5 Population growth and regulation

5.1 Introduction

In this chapter we first describe the fundamental characteristics of population growth and how these vary with body size. We consider the range of processes that can constrain population growth through *population limitation* and ultimately serve to stabilize populations through the density-dependent process of *natural regulation*. We then analyze the processes that can cause fluctuations and population cycles, using models to develop our understanding of them. Finally, we examine one of the major causes of regulation: competition between individuals for resources, or *intraspecific competition*. Other causes of regulation, such as predation, parasites, and disease, will be dealt with in Chapters 7–9.

5.2 Rate of increase

If a population comprising 100 animals on (say) January 1 contains 200 animals on the following January 1 then obviously it has doubled over one year. What will be its size on the next January 1 if it continues to grow at the same rate? The answer is not 300, as it would be if the growth increment (net number of animals added over the year) remained constant each year, but 400, because it is the growth rate (net number of animals added, divided by numbers present at the beginning of the interval) that remains constant. Thus the growth of a population is analogous to the growth of a sum of money deposited at interest with a bank. In both cases, the growth increment each year is determined by the rate of growth and by the amount of money or the number of animals that are there to start with. Both grow according to the rules of compound interest and all calculations must therefore be governed by that branch of arithmetic.

Populations decrease as well as increase. The population of 100 animals on January 1 might have declined to 50 by the following January 1, in which case we would say that the population has halved. If its decline continues at the same rate it will be down to 25 on the next January 1. Halving and doubling are the same process operating with equal force, the only difference being that it is running in opposite directions. The terms by which we measure the magnitude of the process should reflect that equivalence. This is poorly achieved by simply giving the multiplier of the growth, 2 for a doubling and 0.5 for a halving, and it becomes even more confusing when percentages are used. We need a metric that gives exactly the same figure for a halving as for a doubling, but with the sign reversed. This will make it obvious that a decrease is simply a negative rate of increase.

Wildlife Ecology, Conservation, and Management, Third Edition.
John M. Fryxell, Anthony R. E. Sinclair and Graeme Caughley.

Companion Website: www.wiley.com/go/Fryxell/Wildlife

This is achieved by expressing the rate of increase, positive or negative, as a geometric rate according to the following equation:

$$N_{t+1} = N_t\lambda = N_t e^r$$

where N_t is population size at time t, N_{t+1} is the population size a unit of time later, e is the base of natural logs taking the value 2.7182817, and r is the exponential rate of increase. The *finite rate of increase* (λ) is the ratio of the two censuses:

$$\lambda = N_{t+1}/N_t$$

and therefore the *exponential rate of increase* is:

$$r = \log_e(N_{t+1}/N_t) = \log_e\lambda$$

Let us test this on a doubling and a halving.

With a doubling:

$$\lambda = 200/100 = 2$$

and so:

$$r = \log_e\lambda = 0.693.$$

With a halving:

$$\lambda = 50/100 = 0.5$$

and so:

$$r = \log_e\lambda = -0.693.$$

Thus, a halving and a doubling both provide the same exponential rate of increase, 0.693, but in the case of a halving with the sign reversed (i.e. −0.693). This makes the point again that a rate of decrease is simply a negative rate of increase.

The finite rate of increase (i.e. the growth multiplier λ) and the exponential rate of increase r must each have a unit attached to them. In our example, the unit is a year, and so we can say that the population is multiplied by λ per year. The exponential rate r is actually the growth multiplier of $\log_e$ numbers per year. That is something of a mouthful, so we say that the population increased at an exponential rate r on a yearly basis. Note that λ and r are simply different ways of presenting the same rate of change; they do not contain independent information.

Unlike the finite rate of increase, the exponential rate of increase can be changed from one unit of time to another by simple multiplication and division. If $r = -0.693$ on a yearly basis then $r = -0.693/365 = -0.0019$ on a daily basis. This simplicity is not available for λ.

These equations are simplified to embrace only one unit of time. They can be generalized to:

$$N_t = N_0 e^{rt}$$

where N_0 is the population size at the beginning of the period of interest and N_t is the population size t units of time later. The average exponential rate of increase over the period is:

$$r = (\log_e(N_t/N_0))/t$$

which can also be written as:

$$r = (\log_e N_t - \log_e N_0)/t$$

It would be of a waste of data to use only the population estimates at the beginning and end of the period to estimate the average rate of increase between these two dates. If intermediate estimates are available, they can and should be included in the calculation to increase its precision. The appropriate technique is to take natural logarithms of the population estimates and then fit a linear regression to the data points, each comprising $\log_e N$ and t. A linear regression takes the form $y = a + bx$, where y is the dependent variable (in this case logged population size) and x is the independent variable (in this case time measured in units of choice). Our equation thus becomes:

$$\log_e N = a + bt$$

where a is the fitted value of $\log_e N$ when $t = 0$ and b is the increase in $\log_e N$ over one interval of time. Note that this is the definition of r, so $r = b$. The equation for the linear regression may thus be rewritten in the following manner:

$$\log_e N = a + rt.$$

This can be converted back to the notation used in the example where rate of increase was measured between only two points by designating the start of the period as time 0:

$$\log_e N_t = \log_e N_0 + rt$$

which with a little rearranging converts to:

$$r = (\log_e N_t - \log_e N_0)/t$$

as before. Fig. 5.1 shows such use of linear regression to estimate the rate of increase of the George River caribou herd in eastern Canada, yielding $r = 0.11$ (Messier *et al.* 1988).

5.2.1 *Intrinsic rate of increase*

The rate of increase of a population of vertebrates usually fluctuates gently most of the time, around a mean of zero. If conditions suddenly become more favorable, the population increases, the environmental improvement being reflected in a rise in fecundity and a decline in mortality.

The environmental change might be an increase in food supply, perhaps a flush of plant growth occasioned by a mild winter and a wet spring. The rate at which the population increases is then determined by two things: the amount of food available and the intrinsic ability of the species to convert this extra energy into enhanced fecundity and diminished mortality. It thus depends on an environmental effect and

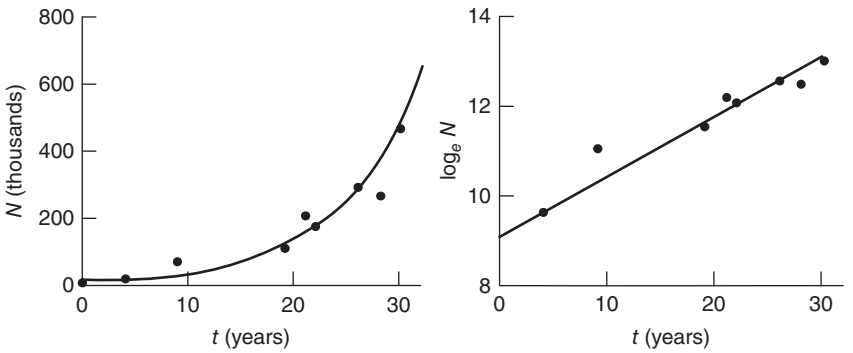

Fig. 5.1 Exponential population growth of the George River caribou herd, as discussed in the text. (After Messier *et al.* 1988.)

an intrinsic effect, but neither is without limit. From the viewpoint of the animal, both are constrained. There comes a point at which the animal has all the food it can eat, any further food having no additional effect on its reproductive rate and probability of survival. Similarly, an animal's reproductive rate is constrained at the upper limit by its physiology. Litters can be only so big and the interval between successive litters cannot be reduced below the gestation period. The potential rate of increase can never be very high, irrespective of how favorable the environmental conditions are, if the period of gestation is long (e.g. 22 months for the African elephant (*Loxodonta africana*)). All species, therefore, have a maximum rate of increase, which is called their *intrinsic rate of increase* (Fisher 1930) and symbolized r_m. This is a particularly important parameter in estimating sustainable yield (see Chapter 18). Populations do not attain this maximum very often. It requires a very high availability of food and a low density of animals, such that there is negligible competition for the food. These conditions are most closely approached when a population is in the early stage of active growth subsequent to the release of a nucleus of individuals into an area from which they were formerly absent. Fig. 5.2 gives the intrinsic rates of increase of several mammals.

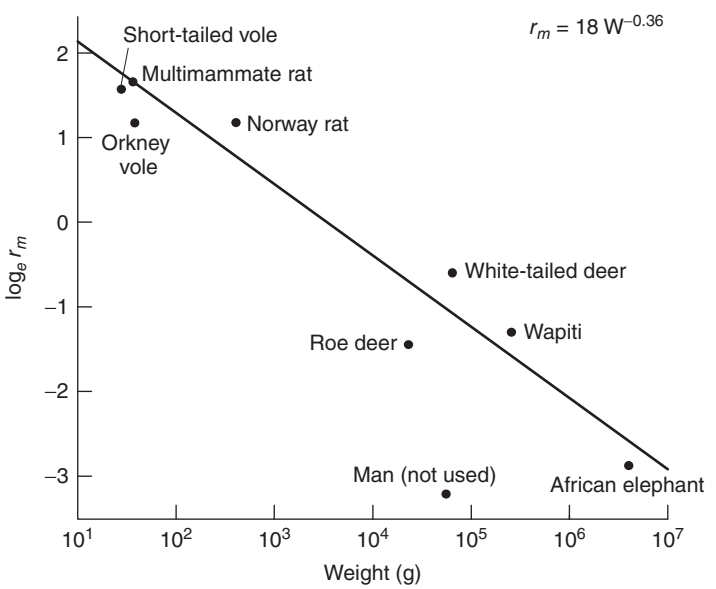

Fig. 5.2 Intrinsic rate of increase of mammals plotted against body weight. (After Caughley and Krebs 1983.)

Table 5.1 Expected intrinsic rates of increase r_m on a yearly basis for herbivorous mammals, as estimated from mean adult live weight.

