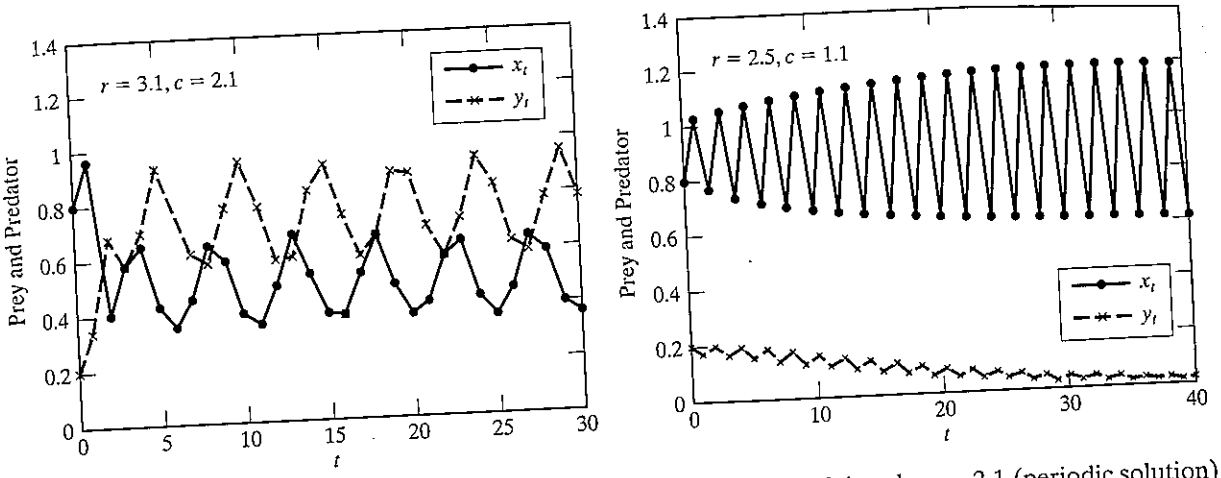

Figure 3.9 Solutions to the normalized predator-prey system when $r = 3.1$ and $c = 2.1$ (periodic solution) and when $r = 2.5$ and $c = 1.1$ (period 2 solutions for the prey and the predator goes extinct).

3.7 Population Genetics Models

Inheritance depends on the information contained in the chromosomes that are passed down from generation to generation. Humans have two sets of 23 chromosomes (diploid), making a total of 46 chromosomes; one set is obtained from each parent. Certain locations along the chromosomes contain the instructions for some characteristic, such as eye or hair color. The locations along the chromosomes are called the *loci* (a single location is called a *locus*). The instructions within the chromosomes are referred to as the *genes*. Each gene gives a unique instruction (for color of eyes, color of hair, etc.) and each human has two genes per locus because there are two sets of chromosomes. The physical characteristics (eye or hair color) unique to each individual are determined by that individual's genes. In simple organisms, such as bacteria, there are 2000 to 3000 genes, whereas in higher organisms such as plants and animals there are 50,000 to 100,000 genes (Clark and Russell, 1997). Each gene has different variant forms (the gene for eye color can be green, blue, brown, etc.). These different variant forms of the genes are referred to as *alleles*. Here, we shall consider the simplest possible case, the case where there are only two different alleles associated with a particular gene.

Suppose there are two alleles for a given gene. The two alleles are denoted a and A. A human with two sets of chromosomes could then have one of three different combinations on his or her chromosome: AA, Aa, or aa. The combinations AA and aa are *homozygous*, whereas the combination Aa is *heterozygous*. The three combinations, AA, Aa, and aa, are called the *genotypes* of the locus.

One of the two alleles may be *dominant*. For example, if A is the dominant allele, then a is referred to as the *recessive allele*. Then genotypes AA and Aa correspond to the same physical trait, but different from that of aa. This is also described as saying that genotypes AA and Aa have *phenotype* A and aa has *phenotype* a (Hoppensteadt, 1975).

