

Differential Equations: Techniques, Theory, and Applications

Barbara D. MacCluer | Paul S. Bourdon | Thomas L. Kriete



AMERICAN
MATHEMATICAL
SOCIETY

We will see that an unanticipated consequence of habitat destruction is the different ways in which the dominant competitor (species 1) and the weaker competitor (species 2) are affected by the loss of habitat.

(a) Find the equilibrium point P_D obtained by solving the pair of equations

$$a_1 - a_1 p_1 - a_1 D - m = 0 \quad \text{and} \quad a_2 - a_2 p_1 - a_2 p_2 - a_2 D - a_1 p_1 - m = 0.$$

(When $D = 0$ your answer should agree with equation (10.50).)

- (b) We know from Example 10.4.1 that when $D = 0$, under certain conditions on the parameters a_1, a_2 , and m , this equilibrium point will lie in the open first quadrant $p_1 > 0, p_2 > 0$. Assuming these conditions hold, how large can D be so that the equilibrium point P_D from

(a) has both coordinates positive? You may find it helpful to write the coordinates of P_D in the form

$$\text{first coordinate} = \frac{a_1 - m}{a_1} + \text{an expression in terms of } D$$

and

$$\text{second coordinate} = \frac{ma_2 - a_1^2}{a_1 a_2} + \text{an expression in terms of } D$$

so that the relationship between the coordinates of P_D and the equilibrium point in equation (10.50) (corresponding to $D = 0$) is clear.

- (c) True or false: The dominant competitor is predicted to become extinct at a lower level of habitat destruction than the weaker competitor.
 (d) Does the phrase “the enemy of my enemy is my friend” have any relevance to this model?

10.5. Modeling the spread of disease

Communicable diseases have had profound impacts on the course of history. The Antonine plague (probably smallpox or measles or both) in AD 165–180 contributed to the demise of the Roman Empire. Bubonic plague caused the death of up to one third of the population of Europe in the fourteenth century. It reappeared again in London in 1665, killing one fifth of the population there. It forced the closure of Cambridge University for a while, where coincidentally Issac Newton was a student. Returning home, Newton had a spectacularly prolific period of scientific work during the university’s closure. The 1918–1919 pandemic influenza affected one third of the world’s population and killed more (up to 50 million) than died in World War I (16 million); healthy young adults were particularly hard hit. HIV, SARS, swine or bird flu, the possibility of weaponized anthrax, or a terrorist release of the smallpox virus are just a few of the current concerns of public health officials.

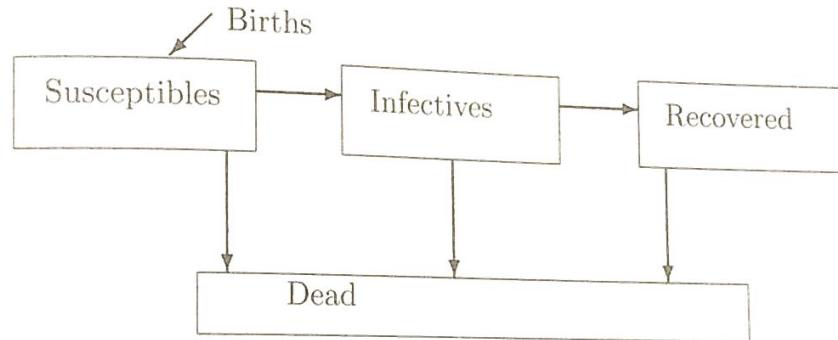
The use of mathematical models to analyze the spread of diseases goes back centuries. In 1760, the mathematician Daniel Bernoulli used a mathematical argument to show that cowpox vaccination against smallpox would significantly increase the average life span—his model showed that if deaths from smallpox could be eliminated, the average life span would increase by about 3 years (from the then-current value of 26.5 years). His work (see Exercise 4) represents the first important use of a mathematical model to address a practical vaccination proposal. In the early twentieth century, differential equation compartment models for disease propagation started to appear and win acceptance. As with other types of mathematical models, there is a trade-off between simple models which incorporate only a few broad details and more refined models whose solutions may be more difficult to obtain and to analyze. In this section we will look at several compartment models and see how even simple models give interesting predictions which can be tested against observed phenomena.

Example 10.5.1. Diseases with permanent immunity.

We'll model the spread of a disease—like measles—which confers immunity. This means, once infected and recovered, a person can never get the disease again. We will also modify this basic model, called an SIR model, to include the possibility of vaccination against the disease, and look at the idea of “herd immunity”.

Setting up the SIR model. Imagine our population divided into three nonoverlapping compartments: the susceptibles, with population $S(t)$ at time t ; the infectives, with population $I(t)$; and the recovered, with population $R(t)$, consisting of those people who have had the disease and recovered (and are furthermore immune from it). There is also a fourth compartment, consisting of people who have died (either from the disease or from other causes). The diagram below shows how people move from one compartment to another. Notice the model allows for births, and all newborns begin life in the susceptible compartment. We will set up our model so that total size of the population is a fixed value K . This means we assume that deaths balance out births. Here are the assumptions we make to describe the movement between the compartments:

- Infectives recover from the disease at a rate proportional to the number of infectives.
- Susceptibles become infective at a rate jointly proportional to the number of infectives and the number of susceptibles.
- The birth rate is constant and proportional to the total size K of the population.
- The total death rate is equal to the birth rate, and the death rate from each compartment is proportional to the size of the compartment, with the same proportionality constant μ for each of the three compartments, S , I , and R .



These assumptions give us the following equations:

$$(10.53) \quad \frac{dS}{dt} = \mu K - \beta SI - \mu S, \quad \frac{dI}{dt} = \beta SI - \mu I - \gamma I, \quad \frac{dR}{dt} = \gamma I - \mu R,$$

where μ , β , and γ are positive constants. Check that $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$, reflecting the assumption that $S + I + R = K$ for all times. Since the equations for $\frac{dS}{dt}$ and $\frac{dI}{dt}$ involve only S and I , we can focus on those so as to have a planar system to analyze.

Nullcline-and-arrow diagrams. From the equation for $\frac{dI}{dt}$ in (10.53) we see that the I -nullclines are the lines with equations $I = 0$ and $S = (\gamma + \mu)/\beta$, shown in Fig. 10.44, along with the relevant up/down arrows. Using the equation for $\frac{dS}{dt}$ in (10.53) we see that the S -nullcline has equation

$$I = \frac{\mu K}{\beta} \frac{1}{S} - \frac{\mu}{\beta},$$

which is shown in Fig. 10.45 along with the relevant right/left arrows. How Figs. 10.45 and 10.44 fit together depend on whether $K \leq (\gamma + \mu)/\beta$ or $K > (\gamma + \mu)/\beta$.

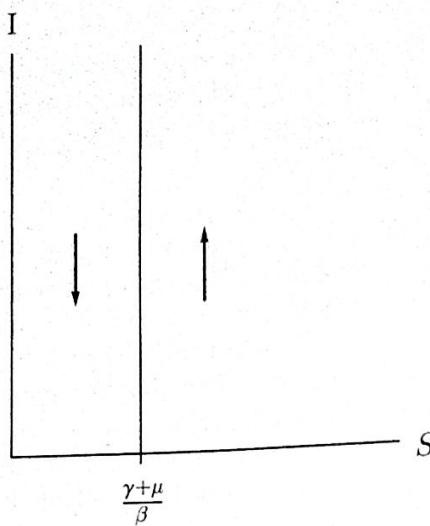


Figure 10.44. The I -nullclines $I = 0$ and $S = \frac{\gamma + \mu}{\beta}$.