Weight (kg)	r_m
1	1.50
10	0.65
100	0.29
1000	0.08

Alternatively, the rate can be estimated from the initial stages of growth of a population recovering from overhunting. This would work for blue whales (*Balaenoptera musculus*), for example, which are presently recovering from intense overharvesting between about 1925 and 1955 (Cherfas 1988).

Intrinsic rate of increase r_m tends to vary with body size. The following relationship has been calculated for herbivorous mammals (Caughley and Krebs 1983; Sinclair 1996):

$$r_m = 1.5W^{-0.36}$$

where W is mean adult live weight in kilograms. Table 5.1 gives r_m calculated according to this equation for a range of body weights. In the absence of other data, it provides an approximation that can be used to make a first estimate of sustained yield (see Chapter 18).

5.3 Geometric or exponential population growth

In 1798, Thomas Malthus recognized that populations have an intrinsic tendency towards exponential or geometric growth, just as a bank account at fixed interest grows geometrically with the amount of money it contains. The growth of such populations can be calculated as either a continuous or a discrete process. For simplicity, we will concentrate on discrete time representations of population growth. Strictly speaking, such models are most applicable to organisms whose patterns of deaths and births follow a seasonal or annual cycle of events, which includes most wildlife species. Consider, for example, a population whose finite growth rate $\lambda = 0.61$ and whose initial density N_0 is 1.5. The geometric growth model predicts subsequent changes in density over time according to the following equation:

$$N_t = N_0\lambda^t$$

The outcome depends on whether λ is larger or smaller than 1: when $\lambda < 1$ (Fig. 5.3), there is a decelerating pattern, while when $\lambda > 1$ (Fig. 5.4), there is an accelerating pattern.The geometric model can be readily translated into the exponential model:

$$N_t = N_0\, e^{r_{\max} t}$$

Hence, it is straightforward to shift between representations of population dynamics in continuous versus discrete time. Such simple models are most appropriate for small populations introduced into a new environment or for a short period following a perturbation. For example, the George River caribou herd in eastern Canada grew exponentially at a rate of $r = 0.11$ during a 30 year period following recovery from overharvesting (Messier *et al*. 1988).

5.4 Stability of populations

If we look at long-term records of animal populations, we see that some populations remain quite constant in size for long periods of time. Records of mute swans (*Cygnus olor*) in England from 1823 to 1872 (Fig. 5.5) show that although the population

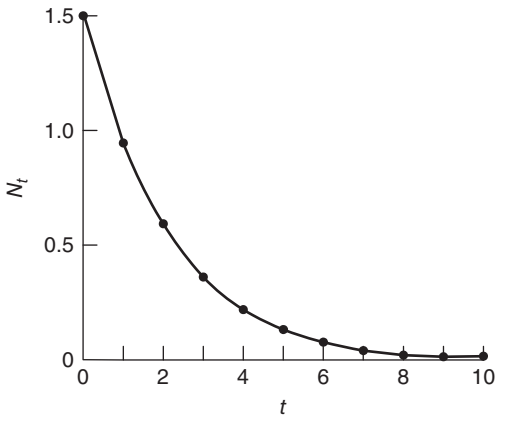

Fig. 5.3 Population changes according to the geometric model with $\lambda = 0.61$.

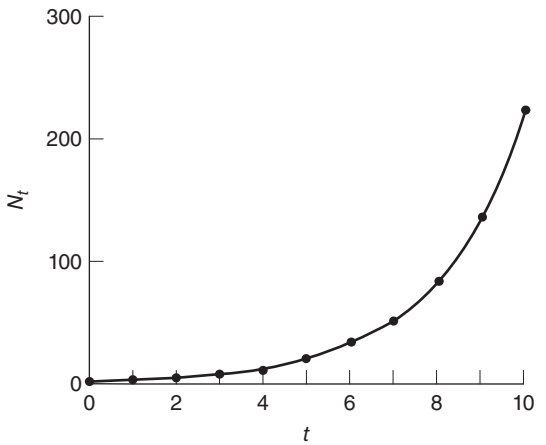

Fig. 5.4 Population changes according to the geometric model with $\lambda = 1.65$.

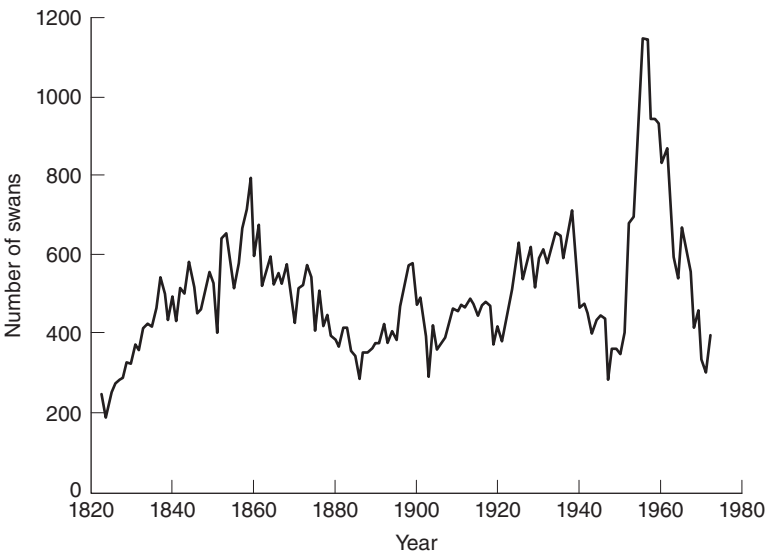

Fig. 5.5 Some populations remain within relatively close bounds over long time periods. The mute swan population of part of the River Thames in England (estimated by total counts) shows a steady level or gentle increase despite some perturbations due to severe winters, for example in 1946–1947 and 1963–1964. (Data from Cramp 1972.)

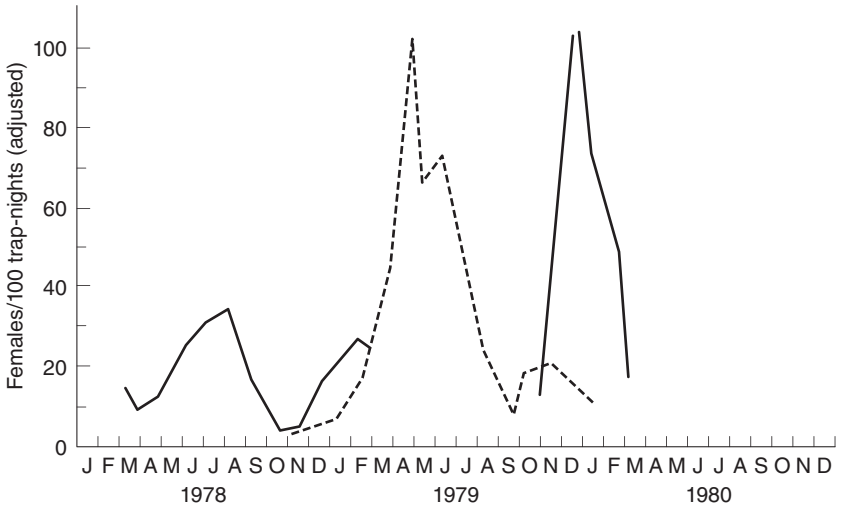

Fig. 5.6 Density indices for old female house mice on contour banks and in stubble fields of rice crops in southeastern Australia. Broken lines distinguish the crop cycle cohort of 1978–1979 from those of 1977–1978 and 1979–1980. The extent of the peak in January 1980 is unknown due to a poisoning campaign. (After Redhead 1982.)

fluctuates, it remains within certain limits (190–1150). Other populations, such as those of insects and house mice (*Mus domesticus*) in Australia (Fig. 5.6), fluctuate to a much greater extent and furnish no suggestion of an equilibrium population size. Nevertheless, such populations do not always go extinct and remain in the community for long periods. Occasionally one finds unusual situations where populations show regular cycles. The snowshoe hare (*Lepus americanus*) in northern Canada has the clearest (Fig. 5.7), as indicated by the furs collected by trappers for the Hudson Bay Company over the past two centuries (MacLulich 1937).

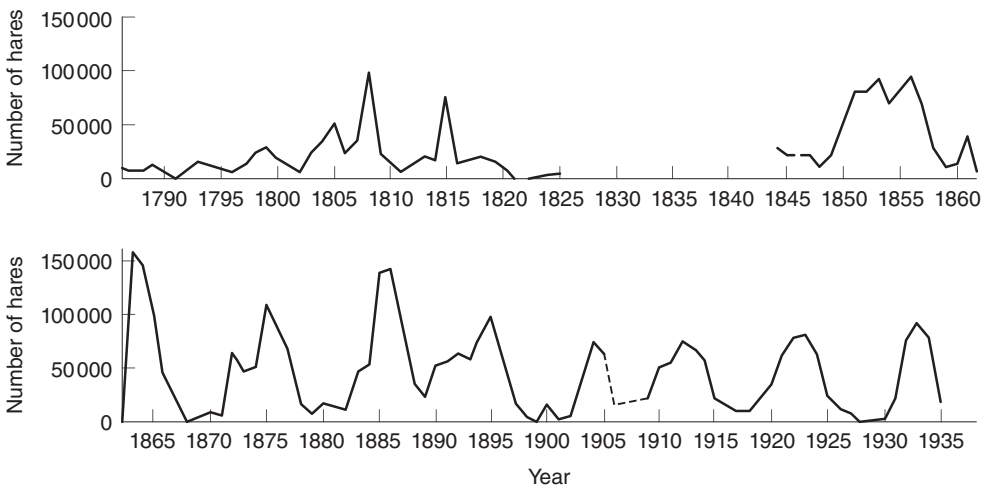

Fig. 5.7 Snowshoe hares in the boreal forest of Canada show regular fluctuations in numbers with a 10-year periodicity. Data are from the Hudson Bay Company fur records up to 1903 and questionnaires thereafter. (After MacLulich 1937.)

