3

Population regulation

- What is population regulation?
- Combining density-dependent and density-independent factors
- Tests of density dependence

3.1 Introduction

Are populations regulated? If so, how? What does population regulation really mean? These questions have been debated for many years and were at the core of a hotly contested difference of opinion between ecologists who emphasized the importance of density-dependent factors versus those who emphasized density-independent factors in population regulation (Turchin 1995). The logistic equation is, of course, based on density dependence. But how do we differentiate density-independent from density-dependent causation in populations with time lags, or in those dominated by stochastic processes?

The ecologists who emphasized the primary role of density-independent factors in population regulation were often those who worked with small animals, especially insects and rodents, and/or in habitats characterized by drought or short growing seasons. Among them were early influential ecologists such as Andrewartha and Birch, who worked on Australian grasshoppers, the distribution of which was determined by the length and intensity of the wet season. The northern boundary was determined by conditions that were too dry for their food plants, and the southern boundary was said to have too much moisture for the grasshoppers.

In the first major ecology textbook, Andrewartha and Birch (1954) concluded that abundance of a population was limited by the same conditions that limited its distribution. A major example used by Davidson and Andrewartha (1948) was the distribution and abundance of thrips (*Thrips imaginis*), tiny insects that feed on pollen and soft tissues of flowers in southern Australia. Populations were said to increase unchecked in the spring, with a sharp decline during the summer drought when flowers were scarce. Andrewartha and Birch (1954) asserted that thrip populations were checked by rainfall, not by their food supply, a potentially density-dependent factor. Advocates of density dependence, such as

Nicholson (1957), Solomon (1957), and Lack (1954, 1966), often cited the importance of competition in population regulation, especially in vertebrate populations.

Turchin (1995, 1999, 2003) and others (Ricklefs 1990) have asserted that this debate is largely artificial and that density dependence has been repeatedly and convincingly demonstrated (see examples from Chapter 2). At the same time, we know that the physical environment and other stochastic factors often reduce population size before density-dependent factors become operational.

When population regulation takes place, what is the dominant mechanism? Competition? Predation? Parasites? We will explore this question in the second half of the book. Note, however, that the answer may differ by trophic level. For example, Hairston *et al.* (1960) proposed that while herbivore populations were mainly limited by predation, producers (green plants), decomposers, and predators were usually limited by competition. This is such a broad generalization that is difficult to imagine how to test it. Nevertheless, this theory resurfaces regularly in modified forms.

3.2 What is population regulation?

One of the problems with this debate is a lack of agreement as to what a "regulated population" is. Given what we learned in the previous two chapters about the behavior of populations with time lags, with high reproductive potentials, or under the influence of demographic and environmental stochasticity, it is not realistic to expect a population to show a simple attraction to a specific number called the carrying capacity. In Chapter 2 we defined a **stable point** as a stable number at the carrying capacity with no oscillations. But we also recognized that the population could be temporarily oscillatory with oscillations dampening and moving toward a stable number or point. We also discussed populations that show regular oscillations around the carrying capacity (**stable cycles**), oscillations between two, four, and eight points, and chaotic behavior. All of these population behaviors are based on variations of the density-dependent logistic model.

Population regulation does not depend on a specific stable equilibrium point, but rather on a "long-term stationary probability distribution of population densities" (Turchin 1995) or a "stochastic equilibrium probability distribution" (May 1973). The key concept is that there is some mean population level around which a regulated population fluctuates. In addition, over time the population does not wander increasingly far away from this level. The variance of the population density is bounded (Royama 1977). All of these definitions relieve us of the expectation of a fixed carrying capacity or of a fixed stable point. The population is not expected to seek a stable point from which it does not wander. Instead, we allow for stochastic variation around the carrying capacity, **which can itself vary over time**. Hence, an ecological equilibrium is not a fixed or stable point, but a cloud of points. However, since it is often difficult to distinguish density dependence from stochastic noise (see below), many populations have been described as "density vague" (Strong 1986).

Fluctuations in the Dow Jones Industrial Average have been used as an example of an unregulated system, with rainfall patterns on Barro Colorado Island in Panama as an example of a regulated system (Turchin 1995). Some economists and meteorologists might argue with these examples, but the general idea is that the Dow Jones average, in spite of short-term ups and down, shows a trend upward over long time spans. A better example might be the carbon dioxide content of the atmosphere from 1850 to the present.

