

## Part II

# Evolution by natural selection



## Chapter 7

# Basic relations

IN THE PRECEDING part we dealt only with some ecological implications of interference competition, and in the rest of the book we will focus mainly on the evolutionary consequences of intra-specific interference competition. But before we begin to analyse the evolutionary modulation of the phenotype, in this part, I will describe some of the basic principles underlying the analysis of theoretical evolutionary biology.

According to the Darwinian hypothesis the evolutionary changes in a trait are induced by natural selection defined by the variation in the rates by which the different variants of that trait increase or decrease in density. This means that evolution by natural selection is defined by the relations that link genetic variation in a trait to variation in the growth rate of that trait. It is the most elementary of these relations that I describe in this chapter where the link between a trait and the growth rate of that trait is partitioned into three subsequent sets of constraints.

Seen from the growth rate the first set of constraints is the demographic constraints that define the growth rate from the demographic traits that are represented by age- or stage-structured reproduction and survival. The second set of constraints is the intrinsic, or physiological and genomic, constraints that define the demographic traits from the amount of resource, or energy, that is available to the individual. And, finally, the third set of constraints is the extrinsic, or ecological, constraints that define the amount of resource available to the individual.

Together with genetic variation it is the constraints at these three levels that define natural selection and, thus also the evolutionary determinants of the organism. This means that the crucial point in the construction of an evolutionary theory is to identify exactly those constraints at these three levels that can explain the evolutionary transitions that have occurred in the phenotypes of natural organisms during their history on Earth. In this and

the following two chapters, I will not deal with the constraints that impose these transitions. Instead, I will describe only the most basic constraints at the three levels, the constraints that I use to construct a model organism and to define the different levels of selection that may operate on that organism.

## 7.1 Age-structured demography

As the fitness of a variant is given by its population growth rate the analysis of natural selection depends on our ability to calculate the Malthusian parameter from basic principles. In this section I will briefly describe the calculus of age-structured demography that, among other things, can be used to define the Malthusian parameter from age-structured reproduction and age-structured survival. This description is a summary of the most basic calculus in this field, and for other and more detailed reviews, you might examine Charlesworth (1994) and Bulmer (1994), or Caswell (1989a) and Van Groenendael et al. (1988) that deal also with stage structure.

In Chapter 2 on Malthusian increase we defined the Malthusian parameter from the demography in two special cases. The first case is the situation where the individuals are potentially immortal and where the instantaneous rates of reproduction ( $m$ ) and mortality ( $d$ ) are constants. In this case the Malthusian parameter is  $r = m - d$ . The second case is the instance where an individual replicates only once and then dies. If, in this latter case,  $R$  is the number of offspring produced per individual and  $p$  is the probability that an offspring will survive and reproduce, then net lifetime reproduction is  $R_0 = pR$ , and this  $R_0$  is also the discrete Malthusian parameter ( $\lambda$ ). However, most organisms replicate more than once and their rates of reproduction and survival also depend upon the age of the individuals. In this more general case the relation between the demographic traits and the Malthusian parameter is more complex as shown below.

In the presence of age structure it is convenient to describe the population by a vector

$$\mathbf{N} = \{N_0, N_1, N_2, \dots, N_T\} \quad (7.1)$$

where  $N_0$  is the number of individuals in age class zero and  $T$  is the lifespan, defined as the period from birth to death by senescence. Then, the population density is

$$N = \sum_{a=0}^T N_a \quad (7.2)$$

The age distribution is the vector of the proportion of the total population in each age class, and it is defined as

$$\mathbf{c} = \{c_0, c_1, c_2, \dots, c_T\} \quad (7.3)$$

where  $c_a = N_a/N$  and  $1 = \sum c_a$  with  $a$  indicating the age, i.e., with  $a \in \{0, 1, 2, \dots, T\}$ . When  $m_a$  is the number of offspring produced per individual in age class  $a$ , lifetime reproduction is

$$R = \sum_{a=0}^T m_a \quad (7.4)$$

When  $p_a$  is the probability to survive from age class  $a$  to age class  $a + 1$ , the probability to survive to the age  $a$  is

$$l_a = \prod_{i=0}^{a-1} p_i \quad (7.5)$$

with  $l_0 = 1$ . Net lifetime reproduction is then

$$R_0 = \sum_{a=0}^T l_a m_a \quad (7.6)$$

and from eqns 7.4 and 7.6 we can define the survival component in  $R_0$  by the survival scalar

$$p = R_0/R \quad (7.7)$$

We can then scale age-structured reproduction with respect to lifetime reproduction and write  $m_a = R\hat{m}_a$  with  $\hat{m}_a = m_a/R$ . Then, if age is scaled with respect to the lifespan, from eqns 7.6 and 7.7, the survival scalar  $p$  is

$$p = \sum_{a=0}^1 l_a \hat{m}_a \quad (7.8)$$

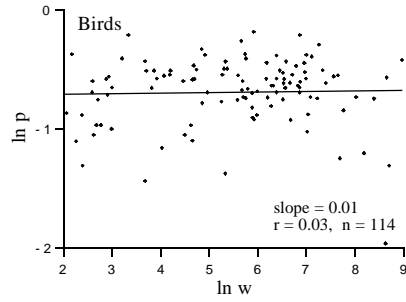
From this expression we notice that  $p$  is independent of both the absolute lifespan and absolute reproduction. This means that  $p$  is defined from the shape of the survival and the fecundity curves. In this sense we expect that  $p$  is invariant among organisms with a similar bauplan, a prediction that holds at least on the body mass axis within birds (Fig. 7.1).

From the equations defined above the population vector can be projected in time. In the case where the population vector is defined immediately posterior to the reproductive pulse the population projection is given as

$$N_{0,t+1} = \sum_{i=1}^T m_{i,t+1} N_{i,t+1} \quad (7.9)$$

with

$$N_{i,t+1} = p_{i-1,t} N_{i-1,t} \quad (7.10)$$



**Fig. 7.1** The survival scalar ( $p$ ) plotted against body mass ( $w$ ) on double logarithmic scale for 114 species of birds, with the line indicating the linear regression.  $p$  is estimated as annual adult survival raised to the power of the age of first reproduction. From Witting (1995).

For the hypothetical situation where age-structured reproduction and survival are constant most populations will converge toward a stable age distribution characterised by exponential growth in all age classes. This growth is described as

$$\begin{aligned} \mathbf{N}_t &= \mathbf{N}_0 e^{rt} \\ &= \{N_{0,0}e^{rt}, N_{1,0}e^{rt}, \dots, N_{T,0}e^{rt}\} \end{aligned} \quad (7.11)$$

where  $N_{a,0}$  denote the number of individuals in age class  $a$  at time zero. To calculate the stable age distribution at this limit of exponential growth we notice that at current time (0)

$$\begin{aligned} N_{a,0} &= l_a N_{0,-a} \\ N_{0,0} &= N_{0,-a} e^{ra} \end{aligned} \quad (7.12)$$

Then, from the bottom equation in eqn 7.12 we have that  $N_{0,-a} = N_{0,0} e^{-ra}$ , and if we insert this expression into the top equation we obtain

$$N_{a,0} = N_{0,0} l_a e^{-ra} \quad (7.13)$$

The sum of eqn 7.13 for all age classes will then give us the population abundance

$$N = \sum_{a=0}^T N_{0,0} l_a e^{-ra} \quad (7.14)$$

which we can divide into eqn 7.13 to obtain the proportion of the population in age class  $a$

$$c_a = \frac{l_a e^{-ra}}{\sum_{a=0}^T l_a e^{-ra}} \quad (7.15)$$

We can now turn to the question that is most important in an evolutionary context, and that is how to calculate the Malthusian parameter, i.e., fitness, from age-structured survival and reproduction. First, we notice that

$$N_0 = \sum_{a=0}^T m_a N_a \quad (7.16)$$

If into this equation we insert  $N_a$ , as defined by eqn 7.13, we obtain

$$N_0 = \sum_{a=0}^T N_0 l_a m_a e^{-ra} \quad (7.17)$$

which reduces to the Euler equation

$$1 = \sum_{a=0}^T l_a m_a e^{-ra} \quad (7.18)$$

from which fitness ( $r$ ) is defined by the age structure in survival and reproduction. Usually this equation cannot be solved directly for  $r$ , but this is of no practical importance since eqn 7.18 is easy to solve numerically as described by Press et al. (1986).