This relative constancy of population size, or at least fluctuation within limits, is in contrast to the intrinsic ability of populations to increase rapidly. The fact that population increase is limited suggests that there is a mechanism that slows down the rate of increase and so regulates the population.

5.5 The theory of population limitation and regulation

Populations have inputs of births and immigrants and outputs of deaths and emigrants. For simplicity, we will confine discussion to a self-contained population having only births B and deaths D per unit time.

5.5.1 *Density dependence*

If either the proportion of the population dying increases or the proportion being born decreases as population density increases then we define these changes as being *density-dependent*. The underlying causes of the changes in these rates are called *density-dependent factors*.

Births and deaths as a proportion of the population (B/N_t, D/N_t) can be related to the instantaneous birth b and death d rates in the following way.The change in population per unit time is:

$$N_{t+1} - N_t = B - D$$

The instantaneous rate of increase r is given by:

$$r = b - d$$

The finite rate of increase R is given by:

$$\lambda = N_{t+1}/N_t = e^r$$

Therefore:

$$e^{b-d} = (N_{t+1}/N_t) = (B - D + N_t)/N_t$$

If $d = 0$, $D = 0$ then:

$$e^b = (B + N_t)/N_t = (1 + (B/N_t))$$

and:

$$b = \ln(1 + (B/N_t))$$

Similarl,y if $b = 0$, $B = 0$, and D/N_t is much less than 1 then:

$$d = \ln(1 + (D/N_t))$$

If B and D fall in the range of 0–20% of the population then b and d are nearly linear on N. They remain approximately linear even if B and D are 20–40% of N. This range covers most of the examples we see in nature, so for our purposes we can say that D/N_t and B/N_t change with density in the same way as do b and d, and both go through the origin.

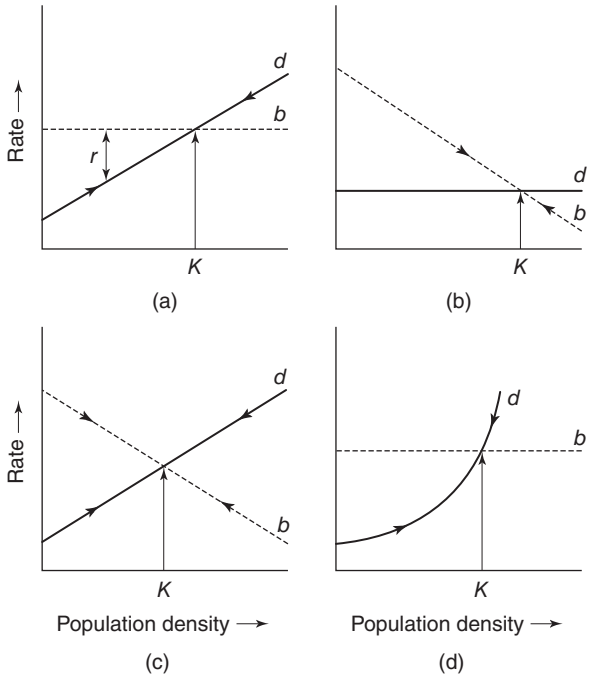

Fig. 5.8 Model of density-dependent and density-independent processes. (a) Birth rate, *b*, is held constant over all densities while mortality, *d*, is density-dependent. The population returns to the equilibrium point, *K*, if disturbed. The instantaneous rate of increase, *r*, is the difference between *b* and *d*. (b) As in (a) but *b* is density-dependent and *d* is density-independent. (c) Both *b* and *d* are density-dependent. (d) *d* is curvilinear so that the density dependence is stronger at higher population densities.

In Fig. 5.8a we plot *b* against density (or population size) *N* as a constant so that it is a horizontal line. If we now plot *d* as an increasing function of density, we see that where the two lines cross, $b = d$ and the population is stationary at the equilibrium point *K*. The difference between the *b* and *d* lines represents *r*, which declines linearly as density increases, in the same way as it does for the logistic curve (see Section 5.6). In Fig. 5.8a, the decline in *r* is due solely to *d* being density-dependent. Since *b* (or B/N_t) is constant in this case, we describe it as *density-independent* (i.e. it is unrelated to density). In real populations, density-independent factors such as weather can affect birth and death rates randomly. Rainfall acted in this way on greater kudu (*Tragelaphus strepsiceros*) in Kruger National Park in South Africa, causing mortality of juveniles (Owen-Smith 1990).

We can apply the same arguments if we assume that *b* is density-dependent and *d* is density-independent (Fig. 5.8b) or if both are density-dependent (Fig. 5.8c). So far we have assumed that the density-dependent factor has a linear effect on rate of increase, as in the logistic curve. However, density-dependent mortality is more likely to be curvilinear, as in Fig. 5.8d.

5.5.2 *Limitation and limiting factors*

In Fig. 5.9 we take the argument a little further. Let us assume a constant (density-independent) birth rate *b*. Shortly after birth, a density-independent mortality d_1 (depicted here as a constant) kills some of the babies so that inputs are reduced to b_1. There follows a density-dependent mortality d_2, and the population reaches an equilibrium at K_3. If mortality d_1 had not occurred (or was smaller), the equilibrium population would be at K_1. Therefore, the presence or absence of the density-independent factor causing d_1 alters the size of the equilibrium population.

The strength or severity of the density-dependent factor is indicated by the slope of d_2. If the density-dependent factor becomes stronger, for example to produce d_3 instead

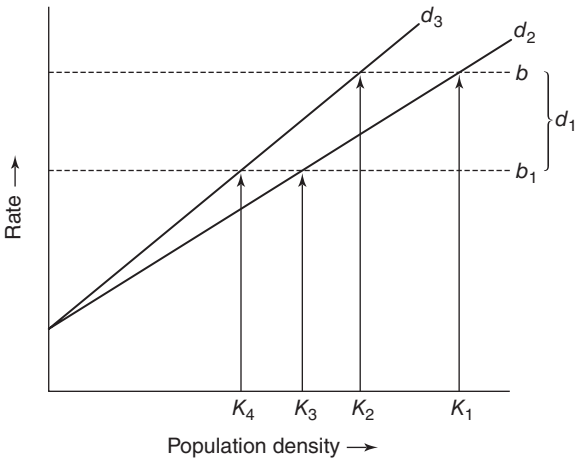

Fig. 5.9 Model showing that the equilibrium point, K, can vary with both density-dependent and density-independent processes. Birth rate, b, is held constant over all densities. In sequence, a density-independent mortality d_1 reduces the input to the population to b_1. There follows a density-dependent mortality d_2 or d_3. The intercept of b or b_1 with d_2 or d_3 determines the equilibrium (K_1–K_4).

of d_2, the slope becomes steeper and the equilibrium population drops from K_3 to K_4 (or from K_1 to K_2 if d_1 is absent). Thus, altering the strengths of density-dependent factors also alters the size of the equilibrium population.

We define the process determining the size of the equilibrium population as *limitation* and the factors producing this as *limiting factors*. We can see therefore that both density-dependent and density-independent factors affect the equilibrium population size; they are thus both limiting factors. Any factor that causes mortality or affects birth rates is a limiting factor.

5.5.3 *Regulation*

Populations are often disturbed from their equilibrium K by temporary changes in limiting factors (a severe winter or drought or an influx of predators might reduce the population; a mild winter or good rains might increase it). The subsequent tendency to return to K is largely due to the effect of density-dependent factors, and this process is called *regulation*. Therefore, *regulation is the process whereby a density-dependent factor tends to return a population to its equilibrium*. We say "tends to return" because the population may be continually disturbed, so that it rarely reaches equilibrium. Nevertheless, this tendency to return to equilibrium results in the population remaining within a certain range of sizes. Superficially, it appears as if the population has a boundary to its size and fluctuates randomly within this boundary. However, it is more constructive to picture random fluctuations in both the density-independent (d_1) and density-dependent (d_2) mortalities as the shaded range in Fig. 5.10a. This results in a fluctuation of the equilibrium population indicated by the range of K. Fig. 5.10a shows that this range of K is relatively small when the density-dependent mortality is strong (steep part of the curve); Fig. 5.10b shows the much greater range of K (which we see in nature as fluctuations in numbers) when the density-dependent mortality is weak. Note in Fig. 5.10a and b that differences in the amplitudes of fluctuations are due to changes in the strength of the density-dependent mortality, as we have held density-independent (random) mortality constant in this case.