Although there are annual fluctuations, CO_2 concentration has steadily increased from around 280 parts per million (ppm) to over 360 ppm. By contrast, ice cores have shown that carbon dioxide concentration in the atmosphere from the year 1000 to 1750 was a good example of a regulated system. With regard to rainfall, records from 1900 to 2000 showed that rainfall fluctuated from year to year in Panama, but showed no overall pattern of increase or decrease.

Population regulation does not occur in the absence of density dependence. Thus a population showing pure exponential growth or decline would be unregulated. If population density had no effect on the per capita growth rate, there could be no range of population densities to which the population would return. In this context, Turchin (1995) uses the Murdoch and Walde (1989) definition of density dependence as "a dependence of per capita population growth on present and/or past population densities." While density dependence is a property of the overall population dynamics, which may involve time lags, no specific mechanism is necessarily responsible (Turchin 2003). That is, one aspect of the life history, such as birth rates, may not be density-dependent.

Although density dependence is necessary for population regulation, it is not a sufficient condition. For regulation to occur the following must also be true: (i) density dependence must be of the right sign (there must be a tendency to return to a carrying capacity); (ii) the return tendency must be strong enough to counteract potential disruptive effects of density-independent or stochastic factors; and (iii) the lag time over which the return tendency operates must not be too long (Turchin 1995).

Given more precise definitions of population regulation, and density dependence, Turchin (1995) found that the other remaining pieces needed are: (i) an acceptable statistical test of density dependence, and (ii) time-series data long enough to detect density dependence. From a review of the literature, Turchin (1995) concluded that whenever adequate data have been gathered for an appropriate period of time and tested with an appropriate method, density dependence has been found. This does not mean that all populations are regulated at all times. Lack of regulation doubtless occurs temporarily, but the probability of detectable density dependence will increase with the length of time that data are collected on a population.

3.3 Combining density-dependent and density-independent factors

In Section 2.7 we added stochasticity to density-dependent models. To simulate density-independent effects we simply added environmental stochasticity by allowing the carrying capacity to randomly vary. We can more explicitly add a stochastic density-independent factor by modifying the Ricker equation (2.12). Let us assume that a certain level of density-independent mortality happens every year, but it varies randomly. We can modify the Ricker equation as shown below (Eqn. 3.1) by simply subtracting the number of deaths from density-independent factors:

$$N_{t+1} = N_t \, e^{r\left(\frac{K-N_t-D}{K}\right)} \tag{3.1}$$

We can also select different levels of density-independent death rates. As shown in Fig. 3.1, the population growth is increasingly affected as we move from zero to increasingly higher amounts of density-independent growth.

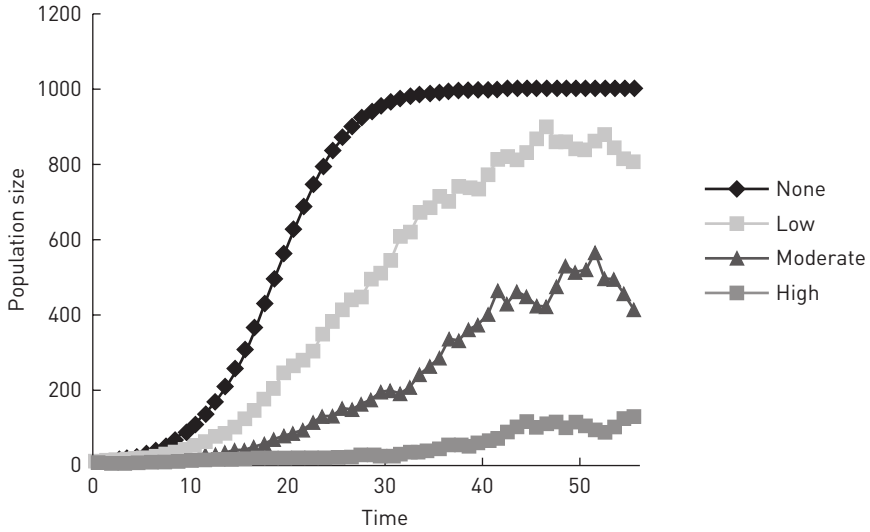

Figure 3.1 Effect of different levels of stochastic density-independent mortality on population growth, based on the Ricker equation (Eqn. 2.12). In all cases $r = 0.25$, $N_0 = 10$, and $K = 1000$. Population growth is increasingly affected as the strength of stochastic density independent mortality increases.