In relation to the calculation of the Malthusian parameter let us first check that eqn 7.18 defines  $r$  correctly for the two situations in Chapter 2. For the case where individuals are potentially immortal and instantaneous reproduction ( $m$ ) and mortality ( $d$ ) are constants, the survival probability declines exponentially with age, i.e.,  $l_a = e^{-da}$ , while reproduction remains constant, i.e.,  $m_a = m$ . Thus eqn 7.18 is equal to

$$\begin{aligned} 1 &= \int_{a=0}^{\infty} e^{-da} m e^{-ra} da & (7.19) \\ &= \frac{-m(e^{-(d+r)\infty} - e^{-(d+r)0})}{d+r} \\ &= \frac{m}{d+r} \end{aligned}$$

which means that eqn 7.18 correctly defines that  $r = m - d$ . For the case where the individuals reproduce only once just before they die, we have that  $m_T = R$ ,  $m_{a \neq T} = 0$  and  $p = l_T$ . Then, on a per lifespan time scale where  $T = 1$ , eqn 7.18 is given as

$$1 = p R e^{-r} \quad (7.20)$$

from which it follows that  $r = \ln(pR) = \ln(R_0) = \ln(\lambda)$  as already defined in Chapter 2. In other words, eqn 7.18 is valid for the two special cases that we used to define exponential and geometrical increase.

In the discrete case with non-overlapping generations the relation  $\lambda = R_0$  is valid. This is, however, generally not the case when the generations are overlapping as they are in most species. To see this consider a simple example where an individual reproduces twice; at the middle (age 1/2) and the end (age 1) of its life. Let  $l_0 = 1$ ,  $l_{1/2} = 0.5$ ,  $l_1 = 0.4$ , and  $m_0 = 0$ ,  $m_{1/2} = 2$ , and  $m_1 = 5$ . Then,  $R = 7$ ,  $R_0 = 3$  and from eqn 7.18

$$1 = e^{-r/2} + 2e^{-r} \quad (7.21)$$

which implies that  $r = \ln 4$  and this is larger than  $\ln R_0 = \ln 3$ . This difference arises because in estimating  $r = \ln \lambda$  from eqn 7.18 the estimated  $r$  and  $\lambda$  incorporates the overlap in generations which  $R_0$  does not.

From Chapter 3 we generally expect that natural populations will be situated at their population equilibria. In these cases we have that  $r = 0$ ,  $\lambda = 1$ , and that eqn 7.18 reduces to

$$1 = \sum_{a=0}^T l_a m_a \quad (7.22)$$

which is also the definition of  $R_0$  (eqn 7.6). Thus, at the population equilibrium the simple relation  $\lambda = R_0$  is valid for all populations. In other words, as we will focus on the plausible hypothesis that populations are in population dynamic equilibria, we can approximate the process of evolution through the fitness values at the immediate limit of the fitness of an average individual with  $\lambda = R_0 = pR = 1$ . Then, the relevant fitness values are defined by  $\ln(pR)$  so that when we analyse the evolution of a particular trait we need only define  $p$  and  $R$  as functionally determined by that trait.

## 7.2 Physiological constraints

In the classical theory it is generally assumed that natural selection is defined by the physiological constraints that link a trait to the demographic parameters  $p$  and  $R$ . This is, e.g., the case in Roff's (1982) hypothesis, where it is a proportional relationship between the reproductive rate and body mass that defines the selection pressure for a large body mass (Chapter 10), and in Lack's clutch size (Lack, 1947), where the rate of reproduction evolves from a trade-off between the reproductive rate and the survival rate (Chapter 12). Although these physiological relations and trade-offs are empirically confirmed we will find that they fail as the evolutionary constraints that can define natural selection, and this is because the relations have evolved by natural selection themselves. To illustrate that it is doubtful that the physiological relations play the primary role in defining natural

selection I would like to give an analogue between a physicist and an evolutionists before I deal more explicitly with the incorporation of physiological constraints into evolutionary models.

### 7.2.1 The physicist and the evolutionist

Consider first a physicist who wants to explain how stones originate from inert matter. The physicist walks along the beach and wonders why there are so many varieties of stone, and to answer the question he collects some and takes them into the laboratory. He wonders why the different stones have different shapes and different masses, and he gets the idea that it must be because each stone is created from its own intrinsic forces. To explore his idea he begins to perturb the shape of each stone by an extrinsic force, and when he relaxes the force he observes that each stone returns to its original shape. The physicist is happy because he believes that he has shown that each stone has its own equilibrium state created by the forces within the stone. To explain why the different stones differ in shape and mass he speculates on the diversity of stones and he reaches the conclusion that each stone is unique because it has its particular spot on the beach, and associated to each spot there exist a specific set of intrinsic forces. If these forces were not specific to each spot the stones would probably be either too big or too small to fill out their particular spots in the pattern of stones on the beach.

We probably all agree that although the physicist may have some interesting ideas he is sidetracked because stones are not created from intrinsic forces, instead they are shaped mainly by geological and other physical forces that exist extrinsic to the stones. The intrinsic forces are only the glue that maintains the shape that has once been created.

Consider now a present day evolutionist who wants to explain the evolutionary structuring of organic matter into species. The evolutionist walks along the beach and wonders why there are so many varieties of shorebirds, and to answer the question he collects some and brings them into the laboratory. He wonders why the different species have different rates of reproduction and different masses, and he gets the idea that it must be because each species has evolved from the physiological trade-offs within the individuals of that species. To explore his idea he begins to perturb the phenotype of each species. He forces some individuals to reproduce at an earlier age than normal, and he observes that such individuals get fewer offspring and that each offspring has a lower chance of survival than normal. He also transfers eggs from one nest to another, and he observes that the parents that have most surviving offspring the next year are the individuals with the original number of eggs. The evolutionist is happy because he believes that he has shown that each species has an equilibrium phenotype

that has evolved from the trade-offs that exist within the individuals of the species. To explain why the different shorebirds have different shapes and different masses he speculates on the diversity of species and he reaches the conclusion that each species is unique because it has its particular niche on the beach and associated to each niche there exist a specific set of trade-offs. If these trade-offs were not specific to each niche the species would probably be either too big or too small to fill out their particular niches in the community of shorebirds.

The analogue between the physicist and the evolutionist is striking. But why is the physicist sidetracked when the evolutionist is not? (At least not according to the classical theory). Might the only reason be that it is because we know better than the physicist, while most of us do not know better than the evolutionist? In the following chapters I hope that I will succeed in convincing you that there is no firm evidence that birds are evolutionarily created primarily from their intrinsic forces. Instead, it seems that they are evolutionarily shaped by the population dynamic forces that exist extrinsic to the birds, and that the intrinsic forces are only the glue that maintains the phenotypic and genotypic traits that has once been created by the process of natural selection.

