

- (e) The nutrient uptake function $p(S)$ can be shown experimentally to be a monotonically increasing function bounded above. Show that a Michaelis-Menten type function

$$p(S) = \frac{mS}{a + S},$$

with m and a positive, non-zero constants, satisfies these requirements. What is the maximum absorption rate? And why is a called the half-saturation constant? (Hint: The maximum absorption rate is the maximum reached by $p(S)$. For the second part consider $p(a)$.)

This system of equations is known as the Monod Model for single species growth and was developed by Jaques Monod in 1950.

- 3.14. Stability of 2-cycles.** Consider the discrete logistic equation (with $K = 1$)

$$X_{n+1} = X_n + rX_n(1 - X_n).$$

- (a) Show that every second term in the sequence X_0, X_1, X_2, \dots satisfies the difference equation

$$\begin{aligned} X_{n+2} &= (1 + 2r + r^2)X_n - (2r + 3r^2 + r^3)X_n^2 \\ &\quad + (2r^2 + 2r^3)X_n^3 - r^3X_n^4. \end{aligned}$$

- (b) For equilibrium solutions, let $S = X_{n+2} = X_n$ and obtain a quartic equation (that is, an equation with the unknown raised to the fourth power, at most). Explain why $S = 0$ and $S = 1$ must be solutions of this quartic equation. Hence show that the other two solutions are

$$S = \frac{(2 + r) \pm \sqrt{r^2 - 4}}{2r}.$$

[Note: Comparing with Figure 3.10 ($r = 2.2$) we see that these two values of the two non-zero equilibrium solutions are the values between which the population oscillates in a two-cycle. Furthermore, when r increases to where these two equilibrium solutions become unstable this corresponds to where the two-cycle changes to a four-cycle.]

- 3.15. Linear differential-delay equation.** Consider the linear differential-delay equation

$$\frac{dX}{dt} = X(t - 1), \quad X(0) = 1.$$

Look for an exponential solution, of the form $X(t) = Ce^{mt}$ where m is a constant you must determine, and C is an arbitrary constant.

Chapter 4

Numerical solution of differential equations

This chapter provides a brief overview of numerical procedures on which we rely when employing software as a tool in the solution and analysis of mathematical models. While developments have ensured that in most cases the methods are robust, it is important to understand the trade-offs we must accept when using them, and the possible errors which could accumulate, making interpretation inaccurate. Perhaps the most important part of this chapter is the final discussion.

4.1 Introduction

In finding solutions to differential equations, plotting trajectories and displaying time-dependent graphs, we have come to rely on the performance of computers and software packages. Some packages use analytical solutions where possible, a symbolic approach such as in **Maple**, but many problems cannot be solved analytically and there is a need to employ numerical schemes to find a solution. In this case the numerical solution, whilst possibly extremely accurate, is an approximation to the exact analytic solution.

There are some major drawbacks in accepting whatever is produced by a computer, particularly when numerical solvers are used, as errors may accrue for the following reasons:

- there are *round-off errors* and these increase with an increase in the number of calculations performed,
- there are *discretisation errors* resulting from the estimation of a solution for a discrete set of points; these may decrease with an increase in the number of points used,
- there are errors due to the *estimating procedure*, or method of approximation, used by the numerical solver.

Many different numerical procedures are available for finding derivatives, integrals, sums, etc. First we provide a very brief outline of a few used in finding solutions to differential equations (the main use of solvers in this book), and we then consider some of the drawbacks in using these numerical approximations.

4.2 Basic numerical schemes

To examine some numerical schemes used in solving differential equations, we consider the general problem $y' = f(t, y)$ with $y(t_0) = y_0$, and apply the schemes which follow to calculate (estimate) $y(t)$ with $t_0 < t < T$. We begin with a very simple method, Euler's method, to gain a basic understanding of the process any method needs to adopt, and then look briefly at a well-known and widely used method, the Runge-Kutta method.

Euler's Method

Clearly a computer cannot calculate every point on a curve as there are infinitely many, so an approximation is made for a discrete set of points. Euler's method provides a very simple way of approximating the solution of a differential equation at a discrete set of points.

The steps in Euler's method are as follows:

- Divide the interval into N equal sections, then $t_n = t_0 + nh$ for $n = 0, 1, \dots, N - 1$ and $h = (T - t_0)/N$ is the step size.
- We know that (t_0, y_0) is on the curve and we approximate $y_n = y(t_n)$. To approximate y_1 we follow the tangent of the curve through (t_0, y_0) (which is known) extending it to t_1 . Then

$$y_1 \approx y_0 + h f(t_0, y_0)$$

or

$$y' = f(t_0, y_0) \approx \frac{y_1 - y_0}{h} = \frac{y_1 - y_0}{t_1 - t_0}.$$

- We follow this procedure repeatedly: $y_2 \approx y_1 + hf(t_1, y_1)$ and so on for y_3, \dots, y_n .

Figure 4.1 illustrates this procedure.

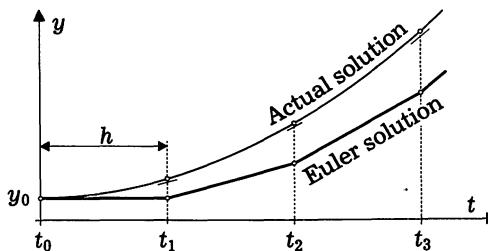


Figure 4.1: Diagram of Euler's method for solving differential equations.

In general Euler's method is the recursive scheme

$$y_{n+1} = y_n + hf(t_n, y_n)$$

with

$$t_{n+1} = t_n + h \quad \text{and} \quad 0 \leq n \leq N - 1.$$

The following example shows how to apply Euler's method.

Example 4.1: Use Euler's method, with step size $h = 0.1$, to find $y(0.2)$ for the differential equation

$$\frac{dy}{dt} = y^2 + t, \quad y(0) = y_0 = 1.$$

Solution: For this equation $f(t, y) = y^2 + t$. Using Euler's method, with $h = 0.1$, and letting $n = 0$, we obtain

$$y_1 = y(0.1) = y_0 + hf(t_0, y_0) = 1 + 0.1 \times (1^2 + 0) = 1.1.$$

Now, with $n = 1$, we obtain

$$y_2 = y(0.2) = y_1 + hf(t_1, y_1) = 1.1 + 0.1 \times (1.1^2 + 0.1) = 1.231.$$

Taylor's Theorem states that

$$y_{n+1} = y_n + h \frac{dy_n}{dt} + \frac{h^2}{2!} \frac{d^2y_n}{dt^2} + \dots$$

(Taylor's Theorem provides an accurate polynomial approximation to a large group of functions. For details see Appendix B.2.)

Comparing this approximation with Euler's method, it is clear that the latter consists of the first two terms in this expansion. We say that Euler's method is a *first-order approximation*. (Including further terms in this series would produce second-order, third-order etc., approximations.)

With any approximation, and thus with each numerical scheme, comes an associated error term. Since the approximation in Euler's method consists of only the first two terms of the Taylor series, it would be useful to be able to calculate the error term and then perhaps

control the size of the error by choice of the step size. From Euler's method we have the estimated value

$$y_{n+1} = y_n + h f(t_n, y_n) \quad n = 0, \dots, N - 1.$$

From Taylor's Theorem we have the actual value

$$y(t_{n+1}) = y(t_n) + h f(t_n, y(t_n)) + \frac{h^2}{2} f'(\xi_n, y(\xi_n))$$

where ξ_n lies between t_n and t_{n+1} .

Subtracting the estimate from the true value we get

$$\begin{aligned} y(t_{n+1}) - y_{n+1} &= y(t_n) - y_n \\ &\quad + h [f(t_n, y(t_n)) - f(t_n, y_n)] + \frac{h^2}{2} f'(\xi_n, y(\xi_n)) \end{aligned}$$

which is the error term E_{n+1} . So for each step of the method an error E_{n+1} is incurred. We can find an upper bound for this error term and it can be shown that

$$E_{n+1} \leq \frac{Dh}{2L} [e^{T-t_0} - 1]$$

where L is an upper bound for f and D is an upper bound for f' on the interval $[t_0, T]$ where $f(t, y)$ is continuously differentiable and $T = t_0 + (N - 1)h$.

Clearly $\lim_{h \rightarrow 0} E_{n+1} = 0$ indicating increasing accuracy of the estimation, with decreasing step size. (For a full analysis see, for example, Kincaid and Cheney (1991).)

However, it should be remembered that every computational operation (for example, addition, division, etc.) comes at a cost, as each may incur an error. This is a result of the fact that a computer can only store a fixed number of digits and so a number may be rounded off at each step. Thus, while decreasing the step size h (or equivalently increasing the number of points in the interval N), we are simultaneously increasing the number of operations or calculations and are thus increasing the impact of round-off errors. For best performance then, we should aim at finding some optimum h such that the combined effect of these two errors is minimised.

Runge-Kutta Methods

One-step algorithms that use averages of the slope function $f(t, y)$ at two or more points over the interval $[t_{n-1}, t_n]$ in order to calculate y_n are called *Runge-Kutta methods*. They are also examples of *predictor-corrector methods* as they make predictions for 'next' values, and then with a series of weights, correct them.

The fourth-order Runge-Kutta method (RK4) is one of the most widely used methods of any step algorithms. It involves weighted averages of slopes at the midpoint and end points of the subinterval. For the IVP $y' = f(t, y)$, $y(t_0) = y_0$, the fourth order RK method is a one step method (that is, y_n depends only on y_{n-1}) with the constant step size h and is given by

$$y_{n+1} = y_n + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4)$$

where

$$k_1 = f(t_n, y_n)$$

$$k_2 = f\left(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_1\right)$$

$$k_3 = f\left(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_2\right)$$

$$k_4 = f(t_n + h, y_n + hk_3).$$

Another Runge-Kutta method, using a simpler scheme of averaging than the RK4 above, is Heun's method. This is illustrated in Figure 4.2.

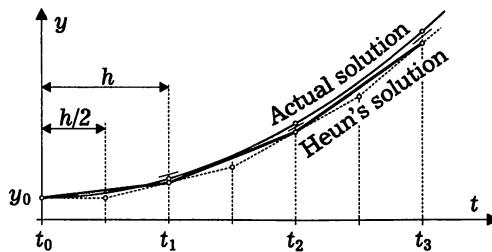


Figure 4.2: Diagram of Heun's method for solving differential equations.

Like Euler's scheme this method is a one-step method, but it is more accurate. With a constant step size h the general form of Heun's method is given by

$$y_{n+1} = y_n + \frac{h}{2}(k_1 + k_2)$$

where

$$k_1 = f(t_n, y_n),$$

$$k_2 = f(t_n + h, y_n + hk_1).$$

4.3 Computer implementation using Maple and MATLAB

Most good ODE solver packages use sophisticated methods of controlling errors, and being efficient most of the time they have widespread acceptance. Many schemes choose their own step-sizes in order to optimise the results, and some vary this step-size on an interval in order to provide an optimal result.

Note, once again, that there is a trade-off between decreasing the step-size and increasing the round-off error, due to an increased number of computations. Another problem is that of

instability over long time periods: sometimes, whilst providing an accurate approximation over an initial period of time, the solution may ‘become’ very inaccurate at later times.

Using **Maple** or **MATLAB**, the method of integration can be stipulated and the results compared. **Maple** or **MATLAB** also allow you to change and choose the *initial* step-size of the default numerical scheme which, as we see below, is sometimes required for adequate resolution. Note however, that with the standard DE solvers `ode45` for **MATLAB** and `DEplot` for **Maple**, adaptive stepping is used. This means the step-size is varied, becoming smaller when the function is changing fast, so as to maintain a specified error, and where the step-size increases when the function is changing slowly, so generally, specifying the step-size only really is useful if the *initial* default step-size happens to be too large. In practice, the default values for **MATLAB** appear to be adequate for most problems, but for **Maple** setting the `stepsize` parameter to 0.1, for example, is sometimes required, as discussed below.

In Chapter 2, in the discussion on finding a computer solution to a radioactive element decay cascade, we mentioned that it would be difficult to find a suitable numerical scheme because of the vastly different half-lives. (For Uranium-238 the half-life is billions of years whereas for the next element in its decay series, Thorium-234, the half-life is in days.) This problem requires large time steps to resolve the Uranium decay process and (comparatively) very small time steps to resolve the Thorium decay: such a problem is known as a *stiff problem*. This discrepancy between rates can be seen clearly in the following example. Suppose a solution to some differential equation $X' = f(x)$ is

$$X(t) = e^{-t} + e^{-1000t}, \quad t \geq 0.$$

Clearly, the second component decays at a much faster rate than the first. When t is small, the value of $X(t)$ is dominated by e^{-1000t} , and small step sizes are required for resolution of this behaviour. Alternatively, for t away from 0, the solution is dominated by e^{-t} and large step sizes (in comparison) can be used for high accuracy. Attempting to solve a stiff problem with a standard adaptive time-stepping method, such as the default in `DEplot` of **Maple** or `ode45` in **MATLAB**, will result in the method taking smaller and smaller time steps, and usually being unable to complete the calculation. For some software this may result in a prompt of an error message, triggered when a specified maximum number of function evaluations has been reached.

Alternative numerical methods, involving implicit solution methods, have been developed to deal specifically with stiff problems, but typically they increase the computation time incurred when the problem is not stiff. Thus, they are usually employed only once a problem is known to be stiff. **Maple** provides a stiff solver which can be used for the solution of such problems by stipulating the numerical method (always after `numeric`) in `dsolve`. Applying this to the example of lake pollution (Section 2.5), which is not a stiff problem, it is easy to establish a substantial increase in computation time. The code below gives an example of one way to specify a stiff solver in **Maple**.

```
> restart:with(plots):
> cin:=3;V:=28;F:=4*12;threshold:=4;init_c:=10;
> de1:=diff(c(t),t)=(F/V)*(cin-c(t));
> soln:=c0->dsolve({de1,c(0)=c0},c(t),numeric,method=lsode);
> plot1:=c0->odeplot(soln(c0),[t,c(t)],0..8):
> list1:=seq(plot1(i/2),i=1..12):
> line1:=plot([[],[0,threshold],[8,threshold]]):
> display({list1,line1});
```

With **MATLAB**, to use a stiff solver, simply replace the call to `ode45` with one to `ode15s`. **MATLAB** provides a few different stiff solvers, of different accuracy, but the `ode15s` is a good first method of choice.

A problem which frequently arises when using **Maple** to sketch solutions in a plane is a very odd-shaped or angular ‘scribble’, rather than a smoothly varying solution. By now,

if you have experimented with **Maple**, you will no doubt have encountered this problem in some implementation. Consider a pair of differential equations in time, $\text{de1}=dX/dt$ and $\text{de2}=dY/dt$. Then the following code fragment below, *without* the step size parameter stipulated, could produce the ‘mess’ in the first diagram of Figure 4.3, while, with the step size included, we obtain the second diagram. When the step-size is specified the problem ‘rights’ itself as displayed in the second diagram of Figure 4.3.

```
> ...
> plot1:=DEplot([de1,de2],[X,Y],t=0..80,{inits},X=0..Xlimit,Y=0..Ylimit,
  scene=[P,H],linecolor=black,stepsize=0.1,arrows=none):
> display(plot1);
```

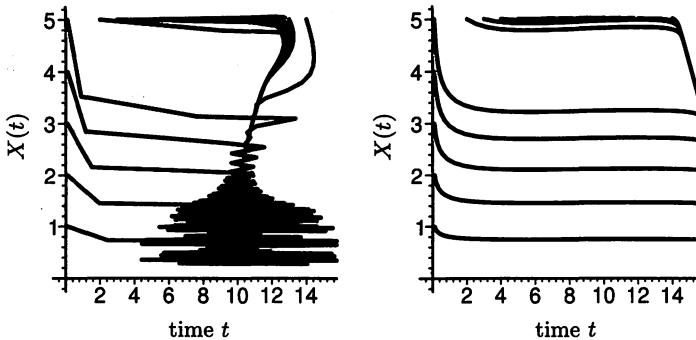


Figure 4.3: Solution of a differential equation for different initial conditions. In the diagram on the left a large step size was used, and instability of the numerical scheme prevents convergence. In the diagram on the right the step size of the solver has been reduced.

This problem is that of *instability* of the numerical scheme. Typically this can be improved with a decrease in the step-size, but sometimes the values of a step-size for which convergence to the solution ensues can be extremely small and depend on the differential equation itself. (We see an example of this in Section 4.4.) Furthermore, as the step-size decreases, there is an increasing error due to a larger number of operations which once again becomes an issue.

Note that it is possible to control the absolute and relative error using **Maple** or **MATLAB** as well as the initial step-size, and several other parameters. The details of how to do this can be found in the help systems for either software package.

4.4 Instability

Recall that Euler’s method is a recursive application of

$$y_{n+1} = y_n + h f(t_n, y_n)$$

where $t_{n+1} = t_n + h$ and $0 \leq N - 1$.

Note also that the logistic equation is given by

$$y' = ry \left(1 - \frac{y}{K}\right) = ry(1 - y) \quad \text{when } K = 1$$

with $y(0) = y_0$ and $r > 0$, $K > 0$. We know that the resulting solution is a smooth monotonically increasing, or decreasing, function approaching $K = 1$ (see Chapter 3).

What we now consider is how well Euler's method predicts or approximates the logistic equation solution as h (the step size) increases. Applying Euler's method to the logistic equation (with $K = 1$) gives successive approximations for y_1, y_2, \dots with

$$y_{n+1} = y_n + hry_n(1 - y_n) \quad n = 0, 1, 2, \dots \quad (4.1)$$

Note here that Euler's equation is a difference equation estimating a differential equation. Furthermore, this equation is identical to that considered in Section 3.6 (to describe discrete population growth) if rh of (4.1) is set equal to parameter r of that section, and the carrying capacity is taken as $K = 1$.

Applying the results of that section to this case, for small rh the estimation approaches the analytic solution smoothly; however, for a small increase in rh , the estimation will oscillate about the analytic solution. And for some further increase, the numerical solution predicts chaos.

This means that when solving the logistic equation with parameter r using Euler's method, if the parameter rh is sufficiently small then the method will converge to the analytic solution. If not, then the method will never converge and the sequence of generated values may oscillate or behave chaotically. For any fixed r , there is not one single value for h (the step size of the method) that will ensure convergence to the analytic solution in all cases. Choice of the step size would need to depend on the parameter r , as otherwise the solution would not necessarily be stable, regardless of how small the step size is taken.

4.5 Discussion

Thus numerical methods need to be chosen carefully for accurate results. Euler's method, for example, while simple to use, is not a good choice for situations in which high accuracy is desired. Likewise, it brings us to the realisation that it would be very valuable to have some prior knowledge of expected behaviour/results in order to interpret and evaluate numerical solutions correctly.

To this end we have already developed some tools, and will develop others, both analytical and graphical, to establish aspects concerning the behaviour of the solution. These can then be compared with the numerical results. When using computational methods, it is commonplace to run experiments determining values which are already known exactly (through some theoretical or experimental means) in order to establish consistency with expected results.

Another essential aspect of generating numerical solutions to be borne in mind is that a single solution to a single set of parameter values, and for a single set of initial values, may impart very little information about the general behaviour of a system. Analytical tools are essential in providing a framework for choosing which numerical solutions to generate in order to gain an understanding of the system dynamics. In Chapter 6, in particular, we see how well analytical and numerical methods can combine to provide this information.

Summary of skills developed here:

- Understand the general principles of applying numerical methods, and how the process contributes towards generating errors in computed solutions.
- Understand the notion of a stable or unstable numerical scheme.
- Understand the difference between an exact and an estimated solution.
- Know how to choose a numerical scheme offered by Maple.

4.6 Exercises for Chapter 4

4.1. Round-off errors. A simple example can illustrate how the round-off errors accumulate to provide inaccuracy. Using Maple set

$$z = (x + 1)^{12} - (4x + 3)^6.$$

- Set $x = 1 + \sqrt{3}$ and evaluate z .
- Simplify z and compare this solution with that in (a).

Their difference arises through round-off errors which accumulate from the calculations required for the evaluation in (a), compared with the analytic approach of (b) in which no numerical calculations occur.

Some useful Maple commands are:

```
x:=(1+sqrt(3));, evalf(z); and simplify(z).
```

4.2. Numerical schemes. A variety of numerical methods can be used to solve the same equation and thus give an indication of their differences, in terms of estimation, compared with the analytical solution. Consider the simple IVP

$$\frac{dy}{dt} = y, \quad y_0 = y(0) = 1.$$

- Using Maple (which uses a symbolic approach) find and plot the analytical solution.
- Now create further plots choosing particular numerical methods as follows:

- Using the code

```
plot2:=DEplot(eqnl, {[y(0)=1]}, [y(t)],
t=0..4,y=0..maxy,iterations=5,
stepsize=0.1, arrows=NONE, linecolour=blue,
method=classical[foreuler]):
```

- and, as above, with

```
> plot3:=DEplot(...,
linecolor=red, method=classical[heunform]):  
> plot4:=DEplot(...,
linecolor=green, method=classical[rk4]):
```

Display these on the same axes to see how they differ from the analytical solution.

- Try changing the step size in the methods above and compare the results.

4.3. Numerical and symbolic solutions. One can get an idea of how the error between the analytical and numerical solutions grows by plotting that error. To this end, for the IVP in Question 2, use the following code in Listing 4.3 to examine these errors, comparing a variety of time steps and methods. Notice the rate of growth in the error functions as t increases.

Listing 4.3: Maple code: c_cn_num_sym.txt

```
> restart:with(plots):
> lasttime:=10:
> timelist:=seq(i,i=1..lasttime):
> deq:=diff(y(x),x)=y(x):
> init:=y(0)=1:
> Digits:=20:
# three methods of solution
>
> ans1:=dsolve({deq,init},y(x),type=numeric,
    method=classical[foreuler],output=array([timelist]),stepsize=0.1):
> ans2:=dsolve({deq,init},y(x),type=numeric,
    method=classical[heunform],output=array([timelist]),stepsize=0.1):
> ans3:=dsolve({deq,init},y(x),type=numeric,
    method=classical[rk4],output=array([timelist]),stepsize=0.1):
# compare the solutions using the difference between solutions
>
> plot1:=plot([seq([ans1[2,1][i,1],ans3[2,1][i,2]-ans1[2,1][i,2]],i=1..lasttime)],
    style=line):
> plot2:=plot([seq([ans2[2,1][i,1],ans3[2,1][i,2]-ans2[2,1][i,2]],i=1..lasttime)],
    style=line,colour=blue):
> display(plot1,plot2,view=[0..lasttime,0..10]);
# compare the numerical solutions with the analytical solution
>
> plot3:=plot([seq([ans1[2,1][i,1],exp(i)-ans1[2,1][i,2]],i=1..lasttime)],
    style=line,colour=red):
> plot4:=plot([seq([ans2[2,1][i,1],exp(i)-ans2[2,1][i,2]],i=1..lasttime)],
    style=line,colour=blue):
> plot5:=plot([seq([ans3[2,1][i,1],exp(i)-ans3[2,1][i,2]],i=1..lasttime)],
    style=line,colour=green):
> display(plot3,plot4,plot5,view=[0..lasttime,0..10]);
```

4.4. Discretisation and round-off errors. The number of steps chosen for a numerical method will also have an impact on the accuracy of the solution, with an increase in the number of steps reducing the discretisation error. However, when the step size approaches the accuracy of the machine (the smallest number representable by the machine) the error produced by the increased number of round-off errors becomes dominant, and the combined error increases reducing the accuracy of the solution. To illustrate this we can use the same IVP as above (Question 2) and set the machine accuracy through the variable `Digits`.

- Using the Maple code as given below, in Listing 4.4 compare the errors in the RK4 method (fourth-order Runge-Kutta method) as the step size decreases. Use step sizes of 0.1, 0.001 and 0.0005 and set the machine accuracy (`Digits`) to 10.
- What happens when the step size is close to the maximum accuracy of the machine? To see this compare the output with the machine accuracy (through `Digits`) set to 10 and set to 20.

