Chapter 10
Ecological Context of Epidemiology

10.1 Infectious Diseases in Animal Populations

Infectious disease pathogens affect numerous animal populations. An animal **population**, by definition, is a collection of individuals of the same species occupying the same habitat. A *species* is a group of individuals who generally breed among themselves and do not naturally interbreed with members of other groups. Fortunately, familiar baseline models of infectious diseases in humans such as the SI, SIS, SIR, SIRS models also can be used to model diseases in animal populations. There are some very important distinctions, however, that we will discuss below.

Why is it important to study diseases in animal populations?

- The simplest and most obvious reason is that a nonhuman species, such as species of fish, birds, and other mammals, represents a much simpler biological system for scientific study than the human species. These species can often be manipulated for better understanding of its properties and dynamics. One such example, in which population dynamics principles were validated through experiments with a beetle population is given in [47].
- There is also a practical reason to study animal diseases. Historically, human diseases have been inextricably linked to epidemics in animal populations. Rapid expansion of civilization in the last few millennia has increased the contact at the human–animal interface, through urban and agricultural expansion that encroaches on wildlife habitats, domestication of cattle and other livestock, or simply by keeping pets. Because of such close intimacy between humans and nonhuman animals, viruses and bacteria that cause various animal diseases continuously "jump across the species barrier" and infect humans. Recent examples include HIV, whict came from monkeys; SARS, which came from bats; and avian flu H5N1, which was first found in wild birds. In fact, about 60% of all human infectious diseases have their origin in animal species, and about three-quarters

M. Martcheva, *An Introduction to Mathematical Epidemiology*,
Texts in Applied Mathematics 61, DOI 10.1007/978-1-4899-7612-3_10

of all *emerging* infectious diseases of humans—those that are occurring for the first time in humans—have been traced back to nonhuman species. Thus, understanding disease dynamics and ways to control disease in animal populations has tremendous human health implications.
- Another practical reason to study disease in natural populations is that the disease can be a regulator of a population, leading to control of dangerous pests [11, 114]. Furthermore, parasites can have substantial effect on community composition [76].

Epidemic models of human populations assume that the population being considered is a closed population, completely separate from the rest of the world. This is rarely true for animal populations. Each animal species is a part of an intricate web of ecological interactions. Interacting populations that share the same habitat form a *community*. Two fundamental community interactions are *competition* for resources and *predation*. Such interspecies interactions impart complex feedback on the dynamics of the diseases in the population that is studied. This necessitates the integration of the principles of infectious disease epidemiology and community ecology.

There are several ways in which the disease dynamics within a population are influenced by the interaction with other species in the community. Here are some of the major ways in which the community interactions influence the host–pathogen dynamics:

- Often, a single pathogen simultaneously infects many different species in the community. Even if the pathogen is completely removed from a given host, it may survive in the other hosts and can reinfect the original host. This is of particular concern if one of the species being infected is humans, while another species is an animal species.
- Intense competition of the focal host species with the other species in the community can bring down the host population size to threateningly small levels, and a pathogen attack can eventually drive it to extinction.
- Predation of the host by other species in the community can regulate pathogen outbreaks in the host species.

Conversely, host–pathogen interactions can feed back and impact the community by weakening the competitive abilities of the affected species.

In this chapter, we will consider the interrelation between infectious diseases and the two principal community interactions: predation and competition. *Predation*, or *predator–prey interactions*, refers to the feeding on the individuals of a *prey* species by the individuals of a *predator* species for the survival and growth of the predator. Some common examples of predator–prey interactions are big fish eating small fish, birds feeding on insects, and cats eating rats and mice. The predator itself can be of two types—a *specialist* predator, whose entire feeding choice is restricted to a single prey species, and a *generalist* predator that feeds on many different prey species. The dynamics of a specialist are strongly coupled to those of the prey, so that any

rise or fall of the population size of the prey triggers a consequent rise or fall in the population size of its predator. The interaction between a specialist predator and its prey is modeled by the familiar Lotka–Volterra predator–prey model. By contrast, the dynamics of a generalist predator are only weakly coupled to any particular focal prey species and may not be influenced by the dynamics of the prey. The predation of a focal species by a generalist predator can be modeled by a predator-added mortality on the prey.

10.2 Generalist Predator and SI-Type Disease in Prey

We can begin studying the effect of predation on disease growth in a host population by considering a host to the disease that is also a prey to a generalist predator. We will model the disease dynamics in the prey by the familiar SI epidemic model that we have used for humans. We will also assume that the predator is a "complete generalist," so that any change of the predator's population size P is caused by other factors and is independent of the dynamics of the focal prey that we are studying. In mathematical terms, we do not need another equation for the rate of change of P, and P is not a dynamic variable but enters the SI model via the death rate of the prey population as a free parameter. Then we have the following system modeling the dynamics of a host–pathogen interaction with predation:

$$\begin{aligned} S'(t) &= \Lambda - \beta SI - \mu(P)S, \\ I'(t) &= \beta SI - \mu(P)I, \end{aligned} \tag{10.1}$$

where Λ is the prey birth rate, β is the transmission parameter, and $\mu(P)$ is the prey death rate, which is split into two components:

$$\mu(P) = \mu_0 + \mu_P(P).$$

The constant μ_0 is a parameter that lumps death rate from the disease, other natural causes, accidental factors, and so on. The function $\mu_P(P)$ is the predation-added mortality of the prey that depends on the predator abundance P, taken as a parameter. For simplicity, we assume that μ_P increases linearly with P, that is, $\mu_P = aP$, where the parameter a denotes the rate at which the predator captures its prey. In general, there should be two different capture rates: a_S and a_I, for susceptible and infected individuals. For instance, the predator may be able to capture physically weak infected prey more easily than susceptible prey, that is, $a_I > a_S$. Here we shall consider two extreme kinds of predation: *selective predation*, in which the predator attacks only one prey type, leaving the other one alone, and *indiscriminate predation*, in which the predator attacks all prey types nonpreferentially. For instance, in selective predation of infected prey alone, we have $a_I > 0$, $a_s = 0$, and in indiscriminate predation, we have $a_S = a_I = a$.

10.2.1 Indiscriminate Predation

In this subsection, we assume that the predator preys indiscriminately on all prey types. Hence,

$$\mu(P) = \mu_0 + aP.$$

We obtain the familiar equation $N'(t) = \Lambda - \mu(P)N$ for the total population size of the prey. From this equation, the equilibrium total population size of the prey becomes

$$N^* = \frac{\Lambda}{\mu} = \frac{\Lambda}{\mu_0 + aP}.$$

So N^* decreases with increasing predator numbers P, as expected. Furthermore, we can solve for the equilibrium number of infected individuals:

$$I^* = \frac{\beta\Lambda - (\mu_0 + aP)^2}{\beta(\mu_0 + aP)}. \tag{10.2}$$

Throughout this chapter, we will call I^* the *disease load*, and the fraction of the infected individuals in the total prey population size will be called the *prevalence*:

$$p = \frac{I^*}{N^*}.$$

From the expressions above for the disease load and the total population size, we have that the prevalence is given by

$$p^* = \frac{\beta\Lambda - (\mu_0 + aP)^2}{\beta\Lambda}. \tag{10.3}$$

From (10.2) and (10.3), we see that both the disease load and the prevalence are decreasing functions of the predator numbers P. The condition for pathogen establishment requires that the numerators of (10.2) and (10.3) be positive, which gives the familiar threshold condition

$$\mathscr{R}_0 = \frac{\beta\Lambda}{(\mu_0 + aP)^2} > 1. \tag{10.4}$$

We see again that the reproduction number of the disease is a decreasing function of the predation level P. We can derive a minimum equilibrium host population size $N^*_{\min}$ that is needed to support the pathogen in the host. To do this, we replace the term $\mu_0 + aP$ in the expression for $\mathscr{R}_0$ above by Λ/N^* and obtain the inequality

$$N^* > N^*_{\min} = \sqrt{\frac{\Lambda}{\beta}}.$$

Thus, $I^* = 0$ and $p^* = 0$ when $N^* < N^*_{\min}$. We have the following interesting result:

While the direct effect of the predator is to harm the prey by increasing its mortality rate, predation can indirectly benefit the prey by keeping its population size low and thereby ruling out epidemic outbreaks.

10.2.2 Selective Predation

In the previous subsection, we assumed indiscriminate predation, whereby the predator's capture rate a is the same for both susceptible and infected prey. In this subsection, we want to see what happens under selective predation, that is, when the predator attacks either the susceptible prey alone $a_S > 0, a_I = 0$, or the infected prey alone, $a_S = 0, a_I > 0$. Even though the first scenario is less likely, it can occur in certain situations such as the infected prey hiding in burrows to avoid predation, or the predator may deliberately avoid infected prey to prevent infecting itself. In the case of selective predation of susceptible prey alone, the model (10.1) becomes

$$\begin{aligned} S'(t) &= \Lambda - \beta SI - (\mu_0 + a_S P)S, \\ I'(t) &= \beta SI - \mu_0 I. \end{aligned} \tag{10.5}$$

The equilibrium disease load is given by

$$I^* = \frac{\beta\Lambda - \mu_0(\mu_0 + a_S P)}{\beta\mu_0}.$$

We see that the equilibrium disease load decreases linearly with increasing predator numbers P. To obtain the equilibrium prey population size, we solve for S^* from the second equation above to obtain

$$S^* = \frac{\mu_0}{\beta}$$

and then use the fact that $N^* = S^* + I^*$. We obtain

$$N^* = \frac{\beta\Lambda - \mu_0 a_S P}{\beta\mu_0}.$$

Hence, the equilibrium prevalence is given by

$$p^* = \frac{I^*}{N^*} = \frac{\beta\Lambda - \mu_0(\mu_0 + a_S P)}{\beta\Lambda - \mu_0 a_S P} = 1 - \frac{\mu_0^2}{\beta\Lambda - \mu_0 a_S P}.$$

From the last expression, we see that p^* is also decreasing with increasing P. The threshold predation level for which $p^* = 0$ is

$$P_{\text{crit}} = \frac{\beta\Lambda - \mu_0^2}{\mu_0 a_S}.$$

We note that the denominator of p^* is positive for all $P < P_{\text{crit}}$.

