

8. (a) Suppose  $\mathbf{Y}_1(t)$  is a solution of an autonomous system  $d\mathbf{Y}/dt = \mathbf{F}(\mathbf{Y})$ . Show that  $\mathbf{Y}_2(t) = \mathbf{Y}_1(t + t_0)$  is also a solution for any constant  $t_0$ .  
 (b) What is the relationship between the solution curves of  $\mathbf{Y}_1(t)$  and  $\mathbf{Y}_2(t)$ ?
9. Suppose  $\mathbf{Y}_1(t)$  and  $\mathbf{Y}_2(t)$  are solutions of an autonomous system  $d\mathbf{Y}/dt = \mathbf{F}(\mathbf{Y})$ , where  $\mathbf{F}(\mathbf{Y})$  satisfies the hypotheses of the Uniqueness Theorem. Suppose also that  $\mathbf{Y}_2(1) = \mathbf{Y}_1(0)$ . How are  $\mathbf{Y}_1(t)$  and  $\mathbf{Y}_2(t)$  related?
10. Consider the system

$$\begin{aligned}\frac{dx}{dt} &= 2 \\ \frac{dy}{dt} &= y^2.\end{aligned}$$

- (a) Calculate the general solution for the system.  
 (b) What solutions go to infinity?  
 (c) What solutions blow up in finite time?
11. Consider the system

$$\begin{aligned}\frac{dx}{dt} &= x^2 + y \\ \frac{dy}{dt} &= x^2 y^2.\end{aligned}$$

Show that, for the solution  $(x(t), y(t))$  with initial condition  $(x(0), y(0)) = (0, 1)$ , there is a time  $t_*$  such that  $x(t) \rightarrow \infty$  as  $t \rightarrow t_*$ . In other words the solution blows up in finite time. [Hint: Note that  $dy/dt \geq 0$  for all  $x$  and  $y$ .]

## 2.7 THE SIR MODEL OF AN EPIDEMIC

H1N1 flu, often called “swine flu,” caused a worldwide pandemic in 2009. The outbreak began in Mexico early in the year, and eventually the Mexican government closed many public and private facilities in Mexico City in an attempt to restrict the spread of the disease. Nevertheless, the virus spread worldwide. Against the advice of public health officials, some summer camps in the U.S. went so far as to use drugs such as Tamiflu in a prophylactic fashion. More typically, we were encouraged to wash our hands frequently, cough into our sleeves, and stay home during exams.

The pandemic seemed to peak in November of 2009, and by spring of 2010 the number of cases was in rapid decline. The World Health Organization announced the end of the pandemic in August of 2010.\*

### Modeling an Epidemic

The spread of a contagious disease through a population involves intricate interactions from the level of populations down to the level of individual cells and viruses. How-

\*To compare this pandemic to others, see the video “Secrets of the Dead: Killer Flu (1918)” at <http://www.pbs.org/wnet/secrets/episodes/preview-of-killer-flu/222/>

ever, it is still possible to learn interesting and useful information from relatively simple models. A classical model, introduced by Kermack and McKendrick in 1927\* is called the SIR model. In this model, a population is divided into three groups—the susceptible individuals, the infected individuals, and the recovered individuals.

In this model  $S(t)$  denotes the fraction of the population that can catch the disease at time  $t$ ,  $I(t)$  denotes the fraction of the population that has the disease and can spread it to the susceptibles, and  $R(t)$  denotes the fraction of the population that has recovered from the disease and cannot catch it again. This model is appropriate for the spread of a flu epidemic since once a person has had a particular strain of flu, their immune system prevents them from catching that strain again. Since flu spreads fairly quickly, we can assume that time is measured in days. While most people recover from the flu fairly easily, there is a low mortality rate. Those who do not survive are included in  $R(t)$ .

We assume everyone in the population is either susceptible, infected, or recovered, that is,

$$S(t) + I(t) + R(t) = 1$$

for all  $t$ . In addition, we assume that the disease spreads relatively quickly, so it is reasonable to assume that the only change in the size of these groups is due to the disease.

To set up the model, we make some more specific assumptions. First, we assume that the rate that susceptible people and infected people interact is proportional to both the number of susceptibles and the number of infecteds, that is, proportional to the product of  $S(t)$  and  $I(t)$ . Some fraction of these interactions lead to a susceptible becoming infected. We also assume that the infected individuals recover at a rate that is proportional to the number of infecteds.