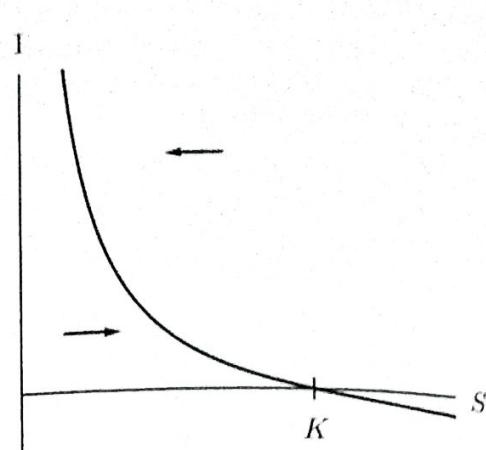


Figure 10.45. The S -nullcline $I = \frac{\mu K}{\beta} \frac{1}{S} - \frac{\mu}{\beta}$.

For the rest of this example, we make the following assumption:

$$(10.54) \quad K > \frac{\gamma + \mu}{\beta}.$$

With this assumption, the S - and I -nullclines intersect in the first quadrant as shown in Fig. 10.46.

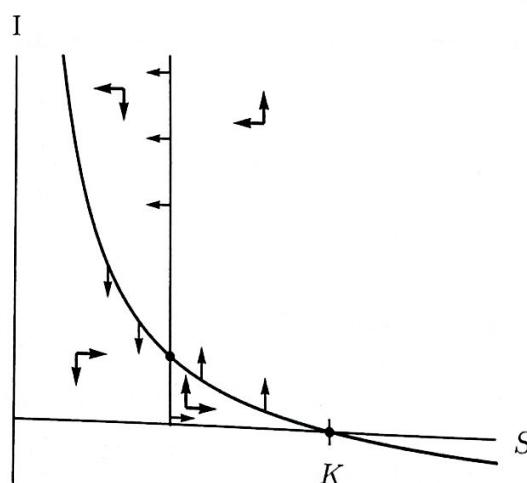


Figure 10.46. Nullcline-and-arrow diagram for first two equations in (10.53) if $\beta K > \gamma + \mu$.

Equilibrium points. Still assuming $\beta K > \gamma + \mu$, we see that there are two biologically relevant equilibrium points, obtained as the intersection of the S -nullcline with an I -nullcline. One is the “disease-free” equilibrium point $(K, 0)$. This should make perfect sense: If there are no infectives and $S = K$, then S should remain K , the total population size, for all times. The second equilibrium point is

$$(10.55) \quad \left(\frac{\gamma + \mu}{\beta}, \frac{\mu}{\beta(\gamma + \mu)}(\beta K - \gamma - \mu) \right).$$

To analyze these equilibrium points we compute the Jacobian matrix for the first two equations in (10.53), obtaining

$$\mathbf{J} = \begin{pmatrix} -\beta I - \mu & -\beta S \\ -\beta I & \beta S - \gamma - \mu \end{pmatrix}.$$

At the equilibrium point $(K, 0)$ this is

$$\begin{pmatrix} -\mu & -\beta K \\ 0 & \beta K - \gamma - \mu \end{pmatrix}.$$

(reported in the last page of the present document)

This matrix has eigenvalues $-\mu$ and $\beta K - \gamma - \mu$. Under assumption equation (10.54), one eigenvalue is positive and one is negative. By Theorem 10.2.3, we have an (unstable) saddle point at $(K, 0)$.

Computing the Jacobian at the second equilibrium point in equation (10.55) looks tedious, but we can extract what we need to know by observing that the 2-2 entry of the Jacobian at this point is 0: $\beta S - \gamma - \mu = 0$ when $S = (\gamma + \mu)/\beta$. From this it is immediate that the trace of the Jacobian is negative and the determinant, which is $\beta^2 SI$, is positive. This tells us that the second equilibrium point is stable (either a node or spiral).

Because we know that the equilibrium point (10.55) is stable, we can predict that the disease will become *endemic* in the population, with $I(t)$ tending to a constant, positive value as $t \rightarrow \infty$. An endemic disease is one that is consistently present in the population. Exercise 1 asks you to predict the long-term behavior if (10.54) does not hold.

The condition of (10.54) can be written as $\beta K / (\gamma + \mu) > 1$, and the qualitative long-term behavior depends on whether the quantity

$$(10.56) \quad R_0 = \frac{\beta K}{\gamma + \mu}$$

is greater than or less than 1. This is an example of a **threshold** quantity; R_0 is sometimes called the **basic reproduction number**.

The effect of vaccination. How can vaccination change the course of the disease? Assume that a fixed fraction ρ of all newborns is vaccinated against the disease, so they are born into the recovered compartment. Since the birth rate is μ , this adds a “rate in” term of $\rho\mu K$ to the equation for $\frac{dR}{dt}$ and modifies the “rate in” term for $\frac{dS}{dt}$ to be $(1 - \rho)\mu K$. Our equations are now

$$(10.57) \quad \frac{dS}{dt} = (1 - \rho)\mu K - \beta SI - \mu S, \quad \frac{dI}{dt} = \beta SI - \mu I - \gamma I, \quad \frac{dR}{dt} = \gamma I - \mu R + \rho\mu K.$$

Again we can focus on just the first two equations in (10.57). Our previous analysis still applies, except where we had “ K ” before, we now have $(1 - \rho)K$. What determines whether we have one equilibrium point or two (in the first quadrant $I \geq 0, S \geq 0$) is whether $\beta(1 - \rho)K > \gamma + \mu$ or not; this is (10.54) with K replaced by $(1 - \rho)K$.

What fraction of newborns needs to be vaccinated so that instead of having a stable endemic equilibrium value, we have only a (stable) disease-free equilibrium? By our analysis and Exercise 1 we know this will happen if $\beta(1 - \rho)K \leq \gamma + \mu$, or equivalently, if the new basic reproduction number

$$\widetilde{R}_0 = \frac{\beta(1 - \rho)K}{\gamma + \mu}$$

is less than 1. (The case of equality is ignored, as exact equality would not be expected to occur in practice.) Some algebra shows $\widetilde{R}_0 < 1$ precisely if

$$(10.58) \quad \rho > 1 - \frac{\gamma + \mu}{\beta K}, \quad \text{or equivalently, if } \rho > 1 - \frac{1}{\widetilde{R}_0}$$

where R_0 is the original basic reproduction number in (10.56). For different diseases, the value of R_0 can be estimated from epidemiological data. For example, measles has a high value, estimated at about 12–18 in urban areas. By equation (10.58) this translates into a need of vaccinating about 91.6–94.4 percent of the newborn population to ensure that measles cannot become endemic. Exercise 2 asks you to verify this and to similarly determine what fraction of newborns needs to be vaccinated to control several other diseases with different basic reproduction numbers. If enough of the population has been vaccinated to prevent the disease from becoming endemic, the population is said to have “herd immunity”.