We explore the question of whether the allele frequencies (associated with a particular gene) change in a given population over time as individuals within that population mate and reproduce. Our population genetics model is a simple one-locus, two-allele model. We assume that during each time step, the population in generation t is replaced by the population in generation $t + 1$.

An important principle in population genetics is known as the *Hardy-Weinberg law*. First, the following assumptions must hold: (1) Mating is random, (2) there is no variation in the number of progeny from parents of different genotypes, (3) all genotypes are equally fit, and (4) there are no mutations. Then the Hardy-Weinberg law asserts that gene frequencies and allele frequencies do not change from one generation to the next. Our population genetics model is based on these assumptions. We will show that our model follows the Hardy-Weinberg law. The name Hardy-Weinberg recognizes the work of G. H. Hardy, a famous English mathematician (number theorist) and Wilhelm Weinberg, a German physician and human geneticist, who independently discovered this result in 1908 (Felsenstein, 2003).

The following definitions and assumptions are needed. Let N be the total population size. Since each individual has two alleles per locus, there are a total of $2N$ alleles in the population. Let

$$p = \text{frequency of allele } A = (\text{total number of } A \text{ alleles})/(2N),$$
$$q = \text{frequency of allele } a = (\text{total number of } a \text{ alleles})/(2N),$$

then $p + q = 1$. Let

$$p_{AA} = \text{frequency of } AA \text{ genotype},$$
$$p_{Aa} = \text{frequency of } Aa \text{ genotype},$$
$$p_{aa} = \text{frequency of } aa \text{ genotype}.$$

Thus, the frequency of A alleles is

$$p = \frac{2Np_{AA} + Np_{Aa}}{2N} = p_{AA} + \frac{p_{Aa}}{2}.$$

The frequency of a alleles is

$$q = 1 - p = \frac{p_{Aa}}{2} + p_{aa}.$$

To determine what happens after one generation of mating, it is necessary to consider all possible matings, their frequency, and all possible offspring and their frequency. The possible matings are obtained by considering all possible pairings of AA, Aa, and aa (representing a genotype for each parent) for which there are $3(2) = 6$:

$$AA \times AA,\ AA \times Aa,\ AA \times aa,\ Aa \times Aa,\ Aa \times aa,\ aa \times aa.$$

Each of these occur with the corresponding frequencies:

$$p_{AA}^2,\ 2p_{AA}p_{Aa},\ 2p_{AA}p_{aa},\ p_{Aa}^2,\ 2p_{Aa}p_{aa},\ p_{aa}^2.$$

This information plus the information needed for the offspring frequencies are given in the following mating and offspring table, Table 3.1 (Hastings, 1998). Note that the sum of the mating frequencies equals $(p_{AA} + p_{Aa} + p_{aa})^2$.

Let p'_{AA}, p'_{Aa}, and p'_{aa} denote the genotypic frequencies in the next generation. Then, applying the results from Table 3.1,

$$\begin{aligned} p'_{AA} &= p_{AA}^2 + p_{AA}p_{Aa} + p_{Aa}^2/4 \\ &= (p_{AA} + p_{Aa}/2)^2 \\ &= p^2, \end{aligned}$$

Table 3.1 Mating and offspring table.

Mating	Mating Frequency	Offspring Fraction			Next Generation		
		AA	Aa	aa	AA	Aa	aa
$AA \times AA$	p_{AA}^2	1	0	0	p_{AA}^2	0	0
$AA \times Aa$	$2p_{AA}p_{Aa}$	1/2	1/2	0	$p_{AA}p_{Aa}$	$p_{AA}p_{Aa}$	0
$AA \times aa$	$2p_{AA}p_{aa}$	0	1	0	0	$2p_{AA}p_{aa}$	0
$Aa \times Aa$	p_{Aa}^2	1/4	1/2	1/4	$p_{Aa}^2/4$	$p_{Aa}^2/2$	$p_{Aa}^2/4$
$Aa \times aa$	$2p_{Aa}p_{aa}$	0	1/2	1/2	0	$p_{Aa}p_{aa}$	$p_{Aa}p_{aa}$
$aa \times aa$	p_{aa}^2	0	0	1	0	0	p_{aa}^2

