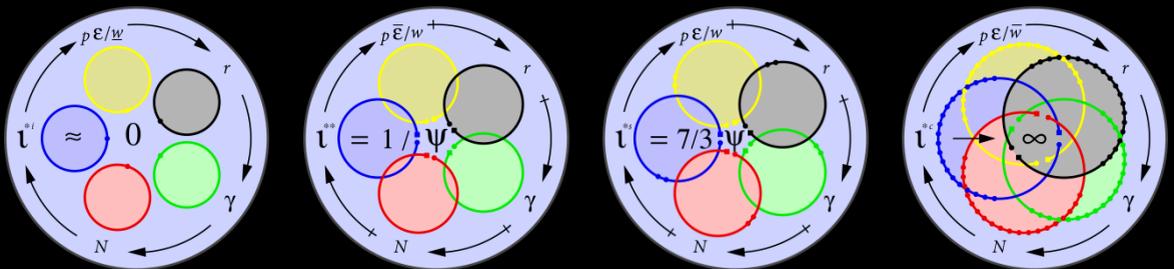


A General Theory of Evolution

By Means of Selection by Density Dependent Competitive Interactions



Lars Witting

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To life

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Preface

SINCE THE early 1990s a large number of books have been published on evolutionary ecology reviewing the major theoretical achievements in this field during the last half century (e.g., Rose, 1991; Roff, 1992; Stearns, 1992; Williams, 1992; Charnov, 1993; Andersson, 1994; Bulmer, 1994; Charlesworth, 1994; Crozier and Pamilo, 1996). From these reviews it is apparent that the classical theories have been established relatively independently of one another, and that they treat evolution by natural selection as an optimisation process that has now been described to the extent of a mature theory covering the major traits of the organism. Unfortunately, as shown in this book, these classical theories do not explain the major evolutionary trajectories that have occurred on Earth, and even more unfortunately the classical predictions are evolutionarily unstable in their phenotypic assumptions.

In this book I have integrated the classical theories with the selection pressure of density dependent competitive interactions, and I have done this to avoid the classical paradox of evolutionarily unstable optima. The result is a radically new theory containing the classical equilibria, but based on a new causality. This theory leads to deterministic (directional) evolution, in contrast to classical Darwinism that is based on historical (non-directional) evolution. In consequence, this book contains the first theory of natural selection suggesting that self-replicating molecules automatically evolve toward the complex organisms on Earth.

At first the readers who are educated in the classical theories might find that my theory is entirely crazy because I argue that the selection pressure is propagated through the population in a direction that is opposite to the direction in classical theories. Nevertheless, the interested reader should give my theory a second thought, a thought that is based on the fact that if we take the traditional approach to evolutionary biology and focus on equilibrium predictions, then the critical scientists would generally be unable to detect whether it is the classical theory or my theory that provides the correct description of evolution by natural selection. In other

words, in order to test the two theories we need to make comparisons at higher levels than traditionally done, and it is at these levels, beyond the classical approach, that the proposed theory is superior to the classical theories of natural selection.

Altogether, the present study suggests that a major change is a necessity in order to obtain a consistent theory of evolutionary biology. In order to anticipate that such a change is indeed needed I have chosen to write this book in a form where it may be useful as a text book in theoretical evolutionary ecology. I have aimed at this by assuming only some familiarity with basic calculus, and by proceeding through a successive construction of the whole theory starting from the principle of the Malthusian law. I have also aimed at making the book readable without reading the mathematics so that the less mathematically minded should be able to follow the essentials of my arguments. I might have failed in both cases, but at least I have done my very best.

For those of my readers who wonder why my study is published in this non-prestigious way there is only to say that the resistance against my theory was too great among the scientists who control the established scientific literature. This is probably best illustrated by the comments made by a highly established university press: “this book was very interesting but the innovative nature of the ideas would make the research community resistant” . . . “In spite of the interesting content, we would not be able to have such a work passed by our editorial board”. Of approximately thirty different submissions of my theory and parts of it to established journals and publishers it was only the deduction of the body mass allometries (Witting, 1995), which is not in itself controversial, that has been accepted for publication. The rest of the studies were rejected for publication even though they were often considered very important, and even though not one single reviewer could detect one single flaw affecting my conclusions. Despite this lack of firm scientific critique, many of the anonymous reviewers did not hesitate to argue against the publication of my studies, and in all instances they succeeded in convincing the editor not to publish them. For further details on the peer-reviews of my studies see the homepage at <http://www.peregrine.dk>.

As my theory has floated around in the scientific community in unpublished versions for quite a while, let me set the record straight on the dates where the different parts were released: The part on the evolution of body mass including the directional change was first submitted for publication on May 11, 1994, and the parts on population dynamics on October 27, 1994. Both of these studies were first presented in public 4-8 September 1995 at the Fifth Congress of the European Society for Evolutionary Biology in Edinburgh, together with some essential parts on the evolution of sexual repro-

duction. In the complete form the theory was first submitted for publication in June 1995, and it was first presented in public in August 1996 at the Fifth International Congress of Systematic and Evolutionary Biology in Budapest, and at the same time some of the essential conclusions were distributed to the members of the mailing list evoldir@evol.biology.mcmaster.ca.

I want to thank Lev R. Ginzburg at Stony Brook for many stimulating discussions that in the early 1990s turned my thoughts to the field of evolutionary ecology, and I am grateful also to my earlier supervisor Volker Loeschcke at Aarhus University. I also want to thank Bernt Guldbrandtsen and reviewers for helpful comments, John Maynard Smith for being the only reviewer who did not remain anonymous, Richard Barlach for checking my English, and the Department of Ecology and Genetics at Aarhus University for providing a desk during most of the period I used on this study.

Lars Witting
Århus, January 1997

Prologue

Chapter 1

Introduction

SINCE DARWIN rejected Lamarck's notion that organisms have an inherent tendency to climb the ladder of nature evolutionists have agreed that, on the theoretical side, there is no reason to expect that evolution is directional generating an increase in complexity with time. Nevertheless, although there is much noise on smaller scales, evidence actually suggests that large scale evolution is directional. Together with the empirical allometries Cope's law (Cope, 1887) suggests that mobile organisms in a stable environment continue to increase in size while their life-history traits evolve in concordance with the exponents of the body mass allometries. Also, a comparison between prokaryotes and the higher eukaryotes suggests that the transition from a negligible to a relatively large body mass is associated with an evolutionary transition from a haploid organism with no soma, no senescence, and no sexual reproduction, to a diploid organisms with soma, senescence, and sexual reproduction between a male and a female. In a few special cases, mostly in insects, there is an additional transition to eusocial communities, a transition that in some instances may coincide also with a transition from a diploid to a haplodiploid genome. As these major evolutionary trajectories tend to summarise the evolutionary process at a very large scale they require an explanation. It is such an explanation that is the major objective with this book where I develop a new and general theory of evolution that is based on an extension of the mathematical framework behind the classical theory of evolution by natural selection.

1.1 The classical theory of natural selection

In 1859 Darwin proposed that organisms on Earth had evolved by natural selection. It was, however, not until the early 1930s that this hypothesis

was developed into a formal mathematical theory by the work of Fisher (1930), Haldane (1932), and Wright (1931). This theory, which became known as the genetical theory of natural selection, is a logical unification between Mendelian inheritance and the Darwinian hypothesis of evolution by natural selection. Since then, the ideas that were laid down mainly by Fisher have grown into a mature theory that today covers the evolution of the major components of the phenotype. In this book I refer to this theory as the classical theory of evolution and it is reviewed in the recent books by Roff (1992), Stearns (1992), Charnov (1993), Bulmer (1994), and Charlesworth (1994).

Broadly speaking, classical theory is based on the assumption that the relative fitnesses are constant among genotypes. This implies that it is also assumed that competition is purely exploitative, and that the classical type of selection can be classified as selection by the intrinsic constraints that are inherently part of the organisms itself. More specifically, this type of selection is the hypothesis that we can partition the phenotype into two different sets of traits, where the first set contains the fundamental traits representing the evolutionary constraints that define natural selection, and the second set contains the derived traits that evolve from the selection pressure defined by the fundamental traits. A few examples will illustrate this more clearly. According to Roff (1981) the body mass is a derived trait that evolves from a fundamental and proportional relation that exists between the reproductive rate and body mass. According to Lack (1947) the reproductive rate is a derived trait evolving from a fundamental trade-off that exists between reproduction and either offspring or parent survival. According to Williams (1957) senescence is a derived trait that evolves from the soma, which is more fundamental. According to Fisher (1930) an even sex ratio is a derived trait that evolves from the diploid zygote and random mating that are more fundamental. And according to Hamilton (1964) eusociality is a derived trait evolving from kin selection and a haplodiploid genome, which are seen to be more fundamental.

1.1.1 Limitations to the classical theory

The classical theory have generally been confirmed to the extent that when the fundamental traits are estimated from the phenotype of a specific organisms, then the predicted setting of the derived traits tends to coincide with the derived traits of that organism. In this sense we might at first think that the classical theory of evolution is firm and solid. However, the interpretation of causality in the classical theory is inherently vulnerable to criticism, and this is because the traits that are assumed to be fundamental in that theory are themselves part of the phenotype. Thus, they have evolved by natural selection, exactly like the traits that are assumed to be

more derived. This means that there is selection on both types of traits and, therefore, it is likely that the fundamental traits are no more evolutionarily constrained than the derived traits.

When both the fundamental and the derived traits evolve by selection we have the general problem that, as long as we focus on equilibrium predictions, it is almost impossible to distinguish the case where it is trait *A* that is fundamental and the cause of the evolution of the derived trait *B*, from the opposing case where it is the trait *B* that is fundamental and the cause of the evolution of the derived trait *A*. In other words, with equal right I can use the classical approach to construct a new theory from which I can propose it is the proportional relation between reproduction and body mass that is the derived trait evolving from the more fundamental selection pressure on body mass. That it is the trade-off between reproduction and survival that evolves from the optimal growth rate that is more fundamental. That it is the soma that is the derived trait evolving from senescence, which is more fundamental. That it is the diploid zygote and random mating that are the derived traits that evolve from the more fundamental sex ratio. And, that it is kin selection and a haplodiploid genome that are the derived traits evolving from eusociality, which is more fundamental.

Such results would not in themselves imply that the classical interpretation of evolutionary causality is wrong. Instead, they imply only that we generally cannot use the classical equilibrium predictions to confirm the traditional explanation instead of the explanation that is diametrically opposite. In other words, we are placed in the uncomfortable situation where we have two opposing theories and where we cannot use simple empirical evidence to confirm which theory is correct and which is false. To avoid this problem I have taken a new approach in this book based on the idea that if we cannot use empirical evidence to confirm whether the fundamental traits in a theory are more fundamental than the derived traits, then we should avoid to base our predictions on the occurrence of fundamental traits that are evolutionarily unexplained.

One way to reach this goal is to extend the theory of selection so that selection operates on all the phenotypic traits that we consider and, then, to show that this complete phenotype is evolutionarily stable given genetic variation in all traits. A theory at this level would be more general than the classical theory, and this is because it will give the same equilibrium predictions as the classical theory while it at the same time will explain also the evolution of the phenotypic assumptions underlying the predictions in the classical theory.

In the construction of such a general or perfect theory we are aiming at a framework predicting evolutionarily stable phenotypes from assumptions that are not in themselves part of the phenotype. Based on this approach

we may conclude that a particular theory fails on an evolutionary scale if the phenotype is not evolutionarily stable with respect to all the traits that we consider. Moreover, if we have two, or more, opposing theories to choose among and we cannot use empirical evidence to confirm which theory is correct, then the hypothesis of a perfect theory suggests that the correct theory is likely to be the theory containing the fewest assumptions, i.e., the theory containing the fewest fundamental traits that are evolutionarily unexplained. In this way the construction of a theory of evolution can be seen as a successive process during which the number of assumptions continuously is reduced until we reach the final stage of perfection where the theory contains no biological assumptions besides those that are associated with the origin of living beings.

In other words, in the construction of a perfect theory of evolution we aim at developing the Darwinian hypothesis into a purely deductive theory that is based only on a single biological assumption, namely the assumption that self-replication is the origin from which all living organisms have evolved. It would be possible to reach this goal from the classical theory if we can prove that all the traits that are assumed to be fundamental in that theory evolve from the principle of self-replication independently of the presence versus absence of the traits assumed to be the derived traits. If this is possible we can always trace the evolution of a particular trait back to the common origin of self-replication and we would have a mechanistic explanation for the evolution of all the traits considered in the classical theory.

Throughout this book I test whether the fundamental traits in the classical theory are evolutionarily stable independently of the derived traits. This is generally done by allowing for genetic variation in the fundamental traits so that their status as evolutionary constraints is relaxed and they evolve by selection, just like the derived traits. When this is done I find that the fundamental traits are evolutionarily unstable, and that the evolutionary predictions of the classical theory collapses in the sense that all organisms evolve to the limit of self-replicating molecules. From these results I conclude that the classical theory fails to explain the evolution of both the fundamental and the derived traits. That is to say that, although the classical explanations are valid according to the traditional framework where it is legitimate to impose evolutionary constraints by assuming the presence of fundamental traits, they fail on an evolutionary scale where it is the complete phenotype that needs to be evolutionarily stable.

It is easy to see why the classical theory fails on an evolutionary scale. This theory has generally been constructed to explain the evolution of traits that require energy that could otherwise be used to enhance numerical replication. Then, as the classical theory defines selection by a continuous increase in the growth rate of the population, the predictions of the theory

depend upon the intrinsic constraints preventing the energy contained in the derived traits from being selected into numerical replication. When there is genetic variation in the fundamental traits there is no longer such constraints, and this implies that the phenotype continues to shrink toward an organism of negligible size that replicates at a high rate.

1.1.2 Historical and non-directional evolution

Closely associated with the classical theory of evolution there is the hypothesis that evolution by natural selection is historical, that is to say that it is non-directional. According to this concept there is an almost infinite number of possible evolutionary trajectories and it is historical incidents that determine the actual evolutionary trajectories that can be observed in the fossil record. According to Wright's (1931) theory of shifting balance, historical accidents may resemble unpredictable rearrangements of the genome that generate brief moments of shifting selection pressures.

In the mathematical version of the classical theory the notion of historical evolution is represented in the form of the fundamental traits. In the classical theory these traits represent history in the sense that they have evolved by an unknown form of natural selection that is not included explicitly in the theory. Then, at the current point in the evolutionary history the fundamental traits are assumed to represent evolutionary constraints. It is of course more elegant if the evolution of all traits is modelled explicitly, but this approach does not work in the classical framework, and this is because if we allow for genetic variation in the fundamental traits, then the predictions of the classical theory collapse.

Because of the particular construction, where the fundamental traits are assumed to be fixed, the mathematical version of the classical theory is static in the sense that it does not allow the phenotype to evolve beyond the equilibrium defined by those fundamental traits. This static view has been challenged in the recent books of Buss (1987) and Maynard Smith and Szathmary (1995) where the authors focus on the evolutionary transitions that have occurred during the history of life on Earth. According to this latter approach evolution is seen as “the elaboration of new self-replicating entities by the self-replicating entities contained within them . . . at each stage in the history of life in which a new self-replicating unit arose—the rules regarding the operation of natural selection changed utterly” Buss (1987:viii). Although this latter approach focuses on transitions instead of static points the concept of evolution remains inherently historical in the sense that the maintenance of the more complex forms of life depends upon evolutionary constraints. In Maynard Smith and Szathmary (1995) these constraints are referred to as contingent irreversibility and central control.

The whole concept of historical evolution seems to be inseparable from

the Darwinian hypothesis of evolution by natural selection. According to Maynard Smith and Szathmáry (1995:4) “It was Lamarck’s notion of an inherent tendency [to climb the ladder of nature], rather than his belief in the inheritance of acquired characters, that Darwin was rejecting”. Today, this rejection is implicit in the thinking of leading evolutionists. For example, by implicitly assuming that evolution on Earth is historical Williams (1992:8) raises the question: “Might there be somewhere a planet on which the biota arises and becomes more complex deterministically?” Along the same line of thought Mayr (1988:20:105) defines natural selection as “a strictly *a posteriori* process” that is not “controlled by any law”. And Maynard Smith and Szathmáry (1995:4) concludes that “On the theoretical side, there is no reason why evolution by natural selection should lead to an increase in complexity”.

The ultimate conclusion from non-directional evolution is that the occurrence of intelligent and large-bodied animals with a high metabolic rate, senescence, soma, and sexual reproduction is more of a coincidence than a consequence of natural selection. This conclusion is somewhat ironical for a theory of evolution by natural selection, and it means that if extraterrestrial life exists, then it may not at all resemble life on Earth. It is obvious that these results are the consequence of an evolutionary theory that lacks a unifying force of selection that can explain the general structuring of organic matter. Just like the structuring of inert matter into planets and solar systems was mysterious prior to Newton’s theory of gravity, so does it seem that the mechanisms behind the structuring of organic matter will remain largely obscure until a unifying force of selection has been identified. With the theory that I propose in this book I have aimed at identifying a unifying force of selection in order to explain the general structuring of organic matter.

1.2 The proposed theory of natural selection

As the predictions of the classical theory fail when there is genetic variation in the fundamental traits we need a new type of selection if we want to explain the evolution of both the fundamental and the derived traits. As the classical theory is based on selection by intrinsic constraints an obvious way to proceed is to include selection by some sort of ecological constraint existing extrinsic to the organism. It is this route I have taken in this book where I develop a new theory of evolution based on a unifying force of selection arising from the density dependent competitive interactions that exist among the individuals within populations. As this leads to a special type of density and frequency dependent relativity among the relative fitness values defined by the Malthusian parameters I refer to my theory as the

theory of Malthusian relativity.

Up to now the hypothesis of evolution by competitive interactions has been treated in relation to specific topics like game theory (e.g., Maynard Smith and Price, 1973; Maynard Smith, 1982; Vincent and Brown, 1988), coevolution (e.g., Lawlor and Maynard Smith, 1976; Brown and Vincent, 1987; Abrams, 1989), the evolution of plant height (e.g., Mirmirani and Oster, 1978; Mäkela, 1985; King, 1990), and the evolution of competitive traits, especially in relation to selection for sexual mates (e.g., Parker, 1979, 1983; Haigh and Rose, 1980; Maynard Smith and Brown, 1986; Abrams and Matsuda, 1994; Day and Taylor, 1996). These earlier studies differ from the theory that I develop in this book in the way that they generally are based on the simplifying assumption that the number of competitive interactions per individual is density independent. They differ also in the sense that they tend to operate with “classical phenotypes” where the evolutionary predictions depend on fundamental traits that are evolutionarily unexplained. Furthermore, in the earlier studies there has been only sporadic interest in developing the hypothesis of evolution by competitive interactions into a general theory of evolution (Day and Taylor, 1996). With this book I have developed a general theory that covers the evolution of many of the major phenotypic patterns observed among and within mobile organisms.

1.2.1 Integrating the two theories

The theory of Malthusian relativity has a restricted or special version and an extended or general version, which are distinguished from one another by the degree to which the intrinsic selection procedures of the classical theory are integrated with the ecological selection pressure of density dependent competitive interactions. In the restricted, or special, form of Malthusian relativity the predictions are based almost exclusively upon the selection pressure of density dependent competitive interactions. This is in contrast to the general form of the theory where the predictions are based also on an integration between the intrinsic selection procedures in the classical theory and the proposed selection pressure of density dependent competitive interactions. The major difference between the predictions made by these two versions of the theory is on the number of phenotypic traits included in the predictions.

In the restricted form of Malthusian relativity the selection pressure of density dependent competitive interactions is used to predict the evolution of the traits that generally are treated as the derived traits in the classical theory. As these predictions are made independently of fundamental traits they are evolutionarily stable, and this is in contrast to the classical predictions, which are evolutionarily unstable in the dimension of the traits that are fundamental in the classical theory.

These new and restricted predictions do not in themselves establish the classical equilibrium relations between the traits that are fundamental and derived in the classical theory, i.e., they do not establish the relations that traditionally have been confirmed by empirical evidence. Instead, these relations are generally established by the transition from the restricted to the general form of Malthusian relativity, a transition carried out by superimposing the intrinsic selection procedures of the classical theory on top of the restricted form of Malthusian relativity.

Although the classical equilibrium relations between the fundamental and the derived traits are reestablished in the general form of Malthusian relativity, there are two major differences between the new and the old form of the classical predictions. The first difference is that the evolutionary causality underlying the new predictions most often is diametrically opposite to the causality underlying the original predictions. That is to say that it is the traits that are fundamental in the classical theory that are the derived traits in Malthusian relativity, while the traits that originally were derived are fundamental.

This change in causality is induced when we apply the classical selection procedures to the restricted form of Malthusian relativity because, then, we have a situation where the derived traits are explained already while the setting of the fundamental traits is unexplained. It is therefore most obvious to let the selection pressure of the classical selection procedures operate, not on the traits that are the derived traits in the classical theory, but instead on the traits traditionally assumed to be fundamental. Then, it is the assumptions in the original version of the classical theory that become the evolutionary predictions in the new version. For example, with Lack's theory on clutch size we will conclude that it is the trade-off between reproduction and survival that evolves from the optimal growth rate, and not the optimal growth rate that evolves from the trade-off as it was originally proposed by Lack.

This change in causality is possible because the causality in the classical selection procedures generally is defined not by the selection procedures in themselves, but by assumptions where it is the traits that are assumed to be fixed that impose selection on the traits allowed to evolve by selection. Hence, when the fixed and the evolving traits are switched around the selection procedure remains the same while the action of selection is turned upside down in the sense that it is now the original prediction that imposes selection on the original assumption.

The second difference between the new and the old forms of the classical predictions is that the new predictions are evolutionarily stable while the original predictions are evolutionarily unstable in the dimension of the traits that are fundamental in the classical theory. This new form of evolutionary

stability arises because the evolution of the traits that are fundamental in the new theory have been explained prior to their use as the assumptions that explain the evolution of the derived traits. In this way the evolutionary stability of the fundamental trait is transferred to the derived trait in the sense that the new prediction is stable in the dimension of the fundamental trait. It is due to this hierarchical propagation of the selection pressure and, thus, also of the evolutionary stability, to the different levels of the phenotype that we can reach a theoretically based conclusion on the causality that links the evolution of the different traits together, namely that it is because we can explain the evolution of trait *A* independently of trait *B*, and because we cannot explain the evolution of trait *B* independently of trait *A*, that it is *A* that induces the evolution of *B*, and not *B* that induces the evolution of *A*.

1.2.2 Deterministic and directional evolution

When we have constructed a theory where the evolutionary optima do not depend on phenotypic assumptions, then we have a situation where phenotypic variation necessarily must be given either by differences in initial conditions, by differences in environmental conditions, and/or differences in the degree to which the organisms have evolved along the evolutionary trajectory defined by natural selection.

The theory in this book suggests that many of the major phenotypic patterns existing among the mobile organisms on Earth are explained by differences in the degree that the organisms have evolved along a major evolutionary trajectory. The proposed theory also suggests that these differences are maintained because the different organisms are exposed to different environmental conditions, and that inter-specific interference competition probably is the major factor maintaining these differences in environmental conditions among species.

This hypothesis implies that evolution is deterministic, or directional, in the sense that organisms in a stable environment have an inherent tendency to evolve in particular directions because natural selection is selecting for directional changes. In the proposed theory the directional component is not in itself the result of selection by density dependent competitive interactions. Instead, the directionality requires the additional notion of selection for an increase in the ability by which an individual exploits the resource. This increase implies an exponential increase in the amount of resource, or energy, that is assimilated by the individual, and it is then selection by density dependent competitive interactions that allocates these resources to the different components of the phenotype generating a major evolutionary trajectory.

For mobile organisms the predicted trajectory includes an exponential

increase in body mass, metabolic rate, and the complexity of behavioural interactions. Associated to the transition from a negligible to a relatively large body mass there is a transition from a haploid organism with no soma, no senescence, and no sexual reproduction, to a diploid organism with soma, senescence, and sexual reproduction between a male and a female. In the special case where the body mass is constrained relatively to the ability by which the individual can assimilate resource the trajectory also includes a transition to eusocial communities, and dependent upon the role played by the sexual male this transition can be associated with a transition from a diploid to a haplodiploid genome. The evolutionary trajectory also explains the across-species exponents of the body mass allometries and the within-species exponent between reproduction and body mass. In other words, the predicted trajectories resemble the major evolutionary trajectories of the evolutionary process that has occurred on Earth. In Table 1.1 the major predictions of Malthusian relativity are listed and compared with the corresponding predictions in the classical theory.

Evidently there are some taxa that have been left aside from the predicted trajectory and this might at first appear to be contradictory to a theory on directional evolution. This is, however, not the case, and this is because the predicted trajectory depends upon an assumption of a stable environment with a sufficiently large resource. If, instead, the resource is extremely sparse, then the upper boundary to resource consumption is low and the organism may not be able to evolve away from a simple self-replicator. Also, for such organisms it might not be possible to invade environments with more abundant resources, and this is because these environments are likely to be dominated by larger species that can exclude the former species by direct interference. In this way there may be a relatively fixed pattern in which the different resources are distributed among different organisms, and this pattern may allow for a variety of variation in the degree to which natural organisms can evolve along the major evolutionary trajectory.

As the predicted evolutionary unfolding depends on a stable environment with a sufficiently high influx of energy to the underlying resource, we will find that the predicted trajectory is reversible in the sense that it may reverse if the influx of energy begins to decline. Also, at points where the influx of energy begins to decline there may be a mass extinction that will eliminate predominantly the larger species. And, if the influx continues to decline we expect a deterministic back-folding characterised by a decline in body mass, metabolic rate, and the complexity of behavioural interactions. As the back-folding continues males and eusocial communities will vanish together with senescence and soma and, given that the physical conditions remain suitable for life, the decline is expected to continue until the point of simple self-replicators.

Table 1.1 Some major predictions of the general theory of Malthusian relativity, and the corresponding predictions of the classical theory. The predictions of Malthusian relativity include all the traits mentioned, while the classical predictions include only the traits marked with a numbered *. These classical predictions depend on the fundamental traits with the superscript # and the corresponding number. A + or – indicates respectively the presence or absence of the main trait.

Main trait	Related patterns
Body mass ^{*1}	Intra-specific relation to reproductive rate ^{#1} , Bergmann's rule, Island rule, Cope's law, Dwarfing
Body mass allometry	For metabolic rate, lifespan, population density, home-range, reproductive rate, intrinsic growth rate, population energy use, biomass, level of sociality
Metabolism	Increase in metabolic rate
Behaviour	Increase in complexity of interactions
Reproductive rate ^{*2}	Trade-offs between reproduction and survival ^{#2} , balance against extrinsic mortality, difference between homeo- and poikilotherms
Population density	Difference between homeo- and poikilotherms
Senescence ^{*3} & soma ^{#3}	- in negligibly sized organisms, + in large organisms
Male individual	- in negligibly sized organisms, + in large mobile organisms, - in large sessile organisms, sex dimorphism's
Sex ratio ^{*4}	Mating structure ^{#4} , parthenogenesis
Sexual reproduction	- in negligibly sized organisms, + in large organisms, differences between sessile and mobile organisms
Genome	Haploid in negligibly sized organisms, diploid or haplodiploid in large organisms ^{#4} , diploid in eusocial termites ^{#4} , haplodiploid in eusocial ants and bees ^{#4}
Eusociality ^{*5}	Offspring workers ^{#5} , kin selection ^{#5} , - in vertebrates and + in insects, female biased sex ratio ^{*6} , female workers ^{#6} , and a haplodiploid genome ^{#6} in ants and bees, even sex ratio ^{*7} , male and female workers ^{#7} , and a diploid genome ^{#7} in termites
Dynamics	Population cycles, phenotypic cycles in, e.g., body mass & sex ratio

1.2.3 Extraterrestrial life

The idea that extraterrestrial intelligent beings may inhabit planets in other stellar systems have been a common subject of books and films, but until recently there has been very little scientific evidence that could support such ideas. Nevertheless, the accumulation of evidence seems now to suggest that extraterrestrial intelligent beings might exist quite commonly on other planets.

In order to conclude on the occurrence of extraterrestrial intelligent life there are at least four independent questions that we need to address. The first three of these belong to the physical sciences, while it is only the last question that belongs to the domain of biology. The first three questions are: *(i)* Whether stars generally have planetary systems, *(ii)* whether such planetary systems generally have planets that are potentially habitable by life, and *(iii)* whether the origin of simple self-replicators is so common a phenomena that they are likely to have arisen on the planets suitable for life. When we know the answer to these questions the final and biological question is whether simple self-replicators on suitable planets will evolve toward complex and intelligent beings.

The theory that is developed in this book is related only to the last question where it suggests that intelligent beings are expected because the predicted evolutionary unfolding is the result of self-replication in a stable environment suitable for life. This suggests that the evolutionary process we know from Earth is only a single example of a general process that is driven by universal laws.

In relation to the three questions in the physical domain there have recently been a number of studies that for the first time suggest that the three physical conditions required for extraterrestrial life may also be fulfilled: *(i)* The observations of a handful of stars with planet-like companions (e.g., Wolszczan and Frail, 1992; Mayor and Queloz, 1995; reviewed by Beckwith and Sargent, 1996) suggest that planetary systems are common, especially when we consider the technical problems associated with the detection of such systems. *(ii)* The theoretical studies of Wetherill (1996) and others (see Black, 1996) suggest that planetary systems have a rather high probability of containing planets that are potentially habitable by living organisms. And finally, *(iii)* if the recent hypothesis of ancient life on Mars (McKay et al., 1996; see also Anders et al., 1997) is true it suggests that the origin of simple self-replicators is a common phenomenon that is not unique to Earth. Hence, it seems to be likely that extraterrestrial intelligent beings are widespread within the universe.

1.3 Evolutionary population dynamics

Apart from defining a new theory of evolution, Malthusian relativity also provides an extension of the classical theory of population dynamics. This latter theory arose from Malthus (1798) and it is based on the assumption of no evolutionary changes in the growth rates of populations. This assumption leads to the population dynamics described by the Malthusian law of exponential increase, the logistic equation, and the Lotka-Volterra predator-prey equations.

In the theory of Malthusian relativity population dynamics is inherently associated with evolutionary changes in the growth rates, and this implies that the proposed theory merges into the classical theory only in the special case where genetic variation is absent. The major implication of the new theory is that it can explain the cyclic dynamics that is widespread throughout the animal kingdom and which have remained a mystery throughout this century. Not only does the predicted dynamics include a cycle in the abundance of the population, but it also includes a cycle in the phenotype. Among other things, this latter cycle can include periodic changes in the body mass and the sex ratio.

1.4 The structure of the book

The book contains six parts that each contain a set of chapters on related subjects. Apart from the deduction of Hutchinson's rule in Chapter 6, the first part is mainly a review describing the classical tradition in theoretical ecology, i.e., the tradition based on the classical theory of population dynamics. This part introduces the inexperienced reader to the theoretical framework used throughout the book. It also illustrates that the structuring of ecological communities is highly influenced by interference competition. The second part is an introduction to evolutionary analysis, and the third part describes the evolution of basic traits like body mass, population abundance, and reproduction. In the last chapter of this part I deduce the exponents of the body mass allometries. Then, in the fourth part I show that the evolutionary process is expected to equilibrate at an evolutionary steady state with exponential increase in the exploitation efficiency, body mass, and metabolic rate. In that part I also focus on evolution during environmental crises and on extinctions. In the fifth part I leave the basic traits and describe the evolution of derived traits like senescence, soma, group size, sexual reproduction, ploidy level of the genome, and eusocial communities. Finally, in the last part the assumption of population dynamic equilibria is relaxed and focus is shifted toward evolutionary population dynamics.

Part I

**Traditional theoretical
ecology**

Chapter 2

Malthusian increase

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

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

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

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

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

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

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

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

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

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

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

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

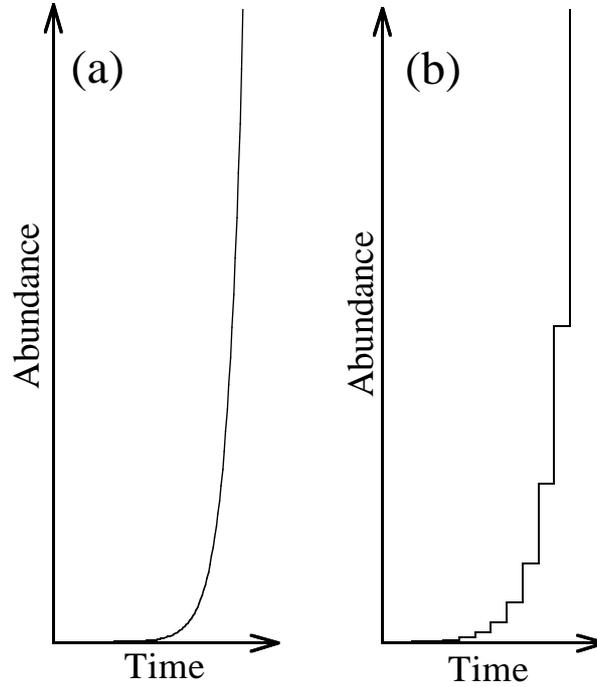


Fig. 2.1 Abundance against time, illustrating exponential (a) and geometrical (b) increase.

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

$$N_t = N_0 \lambda^t$$

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

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

Chapter 3

Density regulation

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

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

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

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

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

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

3.1 Resource regulation

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

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

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

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

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

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

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

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

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

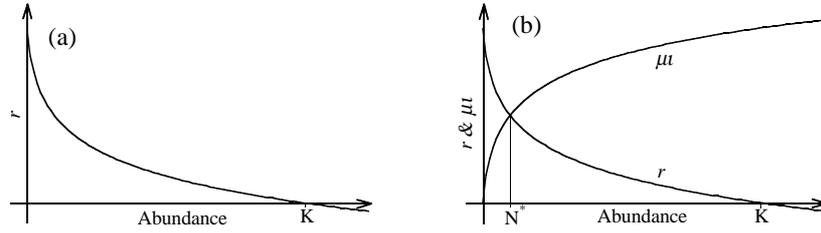


Fig. 3.1 (a) Population regulation according to Lack's hypothesis of resource regulation. The carrying capacity (K) is at the intercept $r = 0$, where r is the intrinsic Malthusian parameter. (b) Population regulation under the hypothesis of interference and resource regulation. The population equilibrium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

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

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

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

3.2 Interference regulation

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

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

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

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

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

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

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

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

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

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

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

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

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

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

from which I can reformulate eqn 3.9 as

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

or simply

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

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

3.3 Continuous logistic growth

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

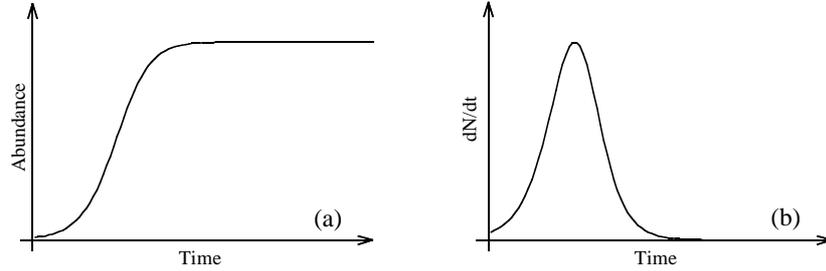


Fig. 3.2 Logistic growth. The abundance (a) and rate of increase in the population (b) plotted against time.

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

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

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

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

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

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

3.4 Discrete logistic growth

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

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

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

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

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

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

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

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

for all densities including the equilibrium.

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

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

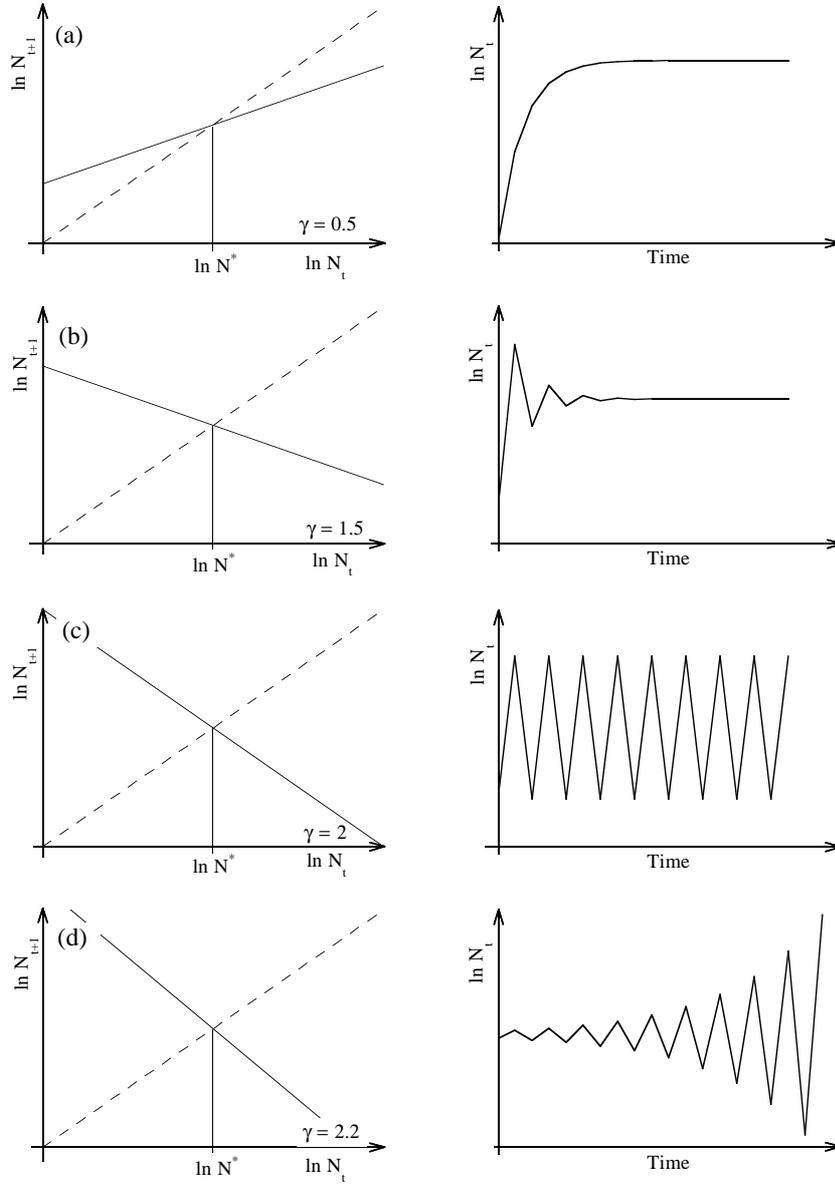


Fig. 3.3 The discrete logistic process of eqn 3.15 on double logarithmic scale for different values of the γ parameter. (Left) Population plots describing the density in generation $t+1$ as a function of the density in generation t . (Right) Population trajectories corresponding to the plots to the left.

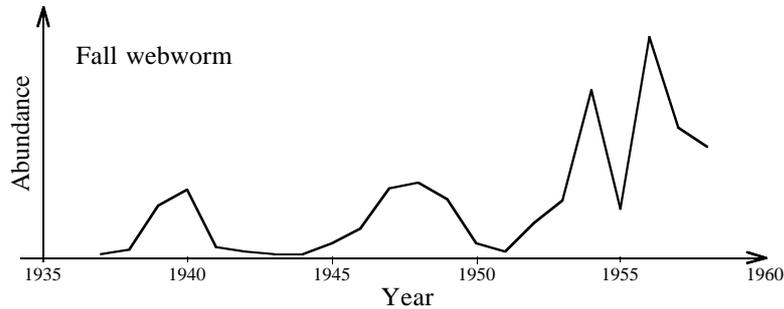


Fig. 3.4 The abundance of the fall webworm plotted against time. Data from Morris (1964).

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

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

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

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

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

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

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

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

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

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

3.5 Delayed density dependence

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

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

while the corresponding discrete model will take the form

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

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

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

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

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

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

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

Chapter 4

Predator-prey

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

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

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

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

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

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

4.1 The Lotka-Volterra equations

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

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

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

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

that is always positive for both populations.

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

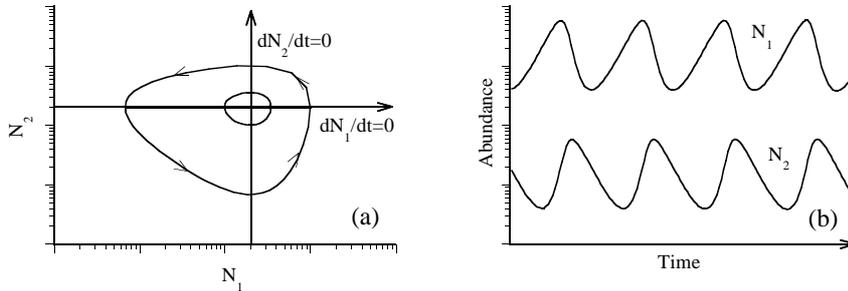


Fig. 4.1 The Lotka-Volterra predator-prey model (eqn 4.1). (a) A plot of the predator abundance (N_2) against the prey abundance (N_1). The two cyclic trajectories illustrate the prediction of a predator-prey driven cycle that is neutrally stable. (b) The abundances of a predator-prey cycle shown against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

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

4.2 Predator caused extinction

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

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

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

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

4.3 Adding interference competition

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

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

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

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

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

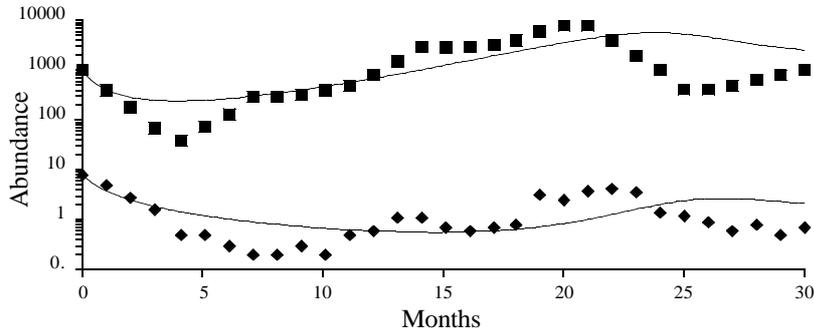


Fig. 4.2 A predator-prey cycle between the California vole (squares) and feral cats (diamonds). The two curves are the trajectories of the model $dN_1/dt = N_1(0.31 + 0.000017N_1 - 0.21N_2)$ and $dN_2/dt = N_2(-0.028 + 0.000088N_1 - 0.13N_2)$ that is estimated from the data by multiple linear regression.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

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

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

Chapter 5

Food chains

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

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

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

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

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

5.1 Exploitative versus interference competition

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

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

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

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

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

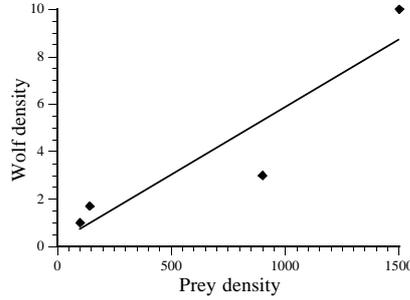


Fig. 5.1 The density, in individuals per 100 square miles, of the wolf against its prey (different species of deer). Data obtained from Ricklefs (1990); originally from Cowan (1947), Thompson (1952), Pimlott (1967), and Kelsall (1968).