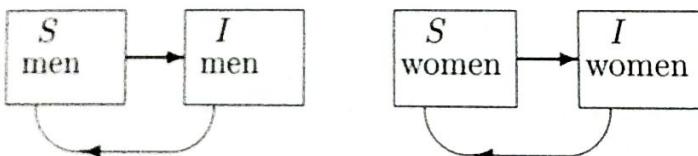
Example 10.5.2. Modeling the spread of a sexually transmitted disease. We start with a fixed population of N at-risk (sexually active) people and assume that men can only be infected by women and women can only be infected by men. Let M be the total number of men, and W the total number of women, so that $M + W = N$; M, W , and N are constants. As is typical of many bacterial STDs, we assume that an infection does not confer immunity, so after a person is infected and recovers, he or she is immediately susceptible to reinfection. At any point in time there will be $x(t)$ infected men and $M - x(t)$ susceptible men, and $y(t)$ infected women and $W - y(t)$ susceptible women. Our model will give differential equations for $\frac{dx}{dt}$ and $\frac{dy}{dt}$. To derive the form of these equations we make several basic assumptions:

- Men are infected at a rate jointly proportional to the number of susceptible men and the number of infected women.
- Women are infected at a rate jointly proportional to the number of susceptible women and the number of infected men.
- Infected men recover at a rate proportional to the number $x(t)$ of infected men.
- Infected women recover at a rate proportional to the number $y(t)$ of infected women.

These assumptions lead to the equations

$$(10.59) \quad \frac{dx}{dt} = a_1(M - x)y - b_1x, \quad \frac{dy}{dt} = a_2(W - y)x - b_2y$$

for some positive constants a_1, a_2, b_1 , and b_2 . You should be able to identify which terms in these equations correspond to which “in” and “out” arrows in the schematic diagram below.



For a number of important STDs, like gonorrhea and chlamydia, women typically show no or few symptoms and thus do not seek (antibiotic) treatment. This fact is reflected in the value of b_2 being relatively small. If men do typically show symptoms and seek treatment (for example, this is the case with gonorrhea), then we would expect b_1 to be significantly larger than b_2 . The reciprocals $1/b_1$ and $1/b_2$ can be interpreted as the average time a man or woman remains infective.

Equilibrium points. The system (10.59) has an equilibrium point at $(0, 0)$. There is a possibility of a second biologically meaningful equilibrium point. Solving the equations

$$a_1(M - x)y - b_1x = 0 \quad \text{and} \quad a_2(W - y)x - b_2y = 0,$$

we get a solution point

$$(10.60) \quad \left(\frac{MWa_1a_2 - b_1b_2}{b_1a_2 + Wa_1a_2}, \frac{MWa_1a_2 - b_1b_2}{b_2a_1 + Ma_1a_2} \right).$$

This will be of interest if it lies in the first quadrant of the xy -plane; this happens if

$$MWa_1a_2 - b_1b_2 > 0.$$

Before going further, let's rewrite the coordinates of the point in (10.60) by dividing each numerator and denominator by a_1a_2 to obtain

$$\frac{MWa_1a_2 - b_1b_2}{b_1a_2 + Wa_1a_2} = \frac{MW - \rho_1\rho_2}{W + \rho_1}, \quad \frac{MWa_1a_2 - b_1b_2}{b_2a_1 + Ma_1a_2} = \frac{MW - \rho_1\rho_2}{M + \rho_2}$$

where $\rho_1 = b_1/a_1$ and $\rho_2 = b_2/a_2$. The equilibrium point

$$\left(\frac{MW - \rho_1\rho_2}{W + \rho_1}, \frac{MW - \rho_1\rho_2}{M + \rho_2} \right)$$

lies in the first quadrant if

$$(10.62) \quad MW - \rho_1\rho_2 > 0.$$

From this point on we assume that (10.62), or equivalently (10.61), holds. The Jacobian matrix for the linear approximation of (10.59) near $(0, 0)$ is

$$\mathbf{J}_1 = \mathbf{J}(0, 0) = \begin{pmatrix} -b_1 & Ma_1 \\ Wa_2 & -b_2 \end{pmatrix}.$$

Under our assumption (10.61), this is a matrix with negative determinant, so according to Section 8.9 and Theorem 10.2.3, $(0, 0)$ is a saddle point for the system (10.59).

The Jacobian matrix at the second equilibrium point is

$$\mathbf{J}_2 = \begin{pmatrix} -a_1y_0 - b_1 & a_1M - a_1x_0 \\ a_2W - a_2y_0 & -a_2x_0 - b_2 \end{pmatrix},$$

where $x_0 = (MW - \rho_1\rho_2)/(W + \rho_1)$ and $y_0 = (MW - \rho_1\rho_2)(M + \rho_2)$ are the coordinates of the nonzero equilibrium point. This is a little unpleasant to deal with, but we can make two observations with minimal calculation:

- The trace of \mathbf{J}_2 is negative, since both entries on the main diagonal are negative.
- The determinant of \mathbf{J}_2 is positive. To see this, compute

$$a_1M - a_1x_0 = a_1 \left(M - \frac{MW - \rho_1\rho_2}{W + \rho_1} \right) = a_1\rho_1 \frac{M + \rho_2}{W + \rho_1}$$

and

$$a_2W - a_2y_0 = a_2 \left(W - \frac{MW - \rho_1\rho_2}{M + \rho_2} \right) = a_2\rho_2 \frac{W + \rho_1}{M + \rho_2}.$$

Using the definitions of ρ_1 and ρ_2 we see that the determinant of \mathbf{J}_2 is

$$(a_1y_0 + b_1)(a_2x_0 + b_2) - a_1a_2\rho_1\rho_2 = a_1a_2y_0x_0 + b_1a_2x_0 + b_2a_1y_0$$

which is clearly positive.

A negative trace and positive determinant tell us that the equilibrium point is stable, either a node or a spiral. We'll return to this shortly.

Nullcline-and-arrow diagrams. What are the nullclines for our system? We are only interested in sketching the phase portrait in the rectangle $0 \leq x \leq M, 0 \leq y \leq W$, since this is the only

portion of the xy -plane that is biologically relevant. The restrictions $x \geq 0, y \geq 0$ are clear, and the restrictions $x \leq M, y \leq W$ follow since the number of infected men (women) cannot exceed the total number of men (women) in the population. The x -nullcline is the curve with equation

$$(10.63) \quad y = \frac{b_1 x}{a_1(M - x)};$$

let's call the right-hand side of (10.63) $F(x)$. To sketch the graph, it's helpful to use a little calculus. From (10.63) we compute

$$F'(x) = \frac{M a_1 b_1}{[a_1(M - x)]^2} \quad \text{and} \quad F''(x) = \frac{2 M b_1}{a_1(M - x)^3}.$$

Since F' is always positive, the graph of F is increasing. We've observed that $x \leq M$, so the graph of F is concave up for $0 < x < M$. Finally, $F(0) = 0$ and $\lim_{x \rightarrow M^-} F(x) = \infty$. The x -nullcline is sketched in Fig. 10.47, where we have also shown the correct right/left arrows on either side of this nullcline. We've only shown the portion of the nullcline in the biologically relevant rectangle.

A similar analysis is possible for the y -nullcline, which has equation

$$y = \frac{W a_2 x}{b_2 + a_2 x}.$$

The graph of this is an increasing, concave down curve, passing through $(0, 0)$ and which has limit W as $x \rightarrow \infty$. You are asked to verify these facts in Exercise 5. Fig. 10.48 shows the y -nullcline and the corresponding up/down arrows in the biologically relevant rectangle.