Listing 4.4: Maple code: c_cn_roundoff.txt

```
> restart:with(plots):
> lasttime:=5:Digits:=10:
> timelist:=seq(i,i=1..lasttime):
> deq:=diff(y(x),x)=y(x):
> init:=y(0)=1:
# Analytical solution
```

```

>
> ans0:=dsolve({deq,init},y(x));
# Numerical solutions with different stepsizes
>
> ans1:=dsolve({deq,init},y(x),type=numeric,
    method=classical[rk4],output=array([timelist]),stepsize=0.1):
> ans2:=dsolve({deq,init},y(x),type=numeric,
    method=classical[rk4],output=array([timelist]),stepsize=0.001):
> ans3:=dsolve({deq,init},y(x),type=numeric,
    method=classical[rk4],output=array([timelist]),stepsize=0.0005):
# Compare the solutions with the analytical solution
>
> plot1:=plot([seq([ans1[2,1][i,1],exp(i)-ans1[2,1][i,2]],i=1..lasttime)],
    style=line,colour=red):
> plot2:=plot([seq([ans2[2,1][i,1],exp(i)-ans2[2,1][i,2]],i=1..lasttime)],
    style=line,colour=blue):
> plot3:=plot([seq([ans3[2,1][i,1],exp(i)-ans3[2,1][i,2]],i=1..lasttime)],
    style=line,colour=green):
> display(plot1,plot2,plot3);
> display(plot2,plot3);

```

4.5. MATLAB comparison of methods. Consider the differential equation and initial condition

$$\frac{dy}{dt} = 3y, \quad y(0) = 1.$$

- (a) Solve this using the MATLAB standard `ode45` function and plot the results. Also plot the exact solution $y = e^{3t}$.
- (b) The code in Listing 4.5 shows how to write a function to solve the differential equation on $[0, 1]$, using Euler's method, with $N = 10$ equally spaced time-steps $h = 0.1$. Using this code, compare on the one plot the solutions graphically for $h = 0.1$, $h = 0.05$ and $h = 0.01$ and also plot the exact solution.

Listing 4.5: MATLAB code: `c_cn_eulersolve.m`

```

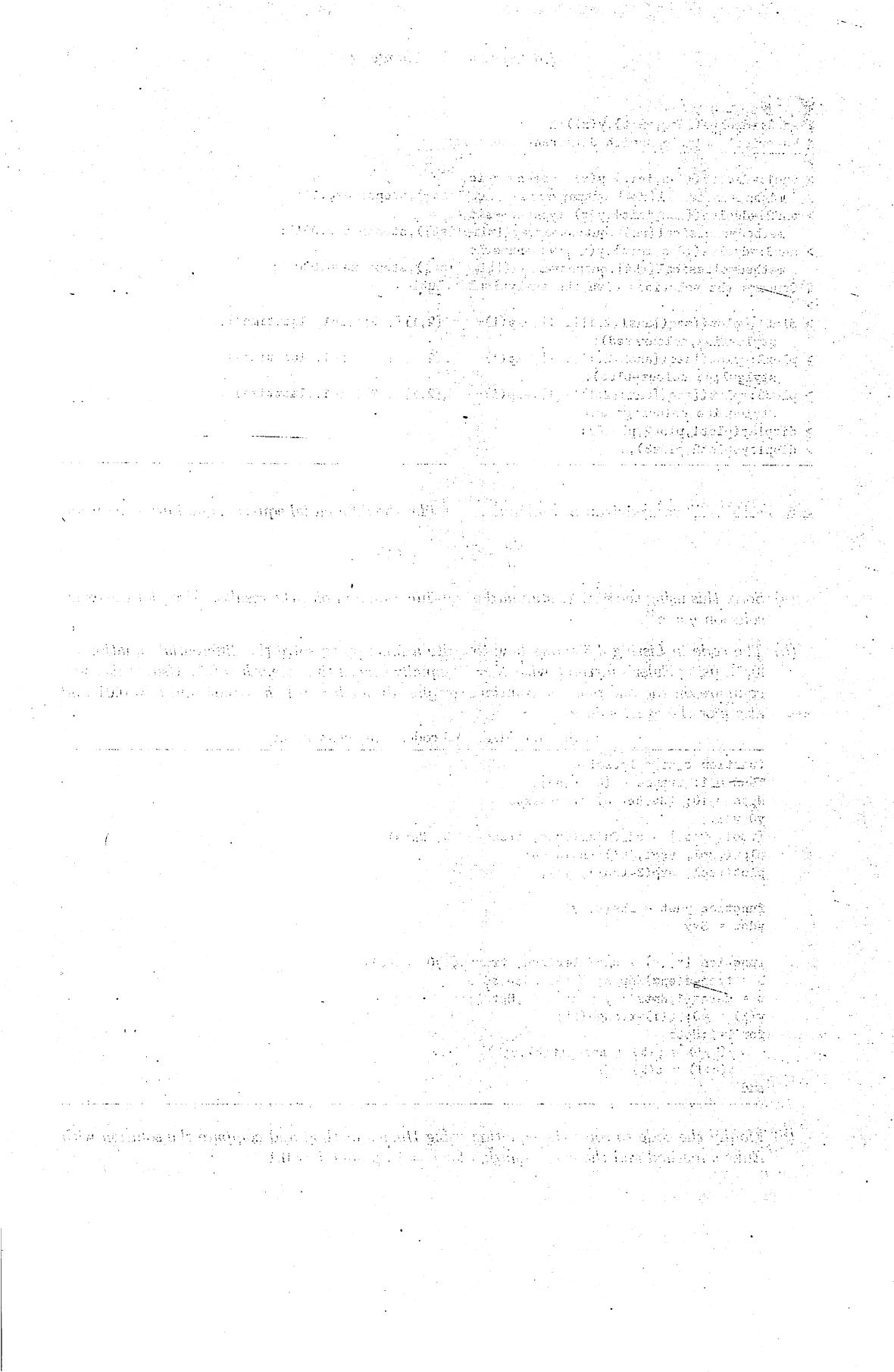
function c_cn_eulersolve
tend = 1; trange = [0, tend];
Npts = 10; %number of time-steps
y0 = 1;
[tsol, ysol] = odeEuler(@rhs, trange, y0, Npts);
plot(tsol, ysol,'b'); hold on;
plot(tsol, exp(3*tsol),'g');

function ydot = rhs(t, y)
ydot = 3*y;

function [t, y] = odeEuler(fcn, trange, y0, Npts)
h = trange(end)/Npts; % the step size
t = zeros(1,Npts); y = zeros(1,Npts);
y(1) = y0; t(1)=trange(1);
for k=1:Npts
    y(k+1) = y(k) + h*fcn(t(k),y(k));
    t(k+1) = t(k) + h;
end

```

- (c) Modify the code to solve the equation using Huen's method and compare the solution with Euler's method and the exact solution for $n = 10$ points, $h = 0.1$.



Chapter 5

Interacting population models

Interacting population models are relevant where two or more populations depend on each other. We study, in detail, four examples of interacting populations: (i) an epidemic model, (ii) a predator-prey interaction, (iii) a competing species interaction, and (iv) a model of a battle between two opposing groups. In this chapter, we concentrate on formulating the differential equation model which is governed by two simultaneous first-order differential equations. We solve the differential equations numerically using Maple. In Chapter 6, we return to the models and develop analytic techniques which give more general insights into the models developed here.

5.1 Introduction

Ecological systems may be extraordinarily complex — an inter-related system of plants and animals, predators, prey, flowering plants, insects, parasites, pollinators, seed dispersing animals, etc. In such systems there is a constant stream of arrivals and departures involving time periods of millions of years. New species evolve or arrive, others decline to extinction or migrate. Human interference has impacted hugely on most of the world's ecosystems, particularly over the last 200 years, with one seemingly insignificant species extinction able to spark a cascade of effects throughout the trophic levels.

Interacting populations

Large numbers of one species may be unaffected by others; however, in some instances removing, introducing or modifying one resource or species through (for example) harvesting or poisoning may have wide ranging ramifications for the system. Evidence suggests that, typically, communities with many interacting species have greater stability than those comprising much simpler systems. That is, while rainforests are stable, cultivated land and orchards are relatively unstable and the populations of species in laboratory controlled predator-prey systems undergo large oscillations.

The relationships between the species within a system are often highly nonlinear, so that it is extremely difficult to establish, with certainty, a precise mathematical model describing the processes involved. However, there is clearly a need to understand these systems, or aspects of them. To this end we develop simple mathematical models where the independent variable is time and the dependent variables are the densities (numbers per area) of the various different interacting populations. We include case studies of interactions where these simple models, or extensions of them, appear to reflect the main processes involved.

The most obvious examples come from nature, where different species interact with each other. Examples of populations which interact include different age groups within a population, predator-prey interactions, the spread of a disease between population groups and battles between opposing groups.

One type of interaction, which occurs within a single species, is the interaction between those who are infected with a *disease* and those who are not. This effectively divides the population into two separate sub-populations, where the growth or decline of one sub-population is affected by the size of the other sub-population. We study, in detail, a simple model for the growth and then decline of the number of individuals in a population infected with a disease, and compare the model predictions with data recorded during a flu epidemic in a boarding school.

There are many different types of interactions between populations in different trophic levels. One such interaction, where some species use other species as their food supply, is the *predator-prey* interaction. We study the simplest example of it in this chapter. Another interaction process between populations on the same trophic level is that of *competing species*, where two or more populations compete for a limited resource, such as food or territory. We model this system in a manner very similar to that developed for the predator-prey system.

The fourth example we study is that of a *battle* between two opposing groups, such as may occur between two insect or human populations. The rate at which soldiers are wounded, or killed, in a battle depends largely on the number of enemy soldiers. We focus on a very simple human war model for aimed fire, where a soldier aims at a target with a given probability of hitting it. The model is compared with an actual battle in World War II for which daily records of the number of casualties were kept.

Modelling assumptions and approach

Different populations are expected to have different birth and death rates. For some populations the size of another population will affect these parameters, and the populations are then said to interact with each other.

In each of the examples which follow, we make some simplifying assumptions and then build the mathematical model on these. We assume that the populations are sufficiently large, so that we can neglect random differences between individuals. Furthermore, we also assume the growth to be continuous, rather than discrete, over time. The model we derive for the rate of change of each population over time is based upon the input-output principle of the balance law: the compartmental model technique. This approach leads to two, or more, simultaneous differential equations, or a system of equations.

Models can describe the number of individuals (population size), as will be the case for the epidemic and battle models we develop. Alternatively, the population density, number per unit area, can be modelled as in the predator-prey and competing species models which follow. For both, the approach is the same.

Systems of differential equations

Many processes are described by more than one differential equation. When these equations need to be satisfied simultaneously the set of equations is known as a *system of differential equations*. Although systems can comprise many differential equations and many unknowns, in this book we consider mainly systems of first-order equations where the number of unknowns is the same as the number of equations in the system. This ensures a unique solution to the system when the initial conditions are specified.

The system of equations is known as a *dynamical system* if it allows prediction of future states given present and/or past states. In most of the models we develop, such as in the growth and decay processes examined in previous sections and the population models we develop here, this is the case.

We also develop systems in which the equations are *nonlinear*: that is, they include products of the dependent variables or their derivatives. While appearing simple, they do not all have analytic solutions and we rely on the approximate solution to these systems obtained by Maple or MATLAB, which employs numerical solvers to find them. Later, in Chapter 7, we develop some theory about linearisation which allows us to predict the behaviour of the system by considering linear approximations to the nonlinear equations.

The systems of equations may be *coupled*, which implies that their solutions are interdependent. For example, in the case of two equations $x'(t) = F(x, y, t)$ and $y'(t) = G(x, y, t)$, if the solution of x' depends on the value of y and the solution of y' depends on x , then the equations are coupled. Alternatively, as was the case in Section 2.7 in the example of drug assimilation, the first equation could be solved independently of the second, and this system is said to be *uncoupled*.

5.2 An epidemic model for influenza

We develop a model to describe the spread of a disease in a population and use it to describe the spread of influenza in a boarding school. To do so the population is divided into three groups: those susceptible to catching the disease, those infected with the disease and capable of spreading it and those who have recovered and are immune from the disease. Modelling these interacting groups leads to a system of two coupled differential equations.

Background

Over the centuries, there have been dramatic examples of how epidemics of various diseases have had a significant effect on the human population. One of the most well known is the Black Death in Europe in the fourteenth century. Today epidemics are still prevalent, the most notable being possibly AIDS and the Ebola virus. If we can understand the nature of how a disease spreads through a population, then we are better equipped to contain it through vaccination or quarantine. Or in the case of the biological control of pests we may wish to determine how to increase the spread of the disease (for example, myxomatosis or calicivirus in rabbits) so as to find an efficient way of reducing the population. Unfortunately, humans themselves have been subjected to this means of ‘control’. In colonial times, the spread of European diseases, such as measles and smallpox, had a disastrous impact on certain indigenous populations who had no resistance to them.

Many diseases are spread by infected individuals in the population coming into close contact with susceptible individuals. These include influenza, measles, chickenpox, glandular fever and AIDS. On the other hand, malaria is transmitted through a host, a mosquito, which carries the disease from individual to individual. Certain diseases are more contagious than others. Measles and influenza are highly contagious, whereas glandular fever is much less so. Many diseases, such as mumps and measles, confer a lifelong immunity; however, influenza and typhoid have short periods of immunity and can be contracted more than once.

There are some natural definitions which we require in order to proceed with our modelling. The *incubation period* of a disease is the time between infection and the appearance of visible symptoms. This should not be confused with the *latent period*, which is the period of time between infection and the ability to infect someone else with the disease. The latent period is shorter than the incubation period so that an individual can be spreading the disease, unaware of having it. For measles the incubation period is approximately 2 weeks and the latent period is approximately 1 week. Below we consider a simple mathematical model for a flu (influenza) epidemic at a boarding school over a period of about 15 days. For this period it is reasonable to assume that reinfection does not occur.

Model assumptions

When considering a disease, the population can be divided into distinct classes: susceptibles $S(t)$ and contagious infectives $I(t)$, where t denotes time. The susceptibles are those liable to catch the disease, while the contagious infectives are those infected with the disease who are capable of communicating it to a susceptible. There are also those who have recovered and who are now immune from further infection of the disease.

Initially, we make some assumptions and then build the model based on them.

- We assume the populations of susceptibles and contagious infectives are large so that random differences between individuals can be neglected.
- We ignore births and deaths in this model and assume the disease is spread by contact.
- We neglect the latent period for the disease, setting it equal to zero.
- We assume all those who recover from the disease are then immune (at least within the time period considered).
- We also assume that, at any time, the population is homogeneously mixed, i.e. we assume that the contagious infectives and susceptibles are always randomly distributed over the area in which the population lives.

Formulating the differential equations

We need to start with an input-output compartment diagram and then describe the rate of change in the number of susceptibles and contagious infectives with word equations. The following example illustrates this process.

Example 5.1: Construct a compartmental diagram for the model and develop appropriate word equations for the rates of change of susceptibles and contagious infectives.

Solution: The only way the number of susceptibles can change is the loss of those who become infected, since there are no births and none of those who become contagious infectives can become susceptibles again. The number of infectives changes due to the susceptibles becoming infected and decreases due to those infectives who die, become immune or are quarantined. The latter cannot become susceptibles again (from the assumptions made). This is illustrated in the compartmental diagram of Figure 5.1.

The appropriate word equations are

$$\begin{aligned} \left\{ \begin{array}{l} \text{rate of} \\ \text{change in no.} \\ \text{susceptibles} \end{array} \right\} &= - \left\{ \begin{array}{l} \text{rate} \\ \text{infected} \\ \text{susceptibles} \end{array} \right\} \\ \left\{ \begin{array}{l} \text{rate of} \\ \text{change in no.} \\ \text{infectives} \end{array} \right\} &= \left\{ \begin{array}{l} \text{rate} \\ \text{infected} \\ \text{susceptibles} \end{array} \right\} - \left\{ \begin{array}{l} \text{rate} \\ \text{have recovered} \\ \text{infectives} \end{array} \right\} \\ \left\{ \begin{array}{l} \text{rate of} \\ \text{change in no.} \\ \text{recovered} \end{array} \right\} &= \left\{ \begin{array}{l} \text{rate} \\ \text{infectives} \\ \text{have recovered} \end{array} \right\} \end{aligned} \quad (5.1)$$

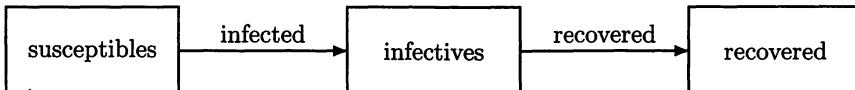


Figure 5.1: Input-output diagram for the epidemic model of influenza in a school, where there is no reinfection.

Note that the term on the RHS of the equation for the susceptibles is exactly the same term as the first term on the RHS for the infectives.

To model the total rate of susceptibles infected, first consider the susceptibles infected by a single infective. It is evident that the greater the number of susceptibles, then the greater the increase in the number of infectives. Thus the rate of susceptibles infected by a single infective will be an increasing function of the number of susceptibles. For simplicity, let us assume that the rate of susceptibles infected by a single infective is directly proportional to the number of susceptibles, with a constant of proportionality β . If we denote the number of susceptibles at time t by $S(t)$ and the number of infectives at time t by $I(t)$, then the rate susceptibles are infected by a single infective is $\beta S(t)$. The total rate of susceptibles infected by $I(t)$ infectives is obtained by multiplying $\beta S(t)$ by the number of infectives, and thus

$$\left\{ \begin{array}{l} \text{rate} \\ \text{susceptibles} \\ \text{infected} \end{array} \right\} = \beta S(t)I(t). \quad (5.2)$$

The constant β is called the *transmission coefficient* or infection rate ¹.

¹An alternative assumption, rather than is to multiply by the *proportion* of the population who are infectives,

We must also account for those removed from the system, in this case, those who have recovered from the disease. More generally, the removed can also consist of fatalities due to the disease, those who become immune to the disease, and those infectives who are quarantined. The number of infectives removed in the time interval should not depend in any way upon the number of susceptibles, but only on the number of infectives. We assume that the rate of infectives recovered is directly proportional to the number of infectives. We write

$$\left\{ \begin{array}{l} \text{rate} \\ \text{infectives} \\ \text{recovered} \end{array} \right\} = \gamma I(t) \quad (5.3)$$

where γ is a positive constant of proportionality, called the *recovery rate*, or more generally, the removal rate. The rate γ is a per-capita rate. Its reciprocal, γ^{-1} , can be identified with the residence time in the infective compartment, i.e. the mean time that an individual is infectious. For influenza the infectious period is typically 1–3 days.

Summarising,

- the rate at which susceptibles become infected is proportional to both the numbers of infectives and susceptibles,
- the rate at which infectives recover and are removed is proportional to the number of infectives only.

The rate of change in the number of susceptibles in the time interval is given by dS/dt and the rate of change in the number of infectives is given by dI/dt . Making use of equations (5.2) and (5.3), the population word equations (5.1) become

$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I, \quad \frac{dR}{dt} = \gamma I, \quad (5.4)$$

subject to the initial conditions $S(0) = s_0$, $I(0) = i_0$ and $R(0) = 0$. Equations (5.4), a coupled system of nonlinear differential equations, were originally derived by Kermack and McKendrick in 1927 (Kermack and McKendrick, 1927). Since the R variable does not appear in the first two differential equations the first two differential equations can be studied as a system on its own.

Numerical solution

We can solve the differential equations (5.4) by a numerical method (e.g. Euler's method, Runge-Kutta methods, or Maple built-in solvers). Using some data from an influenza epidemic at a British boarding school, the results are given in Figure 5.2. The Maple code from which the figure was generated is given in Listing 5.1 and the MATLAB code is given in Listing 5.2.

Listing 5.1: Maple code: c_pe.epidemic.txt

```
> restart:with(plots):with(DEtools):
> unprotect(gamma); gamma:='gamma':
> interface(imaginaryunit=i); I:='I':
> beta:=2.8*10^(-3): gamma:=0.44:
> de1 := diff(S(t),t)=-beta*S(t)*I(t);
> de2 := diff(I(t),t)=beta*S(t)*I(t)-gamma*I(t);
```

giving a term $\beta S(t)I(t)/N(t)$, where $N(t)$ is the total population size. It turns out this makes no difference when N is constant as the constant can be absorbed into the constant β .

```
> inits:=[S(0)=762,I(0)=1];
> myopts:=stepsize=0.1,arrows=NONE:
> plot1:=DEplot([de1,de2],[S,I],t=0..30,[inits],scene=[t,S],linecolour=black,myopts):
> plot2:=DEplot([de1,de2],[S,I],t=0..30,[inits],scene=[t,I],linecolour=red,myopts):
> display(plot1,plot2);
```

Listing 5.2: MATLAB code: c_pe.epidemic.m

```
function c_pe_epidemic
global beta gamma;

tend = 15;%se the end time to run the simulation
u0 = [761; 1]; % set initial conditions as a column vector
beta=2.0*10^(-3); gamma=0.44;
[tsol, usol] = ode45(@rhs, [0, tend], u0);
Ssol = usol(:, 1); Isol = usol(:, 2);
plot(tsol, Ssol, 'r'); hold on; plot(tsol, Isol, 'b');

function udot = rhs(t, u)
global beta gamma;
S=u(1); I=u(2);
Sdash = -beta*S*I;
Idash = beta*S*I - gamma*I;
udot = [Sdash; Idash];
```

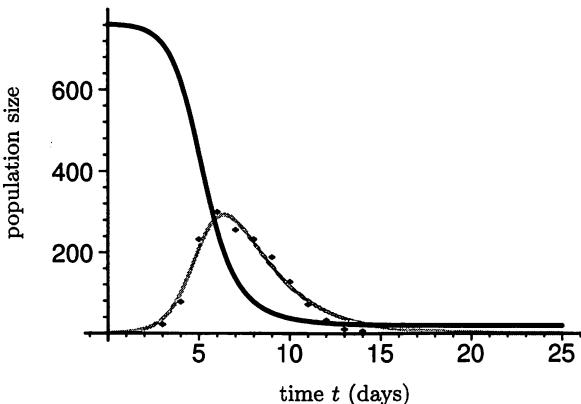


Figure 5.2: Numerical solution of the differential equations for an influenza epidemic in an English boarding school in 1978. The epidemic was started by one infective, thus $i_0 = 1$ and the total number of susceptibles was $s_0 = 762$. The values of the infection rate and removal rate were estimated as $\beta = 2.18 \times 10^{-3}$ susceptibles $^{-1}$ day $^{-1}$ and $\gamma = 0.44$ day $^{-1}$ respectively. The black dots correspond to the original data with the black line, the susceptibles and the grey line, the infectives. (Data from (Murray, 1990).)

The data appear to agree well with the model's predictions. Notice that the number of infectives starts small and increases substantially over 6 days, then decreasing gradually. What is happening is that the number of susceptibles is being 'used up'. Thus, in the latter stages of the epidemic there is a much smaller chance of any given infective coming into contact with someone who has not yet been infected.

Limitations of the model

There are a number of different variations of the basic model to consider: for example, the effect of births which continually introduce more susceptibles into the population, or diseases without immunity, where infectives become susceptible again after removal.

Births will provide a source of additional susceptibles and this can lead to oscillations in the number of infectives. Indeed, before the days of vaccinations, for diseases such as measles regular outbreaks occurred, every 2 years in some countries, and every year in others.

While some diseases confer permanent immunity, (e.g. measles, chickenpox) many others do not (e.g. colds, influenza over a longer period than 25 days). This can easily be modelled, by allowing the infectives to become susceptibles again. Thus the compartmental diagram Figure 5.1 changes as the output from the contagious compartment becomes the input for the susceptible compartment. Another implicit assumption made here is that the rate of new infections is directly proportional to the product $S(t)I(t)$. If the number of infectives became a large proportion of the population then it is likely that there would be a maximum limit to the rate of new infections since there is a limited number of contacts that could be made in any given time interval. The most suitable way to take this into account is by using a ‘contact function’, $\beta = pc(N)/N$ where $c(N)$ is the rate of contacts between individuals, which is dependent on the population size (or population density). See Roberts and Heesterbeek (1993) and Diekmann and Heesterbeek (2000) for an in-depth discussion.

The basic reproduction number

From exploring the numerical solution we observe that the dynamics has a threshold behaviour where, if the initial number of susceptibles, s_0 , is below a certain amount, then the number of infectious $I(t)$ decreases. On the other hand, if s_0 is greater than the threshold then $I(t)$ increases before decreasing again. This appears to be independent of the initial number of infectious individuals, i_0 . We can determine this threshold quantity by defining an important quantity called the *basic reproduction number*, denoted by R_0 .

The basic reproduction number is defined as

the number of new secondary infections resulting from a single infectious individual placed in a completely susceptible population, over the time that individual is infectious.

If $R_0 < 1$ we would expect the disease outbreak to die out ($I(t)$ to decrease) and if $R_0 > 1$ then it would increase initially.

Using the above definition of R_0 we can refer back to our model differential equations and determine a simple formula for R_0 for this SIR model. The instantaneous rate of new infections for the population was βSI . Thus the rate of new infections caused by a single infectious individual is $\beta s_0 \times 1$, where we have set $I = 1$ and $S = s_0$, the initial number of susceptibles. To obtain the number of new infections we multiply this rate by the average time that an individual is infectious for. This time is given by the residence time in the infectious compartment, γ^{-1} (see Section 2.2). Thus we can define R_0 as the quantity

$$R_0 = \frac{\beta s_0}{\gamma}. \quad (5.5)$$

The basic reproduction number is thus a measure of how rapidly an infectious disease spreads through a population when $R_0 > 1$. Some typical estimates of R_0 for some common infectious diseases (in the pre-vaccination era) include $R_0 \approx 3-4$ for influenza, $R_0 \approx 16-18$ for measles, and whooping cough, $R_0 \approx 4$ for smallpox and $R_0 \approx 10-12$ for chickenpox (see Keeling and Rohani (2008) and Anderson and May (1991)).