### **7.2.2 Evolutionary constraints**

As the physiological constraints we can determine empirically often fail as evolutionary constraints the crucial question is how we can identify the physiological constraints that are so fixed that they set limits to the degree that natural selection can modify the organism. As we apparently cannot estimate the relevant constraints empirically there seems to be no simple solution to this question. One obvious approach is though to assume that the different traits are independent of one another unless there is a simple physical constraint that tells us that they are not. We are then faced with the challenge to explain the physiological constraints that we observe from the most limited set of assumptions, instead of using the physiological constraints as the assumptions upon which our evolutionary predictions depend. Below I have taken this approach when I define the physiological model that will be used throughout the rest of the book.

To make this model mathematically simple I will define the physiology in terms of energetics, where the various traits are defined in relation to units of resource, or units of energy, where energy is defined as the energy that organisms can obtain by metabolising the resource that they consume. According to this we may think of the body mass as being measured in joule or units of resource, or simply in grams if we assume that there is a linear transformation between these three units of measurement.

As it is possible to imagine almost any functional relationship between

survival and any trait, as a first approximation, it seems to be fair to assume that the survival scalar  $p$  is physiologically independent of all other traits. By eqn 7.8 and Fig. 7.1 we have already seen that this assumption tends to hold on the body mass axis.

If we make the simplifying assumption that reproduction is independent of age it is easy to see that lifetime reproduction is

$$R(\epsilon) = \epsilon T/q \quad (7.23)$$

where  $\epsilon$  is the amount of energy allocated to reproduction per unit time,  $T$  is lifespan, and  $q$  the energetic quality of an offspring, i.e., the energy used on each offspring. The inverse relationship (trade-off) in eqn 7.23 between lifetime reproduction ( $R$ ) and energetic quality ( $q$ ) exists because energy ( $\epsilon$ ) can be used only once. By eqn 7.23 we have assumed that it is only the reproductive rate that is phenotypically plastic in the sense that it varies with the amount of energy allocated to reproduction.

If we turn to the definition of energetic quality ( $q$ ), then in each offspring the parents will have to invest an energetic quantity that equals the energy in the body mass of the offspring added the energy metabolised by the offspring. The amount of energy that is metabolised by the offspring is likely to be proportional to the metabolic rate per unit body mass ( $B$ ) times the body mass ( $w$ ) times the length of the rearing period, which is expected to be proportional to lifespan ( $T$ ) (e.g., Calder, 1984). Hence,  $q = w(1 + cTB)$ , where  $c$  is a positive constant. To simplify we may assume that  $c = 1$  and approximate energetic quality as  $q = wTB$ . Also, as we will not address any question in relation to the physiological growth of the individual it is reasonable to assume that both  $w$  and  $B$  are age independent. Lifetime reproduction, as it is defined by eqn 7.23, is then

$$R = \epsilon/wB \quad (7.24)$$

and, hence, there is a trade-off between Fisherian fitness and both the body mass and the metabolic rate. Given the present assumptions it follows that the fraction of net assimilated energy that is allocated to reproduction is invariant among organisms with a similar bauplan. To see this we recall that the body mass is assumed to be constant from birth to death. Hence, net assimilated energy must be allocated to either reproduction or to metabolism, i.e.,  $\epsilon_A = \epsilon + \epsilon_B$  where  $\epsilon_A$  is net assimilated energy,  $\epsilon_B$  is metabolised energy, and  $\epsilon$  is energy allocated to reproduction as previously defined. Furthermore, since a population with stable, or damped, population dynamics will be situated at the population equilibrium an average individual will replace itself by a single individual, i.e.,  $R_0 = p\epsilon/wB = 1$  and, consequently,  $\epsilon = wB/p$ . Then, because the energy that is metabolised

is  $\epsilon_B = wB$  we have the following allocation ratio

$$\epsilon/\epsilon_B = (wB/p)/(wB) = 1/p \quad (7.25)$$

where  $p$  is invariant among organisms with a similar bauplan (eqn 7.8). Then, as  $\epsilon_A = \epsilon + \epsilon_B$  we have that  $\epsilon_A/\epsilon = 1 + \epsilon_B/\epsilon$  and, thus, that  $\epsilon = c\epsilon_A$  where  $c = \epsilon/(\epsilon + \epsilon_B)$  is a positive constant. We can then simplify the notation by setting  $c = 1$  so that  $\epsilon$  denotes both net assimilation and the amount of energy allocated to reproduction.

Another constraint that seems to hold on an evolutionary scale was noticed by Pearl in 1928. He saw that the lifespan of an organism is inversely related to the metabolic rate per unit mass of that organism; just like the longevity of a machine is inversely related to the rate by which the machine is used. This principle is illustrated by the fact that the lifespan of, e.g., homiotherms is proportional to the positive 1/4 power of body mass while the metabolic rate per unit mass is proportional to the negative 1/4 power of body mass (e.g., Calder, 1984; Chapter 13). We can formulate this relation between the lifespan ( $T$ ) and the metabolic rate per unit mass ( $B$ ) as

$$T = \omega/B \quad (7.26)$$

where  $\omega$  is a positive constant of senescence that can be modified by selection. Although Pearl's "rate-of-living" has been questioned (Maynard Smith, 1958, 1963; Clarke and Maynard Smith, 1961a,b; reviewed Rose, 1991) it seems to hold in relation to the questions I address.

### 7.3 A few ecological constraints

In relation to the evolutionary theory that I propose in this book it is the extrinsic, or ecological, constraints that are essential for the functional relation between a trait and the demographic parameters  $p$  and  $R$ . These constraints are rather complex with the most essential factors being the density dependence in the number of competitive interactions per individual and the mode by which these interactions partition the resource among individuals. The most essential of these constraints are defined in detail in Section 10.2 that deals mainly with the evolution of body mass. In this section I give only a brief description of the constraints associated with the amount of energy that is consumed, assimilated, and allocated to reproduction.

Let me assume that the energy that is consumed is assimilated and allocated to reproduction without delay so that the energy allocated to reproduction is

$$\epsilon = \kappa E \quad (7.27)$$

where  $E$  is the density of the resource and  $\kappa$  is the realised foraging efficiency of an individual per unit resource density. The dynamics in the resource density is then

$$dE/dt = E(r_e - \gamma_e E - \kappa N) \quad (7.28)$$

where  $r_e$  is the maximal rate of production and  $\gamma_e$  the density regulation. The resource has its maximum equilibrium density ( $E_m$ ) at the limit  $N^* = 0$  where the consumer is absent, the resource is unexploited and given as

$$E_m = r_e/\gamma_e \quad (7.29)$$

When instead the consumer is present, i.e., when  $N^* > 0$ , then the resource equilibrium is

$$\begin{aligned} E^* &= (r_e - \kappa N^*)/\gamma_e \\ &= E_m - \kappa N^*/\gamma_e \end{aligned} \quad (7.30)$$

At the limit  $N^* \approx 0$  the rate of resource assimilation per individual is at its maximum

$$\epsilon_m = \kappa E_m \quad (7.31)$$

and from this expression we find that maximal lifetime reproduction is

$$R_m = \kappa E_m/wB \quad (7.32)$$

More generally, lifetime reproduction is  $R = \kappa E/wB$ , and from this expression and eqn 7.32 we find that

$$R = R_m E/E_m \quad (7.33)$$

From eqns 7.31, 7.32, and  $R_0 = pR$  maximal net lifetime reproduction is

$$R_{0,m} = p\epsilon_m/wB \quad (7.34)$$

From eqn 7.34 it is apparent that the population will persist when  $R_{0,m} \geq 1$  and that it will become extinct when  $R_{0,m} < 1$ . From this extinction criterion we see that the population will become extinct when the survival rate is too low, i.e., when  $p < wB/\epsilon_m$ , and/or when the energetic quality ( $q = wB$ ) is too high compared with the maximal amount of resource that the individuals can consume, i.e., the population will become extinct when  $q > p\epsilon_m$ .