For the case of selective predation on infected prey alone, the system becomes

$$\begin{aligned} S'(t) &= \Lambda - \beta SI - \mu_0 S, \\ I'(t) &= \beta SI - (\mu_0 + a_I P)I. \end{aligned} \tag{10.6}$$

Following steps similar to those have taken previously, we get an expression for the prevalence p^* as follows:

$$p^* = \frac{I^*}{N^*} = \frac{\beta\Lambda - \mu_0(\mu_0 + a_I P)}{\beta\Lambda + a_I P(\mu_0 + a_I P)}.$$

We see again that the prevalence is decreasing with increasing P. So the qualitative nature of the result—predation lowers epidemic outbreaks in the prey—is similar to the case of indiscriminate predation discussed in the previous subsection.

In summary, with an SI model, we see that predation reduces disease load and prevalence in the prey population, irrespective of whether the predator selectively attacks either susceptible or infected prey, or indiscriminately preys on both prey types. The main reason is that the incidence rate βSI, which gives the rate at which new infections appear in the host, depends bilinearly on both the susceptible and infected prey. Therefore, whether the predator eats selectively or indiscriminately, it always reduces the incidence βSI, and hence the disease level in the prey population. In the next section, we discuss disease in prey with permanent recovery, and investigate the impact of the predator on the disease load and prevalence of the disease in the prey population.

10.3 Generalist Predator and SIR-Type Disease in Prey

In this section, we consider an SIR model with predation by a generalist predator. The presence of a recovered class of individuals makes a difference. Since the number of recovered individuals R does not directly contribute to the disease transmission, the predation of the recovered prey can have only an indirect effect on disease growth. The addition of a recovered class will give very different conclusions based on the different dietary choices of the predator. We consider the following SIR model with predation of a generalist predator:

$$\begin{aligned} S'(t) &= \Lambda - \beta SI - \mu(P)S, \\ I'(t) &= \beta SI - (\alpha + \mu(P))I, \\ R'(t) &= \alpha I - \mu(P)R. \end{aligned} \tag{10.7}$$

The prey death rate $\mu(P)$ is defined as before, $\mu(P) = \mu_0 + aP$. This is the death rate for indiscriminate predation. We will consider two cases of selective predation: predation on infected prey alone, $a_I > 0, a_S = a_R = 0$, and predation on recovered prey alone, $a_R > 0, a_S = a_I = 0$.

10.3.1 Selective Predation

We begin with selective predation on infected prey:

$$\begin{aligned} S'(t) &= \Lambda - \beta SI - \mu_0 S, \\ I'(t) &= \beta SI - (\alpha + \mu_0 + a_I P)I, \\ R'(t) &= \alpha I - \mu_0 R. \end{aligned} \tag{10.8}$$

Solving for the disease load in the endemic equilibrium, we have

$$I^* = \frac{\beta\Lambda - \mu_0(\alpha + \mu_0 + a_I P)}{\beta(\alpha + \mu_0 + a_I P)}.$$

We compute the basic reproduction number from the requirement that $I^* > 0$,

$$\mathscr{R}_0 = \frac{\beta\Lambda}{\mu_0(\alpha + \mu_0 + a_I P)}, \tag{10.9}$$

and the condition $\mathscr{R}_0 > 1$. It is clear that both the disease load and the basic reproduction number decrease with predation level P. We need to know the total prey population size at equilibrium N^* to find out the prevalence $p^* = I^*/N^*$. We solve for S^* and R^* and compute $N^* = S^* + I^* + R^*$:

$$N^* = \frac{\beta\Lambda(\alpha + \mu_0) + \mu_0 a_I P(\alpha + \mu_0 + a_I P)}{\beta\mu_0(\alpha + \mu_0 + a_I P)}.$$

The equilibrium prevalence is then given by

$$p^* = \frac{I^*}{N^*} = \frac{\mu_0[\beta\Lambda - \mu_0(\alpha + \mu_0 + a_I P)]}{\beta\Lambda(\alpha + \mu_0) + \mu_0 a_I P(\alpha + \mu_0 + a_I P)}.$$

It can be seen that p^* decreases with increasing predator numbers P at a rate faster than linear. Similar results can be obtained in the case of selective predation on susceptible individuals only, that is, $a_S > 0, a_I = a_R = 0$. Selective predation on susceptible prey decreases the incidence of the disease βSI and therefore also decreases the disease load and the prevalence.

Now we turn to selective predation of the predator on the recovered individuals, that is, $a_R > 0, a_S = a_I = 0$. The model becomes

$$\begin{aligned} S'(t) &= \Lambda - \beta SI - \mu_0 S, \\ I'(t) &= \beta SI - (\alpha + \mu_0) I, \\ R'(t) &= \alpha I - (\mu_0 + a_R P) R. \end{aligned} \tag{10.10}$$

The equilibrium disease load is

$$I^* = \frac{\beta\Lambda - \mu_0(\alpha + \mu_0)}{\beta(\alpha + \mu_0)}.$$

This is an interesting result—the disease load is independent of the predation level P and remains constant with change of P. This happens because both susceptible and infected prey are ignored by the predator, and therefore the incidence rate βSI is unaffected by predation, and so is the disease load I^*. Furthermore, the condition $I^* > 0$ gives the basic reproduction number $\mathscr{R}_0$:

$$\mathscr{R}_0 = \frac{\beta\Lambda}{\mu_0(\alpha + \mu_0)}, \tag{10.11}$$

which is also independent of the predation level P. An implication of this result is that if the inequality $\mathscr{R}_0 > 1$ holds in the absence of predation $P = 0$, it does not change thereafter with increasing P, and thus the pathogen never becomes extinct as a result of predation. The equilibrium prey population size N^* is obtained as the sum $N^* = S^* + I^* + R^*$,

$$N^* = \frac{\beta\Lambda(\alpha + \mu_0 + a_R P) + \alpha a_R(\alpha + \mu_0)}{\beta(\alpha + \mu_0)(\mu_0 + a_R P)},$$

which then gives equilibrium prevalence:

$$p^* = \frac{I^*}{N^*} = \frac{(\mu_0 + a_R P)[\beta\Lambda - \mu_0(\alpha + \mu_0)]}{\beta\Lambda(\alpha + \mu_0 + a_R P) + \alpha a_R P(\alpha + \mu_0)}.$$

Unlike I^* and $\mathscr{R}_0$, the prevalence p^* depends on P, because the equilibrium total prey population size N^* depends on P. The behavior of p^* with increasing P^* is not so obvious, so we consider the derivative of p^* with respect to P:

$$\frac{dp^*}{dP} = \frac{\alpha a_R[\beta\Lambda - \mu_0(\alpha + \mu_0)]^2}{[\beta\Lambda(\alpha + \mu_0 + a_R P) + \alpha a_R P(\alpha + \mu_0)]^2}.$$

This shows that $dp^*/dP > 0$, and therefore, if the prevalence is positive in the absence of predation, i.e., $\mathscr{R}_0 > 1$, it increases continuously with P. This result is not entirely surprising in light of the fact that the disease load I^* remains constant with increasing prevalence. Since we can expect that the total population size N^* decreases with increasing P, we should expect that the ratio, the prevalence p^*, is increasing with P.

Therefore, once the pathogen is established in the prey, $\mathscr{R}_0 > 1$, the prevalence decreases with increasing predation level P when the predator selectively attacks

susceptible or infected prey only. In contrast, prevalence *increases* with increasing predation level P when the predator attacks preferentially the recovered prey. This last result, although counterintuitive, can be explained by the fact that by attacking recovered prey alone, the predator decreases total population size without impacting the incidence of the disease βSI.

10.3.2 Indiscriminate Predation

As a last case, we consider indiscriminate predation, that is, $a_S = a_I = a_R = a$. The system in this case becomes

$$\begin{aligned} S'(t) &= \Lambda - \beta SI - (\mu_0 + aP)S, \\ I'(t) &= \beta SI - (\alpha + \mu_0 + aP)I, \\ R'(t) &= \alpha I - (\mu_0 + aP)R. \end{aligned} \tag{10.12}$$

Solving for the equilibrium level of the disease load, we obtain

$$I^* = \frac{\beta\Lambda - (\mu_0 + aP)(\alpha + \mu_0 + aP)}{\beta(\alpha + \mu_0 + aP)}.$$

The condition that the disease load has to be positive, $I^* > 0$, gives the following basic reproduction number of the disease:

$$\mathscr{R}_0 = \frac{\beta\Lambda}{(\mu_0 + aP)(\alpha + \mu_0 + aP)}.$$

We see that the reproduction number is decreasing with increasing predation level P. In this case, we can obtain an equation for the total population size. Adding all equations in (10.12), we obtain

$$N'(t) = \Lambda - (\mu_0 + aP)N.$$

From the above equation, we obtain the equilibrium total prey population size

$$N^* = \frac{\Lambda}{\mu_0 + aP}. \tag{10.13}$$

We can derive the minimum prey population size $N^*_{\min}$ needed for persistence of the pathogen by plugging (10.13) into the expression for $\mathscr{R}_0$ and using the condition $\mathscr{R}_0 > 1$:

$$N^* > N^*_{\min} = \frac{1}{\beta}(\alpha + \mu_0 + aP).$$

Thus, the pathogen is extinct, $I^* = 0$, when $\mathscr{R}_0 \leq 1$ and equivalently $N^* \leq N^*_{\min}$. We can obtain the equilibrium prevalence:

$$p^* = \frac{I^*}{N^*} = \frac{(\mu_0 + aP)[\beta\Lambda - (\mu_0 + aP)(\alpha + \mu_0 + aP)]}{\beta\Lambda(\alpha + \mu_0 + aP)}. \tag{10.14}$$

It is clear from this expression that the prevalence p^* is not monotone with respect to the predation level P. Looking at the derivative dp^*/dP,

$$\frac{dp^*}{dP} = \frac{\beta\Lambda\alpha - 2(\mu_0 + aP)(\alpha + \mu_0 + aP)^2}{\beta\Lambda(\alpha + \mu_0 + aP)^2},$$

we see that no clear conclusion can be drawn about the sign, and it will depend on the value of the predator population size P. Since for P large enough, the numerator and the whole derivative dp^*/dP are negative, if for $P = 0$ we have

$$\beta\Lambda\alpha > 2\mu_0(\alpha + \mu_0)^2,$$

then $dp^*/dP > 0$ for small P and $dp^*/dP < 0$ for large P. Hence, we can expect a humped-shaped plot of the prevalence p^*, increasing for small P and decreasing for large P.