$$\begin{aligned} p'_{aa} &= p_{Aa}^2/4 + p_{Aa}p_{aa} + p_{aa}^2 \\ &= (p_{aa} + p_{Aa}/2)^2 \\ &= q^2, \end{aligned}$$

and

$$\begin{aligned} p'_{Aa} &= p_{AA}p_{Aa} + 2p_{AA}p_{aa} + p_{Aa}^2/2 + p_{Aa}p_{aa} \\ &= 2(p_{AA} + p_{Aa}/2)(p_{aa} + p_{Aa}/2) \\ &= 2pq. \end{aligned}$$

These results can be used to find the allele frequencies in the next generation, p' and q',

$$\begin{aligned} p' &= p'_{AA} + p'_{Aa}/2 \\ &= p^2 + pq \\ &= p(p + q) = p \end{aligned}$$

and

$$\begin{aligned} q' &= p'_{aa} + p'_{Aa}/2 \\ &= q^2 + pq \\ &= q(p + q) = q. \end{aligned}$$

The frequencies remain constant from generation to generation, that is, from generation t to $t + 1$, $p_{t+1} = p_t$ and $q_{t+1} = q_t$. Hence,

$$p_t = p_0 \quad \text{and} \quad q_t = q_0.$$

The Hardy-Weinberg law has been verified.

Theorem 3.2 **(Hardy-Weinberg Law).** *Assume in a parent population, a particular gene has two alleles A and a, and the initial proportion of allele A is p_0 and the initial proportion of allele a is q_0. In addition, assume (i) mating is random, (ii) there is no variation in the number of progeny from parents of different genotypes, (iii) all genotypes have equal survival probability, (iv) there is no immigration nor*

emigration, (v) there are no mutations, and (vi) generations are nonoverlapping. Then, in generation t, the allele frequencies do not change,

$$p_t = p_0 \quad \text{and} \quad q_t = q_0.$$

In addition, the genotypic frequencies do not change from the second generation onwards,

$$p_{AA} = p_0^2, \quad p_{Aa} = 2p_0q_0, \quad \text{and} \quad p_{aa} = q_0^2. \qquad \square$$

According to the Hardy-Weinberg law, the recessive trait will not die out but remain in the population at a fixed proportion. If the assumptions in Theorem 3.2 are violated, then the Hardy-Weinberg proportions change. We consider a violation of assumption (iii).

Suppose the survival rates depend on genotype. In this case, different genotypes have different fitnesses. The frequencies of allele A and the proportion p are modeled over time. Let p_t be the frequency of allele A in generation t and q_t be the frequency of allele a ($p_t + q_t = 1$). Let w_{AA} and w_{aa} be the constant survival rates of genotypes AA and aa relative to the heterozygote genotype Aa, which is assumed to satisfy $w_{Aa} = 1$. Note that w_{AA} and w_{aa} can be less than or greater than one, but must be nonnegative. Let the *mean fitness* be denoted as

$$w = p^2w_{AA} + 2pqw_{Aa} + q^2w_{aa}.$$

Suppose initially the genotypic frequencies AA, Aa, and aa are in the proportions p^2, $2pq$, and q^2, respectively. Then, it follows from the following genotypic frequency table, Table 3.2, that the next generation satisfies

$$\begin{aligned} p_{t+1} &= p_{AA} + p_{Aa}/2 \\ &= p_t^2w_{AA}/w_t + (1/2)2p_tq_tw_{Aa}/w_t \\ &= p_t(p_tw_{AA} + q_tw_{Aa})/w_t \\ &= p_t(p_tw_{AA} + (1 - p_t)w_{Aa})/w_t, \end{aligned}$$

