in one feeding area, recolonized the next a jump ahead of their predators. Thus, Huffaker had effectively created a metapopulation in the laboratory.

Despite the tenuousness of the predator–prey cycle that was achieved, Huffaker's experiment demonstrated that a spatial mosaic of suitable habitats could enable predator and prey populations to coexist through time. Two kinds of time delays caused the populations to cycle: one resulting from the slow dispersal of predators between food patches, and the other resulting from the time needed for predator numbers to increase. As in Gause's experiments with protozoans, predator and prey could not coexist in the absence of suitable refuges for the prey. Huffaker created those refuges by dispersing food patches and creating barriers to predator movements. When the environment is complex enough that predators cannot easily find scarce prey, stable populations or stable population cycles can be achieved. ▌

Simple mathematical models can reproduce cyclic predator–prey interactions

Even before Huffaker's experimental creation of predator–prey cycles in the laboratory, theoretical biologists had developed mathematical models in an attempt to reproduce this population phenomenon on paper. Alfred J. Lotka and Vito Volterra independently developed the first mathematical descriptions of predator–prey interactions during the 1920s. The **Lotka–Volterra model** predicts oscillations in the abundances of predator and prey populations, with predator numbers lagging behind those of their prey.

The Lotka–Volterra model calculates the rate of change in the prey population and the rate of change in the predator population as each is influenced by the abundance of the other. Following a common convention, we designate the number of predator individuals by P and the number of prey individuals by V (think of V for "victim"). The rate of change in the prey population can be written in words as

$$\begin{bmatrix}\text{the rate of change}\\ \text{in the prey population}\end{bmatrix} = \begin{bmatrix}\text{the intrinsic growth rate}\\ \text{of the prey population}\end{bmatrix} - \begin{bmatrix}\text{the removal of prey}\\ \text{individuals by predators}\end{bmatrix}$$

The first term is the unrestricted exponential growth of the prey population in the absence of predators, which we find by multiplying the exponential growth rate (r) by the number of prey individuals (V). The second term is the removal of prey by predators, over and above other causes of death, cVP. The Lotka–Volterra model assumes that predation varies in direct proportion to the probability of a random encounter between a predator and a prey individual, which is the product of the prey and predator populations, VP. Accordingly, the rate of change of the prey population is given by

$$\frac{dV}{dt} = rV - cVP$$

where c is a coefficient expressing the efficiency of predation (think of c for "capture efficiency").

The growth rate of the predator population also has two components: (1) the birth rate, which depends on number of prey captured; and (2) a death rate imposed from outside the system:

$$\frac{dP}{dt} = acVP - dP$$

The birth rate is the number of prey captured (cVP) multiplied by a coefficient (a) for the efficiency with which food is converted into population growth. The death rate is a constant (d) multiplied by the number of predator individuals. Thus, predators have the same probability of dying during a time interval regardless of the predator population density. The expressions for the growth rates of the prey and predator populations are referred to as differential equations because they describe changes in numbers (dV or dP) with respect to a change in time (dt). The Lotka–Volterra model is therefore a continuous-time model.

For the prey in this model, when the term for population increase (rV) exceeds the removal of individuals by predators (cVP)—that is, when $rV > cVP$—the prey population increases. We can rearrange this inequality to give $P < r/c$. Thus, when the predator population is less than the ratio r/c, the prey increase in number. The inequality represents the number of predators that the population of prey can support and still increase. As you can see, this number is higher when the growth potential (r) of the prey population is higher (as when the prey themselves have more food) and when the predators are less efficient (c) at capturing them. When the terms for prey population increase and removal by predators are exactly balanced, the prey population neither increases nor decreases, and is said to be at equilibrium. At this point, $dV/dt = 0$ and $P = r/c$.

The predator population can increase when its own growth potential exceeds its death rate: $acVP > dP$, which can be rearranged to $V > d/ac$. This inequality represents the number of prey required to support the growth of the predator population. This number is higher when the death rate of predators (d) is higher, and it is lower when predators are more efficient at capturing prey (c) and converting them into offspring (a). The predator population achieves an equilibrium size, $dP/dt = 0$, when $V = d/ac$.