Instead of assuming that density-independent effects are uniformly negative, let us now assume that some years are good years (bonanza years) and others are bad years. That is, environmental stochasticity does not always have a negative effect on our population. We will simply introduce a certain amount of "environmental noise." That is, density-independent effects that can be either beneficial or detrimental. To simulate this we can still use Equation 3.1, but we now introduce stochastic variations that can be positive or negative, but with an average value of $D = 0$. In Fig. 3.2 compare the four stochastic simulations with the deterministic growth curve with no density-independent mortality. Obviously, if we increase the variations around D, the "cloud" of points around K gets larger. The density-independent factors would get increasingly important and obscure the density dependence of the population.

3.4 Tests of density dependence

How can we detect density dependence in the field? For a density-independent population, Tanner (1966) proposed that we can simply use the equation for discrete growth, $N_{t+1} = \lambda N_t$. After taking natural logs of both sides of the equation we can write:

$$\ln N_{t+1} = \ln \lambda + \ln N_t = (1.0) \ln N_t + \ln \lambda \tag{3.2}$$

When we plot $\ln N_{t+1}$ versus $\ln N_t$, if λ is a constant, we should have a straight line with the slope of 1.0 and a y-intercept equal to $\ln \lambda = r$. But if there is density dependence and the growth rate slows with population size, when $\ln N_{t+1}$ is graphed against $\ln N_t$, a linear regression through the data should yield a slope less than 1.0.

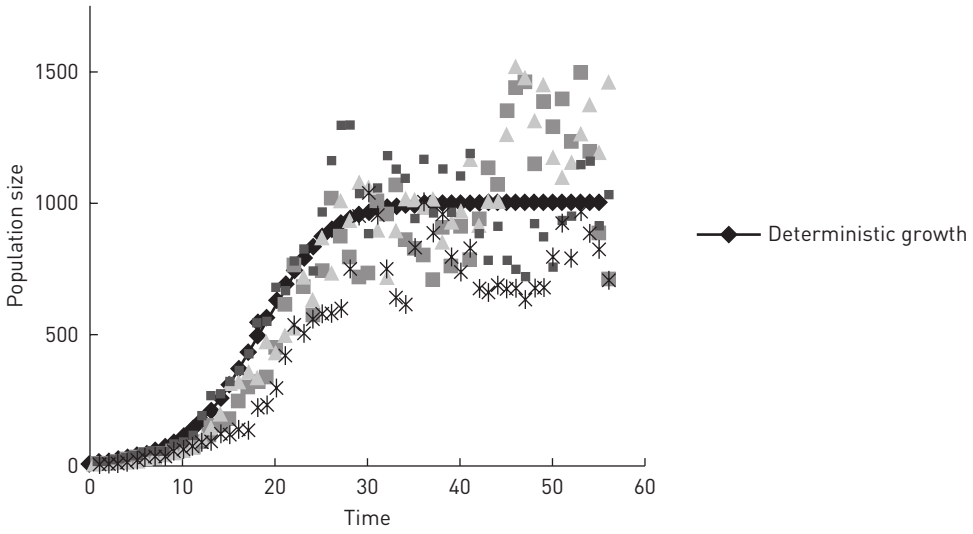

Figure 3.2 Effect of stochastic "environmental noise" on population growth. The deterministic growth curve has no density-independent effects. In all cases $r = 0.25$, $N_0 = 10$, and $K = 1000$.

