11
Applications in Biology

In this chapter we make use of the techniques developed in the previous few chapters to examine some nonlinear systems that have been used as mathematical models for a variety of biological systems. In Section 11.1 we utilize the preceding results involving nullclines and linearization to describe several biological models involving the spread of communicable diseases. In Section 11.2 we investigate the simplest types of equations that model a predator/prey ecology. A more sophisticated approach is used in Section 11.3 to study the populations of a pair of competing species. Instead of developing explicit formulas for these differential equations, we instead make only qualitative assumptions about the form of the equations. We then derive geometric information about the behavior of solutions of such systems based on these assumptions.

11.1 Infectious Diseases

The spread of infectious diseases such as measles or malaria may be modeled as a nonlinear system of differential equations. The simplest model of this type is the SIR model. Here we divide a given population into three disjoint groups. The population of susceptible individuals is denoted by S, the infected population by I, and the recovered population by R. As usual, each of these is a function of time. We assume for simplicity that the total population is constant, so that $(S + I + R)' = 0$.

In the most basic case we make the assumption that, once an individual has been infected and subsequently has recovered, that individual cannot be reinfected. This is the situation that occurs for such diseases as measles, mumps, and smallpox, among many others. We also assume that the rate of transmission of the disease is proportional to the number of encounters between susceptible and infected individuals. The easiest way to characterize this assumption mathematically is to put $S' = -\beta SI$ for some constant $\beta > 0$. We finally assume that the rate at which infected individuals recover is proportional to the number of infected. The SIR model is then

$$S' = -\beta SI$$
$$I' = \beta SI - \nu I$$
$$R' = \nu I$$

where β and ν are positive parameters.

As stipulated, we have $(S + I + R)' = 0$, so that $S + I + R$ is a constant. This simplifies the system, for if we determine $S(t)$ and $I(t)$, we then derive $R(t)$ for free. Hence it suffices to consider the two-dimensional system

$$S' = -\beta SI$$
$$I' = \beta SI - \nu I.$$

The equilibria for this system are given by the S-axis ($I = 0$). Linearization at $(S, 0)$ yields the matrix

$$\begin{pmatrix} 0 & -\beta S \\ 0 & \beta S - \nu \end{pmatrix},$$

so the eigenvalues are 0 and $\beta S - \nu$. This second eigenvalue is negative if $0 < S < \nu/\beta$ and positive if $S > \nu/\beta$.

The S-nullclines are given by the S and I axes. On the I-axis, we have $I' = -\nu I$, so solutions simply tend to the origin along this line. The I-nullclines are $I = 0$ and the vertical line $S = \nu/\beta$. Hence we have the nullcline diagram depicted in Figure 11.1. From this it appears that, given any initial population (S_0, I_0) with $S_0 > \nu/\beta$ and $I_0 > 0$, the susceptible population decreases monotonically, while the infected population at first rises, but eventually reaches a maximum and then declines to 0.

We can actually prove this analytically, because we can explicitly compute a function that is constant along solution curves. Note that the slope of the vector field is a function of S alone:

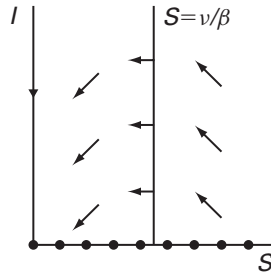$$\frac{I'}{S'} = \frac{\beta SI - \nu I}{-\beta SI} = -1 + \frac{\nu}{\beta S}.$$

Figure 11.1   The nullclines and direction field for the SIR model.

Hence we have

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt} = -1 + \frac{\nu}{\beta S},$$

which we can immediately integrate to find

$$I = I(S) = -S + \frac{\nu}{\beta} \log S + \text{ constant.}$$

Hence the function $I + S - (\nu/\beta) \log S$ is constant along solution curves. It then follows that there is a unique solution curve connecting each equilibrium point in the interval $\nu/\beta < S < \infty$ to one in the interval $0 < S < \nu/\beta$ as shown in Figure 11.2.
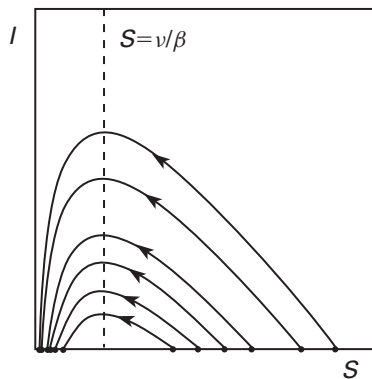

Figure 11.2   The phase portrait for the SIR system.