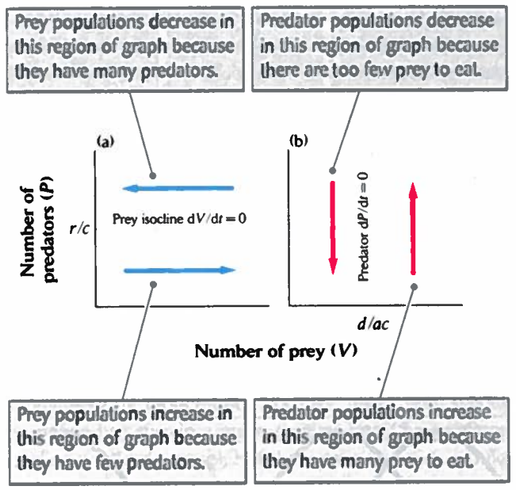

FIGURE 15.13 The equilibrium isoclines for predator and prey populations delineate regions of population increase and decrease. (a) The prey isocline ($dV/dt = 0$ when $P = r/c$) separates regions of prey population increase (low predator numbers) and decrease (high predator numbers). (b) The predator isocline ($dP/dt = 0$ when $V = d/ac$) separates regions of predator population increase (high prey numbers) and decrease (low prey numbers). The two graphs can be superimposed, as in Figure 15.14, to show the pattern of simultaneous change in both populations.

Trajectories of predator and prey populations and the joint equilibrium point

The relationship between predators and prey can be portrayed as a graph with axes representing the sizes of the two populations, as shown in Figure 15.13. By convention, predator numbers increase along the vertical axis and prey numbers along the horizontal axis. The horizontal dotted line at $P = r/c$ in Figure 15.13a represents the condition $dV/dt = 0$ and is called the **equilibrium isocline** (or *zero growth isocline*) for the prey. At any combination of predator and prey numbers that lies in the region below this line, the prey population increases because there are relatively few predators. In the region above the equilibrium isocline, the prey population decreases because predators remove them faster than they can reproduce.

The predator population can increase only when the abundance of prey lies to the right of the vertical dotted line at $V = d/ac$, the equilibrium isocline for the predator (Figure 15.13b). To the right of this line, prey are abundant enough to sustain the growth of the predator population. To the left of the isocline, the predator population decreases because prey are scarce. Thus, the criteria for both predators (P) and prey (V) to remain at equilibrium partition the graph into four regions.

The change in predator and prey populations together follows a closed cycle that combines the individual changes in the predator and prey populations (Figure 15.14). This cycle, called a **joint population trajectory,** can be traced through the four regions of the graph. In the lower right-hand region, for example, both predators and prey increase, and their joint population trajectory moves up and to the right. In the upper right-hand region, prey are still abundant enough that predators can increase, but the increasing number of predators depresses the prey population. Accordingly, the joint population trajectory moves up (more predators) and to the left (fewer prey).

The trajectories in the four regions together define a counterclockwise cycling of predator and prey populations one-fourth cycle out of phase, with the prey population increasing and decreasing just ahead of the predator population (Figure 15.15). Referring back to Figures 15.2

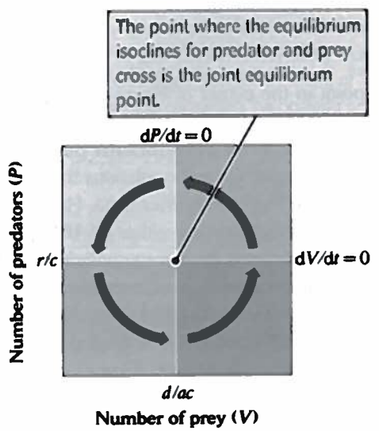

FIGURE 15.14 A joint population trajectory combines the individual changes in predator and prey populations. This trajectory shows the cyclic nature of the predator–prey interaction.