where

$$w_t = p_t^2w_{AA} + 2p_tq_tw_{Aa} + q_t^2w_{aa}$$

is the mean fitness in generation t (see Hastings, 1998). The following difference equation models the change in the allele frequency A from generation t to generation $t + 1$,

$$p_{t+1} = \frac{p_t^2w_{AA} + p_t(1 - p_t)w_{Aa}}{w_t}. \tag{3.7}$$

Table 3.2 Genotypic frequency table, where the mean fitness is given by $w = p^2w_{AA} + 2pq + q^2w_{aa}$.

	Genotype		
	AA	Aa	aa
Juvenile frequencies	p^2	$2pq$	q^2
Relative survival rates	w_{AA}	w_{Aa}	w_{aa}
Relative adult frequencies	p^2w_{AA}	$2pqw_{Aa}$	q^2w_{aa}
Adult frequencies	p^2w_{AA}/w	$2pqw_{Aa}/w$	q^2w_{aa}/w

Note that if $w_{AA} = w_{Aa} = w_{aa} = 1$, then $w_t = 1$ and $p_{t+1} = p_t$.

Suppose that the relative survival rates satisfy

$$w_{AA} = 1 - s, \quad w_{Aa} = 1 \quad \text{and} \quad w_{aa} = 1 - r.$$

Then s and r can be positive or negative but w_{AA} and w_{aa} must be nonnegative so that $r, s < 1$ (but not both zero). Then

$$w_t = p_t^2(1 - s) + 2p_tq_t + q_t^2(1 - r) = 1 - p_t^2s - (1 - p_t)^2r.$$

The difference equation in p satisfies

$$p_{t+1} = \frac{p_t[p_t(1 - s) + (1 - p_t)]}{1 - p_t^2s - (1 - p_t)^2r} = \frac{p_t(1 - p_ts)}{1 - p_t^2s - (1 - p_t)^2r} = f(p_t). \quad (3.8)$$

Next the equilibria are determined for the difference equation (3.8) and their local stability assessed.

There are three equilibria for the difference equation (3.8). They are $\bar{p} = 0$ and the solutions to

$$\bar{p}^2s + (1 - \bar{p})^2r = \bar{p}s.$$

Solutions to the latter equation satisfy $(1 - \bar{p})^2r = s\bar{p}(1 - \bar{p})$. Therefore, $\bar{p} = 1$ is another equilibrium. Dividing by $1 - \bar{p}$ leads to $(1 - \bar{p})r = s\bar{p}$ so that the third equilibrium is

$$\bar{p} = \frac{r}{r + s}.$$

When $\bar{p} = 0$, only the allele a is present in the population. When $\bar{p} = 1$, only allele A is present, and when $\bar{p} = r/(r + s)$ both alleles are present. The local stability of these equilibria are determined next.

The derivative of $f(p) = p(1 - ps)/(1 - p^2s - (1 - p)^2r)$ in simplified form is

$$f'(p) = \frac{1 + p^2s - r + rp^2 - 2ps + 2psr - 2p^2sr}{(1 - p^2s - r + 2rp - rp^2)^2}$$

$$= \frac{(1 - s)p^2 + 2(1 - s)(1 - r)p(1 - p) + (1 - r)(1 - p)^2}{(1 - p^2s - r + 2rp - rp^2)^2}. \quad (3.9)$$

Note that $f'(p) > 0$ for $0 \leq p \leq 1$ and $r, s < 1$.

The equilibrium $\bar{p}$ is locally asymptotic stable if $-1 < f'(\bar{p}) < 1$. However, since $f'(p) > 0$ for $0 \leq p \leq 1$ and $r, s < 1$, we only need to show for local stability that $f'(\bar{p}) < 1$. At $\bar{p} = 0$,

$$f'(0) = \frac{1}{1 - r}.$$

Thus, $\bar{p} = 0$ is locally asymptotically stable if $r < 0$ (i.e., the relative survival rate of aa is $w_{aa} = 1 - r > 1$ or the fitness value of the homozygote aa is greater than that of the heterozygote, $w_{Aa} = 1$).

