

Part I

**Traditional theoretical
ecology**

Chapter 2

Malthusian increase

THE CORNERSTONE of theoretical evolutionary ecology was laid down by Malthus in 1798: In his *Essay on the Principle of Population* Malthus proposed that an unchecked population increases exponentially, or geometrically, in numbers. This type of population growth is now known as Malthusian increase, or the Malthusian law, and it is essential in the way it recognises that self-replication is a fundamental character of all organic beings.

The Malthusian law has played an important role on two major events during the historical development of biological thoughts. The first event was the role that it played in getting Darwin to realise that evolution is driven by natural selection. It was not until Darwin read the essay of Malthus that he realised that natural selection is the consequence of the struggle for existence that “inevitable follows from the high rate at which all organic beings tend to increase” (Darwin, 1859). The second major event was in 1930 when Fisher formulated the fundamental theorem of natural selection by defining natural selection as variation in geometrical, or exponential, increase.

Today, it is generally agreed that Darwin’s idea was the essential observation that initialised the development of evolutionary biology, and that Fisher’s fundamental theorem is the essential hypothesis underlying most of the predictions in the classical theory of evolution by natural selection. This dominating role that the fundamental theorem plays in the classical theory is somewhat of a paradox for Darwin’s hypothesis, and this is because there is almost no resemblance between Darwin’s and Fisher’s definition of natural selection. Where Darwin defined natural selection from the competitive interactions that arise from Malthusian increase, Fisher disregarded these interactions, and defined natural selection from Malthusian growth in itself.

This book deals mainly with the intriguing question that arises from Darwin’s and Fisher’s definition of natural selection: Is it Darwin or Fisher

that is right? Is natural selection defined by competitive interactions, or by Malthusian increase? If none of these relatively simple definitions hold, then what is the essential and mechanistic coupling that we need to make between these two processes before we can construct a theory of natural selection that holds on an evolutionary scale. The theoretical analysis in this book clearly suggests that Darwin was right in the sense that selection by density dependent competitive interactions is essential for our understanding of evolution by natural selection. In retrospect it is also relatively easy to understand why Fisher apparently failed to recognise Darwin's insight: As all other ecologists Darwin saw with his own eyes "the struggle for existence which everywhere goes on" (Darwin, 1958), while as a Mendelian geneticist Fisher focused mainly on the scale of genomic processes where the action of competitive interactions is less obvious.

Before I turn to evolutionary biology I will, in this part of the book, focus only on the ecological implications of Malthusian increase. In this chapter I will even restrict myself to a formulation of the Malthusian laws of exponential and geometrical increase. To do this let us first consider the case where individuals are potentially immortal and instantaneous reproduction (m) and mortality (d) are constants. In this case the instantaneous growth rate, which Fisher (1930) named the Malthusian parameter, is constant and given as $r = m - d$. Hence, the rate of increase in the population abundance (N) is

$$dN/dt = rN \quad (2.1)$$

When the initial abundance (N_0) at time $t = 0$ is given, eqn 2.1 can be solved as

$$\begin{aligned} dN/dt &= rN & (2.2) \\ d \ln N/dt &= r \\ \ln N_t - \ln N_0 &= \int_0^t r dt \\ \ln N_t - \ln N_0 &= rt \\ N_t &= N_0 e^{rt} \end{aligned}$$

This solution is the Malthusian law of exponential increase, and it is illustrated in Fig. 2.1a.

The other simple situation is when the organism has non-overlapping generations, i.e., when the individuals reproduce only once before they die. In this case it is convenient to reformulate the continuous form of the Malthusian law given in eqn 2.2, to the discrete form, and this can be done as

$$N_t = N_0 e^{rt} \quad (2.3)$$

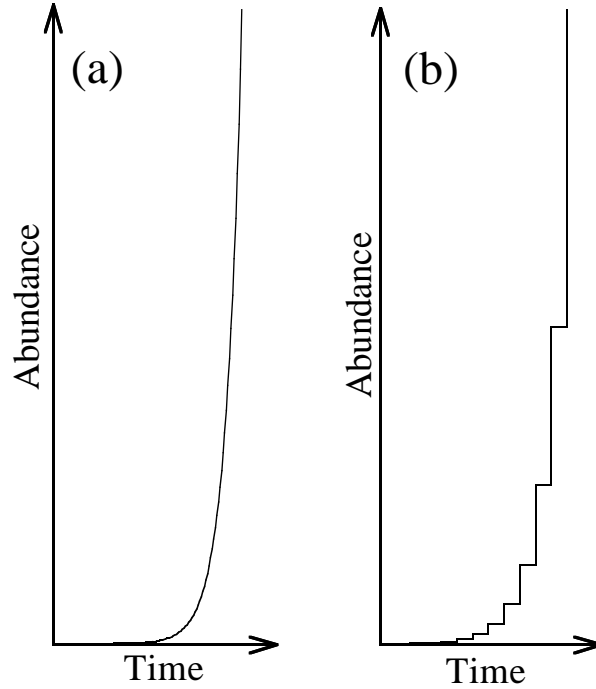


Fig. 2.1 Abundance against time, illustrating exponential (a) and geometrical (b) increase.

$$N_t = N_0 e^{t \ln \lambda}$$

$$N_t = N_0 \lambda^t$$

where t is time in generations and $\lambda = e^r$ is the discrete Malthusian parameter, also known as the discrete growth rate. The discrete version of the Malthusian law is the law of geometrical increase, and it is illustrated in Fig. 2.1b. Note, the non-differentiable steps that distinguish this per-generation process from the continuous process of exponential increase.

When the generations are non-overlapping the discrete growth rate is given as net lifetime reproduction (R_0), which in this special case is lifetime reproduction (R) times the probability (p) that an offspring will survive and reproduce, i.e., we have that $\lambda = R_0 = pR$. This simple relation does not hold when the individuals reproduce more than once and they are structured into age classes. This more complex case is dealt with in Chapter 7, but until then I will consider only relatively simple and unstructured models.

Chapter 3

Density regulation

THE CURVE OF EXPONENTIAL increase is unrealistic because it is based on the assumption of an ecological vacuum. In reality ecological forces are present at all times and these forces will bend the curve of exponential increase. This was recognised by Malthus when in his essay he proposed that animal populations increase up to the limit of their resource where lack of food prevents further increase. This idea is now known as the Malthusian principle.

The Malthusian principle was elaborated in further detail by Nicholson (1933) and Smith (1935). The major point of their studies is that the process that bends the curve of exponential increase is driven by regulating factors that are density dependent (Sinclair, 1989). That is to say that the factors that control the population density are themselves controlled by the population density. The chief of these factors are intra-specific competition, although predators and parasites may also be density dependent. The overall process is named density, or population, regulation and it is this process that determines that the population has a positive equilibrium called the population equilibrium.

In its further development the idea of population regulation became polarised into two opposing schools. On the one hand Lack (1954, 1966) proposed that populations are regulated by factors, or resources, outside the population while, on the other hand, Chitty (1960) and Wynne-Edwards (1962, 1986) proposed that populations are self-regulated by behavioural interactions. Wynne-Edwards idea was closely connected with group selection where animals are hypothesised to restrain from reproduction in order to keep the population from over-exploiting the resource. As this latter idea of natural selection is doubtful (Maynard Smith, 1964; Williams, 1966; Wiens, 1966; Bell, 1987) the idea of self-regulation vanished while it was Lack's idea that became the established hypothesis.

As illustrated below, Lack's idea of resource regulation is too simple because it is based on the invalid assumption that a single population on a single resource is regulated exclusively by the exploitation of that resource. That is to say, it is assumed that the removal of resource from the common resource pool by the consumers is the only regulating factor that, through a shortage of resource, affects the rate of increase in the consumer population. This hypothesis is too simple because "self-regulation" also occurs in natural populations, and this is because the number of competitive interactions per individual is density dependent and because each interaction requires both time and energy that otherwise could be used for undisturbed transformation of resource into numerical replication. Since the term self-regulation has been connected with a variety of more or less doubtful processes I will use the term interference regulation for the regulation that is caused by competitive interactions.

Apart from population regulation there is another process that is essential for the population equilibrium. As it is population regulation that determines that a population has a positive equilibrium, it is the process of population limitation that sets the abundance of that equilibrium. Any factor that when changed changes the equilibrium point is a limiting factor. Regulating factors are generally limiting factors, but limiting factors are not necessarily regulating factors. An example of a limiting factor that is not a regulating factor is a density independent factor that affects the maximal rate of reproduction.

In this chapter I will first describe both resource and interference regulation before I turn to population growth under density regulation, a growth form that is known as logistic growth and which was first studied by Verhulst in 1838. In the original form logistic growth was described by a continuous model predicting a monotonic return to the population equilibrium. In 1974 May found that the time lag in discrete models with non-overlapping generations could result in over-compensation and very complex dynamics, including chaos. Hassell et al. (1976) were fast to show that this form of over-compensatory density dependence generally does not cause complex dynamics in natural populations. In the later studies by, e.g., Turchin (1990), Wittman et al. (1990), and Turchin and Taylor (1992), it was found that models incorporating density regulation that typically is delayed one generation actually can account for much of the complex dynamics in natural populations. These latter studies are based on statistical analysis and they are generally uninteresting from a mechanistic perspective because they do not indicate why the regulatory effects are delayed in one generation when all evidence suggest that "true density regulation" operates without this delay.