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

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

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

if we disregard d_4 . Solving for the equilibrium we obtain

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

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

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

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

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

Chapter 6

Inter-specific competition

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

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

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

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

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

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

6.1 Exploitation: Competitive exclusion

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

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

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

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

6.2 Intra-specific interference: Competitive coexistence

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

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

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

with the equilibrium

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

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

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

with the equilibrium

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

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

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

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

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

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

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

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

and the equilibrium

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

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

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

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

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

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

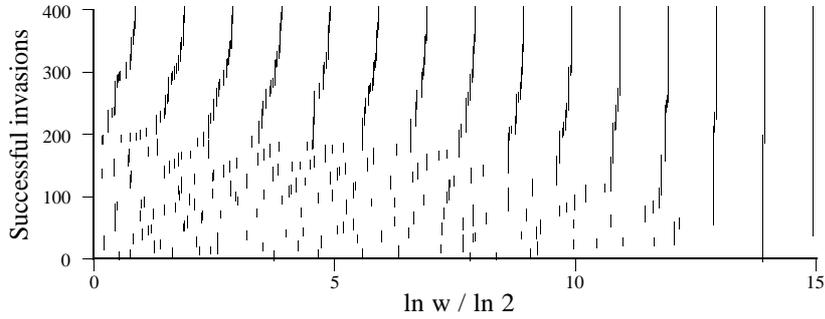


Fig. 6.1 The body mass (w) distribution among species that compete by interference competition as a function of the number of successful invasions into an initially empty locality. The invading species are drawn at random from an infinitely large species set that contains species with body masses that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

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

6.4 Appendix

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

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

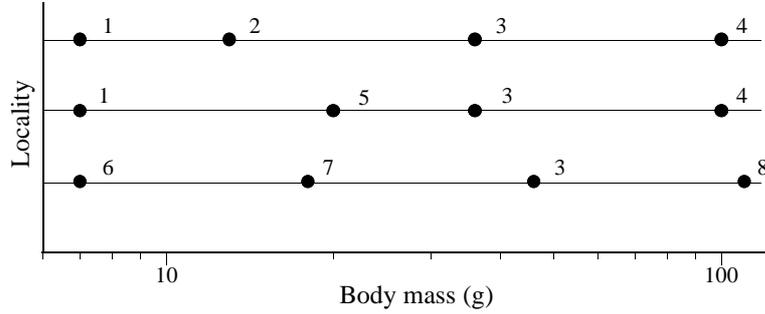


Fig. 6.2 The distribution of body masses among coexisting seed-eating desert rodents on three arbitrarily chosen localities. The scale is logarithmic to illustrate the equal spacing of weight ratios of approximately two. The numbers represent different species. Data from Bowers and Brown (1982).

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

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

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

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

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

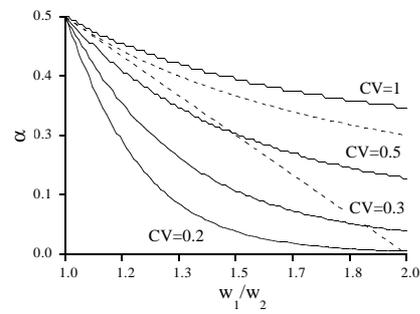


Fig. 6.3 The relationship between the α parameter and the body mass ratio (w_1/w_2) between two species 1 and 2. The area in between the two dashed lines defines the area where coexistence occurs, and the solid lines indicate the expected relationships when the body masses within each species are normally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

Part II

Evolution by natural selection

Chapter 7

Basic relations

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

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

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

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

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

7.1 Age-structured demography

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

with

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

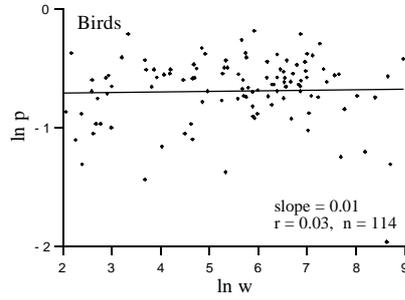


Fig. 7.1 The survival scalar (p) plotted against body mass (w) on double logarithmic scale for 114 species of birds, with the line indicating the linear regression. p is estimated as annual adult survival raised to the power of the age of first reproduction. From Witting (1995).

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

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

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

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

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

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

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

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

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

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

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

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

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

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

which reduces to the Euler equation

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

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

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

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

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

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

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

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

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

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

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

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

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

7.2 Physiological constraints

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

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

7.2.1 The physicist and the evolutionist

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

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

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

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

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

7.2.2 Evolutionary constraints

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

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

As it is possible to imagine almost any functional relationship between

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

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

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

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

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

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

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

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

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

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

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

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

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

7.3 A few ecological constraints

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

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

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

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

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

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

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

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

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

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

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

and from this expression we find that maximal lifetime reproduction is

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

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

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

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

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

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

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

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

$$\epsilon = \alpha f g E \quad (7.35)$$

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

Chapter 8

Fitness and selection

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

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

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

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

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

8.1 Selection at different levels

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

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

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

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

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

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

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

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

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

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

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

8.2 Selection in the classical theory

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

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

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

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

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

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

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

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

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

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

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

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

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

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

8.3 Selection by density dependent competitive interactions

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Chapter 9

Historical versus deterministic evolution

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

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

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

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

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

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

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

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

9.1 Lamarck and Darwin

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

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

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

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

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

9.2 Historicity versus determinism

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

Determinism at this level suggests that selection within a phylogenetic

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

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

9.2.1 A mathematical distinction

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

9.2.2 Dimensionality of theoretical optima

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

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

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

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

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

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

9.3 Integrating the two theories

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

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

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

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

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

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

9.4 Equilibria at different levels

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

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

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

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

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

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

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

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

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

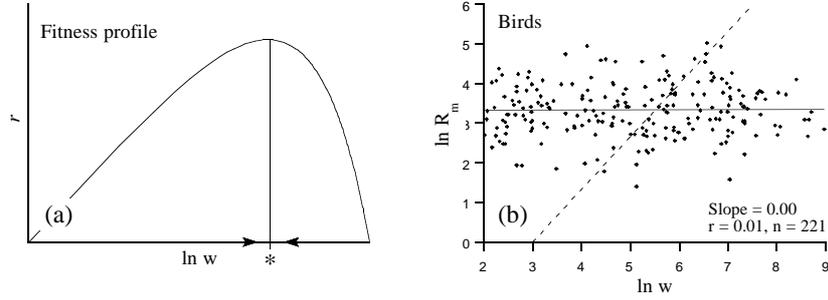


Fig. 10.1 (a) A classical one-dimensional fitness (r) profile for body mass (w), as defined by eqn 10.1. The star indicates the Fisherian equilibrium. (b) Maximal lifetime reproduction (R_m) plotted against body mass on double logarithmic scale for 221 species of birds. The solid line is the linear regression 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

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

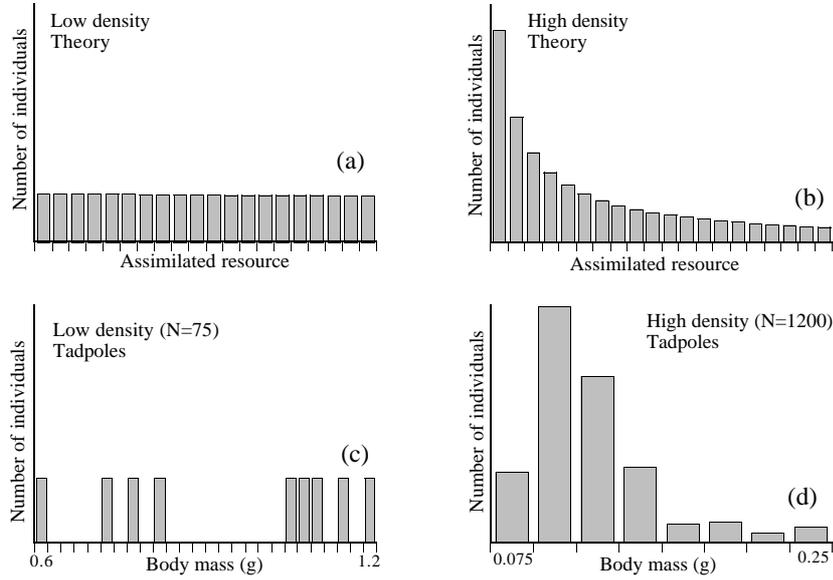


Fig. 10.2 An illustration of the within-population bias in resource assimilation, as generated by the intra-population interference in eqn 10.12 (**a** & **b**), and as seen in a population of tadpoles (**c** & **d**; data from Wilbur and Collins, 1973). For the tadpoles the variation in resource assimilation 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

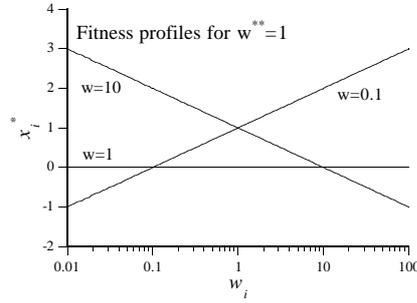


Fig. 10.3 One-dimensional projections of the fitness profile for body mass as defined by density dependent competitive interactions (eqn 10.19) for the situations where the average body mass at the evolutionary equilibrium (w^{**}) is 1, and the actual average body mass (w) is respectively 0.1, 1, and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

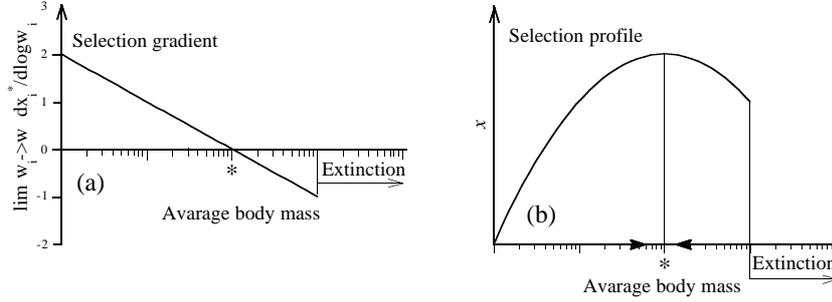


Fig. 10.4 The selection gradient (a) and the selection profile (b) for body mass as defined by density dependent competitive interactions. The stars indicate the evolutionary equilibrium, and the body masses to the right of the curves are so large that the population becomes extinct because the individuals 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.

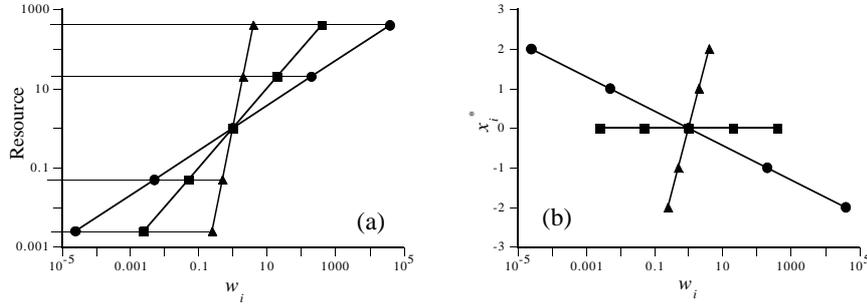


Fig. 11.1 (a) An illustration of the partitioning of resource against body mass (w_i) and (b) its relation to fitness (x_i^*) among five individuals when the partitioning is determined by contest-like interference competition. The squares represent the situation at the evolutionary equilibrium where 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

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

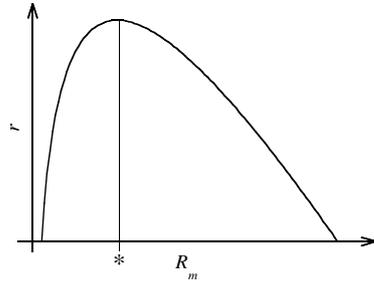


Fig. 12.1 A classical fitness profile for maximal lifetime reproduction (R_m) when the survival scalar p declines exponentially with R_m . The star indicates the Fisherian equilibrium.

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., $N^{**} = E^* e^{1/\gamma \psi}$, 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

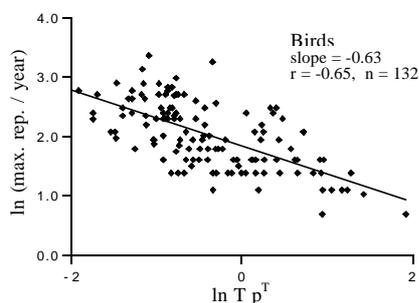


Fig. 12.2 Maximal yearly reproduction against time scaled survival (Tp^T) among 132 species of birds when plotted on double logarithmic scale. In this figure T is age at maturation, p adult annual survival, and maximal yearly reproduction (R_y) is calculated as the maximal clutch size times the number 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,

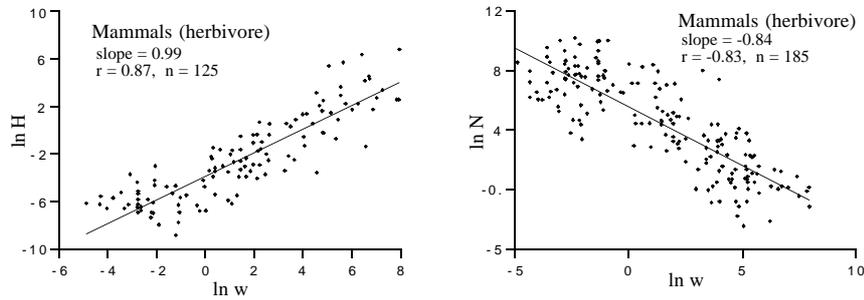


Fig. 13.1 The body mass (w) allometries for home-range area (H) and population density (N) in mammals. Data from Nowak (1991).

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 re-feeding 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

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

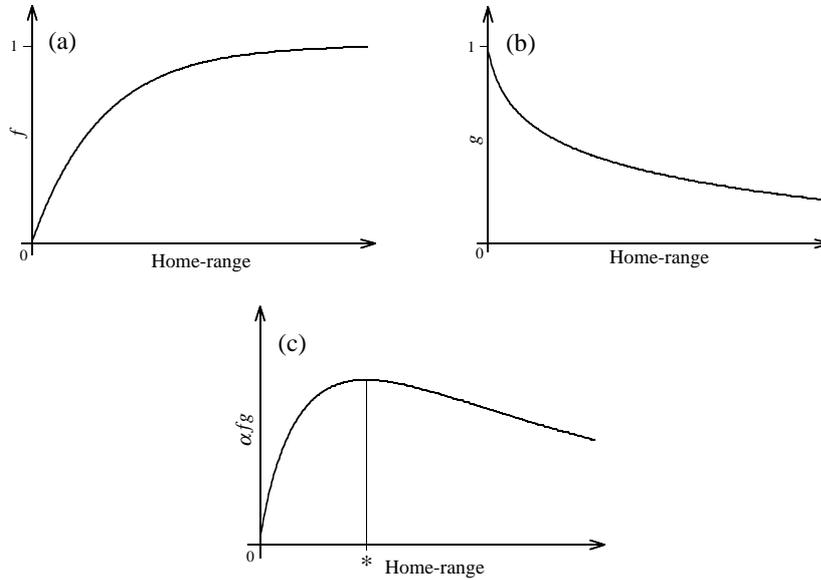


Fig. 13.2 Self-inhibition (a), interference regulation (b), and realised foraging (c) against the home-range. The star indicates the optimal home-range.

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 $p\alpha f g E/wB = 1$ 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

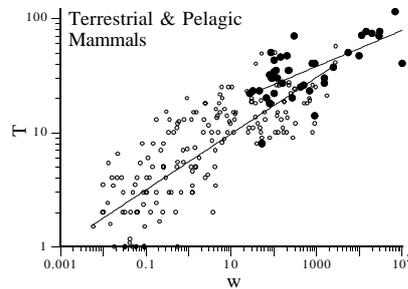


Fig. 13.3 The body mass (w) allometry for lifespan (T) among 195 species of terrestrial mammals where the exponent is 0.25 ± 0.04 (SE) (small open circles), and among 40 species of pelagic mammals (taxa Cetacea, Pinnipedia, and Sirenia) where the exponent is 0.16 ± 0.02 (SE) (large solid circles). Data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

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

Part IV

The evolutionary steady state

Chapter 14

Exponential increase in body mass

COPE (1887) SAW that species in the fossil record continue to increase in size, and this may include up to 90 percent of the mammalian lineages (Maynard Smith and Brown, 1986). Despite this empirical fact main stream evolutionists have continued to reject the idea that evolution is an upward drive. Instead, they have favoured the view that evolution by natural selection is stochastic, historical, and non-directional (e.g., Mayr, 1988; Williams, 1992; Maynard Smith and Szathmáry, 1995). This is probably best illustrated by the recent book of Maynard Smith and Szathmáry (1995:4) in which the authors already in the introduction conclude that “On the theoretical side, there is no reason why evolution by natural selection should lead to an increase in complexity”.

It is also widely accepted that species that increase in size do so only because they arose as small and because evolutionary diffusion will cause the size to increase in at least some species (e.g., Stanley, 1973; Gould, 1988, 1997; McKinney, 1990; Jablonski, 1997). In this and the following chapter I use the framework of Malthusian relativity to show that these traditional propositions on non-directional evolution fail because there *is* a force of selection that causes exponential increase in both the body mass and metabolic rate of mobile organisms. This exponential increase in competitive quality is the evolutionary process occurring in a stable environment when there is no immediate limit to the exploitation efficiency.

The idea of directional evolution is not new, actually we have already seen that the classical theory predicts directional evolution. Fisher’s fundamental theorem of natural selection predicts a continuous increase in the intrinsic Malthusian parameter and this leads to a continuous decline in traits

like body mass and metabolic rate. Today, it seems that the consequences of this straightforward prediction, which obviously does not resemble natural conditions, have been neglected for decades by evolutionists who have defined selection by intrinsic constraints that are evolutionarily unstable.

When we turn to the directional evolution in Malthusian relativity we relax the traditional assumption of evolutionary equilibria. This assumption is problematic because it is based on the generally invalid assumption that the individuals of a given species have a fixed ability to consume resource, i.e., a fixed exploitation efficiency (α) that cannot evolve by natural selection. In this chapter I relax this assumption and let α evolve freed from evolutionary constraints. As it is shown, this implies selection at a steady state that is characterised by exponential increase in both α and body mass. It is shown also that while the body mass increases exponentially the other phenotypic traits will evolve in accordance with the exponents of the body mass allometries. Among other things, this latter result predicts a continuous decline in the intrinsic Malthusian parameter, i.e., it predicts a continuous decline in Fisherian fitness.

The deduction of the steady state also predicts the proportional relation, between lifetime reproduction and body mass, that is used in the classical theory as the intrinsic constraint causing the evolution of large body masses. That is to say, that it turns out that the relation that originally was thought to define natural selection on body mass is the evolutionary result of the ecological constraints that define selection at the evolutionary steady state.

In the analysis of the steady state I will describe selection on the exploitation efficiency before I will deduce the steady state with the exponential increase in body mass. Hereafter, I will show that the across-species allometries, we deduced in Chapter 13, apply also for the steady state. Then, I will deduce the exponent of the within-species allometry between lifetime reproduction and body mass, and use this prediction to confirm that natural populations are situated at the evolutionary steady state. Having done this, I will describe a few relationships between steady state evolution and constraints on the body mass and the exploitation efficiency and, finally, I will conclude that the steady state defines broad scale evolution as a deterministically unfolding process.

14.1 Exponential increase in resource consumption

The first and crucial observation leading to the evolutionary steady state with exponential increase in body mass is that an individual generally does better if it has more resource, or energy, available. That is to say, that

with more resource the individual can produce more offspring and, thus, it can be more fit. This implies that there is selection for an increase in the exploitation efficiency (α). As this parameter has no direct influence on competitive quality selection on α conforms to the classical set-up with Fisherian selection. Then, the fitness profile at the population equilibrium is $\lambda^* = p\alpha E^* e^{-\mu^*} / wB$, and this implies that the selection gradient on $\ln \alpha$ is $\partial x^* / \partial \ln \alpha = 1$. We can then multiply by the genetic variance in $\ln \alpha$ and find that the exploitation efficiency increases exponentially at the following rate

$$d \ln \alpha / dt = \sigma_{\ln \alpha}^2 \quad (14.1)$$

Due to the exponential increase in α the species will have more and more energy available per individual, at least if the resource density remains stable as it is the case at the steady state (shown in Section 14.3). With this increase in the amount of resource per individual the intriguing question is to deduce the allocation of the extra energy among the phenotypic traits.

14.2 Exponential increase in body mass

We now have a situation with exponential increase in the amount of resource consumed by an individual. To analyse the allocation of the extra energy available from this increase, let me first consider selection when the body mass is the only competitive trait. I will then in Chapter 15 extend the analysis to the situation where the metabolic rate per unit body mass acts also as a competitive trait.

Because the rate of reproduction is a very plastic demographic rate and because realised reproduction correlates strongly with fitness we may expect that the extra energy initially will be allocated to an increased rate of reproduction. Then, as the average rate of reproduction increases, the population equilibrium will increase and this will cause an increase in the level of interference competition and, thus, also an increased bias in the access to resource. As this increased bias is in favour of the large-bodied individuals, selection will begin to allocate some fraction of the extra energy from reproduction to body mass. The level of interference will, however, continue to increase as long as the amount of energy selected into body mass is smaller than the amount of energy that continuously is added to reproduction due to the increase in α . At some specific level of interference competition the two rates of allocation will balance and we have the evolutionary steady state that is characterised by exponential increase in both the exploitation efficiency and body mass.

To deduce the evolutionary steady state mathematically we recall that the selection gradient on \ln body mass is given by eqn 10.20. If this gradient

is multiplied by the genetic variance in \ln body mass we find that the rate of evolution is

$$d \ln w / dt = \sigma_{\ln w}^2 [(\psi \gamma_\iota / \gamma) \ln(\rho \alpha / w) - 1] \quad (14.2)$$

with $\rho = pE_m/B$. The evolutionary trajectory described by eqn 14.2 reflects direct selection on body mass, and at the steady state this trajectory is defined also by the evolutionary increase in α together with the steady state relationship between α and body mass within the phylogenetic lineage in time. Although this latter relation between α and body mass is currently unknown to us we can use the unknown function to describe the rate of evolutionary increase in body mass. To do this let us approximate the intra-lineage relation between α and body mass by the power function $\alpha_t \propto w_t^a$, where the exponent a is an unknown constant. From this expression we have that $\ln w_t \propto \ln \alpha_t / a$ and, thus, that

$$d \ln w / dt = a^{-1} d \ln \alpha / dt \quad (14.3)$$

When this expression is combined with eqn 14.1 we find that the rate of evolution in \ln body mass is

$$d \ln w / dt = \sigma_{\ln \alpha}^2 / a \quad (14.4)$$

We now have two independent expressions for the rate of evolution in the natural logarithm of body mass: eqn 14.2 describing the rate of evolution as it is caused by selection on body mass, and eqn 14.4 describing the rate of evolution as it is caused by selection on the exploitation efficiency (α) and the steady state relationship between α and body mass. Where the two rates are balanced against one another we have the evolutionary steady state.

To deduce some of the characteristics of the evolutionary steady state we may begin with a deduction of the body mass. To obtain the body mass at the steady state set the two rates of evolution given by eqns 14.2 and 14.4 equal to one another and solve for w^{*s} . The obtained w^{*s} can then be inserted into the level of interference as defined by the constraint of the population dynamic equilibrium (eqn 10.18). Hereby we obtain the level of interference at the steady state (ι^{*s}). The obtained ι^{*s} can then be inserted into eqn 10.20 to obtain the selection gradient on body mass at the steady state $[(\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i)^{*s}]$. By solving these relations we find that the steady state is characterised by

$$\begin{aligned} w_t^{*s} &= \rho \alpha_t e^{-(1 + \sigma_{\ln \alpha}^2 / \sigma_{\ln w}^2 a) \gamma / \gamma_\iota \psi} & (14.5) \\ \iota^{*s} &= (1 + \sigma_{\ln \alpha}^2 / \sigma_{\ln w}^2 a) / \psi \\ (\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i)^{*s} &= \sigma_{\ln \alpha}^2 / \sigma_{\ln w}^2 a \end{aligned}$$

$$\begin{aligned}(d \ln w / dt)^{*s} &= \sigma_{\ln \alpha}^2 / a \\ (d \ln \alpha / dt)^{*s} &= \sigma_{\ln \alpha}^2\end{aligned}$$

From these equations it is apparent that $\iota^{*s} > \iota^{**} = 1/\psi$, and this means that the level of intra-population interference is higher at the steady state than at the evolutionary equilibrium. When we compare w^{*s} in eqn 14.5 with w^{**} in eqn 10.21 it is apparent that, for a particular exploitation efficiency (α), the body mass at the steady state is smaller than the body mass at the evolutionary equilibrium. Furthermore, from eqn 14.5 we find that the exponential increase in body mass is defined by the exponential increase in the exploitation efficiency in the sense that it is the genetic variance in the exploitation efficiency, and not the genetic variance in body mass, that determines the rate of evolution in body mass. This is because the level of interference will equilibrate so that the selection gradient on body mass is exactly so strong that the body mass absorbs the extra energy that continuously is added from the evolutionary increase in the exploitation efficiency.

Let us now analyse the evolutionary stability of the evolutionary steady state. From the obtained w^{*s} and eqn 14.2 we note that selection on body mass defines the following rates of increase in body mass

$$\begin{aligned}d \ln w / dt &< \sigma_{\ln \alpha}^2 / a \quad \text{for } w > w^{*s} \\ d \ln w / dt &> \sigma_{\ln \alpha}^2 / a \quad \text{for } w < w^{*s}\end{aligned}\tag{14.6}$$

From these equations we have that when $w > w^{*s}$, then selection on body mass causes a smaller increase in body mass than defined by the selection on the exploitation efficiency and the unknown intra-lineage relation between α and w at the steady state. This means that the evolutionary trajectory in body mass will converge toward the trajectory defined by the steady state. The same is true when $w < w^{*s}$ because, then selection on body mass causes a higher increase in the body mass than defined by the steady state trajectory. This implies that the evolutionary trajectory defined by the steady state and an initial value of α will attract all other evolutionary trajectories with the same initial α independently of the initial setting of the body mass. In other words, the evolutionary trajectory of the steady state is the global attractor. In Fig. 14.1a I used eqn 14.5 to plot the evolutionary trajectory of the steady state together with two other trajectories following from differences in the initial body mass.

14.3 Body mass allometries at steady state

We now have a situation where the global attractor is the evolutionary steady state with an exponentially increasing body mass. The intriguing

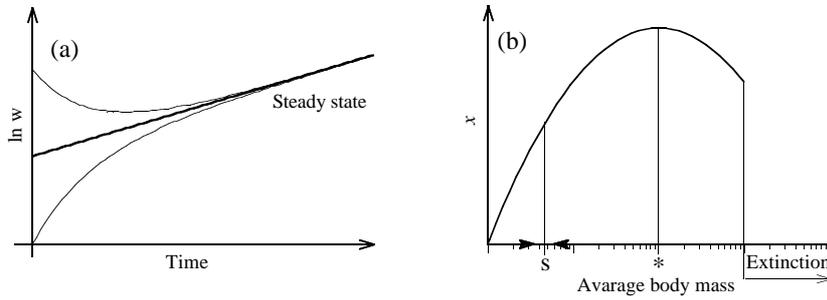


Fig. 14.1 (a) Exponential increase in body mass (w) as defined by selection when $\sigma_{\ln \alpha}^2$ and $\sigma_{\ln w}^2$ are constants. The two different trajectories (thin curves), that are initialised from the same α but two different body masses, converge to the trajectory of the steady state (thick line) for that initial value of α . (b) A selection profile for body mass with the position of the evolutionary equilibrium (*) and the evolutionary steady state (s) for an organism that forages in two dimensions.

questions then are whether the body mass allometries we deduced in Chapter 13 will hold in time for a phylogenetic lineage at steady state, and whether the body mass allometries will hold also across a set of species situated at the evolutionary steady state.

To determine the applicability of the allometric deduction at steady state we recall that the allometric exponents we deduced in Chapter 13 were based on a body mass invariant level of interference. In Chapter 10 we saw that this condition was fulfilled for a body mass in evolutionary equilibrium, but does this assumption also apply to a body mass in evolutionary steady state? From eqn 14.5 we notice that the level of interference at steady state is body mass invariant when the genetic variance in the logarithm to the exploitation efficiency and body mass are independent of respectively the average exploitation efficiency and the average body mass. As this seems to be a reasonable condition we expect that the exponents of the body mass allometries apply also to natural species that are situated at the evolutionary steady state. More exactly, this means that they are valid both across species situated at the evolutionary steady state, and within a phylogenetic lineage evolving along the trajectory of the steady state.

As the deduction of the body mass allometries are valid within a phylogenetic lineage in time we find that when the body mass increases exponentially the other phenotypic traits will evolve in accordance with the exponents of the body mass allometries. Among other things, this implies that the resource density will remain stable, and from the allometric relations $r_m \propto w^{-1/2d}$ and $N^* \propto w^{(1-2d)/2d}$ we find that the Fisherian fitness

defined as r_m and/or N^* will continue to decline. This conclusion is diametrically opposite to the fundamental theorem of natural selection that predicts a continuous increase in the two parameters.

14.3.1 Within-species allometry between reproduction and body mass

The body mass allometries deduced in Chapter 13, and which we have just shown to apply also at the evolutionary steady state, are across-species allometries describing the relationships between the average body mass and the average values of the other traits. At the smaller within-species scale we have a different set of allometries describing the allometric relationships across the individuals within the species. From Chapter 10 on the evolution of body mass, we recall that the within-species allometry between body mass and lifetime reproduction is the essential assumption defining the selection pressure for large body masses in the classical theory. In this section I will show that this classical assumption is explained from the ecological constraints at the steady state, and I will show also that the predicted allometry is confirmed empirically. In order to do this I will examine the selection gradient on body mass at steady state.

To obtain the selection gradient at steady state, from eqn 14.5 we notice that the level of interference and the selection gradient at the steady state are related to the exponent of the body mass allometry for the exploitation efficiency, i.e., related to the exponent a . So far the value of this exponent has been unknown, but as we have just shown that the across-species allometries are valid at the steady state from Chapter 13 we recall that $a = (2d - 1)/2d$, where d is the number of dimensions in which the organism forages. If we insert this a into eqn 14.5 and approximate by setting $\sigma_{\ln w}^2 \approx \sigma_{\ln \alpha}^2$, then we find that the level of interference and the selection gradient at the steady state are

$$i^{*s} = \frac{4d - 1}{2d - 1} \frac{1}{\psi} \quad (14.7)$$

$$\left(\lim_{w_i \rightarrow w} \frac{\partial x_i^*}{\partial \ln w_i} \right)^{*s} = \frac{2d}{2d - 1}$$

From this expression we find that the selection gradient at the steady state is defined by the number of dimensions in which the organism forages. In Fig. 14.1b I used this prediction to plot the position of the steady state on to the selection profile for the body mass defined by eqn 10.22. From the figure it is apparent that the steady state is situated to the left of the evolutionary equilibrium at the position where the slope of the selection

profile is $2d/(2d-1)$. Notice also that as selection increases the body mass the whole selection profile evolves toward the right.

To deduce the within-species allometry between reproduction and body mass let us integrate the selection gradient with respect to the within-population variation in $\ln w$ and obtain

$$\begin{aligned} x_i^* &= \int 2d/(2d-1) \partial \ln w_i \\ &\propto [2d/(2d-1)] \ln w_i \end{aligned} \quad (14.8)$$

Then, as fitness at the population equilibrium is defined as $x_i^* = \ln(p_i R_i)$, we can use eqn 14.8 to find that the within-species allometry between body mass and lifetime reproduction is

$$R_i \propto w_i^{2d/(2d-1)} \quad (14.9)$$

when it is assumed that $p_i \propto w_i^0$. By eqn 7.8 we have that this assumption is likely to hold as long as the energetic differences among the individuals within a population is so small that it affects mainly the rate of reproduction and not the rate of mortality. From Chapter 10 we recall that the within-species allometry of eqn 14.9 resembles the proportional relation between reproduction and body mass that is used as the intrinsic constraint that defines the selection pressure for large body masses in the classical theory. In other words, we can conclude that the classical assumption is explained by the ecological constraints at the evolutionary steady state.

Let us now use the prediction of eqn 14.9 to test whether natural populations are situated near the steady state, with exponential increase in the body mass, or near the evolutionary equilibrium, with a stable body mass. From eqn 14.9 we expect that lifetime reproduction at the steady state is proportional to the positive 4/3rd or 6/5th power of body mass when the organism forages in either 2 or 3 dimensions, i.e., proportional to $R_i \propto w_i^k$ with $k = 1.3$ or $k = 1.2$. In contrast to this we expect from Chapter 10 or Fig. 14.1b that $k = 0$ if the body mass is in evolutionary equilibrium.

From empirical studies it is well-known that the exponent k is positive in most species, and Reiss (1989) and Peters (1983) summarise some estimates of k : The best estimates from fishes are given by Wootton (1979) who used data from 124 studies on 62 species. He estimated an average k of 1.2, as it is predicted by eqn 14.8 if we assume that fishes forage in three spatial dimensions as indicated by Table 13.2. Peterson (1950) estimated a k of 1.03 in spiders, Robertson (1957) a $k \approx 1$ in *Drosophila melanogaster*, and Ridelyand Thompson (1979) estimated k to 0.77 for five species of Asellus (Crustacea; Isopoda). For the house martin I estimated k to 1.4 (data from Bryant, 1988), and for the ant-tended lycaenid butterfly k is approximately

0.9 (data from Elgar and Pierce, 1988). These estimates suggest that natural populations are closer to the evolutionary steady state than they are to the evolutionary equilibrium and, thus, that their body masses tend to increase exponentially.

14.4 Evolutionary constraints

The results that we obtained in the previous sections depend upon the assumption that there are no immediate limit to either the exploitation efficiency or the body mass. These assumptions may hold in a variety of situations. However, in some instances there may be limits that are imposed by the physical constraints of a given biological design. If there is such a constraint to a particular trait, then this limit cannot easily be broken even by artificial selection. This is because the genetic variance in that trait will decline as the trait evolves toward the limit, and this decline arises because it is almost impossible to generate a mutation that can break the barrier imposed by the constraint. In this section I will describe the evolutionary trajectories in the body mass and the level of interference given that this type of constraints operate either on the body mass or on the exploitation efficiency.

In the three subsections below I will examine respectively the evolutionary consequences of respectively a lower constraint on body mass, an upper constraint on body mass, and an upper constraint on the exploitation efficiency. Together with the evolutionary steady state these three situations will define four different levels of interference competition and, as it is apparent from Part V, these different levels of interference will define the selection pressure for many of the phenotypic differences existing between negligibly sized organisms, like prokaryotes and viruses, large-bodied organisms, like the higher eukaryotes, and eusocial organisms, like ants, bees, and termites. These results will indicate that the size of viruses and prokaryotes may coincide with a minimum size that is required for the metabolic and self-replicating processes of those organisms, and that the size of eusocial insects may reflect an upper limit imposed by the ecto-skeleton. If this is the case we have that the body masses of those organisms are defined by their evolutionary constraints, and not by the evolutionary equilibrium or by the evolutionary trajectory of the steady state.

14.4.1 A lower constraint on body mass

If there is a lower limit to the body mass, then the organism will have this minimum size if the individuals can assimilate only the most limited amount of resource. This can, e.g., be the case if the exploitation efficiency

is extremely small, or if the organism due to inter-specific interference competition is excluded from resource rich areas so that it will have to live on marginal resources.

In these situations where the assimilation of resource is extremely limited there is selection for the minimum mass, and this is because the assimilation of resource is so small that the population growth of these organisms cannot generate the amount of intra-population interference required to generate selection for an increase in body mass. In this case, the level of interference will remain approximately zero even though almost all the assimilated energy is allocated to numerical replication. This implies that selection is purely Fisherian and, thus, that the selection gradient on body mass is minus one (from eqn 10.17). From this gradient we expect at first a continuous decline in the body mass, but as the mass is downward constrained there will be no genetic variance upon which selection can operate and, thus, the body mass will remain stable.

From the prediction of Section 14.1, that the exploitation efficiency will increase exponentially, we might at first expect that minimum-sized organisms are evolutionarily unstable because, eventually, the exploitation efficiency will increase to a limit where selection by interference competition will begin to favour a larger body mass. However, in Section 14.4.3 we will find that this is not necessarily the case if the resource density is sufficiently low. In the Chapters 17, 20, and 21 I will consider this situation in further detail in order to show that a downward constrained body mass may be the essential component that can explain that prokaryotes and viruses have no soma, that they do not senesce, that they are haploid, and that they have asexual reproduction.

14.4.2 An upper constraint on body mass

If there is an upper limit to the body mass, then the organism will have this maximum body mass if the individuals pass beyond some upper threshold in their ability to assimilate resource.

In this case, with an upper limit to the body mass, the evolutionary trajectory can be described by the evolutionary steady state when the genetic variance in $\ln w$ declines with an increase in body mass. For example, the upper limit may be defined by the following relation

$$\sigma_{\ln w}^2 = \sigma_{\ln w,0}^2 e^{-hw} \quad (14.10)$$

where $\sigma_{\ln w,0}^2$ is the unconstrained level of genetic variance in $\ln w$, and h is a positive constant.

The situation of eqn 14.10 is shown in Fig. 14.2a where the body mass and the level of interference is plotted against time. Here, the body mass

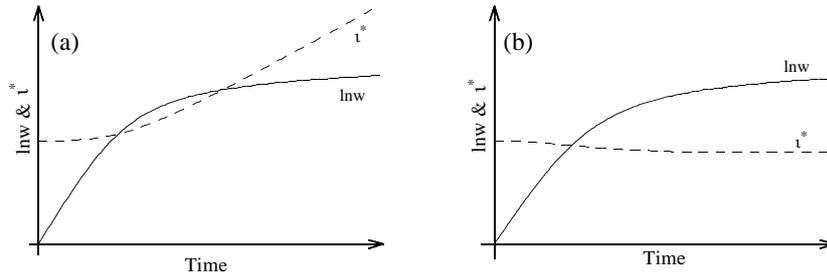


Fig. 14.2 Two evolutionary trajectories in body mass (w) and intra-population interference (t^*). (a) When the body mass is upward constrained by eqn 14.10. (b) When the exploitation efficiency is upward constrained by eqn 14.11.

increases exponentially at first, and then this increase stabilises as an increase in the level of interference appears. This increase in interference is in contrast to both the unconstrained steady state and to the evolutionary equilibrium where the level of interference is constant. With an upward constrained body mass the level of interference increases because at some point the constrained body mass can no longer absorb the free energy that is available from the evolutionary increase in the exploitation efficiency. Hereafter, selection is allocating the extra energy to numerical replication so that the population density and the level of interference increases. This result depends upon the assumption that the exploitation efficiency can evolve beyond the point that is associated with the transition in the allocation of the assimilated resource. As illustrated below, this may not always be the case if the resource density is too low. In Chapter 22 I will consider the situation with an upward constrained body mass in further detail, and this is done in order to show that it may be the essential component promoting the evolutionary transition to the eusocial colonies known from ants, termites, and bees.

14.4.3 An upper constraint on the exploitation efficiency

It is not only the body mass that can be upward constrained, it might also be the case for the exploitation efficiency. If there is such an upper limit to the exploitation efficiency, then the organism is expected to evolve to this limit at which the body mass will cease to increase.

To analyse this situation consider the case where the exploitation efficiency increases in a stable environment. Then, at some point it is likely that it becomes increasingly harder to improve the exploitation efficiency,

and this is because there is expected to be an upper limit to the rate at which the resource can be harvested by a given type of design. In such instances we find that the amount of genetic variation in $\ln \alpha$ will decline with α , e.g., as it is indicated by the relation

$$\sigma_{\ln \alpha}^2 = \sigma_{\ln \alpha, 0}^2 e^{-ha} \quad (14.11)$$

where $\sigma_{\ln \alpha, 0}^2$ is the unconstrained level of variation in $\ln \alpha$ and h is a positive constant.

The situation of eqn 14.11 is shown in Fig. 14.2b where the body mass and the level of interference are plotted against time. Here, the body mass increases exponentially before it stabilises, but in contrast to the constrained body mass the constrained exploitation efficiency causes a decline in the level of interference. From eqn 14.5 we see that this is because as $\sigma_{\ln \alpha}^2 \rightarrow 0$, then the level of interference approaches the level at the evolutionary equilibrium. In relation to this result we recall that the evolutionary equilibrium is defined by a non-evolving α , and by eqn 14.1 α does not evolve when $\sigma_{\ln \alpha}^2 = 0$.

14.5 Evolution as a deterministically unfolding process

We have now a situation where there is a direction to the evolutionary process, and although the direction may not continue indefinitely (Section 14.4), and although the assumption of a constant environment may not strictly apply in most situations, the underlying forces of selection seem to be so general that we may conclude that broad scale evolution is a deterministically unfolding process where living organisms have an inherent tendency to increase in mass and competitive quality.

The inherent tendency to increase in body mass is well documented from the fossil record where it is known as Cope's law (e.g., Cope, 1887; Depéret, 1909; Stanley, 1973; McKinney, 1990). According to Kurtén (1953) this paleontologic rule of evolution "is second in repute only to 'Dollo's law' of . . . irreversibility." In order to explain the increase in body mass many advantages of being big have been proposed (see Newell, 1949; Rensch, 1960; Peters, 1983; McKinney, 1990), and they include that larger individuals are superior in interference competition (e.g., Newell, 1949; Brown and Maurer, 1986; Maynard Smith and Brown, 1986). However, as shown in Chapter 10, it is not the component of competitive superiority that in itself can explain the increase in body mass, and this is because the average body mass will stabilise if α is constant. Instead, it is the combination of density dependent interference competition and the continuous increase in α that generate the evolution of exponential increase in body mass.