At $\bar{p} = 1$,

$$f'(1) = \frac{1}{1 - s}.$$

Thus, $\bar{p} = 1$ is locally asymptotically stable if $s < 0$ (i.e., the relative survival rate of AA is $w_{AA} = 1 - s > 1$ or the fitness value of the homozygote AA is greater than that of the heterozygote, $w_{Aa} = 1$).

Finally, at the equilibrium $\bar{p} = r/(r + s)$,

$$f'\left(\frac{r}{r+s}\right) = \frac{2rs - r - s}{rs - r - s}. \tag{3.10}$$

In order for $\bar{p}$ to be positive and less than one, either both r and s are positive or both are negative, $rs > 0$. Suppose $rs - r - s > 0$; then $f'(r/(r + s)) > 1$, which means for stability $rs - r - s \leq 0$. This means r and s must both be positive for stability.

We will show that if $r, s \in (0, 1)$, then the equilibrium $\bar{p} = r/(r + s)$, where $0 < \bar{p} < 1$, is locally asymptotically stable. First, if $r, s \in (0, 1)$, then $rs < r + s$. Second, it follows from (3.10) that $2rs < r + s$ [because $f'(\bar{p}) > 0$]. Hence $f'(\bar{p}) = (r + s - 2rs)/(r + s - rs) < 1$. Therefore, if $r, s \in (0, 1)$, the equilibrium $\bar{p} = r/(r + s)$ is locally asymptotically stable. This result can be interpreted biologically. When $r, s \in (0, 1)$, the heterozygous genotype has the largest survival rate, $w_{Aa} > \max\{w_{AA}, w_{aa}\}$. Because the heterozygote has an advantage, both alleles persist in the population.

It is interesting to note that in all cases, the mean fitness, $w = w_t$, increases over time until an equilibrium is reached, either $\bar{p} = 0$, $\bar{p} = 1$ or $\bar{p} = r/(r + s)$. This result can be verified mathematically (see Exercise 8). That is, for $t = 0, 1, \ldots,$

$$w_{t+1} \geq w_t.$$

An alternate method to verify stability of the equilibria is to consider the graph of $p_{t+1} = f(p_t)$ and use the cobwebbing method or apply the theorems in Chapter 2. For example, there are four possible configurations for $p_{t+1} = f(p_t)$ in the p_t-p_{t+1} plane. They are graphed in Figure 3.10. In Exercise 9, the stability conditions derived for the positive equilibrium are shown to be global asymptotic stability conditions.