3.1 Resource regulation

Let me now formulate Lack's hypothesis that says that populations are regulated by their exploitation of their resource. The exploitation of the resource is a regulating factor because the degree of exploitation is density dependent and because the degree of exploitation influences the resource density that in turn affects the rate of increase in the consumer population. This type of regulation can be formulated as

$$\lambda = \lambda_m f(N) \quad (3.1)$$

where λ_m is the maximal and discrete growth rate and $f(N)$ is a function that declines monotonically from one to zero as the population density increases from zero to infinity. Instead of representing density regulation by the general model in eqn 3.1, let me disregard the disturbances that arise from non-linearities and focus on the process that is linear on the scale of the population dynamic processes. As these operate multiplicatively this model is linear on a logarithmic scale so that resource regulation is given as

$$\lambda = \lambda_m N^{-\gamma_\alpha} \quad (3.2)$$

where γ_α is the strength of the resource regulation ($\gamma_\alpha > 0$) and the notation is simplified by scaling the consumer density so that eqn 3.2 is defined only for $N \geq 1$ and the resource is unexploited at the lower limit $N = 1$.

In between $N = 1$ and $N \rightarrow \infty$ we have the population equilibrium where an average individual replaces itself by exactly one individual, i.e., where $\lambda = 1$. If I set eqn 3.2 = 1, I can rearrange the equation and obtain the population equilibrium as

$$N^* = \sqrt[\gamma_\alpha]{\lambda_m} \quad (3.3)$$

where the superscript star denotes that equilibrium. From the definition in eqn 3.2 we know that the γ_α parameter is the only regulating factor, and from eqn 3.3 we can see that both the γ_α and the λ_m parameters are limiting factors because together they determine the population equilibrium. Hence, γ_α is both regulating and limiting, whereas λ_m is limiting only. If we think of N^* as the total abundance, a factor that will influence γ_α , but not λ_m , is the geographical distribution of the habitat, where a small habitat implies a large γ_α , but not a small λ_m , at least not if the habitat is considerably larger than the home-range of the animal in question. A factor that will influence λ_m , but not γ_α , is density independent mortality, and a factor that is likely to influence both λ_m and γ_α is the rate of production in the resource, where a low rate of production implies a low λ_m and a high γ_α .

If I take the natural logarithm to λ_m I obtain $r_m = \ln \lambda_m$ that is the maximal Malthusian parameter as it is defined on a per generation time scale.

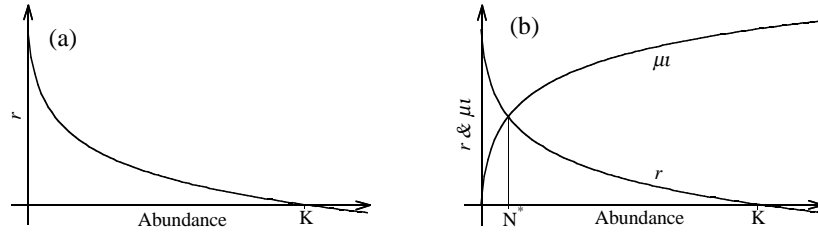


Fig. 3.1 (a) Population regulation according to Lack's hypothesis of resource regulation. The carrying capacity (K) is at the intercept $r = 0$, where r is the intrinsic Malthusian parameter. (b) Population regulation under the hypothesis of interference and resource regulation. The population equilibrium (N^*) is at the intercept between r and the regulation by interference (μ).

If I take the natural logarithm to eqn 3.2 I obtain the intrinsic Malthusian parameter

$$r = r_m - \gamma_\alpha \ln N \quad (3.4)$$

which is the intrinsic potential that an organism has to increase in numbers on a given resource density at given abiotic conditions and given that interference regulation is absent. The relationship in eqn 3.4 between r and N is illustrated in Fig. 3.1a. We see that the rate of increase (r) is density dependent because it declines monotonically as the abundance increases. At the intercept $r = 0$ we have the population equilibrium that, in this case with resource regulation, is the number of individuals that the resource can sustain. This density is often referred to as the carrying capacity.

3.2 Interference regulation

In the section above we assumed that the exploitation of the resource was the only regulating factor. Consider now the regulation that arises because the individuals in the population encounter one another. In this context, let ι be the level of interference in the population, a quantity that we may think of as the number of times, per unit time, that an individual encounters other competing individuals. We then expect that ι will increase monotonically with the population density, and that this relation is convex because there is an upper boundary to the number of encounters that an individual can experience per unit time. I have chosen to approximate this relation as

$$\iota = \gamma_\iota \ln N \quad (3.5)$$

where γ_ι is the strength of the density dependence and the density is scaled so that the regulatory effects vanish at the lower limit $N = 1$. Now, let μ

be the cost per encounter, i.e., the cost that reflects the time and energy that an individual uses in the competitive interactions associated with the encounter. We can then define the extrinsic Malthusian parameter (x) by subtracting the total cost of interference (μI) from the intrinsic Malthusian parameter, i.e., let

$$x = r - \mu I \quad (3.6)$$

In Fig. 3.1b I have plotted the relationship between the population density and both r and μI . We see that the population equilibrium, which is defined at $x = 0$, is given at the intercept of the two curves, and that the equilibrium can be substantially lower than the carrying capacity.

In relation to a population that experiences both resource and interference regulation let me describe a few alternative notation forms that I will use throughout the book. Let me first define

$$I = e^t \quad (3.7)$$

as an alternative measure of the level of interference. Then, as $r = r_m - \gamma_\alpha \ln N$, we find that eqn 3.6 is equivalent to

$$x = r_m - \gamma_\alpha \ln N - \mu \ln I \quad (3.8)$$

If I take the exponential to this expression I find that the discrete growth rate is

$$\lambda = \lambda_m N^{-\gamma_\alpha} I^{-\mu} \quad (3.9)$$

If I then combine eqn 3.7 with eqn 3.5 I obtain the following relation

$$I = N^{\gamma_\ell} \quad (3.10)$$

from which I can reformulate eqn 3.9 as

$$\lambda = \lambda_m N^{-\gamma_\alpha} N^{-\mu\gamma_\ell} \quad (3.11)$$

or simply

$$\lambda = \lambda_m N^{-\gamma} \quad (3.12)$$

with $\gamma = \gamma_\alpha + \mu\gamma_\ell$. From eqn 3.12, it is apparent that the population equilibrium is $N^* = \sqrt[\gamma]{\lambda_m}$, and that the four parameters λ_m , γ_α , γ_ℓ , and μ are the limiting parameters, because together they set the population equilibrium.

3.3 Continuous logistic growth

As the Malthusian law is unrealistic it is preferable to describe population growth by exchanging the density independent r in eqn 2.1 with the density

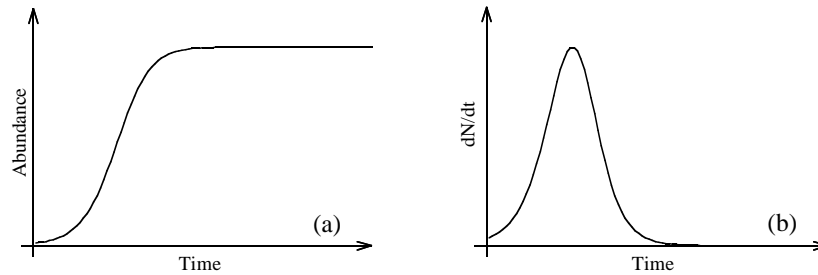


Fig. 3.2 Logistic growth. The abundance (a) and rate of increase in the population (b) plotted against time.

dependent x in eqn 3.8. If I do this, and exchange $\gamma_\alpha \ln N + \mu \ln I$ with $\gamma \ln N$ I obtain the growth equation

$$dN/dt = N(r_m - \gamma \ln N) \quad (3.13)$$

From this equation I can obtain the population equilibrium by setting $dN/dt = 0$ and by solving for N . If I do this I obtain $N^* = \sqrt[\gamma]{\lambda_m}$, which is equal to the equilibrium of the discrete model, as it should be.

The model in eqn 3.13 is only a single of many continuous growth equations describing density regulation. The most famous of these is the linear form

$$dN/dt = N(r_m - \gamma N) \quad (3.14)$$

that was named the logistic equation by Verhulst (1838), who invented it. In this book I use the term logistic as a synonym for the single species models that are based on density regulation. In Fig. 3.2a I have used eqn 3.14 to plot the abundance as a function of time for a hypothetical population. We see that the population increases monotonically until it reaches the population equilibrium. This growth curve is known as logistic growth, and the monotonic return to the equilibrium is a characteristic behaviour of the continuous logistic equations. In Fig. 3.2b I have plotted the rate of increase in total numbers for the curve shown in Fig. 3.2a. We see that the rate of increase has its maximum well below the population equilibrium, and this is another characteristic of logistic growth.