From the differential equation for I ,

$$\frac{dI}{dt} = \beta SI - \gamma I = \gamma I \left(\frac{\beta S}{\gamma} - 1 \right)$$

we also see that $dI/dt > 0$ only if $\beta S/\gamma > 1$ and $dI/dt < 0$ only if $\beta S/\gamma < 0$. Putting $S = s_0$, initially, then this also gives that $dI/dt > 0$ if $R_0 > 1$ and $dI/dt < 0$ if $R_0 < 1$.

As an application of the basic reproduction number we consider the vaccination of a population. Assuming we could instantaneously vaccinate a proportion of a population, what proportion would result in the eradication of the infectious disease?

This is easy to answer using the concept of the basic reproduction number R_0 . Since $R_0 = \beta s_0 / \gamma$ then if we vaccinate a proportion p of the population of susceptibles then this means that the basic reproduction number changes to

$$R_v = \frac{(1-p)s_0}{\gamma} = (1-p)R_0$$

since there is now only $(1-p)s_0$ susceptibles who may potentially catch the disease. Setting $R_v < 1$ for eradication, and solving for p we obtain the simple formula

$$p > 1 - \frac{1}{R_0}. \quad (5.6)$$

For smallpox, for example, where $R_0 \approx 4$, we calculate $p \approx 75\%$ of the population, but for measles, in unvaccinated communities, where typically $R_0 \approx 15$ then $p \approx 93\%$. It is clear from this calculation why smallpox was chosen to be targeted worldwide for eradication.

Endemic diseases

Endemic diseases persist in the population; they are characterised by outbreaks which occur from time to time. There are many examples, including childhood diseases such as measles and chickenpox. These models are usually studied on a larger scale, for example, a population the size of a city or a country.

To model an endemic disease we need to include in the model some of the underlying population dynamics, i.e. we need to include the effects of natural births and deaths. The following model is a relatively simple model for exploring an endemic disease; it is a variant of the SIR model. A compartment diagram is given in Figure 5.3.

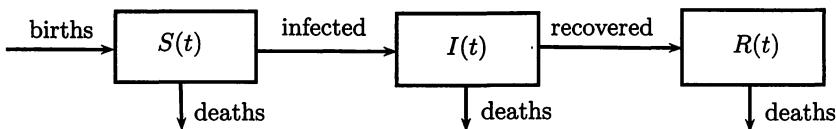


Figure 5.3: Input-output diagram for the epidemic model of influenza in a school, where there is no reinfection.

Let a and b denote the natural per-capita death rate and birth rate of population, where deaths are due to natural causes. The differential equations are

$$\begin{aligned} \frac{dS}{dt} &= bN - \beta SI - aS \\ \frac{dI}{dt} &= \beta SI - \gamma I - aI \\ \frac{dR}{dt} &= \gamma I - aI \end{aligned}$$

where $N(t) = S(t) + I(t) + R(t)$. If we add the three differential equations together, we obtain a differential equation for $N(t)$,

$$\frac{dN}{dt} = (b - a)N.$$

If $b = a$ then the population remains constant. This is sometimes a useful assumption to make provided the time-scale over which the model is applied is sufficiently short that a constant population is a reasonable assumption. A numerical solution, using MATLAB or Maple, is given in Figure 5.4. The number of infectives has initially similar dynamics to that in Figure 5.2 but after some time the number of infectives rebounds instead of dying out.

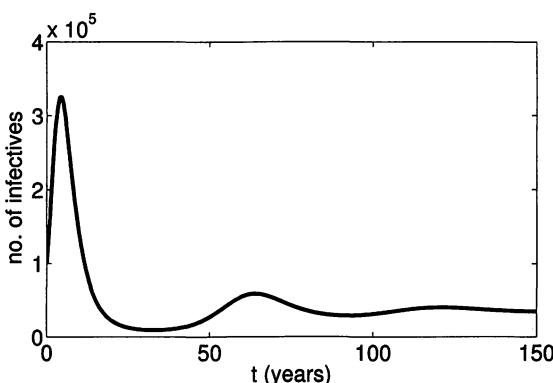


Figure 5.4: Numerical solution of the differential equations for an endemic disease for an infectious disease in a hypothetical population of $N = 10^6$. Parameter values used were $\beta = 10^{-6}$ susceptibles $^{-1}$ day $^{-1}$, $\gamma = 1/3$ years $^{-1}$, $b = a = 1/50$ years $^{-1}$ with initial populations $S(0) = 9 \times 10^5$ and $I(0) = 10^5$.

Initially there are sufficient susceptibles for the infectious disease to spread in the population (i.e. $R_0 > 1$). After a time the number of susceptibles falls so that there are insufficient susceptibles available to sustain the increase (i.e. a single infective cannot infect more than one susceptible while they are still infectious), and the number of infectives begins to fall. However, due to births of new susceptibles, there will come a time when the number of susceptibles again reaches a critical value so that the number of infectives begins to rise again, thus causing a new outbreak. In this model we obtain damped oscillations, and the number of infectives tends to a steady-state. However, in practice, the transmission coefficient β can be seasonally dependent and this can cause sustained oscillations rather than damped oscillations. This additional oscillatory forcing can sometimes also produce chaotic behaviour. This is discussed in Keeling and Rohani (2008), and see also Roberts and Tobias (2000) for an example of this behaviour for recurrent epidemics of measles in New Zealand.

Discussion

One question we might ask is whether a rapid increase in the number of infectives is always followed by a decrease? Also, by adjusting any of the parameters, could we limit the increase or even prevent it? Changing parameters could correspond to, for example, adopting certain vaccination strategies.

To answer these questions it is useful to gain more qualitative information about this epidemic model. We would like to be able to say what happens for any values of the parameters. An exact solution of the simultaneous equations, however, is not easily obtained because the differential equations are nonlinear. An alternative approach is to use the chain rule to eliminate time and reduce the pair of differential equations to a single first order differential equation, from which some insight can be gained. This analysis is covered in the next chapter.

There are a number of extensions of the basic SIR infectious disease model. Some of these are developed in the exercises. One extension is to incorporate a latent period which is neglected in the basic SIR model. A latent period is the time from contact to when an individual is infectious to others. The simplest way to model the latent period is to include an additional compartment, with population size $E(t)$, consisting of those *exposed* who are infected but not yet infectious. The exposed then become infectious at a constant per-capita rate. This leads to an additional differential equation in the system. Such models are known as SEIR models.

Further extensions include continuous vaccination, where susceptibles move into a vaccinated compartment; incorporating population growth into the equations for longer term endemic diseases; sexually transmitted diseases; and disease spread by an animal agent, such as malaria spread by mosquitoes.

A classic reference in the field of mathematical epidemiology is Anderson and May (1991). Murray (1990) discusses the same model developed above, and the influenza epidemic used to validate the model. Also considered are the Black Plague, and rabies in foxes, amongst other examples. Braun (1979) outlines an extension to the model for sexually transmitted diseases. For an introduction to both stochastic approaches to modelling human epidemics, see Daley and Gani (1999) and Allen (2003). For some further extensions of the basic models, see Grenfell and Dobson (1995). Keeling and Rohani (2008) give a comprehensive treatment of the modelling of infectious diseases in human and animal populations, including a chapter on vaccination and other means of controlling infectious diseases and a discussion of density-dependent transmission coefficients (where the rate of contacts between individuals is proportional to population size) and frequency-dependent transmission coefficients (where the rate of contacts is independent of the size of the population). These are particularly significant when the total population size changes with time. Diekmann and Heesterbeek (2000) discuss many fundamental ideas in infectious disease modelling and, in particular, how to compute the basic reproduction number for more complicated models that involve populations structured into several groups, such as age groups or social groups.

Summary of skills developed here:

- Formulate differential equations for variations on the two models presented here, such as, diseases with a latent period, continuous vaccination and diseases without immunity.
- Obtain a numerical solution for a system of differential equations.

5.3 Predators and prey

In the following section, we develop a simple predator-prey model for carnivores using the growth of a population of small insect pests which interact with another population of beetle predators. An example of a model for herbivores is examined in a case-study in Section 8.6, while models for parasitic interactions or cannibalism will be simple to derive from these given examples.

Background

There are several types of predator-prey interactions: that of herbivores which eat plant species, that of carnivores which eat animal species, that of parasites which lives on or in another species (the host), and that of cannibals which eat their own species, often an interaction between the adults and young.

One interesting example of a predator-prey interaction occurred in the late nineteenth century, when the American citrus industry was almost destroyed by the accidental introduction from Australia of the cottony cushion scale insect. To combat this pest its natural predator, the Australian ladybird beetle, was also imported, but this did not solve the problem and finally DDT was used to kill both predator and prey in a bid to eradicate the pest. Surprisingly, application of DDT to the orchards led to an increase in the scale insects, the original pest, suggesting that the use of pesticide is advantageous to the pest!

Model assumptions

We make a few preliminary assumptions on which to build the model.

- Initially we assume the populations are large, sufficiently large to neglect random differences between individuals.
- We ignore the effect of DDT initially, but modify the model later to incorporate its impact on the system.
- We also assume there are only two populations, the predator and the prey, which affect the ecosystem.
- We assume that the prey population grows exponentially in the absence of a predator.

Compartmental model

There are two separate quantities which vary with time: the number of prey and the number of predators. For populations of animals it is common to consider the population density, or number per unit area, rather than population size, as we do here. We need to develop two word equations, one for the rate of change of prey density and one for the rate of change of predator density.

Example 5.2: Determine a compartment diagram and appropriate word equation for each of the two populations, the predator and the prey.

Solution: The only inputs for each population are births and the only outputs are deaths. However, the prey deaths occur due to the predators capturing and eating them. This is illustrated in the input-output compartmental diagram of Figure 5.5. Here we distinguish between natural prey deaths and prey deaths due to predators. We also distinguish between natural predator births, occurring in the absence of prey, and the additional births and that would occur due to the prey

being eaten by the predators. The appropriate word equations are

$$\begin{aligned} \left\{ \begin{array}{l} \text{rate of} \\ \text{change of} \\ \text{prey} \end{array} \right\} &= \left\{ \begin{array}{l} \text{rate of} \\ \text{natural prey} \\ \text{births} \end{array} \right\} - \left\{ \begin{array}{l} \text{rate of} \\ \text{natural prey} \\ \text{deaths} \end{array} \right\} - \left\{ \begin{array}{l} \text{rate of} \\ \text{prey killed} \\ \text{by predators} \end{array} \right\}, \\ \left\{ \begin{array}{l} \text{rate of} \\ \text{change of} \\ \text{predators} \end{array} \right\} &= \left\{ \begin{array}{l} \text{rate of} \\ \text{predator} \\ \text{births} \end{array} \right\} - \left\{ \begin{array}{l} \text{rate of} \\ \text{natural predator} \\ \text{deaths} \end{array} \right\}. \end{aligned} \quad (5.7)$$

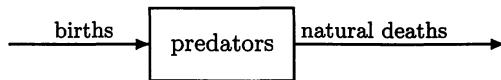
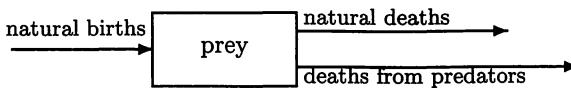


Figure 5.5: Input-output diagram for the 2-species predator-prey model.

Let us assume that the per-capita birth rate for the prey (the scale insect) is a constant b_1 . Remember that the per-capita birth rates give the rate of births from an individual prey. The rate of births for each *individual* scale insect does not depend on the predator density. Similarly the natural per-capita death rate of the scale insect is a constant a_1 . Alternatively, the per-capita death rate of prey due to being killed by the predators will depend on the predator density; the simplest assumption is to assume this per-capita rate is proportional to the predator density, a per-capita rate $c_1 Y$. The greater the density of predators, the more likely it is that an individual prey will be eaten. We assume a constant per-capita death rate for the predators (the ladybird beetles) independent of the prey density. The predator per-capita birth rate is more complicated. We assume the prey are an essential requirement for births of the predator, so the per-capita birth-rate for the predators will be the sum of a natural rate, which is constant, plus an additional rate which is proportional to the rate of prey killed. At any time the per-capita birth rate will increase with more food available and depend heavily on the amount of prey available.

Example 5.3: Let $X(t)$ denote the number of prey per unit area and $Y(t)$ the number of predators per unit area. Using the above assumptions and the word equations (5.7), formulate differential equations for the prey and predator densities.

Solution: First look at the constant per-capita terms, the prey births and predator deaths. (Subscripts 1 and 2 will be used for the parameters associated with X the prey, and Y the predator, respectively.)

Since the overall rates are the per-capita rates multiplied by the respective population densities, we can write,

$$\left\{ \begin{array}{l} \text{rate of} \\ \text{prey} \\ \text{births} \end{array} \right\} = b_1 X(t), \quad \left\{ \begin{array}{l} \text{rate of} \\ \text{prey} \\ \text{natural deaths} \end{array} \right\} = a_1 X(t), \quad \left\{ \begin{array}{l} \text{rate of} \\ \text{predator} \\ \text{deaths} \end{array} \right\} = a_2 Y(t). \quad (5.8)$$

For the prey deaths we denote the per-capita death rate as $c_1 Y(t)$, since it is proportional to the predator density, with c_1 as the positive constant of proportionality. Thus the rate at which prey

are eaten is given by $c_1 Y(t)X(t)$. The predator birth rate has a component which is proportional to this rate of prey eaten, so we write

$$\left\{ \begin{array}{l} \text{rate of} \\ \text{prey killed} \\ \text{by predators} \end{array} \right\} = c_1 Y(t)X(t), \quad \left\{ \begin{array}{l} \text{rate of} \\ \text{predator} \\ \text{births} \end{array} \right\} = b_2 Y + f c_1 Y(t)X(t), \quad (5.9)$$

where f is also a positive constant of proportionality.

Now substitute equations (5.8–5.9) into the word equations (5.7). We obtain the pair of differential equations

$$\frac{dX}{dt} = b_1 X - a_1 X - c_1 XY, \quad \frac{dY}{dt} = b_2 Y + f c_1 XY - a_2 Y.$$

We can combine some parameters. Let $\beta_1 = b_1 - a_1$, $-\alpha_2 = b_2 - a_2$ and $c_2 = fc_1$, then

$$\frac{dX}{dt} = \beta_1 X - c_1 XY, \quad \frac{dY}{dt} = c_2 XY - \alpha_2 Y. \quad (5.10)$$

where we assume that β_1 , a_2 , c_1 and c_2 are all positive constants.

This system of equations is called the *Lotka-Volterra predator-prey system* after the two mathematicians who first worked with them. The parameters c_1 and c_2 are known as *interaction parameters* as they describe the manner in which the populations interact. Since there are positive and negative terms on the RHS of each differential equation, we might anticipate that the populations could either increase or decrease. Further, these differential equations are coupled since each differential equation depends on the solution of the other. The differential equations are also nonlinear since they involve the product XY . One interpretation of the product XY is that it is proportional to the rate of encounters (contacts) between the two species.

We now have a model to which we can apply some simple checks to ascertain whether the equations behave as we might expect. For a two-species model we would expect that, in the absence of any predators, the prey would grow without bound (since we have not included any growth limiting effects other than the predators). Also, in the absence of prey, we would expect the predators to die out. The following example indicates that the model incorporates this behaviour.

Example 5.4: Check the Lotka-Volterra model in the limiting cases of prey with no predators, or predators with no prey.

Solution: Suppose there are no predators so that $Y = 0$. The equations then reduce to

$$\frac{dX}{dt} = \beta_1 X,$$

which is the equation for exponential growth. The prey grows exponentially.

If there are no prey then $X = 0$ and the equations reduce to

$$\frac{dY}{dt} = -\alpha_2 Y,$$

that is, exponential decay, which means that the predator population decreases exponentially and dies out.

Numerical solution

Although the equations may appear simple, they are not simple to solve. In fact we cannot find an analytic solution. However, to solve (5.10) numerically we can use Maple or MATLAB. We have chosen the values of the parameters β_1 , α_2 , c_1 and c_2 arbitrarily, in order to get a feel for how the model behaves. A sample numerical solution is illustrated in Figure 5.6 with the Maple code provided in Listing 5.3, and the MATLAB code provided in Listing 5.4.

Listing 5.3: Maple code: c_pe_predprey.txt

```
> restart; with(plots): with(DETools):
> beta[1]:=1.0: alpha[2]:=0.5: c[1]:=0.01: c[2]:=0.005:
> de1 := diff(X(t),t) = beta[1]*X(t)-c[1]*X(t)*Y(t);
> de2 := diff(Y(t),t) = -alpha[2]*Y(t)+c[2]*X(t)*Y(t);
> inits := [X(0)=200, Y(0)=80];
> myopts := stepsize=0.1, arrows=None:
> plot1 := DEplot([de1,de2], [X,Y], t=0..20,
    [inits], scene=[t,X], linecolor=red, myopts):
> plot2 := DEplot([de1,de2], [X,Y], t=0..20,
    [inits], scene=[t,Y], linecolor=blue, myopts):
> display(plot1,plot2);
```

Listing 5.4: Maple code: c_pe_predprey.m

```
function c_cp_predprey
global betai alpha2 c1 c2;

beta1=1.0; alpha2=0.5; c1=0.01; c2=0.005;
tend = 20;%se the end time to run the simulation
u0 = [200; 80]; % set initial conditions as column vector
[tsol, usol] = ode45(@rhs, [0, tend], u0);
Xsol = usol(:, 1); Ysol = usol(:, 2);
plot(tsol, Xsol, 'b'); hold on; plot(tsol, Ysol, 'r');

function udot = rhs(t, u)
global betai alpha2 c1 c2;
X = u(1); Y=u(2);
Xdot = betai*X - c1*X*Y;
Ydot = -alpha2*Y + c2*X*Y;
udot = [Xdot; Ydot];
```

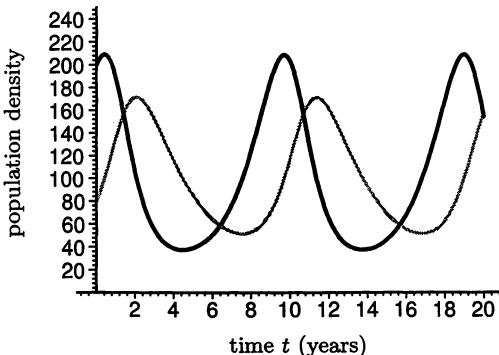


Figure 5.6: Sample numerical solution of the predator-prey equations using Maple. The black curve represents the prey and the grey curve the predator. Parameters here are chosen to illustrate the dynamics, and are not based on data from real populations: $\beta_1 = 1$, $\alpha_2 = 0.5$, $c_1 = 0.01$, $c_2 = 0.005$, with initial populations $x_0 = 200$ and $y_0 = 80$.

The prey population oscillates out of phase with the predator population. Further exploration with other values of the parameters appears to indicate that this model always leads to oscillating populations. In Chapter 6 we use some mathematical analysis to prove this.

We also note that the predator population oscillation lags behind the prey cycles. We show (Chapter 6) that the Lotka-Volterra equations (5.10) always predict oscillations and that the predator oscillations always lag behind those of the prey.

If DDT is sprayed on the crops we need to modify the equations since this represents a different mechanism for prey and predator deaths. We shall assume the per-capita death rates due to DDT are constant, with different per-capita rates p_1 and p_2 for the prey and predator, respectively. This means the DDT has an equal effect on each individual of a given species. The modified differential equations (5.10) including the additional terms $p_1X(t)$ for the prey and $p_2Y(t)$ for the predator are

$$\frac{dX}{dt} = \beta_1 X - c_1 XY - p_1 X, \quad \frac{dY}{dt} = c_2 XY - \alpha_2 Y - p_2 Y. \quad (5.11)$$

Note that the differential equations (5.11) are of the same form as the original system (5.10), with the positive constant β_1 replaced with $(\beta_1 - p_1)$ and the positive constant α_2 replaced with $(\alpha_2 + p_2)$. (Note, there is an extra provision here that $\beta_1 - p_1 > 0$.)

To investigate the effect of DDT on the two populations we run the numerical solution again with $p_1 = p_2 = 0.1$. The results are presented in Figure 5.7. We see that, compared with Figure 5.6, the effect of the pesticide on the predator has been to decrease its overall numbers. It appears, however, that the mean prey population has increased. This is quite the opposite from the desired effect of the DDT and comes as a result of dealing with a nonlinear system. The DDT also reduces the average predator population so that there will be a reduced number of prey deaths due to the prey being eaten by the predators.

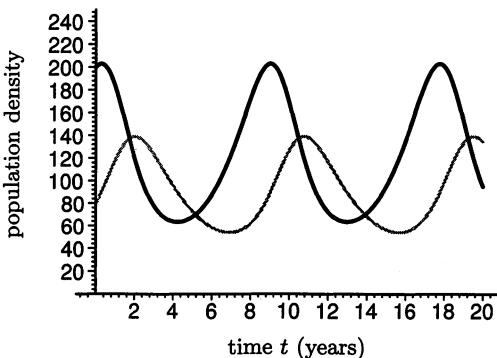


Figure 5.7: The scale insect or prey (black) and beetle population or predator (grey) where DDT has been used. Here we have $\beta_1 = 1$, $\alpha_2 = 0.5$, $c_1 = 0.01$, $c_2 = 0.005$, $p_1 = 0.1$ and $p_2 = 0.1$. The initial conditions are $x_0 = 200$, $y_0 = 80$. (These parameter values are chosen to illustrate the dynamics, and are not based on data from real populations.)

Interpretation of parameters

The differential equations (5.10) have 4 parameters: the prey per-capita birth rate β_1 , the predator per-capita death rate α_2 and the two constants of proportionality c_1 and c_2 . Making some further assumptions allows us to express c_1 and c_2 in terms of biologically relevant parameters.

Choose an arbitrary time interval Δt . We assume that Δt is sufficiently small, so that the populations $X(t)$ and $Y(t)$ do not change significantly over the time interval. We assume that each individual predator can search an area $a'\Delta t$, where a is the area searched per

unit time. We assume the areas covered by each individual predator do not overlap and we neglect the time it takes for the predator to eat the prey. The positive constant a' is called the *searching rate*. For a single predator the number of prey encountered is the prey density (number of prey per unit area) multiplied by the area covered by the predator, which is $X(t)a'\Delta t$. If we assume each encounter results in a kill for the predator, then the rate of prey deaths by a single predator is given by dividing by Δt . So

$$\left\{ \begin{array}{l} \text{rate of} \\ \text{prey killed by} \\ \text{one predator} \end{array} \right\} = a'X(t),$$

and hence the total rate of prey deaths (per unit area) is obtained by multiplying by $Y(t)$,

$$\left\{ \begin{array}{l} \text{rate of} \\ \text{prey killed} \\ \text{by predators} \end{array} \right\} = a'X(t)Y(t).$$

Comparing with equation (5.9) this gives $c_1 = a'$, for constant a' . This provides a biological interpretation for the parameter c_1 as the searching rate, or the rate of area covered by a predator per unit time. In Chapter 8 we improve the model to incorporate the time a predator spends search for and handling the prey.

Recall that $c_1X(t)Y(t)$ represents the rate of prey deaths and $c_2X(t)Y(t) = fc_1X(t)Y(t)$ the rate of predator births, so the constant ratio $f = c_2/c_1$ represents the amount of prey required for a unit predator birth. This parameter f is interpreted as the *predator efficiency parameter*.

Limitations and extensions

A number of attempts have been made to validate the Lotka-Volterra equations. These include experiments by the Russian microbiologist Gause with two different types of protozoa, *Paramecium caudatum* and *Didinium nasutum* (see Kormondy (1976)). Gause found that the prey protozoa died out within only a few oscillations. In Renshaw (1991) a modified version of the Lotka-Volterra equations, which incorporate random births and deaths, is investigated. This model exhibits behaviour similar to the Gause experiments. In general, however, the Lotka-Volterra equations are not widely used in practice, but rather form a sound basis for more complicated models, which we investigate in Chapter 8.

Some mathematics texts point to a well-known data set regarding the number of pelts of lynx and snow-shoe hare as a validation of the Lotka-Volterra equations. This data set has 10-year oscillations. However, Murray (1990) observes that these data are not a validation of the Lotka-Volterra equations at all, since the hare oscillations lag one quarter of a period behind those of the lynx, contradicting what we might expect. It would mean that the hare was eating the lynx, which would be a ridiculous conclusion. In Renshaw (1991), it is pointed out that the hares and the grass they eat show the correct predator-prey lag according to the Lotka-Volterra equations. This simple example demonstrates how easily the over-simplification of an ecosystem, into only two interacting species in this case, can lead to misinterpretation.

An obvious limitation of the model is the inclusion of exponential growth, and in the following section we improve on this by including a density dependent growth rate. Then in Chapter 8 (Section 8.3) we extend this predator-prey model further to improve its performance in predicting the observed behaviour of populations. We consider more realistic functions describing the prey death rate as well as the overall predator growth rate.