Thus, we are faced with an intriguing situation. The basic reproduction number $\mathscr{R}_0$ and the disease load I^* decrease with increasing predation pressure P, which tends to give the impression that the epidemic in the prey is weakened in the presence of predation. However, prevalence p^*, which gives another measure of the disease in the population, increases, at least for low predation level P. The reason is that even though both I^* and N^* both decrease with increasing P, N^* falls faster than I^* for low values of P, and therefore the ratio I^*/N^*, which gives the prevalence, increases for those values of P. In other words, while both the population size of the prey and the number of infective hosts are depressed under indiscriminate predation, the proportion of infective individuals in the population increases for low levels of P. The epidemiological significance of this result is that the overall disease burden in the population is reduced, but the risk that a randomly chosen individual is infected can increase with predation, at least at small predation levels.

10.4 Specialist Predator and SI Disease in Prey

Specialist predators prey on a given species, which is our focal species. The dynamics of a specialist predator are closely linked to those of the prey. These dynamics are those described by the Lotka–Volterra predator–prey model. In the next subsection, we discuss various types of predator–prey models.

10.4.1 Lotka–Volterra Predator–Prey Models

The Lotka–Volterra predator–prey model was initially proposed by Alfred J. Lotka in the theory of autocatalytic chemical reactions in 1910. In 1925, Lotka used the equations to analyze predator–prey interactions in his book *Elements of Physical Biology*, deriving the equations that we know today. Vito Volterra, who was interested in statistical analysis of fish catches in the Adriatic, independently investigated the equations in 1926. The equations are based on the observation that the predator–prey dynamics are often oscillatory. The Lotka–Volterra model makes a number of assumptions about the environment and evolution of the predator and prey populations:

- The prey population finds ample food at all times.
- The food supply of the predator population depends entirely on the prey population.
- The rate of change of population is proportional to its size.
- During the process, the environment does not change in favor of one species, and genetic adaptation is sufficiently slow.

The prey population size $N(t)$ grows exponentially in the absence of the predator and dies only as a result of predation. Thus the prey equation takes the form

$$N'(t) = rN - \gamma NP,$$

where $P(t)$ is the predator population size, and r is the growth rate of the prey. The rate of predation on the prey is assumed to be proportional to the rate at which the predators and the prey meet, and it is described by a mass action term γNP. The predator depends entirely for food on the supply of prey and will die exponentially if no prey is available at a natural death rate d. The equation for the predator takes the form

$$P'(t) = \varepsilon\gamma NP - dP,$$

where ε is the conversion efficiency of the predator, that is, ε is a measure of the predator metabolic efficiency by which the biomass of the prey eaten is converted into biomass of the predator. The Lotka–Volterra predator–prey model takes the form

$$\begin{aligned} N'(t) &= rN - \gamma NP, \\ P'(t) &= \varepsilon\gamma NP - dP. \end{aligned} \tag{10.15}$$

The term γNP is called the *predation term*. The per capita rate at which the predator consumes the prey, γN in this model, is called the *predator's functional response*. The predator's functional response term in this model is linear in the prey population size N. This model also assumes that there is no interference among predators in finding prey. In other words, encounters of predators do not reduce the efficiency of search for prey. Mathematically, this is expressed in the fact that the predation term is linear in P. The combination of the assumption of linear functional response and

that of no interference between predators leads to a term proportional to the product NP, which is the mass action predation term. The predator–prey model (10.15) has an extinction equilibrium $\mathscr{E}_0 = (0,0)$. The model also has one coexistence equilibrium

$$\mathscr{E} = \left(\frac{d}{\varepsilon\gamma}, \frac{r}{\gamma}\right).$$

It can be shown that the orbits of the predator–prey system (10.15) are closed curves around the coexistence equilibrium (see Fig. 10.1). Since the solutions are closed orbits, they are periodic. From the direction of the vector field, it can be seen that the solution curves in the (N,P)-plane run counterclockwise. Thus, the maximum prey population comes about one-quarter of a cycle before the maximum predator population. The predator population's fluctuations follow those of the prey population through time. That is, the prey population begins to increase while the predator population is still decreasing, and the prey population decreases while the predator population is still increasing. The classic (and simplest) explanation of these cycles is that the predator drives the changes in the prey population by catching and killing its members, and the prey as the predator's sole food supply drives the predator's population changes, but a lag between the population responses

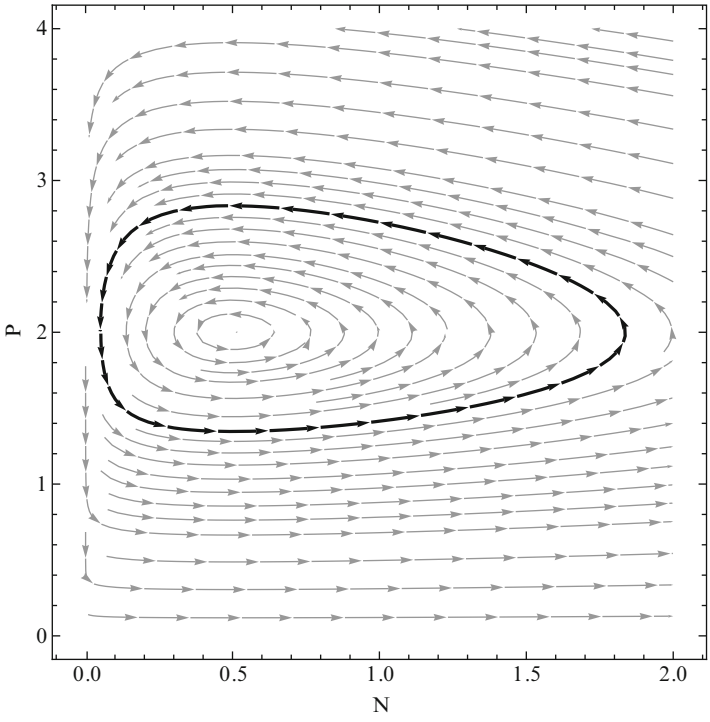

Fig. 10.1 Predator–prey cycles in the (N,P)-plane. The vector field shows that the orbits are traversed counterclockwise

of predator and prey cause the two cycles to be out of phase. However, this explanation has been challenged, and it may not be the only viable explanation for the pattern.

The Lotka–Volterra model represents one of the early triumphs of mathematical modeling, because it captures the oscillatory behavior observed in natural predator–prey systems with a specialist predator. Unfortunately, the model cannot explain these oscillations, because the oscillations in the model are **structurally unstable**, that is, small changes to the model can significantly change the qualitative behavior of the model, e.g., it can stabilize the oscillations. Ideally, we would like the oscillations in the model to be structurally stable, that is, if we make small changes to the model to better reflect reality, the qualitative predictions of the model remain the same, and in particular, the model continues to exhibit oscillations.

The first modification in the Lotka–Volterra model that is natural to be considered is the possibility that the prey population is self-limiting, that is, the prey in the absence of the predator grows logistically. The model becomes

$$\begin{aligned} N'(t) &= rN\left(1-\frac{N}{K}\right)-\gamma NP, \\ P'(t) &= \varepsilon\gamma NP - dP, \end{aligned} \tag{10.16}$$

where K is the carrying capacity of the prey in the absence of the predator. The dynamical behavior of model (10.16) is very different from that of model (10.15).