3.4 Discrete logistic growth

In the continuous model the population returns monotonically to the equilibrium because the growth rate is continuously adjusted to the changes in the population density. When instead the generations are non-overlapping

the adjustment occurs in discrete events that are separated by one generation and, therefore, the population may overshoot or undershoot the equilibrium density when it is returning toward it. This process is known as over-compensation and, unless the density regulation function is strongly non-linear, it will generate oscillatory dynamics where the period of a population cycle is two generations.

The transition to over-compensatory dynamics is best illustrated by a population plot that describes the density in generation $t + 1$ as a function of the density in generation t . To see this let us formulate a discrete logistic equation by inserting the density regulated growth rate from eqn 3.12 into eqn 2.3, which describes the discrete process when the growth rate is constant. Then,

$$N_{t+1} = N_t \lambda_m N_t^{-\gamma} \quad (3.15)$$

Now, take the natural logarithm to eqn 3.15 and obtain the linear relation

$$\ln N_{t+1} = r_m + (1 - \gamma) \ln N_t \quad (3.16)$$

and the left hand plots in Fig. 3.3. Here, the solid lines represent eqn 3.16 for different values of γ , and the population equilibrium is the intercept between the solid lines and the dashed diagonal. The transitions to different types of oscillatory dynamics is then given by changes in the slope of the logistic function at the population equilibrium. To determine these transitions let $F = r_m + (1 - \gamma) \ln N_t$ so that the slope is

$$dF/d \ln N = 1 - \gamma \quad (3.17)$$

for all densities including the equilibrium.

As the biologically valid values of γ are larger than zero, the slope can vary from the limit of one to, at least in principle, negative infinity. When $0 < \gamma \leq 1$ the slope is positive and smaller than one, and this implies that the population returns monotonically to the equilibrium, as it is shown in Fig. 3.3a. If, instead, $1 < \gamma$ the slope is negative and the population will overshoot the equilibrium when the abundance is lower than the equilibrium, while it will undershoot the equilibrium when the abundance is above the equilibrium. Consequently, the dynamics is oscillatory in the sense that the period of a population cycle is exactly two generations. When $1 < \gamma < 2$ the absolute value of the slope is smaller than one so that the oscillatory dynamics is damped and the population returns geometrically toward the equilibrium (Fig. 3.3b). If, instead, $\gamma = 2$ the absolute value is one and the amplitude of the oscillation is stable and given by the initial perturbation of the equilibrium (Fig. 3.3c). Finally, when γ is larger than two the amplitude increases geometrically (Fig. 3.3d).

The fact that over-compensation generates oscillatory dynamics raises the question whether the complex dynamics that exists in many in natural

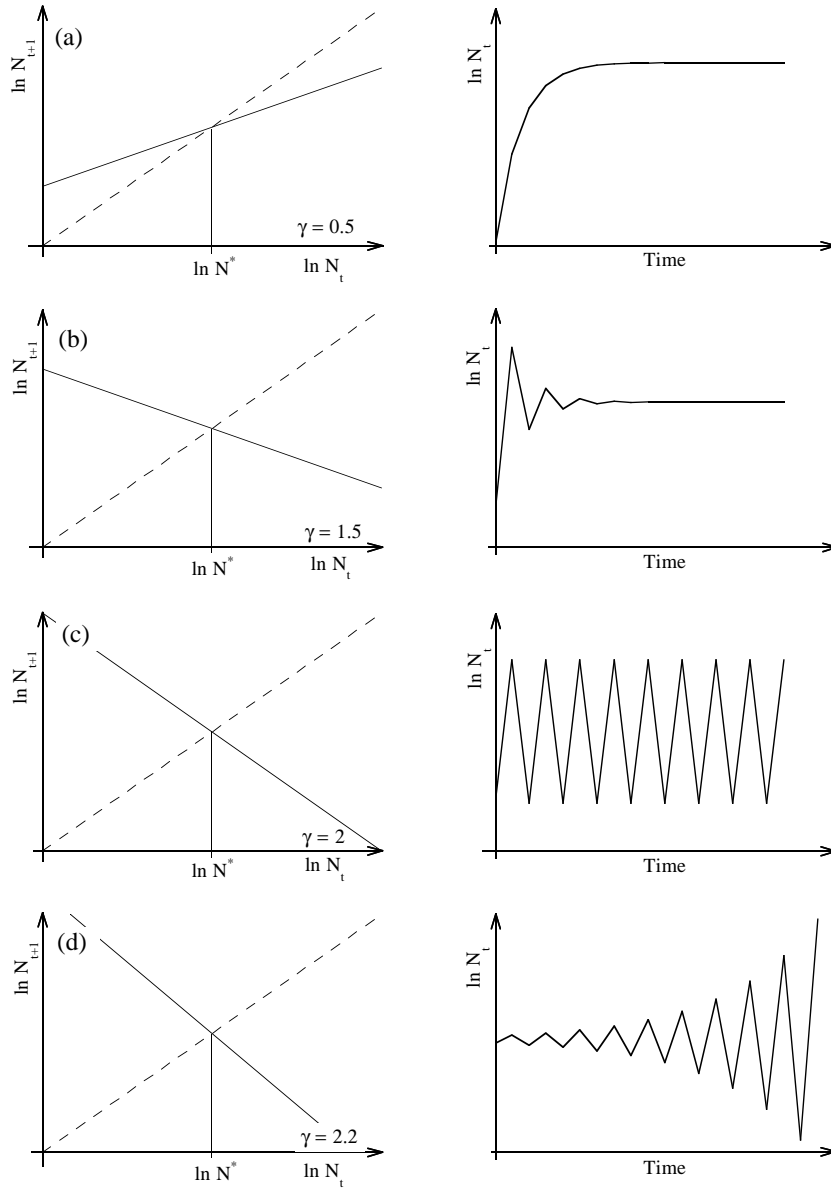


Fig. 3.3 The discrete logistic process of eqn 3.15 on double logarithmic scale for different values of the γ parameter. (Left) Population plots describing the density in generation $t+1$ as a function of the density in generation t . (Right) Population trajectories corresponding to the plots to the left.

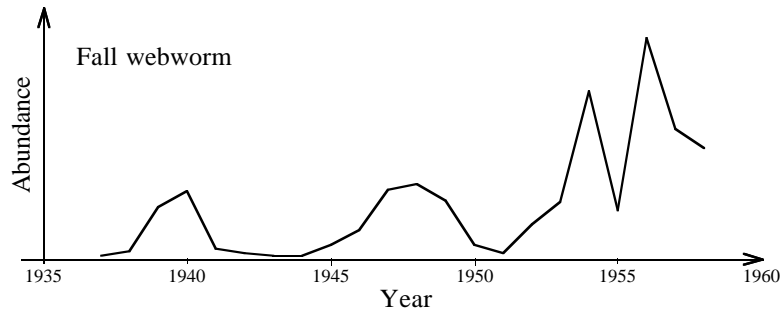


Fig. 3.4 The abundance of the fall webworm plotted against time. Data from Morris (1964).

populations is caused by over-compensatory density regulation. This was first investigated by Hassell et al. (1976) who fitted population data to a discrete logistic equation for 28 populations of insects with non-overlapping generations. For all but one of those populations that breed in natural habitats the fitted equation showed a monotonic return to the equilibrium while the remaining population showed a damped return. It was only in one semi natural and two laboratory populations that the dynamics extended to or beyond a stable oscillation. Later studies by, e.g., Desharnais and Liu (1987) and Costantino et al. (1995) have shown that, although over-compensatory dynamics apparently is rare in natural populations, it is possible to manipulate laboratory populations into the parameter space of over-compensatory dynamics.

Although the study by Hassell et al. did not detect over-compensatory dynamics in natural populations of insects it is well-known that many of these species have non-trivial dynamics that generally is cyclic (e.g., Berryman, 1988; Watt et al., 1990; Turchin, 1990; Witteman et al., 1990; Turchin and Taylor, 1992; Ginzburg and Taneyhill, 1994). By a brief search in the literature I found nine insect species, mainly Lepidoptera, with yearly non-overlapping generations, a cyclic type of dynamics, and with density estimates for periods longer than ten years. The information on these species are listed in Table 3.1 and the data of one species is shown in Fig. 3.4 to illustrate the general type of dynamics in a natural insect population. As these species have been chosen because they have non-trivial dynamics they do not represent the general picture in insects, which appears to be a stable equilibrium (Woiwod and Hanski, 1992).

From Table 3.1 it is apparent that the period in the dynamics of these species generally is larger than two generations, and that the most typical periods are between four and nine generations. In other words, this dynam-

Table 3.1 Data on nine species of insects with periodic cycles in their densities. N_G is the geometric mean in the densities, P is the average length of a population cycles in years, and n is the number of years with estimated densities.