The realised foraging efficiency ( $\kappa$ ) can be partitioned into three multiplicative components. The first component is the maximum value of  $\kappa$ , and this value is the exploitation efficiency ( $\alpha$ ) that defines consumption when competition is purely exploitative and the resource is exploited evenly by

the population. The second component ( $0 \leq g \leq 1$ ) is interference regulation, and this is the downward regulation of  $\alpha$  by the density dependent competitive interactions that exist among the foraging individuals. The third component ( $0 \leq f \leq 1$ ) is self-inhibition, and this is the downward regulation of  $\alpha$  that occurs when an individual exploits its own fraction of the resource more heavily than the population exploits the total resource. The energy consumed by an individual is then

$$\epsilon = \alpha f g E \tag{7.35}$$

The self-inhibition component ( $f$ ) is described in Chapter 13, where together with the interference component ( $g$ ) it defines the constraints used to explain the evolution of the exponents of the body mass allometries. In the other chapters I will generally disregard  $f$  and focus on the evolutionary transitions that occur as a consequence of the presence versus absence of the interference component ( $g$ ).

## Chapter 8

# Fitness and selection

THE STANDARD CONCEPT of fitness dates back to Fisher (1930) who defined it as the intrinsic Malthusian parameter that is given as the solution to the Euler equation (7.18). In his definition Fisher disregarded the effects of competitive interactions and treated the fitness of an individual as an intrinsic property of its genotype. In this way the fitness of an individual was seen as being independent of the genotypes of the other individuals in the population. Under genetic variation this definition leads to frequency-independent selection, the most widespread hypothesis of natural selection forming the basis of the classical theory of evolution (see Roff, 1992; Stearns, 1992; Charnov, 1993; Charlesworth, 1994; Bulmer, 1994).

The alternative to frequency-independent selection is frequency-dependent selection where the fitness of a genotype depends also upon the genotypes of the other individuals in the population. In the traditional treatment of natural selection it is mainly in two special cases that frequency-dependent selection is treated as being essential. The first case is the classical theory on the evolution of sex ratios that was introduced also by Fisher in 1930. The second case is game theory and the approach of evolutionarily stable strategies that were introduced by Maynard Smith and Price (1973) in order to describe the evolution of discrete types of animal behaviour. In these two cases it is only game theory that operates with competitive interactions while the frequency dependence in the traditional sex ratio theory arises from the transmission of genes from parents to offspring. As the two types of selection that disregard competitive interactions were both introduced by Fisher I use the term Fisherian selection for those two modes of selection.

The theory that I propose in this book is based on the frequency dependent selection that arises from the density dependent competitive interactions existing among the individuals within a population. Although

competitive interactions are essential for phenotypic evolution in sessile organisms (e.g., Mirmirani and Oster, 1978; Mäkela, 1985; King, 1990; Vincent and Vincent, 1996), I will generally focus only on mobile organisms where evolution by competitive interactions has been studied by, e.g., Lande (1976), Parker (1979, 1983), Slatkin (1979), Haigh and Rose (1980), Maynard Smith and Brown (1986), Abrams and Matsuda (1994), and Day and Taylor (1996). The major differences between these studies and my study are that the earlier studies generally are based on the unrealistic assumption that the number of competitive interactions per individual is density independent, and that they tend to assume also a “classical phenotype” where the evolutionary predictions depend on fundamental traits that are evolutionarily unexplained. In the earlier studies there has also been only sporadic interest in developing a general theory of phenotypic evolution, while I present a theory that covers the evolution of many of the major phenotypic patterns observed among mobile organisms.

In this chapter I describe some of the basic differences between different types of selection. In the first section I give a brief description of the levels at which selection can operate. Then I describe some basic properties of frequency-independent Fisherian selection, and then I turn to frequency dependent selection under the action of density dependent competitive interactions.

## 8.1 Selection at different levels

When we describe selection from the differences between the growth rates of individuals we deal with individual selection, and when we describe selection from the differences between the growth rates of genes we deal with genomic selection.

There are only a few instances where the distinction, or lack of distinction, between genomic and individual selection is a priori clear. One case is when individuals reproduce asexually. Then, genomic selection is equivalent to individual selection because there is no transfer of genes among individuals. Another case is where a species contains both a sexual and an asexual variant. Then, because there is no transfer of genes between the sexual and the asexual variant, we find that genomic selection will operate within the sexual variant, while it is individual selection that will operate between the sexual and the asexual variant. In this sense the two levels of selection will operate independently of one another.

Apart from these two examples it is generally not easy to distinguish individual selection from genomic selection. This problem will be addressed in detail when, in the Chapters 19 to 22, we turn to the traits connected with sexual reproduction. In this section let us consider only what happens in the

special case with independence between genomic and individual selection. In this case it is individual selection that will dominate over genomic selection, and this is because it is the variant with the highest growth rate that will out-grow the other variant. To see this let  $r_a$  and  $r_s$  be respectively the growth rates of the asexual and the sexual variant. Then, from eqn 2.2,

$$\begin{aligned} N_{a,t} &= N_{a,0}e^{r_a t} \\ N_{s,t} &= N_{s,0}e^{r_s t} \end{aligned} \quad (8.1)$$

and if  $N_{a,0} = N_{s,0}$  we have that

$$N_{a,t}/N_{s,t} = e^{(r_a - r_s)t} \quad (8.2)$$

From this equation we find that  $N_a/N_s \rightarrow \infty$  for  $t \rightarrow \infty$  when  $r_a > r_b$ , whereas  $N_a/N_s \rightarrow 0$  for  $t \rightarrow \infty$  when  $r_a < r_b$ . In other words, it is the variant with the highest growth rate that evolves to fixation.

Kin selection is a special type of genomic selection, described especially by Hamilton (1964, 1972). This form of selection is special because it operates indirectly through the inclusive fitness of related individuals. Under the traditional form of genomic selection fitness is direct in the sense that it is the sexual male or female that copy their own genes to the offspring. Under the operation of kin selection fitness is indirect, or inclusive, in the sense that the fitness of a gene in an individual depends upon the probability by which a related individual copies that gene to its offspring. In this book we will deal with kin selection mainly in Chapter 22 that deals with the evolution of eusociality.

It has been argued, especially by Wynne-Edwards (1962, 1986, 1993), that natural selection occurs mainly at the level of groups instead of at the level of individuals and/or genes. This notion is interesting to the degree that group selection differs from individual selection, and according to Wynne-Edwards this is the case. He argues that group selection is diametrically opposite to individual selection because it operates through group extinctions caused by over-exploitation. This implies that natural selection is hypothesised to favour individuals that depress their individual and/or genomic fitness so as to keep the group from over-exploiting the resource. Today, this idea is largely abandoned and this is because there is no evidence that some groups go extinct due to famine, while the individuals in the remaining groups restrain from reproduction. In Chapters 18 to 22 I will allow individuals to form groups and, therefore, we will necessarily deal with selection at the level of groups. This type of selection though has no resemblance with the ‘‘original’’ type of group selection, instead it has a close resemblance to individual selection, and this is because there is no conflicts between the modes of selection at the two levels. Due to this

resemblance I will not distinguish between individual and group selection in these cases, but instead continue to use the term individual selection although the individuals aggregate into groups, and then reserve the term group selection for the type of selection proposed by Wynne-Edwards.

## 8.2 Selection in the classical theory

In the classical theory it is customary to say that natural selection leads to an evolutionary increase in the fitness of a population or species. This is because the classical theory is based on frequency-independent selection where the relative growth rate of an individual is independent of the other individuals in the population. Hereby the fitness of an individual is defined as a genotypic trait of that individual, a trait that is expressed as the phenotypic trait known as the intrinsic growth rate, or the intrinsic Malthusian parameter ( $r$ ). The fitness of a population is then the population average to the intrinsic growth rate and the increase in fitness is the increase in this average value.