In this chapter we have focused on the evolutionary increase in body mass, but selection by competitive interactions at the steady state implies that it is competitive quality in general, and not only the body mass, that increases over time. Thus, among other things, we expect a continuous increase in the metabolic rate and in the complexity of the behavioural interactions that are used to control conspecifics. As the competitive quality increases we also expect that the overall complexity of the organisms will increase. For example, when the size of the organism increases beyond a certain level we may expect that multi-cellular organisms evolve because a multi-cellular design is more flexible than a single cell design. Also, to maintain a high metabolic rate within a relatively large organism it is a necessity that an intra-individual transportation systems is developed in order to elevate the exchange of chemical compounds above the rate of pure diffusion. Likewise, to perform complex behaviour an information processing system, as the neural-network, must develop. In Chapters 17 to 22 we will also see that the transitions in the level of interference, that we have described, e.g., in Section 14.4, will explain the transition from a haplodiploid and negligibly sized asexual organisms that do not senesce to a large-bodied organism with traits like senescence, soma, males, sexual reproduction, a diploid or a haplodiploid genome, and explain the additional transition to eusocial communities.

In conclusion, let me summarise the predicted evolutionary unfolding from self-replicating molecules to the complex organisms of today. At the origin of life the energetic level of the self-replicating molecules must have been so low that their density and their level of intra-population interference virtually were zero. At that time natural selection resembled Fisherian selection generating a continuous increase in the intrinsic Malthusian parameter through an increase in α and through evolutionary modifications in the allocation of the assimilated resource. As the evolutionary process proceeded through Fisherian selection the population density and the level of interference continued to increase. At a specific time in the history of life the level of interference would have risen to the level that is specified by the evolutionary equilibrium. As interference rose beyond this level the selection pressure changed from Fisherian to that of Malthusian relativity, and this change caused an evolutionary reallocation of the assimilated resource so that antagonistic traits could evolve together with the energy consuming competitive traits. As selection continued to increase α the level of interference would eventually equilibrate at the evolutionary steady state implying a steady increase in the various traits of competitive quality and their associated physiological complexity.

Chapter 15

Exponential increase in metabolic rate

THE ABILITY to metabolise energy into speed and power can be a crucial component for the outcome of a competitive interaction. This means that a high metabolic rate can evolve by density dependent competitive interactions because it can enhance the ability by which the individual can allocate energy to competitive interactions. Due to this type of selection the metabolic rate is expected to increase exponentially in the same way as it was deduced for the body mass in the previous chapter. In this sense it is not only the body mass, but also the metabolic rate, which absorbs the exponentially increasing amount of energy that the average individual has available from the exponential increase in the exploitation efficiency.

It is not surprising that Cope's law was established for body mass and not for the metabolic rate. Unlike body mass it is almost impossible to estimate the metabolic rate from fossil data. We all know that the dinosaurs were very big, but we still do not really know if they were homoio- or poikilotherms. Due to the problems in estimating the metabolic rate from fossils it is difficult to examine whether the prediction of a continuously increasing metabolic rate holds for fossil data. This is nevertheless what I intend to do in this chapter where I construct a model by which we can estimate the rate of increase in the metabolic rate from the rate of increase in body mass. This model can be applied to organisms that have undergone unconstrained evolution in the sense that they have been situated at the evolutionary steady state. As it is shown, when this is the case, then the exponent of the body mass allometry for the rate of increase in body mass is given by the rate of change in the metabolic rate per unit body mass.

One of the best documented candidates for unconstrained evolution is

the fossil horse, where the body mass has increased more or less exponentially for the last 57 million years (MacFadden, 1986). Evidently this lineage has not yet reached the upper limit to body mass, and it is also likely that the exploitation efficiency has evolved without constraints, because the plants eaten by the horse are easy to gather. Furthermore, as the fossil data on this taxon are almost perfect, it is likely that we can get a solid estimate of the rate of change in the metabolic rate. Hereby, it will be possible to estimate both the metabolic rate and the lifespan of the horse that lived 57 million years ago.

In the two sections below I will first describe the theoretical coupling between the evolutionary increase in body mass and metabolic rate, and then I will analyse the data on fossil horses.

15.1 Scaling time with metabolism

The comparisons we have performed in the previous chapters between empirical patterns and theoretical predictions have all been of the kind where we compared instantaneous patterns having no duration in time. For example, when in Chapter 14 we tested whether the body masses of natural species were increasing exponentially we did not look at the evolutionary trajectory in time, instead we examined the selection profile on body mass. In this chapter I will take the alternative approach and test the prediction of an exponentially increasing metabolic rate through an examination of the evolutionary trajectory in the body mass of fossil horses. This cannot be done by a simple projection of the equations that we described in the previous chapter, and this is because we need to consider the relationship between the time scale of the evolutionary process and the astronomical time scale on which empirical measurements are carried out.

As evolutionary changes caused by natural selection occur with a built-in delay of a single generation we have that the scaling of the evolutionary process to astronomical time is changing with evolutionary changes in lifespan. Then, as the lifespan is inversely related to the metabolic rate per unit body mass (eqn 7.26) we find that the evolutionary changes in the metabolic rate will alter the rate of the evolutionary increase in body mass. In the previous chapter we saw that if the body mass is the only competitive trait, then we expect that the intercepts of the body mass allometries are constant and that the evolutionary changes in lifespan and metabolic rate are defined by the evolutionary changes in body mass and the allometric relationships we deduced in Chapter 13. These allometries imply that while the body mass is increasing exponentially the lifespan is increasing exponentially too, while the metabolic rate is decreasing exponentially. In this relatively simple case the time scaling of the evolutionary trajectory in body mass involves only

the allometric relation between lifespan and body mass.

In the more general case where the metabolic rate is also a competitive trait that evolves due to selection by competitive interactions we find that the time scaling of the evolutionary trajectory in body mass becomes more complex, and this is because the intercepts of the body mass allometries for lifespan and metabolic rate are evolving also. For example, if selection on the metabolic rate is so strong that the rate increases exponentially, then the lifespan will decline exponentially and this implies that the evolutionary increase in body mass is accelerating at a faster rate when it is observed on an astronomical time scale. In other words, when the evolutionary trajectory in body mass is observed on an astronomical time scale, we can use the shape of the growth curve to determine whether the lifespan and the metabolic rate is increasing or decreasing exponentially. This is the idea behind the analysis that I will perform in this chapter.

To describe the relationship between the evolutionary trajectory in body mass and the evolutionary trajectories in lifespan and metabolic rate let, on a per generation time scale, the rate of exponential change in the metabolic rate be the rate of increase in body mass (s) times a constant (v). Then

$$\begin{aligned} dw/dt &= sw \\ dB/dt &= vsB \end{aligned} \quad (15.1)$$

and when solved

$$\begin{aligned} w_t &= w_0 e^{st} \\ B_t &= B_0 e^{vst} \end{aligned} \quad (15.2)$$

where w_0 and B_0 are the body mass and metabolic rate at time $t = 0$. Now, solve the growth equation for body mass with respect to time, obtain $t = \ln(w_t/w_0)/s$, and insert this expression into the growth equation for the metabolic rate. Then

$$B_t = (B_0/w_0^v)w_t^v \quad (15.3)$$

From eqn 7.26 we have that the physiological relationship between lifespan and the metabolic rate is $T = \omega/B$, and if this B is exchanged with B in eqn 15.3 we find that

$$T_t = \tau w_t^{-v} \quad (15.4)$$

where $\tau = \omega w_0^v/B_0$. As the growth equations in eqn 15.1 are given on a per generation time scale we need to scale these equations with respect to T_t if we want to predict the evolutionary trajectory on an astronomical time scale. For example, if the growth equation for body mass ($dw/dt = sw$) is scaled with respect the lifespan in eqn 15.4, then we find that the allometric relation for the growth curve in body mass depends upon v relating the

growth rate in the metabolic rate to the growth rate in body mass. That is, we find that

$$dw/dt = \dot{s}w^{1+v} \quad (15.5)$$

with $\dot{s} = s/\tau$. Notice here that this result depends upon the assumption that the senescence parameter ω , that is incorporated in τ , is constant.

If we consider first the case where it is only the body mass that represents competitive quality, whereas the metabolic rate does not, then we expect that the free energy available from the exponential increase in the exploitation efficiency is selected into the body mass while the other traits evolve in concordance with the exponents of the body mass allometries (Chapters 13 and 14). In this case the intercepts of the allometries are constant, and from the allometric deduction in Chapter 13 we have that the lifespan in eqn 15.4 is $T_t = \tau w_t^{1/2d}$, where d is the number of spatial dimensions in which the organism forages. This means that the evolutionary trajectory in the body mass of an organism foraging in two dimensions is described by the allometric relation $dw/dt \propto w^{3/4}$, and that the trajectory for an organism foraging in three dimensions is described by $dw/dt \propto w^{5/6}$. More generally we do not know the allometric scaling of eqn 15.5, but this scaling is easily estimated from evolutionary trajectories in body mass, and this is what we will do for fossil horses in the next section.

15.2 Metabolic rate and lifespan of horses 57 million years ago

Probably the most well-known example of Cope's law is the fossil horses that have increased in size more or less exponentially for millions of years. This increase is well documented especially by MacFadden (1986) who gives estimates of both the body mass and the rate of changes in body mass for a period that reach back 57 million years. In Fig. 15.1a I used his data to plot the rate of evolutionary increase in body mass against body mass on a double logarithmic scale. From the figure we see that the exponent of this relationship is 1.5, and this implies that $v = 0.5$. In other words, while the body mass of the horse has increased exponentially it seems that the metabolic rate per unit mass has increased exponentially at half the rate of the increase in mass and that this latter increase has caused a comparable decline in lifespan. In Fig. 15.1b I show the evolutionary trajectory in the body mass of the fossil horses together with a trajectory of the fitted model.

From eqn 15.3 it is possible to estimate the proportional increase that has occurred in the metabolic rate during the period $t=0$ to $t=t$, and this is done as $B_t/B_0 = (w_t/w_0)^v$. Due to the body mass allometry that we deduced in Chapter 13 for the metabolic rate, i.e., due to the relation $B_t = \beta_t w_t^{-1/2d}$, it is though more intuitively useful to estimate the proportional

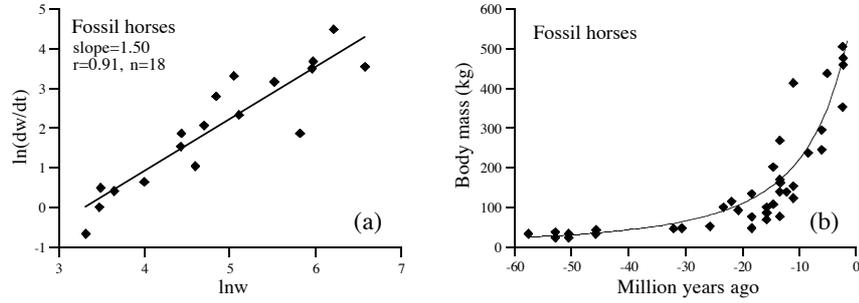


Fig. 15.1 (a) The rate of evolutionary increase in body mass (dw/dt) against body mass (w) on a double logarithmic scale for fossil horses. The diamonds are data from MacFadden (1986), and the line the linear regression. (b) The evolutionary trajectory for the body mass of fossil horses (diamonds), and a projection of eqn 15.5 (curve) with $v = 0.5$.

increase in the intercept of this allometry, i.e., estimate β_t/β_0 . In order to estimate this ratio, insert $B_t = \beta_t w_t^{-1/2d}$ and $B_0 = \beta_0 w_0^{-1/2d}$ into $B_t/B_0 = (w_t/w_0)^v$, rearrange, and obtain

$$\beta_t/\beta_0 = (w_t/w_0)^{v+1/2d} \quad (15.6)$$

From MacFadden we have that the horse weighed ≈ 25 kg 57 million years ago, and today it weighs ≈ 500 kg. Then, as horses forage in two dimensions we have that $d = 2$, and this implies that eqn 15.6 gives us the estimate $\beta_t/\beta_0 = 10$. That is to say that, when we correct for the allometric relationship between the metabolic rate and body mass, then the metabolic rate of the horse is ≈ 10 times larger today than it was 57 million years ago. In comparison, it can be mentioned that the metabolic rate of homoiotherms is ≈ 30 times larger than the metabolic rate of poikilotherms (Peters, 1983).

Due to the inverse relationship between the metabolic rate and lifespan the proportional decline in the intercept of the allometric relation between lifespan and body mass can be estimated as

$$\tau_t/\tau_0 = \beta_0/\beta_t \quad (15.7)$$

For a fixed body mass this implies that the lifespan has been reduced by 90% over the 57 million years. Thus, as today a 500 kg horse lives for ≈ 20 years (Nowak, 1991) we estimate that a hypothetical similarly sized horse that lived 57 million years ago would have had a lifespan of ≈ 200 years. If, instead, we incorporate the evolutionary changes that have occurred in body mass we find that $T_0 = T_t(w_t/w_0)^v$ and, thus, we estimate a lifespan of approximately 90 years for the 25 kg horse that lived 57 million years ago.

Chapter 16

Dwarfing and extinction

FROM THE FOSSIL record we know that the conditions on Earth are not so stable that all organisms tend to increase in size at all times. Instead, at any time there seems to be at least some organisms that dwarf, i.e., decline in size, and at particular events dwarfing even tends to predominate over Cope's rule of size increase. Associated with the events of widespread dwarfing there are normally many extinctions that tend to eliminate predominantly the larger species (Martin and Klein 1984; Donovan, 1989; McKinney, 1990). This type of crisis has occurred on many occasions during the history of the biota on Earth, but it is especially the five major mass extinctions that are well-known. These occurred at the end of Ordovician (≈ 440 million years ago (ma)), Devonian (≈ 360 ma), Permian (≈ 245 ma), Triassic (≈ 210 ma) and Cretaceous (≈ 65 ma), and they were big in the sense that between 70 and 95 percent of the species became extinct on each event (Jablonski, 1995). In this chapter I study the evolutionary processes that can explain the association between dwarfing and widespread extinction that eliminate predominantly the larger species.

A species may dwarf if its individuals have progressively less resource available, because then the rate of reproduction will decline causing a decline in the population equilibrium, the level of intra-population interference, and the biased access to resource. Due to this latter decline selection may allocate energy from body mass to numerical replication. This means that a species may dwarf in a stable environment if a larger species invades and by interference competition excludes the first species from the main habitat so that it will have to live in habitats of lower quality. In the same way, widespread dwarfing can occur if the primary production or the availability of resources decline. Furthermore, if the decline in primary production occurs faster than the phenotype can evolve, then, at a certain stage, the individuals can no longer find sufficient resource for self-replication and the

species becomes extinct. This type of extinction will affect predominantly the larger species, and this is because the rate of evolution is inversely related to the lifespan so that a small species having a short lifespan may be able to keep up with the environmental changes while a larger species with a longer lifespan may not.

So far there have been many proposals for the type of disturbances that might induce mass extinction, and these include meteoric impact, ice-ages, and increased human activity (see Martin and Klein, 1984; Donovan, 1989). There has, however, not yet been a general theory describing the biological processes that can explain why it was the large dinosaurs and not the small mammals that became extinct at the end of the Cretaceous, and why widespread dwarfing tends to co-occur with events of mass extinction. It is such a theory that I develop in this chapter where the mechanism that is involved is selection at steady state during periods with a decline in the primary production.

Apart from the patterns of extinction and dwarfing, the steady state with a declining primary production also generates a disturbance in the regularities behind the body mass allometries. Due to this disturbance it seems that the life-history patterns among organisms during periods of crisis are different from the life-history patterns that we know from the body mass allometries. Also, the steady state with a declining production provides us with a prediction of what is going to happen when the source that provides the energy for the primary production vanishes. In this case we find that, if the physical conditions remain suitable for life, then the body masses of all persisting species are expected to decline until they reach the molecular level. In other words, the evolutionary process seems to be reversible with the actual direction being defined by environmental conditions.

In the four sections below I will first consider the general process of dwarfing, then the allometric disorder during environmental crises, the evolutionary consequences of a continuous decline in the primary production, and finally, why it was the dinosaurs and not the mammals that became extinct during the perturbation at the end of the Cretaceous.

16.1 Dwarfing

From Chapter 14 we have that the steady state in a stable environment will stabilise at a level of interference where the exponentially increasing amount of energy available from the evolutionary increase in the exploitation efficiency is selected into body mass while lifetime reproduction remains constant. However, when the environment is unstable because the primary production is declining, an average individual has less energy available for reproduction, and this implies that the steady state will stabilise at a lower

level of interference where selection allocates less energy from reproduction to body mass, if energy is not allocated in the opposite direction.

To deduce this mathematically let us follow the procedure in Chapter 14 and make the additional assumption that the primary production (r_e) declines exponentially due to some major astronomic, geological, or climatical perturbation. Let this decline be

$$d \ln r_e / dt = -\xi \quad (16.1)$$

where ξ is a positive constant. As the decline in production is induced by abiotic factors it occurs in astronomical time and, thus, to connect the process of selection in the consumer organism to this decline we need to specify the evolutionary process in astronomical time also. The rate of evolution in body mass is then given as the rate defined by eqn 14.2, divided by the lifespan, i.e., as

$$d \ln w / dt = \sigma_{\ln w}^2 [(\psi \gamma_l / \gamma) \ln(\rho \alpha r_e / w) - 1] / T \quad (16.2)$$

where $\rho = p / \gamma_e B$.

The trajectory described by eqn 16.2 reflects direct selection on body mass, and at the steady state this trajectory is defined also by the relation in time between the body mass and the evolutionary increase in α and the environmental decline in r_e . Let us approximate these relations by the two power functions $\alpha_t \propto w_t^a$ and $r_{e,t} \propto w_t^e$, where the exponents a and e are unknown. Then, $\alpha_t r_{e,t} \propto w_t^{a+e}$ and, thus, $w_t \propto (\alpha_t r_{e,t})^{1/(a+e)}$. From this expression we find that $\ln w_t \propto (\ln \alpha_t + \ln r_{e,t}) / (a + e)$ and, thus, that

$$\frac{d \ln w}{dt} = \left(\frac{d \ln \alpha}{dt} + \frac{d \ln r_e}{dt} \right) \frac{1}{a + e} \quad (16.3)$$

From eqn 14.1 it is apparent that $d \ln \alpha / dt = \sigma_{\ln \alpha}^2 / T$, and from eqn 16.1 that $d \ln r_e / dt = -\xi$. When these two expressions are inserted into eqn 16.3 we find that

$$\frac{d \ln w}{dt} = \frac{\sigma_{\ln \alpha}^2 - \xi T}{T(a + e)} \quad (16.4)$$

We now have the two independent expressions (eqns 16.2 and 16.4) that define the rate of evolution in body mass at steady state. By setting the two rates equal to one another, and by following the procedure for eqn 14.5, we find that the steady state is characterised by

$$\begin{aligned} w_t^{*s} &= \rho \alpha_t r_{e,t} e^{-[1 + (\sigma_{\ln \alpha}^2 - \xi T_t) / \sigma_{\ln w}^2 (a + e)] \gamma / \gamma_l \psi} \\ \iota_t^{*s} &= [1 + (\sigma_{\ln \alpha}^2 - \xi T_t) / \sigma_{\ln w}^2 (a + e)] / \psi \\ \left(\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i \right)_t^{*s} &= (\sigma_{\ln \alpha}^2 - \xi T_t) / \sigma_{\ln w}^2 (a + e) \end{aligned}$$

$$\begin{aligned}
(d \ln w / dt)_t^{*s} &= (\sigma_{\ln \alpha}^2 / T_t - \xi) / (a + e) \\
(d \ln \alpha / dt)_t^{*s} &= \sigma_{\ln \alpha}^2 / T_t \\
(d \ln r_e / dt)^{*s} &= -\xi
\end{aligned}
\tag{16.5}$$

If we compare these equations with the steady state in a stable environment (eqn 14.5) it is apparent that both the level of interference and the selection gradient are smaller when the resource is declining than when it is stable, and that the body mass is larger for given values of r_e and α . We also notice that, when lifespan is positively related to body mass, then the rate of evolution in body mass is inversely related to body mass. For example, if the relation between lifespan and body mass is $T = \tau w^c$, with $\tau > 0$ and $c > 0$, then the organisms that are smaller than $\sqrt[c]{\sigma_{\ln \alpha}^2 / \tau \xi}$ will increase in size, while the organisms that are larger will dwarf.

Dwarfing is often observed in the fossil record. For example, in the last interglacial isolated red deer became reduced to one-sixth of their body weight in less than six thousand years (Lister, 1989). Moreover, there is an indirect way to test whether dwarfing in the fossil record has occurred by the action of selection due to density dependent competitive interactions. This is because selection by competitive interactions associates body mass increase with a high level of intra-population interference competition while it associates dwarfing with a low level of interference competition. This implies that there is a differentiated selection pressure on the size of the morphological traits that are used as social weaponry: When the organism increases in size there is a high level of interference competition and it will pay to allocate a relatively large amount of energy to social weaponry, but this energy is more or less wasted when the organism is dwarfing because, then there is only a low level of interference competition. In resemblance with this pattern we have Guthrie's (1984:262) conclusion from the fossil record: "Related to this dwarfing was a decrease in size of antlers, tusks, horns, and other social weaponry."

16.2 Allometric disorder

From Chapter 13 we recall that the deduction of the exponents of the body mass allometries is based on the occurrence of a body mass invariant level of interference competition. Moreover, we have previously shown that this condition was fulfilled both at the evolutionary equilibrium and at the evolutionary steady state in a stable environment. However, when the primary production is declining we find that this assumption no longer is fulfilled. This is because eqn 16.5 predicts that the level of interference is negatively related to lifespan, and because lifespan is expected to be positively related to body mass. Thus, we expect that the level of interference is negatively re-

lated to body mass. So in conclusion, we do not expect that the body mass exponents we deduced in Chapter 13 will apply to systems with a declining primary production. Instead, in this situation there seems to be no simple allometric solution to the life-history patterns among mobile organisms.

This disorder may be temperately only. To see this we note that even during an environmental crisis it seems to be reasonable to assume that lifespan is related positively to the body mass as $T_t = \tau w_t^c$ with $c > 0$. Then, from eqn 16.5, we find that the body mass will increase when $\xi < \sigma_{\ln \alpha}^2/T$, and this will cause an increase in lifespan and, consequently, we find that $\sigma_{\ln \alpha}^2/T \rightarrow \xi$. In comparison, when $\xi > \sigma_{\ln \alpha}^2/T$ we have that the body mass will decline and, thus, we find again that $\sigma_{\ln \alpha}^2/T \rightarrow \xi$. Then, as $\sigma_{\ln \alpha}^2/T$ will converge to ξ , by eqn 16.5, we find that the steady state will converge toward

$$\begin{aligned}
 w_t^{*s} \rightarrow w^{**} &= \rho \alpha_t r_{e,t} e^{-\gamma/\gamma_t \psi} & (16.6) \\
 t_t^{*s} \rightarrow t^{**} &= 1/\psi \\
 \left(\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i \right)_t^{*s} &\rightarrow \left(\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i \right)^{**} = 0 \\
 (d \ln w / dt)_t^{*s} \rightarrow (d \ln w / dt)^{**} &= 0 \\
 (d \ln \alpha / dt)_t^{*s} &\rightarrow \xi \\
 (d \ln r_e / dt)^{*s} &= -\xi
 \end{aligned}$$

This implies that we may expect that the body mass will stabilise at an equilibrium value that is maintained because the evolutionary increase in α is balanced against the exponential decline in r_e so that the product $\alpha_t r_{e,t}$ and the amount of resource consumed per individual, remains constant. For this steady state we note the body mass is independent of the initial body mass being defined only by the initial values of α and r_e . Furthermore, the selection gradient on body mass is zero and the level of interference resembles the level at the evolutionary equilibrium. Therefore, the level of interference is body mass invariant and, consequently, the well-known body mass allometries are reestablished.

16.3 Deterministic back-folding of biological systems

The predictions given by eqns 16.5 and 16.6 are expected to apply only during perturbations that are so short that there are no constraints on the exploitation efficiency. On longer perspectives, the exponential increase in the exploitation efficiency is likely to cease, and this is because of constraints on the magnitude of α in environments with extremely limited resources.

In such instances we expect that the decline in the genetic variance in $\ln \alpha$, which here is described by eqn 14.11, is functionally related to the primary production as indicated by the relation

$$\sigma_{\ln \alpha}^2 = \sigma_{\ln \alpha, 0}^2 e^{-h\alpha/r_e} \quad (16.7)$$

According to this expression we have that $\sigma_{\ln \alpha} \rightarrow 0$ when $r_e \rightarrow 0$, and this implies that the ultimate steady state is characterised as

$$\begin{aligned} w_t^{*s} &= \rho \alpha_t r_{e,t} e^{-[1 - \xi T_t / \sigma_{\ln w}^2 (a+e)] \gamma / \gamma_t \psi} \\ \iota_t^{*s} &= [1 - \xi T_t / \sigma_{\ln w}^2 (a+e)] / \psi \\ \left(\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i \right)_t^{*s} &= -\xi T_t / \sigma_{\ln w}^2 (a+e) \\ (d \ln w / dt)_t^{*s} &= -\xi / (a+e) \\ (d \ln \alpha / dt)_t^{*s} &= 0 \\ (d \ln r_e / dt)_t^{*s} &= -\xi \end{aligned} \quad (16.8)$$

From this expression we find that the level of interference will eventually be both body mass and time dependent and, thus, the well-known exponents to the body mass allometries will be invalid too. By eqn 16.8 we also expect that the body masses of all species will eventually decline if the primary production continues to do so.

The equations we have developed in this chapter consider only the body mass component of competitive quality, but their conclusions also apply to most of the other energy requiring traits that distinguish the complex organisms of today from simple self-replicating entities. This means that a continuous decline in the influx of energy to the biological system is expected to cause a deterministic back-folding, where complex organisms generally evolve toward the origin of life. This process is opposite to the evolutionary and deterministic unfolding that occurs in a stable environment with a sufficiently high influx of energy, and this result suggests that the evolutionary process is reversible, with the actual direction being defined by environmental conditions.

16.4 Why did mammals persist when dinosaurs became extinct?

When an organism dwarfs, selection allocates energy from body mass to numerical reproduction in the sense that an average individual can produce progressively more offspring from the same amount of resource. This implies that dwarfing will promote the persistence of a species, and this is because it can prevent a severe decline in the rate of reproduction when the

primary production continues to decline. But, if the decline in the primary production occurs too fast, then the allocation of energy from body mass to reproduction will be so slow that dwarfing cannot prevent that the reproductive rate will decline below the limit leading to extinction. Moreover, as evolution by natural selection is delayed with one generation, it is especially the larger species with the longer lifespans that are most severely affected by a decline in the primary production and, thus, it is predominantly these organisms that will go extinct.

In order to describe the different effects that an environmental perturbation will have on species that differ in body mass, let us recall from Chapter 14 that the per generation selection gradient on \ln body mass is $4/3$, or $6/5$, at the evolutionary steady state of organisms foraging in respectively two or three spatial dimensions, that it is zero at the evolutionary equilibrium, and that it is minus one at the limit of extinction. As it is shown below, this implies that the degree to which a species is affected by a decline in the primary production can be described by the magnitude of a downward change in the selection gradient from $4/3$ or $6/5$: where a selection gradient of $4/3$ or $6/5$ defines a healthy population in a stable environment, where progressively smaller gradients define progressively more perturbed populations, and where a gradient of approximately minus one defines a population that is so disturbed that it is about to become extinct.

In order to describe the different effects that an environmental perturbation will have on the selection gradient of species with different body masses we need a few simplifying assumptions, and this is because the steady state during an environmental crisis is not so clearly defined as the steady state in a stable environment. As indicated by eqns 16.5, 16.6, and 16.8 there are at least three different types of steady state to choose among, and let us choose the steady state defined by eqn 16.5, because this is the state we expect immediately posterior to the transition from a stable environment to a situation with an exponentially declining primary production. Just prior to this transition the environment is stable, the body mass allometries are valid, and by the prediction of eqn 14.7 there is reason to believe that $\sigma_{\ln \alpha}^2 \approx \sigma_{\ln w}^2$ for many species. If these conditions hold, then, for an organism foraging in two dimensions we have that $a = 3/4$, $e = 0$, and that $T = \tau w^{1/4}$. Thus, the selection gradient in eqn 16.5 is

$$\lim_{w_i \rightarrow w} \partial x_i^* / \partial \ln w_i = 4/3 - \dot{\xi}(w^{*s})^{1/4} \quad (16.9)$$

on a per generation time scale where $\dot{\xi} = 4\xi\tau/3\sigma_{\ln w}^2$ is a positive constant. From this expression we note that the selection gradient is negatively related to both the rate of decline in primary production (ξ in $\dot{\xi}$) and to the average body mass (w^{*s}). As dwarfing occurs when the selection gradient is negative, we find from eqn 16.9 that it occurs only among the species

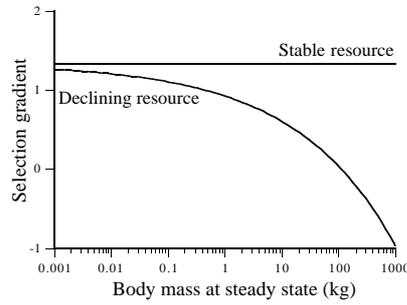


Fig. 16.1 The selection gradient for \ln body mass against the average body mass at the steady state for a stable and an exponentially declining primary production (resource). The rate of decline is so high that it causes the extinction of the animals larger than one ton.

that are larger than $w^{*s} > (4/3\dot{\xi})^4$ and, as a selection gradient of minus one represents the limit of extinction, we find that the species that are larger than $w^{*s} > (7/3\dot{\xi})^4$ become extinct. In Fig. 16.1 I have illustrated the situation where it is the species heavier than ≈ 110 kg that dwarf, and it is the species heavier than one ton that become extinct. This predicted association between dwarfing and extinction that eliminate predominantly the larger species is well-known from the fossil record (e.g., Martin and Klein, 1984; Barnosky, 1989; McKinney, 1990; Guthrie, 1984; King and Saunders, 1984). It is thus not surprising that it was the dinosaurs and not the mammals that became extinct during the perturbation at the end of the Cretaceous.

Part V

Evolution of derived traits

Chapter 17

Senescence and soma

PROKARYOTES ARE known to be potentially immortal whereas the individuals of the higher eukaryotes deteriorate with death being the unavoidable destiny. The classical theory on the evolution of this deterioration, called senescence, has roots all the way back to Weismann (1889). It was, however, especially Medawar (1952) and Williams (1957) who developed it into a formal theory that later was elaborated by, e.g., Hamilton (1966), Kirkwood (1977), and Charlesworth (1994).

According to the traditional view senescence is a derived trait evolving from the presence of a more fundamental somatic tissue from which no part is passed on in either sexual or asexual reproduction. Although this theory does not explain why the higher eukaryotes have evolved a soma, while prokaryotes have not, the predictions coincide with the fact that the non-senescent prokaryotes have no soma, whereas the senescing higher eukaryotes do. This dichotomy has been seen as one of the strongest predictions in evolutionary biology (Rose, 1991), but in this chapter I will aim at an even stronger prediction explaining both why soma and senescence are absent in negligibly sized organisms, like viruses and prokaryotes, and why both of these traits are present in large-bodied mobile organisms, like the higher and mobile eukaryotes.

To explain this pattern I will use an alternative model where the soma is the trait through which senescence is expressed, instead of being the fundamental and unexplained trait upon which the evolution of senescence depends. I will then apply selection by density dependent competitive interactions and show that both senescence and soma can evolve secondarily when selection favours competitive quality over a degree of self-repair in the tissue that will cause potential immortality. I will also show that it is only in the organisms having evolved a non-negligible body mass that the level of interference is so high that selection by competitive interactions will cause

the evolution of senescence and soma. In other words, the proposed hypothesis will explain that prokaryotes, which have a negligible body mass, are expected to be potentially immortal and to have no soma, whereas the individuals of the higher eukaryotes, which have a relatively large body mass, are expected to have a soma and to suffer from senescence.

In the description of the evolution of senescence and soma I will proceed in three successive steps. In the first section I will question the classical hypothesis that the soma is the fundamental trait promoting the evolution of senescence, and I will introduce the alternative hypothesis where the soma is the trait through which senescence is expressed. Then, in the second section, I will consider a physiological model of senescence and, in the third section, I will turn to the evolution of both senescence and soma.

17.1 On soma

In the classical theory the soma is the fundamental trait that promotes the evolution of senescence. The essential idea behind this hypothesis is that senescence evolves from a decline in the force of selection with age and that this decline applies only to the organisms having a soma. The decline in the force of selection with age is due to the fact that when the Malthusian parameter is calculated from age structure it is the changes in reproduction and survival early in life that have the largest impact on the Malthusian parameter. The idea is then that senescence evolves because selection favours early reproduction and/or survival at the cost of a degree of self-repair in the tissue that otherwise would lead to potential immortality (e.g., Kirkwood and Rose, 1991).

The hypothesis that the decline in the force of selection with age applies only to the organisms having a soma is based on the idea that age-structured demography applies only to somatic organisms and, consequently, it is hypothesised that the Malthusian parameter of an asomatic organism cannot be calculated from the age structure of that organism (Rose, 1991:84). The hypothesis that age-structured demography does not apply to asomatic organisms is based on the observation that asomatic organisms often reproduce by binary fission, where it is impossible to distinguish the “mother” from the “offspring”. And when we cannot distinguish the “mother” it is hypothesised that we also cannot define the age structure of the demographic traits of the mother. This latter conclusion is, however, true only when the demographic rates of the “mother” and the “offspring” differ. But, in asomatic organisms the two rates are the same and, therefore, we do not need to distinguish between the “mother” and the “offspring” in order to

determine the demographic rates of the “mother”. In other words, there is no problem in defining the Malthusian parameter from the age-structured demography of a bacterium. Actually, we have already done that when by eqn 7.19 we defined the Malthusian parameter for a potentially immortal organism with constant demographic rates.

We thus expect that the decline in the force of selection with age will apply to both asomatic and somatic organisms. Then, as both senescing and non-senescing organisms exist it cannot be the decline in the force of selection with age that is the essential component that explains the evolutionary transition from a non-senescing to a senescing organism. This implies, that if the essential component triggering the evolution of senescence is the presence versus absence of a soma, then the essential mechanism by which the soma promotes the evolution of senescence will have to be different from the classical mechanism of a decline in the force of selection with age. As no such alternative mechanism seems yet to have been proposed it may not hold that it is the soma that induces the evolution of senescence.

To avoid the dilemma in the classical theory I would like to propose the alternative hypothesis that the soma is the trait by which senescence is expressed. This hypothesis is based on the notion that senescence cannot evolve without a clear separation between a senescing somatic tissue and a non-senescing reproductive tissue. To see this consider the case where there is no clear separation between the two types of tissue. Then, if the tissue is not self-repairing the senescing tissue of the mother is passed on to her offspring and both the mother and her offspring will die from senescence at approximately the same time. It is evident that such lineages cannot persist, nor are they likely to evolve because they cannot produce viable offspring. Thus, if organisms with truly mixed tissues exist they must necessarily have completely self-repairing tissues and be potentially immortal.

Although there cannot be selection for senescence in the tissue that is passed on in reproduction there can be selection for a partitioning of the tissue into reproductive and non-reproductive tissue together with selection for the absence of self-repair in the non-reproductive tissue. This type of selection can occur when, e.g., early reproduction and/or survival is favoured at the cost of self-repair that leads to potential immortality. In such instances selection will favour the co-evolution of senescence and soma. The critical point left to explain is then to determine the dichotomy in the selection pressure that will select against senescence in prokaryotes and for senescence in higher eukaryotes. But before we deal with the identification of this dichotomy let us first consider in more detail the physiology of senescence.

17.2 On senescence

By senescence is understood that the tissue of an individual continues to deteriorate until the point of death where the detrimental effects have accumulated to a level where the individual fails to function. In complex organisms this process is likely to be an accumulation of small and stochastically occurring detrimental effects. Because complex organisms have an almost infinite number of local processes, that might fail due to stochastic malfunctioning, the accumulation of detrimental effects will take the form of an apparently deterministic process. This is not the case for extremely simple organisms such as self-replicating molecules. The individuals of such “organisms” have a very limited number of local processes that can fail due to stochastic malfunctioning. This implies that the “death” of a self-replicating molecule is likely to follow if only a single or a few of these processes are failing. In this case the term senescence is losing its meaning because the molecule is either perfectly functioning or completely failing. Hence, it appears that it is only organisms that have passed a certain level of complexity that can experience senescence, and the dichotomy between the apparently non-senescenting prokaryotes and the senescing higher eukaryotes might simply reflect differences in levels of complexity.

Although differences in complexity might explain why prokaryotes do not senesce, the argument of complexity is too limited to explain why the higher eukaryotes do senesce. This is because the higher eukaryotes theoretically could repair their tissue at so high a rate that the detrimental effects would not accumulate, and then the individuals would be non-senescenting and potentially immortal. In other words, to explain that higher eukaryotes senesce we need to justify that there is selection against a level of self-repair that will cause potential immortality.

If we return to the apparently non-senescenting prokaryotes there are two possible explanations for their lack of senescence. Firstly, as already mentioned, the prokaryotes might be so simple that they do not senesce despite the possible absence of self-repair. If this is the case it does not matter whether there is selection for or against self-repair, since in both cases the prokaryotes will not deteriorate in a continuous manner. The second possibility is the case where the prokaryotes are complex enough to deteriorate continuously in the absence of self-repair. If this is the case, then to explain the pattern in nature I need to explain both that there is selection for a high level of self-repair in prokaryotes and selection against a high level of self-repair in higher eukaryotes. In this chapter I will take this latter approach and show that, in the presence of density dependent competitive interactions, there is no problem in having selection for a high level of self-repair in prokaryotes and selection against a high level of self-repair in higher eukaryotes.

17.3 Evolution of senescence and soma

In this section, where I deal with the evolution of senescence and soma, I will first describe the trade-off between self-repair and senescence, which is essential for the evolution of both the soma and senescence. Then I will use this trade-off and the demographic model we defined in Chapter 7 to show that, when competition is purely exploitative and the force of selection declines with age, selection might not favour early reproduction at the cost of self-repair and late survival. I will then introduce density dependent interference competition in order to predict (i) that the individuals of negligibly sized organisms are expected to have no soma and to be potentially immortal, and (ii) that the individuals of large-bodied mobile organisms are expected to have a soma and to suffer from senescence.

17.3.1 Trade-off between self-repair and senescence

Before we can analyse the degree to which senescence and soma will evolve we need a model that describes the trade-off between self-repair and senescence, i.e., a model where the lifespan is positively related to the degree of self-repair and where selection is operating on the degree of self-repair. From Chapter 7 we have that lifespan is defined as the period from birth to death by senescence, and that it is given as $T = \omega/B$, where B is the metabolic rate per unit body mass, and ω is a constant of senescence that will change with changes in the degree of self-repair.

In order to model the selective modification of ω let us define ω as a function of the amount of energy that is spent on self-repair. If the organism repairs itself maximally, with X amounts of energy spent on repair per unit body mass per unit time, it will have the potential to become infinitely old, i.e., $\omega \rightarrow \infty$. If instead, no energy is spent on self-repair we have that senescence will occur and that ω will take a minimum value which we can denote ω_{min} . From these two limits we may expect that the physiological relationship between self-repair and lifespan will look approximately as illustrated in Fig. 17.1. Mathematically this function can be described through the amount of energy taken from maximal self-repair (X) and used for other purposes. If the energy that is taken from maximal self-repair per unit body mass is s , then the relationship between ω and s can be defined as $\omega = \zeta/s$, where ζ is a positive constant. Now, let $\zeta = \zeta/B$, so that lifespan is defined as

$$T = \zeta/s \quad (17.1)$$

That is, when $s = 0$ the individual may live infinitely because it repairs itself maximally with X amounts of energy used on self-repair per unit body mass, whereas the individual can survive only the period $T_{min} = \zeta/X$

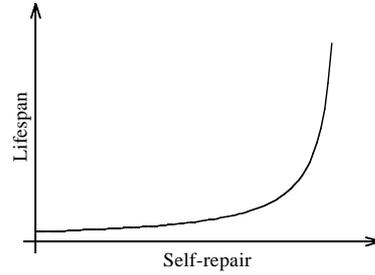


Fig. 17.1 The expected relationship between the lifespan and the degree of self-repair in the tissue of the organism.

if $s = X$ because, then it uses no energy on self-repair.

For the demographic model that was defined in Chapter 7 we have that $\lambda = pT\epsilon/wTB$. The presence of a T in the denominator of this expression is based on the assumption of a constant ω . As in this chapter we are modelling the evolutionary changes in ω this assumption will no longer hold. So, to avoid problems we can exchange the dominator- T with a constant, and to simplify let us set the dominator- T equal to one. Then $\lambda = pT\epsilon/wB$, and with the numerator- T being equal to \dot{z}/s we have that $\lambda = p\dot{z}\epsilon/wsB$. We have now defined the physiological relationship between senescence and self-repair so that we can analyse the evolution of soma and senescence under the classical hypothesis of exploitative competition and the alternative hypothesis of competitive interference.

17.3.2 Classical theory and unclear prediction

In the classical theory with exploitative competition selection will operate on the degree of senescence in the sense that the energy not used on self-repair (sw) can enhance the intrinsic Malthusian parameter if it is used on the demographic traits instead. To describe this mathematically, let me assume that the energy taken from self-repair and used on the demographic traits are allocated to reproduction. Then we have that there is $\epsilon + sw$ amounts of energy available for reproduction and, thus, we obtain the following fitness profile

$$\lambda^* = p\dot{z}(\epsilon + sw)/wsB \quad (17.2)$$

Then, as $r^* = \ln \lambda^*$, the selection gradient on s is

$$\partial r^*/\partial s = -\epsilon/s(\epsilon + sw) \quad (17.3)$$

As this gradient is negative we find that $s \rightarrow 0$ and, thus, we expect maximal self-repair and potential immortality. If instead, it is assumed that p and ϵ

are functionally determined by s , then it is relatively easy to construct models for which senescence will evolve even in the absence of intra-population interference competition (see Kirkwood and Rose, 1991).

If by these results we want to explain the dichotomy in the presence versus absence of senescence and soma between prokaryotes and the higher eukaryotes the crucial point would be to determine why the intrinsic trade-offs in prokaryotes differ from those in the higher eukaryotes so that the latter group evolves senescence, while the former group does not. As I am not aware that such a mechanism has yet been identified I will leave the classical hypothesis at this point.

