

Part III

Evolution of basic traits

Chapter 10

Body mass

MANY ORGANISMS have relatively large body masses and this is a paradox for the classical theory of evolution. This is because the fundamental theorem of natural selection predicts a steady increase in the intrinsic Malthusian parameter and because this increase is expected to select energy from the body mass into numerical replication. Hereby all organisms are expected to remain at the level of self-replicating molecules. This paradox is generally neglected in the scientific literature, and this is partially because the evolutionists dealing with the evolution of body mass tend to focus only locally on the body mass of particular species. On this local scale it is possible to use the classical framework to construct a one-dimensional fitness profile that has an optimum that looks like an evolutionary equilibrium although it is not an evolutionary equilibrium.

In the traditional framework several authors have modelled the evolution of body mass by Fisherian selection and the hypothesis that the body mass is either an exploitative trait enhancing the net assimilation of resource, or a demographic trait that enhances reproduction and/or survival by other means (e.g., McLaren, 1966; Schoener, 1969, 1971; Case, 1979; Roff, 1981, 1986; Gerritsen, 1984; Stearns and Koella, 1986; Lunberg and Persson, 1993). Based on such assumptions it is possible to argue that the intrinsic constraint between lifetime reproduction and body mass is not the inverse relation we defined in Chapter 7 from physical constraints, but instead the approximately proportional relation existing within most natural species (e.g., Peterson, 1950; Robertson, 1957; Wootton, 1979; Roff, 1982; Chapter 14). However, when we compare across species we generally observe a negative relationship between body mass and Fisherian fitness, and this does not coincide with the classical assumption that the approximately proportional relation between reproduction and body mass is evolutionarily fixed.

As the positive relation existing between body mass and reproduction within a species is not evolutionarily fixed it is not an evolutionary constraint. We are then left with the alternative hypothesis that the intrinsic constraint conforms to the inverse relation we defined in Chapter 7. In this situation the classical theory predicts that only negligibly sized organisms can evolve by natural selection and, thus, we need a new mechanism of selection if we want to explain the evolution of large body masses. In this chapter I will apply selection by density dependent competitive interactions in order to obtain a large body mass. Under this hypothesis the body mass becomes a competitive trait that is used to dominate other individuals belonging to the same species.

The body mass is not the only quantitative trait that takes energy from numerical replication. Other traits include the metabolic rate per unit body mass and mental capacity as it is expressed in the form of complex behavioural interactions that require time and energy demanding play in order to develop fully. Exactly like with the body mass these two latter traits tend to correlate negatively with Fisherian fitness across species (e.g., Stearns, 1992; Charnov, 1993). Thus, it is questionable whether they can evolve by the classical hypothesis that metabolism and mental capacity enhance fitness because they enhance either the exploitation of resources, the expansion of niches, or the avoidance of hazards. If, instead, these traits reflect competitive quality that is used to dominate conspecifics their evolution and lack of positive relation to Fisherian fitness is easily explained by selection due to density dependent competitive interactions.

In the sections below I describe first the classical approach to the evolution of body mass and I examine also the degree to which this hypothesis is compatible with patterns on an evolutionary, or across-species, scale. Then, I give a general description of selection by density dependent competitive interactions before I use this type of selection to describe the evolution of body mass. Finally, I consider the evolution of a few body mass trends like Bergmann's rule, the island rule, and a negative relationship between the body mass and the rate of mortality. For an evolutionary deduction of the proportional relationship that exists between body mass and reproduction within a species we will have to wait until Chapter 14, which deals with the evolution of an exponentially increasing body mass.

10.1 The classical theory and no body mass

According to the classical theory it is the approximately proportional relationship between body mass and lifetime reproduction that represents the constraint selecting for a large body mass. Let me here assume that the approximate proportionality reflects the relationship $R \propto w^{2d/(2d-1)}$, where

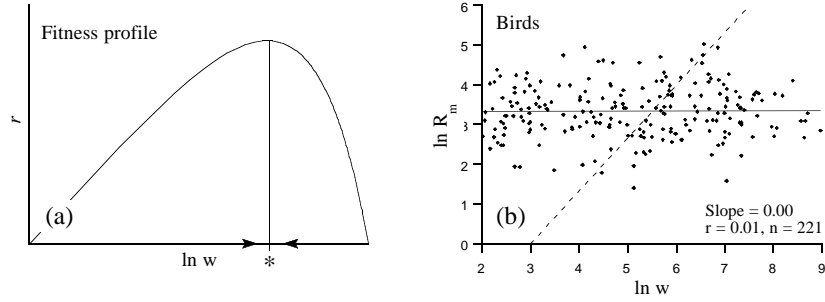


Fig. 10.1 (a) A classical one-dimensional fitness (r) profile for body mass (w), as defined by eqn 10.1. The star indicates the Fisherian equilibrium. (b) Maximal lifetime reproduction (R_m) plotted against body mass on double logarithmic scale for 221 species of birds. The solid line is the linear regression and the dashed line the prediction from the classical theory. R_m is estimated as the maximum lifespan in years subtracted the age of first reproduction and, then multiplied by the maximal clutch size and the number of clutches per year. Data from Cramp and Simmons (1977-1983), Cramp (1985-1992), and Cramp and Perrins (1993).

d is the number of spatial dimensions in which the organisms forage. At present this hypothesis might appear to be relatively obscure, but in Chapter 14 you will find that it nevertheless is plausible. Then, to obtain a body mass in evolutionary equilibrium we need only the additional assumption that survival (p) is negatively related to body mass, e.g., as $p \propto e^{-kw}$, where k is a positive constant. This leads to the fitness profile

$$r \propto \ln(e^{-kw} w^{2d/(2d-1)}) \quad (10.1)$$

illustrated in Fig. 10.1a. The selection gradient is then

$$\partial r / \partial w = 2d / (2d - 1)w - k \quad (10.2)$$

so that the body mass at the Fisherian optimum is $w^{*F} = 2d / (2d - 1)k$.

Let us now examine whether it is a plausible hypothesis that the large body masses of the organisms on Earth have evolved from the intrinsic constraints behind a one-dimensional fitness profile like eqn 10.1. Let us here focus both on the constraint on lifetime reproduction, which we have defined as $R \propto w^{2d/(2d-1)}$, and on the constraint on the exploitation efficiency, which can be deduced from the constraint on lifetime reproduction and the intrinsic relations described in Chapter 7. That is, from eqn 7.24, we have that $R = \epsilon / wB$, and as $\epsilon = \alpha E^*$ we find that $R \propto \alpha / wB$, and this is because all individuals have access to the same resource density E^* when competition is purely exploitative. When this result is combined with the

constraint on lifetime reproduction we have that

$$R \propto \alpha/wB \propto w^{2d/(2d-1)} \quad (10.3)$$

We can then rearrange this equation in order to find that $\alpha \propto w^{(4d-1)/(2d-1)}$ if we assume independence between w and B . This result coincides with the traditional notion that large individuals consume more resource than small individuals.

According to the classical theory we have that the two relationships $R \propto w^{2d/(2d-1)}$ and $\alpha \propto w^{(4d-1)/(2d-1)}$ are evolutionarily fixed so that they are the evolutionary constraints defining selection for a large body mass. As the two relations are fixed they must be valid also across species with a similar bauplan. One such group is birds and in Fig. 10.1b I show the relation between maximal lifetime reproduction (R_m) and body mass for 221 species of birds together with the predicted relation (I have assumed that birds forage in two spatial dimensions as it is indicated by the results in Chapter 13). In the figure there is no resemblance between the prediction and the observation. The expected across-species relation is $R_m \propto w^{4/3}$ whereas the actual relation is $R_m \propto w^0$, and this observation also holds for mammals (e.g., Charnov, 1995). Furthermore, across mammals the exploitation efficiency (α) is proportional to the 3/4 power of body mass (e.g., Brown et al., 1993) and this does not resemble the expected $\alpha \propto w^{7/3}$. These results suggest that the two relations $R \propto w^{2d/(2d-1)}$ and $\alpha \propto w^{(4d-1)/(2d-1)}$ cannot be the intrinsic constraints that define natural selection on the body mass axis, and this is because the relationships between the body mass and respectively the exploitation efficiency and lifetime reproduction are flexible and not evolutionarily fixed as required for the constraints that set the limits to natural selection. Instead, as the relations are flexible, it seems that it is natural selection that determines the particular exponents of the two relations. At this stage we cannot see how natural selection is doing that, but I will return to this problem in Chapter 14.

Another way to examine the degree to which the classical theory is consistent on an evolutionary scale is to focus on the result that the selection profile in the classical theory resembles the fitness profile. This implies that the relation between Fisherian fitness and body mass is given by the across-species allometry between the intrinsic Malthusian parameter and body mass. From Fenchel (1974) we know that this relation is approximately $r \propto w^{-1/4}$. Thus, we obtain the selection gradient $\partial r/\partial \ln w = -1/4$ from which we predict that only negligibly sized organisms can evolve by natural selection. This prediction cannot be true under the Darwinian hypothesis of evolution by natural selection because the prediction is a paradox in the sense that the prediction of negligibly sized organisms is estimated from the occurrence of large-bodied organisms. Thus, we can conclude that the

relative fitnesses are relative and not constant as assumed in the classical theory, and that the classical theory does not explain the evolution of the large body masses in natural organisms.

10.2 Selection by density dependent competitive interactions

As we have now seen that the classical hypothesis of selection by intrinsic constraints fails to explain the evolution of large body masses we need to turn to another type of selection if we want to explain the occurrence of large-bodied organisms. In this and the following sections I will show that selection by density dependent competitive interactions is one such mechanism allowing us to explain the evolution of large-bodied organisms. In the first subsection in this section I will focus on the mechanism by which competitive interactions generate within-population differences in the cost of competitive interference. Then, in Subsection 10.2.2 I will focus on the density dependent bias in the access to resource that is one reflection of these differences, and in Section 10.3 I will show that it is the density dependence in this bias that can explain the evolution on a large body mass.

In order to formulate a general type of selection let us first recall that there is no firm evidence that the within-species proportionality between reproduction and body mass represents an evolutionary constraint. Hence, I will assume that it is the inverse relation $R \propto 1/w$ that we defined in Chapter 7 that represents the evolutionary constraint. I make this assumption because this latter relation reflects the fact that the resource allocated to reproduction can be used only once. Given this basic constraint the classical selection gradient on body mass is negative, and this implies that organisms are expected to remain at the molecular level if competitive interactions did not exist.

For other components of competitive quality, like the metabolic rate and the complexity of the behavioural interactions, we expect also an intrinsic trade-off with the reproductive rate so that these traits will not evolve under the classical hypothesis that disregards competitive interactions. Given these intrinsic trade-offs between competitive quality and reproduction, in this section, I will formulate a general framework that can be used to explain the evolution of competitive quality in the presence of density dependent competitive interactions. This framework is exemplified with body mass, but the trait in question might resemble any other trait of competitive quality.

10.2.1 The cost of competitive interactions

In order to describe the cost of competitive interference let us formulate the fitness of an arbitrary variant i with the body mass w_i in relation to the fitness of an average individual with the body mass w . In Chapter 3 on density regulation we defined the absolute fitness of the average variant as

$$x = r - \mu\iota \quad (10.4)$$

in the presence of intra-population interference, where x is the extrinsic and r the intrinsic Malthusian parameter, μ is the average regulation by interference, and ι the level of intra-population interference. Let us here interpret ι as the number of competitive encounters experienced per individual per unit time. Then, μ is the average cost associated with a competitive encounter. This cost is the cost (μ_l) of a lost encounter subtracted the product between the fitness quantum (Q) gained from a won encounter and the average probability (P) by which an average individual wins a competitive encounter. For the average individual there is, on average, a 50% chance of winning an encounter and, hence, for the average variant the average cost per encounter is

$$\mu = \mu_l - 0.5Q \quad (10.5)$$

Then, on average, let an individual of the i th variant have the probability P_i of winning an encounter which implies that for the i th variant the average cost per encounter is

$$\mu_i = \mu_l - P_iQ \quad (10.6)$$

The deviation in the cost of the i th variant from the cost of the average variant is then

$$\begin{aligned} \Delta\mu_i &= \mu_i - \mu \\ &= (0.5 - P_i)Q \end{aligned} \quad (10.7)$$

Now, to set up a model of selection on competitive quality we need to describe the probability P_i as being positively related to the competitive quality (here body mass) of the i th variant. To do this let us approximate P_i as a linear function on the scale where genetic variation is expected to be additive. For traits like body mass it is most likely that genetic variation is additive on a logarithmic scale so that the coefficient of variation in body mass is body mass invariant if the amount of genetic variation is constant. That is, we can approximate P_i as a linear function of the natural logarithm to the body mass of the i th variant, i.e., as $P_i \propto \ln w_i$.