5.5.4 *Delayed and inverse density dependence*

Some mortality factors do not respond immediately to a change in density but act after a delay. Such *delayed density-dependent factors* might be predators whose populations lag behind those of their prey or a drop in food supply causing the delayed action of starvation. Both causes can have a density-dependent effect on the population, but

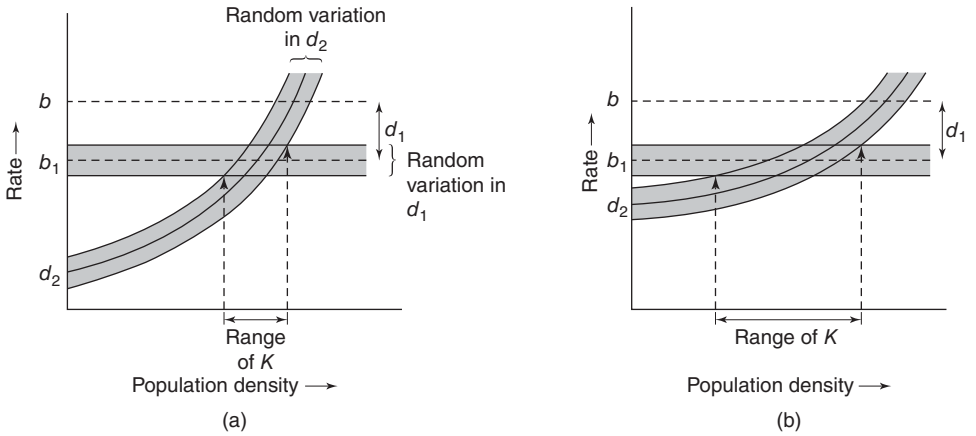

Fig. 5.10 Random variation in the mortalities d_1 and d_2 (indicated by the shaded area) are the same in (a) and (b). In (a) there is stronger density dependence at the intercept of b_1 and d_2 than in (b), and this difference results in a smaller range of equilibria, K, in (a) than in (b).

this effect is related to density at some previous time period rather than the current one. For example, a 34-year study of white-tailed deer in Canada indicated that both the population rate of change and the rate of growth of juvenile animals were dependent on population size several years previously, rather than to current population size (Fryxell *et al*. 1991). A similar relationship was found with winter mortality of red grouse (*Lagopus lagopus*) in Scotland. Delayed density dependence is indicated when mortality is plotted against current density, and the points show an anticlockwise spiral if they are joined in temporal sequence. These delayed mortalities usually cause fluctuations in population size.

Predators can also have the opposite effect to density dependence, termed an *inverse density-dependent* or *depensatory* effect. They take a decreasing proportion of the prey population as it increases, allowing the population to increase faster as it becomes larger. Conversely, if a prey population is declining for some reason, predators will take an increasing proportion and so drive it down even faster towards extinction. In either case we do not see a predator–prey equilibrium. We explore this further in Chapter 9.

5.5.5 *Carrying capacity*

Carrying capacity is one of the most common phrases in wildlife management. It conveys a variety of meanings, however, and unless we are careful and define it precisely, it can be a source of confusion (Caughley 1976, 1981). Some of its more common uses are discussed in this section.

Ecological carrying capacity

This can be thought of abstractly as the K of the logistic equation, which we derive later in this chapter (Section 5.8). In reality it is the natural limit of a population set by resources in a particular environment. It is one of the equilibrium points that a population tends towards through density-dependent effects from lack of food, space (e.g. territoriality), cover, or other resources. As we discussed earlier, if the environment changes briefly, it deflects the population from achieving its equilibrium and so produces random fluctuations about that equilibrium. A long-term environmental change

can affect resources, which in turn alters *K*. Again, the population changes by following or tracking the environmental trend.

There are other possible equilibria that a population might experience through regulation by predators, parasites, or disease. Superficially, they appear similar to the equilibrium produced by a lack of resources, because if the population is disturbed through culling or weather events it may return to the same population size. To distinguish the equilibria produced by predation, by resource limitation, and by a combination of the two, we need to know whether predators or resources or both are affecting *b* and *d*.

Economic carrying capacity

This is the population level that produces the maximum offtake (or maximum sustained yield) for culling or cropping purposes. It is this meaning that is implied when animal production scientists and range managers refer to "livestock carrying capacity." We should note that this population level is well below the ecological carrying capacity. For a population growing logistically, its level is half of *K* (Caughley 1976).

Other senses

We can define carrying capacity according to our particular land use requirements. At one extreme, we can rate the carrying capacity for lions on a Kenya farm or wolves on a Wyoming ranch as zero (i.e. farmers cannot tolerate large predators killing their livestock). A less extreme example is seen where the aesthetic requirements of tourism entail reducing the impact of animals on the vegetation. Large umbrella-shaped *Acacia tortilis* trees make a picturesque backdrop to the tourist hotels in the Serengeti National Park, Tanzania. In the early 1970s, elephants began to knock these trees over. Whereas elephants could be tolerated at ecological carrying capacity in the rest of the park, in the immediate vicinity of the hotels the "carrying capacity" for elephants was much lower and was determined by human requirements for scenery.

5.5.6 *Measurements of birth and death rates*

Birth rates are inputs to the population. Ideally we would like to measure conception rates (*fecundity*), pregnancy rates in mammals (*fertility*), and births or egg production. In some cases it is possible to take these measurements, as in the Soay sheep of Hirta (Clutton-Brock *et al*. 1991). Pregnancies can be monitored in a variety of ways, including ultrasound, x-rays, blood protein levels, urine hormone levels, and rectal palpation of the uterus (in large ungulates). In many cases, however, these are not practical for large samples from wild populations.

Births can be measured reasonably accurately for seal species, in which the babies remain on the breeding grounds throughout the birth season. Egg production, egg hatching success, and fledgling success can also be measured accurately in many bird populations. However, in the majority of mammal species birth rates cannot be measured accurately, either because newborn animals are rarely seen (as in many rodents, rabbits, and carnivores) or because many die shortly after birth and are not recorded in censuses (as in most ungulates). In these cases we are obliged to use an approximation to the real birth rate, such as the proportion of the population consisting of juveniles first entering live traps for rodents and rabbits, or juveniles entering their first winter for carnivores and ungulates. These are valid measures of recruitment.

Death rates are losses to the population. Ideally they should be measured at different stages of the life cycle to produce a life table (see Chapter 13). Once sexual maturity

is reached, age classes often cannot be identified and all subsequent mortality is therefore lumped together as "adult" mortality. Mortality can be measured directly by using mortality radios, which indicate when an animal has died, as Boutin *et al*. (1986) and Trostel *et al*. (1987) did for snowshoe hares in northern Canada. Survivorship can be calculated over varying time periods by the method of Pollock *et al*. (1989).

Mortality caused by predators can also be measured directly if the number of predators (numerical response) and the amount eaten per predator (functional response) are known (see also Chapters 7 and 9). Such measurements are possible for those birds of prey that regurgitate a single pellet containing the bones of their prey each day. With appropriate sampling, the number of pellets indicates the number of predators, while prey per pellet shows the amount they eat. This method was used for raptors (in particular the black-shouldered kite, *Elanus notatus*) eating house mice during mouse outbreaks in Australia (Sinclair *et al*. 1990).

5.5.7 *Implications*

We should be aware of a number of problems associated with the subject of population limitation and regulation:

1 Much of the literature uses the terms "limitation" and "regulation" in different ways. In many cases they are used synonymously, but the meanings differ between authors. Since any factor, whether density-dependent or density-independent, can determine the equilibrium point for a population, any factor affecting b or d is a limiting factor. It is therefore a trivial question to ask whether a certain cause of mortality limits a population: it has to. The more profound question is how mortality and fecundity factors affect the equilibrium.

2 Regulation requires, by our definition, the action of density-dependent factors. Density dependence is necessary for regulation, but it may not be sufficient. First, the particular density-dependent factor that we have measured, such as predation, may be too weak, and other regulating factors may be operating. Second, some density-dependent factors have too strong an effect and thus cause fluctuations, rather than a tendency towards equilibrium (see Section 5.9).

3 The demonstration of density dependence at some stage in the life cycle does not indicate the cause of the regulation. For example, if we find that a deer population is regulated through density-dependent juvenile mortality, this does not give us any indication as to the cause of the mortality. Correlation with population size is merely a convenient abbreviation that hides underlying causes. Density itself is not causing the regulation; the possible underlying factors are competition for resources, competition for space through territoriality, and an effect of predators, parasites, and diseases (see Section 5.10).

5.6 **Evidence for regulation**

There are three ways of detecting whether populations are regulated. First, as we have seen in Section 5.5.3, regulation causes a population to return to its equilibrium after a perturbation. Perturbation experiments should therefore detect this return. Similarly, natural variation in population density, provided it is of sufficient magnitude, can be used to test whether per capita growth rates decline with density (see Chapters 15, 16, and 18). Second, if we plot separate and independent populations at their natural carrying capacity against some index of resource (often a weather factor), there should be a relationship. Third, we can try to detect density dependence in the life cycle.

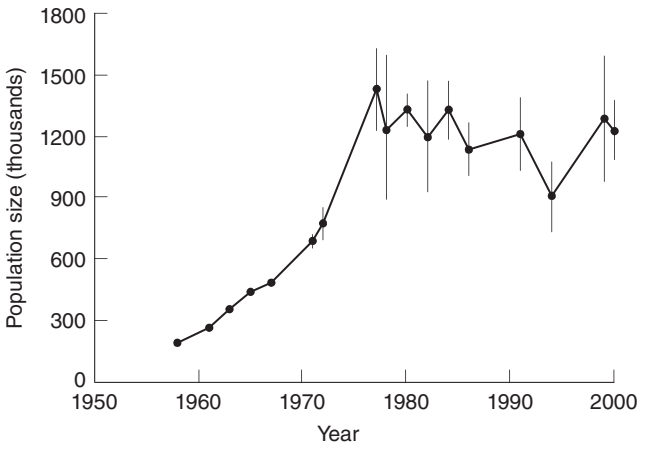

Fig. 5.11 The wildebeest population in the Serengeti increased to a new level determined by intraspecific competition for food after the disease rinderpest was removed in 1963. (After Mduma *et al.* 1999 and unpublished data.)

5.6.1 *Perturbation experiments*

If a population is moved experimentally to either below or above its original density and then returns to this same level, we can conclude that regulation is occurring. An example of downward perturbation is provided by the northern elk herd of Yellowstone National Park (Houston 1982). Prior to 1930, population estimates ranged between 15 000 and 25 000. Between 1933 and 1968, culling reduced the population to 4000 animals. Culling then ceased and the population rebounded to around 20 000 (Coughenour and Singer 1996). This result is consistent with regulation through intraspecific competition for winter food (Houston 1982), since there were no natural predators of elk in Yellowstone until the return of wolves in the early 1990s.

