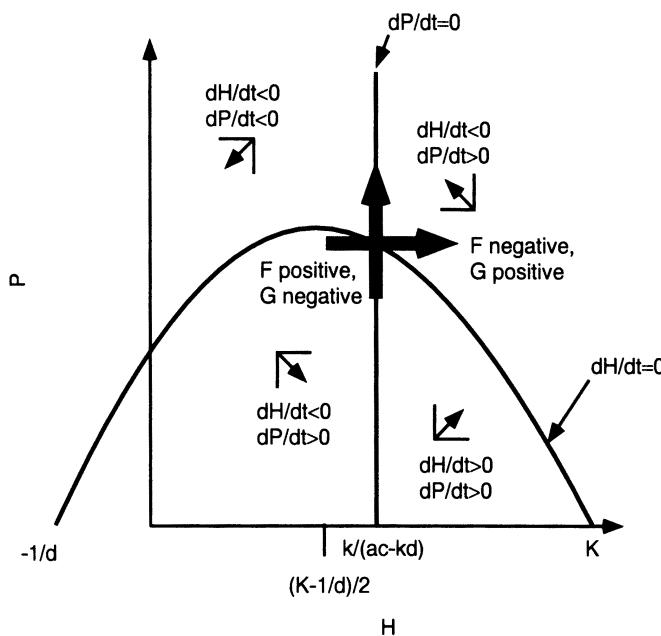


FIGURE 8.12. Phase plane for predator–prey model with type II functional response, stable case. Note that the phase plane near the equilibrium in this case looks like the phase plane depicted in Figure 8.7. The bold arrows in the figure are used to determine the signs of the entries in the linearization (Jacobian) matrix as in Figure 8.7.

where the predator isocline is to the right of the hump (Figure 8.12), the equilibrium point is stable, because in the vicinity of the equilibrium the dynamics are the same as in the case of the Lotka–Volterra response we have already analyzed.

However, if the predator isocline is to the left of the hump, as in Figure 8.13, we have a different situation. We can again determine the signs of the entries in the linearization by following along the bold arrows in the figure, obtaining

$$\begin{pmatrix} + & - \\ + & 0 \end{pmatrix}. \quad (8.39)$$

Now the trace is the sum of a positive number and zero, which is positive. Thus by the criterion for stability outlined in Box 6.2, we conclude that the equilibrium is unstable.

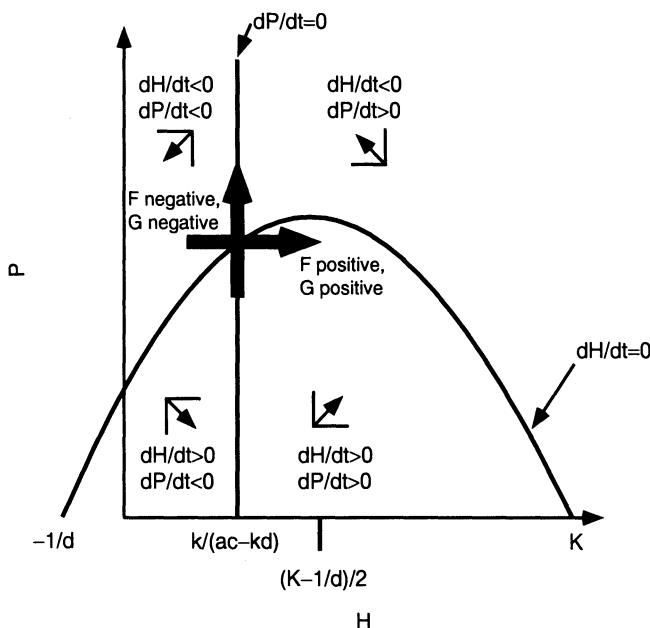


FIGURE 8.13. Phase plane for predator–prey model with type II functional response, unstable case. Contrast the phase plane near equilibrium to the stable case, Figure 8.12.

Ecological implications

We can now discuss what biological changes can cause the system to shift between the stable and unstable cases we have just analyzed. Before doing this we look at the dynamics in the unstable case, which is most easily done by solving the model (8.28)–(8.29) numerically (Figure 8.14). What is important to note from the figure is that in the case where the equilibrium is unstable, the numbers of both species can become very small, so small that if we included any stochastic effects we would expect that one or both species would go extinct. Thus, the inclusion of the functional response produces a model that we can use to explain the observations Gause (1934) made on simple predator–prey systems in the laboratory.

We have seen that the equilibrium of the predator–prey model, when functional response is included, can be either stable or unstable. What are the biological features which lead to stability or instability? The stability of the equilibrium is determined by the

Our deterministic model can never produce the extinction of either species. We need to recognize that when the numbers of either predator or prey become small, we must use intuition and consider that a very small population size in the model may really correspond to extinction if stochastic forces are included.