As the competitively superior individuals will win over the competitively inferior individuals the P_i probabilities are positively related to the within-population rank in the competitive quality of the i th variant. In relation to

this we notice that there is always n ranking levels among n individuals, and this implies that the rank of the j th ranked individual is independent of the amount of variation in the competitive quality among the n individuals. To obtain this invariance we can scale the measure of competitive quality (here, $\ln w_i$) with respect to the standard deviation in the genetic component of competitive quality (here, $\sigma_{\ln w}$). Then, in the immediate surroundings of the average variant we can approximate P_i as

$$P_i = \frac{\nu \ln w_i}{\sigma_{\ln w}} \quad (10.8)$$

where ν is a positive constant.

For the average variant with the body mass $w = w_i$ we know that $P_i = 0.5$ and, thus, that $0.5 = \nu \ln w / \sigma_{\ln w}$. When we insert this expression and eqn 10.8 into eqn 10.7 we find that

$$\Delta\mu_i = \psi(\ln w - \ln w_i) \quad (10.9)$$

where $\psi = \nu Q / \sigma_{\ln w}$ is a positive constant that is likely to be invariant with respect to body mass. Then, as $\mu_i = \mu + \Delta\mu_i$, from eqns 10.4 and 10.9, we find that the fitness of the i th variant is

$$x_i = r_i - [\mu + \psi(\ln w - \ln w_i)]\iota \quad (10.10)$$

By this equation we can see that the cost of interference to a given variant is negatively related to the body mass of that variant, and that it is positively related to the average body mass and to the level of intra-population interference.

10.2.2 Density dependent bias in resource access

Let me now illustrate that the differential cost to interference we described in the preceding subsection reflects a density dependent bias in the access to resource, a bias that is in favour of the large-bodied individuals.

To describe the bias in resource access let us insert $r_i = \ln(p\alpha E/w_i B)$ into eqn 10.10, take the exponential to the obtained expression, and find that the discrete growth rate is

$$\lambda_i = p\alpha E e^{-[\mu + \psi(\ln w - \ln w_i)]\iota} / w_i B \quad (10.11)$$

because $x_i = \ln \lambda_i$. As λ_i is also $p\epsilon_i/w_i B$, I can multiply with $w_i B/p$ on both sides in eqn 10.11 and find that the within-population differentiation in the assimilation of resource is

$$\epsilon_i = \alpha E e^{-[\mu + \psi(\ln w - \ln w_i)]\iota} \quad (10.12)$$

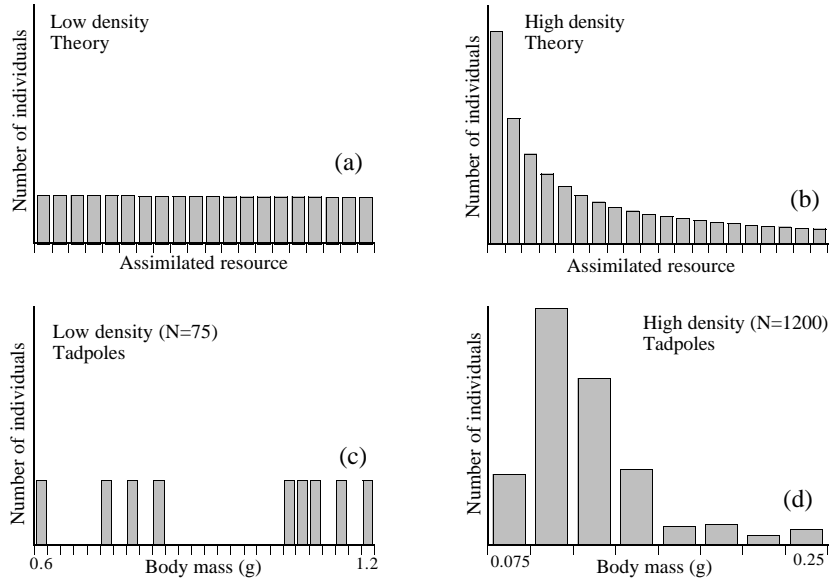


Fig. 10.2 An illustration of the within-population bias in resource assimilation, as generated by the intra-population interference in eqn 10.12 (**a** & **b**), and as seen in a population of tadpoles (**c** & **d**; data from Wilbur and Collins, 1973). For the tadpoles the variation in resource assimilation is described by the phenotypic variation in body mass. At low population densities (**a** & **c**) intra-population interference is nearly absent and all individuals assimilate approximately the same amount of resource. At high population densities (**b** & **d**) interference is present and the assimilation of resource is biased; according to the theory in favour of the competitively superior variants. For the tadpoles, the actual densities are $N = 75$ and $N = 1200$ in a 0.9 cm^3 sized enclosure.

According to eqn 10.12 the assimilation of resource is biased in favour of the individuals with large body masses, and this is because the large-bodied individuals dominate the small-bodied individuals during competitive encounters. This bias is illustrated in Fig. 10.2a,b from which it is apparent that the degree of bias depends upon the level of intra-population interference: with no bias in the absence of interference ($\iota = 0$) and a strong bias when the level of interference is high. As the level of interference is density dependent we find that the bias in resource assimilation will be density dependent, and as shown in Fig. 10.2c,d, such density dependent bias occurs in real populations.

The biased access to resource will, to a larger or smaller degree, act as a counterweight to the trade-off between the intrinsic Malthusian parameter

and competitive quality. When the level of interference is high it pays to be larger than the average because the bias in resource access is stronger than the intrinsic trade-off. On the other hand, it pays to be smaller than the average when the level of interference is low. Then, as selection for a decline in the average size is associated with an increase in the population equilibrium, and as the opposite is the case when selection favours an increase in size, we find that there is an intermediate equilibrium where the resource access is biased exactly so that it out-balances the intrinsic trade-off. In the following section I will formulate this equilibration more explicitly in order to explain the evolution of a large body mass.

10.3 Competitive interactions and a large body mass

To show that a large body mass can evolve from selection due to density dependent competitive interactions, in this section, I will first consider the two-dimensional fitness profile as it arises from the action of competitive interference. Then, I will consider the implausible case where the number of competitive encounters per individual is density independent, and this is done to show that the hypothesis of density independent interference will not explain the evolution of a large body mass. Hereafter, I will add density dependence to the number of competitive interactions and obtain an evolutionarily stable body mass. Finally, I will deal with the evolution of the level of competitive interference, a component that is essential for the further development of a general theory of evolution by density dependent competitive interactions.

The two-dimensional fitness profile on body mass determines the fitness of a particular variant as a function of the body mass of that variant and the body mass of the average variant in the population. From eqn 10.11 we find that the fitness of the average variant is

$$\lambda^* = p\alpha E^* e^{-\mu^*} / wB = 1 \quad (10.13)$$

at the population equilibrium, where ι^* is the level of interference at that equilibrium. From this equation the resource density at equilibrium is $E^* = wBe^{\mu^*} / \alpha p$, so that the fitness of the i th variant, i.e., eqn 10.11, is

$$\lambda_i^* = (w/w_i) e^{\psi \iota^* (\ln w_i - \ln w)} \quad (10.14)$$

when it is scaled with respect to the constraints of the population equilibrium, i.e., scaled with respect to $E^* = wBe^{\mu^*} / \alpha p$ and ι^* . Rearranging eqn 10.14 we find that

$$\lambda_i^* = (w_i/w)^{\psi \iota^* - 1} \quad (10.15)$$

so that we obtain the following fitness profile

$$x_i^* = \ln(w_i/w)(\psi\iota^* - 1) \quad (10.16)$$

when we take the natural logarithm to eqn 10.15. In eqn 10.16 we have scaled explicitly with the density dependence in the exploitation of the resource, whereas the density dependence in the level of interference is represented only implicitly through the superscript star in the term ι^* .

10.3.1 Density independent interference

Although it is unrealistic that ι^* is density independent let us first consider the case with a density independent level of interference, a situation that has been studied in detail by Parker (1979, 1983), Maynard Smith and Brown (1986), and Abrams and Matsuda (1994). In this case we obtain the selection gradient at the limit of the average variant by differentiating eqn 10.16 with respect to $\ln w_i$, and by letting $w_i \rightarrow w$. Then,

$$\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i = \psi\iota^* - 1 \quad (10.17)$$

This gradient implies that the average body mass will decline exponentially when $\iota^* < 1/\psi$, that it will remain stable when $\iota^* = 1/\psi$, and that it will increase exponentially when $\iota^* > 1/\psi$. This latter increase will occur only until the average body mass crosses the limit $w = p\alpha E_m/B$ where the population becomes extinct because $\lambda_m < 1$. In other words, the population will become extinct because the body mass has become so large that the individuals cannot assimilate the amount of resource required for self-replacement, not even in a perfect environment with plentiful resource. This prediction is, however, of no practical importance, and this is because it depends on the invalid assumption that the level of interference is density independent.

So in conclusion we have that it is not the effects of competitive interactions in themselves that will provide us with a general mechanism that can explain the evolution of a large body mass.

10.3.2 Density dependent interference

To explain the evolution of a large body mass we need explicitly to incorporate the component of density dependence into the level of competitive interference. To do this we note that the density dependent constraint on the equilibrium level of interference is $\iota^* = \ln \lambda_m \gamma_\iota / \gamma$, and this is because it is defined by the density dependence in the level of interference ($\iota^* = \gamma_\iota \ln N^*$, eqn 3.5) and the process of population dynamic limitation ($N^* = \sqrt[3]{\lambda_m}$, from eqn 3.12). The equilibrium level of interference is

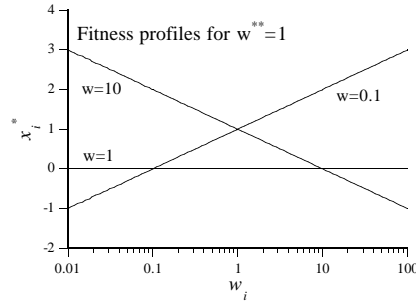


Fig. 10.3 One-dimensional projections of the fitness profile for body mass as defined by density dependent competitive interactions (eqn 10.19) for the situations where the average body mass at the evolutionary equilibrium (w^{**}) is 1, and the actual average body mass (w) is respectively 0.1, 1, and 10.

linked to the average body mass through the intrinsic and inverse relation between the body mass and the maximal growth rate, i.e., through the relation $\lambda_m = p\epsilon_m/wB$. To incorporate this link into the equilibrium level of interference, let $\lambda_m = \rho/w$, with $\rho = p\epsilon_m/B$, and insert this λ_m into $\iota^* = \ln \lambda_m \gamma_\iota / \gamma$ to find that

$$\iota^* = (\gamma_\iota / \gamma) \ln(\rho/w) \quad (10.18)$$

We can then insert this latter expression into eqn 10.16 and obtain the fitness profile

$$x_i^* = \ln(w_i/w) [(\psi\gamma_\iota / \gamma) \ln(\rho/w) - 1] \quad (10.19)$$

In Fig. 10.3 I have plotted three one-dimensional projections of this fitness profiles for the situation where the average body mass at the evolutionary equilibrium is one ($w^{**} = 1$) and the actual average body mass (w) is respectively 0.1, 1, and 10. From the figure it is apparent that the fitness profile rotates with changes in the average trait value, a rotation driven by the density dependent changes in the bias in resource access. Due to the rotation the selection pressure levels off toward the evolutionary equilibrium where the fitness profile is flat and all variants are equally fit even when they vary considerably in body mass. This feature of the evolutionary equilibrium implies that genetic variation can accumulate as long as the average trait value remains constant and the assumption of linearity holds. This accumulation of genetic variation at equilibrium is in contrast to the equilibria in the classical theory that by definition cannot contain any genetic variation.

If I differentiate the fitness profile (eqn 10.19) with respect to the within-population variation in \ln body mass and let $w_i \rightarrow w$ I obtain the selection

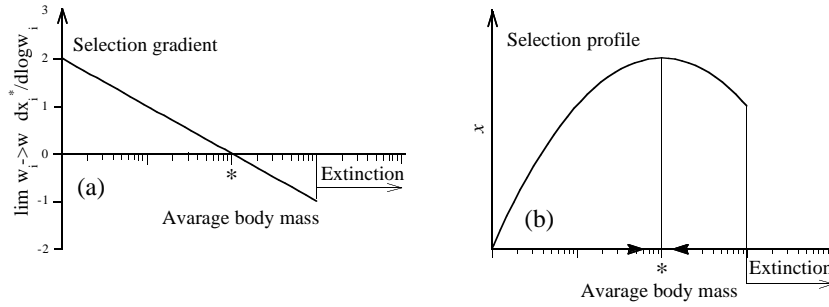


Fig. 10.4 The selection gradient (a) and the selection profile (b) for body mass as defined by density dependent competitive interactions. The stars indicate the evolutionary equilibrium, and the body masses to the right of the curves are so large that the population becomes extinct because the individuals cannot assimilate sufficient resource for self-replacement.

gradient at the limit of the average variant:

$$\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i = (\psi \gamma_i / \gamma) \ln(\rho/w) - 1 \quad (10.20)$$

This gradient is shown in Fig. 10.4a, and it is inversely related to the body mass having an equilibrium defined by the intercept $\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i = 0$. Notice also that if the limit of extinction, i.e., $\lambda_m = \rho/w = 1$, is inserted into the selection gradient of eqn 10.20, then we find that a population is about to become extinct when the within-population slope between fitness and \ln body mass approaches -1.