Density dependent growth

The oscillatory nature of the predator-prey model above is what one might expect intuitively, and together with the phase lag of the predator oscillation behind that of the prey it is

qualitatively what has been observed in some natural systems. However, our model is based on the assumption that the prey grows exponentially in the absence of the predator and this is clearly unrealistic. No matter how abundant the food supply, disease and/or food shortage will eventually curb the growth.

Thus, returning to the case without DDT, we replace the growth term for the prey population in (5.10) with density dependent growth and a carrying capacity of K . (For details on density dependent growth refer to Section 3.2.) The system becomes

$$\frac{dX}{dt} = \beta_1 X \left(1 - \frac{X}{K}\right) - c_1 XY, \quad \frac{dY}{dt} = c_2 XY - \alpha_2 Y. \quad (5.12)$$

Modifying the *Maple* code used to obtain Figure 5.6, and assuming a carrying capacity of $K = 1000$, we get the time-dependent plot of Figure 5.8. Note that the amplitude of the oscillations now decreases with an increase in time, for both populations. In fact, as time increases further, the figure suggests that each population might settle to a fixed population density, that is, stabilise. We look more closely at these results in the next chapter where we develop some analytical tools leading to a better understanding of the processes.

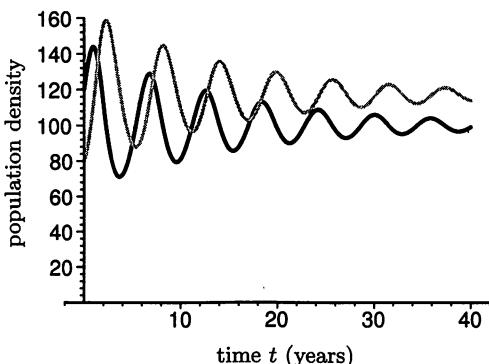


Figure 5.8: The scale insect (black line) and beetle population (grey line) where logistic growth for the scale insect has been included. Here we have $\beta_1 = 1$, $\alpha_2 = 0.5$, $c_1 = 0.01$, $c_2 = 0.005$ and $K = 1000$. (These parameter values are chosen to illustrate the dynamics, and are not based on data from real populations.)

Discussion

The control of pests (plant or animal) through the introduction of a natural enemy has had some remarkable successes and also some disastrous failures. The reasons why some programs succeed and others fail are not always well understood, and thus with current knowledge the outcome is often difficult to predict. One research paper which discusses this issue is Beddington et al. (1978), in which the authors attempt to identify features of the interaction process which have a significant impact on the outcome.

The scale insect/ladybird beetle system discussed in this chapter is just one example of failure. One example of an extremely successful case occurred in Australia in 1925. Prior to this date the prickly-pear cactus had been introduced into Australia with disastrous consequences, as it spread rapidly, thriving in the conditions and rendering thousands of hectares of farmland useless. In 1925, a natural predator moth was introduced to halt the spread of the cacti, and was spectacularly successful, nearly wiping out the weed in just two years. The case study is presented in Chapter 8 and provides a mathematical model which forecasts this success.

Some discussion of more realistic predator-prey models is given in Chapter 8 and also in Murray (1990) and Edelstein-Keshet (1988). For a biological perspective of interactions between a variety of species see (Begon et al., 1990, Chapters 7–12) and also May (1981). Further details of the effect of DDT on scale insects and ladybird beetles is given in MacArthur and Connell (1966). A brief discussion of further examples concerning the use of chemical pesticides on predator-prey systems is given in Keeton (1972).

Summary of skills developed here:

- *Formulate differential equations for two predators and one prey species, or two prey and one predator.*
- *Distinguish between models describing different types of interactions, such as models for symbiosis, parasite-host interactions and competition for the same resource.*
- *Generate numerical solutions to systems of equations.*

5.4 Case Study: Nile Perch catastrophe

We cite here a case where the ramifications of a disturbance within an interacting population system were wide reaching and caused an ecological disaster for the communities living in and around Lake Victoria. It emphasises the responsibility which should accompany any unsuitable modification of environmental processes, and the need for good modelling and forecasting, incorporating a multidisciplinary approach. Adapted from Murray (1990) and Quammen (1997).

Lake Victoria, which feeds into the Nile River, is the largest and most northern of a series of lakes punctuating the Rift Valley in Africa. Round as a pond and nearly as big as Ireland it is bordered by Kenya, Tanzania and Uganda, and until 1960 it supported many communities along its shores providing them with a large and diverse population of fish as well as fresh water. In particular, it supported an abundance of cichlids (tropical fresh water fish of the family *Cichlidae*) in both quantity and species; the sort of fish known for their garish appearance and collected for aquaria.

Because of its scalloped shoreline, its irregular patterns of depth and shallows, and its subjection to periods of intense drought and low water levels during the centuries long history of the lake, pockets of cichlids were cut off from others for long periods. In this way they diverged and speciated. Later reconnection allowed the species to mix spatially, but they had become incompatible reproductively and were now in competition. In order to survive they radiated: that is, they settled in different niches within the environment. Thus the lake represented a kind of underwater Galapagos archipelago with species for every niche: rock-scrappers, sand-digging-insect eaters, plant-scrappers, scale-eaters, nibbler-of-other-fishes'-fins, fish-eaters and the famed *Haplochromis compressiceps* known for biting out eyeballs! Lake Victoria supported about 200 such cichlid species, all of which are thought to have descended from a single ancestral species.

In 1960, supported by the United Nations Food and Agriculture Organisation, the Nile Perch was introduced into the lake to provide an additional source of protein. (The fish can grow to 100 kg or more.) Objections were voiced; however, they were ignored, the

introduction went ahead and the perch thrived. Being large and carnivorous, the perch all but wiped out the smaller fish species; hundreds of varieties unknown elsewhere. Many of these fish also provided the staple foods and economies for the local fishing communities. The markets became flooded with perch and the overall productivity of the lake was reduced by 80% (from the 1960 levels) in 25 years.

The ramifications spread far wider than the local ecological and economic disaster. The Nile Perch are oily and cannot be sun dried, the traditional means by which fish had been preserved. Instead they had to be smoked which resulted in major felling operations in the local environs to provide the fuel.

Furthermore, the cichlids had controlled the level of a particular snail which lives in and around the lake. The snails are an essential link in the spread of the disease bilharzia (liver fluke disease), which can be fatal to humans. Essentially, bilharzia is a disease produced by the larvae of a flatworm. The larvae first infect certain freshwater snails in streams and rivers, but particularly in lakes. They develop in the digestive gland of the snail and are discharged into the lake as larvae. From there they penetrate the skin of animals, such as humans, with a muscular boring action. Once inside the body they mature to an adult fluke and attach to the intestine or bladder and begin producing eggs. The effect on humans is a range of unpleasant problems of the kidney, liver, lung, intestine or central nervous system and can be seriously debilitating or fatal. Without the cichlids there was a marked increase in the number of snails and hence also the disease.

All in all, the perch introduction was catastrophic. Many of the ramifications could have, and should have, been foreseen and avoided. However, in spite of this, further introductions were being planned in the late 1980s for Nile Perch into other African lakes such as Lake Malawi.

5.5 Competing species

Another simple ecosystem to model is that of competing species, where two (or more) populations compete for limited resources such as food or territory. There are two aspects of competition: *exploitation*, when the competitor uses the resource itself and *interference*, where the population behaves in such a manner as to prevent the competitor from utilising the resource. This system is very similar to the predator-prey model of the previous section; however, the terms describing the interaction between the species differ.

Model assumptions

We start with a list of assumptions on which to build our first model.

- We assume the populations to be sufficiently large so that random fluctuations can be ignored without consequence.
- We assume that the two species model reflects the ecosystem sufficiently accurately.
- We assume each population grows exponentially in the absence of the other competitor(s), although we later incorporate density dependent growth for each.

General compartmental model

Let $X(t)$ and $Y(t)$ be the two population densities (number per unit area) where t is again time. As before, we have birth and death rates associated with each population; however, in this case an increase in the number of deaths in one population causes a decrease in

the number of deaths in the other population. The compartmental diagram of Figure 5.9 illustrates the process.

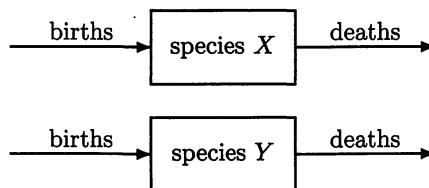


Figure 5.9: Input-output diagram for the 2-species competition model.

In words, we have for each population in the system

$$\left\{ \begin{array}{l} \text{rate of} \\ \text{change of} \\ \text{population} \end{array} \right\} = \left\{ \begin{array}{l} \text{rate of} \\ \text{population} \\ \text{births} \end{array} \right\} - \left\{ \begin{array}{l} \text{rate of} \\ \text{population} \\ \text{deaths} \end{array} \right\}. \quad (5.13)$$

Formulating the differential equations

Since neither population is dependent on the other as far as growth rates are concerned (unlike in the predator-prey example of Section 5.3), we let the positive constants β_1 and β_2 describe the per-capita birth rates for species X and Y , respectively.

Since the two populations are competing for the same resource, the density of each population has a restraining effect, proportional to this density, on the other. So the per-capita death rate for Y is proportional to X , and that for X is proportional to Y , which in symbols is

$$\left\{ \begin{array}{l} \text{rate of} \\ \text{species-}X \\ \text{deaths} \end{array} \right\} = (c_1 Y) X, \quad \left\{ \begin{array}{l} \text{rate of} \\ \text{species-}Y \\ \text{deaths} \end{array} \right\} = (c_2 X) Y.$$

Here c_1 and c_2 are the constants of proportionality for this restraining effect.

Our model becomes

$$\frac{dX}{dt} = \beta_1 X - c_1 XY, \quad \frac{dY}{dt} = \beta_2 Y - c_2 XY. \quad (5.14)$$

While β_1 and β_2 were per-capita birth rates, we may also consider them as overall per-capita growth rates which incorporate deaths (independent of the other species) as well as births. Thus they are per-capita growth rates, or per-capita reproduction rates, while the parameters c_1 and c_2 are the interaction parameters. These equations are known as *Gause's equations* and are a coupled pair of first-order, nonlinear differential equations.

Numerical solution

Again, although they appear reasonably simple, the equations cannot be solved analytically, and we use MATLAB or Maple to draw the time-dependent graphs, obtained using approximate numerical techniques as discussed in Chapter 4. Parameter values have been estimated for two competing species of micro-organisms (see Renshaw (1991)) as

$$\begin{aligned} \beta_1 &= 0.21827, & \beta_2 &= 0.06069, \\ c_1 &= 0.05289, & c_2 &= 0.00459. \end{aligned}$$

The MATLAB code is given in Listing 5.5, the Maple code is given in Listing 5.6 and the results are graphed in Figure 5.10.

Listing 5.5: MATLAB code: c_pe_compet.m

```
function c_pe_compet
global beta1 beta2 c1 c2;
beta1=0.22; beta2=0.06;
c1=0.053; c2=0.0046;
tend = 50; %the end time
u0 = [0.5; 1.5]; %set IC
[tsol, usol] = ode45(@rhs, [0, tend], u0);
Xsol = usol(:,1); Ysol = usol(:,2);
plot(tsol, Xsol, 'b'); hold on;
plot(tsol, Ysol, 'r');
axis([0, tend, 0, 10]);

function udot = rhs(t, u)
global beta1 beta2 c1 c2;
X = u(1); Y=u(2);
Xdot = beta1*X - c1*X*Y;
Ydot = beta2*Y - c2*X*Y;
udot = [Xdot; Ydot];
```

Listing 5.6: Maple code: c_pe_compet.txt

```
> restart; with(plots): with(DEtools):
> beta[1]:=0.22: beta[2]:=0.061: c[1]:=0.053: c[2]:=0.0046:
> de1 := diff(X(t),t) = beta[1]*X(t)-c[1]*X(t)*Y(t);
> de2 := diff(Y(t),t) = beta[2]*Y(t)-c[2]*X(t)*Y(t);
> inits := [X(0)=3, Y(0)=1.5];
> mydeopts := arrows=none, method=rkf45:
> plot1 := DEplot([de1,de2], [X,Y], t=0..50,
    [inits], scene=[t,X], linecolor=black,mydeopts):
> plot2 := DEplot([de1,de2], [X,Y], t=0..50,
    [inits], scene=[t,Y], linecolor=gray,mydeopts):
> display(plot1,plot2,view=[0..50,0..10]);
```

As usual, we are interested in what happens in the long run. From the time-dependent diagram of Figure 5.10 it appears that one of the species dies out over time. Varying the initial conditions illustrates that it is possible to choose values such that the other species dies out instead (e.g. with $X(0) = 2.6$, $Y(0) = 1.6$).

The results suggest that in the case of two species competing for the same resource, in the long run one species will survive and the other become extinct. This is known as *Gause's Principle of Competitive Exclusion*, from a series of experiments conducted by the microbiologist on competing species of yeast cells. Furthermore, unlike in the predator-prey model, the populations do not appear to oscillate with time.

Interpretation of parameters

The system of equations developed so far for the model of competitive species has four parameters: the two per-capita growth rates β_1 and β_2 which are independent of any other species, and the two constants of proportionality c_1 and c_2 which describe the interaction between the species.

The competition interaction can be interpreted as stating that the more deaths there are in species Y , the more resources will be available to species X , and thus the fewer deaths there will be for X . To explain the interaction we consider a small time interval Δt , sufficiently small so that X and Y do not change significantly. Let a' be the rate of area 'used' by one individual in species Y (for food), hence making it unavailable to X (for food). Assume that no such areas can overlap. Then one individual 'uses' an area of $a'\Delta t$,

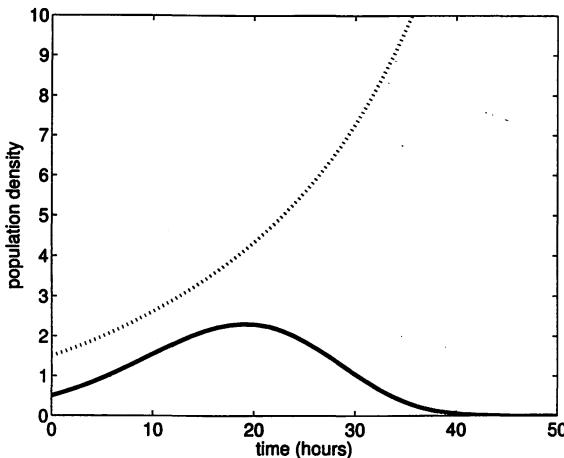


Figure 5.10: A sample numerical simulation of the competing species model with each species having unrestricted growth in the absence of the other species. The parameter values are $\beta_1 = 0.22$, $\beta_2 = 0.060$, $c_1 = 0.053$ and $c_2 = 0.0046$, with the initial condition $x_0 = 0.5$, $y_0 = 1.5$. Species X (black), species Y (grey dotted).

and Y individuals will ‘use’ $a'Y(t)\Delta t$. Assuming that each area of resource ‘used’ by Y implies it is unavailable to X , the rate at which area is made unavailable to X by a single Y individual is the density (number per unit area) of X in an area divided by Δt , which is $a'X(t)$. And hence the total effect of the removal of resources from X by Y becomes $a'X(t)Y(t)$. So a' , or c_1 in our model, can be interpreted as the rate at which X dies off due to the removal of resources by Y .

If we let $f = c_2/c_1$, then f is a measure of the efficiency of the competitive interaction. It can be considered as the number of units of species Y required to reduce species X by one unit.

Limitations of the model

One immediate and obvious limitation of this model is that each population grows exponentially in the absence of the other. In the next section, we improve on this by including density dependent growth.

In the time-dependent diagram, Figure 5.10, it appears that only one of the species will survive and the other will become extinct. (In the next chapter we show this to be the case for the competition model under nearly all conditions.) This idea was emphasised in the results of experimental work carried out by Gause.

Early this century, research tended to accept and support Gause’s principle; however, more detailed research has led to a questioning of this model. How does the degree of competition modify the predictions? What actually constitutes a competitive model when some resources are shared and others are not? By the 1980s, there was a lively debate underway amongst the leaders in the field, some rejecting outright the credibility of such models, while others argued that there was a value in, and a place for, the years of research already accomplished on these competition models. Two articles Lewin (1983a) and Lewin (1983b) provide an informative insight into this controversy and the positions adopted by the various players.

Density dependent growth

In 1932, the Russian microbiologist, Gause, described an experiment with two strains of yeast, *Saccharomyces cerevisiae* and *Schizosaccharomyces kefir*, hereafter called Species X and Species Y. These are described in Renshaw (1991). Gause found that, grown on their own, each species exhibited a logistic growth curve, but when grown together the growth pattern changed with Species X dying out.

We now extend the competition model to account for logistic growth in both species, in the absence of the other species. Let K_1 and K_2 be the carrying capacities for species X and Y respectively. Then including density dependent growth in (5.14) we have

$$\frac{dX}{dt} = \beta_1 X \left(1 - \frac{X}{K_1}\right) - c_1 XY, \quad \frac{dY}{dt} = \beta_2 Y \left(1 - \frac{Y}{K_2}\right) - c_2 XY. \quad (5.15)$$

With $d_1 = \beta_1/K_1$ and $d_2 = \beta_2/K_2$ the system (5.15) becomes

$$\frac{dX}{dt} = \beta_1 X - d_1 X^2 - c_1 XY, \quad \frac{dY}{dt} = \beta_2 Y - d_2 Y^2 - c_2 XY. \quad (5.16)$$

Using Gause's data (Renshaw (1991)), it is also possible to estimate the parameters d_1 and d_2 as in the model as

$$d_1 = 0.017, \quad d_2 = 0.010.$$

Note that these parameters correspond to the carrying capacities (in the absence of the other species) of $K_1 = \beta_1/d_1 = 13.0$ and $K_2 = \beta_2/d_2 = 5.8$.

Re-running the Maple or MATLAB code used to produce Figure 5.10, with an adjustment made for the inclusion of the density dependent growth for both X and Y, and the parameter values from Gause's experiment, produces Figure 5.11.

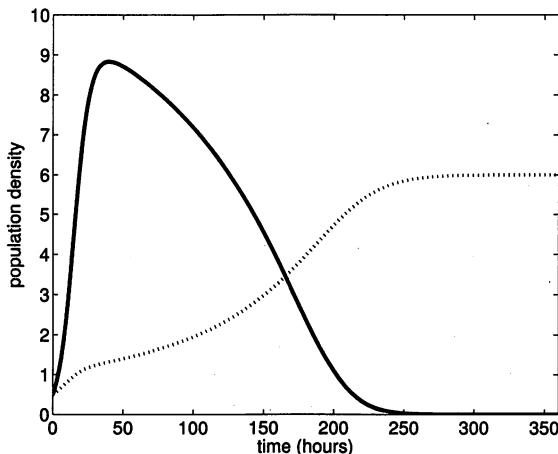


Figure 5.11: Graph of the population density of the two species of yeast populations, species $X(t)$ (black line), and species $Y(t)$ (grey dotted line)) as functions of time for the model with density dependence and with parameter values from Gause's experiment, as given in the text. Here both populations have initial density of 0.5.

We find that after a certain time, the figure suggests that only one of the species survives, stabilising to a fixed density, which is the carrying capacity for that species. This is what Gause observed (see Renshaw (1991) for a comparison between data and numerical solutions).

To get a better understanding of how this model behaves as we change initial conditions, or the parameter values, we could generate further numerical graphs. However, this is a somewhat hit-and-miss approach. For example, does one population always die out, or can they sometimes coexist? We return to a more detailed analysis of the dynamics of this system in Section 6.5, and also in Chapter 7, as we develop some helpful analytical tools.

Renshaw (1991) gives a detailed discussion of the competing species of yeast. See Kormondy (1976) for a discussion of competition between species of microscopic protozoa (*Paramecium aurelia* and *Paramecium caudatum*).

Summary of skills developed here:

- Formulate differential equations for variations on the competition model presented here, including models combining three or more competing species.
- Obtain numerical solutions to the competition model and extensions of it.
- Understand what is meant by, and the implications of, the ‘random’ approach of generating numerical solutions.
- Understand the competitive exclusion principle.

5.6 Case Study: Aggressive protection of lerp and nymphs

The following case study does not incorporate the above theory specifically, but describes the occurrence of a competing species scenario where a colony of birds protects its territory in order to maintain an adequate food supply to support itself. It is included for interest and to provide an example of the value of interspecific territoriality, a system for which the above theory would have relevance. Adapted from the article by Loyn et al. (1983).

In regions of southeastern Australia infestations of psyllids have inflicted severe damage on the eucalypt forests causing deterioration of the foliage and, in extreme cases, completely destroying certain trees. This has prompted an examination of the conditions under which it may occur. Many species of birds live in the canopy of these eucalypts and compete to feed on these insects; the Bell Miners are one such species.

The Bell Miner (*Manorina melanophrys*) is a honeyeater living in large colonies which they defend aggressively against other competing birds, often substantially larger than themselves. They feed on the nymphs, sweet secretions and lerps (protective carbohydrate covers) of psyllids; however, where their colonies occur, the canopy foliage appears unhealthy and infested with these insects. The Miners aggressively protect this abundance of prey. In an experiment a colony of Bell Miners was removed to join another colony 45 km away. They settled into this new location and did not return.

Other bird flocks soon moved into the site of the old colony and rapidly the foliage improved. The insect infestation declined, with the numbers of psyllids remaining very small thereafter.

It appears that the insect infestations can be controlled by the removal of the Bell Miner colonies. The interspecific territorial behaviour of the Miners prevents this control and introduces a control of its own. Miners effectively maintain an abundant and exclusive food supply, albeit detrimental to the eucalypts, enabling a colony to remain within the same territory for up to 40 years.

5.7 Model of a battle

We now consider a novel type of population interaction: ~~a destructive competition~~ or ~~battle between two opposing groups~~. These may be battles between two hostile insect groups, athletic teams or human armies. While the models we derive here apply to the last case, the principles can be generalised and would apply to many other examples. The model we develop turns out to be a system of two coupled, linear differential equations.

Background

Battles between armies have been fought since antiquity. In ancient times battles were primarily hand-to-hand combat. With the development of archery and then gunpowder a crucial feature of battles has been aimed fire. Although many factors can affect the outcome of a battle, experience has shown that numerical superiority and superior military training are critical. The model we present was first developed in the 1920s by F. W. Lanchester who was also well known for his contributions to the theory of flight.

Our aim is to develop a simple model which predicts the number of soldiers in each army at any given time, provided we know the initial number of soldiers in each army. (As with epidemics, we consider the number, rather than the density, of individuals.)

Model assumptions

First we make some basic assumptions and then develop the model based on these.

- We assume the number of soldiers to be sufficiently large so that we can neglect random differences between them.
- We also assume that there are no reinforcements and no operational losses (i.e. due to desertion or disease).

These are assumptions that can easily be relaxed at a later stage if the model is inadequate.

General compartmental model

The first step is to develop two word equations which describe how the two populations change, based on the input-output principle of the balance law. Suppose the two opposing groups or populations are the red army and the blue army.

Example 5.5: Determine the appropriate input-output diagram and associated word equations for the number of soldiers in both the red and blue armies.

Solution: Since there are no reinforcements or operational losses, each population changes by the number of soldiers who are wounded by the other army. Thus we can set up the input-output diagram of Figure 5.12.

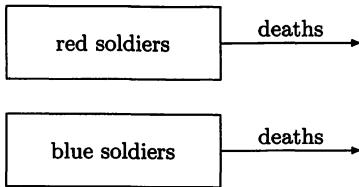


Figure 5.12: Compartmental diagram for the simple battle model.

Thus, in any given instant of time,

$$\begin{aligned} \left\{ \begin{array}{l} \text{rate of} \\ \text{change of} \\ \text{red soldiers} \end{array} \right\} &= - \left\{ \begin{array}{l} \text{rate red soldiers} \\ \text{wounded by} \\ \text{blue army} \end{array} \right\} \\ \left\{ \begin{array}{l} \text{rate of} \\ \text{change of} \\ \text{blue soldiers} \end{array} \right\} &= - \left\{ \begin{array}{l} \text{rate blue soldiers} \\ \text{wounded by} \\ \text{red army} \end{array} \right\}. \end{aligned} \quad (5.17)$$

In a real battle there will be a mixture of shots; those fired directly at an enemy soldier and those fired into an area known to be occupied by an enemy, but where the enemy cannot be seen. Some battles may be dominated by one or the other firing method. We consider these two idealisations of shots fired as *aimed fire* and *random fire*. For the model we assume only aimed fire for both armies.

In the aimed fire idealisation, we assume all targets are visible to those firing at them. If the blue army uses aimed fire on the red army, then each time a blue soldier fires he/she takes aim at an individual red soldier. The rate of wounding of the red army depends only on the number of blue soldiers firing at them and not on the number of red soldiers. We see later that this assumption is equivalent to assuming a constant probability of success (on average) for each bullet fired.