Density is usually recorded as numbers per unit area. If space is the limiting resource (as it might be in territorial animals), or if it is a good indicator of some other resource such as food supply, numbers per unit area will suffice in an investigation of regulation. However, space may not be a suitable measure if density-independent environment effects (e.g. temperature, rainfall) cause fluctuations in food supply. It may be better to record density as animals per unit of available food or per unit of some other resource.

The Serengeti migratory wildebeest experienced a perturbation (Fig. 5.11) when an exotic virus, rinderpest, was removed. The population increased fivefold from 250 000 in 1963 to 1.3 million in 1977 and then leveled out (Mduma *et al.* 1999). This example is less persuasive than that of the Yellowstone elk because the pre-rinderpest density (before 1890) was unknown, but evidence on reproduction and body condition suggests that rinderpest held the population below the level allowed by food supply, a necessary condition for a perturbation experiment implicating a disease.

A case of a population perturbed above equilibrium is provided by elephants in Tsavo National Park, Kenya (Laws 1969; Corfield 1973). From 1949 until 1970, the population was increasing, due in part to immigration from surrounding areas in which human cultivation had displaced the animals. A consequence of this artificial increase in density was depletion of the food supply within reach of water. In 1971, the food supply ran out and there was starvation of females and young around the water holes. After this readjustment of density, the vegetation regenerated and starvation mortality ceased.

5.6.2 *Mean density and environmental factors*

A population that is uninfluenced by dispersal and unregulated (i.e. has no density-dependent factors affecting it) will fluctuate randomly under the influence of weather and will eventually drift to extinction (DeAngelis and Waterhouse 1987).

Just by chance, there may for a time be a correlation between density and environmental factors. However, if we take many separate populations, the probability that all of them are simultaneously correlated with an environmental factor by chance alone is very small. Therefore, if we find a correlation between mean densities from independent populations and environmental factors, a strong inference can be made that weather is influencing some resource for which animals are competing, resulting in regulation about some equilibrium point.

An example of this approach is shown in Schluter's (1988) study of seed-eating finches in Kenya (Fig. 5.12): finch abundance from various populations is correlated with seed abundance. Other examples of density correlated with weather factors are given in Sinclair (1989).

5.6.3 *Examples of density dependence*

As discussed in Section 5.5.7, density dependence is a necessary but not sufficient requirement for demonstrating regulation. There are an increasing number of studies in the bird and mammal literature demonstrating density-dependent stages in the life cycle. For birds (Fig. 5.13a), the long-term study of great tits (*Parus major*) in Oxford, UK has shown that winter mortality of juveniles is related to the number of juveniles entering the winter (McCleery and Perrins 1985). In contrast, (Fig. 5.13b) it is early chick mortality in summer that is density-dependent for the English partridge (*Perdix perdix*) (Blank *et al*. 1967).

For mammals, density-dependent juvenile mortality has been recorded for red deer on the Isle of Rum, UK (Clutton-Brock *et al*. 1985) (Fig. 5.14a), for reindeer in Norway (Skogland 1985) (Fig. 5.14b), for feral donkeys (*Equus asinus*) in Australia (Choquenot 1991), and for greater kudu in South Africa (Owen-Smith 1990). Adult mortality was density-dependent for African buffalo in the Serengeti (Sinclair 1977). In each case, the cause was lack of food at critical times of year. Reproduction is known to be

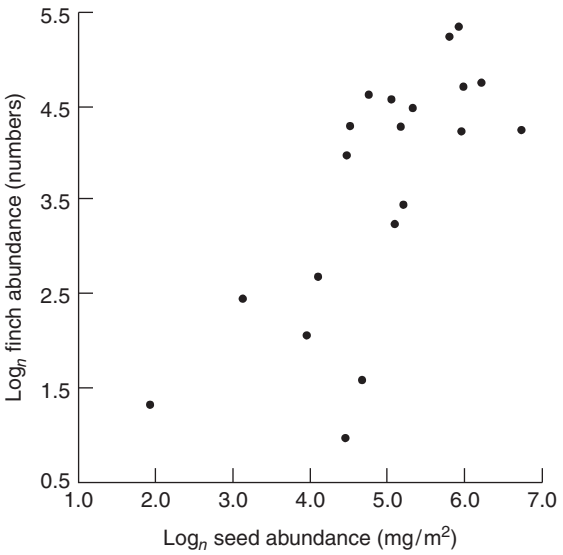

Fig. 5.12 The total abundance of seedeating finches in savanna habitats of Kenya is related to the abundance of the food supply. Such a positive relationship in unconnected populations may demonstrate regulation. (After Schluter 1988.)

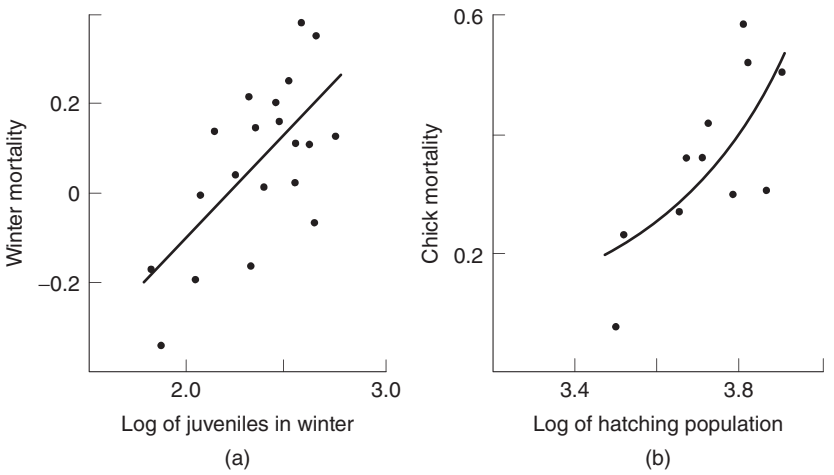

Fig. 5.13 Examples of density-dependent mortality in birds. (a) Great tit (*Parus major*) overwinter mortality (log of [juveniles in winter/first year breeding population]) plotted against log juvenile density in winter. (After McCleery and Perrins 1985.) (b) Chick mortality of European partridge (*Perdix perdix*) (measured as log hatching population/log population at 6 weeks) plotted against log hatching population, in Hampshire, UK. (After Blank *et al.* 1967.)

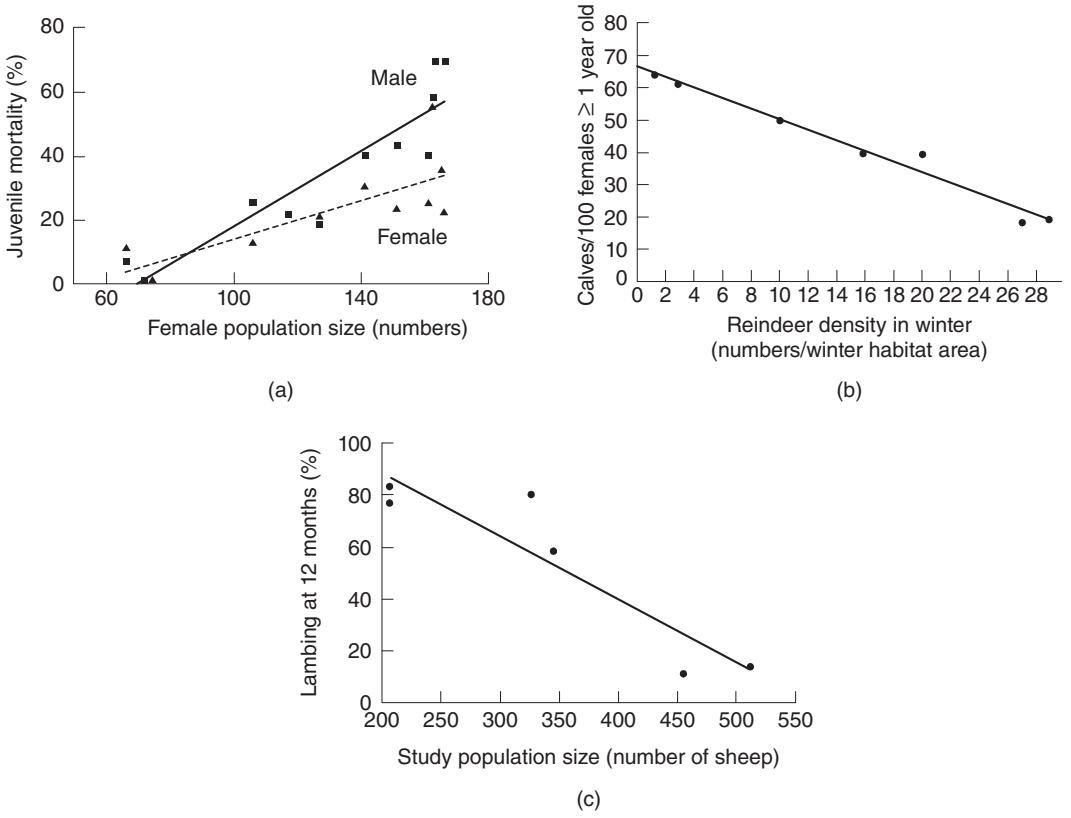

Fig. 5.14 Density dependence in large mammals. (a) Juvenile mortality of male and female red deer on the Isle of Rum, UK. (After Clutton-Brock *et al.* 1985.) (b) Juvenile recruitment per 100 female reindeer older than 1 year in Norway. (After Skogland 1985.) (c) Fertility rate of 1-year-old Soay sheep on St. Kilda island. (After Clutton-Brock *et al.* 1991.)