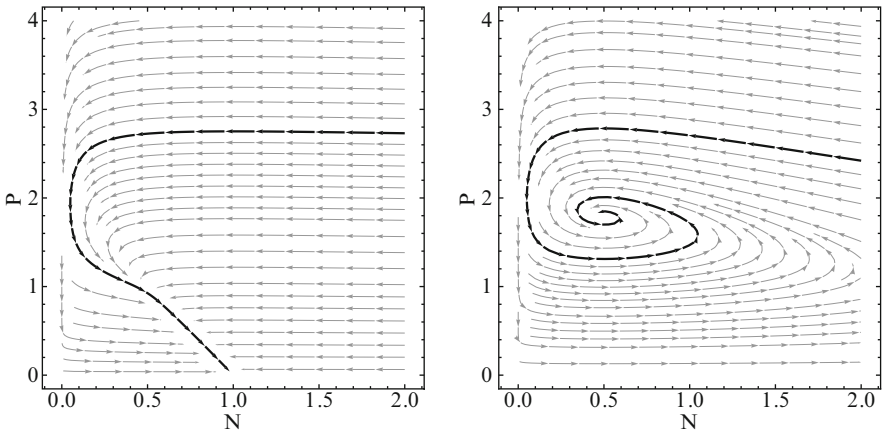

Fig. 10.2 Predator–prey dynamics in the (N,P)-plane. The vector field shows convergence toward the prey-only equilibrium (*left*) or convergence to the predator–prey coexistence equilibrium (*right*)

Model (10.16) has three equilibria. The first equilibrium corresponds to the extinction of both predator and prey, and is called the *extinction equilibrium*, given by $\mathscr{E}_0 = (0,0)$. The Jacobian of the extinction equilibrium, also called the *community matrix*, has one positive and one negative eigenvalue, signifying that the extinction

equilibrium is always a saddle and therefore always unstable. The second equilibrium corresponds to the extinction of the predator only and persistence of the prey population alone. This equilibrium is called the *predator-extinction equilibrium* or semitrivial (boundary) equilibrium, and is given by $\mathscr{E}_1 = (K, 0)$. The community matrix of the prey-only equilibrium is upper triangular and has $\lambda_1 = -r$ and $\lambda_2 = \varepsilon\gamma K - d$. Hence, the prey-only equilibrium is locally asymptotically stable if and only if

$$\frac{\varepsilon\gamma K}{d} < 1. \tag{10.17}$$

The third equilibrium corresponds to a predator–prey coexistence equilibrium. The equilibrium is given by

$$\mathscr{E}^* = \left(\frac{d}{\varepsilon\gamma}, \frac{r}{\gamma}\left(1 - \frac{d}{\varepsilon\gamma K}\right)\right),$$

which exists if and only if $\frac{\varepsilon\gamma K}{d} > 1$. The Jacobian around the coexistence equilibrium has negative trace and positive determinant. Hence, the coexistence equilibrium is locally asymptotically stable. Dulac's criterion can be used to rule out oscillations for this model. It can be shown that if condition (10.17) holds, then the prey-only equilibrium globally stable. If condition (10.17) does not hold, then the predator–prey coexistence equilibrium is globally stable (see Fig. 10.2). Lotka–Volterra predator–prey models have been discussed in multiple texts [90, 27].

10.4.2 Lotka–Volterra Model with SI Disease in Prey

The predator–prey dynamics described by the above models can be impacted by the presence of a disease. The disease may affect the prey, it may affect the predator, or it may affect both the predator and the prey if it is caused by a pathogen that can jump the species barrier. We will consider here a prey population that is subject to predation and impacted by a disease. As a baseline model of the predator–prey dynamics we use the Lotka–Volterra model with self-limiting prey population size and linear functional response. This model exhibits simple dynamics—global convergence to a prey-only equilibrium or to a predator–prey coexistence equilibrium. We will see that the introduction of disease in the prey can lead to much more complex dynamics, namely oscillation and even chaos.

In addition to the assumptions for the predator–prey Lotka–Volterra model with self-limiting prey population size, we also assume the following:

- The disease is transmitted only in the prey and does not affect the predator.
- Infected prey do not recover from the disease—the disease is of SI type for the prey.
- Attack rates of the predator for healthy and infected prey may be different.

- Infected prey does not reproduce but participates in the competition for resources, so it participates in self-limitation.

Assuming that the prey population size N is divided into susceptible S and infective prey I, the Lotka–Volterra model (10.16) with disease in the prey becomes

$$\begin{aligned} S'(t) &= rN\left(1-\frac{N}{K}\right)-\gamma_S SP-\beta SI,\\ I'(t) &= \beta SI-\gamma_I IP-\mu_0 I,\\ P'(t) &= \varepsilon(\gamma_S S+\gamma_I I)P-dP, \end{aligned} \tag{10.18}$$

where γ_S and γ_I are the predation rates of susceptible and infected prey, β is the transmission rate of the disease in the prey, and μ_0 is the natural or disease-induced death rate of infected prey. The natural death rate of susceptible prey is incorporated into the self-limiting logistic term.

Model (10.18) is difficult to analyze, so for analysis, we introduce a slightly simplified model:

$$\begin{aligned} S'(t) &= rS\left(1-\frac{N}{K}\right)-\gamma_S SP-\beta SI,\\ I'(t) &= \beta SI-\gamma_I IP-\mu_0 I,\\ P'(t) &= \varepsilon(\gamma_S S+\gamma_I I)P-dP. \end{aligned} \tag{10.19}$$

Below, we list the equilibria of the system (10.19). The stability of these equilibria depends on the Jacobian (community matrix) evaluated at the corresponding equilibrium. The community matrix at a generic equilibrium is given by

$$J=\begin{pmatrix} r\left(1-\frac{N}{K}\right)-r\frac{S}{K}-\gamma_S P-\beta I & -r\frac{S}{K}-\beta S & -\gamma_S S\\ \beta I & \beta S-\gamma_I P-\mu_0 & -\gamma_I I\\ \varepsilon\gamma_S P & \varepsilon\gamma_I P & \varepsilon(\gamma_S S+\gamma_I I)-d \end{pmatrix}. \tag{10.20}$$

The system (10.19) has four equilibria, three boundary and one interior equilibrium:

1. Extinction or trivial equilibrium: $\mathscr{E}_0=(0,0,0)$. The trivial equilibrium always exists, but it is always unstable, since the community matrix has an eigenvalue $r>0$.
2. Disease-free and predator-free equilibrium: $\mathscr{E}_1=(K,0,0)$. This equilibrium also always exists. If we define a disease reproduction number in the absence of predator

$$\mathscr{R}_0=\frac{\beta K}{\mu_0}$$

and predator invasion number in the absence of disease

$$\mathscr{R}_P^0=\frac{\varepsilon\gamma_S K}{d},$$

then the equilibrium $\mathscr{E}_1$ is locally asymptotically stable if

$$\mathscr{R}_0 < 1 \qquad \text{and} \qquad \mathscr{R}_P^0 < 1$$

and unstable if either inequality is reversed.

3. Predator-free endemic equilibrium in which the disease persists in the prey but the predator dies out, $\mathscr{E}_2 = (S_2, I_2, 0)$, where

$$S_2 = \frac{\mu_0}{\beta} \qquad I_2 = \frac{r\left(1 - \frac{1}{\mathscr{R}_0}\right)}{\frac{r}{K} + \beta}.$$

The equilibrium $\mathscr{E}_2$ exists if and only if $\mathscr{R}_0 > 1$. The equilibrium $\mathscr{E}_2$ is locally asymptotically stable if and only if the invasion number of the predator in the presence of disease is less than one, $\mathscr{R}_p < 1$, where the invasion number of the predator in the presence of the disease is

$$\mathscr{R}_P = \frac{\varepsilon}{d}\left(\gamma_S \frac{\mu_0}{\beta} + \gamma_I \frac{r\left(1 - \frac{1}{\mathscr{R}_0}\right)}{\frac{r}{K} + \beta}\right).$$

4. Predator–prey disease-free equilibrium, where the disease dies out and the predator and the prey coexist disease-free: $\mathscr{E}_3 = (S_3, 0, P_3)$, where

$$S_3 = \frac{d}{\varepsilon \gamma_S} \qquad P_3 = \frac{r}{\gamma_S}\left(1 - \frac{1}{\mathscr{R}_P^0}\right).$$

The disease-free predator–prey coexistence equilibrium $\mathscr{E}_3$ exists if and only if the predator invasion number in the absence of disease satisfies $\mathscr{R}_P^0 > 1$. This equilibrium is locally asymptotically stable if and only if the disease reproduction number in the presence of the predator is less than one: $\mathscr{R}_1 < 1$, where

$$\mathscr{R}_1 = \frac{\beta d}{\varepsilon \gamma_S (\gamma_I P_3 + \mu_0)}.$$

5. Predator–prey–disease coexistence equilibrium: $\mathscr{E}^* = (S^*, I^*, P^*)$. The system for the coexistence equilibrium is given by

$$\begin{aligned} r\left(1 - \frac{N}{K}\right) - \gamma_S P - \beta I &= 0, \\ \beta S - \gamma_I P - \mu_0 &= 0, \\ \varepsilon(\gamma_S S + \gamma_I I) - d &= 0. \end{aligned} \tag{10.21}$$

The system for the interior equilibrium is a linear system, and if it has a nonnegative solution, that solution is unique under the assumption that the determinant

is not zero. The system can be solved, but the expressions obtained do not offer much insight. So we take a different approach. The conditions for existence of a positive equilibrium are stated in the following theorem:

Theorem 10.1. *Assume $\mathscr{R}_0 > 1$ and $\mathscr{R}_P^0 > 1$. Assume also that*

$$\mathscr{R}_p > 1, \qquad \mathscr{R}_1 > 1, \qquad \text{and} \qquad \mathscr{R}_P^0 < \mathscr{R}_0.$$

Then there exists a unique positive interior equilibrium $\mathscr{E}^* = (S^*, I^*, P^*)$.