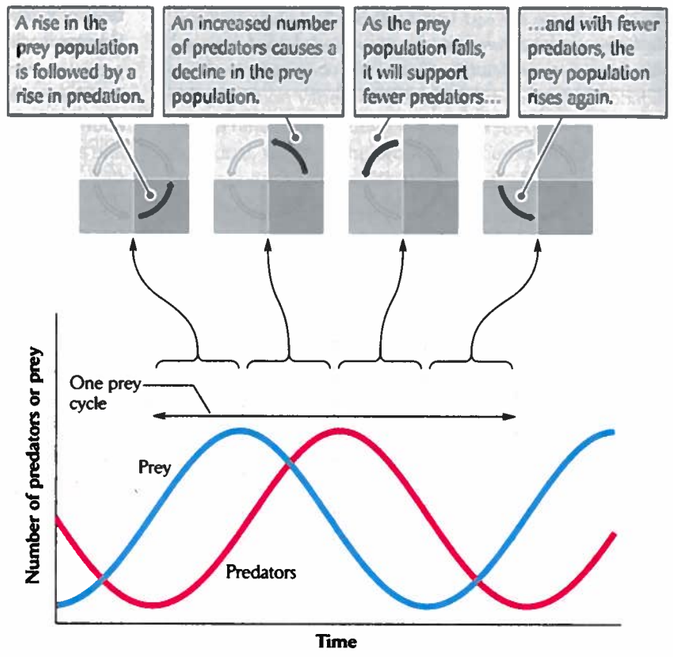

FIGURE 15.15 The Lotka–Volterra model predicts a regular cycling of predator and prey populations. The curves show how predator and prey populations continually cycle out of phase with each other.

and 15.12, for example, you can see that in each cycle, prey populations tend to peak just ahead of predator populations.

LIVING GRAPHS To access an interactive tutorial on the Lotka–Volterra model, go to http://www.whfreeman.com/ricklefs6e.

The point in the center of Figure 15.14, at which the equilibrium isoclines for predator and prey populations cross, is called the **joint equilibrium point.** A combination of predator and prey populations that falls exactly at this point will not change over time. However, in the Lotka–Volterra model, when either of the populations strays ever so little from the joint equilibrium point, they oscillate around it in a continuous cycle rather than returning to it. For this reason, the Lotka–Volterra model is said to exhibit **neutral stability.** The system stays where it is, either at the joint equilibrium point or cycling around it, until it is perturbed. In this sense, the Lotka–Volterra model has no intrinsic stabilizing force. The period of the oscillation (T) is approximately $2\pi/\sqrt{(rd)}$, where π (pi) is a constant, approximately 3.14. For example, if the prey population growth rate were $r = 2$ (200%) per year and the predator death rate were $d = 0.5$ per year, the period of the cycle would be 6.3 years. With a higher prey population growth rate or a higher predator death rate—that is, with a higher rate of population turnover—T would be shorter, and the system would oscillate more rapidly. The amplitude of the cycle depends only on how far the predator and prey populations are displaced from the joint equilibrium point.

It is important to point out that the Lotka–Volterra model is a set of differential (continuous-time) equations, meaning that the populations' responses to change are immediate. Thus, the cycling dynamic of the predator–prey interaction is not caused by time delays in responses, but rather reflects the time required for predator and prey populations to change in size; population responses are immediate, but they are unable to return the system exactly to the joint equilibrium point. The Lotka–Volterra model can also be written in a difference (discrete-time) form that introduces response time delays, but this form of the model produces unstable population cycles and eventual demise of the system. Other models based on difference equations, particularly the Nicholson–Bailey model of parasitoid–host interactions, produce stable cycles, but we will not consider them here.

Returning to the Lotka–Volterra model, the equilibrium isocline for the predator is the minimum number of prey ($V = d/ac$) that can sustain the growth of the predator population. The equilibrium isocline for the prey is the largest number of predators ($P = r/c$) that the prey population can sustain. If the reproductive rate of the prey (r) were to increase, or the capture efficiency of the predators (c) were to decrease, or both, the equilibrium isocline for the prey ($P = r/c$) would move upward. That is, the prey population would be able to bear the burden of a larger predator population, and it would increase. If the death rate of the predators (d) increased and either the prey capture efficiency (c) or the reproductive efficiency (a) of the predators decreased, the equilibrium isocline for the predator ($V = d/ac$) would move to the right, and more prey would be required to support the predator population. Increased predation efficiency (c) alone would simultaneously reduce both isoclines: fewer prey would be needed to sustain a given capture rate (the predator isocline would decrease), but the prey population would be less able to support the more efficient predators.