From eqn 10.20 we find that the average body mass at the evolutionary equilibrium is

$$w^{**} = \rho e^{-\gamma / \gamma_i \psi} \quad (10.21)$$

This equilibrium is also the global attractor because $\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i < 0$ for $w > w^{**}$ and $\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i > 0$ for $w < w^{**}$. The result that the equilibrium mass is the global attractor can depend upon the assumption that the population remains at the population dynamic equilibrium. When this is not the case the attractor might instead be a cyclic body mass, as described in Chapter 24.

When the prediction of eqn 10.21 is compared with the results from eqn 10.17 we can conclude that it is the density dependence in the level of interference and the inverse relation between the level of interference and the average body mass that are the essential components allowing the evolution of a large body mass. By a different approach Abrams and Matsuda (1994) obtained a similar conclusion that density dependent competitive interactions are essential for the evolution of a large body mass.

Let us now examine the selection profile describing the relative fitness of the average variant as a function of the body mass of that variant. I can obtain this profile if I integrate the selection gradient (eqn 10.20) with respect to the natural logarithm of the average body mass. That is, the selection profile is

$$\begin{aligned}
 x &= \int [\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i] \partial \ln w & (10.22) \\
 &= \int [(\psi \gamma_\iota / \gamma) \ln(\rho/w) - 1] \partial \ln w \\
 &= \ln w [(\psi \gamma_\iota / \gamma) \ln(\rho/\sqrt{w}) - 1] + k
 \end{aligned}$$

where k is a constant. I have plotted one version of this profile in Fig. 10.4b where it is apparent that the curve has an optimum that coincides with the evolutionary equilibrium. Notice also that the selection profile differs from the one-dimensional projections of the fitness profile (Fig. 10.3), and this is in contrast to the classical theory where the two profiles are similar.

10.3.3 Evolution of interference

So far in this section we have dealt only with the evolution of body mass, but the equilibrium mass is associated also with equilibrium values in a number of other traits. Some of these traits are the population density and the rate of reproduction, traits that I will consider in detail in the following two chapters. Another component is the level of interference in the population, a trait that I will consider briefly in this subsection. As it is apparent throughout the rest of the book, the equilibrium level of interference is a component that is essential for the further development of a general theory of evolution by competitive interactions. This is because it is this level of interference that will determine the degree to which the different components of competitive quality will evolve. As you will see in Part V, the variation in the equilibrium level of interference is an essential component that, among other things, can explain both why traits like senescence, soma, sexual reproduction, and a diploid genome are present in large-bodied organisms, like the higher eukaryotes, and why these traits are absent in negligibly sized organisms, like prokaryotes and viruses.

The level of intra-population interference for a body mass in evolutionary equilibrium is defined by eqn 10.15 at the limit $\psi \iota^* = 1$, where $\lambda_i^* = 1$ for all the variants within the population. From this equilibrium constraint we find that the level of interference at the evolutionary equilibrium is

$$\iota^{**} = 1/\psi \quad (10.23)$$

This result implies that an individual will experience $1/\psi$ competitive encounters per unit time when the body mass is in evolutionary equilibrium.

This level of interference depends on the assumption that the body mass is in evolutionary equilibrium and, as shown in Chapter 14, this assumption will apparently not hold for many populations because they experience a higher level of interference.

As we recall that $\psi \propto Q$ we find that the evolutionarily determined number of competitive encounters per individual is inversely related to the size of the fitness quanta Q for which the individuals compete. As these quanta can represent anything from a single resource item to whole territories we expect that the level of interference can vary quite a bit in natural populations. For example, when competition is for small and single resource-items we expect many competitive encounters per individuals per unit time. This prediction might resemble pelagic fish that compete for small and single resource items and which experience relatively many competitive encounters when they forage in large and dense schools. In comparison, when competition is for large and defensible territories we expect only few competitive encounters per individual per unit time. This prediction could resemble terrestrial vertebrates that compete for defensible territories and which experience relatively few competitive encounters because the individuals are evenly distributed at relatively sparse densities.

10.4 Some predicted patterns

Let us briefly consider whether the body mass predicted by eqn 10.21 behaves in resemblance with general observations. To do this we notice, from eqn 10.21 and $\rho = p\epsilon_m/B$, that the body mass at the evolutionary equilibrium is

$$w^{**} = p\epsilon_m e^{-\gamma/\gamma_e \psi} / B \quad (10.24)$$

Then, as $\epsilon_m = \alpha E_m = \alpha r_e / \gamma_e$, from eqn 10.24 we obtain

$$\begin{aligned} w^{**} &\propto p\alpha E_m \\ &\propto p\alpha r_e \end{aligned} \quad (10.25)$$

From this equation it is apparent that organisms that are efficient in assimilating resource (large α) will have a large body mass. This prediction coincides with the observation that an elephant eating a large amount of resource is large, whereas a mouse eating only a small amount is small. At first this prediction may appear trivial, but this is not the case. To see this we need only return to the classical framework where we predict the absence of a body mass independently of the amount of resource assimilated. In the following three subsections I will consider some of the other patterns that emerge from eqns 10.24 and 10.25.

10.4.1 Body mass balanced against mortality

From the prediction of eqn 10.25 we have that the body mass is balanced against mortality, i.e., that the mass is negatively related to the rate of mortality ($1 - p$), or positively related to the survival probability (p). That is to say that if the rate of extrinsic imposed mortality is increased, then the body mass will decline.

We can understand this balance between body mass and rate of mortality by considering a population in evolutionary equilibrium. Then, if the rate of mortality is increased the density of the population equilibrium will decline causing a decline in the bias in resource access and this will favour the small variants that have a high intrinsic Malthusian parameter. Selection will then increase numerical replication at the cost of body mass until the population density has increased to the density where the extrinsic bias in resource access is exactly so strong that the different variants in the population have the same extrinsic Malthusian parameter.

The predicted and negative relationship between body mass and the rate of mortality has been observed in guppies where the individuals from localities with a high predation rate (high predation localities) are smaller than the individuals from localities with a low predation rate (low predation localities) (Reznick et al., 1996). Introduction experiments have also been carried out for this species, and they shown that guppies that are transferred from a high predation locality to a low predation locality evolve a significantly larger body mass, both in the juvenile and the adult stages (Reznick et al., 1990).

According to the classical theory we do not expect a negative relationship between body mass and the rate of mortality. Instead, in the classical theory it is size- or age-structured differences in the rate of mortality that will select for differences in body mass (Gadgil and Bossert, 1970; Charlesworth, 1994). This implies that the negative relationship between body mass and the rate of mortality will evolve only when mortality operates differentially, so that it predominantly is the large individuals that die when the rate of mortality is high, while it predominantly is the small individuals that die when the rate of mortality is low. If, instead, it predominantly is the large individuals that die when the mortality rate is low and vice versa, then from the classical framework we expect the opposite relation, i.e., that the individuals are larger the higher the rate of mortality.

At first it was thought that the classical hypothesis could explain the differences in the body mass of guppies (e.g., Reznick et al., 1990). It was assumed that the predators at the high predation localities selected preferentially on the large and adult guppies, while the predators at the low predation localities ate predominantly the small and juvenile guppies. However, a recent study by Reznick et al. (1996) that was designed to test

this assumption, “by directly estimating the mortality rates of guppies in natural populations”, failed to detect the differential mortality that is required before the classical hypothesis will work. Instead, this latter study of Reznick et al. supported the hypothesis of selection by density dependent competitive interactions, and this is because it confirmed that the “guppies from the high predation localities experience significantly higher mortality rates than their counterparts from low predation localities”, and because the “higher mortality rates are uniformly distributed across all size classes, rather than being concentrated in the larger size classes.”

10.4.2 Bergmann’s rule

From the prediction of eqn 10.25 it follows also that the body mass will be positively related to the rate of production in the resource, i.e., that $w^{**} \propto r_e$. In relation to this prediction we have Bergmann’s rule (1847) that states that the body mass within a species is larger toward the polar regions. Bergmann’s explanation for this cline was that it was a reflection of thermal homeostasis so that the animal needed to be larger in colder regions because otherwise it could not maintain the same internal temperature. This explanation has recently been criticised by McNab (1971) and Geist (1986). They found that Bergmann’s explanation is invalid because the body mass in mammals increases only up to 60-65° N after which it decreases with increased latitude. McNab and Geist found that this change in body mass correlated positively with the duration of the productivity pulse in the resource. To the extent that this duration correlates with the density of the edible resource this result resembles the prediction of eqn 10.25.

10.4.3 The island rule

The island rule is a second rule that is related to variation in the body mass within a species. It describes that large animals are smaller on islands than on mainlands while the opposite is the case for small animals (Foster, 1964; Heaney, 1978; Lomolino, 1985). In contrast to Bergmann’s rule it is unlikely that the island rule is the result of variation in the resource density. This is because a lower resource density on islands should result in a decline in body mass independently of the body mass of the animal. In other words, according to the resource hypothesis we would expect that it is not only the large animals, but also the small animals, that are smaller on islands than on mainlands.

So, to explain the island rule we need to focus on mechanisms operating differentially in small and large animals. One such mechanism is related to the home-range area where the size of small islands may limit the home-range of the larger, but not the smaller, animals. Then, in the

smaller home-ranges of the larger animals there will be less resource available, and this would imply that large animals should be smaller on such islands. The smallness in the body mass of large animals on islands may be caused also by a different mechanism. For example, we may expect that the within-population differentiation in resource access is partially due to dispersal of competitively inferior individuals from favourable habitats into less favourable habitats. Such dispersal is expected to be severely restricted on islands. It is then likely that antagonistic behaviour cannot cause the access to resource to differentiate as strongly on islands as on mainlands, i.e., the ψ parameter will be smaller on islands. Then, according to eqn 10.24 we find that the decline in ψ will cause a decline in body mass.

In principle, it is possible to test whether the smallness of large animals on islands is due to a size restriction of the home-range area, or due to restricted dispersal. As restricted dispersal will decline the ψ parameter it will, according to eqn 11.4 ($N^{**} = e^{1/\gamma_i \psi}$), cause the population density to increase. This is in contrast to the situation where the resource availability (E^*) is reduced due to a restricted home-range. Here, eqns 11.4 and 11.10 ($N^{**} = e^{1/\gamma_i \psi}$ and $N^{**} = E^* e^{1/\gamma_i \psi}$) predict that the population density is left unchanged, or even declined, relative to the body mass allometry for the population density.

For small animals the home-range and the migration pattern may not be affected by the size of an island. Instead, on islands small animals can have an increased access to resource if larger species, that are superior in interference competition, are absent. When this is the case we would expect that small animals should have a larger body mass. It is not only the absence of competitors that may lead to an increased body mass for small animals on islands. From eqn 10.24, and the results from guppies, we know that increased survival can enhance the body mass. Hence, if the predators to smaller species are absent on islands we might expect a larger body mass. In principle, it should be possible to test whether the largeness of small animals on islands is caused by the absence of predators, or the absence of competitors. This is because eqn 12.8 ($R_m^{**} = e^{\gamma/\gamma_i \psi}/p$) predicts that decreased predation, i.e., increased p , causes a decline in the maximal rate of reproduction, while eqn 12.11 ($R_m^{**} \propto r_e^\gamma \propto E^{*\gamma}$) predicts that an increase in resource access, due to the absence of a large competitor, would cause an increase in maximal reproduction.

So far we have dealt only with the patterns that are observed in the body mass, but the described theory predicts that we should observe the same patterns in the rate of metabolism or the degree of complexity in behavioural interactions. This coincides with McNab (1994) who found that on small islands where the body size of pigeons, bats, and rodents is reduced their metabolic rate is reduced also.

Chapter 11

Population limitation

THE ABUNDANCES of terrestrial animals are limited to the degree that the world is green. That is to say, animals are so sparse that they do not over-exploit their resources. This fact is often referred to as the balance of nature and it is a paradox for the classical theory. This is because the fundamental theorem of natural selection predicts a steady increase in the population equilibrium, and because this leads to a continuous deterioration of the resource.

Today it is often neglected that the balance of nature is a paradox, and this is partially because the scientists who deal with population limitation tend to operate within the classical theory of population dynamics that disregards evolutionary changes. This approach, which I described in the first part of the book, accounts only for the proximate, or local, setting of the population equilibrium. The global setting is evolutionarily determined, and it is this ultimate determinant of the population equilibrium that is the subject of this chapter.

There has been one major attempt to explain the balance of nature and that is the proposal by Wynne-Edwards (1962, 1986, 1993) that it is group selection that sets an upper limit to reproduction because over-exploitation causes the extinction of the groups having any higher rate of reproduction. Hereby group selection should favour animals that restrain from reproduction in order to keep the population from over-exploiting the resource. Today this idea is largely abandoned, and that is because there is no evidence for the hypothesis that some groups go extinct due to famine while the individuals in the remaining groups restrain from reproduction. On the contrary, the individuals in natural populations tend to compete in order to optimise their reproductive output.