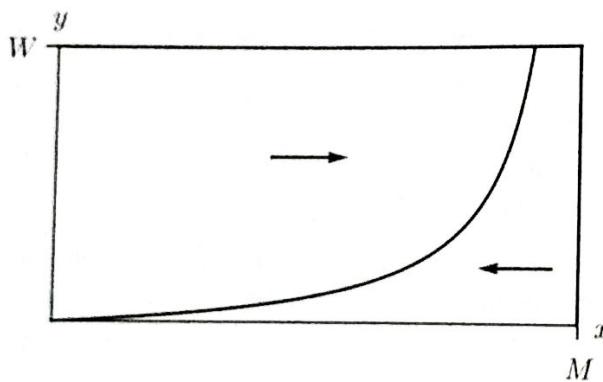


Figure 10.47. The x -nullcline.

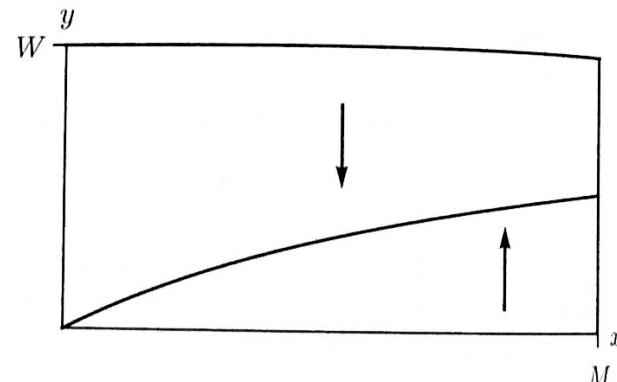


Figure 10.48. The y -nullcline.

We want to put Figs. 10.47 and 10.48 together. There are two possibilities here: Either the x - and y -nullclines intersect in two points, $(0, 0)$ and a second equilibrium point, or they only intersect in $(0, 0)$. From our earlier discussion of the equilibrium points, we know the first case occurs exactly when $M W a_1 a_2 - b_1 b_2 > 0$. (Exercise 5(c) gives another perspective on joining Fig. 10.47 and Fig. 10.48.) We focus on this case, leaving the case $M W a_1 a_2 - b_1 b_2 \leq 0$ for Exercise 5.

The nullcline-and-arrow picture looks like Fig. 10.49. Remembering that $(0, 0)$ is a saddle point, we sketch some trajectories. We hadn't classified the second equilibrium point, since the algebra involved in doing so was a bit off-putting, but we did note it was stable, and we can see from the nullcline-and-arrow diagram that it must be a node: A trajectory cannot spiral about the first quadrant equilibrium point because once it enters the region between the nullclines and to the left of the equilibrium point, it cannot leave. In that basic region trajectories rise, but they cannot rise above the y -nullcline because in so doing they would violate the downward direction-indicator arrow in the region above the y -nullcline.

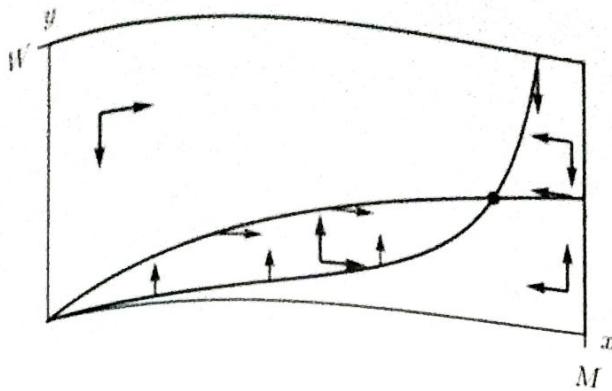


Figure 10.49. Nullcline-and-arrow diagram; $MW a_1 a_2 - b_1 b_2 > 0$.

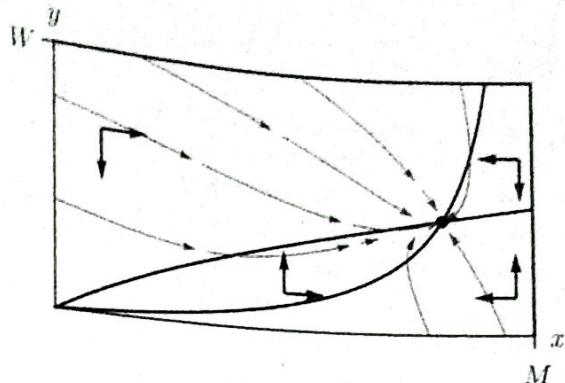


Figure 10.50. Phase portrait; $MW a_1 a_2 - b_1 b_2 > 0$.

What does this predict for the long-term behavior of $x(t)$ and $y(t)$? As $t \rightarrow \infty$,

$$x(t) \rightarrow \frac{MW - \rho_1 \rho_2}{W + \rho_1} \quad \text{and} \quad y(t) \rightarrow \frac{MW - \rho_1 \rho_2}{M + \rho_2}.$$

Since these limiting values are nonzero, the disease will continue at an endemic level in the population.

Example 10.5.3. Modeling smoking. In this example, we model cigarette smoking among high school students. We assume that peer pressure plays some role in recruiting nonsmokers to become smokers. A similar system of equations could be used to model drug or alcohol use. We consider the student population to be divided into three compartments: nonsmokers $x(t)$ (whom we think of as “potential smokers”), smokers $y(t)$, and students who have quit smoking $z(t)$. We will assume that the total population size is fixed. Thus while students graduate, an equal number of new students matriculate, so that $x(t) + y(t) + z(t) = N$, where N is the constant total size of the school. The nonsmoker compartment plays the role of the “susceptible” compartment in the disease models, and we assume that the rate at which nonsmokers become smokers is jointly proportional to the number of smokers and the number of nonsmokers. This is the quantification of the role of peer pressure, and it will give rise to a term $-(\text{constant})xy$ in the differential equation for $\frac{dx}{dt}$ and a corresponding term, with a positive sign, in the differential equation for $\frac{dy}{dt}$. Remembering that N is constant, we will actually write this term in the form

$$-bx \frac{y}{N},$$

where the factor $\frac{y}{N}$ is the *proportion* of smokers in the student body and b is a positive constant that reflects, for example, the overall amount of social interaction and the likelihood of a nonsmoker being influenced to start smoking by the presence of smokers. We will also assume that the graduation rates are the same from each of the three compartments and that all newly matriculated students are initially nonsmokers. Finally we assume that smokers “recover”—that is, move into the “quitters” compartment at a rate proportional to y , the number of smokers. Our first model doesn’t consider the possibility of “relapse” for someone who has quit smoking. With these assumptions, we have the differential equation system

$$(10.64) \quad \frac{dx}{dt} = gN - bx \frac{y}{N} - gx \quad (\text{nonsmokers}),$$

$$(10.65) \quad \frac{dy}{dt} = bx \frac{y}{N} - cy - gy \quad (\text{smokers}),$$

$$(10.66) \quad \frac{dz}{dt} = cy - gz \quad (\text{quitters})$$

for some positive constants g, b , and c . The terms $-gx$, $-gy$, and $-gz$ represent the graduation rates from the three compartments; these are exactly balanced by the term $gN = g(x + y + z)$ in the first equation, representing the addition of new students to the school, all initially in the nonsmoker category. In the equation for $\frac{dy}{dt}$ the term $-cy$ represents smokers quitting. The units on g and c would be, for example, $\frac{1}{\text{years}}$, and their reciprocals $\frac{1}{g}$ and $\frac{1}{c}$ have the meaning of “average time in school” and “average time as a smoker”. Thus if the average time that a student spends in high school is 4 years, we have $g = \frac{1}{4}/\text{year}$. If we take 12 years as the average number of years that a smoker continues to smoke, then $c = \frac{1}{12}/\text{year}$.