Species	N_G	P	n
Douglas-fir tussock moth	4.1	9.0	10
Fall webworm	10.2	7.0	22
Larch budmoth	2.2	9.3	38
Larch cone fly	945	4.5	10
Nun moth	2600	4.1	42
Pine looper moth	0.66	4.9	50
Southern pine beetle	700	7.7	30
Spruce budworm	1.5	21	28
Wasp spp.	10.2	2.2	25

Data from Royama (1984), Turchin et al. (1991), Dahlsten et al. (1990), Baltensweiler and Fischlin (1988), Morris (1964), Bejer (1988), Barbour (1990), Roques (1988), and Southwood (1967).

ics does not conform to the over-compensatory dynamics of eqn 3.15 that is oscillatory with a period of only two generations. However, as the data represent time series of single species let us fit the classical single species equation to those data in order to examine the degree of inconsistency between the data and the hypothesis of density regulation. As indicated by eqn 3.16, the parameters of eqn 3.15 are easily estimated by a linear regression between $\ln N_{t+1}$ and $\ln N_t$. Although this estimation procedure might result in a slightly biased estimate of γ (Pollard et al., 1987; see also Bulmer, 1994) it is sufficiently accurate for an examination of the degree to which the natural data are consistent with the hypothesis of dynamics by density regulation.

The estimated parameters are listed in Table 3.2 together with the dynamics of eqn 3.15 for those estimated parameters. First, we notice that there is generally no consistency between the estimated and the natural equilibrium, when the natural equilibrium is given by the geometric mean in the population data. Also, as eqn 3.15 describes density regulation the estimated γ parameters must be positive before they are biologically valid. For four of the nine species the γ parameter is negative and biologically invalid. For four of the remaining five species the estimated models predict a monotonic return to the equilibrium, and this does not coincide with the cyclic dynamics that is found within those species. It is only for the last species that the prediction coincides with the observation, and here the observed period is 2.2 years while predicted dynamics is a damped return with a period of two years.

Table 3.2 Parameter estimates for the discrete logistic equation (eqn 3.15). The dynamics obtained by eqn 3.15 and the estimated parameters are listed under P : M stands for a monotonic return, and 2 for a damped cycle with a period of 2 years. The column under r list the correlation coefficient between $\ln N_{t+1}$ and $\ln N_t$.

Species	γ	λ_m	N^*	r	P
Douglas-fir tussock moth	-0.48	2.16	(0.20)	0.96	-
Fall webworm	-0.31	4.28	(0.006)	0.82	-
Larch budmoth	0.28	1.38	3.17	0.74	M
Larch cone fly	0.68	97700	$2.4 \cdot 10^7$	0.33	M
Nun moth	0.61	109	2300	0.38	M
Pine looper moth	0.32	0.61	0.21	0.60	M
Southern pine beetle	-0.58	14.4	(0.01)	0.94	-
Spruce budworm	-0.72	1.17	(0.80)	0.94	-
Wasp spp.	1.89	7.28	32.6	-0.57	2

3.5 Delayed density dependence

Another type of models that has been used to explain the occurrence of cyclic dynamics in natural populations are models that incorporate delayed feedback, also known as delayed density dependence (e.g., Hutchinson, 1948; Morris, 1959; Varley et al., 1973; Berryman, 1978, 1995; Berryman et al., 1987; Witteman et al., 1990; Turchin and Taylor, 1992; Hörnfeldt, 1994). In these models it is the custom to incorporate feedback terms that are successively delayed in steps of either one year or one generation, and this is because it usually is on those time scales that the data have been collected. A continuous and linear model with delays that reach back three generations will look like

$$dN/dt = r + \gamma_t N_t + \gamma_{t-1} N_{t-1} + \gamma_{t-2} N_{t-2} + \gamma_{t-3} N_{t-3} \quad (3.18)$$

while the corresponding discrete model will take the form

$$N_{t+1} = \lambda_m N_t^{\gamma_t} N_{t-1}^{\gamma_{t-1}} N_{t-2}^{\gamma_{t-2}} N_{t-3}^{\gamma_{t-3}} \quad (3.19)$$

Such models have been developed to include delays for up to at least eleven successive generations (see Royama, 1992), but generally it is the custom to include only delayed regulation that reaches back a few generations.

It is important to notice that delayed density dependence has nothing to do with the (direct) density dependence I described earlier in this chapter. This is because (direct) density dependence refers to the processes that regulate the population through the exploitation of the resource and the

amount of interference in the population. Although the regulatory effects of these processes might be slightly delayed, e.g., due to physiological delays (Akçakaya et al., 1988), they generally operate instantaneously on a population dynamic time scale. That is to say that there is no evidence that the regulatory effects of (direct) density dependence is delayed in the order of years and/or generations. This implies that delayed density dependence refers to other processes and these are almost never explicitly defined in the studies that deal with delayed density dependence. Due to this lack of a mechanism that can cause the delayed regulation we often have no a priori expectation about the realistic values of the gamma parameters in eqns 3.18 and 3.19 and, therefore, they are allowed to be both positive and negative, which is in contrast to (direct) density dependence where the gamma parameter is always positive.

As the concept of delayed density dependence is uncoupled from specific biological processes the major use of these models is not to explain why the population dynamics behaves in the way it does, but instead to approximate the overall process with models that sidestep the problem of biological causality. These approximations can be used to improve the forecast of the population trajectory of a given species even though we do not know the underlying mechanism generating the trajectory. They can be used also as a statistical tool whereby it is possible to identify the time lags of the essential feedback mechanisms underlying the dynamics in natural populations. This has been done by, e.g., Bjørnstad et al. (1995) and Stenseth et al. (1996), and these studies agree with Turchin and Taylor (1992), Ginzburg and Taneyhill (1994), and Berryman (1996) in the conclusion that the essential feedback mechanism often operate with a time lag of approximately one generation. The interesting biological question is then to quantify a biological mechanism in order to analyse whether it can explain both the observed dynamics and the observed time lag.

In this book I will examine two feedback mechanisms that may explain the cyclic dynamics in natural populations. The first mechanism, which is described in the following chapter, is the hypothesis of a predator-prey driven cycle. This hypothesis was first introduced in the 1920s by Lotka and Volterra and it can generate a time lag of one generation if the generation time is identical for the prey and the predator species. This is, however, often not the case. The second mechanism, which I develop in Chapter 24, is the hypothesis that the dynamics of a single species is inherently cyclic because of the action of selection by density dependent competitive interactions. In contrast to the predator-prey hypothesis this latter hypothesis will always induce a time lag of one generation, and this is because the evolutionary effects of selection are delayed by a single generation. A third mechanism that will induce a delay of one generation is the maternal effect

hypothesis that was introduced by Ginzburg and Taneyhill (1994). This effect may be a special case of the selection hypothesis in the sense that selection in a dynamic system will select for a flexible, or plastic, phenotype, and one way to induce such plasticity is through maternal effects. For a brief discussion of the maternal effect hypothesis, see Berryman (1995) and Ginzburg and Taneyhill (1995).

Chapter 4

Predator-prey

THE STUDY on the interactions between predators and their prey began with the development of the Lotka-Volterra predator-prey equations. These equations were developed independently by Volterra (1926) in Italy and Lotka (1925) in United States in order to explain the widespread occurrence of periodic cycles in the abundances of animal populations. We recall that the continuous form of the logistic equation predicts that populations return monotonically to the equilibrium. Thus, to explain the cyclic dynamics that occur in natural species Lotka and Volterra needed to invent a new mechanism and they chose the interactions between the predator and the prey. Ever since, the study of predator-prey interactions has been inseparable from the study of population cycles, a tradition that is maintained in this chapter.

Apart from the hypothesis that population cycles are caused by predator-prey interactions there have been one major opposing hypothesis. This is the Chitty hypothesis (Chitty, 1960) that states that population cycles are caused by factors intrinsic to the population. Originally, the Chitty hypothesis was relatively vaguely formulated in the sense that the crucial factors were never incorporated into a population dynamic equation that showed cyclic dynamics. Among other things, the hypothesis was based on the concept of self-regulation that was elaborated into group selection by Wynne-Edwards (1962, 1986). Several authors (Dekker, 1975; Stenseth, 1978, 1981; Thue Poulsen, 1979; Hunt, 1982) performed theoretical studies over the Chitty hypothesis, and Stenseth (1981, 1995) concludes that they either failed to explain the occurrence of population cycles or that the studies were burdened by unrealistic assumptions. So although there was some evidence in its favour (Krebs, 1978; Boonstra and Krebs, 1979) the intrinsic hypothesis it is now generally discounted.

Today ecologists are returning to the old idea that population cycles in

animals as diverse as snowshoe hares (Akçakaya, 1992; Krebs et al., 1995), rodents (Hanski et al., 1993; Hanski and Korpimäki, 1995), and insects (Berryman, 1996) are caused by predator-prey interactions in their broadest definition including both host-parasitoid and plant-herbivore interactions. The reason for this is obvious. In the absence of an alternative hypothesis, and with a logistic equation that does not explain the observed dynamics, the straightforward path is to include an extra dimension extrinsic to the population. And here predators and parasites are obvious candidates because their densities tend to correlate positively with the densities of their prey or hosts.