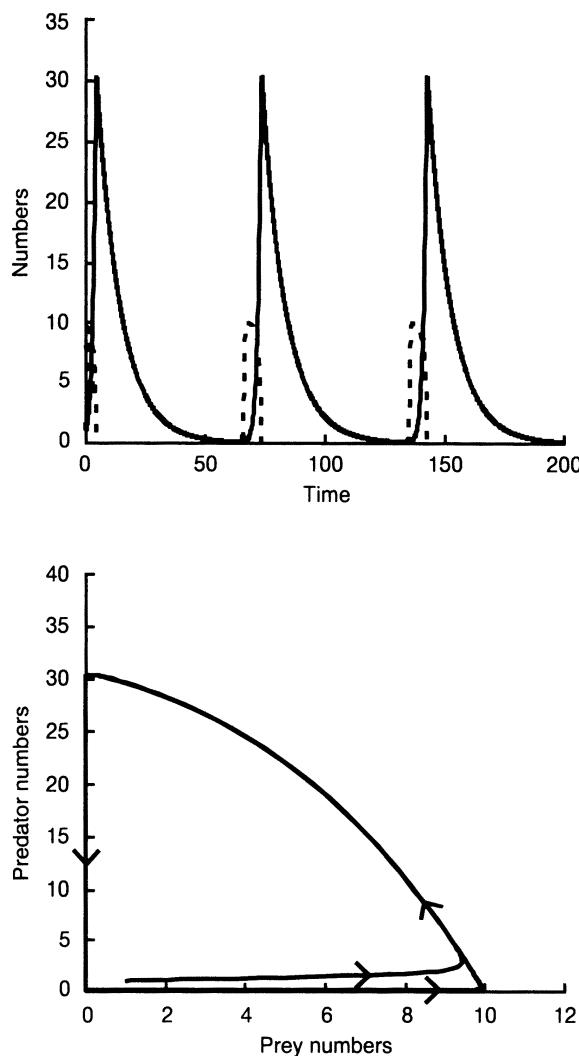


FIGURE 8.14. Dynamics against time (top) and in the phase plane (bottom) for the predator-prey model with a type II functional response, where the equilibrium is unstable. In the plot of numbers against time, the prey is the solid line; the predator is the dashed line. Note how close solutions come to the axes.

location of the predator isocline relative to the ‘hump’ in the prey isocline. The prey ‘hump’ moves to the right if the carrying capacity is increased, so *an increase in the carrying capacity of the prey is a destabilizing influence*. This may at first seem counter-

intuitive, and this concept was originally named the *paradox of enrichment* by Rosenzweig (1971).

Changing the parameter d in the model would also change the location of the ‘hump’ in the prey isocline. To understand the biological meaning of d , note that if $d = 0$, we recover the original type I functional response. Thus, a small value of d corresponds to a predator that is very efficient at consuming prey, a predator that does not require a large handling time (relative to the rate at which prey are captured) to consume the prey. Also notice that reducing d is a stabilizing influence.

Notice that the parameters a and b do not enter at all into stability.

The other way to change stability in the model would be to alter parameters that would move the location of the predator isocline. Increasing k , the death rate of the predator, would move the predator isocline to the right, and would be a stabilizing influence. Increasing c , the ‘efficiency’ of converting captured prey into predators, would be a destabilizing factor.

8.6 Further laboratory study of predation

In a very clever study, Luckinbill (1973) followed up Gause’s (1934) work with *P. aurelia* and its predator *D. nasutum* with a further series of experiments with the same species that provide a striking qualitative fit to the theory we have just developed. Under the standard controlled conditions that Luckinbill used, the predator consumed all the prey in a few hours, just as Gause had observed.

Luckinbill then altered experimental conditions in two ways that corresponded to the stability-changing ideas we have just discussed. He first tried adding methyl cellulose to the experimental media, which increased the viscosity of the medium and thereby reduced the swimming speed of both organisms. This reduced the overall predation rate of *Didinium*. However, the handling time – the time to actually consume an individual captured prey – remained the same. Thus, the effect on the parameters in the model was to reduce d , which would be a stabilizing influence. Indeed, Luckinbill found that the prey population persisted much longer, although eventually all the prey were consumed. Although this

change in conditions was stabilizing, as indicated by the longer persistence time, coexistence was still impossible.

Luckinbill then made one more change in experimental conditions – reducing the food supply for the prey by reducing the density of bacteria (the food for *Paramecium*) in the medium. With this further, stabilizing, change in the experimental conditions, long-term persistence was found. Thus, at least for a simple laboratory system, there is very good *qualitative* agreement between theory and experiment.