DATA ANALYSIS DATA ANALYSIS MODULE *Simulation Models of Predator–Prey Interactions.* Try changing variables in the Lotka–Volterra model to see the effects on the period and the amplitude of predator–prey cycles. You will find this module at http://www.whfreeman.com/ricklefs6e.

ECOLOGISTS IN THE FIELD **Testing a prediction of the Lotka–Volterra model.** One of the more surprising predictions of the Lotka–Volterra model is that an increase in the birth rate of the prey (r) should lead to an increase in the population of predators (P), but not in the prey population (V). It is as if the benefit to the prey of some improvement in their environment—a better supply of their own food, for example—is passed directly to their predators.

This prediction was tested by Brendan Bohannan and Richard Lenski of Michigan State University in a simple microcosm experiment. The prey in their system was the bacterium *Escherichia coli*, and the predator was the bacteriophage T4 (a virus that infects bacteria). Populations of bacteria and phage were maintained in a chemostat, a device in which the culture medium is continually replaced by a fresh supply as old medium is removed. In these experiments, the reproductive rate of *E. coli* was limited by the availability of glucose, which was supplied in concentrations of either 0.1 or 0.5 mg per liter of medium. Because a constant influx of new medium was balanced by removal of old medium, the bacteria and phage populations soon reached equilibrium levels. Consistent with the predictions of the Lotka–Volterra model, the higher rate of food provisioning to the bacteria led to an increase in the population of the phage, but not of the bacteria themselves (Figure 15.16). More rapid food provisioning also increased the amplitude of the population cycles by supporting a more rapid rate of increase of the bacterial population, which carried it to higher densities before the phage could catch up. ▌

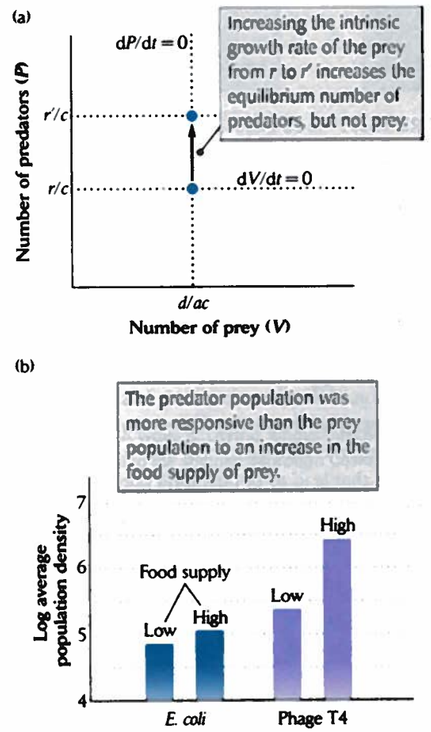

FIGURE 15.16 An increase in the birth rate of prey increases the predator population, but not the prey population. (a) According to the Lotka–Volterra model, an increase in the intrinsic growth rate of the prey population (r) raises the equilibrium isocline for the predator population (r/c), but does not change the equilibrium number of prey. (b) This prediction of the Lotka–Volterra model was tested by increasing the rate of resource (glucose) provisioning to cultures of *E. coli* bacteria in chemostats containing the bacteria and their predators, T4 bacteriophage. After B. J. M. Bohannan and R. E. Lenski, *Ecology* 78:2303–2315 (1997).

Pathogen–host dynamics can be described by the S-I-R model

Relationships between pathogens and their hosts can be built into models that are similar to the Lotka–Volterra predator–prey model. Such models add to our

FIGURE 15.17 The S-I-R model simulates pathogen–host interactions. Individuals in a host population are initially susceptible to a new pathogen (*S*). They become infected (*I*), during which time they can infect other individuals, then recover (*R*) and become resistant to further infection.

understanding of infectious diseases. Pathogens, unlike predators, do not always remove host individuals from a population. However, because hosts may develop immune responses that make some individuals resistant to the pathogen, the pathogen–host interaction can develop time delays that lead to population cycling.