For random fire a soldier firing a gun cannot see his/her target, but fires randomly into an area where enemy soldiers are known to be. The more enemy soldiers in that given area then the greater the rate of wounding. For random fire we thus assume the rate of enemy soldiers wounded is proportional to both the number firing and the number being fired at.

In summary then we make the following further assumptions:

- for aimed fire the rate of soldiers wounded is proportional to the number of enemy soldiers only,
- for random fire the rate at which soldiers are wounded is proportional to both numbers of soldiers.

Formulating the differential equations

Let $R(t)$ denote the number of soldiers of the red army and $B(t)$ the number of soldiers of the blue army. We assume aimed fire for both armies. This information is expressed mathematically by writing

$$\left\{ \begin{array}{l} \text{rate red soldiers} \\ \text{wounded by} \\ \text{blue army} \end{array} \right\} = a_1 B(t), \quad \left\{ \begin{array}{l} \text{rate blue soldiers} \\ \text{wounded by} \\ \text{red army} \end{array} \right\} = a_2 R(t) \quad (5.18)$$

where a_1 and a_2 are positive constants of proportionality. The constants a_1 and a_2 measure the effectiveness of the blue army and red army respectively, and are called *attrition coefficients*.

We thus assume that attrition rates are dependent only on the firing rates and are a measure of the success of each firing.

We now substitute (5.18) into the basic word equation (5.17), where the rate of change in the number of red soldiers is dR/dt and for the blue soldiers it is dB/dt . The two simultaneous differential equations are thus

$$\frac{dR}{dt} = -a_1 B, \quad \frac{dB}{dt} = -a_2 R. \quad (5.19)$$

Numerical solution

During the Battle of Iwo Jima in the Pacific Ocean (1945) daily records were kept of all U.S. combat losses. These data are graphed and referenced in Braun (1979). The values of the attrition coefficients a_1 and a_2 have been estimated from the data² as $a_1 = 0.0544$ and $a_2 = 0.0106$, and the initial numbers in the red and blue armies respectively were $r_0 = 66\,454$ and $b_0 = 18\,274$.

Using MATLAB or Maple we can obtain accurate numerical solutions to the differential equations (5.19). The results are shown in Figure 5.13 with the red army as the U.S. forces and the blue army as the Japanese. The model shows a remarkably good agreement with the data. The MATLAB and Maple code used to obtain the graphs are given in Listing 5.7 and Listing 5.7.

Listing 5.7: MATLAB code: c_pe_combat.m

```
function c_cp_combat
global a1 a2;
tend = 30; %the end time to run the simulation
a1=0.0544; a2=0.0106;
u0 = [66; 18]; % set initial conditions as a column vector
[tsol, usol] = ode45(@rhs, [0, tend], u0);
Rsol = usol(:, 1); Bsol = usol(:, 2);
plot(tsol, Rsol, 'r'); hold on; plot(tsol, Bsol, 'b');

function udot = rhs(t, u)
global a1 a2;
R=u(1); B=u(2);
Rdash = -a1*B;
Bdash = -a2*R;
udot = [Rdash; Bdash];
```

Listing 5.8: Maple code: c_pe_combat.txt

```
> restart:with(plots):with(DEtools):
> a[1]:=0.0544:a[2]:=0.0106:
> de1:=diff(R(t),t)=-a[1]*B(t);
> de2:=diff(B(t),t)=-a[2]*R(t);
> init:= [R(0)=66,B(0)=18]:
> myopts:=steptime=0.1,arrows=NONE:
> plot1:=DEplot([de1,de2],[R,B],t=0..30,[init],scene=[t,R],linecolour=red,myopts):
> plot2:=DEplot([de1,de2],[R,B],t=0..30,[init],scene=[t,B],linecolour=black,myopts):
> display(plot1,plot2);
```

Despite the excellent fit to the data there are still some reservations about using this model for the Battle of Iwo Jima. The values of the parameters indicate the Japanese

²The method of least squares was used to determine the parameter values. It varies the parameters to minimise a function which is the sum of the squares of the errors between the numerical solution and the data points.

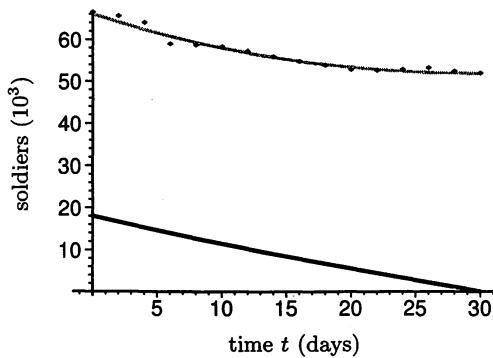


Figure 5.13: Numbers of U.S. soldiers in the battle of Iwo Jima measured in thousands. The solid lines (U.S.-grey, Japanese-black) are the model predictions from Maple and the dots are the measured data, from Braun (1979).

soldiers were approximately five times more effective than the U.S. soldiers, since $a_1 \simeq 5a_2$. In this battle, the Japanese army occupied the island and were well dug in. Perhaps, a better model would be to assume the U.S. soldiers used random fire with the Japanese soldiers using aimed fire. It turns out (see Exercises Question 18) that this model gives an equally good fit to the data.

Interpretation of parameters

We can further refine the model by trying to express the parameters a_1 and a_2 in terms of possible quantities which could be measured. The rate at which soldiers are wounded depends on both the firing rate and the probability of a shot hitting a target.

We return to equations (5.18). Consider a single blue soldier firing at the red army. We assume each blue soldier fires at a constant rate f_b . Then

$$\left\{ \begin{array}{l} \text{rate red soldiers} \\ \text{wounded by} \\ \text{single blue soldier} \end{array} \right\} = \left\{ \begin{array}{l} \text{rate bullets} \\ \text{fired in time} \\ \text{interval} \end{array} \right\} \times \left\{ \begin{array}{l} \text{probability of} \\ \text{a single bullet} \\ \text{hitting target} \end{array} \right\}$$

$$= f_b p_b,$$

where p_b is the probability (constant) that a single bullet from the blue soldier wounds a red soldier. Hence, for the entire blue army we multiply by the number of blue soldiers, $B(t)$, to obtain the total rate of red soldiers wounded by the blue army (per unit time). This gives

$$\left\{ \begin{array}{l} \text{rate red soldiers} \\ \text{wounded by} \\ \text{blue army} \end{array} \right\} = f_b p_b B(t). \quad (5.20)$$

Equating this to (5.18) we obtain the attrition rates, or coefficients, a_1 and a_2 as

$$a_1 = f_b p_b, \quad a_2 = f_r p_r, \quad (5.21)$$

where f_r is the firing rate by a single red soldier and p_r to be the probability that a single red bullet hits its target. We can think of f_b , f_r and p_b , p_r as factors influenced by morale, training and technology.

For random fire we do not assume the probability of a single bullet wounding a soldier to be constant. It will vary depending on the number of target soldiers within a given area. Thus, this probability will depend on both the number of target soldiers and the area into which fire is being directed.

Exact solution

The differential equations (5.19) are linear, unlike the systems of differential equations in previous sections (Sections 5.3, 5.5 and 5.2) and it is possible to obtain an exact solution. We can use the Maple code in Listing 5.9.

Listing 5.9: Maple code: c_pe.combat_sym.txt

```
> restart;
> de1 := diff(R(t),t) = -a[1]*B(t);
> de2 := diff(B(t),t) = -a[2]*R(t);
> inits := R(0)=r[0],B(0)=b[0];
> dsolve({de1,de2, inits}, {R(t),B(t)});
```

The solution, in terms of exponential functions, can be written in a nice compact form as

$$\begin{aligned} R(t) &= r_0 \cosh(\alpha t) - b_0 v \sinh(\alpha t), \\ B(t) &= b_0 \cosh(\alpha t) - r_0 v^{-1} \sinh(\alpha t), \end{aligned} \quad (5.22)$$

where

$$\alpha = \sqrt{a_1 a_2}, \quad v = \sqrt{\frac{a_1}{a_2}}.$$

This solution is also obtained in the exercises (see Question 20) by substitution of one differential equation into the other. For details on the hyperbolic functions (sinh and cosh) see Appendix A.2 and Appendix B.4. (Matrices and eigenvalues may also be used to find this solution, as is explained in Section 7.2.)

Limitations and extensions of the model

The model we have developed here was based on the assumption of aimed fire. More generally, battles occur where one army uses aimed fire and the other uses random fire (for example, guerrilla warfare) or where both armies use random fire (e.g. long-range artillery). These models lead to the differential equations

$$\frac{dR}{dt} = -a_1 B, \quad \frac{dB}{dt} = -c_2 R B,$$

in the case of guerrilla warfare and

$$\frac{dR}{dt} = -c_1 R B, \quad \frac{dB}{dt} = -c_2 R B,$$

for long-range artillery or trench warfare.

There are several other obvious extensions and variations of the basic model. These include incorporating both random and aimed fire, or modelling regular reinforcement and/or operational losses, such as from disease.

Other good references include Taylor (1980), Przemieniecki (1994) and Tung (2007) who discuss further extensions of the basic models to include reinforcements, range dependent firing and geometric mean fire. In Braun (1979) there is a clear discussion of this model and a similar model for guerrilla warfare, where one army is hidden (e.g. jungle warfare). Also discussed is the Battle of Iwo Jima. See Taylor (1980) for a substantial discussion of these types of models, and ways of estimating attrition coefficients. Stochastic (random) effects may also be included into the basic models; see Przemieniecki (1994).

Summary of skills developed here:

- Modify the model to account for one, or both, of the armies using random fire.
- Modify the model to account for loss due to disease and/or gains due to reinforcements.
- Obtain numerical solutions to the model developed and some extensions.

5.8 Case Study: Rise and fall of civilisations

This case study is based on an article by Feichtinger et al. (1996). It illustrates the use of mathematical models to provide a possible explanation for the rise and fall of dynasties in ancient China.

China is one of the oldest human civilisations which can claim to have some degree of continuity. In ancient China there have been many dynasties, such as the Xia, Shang, and Zhou dynasties of ancient times and the Qin and Han and Ming dynasties. Between these dynasties there have been periods of rapid population decline corresponding to the fall of dynasties. During these times of anarchy the ruling classes (which include the soldiers under their command) have been weak and unable to control the numbers of bandits and outlaws. However, over time this state does not persist and the ruling classes are able to suppress the outlaws and bandits and the general population of peasants and farmers increases.

Why do these periods of anarchy occur? There are probably many causes; however, one plausible explanation is that it could be due to the natural dynamics of various interacting groups in the population. It is the aim of a mathematical model to try and explain this behaviour with as few variables as possible. To try and help explain the fall of dynasties, a simple three-population model has been developed.

The three sub-populations are the farmers, $F(t)$, (i.e. the peasant class), the bandits, $B(t)$, and the ruling class, $R(t)$ (which includes the soldiers hired by the emperor). The differential equations are

$$\frac{dF}{dt} = rF \left(1 - \frac{F}{K}\right) - \frac{aFB}{b+F} - hFR, \quad (5.23)$$

$$\frac{dB}{dt} = \frac{eaFB}{b+F} - mB - \frac{cBR}{d+B} \quad (5.24)$$

$$\frac{dR}{dt} = \frac{faFB}{b+F} - gR \quad (5.25)$$

where r , K , a , h , b , d , m , c , f , and g are positive constants in the model. This model is like a predator-prey model where the bandits 'prey on' the farmers and the soldiers (ruling class) 'prey on' the bandits as well as the farmers. Rulers impose taxes on farmers and punish bandits.

In the absence of rulers and bandits the farmers exhibit logistic growth,

$$\frac{dF}{dt} = rF \left(1 - \frac{F}{K}\right).$$

The term $aFB/(b+F)$ is a saturating predation rate of bandits upon farmers. The number of farmers killed by an individual bandit is $aF/(b+F)$ and as F becomes large then this rate tends to a constant rate (i.e. as $F \rightarrow \infty$, then $aF/(b+F) \rightarrow a$, a constant rate); but is approximately proportional to the number of farmers, F , for small numbers of farmers. This saturating rate reflects the fact that bandits must spend some time searching out targets. (See Section 8.3 for a further discussion of models of predation incorporating with searching times.)

Both the bandits and the rulers (soldiers) have a natural mortality term proportional to their numbers, mB and gR . The bandits have an additional mortality term $cBR/(d+B)$ from the contact of bandits with soldiers. This term also incorporates saturation, reflecting the fact that soldiers have to search out bandits, so with increasing numbers of bandits the rate that each individual soldier can kill is $cB/(d+B)$ which tends to a constant as $B \rightarrow \infty$ and is proportional to B for small numbers of bandits.

The term hFR in the first differential equation represents an additional mortality rate for the farmers due to excessive taxing of the farmers by the ruling class. Note that this is proportional to both the number of farmers and the number of the ruling class; the greater the number of soldiers the more taxes are needed to pay for them, and the higher the mortality for each individual farmer.

The parameter values for this model have been listed by Feichtinger et al. (1996) as given in Table 5.1. The populations are measured as fractions of the farmer carrying capacity, so, in these units, the carrying capacity $K = 1$. Similarly, the time is scaled with respect r^{-1} , where r is the farmer intrinsic growth rate, so $r = 1$ here. (See Section D.1 in the appendix for a discussion of scaling.) Using initial conditions $F(0) = 0.7$, $B(0) = 0.1$ and $R(0) = 0.2$ we can easily use Maple or MATLAB to solve the differential equations numerically and to graph the populations, as in Figure 5.14 and Figure 5.15.

Table 5.1: Parameter values for the dynamic cycle model, from Feichtinger et al. (1996). These assume the populations are measured as a fraction of the maximum farmer population, so $K = 1$, and $r = 1$ in these units. These parameters were chosen to illustrate the possibility of cycles of population growth rather than an accurate estimate from historical data.

$r = 1$	$K = 1$	$a = 1$
$b = 0.17$	$h = 0.1$	$d = 0.42$
$m = 0.4$	$c = 0.4$	$f = 0.1$
$g = 0.009$	$e = 1.2$	

With this choice of parameters the populations of farmers and bandits oscillate, with the farmer population near the carrying capacity most of the time, but where the population periodically undergoes a dramatic fall. This directly coincides with the time when the bandit population increases dramatically. The ruling population (the soldiers) peaks at the same time as the peaks of the bandit population then falls slightly until another farmer population collapse.

It is interesting to explore the effect of changing some of the parameters. In Figure 5.15 the parameter h has been changed. This parameter corresponds to the coefficient of the additional mortality rate of farmers due to taxing by the rulers. If this value is increased from $h = 0.14$ to $h = 2.0$ this changes the dynamics of the populations. After some degree of oscillation all three populations then settle into a steady-state, and are in equilibrium. The surprising implication is that a higher degree of severity of taxation of farmers leads to a more stable society, in the long term. A possible interpretation of this change in behaviour is as follows. With a larger exploitation rate, h , the ruling class is more able to maintain

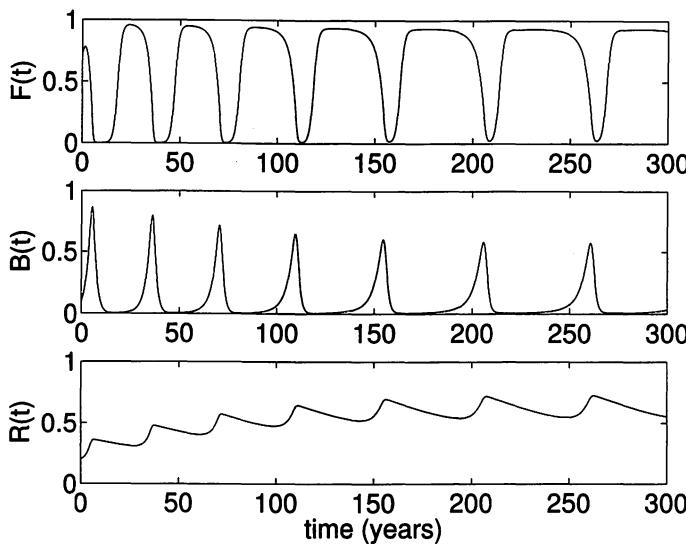


Figure 5.14: Numerical simulation of the farmer-bandit-rulers model for the rise and fall of dynasties in ancient China. Parameter values used are given in Table 5.1 and initial values are $F(0) = 0.7$, $B(0) = 0.1$ and $R(0) = 0.2$, where these values correspond to a fraction of the carrying capacity of the farmer population.

order by having sufficient resources (i.e. numbers of soldiers) to keep the bandit population from becoming too large.

It is also interesting to determine the effect of changing other parameters in the model, and trying to interpret the results. For example, increasing the parameter $c = 0.4$ to $c = 0.8$ causes longer periods between periods of anarchy (see exercises, Question 26). Note that changing the initial conditions changes the graphs, but they eventually settle into the same long-term behaviour.

This is a fairly complex model (with 11 separate parameters) and there is much scope for exploring its behaviour. Some progress can be made by making the observation that the ruling class population generally changes slowly compared with the other populations. Feichtinger et al. (1996) exploit this by holding the variable R constant and analysing the resulting system of two simultaneous differential equations. They do this using bifurcation theory. This is beyond the scope of this book; however, some of the techniques developed in subsequent chapters on phase-plane analysis are used in their analysis.

Despite the apparent complexity of behaviour of this model, it is still of some interest to explore some simple extensions. Foremost is one identified by Feichtinger et al. (1996) who suggest adding an additional term corresponding to a loss of the soldiers from contacts with the bandits. This might be significant where there are large numbers of bandits or if the bandits were sufficiently good fighters compared with the soldiers. Another interesting extension would be to include an additional term in the farmer differential equation corresponding to recruitment of the farmers as soldiers into the ruling class, with a corresponding term in the differential equation for the rulers (soldiers).

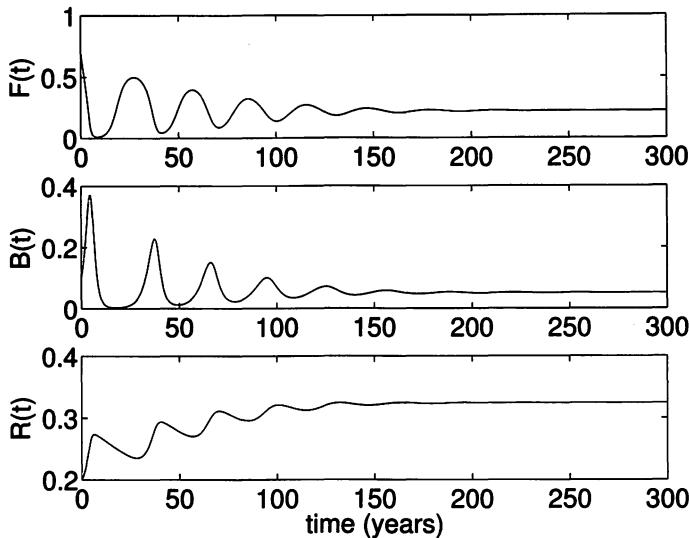


Figure 5.15: Numerical simulation of the farmer-bandit-rulers model. Here the parameter values in Table 5.1 are used, but the parameter h , corresponding to the hiring rate of soldiers, is increased from 0.1 to 2.0.

5.9 Exercises for Chapter 5

5.1. Numerical solution of basic SIR epidemic model. In Section 5.2, a model for an epidemic was developed, which led to the differential equations

$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I.$$

- (a) Use parameter values $b = 0.002$ and $r = 0.4$, and assume that initially there is only one infective but there are 500 susceptibles. Use MATLAB or Maple to generate the time-dependent plot on the interval $t = [0, 20]$.
- (b) How many susceptibles never get infected?
- (c) What happens as time progresses if $S(0) = 100$?
- (d) Suppose the transmission coefficient b is doubled. How does this effect the maximum number of infected individuals? Is this what you expect?

5.2. SI model, Contagious for life. Consider a disease where all those infected remain contagious for life. Ignore all births and deaths.

- (a) Write down suitable word equations for the rate of change of numbers of susceptibles and infectives. Hence develop a pair of differential equations. (Define any notation you introduce.)
- (b) With a transmission coefficient of 0.002, and initial numbers of susceptibles 500 and infectives 1, use Maple or MATLAB to sketch time-dependent plots for the sub-populations (susceptibles and infectives) over time.

5.3. Disease with no immunity. Consider an infectious disease where all those infected become susceptible again upon recovering from the disease. Let $S(t)$ and $I(t)$ denote numbers of infectious

and susceptibles and let β be the transmission coefficient and γ^{-1} the infectious period. Develop a model as two differential equations for S and I .

5.4. Continuous vaccination. Consider a model for the spread of a disease where lifelong immunity is attained after catching the disease. The susceptibles are continuously vaccinated against the disease at a rate proportional to their number. Write down suitable word equations to describe the process, and hence obtain a pair of differential equations.

5.5. SEIR model, disease with a latent period. Many diseases have a latent period, which is when there is a period of time between infection and when an infected individual becomes infectious. One example is measles, where the latent period is approximately 5 days.

Extend the basic epidemic model to one with an additional population class $E(t)$, corresponding to individuals who have been exposed to the disease, so they are no longer susceptibles, but are not yet infectious. You may assume the per-capita rate at which an individual in the exposed class becomes an infective is constant. Also the infectious recover in a mean time γ^{-1} and have lifelong immunity. (Give a suitable compartmental diagram or a set of word equations and define any new parameters you introduce.)

Note that the latent period is not the same as the incubation period (the time from infection to when symptoms appear).

5.6. Two prey and one predator. Develop a model with three differential equations describing a predator-prey interaction, where there are two different species of prey and one species of predator. What assumptions have been made in this model formulation? (Base these on the assumptions used in Section 5.3.)

5.7. Effect of DDT. Consider the predator-prey model developed in Section 5.3, which describes the threat to the American citrus industry posed by the accidental introduction of the Australian scale insect, and the later introduction of the ladybird to combat the pest.

- Use MATLAB or Maple to generate the time-dependent graphs of the populations over time with $\beta_1 = 1$, $\alpha_2 = 0.5$, $c_1 = 0.01$ and $c_2 = 0.005$. Initialise with $(x_0, y_0) = (200, 80)$ and $(x_0, y_0) = (80, 200)$. Comment on any differences in the long-term behaviour.
- Verify that $X' = 0$ and $Y' = 0$, if $X = 100$ and $Y = 100$ simultaneously. Plot these values (as horizontal lines) in the time-dependent diagrams.
(At these values there is no change in the populations over time, so they are equilibrium populations. We see how to find such equilibrium solutions in Chapter 6.)
- Now include the effect of DDT in the model, with $p_1 = 0.1$ and $p_2 = 0.1$. Generate the time-dependent plots as before. Verify that $X' = 0$ and $Y' = 0$ when $X = 120$, $Y = 80$, and plot these values on the time-dependent diagrams.
- Comment on your observations from the above.

5.8. Predator-prey with density dependence. Starting with the Lotka-Volterra model, we wish to include the effect of logistic growth for the prey and DDT on both species. The differential equations are

$$\frac{dX}{dt} = \beta_1 X \left(1 - \frac{X}{K}\right) - c_1 XY - p_1 X, \quad \frac{dY}{dt} = c_2 XY - \alpha_2 Y - p_2 Y.$$

- With parameter values as in Figure 5.7, and also Figure 5.8, use Maple or MATLAB to generate time-dependent graphs for the populations over time.
- Verify (by substitution) that $X' = 0$ and $Y' = 0$, if $X = 50.5$ and $Y = 84.95$. Include them in the time-dependent plots of (a) as horizontal lines.
- Comment on your observations, particularly with respect to Figure 5.8.

5.9. Competing species with no density dependence. In Section 5.5, a simple model describing the interaction between two competing species was developed, where each population exhibits exponential growth.

- Use MATLAB or Maple to generate time-dependent graphs for the populations over time, with parameter values as given in Figure 5.10, but using a variety of initial conditions.
- Show that $X' = 0$ and $Y' = 0$ simultaneously, when $X = 2.5$ and $Y = 1.5$. Include them in the time-dependent plots as horizontal lines.
- In this case it is not so easy to make some ‘general conclusions’ about the population behaviour over long periods of time. Why?

5.10. Competing species with density dependence. Consider the following model for two competing species, with densities, $X(t)$ and $Y(t)$, given by the differential equations

$$\frac{dX}{dt} = X(\beta_1 - c_1 Y - d_1 X), \quad \frac{dY}{dt} = Y(\beta_2 - c_2 X - d_2 Y),$$

with parameter values $\beta_1 = 3$, $\beta_2 = 3$, $c_1 = 2$, $c_2 = 1$, $d_1 = 2$ and $d_2 = 2.5$.

- What is the carrying capacity for each of the species, evaluated for the given parameter values? (Hint: Compare with equations (5.15) in Section 5.5.)
- With the above parameter values, and the initial values $X = 2$ and $Y = 2$, use MATLAB or Maple to draw time-dependent plots for these populations. Over a period of time what population densities do you estimate they will approach?