8.7 Metapopulation models

We have already seen how a ‘patch’ structure led to persistence in the experiments performed by Huffaker. We now discuss a simple model capturing some of the aspects of this experiment. As with competition, another approach to predation is to consider the effects of spatial structure in a patch model. We will be looking at a system where at any one location in space the predator–prey interaction is always unstable, and asking whether in a system of patches the ‘metapopulation’ structure allows predator and prey to coexist. As in our earlier metapopulation models, we ignore any explicit spatial arrangement of the patches we observe. We also look at the fraction of patches in each state, rather than the absolute numbers.

We begin with the possible transitions at one location in space, given in Figure 8.15. In the simplest model each patch is assumed to go through the following sequence: unoccupied, colonization by prey, colonization by predator, extinction of both species, and return to the unoccupied state.

Colonization of empty patches by prey is assumed to be proportional to the fraction of patches occupied only by the prey. Similarly, colonization by the predator of patches occupied by the prey is assumed proportional to the fraction of patches occupied by the predator. Thus the dynamics of the prey are given by the colonization rate of empty patches by the prey minus colonization

Think of how the assumptions of the model relate to the experiment.

Also consider a model where the prey emigrate from patches occupied by the predator.

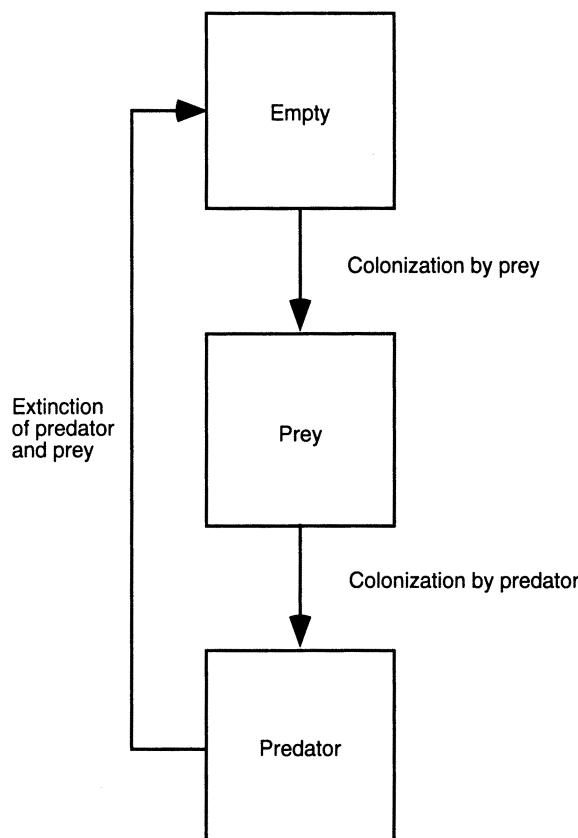


FIGURE 8.15. Transitions among different ‘patch’ states in a simple spatially structured predator–prey model. A ‘patch’ labeled ‘predator’ is one with both predator and prey, where the predator consumes the prey. Here, predators are assumed to be unable to survive in the absence of prey.

of prey patches by the predator;

$$\frac{dp_b}{dt} = m_b p_b (1 - p_b - p_p) - m_p p_b p_p, \quad (8.40)$$

where p_b is the fraction of patches occupied by the prey, m_b is the colonization rate of the prey, and p_p is the fraction of patches occupied by the predator.

The dynamics of the predator are given by the rate of colonization of prey patches minus the extinction rate of patches with the predator. The equation for the dynamics of the predator is

Once again, we assume that predator patches become empty at a fixed rate, recognizing that predator extinction after a fixed time might be more realistic, but more difficult to analyze.

$$\frac{dp_p}{dt} = m_p p_b p_p - e p_p \quad (8.41)$$

where e is the rate at which patches occupied by the predator become empty.

In the problems, you are asked to show that this model invariably leads to a stable equilibrium. Thus, we have shown how a metapopulation structure can lead to persistence of predator and prey that could not coexist within a single habitat. More sophisticated models are needed, however, to produce the oscillatory (rather than equilibrium) dynamics observed by Huffaker (1958).

Think of changes in assumptions that might be destabilizing and lead to oscillations, rather than an equilibrium, in a metapopulation predator-prey model

8.8 Predation in natural systems

How can we use the approach we have developed here to understand natural predator-prey systems? Tanner (1975) attempted to use simple predator-prey models to understand the dynamics of eight pairs of predator and prey. He found rough qualitative agreement between the results from the simple models and whether or not predator-prey cycles were observed in the natural systems.