The idea of an increase in fitness is so essential to the classical theory that it was formulated into the fundamental theorem of natural selection by Fisher himself. The fundamental theorem states that “The rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time.” That is

$$dr/dt = \sigma_r^2 \quad (8.3)$$

where  $\sigma_r^2$  is the genetic variance in  $r$ . This means that selection is hypothesised to increase the population growth rate whenever there is genetic variation and a stable environment. When, instead, the environment is variable due to density dependence the fundamental theorem predicts that the average growth rate will remain stable at approximately zero while selection generates an increase in the population equilibrium ( $K$ ) through an increase in the maximal growth rate ( $r_m$ ) and a deterioration of the environment.

Due to the work of MacArthur (1962) and Roughgarden (1971) the fundamental theorem became partitioned into  $r$  and  $K$  selection that reflect respectively selection in the absence of density dependence and selection at the population equilibrium. This concept developed into two independent branches of theory, the verbal (e.g., Pianka, 1970; Stearns 1976, 1977; Parry, 1981) and the mathematical theory of  $r$  and  $K$  selection (e.g., Anderson, 1971; Roughgarden, 1971; Clarke, 1972). At first it seemed that the verbal version explained many patterns of phenotypic evolution, but these predictions were generally not confirmed by the mathematical version that made only very restricted predictions. Today, it is clear that the concept of  $r$  and  $K$  selection was a mistake, and this is because of the mathematical

fact that the two modes of selection represent the same type of selection (Caswell, 1989b).

The classical form of selection does not only operate on fitness, it operates also on almost all traits, e.g., on the trait  $y$ . With fitness being defined as an absolute quantity of the genotype the relationship between fitness and the trait  $y$  can be defined as a one-dimensional function

$$r = f(y) \quad (8.4)$$

This relation between fitness and a trait was named the fitness profile by Robertson in 1955. If we differentiate the fitness profile with respect to the trait in question we obtain the selection gradient

$$\partial r / \partial y = f'(y) \quad (8.5)$$

where  $f'$  is the partial derivative of  $f$  with respect to  $y$ . Then, if there is one or several solution/s to  $\partial r / \partial y = 0$  for which the second derivative of  $f$  with respect to  $y$  is negative, then the fitness profile has one to several optima that are known as the evolutionary equilibria. If, in this case, the fitness profile has no minima there is only one evolutionary equilibrium and this equilibrium is the global attractor toward which all evolutionary trajectories converge.

If we are interested in the rate of evolution in the average trait value it was derived by Price (1970) and Emlen (1970), and given as the selection gradient multiplied by the genetic variance in that trait ( $\sigma_y^2$ ), i.e., given as

$$dy/dt = f'(y)\sigma_y^2 \quad (8.6)$$

In a slightly different version from Robertson (1968) this theorem is known as the secondary theorem of natural selection. Notice here that when the trait in question is the intrinsic Malthusian parameter ( $y = r$ ), then the secondary theorem (eqn 8.6) reduces to the fundamental theorem (eqn 8.3) because  $\partial r / \partial r = 1$ .

### 8.3 Selection by density dependent competitive interactions

Let us now turn to a different form of selection that is defined by the density dependent competitive interactions that exist among the individuals in a population. In essence this type of selection promotes the evolution of competitive quality that includes traits like body mass, metabolic rate, complex behavioural interactions, and group formation. These traits represent competitive quality in the sense that they can be used to win competitive

interactions so that the winner can prevent that the other competing individuals have access to the resource. In order to win, the large individual will dominate the small individual, the individual that metabolises most energy into the encounter will dominate the individual that metabolises less, the large group will dominate the small group, and the “clever” will outsmart the “stupid”.

The components of competitive quality are more or less equivalent to the components of energetic quality, defined as  $q$  in eqn 7.23. This means that there is a physiological and evolutionarily fixed trade-off between numerical reproduction and competitive quality. In other words, there is a trade-off between the classical definition of fitness and competitive quality, and it is therefore that the classical theory generally fails to explain the evolution of competitive quality. However, when fitness is defined as the extrinsic, instead of the intrinsic, Malthusian parameter the intrinsic trade-off is counterbalanced by an ecological, or extrinsic, gradient where it is the individuals with the largest competitive quality that have access to the largest amount of resource. Thus, dependent upon the ecological conditions, there might be a positive relation between fitness and competitive quality despite the trade-off that exists in the classical framework.

Due to the counteraction between the intrinsic trade-off and the extrinsic access to resource there must be a certain level of interference in the population before the traits of competitive quality can evolve by selection. This is because the fitness gained through interference competition must be equal to or larger than the cost associated with the competitive traits. For a variant that has a competitive quality larger than the average quality in the population, the fitness gained through interference competition is proportional to the number of competitive encounters per individual per unit time. This is because the gain relative to an average individual is proportional to the number of encounters won. If you win 75% of your encounters, but there are no encounters you will win 0 encounters just like an average individual and your increased competitive quality is wasted. But, if there are 100 encounters per day you will win 75 encounters per day, and your gain relative to an average individual that wins only 50 encounters per day may be large enough to out-balance the cost of your increased competitive quality. In other words, the relative fitness of a variant is no longer constant. On the contrary it is highly relative varying with the average quality in the population and the level of intra-population interference. The level of interference is, among other things, determined by the population density, and this density is positively related to the average rate of reproduction, which is negatively related to the average quality in the population. Thus, when the average quality is low we find that the density and the level of interference is high and that it pays to be larger than the average. When,

instead, the average quality is high we find that the density and the level of interference is low and that it pays to be smaller than the average. In between there is an evolutionary equilibrium where the gain from encounters won is balanced against the quality it takes to win.

Due to this relativity of the extrinsic Malthusian parameter we find that the fitness of an individual is no more a property of its own genotype than it is a property of the genotypes in the other individuals in the population. This means that the growth rate of a gene cannot be treated as a character of that gene, not even when the environment extrinsic to the population is constant. Hence, it makes no sense to think of fitness as a genotypic trait that can evolve by natural selection and, therefore, it is meaningless to talk about evolutionary changes in fitness. This implies that the fundamental theorem of natural selection does not apply to the framework of Malthusian relativity. Notice here that the classical definition of fitness is still a genotypic trait, i.e., the trait that is known as the intrinsic Malthusian parameter, but this trait is no longer fitness.

When the fitness of a variant is defined from the relation between the trait values of that variant and the trait values of the other individuals in the population there is no simple one-dimensional function between the fitness of a variant and the trait values of that variant. One way to approximate the true fitness function is to focus on variation around the average trait value in the population. Then, the fitness of the  $i$ th variant can be defined as a two-dimensional function given by the trait value of that variant ( $y_i$ ) and the average trait value in the population ( $y$ ), i.e., given as

$$x_i = f(y_i, y) \quad (8.7)$$

where  $x$  is the extrinsic Malthusian parameter. Due to the two-dimensionality of this fitness profile it is crucial to notice that selection is caused by intra-population variation in the fitness values. This implies that we differentiate  $x_i$  with respect to  $y_i$  when we determine the selection gradient:

$$\partial x_i / \partial y_i = f'(y_i, y) \quad (8.8)$$

where  $f'$  is the derivative of  $f$  with respect to  $y_i$ . Then, since we generally are interested in the effects of selection on the average variant we let  $y_i \rightarrow y$  in order to obtain the selection gradient at the limit of the average variant

$$\lim_{y_i \rightarrow y} \partial x_i / \partial y_i = f'(y, y) \quad (8.9)$$

The evolutionary equilibrium is then the solution/s to  $\lim_{y_i \rightarrow y} \partial x_i / \partial y_i = 0$  that has/have a negative derivative in the dimension of the average trait, i.e.,  $\partial f'(y, y) / \partial y < 0$ .