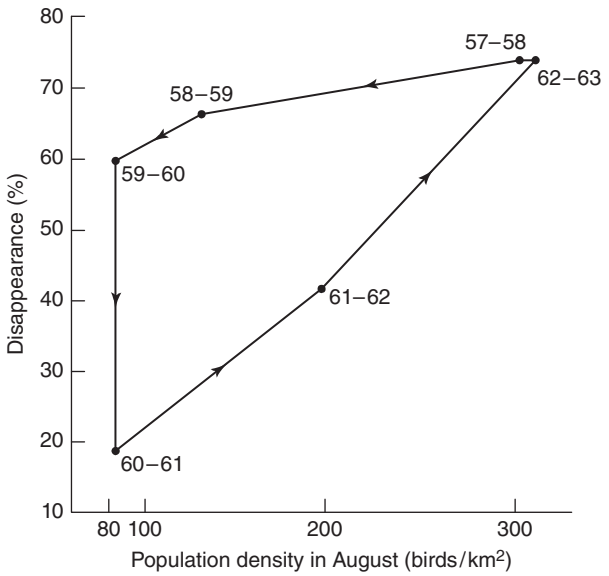

Fig. 5.15 The proportion of a red grouse population in Scotland which disappears over winter (August–April) is related to population density in the previous August in a complex way. Mortality varied according to whether the population was increasing or decreasing. By joining the points sequentially an anticlockwise cycle is produced, indicating a delayed density-dependent effect in the cause of the mortality. By plotting the percentage disappearance against density 1 year earlier, a closer fit can be obtained for a regression line. Thus the delay is 1 year. Numbers at the points are years. (After Watson and Moss 1971.)

density-dependent in both birds (Arcese *et al*. 1992) and mammals (Clutton-Brock *et al*. 1991). Fig. 5.14c shows that the proportion of Soay sheep that give birth at 12 months of age declines with density. Fowler (1987) reports over 100 studies of terrestrial and marine mammal populations in which density dependence was detected.

Delayed density dependence has been recorded in winter mortality of snowshoe hares in the Yukon and in overwinter mortality of red grouse in Scotland (Watson and Moss 1971) (Fig. 5.15). For the hares, the delay appears to have been due to a lag of 1–2 years in the response of predator populations to changing hare numbers (Trostel *et al*. 1987), while for the grouse the delay came from the density response to food conditions in the previous year (see Section 5.10.3).

5.7 Applications of regulation

Causes of population change can be divided into (i) those that disrupt the population and often result in "outbreaks," which can be either density-dependent or density-independent, and (ii) those that regulate and therefore return the population to original density after a disturbance. These are always density-dependent.

Knowledge of regulation may be useful for management of house mice (*Mus domesticus*) plagues in Australia. In one experimental study (Barker *et al*. 1991), mice in open-air enclosures were contained by special mouse-proof fences. The objective was to create high densities, mimicking plague populations, in order to test the regulatory effect of a nematode parasite (*Capillaria hepatica*). It turned out that the effect of the parasite could not be tested because other factors regulated the population and thus obscured it. The replicated populations declined simultaneously. Why did this happen? By dividing up the life cycle into stages, the researchers found that late juvenile and adult mortality were strongly density-dependent but that other stages, including fertility and newborn mortality, were not. This allowed them to discount causes that would affect reproduction and focus more closely on what was happening among adults, particularly in terms of social interactions.

Other studies suggest that mouse populations in Australia may be regulated by predators, disease, and juvenile dispersal (Redhead 1982; Sinclair *et al*. 1990). Under conditions of superabundant food following good rains, the reproductive rate of females increased faster than the predation rate and an outbreak of mice occurred. The implication of these results for management is that if reproduction can be reduced, for example through infections of the *Capillaria* parasite, then predation may be able to prevent outbreaks even in the presence of abundant food for the mice.

5.8 Logistic model of population regulation

At the beginning of this chapter, we derived the geometric and exponential models of population growth. In 1838, Pierre-Francois Verhulst published a paper that challenged the assumption of unlimited growth implicit in these models. Verhulst argued that the per capita rate of change (dN/Ndt) should decline proportionately with population density, simply due to a finite supply of resources being shared equally among individuals. If each individual in the population gets a smaller slice of the energy "pie" as N increases, this will prevent them from devoting as much energy to growth, reproduction, and survival as would be possible under ideal conditions. Changes in demographic parameters lead to corresponding changes in the finite rate of population growth λ_t or its equivalent exponential rate r_t, where t denotes a specific point in time. Other factors, such as risk of disease, shortage of denning sites, and aggressive interactions among population members may also cause the rate of population growth to decline with population size. The simplest mathematical depictions of such phenomena are commonly termed "logistic" models.

There are numerous ways to represent logistic growth. For simplicity, we will focus on population growth modeled in discrete time, which is often a reasonable approximation for species that live in a seasonal environment. One of the most commonly used forms is called the Ricker equation, in honor of the Canadian fisheries biologist, Bill Ricker, who first suggested its application to salmon stocks (Ricker 1954):

$$N_{t+1} = N_t e^{r_{\max}\left(1-\frac{N_t}{K}\right)}$$

The Ricker logistic equation represents the exponential rate of increase under ideal conditions as $r_{\max}$, with a proportionately slower rate of increase with each additional individual added to the population. When the rate of increase has slowed to the point that births equal deaths, the population has reached its carrying capacity K. These two population parameters ($r_{\max}$ and K) dictate how fast the population recovers from any perturbation to abundance.

A population growing according to the logistic equation will have slow growth when N is small, will grow most rapidly when N is of intermediate abundance, and will grow slowly again as N approaches carrying capacity K (Fig. 5.16). This kind of sigmoid or S-shaped pattern is often termed *logistic* growth.

At first, it may seem somewhat counterintuitive that a proportional decline in per capita demographic rates should produce the nonlinear growth pattern seen in Fig. 5.16. The reason lies in the fact that population changes are dependent on both population size and per capita growth rate, in much the same way that the growth of a bank account depends on both the money already in the account and the interest rate. When a population is small, the per capita rate of change will tend to be large, in fact close to $r_{\max}$, either because birth rates are high or because mortality rates are low. Nonetheless, the population will still display a slight change from one year to the

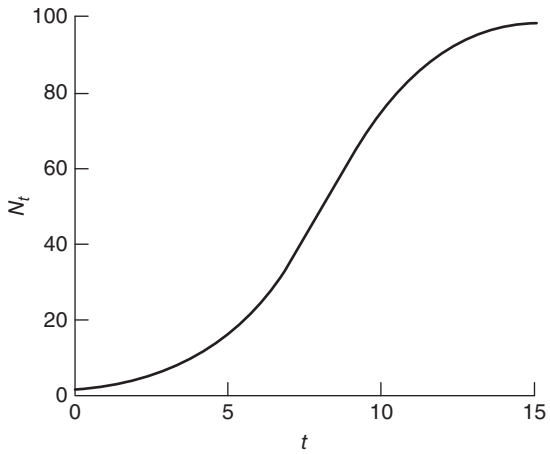

Fig. 5.16 Population growth according to the logistic equation, with $r_{max} = 0.5$, initial population density $N_0 = 1.5$, and carrying capacity $K = 100$.

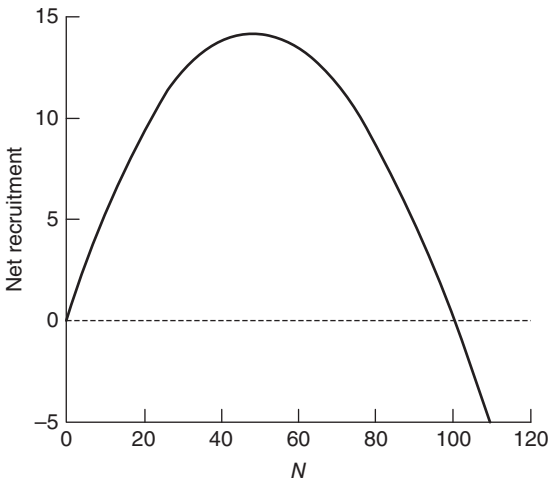

Fig. 5.17 Net recruitment ($N_{t+1} - N_t$) as a function of population density N_t, according to the Ricker logistic growth model, with $r_{max} = 0.5$ and $K = 100$.

next. At the other end of the spectrum, even when N is enormous the population will similarly display only modest change from year to year. This is because the per capita rate of growth is small, due either to low birth rates or high mortality rates. It is only when the population is of intermediate size and growing at intermediate per capita rate that growth is maximized (Fig. 5.17).

Population data displaying the classic sigmoid pattern of change are rare. They will only be seen when a population is reduced to very low initial density and then monitored closely over an extended period. So, logistic growth will not be obvious in most populations that we might see around us in nature, which are presumably close to their carrying capacity. In some cases, however, populations have been perturbed (reduced) to low densities, giving us a rare glimpse of logistic growth in the field. For example, as we discussed earlier, the Yellowstone elk herd has been aggressively culled at various times in the past, particularly in the late 1960s. Cessation of culling operations, stimulated by a new policy of natural regulation in US National Parks, led to a subsequent pattern of elk recovery reminiscent of the sigmoid pattern predicted by the logistic model (Fig. 5.18). Similarly, release of the Serengeti wildebeest population from the

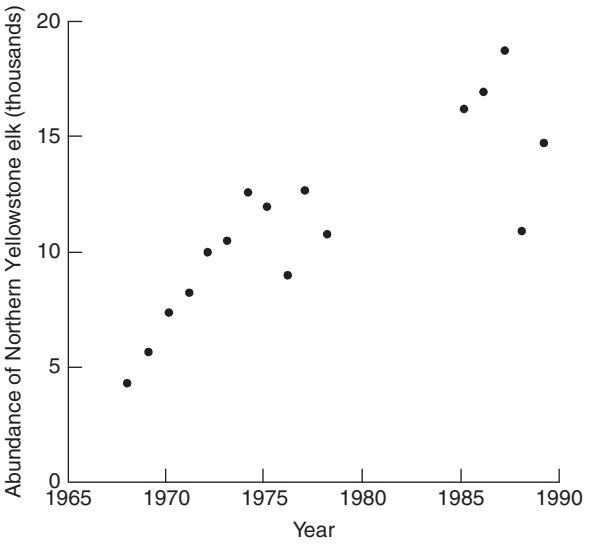

Fig. 5.18 Population dynamics of northern Yellowstone elk between 1968 and 1989. (Data from Coughenour and Singer 1996.)

exotic disease rinderpest led to a subsequent sigmoid pattern of change (Fig. 5.11) reminiscent of the logistic model. Indeed, perturbation is an important ingredient in detecting natural regulation and logistic growth because it gives us evidence to work with, unlike populations kept close to their ecological carrying capacity. We demonstrate how to estimate the parameters for the Ricker logistic model, and compare it to other possible population growth models, in Chapter 15.