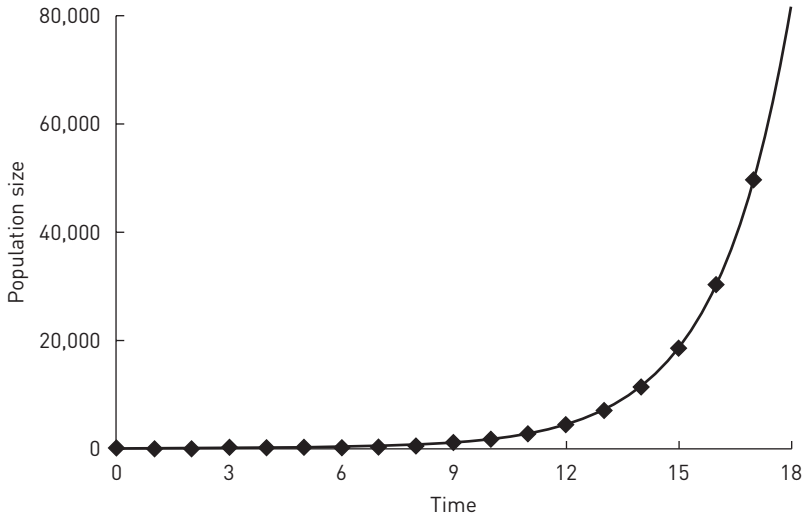

Figure 3.3 Exponential growth. Initial population size = 10 and $r = 0.50$.

For example, consider Figs. 3.3 and 3.4. In Fig. 3.3 we have an exponentially growing population with $\ln \lambda = r = 0.50$ and an initial population size of 10. In Fig. 3.4 we have graphed $\ln N_{t+1}$ versus $\ln N_t$. Since this population is not showing density dependence we get what we expect, a positive slope equal to 1.0. Now contrast this with a hypothetical yeast population showing density-dependent growth (Fig. 3.5, based on Pearl 1927). If we take natural logs and graph the data as we did in Fig. 3.4, we find that the slope of the

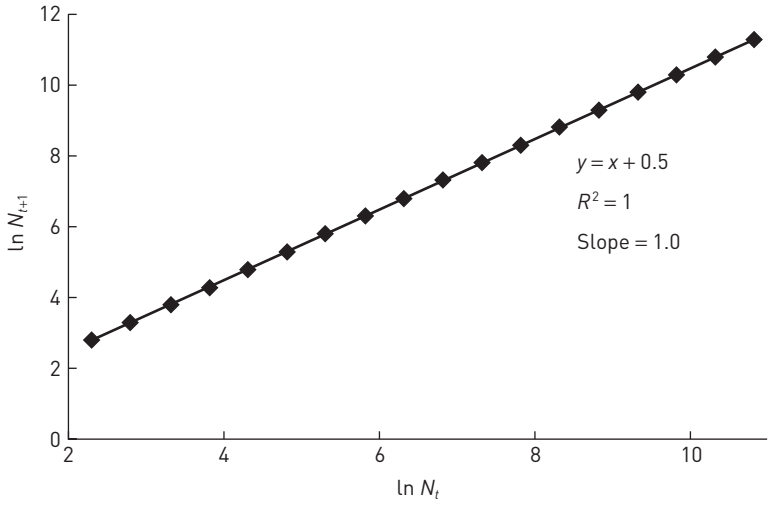

Figure 3.4 Density-dependence test for data from Fig. 3.3.

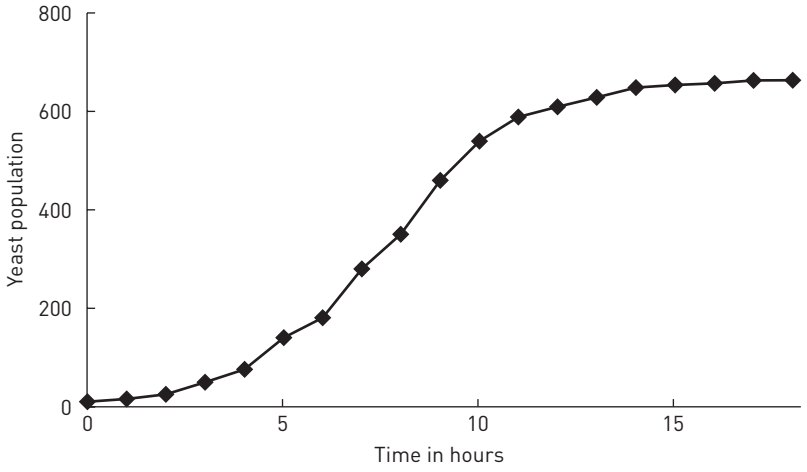

Figure 3.5 Hypothetical yeast population growing in the laboratory. Adapted from Pearl (1927).

line is indeed less than one (Fig. 3.6). The question remains, however, how far less than one should a regression slope be before we consider it significantly different?