The two-dimensional fitness profile in eqn 8.7 can be projected down into a one-dimensional fitness profile when the average trait value is constant. These one-dimensional projections of the fitness profile are always local because they describe only the within-population variation in fitness. Instead of this local function we might be interested in a global one-dimensional relationship that holds across populations so that it can describe the relative fitness of the average variant as a function of the trait value of that variant. To obtain this relation we can calculate the selection profile, and this is done by integrating the selection gradient at the limit of the average variant with respect to the trait value of that variant. That is to say that we need to integrate eqn 8.9 with respect to  $y$ :

$$\begin{aligned} x &= \int \left( \lim_{y_i \rightarrow y} \partial x_i / \partial y_i \right) \partial y \\ &= F(y) + k \end{aligned} \quad (8.10)$$

where  $k$  is an arbitrary constant. For each evolutionary equilibrium the selection profile will have an optimum. When there is only one optimum the evolutionary process will proceed toward that optimum, and when there are several optima it is initial conditions that will determine the optimum that the evolutionary process will proceed toward.

The distinction between the fitness and the selection profile is crucial in Malthusian relativity where the one-dimensional selection profile generally has a different shape than the one-dimensional projection of the fitness profile. This distinction is irrelevant in the classical theory where the shape of the two profiles is identical due to the one-dimensionality of the fitness profile.

If we want to predict the rate of evolutionary change in a trait we proceed as in the classical theory, i.e., we multiply the selection gradient at the limit of the average variant with the genetic variance in the trait. For example, if the trait in question is the intrinsic Malthusian parameter the rate of evolution is

$$dr/dt = \sigma_r^2 \lim_{r_i \rightarrow r} \partial x_i / \partial r_i \quad (8.11)$$

This means that Fisherian fitness will increase only when the correlation between the extrinsic and the intrinsic Malthusian parameter is positive. As we will see in Chapter 14 it turns out that the correlation between  $x$  and  $r$  is negative in a stable environment and, thus, Fisherian fitness will continue to decline, and not increase as it is hypothesised by Fisher's fundamental theorem.

## Chapter 9

# Historical versus deterministic evolution

WHEN DARWIN introduced the concept of evolution by natural selection he disregarded determinism focusing instead on the probabilistic events of chance and history (Mayr, 1991). This allowed him to propose the general mechanism of natural selection, but it also implied that he generally did not identify universal laws of selection. In consequence, Darwin did not establish a theory that made explicit predictions on the evolution of life-history traits in natural organisms. Instead, he proposed a concept, or a working hypothesis, which suggested that evolution in natural species is driven by natural selection that can be understood only in the light of past historical events. This probabilistic thinking was completely new to the scientists at the time who were strict determinists believing that exact predictions were the only way to test the validity of a theory. According to Mayr (1991) Darwin's approach was even so alien that Herschel referred to natural selection as the theory of the "higgledy-piggledy" (F. Darwin, 1888, 2:240).

Since Herschel, leading evolutionists have emphasised that this "higgledy-piggledy" is something special to evolutionary biology and which separates that field from the other disciplines of the natural sciences. At the same time the idea of a deterministic and directional form of evolution that is driven by universal laws has remained an almost forbidden concept. For example, Mayr (1988:20:105) defines natural selection as "a strictly *a posterior* process ... not controlled by any law". Maynard Smith and Szathmáry (1995:4) concludes that "On the theoretical side, there is no reason why evolution by natural selection should lead to an increase in complexity". And Williams (1992:3) writes that the "term evolution in its original sense

of an unfolding or development, analogous to the development of an individual animal, is misleading (Salthe, 1989).” “As S. J. Gould (1989:48) forcefully expressed it, if we could rewind the tape of evolutionary history to the remote past and play it again, it would turn out entirely different.”

Empirically we know that this classical hypothesis of historicity and non-directional evolution holds at least to the degree that all organisms do not proceed along a single evolutionary trajectory. Depending on the trait in question there is a tremendous amount of variation among natural species, and the evolutionary process is better described as a tree than as a single lineage. However, this observation does not imply that natural selection is not driven by universal laws, nor does it imply that there is not an overall direction to the evolutionary process. Below all the variation, evolution in a stable environment might be inherently directional and this might explain the arrow so apparent to the evolutionary process that has occurred on Earth. According to this arrow, organisms tend to become larger and more complex in the sense that multicellular organisms evolved from unicellular organisms, that sexual reproduction evolved from asexual reproduction, and that a diploid genome evolved from a haploid genome.

As evidence does not give us a clear answer to the degree that the classical propositions on non-directional evolution hold we may turn to theoretical considerations instead. As scientists, most of us believe that there is a natural explanation to all the phenomena that we observe and that these are made up by causal relations and initial conditions. This hypothesis implies that there is no such thing as a natural process that is truly stochastic. Instead, what we treat as random in our description of nature is itself determined by causal relations, and it is only because our conception of the natural world is incomplete that we lump these processes into random variables. Hence, given initial conditions, we expect that the trajectory is fixed, even in the instance where the trajectory is completely unpredictable because it is inherently chaotic. This suggests that Gould’s hypothesis holds only in a very restricted version, where it is only when the rewinding of the historical processes is imperfect that we expect the evolutionary trajectories will turn out to be different.

The result that the evolutionary trajectory is fixed for fixed initial conditions does not answer the question whether the apparent arrow to the evolutionary process on Earth is caused by a directional form of selection. The apparent arrow on Earth might only belong to our particular trajectory, being different for other initial conditions. Evidently, there is no practical way to test this directly. So in order to answer the question, whether the arrow on Earth is given by natural selection or not, we may turn to theoretical considerations and try to determine the degree to which the evolutionary trajectories can be explained by general laws of selection, versus the de-

gree to which they depend upon historical events caused either by initial conditions or by the physical laws that lie outside the domain of natural selection. If it is these latter conditions and laws that are essential for our understanding of the biological world, then it is true that the process of evolution by natural selection is non-directional and driven mainly by chance. If, instead, the major evolutionary trajectories are explained by general laws of selection, then we may conclude that the process of evolution by natural selection is inherently directional, or in other words, deterministic.

In this chapter I give a formal description of the types of selection that can be used to distinguish the classical hypothesis of historical and non-directional evolution from the proposed hypothesis of deterministic and directional evolution. As Darwin's introduction of non-directional evolution was associated with a rejection of Lamarck's notion of directional evolution I will initiate my description by comparing the ideas of these two great men.

## 9.1 Lamarck and Darwin

With the publication of his *Philosophie zoologique* in 1809 Lamarck was the first scientist to develop a consistent theory of evolution that could replace the view of a static nature based on the two concepts of *scala naturae* and plenitude. The concept of *scala naturae*, or the great chain of being, goes back to Plato, and it is the idea that organic beings were created on a continuous, or linear, scale from inert matter through plants to lower and higher animals and, finally, to Man and continuing through angels to God. The idea of plenitude was an additional concept, which postulated that the *scala naturae* was completely represented in the way that everything that is possible actually exists.

The essence of Lamarck's theory of evolution is that he accepted the hierarchical arrangement of living beings but rejected creationism proposing instead that the lower organisms arose by spontaneous generations and, then they evolved upward along the great chain of being reaching ever higher levels of perfection. In fact, this is not completely true because Lamarck actually accepted the existence of a few different chains of beings, but these chains were seen as being independent of one another in the sense that each chain had its own origin and its own route toward perfection.

According to Lamarck, all present day organisms belonged to these few chains, being scattered along their linear dimensions with some variation induced by special adaptations to an ever changing environment. The evolutionary process toward perfection was then driven by an inherent tendency that all organisms have to evolve along the axis of their chain, toward perfection. This tendency was hypothesised to be so strong that the present day species that belong to the same chain differed from one another mainly

by the degree to which they had evolved along their axis of perfection. In consequence, the present-day species that belong to the same chain are different from one another mainly because their phylogenetic lineages originated from inert matter at different times during the history of Earth. As it has been expressed by Sober (1993), present-day human beings are not related to present-day earthworms, instead they descended from earthworms that lived long ago.