17.3.3 Competitive interactions and a clear transition

In the presence of density dependent competitive interactions the energy not used for self-repair (sw) can be allocated to competitive interactions. In this case we have that the parameter s will represent competitive quality and, therefore, if we follow the procedure in Chapter 10, we find that the discrete growth rate of the i th variant in s is $\lambda_i^* = p\zeta\epsilon e^{\psi\iota^{*e}(\ln s_i - \ln s)}/ws_iB$, where ι^{*e} is the evolutionarily determined level of intra-population interference as it is determined by selection on the major traits like body mass and metabolic rate. Now, because the discrete growth rate of the average variant is one at the population equilibrium, i.e., because $\lambda^* = 1$, we can divide $\lambda^* = p\zeta\epsilon/wsB$ into λ_i^* and rearrange and find that the discrete growth rate of the i th variant reduces to

$$\lambda_i^* = (s_i/s)^{\psi\iota^{*e}-1} \quad (17.4)$$

Then, as $x_i^* = \ln \lambda_i^*$, the selection gradient at the limit of the average variant is

$$\lim_{s_i \rightarrow s} \partial x_i^* / \partial s_i = (\psi\iota^{*e} - 1)/s \quad (17.5)$$

From this equation we have that the selection gradient for senescence is positively related to the evolutionarily determined level of interference in the population. We may then recall from Chapter 14 that the body masses of small organisms, like prokaryotes, are likely to be evolutionarily constrained to a lower limit where intra-population interference is almost absent ($\iota^{*e} \approx 0$) and there is constant selection for a decline in body mass. If this interpretation is correct, then by eqn 17.5 we predict that prokaryotes are exposed to selection against senescence. If instead, the body mass is large the organism is expected to be situated at the evolutionary steady state where $\psi\iota^{*e} \approx 2$ (eqn 14.7). By eqn 17.5 this implies that large-bodied organisms are exposed to selection for senescence and, thus, also to selection for a soma. That is to say that a large body mass, a soma, and senescence are co-occurring traits, as is the case in the higher and mobile eukaryotes.

Chapter 18

Group size

IT IS ONLY rarely that group formation is absent. In many species a male and a female form a pair, and pairs may be uniformly distributed, or they may aggregate into larger groups, such as colonies. In other species, like cooperatively breeding birds, a breeding pair is surrounded by one to several helpers, and in eusocial ants, termites, and bees the helpers include thousands, or even millions, of non-breeding siblings. In other words, there is a variety of deviations from the simple pattern of uniformly distributed individuals.

In relation to the evolution of these different types of groups we notice that there are three different levels of phenotypic traits upon which selection can operate. The first level is simply the group size. The second level is the partitioning of the group into individuals that perform different tasks, i.e., the partitioning of pairs into males and females, the partitioning of colonies into pairs, the partitioning of cooperative groups into pairs and helpers, and the partitioning of eusocial colonies into queens, sexual males, and workers. And finally, the third level is the genetical structure by which the different individual within the groups are related to one another, i.e., the structure of sexual reproduction where the offspring receives half of its genes from the mother and the other half from the father, and the structure that both the helpers in cooperative breeders and the workers in eusocial colonies are the offspring of the sexuals that reproduce.

In the framework of the classical theory of evolution it has been the custom to regard the propagation of the selection pressure through these three phenotypic levels as being “bottom-up”, or “inside-out”, in the sense that it is the genetical structure that is most fundamental promoting the evolution of the traits at the higher phenotypic levels. This hypothesis is probably most strongly expressed in the Hamlintonian hypothesis that kin selection is the essential component driving the evolution of cooperative

breeding and the evolution of eusocial colonies, but it is equally present in the fact that we tend to regard pair formation as a consequence of sexual reproduction.

In this and the following four chapters I will show that it is more likely that the propagation of the selection pressure through the three levels of the phenotype actually resembles a reverse “top-down”, or “outside-in”, structure. This conclusion is reached because it generally is shown that the predictions from the classical “bottom-up” propagation are evolutionarily unstable, while the predictions from the reverse “top-down” propagation are evolutionarily stable.

By a “top-down” propagation I mean that it is the selection pressure on group size that is most fundamental, that individuals that perform different tasks are the evolutionary consequence of group formation, and that it is the selection pressure generated at these two levels that promotes the evolution of the underlying genetical structure, including both the degree of relatedness among the individuals in the group and the degree to which different types of sexual reproduction evolves. According to this latter hypothesis it is, among other things, kin selection that is the evolutionary consequence of eusociality, and sexual reproduction that is the evolutionary consequence of pair formation.

In order to show all this I will first proceed through three chapters: each chapter describing evolution at a phenotypic level that is successively more intrinsic to the organisms. In this first chapter I will deal only with the evolution of group size. In Chapter 20 I will deal with the evolutionary partitioning of the optimal group into male and female individuals and then, in Chapter 21, I will deal with the evolution of sexual reproduction, including the evolution of the diploid and the haplodiploid genome. Having then described evolution at the three phenotypic levels for organisms that breed solitarily or in pairs, in Chapter 22 I will deal with evolution at all three levels for the two special situations with cooperative breeding and eusocial colonies. In all these chapters it is selection by the ecological constraints of density dependent competitive interactions that is the driving force of selection promoting the evolutionary stability of the different structures at all phenotypic levels. In other words, if the action of density dependent competitive interactions vanishes, e.g., due to a long period with a declining primary production, then the organisation at the three phenotypic levels is expected to vanish in the sense that all that will remain on a uniform resource is uniformly distributed individuals that have a haploid genome and asexual reproduction.

In this chapter where I am interested only in the evolution of group size the intriguing question is why the individuals on a uniform resource will aggregate into groups instead of being uniformly distributed. Actually, it is

not that easy to explain why groups will evolve in a uniform environment, and this is because both the local exploitation of the resource and the local level of interference will increase when the individuals aggregate into groups. This means that an individual that joins a group is exposed to a cost, and that there is selection for uniformly distributed individuals unless the group will also provide a gain that can out-balance the cost.

In this chapter I will use the gain that arises from interference competition among groups to explain the evolution of groups. This gain arises because larger groups are competitively superior to smaller groups and, thus, the large group can prevent that the small group has access to the resource during the competitive encounter. The success by which the large group will dominate the small group will depend upon the number of encounters between the groups. If the groups do not encounter, the larger groups cannot dominate the smaller groups, and this implies that the larger group cannot provide a fitness gain and, thus, there is selection for uniformly distributed individuals. If, instead, the groups encounter one another in rapid succession, then the larger groups can dominate the smaller groups and, thus, there is selection for the formation of groups. More generally, we will see that the optimal group size is positively related to the number of competitive encounters between groups, and that it is inversely related to the cost associated with the formation of groups. But before I analyse the evolution of group size I will first describe the cost associated with them.

18.1 Cost of grouping

Let us consider the cost of grouping when it is defined relative to the background of a uniform resource with uniformly distributed individuals. Let z be the tightness of an average group, with $z = 0$ describing the limit where no groups are formed and the individuals are uniformly distributed, and $z = 1$ describing the limit of completely formed groups. Then, let $\dot{\epsilon}$ be the assimilated resource available for reproduction per individual when the individuals are uniformly distributed, and let $k\dot{\epsilon}$, with $0 \leq k \leq 1$, be the energetic cost per individual in a completely formed group. Then, we find that the energy available per individual (ϵ) during the transition from uniformly distributed individuals ($z = 0$) to completely formed groups ($z = 1$) can be approximated by the linear function

$$\epsilon = \dot{\epsilon}(1 - kz) \quad (18.1)$$

Apart from being a function of the tightness of the group, the cost of group formation is also a function of the number of individuals (n) that form the group. This is because the higher the number of individuals in the group, the higher the local level of exploitation and the local level of interference and,

thus, the larger the cost. To describe this let us first consider the case where a tight group of n individuals have the same amount of resource available as a single individual when the individuals are uniformly distributed. Then, $n\epsilon = \dot{\epsilon}$ for $z = 1$. From this expression we obtain $\epsilon = \dot{\epsilon}/n = \dot{\epsilon}[1 - (n - 1)/n]$, and this expression can be extended to any level of tightness so that the energy available per individual is

$$\epsilon = \dot{\epsilon}[1 - (n - 1)z/n] \quad (18.2)$$

Let us now extend eqn 18.2 to the general case where a tight group of n individuals is v times better to assimilate resource than a single individual, when the individuals are uniformly distributed. That is, let $n\epsilon = v\dot{\epsilon}$ for $z = 1$. Then, when $v = n$ we find that the individuals are equally efficient to assimilate resource, whether they are in groups or not. If instead, $v > n$ we have a situation where group formation can enhance the individual's ability to assimilate resource, and this can, e.g., be the case when the individual's ability to utilise the resource depends upon cooperation with other individuals. The most general situation is, though, expected to be the case $v < n$, where group formation is costly because it increases both the local exploitation of the resource and the local level of interference.

In order to extend eqn 18.2 to the general situation with a variable v let us divide $n\epsilon = v\dot{\epsilon}$ by n so that we obtain $\epsilon = (v/n)\dot{\epsilon}$, which is the amount of resource assimilated by an average individual in a tight group of size n . Then, by following the procedure for eqn 18.2, we can extend the expression $\epsilon = (v/n)\dot{\epsilon}$ so that it will apply to any level of group tightness:

$$\epsilon = \dot{\epsilon}[1 - (n - v)z/n] \quad (18.3)$$

From this equation we have that the energetic cost of an individual joining a group is $k = (n - v)z/n$. As this cost is largest in the point $z = 1$, we find that a tight group with a given n and v can evolve when selection is strong enough to carry the population from $z = 0$ to $z = 1$ for those values of n and v . In the following section we will analyse when this transition is possible.

18.2 Evolution of group size

In the description of the evolution of group size let us first examine the simple situation where the conditions for group formation is given by the exploitation of the resource. Then, on a uniform resource, we expect that the individuals will be uniformly distributed when $v < n$ and group formation is costly due to the increase in the local exploitation of the resource. When instead $v > n$, and the individual's ability to utilise the resource is increased

by cooperation, we expect that the individuals will aggregate into groups. In this situation we also expect that the optimal group size will be the size where the ability of each individual to utilise the resource is at its maximum.

To shown mathematically this aggregation of individuals into groups let us insert eqn 18.3 into the growth rate $\lambda^* = p\epsilon/wB$ and obtain

$$\lambda^* = p\epsilon[n - (n - v)z]/wBn \quad (18.4)$$

Then the selection gradient on group tightness is

$$\frac{\partial r}{\partial z} = -\frac{n - v}{n - (n - v)z} \quad (18.5)$$

This gradient is negative when group formation is costly, i.e., when $v < n$, and it is positive when $v > n$, i.e., when group formation is beneficial due to cooperation in relation to the exploitation of the resource. Now, as this latter situation is a relatively special, although probably common, situation we may generally expect that group formation will not occur on a uniform resource when competition is purely exploitative.

Let us now turn to the case with density dependent interference competition. In this situation we have that a large group generally can dominate a small group during a competitive encounter and we thus expect that individuals will aggregate into groups and cooperate in inter-group interference competition. To describe in more detail in which circumstances this is possible let us first assume a constant group size, and then let us describe the degree to which the differences in the tightness of the different groups causes energetic differences among the groups. As, on a uniform resource, the energetic differentiation among groups is caused by interference competition between groups we have that the energetic differentiation generally will be in favour of the tighter groups, i.e., larger z , and this is because it is these groups that are expected to be better to defend their resource items. Then, if we assume that the genetic variation in $\ln z$ is invariant with respect to z , we can follow the procedure in Chapter 10 and describe the discrete growth rate of the i th variant as $\lambda_i^* = p\epsilon_i e^{\psi\iota^{*e}(\ln z_i - \ln z)}/wB$, where ϵ_i is given by eqn 18.3 and ι^{*e} is the evolutionarily determined level of inter-group interference. Then, to reduce this two-dimensional fitness profile, we recall that the discrete growth rate of the average variant is one, and therefore we can divide the average growth rate into the growth rate of the i th variant and obtain

$$\lambda_i^* = \frac{n - (n - v)z_i}{n - (n - v)z} e^{\psi\iota^{*e}(\ln z_i - \ln z)} \quad (18.6)$$

Then the selection gradient on average group tightness is

$$\lim_{z_i \rightarrow z} \frac{\partial x_i^*}{\partial z_i} = \frac{\psi\iota^{*e}}{z} - \frac{n - v}{n - (n - v)z} \quad (18.7)$$

As larger groups generally are competitively superior to smaller groups we expect that the group size will evolve to be as large as possible, and in terms of eqn 18.7 this implies that the optimal group size is the size that is in evolutionary equilibrium for $z = 1$. That is, by eqn 18.7, we find that the optimal group size is

$$n^{**} = v(\psi\iota^{*e} + 1) \quad (18.8)$$

Hence, the optimal group size is positively related to v , i.e., to the degree by which a tight group of n individuals is better to assimilate resource than a single individual, and to ι^{*e} , i.e., to the evolutionarily determined level of inter-group interference.

In order to analyse the relationship between the group size and the evolutionarily determined level of inter-group interference let us note (*i*) that if the resource is divided evenly among the individuals within a group, (*ii*) or if the group resembles the replicating unit, as is the case when the group is either an individual, a pair, a group of cooperative breeders, and a eusocial colony, then the level of inter-group interference (ι^{*e}) is defined by the degree to which the major traits, like body mass and metabolic rate, are evolutionarily equilibrated. Then, from the chapters on body mass we recall that $\psi\iota^{*e} = 0$ when the body mass is at its lower limit (Section 14.4.1), that $\psi\iota^{*e} = 1$ when the body mass is in evolutionary equilibrium (eqn 10.23), that $\psi\iota^{*e} \approx 2$ when the body mass is in evolutionary steady state (eqn 14.7), and that $\psi\iota^{*e} \rightarrow \infty$ when the body mass is upward constrained and there is plenty of resource (Section 14.4.2). In Fig. 18.1a-d I have illustrated the group formation evolving in these four situations, given that the cost of group formation is close to its maximum, i.e., $v \approx 1$, and given that the resource is uniform. In Chapters 21 and 22 I will show that these four situations coincide with a uniform distribution of respectively asexual individuals, sexually reproducing pairs, cooperative breeders, and eusocial colonies.

The situation with a constrained body mass, a high level of interference competition, and a eusocial colony is not the only way to obtain a large group size from selection by density dependent competitive interactions. The other way is when there is only a small cost associated with the formation of the group, i.e., when there is an almost proportional relation between v and n . In this latter case we find, from eqn 18.8, that the group size is proportional to $2v$ or $3v$ when the body mass is in either evolutionary equilibrium or in evolutionary steady state. In Chapter 22 we will find that this situation resembles a colony with sexually reproducing pairs that may or may not have a helping offspring attached.

Let us now estimate the energetic cost imposed on the individual by the formation of an optimally sized group. By eqn 18.3 this cost is $(n -$

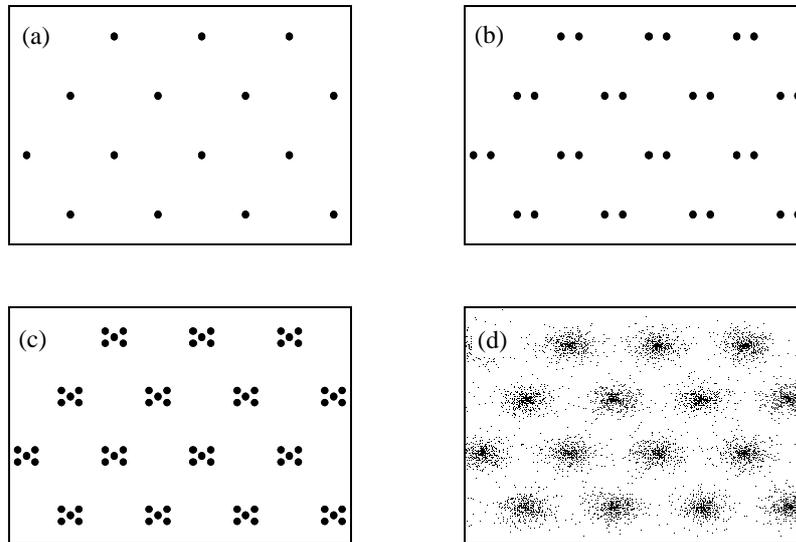


Fig. 18.1 The distribution of individuals on a uniform resource as predicted by eqn 18.8 in the absence (a) and presence (b - d) of intra-population interference. (a) Uniformly distributed individuals. (b) Uniformly distributed pairs. (c) Uniformly distributed cooperative breeders. (d) Uniformly distributed eusocial colonies.

$v)/n$, and this implies that it is 50% when the body mass is in evolutionary equilibrium, $\approx 70\%$ at the evolutionary steady state, and close to 100% when a large group is formed because the body mass is upward constrained. Despite these heavy costs it is still evolutionarily beneficial for the individual to be in the group, and this is because the group is better to compete by interference than is the single individual.

We have now seen that selection by density dependent competitive interactions can explain the general tendency that individuals have to aggregate into groups, although in some instances group formation may also be explained by cooperation in relation to the exploitation of the resource, or simply as protection against predators. Having obtained these results, in Chapters 20 to 22, I will show how the formation of optimally sized groups is associated with a “top-down” form of selection pressure explaining both (i) the evolution of individuals that perform different tasks, like males, females, and eusocial workers, and (ii) the evolution of the genetical structure underlying the four situations illustrated in Fig. 18.1. But before this is done I will deal with the classical theory based on the diametrically opposite hypoth-

esis, where it is the genetical structure that imposes a “bottom-up” form of selection that should be able to explain both the evolution of individuals performing different tasks and the aggregation of these individuals into groups. This is done especially in the next chapter where I show that the classical hypothesis is unlikely to hold because (i) the genetical structure in sexuals cannot explain the evolution or maintenance of the male individual, and because (ii) the genetical structure tends to be evolutionarily unstable in itself.

Chapter 19

Fisherian sex ratios

THE SEX RATIO is the number of males per female, and its evolution has traditionally been modelled by the classical sex ratio theory that arose from Fisher (1930). According to this theory the sex ratio is a derived trait that evolves from the constraints of genomic selection, and these constraints are defined by the differences in the rates whereby the genes in females and males are transmitted to the next generations through sexual reproduction. This type of selection depends on a variety of assumptions that, among other things, include the ploidy level of the genome, the mating structure, and the relative mass of males to females.

Since Fisher considered the evolution of sex ratios his theory has been extended in a variety of directions, and today it is often seen as one of the most successful applications of theoretical evolutionary biology (e.g., Charnov, 1993; Bulmer, 1994). This is largely because the selection pressure can be calculated from first principles without the use of empirical estimates, and because this procedure often leads to strikingly exact predictions. Despite this success the Fisherian sex ratio theory suffers from the same problem as the rest of the classical theory of evolution. That is to say that the theory fails on an evolutionary scale because it produces predictions that are evolutionarily unstable.

The predictions of the Fisherian sex ratio theory are evolutionarily unstable because sexual reproduction is costly in the framework of the classical theory. Here, sexual reproduction is four-fold costly, and this is because both the sexual male and the meiotic separation of the diploid set of genes into haploid gametes are two-fold costly. Due to these costs there is selection for asexual reproduction, and this implies that the predictions of the Fisherian sex ratio theory fail because males do not exist at the evolutionary equilibrium.

In this chapter I will first review some of the major predictions of the

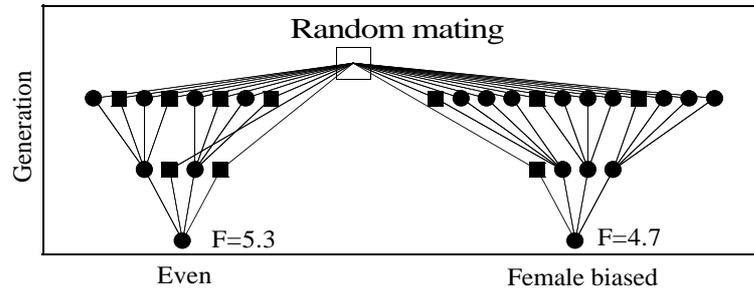


Fig. 19.1 An illustration of the fitness of two variants with respectively an even and a female biased sex ratio, when mating occurs at random at the population level and the two variants are equally abundant. The circles represent females, the squares represent males, and the lines indicate the transfer of genes with the open square indicating random mating. The fitness (F) of a particular gene in each grandmother is given as the average number of copies of that gene in the grand-offspring. Note, that the variant with the even sex ratio has the highest fitness even though the intrinsic growth rate in terms of individuals is highest in the lineage with a female biased sex ratio (compare with Fig. 19.3 where this is not the case).

Fisherian sex ratio theory, starting with the standard condition with one male per female, and then dealing with investment sex ratios, sex ratios in eusocial species, and finally female biased sex ratios due to the action of local mating. Then, having described some of the classical principles, I will focus on the four-fold cost of sex and show that the Fisherian equilibria fail as evolutionary predictions due to the action of both individual and genomic selection.

19.1 One male per female

According to Fisher (1930) there is one male per female because each offspring has one mother and one father. That is to say that it pays to invest in the rare sex because it is an individual of that sex that transmits most genes to the following generation. When there is one male per female there is an equilibrium where an average female and an average male transmit the same amount of genes to the next generation. This type of selection is illustrated in Fig. 19.1.

To analyse this type of selection mathematically let us follow the general consensus and assume that it is the female that controls the sex ratio. We can then define the fitness of the i th variant as the rate by which the offspring of the female copies her genes into the future. This rate is the

sum between the rate by which the mother's genes are copied through her daughters and the rate by which her genes are copied through her sons, and for each offspring sex this rate has three multiplicative components. For the daughters these three components are (i) the number of daughters per female, (ii) the relative value of a daughter compared with a son in projecting genes into the future [denoted $v_d = r_d/(r_d + r_s)$, where the subscript s stands for son, and r_d and r_s are the value of respectively a daughter and a son], and (iii) the transcription probability of the daughter relative to the mother, defined as the probability that a gene copied from a daughter to an offspring of that daughter is identical by descent to a gene in the mother [denoted $p_{d,m}$, where the subscript d stands for daughter and subscript m stands for mother]. For the sons the three multiplicative components are (i) the number of females inseminated by sons, (ii) the relative value of a son compared to a daughter in projecting genes into the future [denoted $v_s = r_d/(r_d + r_s)$], and (iii) the transcription probability of the son relative to the mother, defined as the probability that a gene that is copied from a son to an offspring of that son is identical by descent to a gene in the mother [denoted $p_{s,m}$, where the subscript s stands for son]. Note, that these expressions are essentially kin selection arguments, and that the transcription probabilities resemble the Hamiltonian relatedness coefficients that are usually used in connection with kin selection arguments. For a review on Fisherian sex ratios based on the relatedness coefficients see, e.g., Bulmer (1994).

To obtain the fitness of the i th variant, let θ be the proportion of females in the population, let $\phi = 1 - \theta$ be the proportion of males, let θ_i and ϕ_i be respectively the proportion of females and males for the i th variant in that population, and let n be the number of offspring per female, assumed to be the same for the different variants. Then, for a female of the i th variant, the number of daughters is $n\theta_i$, and the number of females inseminated by sons is $n(\theta/\phi)\phi_i$. Hence, the fitness of the i th variant is

$$\begin{aligned}\lambda_i &= n[X(1 - \phi_i) + Y(\theta/\phi)\phi_i] \\ X &= p_{d,m}v_d \\ Y &= p_{s,m}v_s\end{aligned}\tag{19.1}$$

because $1 - \phi_i = \theta_i$. The selection gradient on the average proportion of males is then the partial derivative of eqn 19.1 with respect to ϕ_i at the limit $\phi_i \rightarrow \phi$. Hence,

$$\lim_{\phi_i \rightarrow \phi} \partial\lambda_i/\partial\phi_i = n[Y(\theta/\phi) - X]\tag{19.2}$$

Then, from eqn 19.2, we find that the equilibrium, or Fisherian, sex ratio is $(\phi/\theta)^{*F} = Y/X$, and that the Fisherian proportion of females is $\theta^{*F} = X/(X + Y)$.

In diploids the transfer of genes through daughters and sons is symmetrical because half of the genes in the diploid zygote come from the father while the other half come from the mother. Due to this symmetry it follows that $p_{d,m} = p_{s,m} = 1/2$ and that $v_d = v_s = 1/2$. Thus, the two products $X = p_{d,m}v_d$ and $Y = p_{s,m}v_s$ are the same ($1/4$) and, thus, the Fisherian sex ratio is one [$(\phi/\theta)^{*F} = 1$], as predicted by Fisher in 1930.

In haplodiploids the transfer of genes through daughters and sons is asymmetrical, and this is because the diploid female transmits genes to all offspring, whereas the haploid male transmits genes only to daughters. Due to this asymmetry it follows that some of the $p_{i,j}$ and v_j terms will differ from $1/2$, their value in diploids. In haplodiploids the transcription probability of the daughter relative to the mother remains one half, i.e., $p_{d,m} = 1/2$. But, as it is only mothers that transmit genes to sons it follows that $p_{s,m} = 1$. Furthermore, because a son transmits genes only to the granddaughters of the mother, while a daughter transmits genes to both granddaughters and grandsons, it follows that the daughter is twice as efficient as the son in transmitting genes, i.e., it follows that $v_d = 2/3$ and $v_s = 1/3$ [for a more formal deduction see Taylor (1988) or Bulmer (1994:190)]. This implies that the two products $X = p_{d,m}v_d$ and $Y = p_{s,m}v_s$ are the same ($1/3$) and, thus, the Fisherian sex ratio is one, as in diploids.

19.2 Investment sex ratios

A Fisherian sex ratio of one depends upon a variety of implicit assumptions. As shown by Fisher (1930) and Kolman (1960), one of the more important assumptions is that the parents invest the same amount of resource in a daughter as they do in a son. In eqn 19.1 this assumption is expressed through the constraint that the total number of offspring per female is constant independently of the sex ratio.

In the more general situation we have that the mass of a female (w_θ) and a male (w_ϕ) offspring may differ, and that a female has a given amount of resource (ϵ) that she can invest in her offspring. This implies that it is the resource invested in the male offspring plus the resource invested in the female offspring that is constant, and not the total number of offspring. In mathematical terms this constraint can be expressed as $n\phi w_\phi + n\theta w_\theta = \epsilon$, if it is assumed that the body mass of an offspring (w) represents the resource invested in that offspring. If we normalise with respect to the body mass of the male we find that $n\phi + (w_\theta/w_\phi)n\theta = c$, with $c = \epsilon/w_\phi$ being a constant. Hence, for the i th variant we find that the number of daughters produced per mother is $n_i\theta_i = (c - n_i\phi_i)(w_\phi/w_\theta)$. Then, as the number of females inseminated by sons is unaffected by the transition to investment sex ratios,

we find that the fitness expression of eqn 19.1 is

$$\lambda_i = X(c - n_i\phi_i)(w_\phi/w_\theta) + Y(\theta/\phi)n_i\phi_i \quad (19.3)$$

This implies that the selection gradient is

$$\lim_{n_i\phi_i \rightarrow n\phi} \frac{\partial \lambda_i}{\partial n_i\phi_i} = Y \frac{\theta}{\phi} - X \frac{w_\phi}{w_\theta} \quad (19.4)$$

Hence, when $X = Y$ as it is the case in diploids and haplodiploids, the Fisherian sex ratio is

$$\left(\frac{\phi}{\theta}\right)^{*F} = \frac{w_\theta}{w_\phi} \quad (19.5)$$

This means that it is the energetic investment in each sex that is even, i.e., that $\phi^{*F}w_\phi = \theta^{*F}w_\theta$. Empirical studies indicate that it is the theory on investment sex ratios, rather than the theory on numerical sex ratios, that is confirmed by empirical evidence (e.g., Metcalf, 1980; Trivers and Hare, 1976).

19.3 Sex ratios in eusocial species

In eusocial insects the majority of a colony are sterile workers, and the sex ratio refers to the number of sexual males over sexual females (queens). If the relative production of sexual males and females from a eusocial colony is determined by the queen, then the Fisherian sex ratio is one, as it was shown in the previous sections. However, Trivers and Hare (1976) and Charnov (1978) noticed that the sex ratio in the sexuals that are produced by a eusocial colony is likely to be controlled by the workers, and this is because the workers are numerically far superior to the queen. This type of worker control is possible if the workers allocate the resources differentially between the two sexes that are laid by the queen. When this is the case we have that the Fisherian sex ratio might differ from one, and that this sex ratio can be determined by an elaboration of the inclusive fitness argument behind eqn 19.1.

To elaborate on eqn 19.1 so that it applies to eusocial species we can change the transcription probabilities of respectively daughters and sons, so that they become relative to a worker instead of being relative to the mother. These new transcription probabilities describe the probabilities that a gene which is copied from a sexual daughter or son to a sexual grand-offspring of the mother (queen) is identical by descent to a gene in a worker, and they are denoted respectively $p_{d,w}$ and $p_{s,w}$, where the w subscript indicates the worker. The relevant fitness expression is then

$$\begin{aligned}
\lambda_i &= n[X(1 - \phi_i) + Y(\theta/\phi)\phi_i] \\
X &= p_{d,w}v_d \\
Y &= p_{s,w}v_s
\end{aligned}
\tag{19.6}$$

where the two new transcription probabilities are defined as

$$\begin{aligned}
p_{d,w} &= p_{d,m}p_{m,w} + p_{d,f}p_{f,w} \\
p_{s,w} &= p_{s,m}p_{m,w} + p_{s,f}p_{f,w}
\end{aligned}
\tag{19.7}$$

where $p_{d,m}$ and $p_{s,m}$ are the transcription probabilities in eqn 19.1, $p_{d,f}$ and $p_{s,f}$ are the transcription probabilities of daughters and sons relative to the father (subscript f), and $p_{m,w}$ and $p_{f,w}$ are the transcription probabilities of the mother and father relative to the worker, i.e., the probabilities that the gene in question was copied also from the mother or father to the worker.

In eusocial termites the genomes in both males and females are diploid, and the workers are both sterile females and sterile males. Due to the symmetry of this system it follows that all the probabilities in eqn 19.7 are one half. Hence, as $v_d = v_s = 1/2$ in diploids, we find that $X = Y = 1/4$ and, thus, that the Fisherian sex ratio $(\phi/\theta)^{*F} = Y/X$ is one under worker control. In other words, there is no conflict between the queen and the workers over the sex ratio.

This is in contrast to eusocial hymenoptera, like ants and bees, where there is a conflict because the genome is haplodiploid and all the workers are sterile females. Here, both a worker and a sexual daughter have a copy of all the genes in their common father and a copy of half of the genes in their common mother. This implies that we obtain the following transcription probabilities: $p_{d,m} = 1/2$, $p_{m,w} = 1/2$, $p_{d,f} = 1/2$, $p_{f,w} = 1$, and $p_{d,w} = 3/4$. Also, as a sexual male has a copy of half of the genes in his mother and no copies of any of the genes in his “father”, it follows that $p_{s,m} = 1$, that $p_{m,w} = 1/2$, that $p_{s,f} = 0$, that $p_{f,w}$ is undefined and, thus, that $p_{s,w} = 1/2$. Then, as $v_d = 2/3$ and $v_s = 1/3$ in haplodiploids, we find that $X = (3/4)(2/3) = 1/2$ and that $Y = (1/2)(1/3) = 1/6$. Hence, the Fisherian sex ratio $(\phi/\theta)^{*F} = Y/X$ is female biased and given as

$$\left(\frac{\phi}{\theta}\right)^{*F} = \frac{1}{3}
\tag{19.8}$$

A female biased sex ratio of $\approx 1/3$ is often found in eusocial hymenoptera (e.g., Trivers and Hare, 1976; Nonacs, 1986; Boomsma, 1989; Crozier and Pamilo, 1996), whereas an even sex ratio appears to be the case in many termites.

19.4 Local mating and female biased sex ratios

A Fisherian sex ratio of one also depends upon an assumption of population-wide random mating. As shown by Hamilton (1967), when mating occurs more locally the Fisherian sex ratio is female biased. By local mating we mean that individuals tend to mate with a relatively permanent set of neighbours that are more closely related to one another than the average relatedness in the population. In such cases we have that the sons of a single female will compete with one another for a limited number of matings. Hence, it will pay to invest less in sons because the more sons a female produces the fewer matings each of them will get.

To analyse the case with local mating let mating occur at random within groups that are founded by s females that each produce n offspring. Then, for the i th variant the number of daughters per female is $n\theta_i$, while the number of females inseminated by sons is $n\phi_i[(s-1)\theta + \theta_i]/[(s-1)\phi + \phi_i]$. Notice, that when $s \rightarrow \infty$, i.e., when the mating structure converges to population wide mating, then the number of females inseminated by sons converges to $n(\phi/\theta)\phi_i$, which is the expression used in eqn 19.1. Thus, in the general case with an arbitrary subdivision of the population, the fitness of the i th variant is

$$\lambda_i = nX \left(1 - \phi_i + \frac{[(s-1)(1-\phi) + 1 - \phi_i]\phi_i}{(s-1)\phi + \phi_i} \right) \quad (19.9)$$

if we assume that $X = Y$ as is the case in diploids and haplodiploids. The selection gradient then is

$$\lim_{\phi_i \rightarrow \phi} \frac{\partial \lambda_i}{\partial \phi_i} = nX \frac{s - 2s\phi - 1}{s\phi} \quad (19.10)$$

and the Fisherian sex ratio

$$\left(\frac{\phi}{\theta} \right)^{*F} = \frac{s-1}{s+1} \quad (19.11)$$

This implies that the sex ratio is zero at the limit $s = 1$, where brothers mate with sisters, and that it is one at the other limit $s \rightarrow \infty$, where mating is random at population level.

The result of eqn 19.11 depends upon an assumption of no inbreeding (Hamilton, 1967, 1972), and this does not hold when breeding occurs locally, because in such cases there is an increased chance that brothers will mate with sisters. These matings imply that the probability that a gene copied from a diploid offspring is identical by descent to a gene in the mother is

increased above one half. In diploids this effect will not affect the sex ratio, and this is because both daughters and sons are diploid, so that inbreeding operates symmetrically on both sexes. More explicitly, the impact of inbreeding in diploids implies that $p_{d,m} = p_{s,m} > 1/2$, and thus X is still equal to Y and the prediction of eqn 19.11 applies.

This is not the case in haplodiploids where all the genes in sons come from the mother independently of the degree of inbreeding, i.e., $p_{s,m} = 1$ always. Hence, inbreeding in haplodiploids will operate only through daughters, and this implies that $p_{d,m} > 1/2$ and, thus, that the sex ratio will be distorted in relation to the prediction of eqn 19.11. It has been shown, e.g., by Bulmer (1994:237), that when inbreeding occurs through local mating, then $p_{d,m} = s/(2s - 1)$ in haplodiploids. This implies that $X = (s/[2s - 1])(2/3)$, while Y is still $1/3$ and, thus, the fitness expression in eqn 19.9 becomes

$$\lambda_i = \frac{n}{3} \left(\frac{2s(1 - \phi_i)}{2s - 1} + \frac{[(s - 1)(1 - \phi) + 1 - \phi_i]\phi_i}{(s - 1)\phi + \phi_i} \right) \quad (19.12)$$

The selection gradient on the fraction of males is then

$$\lim_{\phi_i \rightarrow \phi} \frac{\partial \lambda_i}{\partial \phi_i} = \frac{n}{3} \frac{(s - 1)(2s - 1) - s\phi(4s - 1)}{s\phi(2s - 1)} \quad (19.13)$$

and the optimal sex ratio

$$\left(\frac{\phi}{\theta} \right)^{*F} = \frac{(s - 1)(2s - 1)}{s(4s - 1) - (s - 1)(2s - 1)} \quad (19.14)$$

The inverse relations in eqns 19.11 and 19.14 between the sex ratio and the degree of local mating, has been confirmed in a variety of insects that have female biased sex ratios (e.g., Hamilton, 1967; Werren, 1983; Herre, 1985).

19.5 Four-fold cost of sex and limits to Fisherian sex ratios

In the previous sections in this chapter we examined some of the major predictions in the classical theory on the evolution of sex ratios. In this section I will show that these predictions fail on an evolutionary scale because they are evolutionarily unstable. The classical predictions are evolutionarily unstable because sexual reproduction between a female and a male is costly in the classical theory and, thus, there is selection for the absence of males, and when males are absent the term “sex ratio” is losing its meaning.

Today, it is generally believed that the cost of sexual reproduction between females and males is two-fold (e.g., Maynard Smith, 1971; Michod

and Levin, 1988; Bulmer, 1994). But this holds only at the limit where the investment sex ratio is completely female biased, and males and females still exist. Such a strong bias can arise only when the numerical sex ratio is extremely female biased, or when the male individual is extremely small compared with the female. In these organisms sex is two-fold costly because they experience only the two-fold cost of meiosis, and not the two-fold cost of the male. More generally, sexual reproduction between males and females is four-fold costly because organisms that allocate an equal amount of resource to males and females experience both the two-fold cost of meiosis and the two-fold cost of the male. In this section I will focus first on individual selection and the two-fold cost of the male and, then on genomic selection and the two-fold cost of meiosis. This is done in order to show, respectively, that the Fisherian sex ratios are evolutionarily unstable due to the action of both individual and genomic selection.

19.5.1 Two-fold cost of males

In the traditional treatment of the Fisherian sex ratios it is the custom to disregard individual selection that is defined by the absolute rates by which the individuals of the different variants increase in numbers. In other words, it is implicitly assumed that it is genomic and not individual selection that determines the evolutionary setting of the sex ratio in natural population. This assumption is unlikely to hold, and this is because in the situation with independence between the two types of selection it is individual, and not genomic, selection that determines the evolutionary setting of the phenotype (see Chapter 8).

In relation to Fisherian sex ratios the case with independence between genomic and individual selection resembles the example in Fig. 19.2. Here, genomic selection is operating only within the sexual lineage, while it is individual selection that operates between the asexual and the sexual variants. This figure is based on the traditional reasoning where the fitness of the male is given by the number of females he inseminates. According to this hypothesis the absolute growth rate of a variant is proportional to the proportion of females in the offspring, and this is because each female produce a given number of offspring. This implies that the male is two-fold costly in the sense that the sexual pair produces only half the number of offspring produced by the asexual pair. Evidently, in this case, the asexual variant will out-reproduce the sexual variant, and this implies that if asexual or parthenogenic mutants arises, as they do in many natural populations, then the classical sex ratio theory does not explain the evolution of sex ratios, because it predicts only the evolution of asexual or parthenogenic species.

Another way to show that the Fisherian sex ratio theory is evolutionarily

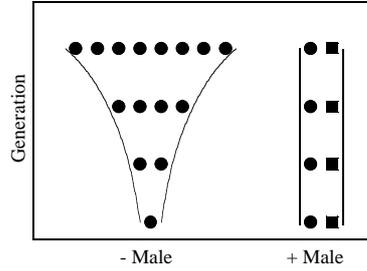


Fig. 19.2 An illustration of the two-fold cost of the male when females (circles) produce two offspring per lifetime. The lineage to the left reproduces asexually without males, whereas the sexual lineage to the right requires males (squares) for reproduction.

unstable is to examine the evolutionary consequences of individual selection on the mating structure (s). From the previous section we know that the individuals of the i th variant in s will tend to mate with each other because they tend to mate at random within subunits founded by s_i females, while the other variants in s tend to mate within subunits founded by a different number of females. Hence, we expect that genomic selection will operate relatively independently within each variant in s , while individual selection will operate among those variants favouring the variant that have the highest intrinsic growth rate. This implies that we can model the simultaneous action of genomic and individual selection by assuming that the sex ratio within a variant in s is approximately equilibrated at the Fisherian optimum, and by assuming that individual selection will favour the Fisherian optimum with the highest intrinsic growth rate.

In order to describe this type of individual selection that operates on the Fisherian optima let us recall that individual selection is defined by the rate of increase in the number of individuals of the different variants. In the current case, where selection operates on s , we have that individual selection is defined by the fitness expression of eqn 19.9 at the limit $\phi_i \rightarrow \phi \rightarrow \phi^{*F}$, where the sex ratio is defined by the Fisherian optimum to eqn 19.11. Thus, as $\theta^{*F} = 1 - \phi^{*F}$, the fitness expression of eqn 19.9 reduces to the following one-dimensional fitness profile

$$\lim_{\theta_i \rightarrow \theta \rightarrow \theta^{*F}} \lambda_i = \lambda^{*F} = \dot{n}\theta^{*F} \quad (19.15)$$

where $\dot{n} = nX2$, and $\theta^{*F} = (s + 1)/2s$ by eqn 19.11. Note, that eqn 19.15 contains the two-fold cost of the male in the sense that $\lambda_{\theta^{*F}=1}^{*F} / \lambda_{\theta^{*F}=1/2}^{*F} = 2$, where $\lambda_{\theta^{*F}=1}^{*F}$ is the growth rate of a variant with pure females and $\lambda_{\theta^{*F}=1/2}^{*F}$

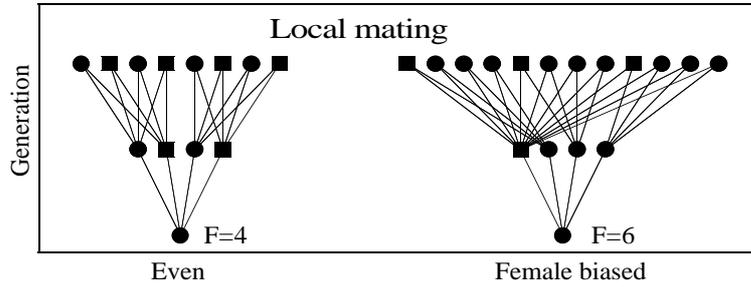


Fig. 19.3 An illustration of the fitness of two variants with respectively an even and a female biased sex ratio, when mating is strictly local ($s = 1$). The fitness (F) of a particular gene in each grandmother is given as the average number of copies of that gene in the grand-offspring. Note, that the fitnesses of the two variants relative to one another are the same at the level of both genes and individuals, and that this is because there is no exchange of genes between the two variants (compare with Fig. 19.1 where this is not the case). Note also, that it is the variant with the female biased sex ratio that is most fit because it has the highest intrinsic growth rate.

is the growth rate of a variant with an even sex ratio. Now, if we substitute $\theta^{*F} = (s + 1)/2s$ into the fitness profile of eqn 19.15 we obtain

$$\lambda^{*F} = \dot{n}(s + 1)/2s \tag{19.16}$$

Hence, the selection gradient on the mating structure is negative

$$\partial\lambda^{*F}/\partial s = -\dot{n}/2s^2 \tag{19.17}$$

and this implies that s evolves to the lower limit $s = 1$ where only females exist because the sex ratio given by eqn 19.11 is zero. This result holds also for the more complex case with inbreeding in haplodiploids. In other words, independently of the particular mode of sexual reproduction, the unbeatable strategy is parthenogenesis, or some other form of asexual reproduction. The evolutionary benefit to a female biased sex ratio is illustrated in Fig. 19.3 for the situation where $s = 1$.