Proof. To see the claim, notice that from the second and third equations, we can express I and P as functions of S:

$$P(S) = \frac{\beta S - \mu_0}{\gamma_I}, \qquad I(S) = \frac{d - \varepsilon\gamma_S S}{\varepsilon\gamma_I}. \tag{10.22}$$

We use the first equation in system (10.21) to define the following function:

$$f(S) = r\left(1 - \frac{S + I(S)}{K}\right) - \gamma_S P(S) - \beta I(S).$$

Since $\mathscr{R}_P > 1$, we have $I(S_2) \le I_2$ and $P(S_2) = 0$. Hence,

$$f(S_2) = r\left(1 - \frac{S_2 + I(S_2)}{K}\right) - \gamma_S P(S_2) - \beta I(S_2) \ge r\left(1 - \frac{S_2 + I_2}{K}\right) - \beta I_2 = 0. \tag{10.23}$$

Similarly, since $\mathscr{R}_1 > 1$, we have $I(S_3) = 0$, $P(S_3) = \frac{\beta S_3 - \mu_0}{\gamma_I} \ge P_3$. Hence,

$$f(S_3) = r\left(1 - \frac{S_3}{K}\right) - \gamma_S P(S_3) \le r\left(1 - \frac{S_2}{K}\right) - \gamma_S P_3 = 0. \tag{10.24}$$

Therefore, $f(S_2) \ge 0$, $f(S_3) \le 0$, and there must be a unique solution S^* in the interval (S_2, S_3). The condition $\mathscr{R}_P^0 < \mathscr{R}_0$ implies that $S_2 < S_3$ and $S_2 < S^* < S_3$. Since $I(S)$ is a decreasing function of S, we have $I(S^*) > I(S_3) = 0$. At the same time, $P(S)$ is an increasing function of S, so we have $P(S^*) > P(S_2) = 0$. Hence, $I^* = I(S^*) > 0$ and $P^* = P(S^*) > 0$. This completes the proof. □

We note that an interior equilibrium may exist if

$$\mathscr{R}_p < 1, \qquad \mathscr{R}_1 < 1, \qquad \text{and} \qquad \mathscr{R}_P^0 > \mathscr{R}_0.$$

Concerning the stability of the coexistence equilibrium, we consider that the characteristic equation of the Jacobian J is given by $|J - \lambda I| = 0$. We obtain the following cubic polynomial:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where

$$\begin{aligned} a_1 &= rS^*/K, \\ a_2 &= \gamma_S S^* \varepsilon \gamma_S P^* + \varepsilon \gamma_I P^* \gamma_I I^* + \beta I^* (rS^*/K + \beta S^*), \\ a_3 &= \varepsilon P^* \gamma_I I^* r S^*/K(\gamma_I - \gamma_S). \end{aligned} \tag{10.25}$$

The following result is immediate:

Theorem 10.2. *The interior equilibrium is locally stable if $\gamma_I > \gamma_S$. If $\gamma_I < \gamma_S$, the interior equilibrium is unstable.*

We highlight the main conclusion:

> The presence of a disease in the prey and a preferential predation of susceptible individuals can destabilize otherwise stable predator–prey dynamics.

In the case $\gamma_S > \gamma_I$, model (10.18) can exhibit very complex behavior. Since our premise is that the disease destabilizes the otherwise stable predator–prey dynamics, we investigate how the dynamics of the predator–prey-disease system change when the transmission rate is varied while all other parameters are kept fixed. The fixed parameters are listed in Table 10.1.

Table 10.1 Fixed parameter values used in simulations

Parameter	Interpretation	Value
r	Prey growth rate	2
K	Prey carrying capacity	1,000
μ_0	Disease-induced death rate	0.001
ε	Predator conversion efficiency	0.2
γ_I	Attack rate on infectious prey	1.0
γ_S	Attack rate on susceptible prey	9.1
d	Predator death rate	0.2

For $\beta = 6.2$, we observe a closed periodic orbit of period one, which signifies the fact that the periodic orbit makes one loop around the central point (equilibrium) before starting to repeat itself. We illustrate this situation in Fig. 10.3.

As the transmission rate of the disease increases, the dynamics of the system become more and more complex. The system undergoes a process called period-doubling. A period-one periodic solution bifurcates into a period-two periodic solution, that is, a solution that loops twice around the central point. A period-two solution bifurcates into a period-four solution. Such a solution is exhibited in Fig. 10.4. Period-doubling is a common route to chaotic behavior. The period-doubling bifurcations are usually depicted by a bifurcation diagram in which all

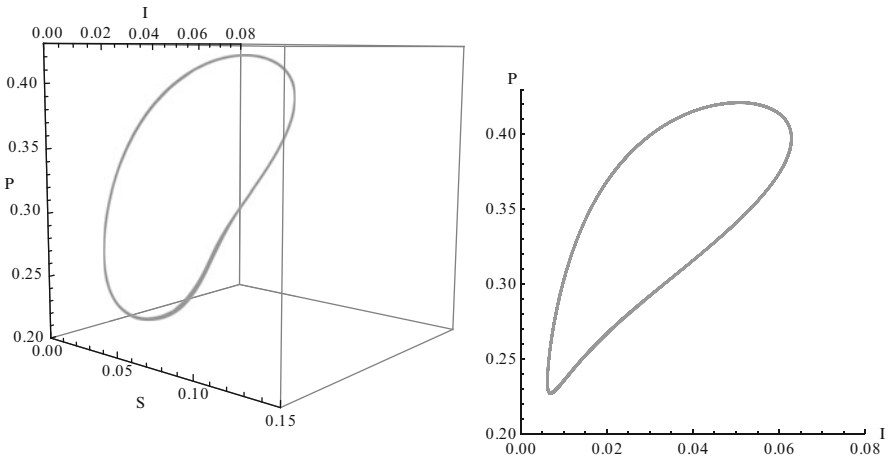

Fig. 10.3 Period-one cycle. The *left figure* shows (S,I,P)-space. The *right figure* shows the (I,P)-plane. Parameters taken from Table 10.1; $\beta = 6.2$

parameters are held fixed except one. Bifurcation diagrams are a common tool for analyzing the behavior of dynamical systems. They are created by running the equations of the system, holding all but one of the variables constant and varying the last one. Then a graph is plotted of the points that a particular value for the changed variable visits after transient factors have been neutralized. Chaotic regions are indicated by filled-in regions of the plot. A bifurcation diagram for the predator–prey–disease dynamical system is shown in Fig. 10.5. For β large enough, the system

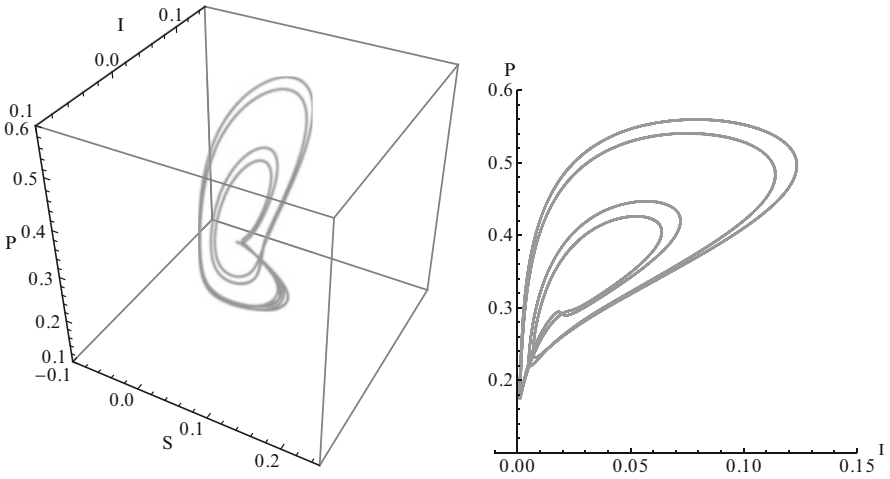

Fig. 10.4 Period-four cycle. The *left figure* shows the (S,I,P)-space. The *right figure* shows the (I,P)-plane. Parameters taken from Table 10.1; $\beta = 6.54$

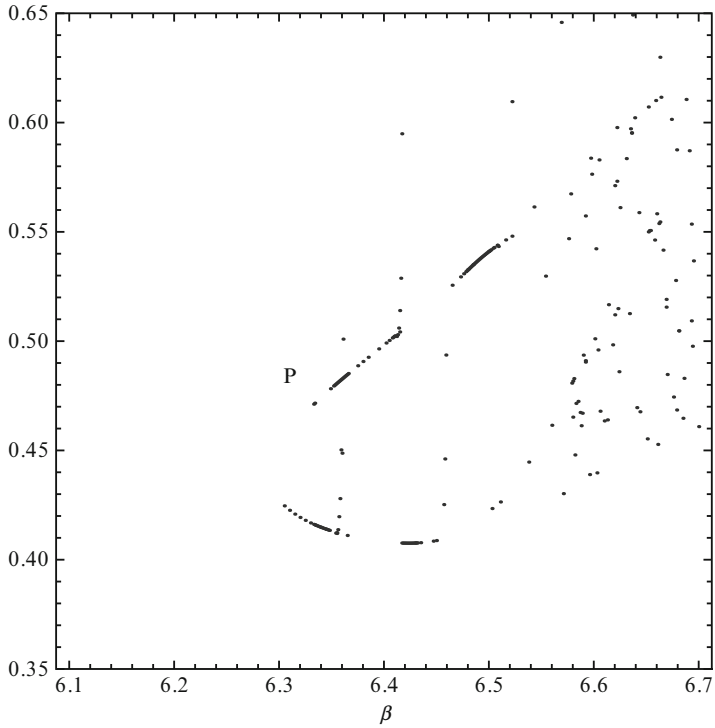

Fig. 10.5 Bifurcation diagram for the system (10.18). Parameter β is plotted on the horizontal axis; P is plotted on the vertical axis. Parameters taken from Table 10.1

exhibits chaos, which is characterized by aperiodic behavior and sensitive dependence on the initial data (Chap. 4). Orbits converge to a chaotic attractor, which is plotted in Fig. 10.6. The attractor has two wings, one of which largely resides in the (S,P)-plane and the other in the (I,P)-plane. Projection of the attractor on the (S,I)-plane is minimal. The dynamics of infected individuals exhibit random spikes modeling outbreak disease, rather than endemicity. The predator persists but also exhibits spikes that coincide with the spikes of the infected prey.

10.5 Competition of Species and Disease

Predation, which we considered in the previous sections, is one of the interactions in the ecological community, and it is certainly the most dynamic interaction. For many years, however, *competition* has been thought to be the main mode of interaction. There is no question that competition is a very important community interaction.