Tanner (1966) examined 70 data sets for animal populations and found slopes significantly different from one in 63 of them. However, this method for detecting density dependence is fundamentally flawed. First, a linear regression assumes data points are independent. In this analysis the *x*-value in one time series becomes the *y*-value in the next time series. Second, measurement error in the population estimates inevitably leads to a slope of less than one. Therefore a slope of less than one is often just an artifact of measurement error and not evidence for density dependence. Finally, the expected relationship between N_{t+1} and N_t is not necessarily linear if there is environmental variability

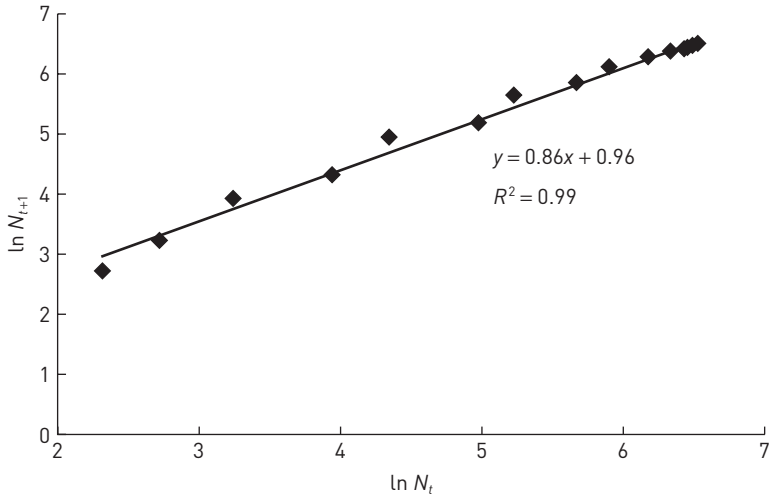

Figure 3.6 Density-dependence test for a yeast population. Based on data from Fig. 3.5.

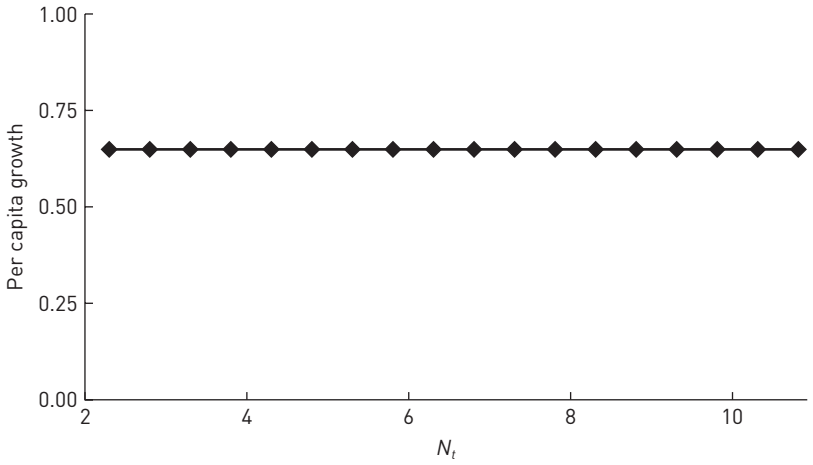

Figure 3.7 Per capita growth test for density dependence in a population with exponential growth and no carrying capacity. Based on data from Fig. 3.3.

or if the population has such a high growth rate that it approximates chaos (as described in Chapter 2).