5.9 **Stability, cycles, and chaos**

Paradoxically, the same density-dependent processes that are responsible for natural regulation can also induce population fluctuations, at least under special circumstances. One way that this can happen is when the maximum rate of growth is particularly high. For example, consider the dynamics of a hypothetical population whose maximum rate of increase $r_{max} = 3.3$ and carrying capacity $K = 100$ (Fig. 5.19). In this case the population does not increase smoothly over time and level off at the carrying capacity but rather fluctuates erratically over time, with no apparent repeated pattern. Such a pattern of population change is known as *deterministic chaos* (May 1976). It arises because the population grows so fast that it tends to overshoot the carrying capacity, a process known as *overcompensation* (May and Oster 1976). Once above the carrying capacity, the net recruitment is negative (Fig. 5.19), so the population declines rapidly. Repetition of this boom–bust pattern of overshooting and subsequent decline results in the erratic fluctuations of deterministic chaos seen in Fig. 5.19. For lower rates of increase ($2.0 < r_{max} < 2.7$), the pattern of fluctuation will be regular cycles rather than deterministic chaos, but the underlying cause is still overcompensation.

The underlying cause of instability due to overcompensatory density dependence can be better appreciated by plotting the population dynamics over time on a graph with N_t on the horizontal axis and N_{t+1} on the vertical (Fig. 5.20). The diagonal identifies potential points of equilibria, at which $N_{t+1} = N_t$. We will also plot the recruitment curve. Dynamics are plotted by starting at a particular value of N_0, projecting upwards to the recruitment curve (which identifies the next year's population density), and

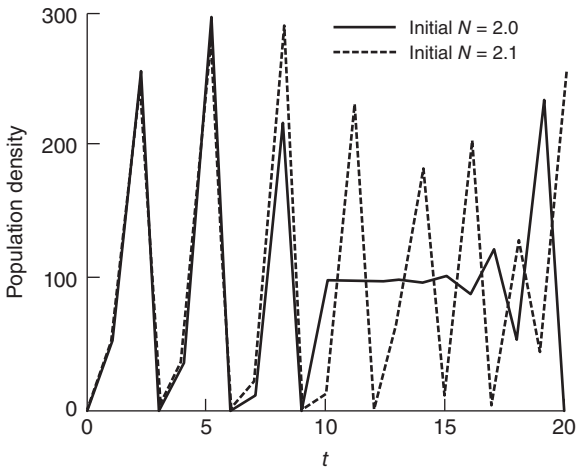

Fig. 5.19 Simulated dynamics over time of two different populations growing according to the Ricker logistic equation, with $r_{max} = 3.3$ and $K = 100$. The first population was initiated at a density of 2.0 individuals per unit area, whereas the second population was initiated at a slightly higher density of 2.1 individuals per unit area. The rapid divergence in population dynamics due to slight changes in starting conditions is typical of deterministic chaos.

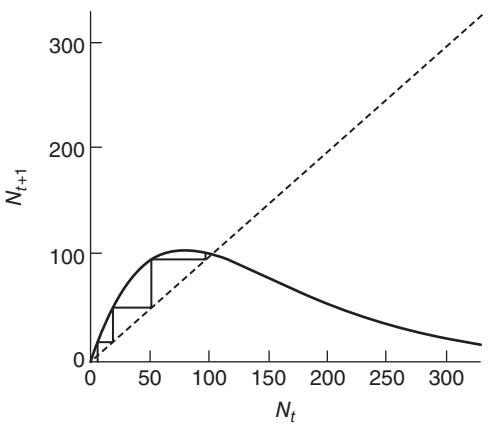

Fig. 5.20 Plot of predicted recruitment (N_t+1) relative to N_t (the heavy curve), equilibrium line at which N_t+1 = N_t (thin broken line), and trajectory of population dynamics over time for a simulated population following the Ricker logistic model, with r_{max} = 1.3 and $K = 100$ (thin solid line).

then projecting horizontally to the dotted equilibrium line, before repeating the process. At modest values of r_{max}, the recruitment curve is low and has a shallow angle of incidence as it intersects the equilibrium line. The result is that the population trajectory becomes pinched between the recruitment curve and the equilibrium line as it converges on K. This leads to stability.

Now, let us consider the pattern arising when $r_{max} = 3.3$ (Fig. 5.21). The recruitment curve has a pronounced hump and intersects the equilibrium line at a sharp angle (> 90°). The recruitment curve is so sharply peaked that recruitment events tend to overshoot the carrying capacity. This leads to the population collapsing to well below the carrying capacity, where the boom–bust cycle begins anew. In this way, the population never reaches an equilibrium, despite the fact that there is strong density dependence. This example demonstrates overcompensation, and it occurs when the angle of incidence of the recruitment curve exceeds 90° as it approaches the equilibrium line (May 1976; May and Oster 1976).

A diagnostic feature of deterministic chaos is that slight changes in starting conditions lead to quite different population dynamics over time. In Fig. 5.21, the simulated dynamics of the two hypothetical populations, begun at slightly different densities,

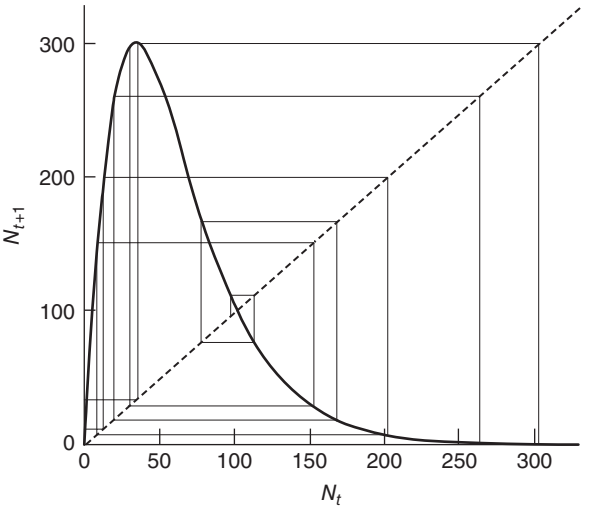

Fig. 5.21 Plot of predicted recruitment (N_t+1) relative to N_t (the heavy curve), equilibrium line at which N_t+1 = N_t (thin broken line), and trajectory of population dynamics over time for a simulated population following the Ricker logistic model, with rmax = 3.3 and K = 100 (thin solid line).

become quite different later on, illustrating their sensitivity to initial conditions. The two populations go through similar changes in the first few years but rapidly diverge thereafter, displaying different patterns of fluctuation.

We have thus far limited our discussion to the simplest pattern of density dependence: linear changes in per capita rates of reproduction or survival. We saw earlier (Figs 5.8 and 5.10) that there is no reason to expect natural regulation to be linearly density-dependent. Some wildlife biologists have even argued that this may be the exception rather than the rule (Fowler 1981); adult mortality in Serengeti wildebeest is a good example (Mduma *et al*. 1999).

5.10 **Intraspecific competition**

Regulation can occur through a number of mechanisms, such as predation or parasitism, but the most common cause is competition between individuals for resources. Such resources might be food, shelter from weather or from predators, nesting sites, or space to set up territories. We have seen some examples already in Figs 5.13 and 5.14.

5.10.1 *Definition*

Intraspecific competition occurs when individuals of the same species utilize common resources that are in short supply; or, if the resources are not in short supply, when the organisms seeking the resource nevertheless harm one another in the process (Birch 1957).

5.10.2 *Types of competition*

When individuals use a resource so that less of it is available to others, we call this type of competition *exploitation*. This includes both removal of a resource (consumptive use), as when food is consumed, and occupation of a resource (pre-emptive use), as when resources such as nesting sites are used. Individuals competing for food need not be present at the same time: one ungulate can reduce the food supply of another that arrives later.

Another type of competition involves the direct interaction of individuals through various types of behavior. This is called *interference* competition. One example of behavioral interference is the exclusion of some individuals from territories; another is the displacement of subordinate individuals by dominants in a behavioral hierarchy.

5.10.3 *Intraspecific competition for food*

Experimental alteration of food supply

Food addition experiments provide the best evidence for intraspecific competition. Krebs *et al*. (1986) supplied extra food to snowshoe hares in winter from 1977 to 1985. This raised the mean winter density fourfold at the peak of the 10-year population cycle. Similarly, Taitt and Krebs (1981) increased the density of vole populations (Fig. 5.22) by giving them extra food. The elk population at Jackson Hole, Wyoming, is kept at a higher level than would otherwise be the case by supplementary feeding in winter (Boyce 1989). These examples show that food is one of the factors limiting density.