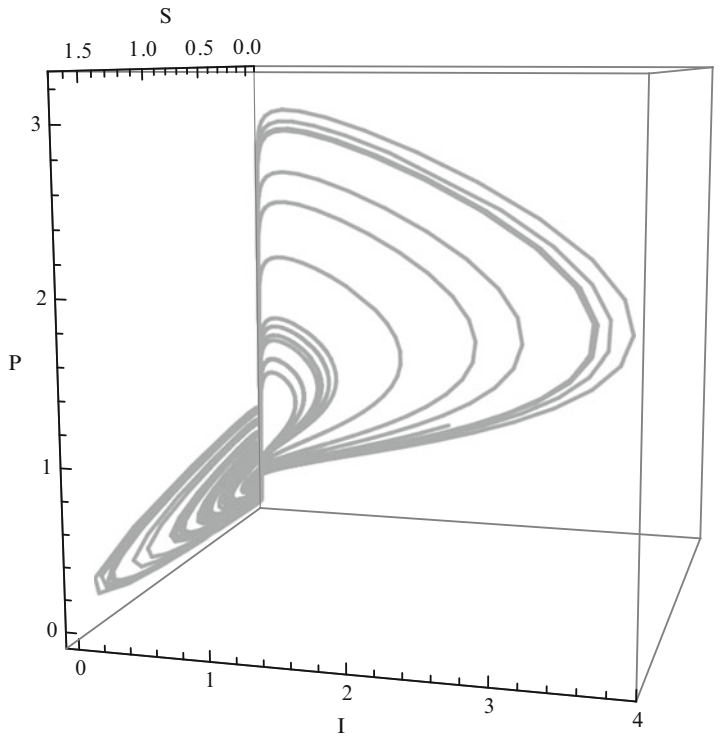

Fig. 10.6 Strange attractor for the system (10.18). Parameter $\beta = 9.5$. Other parameters taken from Table 10.1

10.5.1 Lotka–Volterra Interspecific Competition Models

Lotka and Volterra also developed competition models. Lotka–Volterra competition models describe the competition between two or more species for limited resources. Such competition is called *interspecific competition*, which is contrasted with *intraspecific competition*, which is competition among individuals of one species for limited resources. Lotka–Volterra models are representatives of the **interference competition models**, whereby the increase in the size of one species is assumed to decrease the other species per capita growth rate [90].

We review here the classical Lotka–Volterra competition model considered in [90]. Readers are directed to that source for more detailed discussion of mathematical models of ecology. To introduce the classical Lotka–Volterra model, consider two species with population size $N_1(t)$ and $N_2(t)$ respectively. Each species is assumed to grow logistically in the absence of the other with a growth rate r_i and carrying capacity K_i, $i = 1, 2$:

$$\begin{aligned} N_1'(t) &= r_1 N_1 \left(1 - \frac{N_1 + \alpha_{12} N_2}{K_1}\right), \\ N_2'(t) &= r_2 N_2 \left(1 - \frac{N_2 + \alpha_{21} N_1}{K_2}\right). \end{aligned} \tag{10.26}$$

In these equations, individuals of the second species decrease the per capita growth rate of the individuals of the first species and vice versa. Because the two species are different, the effect of the second species on the first may be stronger or weaker than the effect of the first species on the second. To account for this effect, a pair of competition coefficients α_{12} and α_{21} that describe the strength of the effect of species two on species one and vice versa are introduced.

The system has at most four equilibria. Clearly, it has the extinction equilibrium $\mathscr{E}_0 = (0,0)$ and two semitrivial equilibria corresponding to the dominance of each species: $\mathscr{E}_1 = (K_1,0)$ and $\mathscr{E}_2 = (0,K_2)$. Finally, under appropriate conditions, there is a unique coexistence equilibrium satisfying the system

$$\begin{aligned} N_1 + \alpha_{12} N_2 &= K_1, \\ \alpha_{21} N_1 + N_2 &= K_2. \end{aligned} \tag{10.27}$$

This is a linear system in the unknowns $N_1 \neq 0$ and $N_2 \neq 0$. The solution is given by

$$N_1^* = \frac{\alpha_{12}K_2 - K_1}{\alpha_{12}\alpha_{21} - 1}, \qquad N_2^* = \frac{\alpha_{21}K_1 - K_2}{\alpha_{12}\alpha_{21} - 1}.$$

This solution exists and is positive under appropriate conditions, which we will discuss later. Local stability of equilibria is determined from the community matrix, which in the general case at arbitrary equilibrium is given by the matrix

$$J = \begin{pmatrix} \frac{r_1}{K_1}(K_1 - N_1 - \alpha_{12}N_2) - \frac{r_1}{K_1}N_1 & -\alpha_{12}\frac{r_1}{K_1}N_1 \\ -\alpha_{21}\frac{r_2}{K_2}N_2 & \frac{r_2}{K_2}(K_2 - N_2 - \alpha_{21}N_1) - \frac{r_2}{K_2}N_2 \end{pmatrix}. \tag{10.28}$$

The community matrix for the extinction equilibrium has the eigenvalues $\lambda_1 = r_1$ and $\lambda_2 = r_2$, which are both real and positive. Thus the extinction equilibrium is always an unstable node. The community matrix at the dominance equilibrium of species one is

$$J(K_1,0) = \begin{pmatrix} -r_1 & -\alpha_{12}r_1 \\ 0 & \frac{r_2}{K_2}(K_2 - \alpha_{21}K_1) \end{pmatrix}. \tag{10.29}$$

Thus, one of the eigenvalues is $\lambda_1 = -r_1$, and it is negative. The other eigenvalue is $\lambda_2 = \frac{r_2}{K_2}(K_2 - \alpha_{21}K_1)$. The sign of this eigenvalue depends on the sign of $K_2 - \alpha_{21}K_1$. Thus, the equilibrium $\mathscr{E}_1$ is a stable node or a saddle. Symmetrically, the eigenvalues of the community matrix of the equilibrium $\mathscr{E}_2$ are $\lambda_1 = \frac{r_1}{K_1}(K_1 - \alpha_{12}K_2)$ and $\lambda_2 = -r_2$. The sign of the first eigenvalue depends on the sign of $K_1 - \alpha_{12}K_2$. The equilibrium $\mathscr{E}_2$ is also either a stable node or a saddle. Finally, to simplify the community matrix of the coexistence equilibrium, we notice that at the coexistence equilibrium, we have

$$K_1 - N_1^* - \alpha_{12}N_2^* = 0, \qquad K_2 - N_2^* - \alpha_{21}N_1^* = 0.$$

Thus, the community matrix becomes

$$J(N_1^*, N_2^*) = \begin{pmatrix} -\dfrac{r_1}{K_1}N_1 & -\alpha_{12}\dfrac{r_1}{K_1}N_1 \\ -\alpha_{21}\dfrac{r_2}{K_2}N_2 & -\dfrac{r_2}{K_2}N_2 \end{pmatrix}. \tag{10.30}$$

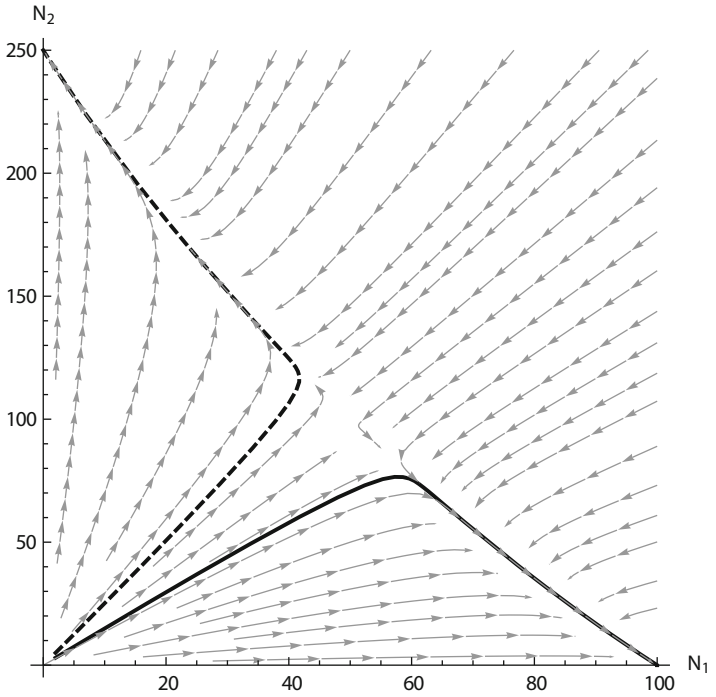

Fig. 10.7 Phase portrait of two competing species. Species one has $K_1 = 100$. Species two has $K_2 = 250$. The *black continuous* trajectory converges to equilibrium $\mathscr{E}_1 = (100, 0)$. The *black dashed* trajectory starts from a different initial condition and converges to equilibrium $\mathscr{E}_2 = (0, 250)$

The trace of the above matrix is clearly negative. The determinant is given as follows:

$$\det J = \frac{r_1}{K_1}\frac{r_2}{K_2}N_1^*N_2^*(1 - \alpha_{12}\alpha_{21}).$$

Thus, the sign of the determinant depends on the sign of the expression $1 - \alpha_{12}\alpha_{21}$. If $1 - \alpha_{12}\alpha_{21} > 0$, then the coexistence equilibrium is a stable node or a stable focus. It can be checked that the discriminant of the characteristic polynomial $\lambda^2 + p\lambda + q = 0$, is given by

$$\Delta = p^2 - 4q = \left(\frac{r_1}{K_1}N_1^* + \frac{r_2}{K_2}N_2^*\right)^2 - 4\frac{r_1}{K_1}\frac{r_2}{K_2}N_1^*N_2^*(1 - \alpha_{12}\alpha_{21}) > 0.$$

Hence, the coexistence equilibrium in this case is a stable node. If $1-\alpha_{12}\alpha_{21}<0$, then the coexistence equilibrium is a saddle. There are four distinct cases that encompass all possibilities. They are centered on the position of the nullclines. Species-one nullclines (x-nullclines) are $N_1=0$ and $N_1+\alpha_{12}N_2=K_1$. Symmetrically, species-two nullclines (y-nullclines) are $N_2=0$ and $\alpha_{21}N_1+N_2=K_2$. There are four cases.