5.11. Symbiosis. Symbiosis is where two species interact with each other, in a mutually beneficial way. Starting with a compartmental diagram, formulate a differential equation model describing this process, based on the following. Assume the per-capita death rate for each species to be constant, but the per-capita birth rate to be proportional to the density of the other species. In other words, the presence of the other species is necessary for continued existence. (Define all parameters and variables of the model.)

5.12. Simple age-based model. Consider a population split into two groups: adults and juveniles, where the adults give birth to juveniles but juveniles are not yet fertile. Eventually juveniles mature into adults. You may assume constant per-capita birth and death rates for the population, and also assume that the young mature into adults at a constant per-capita rate σ .

Starting from suitable word equations or a compartment diagram formulate a pair of differential equations describing the density of adults, $A(t)$, and the density of juveniles, $J(t)$. Define all variables and parameters used.

5.13. Beetle populations. A population of beetles has three different age stages: larvae (grub), pupae (cocoon), and adult. Assume constant per-capita death rates for each population class of a_1 for larvae, a_2 for pupae and a_3 for adults. Also assume adults produce larvae at a constant per-capita birth rate of larvae b_1 . The larvae turn into pupae at a constant per-capita rate σ_1 and pupae turn into adults at a constant per-capita rate σ_2 .

Let $A(t)$ denote the number of adults, $L(t)$ the number of larvae and $P(t)$ the number of pupae at time t and formulate a mathematical model in the form of three differential equations.

5.14. Wine fermentation. In the fermentation of wine, yeast cells digest sugar from the grapes and produce alcohol as a waste product, which is toxic to the yeast cells. Develop a model consisting of three coupled differential equations for the density of yeast cells, the amount of alcohol and the amount of sugar. In the model assume the yeast cells have a per-capita birth rate proportional to the amount of sugar, and a per-capita death rate proportional to the amount of alcohol present. Assume the rate of alcohol produced is proportional to the density of yeast cells, and the rate of sugar consumed is proportional to the density of yeast cells.

5.15. Cycles of measles epidemics. For the spread of measles it is thought that the effect of births, continuously refreshing the number of susceptibles, is an important contributing factor towards the periodic outbreaks of the disease.

Develop a model for the spread of measles which incorporates this refreshing of the susceptible numbers, assuming a constant per-capita birth rate for susceptibles, and also that all those infected become immune. Start with suitable word equations and define all symbols introduced.

5.16. Density dependent contact rate. For a fatal disease, if the basic epidemic model of Section 5.2 is modified to include density dependent disease transmission, the resulting differential equations are

$$\frac{dS}{dt} = -p \frac{c(N)}{N} SI, \quad \frac{dI}{dt} = p \frac{c(N)}{N} SI - \gamma I,$$

where $N = S + I$, p is a constant (the probability of infection) and the contact rate function $c(N)$ is given by

$$c(N) = \frac{c_m N}{K(1 - \epsilon) + \epsilon N},$$

where ϵ is a positive constant between 0 and 1 and K is a positive constant.

Consider $\epsilon = 0.5$, and using Maple or MATLAB with $pc_m = 1.62$, $r = 0.44$, $K = 1000$, and initial values $i_0 = 1$, $s_0 = 762$ (time is measured in days) graph the number of susceptibles over time and determine when the number of infectives is at a maximum. Compare this model to those with $\epsilon = 1$ and $\epsilon = 0$, and discuss the differences.

5.17. Battle loss due to disease. Develop a model (a pair of differential equations) for a battle between two armies where both groups use aimed fire. Assume that the red army has a significant loss due to disease, where the associated death rate (from disease) is proportional to the number of soldiers in that army.

5.18. Jungle warfare. In Section 5.7, we developed a simple model for a battle between two armies. We assumed that the probability of a single bullet hitting its target is constant. This is not a good assumption in jungle warfare or guerrilla warfare where one, or both, of the soldiers may be hidden from view of the other.

Suppose that soldiers from the red army are visible to the blue army, but soldiers from the blue army are hidden. Thus, all the red army can do is fire randomly into an area and hope they hit something. The blue army uses aimed fire.

- (a) Write down appropriate word equations describing the rate of change of the number of soldiers in each of the armies.
- (b) By making appropriate assumptions, obtain two coupled differential equations describing this system.
- (c) Extend the model to include reinforcements if both of the armies receive reinforcements at constant rates.

5.19. Jungle warfare (continued). This question refers to the differential equations model, developed in Question 18, and gives estimates of the parameters. Suppose the blue army defends an area of $A = 10^5 \text{ m}^2$, with an initial number of 150 soldiers. The red army has 500 soldiers initially, all of whom are exposed to fire from the blue army. Each soldier, in either army, fires at the same rate of $f_r = f_b = 400$ bullets per day. Field data have shown that each single bullet fired from the blue army has the constant probability 1/100 of one in one hundred of wounding a red soldier.

- (a) Write down a formula for the probability of a single bullet fired from a single red soldier wounding a blue soldier in terms of the total area A and the area exposed by a single blue soldier $A_b = 0.1 \text{ m}^2$.

- (b) Hence, estimate the coefficients in your model (i.e. write the rate of wounding in terms of the probability in (a) and the firing rate.)
- (c) With your estimates in (b), use MATLAB or Maple to calculate the number of soldiers left in each army after 5 days.

5.20. Exact solution for battle model. Consider the aimed fire battle model developed in the text

$$\frac{dR}{dt} = -a_1 B, \quad \frac{dB}{dt} = -a_2 R.$$

The exact solution can be found using theoretical techniques as follows:

- (a) Take the derivative of the first equation to get a second-order differential equation, and then eliminate dB/dt from this equation by substituting the second equation (given above) into this second-order equation.
- (b) Now assume the solution to be an exponential of the form $e^{\lambda t}$. Substitute it into the second-order equation and solve for the two possible values of λ . The general solution for R will be of the form

$$R(t) = c_1 e^{\lambda_1 t} + c_2 e^{\lambda_2 t},$$

where c_1 and c_2 are the arbitrary constants of integration. The solution for B is then found using the equation $dR/dt = -a_1 B$.

- (c) Now find the arbitrary constants by solving the simultaneous equations for $R(0) = r_0$ and $B(0) = b_0$, when $t = 0$. The final solution is given in the text in Section 5.7.
- (d) Using Maple or MATLAB (with symbolic toolbox) check the solution above. Use the `dsolve` command or just substitute back into the original differential equations.

(Further details about methods for solving second-order differential equations, in particular for differential equations with constant coefficients, as used here, can be found in Appendix A.5.)

5.21. Spread of malaria by mosquitoes. With the disease malaria, in humans, the disease is carried by mosquitoes who also cannot infect each other. Infectious mosquitoes can only infect susceptible humans and infected humans can only infect susceptible mosquitoes when they are bitten by a susceptible mosquito. Assume the rate of transmission is proportional to both numbers of mosquitoes and number of humans for transmission in both directions, and assume once infected, both humans and mosquitoes never recover.

Ignoring any births and deaths develop a mathematical model for susceptible and infected humans $S_h(t)$, $I_h(t)$, and susceptible and infected mosquitoes $S_m(t)$, $I_m(t)$.

5.22. Spread of a religion. A new religion is spreading through a community in a remote country. The community is made up of unbelievers (with numbers denoted by $U(t)$), converts (numbers $C(t)$) and missionaries (numbers $M(t)$). Assume only contacts between missionaries and unbelievers result in an unbeliever becoming a convert. A constant proportion of converted each year become ordained as missionaries.

Formulate a system of differential equations for these populations. Your model should have the property that the total population remains constant over time. Births and deaths may be ignored and relapses to unconverted of either converts or missionaries may be neglected.

5.23. Predator-prey with protection of young prey. Formulate a mathematical model for a predator-prey system where the prey protect their young from the predators. The model should have three dependent variables: $X_1(t)$, the juvenile prey numbers; $X_2(t)$, the adult prey numbers; and $Y(t)$, the predator numbers. In your model assume the juvenile prey are completely sheltered from the predators.

5.24. Diseases with carriers. Develop a model for an infectious disease where there is immunity for only some of those who recover; others ‘recover’ to become permanent carriers, who can still cause infections. Thus susceptibles, $S(t)$, may be infected by either infectious individuals, $I(t)$, or carriers, $C(t)$. A carrier can infect others at a reduced rate compared to infectious individuals but shows no symptoms.

- (a) Give a suitable compartment diagram for this model.
- (b) Assume there is a fixed proportion q of those recovering from the infection become carriers. Assume transmission rates β_1 for normal infectives and β_2 for carriers and assume that individuals remain infective for a mean time γ^{-1} . Give equations for the number of susceptibles, $S(t)$, the number of infectious, $I(t)$, the number of carriers, $C(t)$ and the number of recovered who are immune, $R(t)$.
- (c) Give at least one example of an infectious disease that could be modelled by the equation you have developed.

5.25. Basic reproduction number. The basic reproduction number, R_0 , for an infectious disease is defined as:

the number of secondary infections caused by a single infective in a completely susceptible population, over the time that individual is infectious.

We found in this chapter that the basic reproduction number for the SIR model, with $S' = -\beta SI$, and $I' = -\beta SI - \gamma I$, given by

$$R_0 = \frac{\beta s_0}{\gamma}$$

where β is the transmission coefficient and γ is the recovery rate.

- (a) What would R_0 be for a chronic disease, where there is no recovery from the disease?
- (b) Consider an SEIR model (see Question 5), for a disease with a latent period, where once infected, an individual goes into an exposed compartment for a time σ^{-1} before becoming infectious. Argue that the expression for R_0 for this model is the same as for the SIR model.
- (c) Develop equations for an SEIR model which includes natural deaths, where each class has the same per-capita death rate a .
- (d) How would the expression for R_0 change for the model in (c)? Give an interpretation of this.

5.26. Farmers, bandits and soldiers. Read over the case study Section 5.8 for a model of farmer, bandit and soldier population in ancient China.

- (a) Write a Maple or MATLAB program to reproduce the graph in Figure 5.14.
- (b) Change the parameter c from $c = 0.4$ to $c = 0.8$ and plot the graph. Describe the changes and interpret.

(3) *Exercitii de lucru cu caiete*
Experimentul si exercitiile de lucru cu caiete sunt o parte esentiala din programul de
matematica. Exercitiile pot fi realizate individual sau in grupuri de 2-3 elevi. Ele pot fi de
tipul (1), de tipul (2) sau de tipul (3). Exercitiile de lucru cu caiete pot fi realizate in
sunt elaboratii specialelor si principale exercitii sau sunt realizate in secventa
exercitiilor de lucru cu caiete.

(4) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care permit elevilor sa realizeze in mod
individual sau in grupuri de 2-3 elevi, sau in grupuri de 4-5 elevi sau in grupuri de
6-7 elevi. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.
Exercitiile de lucru cu caiete pot fi de tipul (1), de tipul (2) sau de tipul (3).

(5) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care pot fi realizate in secventa exercitiilor
de lucru cu caiete. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.

(6) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care pot fi realizate in secventa exercitiilor
de lucru cu caiete. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.

(7) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care pot fi realizate in secventa exercitiilor
de lucru cu caiete. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.

(8) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care pot fi realizate in secventa exercitiilor
de lucru cu caiete. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.

(9) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care pot fi realizate in secventa exercitiilor
de lucru cu caiete. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.

(10) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care pot fi realizate in secventa exercitiilor
de lucru cu caiete. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.

(11) *Exercitii de lucru cu caiete*
Exercitiile de lucru cu caiete sunt exercitii care pot fi realizate in secventa exercitiilor
de lucru cu caiete. Aceste exercitii sunt realizate in secventa exercitiilor de lucru cu caiete.

Chapter 6

Phase-plane analysis

For pairs of coupled first-order differential equations it can be useful to use the chain rule to eliminate time and reduce the pair of equations to a single first-order differential equation. The graph of all the solutions to this single equation is called the phase-plane. We apply this method to the basic models from Chapter 5. Consequently, we are able to draw some general conclusions about the models and confirm the observations which were made using only the numerical solutions.

6.1 Introduction

In the previous chapter, we developed models for interacting populations which resulted in pairs of nonlinear, coupled differential equations and we then used Maple to solve the equations numerically. However, each solution was for a particular pair of initial conditions and a particular combination of the parameters. Clearly there are infinitely many such combinations and we need some other tools to understand the system behaviour as a whole. What would be valuable is to understand how any changes in the initial conditions or values of the parameters might affect these solutions and the subsequent system dynamics.

We see how to gain insight into the behaviour of the solutions to these systems by finding the equilibrium points (points corresponding where the derivatives are zero) and eliminating time from the differential equations (by converting the coupled differential equations to a single first-order differential equation). Further, we are interested in the long-term behaviour of the systems. We ask questions such as: does a population die out or settle down to some fixed size, or, can a fatal disease wipe out a population completely or could it recur periodically? The techniques we develop to deal with such questions are designed to investigate the behaviour of solutions to the systems and require an understanding of equilibrium points and the phase-plane. First, we introduce these techniques using a very simple example system and then apply them to the systems developed in the previous chapter.

Consider the coupled pair of first-order (linear) differential equations

$$\frac{dX}{dt} = Y, \quad \frac{dY}{dt} = -X. \quad (6.1)$$

Equilibrium points

Equilibrium points correspond to solutions of a coupled system of differential equations where the solutions are constant, i.e. where $dX/dt = 0$ and $dY/dt = 0$, simultaneously. Hence, for the differential equations (6.1) we obtain

$$Y = 0, \quad X = 0$$

and so $(X, Y) = (0, 0)$ is the only equilibrium solution.

Trajectories and the phase-plane diagram

Let us consider the (X, Y) -plane: this is called the *phase-plane*. Dividing the plane into four quadrants in the manner illustrated in Figure 6.1, we have, in the first quadrant where $X > 0$ and $Y > 0$, that $dX/dt = Y > 0$ and $dY/dt = -X < 0$. Thus $X(t)$ is increasing and $Y(t)$ is decreasing and we obtain a direction vector for any solution in that quadrant, given by the arrow in Figure 6.1. Each quadrant can be considered in the same manner. We can thus infer that the solutions, that is the *phase-plane trajectories*, move in a clockwise direction.

Using the chain rule

We note that neither of the differential equations involves the time variable t explicitly. We mean by this that t does not appear on the RHS of either equation. (Of course, the solutions will be time dependent, since the derivatives on the LHS are with respect to time.) This means that we should be able to eliminate the time variable and find an expression, independent of t , which relates X and Y . Alternatively stated, we express Y as a function

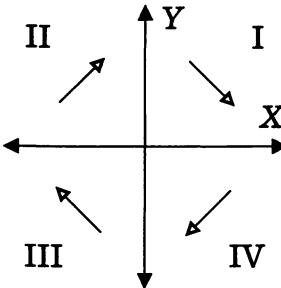


Figure 6.1: Direction vectors for the trajectories in the phase-plane cf example equations (6.1).

of X . That is, we are making X the independent variable where it was previously a variable dependent on t .

An expression for the chain rule is

$$\frac{dY}{dt} = \frac{dY}{dX} \frac{dX}{dt}$$

which gives the derivative of Y with respect to t in terms of the derivative of Y with respect to X and the derivative of X with respect to t . Dividing by the derivative of X with respect to t gives

$$\frac{dY}{dX} = \frac{dY/dt}{dX/dt}. \quad (6.2)$$

We substitute from the coupled pair of differential equations (6.1) into (6.2) to give

$$\frac{dY}{dX} = -\frac{X}{Y}. \quad (6.3)$$

Here we have a first-order differential equation with Y a function of X .

It is not always a simple matter to solve the differential equation; however, in this case the differential equation (6.3) is separable since we can write it in the form

$$Y \frac{dY}{dX} = -X. \quad (6.4)$$

Example 6.1: Solve the differential equation (6.4) using the separation of variables technique.

Solution: Integrate both sides with respect to the independent variable X to obtain

$$\int Y \frac{dY}{dX} dX = \int -X dX.$$

By the substitution rule for integration this simplifies to

$$\int Y dY = \int -X dX.$$

Carrying out both integrations gives

$$\frac{1}{2}Y^2 = -\frac{1}{2}X^2 + C$$

where C is the constant of integration. Multiplying throughout by 2, and rearranging the terms, we have that

$$X^2 + Y^2 = K$$

with $K = 2C$. The value of K will be determined by the initial conditions.

This solution equation is that of a circle. It describes the paths traced out by the (X, Y) pair over time, depending on the initial values or starting conditions. These are the exact solutions to the phase-plane trajectories.

Interpretation of the phase-plane

As an interpretation of these trajectories, if a system has initial values x_0 and y_0 then the system starts at the point (x_0, y_0) in the phase-plane and, as time evolves, it traces out the trajectory curve (in this case a circle) in a clockwise direction as was established in Figure 6.1. At any subsequent time the values of $X(t)$ and $Y(t)$ will be the coordinates of this trajectory. Since, for this example, the trajectory is a circle the motion is repeated continuously in time. In fact, this would be the case for any closed trajectory.

To see how the system evolves in time we would normally need an exact solution of the original coupled equations giving both Y and X as functions of time. Often this is not possible. Nevertheless, we can still use the chain rule to infer useful information about the system. We see how to do this in the following sections which examine the systems of differential equations for populations, developed in the previous chapter.

In essence, the idea of phase-plane analysis is to draw a phase-plane diagram together with the phase-plane trajectories in order to understand some general features of the system. (The terminology arises from the use of these ideas to calculate planetary orbits.) If the differential equations are sufficiently simple, we can go further and use the chain rule to eliminate time. In this way, we may obtain an exact expression relating the two dependent variables which describes the trajectory path.

The phase-plane diagram is useful for determining the behaviour of solutions for a variety of initial conditions. In the above example, we saw that all solutions of the differential equations have phase-trajectories which are circles. This means that the plots for both variables as functions of time must be oscillations, which follows because X and Y must always return to their original values as we move along the trajectory. Furthermore, as the initial point approaches the equilibrium point the amplitude of the oscillation is reduced, with the equilibrium point itself corresponding to a solution which is constant in time.

Note, however, that in this procedure we have lost information about time. This was the price for reducing the coupled, first-order differential equations to a single first-order equation.

Summary of skills developed here:

- Understand the concept of equilibrium solutions.
- Establish the directions of trajectories.
- Use the information on equilibrium points and trajectory directions to draw a phase-plane.
- Understand how the chain rule can eliminate time and reduce a coupled pair of differential equations to a single differential equation.

6.2 Phase-plane analysis of epidemic model

Previously, in Section 5.2, we developed a model for an epidemic of an infectious disease. We now use the chain rule and some analysis to prove that the disease, described by this model, can never infect the entire population.

Review of the model

The epidemic model, developed in Section 5.2, assumed the population N was divided into susceptibles, denoted by $S(t)$, infectives, denoted by $I(t)$ and removals $N - S(t) - I(t)$. We assumed the disease to confer life-long immunity and we neglected to include natural births and deaths. The model pair of differential equations we obtained was

$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I. \quad (6.5)$$

The parameter β is the transmission coefficient and γ is the recovery (or removal) rate.

For the parameter values we considered in the example of Section 5.2, our results indicated that the number of infectives always tended to zero while the number of susceptibles approached some finite number. It seemed there were always certain individuals who never contracted the disease. It is interesting to speculate whether, according to our model, this is always the case. We use the chain rule below to show this is indeed so.

A picture of the phase-plane plot can be obtained by solving the differential equations numerically. This allows us to determine the behaviour of the model for a range of initial conditions, but for a fixed set of parameter values. Some Maple code to give the phase-plane diagram in Figure 6.2 is given in Listing 6.1. Similarly, some MATLAB code for a similar diagram is given in Listing 6.2.

Listing 6.1: Maple code: c_ps.epidemic.txt

```
> restart:with(plots):with(DEtools):
> unprotect(gamma): gamma :='gamma';
> interface(imaginaryunit=i); I='i';
> beta:=2.18*10^(-3): gamma:=0.44: beta
> de1:=diff(S(t),t)=beta*S(t)*I(t);
> de2:=diff(I(t),t)=beta*S(t)*I(t)-gamma*I(t);
> inits:=[0,762,1],[0,600,20],[0,400,50]:
> plot1:=DEplot([de1,de2],[S(t),I(t)],t=0..30,
  [inits],scene=[S,I],stepsize=0.1,dirgrid=[10,10],
  arrows=medium,linecolour=black):
> display(plot1);
```

Listing 6.2: MATLAB code: c_ps_epidemic.m

```
function c_ps_epidemic
global beta gamma;

tend = 15; %the end time
beta=2.0*10^(-3); gamma=0.44;
u0vec = [762, 600, 400]; % make a matrix of 3 ICs
                           1, 20, 50];
u0size = size(u0vec); % size of the matrix of ICs
numICs = u0size(2); % number of ICs

for k = 1:numICs %loop over each case of ICs
    u0 = u0vec(:, k); %extract the kth column of matrix
```

```

[tsol, usol] = ode45(@rhs, [0, tend], u0); %solve the DE
Ssol = usol(:, 1); Isol = usol(:, 2);
plot(Ssol, Isol); hold on; %plot each trajectory
end
%produce arrows (see appendix for code for this function)
c_dirplot(@rhs, 0, 800, 0, 300, 10);
axis([0,800, 0, 300]);
function udot = rhs(t, u)
global beta gamma;
S=u(1); I=u(2);
Sdot = -beta*S*I;
Idot = beta*S*I - gamma*I;
udot = [Sdot; Idot];

```

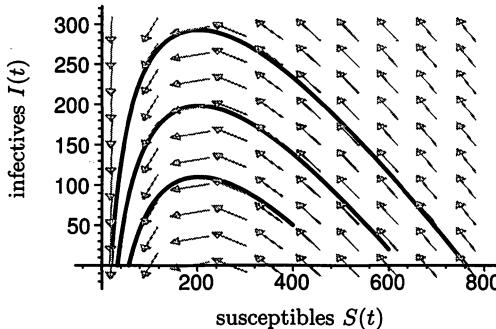


Figure 6.2: Maple generated phase-plane diagram, with $\beta = 2.18 \times 10^{-4}$ and $\gamma = 0.44$ with a variety of initial values. This shows the behaviour for the given parameter values but for different initial conditions.

In Figure 6.2 we follow the curve (a trajectory) from the initial values in the direction of the arrows. The arrows come from the direction field as determined by the differential equation for each value of S and I . We see from this diagram that as $I \rightarrow 0$ that S does not tend to zero — there are some susceptibles left uninfected. Of course this conclusion is only valid for the given parameter values for β and α . We can form conclusions for general values of parameter values, in this model, to obtain analytic expressions for the trajectories.

Applying the chain rule

For the simple differential equations comprising this model it is possible to obtain an analytic expression for the trajectories. We can do this by eliminating time (using the chain rule) which results in a differential equation involving the variables I and S . The solution to the differential equation establishes a relation between these two variables, as derived in the following example.

Example 6.2: Use the chain rule to find I in terms of S , given that the initial number of susceptibles is s_0 and the initial number of contagious infectives is i_0 .

Solution: Using the chain rule

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt}.$$

Substituting from the differential equations (6.5) we eliminate the time variable, obtaining the

single differential equation

$$\frac{dI}{dS} = \frac{\beta SI - \gamma I}{-\beta SI}$$

which simplifies to

$$\frac{dI}{dS} = -1 + \frac{\gamma}{\beta S}. \quad (6.6)$$

This differential equation relates the number of infectives I to the number of susceptibles S but does not retain explicit information about time.

The differential equation (6.6) is a first-order separable differential equation of a trivial kind and to solve it we have only to integrate both sides with respect to S . The solution is

$$I = -S + \frac{\gamma}{\beta} \ln(S) + K \quad (6.7)$$

where K is an arbitrary constant of integration.

The initial numbers of susceptibles and infectives are s_0 and i_0 , respectively, and hence the initial condition for the differential equation (6.6) is

$$I(s_0) = i_0.$$

Applying this to the general solution (6.7) gives an equation for K from which we deduce that

$$K = i_0 + s_0 - \frac{\gamma}{\beta} \ln(s_0).$$

Sketching the phase-plane trajectories

The solution to the differential equation (6.6), given by (6.7), is not very complicated so it is worthwhile to try and obtain a general sketch of I versus S .

Example 6.3: Determine and sketch the family of phase-plane curves given by (6.7).

Solution: To find turning points we set $dI/dS = 0$. That is

$$-1 + \frac{\gamma}{\beta S} = 0.$$

Solving for S gives

$$S = \frac{\gamma}{\beta},$$

corresponding to a potential turning point. Since there is only one potential turning point we do not need to find a second derivative to determine whether it is a maximum, minimum or inflection point. Instead we can use the values of the function at $S = 0$ and $S \rightarrow \infty$.

As $S \rightarrow 0$ we see that $I \rightarrow -\infty$ since $\ln(S) \rightarrow -\infty$. As $S \rightarrow \infty$ the limit is harder to work out. Note that $\ln(S) \rightarrow \infty$ and $-S \rightarrow -\infty$ as $S \rightarrow \infty$. To determine the limit let us consider the derivative dI/dS . Note that from (6.7), $dI/dS \rightarrow -1$ as $S \rightarrow \infty$. This implies that $I \rightarrow -\infty$ since S grows faster than $\ln S$ for large S . (A formal method for showing this is to use l'Hôpital's rule.)