You may notice that the form of equations (10.64)–(10.66) is exactly the same as in the SIR disease model in Example 10.5.1. So the analysis we did there applies here as well. However, we are going to proceed a little differently and begin by using the relation $x(t) + y(t) + z(t) = N$, to reduce the system to a planar system, with dependent variables y and z . Making the substitution $x = N - y - z$ in equation (10.65), we obtain the equations

$$\begin{aligned} \frac{dy}{dt} &= b \frac{y(N - y - z)}{N} - (g + c)y, \\ \frac{dz}{dt} &= cy - gz. \end{aligned}$$

Next we will make the substitutions $S = \frac{y}{N}$ and $Q = \frac{z}{N}$, so that S and Q are the proportions of smokers and of smokers who have quit, respectively. These are “dimensionless” variables; they have no units. Since $y = NS$ and $z = NQ$, where N is constant, this gives

$$\begin{aligned} N \frac{dS}{dt} &= b \frac{NS(N - NS - NQ)}{N} - (g + c)NS, \\ N \frac{dQ}{dt} &= cNS - gNQ, \end{aligned}$$

or simply

$$(10.67) \quad \frac{dS}{dt} = bS(1 - S - Q) - (g + c)S, \quad \frac{dQ}{dt} = cS - gQ.$$

Because $\frac{x(t)}{N} + \frac{y(t)}{N} + \frac{z(t)}{N} = 1$ for all t , the only relevant part of the SQ -plane is the triangular region $S \geq 0, Q \geq 0, S + Q \leq 1$ shown in Fig. 10.51.

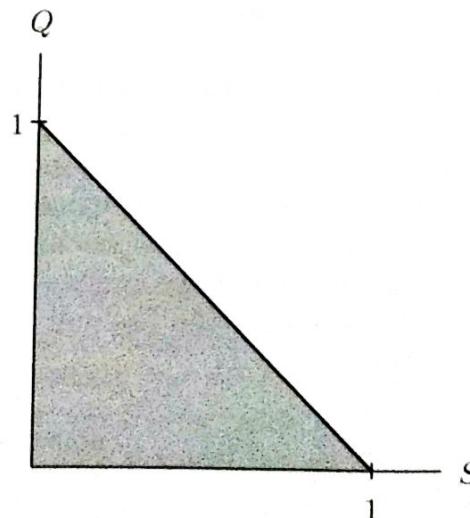


Figure 10.51. The biorelevant region $S \geq 0, Q \geq 0, S + Q \leq 1$.

Note that the Jacobian matrix for the system in equation (10.67) is

$$\begin{pmatrix} b - 2bS - bQ - (g + c) & -bS \\ c & -g \end{pmatrix}.$$

To proceed with our analysis of this system, we will assign values to the constants b , g , and c . As previously discussed, we will use $g = \frac{1}{4}$ and $c = \frac{1}{12}$, and we will set $b = \frac{1}{2}$. With these values our system becomes

$$(10.68) \quad \frac{dS}{dt} = \frac{1}{2}S(1 - S - Q) - \frac{1}{3}S, \quad \frac{dQ}{dt} = \frac{1}{12}S - \frac{1}{4}Q.$$

Nullclines and arrows. Since $\frac{dS}{dt} = S(\frac{1}{6} - \frac{1}{2}S - \frac{1}{2}Q)$, the S -nullclines are the pair of lines $S = 0$ and $S + Q = \frac{1}{3}$. The Q -nullcline is the line $Q = \frac{1}{3}S$. Fig. 10.52 shows the nullcline-and-arrow picture in the biologically relevant triangle.

Equilibrium points. Equilibrium points for the system (10.68) appear as the intersection of an S -nullcline with a Q -nullcline. One equilibrium point is $S = 0, Q = 0$, which corresponds in the original system (10.64)–(10.66) to $x = N, y = 0, z = 0$. A second equilibrium point is $S = \frac{1}{4}, Q = \frac{1}{12}$. To classify these equilibrium points we use the Jacobian matrix

$$\begin{pmatrix} \frac{1}{6} - S - \frac{1}{2}Q & -\frac{1}{2}S \\ \frac{1}{12} & -\frac{1}{4} \end{pmatrix}.$$

Evaluating the Jacobian matrix at the equilibrium point $(0, 0)$ gives

$$\mathbf{J}_1 = \begin{pmatrix} \frac{1}{6} & 0 \\ \frac{1}{12} & -\frac{1}{4} \end{pmatrix},$$

and the Jacobian matrix at the equilibrium point $(\frac{1}{4}, \frac{1}{12})$ is

$$\mathbf{J}_2 = \begin{pmatrix} -\frac{1}{8} & -\frac{1}{8} \\ \frac{1}{12} & -\frac{1}{4} \end{pmatrix}.$$

Since \mathbf{J}_1 has one positive eigenvalue $(\frac{1}{6})$ and one negative eigenvalue $(-\frac{1}{4})$, we conclude from Theorem 10.2.3 that $(0, 0)$ is a saddle point and thus unstable. Since \mathbf{J}_2 has trace $\tau = -\frac{3}{8} < 0$ and determinant $\Delta = \frac{1}{24} > 0$ with $\tau^2 - 4\Delta = \frac{9}{64} - \frac{1}{6} < 0$, Table 8.2 from Section 8.9 together with Theorem 10.2.3 tell us that $(\frac{1}{4}, \frac{1}{12})$ is a stable spiral point. Figs. 10.52 and 10.53 show the nullcline-and-arrow diagram and a phase portrait. Trajectories approach the equilibrium point $(\frac{1}{4}, \frac{1}{12})$, and long term we expect $S(t) \rightarrow \frac{1}{4}$ and $Q(t) \rightarrow \frac{1}{12}$.

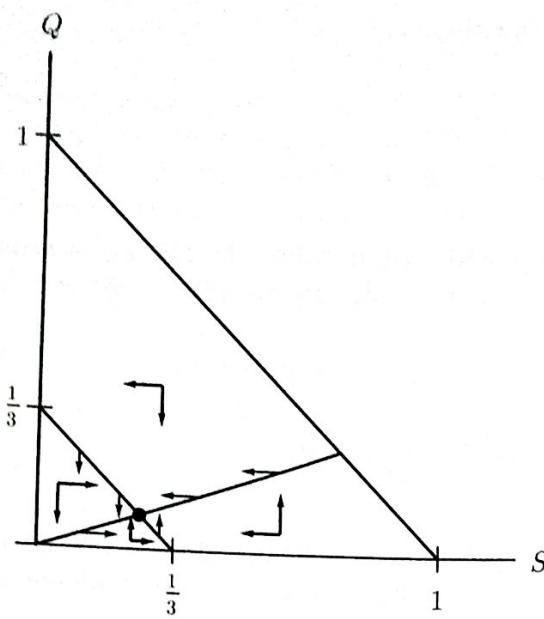


Figure 10.52. Nullcline-and-arrow diagram.