A better test for density dependence is to examine the per capita growth rate versus population size (Turchin 1995, Case 2000). In the logistic equation we expect the per capita growth rate to have a negative slope when graphed against population number (Fig. 2.2 from Chapter 2). By contrast, in exponential growth, the per capita rate remains steady. For example, examine Figs 3.7 and 3.8. In Fig. 3.7 (based on exponential-growth data from Fig. 3.3), the slope equals zero, indicating no change in the per capita growth rate with population density. By contrast, for our yeast population graphed in Fig. 3.5 we find

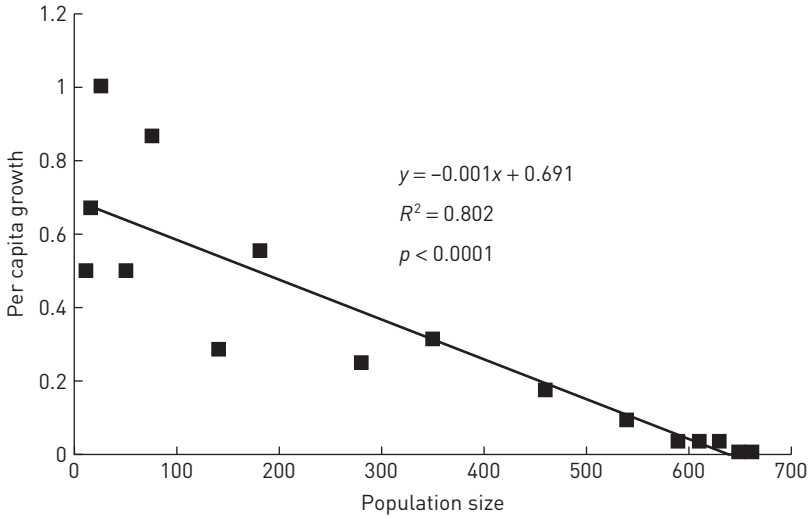

Figure 3.8 Per capita growth test for density dependence in a yeast population. Based on data from Fig. 3.5.

a significant negative slope for the same analysis (Fig. 3.8). Examining plots of per capita growth versus N have many advantages, including the detection of density dependence in populations with environmental noise (Case 2000).

Let us now look at some field data. The following is based on long-term Christmas Bird Counts of waterfowl populations in the Chesapeake Bay area of Maryland and on the Piedmont of Virginia. These data were obtained by Heath (2002) from the United States Fish and Wildlife Service and from the Virginia Society for Ornithology. Christmas Bird Counts (CBC) were initiated in the late 1800s as a replacement for the traditional Christmas hunt, and may be the oldest wildlife census in the world. The CBC, however, depends on volunteers, many of whom are not professional biologists, and the use of CBC data in scientific studies is problematic. Nevertheless, the CBC often represents the only long-term data on waterfowl in regions such as the Virginia Piedmont. In addition, Maryland and Virginia waterfowl populations have been affected by habitat loss, hunting pressure, and environmental degradation in the Chesapeake Bay. On the other hand, due to land-use changes including the creation of new reservoirs and wetlands, waterfowl populations may be increasing on the Virginia Piedmont (Heath 2002).

Indeed, if we examine CBC estimates of Canada geese (*Branta canadensis*) populations from 1958 to 2001, there is a distinct increase (estimated $r = 0.17$) in the Piedmont population, while the Coastal Plain population has no distinct trend other than that of increasing oscillations (Fig. 3.9). In Fig. 3.10 we have tried to test for density dependence in the Piedmont population by the first method, graphing $\ln N_{t+1}$ versus $\ln N_t$. The resultant regression is so close to one that the conclusion is inescapable that the Piedmont goose population is **not** density-dependent at this time. By contrast, in Fig. 3.11 we see that the regression slope departs radically from one in the Coastal Plain population. In fact the regression is so weak that the slope is not significantly different from zero. How do we interpret this? On the one hand we might conclude that the Coastal Plain population is very density-dependent. Or we might conclude that this is just an unreliable set of data.