Since $I \rightarrow -\infty$ as $S \rightarrow 0$ and $S \rightarrow \infty$ then the point corresponding to $S = \gamma/\beta$ must be a local maximum. Hence the curve must cross the positive S -axis twice, for sufficiently large K . This is sketched in Figure 6.4.

Direction of trajectories

The direction of trajectories in the phase-plane is determined from the differential equations, as below.

Example 6.4: Determine the directions of trajectories in the phase-plane.

Solution: From $dS/dt = -\beta SI$ we see that for $S > 0$ and $I > 0$, dS/dt is always negative. This means that $S(t)$ is always decreasing. Similarly, from the other differential equation, dI/dt is positive provided $I(\beta S - \gamma) > 0$. Thus $dI/dt > 0$ if $S > \gamma/\beta$ and negative if $S < \gamma/\beta$. The phase-plane, for positive S and I , is therefore divided into two regions, as shown in Figure 6.3.

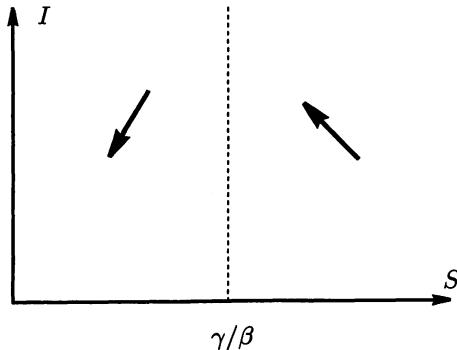


Figure 6.3: Diagram showing directions of trajectories in the phase-plane for the epidemic model.

The resulting sketch of Figure 6.4 agrees with the earlier theory, which indicated (see Figure 6.3) that from any initial condition (s_0, i_0) , the trajectory of $S(t)$ decreases continuously.

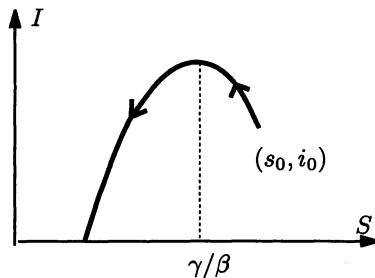


Figure 6.4: General sketch of the phase-plane diagram, illustrating a typical phase-plane curve with the direction in which the curve is traced indicated by the arrow.

Interpretations

A useful conclusion from Figure 6.4 concerns the number of susceptibles left after the disease has run its course. We see that it is impossible for the disease to infect all of the susceptibles. (Although, the number of susceptibles may become sufficiently small for it to be regarded as effectively zero.)

We can interpret some interesting results from Figure 6.4. Note that if s_0 is greater than the critical value γ/β then the number of infectives must increase for a time before it decreases, whereas if $s_0 < \gamma/\beta$ then the disease dies out. Thus for an epidemic to occur, in which the number of infectives increases from an initial number i_0 , there needs to be at least some threshold number of susceptibles present prior to the outbreak, that is $s_0 > \gamma/\beta$. The combination $\beta s_0 / \gamma$ is known as the *basic reproduction ratio*, often denoted by the symbol R_0 , as introduced in Section 5.2. It is a measure of the number of infections caused by a single infective in a fully susceptible population. This is an important quantity in epidemiology: a basic reproduction ratio of less than one indicates the propensity of the disease to die out. This can be exploited to determine control strategies to reduce the impact and even eradicate an infectious disease.

Chapter 8 includes a case study where different strategies are tested in a bid to eradicate bovine tuberculosis in possums in New Zealand. However, the analysis in this case is a little different from models of diseases in humans; for the possums (an introduced and destructive feral pest) we are unconcerned if the population is eradicated in the process!

Summary of skills developed here:

- Use the chain rule to eliminate time from any epidemic model.
- Further practice at graph sketching.
- Use this model (and extensions) to examine vaccination or other disease eradication strategies.

6.3 Analysis of a battle model

We apply the chain rule to the coupled system of differential equations for the Lanchester battle model developed in Section 5.7. This reduces the coupled equations to a single first-order differential equation. By solving this, we obtain an expression which relates the numbers of soldiers in one army to the numbers of those in the other. This in turn provides a more general understanding of the system dynamics.

Review of the model

In the previous chapter we developed a model describing a battle between two armies. The model resulted in the pair of coupled differential equations, where $R(t)$ and $B(t)$ denote the numbers of soldiers in the red army and blue army, respectively. We assumed both armies to use only aimed fire. We derived the pair of differential equations

$$\frac{dR}{dt} = -a_1 B, \quad \frac{dB}{dt} = -a_2 R, \quad (6.8)$$

where a_1 and a_2 are positive constants (attrition coefficients). Recall that these attrition coefficients can also be expressed in terms of firing rates and probabilities of hitting a target.

The phase-plane diagram from Maple

We can use Maple to plot some typical phase-plane trajectories, see Listing 6.3, or MATLAB, see Listing 6.4. We let $a_1 = 0.0544$ and $a_2 = 0.0106$. The phase-plane trajectories corresponding to three different initial conditions are shown in Figure 6.5.

Listing 6.3: Maple code: c_ps_combat.txt

```
> restart:with(plots):with(DETools):
> a[1]:=0.0544:a[2]:=0.0106:
> de1:=diff(R(t),t)=-a[1]*B(t);
> de2:=diff(B(t),t)=-a[2]*R(t);
> inits:=[R(0)=66,B(0)=18],[R(0)=45,B(0)=18],[R(0)=30,B(0)=18]:
> plot1:=DEplot([de1,de2],[R,B],t=0..30,[inits],scene=[R,B],linecolour=black):
> display(plot1);
```

Listing 6.4: MATLAB code: c_ps_combat.m

```
function c_cp_predprey
global a1 a2;

tend = 30;%se the end time to run the simulation
a1=0.0544; a2=0.0106;
u0vec = [66 45 30; % make a matrix of 3 ICs
          18 18 18];
u0size = size(u0vec);
numICs = u0size(2);

% plot phase-plane curve for each Init Cond
for k = 1:numICs
    u0 = u0vec(:,k); %choose kth col for init cond
    [tsol, usol] = ode45(@rhs, [0, tend], u0);
    Rsol = usol(:, 1); Bsol = usol(:, 2);
    plot(Rsol, Bsol); hold on;
end
% makearrows, see Appendix for this function
c_dirplot(@rhs, 0, 70, 0, 20, 10);
axis([0,70, 0, 20]);

function udot = rhs(t, u)
global a1 a2;
R=u(1); B=u(2);
Rdash = -a1*B;
Bdash = -a2*R;
udot = [Rdash; Bdash];
```

Equilibrium points

Typically we set the two rates of change to zero and solve the equations simultaneously. In this case, the exercise is trivial and the only equilibrium solution is $(R, B) = (0, 0)$.

Direction of trajectories

The direction of the trajectories can be obtained from the original differential equations (6.8). This is done in the following example.

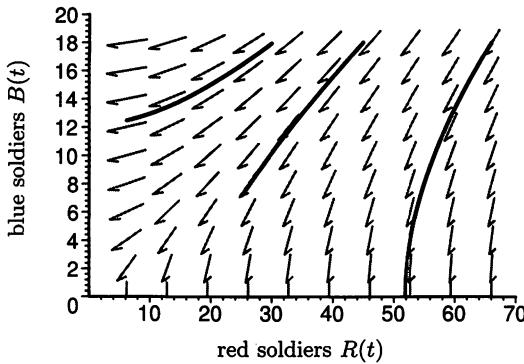


Figure 6.5: Phase-plane trajectories generated by Maple using parameter values $a_1 = 0.0544$ and $a_2 = 0.0106$ with various initial conditions.

Example 6.5: Determine possible directions of phase-plane trajectories in the phase-plane.

Solution: From these equations, since R and B are both positive, and since the constants a_1 and a_2 are positive, then dX/dt is always negative and dY/dt is always negative. Hence $R(t)$ and $B(t)$ are always decreasing with time.

Because of the directions in the phase-plane, it is clear that the trajectory must head towards one of the axes. Since the only equilibrium point is the origin, the trajectory (unless it passes through the origin) must cross the R or B axis in finite time, as the rate of change is not zero there. This means that the battle would be over in finite time. To determine where the trajectory intersects the axes we need to determine the explicit form of the trajectories, and fortunately we can do this using the chain rule.

Applying the chain rule

We use the chain rule as before to eliminate the time variable t . Substituting from the differential equations (6.8) we obtain

$$\frac{dB}{dR} = \frac{dB/dt}{dR/dt} = \frac{a_2}{a_1} \frac{R}{B}. \quad (6.9)$$

Thus we obtain a single first order differential equation which relates B and R , but does not involve t . The solution is given by the following example.

Example 6.6: Solve the differential equation (6.9).

Solution: The solution of the first-order differential equation (6.9) is obtained by separating the variables and then integrating with respect to the independent variable R . This gives

$$\int B \frac{dB}{dR} dR = \int \frac{a_2}{a_1} R dR.$$

Using the substitution rule for integration, the LHS can be converted to an integral involving the variable B ,

$$\int B dB = \int \frac{a_2}{a_1} R dR.$$

Carrying out the integrations gives the equation

$$\frac{1}{2} B^2 = \frac{a_2}{2a_1} R^2 + C,$$

where C is an arbitrary constant of integration. Multiplying both sides of the equation by 2 we obtain

$$B^2 = \frac{a_2}{a_1} R^2 + K, \quad (6.10)$$

where $K = 2C$ is also an arbitrary constant.

Applying the initial conditions $R(0) = r_0$ and $B(0) = b_0$ we obtain

$$b_0^2 = \frac{a_2}{a_1} r_0^2 + K$$

so that

$$K = b_0^2 - \frac{a_2}{a_1} r_0^2. \quad (6.11)$$

If we suppose the battle is fought until one of the sides is wiped out, then we can use (6.10) to determine who wins the battle. We can also see this graphically by plotting the trajectories described by (6.10).

Returning to the phase-plane

Let us now examine the solution (6.10) graphically. From (6.10) we have

$$B = \sqrt{\frac{a_2}{a_1} R^2 + K} \quad (6.12)$$

where the positive square root is the only valid one (as it only makes sense to have a positive number of soldiers), and K is a constant which is determined by the initial conditions.

To sketch the family of phase-plane trajectories satisfying (6.12) we use standard graph sketching techniques. First we look for possible turning points, then we examine the asymptotic behaviour for large R and finally, look for intercepts on the axes.

Example 6.7: Sketch the phase-plane trajectories.

Solution: For turning points we require $dB/dR = 0$. Rather than differentiate (6.12) directly we can use the differential equation (6.9). For R and B both positive, dB/dR is positive and so B is always an increasing function of R and there are no turning points.

We can also check the behaviour of B as R becomes large. For sufficiently large R the term R^2 will always be large compared with K . So for large R the trajectory asymptotes to $B = \sqrt{a_2/a_1} R$, which is the equation of a straight line.

We also calculate where the curves cross the axes. For B -intercepts, we set $R = 0$ and then solve for B to obtain the intercept $B = \sqrt{K}$, for $K > 0$. This only makes sense if K is positive. Similarly, for R -intercepts, we set $B = 0$ and then solve for R to obtain the intercept $R = \sqrt{-a_1 K / a_2}$, for $K < 0$. There is a real solution only if K is negative. We thus get a different family of curves, depending on whether K is positive or negative.

Putting together the information that B is an increasing function of R , the fact that the curves all approach a straight line asymptotically, and the known values of the intercepts, allows us to infer the general form of the phase-plane curves. A sketch of these is given below in Figure 6.6.

For a given initial condition (r_0, b_0) we start at that point on one of the curves. As time increases we move along the curve in a direction towards one of the axes (since the number of soldiers can only decrease in this model).

From Figure 6.6 we see that if $K > 0$ then $R \rightarrow 0$ and the blue army wins. However, if $K < 0$ then $B \rightarrow 0$ and the red army wins.

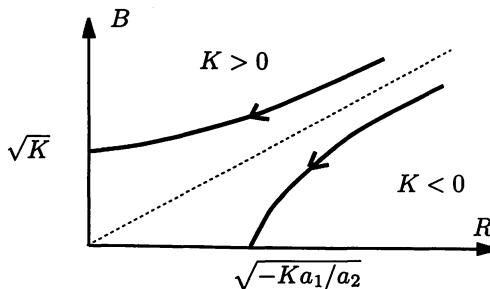


Figure 6.6: Typical phase-plane trajectories for the combat model with two armies exposed to each other's fire. The directions along the trajectories are indicated.

Discussion

An interesting use of this battle model is for the analysis of tactics. Although the model studied here is probably too simple to represent most real battles, it is still useful in understanding why some battle tactics work. One such example is the 'divide and conquer' strategy. In this strategy you divide your enemy's force, while not dividing your own, so that you fight two battles against two smaller forces. It has been used by many famous generals including Napoleon. Why should the outcome of this be any different from fighting a single battle against the total force? The following example illustrates the reason.

Example 6.8: For simplicity let us assume that both armies have equal attrition coefficients $a_1 = a_2$. Let us suppose the red army has 10 000 soldiers initially and the blue army has 8000. Determine who wins if (i) there is one battle between the two armies, and (ii) there are two battles: the first with half the red army against the entire blue army and the second with the other half of the red army against the blue army survivors of the first battle.

Solution: If there is only one battle the red army wins since $a_1 = a_2$ and $r_0 > b_0$. In other words, $a_1 K = a_1 \times (8 \times 10^3)^2 - a_2 \times (10 \times 10^3)^2 = -36 \times 10^6 a_1$ so $K < 0$ which is the condition for the red army to win.

Instead, suppose that half the red army meets the entire blue army in a first battle and then the remaining half of the red army meets the remaining blue army soldiers in a second battle. For the first battle, we can calculate the number of blue soldiers left. For this calculation we use equation (6.10) with $r_0 = 5000$, $b_0 = 8000$ and obtain the surviving number of blue soldiers (as $R \rightarrow 0$)

$$B \rightarrow (b_0^2 - r_0^2)^{1/2} = (64 \times 10^6 - 25 \times 10^6)^{1/2} \approx 6.25 \times 10^3.$$

In the second battle we now have $r_0 = 5 \times 10^3$ and $b_0 = 6.25 \times 10^3$. Clearly, the blue army now wins the second battle since it has the greater numbers (and $a_1 = a_2$).

This example demonstrates how an initially inferior army is able to defeat a superior army by forcing the superior army to engage in two separate battles. It illustrates the important military concept of concentrating forces and provides an indication of the role of this type of theory in the design of battle tactics and strategies.

Summary of skills developed here:

- Apply the chain rule method to coupled systems.
- Draw and interpret phase-plane diagrams for systems of differential equations.
- Extend the results of this and more complicated battle models to give tactical advice.

6.4 Analysis of a predator-prey model

We explore the Lotka-Volterra predator-prey equations with the aim of gaining a more general understanding of the dynamics using phase-plane analysis. We first obtain the phase-plane diagram for a specific set of parameters, using **Maple** to generate a numerical solution, and thereafter we infer what the phase-plane looks like for a general parameter set.

Review of model

In Section 5.3, we formulated a simple model for predator-prey interactions. The resulting differential equations were

$$\frac{dX}{dt} = \beta_1 X - c_1 XY, \quad \frac{dY}{dt} = -\alpha_2 Y + c_2 XY. \quad (6.13)$$

Recall that X denotes the prey population and Y denotes the predator population. For the positive constant parameters we had c_1 and c_2 as the interaction parameters, β_1 as the prey per-capita birth rate and α_2 as the predator per-capita death rate.

We found these equations produced oscillations in time for the various initial conditions and parameter combinations considered. Here we address the question of whether this model always predicts oscillations for any (positive) values of the parameters.

The phase-plane diagram from Maple

We let $\beta_1 = 1$, $\alpha_2 = 0.5$, $c_1 = 0.01$ and $c_2 = 0.005$, which are the same values for the parameters we used in Section 5.3. We can use **Maple** or MATLAB to solve the differential equations numerically and plot some phase-plane trajectories for this set of parameter values for some different initial conditions; see Listing 6.5 and Listing 6.6.

Listing 6.5: Maple code: c_ps_predprey.txt

```
> restart:with(plots):with(DEtools):
> beta[1]:=1.0:alpha[2]:=0.5:c[1]:=0.01:c[2]:=0.005:
> de1:=diff(X(t),t)=beta[1]*X(t)-c[1]*X(t)*Y(t);
> de2:=diff(Y(t),t)=-alpha[2]*Y(t)+c[2]*X(t)*Y(t);
> inits:=[0,100,80],[0,50,50],[0,100,170];
> DEplot([de1,de2],[X,Y],t=0..50,[inits],scene=[X,Y],linecolour=black,stepsize=0.1);
```

Listing 6.6: MATLAB code: c.ps.predprey.m

```

function c_ps_predprey
global beta1 alpha2 c1 c2;

beta1=1.0; alpha2=0.5; c1=0.01; c2=0.005;
tend = 20;%the end time to run the simulation
% as a matrix
u0vec = [100, 50, 100; %make a matrix of ICs
          80, 50, 170];
u0size = size(u0vec); %size of the matrix
numICs = u0size(2); % extract number of ICs

for k = 1:numICs %loop over each IC
    u0 = u0vec(:, k); %extract the kth column
    [tsol, usol] = ode45(@rhs, [0, tend], u0);
    Xsol = usol(:, 1); Ysol = usol(:, 2);
    plot(Xsol, Ysol);
    hold on;
end
% makearrows, see Appendix for this function
c_dirplot(@rhs, 0, 800, 0, 300, 11);
axis([0,300, 0, 200]);

function udot = rhs(t, u)
global beta1 alpha2 c1 c2;
X = u(1); Y=u(2);
Xdot = beta1*X - c1*X*Y;
Ydot = -alpha2*Y + c2*X*Y;
udot = [Xdot; Ydot];

```

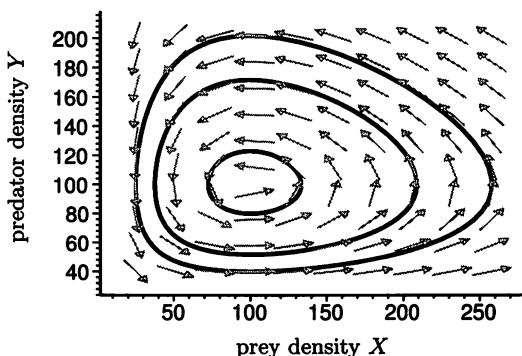


Figure 6.7: Maple generated phase-plane plot for the Lotka-Volterra equations. The parameter values $\beta_1 = 1$, $c_1 = 0.01$, $\alpha_2 = 0.5$ and $c_2 = 0.005$ have been used with various initial conditions.

The fact that the curves which appear in this phase-plane diagram of Figure 6.7 are closed is very significant. (Closed trajectories do not occur in all phase-plane diagrams.) Recall that as time evolves we travel along the curve. From the arrows in Figure 6.7 (the direction field) the direction of travel is in the anticlockwise direction. Thus, if initially the populations are x_0 and y_0 then as we travel along the curve we eventually return to the initial values x_0 and y_0 . In fact every point on the curve will be repeated each time we make a complete circuit. These closed curves imply that both populations must follow *periodic cycles*. This is consistent with the time-dependent graphs obtained in Section 5.3.

We now see what we can deduce about the model for general parameter values. We begin by finding a general expression for the equilibrium points. Then we analyse the directions which the trajectories follow in the phase-plane.

Equilibrium populations

As we saw from Figure 6.7, for different initial values we get different phase-plane trajectories enclosed within one another. We can imagine the trajectories shrinking to a single point. This point would correspond to equilibrium populations, where the populations do not change with time. The equilibrium solutions correspond to those values where the rates of change are zero. These are the constant solutions of the differential equations (6.13).

We can find these values analytically, as the following example shows, by solving a pair of simultaneous equations. When solving simultaneous nonlinear equations it is important to ensure you have found all the possible solutions and to this end it is often useful to factorise, as in the following example.

Example 6.9: Find the equilibrium solutions of the differential equations (6.13).

Solution: We set $dX/dt = 0$ and $dY/dt = 0$ in (6.13) and obtain the equations

$$\beta_1 X - c_1 XY = 0, \quad -\alpha_2 Y + c_2 XY = 0.$$

To find solutions we first write the two simultaneous equations in factored form as

$$X(\beta_1 - c_1 Y) = 0, \tag{6.14}$$

$$Y(-\alpha_2 + c_2 X) = 0. \tag{6.15}$$

From (6.14) there are two possible solutions: $X = 0$ or $\beta_1 - c_1 Y = 0$. We need to look at each case. Recall that the parameters β_1 , α_2 , c_1 and c_2 are positive (non-zero) constants.

If $X = 0$, then substituting this into (6.15) gives $-\alpha_2 Y = 0$ so that $Y = 0$. This gives one possible solution of both equations at $(X, Y) = (0, 0)$.

Taking the other case, $\beta_1 - c_1 Y = 0$, then $Y = \beta_1/c_1$. Substituting this into (6.15) gives $-\alpha_2 + c_2 X = 0$, for which the only solution is $X = \alpha_2/c_2$. Hence we obtain a second solution to both equations $(X, Y) = (\alpha_2/c_2, \beta_1/c_1)$.

To ensure we have all the solutions, we should also solve the second equation and substitute the solution into the first, but this yields exactly the same two solutions as we obtained above.

Summarising, we have obtained two equilibrium solutions

$$(X, Y) = (0, 0) \quad \text{and} \quad (X, Y) = \left(\frac{\alpha_2}{c_2}, \frac{\beta_1}{c_1} \right).$$

Another way of being sure we have all the solutions is to think of the problem of finding the solutions geometrically. Solving the equation $dX/dt = 0$ gave $X = 0$ and $Y = \beta_1/c_1$ which represents the equations of two lines, which we denote as L_0 and L_1 , in the (X, Y) -plane. Similarly, $dY/dt = 0$ yields two lines, L_2 and L_3 , given by $X = \alpha_2/c_2$ and $Y = 0$, respectively. The equilibrium points occur where the pair L_0 and L_1 intersect with L_2 and L_3 (not L_0 with L_1 , or L_2 with L_3). These lines (or curves) are known as *nullclines* and their points of intersection are illustrated in Figure 6.8.

We can also use **Maple** or **MATLAB** (with symbolic toolbox) to solve the equations for the equilibrium solutions. The **Maple** code is given in Listing 6.7.

Listing 6.7: Maple code: c_ps_predprey_eqmpts.txt

```
> restart;
> eq1 := beta[1]*X(t) - c[1]*X(t)*Y(t);
> eq2 := -alpha[2]*Y(t) - c[2]*X(t)*Y(t);
> solve({eq1,eq2}, {X(t),Y(t)});
```

Consider the phase-plane trajectories, which are equivalent to the oscillations in the time-dependent diagrams. We may regard the equilibrium populations in this case as the average density of the prey and predator populations, about which they oscillate.

Direction of trajectories

In order to examine the direction vectors in the phase-plane, we first rewrite the original differential equations (6.13) in the factored form

$$\frac{dX}{dt} = X (\beta_1 - c_1 Y), \quad \frac{dY}{dt} = Y (-\alpha_2 + c_2 X). \quad (6.16)$$

We draw the nullcline curves corresponding to $dX/dt = 0$ and $dY/dt = 0$. They divide the phase-plane into regions where the trajectories have different directions.

The nullcline curves here are the lines $X = 0$, $Y = \beta_1/c_1$ (for $dX/dt = 0$) and the lines $Y = 0$, $X = \alpha_2/c_2$ (for $dY/dt = 0$). Effectively they divide the phase-plane into the four regions labelled I, II, III and IV in Figure 6.8.

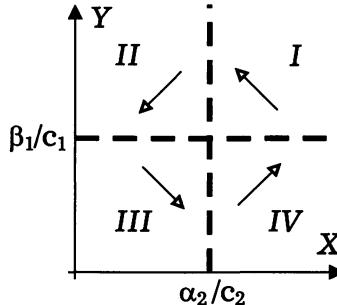


Figure 6.8: Regions in the phase-plane illustrating the trajectory directions. The dashed lines are the non-zero nullclines.

From the differential equations (6.16) we can establish whether $X(t)$ and $Y(t)$ are increasing or decreasing in each of the regions. The following example illustrates how this can be done.

Example 6.10: Determine the trajectory directions for region II in Figure 6.8.

Solution: Everywhere in region II, $X < \alpha_2/c_2$ and $Y > \beta_1/c_1$.

From (6.16), $dX/dt < 0$ everywhere in region II, since $\beta_1 - c_1 Y < 0$ if $c_1 Y > \beta_1$.

Similarly, $dY/dt < 0$ everywhere in region II, since $-\alpha_2 + c_2 X < 0$ when $c_2 X < \alpha_2$.