A slightly more complicated model for infectious diseases arises when we assume that recovered individuals may lose their immunity and become reinfected with the disease. Examples of this type of disease include malaria and tuberculosis. We assume that the return of recovered individuals to the class S occurs at a rate proportional to the population of recovered individuals. This leads to the SIRS model (where the extra S indicates that recovered individuals may reenter the susceptible group). The system becomes

$$S' = -\beta SI + \mu R$$
$$I' = \beta SI - \nu I$$
$$R' = \nu I - \mu R.$$

Again we see that the total population $S + I + R$ is a constant, which we denote by τ. We may eliminate R from this system by setting $R = \tau - S - I$:

$$S' = -\beta SI + \mu(\tau - S - I)$$
$$I' = \beta SI - \nu I.$$

Here β, μ, ν, and τ are all positive parameters.

Unlike the SIR model, we now have at most two equilibria, one at $(\tau, 0)$ and the other at

$$(S^*, I^*) = \left(\frac{\nu}{\beta}, \frac{\mu(\tau - \frac{\nu}{\beta})}{\nu + \mu} \right).$$

The first equilibrium point corresponds to no disease whatsoever in the population. The second equilibrium point only exists when $\tau \geq \nu/\beta$. When $\tau = \nu/\beta$, we have a bifurcation as the two equilibria coalesce at $(\tau, 0)$. The quantity ν/β is called the *threshold level* for the disease.

The linearized system is given by

$$Y' = \begin{pmatrix} -\beta I - \mu & -\beta S - \mu \\ \beta I & \beta S - \nu \end{pmatrix} Y.$$

At the equilibrium point $(\tau, 0)$, the eigenvalues are $-\mu$ and $\beta\tau - \nu$, so this equilibrium point is a saddle provided that the total population exceeds the threshold level. At the second equilibrium point, a straightforward computation shows that the trace of the matrix is negative, while the determinant is positive. It then follows from the results in Chapter 4 that both eigenvalues have negative real parts, and so this equilibrium point is asymptotically stable.

Biologically, this means that the disease may become established in the community only when the total population exceeds the threshold level. We will only consider this case in what follows.

Note that the SIRS system is only of interest in the region given by $I, S \geq 0$ and $S + I \leq \tau$. Denote this triangular region by Δ (of course!). Note that the I-axis is no longer invariant, while on the S-axis, solutions increase up to the equilibrium at $(\tau, 0)$.

Proposition. *The region Δ is positively invariant.*

Proof: We check the direction of the vector field along the boundary of Δ. The field is tangent to the boundary along the lower edge $I = 0$ as well as at $(0, \tau)$. Along $S = 0$ we have $S' = \mu(\tau - I) > 0$, so the vector field points inward for $0 < I < \tau$. Along the hypoteneuse, if $0 < S \leq \nu/\beta$, we have $S' = -\beta S I < 0$ and $I' = I(\beta S - \nu) \leq 0$ so the vector field points inward. When $\nu/\beta < S < \tau$ we have

$$-1 < \frac{I'}{S'} = -1 + \frac{\nu}{\beta S} \leq 0$$

so again the vector field points inward. This completes the proof. ∎

The I-nullclines are given as in the SIR model by $I = 0$ and $S = \nu/\beta$. The S-nullcline is given by the graph of the function

$$I = I(S) = \frac{\mu(\tau - S)}{\beta S + \mu}.$$

A calculus student will compute that $I'(S) < 0$ and $I''(S) > 0$ when $0 \leq S < \tau$. So this nullcline is the graph of a decreasing and concave up function that passes through both $(\tau, 0)$ and $(0, \tau)$, as displayed in Figure 11.3. Note that in this phase portrait, all solutions appear to tend to the equilibrium point (S^*, I^*); the proportion of infected to susceptible individuals tends to a "steady state." To prove this, however, one would need to eliminate the possibility of closed orbits encircling the equilibrium point for a given set of parameters β, μ, ν, and τ.

11.2 Predator/Prey Systems

We next consider a pair of species, one of which consists of predators whose population is denoted by y and the other its prey with population x. We assume that the prey population is the total food supply for the predators. We also