The simplest model of infectious disease transmission that incorporates immunity is the **S-I-R model.** The *S* in S-I-R stands for susceptible individuals, *I* for infected individuals, and *R* for recovered individuals with acquired immunity (Figure 15.17). We can use this model to examine the course of a short-lived epidemic as it moves through a population.

A host individual infected by a pathogen (the primary case of the disease) will spread the disease to others, creating secondary cases. The course of the epidemic depends on two opposing factors: the rate of transmission (b) and the rate of recovery (g). The variable b includes the rate of contact of susceptible individuals with an infectious individual as well as the probability of infection given contact. The variable g determines the period over which an individual is infectious. The reproductive ratio, R_0, is defined as the number of secondary cases produced by a primary case during its period of infectiousness, where $R_0 = (b/g)S$. Thus, R_0 is the ratio of the rate of transmission (b) to the rate of recovery (g) times the number of susceptible individuals in the population (S).

Using the S-I-R model, we can ask whether the introduction of a small number of infectious individuals into a susceptible population at time 0 will cause an epidemic of the disease. If $R_0 > 1$, then a chain reaction will occur, and an epidemic will ensue, because each infected individual infects more than one other host individual before it recovers from the disease. When $R_0 < 1$, the infection fails to take hold in the host population because infected individuals fail to generate a single new infection, on average, before they recover. Even when an epidemic begins, as more individuals are infected and subsequently recover and become resistant (R), the number of susceptible individuals (S) decreases, and the value of R_0 decreases in parallel. When it reaches $R_0 < 1$, the epidemic can no longer sustain itself.

Typical values for R_0 in childhood diseases of humans (measles, chicken pox, mumps, etc.) range from 5 to as high as 18. HIV, which is limited primarily by its mode of transmission by direct sexual contact or blood transfusion, has an R_0 value of 2–5. At the other extreme, malaria, which is transmitted by a mosquito vector, has an R_0 value greater than 100 in crowded human populations; infected people remain infectious for long periods, and mosquitoes are efficient vectors. The course that a typical disease epidemic might take is illustrated in Figure 15.18.

The basic S-I-R model includes no births of new susceptible individuals, nor loss of resistance among previously infected individuals, so the epidemic simply runs its course until all the individuals in the population are

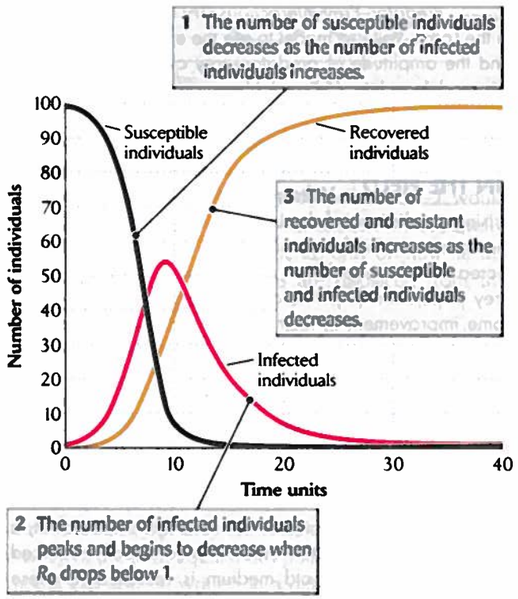

FIGURE 15.18 The S-I-R model can predict the spread of an epidemic through a host population. For this simulation, the size of the host population is arbitrarily set at 100 individuals, so that *S*, *I*, and *R* are expressed as numbers of individuals and also as percentages of the host population. R_0 is expressed ×100. At the beginning of the epidemic, when *S* is close to 1, $R_0 = 5$. The infection rate (b) is 1, the recovery rate (g) is 0.2, and the duration of infectiousness ($1/g$) is 5 time units.

recovered and resistant or too few susceptible individuals remain to sustain the spread of the disease. Influenza viruses spread through the human population in this manner. The effect of vaccination in this model is to remove individuals from the susceptible population, thus reducing the value of R_0 and reducing the probability that an epidemic can sustain itself.