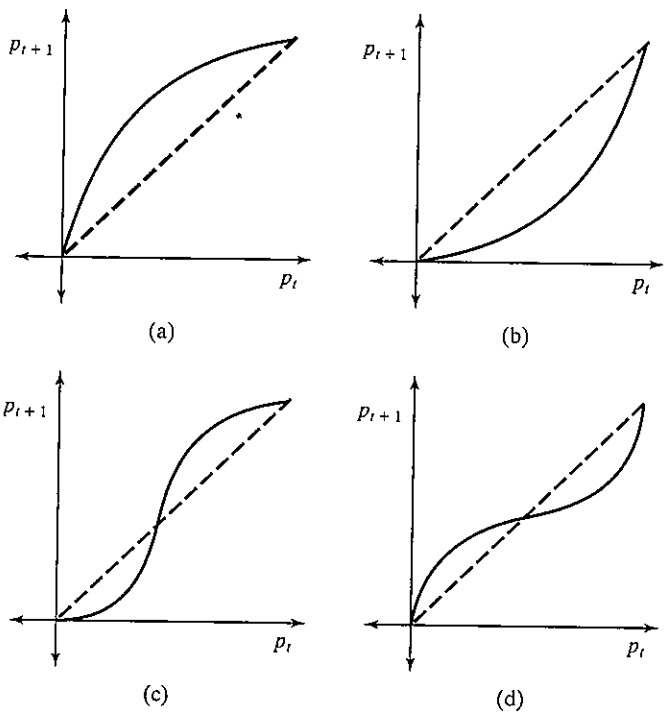

Figure 3.10 Graphs of the function $p_{t+1} = f(p_t)$ in p_t-p_{t+1} plane. The solid curve is the function $p_{t+1} = f(p_t)$ and the dotted curve is the line, $p_{t+1} = p_t$. The intersection points of these two curves represent the equilibria: 0, 1, and $\bar{p}$. In (a), $s < 0$ and $0 < r < 1$, $\lim_{t\to\infty} p_t = 1$. In (b), $r < 0$ and $0 < s < 1$, $\lim_{t\to\infty} p_t = 0$. In (c), $r, s < 0$, so that the limit depends on initial conditions. Solutions approach one of the two equilibria, 0 or 1. In (d), $0 < r, s < 1$, $\lim_{t\to\infty} p_t = \bar{p}$, the positive polymorphic equilibrium.

Selection depends on many different factors, and the importance of these factors differs significantly between animal and plant populations. Hedrick (2000) classifies the types of selection into several categories based on the underlying biological principles: viability selection (dependent on survival such as in the previous model), fecundity selection (differential production of offspring), sexual selection (males or females have preferential mating), and gametic selection (for example, equal proportion of alleles may not be produced). In addition, population size may affect selection for mates. For example, when the fitnesses w_{AA} and w_{Aa} depend on the frequency p of allele A, this is referred to as *frequency-dependent selection* (Nagylaki, 1992). According to Nagylaki (1992), polymorphism can be maintained by frequency-dependent selection which favors rare genotypes.

Example 3.2 Assume $w_{Aa} = 1$ and $w_{AA}(p)w_{aa}(p) = 1$ so that the heterozygote has fitness equal to the geometric mean of the homozygotes (Elaydi, 2000). In addition, assume that the frequency-dependent fitnesses are symmetric, $w_{AA}(p) = w_{aa}(1 - p) = w_{aa}(q)$ and $w_{AA}(p) = f(p) = 1/w_{aa}(p)$. The function $f(p)$ is a positive, strictly decreasing function with $f(0) > 1$ and f' continuous. Thus, when p is small or A is rare, the fitness $w_{AA}(p)$ is large and when p is large, $1 - p$ is small, a is rare, the fitness $w_{aa}(p)$ is large. The population genetics model (3.7) has the form

$$p_{t+1} = \frac{p_t f(p_t)[p_t + (1 - p_t)/f(p_t)]}{(p_t f(p_t) + 1 - p_t)[p_t + (1 - p_t)/f(p_t)]} = \frac{p_t f(p_t)}{p_t f(p_t) + 1 - p_t} = F(p_t).$$

This model has three equilibria, $\bar{p} = 0$, $\bar{p} = 1$, and the polymorphic equilibrium p^* satisfying $f(p^*) = 1$. The polymorphic equilibrium is unique because of the assumptions on f. Now,

$$F'(p) = \frac{f(p) + p(1 - p)f'(p)}{[pf(p) + 1 - p]^2},$$

so that $F'(0) = f(0) > 1$ and $F'(1) = 1/f(1) = w_{aa}(1) = w_{AA}(0) = f(0) > 1$. The equilibria $\bar{p} = 0$ and $\bar{p} = 1$ are unstable. Since the derivative $F'(p^*) = 1 + p^*(1 - p^*)f'(p^*) < 1$, for stability of p^*, it is only necessary to show that $F'(p^*) > -1$ or $p^*(1 - p^*)f'(p^*) > -2$. For example, if $f(p) = \exp(1 - 2p)$, then $p^* = 1/2$ and $p^*(1 - p^*)f'(p^*) = -1/2 > -2$ so that the polymorphic equilibrium is locally asymptotically stable. Elaydi (2000) has shown that for suitably chosen $f(p)$, the model can exhibit period-doubling behavior (see Exercise 10). ■