Apart from the idea of Wynne-Edwards the classical approaches to population limitation are based on the population dynamic equations that are

described in the first part of this book. In this chapter I will briefly summarise these ideas and relate them to the classical theory of selection where they do not prevent the evolution of over-exploitation. Thereafter I will show that selection by density dependent competitive interactions predicts a nature in balance. As we move from classical population limitation to limitation by selection due to density dependent competitive interactions the process of limitation changes completely. It is only when genetic variation is absent and evolution does not occur that the new form of limitation merges into the classical form.

11.1 The classical theory and no limit

In the absence of evolutionary changes the population equilibrium is determined by the population dynamic processes that are described in the first part of the book. For our single species model these processes set the following limit to the population equilibrium

$$N^* = \sqrt[3]{\lambda_m} \quad (11.1)$$

where $\lambda_m = pR_m$ at this equilibrium. From these two equations we see that a limiting factor is any factor that when changed causes a change in either production (R_m), loss ($1-p$), or regulation (γ). This rule is the core of population limitation in the classical theory that disregards evolutionary changes, and it is maintained when Fisherian selection is added. The only change is that especially the physiological, or intrinsic, determinants of R_m and p can be modified by selection while the ecological, or extrinsic, constraints on R_m , p and γ are less modifiable.

In the classical theory where selection is defined from the genomic and physiological constraints intrinsic to the organism the evolutionary changes in the population equilibrium are secondary phenomena following from the primary changes in the demographic traits R_m and p . As it is described in the following chapter, these latter changes are a continuous increase in maximal reproduction and a simultaneous optimisation of the physiological trade-offs between survival and reproduction. This leads to the fundamental theorem of natural selection predicting that the population equilibrium continues to increase under Fisherian selection. This increase will inevitably lead to an over-exploited resource, and this is because the resource abundance at equilibrium is $E^* = E_m - \alpha N^* / \gamma_e$ by eqn 7.30. That is to say that when $N^* \rightarrow \infty$, then $E^* \rightarrow 0$.

This classical prediction of an over-exploited resource does not resemble reality where positive resource equilibria exist. Especially in terrestrial habitats, where herbivores generally eat less than 10% of the edible plant

biomass, the primary producers are almost unaffected by the presence of herbivores (Wiegert and Owen, 1971; Ricklefs, 1990; Hairston and Hairston, 1993). To avoid this paradox Hairston et al. (1960) proposed, and Hairston and Hairston (1993) elaborated, the hypothesis that it is the inter-specific interactions of the food web that maintains nature in a state of balance where the resource is almost unexploited. In short, their idea is that if we add a predator on top of the herb-herbivore system, then the predator will limit the density of the herbivore to the extent that it no longer over-exploits the herb.

This traditional argument of limitation by a predator holds only in the absence of evolutionary changes. With Fisherian selection the density of at least one of the involved species is expected to increase in an uncontrolled manner. It is thus very understandable that Wynne-Edwards (1962, 1986, 1993) continues to argue that in order to explain the observed balance we need group selection where the individuals scarify their own fitness for the benefit of the group and/or the population. In the following section we will see that group selection is not a necessity since individual selection by density dependent competitive interactions predicts a nature in balance.

11.2 Competitive interactions and a nature in balance

Before I describe the balanced equilibria evolving from selection by density dependent competitive interactions let us consider the differences between the classical theory and Malthusian relativity in the process of population limitation. In the previous section we saw that the evolutionary setting of the population equilibrium in the classical theory is a secondary phenomenon following from the primary changes in the demographic traits, changes that are driven by selection on intrinsic constraints. That is to say, the demographic traits are evolutionarily determined independently of the population equilibrium, and that the population equilibrium follows from this determinants of the demographic traits. This causality is turned upside down when we turn to population limitation under the theory of Malthusian relativity. That is to say, in Malthusian relativity it is the evolutionary setting of the demographic traits that is the secondary phenomenon following from the primary changes in the population equilibrium. In other words, it is the evolutionarily determined population equilibrium that is given by extrinsic, or ecological, constraints independently of the demographic traits, and it is then the demographic traits that are evolutionarily adjusted so that they conform to the evolutionary determinants of the population equilibrium.

To see why the causality in Malthusian relativity is the reverse of the causality in the classical theory recall from Chapter 10 on body mass that the evolutionary equilibration by density dependent competitive interactions occurs through an adjustment of the level of intra-population interference. This implies that the evolutionary equilibrium is defined primarily by the level of interference that is exactly so high that the extrinsic bias in the access to resource is balanced against the intrinsic trade-off between the intrinsic Malthusian parameter and competitive quality. This level of interference is determined by the ecological constraints summarised in the ψ parameter, i.e., from eqn 10.23 the level of interference at equilibrium is $\iota^{**} = 1/\psi$, and this is equivalent to

$$I^{**} = e^{1/\psi} \quad (11.2)$$

since by definition $I = e^{\iota}$. Then, from Chapter 3, we have that the density dependence of the level of interference is

$$I = N^{\gamma\iota} \quad (11.3)$$

We can then combine eqns 11.2 and 11.3 and find that the evolutionarily determined population equilibrium is given as

$$N^{**} = e^{1/\psi\gamma\iota} \quad (11.4)$$

independently of the demographic traits.

This equilibrium implies that the Malthusian parameter (λ_m) and its two components, reproduction (R_m) and survival (p), are absent from the set of limiting parameters. This is very different from limitation in the classical theory where any change in λ_m , R_m , and p is transformed directly into changes in the population equilibrium (see eqn 11.1). In other words, the equilibria in the two theories behave distinctively different: If in the classical theory we begin to remove individuals at a constant rate from the population the equilibrium density declines. If in Malthusian relativity we begin to remove individuals at a constant rate the equilibrium density remains the same (a first approximation). The density remains the same because the extra energy taken from the population by the removal of individuals causes a decline in the competitive quality of an average individual instead of a decline in the equilibrium density. This prediction is an equilibrium prediction, which means that as long as the evolutionary equilibrium is not yet reached the removal of individuals will cause a depression in the population density.

According to selection by density dependent competitive interactions the limiting factors are the factors that influence the balance between the extrinsic gradient, between resource access and competitive quality, and the

intrinsic trade-off, between the demographic traits and competitive quality. As the extrinsic gradient is caused by interference competition the limiting factors include the density dependence in the level of intra-population interference (γ_i), and the degree to which a competitive encounter generates differences in resource access (ψ). In other words, when the density dependence in the level of interference rises the population equilibrium declines, and this is because fewer individuals are needed to generate the level of interference that generates the appropriate bias in resource access. When, instead, the differentiation in resource access caused by a competitive encounter rises, the population equilibrium declines, and this is because less interference and, thus, fewer individuals are needed to balance the extrinsic gradient in resource access against the intrinsic trade-off between the demographic traits and competitive quality.

Let us now briefly consider how the interplay between the evolutionary and the population dynamic processes will influence the degree to which the actual form of population limitation will resemble classical limitation or limitation by selection due to density dependent competitive interactions. Given a change in the environment and given that the population dynamics is damped, the time that it will take to return to the evolutionary equilibrium is inversely related to the amount of genetic variation, while the time it will take to return to the population equilibrium is relatively independent of the amount of genetic variation. This means that the shorter the time span of observation and the smaller the amount of genetic variation the more the observed abundances will tend to behave as predicted by the classical equilibrium. On the other hand, the longer the perspective the more the prediction of eqn 11.4 will take over. In Chapter 24 I will return to such dynamic comparisons, but in the sections below I focus only on the evolutionary equilibrium and how it is influenced by variation in different factors.

11.2.1 The size of resource quanta

Let us first examine how the evolutionary population equilibrium is influenced by the size of the resource quanta for which the individuals compete. From the definition in Chapter 10 the ψ parameter in eqn 11.4 is proportional to the resource, or fitness, quanta (Q) so that the population equilibrium is inversely related to Q as

$$N^{**} \propto e^{1/Q} \quad (11.5)$$

Hence, if competition is for large quanta, like defensible territories, the evolutionarily determined population density is relatively low, while it is high when competition is for small and single resource items. If we combine this prediction with the equilibrium density of the resource, i.e., with

$E^* = E_m - \kappa N^{**}$, we find that systems with competition for small resource items have a relatively over-exploited resource, whereas systems with competition for large and defensible resource quanta have a relatively unexploited resource. It might be this mechanism that explains the pattern that is apparent between pelagic and terrestrial systems. If so, the herbivores in pelagic systems eat between 60 and 99% of the primary production because they compete for small resource items, whereas the herbivores in terrestrial systems eat less than 10 to 15% of the primary production because they compete for larger resource quanta, e.g., for territories (data from Wiegert and Owen, 1971; Ricklefs, 1990; Hairston and Hairston, 1993).

11.2.2 Genetic variation

The amount of genetic variation in competitive quality may also affect the evolutionary setting of the population equilibrium. From the definition in Chapter 10 the ψ parameter in eqn 11.4 is inversely proportional to the amount of genetic variation in competitive quality ($\sigma_{\ln q}$). Hence, by eqn 11.4, the evolutionarily determined population density is positively related to the level of genetic variation, i.e.,

$$N^{**} \propto e^{\sigma_{\ln q}} \quad (11.6)$$

To understand this relationship between the equilibrium point and the amount of genetic variation let us consider a population in evolutionary equilibrium as it is described by the squares in Fig. 11.1. They represent five individuals in an equilibrium population; two with body masses below the population average, one at the average, and two above the average. As it is illustrated in Fig. 11.1a, at the evolutionary equilibrium the resource access among the individuals is proportional to the body mass of the individuals, and this implies that the five individuals are equally fit (Fig. 11.1b).

If the variation in the body mass among the five individuals is increased we have a situation like the one illustrated by the circles. Due to the contest-like type of competition the distribution of resource over the five individuals will remain the same, which implies that the slope of resource access over body mass will decline (Fig. 11.1a). Due to this decline in the bias in resource access the two smaller individuals now have relatively more resource available and this will cause selection for a decline in body mass (Fig. 11.1b). Then, because $N^* \propto \sqrt{\lambda_m}$, by eqn 11.1, and because $\lambda_m = p\epsilon_m/wB$, the decline in body mass will generate an increase in the population equilibrium. The triangles in Fig. 11.1 represent a situation where the variation in body mass has declined relative to the equilibrium situation described by the squares. In this situation the slope of resource access over body mass is increased relative to the equilibrium situation, and thus there is selection for an increase in the body mass and a decline in the population equilibrium.

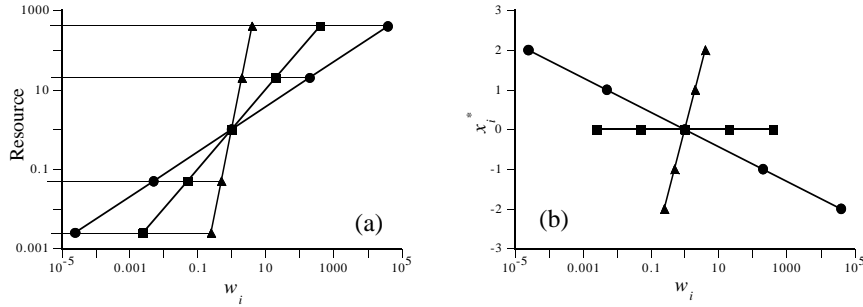


Fig. 11.1 (a) An illustration of the partitioning of resource against body mass (w_i) and (b) its relation to fitness (x_i^*) among five individuals when the partitioning is determined by contest-like interference competition. The squares represent the situation at the evolutionary equilibrium where the resource access across the individuals is proportional to the body mass of the individuals (a), and where all individuals are equally fit (b). When the level of variation in body mass is increased (circles) compared with the equilibrium (squares) the small individuals are most fit and selection will cause an increase in the population equilibrium. When, instead, the variation is decreased (triangles) relative to the equilibrium the large individuals are most fit and selection will cause a decline in the population equilibrium.

11.2.3 Metabolic rate

Among terrestrial vertebrates ectotherm reptiles are ≈ 30 times more abundant than endotherm mammals (e.g., Currie and Fritz, 1993). This relationship can be explained by the metabolic rate that is ≈ 30 times higher in endotherms than in ectotherms (Peters, 1983).

Because ectotherms metabolise ≈ 30 times less energy than endotherms they need ≈ 30 times less resource and, thus, they need to forage ≈ 30 times less often. Due to this reduced foraging activity the level of interference will be reduced. Therefore, at a given density the number of competitive encounters per individual per unit time is expected to be positively related to the metabolic rate per unit mass (B). Hence, we have the relationship

$$I = (BN)^{\gamma_\iota} \quad (11.7)$$

if we assume that the process determining non-proportionality in the level of interference, i.e., deviations in γ_ι from one, is associated to the process of interference. When eqn 11.7 is combined with the equation for the density dependence in the level of interference (eqn 11.3) we find that the population density at the evolutionary equilibrium is

$$N^{**} = e^{1/\psi\gamma_\iota} / B \quad (11.8)$$

From this expression the population density is inversely related to the metabolic rate, and this implies that ectotherms are expected to be ≈ 30 times more abundant than endotherms because their metabolic rate is ≈ 30 times smaller.