Based on these assumptions, our model is

$$\begin{aligned}\frac{dS}{dt} &= -\alpha SI \\ \frac{dI}{dt} &= \alpha SI - \beta I \\ \frac{dR}{dt} &= \beta I,\end{aligned}$$

where  $\alpha$  is the “contagion” parameter and  $\beta$  is the “recovery” parameter. If we know  $S(t)$  and  $I(t)$ , then  $R(t) = 1 - (S(t) + I(t))$  (see Exercise 1). Consequently, we need only keep track of  $S(t)$  and  $I(t)$ , and we can consider the planar system

$$\begin{aligned}\frac{dS}{dt} &= -\alpha SI \\ \frac{dI}{dt} &= \alpha SI - \beta I.\end{aligned}$$

The equilibria of this system are the solutions of the simultaneous system of equa-

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\*See “A contribution to the mathematical theory of epidemics” by W. O. Kermack and A. G. McKendrick, *Proceedings Royal Society of London A* 115, 1927, pp. 700–721.

tions

$$\begin{cases} -\alpha SI = 0 \\ (\alpha S - \beta)I = 0, \end{cases}$$

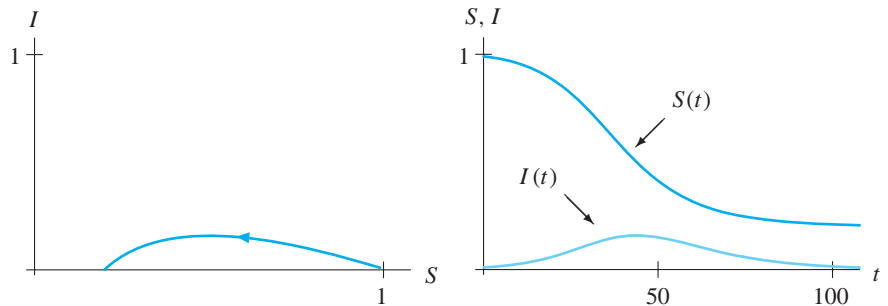
which is precisely the line  $I = 0$ . This makes sense since if there are no infecteds, then no one can catch the disease. The next step is to sketch the direction field, and to do so, we must choose values for the parameters. The recovery parameter  $\beta$  gives the rate at which infecteds recover. If we assume that an infected person is contagious for an average of ten days, then roughly 10% of the infecteds recover each day and  $\beta = 0.1$ .

Choosing  $\alpha$  is more difficult since it contains the proportionality constant that measures the likelihood of interaction within the population as well as the likelihood of the disease spreading during an interaction. We test several different values of  $\alpha$  starting with  $\alpha = 0.2$ . We focus on an initial condition  $(S(0), I(0)) \approx (1, 0)$  with  $I(0) > 0$ . It corresponds to a few infecteds existing in a population that is otherwise entirely susceptible. In fact, we use  $(S(0), I(0)) = (0.99, 0.01)$ , that is, one person in 100 is infected.

Note that the smaller we make  $I(0)$ , the longer it takes the epidemic to manifest itself. Both  $S(t)$  and  $I(t)$  change very slowly near the line of equilibrium points along the  $S$ -axis.

The solution shows very interesting behavior (see Figure 2.58). The number  $I(t)$  of infecteds grows initially. It peaks near  $t = 45$  with  $I(45) \approx 0.15$ . Finally,  $I(t) \rightarrow 0$  as  $t \rightarrow \infty$ . The number  $S(t)$  of susceptibles initially decreases and then almost levels off as  $t \rightarrow \infty$ . However, note that  $S(t)$  does not tend to zero as  $t \rightarrow \infty$ . Rather, it tends toward  $S \approx 0.2$ . In terms of the disease, the model predicts that the percentage of the population that is infected will reach a maximum of approximately 15% after 45 days and then quickly decrease to close to zero after 100 days. The fraction of the population that contracts the disease during the epidemic is approximately 80%. Approximately 20% of the population never gets the disease.