A few years ago Ginzburg (1992) pointed out that the conclusion that predator-prey interactions are essential for cyclic dynamics is based on the implicit assumption that the maximal growth rate is density independent. In the last part of this book I will show that when we incorporate the effects of density dependent selection the maximal growth rate becomes density dependent and the logistic equation extends so that it can easily explain the observed population cycles. The idea of this chapter is to review the classical theory behind the predator-prey driven cycles keeping in mind that the predator-prey interaction is not necessarily for the occurrence of cyclic dynamics.

Before I describe the theory let me briefly consider the degree to which predator-prey driven cycles have been confirmed empirically. Early studies that were conducted by, e.g., Gause (1934), Utida (1957), and Huffaker (1958), showed that it apparently is possible to manipulate laboratory systems into the parameter space of a predator-prey cycle. However, a later study by Murdoch and McCauley (1985), on an isolated *Daphnia*-algae system showed that the interpretation of laboratory results may be extremely difficult. In this latter study the *Daphnia* cycled with a relatively fixed period independently of the presence versus absence of a cycle in its prey. The conclusion to this study is that periodic dynamics can be caused by a mechanism intrinsic to the population, and that some intrinsic driven cycles apparently look like predator-prey cycles because of the predator-prey interactions although it may not be these interactions that drive the cycle. In this light it is not surprising that the snowshoe hare apparently continues to cycle in the absence of the lynx (Keith, 1963), that predation often cannot account for the recurrent declines in vole numbers (Godfrey, 1955; Lockie, 1955; Chitty, 1960), and that the best documented cycle in forest insects lacks a firm predator-prey interaction (Berryman, 1996). In Chapter 24 I will return to this latter example that is the larch budmoth that “goes through 10000-fold changes in density during its very regular $[8.24 \pm 0.27$ (SE) years] cycle in the Alps” (Berryman, 1996).

4.1 The Lotka-Volterra equations

In the original Lotka-Volterra equations the density regulation of the predator and the prey is modelled explicitly by the predator's exploitation of the prey. This implies that the predator is resource (prey) regulated while the prey is predator regulated. That is to say that the prey is the only regulating factor that affects the predator, and the predator is the only regulating factor that affects the prey. This is done by assuming that the prey (N_1) will increase exponentially with a characteristic rate (r_1) whenever the predator (N_2) is absent. It is then assumed that each predator will consume $\gamma_{12}N_1$ prey per unit time, and that the predator feeds exclusively upon the prey species that is included in the equations. This implies that in the absence of prey the predators will die exponentially with a rate d_2 . As each predator consumes prey at the rate $\gamma_{12}N_1$, a predator produces offspring at the rate $\gamma_{21}N_1$, where $\gamma_{21} = a\gamma_{12}$ and a is the number of predators produced per consumed prey. Given these conditions the predator-prey equations are:

$$\begin{aligned} dN_1/dt &= N_1(r_1 - \gamma_{12}N_2) \\ dN_2/dt &= N_2(\gamma_{21}N_1 - d_2) \end{aligned} \quad (4.1)$$

Having obtained the equations we want to determine whether the predator and the prey can coexist at a stable equilibrium. At the equilibrium we have that $dN_1/dt = 0$ and that $dN_2/dt = 0$. These relations are easy to solve so that we obtain the equilibrium

$$\begin{aligned} N_1^* &= d_2/\gamma_{21} \\ N_2^* &= r_1/\gamma_{12} \end{aligned} \quad (4.2)$$

that is always positive for both populations.

The dynamics of the predator-prey system is usually illustrated on a population graph where each axis describes the density of one of the two species. On this graph we can plot the isoclines of the two species, i.e., the curves at which the rate of increase in each species is zero. I have done this in Fig. 4.1a where it is apparent that the isocline of the predator is independent of the predator density, that the isocline of the prey is independent of the prey density, and that the equilibrium densities are given at the intercept between the two lines. The independence between the isocline of a species and the density of that species reflects the assumption that the predator is purely prey regulated, and that the prey is purely predator regulated. Hereby the rate of increase in each species depends only upon the density of the other species. This implies that when the prey density is high the predator density will increase, and when the prey density is low the predator density will decline. Likewise, the prey is declining when there

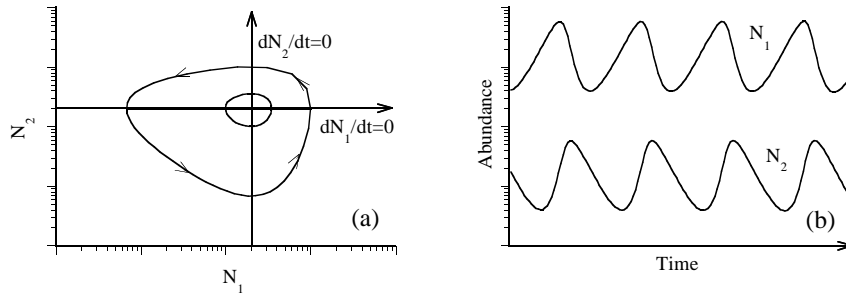


Fig. 4.1 The Lotka-Volterra predator-prey model (eqn 4.1). (a) A plot of the predator abundance (N_2) against the prey abundance (N_1). The two cyclic trajectories illustrate the prediction of a predator-prey driven cycle that is neutrally stable. (b) The abundances of a predator-prey cycle shown against time.

are many predators and the prey is increasing when there are few predators. These patterns of increase and decrease imply that populations away from the equilibrium point will continue to cycle counterclockwise in a neutrally stable orbit, as it is illustrated by the two simulations in Fig. 4.1a. That the orbit is neutrally stable means it is given by initial conditions, that is to say that the orbit is determined by the initial densities of the two populations. If the density of one or both of the populations is perturbed by a single fluctuation in the environment the populations will jump into a new orbit in which they will remain until one or both of the populations are perturbed again. In Fig. 4.1b the abundances of such a cycle are plotted against time.

4.2 Predator caused extinction

The equilibrium prediction of the original predator-prey equations has generated the hypothesis that “neither predators parasitoids nor pathogens can depress their prey or host populations to the point of eradication” (Hassell and Anderson, 1989:185). This hypothesis depends upon the assumption that the predator is a specialist that in the absence of the prey is doomed to die exponentially (the term $-d_2$). In many cases the predator is more of a generalist that preys upon several species and, then, the predator is likely to have a positive rate of increase just like its prey. Thus, instead of the classical set of equations we may expect the following equations

$$\begin{aligned} dN_1/dt &= N_1(r_1 - \gamma_{12}N_2) \\ dN_2/dt &= N_2(r_2 + \gamma_{21}N_1) \end{aligned} \quad (4.3)$$

If I try to solve the equilibrium by setting $dN_1/dt = 0$ and $dN_2/dt = 0$ I find that there is no equilibrium. The equilibrium density of the prey is

always negative ($N_1^* = -r_2/\gamma_{21}$), which implies the extinction of the prey, whereas the predator continues to increase exponentially ($dN_2/dt = r_2N_2$). This prediction, that a predator will always drive its prey to extinction and then continue to increase exponentially, is unrealistic, and it is clear that the unrealistic assumption is the exponential increase in the predator. Although this prediction appears to be more unrealistic than the classical predictions this is not necessarily the case, and this is because the assumption of a positive r_2 is not more unrealistic than the four assumptions in the original Lotka-Volterra equations, which are, the assumptions of exponential increase in the prey, exponential decline in the predator, pure prey regulation of the predator, and pure predator regulation of the prey.

4.3 Adding interference competition

Since most species feed upon a variety of resources and since all populations, at least to some degree, experience intra-specific interference, a more general approach is to use the logistic equation as a base on which we can add the explicit interactions between the two species in study. If we do this we obtain the following equations

$$\begin{aligned} dN_1/dt &= N_1(r_1 - \gamma_{11}N_1 - \gamma_{12}N_2) \\ dN_2/dt &= N_2(r_2 + \gamma_{21}N_1 - \gamma_{22}N_2) \end{aligned} \quad (4.4)$$

In these equations the logistic terms ($-\gamma_{11}N_1$ & $-\gamma_{22}N_2$) include both regulation by intra-specific interference and regulation by interactions with unspecified species. Solving for the equilibrium we find that

$$\begin{aligned} N_1^* &= \frac{\gamma_{22}r_1 - \gamma_{12}r_2}{\gamma_{11}\gamma_{22} + \gamma_{12}\gamma_{21}} \\ N_2^* &= \frac{\gamma_{21}r_1 + \gamma_{11}r_2}{\gamma_{11}\gamma_{22} + \gamma_{12}\gamma_{21}} \end{aligned} \quad (4.5)$$

We see that the abundance of the prey is positively related to the growth rate in the prey (r_1) and the logistic regulation in the predator (γ_{22}), whereas it is negatively related to the growth rate in the predator (r_2), the exploitation of the prey by the predator (γ_{12} & γ_{21}), and the regulation in the prey (γ_{11}). All these relations make intuitive sense. So does the extinction of the prey when the predator growth rate (r_2) and/or consumption rate (γ_{12}) are high, or when the predator regulation (γ_{22}) and/or the prey growth rate (r_1) are low. The positive relationship between the predator equilibrium abundance (N_2^*) and the growth rate of both species (r_1 & r_2) is also intuitively appealing, like the inverse relationship between the predators equilibrium abundance (N_2^*) and the regulation in the predator (γ_{22}),