Where Lamarck believed that each species belongs to its own phylogenetic lineage and that new phylogenetic lineages continue to originate from inert matter, Darwin believed the transition from inert matter to life took place only once and, then, the phylogenetic lineage diversified into a tree containing all present-day species. The evidence that Darwin presented clearly suggested that each species of today belongs to its own little branch on a single, or a few, phylogenetic tree/s, instead of being arranged along a few axes of perfection. This result was incompatible with Lamarck's theory, and this is because a continuous generation of lower species followed by directional modification simply does not allow for the generation of a single, or a few, phylogenetic tree/s. Instead, a common tree is what we expect from the process of natural selection proposed by Darwin.

## 9.2 Historicity versus determinism

Although the transition from Lamarck's mechanism of an intrinsic drive toward perfection, to Darwin's mechanism of natural selection, allowed for a transition from independent phylogenetic lineages to a single phylogenetic tree, it did by no means in itself imply also a transition from directional to non-directional, or historical, evolution. This latter transition is not a consequence of natural selection per se, but instead of the particular treatment of this process by Darwin, and nearly all subsequent authors, which focused primarily on the probabilistic events of chance instead of identifying the universal laws that in a stable environment underlies also the process of natural selection. In consequence, there is no conflict between a directional, or deterministic, theory of evolution and a single phylogenetic tree, at least not as long as evolution is driven by natural selection. This is because as long as it is only the selection pressure in a stable environment that selects for directional changes we find that speciation and the diversification among species can easily be driven by population specific environmental conditions. Variation in these conditions may, among other things, arise from geographical variation and interactions among species. It is this degree of determinism that underlies the theory of Malthusian relativity that I propose in this book.

Determinism at this level suggests that selection within a phylogenetic

lineage is directional, but it does not suggest that different lineages that experience the same environmental conditions evolve along exactly the same evolutionary trajectory. Even in a stable environment natural selection will inevitably be historical in the restricted sense where it depends on initial conditions and upon the historical generation of genetic variation by mutation. Hence, different lineages will be characterised by different phylogenetic histories and, they will thus contain different phylogenetic constraints that, at least to some extent, will influence the future direction of the evolutionary trajectory. However, as there generally is plenty of genetic variation, we also expect that there is a major long-term trajectory determined mainly by natural selection, and not by genetic, developmental, or phylogenetic constraints. It is at this level, above local constraints, that the directionality of Malthusian relativity differs from the non-directionality of the classical hypothesis of evolution by natural selection.

In the classical theory of evolution the historical events are not only restricted to environmental, genetic, and phylogenetic constraints. Instead, they include also the fundamental traits that define the selection pressure for the evolution of the derived traits. The fundamental traits represent historical events in the sense that they are given by assumption, and not by a type of selection that is included explicitly in the classical theory. This is in contrast to a deterministic theory of evolution by natural selection that contains no fundamental traits and, therefore, contains historical events only at the environmental, genetic, and phylogenetic level. Below, I will specify these differences in a more detailed form.

### 9.2.1 A mathematical distinction

Let us consider, in mathematical terms, how we can distinguish the classical hypothesis of historical and non-directional evolution from the proposed hypothesis of deterministic and directional evolution. Let us assume that the environmental, genetic, and phylogenetic constraints are constant, and let us first focus on the classical hypothesis of non-directional evolution. Then, let  $\mathbf{P}$  be the complete set of phenotypic traits that we consider and let these traits be partitioned into the fundamental,  $\mathbf{F}$ , and the derived,  $\mathbf{D}$ , traits, so that  $\mathbf{P} = \mathbf{F} \cup \mathbf{D}$ . The Fisherian fitness profile on the derived trait  $D_i \in \mathbf{D}$  is then

$$r = g_i(\mathbf{F}_i, D_i) \quad (9.1)$$

where  $\mathbf{F}_i \subset \mathbf{F}$  are the fundamental traits that define the selection pressure on the derived trait  $D_i$ ,  $\mathbf{F} \setminus \mathbf{F}_i$  are the fundamental traits that do not influence the evolutionary setting of  $D_i$ , and the function  $g_i$  represents the constraints that link the different traits together and defines the selection

pressure on  $D_i$ . The selection gradient on the derived trait  $D_i$  is then

$$\partial r / \partial D_i = g'_i(\mathbf{F}_i, D_i) \quad (9.2)$$

where  $g'_i$  is the partial derivative of  $g_i$  with respect to  $D_i$ . If we now solve the selection gradient at the intercept  $\partial r / \partial D_i = 0$ , where the second derivative is negative, we find that the equilibrium of the derived trait  $D_i$  is given as a function of the fundamental traits, i.e., we find

$$D_i^{*F} = G_i(\mathbf{F}_i) \quad (9.3)$$

where the function  $G_i$  represents selection and the superscript  $*F$  indicates an equilibrium under Fisherian selection. This optimisation procedure can be extended so that it includes all the derived traits that we consider, and then we find that the Fisherian optimum to the derived set  $\mathbf{D}$  is functionally determined by the fundamental set  $\mathbf{F}$ , i.e., we find

$$\mathbf{D}^{*F} = G(\mathbf{F}) \quad (9.4)$$

This classical equilibrium, where we explain the evolution of the derived traits from the fundamental traits, is different from the equilibrium under the hypothesis of deterministic evolution. This is because under the latter hypothesis we explain the evolution of all the phenotypic traits that we consider. More explicitly, under the hypothesis of deterministic evolution the complete phenotype is given by selection ( $G$ ), initial conditions ( $c$ ), and the time ( $t$ ) elapsed since the initial point  $t = 0$ , i.e.

$$\mathbf{P}^{*s} = G(t, c) \quad (9.5)$$

where the superscript  $*s$  indicates that the phenotype is in an evolutionary steady state where it continues to evolve.

By comparing eqns 9.4 and 9.5 it is apparent that the predictions of eqn 9.4 resemble Salthe (1989) and Williams (1992) hypothesis that it is misleading to think of evolution by natural selection as an unfolding process. This is because the phenotype of eqn 9.4 does not evolve in any particular direction, instead it is static and given by the historical events represented by the fundamental traits  $\mathbf{F}$ . In comparison, the prediction of eqn 9.5 is the diametrically opposite, i.e., that evolution is a deterministically unfolding process. This is because when the initial conditions  $c$  are set to the origin of self-replication, then the phenotype is given as a function of time.

In reality we do not need to incorporate the time component explicitly in order to construct a deterministic theory. As shown in Part IV, this is because the time component can be incorporated into the phenotype through an evolutionary increase in the exploitation efficiency  $\alpha$ . Hence,

at first, we may choose to disregard selection on the exploitation efficiency and let  $\alpha$  represent both the time component  $t$  and the initial conditions  $c$ . Then, it is possible to remove the time component from the deterministic hypothesis so that eqn 9.5 is represented as

$$\mathbf{P}^{**} = G(\alpha) \quad (9.6)$$

where the phenotype is given as a function of  $\alpha$ , and the superscript \*\* indicates that the complete phenotype is now in evolutionary equilibrium because  $\alpha$  does not evolve.

### 9.2.2 Dimensionality of theoretical optima

Another way to distinguish between the classical theory of evolution and the theory of Malthusian relativity is on a scale that describes the dimensionality of the optima, or equilibria, in the two theories. Here, a dimension refers to a trait that has evolved by natural selection, i.e., a trait that is not an evolutionary constraint, at least not on a very long time scale. The dimensionality then refers to the number of traits that are in equilibrium at the theoretical optimum instead of being given as an assumption, i.e., as a fundamental trait.