Before we leave this section let us notice that the two-fold cost of the male often does not apply to hermaphrodites. This is because the energy a hermaphrodite allocates to the male function (spermatozoons or pollen) often is negligible compared with the energy it allocates to the offspring (e.g., the seed or fruit). Thus, these hermaphrodites are expected to produce close to the same number of offspring as females, and this implies that a pair of hermaphrodites have the potential to produce approximately twice as many offspring as a pair containing one female and one male.

This example with the two-fold cost of the male illustrates that it is crucial to take into account the simultaneous action of both individual and genomic selection when we deal with the evolution of sex ratios. However, individual selection is not the only reason why the Fisherian sex ratio theory is insufficient on an evolutionary scale: As shown in the following subsection, the Fisherian equilibrium is evolutionarily unstable also at the level of genomic selection.

19.5.2 Two-fold cost of meiosis

To see that the Fisherian sex ratio theory is evolutionarily unstable at the level of genomic selection we can examine the cost of sexual reproduction at the level of genes. At this level sexual reproduction is two-fold costly because of the two-fold cost of meiosis. This cost is the fact that the meiotic separation of the diploid set of genes into haploid gametes causes a reduction from one to one half in the probability by which a particular gene in the mother is copied to a particular offspring of that mother. In other words, if the female reproduces asexually, each gene in the female would be copied to all her offspring, and not only to half of them as is the case with sexual reproduction. These arguments hold only in sexual species with separate sexes, and this is because in hermaphrodites the male function will distribute genes to approximately the number of offspring produced by the female function and, thus, the rate at which a gene will spread in a hermaphrodite is comparable to the rate of spread in an asexual species. In other words, a hermaphrodite has the potential to bypass both the two-fold cost of the male and the two-fold cost of meiosis.

If we assume that the sexes are separate, then, in the framework of the classical sex ratio theory the two-fold cost of meiosis implies selection for asexual reproduction so that the classical equilibria are evolutionarily unstable. To illustrate this let $0 \leq f \leq 1$ be the fraction of the genome that an offspring receives from the father, and let $1 - f$ be the fraction that the offspring receives from the mother. Then $f = 0$ corresponds to the case of asexual reproduction where the offspring receives all its genes from its mother, $f = 1/2$ corresponds to the usual form of sexual reproduction where the offspring receives half of its genes from the mother and the other half from the father, and $f = 1$ corresponds to the unusual form of sexual reproduction where the offspring receives all its genes from the father. Note also that the common solutions to f in natural populations are the haploid genome for $f = 0$, and the diploid genome with meiosis for $f = 1/2$.

Given these definitions we can analyse the evolutionary determinants of the sex ratio, taking into account the two-fold cost of meiosis. To do this let us consider the standard situation where the Fisherian sex ratio is given by the fitness profile of eqn 19.1. According to this equation the

essential terms determining the Fisherian sex ratio are the transcription probabilities of the daughter or son relative to the mother, and the relative value of a son and a daughter in copying genes into the future. Under the current framework with variation in the fraction f it is easily seen that the transcription probability of the daughter relative to the mother is $p_{d,m} = 1 - f$, and thus also that $p_{s,m} = f$. Likewise, the relative value of a daughter compared with a son in projecting genes into the future is $v_d = 1 - f$, while the relative value of the son is $v_s = f$. From these estimates we have that $X = (1 - f)^2$, and that $Y = f^2$, and thus, the equilibrium relation between f and the Fisherian sex ratio $(\phi/\theta)^{*F} = Y/X$ is

$$\left(\frac{\phi}{\theta}\right)^{*F} = \frac{f^2}{(1-f)^2} \quad (19.18)$$

Hence, at the limit of asexual reproduction ($f = 0$) the Fisherian sex ratio is zero, while it is even in the presence of the usual form of sexual reproduction, where $f = 1/2$ and the offspring receives half of its genes from each parent, and infinity at the unusual form of sexual reproduction, where $f = 1$ and the offspring receives all its genes from the father.

Now, by the prediction of eqn 19.18 we have that the fraction f is neutrally stable in the sense that it is not affected by Fisherian selection but instead given as an initial condition. This may imply that f is given by another form of selection, and here let us examine if the two-fold cost of meiosis sets any evolutionary constraints on the value of f . One type of selection that is defined by the cost of meiosis is the type defined from variation in the rate or probability, by which a particular gene is copied into the future, given that the growth rate at the individual level is constant. In the fitness profile of eqn 19.1 this rate is given by the two terms X and Y and, thus, genomic selection on f can be defined by eqn 19.1 at the limit $\phi_i \rightarrow \phi \rightarrow \phi^{*F}$, where the sex ratio is defined by the Fisherian optimum of eqn 19.18. Thus, as $\theta^{*F} = 1 - \phi^{*F}$, the fitness profile of eqn 19.1 reduces to the one-dimensional profile

$$\lim_{\theta_i \rightarrow \theta \rightarrow \theta^{*F}} \lambda_i = \lambda^{*F} = n\theta^{*F}[X + Y] \quad (19.19)$$

where $\theta^{*F} = (1 - f)^2 / [(1 - f)^2 + f^2]$ by eqn 19.18. Notice here that eqn 19.19 contains both the two-fold cost of the male and the two-fold cost of meiosis. From the previous subsection we have that the two-fold cost of the male is given as $\lambda_{\theta^{*F}=1}^{*F} / \lambda_{\theta^{*F}=1/2}^{*F} = 2$, and eqn 19.19 shows that the two-fold cost of meiosis is given as $\lambda_{f=0}^{*F} / \lambda_{f=1/2}^{*F} = 2$, where $\lambda_{f=0}^{*F}$ is the growth rate of the asexual variant with $f = 0$, and $\lambda_{f=1/2}^{*F}$ is the growth rate of the sexual variant with $f = 1/2$. In the same way we find that the overall cost of

sexual reproduction between a male and a female is four-fold, and this is because $\lambda_{\theta^{*F}=1, f=0}^{*F} / \lambda_{\theta^{*F}=1/2, f=1/2}^{*F} = 4$.

Now, to determine the Fisherian equilibrium to f let us substitute both $\theta^{*F} = (1-f)^2 / [(1-f)^2 + f^2]$, $X = (1-f)^2$, and $Y = f^2$ in the fitness profile of eqn 19.19. Then, we have the following fitness profile

$$\lambda^{*F} = n(1-f)^2 \quad (19.20)$$

Hence, the selection gradient on the males fraction of the offspring's genome is negative

$$\partial \lambda^{*F} / \partial f = -2n(1-f) \quad (19.21)$$

which implies that f evolves to the lower limit $f = 0$, where only asexually reproducing females exist.

These results show that the Fisherian sex ratio theory is incomplete in the sense that in itself it does not explain the evolution of sex ratios even though it applies on a local scale, where it often predicts the correct relationship between the sex ratio and the fundamental traits that include the ploidy level of the genome, the relative mass of males to females, and the mating structure. Hence, to explain the evolution of sex ratios and their causal relationship to the traits that traditionally have been treated as fundamental, we need to use an other type of selection. This is what I will do in the following two chapters where I show that a theory based on selection by density dependent competitive interactions can explain both the evolution of sex ratios and the evolution of the traditional assumptions, including the mating structure and the ploidy level of the genome.

Chapter 20

Males and sex ratios

MALES ARE individuals producing microgametes, or spermatozoons, that fertilise the female's macrogamete, or egg. As these male individuals are two-fold costly when there is one male per female, and as they are also the crucial component inducing the two-fold cost of meiosis in sexually reproducing species, their evolution has remained a paradox in evolutionary biology. More explicitly, why do sexual reproduction in mobile organisms generally occur between males and females when the four-fold cost of sex can be avoided in hermaphrodites?

So far there have been some attempts to explain the evolution of males, e.g., through disruptive selection on gamete size (Parker et al., 1972; Maynard Smith, 1978) or through uni-parental cytoplasmic inheritance (Cosmides and Tooby, 1981; Law and Hutson, 1992). As these models assume that the fully evolved male contributes to reproduction only by his transmission of microgametes to the female, their predictions are evolutionarily unstable in the sense that they are easily invaded and out-reproduced by an asexual, or hermaphroditic, variant. In this chapter, I will instead focus on an explanation that is evolutionarily stable in the sense that the fitness of the male individual will out-balance his two-fold cost. Then, in the following chapter I will focus on a mechanism that can out-balance the two-fold cost of meiosis and explain the evolution of the optimal mode of sexual reproduction between males and females.

To explain the evolution of males I will turn to selection by density dependent competitive interactions. In this framework the fitness of the male will depend not only on his ability to transmit gametes to females, but also on his ability to defend the resource utilised by his female. Due to this extra component to male fitness I will tread the evolution of the male as the evolution of an individual that invests energy and time in interference competition instead of using them on reproduction, as it is done by the

female. In this sense the male individual gains fitness not only through fertilisation, but also from interference competition where he, among other things, can prevent that the asexual variant has access to the resource. Then, due to the bias in the access to resource between the sexual and the asexual variant, the sexual variant can replicate at a faster rate than the asexual variant.

Apart from the evolution of males I will in this chapter develop also a new theory that predicts evolutionarily stable sex ratios. To do this I note that the fitness that the male gains from interference competition depends on the average number of competitive encounters that a pair experiences. If pairs do not encounter, it is impossible to gain fitness through interference competition between pairs and, consequently, males cannot evolve by these means. If instead pairs encounter one another at a high rate it might pay to let the male specialise in interference competition at the cost of self-replication at the physiological level. Among other things, I will show that the fraction of the individuals that in an optimally sized group will specialise in interference competition and evolve non-replication is positively related to the evolutionarily determined level of inter-group interference. More specifically, I find that when the body mass is negligible, as is the case in prokaryotes and viruses, the level of interference is so low that there is selection for the absence of males. If instead the body mass is large, as is the case in many eukaryotes, the evolutionarily determined level of interference is exactly so high that there is selection for a single or a few male/s per female.

Having developed the new sex ratio theory in Section 20.3, I will in Section 20.4 superimpose the framework of the classical sex ratio theory on top of the new theory and show that, although the classical sex ratio theory apparently fails to explain the evolution of sex ratios, it can be used to explain the evolution of local mating. In this new form of the old theory the equilibrium relation between the mating structure and the sex ratio is the same as in the old theory. The differences between the two theories are instead (i) that the predictions of the old theory are evolutionarily unstable while the new predictions are evolutionarily stable, and (ii) that the causality between the fundamental and the derived trait is turned upside down. This implies that it is the mating structure that evolves from the sex ratio in the new theory, and not the sex ratio that evolves from the mating structure, as it traditionally has been thought.

In the two last sections in this chapter I will take a closer look at the male himself. Here, I will first deal with the evolution of the body mass of the male relative to the body mass of the female, and then I will focus on the evolution of male characters that, among other things, include ritualised fighting tactics, physiological ornaments, and song. But before I will turn to

the evolution of the male and his associated characters, I will first describe the cost of the male in more detail than it was done in Section 19.5.1.

20.1 Cost of males

The male is energetically costly when his contribution to reproduction is restricted to the fertilization of the females' eggs, and from Fig. 19.2 we know that this cost is 50% when there is one male per female. In this section I will quantify this cost in the general case where an optimally sized group of n individuals contains u females and $n - u$ other individuals that are defined on a continuous scale including females and males as the two extremes. To define this continuum from females to males, let us deal with the analysis on sexual reproduction in the following chapter and assume that sexual reproduction is absent and that the individuals that form a group belong to the same clone.

In this framework the differences between males and females are defined in terms of energetics, where the female component of an individual can be characterised as the amount of energy and time that the individual allocates to self-replication, while the male component can be characterised as the diametrically opposite component, i.e., as the amount of energy and time taken from self-replication and used for other purposes, e.g., on inter-group interference competition. On this scale an individual is a male when it does not replicate, because it does not allocate any energy and time to self-replication.

To describe this continuum from females to males in mathematical terms let $\dot{\epsilon}$ be the energy that an individual can assimilate and allocate to reproduction, and let ϵ_i be the amount of this energy that a potential male uses up in inter-group interference competition instead. Then, a potential male is a male when $\epsilon_i = \dot{\epsilon}$, while it is a female when $\epsilon_i = 0$. Hence, for a group with n individuals and $n - u$ potential males, the average cost of the male component per individual in that group is $\epsilon_i(n - u)/n$, and that is because this is the amount of energy taken from reproduction on average. Thus, on average, the amount of energy that an individual has available for reproduction is

$$\epsilon = \dot{\epsilon} - \epsilon_i(n - u)/n \quad (20.1)$$

From this equation we have that at the limit $\epsilon_i = 0$ all n individuals are females having $\dot{\epsilon}$ amounts of energy available for reproduction, whereas at the other limit $\epsilon_i = \dot{\epsilon}$ there are $n - u$ males that do not replicate because they have used all their free energy in inter-group interference competition. At this latter limit eqn 20.1 is equivalent to $\epsilon = \dot{\epsilon}[1 - (n - u)/n]$, where $(n - u)/n$ is the average and proportional cost of the male component of

non-replication. For the case with one male per female we find that $n-u = 1$ and that $n = 2$ and, thus, that the cost of the male component is 50%, or two-fold. When instead there are no males the cost of non-replication is zero, while it is 100% when there are no females.

20.2 Evolution of males

Let us now examine how selection by the density dependent competitive interactions that exists among groups can offset the cost of non-replication so that male individuals can evolve by natural selection. From Chapter 18 we know that the optimal group size under inter-group interference is $n^{**} = v(\psi\iota^{*e} + 1)$, where v is the cost of group formation and ι^{*e} is the evolutionarily determined level of inter-group interference. Then, to explain the evolution of the male we need to show how the male component of the potential males, i.e., how ϵ_{ι} , can evolve from zero to $\dot{\epsilon}$.

To describe the evolution of males by this form of selection we can focus on the bias in the access to resource as it is caused by the intensity by which the $n - u$ males participate in inter-group interference competition, i.e., as it is caused by the differentiation in the parameter ϵ_{ι} . This means that we can model the discrete growth rate of the i th variant as $\lambda_i^* = p\epsilon e^{\psi\iota^{*e}(\ln \epsilon_{\iota,i} - \ln \epsilon_{\iota})} / wB$, where ϵ is given by eqn 20.1. By inserting eqn 20.1 into this expression, and by dividing the obtained growth rate by the growth rate of the average variant, we find that the growth rate of the i th variant is

$$\lambda_i^* = \frac{\dot{\epsilon}n - \epsilon_{\iota,i}(n-u)}{\dot{\epsilon}n - \epsilon_{\iota}(n-u)} e^{\psi\iota^{*e}(\ln \epsilon_{\iota,i} - \ln \epsilon_{\iota})} \quad (20.2)$$

The selection gradient at the limit of the average variant is then

$$\lim_{\epsilon_{\iota,i} \rightarrow \epsilon_{\iota}} \frac{\partial x_i^*}{\partial \epsilon_{\iota,i}} = \frac{\psi\iota^{*e}}{\epsilon_{\iota}} - \frac{n-u}{\dot{\epsilon}n - \epsilon_{\iota}(n-u)} \quad (20.3)$$

From this selection gradient we find that selection by inter-group interference is strong enough to cause the evolution of non-replication in the $n - u$ potential males when $\lim_{\epsilon_{\iota,i} \rightarrow \epsilon} \partial x_i^* / \partial \epsilon_{\iota,i} \geq 0$ for $\epsilon_{\iota} = \dot{\epsilon}$. By exchanging ϵ_{ι} in eqn 20.3 with $\dot{\epsilon}$, setting $\lim_{\epsilon_{\iota,i} \rightarrow \epsilon} \partial x_i^* / \partial \epsilon_{\iota,i} \geq 0$, and solving for n/u , we find that males evolve when

$$n/u \leq \psi\iota^{*e} + 1 \quad (20.4)$$

To analyse when the conditions of this inequality is fulfilled we notice that when males exist, then the left-hand side of eqn 20.4 is always larger than one, and it is two for the special case with one male per female. Then, to determine the right-hand side we recall from Chapter 14 that $\psi\iota^{*e} = 0$ when

the body mass is negligible and situated at a lower evolutionary limit. In other words, the inequality cannot be fulfilled and, thus, we can conclude that a negligible body mass and the absence of males are co-occurring traits, as it is the case in both prokaryotes and viruses. Also from Chapter 14 we recall that $\psi\iota^{*e} \geq 1$ when the body mass is large and situated at the evolutionary equilibrium, or at the evolutionary steady state. In this case the inequality for a single male per female is always fulfilled and, thus, we can conclude that a large body mass and the presence of males are co-occurring traits as it is the case in most of the higher eukaryotes that are mobile.

20.3 Evolution of sex ratios

As we have now explained the evolution of the male individual, let us assume that an individual is either a male or a female, and then let us focus on the sex ratio and analyse the selection pressure on the number of males per female.

If we follow the present notation we have that the proportion of males is $\phi = (n - u)/n$, that the proportion of females is $\theta = u/n$, and that $\epsilon_\iota = \dot{\epsilon}$ and $\epsilon_{\iota,i} = \dot{\epsilon}$ because we assume that all males are fully developed on the continuum from females to males. Then, in the dimension with variation in the proportion of males it can be seen that the fitness expression of eqn 20.2 turns into

$$\lambda_i^* = \frac{1 - \phi_i}{1 - \phi} e^{\psi\iota^{*e}(\ln \phi_i - \ln \phi)} \quad (20.5)$$

where the $\ln \phi$ terms in the exponent indicate that it is the group containing most males that will dominate the other groups during the competitive encounters. The selection gradient on the proportion of males is then

$$\lim_{\phi_i \rightarrow \phi} \frac{\partial x_i^*}{\partial \phi_i} = \frac{\psi\iota^{*e}}{\phi} - \frac{1}{1 - \phi} \quad (20.6)$$

and the equilibrium sex ratio

$$(\phi/\theta)^{**} = \psi\iota^{*e} \quad (20.7)$$

This sex ratio is positively related to the evolutionarily determined level of inter-group interference, with the ratio approaching zero at the limit of no interference, and infinity at the limit of infinite interference. Then, as the level of interference is density dependent we expect that the sex ratio is positively related to the population density. As illustrated in Fig. 20.1, this prediction is confirmed by recent data on the sex ratio in new-born spotted hyenas.

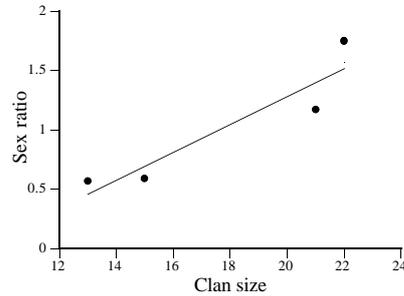


Fig. 20.1 The relationship between the sex ratio in new-born spotted hyena and the population density (clan size). The dots are data and the line their linear regression. Data from Holekamp and Smale (1995).

To determine the theoretical predictions on the absolute value of the sex ratio recall that $\psi l^{*e} = 1$ for large-bodied organisms with a body mass in evolutionary equilibrium, and that $\psi l^{*e} \approx 2$ at the evolutionary steady state. This implies that we predict that a large body mass and the presence of one or two males per female are co-occurring traits, as the case in many of the higher eukaryotes. Although the sex ratio is often male biased in mammals and birds (Charnov, 1982), and although Darwin (1871) saw that “there are certain animals (for instance, fishes and cirripedes) in which two or more males appear to be necessary for the fertilization of the female, and the males accordingly largely preponderate”, the widespread case appears to be approximately one male per female. In this context we notice that under the current model the second interfering individual need not necessarily be a male. Instead, the second individual can be a related helper like an offspring of the previous year. This, for example, is often the case for cooperatively breeding birds, where the average number of interfering individuals (male + helpers) per replicating female is 1.8 for 17 species in Stacey and Koenig (1990). This number is reasonably close to the expected 2.3 at the steady state for organisms foraging in two dimensions. In Chapter 22, on the evolution of eusocial communities, I will examine why the extra individual in cooperative breeders is an offspring, and not an extra male that mate with the female.

Natural populations of large-bodied species may not always be situated at the evolutionary steady state. Instead, under relatively extreme density independent conditions, such as in marginal habitats, the population density is often substantially declined and this will cause a decline in the level of intra-population interference and, thus, also a decline in the sex ratio. Here, we notice that we expect that it is the organisms with relatively small body

masses and low metabolic rates that are most severely affected by variation in density independent factors, and this is because it is the individuals of these organisms that have the smallest energetic buffers that can be used to compensate against environmental changes. Thus, we expect that it is the densities of the small organisms with low metabolic rates that generally will be most severely affected by variation in density independent factors. This coincides with the fact that female biased sex ratios are relatively common in insects and poikilotherm vertebrates, while they are relatively rare among birds and mammals (Charnov, 1982; Wrensch and Ebbert, 1993).

As parthenogenesis represents the most extreme form of a female biased sex ratio, it is not surprising that parthenogenesis is relatively common among insects and poikilotherm vertebrates, and that it is almost unknown in both mammals and birds. Also, as expected, there is an inverse correlation between parthenogenesis and the level of intra-population interference. That is, within species containing both sexually and parthenogenetically reproducing variants, parthenogenesis occurs mainly at the geographical limits of the species' distribution, where the population densities are considerably below the densities in the centers of the distributions (Vandel, 1928; Cuellar, 1977; Lynch, 1984).

Here it might, of course, be argued that species are parthenogenic when the densities are low simply because it is difficult to find a mate at such densities. Note, however, that this simple argument is not simplifying, and this is because it introduces a lot of new complexities that are not needed. First of all, this latter hypothesis is based on the assumption that it is difficult to find a mate when the density is low, but this is far from certain, at least when we deal with mobile organisms. Also, there is no need to include an additional mechanism to explain the evolution of parthenogenesis at low densities, and this is because the difficult task is not to explain the evolution of parthenogenesis, but instead to explain the evolution of the male. Hence, when the model explaining the evolution of the male also predicts that males are present mainly at the centres of the species' distributions, then there is no real need for another mechanism that can explain that parthenogenesis is present in the areas where we already expect it to be present.

In the present model, males can avoid their two-fold cost and evolve by selection because they can help the female during moments of intra-population interference competition. According to this view it is cooperation in relation to inter-pair interference competition that is the essential fundamental trait leading to the evolution of males, whereas the various other forms of cooperation that can exist between a male and a female are considered to be derived traits evolving only secondarily once the transition to pairwise reproduction has occurred. In relation to this hypothesis, and the distinction between interference and exploitative competition, the

alternative hypothesis would be that the essential factor that promotes the evolution of the male is that the male can avoid the two-fold cost by collecting resource that the female can use for reproduction. This latter hypothesis seems to fail because it does not really explain why this sort of helping behaviour is beneficial only in large, but not in negligibly sized, organisms, and why it is more beneficial in a stable than in a harsh environment, and why it is beneficial at the centre of the species' distribution, but not at the edges.

20.4 Evolution of local mating

We now have a balanced sex ratio that evolves by individual selection independently of the constraints associated with the genomic type of selection that underlies the classical, or Fisherian, sex ratio theory. This implies that we have explained the evolution of the sex ratio independently of the classical assumptions that, among other things, include the mating structure, when it is given as the degree of local mating. In this section I will show how we can explain the evolution of the classical assumptions on the degree of local mating by integrating the classical sex ratio theory with the sex ratio theory I developed in the preceding section.

To explain the evolution of the mating structure, given as the degree of local mating, let us maintain the classical assumption that sexual reproduction includes a diploid or a haplodiploid genome. Then, as long as the body mass is in evolutionary equilibrium and there is one male per female, we know from Chapter 19 that genomic selection is in equilibrium if mating occurs at random at the population level. However, if the sex ratio, which is optimal under the action of competitive interactions, is changed from one toward pure females and the ploidy level of the genome remains the same, then the Fisherian form of genomic selection will impose some constraints on the way by which the new optimum can be reached. In this section I show that these constraints can lead to the evolution of local mating. Notice here, that I assume that the genome is more conservative than the mating structure in the sense that selection will alter only the mating structure and not the ploidy level of the genome. It is not until in the next two chapters that I will relax the constraint of the genome in order to illustrate how a diploid and a haplodiploid genome can evolve under the hypothesis of selection by competitive interactions.

To explain the evolution of local mating let us use the model we defined in Section 19.4. Here s is the degree of local mating, with $s = 1$ being the limit with mating among brothers and sisters, and $s = \infty$ being the limit of population wide random mating. In this model we expect that the individuals of the i th variant in s will tend to mate only with each other,

and this is because they will mate at random within subunits founded by s_i females, while the other variants in s will mate within subunits founded by a different number of females. Hence, genomic selection is expected to operate relatively independently within each variant in s and, thus, for the i th variant the sex ratio will evolve toward the Fisherian sex ratio given by eqn 19.11 for diploids, and by eqn 19.14 for haplodiploids.

While genomic selection operates within each variant in s , individual selection will operate among those variants. That is to say that it is the variant with the highest growth rate that will out-grow all other variants and, thus, the optimal proportion of males is given by individual selection. As, for a given s , the proportion of males will be given also by genomic selection we can exchange the proportion of males in the fitness expression of competitive interactions (eqn 20.5) with its functional relation to s , as defined by genomic selection, i.e., as it is defined by eqns 19.11 and 19.14. Here, let us formulate the case in diploids, where $\phi = (s-1)/2s$ by eqn 19.11. Substituting this expression into eqn 20.5, we find that the selection gradient on s is

$$\lim_{s_i \rightarrow s} \frac{\partial \lambda_i}{\partial s_i} = \frac{\psi \iota^{*e} (s+1)}{s(s-1)^2} - \frac{1}{s(s-1)} \quad (20.8)$$

Hence, the number of foundresses at the evolutionary equilibrium is

$$s^{**} = \frac{\psi \iota^{*e} + 1}{1 - \psi \iota^{*e}} \quad (20.9)$$

Thus, when $\psi \iota^{*e} = 1$ and the body mass is in evolutionary equilibrium we find that $s \rightarrow \infty$, and this implies that mating will occur at random at the population level. If, instead, $0 < \psi \iota^{*e} < 1$ we find that some intermediate degree of local mating will evolve.

Let us now show that the equilibrium of eqn 20.9 resembles the Fisherian equilibrium with local mating. To do this we note that the sex ratio of eqn 20.7 is given as a function of $\psi \iota^{*e}$, and when this relation is substituted into eqn 20.9 the degree of local mating is

$$s^{**} = \frac{(\phi/\theta)^{**} + 1}{1 - (\phi/\theta)^{**}} \quad (20.10)$$

If I rearrange eqn 20.10 I find that $(\phi/\theta)^{**} = (s^{**} - 1)/(s^{**} + 1)$, and this is the expression in eqn 19.11 representing the prediction in the classical sex ratio theory. In other words, the relation between the mating structure and the sex ratio is the same in the two theories. The only differences are (i) that the dependent and the independent variables are changed around, and (ii) that the sex ratio predicted by the classical theory is evolutionarily unstable while the local mating predicted by competitive interactions and the new version of the classical theory is evolutionarily stable. In Fig. 20.2 I

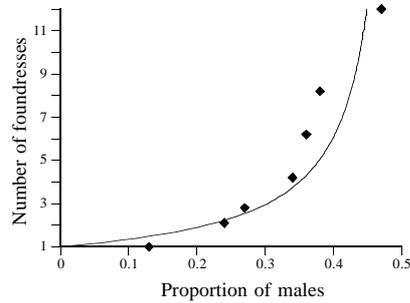


Fig. 20.2 The number of foundresses against the proportion of males in haplodiploids. The curve represents the theoretical prediction, and the diamonds are Werren's (1983) data on the parasitic wasp *Nasonia vitripennis*.

show the predicted relation for haplodiploids together with Werren's (1983) data on a parasitic wasp.

20.5 Evolution of male and female size

So far in this chapter it has been assumed that the amount of resource that the parents invest in a male offspring is equal to the amount that they invest in a female offspring. Under the current notation this implies that the body mass represented by the parameter w is the same in the two sex, although the actual size of the male might be larger than the size of the female if a fraction of the resource ϵ_l , which the male uses on interference competition at the cost of reproduction, is allocated to body mass. In this section I will disregard the effects that the component ϵ_l may have on the body mass of the male, and describe the evolution of male and female size through the differentiation in the amount of resource the parents allocate to a male versus a female offspring. In this description I will consider first the total amount of mass invested in the replicating unit, and thereafter the distribution of this mass between the male and the female component.

In Chapters 10 and 14 I described the evolution of body mass under the assumption that the replicating unit is a single replicating individual, which for the sake of simplicity, we may think of here as a female that can replicate with and without males. More generally, the replicating unit contains a single replicating female and from zero to a few non-replicating males. In this general situation the total amount of mass invested in a replicating unit (w_u) is the mass of the female (w_θ), plus the mass of the male (w_ϕ) times the number of males per female (ϕ/θ), i.e., the mass of the replicating unit is $w_u = w_\theta + (\phi/\theta)w_\phi$. In this way the body mass w , that was used in

Chapters 10 and 14, is a special case of w_u , a case defined at the limit $\phi \rightarrow 0$, where males are absent. The resemblance between the situations with and without males is so general that we can use the models in Chapters 10 and 14 to model the evolution of w_u . This is because, in Chapters 10 and 14, it is the mass w that defines both the competitive quality of the replicating unit and the trade-off between numerical replication and competitive quality, while these two components are defined by the mass w_u in the general situation that allows for the existence of males.

Due to this resemblance between the two situations we can transfer the predictions on body mass from the special situation, where males are absent, to the general situation, which also includes the presence of males and variation in the sex ratio. The only thing we need to do is to exchange the mass of the replicating unit (w_u) with the body mass (w) in the equations in Chapters 10 and 14, and then notice that the exploitation efficiency in those chapters have now become a parameter of the replicating unit, instead of a parameter of the individual. In other words, the predictions on the evolution of body mass, and on the evolution of the level of interference, hold also for the general case with males, as it already has been assumed in the previous sections in this chapter.

We now have a set of predictions on the mass of the replicating unit so that we can turn to the distribution of this mass between the female and the male offspring. To describe this distribution let us follow the approach in Section 19.2 on Fisherian investment sex ratios. In this situation the replicating unit has a given amount of resource (ϵ) that is allocated to reproduction, and the distribution of this resource between the female and male offspring is described by the following constraint $\phi w_\phi + \theta w_\theta = \epsilon/m$, where m is the total number of offspring produced. From this constraint there is a trade-off between the body mass of the male and the proportion of females in the offspring, a trade-off that is described by the relation $\theta = (\epsilon/m - \phi w_\phi)/w_\theta$. This trade-off also represents a trade-off between competitive quality and the intrinsic growth rate, where it is the body mass of the male that is positively correlated with the competitive quality of the replicating unit, and it is the proportion of female offspring that is positively correlated with the potential rate whereby the replicating units can increase in numbers. In other words, in the absence of competitive interactions we can approximate the discrete growth rate as $\lambda = m\theta$, and this implies that it is $\lambda = m(\epsilon/m - \phi w_\phi)/w_\theta$ for the current model. Then, in the presence of density dependent competitive interactions we obtain the following fitness expression for variation in the mass of the male

$$\lambda_i^* = \frac{\epsilon/m - \phi w_{\phi,i}}{\epsilon/m - \phi w_\phi} e^{\psi_i^* e (\ln w_{\phi,i} - \ln w_\phi)} \quad (20.11)$$

Hence, remembering that $\theta = (\epsilon/m - \phi w_\phi)/w_\theta$, we find that the selection

gradient on male mass is

$$\lim_{w_{\phi,i} \rightarrow w_{\phi}} \frac{\partial x_i^*}{\partial w_{\phi,i}} = \frac{\psi \iota^{*e}}{w_{\phi}} - \frac{\phi}{\theta w_{\theta}} \quad (20.12)$$

so that the evolutionary equilibrium for the ratio of male to female mass is

$$(w_{\phi}/w_{\theta})^{**} = \psi \iota^{*e} (\theta/\phi)^{**} \quad (20.13)$$

where $(\theta/\phi)^{**}$ is the inverse to the numerical sex ratio at its evolutionary equilibrium. As the numerical sex ratio at this equilibrium is $(\phi/\theta)^{**} = \psi \iota^{*e}$ by eqn 20.7, the ratio of male to female mass reduces to unity at equilibrium, i.e., we have that

$$(w_{\phi}/w_{\theta})^{**} = 1 \quad (20.14)$$

In other words, the mass of the male is equal to the mass of the female, independently of whether the body mass is in evolutionary equilibrium or in evolutionary steady state.

The result that the body masses of the male and the female are the same relates only to the parents' investment in the offspring. Moreover, we notice that the result of an even body mass ratio is an equilibrium prediction, and that the conditions behind this equilibrium are likely to be violated when the optimal sex ratio is either female or male biased. For example, when it is obligate that sexual reproduction occurs between a female and a male, and the optimal numerical sex ratio is female biased, then there may be too few males to copulate with all the females and this will impose selection for a sex ratio that is less female biased than indicated by eqn 20.7. On the other hand, when the optimal sex ratio is male biased it is likely that the stronger males will exclude the smaller males from copulation and this will induce selection for a sex ratio that is less male biased than indicated by eqn 20.7. In other words, the real sex ratio is expected to be conservative toward a more even sex ratio than indicated by eqn 20.7. Then, from eqns 20.13 and 20.7, it is apparent that this conservatism will induce selection on the body mass ratio, and this will imply that males are larger than females in systems with a male biased sex ratio, and that males are smaller than females in systems with a female biased sex ratio. This differentiation seems to hold in many natural systems: For example, males are often larger than females in birds and mammals (reviewed by Andersson, 1994), and birds and mammals tend to have male biased sex ratios (e.g., Charnov, 1982). At the other end of the spectrum, males are often smaller than females in insects (e.g., Andersson, 1994), and insects tend to have female biased sex ratios (e.g., Charnov, 1982). Moreover, males may be extremely small, especially in insects with highly female biased sex ratios (e.g., Hamilton, 1967).

Finally, it should be noticed that body mass is only one component of competitive quality, which also includes the metabolic rate and the complexity of behavioural interactions. In other words, males are expected to have a higher metabolic rate and more complex interactions than females, when the sex ratio is male biased, while the reverse may be true when the sex ratio is female biased.

20.6 Male characters and sexual versus non-sexual selection

So far in this chapter we have not dealt explicitly with the mode by which the male allocates the resource ϵ_i into competitive interactions at the cost of reproduction. However, when males evolve by competitive interactions there is selection also on the mode by which they use the extra energy and time in interference competition. Due to the benefit of winning competitive encounters it might, at first, be expected that selection would allocate the free energy and time to the development of effective weapons and direct combats without rules. However, Maynard Smith and Price (1973) showed that, because effective weapons and direct combats are associated with costs of injury, selection will often favour inefficient weaponry and ritualised tactics. Apart from ritualised fighting tactics the rules and traits that are used for communication between the competing males can include song, which can signal territory boundaries, and traits like physiological ornaments or colourful plumages, which can signal energetic superiority. In natural species these forms of traits are often breeding traits that appear only during the breeding season, and together they are known as male characters because they generally are the traits by which males can be distinguished from females. However, not all breeding traits are male characters. In some species it is both the male and the female that develop these conspicuous traits during the breeding season.

The evolution of male characters have traditionally been explained by sexual selection, which was first introduced by Darwin (1859, 1871) in order to explain the evolution of extravagant male characters that appeared to decrease the survival of the male (reviewed by, e.g., Andersson, 1994; Møller, 1994; Bulmer, 1994; Andersson and Iwasa, 1996). This hypothesis is based on female choice where male characters evolve because the females are selecting the more expressive males, either by comparing males independently of one another, or by choosing the winner from contests among males. When, in such instances, the females have a common preference for special characters, then these characters may evolve even to the extent where the survival of the male is depressed (Fisher, 1930; O'Donald, 1962,

1980; Lande, 1981; Kirkpatrick, 1982).

The critical point in the hypothesis of sexual selection is to explain why females have the preferences they have, and especially, why they would select for traits that may even cause a decline in the survival of the male. This latter point is extremely critical because a trait that declines the fitness of the male generates selection against a female choice for that trait. Hence, there is no direct way in which the preference for a truly detrimental trait can invade and evolve to fixation. In short, this implies that a widespread female preference that might explain the maintenance of a detrimental male trait must be given by initial conditions. These initial conditions can, e.g., at least hypothetically, arise through the hypothesis of the Fisherian runaway process, where the preference for a trait evolves before that trait becomes detrimental. But even then, there is a good chance that there will be selection against that preference once the detrimental male character has evolved, and in these instances sexual selection thus cannot explain even the maintenance of detrimental male characters.

The more simple hypothesis is that, disregarding the males mating opportunities, the male characters evolve because they increase the overall fitness of the male (e.g., Williams, 1975; Heywood, 1989; Hoelzer, 1989; Graften, 1990), even though they may also decrease his survival probability. This may, e.g., be the case if the male characters are used for communication among males, when they compete for other resources than females. In this latter case the male characters will evolve independently of sexual selection, and superimposed on the evolution of the male characters there will be selection for a female choice for the most expressive males, simply because these males are most fit. Note here that this alternative hypothesis actually is diametrically opposite to the original hypothesis proposed by Darwin. According to Darwin it is the female choice that is the fundamental trait inducing the evolution of the male characters, while according to the alternative hypothesis it is the male characters that are most fundamental inducing the evolution of the female choice for those characters. Then, when this female choice has evolved it may, of course, induce an additional level of selection that might modify the male characters as long as this modification coincides with the rules on the competitive interactions between males.

In order to test the Darwinian hypothesis that male characters follow from female choice and sexual selection, against the opposing hypothesis, that female choice and sexual selection follow from male characters and interference competition for resource, let us first examine in more detail the differences between the two modes of selection. A superficial comparison might first suggest that sexual selection through female choice and male contests for females is approximately similar to the type of selection by competitive interactions that I used in this chapter to explain the evolution

of the male individual, including the evolution of male characters. This is, however, not the case. In sexual selection the contests are male contests for females, and the male characters evolve because the successful male can mate with the female. Instead, in the non-sexual form of interference competition the contests are for essential resources, like food or nesting sites, and the essential units that compete against one another are the reproducing units, usually pairs, and this holds even though in many instances it may only be the male individuals that directly compete. In this latter system the male characters evolve because the successful pair has access to the best and/or the most plentiful resource, so that their reproductive success can be enhanced relative to the unsuccessful pair. In the rest of this section I will refer to these two types of competition as respectively male contests for females and pair contests for resource.

In pair contests for resource the evolution of male characters depends on an asymmetrical system where it is beneficial to let the male individual play the most important role in interference competition. This assumption is likely to hold when the pair breeds in a territory in which the essential resource is contained. In this case both the male and the female will be in the territory, so that the male can specialise in interference competition while the female can specialise in the skills of reproduction. This partitioning of labour is not always possible, especially not if the breeding site and the essential resource are widely separated, as often is the case for colonial breeders. In this latter case, competition for resource and competition for breeding sites will occur in two different places, and this imposes the constraint that it is the individual that is present at a particular site that will have to compete by interference at that site. Hence, if the pair will defend the breeding site they need to have one individual at that site, while the other individual may leave the site to forage. This implies that if both males and females forage for approximately the same amount of time, then the system will be symmetrical in the sense that both the female and the male are constrained to allocate approximately the same amount of energy into interference competition. In other words, true male characters are not expected to evolve, but energy requiring ornaments may evolve in both sexes if they are used to signal competitive quality in the defence of the breeding site and/or in the defence of resource items at the foraging site.

Unlike pair contest for resource, male contests for females and female choice do not depend upon an overlap between the breeding site and the essential resource. This implies that the relative importance of sexual selection versus pair contests for resource in determining the evolution of ornaments can be determined by comparing the covariance between male characters and the degree of overlap between the breeding sites and the essential resource. If male characters evolve primarily by sexual selection we expect

that male characters will be present at approximately the same frequency in the systems with overlap and no overlap between the breeding site and the foraging site, while male characters will exist much more frequently in the system with overlap, if they evolve by pair contests for resource. However, in this latter case ornaments may be present in both sexes in the systems with no overlap between the breeding and the foraging site.

Ornaments and male characters are probably best developed in birds, and it is also here that we find some of the best examples with overlap and no overlap between the breeding and the foraging site. On a very big scale breeding sites are separated from foraging sites in seabirds, because they generally forage over the ocean and breeds on the shore, often in colonies on either islands or cliffs. This is in contrast to many of the other bird taxa that breed in habitats where the breeding site is contained within the foraging site. If we compare seabirds with other birds we find that male ornaments are relatively common among terrestrial birds, while they are almost completely absent among seabirds. More accurately, among the 320 species of seabirds listed in Harrison (1985) it is only the five species of frigatebirds that have conspicuous differences in the plumage between males and females, while ornaments are found in both sexes in other species of seabirds, e.g., in penguins, puffins, and tropicbirds. If we allow also for minor differences in size and structure we find that male-female differences are present in 15% of all seabird species.

If we take a closer look at the birds that are not seabirds we have that the breeding and foraging sites often are non-overlapping in colony breeders, while the two sites tend to overlap in the non-colonial breeders. One bird taxa that contains both colonial and non-colonial breeders are herons where differences between males and females are present only in 12% of 33 colonial species while they are present in 71% of the 17 species that breed solitarily (data from del Hoyo et al., 1992). Among the birds that have highly conspicuous plumages we have kingfishers and bee-eaters, where kingfishers are strictly solitary breeders while bee-eaters often breed in colonies. For the 87 species of kingfishers in the world there are male-female differences in 60% of the cases, while male-female differences are found only in 33% of the 24 species of bee-eaters in the world (data from Fry et al., 1992). Moreover, if we examine the pattern within bee-eaters we find that all 8 species with male-female differences are contained within a set of 15 species that breed solitarily or at least often do so, while not a single case with male-female differences is found within the set of 8 species that breed only in colonies. Also, it is peculiar that the barn swallow, which has been the subject for some of the most extensive studies on sexual selection (Møller, 1994), is the only non-colonial species and the only species with sex dimorphism among the three species of swallows breeding in northern Europe.

Finally, Andersson (1994) lists data on 24 species, ranging from insects to mammals, where it has been examined whether the male character of female preference coincides with the male character that is used in interference competition between males. In 18 of these species the characters were the same, and in 2 of the remaining 6 species the character of female choice was the male territory in itself rather than the character that the male used to defend the territory.

These results suggest that male characters evolve primarily by pair contests for resource, and that female choice and sexual selection are secondary conditions, or derived traits, that evolve due to the presence of male characters that indicate the competitive quality of the male. The conclusion that the evolutionary causality between male characters and female choice is changed around relative to the original proposal by Darwin is not the only radical change that seems to apply to the traditional notion of sexual selection. Another change, which seems to be even more radical, is concerned with the causal relation between sexual selection and sexual reproduction. According to the original proposal by Darwin, and apparently all subsequent treatments of sexual selection, sexual reproduction is the fundamental trait inducing sexual selection for sex dimorphisms, including male characters. However, in the following chapter we will see that it is probably more likely that it is the functional differences between the two sexes that induce sexual selection on the mode of sexual reproduction.