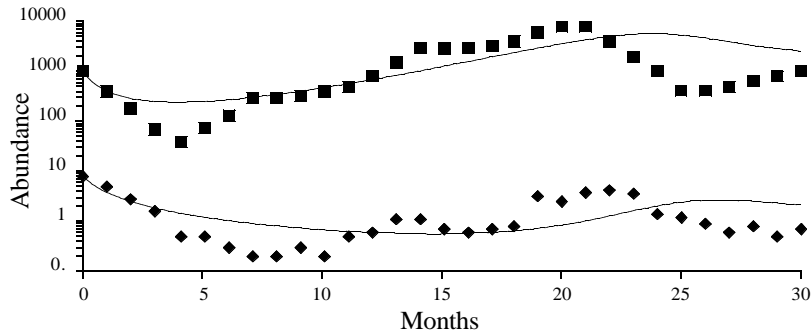


Fig. 4.2 A predator-prey cycle between the California vole (squares) and feral cats (diamonds). The two curves are the trajectories of the model $dN_1/dt = N_1(0.31 + 0.000017N_1 - 0.21N_2)$ and $dN_2/dt = N_2(-0.028 + 0.000088N_1 - 0.13N_2)$ that is estimated from the data by multiple linear regression. Data from Pearson (1966).

and that the extinction of the predator requires a negative growth rate in the predator. Less straightforward is the more complex relation between the predators equilibrium abundance and its exploitation of the prey.

If we turn to population dynamics it has been known since Lotka (1925) that logistic terms in predator-prey equations will stabilise the system creating dynamics in between a monotonic return to the equilibrium and a neutrally stable orbit, with inward spiralling trajectories being one possibility. For such a flexible model (eqn 4.4) it is essential to find out which of the two forces is the stronger in natural populations; the destabilising predator-prey interactions or the stabilising terms of logistic regulation. This is not an easy task and to my knowledge such a study has not yet been conducted for a natural population. Here, let me restrict myself to a simple fitting of the general predator-prey equation to the data from Pearson's studies (1966) on the cyclic dynamics in voles and cats. Using standard multiple linear regression, as it is described by Sokal and Rohlf (1981), the dynamics of the estimated model is shown in Fig. 4.2 together with the original data.

Chapter 5

Food chains

IN THE PREVIOUS chapter we saw that the predator-prey equations were constructed to explain the occurrence of cyclic dynamics, rather than being constructed to explain the setting of the population equilibrium. In this chapter I will arrange several predator-prey equations on top of one another in order to examine the equilibration of food chains.

From empirical evidence we know that food chains generally include three or four species in terrestrial systems and up to seven species in marine plankton-based systems (Ricklefs, 1990). We also know that the biomass at each trophic level is positively correlated with the primary production (e.g., Arditi et al., 1991; Ginzburg and Akçakaya, 1992). This positive relationship makes so much intuitive sense that it appears trivial. Nevertheless, it is a major problem for the original predator-prey equations that are based on pure exploitative competition, a problem that is known as the “paradox of enrichment”. This paradox was generally neglected until Arditi and Ginzburg (1989) pointed out that the traditional food chain models predict that the response varies with the length of the food chain, and that the only level that always responds proportionally to an increase in primary production is the top predator, whereas the next last level is always constant, and at lower trophic levels the biomass may even decline.

To avoid the paradox of enrichment Arditi and Ginzburg (1989) introduced ratio-dependent predator-prey equations where the predator consumption of prey depends upon the prey/predator ratio instead of on the abundance of the prey, as it is assumed in the Lotka-Volterra equations. The rationale behind the ratio-dependent response is that it may be the result from the fact that the time scale of foraging often is faster than the time scale of population dynamics, or the result from interactions among predators that divide up the resource before they consume it. Since their introduction the ratio dependent models have been the centre of a lively de-

bate where some ecologists defend the traditional Lotka-Volterra equations by arguing that the ratio-dependent models are non-causal. It is, though, fair to say that no firm conclusion has yet been reached, and this is because the choice of model depends upon the scale of modelling (see, e.g., Matson and Berryman, 1992; Arditi et al., 1992; Oksanen et al., 1992; Ruxton and Gurney, 1992; Abrams, 1994; Gleeson, 1994; Sarnelle, 1994; McCarthy et al., 1995; Akçakaya et al., 1995; Berryman et al., 1995).

In this chapter we will see that an alternative way to obtain the proportional relation between the population densities in a food chain and the primary production is by adding the regulatory component of intra-specific interference competition to each species in the chain. But first I will show that food chains that are based on the idea of exploitative competition cannot explain the observed pattern.

5.1 Exploitative versus interference competition

The building blocks in food chain models are predator-prey equations, and under the hypothesis of regulation by exploitation these equations are the original Lotka-Volterra equations. A four-species chain will then resemble

$$\begin{aligned} dN_1/dt &= N_1(r_1 - \gamma_{12}N_2) \\ dN_2/dt &= N_2(\gamma_{21}N_1 - \gamma_{23}N_3) \\ dN_3/dt &= N_3(\gamma_{32}N_2 - \gamma_{34}N_4) \\ dN_4/dt &= N_4(\gamma_{43}N_3 - d_4) \end{aligned} \quad (5.1)$$

if we disregard density independent mortality (d) for the species that are regulated by a predator. By setting the four equations equal to zero we obtain the equilibrium

$$\begin{aligned} N_1^* &= c_1 \\ N_2^* &= c_2r_1 \\ N_3^* &= c_3 \\ N_4^* &= c_4r_1 \end{aligned} \quad (5.2)$$

where c_1 to c_4 are the constants $c_1 = c_3\gamma_{23}/\gamma_{21}$, $c_2 = 1/\gamma_{12}$, $c_3 = d_4/\gamma_{43}$, and $c_4 = c_2\gamma_{32}/\gamma_{34}$. From eqn 5.2 it is apparent that when the primary production (r_1) is increased the changes in the equilibrium points do not resemble the observed proportional increase at all trophic levels. Instead, the response varies with trophic level so that only the biomass at the second and fourth trophic levels correlate positively with the primary production,

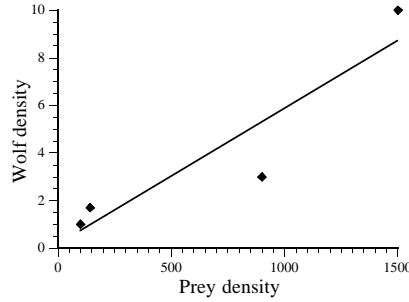


Fig. 5.1 The density, in individuals per 100 square miles, of the wolf against its prey (different species of deer). Data obtained from Ricklefs (1990); originally from Cowan (1947), Thompson (1952), Pimlott (1967), and Kelsall (1968).

whereas the biomass of the first and third trophic levels is independent of the primary production.

If we add the regulatory component of intra-specific interference competition to each species in the chain we find that eqn 5.1 extends to

$$\begin{aligned}
 dN_1/dt &= N_1(r_1 - \gamma_{11}N_1 - \gamma_{12}N_2) & (5.3) \\
 dN_2/dt &= N_2(\gamma_{21}N_1 - \gamma_{22}N_2 - \gamma_{23}N_3) \\
 dN_3/dt &= N_3(\gamma_{32}N_2 - \gamma_{33}N_3 - \gamma_{34}N_4) \\
 dN_4/dt &= N_4(\gamma_{43}N_3 - \gamma_{44}N_4)
 \end{aligned}$$

if we disregard d_4 . Solving for the equilibrium we obtain

$$\begin{aligned}
 N_1^* &= c_1 r_1 & (5.4) \\
 N_2^* &= c_2 r_1 \\
 N_3^* &= c_3 r_1 \\
 N_4^* &= c_4 r_1
 \end{aligned}$$

where c_1 to c_4 are the constants $c_1 = 1/(\gamma_{11} + c_5\gamma_{12})$, $c_2 = c_1c_5$, $c_3 = c_2c_6$, and $c_4 = c_3c_7$, with $c_5 = \gamma_{21}/(\gamma_{22} + c_6\gamma_{23})$, $c_6 = \gamma_{32}/(\gamma_{33} + c_7\gamma_{34})$, and $c_7 = \gamma_{43}/\gamma_{44}$. From eqn 5.4 it is apparent that the regulatory effects of intra-specific interference has restructured the chain so that the abundance at all trophic levels has become proportional to the primary production (r_1). This implies that the ratios of predators over prey have the characteristic values

$$\begin{aligned}
 N_2^*/N_1^* &= c_5 & (5.5) \\
 N_3^*/N_2^* &= c_6 \\
 N_4^*/N_3^* &= c_7
 \end{aligned}$$

This constancy in the ratio of predators to prey is illustrated in Fig. 5.1 for four populations of wolves feeding on deer.

As interference competition is present in any population it is not surprising that we need to add it to the equations in order to explain the equilibrium. Notice in this context that when we explain the setting of the equilibrium we tend to lose the classical prediction of cyclic dynamics. This is because the addition of interference competition to the predator-prey equations makes the equilibrium stable and the dynamics damped.