The dense shrubland (chaparral) of northern California contains two shrubs, chamise (*Adenostema taxiculatum*) and oak (*Quercus wislizenii*), that are preferred food for black-tailed deer (*Odocoileus hemionus*). These shrubs resprout from root stocks after burning to provide the new shoots that the deer consume. Taber (1956) showed that on plots thinned by experimental burning, herbaceous food supply increased to 78 kg/ha from the 4.5 kg/ha found on control plots, while the shrub component increased from 165 to 460 kg/ha. Deer densities consequently increased from 9.5 km^{-2} on the experimental controls to 22.9 km^{-2} on the treatment plots, while fertility increased from 0.77 to 1.65 young per adult female.

Red grouse (*Lagopus lagopus*) live year round on heather (*Calluna vulgaris*) moors in Scotland. Their diet consists almost entirely of heather shoots. Watson and Moss (1971) describe experiments in which some areas were cleared of grouse, fertilized with nitrogen in early summer, and then left to be recolonized. Fertilizing increased the growth and nutrient content of the heather. The size of their territories did not differ between fertilized and control areas when the grouse set them up in autumn. However, territorial grouse that had been present all winter reared larger broods on the fertilized than on the control areas, indicating that reproduction was affected by overwinter nutrition. Territory sizes did decline in the following autumn, and densities increased, showing the 1-year lag of density response to nutrition. On other areas, old heather was burned every 3 years, creating a higher food supply of young regenerating heather. Territory size on these plots decreased (as density increased) in the same year as the treatment, so there was a more immediate response than on the fertilized plots.

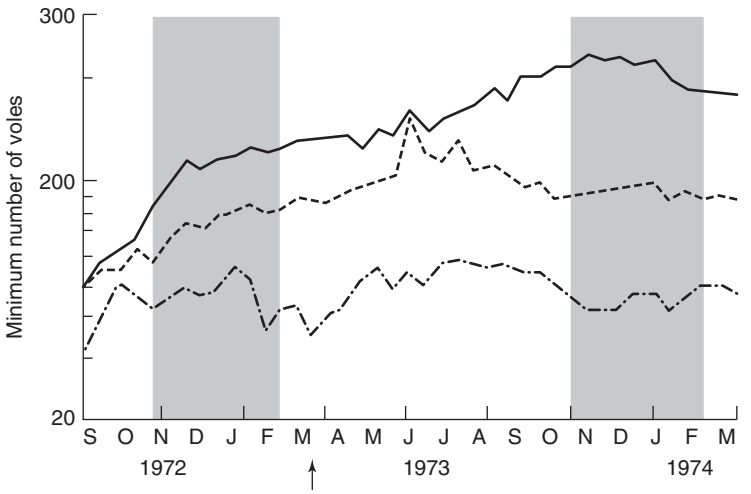

Fig. 5.22 The numbers of Townsend's voles on trapping grids increase in proportion to the amount of food that is provided, indicating that intraspecific competition regulates the population. Dashed-dotted line: control; dashed line: low food addition; solid line: high food addition; shaded area: winter. (After Taitt and Krebs 1981.)

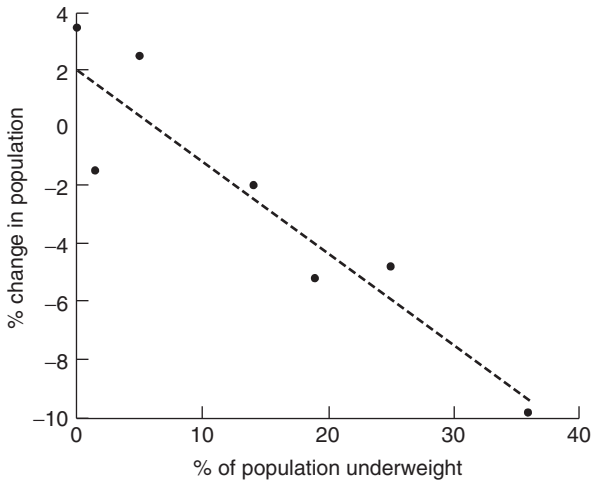

Fig. 5.23 The percentage change in a wood pigeon population in England is related to the proportion of the population that is underweight. (Data from Murton *et al*. 1966.)

Direct measures of food

Snowshoe hare populations in the boreal forests of Canada and Alaska reach high numbers every 10 years or so. Measurement of known food plants and feeding experiments suggest that the animals run short of food at peak numbers (Pease *et al*. 1979). Other measures, such as the amount of body fat (Keith *et al*. 1984) and fecal protein levels (Sinclair *et al*. 1988), also identify food shortage at this time (see Section 2.9).

African buffalo graze the tropical montane meadows of Mount Meru in northern Tanzania, keeping the grass short. Grass growth rates and grazing offtake were measured by use of temporary exclosure plots. Growth in the rainy season was more than sufficient for the animals, but in the dry season available food fell below maintenance requirements (Sinclair 1977).

Murton *et al*. (1966) measured the impact of wood pigeons (*Columba palumbus*) on their clover (*Trifolium repens*) food supply. Food supply was measured directly by counting clover leaves in plots. Pigeons consumed over 50% of the food supply during winter. They fed in flocks, those at the front obtaining more food than those in the middle or at the back. The proportion of underweight birds (under 450 g) was related directly to the overwinter change in numbers (Fig. 5.23) and inversely related to the mid-winter food. Thus, competition within flocks resulted in some animals starving, and the change in numbers was related to the proportion that starved.

Indirect measures of food shortage

Indirect evidence for competition for food comes from indices of body condition (see Section 2.9). The last stores of body fat that are used by ungulates during food shortages are in the marrow of long bones such as the femur. Bone marrow fat can be measured directly by extraction with solvents. However, since there is an almost linear relationship between fat content and dry weight (Hanks 1981) (see Section 2.9.3), it is easier to collect a sample of marrow from carcasses found in the field and oven dry it. A cruder but still effective method, introduced by Cheatum (1949), is to describe the color and consistency of the marrow.

Other fat stores, such as those around the heart, mesentery, and kidney, are used up before the bone marrow fat starts to decline (see Section 2.9). The relationship between kidney and marrow fat holds for many ungulate species (see Fig. 2.11). If both kidney

and marrow fat can be collected, a range of body conditions can be recorded. However, often the marrow fat is all that is found in carcasses, because scavengers have eaten the internal organs.

Klein and Olson (1960) used bone marrow condition indices to conclude that deer in Alaska died from winter food shortage, as did Dasmann (1956) for deer in California. Similarly, migratory wildebeest in the Serengeti that died in the dry season were almost always in poor condition, as judged by the bone marrow, and this was correlated with the protein level in their food (see Fig. 2.12). This dry-season mortality was density-dependent and was sufficiently strong to allow the population to level out (Sinclair *et al*. 1985; Mduma *et al*. 1999).

Problems with measurement of food supply

To determine whether competition for resources such as food is the cause of regulation, we need to know what type of food is eaten, how much is needed, and how much is available. What is needed must exceed what is available in order for competition to occur. The types of food eaten form the basis for many studies on diet selection, sometimes called *food habit studies*. These in themselves do not tell us what is needed in terms of digestible dry matter, protein, and energy. We should note that such requirements are unknown for most wild species and we have to use approximations from other, often domestic, species. The amount of food available to animals is particularly difficult to assess because we are unlikely to measure potential food in the same way as does the animal. Animals are likely to be far more selective than our crude sampling, and so we are likely to record more "food" than the animal sees. Our measures of food supply are often seriously flawed, which is one of the reasons why direct evidence for intraspecific competition for food is rare. There is far more indirect evidence for competition, provided by indicators such as body condition.

5.11 Interactions of food, predators, and disease

The effect of limited food on population demography can go beyond the direct effects of undernutrition: there can also be synergistic interactions with predation and disease. Animals may alter their behavior when food becomes difficult to find in safe areas, searching increasingly in areas where they are at risk of predation in order to avoid eventual starvation (Lima and Dill 1990; McNamara and Houston 1987). This is called *predator-sensitive foraging* and has been observed in snowshoe hare feeding (Hik 1995; Hodges and Sinclair 2003). Such behavior can result in increased predation well before starvation takes effect, as seen in wildebeest (Sinclair and Arcese 1995).

Disease can also interact synergistically with food, pathological effects suddenly becoming apparent at a certain, sometimes early, stage of undernutrition (see Chapter 11). Sometimes food, disease, and predators all interact. Wood bison numbers in the Wood Buffalo National Park, Canada, switch suddenly from a high-density food-regulated state to a low-density predator-regulated one when diseases such as tuberculosis and brucellosis affect the population (Joly and Messier 2004).

5.12 Summary

Regulation is a biotic process that counteracts abiotic disturbances affecting an animal population. Two common biotic feedback processes are predation and intraspecific competition for food. These are called density-dependent factors if they act as negative feedbacks. Negative feedback imparts stability to the population. Disturbances are provided by fluctuating weather or other environmental conditions or by chance effects on

reproduction and survival. These are called density-independent factors and will cause populations to drift to extinction if there are no counteracting density-dependent processes operating. For wildlife management, it is necessary to know (i) the causes of the density-dependent processes that stabilize the population and of fluctuations and instability, and (ii) which age and sex groups are most influenced by these stabilizing or destabilizing processes.

One way to understand such effects is to model density-dependent changes in population growth rate using logistic models. Application of such models shows that whereas density dependence is often stabilizing, overcompensatory density dependence can itself encourage population fluctuation, beyond the degree we would expect from demographic or environmental stochasticity. A common cause of regulation is intraspecific competition for food.

Competition occurs if the needs of the population exceed availability. To measure such competition, we need to know how much food is available and how much is needed, and whether it is density-dependent. Food can also interact with predation and disease to regulate populations.