Note. The idea for this section on sexual versus non-sexual selection for male ornaments was given to me by Jørn Madsen and Kim Moritsen in the fall of 1996 during a seabird excursion to Blåvand at the west coast of Denmark.

Chapter 21

Sexual reproduction and ploidy level

SEXUAL REPRODUCTION occurs when the offspring receives genes from more than one parent. In most sexually reproducing species the offspring receives half of its genes from the mother and the other half from its father. This type of reproduction is, though, only a special case. More generally, we can imagine situations where the offspring receives most of its genes from the mother and only a tiny fraction from its father, or situations where the offspring receives genes from a single mother and up to several fathers. But why is sexual reproduction in natural species of the kind where the offspring has only two parents that each contribute the same number of genes to the offspring? Also, why is sexual reproduction absent in negligibly sized organisms, like viruses and prokaryotes, and why does it exist in large-bodied organisms, like the higher eukaryotes? And finally, why in this latter case does sexual reproduction generally occur among hermaphrodites when the organism is sessile, while it generally occurs between a male and a female when the organism is mobile? These are some of the questions that I attempt to answer in this chapter.

From Chapter 19 on Fisherian sex ratios we know that the difficulty with sexual reproduction is that the two-fold cost of meiosis selects for a haploid genome and asexual reproduction, when sexual reproduction occurs between males and females. Due to this paradox the evolution of sexual reproduction in mobile organisms has remained largely obscure although there has been at least twenty different attempts to explain it (Kondrashov, 1994). Among the proposed theories there are the various versions of the Fisher-Muller hypothesis (Fisher, 1930; Muller, 1932; Manning and Thompson, 1984; Peck, 1994), which suggests that sex and recombination speed up evolution, Kon-

drashov's (1982) hypothesis that sexual reproduction evolved to protect against a special type of deleterious genes acting together so that each gene becomes increasingly deleterious as the number of deleterious genes increases, the sib competition models of Williams (1975) and Young (1981) suggesting that sexual reproduction evolved because competition is more severe between asexual sibs, which are genetically identical, than between sexual sibs, which are genetically diverse, and the host-parasite coevolution model of Hamilton et al. (1990) suggesting that sexual reproduction evolved because it stores genes that currently are bad, but protect against future mutant parasites (reviewed by Bulmer, 1994; Ebert and Hamilton, 1996; Hurst and Peck, 1996).

Common to nearly all these models is that they are based on the idea that genetic diversity is beneficial to the organism per se and, thus, diploid organisms with sexual reproduction are expected to be more fit than haploid organisms with no sex, simply because the diploid organisms are genetically more diverse. These hypotheses will, however, not explain the difficult case with sexual reproduction between males and females, and this is because, even in the presence of genetic variation, the male is still four-fold costly compared with the hermaphrodite. Moreover, the traditional hypotheses will generally not explain why the natural solution to the problem of genetic diversity should be exactly a diploid genome with haploid gametes. Actually, it seems that only a small degree of diversity will do just fine (Hurst and Peck, 1996), and this suggests that the small degree of gene exchange occurring among the individuals of haploid prokaryotes may in most cases be sufficient to account for most of the genetic diversity hypotheses. If this is true, the paradox of the diploid genome and the two-fold cost of meiosis remains the major unsolved question. Also, in this context it remains to be explained why the diploid genome with haploid gametes generally is beneficial only in large-bodied organisms, while the haploid genome seems to do just fine in negligibly sized organisms like prokaryotes and viruses.

In this chapter I will disregard the effects of genetic diversity showing that selection by density dependent competitive interactions explains both why the haploid genome is optimal in negligibly sized organisms and why the diploid genome with the two-fold cost of meiosis is optimal in large-bodied mobile organisms. In order to show this, I will turn the evolutionary causality in the theory of sexual selection upside down. Ever since Darwin introduced the notion of sexual selection it has been seen as a type of selection that arises from the presence of sexual reproduction and which can explain the evolution of male characters, including the evolution of extravagant male ornaments. However, in Section 20.6 we saw that under the hypothesis of density dependent competitive interactions, we expect that male characters will evolve independently of sexual selection and that this

will induce the evolution of female choice for competitively superior males. In this chapter I will combine this female choice with a special type of male choice in order to show that sexual selection can explain the evolution of sexual reproduction, including the evolution of the ploidy level of the genome.

More explicitly, in order to explain the evolution of sexual reproduction, I will let the competitively superior males choose the females that pass the largest fraction of the males genome on to the offspring. I can then determine the fraction of the males genome that the female should accept and pass on to her offspring in order to optimise the replication of her own genome. I will find that this fraction is positively related to the level of intra-population interference and that, when the body mass is negligible, the level of interference is so low that there is selection for the absence of sexual reproduction. If, instead, the body mass is large, and in evolutionary equilibrium, then the level of interference is exactly so high that the females' genome will replicate at the fastest rate when half of the genes in the offspring come from the father and the other half from the mother. A diploid and a haplodiploid genome with a meiotic division into haploid gametes are two optimal solutions to this mode of sexual inheritance.

Also, I will show that the hypothesis of competitive interactions will explain why sexual reproduction in large organisms tends to occur among hermaphrodites when the organism is sessile, and why it tends to occur between a male and a female when the organisms are mobile. These latter questions are also left unanswered by the traditional hypotheses on genetic diversity. But before I deal with the deduction of the particular mode of sexual reproduction, let me first consider why we expect sexual reproduction between males and females at all.

21.1 Sexual reproduction

Let us here consider why, in large-bodied mobile organisms, we expect at least some degree of sexual reproduction so that the evolutionarily stable strategy, under the hypothesis of selection by density dependent competitive interactions, is not a clonal pair containing one non-replicating male and one replicating female.

To show this let us consider two variants that reproduce in pairs. Let reproduction in the first variant be purely clonal in the sense that the female can replicate only asexually and that the male cannot replicate, nor can he transfer genes to a female. Then, let the second variant be facultatively sexual in the sense that the female can reproduce both with and without sex and that the male cannot replicate, but he has the ability to transfer genes to a female willing to accept his genes and transmit some of them to

her offspring.

As long as the females and the males of the two variants remain together in pairs we expect that the females of the two variants will replicate at the same rate. The female and the male in a pair may, however, lose contact to the degree that they are unable to join thereafter. When a pair separates the female will replicate only at a reduced rate, and this is because a lonely female has a low competitive quality when compared to a pair. There is, however, a certain probability that a lonely female will find another lonely male and that they can form a new pair so that the female can replicate at the high rate again. This latter probability is highest in the sexual variant since it is only the sexual females that can attract an unrelated male and receive his help by transferring his genes to her offspring. This means that the sexual female will replicate at the fastest rate, and we thus expect that the sexual variant will evolve to fixation.

21.2 Evolution of sexual inheritance

We now have a situation where there is some degree of sexual reproduction, and then there is selection on the degree to which the heritable codes of the two parents are transmitted to the offspring. The female would prefer if she could continue to replicate asexually transmitting only her own genes, while at the same time she receives help from an unrelated male. However, in order to attract the unrelated male and receive his help the female must increase the male's fitness. She can do this by accepting some fraction of the male's genome and by passing that fraction on to her offspring. The relevant question is then to determine the fraction of the male's genome that the female shall pass on to her offspring in order to optimise the replication of her own genome.

To determine this function, we recall the model in Section 19.5.2 where the father's fraction of the offspring's genome is given as f , with $f = 0$ representing the limit of asexual reproduction and $f = 1/2$ representing the usual form of sexual reproduction where the offspring receives half of its genes from the mother and the other half from the father. To recapitulate the situation with exploitative competition, let $R_0^* = p\epsilon^*/wB$ be net lifetime reproduction of the female so that the rate at which a given gene in the female will spread is

$$\lambda^* = (1 - f)R_0^* \quad (21.1)$$

Hence, the selection gradient on f is negative ($\partial\lambda^*/\partial f = -R_0^*$), and f will evolve to the lower limit $f = 0$ where the genome is haploid and reproduction is purely asexual.

Now, to determine the optimal value of f under the action of density dependent competitive interactions we first notice that there is mutual se-

lection for a female choice for the competitively superior males, and for a male choice for the females that pass the largest fraction of the male's genome on to the offspring. That is to say that it is in the female's interest to advertise her attractiveness, i.e., her fraction f , so that the most dominant males will choose the most attractive females, and it is in the male's interest to advertise his ability and willingness to dominate other males so that the most attractive females will choose the most dominating males. This implies that there will be a positive correlation between the female's fraction f and the competitive quality of the male and we may thus let f define the competitive quality of the female. Hence, the discrete growth rate of the genome of the i th female can be given as

$$\lambda_i^* = (1 - f_i)R_0^*e^{\psi\iota^{*e}(\ln f_i - \ln f)} \quad (21.2)$$

and the selection gradient on f as

$$\lim_{f_i \rightarrow f} \frac{\partial x_i^*}{\partial f_i} = \frac{\psi\iota^{*e}}{f} - \frac{1}{1-f} \quad (21.3)$$

Hence, the equilibrium in the father's fraction of the offspring's genome is

$$f^{**} = \frac{\psi\iota^{*e}}{1 + \psi\iota^{*e}} \quad (21.4)$$

Then, as $\psi\iota^{*e} = 0$ in negligibly sized organisms we have that they will reproduce asexually ($f^{**} = 0$), like it is the case with viruses and prokaryotes. If instead, the body mass is large and in evolutionary equilibrium, then $\psi\iota^{*e} = 1$ so that $f^{**} = 1/2$, by eqn 21.4. This implies that such organisms are predicted to reproduce by sex and that one half of the genes in the offspring comes from the father while the other half comes from the mother. This prediction resembles the type of sexual reproduction that exists in most of the mobile eukaryotes with large body masses. For organisms at the evolutionary steady state, or with an upward constrained body mass, we have that $\psi\iota^{*e} > 2$ so that eqn 21.4 predicts that the father's fraction of the offspring's genome is larger than one half in these organisms. I will return to this latter case in Chapter 22 where I show that the prediction $f^{**} > 1/2$ does not hold. Instead, even in these latter cases the optimum of sexual reproduction is $f^{**} = 1/2$.

21.2.1 Evolution of diploid and haplodiploid genomes

One solution to the $f^{**} = 1/2$ pattern of inheritance is diploids, where the meiotic division divides the diploid genome into haploid gametes so that each parent transmits a single copy of all genes to each offspring. This is the widespread solution found in most sexually reproducing species.

A less common system, found mainly in hymenoptera, is the haplodiploid genome where haploid males transmit genes only to daughters, while diploid females transmit genes to both daughters and sons. Although, in this system, a son does not receive genes from his father, the haplodiploid genome conforms to the $f^{**} = 1/2$ equilibrium. This is because when the sex ratio is one, then a gene in the father has, on average, the same chance of being copied to an offspring as a gene in the mother.

Although the diploid and the haplodiploid systems are the widespread forms of inheritance in natural species there are other theoretical possibilities fulfilling the $f^{**} = 1/2$ criterion. These include a diplohaploid system, where diploid males transmit genes to both sons and daughters and haploid females transmit genes only to sons. Another possibility is serial inheritance, where both the parents and the zygote of the offspring are haploid and where the mother transmits one half of the genes to the zygote while the father transmits the other half.

From Chapter 19 on Fisherian sex ratios we recall that classical sex ratio theory also predicted the co-occurrence of a sex ratio of one and a diploid, or a haplodiploid, genome. In the traditional theory it is assumed that it is the sex ratio that is the derived trait that evolves from the ploidy level of the genome. When instead the assumption on the ploidy level is relaxed, as done in this chapter, we find that the hypothesis of density dependent competitive interactions predicts that the ploidy level evolves from the sex ratio. In other words, the evolutionary causality is changed 180 degrees between the two theories. Another difference between the two theories is that the prediction of the classical theory is evolutionarily unstable, while the prediction based on competitive interactions is evolutionarily stable.

21.3 Sex in sessile organisms

So far I have dealt only with sexual reproduction in mobile organisms, where an ultimate constraint behind the evolution of the male is that he can help the female. Not only can the male help the female but the female needs help because there is a trade-off between exploitative and interference competition. This trade-off exists because, at the same time, a single mobile individual cannot devote itself completely to both resource consumption and competitive interactions. If this trade-off did not exist the female needed not have the male to interfere for her, because then the female could continue to consume resource while competing through interference also.

This latter type of competition is actually occurring in sessile organisms where the individuals compete by interference through the exploitation of resource: For example, plants interfere by shading, i.e., by absorbing the solar flux upstream to their neighbours, and the same is true for the aquatic

filter feeders that interfere by assimilating food particles upstream to their neighbours. Also, in sessile organisms a male individual cannot really help a female, because if he casts shadows upon the competitors to the female, then he casts shadows upon the female also. In other words, if the male competes for the female, then, due to the sessility he competes also against her. This means that the task of the sessile male necessarily is reduced to the transmission of gametes to the female, and there is thus selection against the male individual so that we predict either asexual reproduction, or hermaphroditic individuals that do not experience the four-fold cost to sexual reproduction between males and females.

As predicted, males are generally absent in sessile organisms where the two sexes tend to co-occur on hermaphroditic individuals, and this is especially true for plants. Some sessile animals that are not hermaphroditic, have solved the problem in a different way. They produce dwarf males that are far from two-fold costly and, to the degree that these dwarf males function as sperm packages, these organisms can actually also avoid the two-fold cost of meiosis. Dwarf males are produced, e.g., by certain barnacles (e.g., Andersson, 1994) and by many sessile, but not motile, parasites (e.g., Ghiselin, 1974). Dwarf males are present also in angler fish (e.g., Berthelsen, 1951; reviewed Andersson, 1994), that functionally are sessile because they are sit-and-wait predators, and in many spiders that spin webs or sit-and-wait on flowers (e.g., Bristowe, 1929; Vollrath, 1980).

The distinction between motility and sessility does not only imply differences in the absence versus presence of the male, but also in the degree to which self-fertilisation and asexual replication will evolve in the female, or the hermaphrodite: in mobile organisms the males choose the females that have the purest form of sexual reproduction because the males gain no fitness through the female if she replicates asexually. This means that the male choice will select against both self-fertilization and asexual reproduction in the female. In contrast, in sessile organisms there is no selective male choice that can prevent that self-fertilization and asexual reproduction evolve in order to enhance the reproductive rate in the hermaphrodite. These predictions coincide with the fact that self-fertilization and asexual reproduction are almost absent in the sexually reproducing mobile organisms, while the sessile hermaphrodites often self-fertilise and reproduce without sex.

In a few instances there are organisms that are facultatively sessile and mobile, and in such cases we may expect that the sessile stage contains pathogenetic females, whereas the mobile stage contains both males and females that reproduce by sex. One such group is aphids that virtually are sessile during their pathogenetic stage, while they are sexual when they are winged and mobile.

So in conclusion, the proposed model suggests that both male individ-

uals, sexual reproduction, and a diploid genome evolved in relatively large-bodied mobile organisms, where selection by density dependent competitive interactions is exactly so strong that it out-balances both the two-fold cost of the male and the two-fold cost of meiosis. Thereafter, some of these organisms became sessile and, because density dependent competitive interactions in sessile organisms cannot out-balance the four-fold cost of sex, these organisms evolved to the stage of hermaphrodites in order to avoid the four-fold cost of sex.

Chapter 22

Eusociality

A FULLY DEVELOPED eusocial colony contains thousands to millions of sterile workers and/or soldiers that are the offspring of a single, or a few, queen/s that is/are mated with a single, or a few, sexual male/s. In the more moderate form a eusocial colony contains fewer workers, and at the lower limit of eusociality there is cooperative breeding where a single, or a few, offspring stay/s behind to help their parents to raise more young even though the helpers are potentially capable of breeding themselves. While cooperative breeding is common among birds and mammals, eusocial colonies are present mostly in insects where full-blown eusociality is known from termites, ants, and bees. In the less developed form with fewer workers eusociality is present also in gall-making aphids (Itô, 1989) and thrips (Crespi, 1992), and this form has been found also in taxa other than insects, e.g., in a single crustacea, the snapping shrimp (Duffy, 1996), and in a single mammal, the naked mole rat (Jarvis, 1981).

When we turn to a theory on the evolution of eusociality there are many patterns of co-occurring traits that a successful theory needs to explain. First of all the theory should explain the transition from a sexually reproducing pair over cooperative breeding to the fully developed eusocial colony containing thousands of non-replicating workers. At its best the theory should also indicate why this transition has occurred mainly in insects. Apart from this the theory should explain why kin selection is operating in eusocial species, i.e., it should explain why the workers are the offspring of the queen, and why these offspring workers are females in ants and bees while they include both females and males in termites. In connection with this latter difference the theory should also explain why the sex ratio in the sexuals tends to be three females per male in ants and bees, while it tends to be one male per female in termites. Finally, the successful theory should also explain why ants and bees are haplodiploid while termites are diploid.

The classical theory on the evolution of eusociality (reviewed by, e.g., Bulmer, 1994; Crozier and Pamilo, 1996) explains only a small subset of these patterns of co-occurring traits, and these predictions are based on assumptions with respect to the other half of the co-occurring traits. Broadly speaking, the classical theory on the evolution of eusociality is formed primarily by Hamilton's (1964, 1972) notion of kin selection, and Trivers and Hare's (1976) elaboration on the Fisherian sex ratio theory. The former of these two sub theories explains the evolution of eusociality from the assumption of offspring workers and kin selection, and the latter of the two sub theories explains the evolution of the sex ratio in the sexuals from assumptions on the ploidy level of the genome and assumptions on the sex ratio in the worker caste. Although these assumptions, or fundamental traits, are confirmed, in the sense that they exist in the organisms of interest, we cannot know their evolutionary significance before the evolution of these traits are modelled explicitly. This implies that there are several assumptions that we need to relax before the theory of eusociality can be considered to be successful on an evolutionary scale.

In this chapter I elaborate on the classical theory in order to develop a new theory of eusociality that does not contain the traditional assumptions. Hereby, the proposed theory explains not only the evolution of eusociality and the sex ratio in the sexuals, but also why kin selection is operating, why the workers are the offspring of the queen, why these workers are females in ants and bees, why they include both males and females in termites, why ants and bees are haplodiploid and, finally, why termites are diploid. In the development of this theory I will first consider the evolutionary transition from pairwise to eusocial reproduction, and then the evolution of kin selection and offspring workers. Hereafter, I consider the evolution of the sex ratio in both the worker and the sexual caste, before I consider the evolution of the ploidy level in eusocial species.

22.1 Evolution of eusociality and worker caste

Since Hamilton (1964, 1972) it has been assumed that kin selection is fundamental for the evolution of cooperative breeding and eusociality. The essentials of this idea is that an offspring will stay behind and help its parents if by this choice it gains more inclusive fitness than it gains direct fitness if it tried to reproduce. Originally, this idea was coupled to the observation that eusociality seemed to have evolved mainly in the haplodiploid hymenoptera, and that haplodiploids seemed to be predisposed for eusociality when they were compared with diploids. In consequence, the case with eusociality in

the diploid termites was soon seen as a puzzle, but Trivers and Hare (1976) pointed out that haplodiploids are predisposed for eusociality only if the sex ratio in the sexuals in the ancestors is female biased. So it might, in fact, be that in most cases eusociality is equally likely to evolve in both haplodiploids and diploids.

Both the fact that eusocial diploids and haplodiploids exist, and the result that haplodiploids and diploids may not differ that much in the strength of kin selection during the transition to eusociality, suggest that the essential factor that promotes the evolution of eusociality is not the genetic system. This is because, although it is clear that kin selection must operate before an offspring can specialise in helping its parents at the cost of its own reproduction, it is equally clear that kin selection does not explain why it is beneficial that the offspring stays behind only in some instances and not in others. To answer this latter question we may disregard the genomic system, and then, first of all, explain the transition from pairwise reproduction to eusociality by a transition in the optimal number of workers per female. It is this route that I will take in this section in order to show (*i*) that full-blown eusociality evolves by density dependent competitive interactions when the body mass is upward constrained relative to the exploitation efficiency, and (*ii*) that cooperative breeding is evolutionarily optimal when the body mass is in evolutionary steady state.

To explain the evolutionary transition from a sexually reproducing pair to a eusocial colony, let me define a eusocial colony as a colony containing a single, or a few, sexually reproducing females (queens) and a large caste of workers (including soldiers) that are defined as physiologically non-replicating individual. According to this definition a worker can be either a sexual male, which mates with the queen, or a male or female that does not reproduce but are related to the queen, e.g., by being her offspring. In this section I am, however, interested only in the optimal size of the colony and in the optimal number of workers per queen, and therefore, let me characterise the eusocial colony by the size (n), the number of sexual females (n_θ), and the number of workers ($n_w = n - n_\theta$).

To describe this optimum I will turn to the results that we obtained in Chapters 18 and 20. In those chapters we deduced both the optimal colony, or group, size and the optimal composition of a colony into physiologically non-replicating individuals (workers) and replicating females (queens). Summarising from those chapters we have that

$$\begin{aligned} n^{**} &= v(\psi\iota^{*e} + 1) \\ n_\theta^{**} &= v \\ (n_w/n_\theta)^{**} &= \psi\iota^{*e} \end{aligned} \tag{22.1}$$

where ι^{*e} is the evolutionarily determined level of interference among colo-

nies and v represents the cost of colony formation, where the cost is low when v is high and at its maximum when v is one. From eqn 22.1 we have that there is one worker, e.g., a male, per female when $\psi\iota^{*e} = \psi\iota^{**} = 1$, i.e., we expect one worker (male) per female when the body mass is in evolutionary equilibrium. When instead the body mass is in evolutionary steady state, where $\psi\iota^{*e} = \psi\iota^{*s} \approx 2$, we have that eqn 22.1 predicts that there is approximately two workers per female, a situation that resembles cooperative breeding among a male, a female, and an offspring. From eqn 22.1 we also have that there is only a single female per colony when the cost of grouping is maximal, i.e., when $v = 1$. In other words, in this instance we have a situation with uniformly distributed pairs, or uniformly distributed cooperative families, depending upon whether the body mass is in evolutionary equilibrium or in evolutionary steady state. If instead the cost of grouping is low, i.e., if v is high, then, from eqn 22.1 we expect that these pairs or families will aggregate into larger colonies. These colonies are not eusocial, and this is because they lack the worker caste and because they are made up of pairs or cooperative families that reproduce relatively independently of one another. From eqn 22.1 we have that a typical eusocial colony will evolve when the cost of grouping is maximal ($v = 1$) and the body mass is upward constrained, i.e., when $\psi\iota^{*e} \rightarrow \infty$. Then, we predict that the colony contains only a single female (queen) and close to infinitely many workers.

The prediction of a single queen per eusocial colony depends upon the assumption that the cost of grouping is maximal. From Chapter 18 we recall that the cost of grouping is maximal when the complete colony is no better than a single individual in finding resource. Normally, we expect that a large colony is much better in finding resource than a single individual, i.e., we expect that v is much larger than one so that eqn 22.1 predicts that there is more than a single queen per colony. This latter result with only one queen per colony, depends upon the implicit assumption that a worker cannot gather resource that the queen can use for reproduction. In many instances this is not true, and in such cases there may be only a single queen even when the colony is much more efficient in gathering resource than a single individual.

In the present model the evolutionary transition to eusociality is driven by the specific form of cooperation where the individuals within a colony cooperate in relation to interference competition among colonies. According to this view it is cooperation in relation to inter-group interference competition that is the fundamental trait that leads to the evolution of eusociality, whereas the various other forms of cooperation observed in eusocial colonies are derived traits evolving secondarily from the transition to eusocial reproduction. This view is somewhat different from the traditional view where

the transition to eusociality is often seen as a result of the benefits related to cooperation in general, i.e., the benefits related to a general partitioning of the different tasks of work. According to this traditional view it is all forms of cooperative behaviour that can induce the evolution of eusociality. A way to test these two hypotheses against one another is to analyse the major patterns in which eusociality occurs in natural species. In the extreme form of the traditional hypothesis the partitioning of work tasks is seen to be beneficial in general, and this implies that eusociality is expected to be widespread across most taxa. As this is generally not the case it seems that cooperation at the level of eusociality is generally not advantageous, and therefore we expect that it requires a specific set of conditions before it will evolve by natural selection.

In relation to the proposed model, the specific condition promoting the evolution of eusociality is a high level of interference competition that is induced by an upward evolutionary constraint on the body mass. This hypothesis is not that unrealistic because full-blown eusociality with thousands of workers per colony is known only from insects where it is likely that the ecto-skeleton imposes some constraints on the upper limit to the body mass. The evolutionary constraint on body mass does, however, not need to be physiological or genetical, it can instead be environmentally imposed if, e.g., the species live in narrow canals where selection may operate against large-bodied individuals. This type of an environmentally imposed constraint might explain why eusociality has evolved in, e.g., the naked mole rat that lives in subterranean canals, and in snapping shrimps that live in sponge canals in coral-reefs. Also, as the crucial factor promoting the evolution of eusociality in Malthusian relativity is not an absolute constraint on body mass, but instead a body mass that is constrained relative to the rate by which the organism utilises the resource, it is not surprising that eusociality tends to evolve mainly in those insect taxa that explore plentiful resources that are relatively easy to collect.

If eusociality evolves from a body mass that is constrained relative to the utilisation of resource we have that eusociality should co-occur with a high level of interference competition among colonies and with a high population density. This co-occurrence seems to be the case: it has been estimated that one-third of the animal biomass in the Amazonian forest is composed of ants and termites, and that social insects all together account for more than 75% of the total insect biomass (Beck, 1971; Fitthau and Klinge, 1973). Furthermore, in connection to these studies, Hölldobler and Wilson (1990:1) conclude that social insects are “comparably abundant in most other principal habitats around the world.” It is also well-known that aggression is extremely common among colonies of ants. For the harvesting ant *De Vita* (1979) has estimated that the cost of interference competition

among colonies is a 6% chance of being killed per ant foraging hour. In other words, if a worker ant forages for 48 hours it has only a 5% chance of not being killed by conspecifics. It is no wonder that it takes thousands of soldiers to protect the queen.

When the body mass is upward constrained it cannot act as the energetic buffer that in an evolutionary context reflects the amount of resource that the individual can assimilate. Instead, it will be the number of individuals in the colony that will adjust to the amount of resource collected by the colony. This has been observed in a recent study by Kaspari and Vargo (1995) who found that the latitude trend in body mass (Bergmann's rule) we described in Chapter 10 is present also in the colony size of ants.

22.2 Evolution of kin selection and offspring workers

The Hamiltonian hypothesis that kin selection is essential for the evolution of eusociality depends upon the assumption that the workers and soldiers are the offspring of the queen. If instead the workers and soldiers were the sexual partners of the queen we would have a situation where eusocial species can evolve independently of kin selection. A solution to this latter system would be that the queen needed to mate with all, or nearly all, the workers and soldiers, and that each parent would transmit a tiny fraction of its genome to each offspring.

This type of eusocial colony, where the workers are the sexual partners of the queen, are not known from natural species. The absence of such colonies does not imply that they are irrelevant in relation to an evolutionary theory, and this is because we expect that they would exist if natural selection is selecting for them. This implies that if we want to understand the evolution of, or at the very least the evolutionary maintenance of, the type of eusociality that occurs among natural organisms, then it is essential to show that selection is promoting the maintenance of offspring workers at the cost of sexual workers. In this section I address this question by relaxing the assumption of kin selection, and by using a broader definition of genomic selection to determine the optimal ratio of offspring to sexual workers in eusocial colonies. In resemblance with observations I will find that it is optimal that the workers and soldiers are the offspring of the queen, instead of being her sexual partners. The implication of this result is that it is kin selection that is the derived trait evolving from, or being maintained by, the action of genomic selection in eusocial species. This conclusion goes contrary to the Hamiltonian view where it is kin selection that causes the evolution of eusociality.

To describe the evolution of kin selection let me elaborate on the predictions from eqn 22.1 by describing the optimal composition of the workers into sexual males versus sterile offspring. This can be done by letting genomic selection favour the variant where each gene in each individual in the colony has the highest probability of being copied to a sexual offspring. For a gene in a sexual male this probability is determined by direct inheritance, i.e., by sexual reproduction, while for a gene in a sterile offspring it is determined by indirect inheritance, i.e., by inclusive fitness that leads to kin selection. This means that the degree of kin selection in eusocial species is positively related to the fraction of the workers that are offspring workers that do not reproduce: At the limit where all workers are sexual males kin selection is absent, while it is fully developed when all the workers are offspring. Thus, when we determine the evolutionary partitioning of workers into non-reproducing offspring versus sexual males we also determine the degree to which kin selection evolves in eusocial species.

To determine the optimal degree of kin selection, consider a colony of size n containing a single queen, n_w offspring workers, and $n_\phi = n - 1 - n_w$ sexual male workers. Now, let the queen mate with all the males, and let each of the sexuals transmit on average the fraction $1/(n_\phi + 1)$ of its genome to an offspring. Genomic selection will then favour the mode of sexual reproduction where each gene in each individual has the highest probability of being copied to a sexual offspring. For a particular gene in one of the $n_\phi + 1$ sexuals the probability of being copied to a sexual offspring is $1/(n_\phi + 1)$. For a particular gene in a sterile worker the probability of being copied to a sexual offspring is also $1/(n_\phi + 1)$. This is because the worker received that gene from one of the $n_\phi + 1$ sexuals and with the probability $1/(n_\phi + 1)$ that same sexual will transmit that gene to each of the sexual offspring. In other words, for a gene in each of all the individuals in the colony the probability of being copied to a sexual offspring is

$$p = 1/(n_\phi + 1) \quad (22.2)$$

Hence, the selection gradient on the number of sexual male workers is

$$\partial p / \partial n_\phi = -1/(n_\phi + 1)^2 \quad (22.3)$$

As this gradient is negative there is selection against sexual male workers and the workers will thus be the offspring of the queen, as is the case in eusocial species. This prediction also implies that the helpers in cooperative breeders are expected to be offspring instead of extra males that mate with the female.

22.3 Sex ratios in eusocial species

In Section 19.3 we saw that Trivers and Hare (1976) combined the Fisherian sex ratio theory with the Hamiltonian notion of inclusive fitness in order to predict the average sex ratio in sexuals produced by eusocial colonies. The predictions from these models are based on genomic selection and on assumptions with respect to the ploidy level of the genome and the sex ratio in the worker caste. For example, the prediction of a sex ratio of three queens per sexual male in ants and bees is based on the assumptions that ants and bees are haplodiploid and that the worker caste in these taxa contains only female offspring. Likewise, the prediction of an even sex ratio in the sexuals in termites is based on the assumption that termites are diploid.

One reason why the worker sex ratio is treated as an assumption in the classical theory is that in that framework there is no genetical bias against the evolution of male workers in either haplodiploid or diploid eusocials (e.g., Crozier and Pamilo, 1996). So in order to “explain” the difference in the worker sex ratio between diploids and haplodiploids it has been assumed (*i*) that the male individuals in the ancestors to ants and bees lacked the preadaptation to become workers, and (*ii*) that the male individuals in the ancestors to termites had the preadaptation to become workers (e.g., Alexander, 1974; West-Eberhard, 1975; Starr, 1985; Kukuk et al., 1989; Kerr, 1990; Crozier and Pamilo, 1996). Note, that this hypothesis does not extend the explanatory power of the classical theory, and this is because it is impossible to test both whether the males in the ancestors to ants and bees lacked the preadaptation to become workers, and whether the males in the ancestors to termites had this preadaptation. In other words, I could with equal right claim the opposite, and then we would have a “paradox”, and not an “explanation”.

In this section I will provide a firm explanation for the evolution of worker sex ratios. In order to do this I will relax the constraints on the sex ratio in the worker caste and extend the classical model so it predicts not only the optimal sex ratio in the sexuals, but also the optimal sex ratio in the worker caste, given the functional role of the sexual male. In the first subsection, I will extend the Fisherian sex ratio theory of Trivers and Hare (1976) so it applies to the situation with variation in the sex ratio in the worker caste. Then, in the second subsection, I will superimpose individual selection on the sex ratio in the sexuals on top of the extended Fisherian sex ratio theory in order to determine the sex ratio in the worker caste, given the ploidy level of the genome and the functional role of the sexual male. Then, in the third subsection, I will combine the action of genomic and individual selection on the sex ratio in the sexuals in order to re-analyse the evolutionary determinants of the average sex ratio in the sexuals produced by eusocial colonies.

22.3.1 Fisherian sex ratio with variation in worker sex ratio

In order to describe selection on the sex ratio in both the sexuals and the workers in a eusocial colony, let me in this subsection extend Trivers and Hare's (1976) model for Fisherian sex ratios so that it applies also to a worker sex ratio that can vary continuously from zero to infinity. The model that I will develop in this section is purely Fisherian in the sense that it is based exclusively on genomic selection, and that it is the sex ratio in the sexuals that is treated as the derived trait evolving from the sex ratio in the worker caste and the ploidy level of the genome. It is not until in the following two subsections that I will relax the assumption on the worker sex ratio by superimposing individual selection on the sex ratio in the sexuals on top of the model that I develop in this subsection.

From Section 19.3 we recall that the model of Trivers and Hare is based on the assumption that the sex ratio in the sexuals produced by a eusocial colony is controlled by the workers. Due to this assumption we have that the transcription probabilities in the Fisherian fitness expression on the sex ratio in the sexuals are $p_{d,w}$ and $p_{s,w}$, i.e., respectively the probabilities that a gene copied from a sexual daughter or son of the queen to a sexual offspring of that daughter or son is identical by descent to a gene in a worker. In this section where we deal with variation in the worker sex ratio we can define the $p_{d,w}$ probability by the term $\theta_w p_{d,d} + \phi_w p_{d,s}$, where θ_w and ϕ_w respectively are the proportion of the workers that are females and males, and $p_{d,d}$ and $p_{d,s}$ respectively are the probabilities that a gene copied from a sexual daughter to a sexual offspring of that daughter is identical by descent to a gene in a worker female (daughter), or worker male (son). Likewise, $p_{s,w} = \theta_w p_{s,d} + \phi_w p_{s,s}$. According to these expressions we have that each worker individual plays an equal role in controlling the sex ratio so that the overall worker control reflects the composition of the workers into males and females. If we redefine the fitness expression of eqn 19.6 in accordance with this we have that

$$\begin{aligned}\lambda_i &= \frac{n[X(1 - \phi_i) + Y(\theta/\phi)\phi_i]}{X + Y} & (22.4) \\ X &= (\theta_w p_{d,d} + \phi_w p_{d,s})v_d \\ Y &= (\theta_w p_{s,d} + \phi_w p_{s,s})v_s\end{aligned}$$

The Fisherian sex ratio in the sexuals is then

$$\left(\frac{\phi}{\theta}\right)^{*F} = \frac{Y}{X} \quad (22.5)$$

and the Fisherian proportion of females $\theta^{*F} = X/(X + Y)$. We can then

follow the definitions and notations in Chapter 19, and find that the probabilities in eqn 22.4 are defined as

$$\begin{aligned}
 p_{d,d} &= p_{d,m}p_{m,d} + p_{d,f}p_{f,d} \\
 p_{d,s} &= p_{d,m}p_{m,s} + p_{d,f}p_{f,s} \\
 p_{s,s} &= p_{s,m}p_{m,s} + p_{s,f}p_{f,s} \\
 p_{s,d} &= p_{s,m}p_{m,d} + p_{s,f}p_{f,d}
 \end{aligned} \tag{22.6}$$

For diploids it can be seen that all these probabilities are $1/2$. Then, as $v_d = 1/2$ and $v_s = 1/2$ in diploids, it follows that $X = 1/4$ and $Y = 1/4$. Consequently, the Fisherian proportion of females in sexual diploids is one half, i.e., $\theta^{*F} = 1/2$, independently of the proportion of females in the worker caste.

For haplodiploids it can be seen that the transcription probabilities are

$$\begin{aligned}
 p_{d,m} &= 1/2 & p_{s,m} &= 1 & p_{d,d} &= 3/4 \\
 p_{d,f} &= 1/2 & p_{s,f} &= 0 & p_{d,s} &= 1/4 \\
 p_{m,d} &= 1/2 & p_{f,d} &= 1 & p_{s,s} &= 1/2 \\
 p_{m,s} &= 1/2 & p_{f,s} &= 0 & p_{s,d} &= 1/2
 \end{aligned} \tag{22.7}$$

Then, as $v_d = 2/3$ and $v_s = 1/3$ in haplodiploids, it follows that $X = \theta_w/3 + 1/6$ and that $Y = 1/6$. Consequently, the Fisherian proportion of females in sexual haplodiploids is $\theta^{*F} = (\theta_w + 1/2)/(\theta_w + 1)$. This implies that the predicted sex ratio in the sexuals depends upon the proportion of females in the worker caste. For example, if the proportion of females in the worker caste is one, as is the case in ants and bees, the proportion of females in the sexuals is $3/4$, while it is respectively $2/3$ and $1/2$ if the proportion of females in the worker caste is respectively $1/2$ and zero. Note also that the Fisherian sex ratio in the sexuals in haplodiploids is a monotonic function of the worker sex ratio, a result that we will use in Section 22.3.3.

22.3.2 Evolution of sex ratios in the worker caste

We now have a classical model based on genomic selection, and this model will give us the Fisherian sex ratio in the sexuals as a function of the sex ratio in the workers and the ploidy level of the genome. In this section I will show that we can relax this classical assumption on the worker sex ratio and analyse the evolutionary modification of that sex ratio by superimposing individual selection on the sex ratio in the sexuals on top of the classical model with genomic selection. To do this I will first describe the overall principle from a general model. Thereafter, I will consider why the sex ratio in the worker caste in diploids is unaffected of the individual selection that

operates on the sex ratio in the sexuals. I will then turn to haplodiploids and analyse the evolutionary modification of their worker sex ratio, given that the only task of the sexual male is to transmit genes through sexual reproduction.

In order to see how individual selection on the sex ratio in the sexuals can determine the evolutionary optimum to the sex ratio in the workers, let us first consider a general model. For this model let the two-dimensional fitness profile of genomic selection on the sex ratio in the sexuals take the form

$$\lambda_i = f(\theta, \theta_i) \quad (22.8)$$

where f is some undefined function that may take a variety of shapes depending upon the actual form of individual selection, e.g., it may take the form of eqn 22.4. Then, because Fisherian selection is operating at the level of genes, we expect that the average proportion of females (θ) will be situated at the Fisherian optimum (θ^{*F}), which is defined at the limit $\theta_i \rightarrow \theta \rightarrow \theta^{*F}$. Hence, for a group of individuals that do not interbreed with other individuals we find that the fitness of an average individual can be defined as

$$\lim_{\theta_i \rightarrow \theta \rightarrow \theta^{*F}} \lambda_i = \lambda^{*F} = f(\theta^{*F}, \theta^{*F}) \quad (22.9)$$

Now, if the population is substructured into groups that do not interbreed we have a situation with independence between genomic and individual selection. For this special situation it is evident that the fitness profile at the level of individual selection is given by eqn 22.9. This implies that the partial derivative of eqn 22.9 with respect to the Fisherian proportion of females, i.e., $\partial \lambda^{*F} / \partial \theta^{*F}$, will give us the selection gradient of individual selection on the Fisherian optimum to the sex ratio in the sexuals. Now, the evolutionary changes that this selection gradient imposes on the Fisherian optimum will necessarily be driven by evolutionary changes in the parameters that determine the Fisherian optimum to the sex ratio in the sexuals. From the previous subsection we know that one of these parameters is the sex ratio in the worker caste, and individual selection on the sex ratio in the sexuals may thus impose evolutionary changes in the sex ratio in the worker caste. Following this line of thought we find that the selection gradient on the proportion of workers can be given as

$$\frac{\partial \lambda}{\partial \theta_w} = \frac{\partial \lambda^{*F}}{\partial \theta^{*F}} \frac{\partial \theta^{*F}}{\partial \theta_w} \quad (22.10)$$

More generally, we do not expect that populations are perfectly substructured into groups that do not interbreed. Instead, there will nearly always be some degree of gene flow between groups and this will tend to diminish

the strength of individual selection relative to the strength of genomic selection. It will, however, not alter the direction, nor the optimum to individual selection. Hence, if a trait is affected simultaneously by individual and genomic selection and if the two types of selection do not select toward the same optimum and we want to determine the evolutionary optimum, then we need to quantify the degree to which the strength of individual selection is diminished by the spread of genes. However, as we saw in the previous subsection that the worker sex ratio is unaffected by genomic selection, we have that the worker sex ratio will be determined exclusively by individual selection, if it is operating on that sex ratio, and we may thus use eqn 22.10 to determine the evolutionary optimum to the sex ratio in the worker caste.

Worker sex ratio in diploids

Let us now examine why individual selection on the sex ratio in the sexuals does not affect the worker sex ratio in diploids. From Section 22.3.1 we have that $\theta^{*F} = 1/2$ in diploids, independently of variation in the proportion of worker females (θ_w). By eqn 22.10 this implies that $\partial\lambda/\partial\theta_w = 0$ for all θ_w because $\partial\theta^{*F}/\partial\theta_w = 0$ for all θ_w . Thus, in diploids, individual selection on the sex ratio in the sexuals will not affect the sex ratio in the worker caste. In other words, under the current model the sex ratio in the workers of eusocial diploids is neutrally stable in the sense that it is given by initial conditions. As the worker sex ratio is neutrally stable we expect that it will often contain both female and male offspring although in some instances, the worker caste might be fixed for either males or females. Hence, it is not surprising that the worker caste in the diploid mole rats contains an approximately even number of females and males, and that this is most often also the case in the diploid termites, although in this latter taxon, the workers are only males in some species and only females in other species (Noirot and Pasteels, 1987).

Worker sex ratio in haplodiploids

In contrast to the situation in diploids, in haplodiploids individual selection on the sex ratio in the sexuals cause evolutionary changes in the worker sex ratio. This is because the Fisherian sex ratio in the sexuals in haplodiploids is functionally determined by the sex ratio in the workers.

To analyse the evolutionary changes in the worker sex ratio in haplodiploids, we recall from Section 22.3.1 that the relationship between the sex ratio in the sexuals and the worker sex ratio is $\theta^{*F} = (\theta_w + 1/2)/(\theta_w + 1)$ at the Fisherian optimum. This implies that the partial derivative of the Fisherian proportion of sexual females with respect to the proportion of

female workers is

$$\frac{\partial \theta^{*F}}{\partial \theta_w} = \frac{1}{2(\theta_w + 1)^2} \quad (22.11)$$

Then, from eqns 22.9, 22.10, and 22.11 we find that the evolutionary optimum of the worker sex ratio in haplodiploids will depend on the particular form of the function f , i.e., on the particular form of the fitness profile of individual selection on the sex ratio in the sexuals.