Case 1. $K_1>\alpha_{12}K_2$ and $K_2<\alpha_{21}K_1$. In this case, there is no interior equilibrium, since the numerators of N_1^* and N_2^* have opposite signs. The boundary equilibrium $\mathscr{E}_1$ is a stable node, while the boundary equilibrium $\mathscr{E}_2$ is a saddle (unstable). All orbits tend to $(K_1,0)$ as $t\to\infty$. Thus, species one persists at carrying capacity, while species two becomes extinct.

Case 2. $K_1<\alpha_{12}K_2$ and $K_2>\alpha_{21}K_1$. This is a symmetric case to Case 1. In this case, again there is no interior equilibrium. The boundary equilibrium $\mathscr{E}_1$ is a saddle (unstable), while the boundary equilibrium $\mathscr{E}_2$ is a stable node. All orbits tend to $(0,K_2)$ as $t\to\infty$. Thus, species two persists at carrying capacity, while species one becomes extinct.

Case 3. $K_1<\alpha_{12}K_2$ and $K_2<\alpha_{21}K_1$. These two inequalities imply that $1<\alpha_{12}\alpha_{21}$. Thus the interior equilibrium exists. However, since the determinant of the community matrix evaluated at the interior equilibrium is negative,

$$\mathrm{Det}J(N_1^*,N_2^*)<0,$$

the community matrix has two real eigenvalues of opposite sign ($q<0$). Therefore, the coexistence equilibrium is a saddle. At the same time, both semitrivial equilibria $\mathscr{E}_1$ and $\mathscr{E}_2$ are stable nodes. In this case, the coexistence of the two species is again impossible. One of the species always outcompetes and eliminates the other. However, the winner of the competition is determined by the initial conditions. We recall that this dependence on the initial conditions is called the *founder effect*. This is another example of bistability. Solution orbits that start from the upper part of the plane converge to the equilibrium $\mathscr{E}_2$, while those that start from the lower part converge to the equilibrium $\mathscr{E}_2$ (see Fig. 10.7).

Case 4. $K_1>\alpha_{12}K_2$ and $K_2>\alpha_{21}K_1$. In this case, $1>\alpha_{12}\alpha_{21}$, and the interior equilibrium also exists. The community matrix at the interior equilibrium has negative trace and

$$\mathrm{Det}J(N_1^*,N_2^*)>0.$$

Therefore, the coexistence equilibrium is locally asymptotically stable. It can be further shown that it is a stable node. The community matrices of the two semitrivial equilibria have one positive eigenvalue and one negative eigenvalue. Hence, the two semitrivial equilibria are saddle points. In this case, every orbit that starts from the interior tends to the coexistence equilibrium as $t\to\infty$.

In Cases 1 and 2, we say that *competitive exclusion* occurs. That means that one of the species excludes the other and dominates by itself. We recall that the principle of competitive exclusion was first formulated by Gause in 1934 [64] on the basis of experimental evidence.

10.5.2 Disease in One of the Competing Species

How will the outcome of the competition between two species be influenced if one of the species is plagued by a disease? Models of two competing species with disease have been considered before and they have shown that the presence of the disease tends to destabilize the dynamics of the interaction of the species [160]. We consider model (10.26), and we assume that a disease is spreading among species one. Then the population of species one $N_1(t)$ is split into the number of susceptible individuals $S(t)$ and the number of infected individuals $I(t)$. We have $N_1(t) = S(t) + I(t)$. We assume that the growth rate of species one, r_1, is the growth rate of the susceptible individuals but that infected individuals have a different, possibly lower, growth rate r_I ($r_I < r_1$). The transmission of the disease happens at a rate βSI. Model (10.26) can be modified as follows:

$$\begin{aligned} S'(t) &= S\left(r_1 - \frac{r_1 N_1 + r_1 \alpha_{12} N_2}{K_1}\right) - \beta SI, \\ I'(t) &= I\left(r_I - \frac{r_1 N_1 + r_1 \alpha_{12} N_2}{K_1}\right) + \beta SI, \\ N_2'(t) &= r_2 N_2 \left(1 - \frac{N_2 + \alpha_{21} N_1}{K_2}\right). \end{aligned} \tag{10.31}$$

We notice that in the equation for S, we have multiplied through by r_1. In the equation for I, we first multiply by r_1 and then replace the first occurrence by the reproduction rate r_I. However, the intraspecies interference term remains the same. To simplify the appearance of system (10.31), we rewrite it in the form

$$\begin{aligned} S'(t) &= S(r_1 - a_{11} N_1 - a_{12} N_2) - \beta SI, \\ I'(t) &= I(r_I - a_{11} N_1 - a_{12} N_2) + \beta SI, \\ N_2'(t) &= N_2(r_2 - a_{22} N_2 - a_{21} N_1), \end{aligned} \tag{10.32}$$

where we have set $a_{11} = r_1/K_1$, $a_{12} = r_1 \alpha_{12}/K_1$, $a_{21} = r_2 \alpha_{21}/K_2$, and $a_{22} = r_2/K_2$.

System (10.32) is a three-dimensional competitive Lotka–Volterra system. Notice that if we set $I = 0$, the second equation is trivially satisfied, and system (10.31) reduces to system (10.26). We consider the equilibria of the system (10.32). First, there is an extinction equilibrium of the system $\mathscr{E}_0 = (0,0,0)$. Next, there are three vertex equilibria: $\mathscr{E}_1 = (K_1,0,0)$, $\mathscr{E}_2 = (0, r_I K_1/r_1, 0)$, and $\mathscr{E}_3 = (0,0,K_2)$. Next we investigate equilibria that have one component equal to zero:

- Equilibrium $\mathscr{E}_{12}$ is an equilibrium in which the disease is present in species one but species two is absent:

$$\mathscr{E}_{12} = \frac{1}{\beta^2}((r_1 - r_I)a_{11} - r_I\beta, (r_I - r_1)a_{11} + r_1\beta, 0).$$

  This equilibrium exists when the following inequalities are satisfied:

$$r_I\beta < (r_1 - r_I)a_{11} < r_1\beta.$$

  See Problem 10.4 for further details.
- Equilibrium $\mathscr{E}_{13}$ is an equilibrium in which species one is present with susceptible individuals only and species two is also present:

$$\mathscr{E}_{13} = \frac{1}{\Delta}(r_1a_{22} - r_2a_{12}, 0, r_2a_{11} - r_1a_{21}) = \left(\frac{K_1 - \alpha_{12}K_2}{1 - \alpha_{12}\alpha_{21}}, 0, \frac{K_2 - \alpha_{21}K_1}{1 - \alpha_{12}\alpha_{21}}\right),$$

  where $\Delta = a_{11}a_{22} - a_{12}a_{21}$ is the determinant of the matrix of coefficients. Hence, the existence and stability of equilibrium $\mathscr{E}_{13}$ is exactly the same as the existence and stability of the coexistence equilibrium of the two species.
- Equilibrium $\mathscr{E}_{23}$ is an equilibrium in which species one is present with infected individuals only and species two is also present:

$$\mathscr{E}_{23} = \frac{1}{\Delta}(0, r_Ia_{22} - r_2a_{12}, 0, r_2a_{11} - r_Ia_{21}) = \left(0, \frac{\frac{r_I}{r_1}K_1 - \alpha_{12}K_2}{1 - \alpha_{12}\alpha_{21}}, \frac{K_2 - \frac{r_I}{r_1}\alpha_{21}K_1}{1 - \alpha_{12}\alpha_{21}}\right),$$

  where $\Delta = a_{11}a_{22} - a_{12}a_{21}$ has the same meaning as above. Hence, the existence and stability of equilibrium $\mathscr{E}_{23}$ can be derived in a similar way to that of the existence and stability of the coexistence equilibrium of the two species. See Problem 10.5.

The system has a unique interior equilibrium $\mathscr{E}^* = (S^*, I^*, N_2^*)$, where (see [160])

$$\begin{aligned} S^* &= \frac{1}{a_{22}\beta^2}(\Delta(r_1 - r_I) - \beta(a_{22}r_I - a_{12}r_2)), \\ I^* &= \frac{1}{a_{22}\beta^2}(\beta(a_{22}r_1 - a_{12}r_2) - \Delta(r_1 - r_I)), \\ N_2^* &= \frac{1}{a_{22}\beta}(\beta r_2 - a_{21}(r_1 - r_I)). \end{aligned} \tag{10.33}$$

We notice that $S^* + I^* = (r_1 - r_I)/\beta$. The interior equilibrium is feasible if the reproduction number of the second species satisfies

$$\mathscr{R}_2 = \frac{\beta r_2}{a_{21}(r_1 - r_I)} > 1.$$

In addition, we need the following double inequality to hold:

$$a_{22}r_1 > \Delta\frac{r_1 - r_I}{\beta} + a_{12}r_2 > a_{22}r_I.$$

To investigate the stability of the interior equilibrium, we consider the community matrix at the interior equilibrium:

$$J = \begin{pmatrix} -a_{11}S^* & -a_{11}S^* - \beta S^* & -a_{12}S^* \\ -a_{11}I^* + \beta I^* & -a_{11}I^* & -a_{12}I^* \\ -a_{21}N_2^* & -a_{21}N_2^* & -a_{22}N_2^* \end{pmatrix}. \tag{10.34}$$

Here we have used the equations of the equilibria to simplify the Jacobian. Namely, we have used

$$\begin{aligned} (r_1 - a_{11}N_1 - a_{12}N_2) - \beta I &= 0, \\ (r_I - a_{11}N_1 - a_{12}N_2) + \beta S &= 0, \\ r_2 - a_{22}N_2 - a_{21}N_1 &= 0. \end{aligned} \tag{10.35}$$