Chapter 6

Inter-specific competition

IN THE STUDY of competition among species the focus is directed toward the conditions that allow the different species to coexist. Volterra (1926) was the first to show that two competing species can coexist at equilibrium only if the regulatory effects of intra-specific competition is larger than the regulatory effects of inter-specific competition. Niche differentiation is one mechanism allowing this to occur and Volterra's deduction was soon seen as support for the hypothesis that two co-occurring species must in some sense be occupying different niches. This view was supported by the famous experiments of Gause (1934) that showed competitive exclusion between *Paramecium aurelia* and *Paramecium caudatum*. Hereafter, the niche hypothesis became almost ordained in what is known as the Gause axiom, or the Volterra-Gause principle of competitive exclusion among species that occupy similar niches.

The Volterra-Gause principle is still treated as a fundamental law in most text books (e.g., Begon et al., 1990; Ricklefs, 1990) and in recent reviews (Chesson 1991; Mathew, 1995). The principle is, nevertheless, basically wrong. Although niche differentiation promotes, it is by no means a necessity for, the coexistence of species. It is only a necessity if competition is purely exploitative, but competition always includes some degree of interference. Schoener (1976) and Vance (1985) analysed the situation with interference competition and showed that although coexistence within a single niche is not guaranteed it can occur under many circumstances. If two species with completely overlapping niches cannot coexist due to interference competition there is also the chance that they might coexist in a seasonal (Stewart and Levin, 1973), fluctuating (Chesson and Warner, 1981), or patchy (Horn and MacArthur, 1972) environment.

In the study of species coexistence the interesting goal is to find the rules determining the patterns in which natural species coexist, i.e., the

rules that determine which species can coexist and which cannot. This is the study of the assembly of natural communities, a field in which there has been only relatively moderate theoretical achievements (Roughgarden, 1989).

Probably the most clear-cut pattern in natural communities is the pattern that is described by Hutchinson's rule (1959) that has been observed in a variety of taxa ranging from mammals over birds to insects (Hutchinson, 1959; Diamond, 1973, 1975; Cody, 1974; Emmons, 1980; Bowers and Brown, 1982; Pyke, 1982; Begon et al., 1990). According to this rule two, or more, strongly competing species can coexist when the weight ratio of neighbouring species is larger than two.

Unlike the Gause axiom there has never been a theoretical explanation of Hutchinson's rule. In the studies of coexistence through niche differentiation made by MacArthur and Levins (1967), May and MacArthur (1972), and Roughgarden (1976) a theoretical basis for Hutchinson's rule never emerged, and it is probably therefore that this rule often is considered to be insignificant, or even false (Roth, 1981; Lewin, 1983; Begon et al., 1990). In this chapter I will, however, deduce Hutchinson's rule from the regularities by which the inter-specific competitive interactions partition the resource among species. To do this I will proceed in three sections that resemble respectively the absence of competitive interactions, the presence of intra-specific interactions, and the presence of both intra- and inter-specific interactions. The two first sections are brief reviews describing the deduction and falsification of the Gause axiom, and in the last section I deduce Hutchinson's rule from inter-specific interactions.

6.1 Exploitation: Competitive exclusion

When competition is purely exploitative we can describe the case with two competing species (N_1 & N_2) on a single resource (E) by the Lotka-Volterra predator-prey equations. That is

$$\begin{aligned} dE/dt &= E(r_e - \gamma_{e1}N_1 - \gamma_{e2}N_2) \\ dN_1/dt &= N_1(\gamma_{1e}E - d_1) \\ dN_2/dt &= N_2(\gamma_{2e}E - d_2) \end{aligned} \quad (6.1)$$

Let us ignore the infinitely small possibility that $\gamma_{1e}/d_1 = \gamma_{2e}/d_2$, since in this case there is infinitely many equilibria [$E^* = \gamma_{1e}/d_1 = \gamma_{2e}/d_2 \wedge N_1^* = (r_e - \gamma_{e2}N_2^*)/\gamma_{e1}$] at which the two species can coexist. In general, where $\gamma_{1e}/d_1 \neq \gamma_{2e}/d_2$, eqn 6.1 predicts that the species with a positive growth rate at the lowest resource density persists, while the other species becomes extinct, i.e., if $\gamma_{1e}/d_1 < \gamma_{2e}/d_2$ we find that species 2 persists while species 1 goes extinct.

In order to explain the equilibrium coexistence of multiple competing species the classical approach with pure exploitative competition requires the additional concept of niche differentiation. However, it has been known for some time that it is not the number of niches that allow a set of species to coexist, instead it is the number of regulating factors where n species require at least n regulating factors (Levin, 1970). When competition is purely exploitative the exploitation of resources is the only regulating factor and, thus, if the use of resource is identical among species only a single regulating factor exist and only a single species can persist at an equilibrium, as shown by the equations above.

6.2 Intra-specific interference: Competitive coexistence

If to the system where competition is purely exploitative we add the regulatory component of intra-specific interference competition we find that each species will be regulated by a factor that is a combination of at least two underlying regulatory factors: the exploitation of the resource and intra-specific interference. Thus, a set of n competing species that feed upon a single resource will contain at least $n + 1$ regulating factors, which implies that the minimum requirement for coexistence is fulfilled.

To illustrate that there is no problem in having several coexisting species on a single resource let us first consider the case with two species on a single resource. With intra-specific interference eqn 6.1 extends to

$$\begin{aligned} dE/dt &= E(r_e - \gamma_{ee}E - \gamma_{e1}N_1 - \gamma_{e2}N_2) \\ dN_1/dt &= N_1(\gamma_{1e}E - \gamma_{11}N_1 - d_1) \\ dN_2/dt &= N_2(\gamma_{2e}E - \gamma_{22}N_2 - d_2) \end{aligned} \quad (6.2)$$

with the equilibrium

$$\begin{aligned} E^* &= \frac{r_e + d_1\gamma_{1e}/\gamma_{11} + d_2\gamma_{2e}/\gamma_{22}}{\gamma_{ee} + \gamma_{e1}\gamma_{1e}/\gamma_{11} + \gamma_{e2}\gamma_{2e}/\gamma_{22}} \\ N_1^* &= (E^*\gamma_{1e} - d_1)/\gamma_{11} \\ N_2^* &= (E^*\gamma_{2e} - d_2)/\gamma_{22} \end{aligned} \quad (6.3)$$

Hence, the two species can coexist when $d_1 < E^*\gamma_{1e}$ and $d_2 < E^*\gamma_{2e}$, i.e., when density independent mortality is low relative to the efficiency by which the organisms utilise the resource at equilibrium. This situation is easily extended to include any number (n) of species

$$\begin{aligned} dE/dt &= E(r_e - \gamma_{ee}E - \sum_{i=1}^n \gamma_{ei}N_i) \\ dN_i/dt &= N_i(\gamma_{ie}E - \gamma_{ii}N_i - d_i) \end{aligned} \quad (6.4)$$

with the equilibrium

$$\begin{aligned} E^* &= \frac{r_e + \sum_{i=1}^n d_i \gamma_{ei} / \gamma_{ii}}{\gamma_{ee} + \sum_{i=1}^n \gamma_{ei} \gamma_{ie} / \gamma_{ii}} \\ N_i^* &= (E^* \gamma_{ie} - d_i) / \gamma_{ii} \end{aligned} \quad (6.5)$$

For this equilibrium we find that competitive exclusion by the Gause rule is the extreme that occurs when density independent mortality is high relative to resource consumption at equilibrium. At the other extreme where density independent mortality is absent all species will persist on the single resource.

6.3 Intra- and inter-specific interference: Hutchinson's rule

In systems that are biased toward competitive coexistence, competitive exclusion can occur because of inter-specific interference competition, a component that is absent from eqns 6.2 and 6.4. In this section I will show that competitive exclusion by inter-specific interference can explain Hutchinson's rule that two strongly competing species can coexist when their body mass ratio is larger than two.

To deduce Hutchinson's rule let us describe the conditions for which two species with different body masses can coexist on a single resource. And, let us focus on the case where the two species are equally suited to assimilate resource in the absence of competitive interactions, i.e., where $\gamma_{e1} = \gamma_{e2}$. Then, because the number of offspring that can be produced from a given amount of resource is inversely proportional to the mass of each offspring we may expect that the intrinsic growth rate is inversely proportional to body mass (w), i.e., that $r_1 = r/w_1$ and that $r_2 = r/w_2$ with $r = \gamma_{1e}E - d_1 = \gamma_{2e}E - d_2$. Here, it is also assumed that the scaling between body mass and respectively reproduction and survival is the same, and this seems to hold in the light of the body mass allometries in Chapter 13.