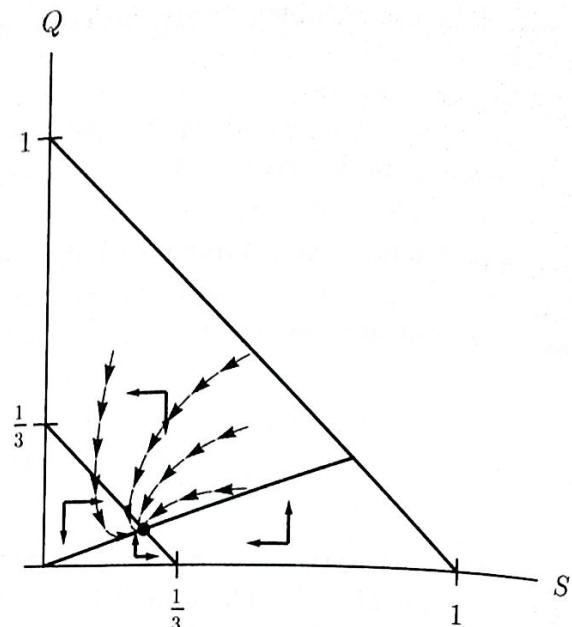
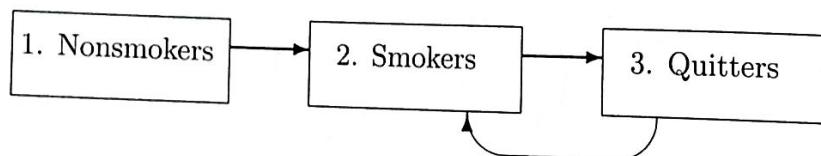


Figure 10.53. A phase portrait.

Allowing for relapse. Let's make this model more realistic. Smokers who have quit may very well start smoking again; this adds the arrow from compartment 3 back to compartment 2 in the diagram below.



We assume that the rate at which quitters relapse is jointly proportional to z , the number of students in compartment 3, and y/N , the proportion of smokers in the school population. So we modify equations (10.64)–(10.66) to

$$(10.69) \quad \frac{dx}{dt} = gN - bx\frac{y}{N} - gx, \quad \frac{dy}{dt} = bx\frac{y}{N} - (g + c)y + rz\frac{y}{N}, \quad \frac{dz}{dt} = cy - gz - rz\frac{y}{N}.$$

As before, we focus on the second two equations (substituting $x = N - y - z$) and make the change of variable $S = y/N$, $Q = z/N$ to obtain (see Exercise 8)

$$(10.70) \quad \frac{dS}{dt} = bS(1 - S - Q) - (g + c)S + rQS, \quad \frac{dQ}{dt} = cS - gQ - rQS.$$

The Jacobian matrix for this new system is

$$\begin{pmatrix} b - 2bS - bQ - (g + c) + rQ & -bS + rS \\ c - rQ & -g - rS \end{pmatrix}.$$

Analyzing this model with values for the parameters. As before, we will continue our analysis using specific values for the parameters. Now we will use the values $b = 0.62$, $g = 0.25$,

$c = 0.06$, and $r = 1.4$.¹² With these values the S -nullclines are the pair of lines

$$S = 0 \quad \text{and} \quad Q = \frac{0.62}{0.78}S - \frac{0.31}{0.78}$$

and the Q -nullcline is the curve with equation

$$Q = \frac{0.06S}{0.25 + 1.4S}.$$

This is a curve passing through $(0, 0)$ which is increasing and concave down for S in the relevant range $0 \leq S \leq 1$. These nullclines are shown separately in Figs. 10.54–10.55. (Note the scale on the Q -axis in Fig. 10.55.) In Exercise 11 you are asked to show the appropriate left/right and up/down arrows to complete this nullcline-and-arrow diagram and to finish the analysis of the phase portrait for these particular parameters.

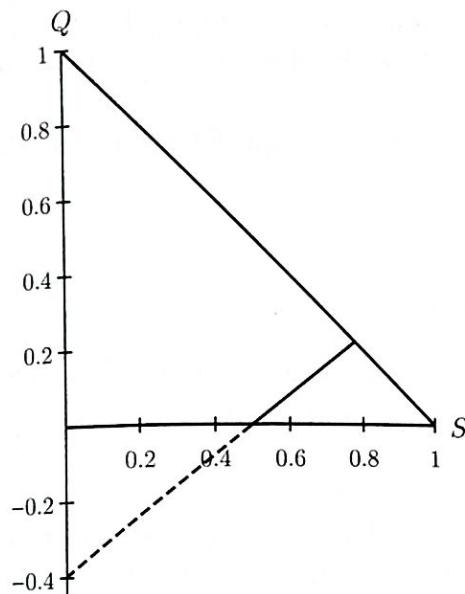


Figure 10.54. The S -nullcline in the biorelevant triangle.

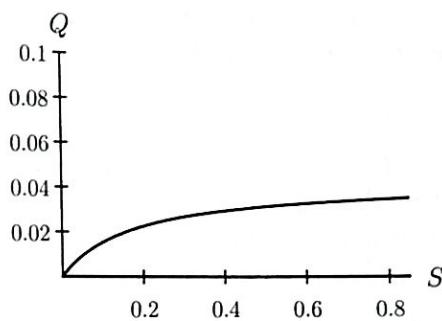


Figure 10.55. The Q -nullcline in a portion of the biorelevant triangle.

10.5.1. Exercises.

- Suppose that in Example 10.5.1 we assume that $\beta K < \gamma + \mu$, so that the only biologically relevant equilibrium point is $(K, 0)$.
 - Show that this equilibrium point is a stable node.
 - Give the nullcline-and-arrow sketch for this case, showing several trajectories.
 - What happens to $S(t)$ and $I(t)$ as $t \rightarrow \infty$?
- Use the following estimates for the basic reproduction number for the listed diseases (all are diseases where recovery confers immunity) to estimate what percentage of newborns need to be successfully immunized to prevent the disease from becoming endemic.

¹²The values of b and r are taken from *Mathematical models for the dynamics of tobacco use, recovery, and relapse by C. Castillo-Garsow, G. Jordan-Salivia, and A. Rodriguez-Herrera, Biometrics Unit Technical Reports, Number BU-1505-M, Cornell University, Ithaca, 1997.*

Table 10.3. Comparing R_0 .

Disease	Basic reproduction number R_0
measles	12–18
mumps	5–7
diphtheria	6–7
pertussis	12–17
smallpox	5–7

Only smallpox has been eliminated on a worldwide basis, by intensive vaccination efforts. The last known case was in 1977 in Somalia. Now it exists only in laboratories, and routine smallpox vaccination is no longer done. The deliberate release of smallpox as a terrorist act has been a recent concern, especially since 9/11/2001. Mathematical models are an important tool in planning for a response to such a bioterrorism attack. One recent such model assumes the release of smallpox in the New York City subway.¹³ While the model is more complicated than that considered in Example 10.5.1 (in particular it separates “subway users” from “nonsubway users”, and it considers behavioral changes people might make after such an attack), many of the basic features of our model are still present.

3. For endemic diseases that confer immunity, there is a rule of thumb for estimating the basic reproduction number R_0 :

$$R_0 \approx 1 + \frac{L}{A}$$

where L is the average life span and A is the average age of contracting the disease. Suppose that in 1955 the average life span in the US was 70 and the average age of contracting polio was 17.9. What percentage of the population would have to be vaccinated for herd immunity?

4. This problem outlines Daniel Bernoulli’s work on smallpox in 1760. We start with a group of people all born at the same time $t = 0$.

- Let $x(t)$ denote the number of this original group who are still alive t years later.
- Let $y(t)$ be the number who are still alive at time t and have not yet had smallpox.