Other factors can be added to the model, including births of susceptible infants, latency between infection and infectiousness, disease-dependent host mortality, host population dynamics, and vertical transmission of disease from parent to offspring. When recovered individuals lose their immunity and become susceptible again, the pathogen can produce periodic epidemics within the host population. For example, if an infected individual is infectious for 1 week and retains immunity for 5 years thereafter, the period between outbreaks of a disease with $R_0 = 5$ is almost exactly 1 year. Births of newly susceptible individuals have the same effect of increasing the number of susceptible individuals and creating periodic epidemics, as shown in Figure 15.9 for measles.

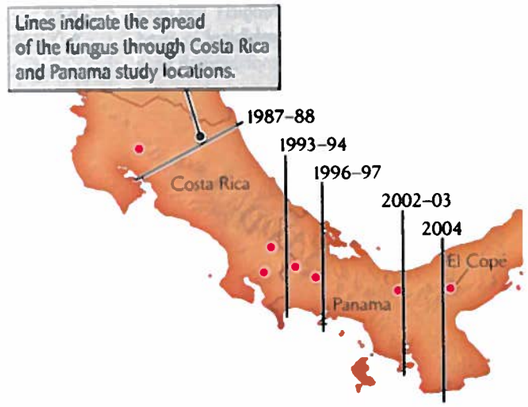

FIGURE 15.19 A wave of chytrid fungus infection spread from the northwest to the southeast through Costa Rica and Panama from 1987 to 2004. The red dots indicate locations sampled for infected amphibians. From K. Lips, et al., *Proc. Natl. Acad. Sci. USA* 103:3165–3170 (2006).

ECOLOGISTS IN THE FIELD

The chytrid fungus and the global decline of amphibians. Most species of amphibians are declining worldwide, and many have already gone extinct. Amphibians are particularly sensitive to pollutants and changes in climate, but the most important cause of this population decline appears to be a fungal pathogen. As predicted by the S-I-R model, we would expect a typical pathogen to cause a host species to decline until there are too few susceptible hosts to support the continued spread of the pathogen. Accordingly, we would not expect a pathogen to drive a host species to extinction. However, the recently discovered pathogen in amphibians doesn't seem to follow this expectation.

In 2006, Karen Lips, of Southern Illinois University, and her colleagues documented the spread of the pathogenic fungus *Batrachochytrium dendrobatidis*, commonly called the chytrid fungus, throughout Central America. The origin of this fungus is not yet known, but it appears to be a recent arrival in Central America. Unlike many other pathogens, *B. dendrobatidis* can infect a wide variety of amphibian species. Thus, if the fungus kills off one host species, it can persist by infecting alternative host species. Such a pathogen poses a major threat to the persistence of entire groups of species.

Lips's team of researchers decided to document the spread of *B. dendrobatidis* among amphibians in Central America (Figure 15.19). They sampled more than 1,500 amphibians at a site in El Copé, Panama, where the fungus had not yet arrived. Between 2000 and July 2004, not one individual tested positive for *B. dendrobatidis*. By October 2004, however, 21 of 27 species sampled had a greater than 10% prevalence of the fungus in their populations. By December 2004, 40 species tested positive for the fungus.

During their years of testing species for the presence of the fungus, the researchers had also been counting amphibians in El Copé as they walked transects through amphibian habitat to estimate the population sizes of each species. At the end of 2004, however, coincident with the arrival of the fungus, the numbers of live amphibians that were counted along the transects declined sharply (Figure 15.20), while the number of dead amphibians increased. The dead amphibians included 38 different frog species, and 99% of the 318 dead individuals collected had moderate to severe chytrid infections.

While it remains unknown exactly how *B. dendrobatidis* kills its hosts, the fungus clearly has been responsible for the massive die-off of frogs in Central America. Extinction is always difficult to prove because a few individuals may still exist in an area undetected. However, many of the species involved in the die-off have not been seen for several years, and they are almost certainly extinct. The fungus is also appearing in other parts of the world, with similar effects. Hence, *B. dendrobatidis* poses a major threat to amphibian conservation around the world. An important message that emerges from this research is that when a pathogen is not restricted to a single host species, it has the ability to persist and spread even after it drives one of its hosts extinct.