Example 3.3 We formulate a population genetics model for two populations. Assume the two populations are diploid. We model one gene in each population and assume there are only two alleles. In the first population, the two alleles are V and v and in the second population, the two alleles are R and r. We model the frequency of alleles V and R for the first and second population, respectively. Let the proportion of allele V in the first population be denoted as n, and the proportion of allele R in the second population be denoted as p. Then the model takes the form

$$n_{t+1} = \frac{n_t^2 w_{VV} + n_t(1 - n_t)w_{Vv}}{n_t^2 w_{VV} + 2n_t(1 - n_t)w_{Vv} + (1 - n_t)^2 w_{vv}} = f(n_t, p_t),$$

$$p_{t+1} = \frac{p_t^2 w_{RR} + p_t(1 - p_t)w_{Rr}}{p_t^2 w_{RR} + 2p_t(1 - p_t)w_{Rr} + (1 - p_t)^2 w_{rr}} = g(n_t, p_t). \quad (3.11)$$

Population genetics models of this form have been studied in relation to plant pathogens. The first population represents a pathogen, whereas the second population represents a plant that is attacked by the pathogen. Allele V represents a virulent allele and v an avirulent allele in the pathogen population and R represents a resistant allele and r a susceptible allele in the host plant. A virulent gene in the pathogen population is matched by a resistant gene in the plant population. Such types of gene relationships are referred to as *gene-for-gene* systems and have been studied by Leonard (1977, 1994) and many others (see, e.g., Sasaki, 2002; Kesinger and Allen, 2002 and references therein). The gene-for-gene hypothesis states that for each gene determining resistance in the host there is a corresponding gene for avirulence in the parasite with which it interacts (Thompson and Burdon, 1992). This hypothesis was originally applied to flax and flax rust (Flor, 1956) but has been applied to variety of plant pathogens including wheat stem rust and potato late blight (Vanderplank, 1984).

The fitnesses of the various pathogen genotypes, w_{VV}, w_{Vv}, and w_{vv}, depend on the frequency of the plant resistance allele p. The fitnesses of the plant genotypes, w_{RR}, w_{Rr}, and w_{rr}, depend on the frequency of the pathogen virulence gene n. Model (3.11) is studied in more detail in Exercise 12. ■

The subject of inheritance and population genetics is much more complicated than the short introduction we have given here. For example, selection, mutation, nonrandom mating, migration, recombination, and gene linkage affect the outcome of the genetic makeup of a population. Please consult population genetics textbooks Hartl and Clark (1997) or Hedrick (2000) for a wealth of biological examples.

3.8 Nonlinear Structured Models

Two theoretical nonlinear Leslie matrix models and two structured models applied to specific populations are studied. The nonlinear Leslie matrix models are presented in the next subsection. Then the two structured models are presented in the next two subsections. The structured models are applied to a flour beetle population and to the northern spotted owl. The final example in this section is a generalization of the Leslie matrix model to a two-sex model.

3.8.1 Density-Dependent Leslie Matrix Models

Assume that the population size or density affects the survival and/or fecundity of each age class. Assume that as the total population size or density increases, food resources are depleted resulting in a decrease in survival and/or fecundity. Competition, cannibalism, and predation also tend to increase with population density, which ultimately leads to decreased survival and fecundity.

Recall that the Leslie matrix model has the form $X(t+1) = LX(t)$, where

$$L = \begin{pmatrix} b_1 & b_2 & \cdots & b_{m-1} & b_m \\ s_1 & 0 & \cdots & 0 & 0 \\ 0 & s_2 & \cdots & 0 & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & \cdots & s_{m-1} & 0 \end{pmatrix},$$