11.2.4 Rate of production in the resource

In Chapter 5 we saw that the population density is proportional to the rate of production in the resource. To investigate whether this relation holds at the evolutionary equilibrium let us consider the relationship between the foraging distance and the density of the resource. Here, we find that the distance that an individual covers while it forages is inversely related to the density of the resource, i.e., if the resource is sparse an individual needs to cover a large distance in order to find sufficient food, whereas if the resource is abundant the individual needs hardly move while it is foraging. Then, as the number of competitive encounters per individual per unit time is proportional to the average distance travelled per individual during that unit of time, we find that the level of interference is expected to be inversely related to the density of the resource, i.e.

$$I = (N/E)^{\gamma_\iota} \quad (11.9)$$

At the evolutionary equilibrium the level of interference in the consumer population is given also by eqn 11.2. Combining this level with the functional determinants of interference given by eqn 11.9, we find that the consumer density at the evolutionary equilibrium is

$$N^{**} = E^* e^{1/\psi\gamma_\iota} \quad (11.10)$$

Hence, the density of the consumer species is proportional to the density of its resource. Then, as $E^* \propto r_e$ (eqn 7.30), the population equilibrium is proportional to the rate of production in the resource, exactly like the body mass and the metabolic rate (Chapter 10).

Chapter 12

Reproduction

THE RATE OF reproduction can vary from thousands of eggs in many species of fish to a minimum clutch of one in many birds and mammals. The evolution of these most limited rates of reproduction is a paradox for the classical theory of evolution. This is because the fundamental theorem of natural selection predicts a continuous increase in the growth rate and because the obvious solution to this problem is a continuous increase in reproduction. Today this paradox is generally neglected and this is partially because the scientists who deal with the evolution of the reproductive rate tend to focus only locally on predictions within species. On this local scale it is possible to use the classical framework to construct a one-dimensional fitness profile that has an optimum that looks like an evolutionary equilibrium although it is not an evolutionary equilibrium.

The classical approach to the evolution of the reproductive rate is based on Lack's clutch size. In 1947 Lack published an influential paper in which he proposed that optimal reproduction occurs at the rate where most offspring survive. In this limited version, where Lack's idea is seen only as an optimisation of reproduction against survival, Lack's clutch size is not contradictory to a continuous increase in reproduction. This is because no matter the level at which reproduction occurs it can be optimised against mortality. However, starting with Lack himself there has been a growing tradition to use Lack's clutch size as an explanation for the evolution of the optimal rate of reproduction. It is on this global, or evolutionary, scale that the classical tradition fails.

When Lack published his paper he realised that the reproductive rate is equilibrated at different levels. At the most proximate level phenotypic plasticity is adjusting reproduction to the amount of resource available. According to the $pR = 1$ constraint of the population equilibrium this is the adjustment from R_m to $R = 1/p$, an adjustment that is caused mainly by

density regulation. At the other extreme there is the ultimate, or evolutionary, setting of R_m .

According to Lack's theory the ultimate setting of R_m evolves from physiological trade-offs that exist between reproduction and offspring (Lack, 1947) or parent (Charlesworth, 1994; Schaffer, 1983) survival (reviewed by Godfray et al., 1991; Stearns 1992; Bulmer, 1994). In Section 12.1 we will find that this hypothesis fails because the predicted rates of reproduction are evolutionarily unstable. This implies that if we want to predict the evolution of the reproductive rate, then we need to include selection by another dimension, e.g., by density dependent competitive interactions. When this is done in Section 12.2 we will find that R_m can evolve from ecological, or extrinsic, constraints instead of being defined by physiological, or intrinsic, constraints as it traditionally has been thought.

When an evolutionarily stable rate of reproduction has been obtained through the density dependent competitive interactions in Malthusian relativity it is possible to reinterpret Lack's equilibrium as a secondary phenomenon defined from the equilibrium phenotype of Malthusian relativity. When this is done we obtain the result that it is the physiological trade-offs between reproduction and survival that are the derived traits evolutionarily determined by the optimal growth rate, which is more fundamental. In other words, we will find that the intrinsic trade-offs are no longer the primary forces defining natural selection, instead they are the evolutionary result of natural selection that is defined by ecological constraints.

In the classical theory based on Lack's optimum there is no general and simple mechanism that can explain why the evolutionarily determined rate of reproduction in natural species tends to be balanced against the level of mortality. It is widely known that species that experience high mortality often have higher rates of reproduction than species that experience lower mortality. For example, the ostrich, that lives on the savanna in Africa where there are many predators, lay at least 5-11 eggs per clutch, whereas the kiwi, that belongs to the same order but which lives in New Zealand where there were no predators, lays only a single egg.

Also, among the terrestrial vertebrates on the northern hemisphere, northern races generally have higher reproductive rates than southern races. For example, the wren lays 3 eggs on Sicily, 5-6 in England, and 6-8 in Russia (Klomp, 1970). It has often been suggested that such trends exist because the rate of reproduction is adjusted to balance the rate of mortality that is expected to increase toward the polar regions. But according to the classical theory and Lack's clutch size this is generally not possible, and the widespread consensus among the evolutionists of today seems to be that natural selection does not balance reproduction against mortality (see Bulmer, 1994). That is to say, we have another paradox between the classical

theory of evolution and empirical evidence.

In the sections below I will, among other things, show that selection by density dependent competitive interactions implies that the maximal rate of reproduction is balanced against the rate of mortality. First, however, I will give a short summary of the classical approach and show why it fails on an evolutionary scale. At the end of the chapter I will summarise the evolutionary findings of the last three chapters in a table.

12.1 The classical theory and unlimited reproduction

When we want to predict the evolution of the reproductive rate from the classical framework we quantify trade-offs that exist between reproduction and either offspring or parent survival. There are plenty of reasons why a given individual cannot reproduce with an increased rate unless its, or its offspring's, survival will decline. For example, due to the evolutionary setting of the phenotype an offspring needs to obtain a certain size before it can survive and, thus, if there are too many offspring per female the parents cannot gather sufficient food, the offspring will be too small, and they will die at an increased rate. Also, if the parents invest too much energy in current reproduction their condition may decline and their mortality increase. In other words, due to purely physiological constraints we expect a trade-off between reproduction and both offspring and parent survival.

At the level of lifetime reproduction (R_m) and the survival scalar (p) the physiological trade-offs can be represented by describing p as functionally and inversely related to reproduction, e.g., as

$$p = ke^{-cR_m} \quad (12.1)$$

where k is a positive constant reflecting mainly extrinsically caused mortality and c is a positive constant describing the absolute value of the physiological trade-off. Fisherian fitness is then $r^* = \ln(ke^{-cR_m} R_m N^{*- \gamma})$, with $r^* = 0$ for an average variant. This implies that we have a fitness profile like the one shown in Fig. 12.1 and where the Fisherian optimum to the rate of reproduction is intermediate and given as

$$R_m^{*F} = 1/c \quad (12.2)$$

Then, by inserting R_m^{*F} into eqn 12.1, we find that the rate of mortality at the Fisherian optimum is

$$p^{*F} = k/e \quad (12.3)$$

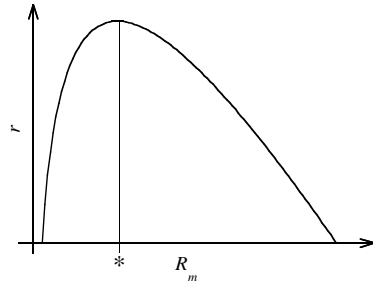


Fig. 12.1 A classical fitness profile for maximal lifetime reproduction (R_m) when the survival scalar p declines exponentially with R_m . The star indicates the Fisherian equilibrium.

Fitness profiles resembling the profile in Fig. 12.1 have been found in several species including birds like the common swift (Lack, 1954) and the great tit (Boyce and Perrins, 1987).

From Chapter 8 on fitness and selection we know that a one-dimensional fitness profile does not necessarily tell us anything about the evolutionary equilibrium, and this conclusion holds even though the profile has an apparent optimum. The reason for this is that the fitness profile describes only the differentiation in fitness within a population at a given moment in time and that this function is likely to change with evolutionary changes in the phenotype. This means that the optimum illustrated in Fig. 12.1 and given by eqns 12.2 and 12.3 may not indicate the evolutionary equilibrium.

To see that the prediction given by eqn 12.2 fails as an evolutionary prediction of R_m , recall that the essential trade-off behind the profile in Fig. 12.1 is the physiological trade-off given by eqn 12.1. That the trade-off is physiological implies that it belongs to the phenotype and, thus, natural selection will operate also on the trade-off. In other words, Fisherian selection is given by the partial derivatives

$$\begin{aligned}\partial r^*/\partial R_m &= 1/R_m - c & (12.4) \\ \partial r^*/\partial c &= -R_m\end{aligned}$$

This means that the intrinsic trade-off (c) continues to decline, while the Fisherian optimum to the reproductive rate ($R_m^{*F} = 1/c$) continues to increase. This increase could, e.g., be obtained through the continuous decline in body mass we predicted from the classical theory in Chapter 10. The continuous increase in R_m^{*F} is in perfect agreement with the fundamental theorem of natural selection and it is in perfect disagreement with natural conditions where highly limited rates of reproduction are widespread. That is to say, there seems to be no justification for Lack's hypothesis that

the reproductive rate evolves from a trade-off between reproduction and survival.

12.2 Competitive interactions and balanced reproduction

When we turn to selection by density dependent competitive interactions we find that a limited rate of reproduction evolves by selection independently of the physiological trade-offs between reproduction and survival. To see this recall that in the previous chapter we determined that the evolutionary limit to the population equilibrium is given as

$$N^{**} = e^{1/\psi\gamma_i} \quad (12.5)$$

In that chapter we also found that this limit is primarily in the sense that it is set by ecological constraints independently of the demographic traits. It is then through the population dynamic processes, which determine the population equilibrium, that the demographic traits are linked to the constraint of the evolutionarily determined population equilibrium. The population dynamic constraint defining this link is given by the well-known population equilibrium:

$$N^* = \sqrt[\gamma]{\lambda_m} \quad (12.6)$$

We can then combine the evolutionarily determined equilibrium (eqn 12.5) with the population dynamic equilibrium (eqn 12.6) and find that the maximal growth rate is

$$\lambda_m^{**} = e^{\gamma/\gamma_i\psi} \quad (12.7)$$

at the evolutionary equilibrium. It is due to this evolutionary boundary to the maximal growth rate that there is also an upper boundary to maximal lifetime reproduction. To obtain this evolutionary determinant of R_m , recall that $\lambda_m = pR_m$ at the population equilibrium. Hence, if we combine this constraint with eqn 12.7, we find that the evolutionary determinant of maximal lifetime reproduction is

$$R_m^{**} = e^{\gamma/\gamma_i\psi}/p \quad (12.8)$$

This means that the optimal rate of reproduction is inversely related to the rate of survival, and that it is given by the ecological constraints γ , γ_i and ψ independently of the physiological trade-offs that determine λ_m in the classical theory.

12.2.1 The evolution of Lack's optimum

We have now a situation where we have explained the evolution of limited reproduction independently of the classical selection mechanism that is based on the physiological trade-offs between the rate of reproduction and the rate of survival. In this subsection I will describe how we can superimpose the classical selection mechanism on top of this prediction in order to describe the evolutionary determinants of the absolute value of the physiological trade-off between reproduction and survival.

In order to understand this integration between the two theories let us first consider the classical hypothesis in its most limited version. Here, the classical hypothesis represents only the observation that the physiology is optimised so that energy is not wasted on traits that are irrelevant for fitness. As this observation holds independently of whether natural selection is based on competitive interactions or not we find that the physiological trade-off between reproduction and survival will be optimised in the sense that the maximal growth rate is optimal for a given investment of energy in reproduction. In other words, we expect that the population will be situated both at the Fisherian optimum, given by eqns 12.2 and 12.3, and at the evolutionary optimum, given by eqns 12.7 and 12.8.

In order to test the expectation that both equilibria apply let us examine if we can deduce Lack's optimum from the evolutionary equilibrium given by eqn 12.8. By this I mean if we can interpret the fitness profile in Fig. 12.1 as being the evolutionary result of the phenotypic optimum that is defined by the ecological constraints of eqn 12.8. This is possible if the parameter c , which defines the absolute value of the physiological constraint of eqn 12.1, is given by the ecological constraints of eqn 12.8.

If it is true that the physiological trade-off that defines Lack's optimum is defined by the evolutionary equilibrium I can deduce c from eqn 12.8, insert the obtained c into the trade-off that determines Lack's optimum (eqn 12.1), and obtain a true expression. If, on the other hand, the obtained expression is false, then the evolutionary equilibrium is inconsistent with Lack's optimum.