Thus both X and Y are decreasing in region II. This gives a vector with negative x -component and negative y -component, as shown in Figure 6.8.

By a similar argument

- in region I, X is decreasing and Y is increasing,

- in region III, X is increasing and Y is decreasing,
- in region IV, X is increasing and Y is increasing.

This tells us that as we move along a phase-trajectory we must move in an anticlockwise direction. However, it does not tell us whether this is a closed curve or a spiral. In Section 6.6, we use the chain rule to prove that all trajectories for this system are closed.

Furthermore, for the equilibrium point $(0, 0)$, we note from Figure 6.8 that the trajectories move towards it in one direction and away from it in another. It is thus an unstable equilibrium point in the sense that trajectories move away from it with time and we show in Section 6.6 that this is always the case, regardless of the parameter values. It is called a saddle point, which we discuss in detail in Chapter 7.

Density dependent growth

In Chapter 5, the basic predator-prey model was extended to include density dependent growth (logistic) for the prey species in order to curb the unrealistic exponential growth. It is left as an exercise (Question 6) to generate the associated phase-plane diagram, and examine how this change in growth alters the equilibrium points and/or the direction vectors of the system.

Summary of skills developed here:

- Find equilibrium solutions.
- Establish and draw the nullclines for a system.
- Find directions of trajectories in the phase-plane.
- Deduce how populations behave in time from their phase-plane diagram.

6.5 Analysis of competing species models

We now apply the phase-plane theory to the model we developed to describe the interaction between competing species. Again, we first look at the phase-plane obtained from numerical solutions (using Maple) for a specific set of parameter values and then extend the analysis to consider general values of the parameters, finding equilibrium points and trajectory directions.

Review of model

In Section 5.5, we formulated two models for competing species. The first model assumed exponential growth in the absence of a competitor, and later we introduced an improved model which assumed density-dependent growth for both species in the absence of a competitor. We consider the second improved model here, leaving analysis of the first model as an exercise (Question 10).

From Section 5.5 our model is

$$\frac{dX}{dt} = \beta_1 X - d_1 X^2 - c_1 X Y, \quad \frac{dY}{dt} = \beta_2 Y - d_2 Y^2 - c_2 X Y \quad (6.17)$$

where β_1 and β_2 are per-capita birth rates or growth rates, d_1 and d_2 are density dependent coefficients (independent of the other species) and c_1 and c_2 are interaction coefficients (the effect of species interaction on the death rates). In the absence of species Y , species X has logistic growth with carrying capacity $K_1 = \beta_1/d_1$. Similarly, if $X = 0$ then species Y has logistic growth with carrying capacity $K_2 = \beta_2/d_2$.

The phase-plane diagram from Maple

We can use Maple to draw the phase-plane, but only for a given set of parameter values. Take, for example, per-capita birth rates $\beta_1 = 0.22$, $\beta_2 = 0.061$, interspecies interaction parameters $c_1 = 0.053$, $c_2 = 0.0046$ and intraspecies interaction parameters $d_1 = 0.017$, $d_2 = 0.010$, which are those from Gause's experiments with yeast (see Section 5.5 and Edelstein-Keshet (1988)).

The results in Figure 6.9 show clearly that the trajectories approach equilibrium populations on the X and Y axes, for these values of the parameters. See Listing 6.8 and Listing 6.9 for sample Maple and MATLAB code.

Listing 6.8: Maple code: c_ps_competlogistic.txt

```
> restart:with(plots):with(DEtools):
> tend:=350:
> beta[1]:=0.22: beta[2]:=0.061:
> d[1]:=0.017: d[2]:=0.010:
> c[1]:=0.053: c[2]:=0.0046:
> de1:=diff(X(t),t)=beta[1]*X(t)-c[1]*X(t)*Y(t)-d[1]*X(t)*X(t);
> de2:=diff(Y(t),t)=beta[2]*Y(t)-c[2]*X(t)*Y(t)-d[2]*Y(t)*Y(t);
> inits:=[0,0.5,0.5],[0,20,4],[0,20,8],[0,20,10];
> plot1:=DEplot([de1,de2],[X,Y],t=0..tend,[inits],
  X=0..20,Y=0..10,scene=[X,Y],stepsize=0.05,dirgrid=[10,10],
  linecolour=black,arrows=medium):
> display(plot1);
```

Listing 6.9: MATLAB code: c_ps_competlogistic.m

```
function c_ps_compet
global betal betat2 c1 c2 d1 d2;

betal=0.22; betat2=0.06;
c1=0.053; c2=0.0046;
d1=0.017; d2=0.010;
tend = 360; %the end time
u0vec = [0.5, 20, 20, 20]; % make matrix of ICs
    0.5, 4, 8, 10];
u0size = size(u0vec); %size of the matrix
numICs = u0size(2); % extract number of ICs

for k = 1:numICs
    u0 = u0vec(:, k); %extract the kth column
    [tsol, usol] = ode45(@rhs, [0, tend], u0);
    Xsol = usol(:, 1); Ysol = usol(:, 2);
    plot(Xsol, Ysol, 'b'); hold on;
end
% makearrows, see Appendix for this function
c_dirplot(@rhs, 0, 20, 0, 10, 10);
axis([0, 22, 0, 10]);

function udot = rhs(t, u)
global betal betat2 c1 c2 d1 d2;
X = u(1); Y=u(2);
Xdot = betal*X - c1*X*Y - d1*X^2;
Ydot = betat2*Y - c2*X*Y - d2*Y^2;
udot = [Xdot; Ydot];
```

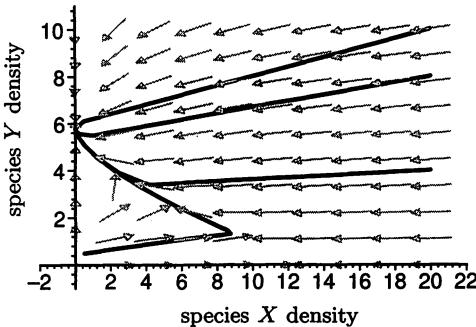


Figure 6.9: Maple produced phase-plane diagram for the competing species model including density dependent growth, with parameter values as given for Gause's experiment.

Note that the trajectory which starts at $(0.5, 0.5)$ moves to the right before turning around and heading towards an equilibrium point at approximately $(0, 6)$. The other trajectories, from a variety of initial conditions, tend to the same equilibrium point. This is consistent with the time-dependent graph in Section 5.5 where one species died out and the other tended to an equilibrium point.

Equilibrium populations

As in Section 6.1 and Section 6.4, we find the equilibrium points for arbitrary parameter values by setting the rates of change to 0. This is done in the following example.

Example 6.11: Find all the equilibrium points for the system of differential equations (6.17).

Solution: We need to solve (simultaneously) $dX/dt = 0$ and $dY/dt = 0$. This implies (after factorising)

$$X(\beta_1 - c_1 Y - d_1 X) = 0 \quad (6.18)$$

$$Y(\beta_2 - c_2 X - d_2 Y) = 0. \quad (6.19)$$

From (6.18) we have two possibilities: $X = 0$ or $\beta_1 - c_1 Y - d_1 X = 0$. If $X = 0$, then (6.19) simplifies to $Y(\beta_2 - d_2 Y) = 0$ which has two possible solutions, $Y = 0$ or $Y = \beta_2/d_2$. Thus we obtain two solutions to the equations, $(X, Y) = (0, 0)$ and $(0, \beta_2/d_2)$.

From (6.19) we have two possibilities: $Y = 0$ or $\beta_2 - c_2 X - d_2 Y = 0$. If $Y = 0$, then (6.18) simplifies to $X(\beta_1 - d_1 X) = 0$. This has two solutions $X = 0$ and $X = \beta_1/d_1$. Thus, simultaneous solutions (X, Y) of both equations are $(0, 0)$ (already obtained above) and $(\beta_1/d_1, 0)$.

One further possibility not yet considered is where (6.18) is satisfied by letting $\beta_1 - d_1 X - c_1 Y = 0$ and where (6.19) is satisfied by letting $\beta_2 - c_2 X - d_2 Y = 0$. By substituting one equation into the other it can be shown that these two equations are satisfied when

$$X = \frac{c_1 \beta_2 - d_2 \beta_1}{c_1 c_2 - d_1 d_2}, \quad Y = \frac{c_2 \beta_1 - d_1 \beta_2}{c_1 c_2 - d_1 d_2}.$$

Summarising, we have found (at most) four equilibrium solutions

$$(0, 0), \quad \left(0, \frac{\beta_2}{d_2}\right), \quad \left(\frac{\beta_1}{d_1}, 0\right), \quad \left(\frac{c_1 \beta_2 - d_2 \beta_1}{c_1 c_2 - d_1 d_2}, \frac{c_2 \beta_1 - d_1 \beta_2}{c_1 c_2 - d_1 d_2}\right),$$

where the fourth equilibrium point is relevant only if both components are positive. Two of the equilibrium points suggest the extinction of one species and the survival and stabilisation of the other.

We can also obtain the equilibrium points using **Maple**. The code for this is given below in Listing 6.10. Similar code for calculating the equilibrium points using **MATLAB** (with symbolic toolbox) is given in Listing 6.11.

Listing 6.10: Maple code: c_ps_compete_eqm.txt

```
> restart;
> eq1 := beta[1]*X(t) - c[1]*X(t)*Y(t) -d[1]*X(t)*X(t);
> eq2 := beta[2]*Y(t) - c[2]*X(t)*Y(t) -d[2]*Y(t)*Y(t);
> solve( {eq1,eq2}, {X(t),Y(t)} );
```

Listing 6.11: Maple code: c_ps_compete_eqm.txt

```
> restart;
> eq1 := beta[1]*X(t) - c[1]*X(t)*Y(t) -d[1]*X(t)*X(t);
> eq2 := beta[2]*Y(t) - c[2]*X(t)*Y(t) -d[2]*Y(t)*Y(t);
> solve( {eq1,eq2}, {X(t),Y(t)} );
```

As before, define the nullcline lines L_0 , L_1 , L_2 , L_3 as

$$\begin{aligned} L_0 : \quad X &= 0, & L_1 : \quad \beta_1 - d_1 X - c_1 Y &= 0, \\ L_3 : \quad Y &= 0. & L_2 : \quad \beta_2 - c_2 X - d_2 Y &= 0, \end{aligned}$$

There is an equilibrium point corresponding to the intersection of the pair of lines L_0 and L_1 with the pair of lines L_2 and L_3 (but not L_0 with L_1 or L_2 with L_3). This is shown in Figure 6.10. The diagrams confirm that there are four equilibrium points if the lines L_1 and L_2 intersect in the positive quadrant of the (X, Y) -plane and only three equilibrium points otherwise.

Direction of trajectories

As usual, we are interested in the predictions of the model over time. We establish the trajectory behaviour by determining trajectory directions and considering only positive values of X and Y . The linear nullclines, L_0 , L_1 , L_2 and L_3 , divide the phase-plane (with X and Y both positive) into different regions in four different ways. These four cases are illustrated in Figure 6.10.

We determine the directions of the trajectories (seen in Figure 6.10) by looking at a typical point in each of the regions bounded by the nullclines. The example below illustrates this process for one of the cases in Figure 6.10.

Example 6.12: Find the directions of the trajectories in each of the regions bounded by the nullclines for Case 3 of Figure 6.10.

Solution: We examine each of the four regions of Case 3 separately. The differential equations (6.17) are written as

$$X' = X(\beta_1 - d_1 X - c_1 Y), \quad Y' = Y(\beta_2 - d_2 Y - c_2 X)$$

where X' denotes dX/dt and Y' denotes dY/dt .

Consider the region below both L_1 and L_2 . From the equations for L_1 and L_2 ($\beta_1 - d_1 X - c_1 Y = 0$ and $\beta_2 - d_2 Y - c_2 X = 0$) a point in the region below L_1 has $Y < (\beta_1 - d_1 X)/c_1$ and a point in the region below L_2 has $Y < (\beta_2 - c_2 X)/d_2$. Thus

$$\begin{aligned} c_1 Y < \beta_1 - d_1 X &\Rightarrow 0 < \beta_1 - d_1 X - c_1 Y &\Rightarrow X' > 0, \\ d_2 Y < \beta_2 - c_2 X &\Rightarrow 0 < \beta_2 - c_2 X - d_2 Y &\Rightarrow Y' > 0. \end{aligned}$$

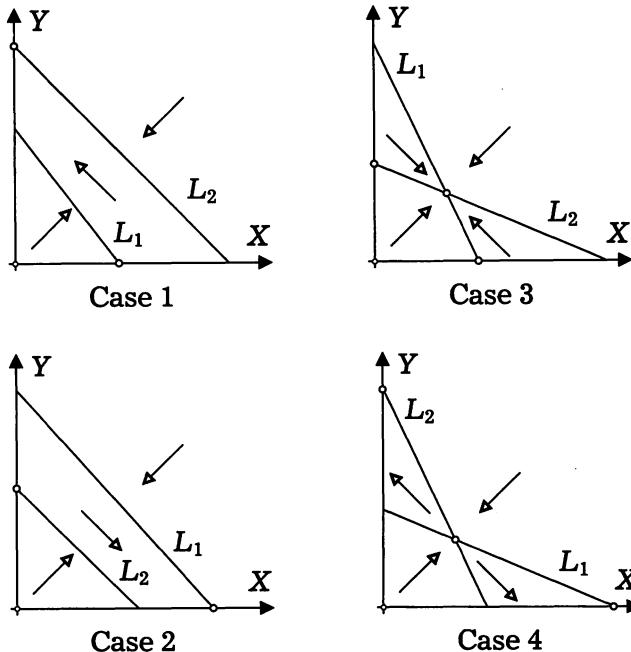


Figure 6.10: The four cases of the phase-plane diagram for competing species including density dependent growth. The direction vectors for the trajectories are illustrated with equilibrium points marked with open circles.

Consider the region below L_1 and above L_2 . Here

$$\begin{aligned} c_1Y < \beta_1 - d_1X &\Rightarrow 0 < \beta_1 - d_1X - c_1Y &\Rightarrow X' > 0, \\ d_2Y > \beta_2 - c_2X &\Rightarrow 0 > \beta_2 - c_2X - d_2Y &\Rightarrow Y' < 0. \end{aligned}$$

Similarly, for the region above L_1 and below L_2

$$\begin{aligned} c_1Y > \beta_1 - d_1X &\Rightarrow 0 > \beta_1 - d_1X - c_1Y &\Rightarrow X' < 0, \\ d_2Y < \beta_2 - c_2X &\Rightarrow 0 < \beta_2 - c_2X - d_2Y &\Rightarrow Y' > 0. \end{aligned}$$

Finally, for the region above both L_1 and L_2

$$\begin{aligned} c_1Y > \beta_1 - d_1X &\Rightarrow 0 > \beta_1 - d_1X - c_1Y &\Rightarrow X' < 0, \\ d_2Y > \beta_2 - c_2X &\Rightarrow 0 > \beta_2 - c_2X - d_2Y &\Rightarrow Y' < 0. \end{aligned}$$

This provides all the information required to establish the direction vectors of Case 3.

In Figure 6.10 we show the directions for each of the four different ways of sectioning the phase-plane with nullclines. In Case 1, the trajectories all move towards the equilibrium point on the Y -axis. This corresponds to extinction of the X -species, in the long term. In Case 2, the trajectories all move towards the equilibrium point on the X -axis. This corresponds to extinction of the Y -species, in the long term. In Case 3, the trajectories move to the equilibrium point where the nullclines L_1 and L_2 intersect. This corresponds to a coexistence of the two species, in the long term. In Case 4, the trajectories may end up at either of the equilibrium points on the axes, but not at the equilibrium point where the nullclines L_1 and L_2 intersect. Which equilibrium point is approached, or which species survives, will depend on the initial conditions.

We can use the differences between the intercepts on the X and Y axes to determine which of the above cases provides the solution. Let

$$\text{Int}_X = \frac{\beta_1}{d_1} - \frac{\beta_2}{c_2} \quad \text{and} \quad \text{Int}_Y = \frac{\beta_2}{d_2} - \frac{\beta_1}{c_1},$$

the signs of which establish the equilibrium point towards which the trajectories move. The results are displayed in Table 6.1. (In Chapter 7, we develop further theory to establish which of these equilibrium points will attract or repel the trajectories.)

Table 6.1: Predicting the equilibrium point from the sign of Int_X and Int_Y .

Int_X	Int_Y	equilibrium point approached
+	-	$\left(\frac{\beta_1}{d_1}, 0\right)$
-	+	$\left(0, \frac{\beta_2}{d_2}\right)$
-	-	$\left(\frac{c_1\beta_2 - d_2\beta_1}{c_1c_2 - d_1d_2}, \frac{c_2\beta_1 - d_1\beta_2}{c_1c_2 - d_1d_2}\right)$
+	+	$\left(\frac{\beta_1}{d_1}, 0\right)$ or $\left(0, \frac{\beta_2}{d_2}\right)$ depending on starting conditions

Interpretation of parameters

Above we established that β_1/d_1 was the carrying capacity K_1 for species X and that β_2/d_2 was the carrying capacity K_2 for population Y . Thus we can interpret the trajectory approach to $(\beta_1/d_1, 0) = (K_1, 0)$ as the survival of X and the extinction (or migration) of Y . Likewise, an approach to $(0, \beta_2/d_2) = (0, K_2)$ can be seen as the survival of Y and the extinction of X .

Initially, when our model allowed for only exponential growth (that is, infinite carrying capacity in the absence of the other species) it can be shown there is an equilibrium point at $(\beta_2/c_2, \beta_1/c_1)$, the only point for which both species can possibly exist simultaneously. Now Int_X is defined as the difference between the X coordinate β_2/c_2 and the carrying capacity of the species as was introduced with logistic growth. (Similarly for Int_Y .) Thus Int_X and Int_Y can be considered as *interference factors*, or a measure of how much one species interferes with the other.

Discussion

Our competition model predicts, in almost all cases, the extinction of one species and the survival of the other. However, observations indicate that there is substantial diversity in many ecosystems with competing species coexisting. As we have discussed previously, these ecosystems do not exist in isolation but are integral components of some much larger system.

In Chapter 8, we present a system which uses the competition model developed here, and embeds it within another system, a predator-prey model, establishing that in this more complicated system the two competing species can coexist. This lends credence to the earlier assertion of larger systems having greater stability than isolated ecosystems.

Summary of skills developed here:

- Reinforcement of methods for finding equilibrium points by solving simultaneous equations.
- Reinforcement of methods for finding direction vectors for trajectories and establishing the general behaviour of a system from the phase-plane.

6.6 Closed trajectories for the predator-prey

We return to the analysis of the basic predator-prey model, the Lotka-Volterra system, introduced in Section 6.4. We show by applying the chain rule that, for all parameter values in this model, the phase-plane curves are always closed curves. This allows us to infer that, according to this model, both populations always oscillate over time.

Review of model

The Lotka-Volterra predator-prey equations are

$$\frac{dX}{dt} = \beta_1 X - c_1 XY, \quad \frac{dY}{dt} = -\alpha_2 Y + c_2 XY. \quad (6.20)$$

where the parameters β_1 , α_2 , c_1 and c_2 are all positive constants. We also found in Section 6.4 that the equilibrium points were given by

$$(X, Y) = (0, 0) \quad \text{and} \quad (X, Y) = \left(\frac{\alpha_2}{c_2}, \frac{\beta_1}{c_1} \right).$$

Furthermore, we deduced the directions of the phase-plane trajectories from which we could infer that the trajectories were either closed curves or spirals, traversed in an anti-clockwise direction. We show, by applying the chain rule to find a relation between X and Y , that we can prove there is never a spiral, regardless of the parameters we may choose.

Applying the chain rule

We use the chain rule to eliminate time from the differential equations, thus obtaining a differential equation relating the predator population Y to the prey population X .

Example 6.13: Use the chain rule to find an equation relating X and Y .

Solution: By the chain rule

$$\frac{dY}{dX} = \frac{dY/dt}{dX/dt}.$$

Hence, using (6.20) we obtain the first-order differential equation

$$\frac{dY}{dX} = \frac{Y(-\alpha_2 + c_2 X)}{X(\beta_1 - c_1 Y)}. \quad (6.21)$$

This differential equation is separable. Separating the variables and integrating both sides with respect to X we obtain

$$\int \left(\frac{\beta_1}{Y} - c_1 \right) dY = \int \left(-\frac{\alpha_2}{X} + c_2 \right) dX.$$

Carrying out both integrations yields

$$\beta_1 \ln(Y) - c_1 Y = -\alpha_2 \ln(X) + c_2 X + K \quad (6.22)$$

where K is an arbitrary constant of integration.

Proving all trajectories are closed

From the direction vector diagram of the earlier section (Figure 6.8) it is clear that the trajectories must be closed curves or spirals. We now provide an argument that the phase-plane trajectories are closed curves for all positive values of the constant parameters.

To get further information we return to the formula (6.22). Suppose we hold the variable X constant, $X = X_1$. Then we have the equation

$$\ln(Y) = \frac{c_1}{\beta_1} Y + K_1 \quad (6.23)$$

where K_1 is the constant defined by $K_1 = (-\alpha_2 \ln(X_1) + c_2 X_1 + K)/\beta_1$. If the phase-plane curve were a closed curve then it could have, at most, two intersection points with any vertical line, in particular $X = X_1$. On the other hand, if it were a spiral it would have an infinite number of intersection points. By sketching the curves $Z = \ln(Y)$ and $Z = (c_1/\beta_1)Y + K_1$ we see there are, at most, two solutions. This is illustrated in Figure 6.11.

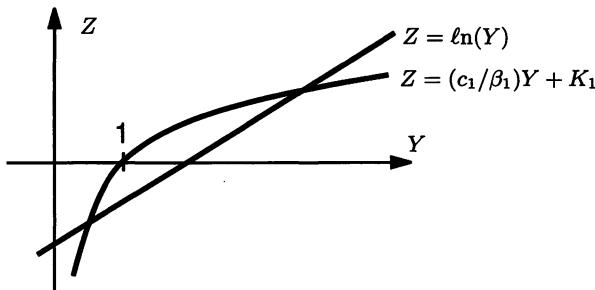


Figure 6.11: A general sketch of the intersection between the two curves $Z = \ln(Y)$ and $Z = (c_1/\beta_1)Y + K_1$ showing at most two intersection points.

Returning to the phase-plane

We have concluded that all the phase-plane curves are closed curves. Using this, together with Figure 6.8 and the fact that X and Y are positive, we can sketch the general form of a typical phase-plane trajectory. Recall, from Section 6.4, that the trajectory is traversed in an anticlockwise direction. This is illustrated in Figure 6.12.

All the phase-plane curves are traversed in the anticlockwise direction along closed trajectories enclosing an equilibrium point, which in this case represents average population densities for the two populations. We note that as we trace out the trajectory, the prey (X) reach their maximum population before the predator population (Y) reaches its maximum

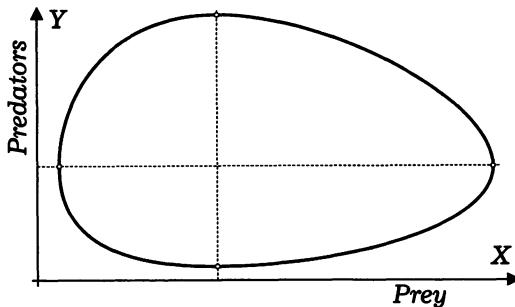


Figure 6.12: Phase-plane diagram showing a typical trajectory for the predator-prey problem enclosing the equilibrium point.

value. Thus the predator oscillation cycle always lags behind the prey oscillation, no matter what parameter values or initial conditions we use.

A feature of this model is that if you suddenly perturb the populations, they begin to move on a different phase-plane trajectory. This is one undesirable feature of the Lotka-Volterra equations from a biological perspective. Biologists would prefer a more realistic model where the populations tend to return to the original phase-plane curve after they have been perturbed slightly. This can be achieved by using a modified version of the Lotka-Volterra equations, which we consider in Chapter 8.

Summary of skills developed here:

- Use the chain rule to prove that the trajectories of the the Lotka-Volterra equations are always closed.
- Consider the points of intersection between two simple graphs to infer information about solutions to a more complicated equation.

6.7 Case Study: Bacteria battle in the gut

Simple models can often be applied to complex systems and successfully predict phenomena that have been observed. In the following case study we model, using the mechanisms discussed in this book, the interaction between different strains of *Escherichia coli* in the gut of animals. With this simple approach we show that a gut with a slow turnover rate favours different strains from one with a fast turnover rate, where these rates of turnover relate to diet. Adapted from Barnes et al. (2007).

Colicins are a class of protein antibiotics known as bacteriocins that are produced by certain strains of *Escherichia coli* (*E. coli*). They are produced through a process called cell lysis, where a single cell of bacteria produces many colicins. The advantage of producing colicins is to kill off other competing strains of *E. coli* in the same environment, although in doing so the colicin itself is destroyed. Thus colicins are important in mediating intra-specific interactions.