The y -population is thus the susceptibles who are alive at time t . There are two “rate out” terms for the y -population, corresponding to the fact that some members contract smallpox and others die from other causes. Bernoulli assumes that susceptibles contract smallpox at a rate proportional to the y -population and deaths occur from nonsmallpox causes at a rate which depends on time t but is the same for both the y - and x -populations. These assumptions give the equation

$$(10.71) \quad \frac{dy}{dt} = -\beta y - d(t)y$$

where β is a positive constant. The x -population changes for two reasons: Some people die from smallpox, and some die from other causes. The “die from other causes” factor gives rise to a term $-d(t)x$ in the differential equation for $\frac{dx}{dt}$. The people who die from smallpox are some fraction δ of those who contract the disease. Thus the differential equation for the x -population is

$$(10.72) \quad \frac{dx}{dt} = -\delta\beta y - d(t)x.$$

¹³G. Chowell, A. Cintron-Arias, S. Del Valle, F. Sanchez, B. Song, J. Hyman, H. Hethcote, and C. Castillo-Chavez, *Mathematical applications associated with the deliberate release of infectious agents*, in *Mathematical Studies on Human Disease Dynamics*, Contemporary Mathematics, Vol. 410, A. Gumel, Editor-in-Chief, American Mathematical Society, 2006, pp. 51–71.

Since t appears explicitly in the equations for $\frac{dx}{dt}$ and $\frac{dy}{dt}$ and we don't know what the function $d(t)$ is, we will need to do something clever to solve the system (10.71)–(10.72).

- (a) Set $z = y/x$ and show that $\frac{dz}{dt} = -\beta z + \delta \beta z^2$.
- (b) Solve the equation in (a) for z , with the initial condition $z(0) = 1$. The rationale for this initial condition is that at a very young age, no survivors have smallpox, since it is almost always fatal to infants. So $z(t) = \frac{y(t)}{x(t)} = 1$ as $t \rightarrow 0^+$.
- (c) In your answer to (b), use the values $\beta = \delta = \frac{1}{8}$, estimated by Bernoulli, and compute $z(10)$ and $z(20)$. What percentage of 10-year-olds have not had smallpox? What percentage of 20-year-olds have not?

5. (a) In Example 10.5.2, suppose t is measured in days. What are the units on $a_1, b_1, a_2, b_2, \rho_1$, and ρ_2 ?
 (b) Sketch the graph of

$$y = \frac{W a_2 x}{b_2 + a_2 x}$$

showing only the portion that lies in the rectangle $0 \leq x \leq M, 0 \leq y \leq W$.

- (c) For the functions

$$F_1(x) = \frac{b_1 x}{a_1(M-x)} \quad \text{and} \quad F_2(x) = \frac{W a_2 x}{b_2 + a_2 x}$$

compute $F'_1(0)$ and $F'_2(0)$. Under what condition is $F'_1(0) > F'_2(0)$? How does this help explain how to fit together the two pictures in Figs 10.47 and 10.48?

- (d) Give the nullcline-and-arrow diagram for equation (10.59) under the assumption $M W a_1 a_2 - b_1 b_2 < 0$. Predict the course of the disease in this case.
6. Suppose we model the spread of gonorrhea in a population of 1,000 sexually active women and 1,000 sexually active men by the system in equation (10.59), with $b_1 = 1/50$ days and $b_2 = 1/10$ days (this corresponds to an average infective period of 50 days for women and 10 days for men). Also suppose that $a_1 = 1/5,000$ and $a_2 = 1/25,000$.
- (a) Find the equilibrium point that has both coordinates positive. What are the total number of infected persons (men + women) corresponding to this equilibrium solution?
- (b) Suppose there is a public health policy that would halve the number of at-risk women. What is the new first quadrant equilibrium point, and is the total number of infected persons corresponding to this equilibrium?
- (c) Next suppose there is a different public health policy that would instead halve the number of at-risk men. Find the equilibrium point in the first quadrant and the total number of infected persons corresponding to this equilibrium.
- (d) Of the two public health policies just discussed, which is more effective in reducing the number of infected individuals at the equilibrium solution?

7. In equation (10.59), show that the substitutions $X = \frac{x}{M}$ and $Y = \frac{y}{W}$ lead to the system

$$(10.73) \quad \frac{dX}{dt} = a_1(1-X)WY - b_1X \quad \text{and} \quad \frac{dY}{dt} = a_2(1-Y)MX - b_2Y.$$

Notice that X represents the proportion of the male population that is infected and Y represents the proportion of the female population that is infected.

8. Show the details of obtaining the equations (10.70) from the second two equations in (10.69).
9. Show that for the system in equation (10.67), the equilibrium point $(0, 0)$ is a saddle point if $b > g + c$ and a stable node if $b < g + c$.

10. In the model for “smoking with relapse” do you think it is more likely for a “quitter” to start smoking again or for a nonsmoker to start smoking? Based on your answer, would you have $b > r$ or $b < r$?
11. In this problem you will further analyze the smoking with relapse model in equation (10.70) with the values $b = 0.62$, $g = 0.25$, $c = 0.06$, and $r = 1.4$.
 - (a) Using Figs. 10.54–10.55 give the nullcline-and-arrow diagram in the biologically relevant region $S \geq 0$, $Q \geq 0$, $S + Q \leq 1$.
 - (b) Find (approximately) all biorelevant equilibrium points and classify them according to type and stability.
 - (c) Sketch a phase portrait using (a) and (b). What do you predict for the long-term behavior of $S(t)$ and $Q(t)$?
12. Consider the initial value problem consisting of the system in equation (10.70), with the values for b , g , c , and r as given in the preceding exercise and initial condition $S(0) = 0.2$, $Q(0) = 0.01$.
 - (a) Sketch the solution curve of this initial value problem based on your nullcline-and-arrow diagram from Exercise 11.
 - (b) (CAS) Using a computer algebra system, solve this initial value problem numerically, and plot the corresponding trajectory over the time interval $[0, 18]$.
 - (c) Using your numerical solution, compute $S(6)$, $S(12)$, and $S(18)$, as well as $Q(6)$, $Q(12)$, and $Q(18)$.
13. Suppose in Example 10.5.3 we model relapse in a different way, by assuming that quitters become smokers again at a rate proportional to the number of people who have quit.
 - (a) What system of equations does this assumption give?
 - (b) Convert your model in (a) to a planar system for $S = y/N$ and $Q = z/N$. When is the equilibrium point $S = 0$, $Q = 0$ stable? Unstable?
14. Norovirus (“stomach flu”) is a common and unpleasant illness caused by a virus.¹⁴ College students, especially those living in dorms, are frequent victims, as are cruise ship passengers. In this problem we will look at a model for the spread of this disease which divides the population into 5 compartments: (S) susceptibles, (E) exposed (these are people who do not yet show symptoms but soon will), (I) infected individuals showing symptoms, (A) asymptotic infected people (these are people who do not have symptoms but are still shedding the virus), and (R), the recovered class of people with temporary immunity to norovirus. Movement between these compartments follows these rules:
 - (1) All births go into the susceptible category with constant births of B per day.
 - (2) Susceptibles move into the exposed compartment at a rate proportional to the product of the number of susceptibles ($S(t)$) and the number of symptomatic infectives ($I(t)$), with proportionality constant β . Notice this means we are assuming only symptomatic infected people are infectious, not the “presymptomatic” exposed or “postsymptomatic” asymptomatic infected people.
 - (3) Exposed individuals move into the symptomatic infected compartment at a rate proportional to the number of exposed ($E(t)$), with proportionality constant $1/\mu_s$, where μ_s is the average length of the incubation period in days.
 - (4) Symptomatic individuals move into the asymptomatic infected compartment at a rate proportional to the number of (symptomatic) infecteds ($I(t)$), with proportionality constant $1/\mu_a$, where μ_a is the average length of symptoms in days.
 - (5) Asymptomatic infected individuals move into the temporarily immune category (R compartment) at a rate proportional to the number of asymptomatic infected people ($A(t)$),

¹⁴The model discussed in this exercise comes from the article *Duration of Immunity to Norovirus Gastroenteritis* by K. Simmons, M. Gambhir, J. Leon, and B. Lopman, Emerging Infectious Diseases, Aug. 2013, 19(8), 1260–1267.

with proportionality constant $1/\rho$, where ρ is average number of days a person sheds the virus without showing symptoms.