In the classical theory of evolution it is often the custom to operate with single-dimensional optima (SDOs). For example, in the Fisherian sex ratio theory the sex ratio is the single trait that has an evolutionary optimum, and this optimum depends on at least four fundamental traits, namely the presence of males, the ploidy level of the genome, the presence of sexual reproduction, and the degree of local mating. If, instead, like it is the case in Section 20.4, we have a model explaining both the presence of males, the sex ratio, and the degree of local mating from the ploidy level of the genome, and the presence of sexual reproduction, then we would have a three-dimensional optimum, or more generally, a multi-dimensional optimum, i.e., a MDO.

Now, the best we can do with any set of traits is to make a single model that has an all-dimensional optimum, i.e., an ADO, where all the traits in the set are explained without making explicit assumptions with respect to the other traits that have evolved by natural selection. For the current example the ADO is reached in Chapter 21 where the presence of males, the sex ratio, the presence of sexual reproduction, the ploidy level of the genome, and the degree of local mating have been explained from the assumption of self-replication, including the generation of density dependent competitive interactions.

Note, that an ADO does not provide us with an explanation for the evolutionary determinants of the complete organism, for that we would need a complete-dimensional optimum, i.e., a CDO that has evolutionary optima

in all the dimensions included in the phenotype of a particular organism. Although CDOs most probably cannot be obtained even for the most simple organisms they provide us with the ultimate goal in theoretical evolutionary biology. To approach this goal we can proceed in successive steps making ADO models that include an increasing number of traits. In this book I have aimed at a single ADO model that can explain the evolutionary equilibration of most of the essential life-history traits in mobile organisms. To describe this overall ADO model I will proceed by describing the evolutionary equilibration of only a single dimension at the time. If, instead, you want to understand the totality of the model I propose you should try and imagine the evolutionary equilibration in all dimensions at the same time.

Due to the reduction in the number of assumptions between a SDO model and an ADO model, we would prefer a deterministic theory of evolution over a historical theory. It is therefore interesting to examine whether the classical hypothesis of evolution by intrinsic constraints can be converted into a deterministic theory, and if this is not the case, to examine whether a deterministic theory can be constructed by other means.

### 9.3 Integrating the two theories

In the rest of the book I use a theoretical approach to analyse the degree to which we can construct a deterministic theory of evolution. The results of this analysis will generally show (*i*) why the classical hypothesis of evolution by intrinsic constraints cannot be converted into a deterministic theory of evolution, (*ii*) how a deterministic theory can be constructed from the hypothesis of selection by density dependent competitive interactions, and (*iii*) how this latter theory can be extended through an integration with the framework of the classical theory.

In more detail, the first step in this analysis is to examine whether the predictions of the classical theory can be converted into a deterministic theory of evolution, i.e., in resemblance with eqns 9.5 and 9.6 to analyse whether the complete phenotype is evolutionarily stable under the classical hypothesis of evolution by intrinsic constraints. When this is done we find that the classical hypothesis fails as a theory of deterministic evolution, and that this generally is because the original predictions are evolutionarily unstable in the dimensions of the fundamental traits. That is to say that the classical fitness profiles do not contain optima that can explain the evolution of the fundamental traits and, hence, the classical predictions are evolutionarily unstable when we allow for genetic variation in all traits.

The second step in the construction of a deterministic theory is the development of the restricted form of Malthusian relativity where the derived traits in the classical theory are explained by selection due to density depen-

dent competitive interactions. This is done independently of fundamental traits and, therefore, we obtain a deterministic theory, or an ADO model, on the evolution of the traits that traditionally have been considered to be derived.

The third step is the development of the general form of Malthusian relativity where the traits that are fundamental in the classical theory generally are explained by superimposing the classical form of selection on top of the restricted form of Malthusian relativity. Hereby the classical theory of selection by intrinsic constraints is integrated with the selection mechanism of density dependent competitive interactions. The result is a deterministic theory, or an ADO model explaining the evolution of both the assumptions and the predictions in the classical theory.

Both the equilibria in the general form of Malthusian relativity and the equilibria in the classical theory of evolution are of the form where the selection pressure for the evolution of the most derived traits depends upon the presence of other traits that are treated as being more fundamental. Moreover, the equilibria in the two theories are similar in the restricted sense that the pattern of co-occurrence between fundamental and derived traits generally is the same in the two theories. There are, however, two major differences between the equilibria in the two theories. The first major difference is that the equilibria in the classical theory are evolutionarily unstable because the fundamental traits in that theory are assumptions that are evolutionarily unexplained, while the equilibria in the general form of Malthusian relativity are evolutionarily stable because the fundamental traits in that theory have been evolutionarily explained prior to their use as the assumptions upon which the predictions of the derived traits depend. The second major difference between the equilibria in two theories is that the traits that are the derived traits in the classical theory generally are the fundamental traits in the general form of Malthusian relativity, and vice versa.

## 9.4 Equilibria at different levels

Due to the integration described in Section 9.3, I adopt the following notation in relation to the equilibria in the different theories. As the classical theory is based on Fisherian selection and as its equilibria are in themselves evolutionarily unstable I refer to these equilibria as Fisherian equilibria, or Fisherian optima. Then, as the equilibria of both the restricted and the general form of Malthusian relativity are evolutionarily stable under the hypothesis of deterministic evolution I refer to these equilibria as evolutionary equilibria, and these equilibria can be equilibrated at three levels.

The first level of equilibration that I refer to as the evolutionary equilib-

**Table 9.1** The superscripts ( $S$ ) used to indicate equilibria at different levels. Here shown for population density ( $N$ ).

$S$	Equilibrium
$N^*$	Population equilibrium
$N^{*F}$	Fisherian equilibrium
$N^{**}$	Evolutionary equilibrium
$N^{*s}$	Evolutionary steady state
$N^{*e}$	Unspecified evolutionary equilibrium

rium, is the equilibrium where the forces of selection are balanced against one another so that selection does not exist. This equilibrium is characterised by the absence of evolutionary changes in the phenotype. The second level is the constrained equilibrium that resembles the evolutionary equilibrium in the sense that it also contains no evolutionary changes in the phenotype. However, at the constrained evolutionary equilibrium the forces of selection are not balanced against one another and selection still occurs. The reason that this selection does not generate evolutionary changes is that it selects on traits that do not contain genetic variation. The evolutionary constraint is then the factor that prevents that genetic variation arises in the relevant traits.

The third level of equilibration is the evolutionary steady state, which we may also refer to as the dynamic evolutionary equilibrium. In contrast to both the evolutionary equilibrium and the constrained evolutionary equilibrium the evolutionary steady state is characterised by evolutionary changes in the phenotype. These changes arise because there is simultaneous selection on both the exploitation efficiency and the phenotypic allocation of the assimilated energy. Although the phenotype is evolving the steady state it is an equilibrium in the sense that the rate of evolution in the allocation of the assimilated energy is balanced against the rate of evolution in the exploitation efficiency.

In the next part of the book I will focus on the evolutionary equilibrium in order to explain the evolution of the basic traits we used to define the demographic model in Section 7.2. Then, in Part IV I will focus on the evolutionary steady state and I will also consider some implications of constrained equilibria. In Part V I will show that the evolution of derived traits, like senescence, sexual reproduction, and the ploidy level of the genome, depend upon the level at which the fundamental traits, like body mass and metabolic rate, are evolutionarily equilibrated. Finally, in the last part of the book I will leave the evolutionary equilibria and describe the evolutionary dynamics that arise when a population is perturbed away

from the population dynamic equilibrium. The superscripts that I use to distinguish between the different equilibria are summarised in Table 9.1.