One of the difficulties of relating the theory we have developed here to interactions between predator and prey in natural systems is finding a relatively contained (in space) system where there is a single predator feeding on a single prey. In most natural systems, predators have alternate prey, and the spatial extent is important, as suggested by our patch model and Huffaker's (1958) experiment. A way to limit the role of spatial extent is to look at an island, which may also serve to limit the number of alternate prey.

The predator-prey interaction between wolves and moose on Isle Royale (located in Lake Superior) has been long studied (Mech, 1966; Taylor, 1984) and can provide insight into the dynamics of predator-prey systems in nature. It is relatively easy to demonstrate that wolves in fact kill moose, but showing that wolves are responsible for regulating the moose population is more difficult; the wolves might only be killing moose that would die anyway. To demonstrate that wolves regulate the moose pop-

ulation, one must show that, as the density of moose increase, the number of deaths from wolf predation also increases. (In all the models, the predation terms include this effect.) The data do not provide a clear-cut answer.

What can essentially be viewed as a field follow-up to Huffaker's study was conducted by Walde et al. (1992), who looked at the dynamics of an herbivorous mite and its predator at two scales: an individual apple tree and the orchard. The study provided strong evidence that spatial structure of the kind studied by Huffaker is important for the persistence of this system. The study also demonstrated that persistence of the predator depended upon alternate prey, again confirming our conclusion that one-predator-one-prey systems are difficult to maintain.

Better evidence for interaction between a single exploiter species and a single victim species comes from the host-parasitoid interactions described in the next chapter.

Problems

1. In this problem we examine the effects of interactions among predators on the stability of a predator–prey system.

We will modify the basic Lotka–Volterra model without density dependence in the prey, equations (8.3) and (8.4). We will be changing the assumption that the per predator predation rate is independent of the number of predators.

We assume that predators help each other, as in the case in which many insects can overwhelm the defenses of a plant (which may be mechanical, such as bark on a tree) only if the density of insects is sufficiently high.

- (a) Write down a modified version of the predator–prey model where the predation term (in both the prey and the predator equations) has the per predator predation rate an increasing function of the number of predators. The exact form of the predation rate is not critical. Leave the other two terms alone.
- (b) Draw the prey isocline. (Will it depend on the number of prey?)

- (c) Draw the predator isocline; a rough sketch will do. (Which way will it ‘tilt’ compared to the Lotka–Volterra case?)
- (d) Use the graphical technique to determine the signs of the terms in the Jacobian (community) matrix.
- (e) Determine the stability of the equilibrium by looking at the sign of the determinant and the trace.
- (f) Discuss (verbally) whether this effect of predator interaction makes sense to you – do insects show outbreaks on plants? What role does the fact that the exact form of the predation rate is not critical here have on your drawing conclusions from this model?
2. Here we analyze the metapopulation model, equations (8.40) and (8.41).
- First find the equilibria of the model.
 - Then find the stability of the equilibria.
- Does the finding that a stable equilibrium always results correspond to the experimental observations of Huffaker? What might explain the differences?
3. A farmer discovered a pest eating his crops. After spraying with a pesticide, the farmer found that the level of the pest increased! Assume that the pest was being eaten by a predator and that the pesticide affected both the predator and the pest. Also assume that the interaction between the pest and the predator can be described by the continuous time Lotka–Volterra model with density dependence in the prey, equations (8.22) and (8.23). Explain the observed results with this model, by adding to each equation a term representing the effect of the pesticide, an additional source of density-independent mortality for both species.
4. Develop a very simple model where the prey have a refuge by modifying the basic Lotka–Volterra model without density dependence in the prey, equations (8.3) and (8.4).
- For the case where a fraction of the prey are free from predation, first write down the modified model. Then

show that the modified model is *mathematically* equivalent to a simple model you have already analyzed: by redefining parameters you recover a model you have seen previously.

- (b) For the case where a fixed number of the prey population is free from predation, analyze the model using a phase plane.
- (c) Discuss the effects of the refuges on stability, comparing the models to the work of Gause.

Suggestions for further reading

The book edited by Crawley (1992) is a recent, in depth summary of predation (as well as parasitism and disease, which we study next). Gause's original (1934) work and the follow-up by Luckinbill (1973, 1974, 1979) are worth reading. Harrison (1995) summarizes the interplay between theory and Luckinbill's 1973 work.

The paper by Huffaker (1958) is an important classic study. The model for patch dynamics is from Hastings (1977), where more complex models are also considered. See also the models in Gurney and Nisbet (1978), and the greenhouse experiments described by Nachman (1981). Walde et al. (1992) studied a mite predator–prey system in the field analogous to the system looked at by Huffaker in the lab.