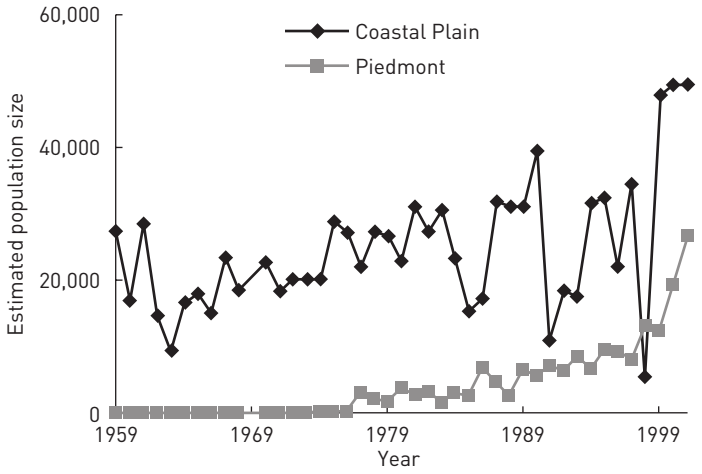

Figure 3.9 Canada goose (*Branta canadensis*) population on the Coastal Plain of the Chesapeake Bay and on the Virginia Piedmont. Based on Christmas bird counts.

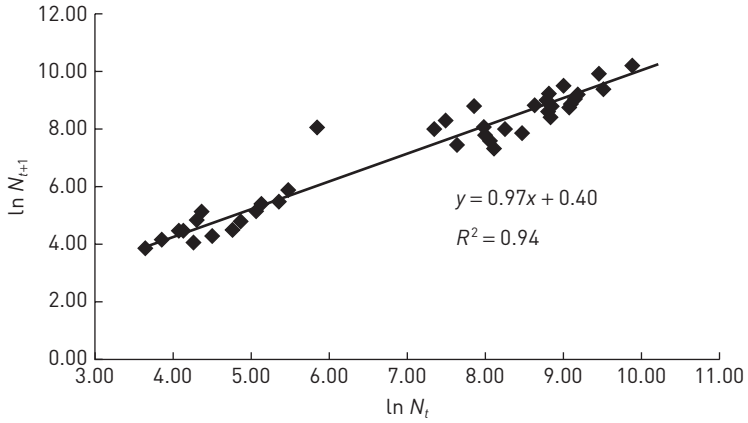

Figure 3.10 Test of density dependence of the Piedmont Canada goose population. Based on Christmas bird counts.

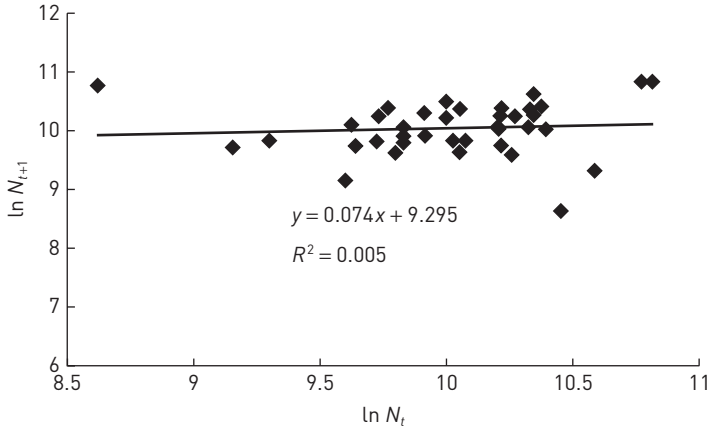

Figure 3.11 Test for density dependence of the Coastal Plain Canada goose population. Based on Christmas bird counts.

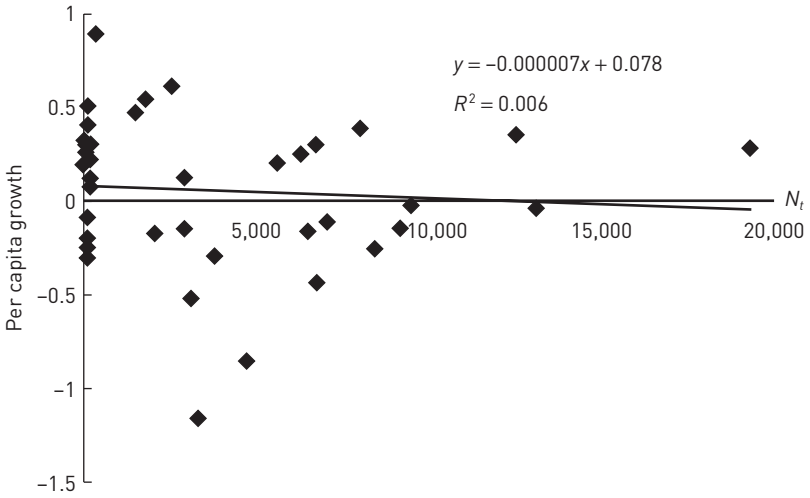

Figure 3.12 Per capita growth versus population size in the Piedmont Canada goose population.