If we assume that Lack's optimum is given by the evolutionary equilibrium we have that $R_m^{*F} = R_m^{**}$, and that the survival scalar p in eqn 12.8 is identical to $p = ke^{-cR_m^{**}}$ as it is defined by eqn 12.1 at Lack's optimum. From this latter constraint and the $pR_m^{**}N^{**-\gamma} = 1$ constraint of the evolutionary population equilibrium we have that $ke^{-cR_m^{**}}R_m^{**}N^{**-\gamma} = 1$ and, thus, that

$$c = \ln(kR_m^{**}N^{**-\gamma})/R_m^{**} \quad (12.9)$$

From the evolutionary equilibrium we have $N^{**-\gamma} = e^{-\gamma/\gamma_i\psi}$ by eqn 12.5, and that R_m is given by eqn 12.8. If I insert these two expressions into

eqn 12.9 we find that the equilibrium estimate of c is

$$c^{**} = -\ln(kp)/R_m^{**} \quad (12.10)$$

When this estimate is inserted into the trade-off that defines Lack's optimum (eqn 12.1) we find that $p = p$. As this is true we can conclude that the physiological trade-off that defines Lack's optimum can be defined by the ecological constraints behind the evolutionary equilibrium of Malthusian relativity. The conclusion is then that Lack's optimum is consistent with the evolutionary equilibrium of Malthusian relativity.

Although Lack's optimum is consistent with the evolutionary equilibrium there are two major differences between the original proposal by Lack and the new version of Lack's optimum. The first difference is that the evolutionary causality is changed around between the two versions of the optimum: in the original version it is the maximal growth rate, the rate of survival, and the maximal rate of reproduction that evolve from the physiological trade-off between reproduction and survival, whereas in the new version it is the absolute value of the physiological trade-off that evolves from the optimal growth rate that is more fundamental and given by ecological constraints. The other difference is that in its original version Lack's optimum is evolutionarily unstable in the dimension of the fundamental trait, i.e., in the dimension of the physiological trade-off, whereas Lack's optimum is evolutionarily stable in the new version. This latter prediction is evolutionarily stable because the maximal growth rate is evolutionarily explained prior to its use as the fundamental trait upon which Lack's optimum depends.

12.2.2 Metabolic rate, resource quanta and production

Let us now consider whether the reproductive rate that is predicted by density dependent competitive interactions behave in resemblance with general expectations and observations.

If, e.g., we are interested in the evolutionarily determined relation between the rate of reproduction and the rate of production in the resource we can use eqn 11.10, i.e., $N^{**} = E^* e^{1/\gamma_e \psi}$, to describe the evolutionary setting of the population equilibrium. Then, as $E^* \propto r_e$, we have that $N^{**} \propto r_e$ and thus, if we follow the procedure for eqns 12.6 to 12.8 we find that

$$R_m^{**} \propto r_e^\gamma \quad (12.11)$$

That is to say, the maximal rate of reproduction is predicted to be higher in the more productive areas than in the less productive areas. This prediction depends upon the assumption that the less productive area is not so harsh that fluctuations in abiotic factors affect the reproductive success. In such

fluctuating environments the fluctuations will reduce the average rate of successful reproduction and, thus, an increased reproductive potential may evolve in order to maintain the same average reproductive success.

If, instead, we are interested in the effects that the metabolic rate (B) has on the reproductive rate we can use eqn 11.8, i.e., $N^* = e^{1/\gamma_i \psi} / B$, as the evolutionary determinants of the population equilibrium. Then, following the procedure for eqns 12.6 to 12.8 we find that

$$R_m^{**} \propto 1/B^\gamma \quad (12.12)$$

That is, maximal lifetime reproduction is inversely related to the metabolic rate raised to the power of the curvature in the density regulation. This prediction coincides with the observation that many poikilotherm vertebrates reproduce at a higher rate than homoiotherm vertebrates.

We may also consider the evolutionary relation between R_m and the resource quanta (Q) for which the individuals compete. This can be done directly from eqn 12.8 and the proportionality between ψ and Q that I described in Section 11.2.1. From these relations we find that

$$R_m^{**} \propto e^{1/Q} \quad (12.13)$$

Hence, if competition is for large quanta, like defensible territories, the evolutionarily determined rate of reproduction is low, while it is high when competition is for small and single resource items. This differentiation may in part explain some of the differentiation between pelagic and terrestrial systems where many pelagic fishes lay thousands of eggs while terrestrial vertebrates produce only relatively few offspring per lifetime. These differences are likely to reflect ecological differences because the polarisation in the reproductive rate is present also within the few phylogenetic lineages that contain both terrestrial and pelagic species. Marine turtles, for example, lay ≈ 200 eggs per annum whereas the two terrestrial species, which have a similar body size, lay only ≈ 10 eggs per annum (Wilbur and Morin, 1988).

The polarisation in the reproductive rate between pelagic and terrestrial animals is likely to reflect also other differences in the ecological conditions between pelagic and terrestrial systems. For example, it may reflect that the extrinsic imposed rate of mortality is higher in pelagic systems than in terrestrial systems, an expectation following from Section 11.2.1 where we predicted that the exploitation of the resource is expected to be higher in pelagic systems than in terrestrial systems. In the following subsection I will describe in more detail how this type of extrinsic imposed variation in the rate of mortality can explain variation in the rate of reproduction.

12.2.3 Reproduction balanced against mortality

From eqn 12.8 we have that the evolutionarily determined rate of reproduction is balanced against the extrinsic imposed level of mortality, i.e., we have that

$$R_m^{**} \propto 1/p \quad (12.14)$$

The evolutionary mechanism behind this relationship is slightly different from the mechanisms behind the three relations that we predicted in the previous subsection, and this is because the former relation depends upon the existence of an additional constraint. For the three relations that are described by the eqns 12.11, 12.12, and 12.13 the essential constraints that cause their evolution are the constraints that exist between the evolutionary and the population dynamic setting of the population equilibrium. In comparison to this we find that the evolutionary balance between maximal reproduction and the rate of mortality depends also upon the trade-off that exists between net lifetime reproduction (R_0) and mortality ($1-p$). That is to say that it is because of the definition $R_{0,m} = pR_m$ and the $\lambda_m = R_{0,m}$ constraint at the population equilibrium that we obtain $R_m^{**} \propto 1/p$ from eqn 12.8.

To get a more intuitive understanding of why the maximal rate of reproduction is balanced against mortality let us consider a population in evolutionary equilibrium. If the level of mortality is increased the population dynamic equilibration will cause the population equilibrium to decline below the evolutionary equilibrium. Then, intra-population interference will decline, and the extrinsic gradient in resource access will be less biased in favour of competitive quality inducing selection that allocates energy from competitive quality to numerical reproduction. This increase in reproduction will continue until the population equilibrium has reached the density of the evolutionary equilibrium. If, instead, the population in evolutionary equilibrium experiences a decline in mortality the density will rise, and the extrinsic gradient in resource access will become more biased in favour of competitive quality causing selection that allocates energy from numerical reproduction into competitive quality. In this way the rate of reproduction will be balanced against the level of mortality.

It is due to this balance between maximal reproduction and extrinsic mortality that populations can persist at balanced equilibria. If maximal reproduction is not balanced against mortality, then the population is likely to become extinct. This is because if the reproductive rate is too limited the population becomes extinct, because it cannot out-reproduce mortality, and if the reproductive rate is too high extinction may follow from over-exploitation.

The predicted balance between the maximal rate of reproduction and the rate of mortality is confirmed in Fig. 12.2 for 132 species of birds. The

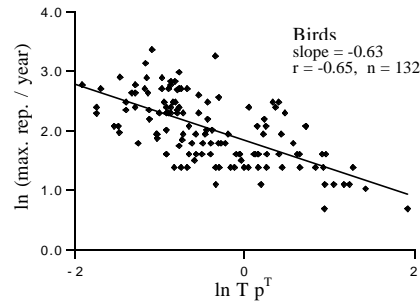


Fig. 12.2 Maximal yearly reproduction against time scaled survival (Tp^T) among 132 species of birds when plotted on double logarithmic scale. In this figure T is age at maturation, p adult annual survival, and maximal yearly reproduction (R_y) is calculated as the maximal clutch size times the number of clutches per year. The negative correlation between $\ln Tp^T$ and $\ln R_y$ confirms the prediction from eqn 12.14. This is because p^T is an estimate of the survival scalar in eqn 12.14 and because R_m is proportional to TR_y because the age at maturity (T) is proportional to lifespan (Peters, 1983; Calder, 1984). Data from Cramp and Simmons (1977-1983), Cramp (1985-1992), and Cramp and Perrins (1993).

predicted balance has been observed also on a smaller scale between different populations of guppies. Here the individuals from localities with a high predation rate (high predation localities) have a higher reproductive rate than the individuals from localities with a low predation rate (low predation localities) (Reznick et al., 1996). Introduction experiments for this species have shown also that guppies that are transferred from a high predation locality to a low predation locality evolve a significantly lower rate of reproduction (Reznick and Bryga, 1987; Reznick et al., 1990).

From the classical theory that is based on Lack's clutch size we do not expect a positive relationship between the rate of reproduction and the rate of mortality (see Bulmer, 1994). Instead, in the classical theory it is differential mortality, and not the absolute rate of mortality, that can select for differences in the rate of reproduction (Gadgil and Bossert, 1970; Charlesworth, 1994). This implies that the positive relationship between the rate of reproduction and the rate of mortality can evolve only when it predominantly is the individuals with a low rate of reproduction that die when the rate of mortality is high, while it predominantly is the individuals with a high rate of reproduction that die when the rate of mortality is low. Due to intrinsic and physiological constraints between reproduction and body mass this type of selection might operate indirectly in the way that age- or size-structured mortality may generate evolutionary differences in the rate of reproduction. In this latter case an increase in juvenile mortality

Table 12.1 Three major traits (body mass, population density, and reproductive rate) as evolutionarily determined by the primary production (r_e), the survival rate (p), the metabolic rate (B), the resource quanta (Q), and the genetic variation in competitive quality (σ).

Trait	r_e	p	B	Q	σ
Body mass	r_e	p	-	$e^{-1/Q}$	$e^{-\sigma}$
Density	r_e	p^0	$1/B$	$e^{1/Q}$	e^σ
Reproduction	r_e^γ	$1/p$	$1/B^\gamma$	$e^{1/Q}$	e^σ

rates may favour delayed maturity and a reduced rate of reproduction.

At first it was thought that the classical hypothesis with differentiated mortality rates could explain the differences in the reproductive rates of guppies (e.g., Reznick and Bryga, 1987; Reznick et al., 1990). It was assumed that the predators at the high predation localities selected preferentially on the large and adult guppies, while the predators at the low predation localities predominantly ate the small and juvenile guppies. A recent study of Reznick et al. (1996) that was designed to test this assumption did, however, fail to support the classical hypothesis because it did not detect the presence of differential mortality. Instead, this study supported the hypothesis of selection by density dependent competitive interactions because it confirmed that the mortality rate is significantly higher in the high predation localities than in the low predation localities.

In Table 12.1 I have summarised the predictions we have made during the last three chapters from the selection mechanism of density dependent competitive interactions.

Chapter 13

Body mass allometries

IN THE PRECEDING chapters I described the evolution of the different traits relatively independently of one another. In this chapter, which is adopted from Witting (1995), I use the constraints of the foraging process to deduce the across-species relationships existing among the different traits. In order to relate the theoretical results to empirical evidence I take an allometric approach and describe the traits (Y) as power functions of body mass (w), i.e., as $Y \propto w^k$, where the exponent k is the unknown parameter I deduce for each trait. We can then compare the theoretically predicted exponents with the exponents of the empirical across-species allometries that are reviewed in books by Calder (1984), Peters (1983), and Reiss (1989).

The empirical exponents have been estimated by linear regression as it is illustrated in Fig. 13.1 for the home-range area and the population density in mammals. When rounded, these exponents are respectively 1 for the home-range area (Schoener, 1968; Turner et al., 1969; Harestad and Bunnell, 1979; Calder, 1984) and $-3/4$ for the population density (Damuth, 1981, 1987). Among the most famous exponents there are the positive $1/4$ exponent for lifespan (Bonner, 1965), the negative $1/4$ exponent for the maximal growth rate in the population density (Fenchel, 1974), and the negative $1/4$ exponent for the metabolic rate per unit body mass (Kleiber, 1932).

A number of mechanisms have been proposed in order to explain the empirical exponents, and these proposals have generally been based on the assumption that it is the exponent for the metabolic rate that is most fundamental. In 1883 Rubner proposed that this exponent is explained by the surface rule that was meant to cause thermal homeostasis. However, the surface rule predicts an exponent of $2/3$, while the observed exponent *is* $3/4$ (Kleiber, 1932). Blum (1977) pointed out that the surface rule actually predicts $3/4$, if the world has four spatial dimensions instead of three. But,

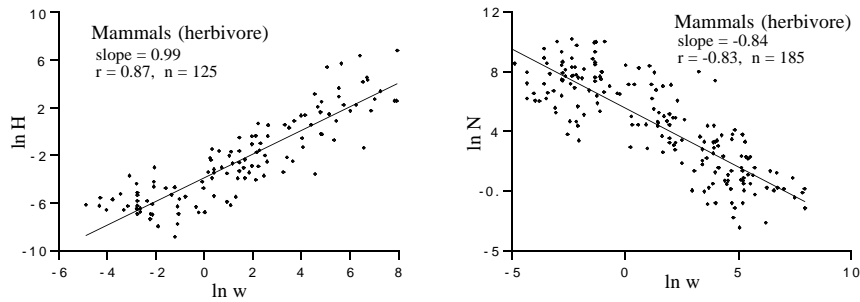


Fig. 13.1 The body mass (w) allometries for home-range area (H) and population density (N) in mammals. Data from Nowak (1991).

the fourth dimension remains to be discovered. McMahon (1973, 1975) argues that the metabolic rate is proportional to muscle cross-sectional area, that due to elastic similarity is expected to be proportional to the 3/4th power of body mass. However, “body support for animals other than the hoofed mammals does not conform to the elastic similarity model” (Calder, 1984:85; see also LaBarbera, 1986). More recent work, which includes Reiss (1989), Charnov (1993), and related studies that are reviewed by Calder (1984) and Peters (1983), have been restricted to the deduction of some allometric exponents from other allometric exponents and, thus, this work does not attempt to explain why 3/4? As LaBarbera (1986:79) concludes, “at present there is no general explanation for the 3/4 mass exponent for metabolic rate; the most all-encompassing of design generalities in biology must, at present, be treated simply as an empirical fact.”