- (6) People leave the R compartment in one of two ways: Either they lose immunity entirely, moving into the S (susceptible) compartment, or they “lose immunity to infection without losing immunity to disease”; this means they move back into the A compartment, shedding the virus but not showing any symptoms. The first case occurs at a rate proportional to R with proportionality constant $1/\theta$ where θ is the average duration of temporary immunity. The second case occurs at a rate jointly proportional to $R(t)$ and $I(t)$, with proportionality constant β as in (2). Moving from R to A has the effect of temporarily boosting immunity again, and these individuals eventually move back into the R compartment.
- (7) Deaths occur from all compartments, at a rate proportional to the compartment size with proportionality constant δ , which we assume is the same for all compartments.
 - (a) Draw a compartment diagram, labeled S, E, I, A, and R, with arrows showing what transfers we have between compartments. You may include a sixth compartment, D, for dead, or you may omit this.
 - (b) Using your answer to (a), give a system of differential equations for $\frac{dS}{dt}$, $\frac{dE}{dt}$, $\frac{dI}{dt}$, $\frac{dA}{dt}$, and $\frac{dR}{dt}$, using the following facts: The average incubation period for norovirus is 1 day, the average duration of symptoms is 2 days, the average duration of asymptotic virus shedding is 10 days, and the average duration of temporary immunity is 5 years. Your equations will include the parameter β from (2) above; we do not give a numerical value for this. Whether or not you included a “dead” compartment in (a), your equations should include births of B people per day and deaths as described in (7) above. We do not give a numerical value for δ .
 - (c) Another model allows for susceptibles to become infected by people in the exposed and asymptomatic compartments, but at a lower rate than by symptomatic infected persons. This changes the “rate in” terms for $\frac{dE}{dt}$ to be $\beta_1 S(t)E(t) + \beta_2 S(t)I(t) + \beta_3 S(t)A(t)$ with β_1 and β_3 to be smaller than β_2 . What are the corresponding changes in $\frac{dS}{dt}$? In $\frac{dR}{dt}$? In $\frac{dA}{dt}$?

10.6. Hamiltonians, gradient systems, and Lyapunov functions

Conserved quantities. In Section 4.8 we used the second-order equation $mv'' + ky = 0$ to model an undamped mass-spring system, where m is the mass, k is the spring constant, and $y(t)$ is the displacement of the mass from equilibrium at time t . We can convert this to a first-order system, with dependent variables y and v , using the substitution $y' = v$:

$$(10.74) \quad y' = v, \quad v' = -\frac{\kappa}{m}y.$$

This linear system has a center at $(0, 0)$. The orbits are ellipses in the vy -plane with equations determined by

$$\frac{dv}{dy} = \frac{dv/dt}{dy/dt} = -\frac{ky}{mv}.$$

Separating variables in this first-order equation gives $\frac{1}{2}mv^2 + \frac{1}{2}ku^2 = C$; for $C > 0$ these describe ellipses as shown in Fig. 10.56.

that of the related linear system $\frac{d\mathbf{Y}}{dt} = \mathbf{J}(\mathbf{X}_e)\mathbf{Y}$ near its equilibrium point $(0, 0)$, where $\mathbf{J}(\mathbf{X}_e)$ is the Jacobian matrix of \mathbf{F} evaluated at \mathbf{X}_e . We use the familiar classification terminology from Chapter 8 of “saddle point”, “node”, and “spiral point”, and so on to capture this idea. Recall that nondegenerate nodes correspond to two different real eigenvalues with the same sign, while a planar linear system has a degenerate node at $(0, 0)$ if the associated matrix has a repeated eigenvalue. We will refer to saddle points, spiral points, and nondegenerate nodes as **major types** and get the best information in these cases:

Theorem 10.2.3. *Suppose we have an autonomous nonlinear system*

$$(10.27) \quad \frac{dx}{dt} = f(x, y), \quad \frac{dy}{dt} = g(x, y),$$

where $f(x, y)$ and $g(x, y)$ are continuously differentiable. Suppose (x_e, y_e) is an isolated equilibrium point for (10.27). Consider the related linear system

$$(10.28) \quad \mathbf{X}' = \mathbf{J}\mathbf{X}$$

where \mathbf{J} is the Jacobian matrix

$$\begin{pmatrix} \frac{\partial f}{\partial x}(x_e, y_e) & \frac{\partial f}{\partial y}(x_e, y_e) \\ \frac{\partial g}{\partial x}(x_e, y_e) & \frac{\partial g}{\partial y}(x_e, y_e) \end{pmatrix},$$

and assume that $(0, 0)$ is an isolated equilibrium point for this linear system (this is the same as saying $\det \mathbf{J} \neq 0$).

If $(0, 0)$ is one of the major types (saddle point, spiral point, or nondegenerate node) for the linear system (10.28), then (x_e, y_e) is the same type for the nonlinear system. Moreover, for these major types, the stability of the equilibrium point is the same for the nonlinear system as it is for the linear system.

Borderline types. Note that Theorem 10.2.3 does not apply if $(0, 0)$ is a center or degenerate node for the linear system $\mathbf{X}' = \mathbf{J}\mathbf{X}$. These cases are discussed in the next theorem, which will be further explored in Section 10.3.

Theorem 10.2.4. *Under the same hypotheses as Theorem 10.2.3, we have the following:*

If the linear system $\mathbf{X}' = \mathbf{J}\mathbf{X}$ has a degenerate node at $(0, 0)$, then the nonlinear system has either a node or spiral at (x_e, y_e) , and the stability is the same for both systems.

If the linear system $\mathbf{X}' = \mathbf{J}\mathbf{X}$ has a center at $(0, 0)$, then the nonlinear system has either a center, a spiral, or a hybrid center/spiral at (x_e, y_e) . In this case, we cannot predict the stability of (x_e, y_e) for (10.27).

It is helpful to keep the picture of the trace-determinant plane from Section 8.9 in mind here. Degenerate nodes correspond there to points on the parabola $4\Delta = \tau^2$; to either side of this parabola are regions corresponding to spirals and nodes. Centers correspond to the positive half of the Δ -axis; the bordering regions on either side of this ray correspond to asymptotically stable spirals or unstable spirals.

The terminology “hybrid center/spiral” refers to a situation where in each disk centered at the equilibrium point there are closed curve orbits surrounding the equilibrium point, but between two such orbits there may be spiral trajectories. We’ll see an example in Exercise 8 of Section 10.3. This situation does not occur if the component functions $f(x, y)$ and $g(x, y)$ are sufficiently nice (as will be the case for nearly every example we consider)⁵.

⁵In particular, it cannot occur if f and g are polynomials in x and y .