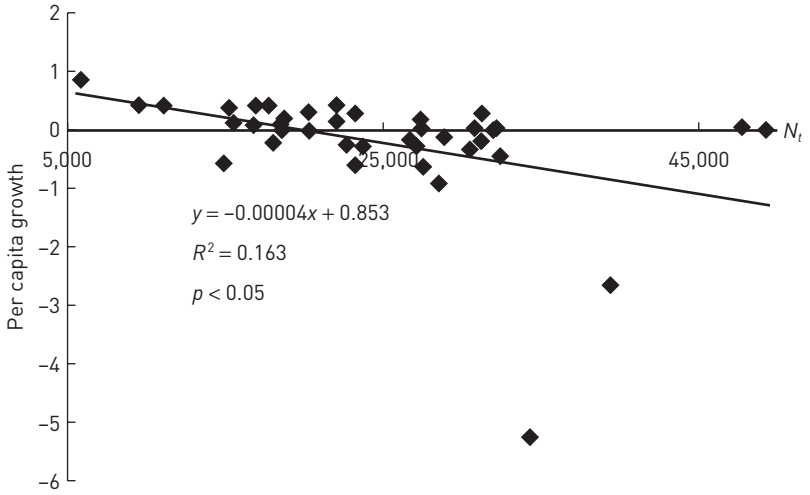

Figure 3.13 Per capita growth versus population size in the Coastal Plain Canada goose population.

Now, let's try the second method for determining density dependence, using per capita growth rates. From Fig. 3.12 we learn that the regression line for the Piedmont population is not significantly different from zero. This means that the Piedmont goose population has shown no decrease in per capita growth through 2001. Therefore, both tests tell us that the Piedmont population is not density-dependent at this time.

On the Coastal Plain (Fig. 3.13), although the slope is very small, it is negative and the regression is significant. Therefore, although the data are rather weak, it does appear that this population shows density dependence.

3.5 Conclusions

Populations are affected by their own life histories and vital rates, and by feedback from the environment. Such feedback components, which may involve time lags, are termed **endogenous** by Turchin (2003). As we have reviewed before, demographic stochasticity can also seriously affect population behavior in spite of density-dependent feedback. By contrast, **exogenous** factors refer to environmentally related density-independent factors that affect population density, but are not, in turn, affected by it (Turchin 2003).

Our main conclusions are:

1. The per capita rate of change, r, is affected by both endogenous and exogenous factors, and both should be examined when attempting to understand population behavior (Turchin 2003).
2. As stated above, negative feedback between the realized growth rate of the population and its density is a necessary but not sufficient condition for population regulation.
3. Population dynamics are nonlinear, and exogenous (density-independent) factors may dominate population behavior.
4. A more reliable method for detecting density dependence is to plot per capita growth against N, as opposed to $\ln N_{t+1}$ versus $\ln N_t$.

Assuming we can reliably demonstrate population regulation, is it due primarily to competition or to predation/parasitism? Is regulation in many populations simply a reflection of habitat loss? How often is regulation due to local processes as opposed to metapopulation dynamics (see Chapter 5) (Murdoch 1994)? Are populations under the influence of stochastic environmental factors to the extent that density-dependent population regulation is largely irrelevant?

Leirs *et al*. (1997) have shown that the population of the African rodent *Mastomys natalensis* is regulated by an interaction between stochastic and deterministic seasonality and nonlinear density-dependent factors. Using a variety of statistical techniques, they analyzed population data based on a multiple mark–recapture method. The best fit to the data came from a model encompassing both density-independent factors (previous three months' rainfall data), and density-dependent factors (a nonlinear demographic model). This example affirms that population regulation is often a combination of stochastic density-independent as well as density-dependent factors operating simultaneously.