In this chapter I will show that the empirical exponents are explained primarily by the constraints by which the foraging individuals encounter one another together with the constraints by which the exploitation of an individual inhibits the foraging of that individual. Both these processes regulate the foraging efficiency downward from the expectation of an ideal free distribution where a uniform resource is exploited evenly and the individuals do not interfere. Moreover, the regulation of these two processes are inversely related. That is, to avoid regulation by interference competition all individuals should forage in small home-ranges that do not overlap so that the individuals do not encounter one another. On the contrary, to avoid self-inhibition the individuals should exploit infinitely large home-ranges so that the individuals do not cover the same area more than once. This implies that there is an intermediate home-range size that will be maintained by natural selection because the foraging efficiency is optimal at that home-range size.

According to the constraints of the home-range optimum we will find

that the well-known exponents $\pm 1/4$ and $\pm 3/4$ apply only to organisms that forage in two dimensions, while the exponents are $\pm 1/6$ and $\pm 5/6$ for organisms that forage in three dimensions, and $\pm 1/2$ for organisms foraging in one dimension. These differences imply that we have an extra dimension in which we can test the allometric deduction. That is to say that the proposed mechanism might provide the true explanation if terrestrial organisms, which forage in two dimensions, conform to the two-dimensional prediction while pelagic organisms, which have an extra vertical dimension in which to forage and interact, conform to the three-dimensional prediction.

In the sections below I use the allometric traits to describe the constraints of respectively self-inhibition and regulation by intra-population interference. Hereby I can optimise the foraging process with respect to the home-range area and combine the constraints of the foraging process with constraints at other levels in order to deduce the unknown exponents in the body mass allometries. In this process I will assume that the population is in evolutionary equilibrium and in population dynamic equilibrium, and I will not use superscripts to indicate the occurrence of equilibria.

13.1 Foraging self-inhibition

To formulate the allometric constraint associated with the process of self-inhibition let us consider foraging as it occurs within a home-range. In this instance the organism will forage along some more or less well defined foraging tracks, and the length (L) of these tracks is expected to be proportional to the d th root of the d -dimensional home-range (H), i.e., $L \propto H^{1/d}$, with $d \in \{1, 2, 3\}$. At least for mammals this expectation is not falsified since the length of their foraging bouts scale to the square root of their home-range area (Garland, 1983; Calder, 1984). The time interval between track reuse (T_V) is then the track distance (L) divided by the foraging speed (V):

$$T_V = L/V \propto H^{1/d}/V \quad (13.1)$$

On the body mass axis the foraging speed is proportional to lifespan (Garland, 1983; Calder 1984) and, thus, the time interval between track reuse scales as

$$T_V \propto H^{1/d}/T \quad (13.2)$$

The availability of food along the foraging track can then be considered to be proportional to the time interval between track reuse, and this is because the longer the time period between foraging events the more time there is available for the resource to regrow or to disperse into the foraged area. Because of this regrowth/dispersal delay we find that the frequency of track

reuse ($1/T_V$) will describe the degree to which an individual will inhibit its own foraging.

In eqn 13.1 the time interval between reforaging is described as an absolute measure in the sense that it is given in absolute time. This assumption will, however, not hold, and this is because self-inhibition is a term that is relative to the situation where no individuals reuse their foraging tracks, i.e., a term that is defined relative to the situation with infinitely large home-ranges. In this situation with no self-inhibition the resource is re-harvested with a given frequency. This means that in order to describe self-inhibition by the time interval between track reuse (T_V) we need to scale this interval by the interval between resource re-harvesting when the home-ranges are infinitely large. From the principle of physiological time (Brody, 1945; Calder, 1984) we may expect that the time available for regrowth/dispersal in the absence of self-inhibition is inversely proportional to the metabolic rate per unit body mass, i.e., proportional to lifespan (T). That is, smaller organisms are expected to re-harvest an area at a faster pace than larger organisms are. Accepting this scaling the scaled T_V is

$$T_{V,S} \propto H^{1/d}/T^2 \quad (13.3)$$

The foraging efficiency in the presence of self-inhibition can then be described as

$$\kappa = \alpha f(H^{1/d}/T^2) \quad (13.4)$$

where f is the self-inhibition function that is expected to be convex and to increase monotonically from zero to unity as the home-range size increases from zero to infinity (Fig. 13.2a). Hence, in the absence of intra-population interference we find that foraging is optimal in infinitely large home-ranges where self-inhibition is absent.

13.2 Intra-population interference

To formulate the allometric constraint associated with interference competition let us consider the rate at which the different individuals encounter one another. If the home-ranges are non-overlapping the individuals cannot meet and the probability that two individuals will encounter one another is zero. More generally the probability of an encounter between two individuals will be proportional to the overlap of their home-ranges. This overlap (O) can be described as the average home-range (H) divided by the per capita availability of space ($1/N$), i.e., as

$$O \propto HN \quad (13.5)$$

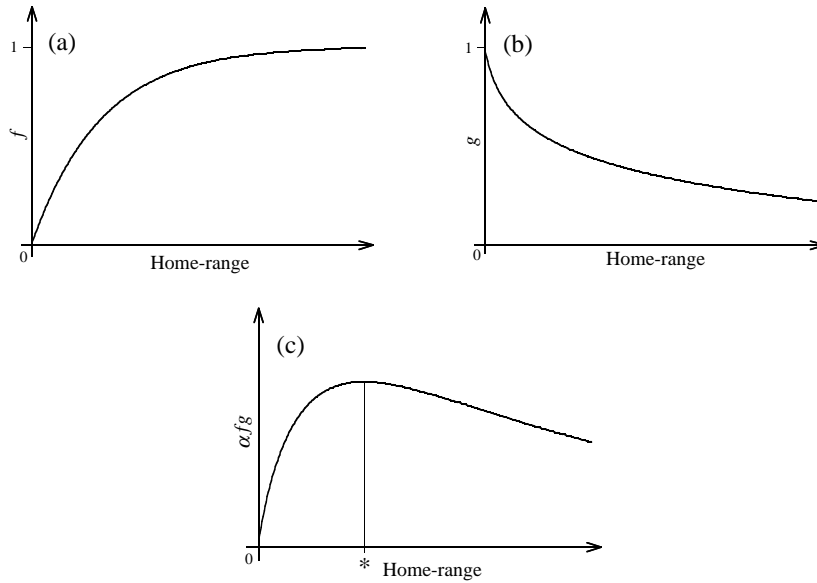


Fig. 13.2 Self-inhibition (a), interference regulation (b), and realised foraging (c) against the home-range. The star indicates the optimal home-range.

where both the home-range and the population density (N) is measured in d dimensions.

The level of interference also depends upon the rate of foraging, and this is because the faster the foraging tracks are covered the faster the individuals will encounter one another. In other words, the number of competitive encounters per individual per unit time is proportional to the frequency at which the foraging tracks are reused and, thus, remembering eqn 13.5, we find that the number is proportional to the home-range overlap divided by the time interval between track reuse (eqn 13.2). Hence, the level of interference is proportional to

$$I \propto TH^{(d-1)/d}N \tag{13.6}$$

The number of competitive encounters also depends upon the density of the resource. If the resource density is low the individual will have to cover its foraging tracks at a fast rate to find sufficient resource, whereas if the resource density is high the individual need hardly move in order to find sufficient resource. The level of interference is then expected to be inversely proportional to the resource density and, thus, we obtain

$$I = TH^{(d-1)/d}N/E \tag{13.7}$$

From Chapter 7 we have that regulation by intra-population interference is given by the function $g(I)$, and this function is inversely related to self-inhibition in the sense that interference regulation is absent when the home-ranges are infinitely small, while it is at its maximum when the home-ranges are infinitely large (Fig. 13.2b). Hence, in the absence of self-inhibition we find that foraging is optimal in infinitely small home-ranges where the individuals do not encounter one another. When, instead, we take into consideration the simultaneous action of both intra-population interference and self-inhibition, then we find that the realised foraging efficiency takes its optimal value at some intermediate home-range size (Fig. 13.2c).

13.3 The allometric deduction

We have now enough information to deduce the allometric exponents for resource density (E), lifespan (T), home-range (H), population density (N), and the exploitation efficiency (α). To do this let these traits be given by the following power functions: $E \propto w^e$, $T \propto w^t$, $H \propto w^h$, $N \propto w^n$, and $\alpha \propto w^a$. We can then combine the constraints of the foraging process with constraints at other levels and obtain five equations from which the five unknown exponents can be deduced.

We obtain the first equation from Chapter 10, where it is given from the prediction that the level of interference is body mass invariant when the body mass is in evolutionary equilibrium. As the level of interference is $I = TH^{(d-1)/d}N/E$ by eqn 13.7, this invariance gives us the following equation

$$t + h(d-1)/d + n - e = 0 \quad (13.8)$$

We obtain the second equation from the optimum of the realised foraging efficiency: As shown in the first section in the appendix to this chapter this foraging optimum imposes the constraint that the level of self-inhibition is body mass invariant. As the level of self-inhibition is defined as $f(H^{1/d}/T^2)$, by eqn 13.4, this invariance gives us the following equation

$$h/d - 2t = 0 \quad (13.9)$$

We obtain the third equation from the constraints on the exploitation of the resource: From eqn 7.30 the resource density at the equilibrium is $E = E_m - \kappa N/\gamma_e$. Then, as by definition we have that $E_m \propto w^0$ and that $\gamma_e \propto w^0$ we find that $E \propto \kappa N$ and, thus, it follows that $E/\kappa N \propto w^0$. If into this equation we insert $\kappa = \alpha fg$, with $f \propto w^0$ and $g \propto w^0$, we find that

$$e - a - n = 0 \quad (13.10)$$

The fourth equation is obtained by combining the constraint on the level of interference with the constraint on the exploitation of the resource: Let, $I = 1/ZE$ with $Z = 1/TH^{(d-1)/d}N$. We can then insert $E = E_m - \alpha fgN/\gamma_e$ into $1/I = EZ \propto w^0$ and obtain $E_m Z - \alpha fgNZ/\gamma_e \propto w^0$, from which it follows that $E_m \propto \alpha fgN/\gamma_e$. If into this equation we insert $E_m \propto w^0$, $\gamma_e \propto w^0$, $f \propto w^0$, and $g \propto w^0$ we obtain $\alpha N \propto w^0$ from which it follows that

$$n = -a \quad (13.11)$$

The fifth equation is obtained from the demographic constraint at the population equilibrium: At equilibrium we have that $p\alpha fgE/wB = 1$ and, as $p \propto w^0$ is a reasonable assumption (Fig. 7.1), the scaling of this expression reduces to $T\alpha E/w \propto w^0$, since $T \propto 1/B$ and $f \propto g \propto w^0$. Then, from $T\alpha E/w \propto w^0$, we have

$$t + a + e = 1 \quad (13.12)$$

We now have the five equations (eqns 13.8 to 13.12) with the five unknown exponents: e, t, h, n , & a . These equations are solved in the second section in the appendix to this chapter, and in the third section these solutions are combined with additional constraints to deduce the allometric relations for maximal lifetime reproduction, the maximal growth rate, the biomass of the population, the amount of energy that is metabolised per population, and the level of social behaviour when it is defined by the de-

Table 13.1 The exponents of the body mass allometries as theoretically deduced for mobile organisms that forage in one (1D), two (2D), three (3D), and d (dD) spatial dimensions. α : Exploitation efficiency. B : Metabolic rate per unit body mass. T : Lifespan. N : Population density given in d -dimensions ($d \in \{1, 2, 3, d\}$). H : Home-range size in d -dimensions. U : Energy used per population. M : Biomass. S : Armitage's sociality index. R_m : Maximal lifetime reproduction. r_m : Maximal rate of increase in population.

Trait	1D	2D	3D	dD
α	1/2	3/4	5/6	$(2d - 1)/2d$
B	-1/2	-1/4	-1/6	$-1/2d$
T	1/2	1/4	1/6	$1/2d$
N	-1/2	-3/4	-5/6	$(1 - 2d)/2d$
H	1	1	1	1
U	0	0	0	0
M	1/2	1/4	1/6	$1/2d$
S	1/2	1/4	1/6	$1/2d$
R_m	0	0	0	0
r_m	-1/2	-1/4	-1/6	$-1/2d$

Table 13.2 The allometric exponents for the relationship between metabolic rate and body mass as observed within mobile chordates. The estimated exponents are grouped according to whether they resemble the exponent deduced for organisms that forage in two ($2D$) or three ($3D$) dimensions. The observed exponents are estimated by linear regression on double logarithm scale.