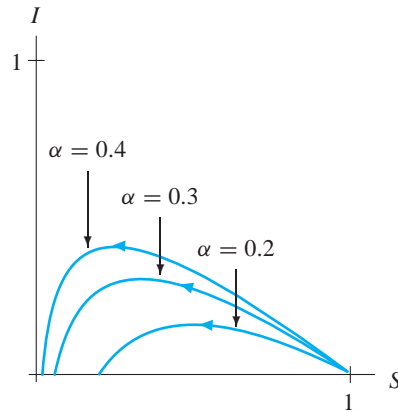
If we try different values of the parameter  $\alpha$ , we see that the predictions made by the model vary quantitatively. As  $\alpha$  increases, the maximum number of infecteds



**Figure 2.58**

The solution curve and  $S(t)$ - and  $I(t)$ -graphs for the initial condition  $(S(0), I(0)) = (0.99, 0.01)$  for  $\alpha = 0.2$  and  $\beta = 0.1$ .

increases while the number of susceptibles that avoid the disease decreases. We can use the model to predict the effect of public health measures that alter the values of the parameters  $\alpha$  and  $\beta$  (see Figure 2.59).



**Figure 2.59**

Solution curves in the  $SI$ -phase plane that correspond to the initial condition  $(S(0), I(0)) = (0.99, 0.01)$  with  $\beta = 0.1$  and  $\alpha = 0.2, 0.3,$  and  $0.4$ . As  $\alpha$  increases, the maximum number of infecteds increases while the number of susceptibles that avoid the disease decreases. If you compare this figure with Figure 2.58, the solution curves corresponding to  $\alpha = 0.2$  look different. This apparent difference is caused by the fact that the distance between 0 and 1 is the same on both axes in this figure while the distance between 0 and 1 is smaller on the vertical axis than on the horizontal axis in Figure 2.58.

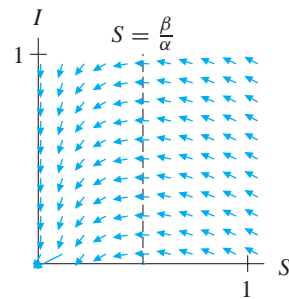
### A little phase plane analysis

If we write the SIR system as

$$\begin{aligned} \frac{dS}{dt} &= -\alpha SI \\ \frac{dI}{dt} &= (\alpha S - \beta)I, \end{aligned}$$

we see that  $dI/dt = 0$  if  $\alpha S - \beta = 0$ . In other words, if  $S = \beta/\alpha$ , the vectors in the vector field are horizontal. To the right of the vertical line in the  $SI$ -phase plane,  $dI/dt > 0$ , and the disease is spreading. To the left of this line,  $dI/dt < 0$ , and the disease is decreasing. Hence, the value  $S = \beta/\alpha$  plays an important role in the evolution of the disease. It is called the **threshold value** of the model. Given values of  $\alpha$  and  $\beta$ , if  $S(0) > \beta/\alpha$ , then an epidemic occurs. If  $S(0) < \beta/\alpha$ , then there is no epidemic (see Figure 2.60).

The line  $S = \beta/\alpha$  along which the vector field is horizontal is one example of what is called a “nullcline.” In Section 5.2, we will study nullclines in great detail.



**Figure 2.60**

The threshold value for  $\alpha = 0.2$  and  $\beta = 0.1$

### An analytic description of the solution curves

Phase plane analysis and numerical solutions for the SIR model give insight into the evolution of a flu epidemic. Because the equations in this system are relatively simple from an algebraic point of view, we can go one step further in describing the solutions curves precisely. The technique we use is one of those ideas that is good to remember. When it works, it gives a great deal of insight into the behavior of solutions.

Because both  $S(t)$  and  $I(t)$  are nonnegative, we see that  $dS/dt < 0$ , and  $S(t)$  decreases monotonically as  $t \rightarrow \infty$ . As a result, we can view the solution curves as

graphs of functions of the variable  $S$ . Moreover, we have

$$\begin{aligned}\frac{dI}{dS} &= \frac{dI/dt}{dS/dt} \\ &= \frac{\alpha SI - \beta I}{-\alpha SI} \\ &= -1 + \left(\frac{\beta}{\alpha}\right) \frac{1}{S}.\end{aligned}$$

This differential equation is one that we can solve by integrating both sides with respect to  $S$ . We get

$$I(S) = -S + \frac{\beta}{\alpha} \ln(S) + c,$$

where  $c$  is a constant of integration.

When an epidemic starts, there are only a few infected individuals, and almost the entire population is susceptible. That is,  $S \approx 1$ , and we have

$$0 \approx I(1) = -1 + \frac{\beta}{\alpha} \ln(1) + c = -1 + c.$$

In this case, it makes sense to take  $c = 1$ . We obtain the function

$$I(S) = -S + \frac{\beta}{\alpha} \ln(S) + 1.$$

The graph of this function  $I(S)$  for  $\alpha = 0.2$  and  $\beta = 0.1$  is almost identical to the solution curve that is shown in Figure 2.58.