For the model we developed in Section 22.3.1 the function f is defined by eqn 22.4, and hence, the fitness profile of individual selection is given by eqn 22.4 at the limit $\theta_i \rightarrow \theta \rightarrow \theta^{*F}$ where

$$\lambda^{*F} = n\theta^{*F} \quad (22.12)$$

This profile resembles the profile in eqn 19.15, and it implies selection for a female biased sex ratio in the sexuals with the two-fold cost of the male being defined by the special case $\lambda_{\theta=1}^{*F}/\lambda_{\theta=0.5}^{*F} = 2$, where $\lambda_{\theta=1}^{*F}$ is the growth rate of a variant with pure females and $\lambda_{\theta=0.5}^{*F}$ is the growth rate of a variant with an even sex ratio. For the one-dimensional fitness profile in eqn 22.12, the selection gradient on the Fisherian proportion of sexual females is $\partial \lambda^{*F}/\partial \theta^{*F} = n$ and the selection gradient on the proportion of female workers thus is

$$\partial \lambda / \partial \theta_w = n/2(\theta_w + 1)^2 \quad (22.13)$$

As this gradient is always positive it follows that the worker caste will contain only female offspring at the evolutionary equilibrium, i.e., $\theta_w^{**} = 1$.

The selection profiles of eqns 22.4 and 22.12 are based on the assumption that successful sexual reproduction depends only on the transmission of the sexual male's gametes to the queen. This is in contrast to the model in Chapter 20 where the male individual evolved because he gained fitness through interference competition. The assumption behind eqns 22.4 and 22.12 is, though, expected to be met in many eusocial species, and this is because the workers, due to their numerical superiority, are expected to take over the fitness component of interference quality from the sexual male. This, e.g., is the case in ants and bees where the sexual males participate only in the act of mating, and thereafter they die while the queens establish the eusocial colonies on their own. Hence, it is not surprising that the worker caste in ants and bees contains only female offspring.

22.3.3 Evolution of sex ratios in the sexual caste

Let us now analyse the evolutionary optimum of the sex ratio in the sexuals under the simultaneous action of both genomic and individual selection.

This optimum will depend upon the form of individual selection, and in this subsection I will consider two different types of individual selection on the sex ratio in the sexuals. The first type corresponds to the form that we considered in the previous subsection, where the task of the sexual male is reduced to the transmission of gametes through sexual reproduction and the two-fold cost of the male selects for a female biased sex ratio. The second type corresponds to a situation where successful reproduction depends not only on the transmission of the male's gametes to the female, but also upon the formation of a pair between the sexual male and the sexual female. In this latter form of individual selection there is selection for an even sex ratio in the sexuals. It is the former of these two types of individual selection that appears to operate in ants and bees, and it seems that it is the latter form that operates in termites. This is because the behaviour of the sexual male in termites is different from the behaviour of the sexual male in ants and bees. While the sexual male and the queen in ants and bees leave one another after they have mated, there seems to be an obligate pair bond in termites where the sexual male, i.e., the king, stays with the queen and helps her during the construction of the eusocial colony.

Selection by two-fold cost of sexual males

Let me first consider the case where the sexual male is two-fold costly, and this cost selects for a female biased sex ratio in the sexuals. In this situation the Fisherian optimum of genomic selection cannot coincide with the optimum of individual selection, and this is because the optimum of individual selection is a sex ratio that is biased to the limit of no males, while the maximal female bias in the Fisherian optimum for the model in Section 22.3.1 is 50% females in diploids, and 75% females in haplodiploids. These differences in the optima of the two levels of selection implies that the evolutionary optimum of the sex ratio in the sexuals is given at the point where the two forces of selection are balanced against one another, and this point will depend on the degree to which the two types of selection operate independently of one another.

To determine this point, where genomic and individual selections on the sex ratio in the sexuals are balanced against one another, we have that genomic selection on the sex ratio is given by the selection gradient $\lim_{\theta_i \rightarrow \theta} \partial \lambda_i / \partial \theta_i = n(X - Y[\theta/\phi]) / (X + Y)$ (from eqn 22.4), and that individual selection on the sex ratio is given by the selection gradient $\partial \lambda / \partial \theta = n$ (from eqn 22.12). The current scaling of these two gradients with respect to one another is based on the assumption that individual selection is operating independently of genomic selection. This assumption will generally not hold, and this is because the spread of genes in the population will tend to minimise the degree to which individual selection can operate in-

independently of genomic selection. One way to model this without making a complex model at the genomic level, is to scale the selection gradient of individual selection by a fraction ($0 \leq \rho \leq 1$) corresponding to the degree that the action of individual selection is diminished by the spread of genes.

When individual selection is scaled by the fraction ρ , the optimal sex ratio in the sexuals is given by the solution to the equation $\lim_{\theta_i \rightarrow \theta} \partial \lambda_i / \partial \theta_i = -\rho \partial \lambda / \partial \theta$, where the left-hand expression is the selection gradient of genomic selection and the right-hand expression represents the scaled selection gradient of individual selection. When these two selection gradients are obtained from eqns 22.4 and 22.12, we find that the equation is equivalent to

$$\frac{n[X - Y(\theta/\phi)]}{X + Y} = -\rho n \quad (22.14)$$

so that the sex ratio at the evolutionary equilibrium is

$$\left(\frac{\phi}{\theta}\right)^{**} = \frac{Y}{X + \rho(X + Y)} \quad (22.15)$$

Hence, for diploids, where $X = Y = 1/4$, the optimal sex ratio in the sexuals approaches one at the limit $\rho \rightarrow 0$, where the action of individual selection is almost completely diluted by the spread of genes, and the optimal sex ratio approaches $1/3$ at the other limit $\rho \rightarrow 1$, where individual selection operates independently of genomic selection. In comparison, for haplodiploids, where $X = 1/2$ and $Y = 1/6$ at the predicted limit where the worker caste contains only female offspring, the optimal sex ratio in sexuals approaches $1/3$ at the limit $\rho \rightarrow 0$, and $1/7$ at the other limit $\rho \rightarrow 1$.

These results suggest that it is possible to estimate the ρ parameter from the degree to which the average sex ratio in the sexuals is female biased in comparison with the classical predictions with a sex ratio of one in diploids and $1/3$ in haplodiploids. More specifically, from eqn 22.15, the parameter ρ can be estimated as $\rho = [(\theta/\phi) - 1]/2$ in eusocial diploids and as $\rho = [(\theta/\phi) - 3]/4$ in eusocial haplodiploids. These estimates are based on a set of simplifying assumptions that may not hold for natural populations. For example, in relation to the estimates from eqn 22.15 the sex ratio in many eusocial species is slightly male biased due to the action of, e.g., multiple matings and worker laying (reviewed in Bulmer, 1994). As most of these biases tend to produce a more male biased sex ratio it follows that eqn 22.15 underestimates the degree of independence between the two levels of selection.

From the data reviewed by Crozier and Pamilo (1996), it is apparent that there is a high degree of variation in the sex ratio estimates for the sexual caste in eusocial haplodiploids, and this makes it difficult to make a clear and firm conclusion on the degree of independence between the two

levels of selection. For 40 species of monogynous ants listed by Crozier and Pamilo (1996) the investment sex ratio, given by the dry weight of the sexual offspring, is $\phi/\theta = 0.5 \pm 0.2$ (SE), and this is more male biased than the predicted $1/3$ at the limit where the force of individual selection is completely diluted by the spread of genes. This result suggests that the force of individual selection is low compared with genomic selection, but, as sex ratios that are much more female biased than $1/3$ exists also, there may be deviations from this suggestion.

Selection by pair formation

Let us now consider the situation where successful reproduction depends upon the formation of a pair between the sexual male and the sexual female, a situation that seems to resemble the case in termites. In this situation the constraints on both genomic and individual selection are different from the situation we dealt with so far. To describe these differences I will first consider genomic selection, and then individual selection, before I consider the evolutionary determinants of the sex ratio under the action of selection at both levels.

For the case with genomic selection by the constraint of pair formation the Fisherian optimum is no longer determined by eqn 22.4, and this is because that equation is based on the assumption that successful sexual reproduction depends only on the transmission of the males gametes to the female. When instead pair formation is essential, an unmated individual is likely to have almost no fitness, and this implies that selection on the sex ratio is determined by selection on the ability to form pairs. That is to say, when the sex ratio in the population is female biased, then there is a mate for all males, while the chance that a female obtains a mate is only ϕ/θ . Likewise, when the sex ratio is male biased there is a mate for all females, while the chance that a male obtains a mate is only θ/ϕ . Thus, with worker control on the production of sexuals, we obtain the following fitness expression

$$\lambda_i = \frac{n[X\theta_i + Y(\theta/\phi)\phi_i]}{X + Y} \quad \text{for } \theta \leq 1/2 \quad (22.16)$$

$$\lambda_i = \frac{n[X(\phi/\theta)\theta_i + Y\phi_i]}{X + Y} \quad \text{for } \theta > 1/2$$

where X and Y are defined in eqn 22.4. For eqn 22.16 we find that the proportion of females at the Fisherian equilibrium is $\theta^{*F} = X/(X + Y)$, exactly as predicted by the traditional eqn 22.4, which is based on a different form of sexual reproduction.

Although the optimum of genomic selection is the same for eqns 22.4 and 22.16, the optimum of individual selection is different. In the previous

subsubsection we saw that in the traditional setup, where successful sexual reproduction depends only on the transmission of the male's genes to the female, the optimal sex ratio under individual selection is the limit with no sexual males. From that section we also recall that the fitness profile of individual selection on the sex ratio in the sexuals is defined by the Fisherian fitness profile (eqn 22.4) at the limit $\theta_i \rightarrow \theta \rightarrow \theta^{*F}$, where the profile of eqn 22.4 reduces to $\lambda^{*F} = n\theta^{*F}$. Using the same approach for eqn 22.16, we find that the fitness profile of individual selection is

$$\begin{aligned} \lambda^{*F} &= n\theta^{*F} & \text{for } \theta^{*F} &\leq 1/2 \\ \lambda^{*F} &= n\phi^{*F} & \text{for } \theta^{*F} &> 1/2 \end{aligned} \quad (22.17)$$

In other words, when the sex ratio is female biased there is individual selection for a more male biased sex ratio, and when the sex ratio is male biased there is individual selection for a more female biased sex ratio. Thus, individual selection by pair formation selects for an even sex ratio in the sexuals, and not a female biased sex ratio as is the case with individual selection by the two-fold cost of the male.

Now, let us examine the optima of the two sex ratios in diploid eusocials when individual selection is defined by pair formation. As the Fisherian optimum of eqn 22.16 corresponds to the optimum of eqn 22.4 we find that an even sex ratio in the sexuals is obtained by genomic selection in diploids independently of the sex ratio in the worker caste. In other words, as the optimal sex ratio in the sexuals is the same for selection at both the level of genes and individuals, there is no conflict between the two levels of selection, and this implies that the evolutionary optimum is an even sex ratio in the sexuals. Then, as the selection gradient of individual selection on the sex ratio in the sexuals is independent of the worker sex ratio, we have that the worker sex ratio is given by initial conditions. Hence, it is not surprising that the sex ratios in the sexuals and the worker caste in termites tend to be approximately even.

The situation with pair formation in haplodiploids is slightly different from the situation in diploids. This is because the Fisherian optimum to the sex ratio in sexual haplodiploids generally differs from one, as it is functionally determined by the sex ratio in the workers. Then, as individual selection will dominate over genomic selection, individual selection will change the sex ratio in the worker caste, so that the Fisherian optimum to the sex ratio in the sexuals is carried toward the optimum of individual selection, i.e., toward a sexual sex ratio of unity. From Section 22.3.1 we know that the Fisherian sex ratio in the sexuals in haplodiploids is a monotonic function of the worker sex ratio, and that it is even only in the situation where the worker caste contains only male offspring. Hence, the evolutionary equilibrium determined by pair formation in eusocial haplodiploids is characterised

by an even sex ratio in the sexuals and a worker caste containing only male offspring. This prediction does not correspond to the situation in ants and bees, and the current model suggests that this is because pair formation generally does not occur between the sexuals in these two taxa.

22.4 Diploid and haplodiploid eusocial species

In the introduction to the previous section we saw that there have been a few attempts to explain the classical assumptions on the worker sex ratio. This does not seem to have been the case with the ploidy level of the genome that has been treated as an unquestionable assumption, exactly as is the case with kin selection (Section 22.2). But, although there seems to be no haplodiploid termites, or no diploid ants and bees, their hypothetical occurrence are not irrelevant in relation to an evolutionary theory. This is because we expect their existence if there were selection for them. This implies that if we want to understand the evolution of, or at the very least the evolutionary maintenance of, the ploidy level in eusocial species, then it is crucial to show theoretically that there is selection for a haplodiploid genome in ants and bees, and that there is selection for a diploid genome in termites. This is what I will do in this section.

To explain the ploidy level of the genome I will extend the model that I developed in the previous section. In that section we saw that we can avoid the traditional assumptions on the sex ratio in the worker caste by superimposing individual selection on the sex ratio in the sexuals on top of the classical model with genomic selection on the sex ratio in the sexuals. In this section I extend this principle in order to avoid the classical assumptions on the ploidy level of the genome. Hereby, the extended version of the sex ratio model can explain not only the evolution of the sex ratio in the sexuals, but also the evolution of the sex ratio in the worker caste together with the evolution and/or maintenance of the ploidy level of the genome.

To extend the theory on eusocial colonies so that it also includes selection on the ploidy level of the genome I will first describe the Fisherian sex ratio for a model with a variable structured genome. Thereafter, I will link this extended version of the Fisherian sex ratio model to the two different modes of individual selection that we analysed in the previous section. Based on this latter model I will show that the two different types of individual selection on the sex ratio in the sexuals will explain the major differences between eusocial termites on one side, and eusocial ants and bees on the other.

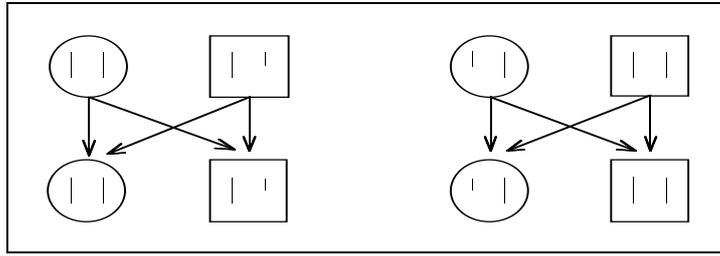


Fig. 22.1 An illustration of a haplodiploid (left) and a diplohaploid (right) biased genomic system. Circles are females and squares males. For details, see the text.

22.4.1 Fisherian sex ratio with variation in ploidy level

The original form of the Fisherian sex ratio model for eusocial species is based on the assumption that the genome is either diploid or haplodiploid. In this subsection I extend this model so that it applies also for a continuous genome that has a haplodiploid and a diplohaploid genome as the two extremes on a continuum with the diploid genome as the intermediate type. The model developed is Fisherian in the sense that it is based exclusively on genomic selection, and that it is the sex ratio in the sexuals that is treated as the derived trait evolving from the ploidy level of the genome, which is treated as being more fundamental. It is not until in the next subsection where I incorporate the action of individual selection that we will deal with the evolution of the ploidy level of the genome.

In the situation where the genome is continuous on a scale from a haplodiploid to a diplohaploid genome the females and males can have from a single to two homologous sets of genes. To describe this let me assume that one of these homologous sets of genes is always complete while the other can vary in the number of homologous genes that it contains. Let h be the fraction of the total number of genes present in the variable set in the female, and let m be the fraction present in the variable set in the male. Also let, for a given variant, h and m be fixed, and let the probability that a gene is present at any particular locus in the variable set be h in females and m in males. Then, let a sexual female transmit a complete set of genes to sons, but only the fraction h to daughters, and let a sexual male transmit a complete set of genes to daughters, but only the fraction m to sons. This genome is illustrated in Fig. 22.1, and it is biased toward haplodiploidy when $h = 1 \wedge 0 < m < 1$, and it is haplodiploid when $h = 1 \wedge m = 0$ and diploid when $h = 1 \wedge m = 1$. Likewise, the genome is biased toward diplohaploidy when $m = 1 \wedge 0 < h < 1$, and it is diplohaploid when $m = 1 \wedge h = 0$.

We can now define the terms in eqns 22.4 and 22.6 as functions of h and m , and in order to do this let us first consider the haplodiploid biased case. In this case a sexual female will copy two sets of genes to a pair containing a son and a daughter, while a sexual male will copy only an average of $1 + m$ sets of genes to that same pair. This implies that the relative value of a sexual daughter in copying genes is $v_d = 2/(3 + m)$, and that the relative value of a sexual son is $v_s = (1 + m)/(3 + m)$. Also, the probability that a sexual daughter copies a gene that is present at a particular locus in the mother to a grand-offspring of that mother is one half ($p_{d,m} = 1/2$), and the probability that a sexual son copies a gene that is present at a particular locus in the mother to a grand-offspring is $p_{s,m} = 1/(1 + m)$. Likewise, it can be seen that

$$\begin{aligned}
 p_{d,m} &= 1/2 & p_{s,m} &= 1/(1 + m) & p_{d,d} &= (3 + m)/4(1 + m) & (22.18) \\
 p_{d,f} &= 1/2 & p_{s,f} &= m/(1 + m) & p_{d,s} &= (1 + 3m)/4(1 + m) \\
 p_{m,d} &= 1/2 & p_{f,d} &= 1/(1 + m) & p_{s,s} &= (2m^2 + m + 1)/2(1 + m)^2 \\
 p_{m,s} &= 1/2 & p_{f,s} &= m/(1 + m) & p_{s,d} &= (1 + 3m)/2(1 + m)^2 \\
 & & v_d &= 2/(3 + m) & v_s &= (1 + m)/(3 + m)
 \end{aligned}$$

when the genome is diploid to haplodiploid. As the diplohaploid case is the mirror image of the haplodiploid case, it can be seen that

$$\begin{aligned}
 p_{d,m} &= h/(1 + h) & p_{s,m} &= 1/2 & p_{d,d} &= (2h^2 + h + 1)/2(1 + h)^2 \\
 p_{d,f} &= 1/(1 + h) & p_{s,f} &= 1/2 & p_{d,s} &= (1 + 3h)/2(1 + h)^2 \\
 p_{m,d} &= h/(1 + h) & p_{f,d} &= 1/2 & p_{s,s} &= (3 + h)/4(1 + h) \\
 p_{m,s} &= 1/(1 + h) & p_{f,s} &= 1/2 & p_{s,d} &= (1 + 3h)/4(1 + h) \\
 v_s &= 2/(3 + h) & & & v_d &= (1 + h)/(3 + h) & (22.19)
 \end{aligned}$$

when the genome is diploid to diplohaploid. If we insert these values into eqns 22.4 and 22.6, and solve for the Fisherian equilibrium, we find that

$$\theta^{*F} = \frac{2\theta_w(1 - m) + 1 + 3m}{2(m + 1)(\theta_w[1 - m] + m + 1)} \quad (22.20)$$

for a haplodiploid biased genome, and that

$$\theta^{*F} = \frac{2h\theta_w(h - 1) + 1 + 3h}{2\theta_w(h + 1)(h - 1) + 4(1 + h)} \quad (22.21)$$

for a diplohaploid biased genome. By eqns 22.20 and 22.21 we have obtained the Fisherian optimum as a function of the worker composition (θ_w) and the ploidy level of the genome (m, h). In Fig. 22.2 I show the Fisherian sex ratio in the sexuals for the complete range of ploidy levels, given that

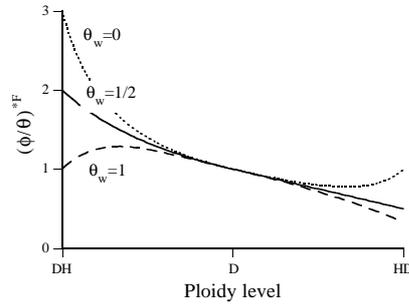


Fig. 22.2 The Fisherian sex ratio in the sexuals $[(\phi/\theta)^*F]$ as a function of the ploidy level of the genome, given that the proportion of female workers (θ_w) is respectively zero, one half, and one. *DH* indicates a diplohaploid, *D* a diploid, and *HD* a haplodiploid genome.

the worker sex ratio is either even or biased to the limits of pure females or pure males. From each of the three curves in the figure it is apparent that a diplohaploid biased genome generates a male bias in the Fisherian sex ratio, and that a haplodiploid biased genome generates a female biased sex ratio. Also, by comparing across the three curves, it is apparent that the Fisherian sex ratio in the sexuals becomes more female biased when the worker caste is female biased, and that the Fisherian sex ratio in the sexuals becomes more male biased when the worker sex ratio is male biased.

22.4.2 Evolution of ploidy level

By eqns 22.18 to 22.21 we have a classical model based on genomic selection giving us the Fisherian sex ratio in the sexuals as a function of the ploidy level of the genome and the sex ratio in the workers. In this subsection I superimpose individual selection on the sex ratio in the sexuals on top of this model. I do this in order to show that a male that is two-fold costly imposes selection for a haplodiploid genome and a worker sex ratio of pure females, while pair formation between a sexual male and a sexual female imposes selection for a diploid genome and a worker sex ratio given by initial conditions.

To incorporate the effects of individual selection we notice that the genomic selection in eqns 22.4 and 22.16 does not affect either the ploidy level of the genome or the sex ratio in the workers, and this is because these traits are the fundamental traits determining the Fisherian sex ratio in those models. This implies that the evolutionary optima to the ploidy level and the worker sex ratio are independent of genomic selection and, hence, their evolution can be analysed by the model of individual selection

on the sex ratio in the sexuals that eqn 22.10 illustrates for the evolution of the sex ratio in the worker caste. This implies that the selection gradient on the ploidy level is given by the following two equations

$$\begin{aligned}\frac{\partial \lambda}{\partial h} &= \frac{\partial \lambda^{*F}}{\partial \theta^{*F}} \frac{\partial \theta^{*F}}{\partial h} \quad \text{for } m = 1 \\ \frac{\partial \lambda}{\partial m} &= \frac{\partial \lambda^{*F}}{\partial \theta^{*F}} \frac{\partial \theta^{*F}}{\partial m} \quad \text{for } h = 1\end{aligned}\quad (22.22)$$

where $\partial \theta^{*F} / \partial h$ and $\partial \theta^{*F} / \partial m$ are the partial derivatives of respectively eqns 22.20 and 22.21, and $\partial \lambda^{*F} / \partial \theta^{*F}$ is the partial derivative of either eqn 22.4 or 22.16, at the limit $\theta_i \rightarrow \theta \rightarrow \theta^{*F}$, depending upon the particular mode of individual selection: If individual selection on the sex ratio in the sexuals is defined by the two-fold cost of the male, then the partial derivative $\partial \lambda^{*F} / \partial \theta^{*F}$ is given by eqn 22.4, while it is given by eqn 22.16 if individual selection is defined by the formation of sexual pairs.

Two-fold cost males and a haplodiploid genome

In the case where individual selection on the sex ratio in the sexuals is defined by the two-fold cost of the sexual male the two-dimensional fitness profile for the ploidy level and the worker sex ratio is given by eqns 22.12, 22.20, and 22.21. In Fig. 22.3a I show three one-dimensional projections of this profile on the ploidy level, given that the worker sex ratio is respectively zero, one, and infinity. From each of these projections it is apparent that it is a general tendency that variants with a haplodiploid biased genome tend to be more fit than variants with a diplohaploid biased genome. Also, by comparing across the three curves, it can be seen that variants with a female biased worker sex ratio tend to be more fit than variants with a male biased worker sex ratio, at least as long as the ploidy level is biased toward either a diplohaploid or a haplodiploid genome. These results suggest that the evolutionary equilibrium is a haplodiploid genome and a worker caste containing only female offspring.

This result is confirmed in Fig. 22.3b, which shows a variety of simulated evolutionary trajectories when individual and genomic selections are operating simultaneously on the two sex ratios and the ploidy level of the genome. From the figure it is apparent that individual selection for a female biased sex ratio in the sexuals implies that the evolutionary trajectories end up with a haplodiploid genome and a worker caste containing only female offspring. Then, from the results in Section 22.3.3 we obtain the additional result that the equilibrium sex ratio in the sexuals is between three and seven females per male, depending upon the degree of independence between the two levels of selection. Given these results it is not surprising

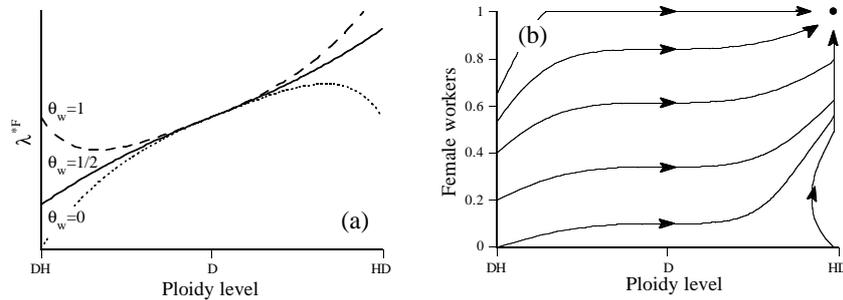


Fig. 22.3 The ant- and bee-like case where individual selection on the sex ratio in the sexuals is defined by the two-fold cost of the sexual male. **(a)** Fitness profiles on the ploidy level of the genome, given that the proportion of female workers (θ_w) is respectively zero, one half, and one. **(b)** Evolutionary trajectories in the proportion of female workers and the ploidy level of the genome. The filled circle indicates that the evolutionary equilibrium is a haplodiploid genome and 100% female workers. *DH* indicates a diplohaploid, *D* a diploid, and *HD* a haplodiploid genome.

that ants and bees are haplodiploid with a worker caste containing only female offspring.

Pair formation and a diploid genome

Compared with the situation where individual selection on the sex ratio in the sexuals is defined by the two-fold cost of the sexual male, the evolutionary equilibria of the ploidy level and the worker sex ratio are different if individual selection is defined by the formation of a pair between the sexual male and the sexual female.

In this latter case the selection pressure of individual selection is defined by eqn 22.17 instead of eqn 22.12, and the two-dimensional fitness profile on the ploidy level and the worker sex ratio is given by eqns 22.17, 22.20, and 22.21. In Fig. 22.4a I show three one-dimensional projections of this profile on the ploidy level, given that the worker sex ratio is respectively zero, one, and infinity. From each of these projections it is apparent that it is a general tendency that variants with a diploid genome tend to be more fit than variants with a diplohaploid or haplodiploid biased genome. Also, by comparing across the three curves, it can be seen that, when the ploidy level is biased toward a diplohaploid genome, then the variants with a female biased worker sex ratio tend to be more fit than the variants with a male biased worker sex ratio, while the opposite is true when the ploidy level is biased toward a haplodiploid genome. These results suggest that the

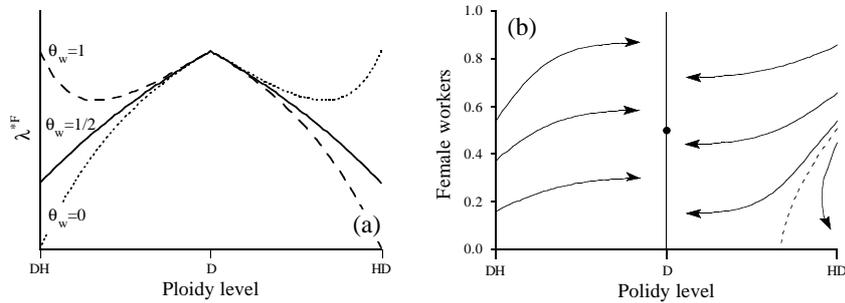


Fig. 22.4 The termite-like case where individual selection on the sex ratio in the sexuals is defined by the pair that is formed by the king and the queen. (a) Fitness profiles on the ploidy level of the genome, given that the proportion of female workers (θ_w) is respectively zero, one half, and one. (b) Evolutionary trajectories in the proportion of female workers and the ploidy level of the genome. The dashed line is the borderline that separates fixation at a diploid genome from fixation at a haplodiploid genome, and the filled circle indicates the expected equilibrium when eusociality evolves from a sexual pair that is diploid. *DH* indicates a diplohaploid, *D* a diploid, and *HD* a haplodiploid genome.

evolutionary equilibrium is a diploid genome and a worker sex ratio that depends on initial conditions.

This result is confirmed in Fig. 22.4b, which shows a variety of simulated evolutionary trajectories when individual and genomic selections are operating simultaneously on the two sex ratios and the ploidy level of the genome. Then, by comparing Fig. 22.3 with Fig. 22.4, it is apparent that the transition from an ant- or bee-like colony formed by a queen to a termite-like colony formed by a king-queen pair, generates an evolutionary transition from a haplodiploid to a diploid genome. The transition to a termite-like colony also implies that the proportion of females in the workers shifts from being obligate one to being determined by initial conditions, with an even sex ratio being a special case that evolves when, among other things, the transition to eusociality occurs from an organism that initially is diploid. As this is the most likely evolutionary trajectory for termites it is not surprising that they tend to have an even sex ratio in their worker caste. Finally, from the results in Section 22.3.3, we have that the evolutionary equilibrium in the sex ratio in the sexuals is one, as it is indicated also by the data on termites.

At this point you may note that, like the rest of the theory on selection by density dependent competitive interactions, the model on eusocial haplodiploids is very much an ADO model, as they are defined in Section 9.2.2. This is because we expect individual selection by the two-fold cost of the

male when the workers, due to their numerical superiority, take over the fitness component of interference quality from the sexual male. On the other hand, the model on eusocial diploids is not an ADO model, and this is because the constraint on pair formation is given by assumption instead of being evolutionarily explained. However, as there seems to be no obvious way to explain the constraint of pair formation from the assumption of eusociality and a diploid genome, the proposed model might provide the true evolutionary explanation for the co-occurrence of pair formation and a diploid genome in eusocial species.

The evolutionary causality underlying the predictions in Sections 22.3 and 22.4 is quite different from the causality underlying the original prediction of Trivers and Hare (1976). In the original explanation it is assumed that it is the ploidy level of the genome and the sex ratio in the worker caste that induce the evolution the sex ratio in the sexuals. When instead these two assumptions are relaxed, as it has been done in this chapter, we find that both the haplodiploid genome and the worker caste of purely females in ants and bees, and the diploid genome in termites can be seen as the derived traits that either evolve from, or are maintained by, a more fundamental form of individual selection operating on the sex ratio in the sexuals. In this sense it is the optimal sex ratio in the sexuals that defines the selection pressure on the ploidy level of the genome and the sex ratio in the worker caste, and not the other way around as it traditionally has been thought.

Although, in the proposed theory, the propagation of the selection pressure through the population is reversed in relation to the classical theory, it is highly unlikely that the diploid genome is not more ancestral than the occurrence of eusociality in termites. This result is not in contradiction with the proposed theory, and this is because the optimality model describes only the direction in which the selection pressure is propagated through a particular population, and not necessarily the chronological order in which the different traits will evolve. Hence, the model in this chapter may explain only the evolutionary maintenance of the diploid genome in termites, while the evolution of the diploid genome could be explained, e.g., by the model in Chapter 21. On the other hand, although the model in Chapter 21 may also lead to the evolution of a haplodiploid genome, in relation to the haplodiploid hymenoptera the chronological order might be the reverse so that the haplodiploid genome evolved as a consequence of eusociality in a diploid ancestral species. This might be possible because the basic prediction of the ADO model is that a haplodiploid genome can follow from the evolution of eusociality. At first, this hypothesis might appear paradoxical in relation to the widespread consensus that the haplodiploid genome is the ancestral trait in hymenoptera, and that eusociality evolved later in at least eleven or twelve different cases. However, here it is worth to notice that the

proposed theory predicts a reversion to pair-wise reproduction if a eusocial species experiences a drastic decline in resource density, and such a decline can arise from a variety of mechanisms including an overall environmental crisis and inter-specific interference competition. In other words, the haplodiploid genome might have evolved from eusociality in an early ancestral species that thereafter returned to pair-wise reproduction during an environmental crisis, and thereafter this haplodiploid lineage could diversify into new lineages containing eusocial species.

Part VI

Evolutionary population dynamics

Chapter 23

Fundamental theorem replaces Malthusian law

DURING THE preceding parts of the book it has become clear that the fundamental theorem of natural selection does not apply to natural populations in the form proposed by Fisher in 1930. Originally, Fisher's theorem was meant to define natural selection where the intrinsic Malthusian parameter defined fitness so that the maximal and the intrinsic growth rate of the population continued to increase. However, in Chapters 10 to 12 we saw that the fundamental theorem does not apply to natural populations, and this is because the theorem, among other things, is inconsistent with the occurrence of both large-bodied organisms and with the occurrence of highly limited rates of reproduction. Moreover, we saw in Chapter 14 that the intrinsic growth rate is predicted to decline when the environment is stable, a result that is diametrically opposite to the fundamental theorem. More generally, the fundamental theorem holds only in the absence of competitive interactions, a hypothetical case that is fulfilled only at the limit of zero density.

In the same way as Fisher's fundamental theorem of natural selection does not apply as the fundament of natural selection, you might also have noticed that the Malthusian law of exponential increase in a density independent environment generally does not apply as the first law of population dynamics. This is because the Malthusian law is based on the assumption that the intrinsic growth rate is constant. But, this is generally not true. For example, from Fishers fundamental theorem we know that if competitive interactions and density dependence are absent, then the intrinsic growth rate is expected to increase at a rate given by the genetic variance in that growth rate. In other words, the fundamental assumption behind the

classical theory of population dynamics is invalid whenever there is genetic variation in the intrinsic growth rate.

In this chapter I will use Fisher's fundamental theorem to extend the first law of population dynamics so that it also includes the situation with genetic variation in the intrinsic growth rate. This implies that the fundamental theorem will replace the Malthusian law as the cornerstone of the population dynamic theory, and that the classical theory is valid only in the absence of genetic variation.

23.1 Fundamental theorem leads to hyper-exponential increase

Let me in this section describe the population dynamics that follow from the fundamental theorem and the hypothetical case with a density independent environment where competitive interactions are absent. In this environment the fundamental theorem will apply, and this implies that the rate of increase in the intrinsic growth rate (r) is equal to the genetic variance in that growth rate (σ_r^2), i.e., that

$$dr/dt = \sigma_r^2 \quad (23.1)$$

By integrating eqn 23.1 we obtain

$$r_t = r_0 + \sigma_r^2 t \quad (23.2)$$

where r_t and r_0 are the growth rates at time t and time zero. As r_t is the average rate of increase per individual at time t , we have that

$$dN/dt = (r_0 + \sigma_r^2 t)N \quad (23.3)$$

When this expression is solved for the abundance we obtain the law of hyper-exponential increase in the abundance, i.e., we find that

$$N_t = N_0 e^{r_0 t + \sigma_r^2 t^2 / 2} \quad (23.4)$$

This equation indicates that the potential rate of increase in the population abundance may be much higher than it is the case under the classical hypothesis, and it also shows that the new law of hyper-exponential increase reduces to the Malthusian law of exponential increase (i.e., to $N_t = N_0 e^{r t}$) when genetic variation is absent, i.e., when $\sigma_r^2 = 0$. A comparison between the new and the classical law is shown in Fig. 23.1.

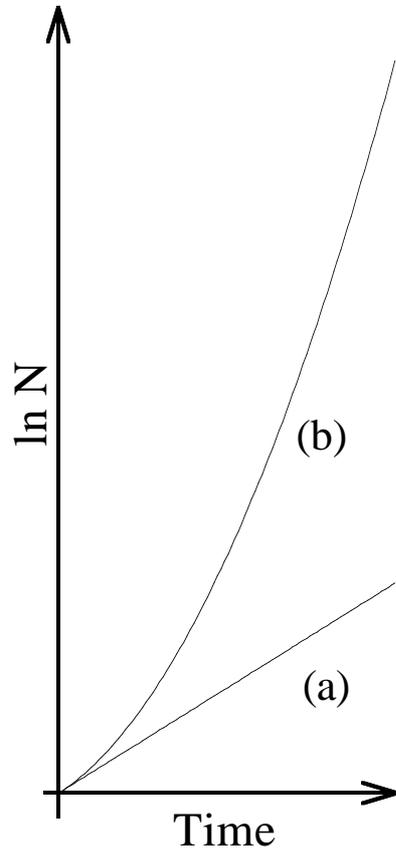


Fig. 23.1 An illustration of exponential (a) and hyper-exponential increase (b).

Chapter 24

Single species cycles

CYCLIC POPULATION DYNAMICS is widespread among the species within the animal kingdom. Who is not familiar with the cyclic outbreak dynamics in small herbivores, like rodents and snowshoe hares, but the dynamics of crustaceans, like *Daphnia*, or insects, like Lepidoptera, also tend to be inherently cyclic. This is unlike the dynamics of the classical theory of population dynamics, which is inherently non-cyclic unless there is some sort of delayed regulation caused by, e.g., over-compensation in age- or stage-structured populations or by interactions with other species.

From the first part of the book we recall that over-compensation in structured populations does not explain the general tendency for cyclic dynamics in natural populations, and that there have been two major hypotheses for the widespread occurrence of cyclic dynamics. On the one hand, it has been argued that the cycles arise from factors intrinsic to populations while on the other hand it has been argued that they arise from interactions between different species, especially between predators and their prey. The former of these two hypotheses is the Chitty hypothesis, which was never confirmed theoretically, while the latter hypothesis is predicted by the Lotka-Volterra predator-prey equations. Hence, as there seems to have been only one plausible hypothesis for cyclic dynamics, it is not surprising that the ecologists of today are returning to the old idea that population cycles in animals as diverse as the snowshoe hare, rodents, and insects are caused by predator-prey interactions in their broadest definition that includes both host-parasitoid and plant-herbivore interactions (e.g., Akçakaya, 1992; Hanski et al., 1993; Hanski and Korpimäki, 1995; Krebs et al., 1995; Berryman, 1996).

This necessity to incorporate predator-prey interactions in order to obtain cyclic dynamics is, though, a limitation imposed by the Malthusian law and the assumption that the maximal growth rate is constant (Ginzburg, 1992). If instead the maximal growth rate is density dependent the dynam-

ics of a single species can be inherently cyclic. This is what I will show in this chapter where I predict cyclic dynamics by exchanging the Malthusian law with the fundamental theorem of natural selection and by incorporating selection by density dependent competitive interactions. Then, the maximal growth rate becomes density dependent, and the dynamics becomes inherently cyclic with periods that resemble the periods in the dynamics of the forest insects we examined in Chapter 3.

The proximate force behind the cyclic dynamics that I deduce in this chapter is an evolutionary and cyclic modulation of the population equilibrium. Due to this modulation the population will cycle because the abundance is always moving toward the population equilibrium, but as this equilibrium is constantly changing the population cannot settle at that equilibrium. It is only at the evolutionary equilibrium that the abundance is stable, and this is because it is only at that particular population equilibrium that there is no selection for a change in the population equilibrium. Dependent of the particular conditions the evolutionary equilibrium may be stable or unstable, but in nearly all cases the dynamics following from a perturbation will cycle around this equilibrium in an either damped, stable, or exploding fashion.

The ultimate force that drives the population cycles, including the cycles in the population equilibrium, is an evolutionary and cyclic modulation of the phenotype that in turn is driven by a cyclic form of selection that arises from cyclic and density dependent changes in the number of competitive interactions per individual. The cyclic changes in the phenotype can, among other things, include changes in the body mass, the intrinsic growth rate, and the sex ratio, and these sorts of changes are often observed in the small rodents that have cyclic dynamics.

In order to give a full description of the dynamics following from density dependent competitive interactions I have partitioned this chapter into eight sections and one appendix with three subsections. In the first section I deduce the logistic equation with density dependent selection and I also describe the cyclic dynamics that follow. In Section 24.2 I give a brief description of the dynamics in order to improve the readers intuitive understanding of the system, and in Sections 24.3 and 24.4 I describe the cyclic phenotypes and I examine also the degree to which these phenotypes are observed in natural populations with cyclic dynamics. Then, in Section 23.5, I test the developed model against the dynamics in forest insects, and in the following three sections I deal with respectively a deduction of the Calder hypothesis on the body mass allometry for the population cycle, the implication of the neutral stability of the predicted cycles, and the dynamics that are associated with extreme perturbations, like the introduction of exotic species into new areas.

24.1 Logistic equation with density dependent selection

Until this last part of the book we have dealt either with population dynamics under the assumption that evolutionary changes are absent, or with evolutionary changes under the assumption that population dynamics is absent, i.e., under the assumption that the population is situated at the population equilibrium. In this section I will relax these assumptions and describe population dynamics under the action of selection by density dependent competitive interactions.

To describe this form of dynamics let me focus on the logistic equation that we developed in Chapter 3 for the case with non-overlapping generations. That is, let me use the model

$$N_{t+1} = N_t \lambda_m N_t^{-\gamma} \quad (24.1)$$

When genetic variation is present this equation is no longer valid because selection will alter the maximal growth rate (λ_m) so that it becomes both time and density dependent. The mathematics of this evolutionary modulation is described in the first appendix to this chapter. There, I use selection by density dependent competitive interactions to derive the evolutionary changes in the maximal growth rate, when the abundance is no longer in population equilibrium. As it is shown, this implies that the logistic eqn 24.1 extends to the following two equations

$$\begin{aligned} \lambda_{m,t} &= \lambda_{m,t-1} N_{t-1}^{-\gamma_q} e^{\sigma_{\ln q}^2} \\ N_{t+1} &= N_t \lambda_{m,t} N_t^{-\gamma} \end{aligned} \quad (24.2)$$

where $\gamma_q = \gamma_t \psi \sigma_{\ln q}^2$. These equations are based on the assumption that the phenotypic plasticity in reproduction and survival is environmentally induced only. However, when a population experiences a cyclic selection pressure, as is the case with eqn 24.2, then there is selection for a more flexible phenotype that can be adjusted according to the current selection pressure. This means that cyclic changes in λ_m may reflect not only evolutionary changes, generated by the immediate selection pressure, but also plastic responses, as they have been selected for during larger time spans. When this is the case the parameter $\sigma_{\ln q}^2$ in eqn 24.2 will not only stand for genetic variance, but it will include also a component of phenotypic plasticity.

Before I describe the cyclic dynamics of eqn 24.2, let me briefly consider the two special cases that represent respectively the absence of genetic variance (and a flexible phenotype) and the absence of density regulation. When genetic variance is absent we have that $\gamma_q = 0$ because $\sigma_{\ln q}^2 = 0$

and, thus, the top equation in eqn 24.2 reduces to $\lambda_{m,t} = \lambda_{m,t-1}$. This means that the maximal growth rate is constant so that eqn 24.2 reduces to the logistic equation in the classical theory (eqn 24.1). In other words, the deduced theory reduces to the classical theory when genetic variation is absent.