Group	$2D$	$3D$
Deduced	0.75	0.83
Mammals ¹	0.74	
Bats ²	0.74	
Birds ³	0.74	
Reptiles ⁴	0.76	
Snakes ⁵	0.74	
Lizards ⁶		0.82
Turtles ⁷		0.86
Amphibians ⁸	0.77	
Frogs ⁹	0.71	
Salamanders ¹⁰		0.82
Freshwater fishes ¹¹		0.81
Marine fishes ¹¹		0.79
Lampreys ⁸		0.81
Lancelets ⁸		0.91

¹Stahl (1967). ²McNab (1969) and Konoplev et al. (1978). ³Calder (1974). ⁴Kayser and Heusner (1964), Bennett and Dawson (1976), and Zotin and Konoplev (1978). ⁵Bennett and Dawson (1976). ⁶Bennett and Dawson (1976) and Bartholomew and Tucker (1964). ⁷Bennett and Dawson (1976) and Kayser and Heusner (1964). ⁸Zotin and Konoplev (1978). ⁹Hutchinson et al. (1968). ¹⁰Whitford and Hutchinson (1967) and Feder (1976). ¹¹Winberg (1960).

gree of home-range overlap. The solutions to these allometric exponents are summarised in Table 13.1. From the table it is apparent that some of the exponents are given as a function of the number of spatial dimensions in which the organism forages. More accurately, the well-known exponents $\pm 1/4$ and $\pm 3/4$ apply only to organisms that forage in two dimensions, while these exponents are $\pm 1/6$ and $\pm 5/6$ for organisms that forage in three dimensions, and $\pm 1/2$ for organisms that forage in one dimension.

13.4 Empirical evidence

It is the allometric relationship between the metabolic rate and body mass that has been studied in greatest detail, and this relation is listed in Table 13.2 for a variety of mobile chordates. From the table it is apparent that the metabolic exponents for almost all terrestrial taxa resemble the

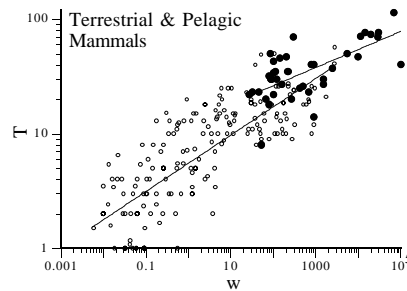


Fig. 13.3 The body mass (w) allometry for lifespan (T) among 195 species of terrestrial mammals where the exponent is 0.25 ± 0.04 (SE) (small open circles), and among 40 species of pelagic mammals (taxa Cetacea, Pinnipedia, and Sirenia) where the exponent is 0.16 ± 0.02 (SE) (large solid circles). Data from Nowak (1991).

theoretical deduction for foraging in two dimensions, while the metabolic exponents for all pelagic taxa resemble the theoretical deduction for foraging in three dimensions. This overall separation is likely to reflect the fact that most terrestrial vertebrates are constrained to foraging in the two horizontal dimensions while pelagic organisms have an extra vertical dimension in which to forage and interact. As shown in Fig. 13.3 this separation also exists between the terrestrial and the pelagic mammals.

Other interesting patterns that emerge from Table 13.2 include that the spatial dimensionality of the behaviour in birds and bats apparently is restricted mainly to the two horizontal dimensions, although these taxa are capable of vertical movements. Also, frogs seem to resemble two dimensions, while salamanders apparently conforms to three dimensions. This separation may arise because salamanders tend to be more dependent upon ponds and lakes than frogs. Likewise, turtles are more dependent upon ponds, lakes, and rivers than are snakes. The observed exponent for turtles resembles three dimensions, while the observed exponent for snakes resembles two dimensions. It does however remain unclear why the metabolic exponent of lizards apparently conforms to a three-dimensional system. These latter separations should not be taken too literally because the empirical studies were not conducted to distinguish three-dimensional systems from two-dimensional systems.

Among invertebrates the exponent for the metabolic rate lies between 0.71 and 0.85 (reviewed Peters 1983) as it is expected for organisms foraging in either two or three dimensions. Metabolic exponents that resemble a two-dimensional system have been found in terrestrial taxa like insects [0.76 by Zontin and Konoplev (1978)], spiders [0.71 by Greenstone and

Table 13.3 The deduced ($2D$) and observed exponents for the body mass allometries for terrestrial vertebrates. The abbreviations resemble those in Table 13.1.

Trait	$2D$	Mammals	Reptiles	Birds
B	-0.25	-0.26 ¹	-0.24 ⁹	-0.26 ¹²
T	0.25	0.25 ²		0.18 ¹³
N	-0.75	-0.78 ³	-0.77 ¹⁰	-0.75 ¹⁴
H	1.00	0.99 ⁴	0.95 ¹¹	1.16 ¹⁵
U	0.00	-0.08 ⁵		
S	0.25	0.22 ⁶		
R_m	0.00	-0.03 ⁷		0.00 ¹⁶
r_m	-0.25	-0.27 ⁸		-0.14 ¹⁷

¹Stahl (1967). ^{2,4,6,7,8}Data from Nowak (1991). ^{3,5}Damuth (1987). ⁹Kayser and Heusner (1964), Bennett and Dawson (1976), and Zotin and Konoplev (1978). ¹⁰Peters (1983). ¹¹Turner et al. (1969). ¹²Zar (1969). ^{13,16,17}Data from Cramp and Simmons (1977-1983), Cramp (1985-1992), and Cramp and Perrins (1993). ¹⁴Nee et al. (1991). ¹⁵Schoener (1968). The number of species involved in each estimate are ¹349, ²235, ³467, ⁴125, ⁵63, ⁶210, ⁷96, ⁸174, ⁹128, ¹⁰11, ¹¹29, ¹²130, ¹³242, ¹⁴147, ¹⁵75, ¹⁶221 and ¹⁷221.

Bennett (1980)], and moths [0.78 by Bartholomew and Casey (1978)]. In comparison, it can be mentioned that metabolic exponents that resemble the three-dimensional picture have been found in pelagic taxa like unicells [0.83 by Robinson et al. (1983)] and unicellular algae [0.90 by Banse (1976)].

For any group of organisms it is the terrestrial vertebrates that have been subjected to far the most detailed allometric studies. The estimated exponents from some of these studies are listed in Table 13.3. For all the traits in the table it can be concluded that a reasonable resemblance exists between the deduced and the observed exponents.

The deduced exponents are based on the assumption that interactions among the individuals of different species are insignificant. This assumption is expected to be fulfilled for the empirical exponents in the present study, and this is because these exponents have been estimated at geographical scales above communities and among species independently of competitive guilds. In comparison, when the allometric exponents are empirically established at smaller scales, as within competitive guilds, deviations have been found from the exponents that are listed in the present study. For example, within the genae and tribes of British birds Nee et al. (1991) found a positive exponent for the population density allometry, whereas the exponent decreased to the expected $-3/4$ when the same species were compared across larger taxonomic units. The positive relation within competitive guilds is probably the result of inter-specific interference competition where the individuals of the larger species monopolise essential resources.

13.5 Appendix

13.5.1 The foraging optimum

In this section I deduce a constraint that is associated with the optimum in the realised foraging efficiency. From Section 13.1 and Section 13.2 on self-inhibition and intra-population interference we have that the realised foraging efficiency is

$$\kappa = \alpha f(H^{1/d}/T^2)g(TH^{(d-1)/d}N/E) \quad (13.13)$$

As shown in Fig. 13.2c, this efficiency has an optimum at an intermediate home-range. We can determine this optimum by differentiating the realised foraging efficiency (eqn 13.13) with respect to the home-range, and by setting the derivative equal to zero. To do this let

$$f(X) = f(H^{1/d}/T^2) \quad (13.14)$$

then, we obtain

$$\begin{aligned} \partial\kappa/\partial H &= (\alpha/d)H^{(1-d)/d}T^{-2}f'(X)g(I) \\ &+ [\alpha(d-1)/d]H^{-1/d}TNE^{-1}f(X)g'(I) \end{aligned} \quad (13.15)$$

where f' and g' are the derivatives of f and g with respect to X and I . Now, set $\partial\kappa/\partial H = 0$. Then, from eqn 13.15 we find

$$\frac{f(X)}{f'(X)} + \frac{g(I)}{g'(I)} \frac{1}{d-1} \frac{EH^{(2-d)/d}}{T^3N} = 0 \quad (13.16)$$

From eqn 10.23 we have that at the level of interference at the evolutionary equilibrium is expected to be body mass invariant, and this implies that $g(I) \propto w^0$. Thus, the allometric scaling of the fraction $EH^{(2-d)/d}/T^3N$ in eqn 13.16 will remain the same if it is multiplied by $I = TH^{(d-1)/d}N/E$. For $T \propto w^t$, $H \propto w^h$, $N \propto w^n$, and $E \propto w^e$, the exponent of this product ($IEH^{(2-d)/d}/T^3N$) is

$$\begin{aligned} &t + h(d-1)/d + n - e + e + h(2-d)/d - 3t - n \\ &= h/d - 2t \end{aligned} \quad (13.17)$$

This exponent is equal to the exponent of X (eqn 13.14) and, thus,

$$X \propto EH^{(2-d)/d}/T^3N \quad (13.18)$$

Then, because $g(I)/g'(I)$ is body mass invariant eqn 13.16 is reduced to

$$f(X)/f'(X) - \kappa X = 0 \quad (13.19)$$

where k is a positive constant. Recall that f is a convex and monotonically increasing function of X . Consequently, f/f' is monotonically increasing with X . Then, at the most, eqn 13.19 can have two roots and thus only one optimum with a limited home-range. As individual selection will optimise the foraging efficiency it will maintain the individuals at the home-range optimum implying that there is selection for a body mass invariant X .

13.5.2 The solution to five allometric equations

In this section I derive the solutions to the five unknown exponents e, t, h, n , & a that are contained in eqns 13.8 to 13.12. From $n = -a$ (eqn 13.11) and $e - a - n = 0$ (eqn 13.10) we have

$$e = 0 \quad (13.20)$$

From $h/d - 2t = 0$ (eqn 13.9) it follows that

$$h = 2td \quad (13.21)$$

When this $h = 2td$ is inserted into eqn 13.8 together with $e = 0$ (eqn 13.20) we get $t + 2t(d - 1) + n = 0$ and, thus,

$$t = n/(1 - 2d) \quad (13.22)$$

Inserting this $t = n/(1 - 2d)$, $a = -n$ (eqn 13.11), and $e = 0$ (eqn 13.20) into eqn 13.12 we have

$$n/(1 - 2d) - n = 1 \quad (13.23)$$

which can be solved for n

$$n = (1 - 2d)/2d \quad (13.24)$$

Then, from eqns 13.11 and 13.24

$$a = (2d - 1)/2d \quad (13.25)$$

From eqns 13.22 and 13.24 we have

$$t = 1/2d \quad (13.26)$$

From $B \propto w^b$, $B \propto 1/T$, and eqn 13.26 we have

$$b = -1/2d \quad (13.27)$$

From eqns 13.21 and 13.26 we have

$$h = 1 \quad (13.28)$$

13.5.3 Additional allometries

In this section I use the results from eqn 13.20 and eqns 13.24 to 13.28 as a set of basic allometric exponents from which I can deduce the exponents of other body mass allometries.

Because the population density scales as $N \propto w^{(1-2d)/2d}$ (eqn 13.24), and because the metabolic rate per unit body mass scales as $B \propto w^{-1/2d}$ (eqn 13.27), we find that the energy that is metabolised per population (U) is body mass invariant, i.e.

$$U = NBw \propto w^{(1-2d)/2d} w^{-1/2d} w^1 \propto w^0 \quad (13.29)$$

and that the biomass (M) of the consumer organism will scale as

$$M = Nw \propto w^{(1-2d)/2d} w^1 \propto w^{1/2d} \quad (13.30)$$

Lifetime reproduction is maximal at the limit $N \rightarrow 0$, where $E \rightarrow E_m$, $f \rightarrow 1$, and $g \rightarrow 1$. Then, as maximal lifetime reproduction is given as $R_m = \alpha E_m / wB$ we find that it will scale as

$$R_m \propto \alpha / wB \propto w^{(2d-1)/2d} w^{-1} w^{1/2d} \propto w^0 \quad (13.31)$$

Hence the maximal rate of increase in the population will scale as

$$r_m = \ln(pR_m) / T \propto w^{-1/2d} \quad (13.32)$$

Sociality has been defined as the “state of group formation when members of a population” . . . “have markedly overlapping home ranges” (Armitage, 1981). Hence, Armitage’s sociality index (S) is given by the home-range overlap

$$S \propto HN \propto w^1 w^{(1-2d)/2d} \propto w^{1/2d} \quad (13.33)$$