For any values of the parameters  $\alpha$  and  $\beta$ , we can explicitly compute the maximum value of  $I(S)$ , that is, the maximum fraction of the population that is ill during the epidemic by doing a maximization problem (see Exercise 4). We can also compute the fraction of the population that completely avoids getting the disease by computing the value of  $S$ ,  $0 < S < 1$ , such that  $I(S) = 0$ . Unfortunately, we cannot solve this equation algebraically for  $S$ , but we can understand the behavior of its roots by graphing the function  $I(S)$  for various values of  $\alpha$  and  $\beta$  (see Exercise 5).

Perhaps the most interesting consequence of the computation of the function  $I(S)$  is the fact that  $I(S)$  is determined by the ratio of  $\beta$  to  $\alpha$ . In other words, if two choices of  $\alpha$  and  $\beta$  have the same ratio  $\beta/\alpha$ , then the maximum number of infecteds and the number who escape infection altogether are the same.

## Concluding Remark

A dose of reality is in order. We have made a number of simplifying assumptions while setting up the SIR model. The situations where this model gives precise, quantitative predictions are limited to closed environments with simple social dynamics and limited geographical separation (see Exercises 9 and 10). For an epidemic spreading around the world, we must include geographical effects as well as the differences in rates of contact among and within different groups of people. In these cases, the SIR model is the starting point for more involved models.

## EXERCISES FOR SECTION 2.7

- For the SIR-model, show that  $S(t) + I(t) + R(t) = 1$  for all  $t$  directly from the system of differential equations.
- In the SIR model, we assume that everyone in the population is susceptible at time  $t = 0$  except the very small fraction that is already infected. Suppose that some fraction of the population has received a vaccine, so they cannot catch the disease. The vaccine makes the fraction of the population that is susceptible at time  $t = 0$  smaller.
  - Using `HPGSystemSolver` applied to the SIR model with  $\alpha = 0.25$  and  $\beta = 0.1$ , describe the behavior of the solutions with  $I(0) = 0.01$  and  $S(0) = 0.9, 0.8, 0.7, \dots$ . Pay particular attention to the maximum of  $I(t)$ , that is, the maximum number of infecteds for each choice of  $S(0)$ . Also, note the limit of  $S(t)$  as  $t \rightarrow \infty$ . (This limit is the fraction of the population that does not catch the disease during the epidemic.)
  - If  $\alpha = 0.25$  and  $\beta = 0.1$ , how large a fraction of the population must be vaccinated in order to keep the epidemic from getting started with  $I(0) = 0.01$ ?
- Vaccines make it possible to prevent epidemics. However, the time it takes to develop a vaccine may make it impossible to vaccinate everyone in a population before a disease arrives.
  - For the SIR model, which initial conditions guarantee that  $dI/dt < 0$ ? [*Hint:* Your answer should be expressed in terms of the parameters  $\alpha$  and  $\beta$ .]
  - For given values of  $\alpha$  and  $\beta$ , what fraction of a population must be vaccinated before a disease arrives in order to prevent an epidemic?
- In this section we showed that solution curves of the SIR model with  $S(0) \approx 1$  and  $I(0) \approx 0$  are graphs of the function

$$I(S) = -S + \frac{\beta}{\alpha} \ln(S) + 1.$$

(Note that the graph depends only on the ratio  $\rho = \beta/\alpha$  of the parameters. Different values of the parameters can give the same value of  $\rho$ .)

- Determine the maximum value of  $I(S)$  in terms of  $\rho$ .
  - Is the statement “The epidemic cannot get started if  $\beta > \alpha$ ” true or false? Justify your answer.
- Let  $\rho$  denote the ratio  $\beta/\alpha$  of the parameters  $\alpha$  and  $\beta$  in the SIR model. Then
 
$$I(S) = -S + \rho \ln(S) + 1.$$
    - Using graphing technology, graph  $I(S)$  over the interval  $0 < S \leq 1$  for various values of  $\rho$  between 0.1 and 1.0.
    - Using the graphs that you produced in part (a), graph the solution of  $I(S) = 0$  for  $0 < S < 1$  as a function of  $\rho$ .

(c) What does the graph that you produced in part (b) tell you about the long-term predictions of the SIR model in terms of the ratio  $\rho$ ?