To determine the γ parameters that describe the regulation by interference competition let us consider contest competition where the winner gets access to a resource item while the loser is prevented access. Then, the γ parameters are proportional to the fraction of the competitive encounters that are lost. Now, let $w_1 > w_2$. Then, when an individual of the large species 1 encounters an individual of the small species 2, the small individual is expected to lose and, thus, $\gamma_{21} = \gamma$ and $\gamma_{12} = 0$, where γ is a

positive constant. When the encountering individuals belong to the same species there is, on average, a 50% chance of winning the encounter. This implies that $\gamma_{11} = \gamma_{22} = \gamma/2$, and that the population equations are

$$\begin{aligned} dN_1/dt &= N_1(r/w_1 - \gamma N_1/2) \\ dN_2/dt &= N_2(r/w_2 - \gamma N_1 - \gamma N_2/2) \end{aligned} \quad (6.6)$$

and the equilibrium

$$\begin{aligned} N_1^* &= \frac{2r^*}{\gamma w_1} \\ N_2^* &= \frac{2r^*}{\gamma w_2} - \frac{4r^*}{\gamma w_1} \end{aligned} \quad (6.7)$$

with $r^* = \gamma_{1e}E^* - d_1 = \gamma_{2e}E^* - d_2$, and given that $E^* > 0$. Hence, when the intrinsic growth rates are positive at equilibrium, the large species 1 persists whereas the small species 2 may be competitively excluded by the large species. Furthermore, the two species can coexist when $N_2^* > 0$, i.e., when

$$w_1/w_2 > 2 \quad (6.8)$$

and they are equally abundant when $w_1/w_2 = 3$.

In order to extend to an arbitrary number of species let us consider the case where the competitive interactions are ranked in the sense that a species competes mainly with the two species of neighbouring sizes. This can, e.g., be the case when the inter-specific interactions rank the different species into different niches where each species get into contact mainly with the two species of neighbouring sizes. This type of ranking may be expected, and this is because the interactions with the individuals of the neighbouring species will remind the individuals of each species of their position in the inter-specific hierarchy, and at the same time the niche separation will ensure that the smaller species avoid getting into unnecessary conflicts with species that are much larger than themselves. In this system we may use eqn 6.6 to describe pairwise competition. Then, for the three species 1, 2 and 3 with $w_1/w_2 > 2 \wedge 1 < w_2/w_3 < 2$ we find that species 2 will exclude species 3. This implies that the set of species that coexist after sufficiently many invasions will be regularly packed in the sense that $w_1/w_2 \approx \dots w_{i-1}/w_i \approx \dots w_{n-1}/w_n \approx 2$ when $w_1 > w_2 > \dots w_{i-1} > w_i > \dots w_{n-1} > w_n$. Based on a computer simulation this type of species packing is illustrated in Fig. 6.1, and the result of equal weight-spacing on a logarithmic scale is shown in Fig. 6.2 for seed-eating desert rodents.

What happens when the assumptions behind the deduction of Hutchinson's rule fail? (i) If the significant interactions at the point of invasion are not restricted to interactions between neighbouring species, then it is

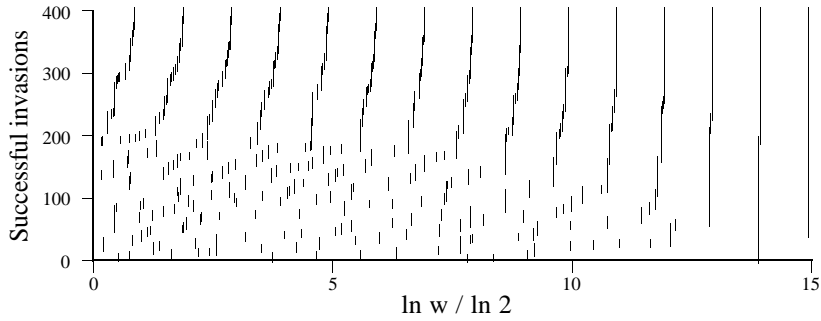


Fig. 6.1 The body mass (w) distribution among species that compete by interference competition as a function of the number of successful invasions into an initially empty locality. The invading species are drawn at random from an infinitely large species set that contains species with body masses that are uniformly distributed on a logarithmic scale within the range of $2^0 \leq w \leq 2^{14}$ weight units. For detail on the model, see the text.

unlikely that the rule will apply to sets with more than two species. This is easily seen by extending eqn 6.6 to sets with any higher number of species. (ii) If the intrinsic growth rates are not inversely related to body mass, but still approximately a power function, then the weight ratio can differ from two. (iii) If the body mass distributions of the competing species overlap, then some of the individuals of the small species can beat some of the individuals of the large species. This implies that $\gamma_{12} \rightarrow \gamma_{21}$ and that coexistence becomes more likely. As body mass overlap occurs only when the two species are not too different in size, the critical question is whether it can promote coexistence between species with a weight ratio that is smaller than two. As shown in the appendix to this chapter, this seems not to be the case if the coefficient of variation in body mass is smaller than 0.2 as it usually is, at least in mammals and birds. The conclusion then is that deviations in the weight ratio from two is most likely caused either by deviations in the intrinsic growth rates, by niche differentiation, or by a small number of invasions into the community.

6.4 Appendix

When the body mass distributions of the two competing species overlap we find that eqn 6.6 extends to

$$\begin{aligned} dN_1/dt &= N_1(r/w_1 - \gamma N_1/2 - \alpha \gamma N_2) \\ dN_2/dt &= N_2(r/w_2 - \gamma N_2/2 - (1 - \alpha)\gamma N_1) \end{aligned} \quad (6.9)$$

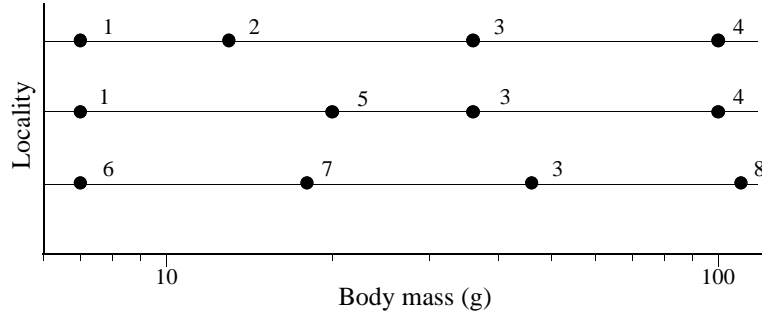


Fig. 6.2 The distribution of body masses among coexisting seed-eating desert rodents on three arbitrarily chosen localities. The scale is logarithmic to illustrate the equal spacing of weight ratios of approximately two. The numbers represent different species. Data from Bowers and Brown (1982).

where $\alpha \leq 0.5$ when $w_1 \geq w_2$. This system has the following equilibrium

$$\begin{aligned} N_1^* &= c(2r/w_1\gamma - 4\alpha r/w_2\gamma) \\ N_2^* &= c(2r/w_2\gamma - 4(1-\alpha)r/w_1\gamma) \end{aligned} \quad (6.10)$$

with $c = 1/[1 - 4\alpha(1 - \alpha)]$. For the equilibrium eqn 6.10 we find that coexistence occurs when $N_1^* > 0$ and $N_2^* > 0$, i.e., when

$$1 - w_1/2w_2 \leq \alpha \leq w_2/2w_1 \quad (6.11)$$

For eqn 6.9 we can define α as the probability that an average individual in species 1 has a body mass that is smaller than an individual chosen at random from species 2. Thus, when there is no overlap in the distributions we have that $\alpha = 0$ and that eqn 6.9 = eqn 6.6, whereas when the two distributions are identical and symmetrical we find that $\alpha = 1/2$, and that inter-specific interference does not differ from intra-specific interference. More generally, if the coefficient of variation in body mass (CV) is the same for the two species, we can define $\alpha = f(w_1, w_2, CV)$ where f is a function that depends on the body mass distributions. In Fig. 6.3 I have shown the case where f is defined from normal distributions, and here we have that $f < 1 - w_1/2w_2$ for $1 < w_1/w_2 < 2$ when $CV < \approx 0.2$. By eqn 6.11 this implies that species 2 is excluded by species 1. For normally distributed body masses we find also that coexistence can occur when, e.g., $1.9 \leq w_1/w_2 \leq 2$ and $CV = 0.3$, and when $1.5 \leq w_1/w_2 \leq 2$ and $CV = 0.5$. When, instead, $CV = 1$ we find that species 1 is excluded by species 2, and this is because $f > w_2/2w_1$ for $1 < w_1/w_2 < 2$.

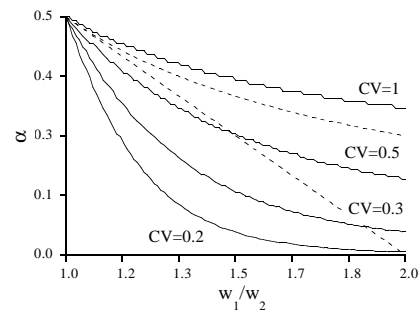


Fig. 6.3 The relationship between the α parameter and the body mass ratio (w_1/w_2) between two species 1 and 2. The area in between the two dashed lines defines the area where coexistence occurs, and the solid lines indicate the expected relationships when the body masses within each species are normally distributed and the coefficient of variation in the body mass of a species (CV) is respectively 0.2, 0.3, 0.5, and 1 in both species.