Next, we consider the characteristic equation $|J - \lambda I| = 0$. Expanding the determinant, we obtain the following cubic characteristic polynomial:

$$\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0,$$

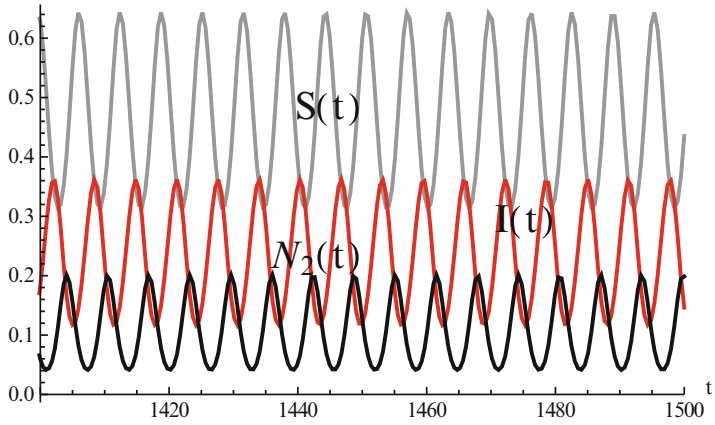

Fig. 10.8 Oscillatory simulation of susceptible species one, $S(t)$, infected species one, $I(t)$, and species two, $N_2(t)$. The susceptible species one have been shifted in the plot with 1.5 units down, that is, what is plotted is $S(t) - 1.5$. Parameters are $r_1 = 25$, $r_I = 14$, $r_2 = 36$, $a_{11} = 10$, $a_{12} = 17$, $a_{21} = 15.3$, $a_{22} = 21.6$, $\beta = 5$ [160]. The oscillatory solution is stable for some initial conditions. Those used to produce the figure are $S(0) = 2$, $I(0) = 0.231667$, $N_2(0) = 0.108333$

where

$$c_1 = a_{22}N_2^* + a_{11}\frac{r_1 - r_I}{\beta},$$
$$c_2 = \Delta N_2^* \frac{r_1 - r_I}{\beta} + \beta^2 S^* I^*,$$
$$c_3 = \beta^2 S^* I^* a_{22} N_2^*. \tag{10.36}$$

Clearly, if $\Delta > 0$, then $c_1 > 0$, $c_2 > 0$, and $c_3 > 0$. Furthermore, it is not hard to see that $c_1 c_2 > c_3$. The Routh–Hurwitz criterion then implies that all roots of the characteristic polynomial have negative real part. Hence the interior equilibrium is locally asymptotically stable. However, if $\Delta < 0$, there is a possibility of Hopf bifurcation and the emergence of a periodic solution. The locally stable periodic solution is illustrated in Fig. 10.8.

Animal populations are subject to a number of ecological interactions. Introducing disease in one or more of the interacting populations is an interesting area of exploration often referred to as **ecoepidemiology**.

Acknowledgements The first part of this chapter is based on lecture notes that Manojit Roy developed and delivered in the Biomathematics Seminar.

Problems

10.1. Competition of Strains under Predation
Consider that a generalist predator is feeding on a prey infected by a pathogen represented by two strains. The model takes the form

$$S'(t) = \Lambda - \beta_1 S I_1 - \beta_2 S I_2 - (\mu_0 + a_S P)S,$$
$$I_1'(t) = \beta_1 S I_1 - (\mu_1 + a_1 P) I_1,$$
$$I_2'(t) = \beta_2 S I_2 - (\mu_2 + a_2 P) I_2. \tag{10.37}$$

(a) Is coexistence of the strains possible in this model? Show competitive exclusion.
(b) Determine which strain dominates depending on the predation level P.

10.2. Competition of Strains under Predation
Consider that a specialist predator is feeding on a prey infected by a pathogen represented by two strains. The model takes the form

$$\begin{aligned} S'(t) &= \Lambda - \beta_1 S I_1 - \beta_2 S I_2 - (\mu_0 + \gamma_S P)S, \\ I_1'(t) &= \beta_1 S I_1 - (\mu_1 + \gamma_1 P) I_1, \\ I_2'(t) &= \beta_2 S I_2 - (\mu_2 + \gamma_2 P) I_2, \\ P'(t) &= \varepsilon(\gamma_S P S + \gamma_1 P I_1 + \gamma_2 P I_2) - dP. \end{aligned} \tag{10.38}$$

(a) Is coexistence of the strains possible in this model? Determine the coexistence equilibrium and the conditions under which it exists.
(b) Use a computer algebra system to simulate the coexistence of the strains. How does changing the predator's predation rates γ_1 and γ_2 affect the competition of the strains?

10.3. Specialist Predator with Disease in Predator
Consider the following model of a specialist predator with disease in the predator. The number of prey is given by $N(t)$. The susceptible predators are given by $S(t)$, and the infected predators by $I(t)$:

$$\begin{aligned} N'(t) &= rN\left(1-\frac{N}{K}\right)-\gamma_S NS-\gamma_I NI,\\ S'(t) &= \varepsilon_S\gamma_S NS-\beta SI-d_S S,\\ I'(t) &= \varepsilon_I\gamma_I NI+\beta SI-d_I I. \end{aligned} \tag{10.39}$$

(a) Find the equilibria of the system.
(b) Compute the reproduction number of the disease in the predator. Determine the stability of the semitrivial equilibria.
(c) Compute the interior equilibrium. When is the interior equilibrium locally asymptotically stable? Does Hopf bifurcation occur?

10.4. Epidemic Model with Vertical Transmission
Consider model (10.32) with species two absent. Assume $r_1 > r_I$:

$$\begin{aligned} S'(t) &= S(r_1-a_{11}N_1)-\beta SI,\\ I'(t) &= I(r_I-a_{11}N_1)+\beta SI. \end{aligned} \tag{10.40}$$

(a) Find the equilibria of model (10.40). Under what conditions does each equilibrium exist?
(b) Determine the local stabilities of each equilibrium.
(c) Use a computer algebra system to draw the phase portrait in each of the cases above.
(d) Explain how the vertical transmission is incorporated in the model.

10.5. Equilibria of Lotka–Volterra Competition Model with Disease
Consider the model (10.32). Assume $r_1 > r_I$.

(a) Find the trivial and semitrivial equilibria of model (10.32). Under what conditions does each equilibrium exist?
(b) Determine the local stabilities of each equilibrium. Determine the corresponding conditions for stability/instability.
(c) Use a computer algebra system to simulate model (10.32). Set $\beta = 0$. Determine parameter values such that $\mathscr{E}_{13}$ is locally stable. Start increasing β. How does the increase in the prevalence of the disease affect the competitive ability of species one?

10.6. Mutualism
Mutualism is an interaction between species that is mutually beneficial for the species involved [90]. Consider the following Lotka–Volterra mutualism model:

$$\begin{aligned} N_1'(t) &= r_1 N_1 \left(1 - \frac{N_1 - \alpha_{12} N_2}{K_1}\right), \\ N_2'(t) &= r_2 N_2 \left(1 - \frac{N_2 - \alpha_{21} N_1}{K_1}\right). \end{aligned} \tag{10.41}$$

Assume $r_1 > 0, r_2 > 0, K_1 > 0, K_2 > 0$. In this case, it is said that the mutualism is facultative.

(a) Find the equilibria of model (10.41). Under what conditions does each an equilibrium exist?
(b) Show that in the coexistence equilibrium, species persist at densities larger than their respective carrying capacities. What does that mean biologically?
(c) Determine the local stabilities of each equilibrium. Determine the corresponding conditions for stability/instability.
(d) Draw a phase portrait in each of the cases $\alpha_{12}\alpha_{21} < 1$ and $\alpha_{12}\alpha_{21} > 1$. Show that in the case $\alpha_{12}\alpha_{21} > 1$, orbits may become unbounded.

10.7. Mutualism with Disease
Consider the mutualism model with disease in one of the species [161]. Let $N_1(t)$ be the density of species one, $S(t)$ the number of susceptible individuals, and $I(t)$ the number of infected individuals of species two:

$$\begin{aligned} N_1'(t) &= N_1(r_1 - a_{11}N_1 + a_{12}(S+I)), \\ S'(t) &= S(r_S + a_{21}N_1 - a_{22}(S+I)) - \beta SI, \\ I'(t) &= I(r_I + a_{21}N_1 - a_{22}(S+I)) + \beta SI, \end{aligned} \tag{10.42}$$

where $r_S > r_I$, and all parameters are positive.

(a) Find the trivial and semitrivial equilibria of model (10.42). Under what conditions does each an equilibrium exist?
(b) Determine the local stabilities of each equilibrium. Determine the corresponding conditions for stability/instability.
(c) Determine the interior equilibrium of the system.
(d) Determine the stability of the interior equilibrium.

10.8. Specialist Predator with a Two-Strain Disease in Predator
Consider the following model of a specialist predator with disease in predator represented by two strains. The number of prey is given by $N(t)$. The susceptible predators are given by $S(t)$, the predators infected by strain one are denoted by $I_1(t)$, and the predators infected by strain two are denoted by $I_2(t)$:

$$
\begin{aligned}
N'(t) &= rN\left(1-\frac{N}{K}\right) - \gamma_S NS - \gamma_{I1} NI_1 - \gamma_{I2} NI_2,\\
S'(t) &= \varepsilon_S \gamma_S NS - \beta_1 SI_1 - \beta_2 SI_2 - d_S S,\\
I_1'(t) &= \varepsilon_{I1} \gamma_{I1} NI_1 + \beta_1 SI_1 - d_{I1} I_1,\\
I_2'(t) &= \varepsilon_{I2} \gamma_{I2} NI_2 + \beta_2 SI_2 - d_{I2} I_2.
\end{aligned} \tag{10.43}
$$

(a) Find the semitrivial equilibria of the system above.
(b) Compute the reproduction numbers of the two strains in the predator. Determine the stability of the semitrivial equilibria.
(c) Is there a coexistence equilibrium for that system?