6. One of the basic assumptions of the SIR model is that individuals who recover from the disease never get it again. However, diseases continually evolve, and new strains can emerge that can infect those who have recovered from the previous strain. In this exercise, we modify the SIR model so that recovereds become susceptible again in a linear rate. We obtain the system of equations

$$\begin{aligned}\frac{dS}{dt} &= -\alpha SI + \gamma R \\ \frac{dI}{dt} &= \alpha SI - \beta I \\ \frac{dR}{dt} &= \beta I - \gamma R\end{aligned}$$

- (a) Show that the sum  $S(t) + I(t) + R(t)$  is constant as a function of  $t$  for this model.
- (b) Derive a system in the two dependent variables  $S$  and  $I$  using the fact that  $R = 1 - (S + I)$ .
- (c) What are the equilibrium points for this model of the two variables  $S$  and  $I$ ? (*Hint*: Both  $S$  and  $I$  are nonnegative, and  $S(t) + I(t) \leq 1$  for all  $t$ .)
- (d) Fix  $\alpha = 0.3$ ,  $\beta = 0.15$ , and  $\gamma = 0.05$  and use `HPGSystemSolver` to sketch the phase portrait. Describe the behavior of solutions.
- (e) How does the system change if we fix  $\alpha = 0.3$  and  $\beta = 0.15$ , but vary  $\gamma$  over a small interval surrounding  $\gamma = 0.05$ ?
7. In the movie *I Am Legend*, the infecteds work together to increase the number of infecteds. We can modify the SIR model to include the assumption that zombies actively infect susceptibles by replacing  $I$  by  $\sqrt{I}$  in the interaction term. (Note that  $0 \leq I \leq 1$ , so  $\sqrt{I} \geq I$ .) We obtain the system

$$\begin{aligned}\frac{dS}{dt} &= -\alpha S\sqrt{I} \\ \frac{dI}{dt} &= \alpha S\sqrt{I} - \beta I.\end{aligned}$$

- (a) Calculate the equilibrium points of this model.
- (b) Find the region of the phase plane where  $dI/dt > 0$ .
- (c) Use  $\alpha = 0.2$  and  $\beta = 0.1$  and sketch the phase portrait. What does the model predict for the spread of the zombies in this case?
8. Many zombie movies are based on the premise that zombies do not stop infecting new victims until they are destroyed by a susceptible. In addition, the susceptibles destroy as many zombies as they can. We can model the spread of zombies in such a movie by assuming that infecteds (zombies) become recovered (zombies who can

not infect susceptibles) at a rate proportional to the size of the remaining susceptible population. We obtain the system

$$\begin{aligned}\frac{dS}{dt} &= -\alpha SI \\ \frac{dI}{dt} &= \alpha SI - \gamma S.\end{aligned}$$

- (a) Calculate the equilibrium points of this model.
- (b) Find the region of the phase plane where  $dI/dt > 0$ .
- (c) Use  $\alpha = 0.2$  and  $\gamma = 0.1$  and sketch the phase portrait. What does the model predict for the spread of the zombies in this case?

The SIR model is particularly relevant to a homogenous population in an environment with little geographic distribution. A famous example of exactly this situation occurred in 1978 at a British boarding school.\* A single boy in the school of 763 students contracted the flu and the epidemic spread rapidly, as shown in Table 2.3. (We are assuming that the number of students confined to bed was the same as the number of infected students.)

**Table 2.3**

The daily count of the number of infected students.

$t$	Infected	$t$	Infected	$t$	Infected
0	1	5	222	10	123
1	3	6	282	11	70
2	7	7	256	12	25
3	25	8	233	13	11
4	72	9	189	14	4

9. Assume that the parameter  $\alpha = 1.66$  in the SIR model for the data in Table 2.3.
  - (a) Using whatever technology that is most convenient, determine an appropriate value of  $\beta$  that matches the data in Table 2.3.
  - (b) Using the value of  $\beta$  that you computed in part (a), calculate the total number of students who caught the flu during the epidemic.
  - (c) Interpret the value of  $\beta$  that you computed in part (a) in terms of the length of time that students with the flu remained infected.
10. Using  $\alpha = 1.66$  and the value of  $\beta$  that you determined in Exercise 9, how would the progress of the epidemic have changed if 200 students had been vaccinated before the disease started? (Give as precise an answer as possible.)

\*Anonymous, "Epidemiology: Influenza in a boarding school," *British Medical Journal*, Vol. 4, 1978, p. 587.