When instead it is density regulation that is absent we have that $\gamma = 0$, and that $\gamma_q = 0$, because $\gamma_\iota = 0$. In this situation, which is characterised by Fisherian selection, eqn 24.2 reduces to

$$\begin{aligned}\lambda_t &= \lambda_0 e^{\sigma_{\ln q}^2 t} \\ N_t &= N_0 \lambda_0^t e^{\sigma_{\ln q}^2 \sum_{\tau=0}^t \tau}\end{aligned}\quad (24.3)$$

a situation with geometrical increase in the growth rate and hyper-geometrical increase in the abundance. From the earlier parts of the book we recall that this situation is characterised also by a geometrical decline in competitive quality.

In natural situations there are both density regulation and genetic variation, and then the dynamics of the model with density dependent selection is inherently cyclic with an either stable or unstable equilibrium. As it is shown in the first appendix to this chapter, the evolutionarily determined population equilibrium is unstable when $\gamma_q \geq \gamma$, or when $\gamma \geq 2$ and $\gamma_q \leq \gamma - 4$. The latter of these two criteria is associated with dynamics that generally is oscillatory with a period of two generations, and this type of dynamics is driven primarily by over-compensatory density regulation. The former criterion is instead associated with non-over-compensatory dynamics that is cyclic with a highly variable period, and this dynamics is caused primarily by the evolutionary modulation of the phenotype.

In relation to the stability of these latter cycles they are damped when $\gamma_q < \gamma$, stable when $\gamma_q = \gamma$, and repelling or exploding, when $\gamma_q > \gamma$. To examine the conditions that determine this instability recall that $\gamma_q = \gamma_\iota \psi \sigma_{\ln q}^2$ and that $\gamma = \gamma_\alpha + \mu \gamma_\iota$. Therefor, the condition $\gamma_q \geq \gamma$ resembles

$$\gamma_\alpha / \gamma_\iota \leq \psi \sigma_{\ln q}^2 - \mu \quad (24.4)$$

This implies that the dynamics become more stable when the resource regulation (γ_α) or the average cost of interference (μ) are increased. Moreover, the dynamics become more unstable when the density dependence in the level of interference (γ_ι), the genetic variance ($\sigma_{\ln q}^2$), and the intra-population differentiation in interference regulation (ψ) are increased.

When the cyclic dynamics generated by density dependent selection are stable, i.e., when $\gamma_q = \gamma$, then the period is determined by the γ parameter, and it declines from an infinite number of generations for $\gamma = 0$ to two generations for $\gamma = 4$. On the continuum from $\gamma = 3.5$ to $\gamma = 4$ the cycle

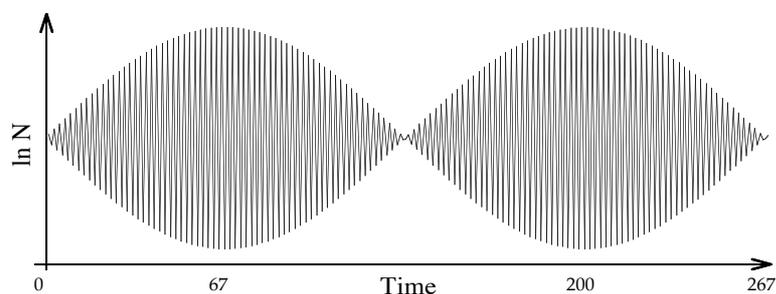


Fig. 24.1 A projection of the logistic equation with selection that shows the occurrence of a cycle in the amplitude of the population cycle; the population cycle has a period of ≈ 2 generations, while the amplitude has a period of ≈ 133 generations.

is dominated increasingly by a cyclic change in the amplitude of the cycle. This latter type of dynamics is illustrated in Fig. 24.1 where the amplitude cycle has a period of approximately 133 generations. As $\gamma \rightarrow 4$ the period of the amplitude cycle continues to increase, from ≈ 10 generations for $\gamma = 3.9$ to an infinite number of generations at the limit $\gamma = 4$. Beyond $\gamma = 4$ the population period is two generations, while the amplitude increases geometrically.

24.2 Population cycle driven by a cyclic population equilibrium

To understand the evolutionary forces underlying the cyclic dynamics consider a downward perturbation of the evolutionary equilibrium, a situation that is illustrated in Fig. 24.2a. Following this perturbation the density regulation is relaxed and, consequently, the reproductive rate will rise and the abundance will increase toward the evolutionary equilibrium.

Associated with the relaxed density regulation there is a decline in the level of interference, and this decline selects for an increase in the intrinsic growth rate at the cost of competitive quality. Thus, when the abundance has reached the evolutionary equilibrium the population will no longer be in population equilibrium at that equilibrium, and this is because the increased growth rate induces an increase in the population equilibrium. Consequently, the population will increase beyond the evolutionary equilibrium heading toward the new population equilibrium. As is illustrated in Fig. 24.2a, the population will never reach this equilibrium, and this is

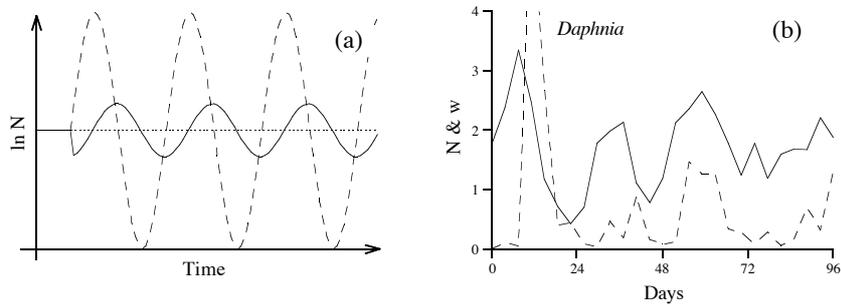


Fig. 24.2 (a) Projection of the logistic equation with selection. The solid curve is the population density, the dashed curve the population equilibrium, and the dotted line the evolutionary equilibrium. (b) The dynamics in the density (N) and body mass (w) of a *Daphnia* population against time. The solid curve is the population density (10^5 *Daphnia* per m^{-3}) and the dashed curve the body mass (μg dry weight). Data from Murdoch and McCauley (1985).

because as soon as the abundance increases beyond the evolutionary equilibrium, then the competitive interactions will select for an increase in competitive quality at the cost of the intrinsic growth rate. Thus, the population equilibrium evolves downward toward the increasing population.

At some intermediate abundance the downward evolving equilibrium and the upward increasing population will intercept, and the population will be in population equilibrium with no change in abundance. At this equilibrium the abundance is at its peak, and selection for competitive quality is thus at its strongest. Hence, the population equilibrium will continue to evolve downward with the result that the abundance will decline in order to keep up with the downward evolving equilibrium.

In Fig. 24.2a the abundance cannot keep up with the equilibrium, and consequently the distance between the abundance and the equilibrium increases until the population intercepts the evolutionary equilibrium with the fastest decline in density that the population encounters during the cycle. Having crossed the evolutionary equilibrium selection reverses, so that energy is allocated from competitive quality to the intrinsic growth rate, and the population equilibrium evolves upward toward the declining population. At some intermediate density the declining population intercepts the upward evolving equilibrium, the rate of change in abundance changes from negative to positive, and the next period begins.

24.3 Cyclic phenotypes

Associated with the population cycle predicted from selection by density dependent competitive interactions, there is a cycle in the phenotype. We have already seen that when the density is above the evolutionary equilibrium, then selection is increasing the competitive quality at the cost of the intrinsic growth rate, while the opposite is the case when the density is below the evolutionary equilibrium.

This cycle in the phenotype resembles the phenotypic cycles found in the small rodents that have cyclic dynamics. Here, it is generally observed that when the densities are low and increasing, then the rodents are small, non-aggressive, and they have a high rate of reproduction. When, instead, the densities are high and declining the rodents are aggressive, 20-30% larger than at low densities, and they have a low rate of reproduction (Krebs, 1978; Boonstra and Krebs, 1979; Stenseth, 1982). Moreover, according to the developed theory we expect that there is strong selection at the peak and bottom densities, and this also coincides with rodent evidence. For example, at the peak density in a rodent cycle Krebs et al. (1973) found that the frequency of the LAPs allele changed from ≈ 0.70 to ≈ 0.40 in less than one generation. These results suggest that selection by density dependent competitive interactions play a role in the generation of cyclic dynamics in small rodents.

You may recall from Chapter 4 that Murdoch and McCauley (1985) observed cyclic dynamics in a *Daphnia* population, and that this cycle occurred even though there were no predators and even though the density of the resource (algae) remained stable. This observation does not fit the hypothesis of a predator-prey driven cycle, but it would fit the current hypothesis if we could detect cyclic changes in the body mass, which are expected to represent competitive quality. From Murdoch and McCauley's Fig. 3 I could calculate the average body mass in the *Daphnia* population, and in Fig. 24.2b I have plotted both the average body mass and the population density against time. From the figure it is apparent that the body mass cycles, and that this cycle is lagging behind the cycle in density, as predicted by the logistic equation with density dependent selection. From eqn 24.14 the predicted relation between the body mass and the population density is

$$\ln w_t - \ln w_{t-1} = \gamma_q \ln N_{t-1} + c \quad (24.5)$$

where c is a constant. This relation is significantly present in the *Daphnia* population ($r = 0.49$, $n = 26$, $\gamma_q = 1.7 \pm 0.5(\text{SE})$, and $c = -0.55 \pm 0.36$), suggesting that selection by density dependent competitive interactions play a role in the cyclic dynamics of *Daphnia* populations.

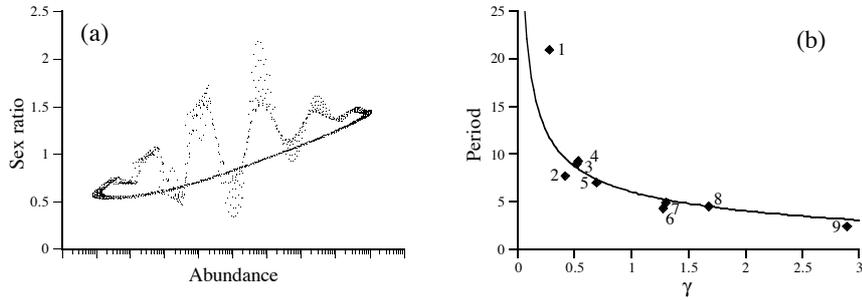


Fig. 24.3 (a) A projection of eqn 24.6 that shows a phase portrait between the sex ratio and the abundance. (b) The period of the population cycle in generations against the γ parameter (assuming $\gamma_q = \gamma$). The curve is defined by eqn 24.2, and the numbered diamonds represent the following species: 1: Spruce budworm. 2: Southern pine beetle. 3: Douglas-fir tussock moth. 4: Larch budmoth. 5: Fall webworm. 6: Nun moth. 7: Pine looper moth. 8: Larch cone fly. 9: Wasp spp.

24.4 The sex ratio cycle

According to the mechanism of selection by density dependent competitive interactions, the cyclic changes in competitive quality include phenotypic traits as diverse as body mass, metabolic rate, antagonistic behaviour, and group size. The sex ratio is also included because the male often is competitively superior to the female. In this section I deal with the incorporation of the sex ratio into the population dynamic equation, a situation that conforms to the transition from an asexual to a sexual species.

The mathematical deduction of the population model for a species with non-overlapping generations, sexual reproduction between males and females, and selection due to density dependent competitive interactions is carried out in the second appendix to this chapter. As it is shown, the incorporation of sexual reproduction leads to the following population equations

$$\begin{aligned}
 \phi_t &= \phi_{t-1} N_{t-1}^{\gamma_\phi} e^{-\phi_t \sigma_{\ln \phi}^2 / (1 - \phi_t)} & (24.6) \\
 \lambda_{m,t} &= \lambda_{m,t-1} N_{t-1}^{-\gamma_q} e^{\sigma_{\ln q}^2} \\
 N_{t+1} &= N_t \lambda_{m,t} (1 - \phi_t) N_t^{-\gamma}
 \end{aligned}$$

This model can generate a variety of dynamics, and in the absence of density dependence, i.e., when $\gamma = \gamma_\phi = \gamma_q = 0$, the model reduces to an asexually reproducing organisms with a hyper-geometrically increasing abundance and a geometrically declining competitive quality. In the realistic situations with density dependence the dynamics is generally cyclic, and

Table 24.1 The parameter estimates of the logistic equation with selection (eqn 24.2, assuming $\gamma_q = \gamma$) for the nine insect species in Table 3.1. N_G is the geometric mean in the data, and r the correlation coefficient of the regression (eqn 24.39) that was used to estimate the parameters.

Species	N_G	P	r	γ	$\sigma_{\ln q}^2$	N^{**}	λ_m^{**}
Tussock moth	4.1	9.0	0.96	0.52	0.77	4.39	2.16
Fall webworm	10.2	7.0	0.82	0.69	1.54	9.83	4.82
Larch budmoth	2.2	9.3	0.96	0.53	0.51	2.62	1.66
Larch cone fly	945	4.5	0.32	1.68	11.5	950	97500
Nun moth	2600	4.1	0.55	1.28	10.0	2630	22900
Pine looper moth	0.66	4.9	0.62	1.30	-0.48	0.69	0.62
Pine beetle	700	7.7	0.94	0.42	2.67	605	14.4
Spruce budworm	1.5	21	0.94	0.28	0.16	1.76	1.17
Wasp spp.	10.2	2.2	-0.57	2.89	6.59	9.78	730

Fig. 24.3a illustrates a stable cycle with a phase portrait between the sex ratio and the population density. This theoretical cycle coincides with the results of the empirical study of Naumov et al. (1969). They found that the percentage of males in small rodents increases when the densities are high, while females predominated during the years with low densities. This resemblance suggests that selection by density dependent competitive interactions plays a role in the cyclic dynamics of small rodents.

24.5 Forest insects

Forest insects, especially Lepidoptera, are known for their very pronounced population cycles, and from Chapter 4 on predator-prey interactions we recall that at least some of these cycles tend to lack a firm coupling to the dynamics of other species. Then, as these insects have non-overlapping generations, like the model in eqn 24.2, it is interesting whether the dynamics of forest insects resemble the dynamics we predict from selection due to intra-specific competitive interactions.

Recall that in Chapter 3 we analysed the data of nine species of forest insects, and that we concluded that their dynamics was not explained by over-compensatory density regulation. As these insects were chosen because they showed cyclic dynamics we can assume that the cycles are stable so that the parameters of eqn 24.2 for those species are easily estimated by the regression in the third appendix to this chapter. For this case, where $\gamma_q = \gamma$, I have listed the estimated parameters in Table 24.1. Before we compare the dynamics between the estimated models and the insect species,

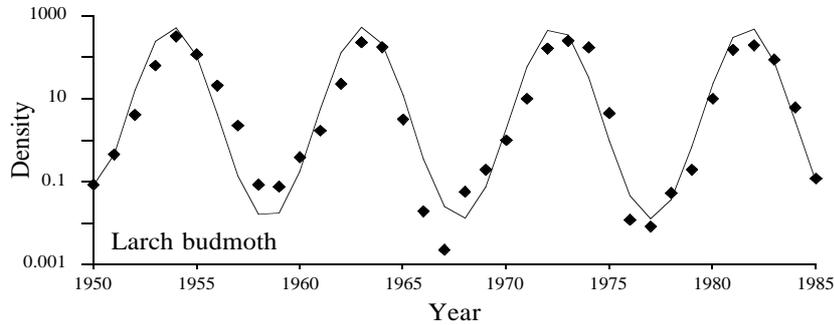


Fig. 24.4 The curve is a projection of eqn 24.2 and the diamonds the yearly densities of the larch budmoth in the Upper Engadine valley from 1950 to 1985. The projection is initialised from the density estimates of 1949, 1950, and 1951. The γ and $\sigma_{\ln q}^2$ parameters of eqn 24.2 is adjusted from those given in Table 24.1 to $\gamma = 0.44$ & $\sigma_{\ln q}^2 = 0.42$ so that the equilibrium is the same and the period resembles the observed period. Data from Baltensweiler and Fischlin (1988).

we may notice that the estimated evolutionary equilibria (N^{**}) resemble the geometric mean of the densities in the time series (N_G), a result that suggests that the assumption of linearity on logarithmic scale is reasonable.

As it has already been mentioned, the period in the dynamics of eqn 24.2 is given by the γ parameter, and in Fig. 24.3b this relationship is shown by the curve that is obtained by numerical simulations. In the same figure the diamonds represent the nine species, where the γ parameter is estimated by the regression in the third appendix and the period is the average period in the data. The resemblance between the theoretically deduced and the observed periods is reasonable, suggesting that selection by density dependent competitive interactions play a role in the cyclic dynamics of forest insects.

Let me now focus on the larch budmoth that lacks a firm predator-prey interaction although it has the best documented cycle among forest insects (Berryman, 1996). The data on one population of this species are shown by the diamonds in Fig. 24.4, where the curve represents a projection of the model in eqn 24.2. Again, the resemblance between theory and data is reasonable, suggesting that selection by density dependent competitive interactions play a role in the cyclic dynamics of the larch budmoth.

24.6 Population cycle allometry

The essential time lag that generates the cyclic dynamics of eqn 24.2 is one generation, and this is because evolution by natural selection is operating with a built-in delay of one generation. In eqn 24.2 this time lag is implicit

in the sense that the model is developed for organisms with non-overlapping generations. This implies that the period of eqn 24.2 is given in generations and, thus, for a given γ the period in astronomical time is proportional to the lifespan (T).

From the allometric deduction in Chapter 13, we have that $T \propto w^{1/2d}$, where w is the body mass and d is the number of dimensions in which the organism forages. Hence, the period of the population cycle is expected to have a similar scaling, and this means that it is expected to be proportional to the positive 1/4 power of body mass, at least for the terrestrial organisms that forage in two spatial dimensions. On empirical grounds this scaling is known as the Calder hypothesis, and it is shown in Fig. 24.5a where it is confirmed for terrestrial homiotherms (Calder, 1983, 1984; Peters, 1983; Peterson et al., 1984; Krukoni and Shaffer, 1991).

24.7 Implications of neutral stability

The cycles generated by selection due to density dependent competitive interactions are neutrally stable in the sense that their amplitude is given by initial conditions even though the period is fixed and given by the particular value of γ . The alternative to a neutrally stable cycle is a limit cycle where both the amplitude and the period is fixed and given by parameter values. In this latter case it is the whole cycle of a given population that is fixed being the global attractor toward which all initial perturbations of the population equilibrium converges.

It is relatively easy to determine whether the population cycles of natural species are neutrally stable, or limit, cycles. If the cycles are limit cycles we expect only a small and relatively similar degree of variation in both the period and the amplitude of the cycle. If, on the other hand, the cycles are neutrally stable, as it is expected by eqn 24.2, then, for a given species the amplitude is expected to be much more variable than the period. This prediction coincides with Batzli's (1981) conclusion stating that for small mammals in any given place the period of the cycle is more regular than its amplitude, and the same is true for the cycle of the Canadian lynx (Moran 1953; Bulmer, 1974).

Also, if the cycle is neutrally stable, then the amplitude is determined by the magnitude of the environmental perturbations. This implies that the amplitude will increase on a cline from a stable to a fluctuating environment. One such cline is from temperate areas toward the polar regions where an increased frequency and severity of unfavourable climatic conditions occur. It is thus not surprising that population cycles generally vanish toward the south while they become progressively more pronounced with progressively larger amplitudes toward Arctic regions (Howell, 1923; Hansson and Henttonen, 1985; Hansson, 1987; Akçakaya, 1992).

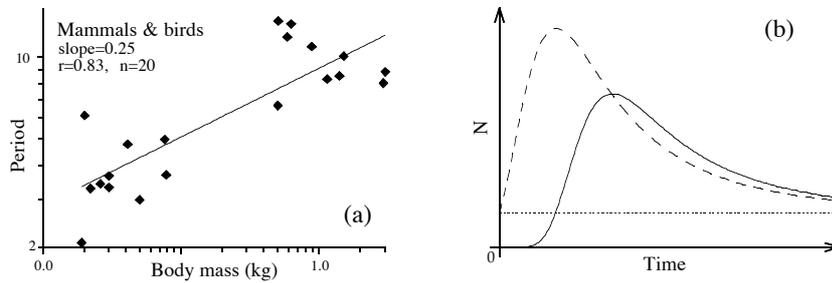


Fig. 24.5 (a) The period of population cycles against body mass for terrestrial homiotherms on double logarithmic scale. Data from Peterson et al. (1984), obtained through Krukoniis and Shaffer (1991). One outlier removed. (b) A projection of eqn 24.2 to illustrate the dynamics of an introduced species with strongly damped dynamics ($\gamma = 0.07$ & $\gamma_a = 0.01\gamma$). At time zero the species is introduced at a density that is 10^{-7} times smaller than the density at the evolutionary equilibrium. The solid curve is the population density, the dashed curve the population equilibrium, and the dotted line the evolutionary equilibrium.

24.8 Extreme perturbations

As the amplitude of the population cycle depends on the magnitude of the environmental perturbation it follows that a special type of dynamics can arise from extreme perturbations of relatively stable equilibria. To illustrate this, consider a species that invades or is introduced into a new area from which it previously has been absent. If such invasions are successful there is often an extreme perturbation with an extraordinarily low population density. This implies that interference competition is almost absent and that there is strong selection for an increase in the intrinsic growth rate. In Fig. 24.5b I have simulated such an introduction of a species with strongly damped dynamics. From the figure it is apparent that, following the introduction, there is a lack phase with only a marginal increase before the evolutionary increase in the intrinsic growth rate causes the population to explode to extreme densities. These densities are associated with high levels of interference where antagonistic behaviour selects energy from the demographic traits to competitive quality, and this causes the population to decline slowly to the normal and stable density given by the evolutionary equilibrium. This type of dynamics with a single, or a few, over-shoots are often observed when humans release exotic species into new areas (e.g., Elton 1927; Adam et al., 1993).

In the extreme case, the dynamics may be so unstable that a severe

environmental perturbation might “kick” a species into an orbit that will lead of extinction. If such cases occur, the extinction event will follow after an extreme peak density. I can recall one such extinction; the passenger pigeon in America. Indeed the densities of this species were extreme prior to the extinction: “a single town in Michigan marketed 15,840,000 birds in two years, while another town sold 11,880,000 in forty days” Elton (1927:105). As the passenger pigeon went extinct during the settlements in America it might be that it experienced a major change in the habitat, and that this change “kicked” this species into an orbit of extinction.

Not only might such extinctions be caused directly by a large environmental perturbation, but maybe even more likely, they might be the result of an environmentally induced transition from a damped to a repelling or exploding cycle. This could, e.g., be the case if an environmental alternation induces changes in the parameters that determine the stability of the population cycle. As it is indicated by eqn 24.4, such an environmental transition to a repelling cycle may occur if the mode of density regulation is shifted toward exploitative competition, or if the biased access to resource at a given density is increased.

Even if the cycle is repelling the environment may be sufficiently heterogeneous to ensure that at least a few individuals will survive during the lows of the population cycle. In such cases a species with a repelling cycle will be characterised by many local extinctions after which it will spread from “epidemic centers” during the increasing phase of its cycle. This pattern of expiration and colonisation is observed for many of the forest insects with cyclic dynamics (see Berryman, 1988; Watt et al., 1990).

24.9 Appendix

24.9.1 Population equation with selection

In this appendix I develop and analyse a population dynamic equation for a species with non-overlapping generations and selection due to density dependent competitive interactions. This is done in three steps. First, I describe the evolutionary changes in the maximal growth rate in order to develop the population equation. Second, I show that the evolutionary equilibrium is the equilibrium of the population equation, and third I analyse the stability of the equilibrium and the dynamic behaviour of the equation.

Evolutionary changes in the growth rate

Let me use the logistic equation eqn 24.1 that we developed in Chapter 3 for a species with non-overlapping generations, and let us assume that the

exploitation efficiency (α) is stable so that the evolutionarily determined population equilibrium resembles the evolutionary equilibrium. For this equilibrium we have previously shown that

$$\begin{aligned}\lambda &= \lambda_m^{**} N^{**-\gamma} = 1 & (24.7) \\ N^{**} &= \sqrt[\gamma]{\lambda_m^{**}} \\ N^{**} &= e^{1/\gamma\iota\psi} \\ \lambda_m^{**} &= e^{\gamma/\gamma\iota\psi}\end{aligned}$$

To predict the evolutionary change in λ_m let

$$\lambda_m = \rho/q \quad (24.8)$$

where ρ is a positive constant and q is the energetic and competitive quality of an average individual. Then the discrete growth rate of the population in the t th generation is

$$\lambda_t = \rho N_t^{-\gamma}/q_t \quad (24.9)$$

while it for the i th variant is

$$\begin{aligned}\lambda_{i,t} &= \rho N_t^{-\gamma_i}/q_{t,i} & (24.10) \\ &= \lambda_t(\rho N_t^{-\gamma_i}/q_{t,i})/(\rho N_t^{-\gamma}/q_t) \\ &= \lambda_t(q_t/q_{i,t})N_t^{\gamma\iota\psi(\ln q_{i,t} - \ln q_t)}\end{aligned}$$

with $\gamma_i = \gamma_\alpha + \mu_i\gamma_\iota$ and $\Delta\mu_i = \psi(\ln q - \ln q_i)$. This equation can be rearranged to

$$\lambda_{i,t} = \lambda_t e^{(\gamma_\iota\psi \ln N_t - 1)(\ln q_{i,t} - \ln q_t)} \quad (24.11)$$

If we differentiate this equation with respect to $\ln q_{i,t}$ and let $q_{i,t} \rightarrow q_t$ we obtain the per generation selection gradient on the average of $\ln q$, i.e.

$$\lim_{q_{i,t} \rightarrow q_t} \partial\lambda_{i,t}/\partial \ln q_{i,t} = \lambda_t(\gamma_\iota\psi \ln N_t - 1) \quad (24.12)$$

When this selection gradient is combined with Robertson (1968) the per generation change in $\ln q$ is

$$\begin{aligned}\Delta \ln q_t &= \frac{\sigma_{\ln q}^2}{\lambda_t} \lim_{q_{i,t} \rightarrow q_t} \frac{\partial\lambda_{i,t}}{\partial \ln q_{i,t}} & (24.13) \\ &= \sigma_{\ln q}^2(\gamma_\iota\psi \ln N_t - 1)\end{aligned}$$

Then, from eqn 24.13 and $\ln q_t = \ln q_{t-1} + \Delta \ln q_{t-1}$ the average quality at time t is

$$q_t = q_{t-1} N_{t-1}^{\gamma_\iota\psi\sigma_{\ln q}^2} e^{-\sigma_{\ln q}^2} \quad (24.14)$$

and from eqns 24.8 and 24.14, the maximal growth rate in generation t is

$$\begin{aligned}\lambda_{m,t} &= \rho N_{t-1}^{-\gamma_i \psi \sigma_{\ln q}^2} e^{\sigma_{\ln q}^2} / q_{t-1} \\ &= \lambda_{m,t-1} N_{t-1}^{-\gamma_q} e^{\sigma_{\ln q}^2}\end{aligned}\quad (24.15)$$

with $\gamma_q = \gamma_i \psi \sigma_{\ln q}^2$. Then, from eqns 24.1 and 24.15 the population dynamic equation with density dependent selection is

$$\begin{aligned}\lambda_{m,t} &= \lambda_{m,t-1} N_{t-1}^{-\gamma_q} e^{\sigma_{\ln q}^2} \\ N_{t+1} &= N_t \lambda_{m,t} N_t^{-\gamma}\end{aligned}\quad (24.16)$$

This equation has three parameters (γ , γ_q , & $\sigma_{\ln q}^2$) and two initial conditions (N_t & $\lambda_{m,t}$).

The evolutionary equilibrium

If the evolutionary equilibrium eqn 24.7 is the equilibrium of the population equation eqn 24.16, then it must satisfy the conditions $\lambda_{m,t+1} = \lambda_{m,t}$ and $N_{t+1} = N_t$ for all t . From eqn 24.16 the condition $N_{t+1} = N_t$ is satisfied when

$$\lambda_{m,t} N_t^{-\gamma} = 1 \quad (24.17)$$

If we insert λ_m^{**} and N^{**} from eqn 24.7 into eqn 24.17 we have

$$e^{\gamma/\gamma_i \psi} e^{-\gamma/\gamma_i \psi} = 1 \quad (24.18)$$

which reduces to $1 = 1$. Hence, for the evolutionary equilibrium the condition $N_{t+1} = N_t$ is satisfied for all t when the condition $\lambda_{m,t+1} = \lambda_{m,t}$ is satisfied also. From eqn 24.16 we see that the condition $\lambda_{m,t+1} = \lambda_{m,t}$ is satisfied when

$$N_t^{-\gamma_q} e^{\sigma_{\ln q}^2} = 1 \quad (24.19)$$

If we insert N^{**} from eqn 24.7 into eqn 24.19 we have

$$e^{-\gamma_q/\gamma_i \psi} e^{\sigma_{\ln q}^2} = 1 \quad (24.20)$$

which for $\gamma_q = \gamma_i \psi \sigma_{\ln q}^2$ is equivalent to

$$e^{-\gamma_i \psi \sigma_{\ln q}^2 / \psi \gamma_i} e^{\sigma_{\ln q}^2} = 1 \quad (24.21)$$

which reduces to $1 = 1$. Hence, the condition $\lambda_{m,t+1} = \lambda_{m,t}$ is satisfied and the evolutionarily determined population equilibrium (eqn 24.7) is the equilibrium of the population dynamic model eqn 24.16.

Stability and dynamic behaviour

In this subsection I analyse the stability of the evolutionary equilibrium, and the dynamics following from a perturbation of that equilibrium. To do this let $r = \ln \lambda_m$ and $n = \ln N$. Then, take the natural logarithm to eqn 24.16 and obtain $r_t = G(r_{t-1}, n_{t-1})$ and $n_{t+1} = F(r_t, n_t)$ where the two functions G and F are defined as

$$\begin{aligned} G &= r_{t-1} - \gamma_q n_{t-1} + \sigma_{\ln q}^2 \\ F &= r_t + (1 - \gamma)n_t \end{aligned} \quad (24.22)$$

If we now follow the procedure in Bulmer (1994) the stability of the evolutionary equilibrium is given by the eigenvalues of the Jacobian matrix

$$\begin{bmatrix} \partial G/\partial r & \partial G/\partial n \\ \partial F/\partial r & \partial F/\partial n \end{bmatrix} \quad (24.23)$$

where the equilibrium is unstable when the absolute value of the dominant eigenvalue is larger than or equal to one. For the two-dimensional matrix eqn 24.23 the eigenvalues are

$$(T \pm \sqrt{T^2 - 4D})/2 \quad (24.24)$$

where D is the determinant and T the trace of that matrix. To determine this D and T we have that $D = (\partial G/\partial r)(\partial F/\partial n) - (\partial F/\partial r)(\partial G/\partial n)$, that $T = \partial G/\partial r + \partial F/\partial n$, and that $\partial G/\partial r = 1$, $\partial G/\partial n = -\gamma_q$, $\partial F/\partial r = 1$, and $\partial F/\partial n = 1 - \gamma$ for eqn 24.22 for all r and n including the equilibrium. Hence, we find that

$$\begin{aligned} D &= 1 - \gamma + \gamma_q \\ T &= 2 - \gamma \end{aligned} \quad (24.25)$$

so that the eigenvalues are

$$(2 - \gamma \pm \sqrt{\gamma^2 - 4\gamma_q})/2 \quad (24.26)$$

for eqn 24.16 on logarithmic scale. This means that the eigenvalues are real when $\gamma^2 \geq 4\gamma_q$ and complex when $\gamma^2 < 4\gamma_q$. Then, for the situation $\gamma^2 \geq 4\gamma_q$ with real eigenvalues we find that the equilibrium is unstable when $\gamma > 4$ or when $\gamma_q \leq 2\gamma - 4$ and $2 \leq \gamma \leq 4$ while it is otherwise stable. For the situation $\gamma^2 < 4\gamma_q$ with complex eigenvalues the absolute value of the eigenvalues is $\sqrt{1 - \gamma + \gamma_q}$ so that the evolutionary equilibrium is unstable when $\gamma \leq \gamma_q$.

When genetic variation is absent, i.e., when $\sigma_{\ln q}^2 = 0$, the instability criterion associated with the real eigenvalues merges into the instability

criterion of the logistic equation without selection, i.e., it merges into the instability criterion of the equation we analysed in Chapter 3. This implies that the instability criterion $\gamma_q \leq 2\gamma - 4$ reduces to $\gamma \geq 2$ because $\gamma_q = 0$ at the limit where genetic variation is absent. In this sense the instability connected with real eigenvalues is generated by over-compensatory density regulation. This is in contrast to the instability criterion associated with the complex eigenvalues, where the instability arises from the evolutionary modulation of the growth rate. This latter type of instability is impossible when genetic variation is absent, and this is because the absence of genetic variation imposes the constraint $\gamma_q = 0$ so that the inequality $\gamma \leq \gamma_q$ is false because γ is positive for natural situations.

Let us now turn to the dynamics that follow from a perturbation of the evolutionary equilibrium of eqn 24.16. For the situation where genetic variation is absent, i.e., when $\gamma_q = 0$, the dynamics are described in Chapter 3, and this is because eqn 24.16 reduces to eqn 3.15 at the limit with no genetic variation. When instead genetic variation is present the long-term dynamics, i.e., for $t \rightarrow \infty$, is given by the eigenvalues of the Jacobian matrix eqn 24.23. If these eigenvalues are real and their absolute values are smaller than one, then the perturbation will decline geometrically toward the equilibrium, while it will increase geometrically if the absolute value of the dominant eigenvalue is larger than one. Hence, for eqn 24.16 the perturbation will increase geometrically when $\gamma > 4$ or when $\gamma_q \leq 2\gamma - 4$ and $2 \leq \gamma \leq 4$, while the dynamics are damped otherwise. Moreover, if the sign of the dominant eigenvalue is negative the long-term dynamics is oscillatory with a period of two generations, while it is monotonic if the value is positive. Hence, the long-term dynamics of eqn 24.16 are oscillatory when $\gamma > 2$ while it is monotonic for $\gamma < 2$. Numerical simulations though do show that the short-term dynamics following from a perturbation of the equilibrium often is oscillatory even when $\gamma < 2$.

If instead the eigenvalues are complex they will induce a cycle with a highly variable period. To describe this cycle, let a complex eigenvalue be given by the polar coordinates in the Argand diagram, i.e., let x be its absolute value and θ its argument. Then the period of the cycle is $P = 2\pi/\theta$ while the amplitude is proportional both to the initial perturbation and to x . This amplitude is stable if $x = 1$, it declines geometrically if $x < 1$, and it increases geometrically if $x > 1$. That is, $x < 1$ corresponds to a damped cycle, $x = 1$ to a stable, and $x > 1$ to a repelling cycle. For eqn 24.16 we find that

$$\begin{aligned} x &= \sqrt{1 - \gamma + \gamma_q} \\ P &= 2\pi / \arctan \left| \frac{\sqrt{4\gamma_q - \gamma^2}}{2 - \gamma} \right| \end{aligned} \quad (24.27)$$

This implies that the cycle caused by the evolutionary modulation of the growth rate is damped when $\gamma < \gamma_q$, stable when $\gamma = \gamma_q$, and repelling when $\gamma > \gamma_q$. Also, when the cycle is stable the period has a minimum of 4 generations at $\gamma = 2$, while the period increases monotonically to infinity as γ goes toward either 0 or 4. For $\gamma < 0$ the system is not biologically defined, and at the limit of, and beyond, $\gamma = 4$ the eigenvalues are real with the dominant eigenvalue being smaller than -1 so that an initial perturbation increases geometrically and oscillatorily without limits.

Now, let the period of the population cycle be defined as the time between two neighbouring events, where the population abundance crosses the equilibrium in the same direction. Then numerical simulations show that the population period has a close resemblance to the period defined by eqn 24.27 when $\gamma_q \approx \gamma < 2$. When instead $\gamma_q \approx \gamma \geq 2$ and $\gamma_q \approx \gamma \rightarrow 4$ the population period declines monotonically from 4 to 2 generations, while the increasing period given by eqn 24.27 has a close resemblance to a period that turns up in the amplitude of the population cycle. The period of the population cycle is shown in Fig. 24.3b together with data from forest insects. An example of the period in the amplitude is shown in Fig. 24.1.

24.9.2 Population equation with selection on the sex ratio

In the population model eqn 24.16 it is not taken into account that a species might reproduce sexually. In such instances we may also want to model the evolutionary changes in the sex ratio, and this is because the sex ratio is expected to be modulated also by selection due to density dependent competitive interactions.

If we assume a stable α and non-overlapping generations, then the evolutionary equilibrium of a sexual mobile organism is

$$\begin{aligned} \lambda &= \lambda_m^{**}(1 - \phi^{**})N^{**-\gamma} = 1 & (24.28) \\ N^{**} &= \sqrt[\gamma]{\lambda_m^{**}(1 - \phi^{**})} \\ N^{**} &= e^{1/\gamma \cdot \psi} \\ \lambda_m^{**} &= e^{\gamma/\gamma \cdot \psi} / (1 - \phi^{**}) \\ \phi^{**} &= 1/2 \end{aligned}$$

where λ_m^{**} represents maximal net reproduction per female and ϕ^{**} is the proportion of males. From eqn 24.28 we find that the projection of the population density for a single time step is

$$N_{t+1} = N_t \lambda_{m,t} (1 - \phi_t) N_t^{-\gamma} \quad (24.29)$$

To predict the population trajectory from this equation we also need to predict the evolutionary trajectory in both maximal reproduction and the

proportion of males. To predict these trajectories let $\lambda_m = \rho/q$ so that the discrete growth rate of an average individual is

$$\lambda_t = \rho(1 - \phi_t)N_t^{-\gamma}/q_t \quad (24.30)$$

We can then follow the procedure for eqns 24.10 to 24.15 and find that maximal reproduction at time t is given by eqn 24.15. If we now assume independence between q and the proportion of males (ϕ) we can follow the same procedure to determine ϕ at time t . When we do this we find, in resemblance with eqn 24.10, that the discrete growth rate of the i th variant is

$$\begin{aligned} \lambda_{i,t} &= \rho(1 - \phi_{t,i})N_t^{-\gamma_i}/q_t & (24.31) \\ &= \lambda_t[\rho(1 - \phi_{t,i})N_t^{-\gamma_i}/q_t]/[\rho(1 - \phi_t)N_t^{-\gamma}/q_t] \\ &= \lambda_t[(1 - \phi_{t,i})/(1 - \phi_t)]N_t^{\gamma_i\psi(\ln \phi_{i,t} - \ln \phi_t)} \end{aligned}$$

with $\gamma_i = \gamma_\alpha + \mu_i\gamma_\iota$ and $\Delta\mu_i = \psi(\ln \phi - \ln \phi_i)$. If we differentiate this equation with respect to $\ln \phi_{i,t}$ and let $\phi_{i,t} \rightarrow \phi_t$ we obtain the per generation selection gradient on the population average of $\ln \phi$

$$\lim_{\phi_{i,t} \rightarrow \phi_t} \partial \lambda_{i,t} / \partial \ln \phi_{i,t} = \lambda_t[\gamma_\iota \psi \ln N_t - \phi_t / (1 - \phi_t)] \quad (24.32)$$

and thus the per generation change in $\ln \phi$

$$\begin{aligned} \Delta \ln \phi_t &= \frac{\sigma_{\ln \phi}^2}{\lambda_t} \lim_{\phi_{i,t} \rightarrow \phi_t} \frac{\partial \lambda_{i,t}}{\partial \ln \phi_{i,t}} & (24.33) \\ &= \sigma_{\ln \phi}^2 [\gamma_\iota \psi \ln N_t - \phi_t / (1 - \phi_t)] \end{aligned}$$

Then, from eqn 24.33 and $\ln \phi_t = \ln \phi_{t-1} + \Delta \ln \phi_{t-1}$ the proportion of males at time t is

$$\phi_t = \phi_{t-1} N_{t-1}^{\gamma_\phi} e^{-\phi_t \sigma_{\ln \phi}^2 / (1 - \phi_t)} \quad (24.34)$$

with $\gamma_\phi = \gamma_\iota \psi \sigma_{\ln \phi}^2$. The population equation for a sexually reproducing mobile organisms is then given by the eqns 24.15, 24.29, and 24.34, which is summarised by the following population equation

$$\begin{aligned} \phi_t &= \phi_{t-1} N_{t-1}^{\gamma_\phi} e^{-\phi_t \sigma_{\ln \phi}^2 / (1 - \phi_t)} & (24.35) \\ \lambda_{m,t} &= \lambda_{m,t-1} N_{t-1}^{-\gamma_q} e^{\sigma_{\ln q}^2} \\ N_{t+1} &= N_t \lambda_{m,t} (1 - \phi_t) N_t^{-\gamma} \end{aligned}$$

This equation has five parameters ($\gamma, \gamma_\phi, \gamma_q, \sigma_{\ln \phi}^2$, & $\sigma_{\ln q}^2$) and three initial conditions (N_t, ϕ_t & $\lambda_{m,t}$).

24.9.3 Parameter estimation

Given the assumption of a stable cycle, i.e., given $\gamma_q = \gamma$, in this appendix I describe how to estimate the parameters of the population model eqn 24.2. By eqn 24.2 we have assumed that the effects of density regulation vanishes at the density $N = 1$. This is generally not the case when densities are scaled arbitrarily, and therefore we need an extra constant (c) so that $\lambda_t = \lambda_{m,t}cN^{-\gamma}$. Then, to estimate $\lambda_{m,t}$ and c from time series of densities we need an independent estimate of either $\lambda_{m,t}$ or c . As such estimates are generally not available let me simplify and exchange $\lambda_{m,t}$ in eqn 24.2 with $\lambda_{c,t} = \lambda_{m,t}c$. For this definition of density regulation it can be shown that the term $e^{\sigma_{\ln q}^2}$ in eqn 24.2 is exchanged with e^δ when $\delta = \gamma_q \ln c/\gamma$. Then, from the assumptions $\gamma_q = \gamma$ and $\lambda_t = \lambda_{c,t}N_t^{-\gamma}$ the population model of eqn 24.2 is equivalent to

$$\begin{aligned}\lambda_{c,t} &= \lambda_{c,t-1}N_{t-1}^{-\gamma}e^\delta \\ N_{t+1} &= N_t\lambda_{c,t}N_t^{-\gamma}\end{aligned}\quad (24.36)$$

To estimate the two parameters γ and δ , from the bottom equation of eqn 24.36, we have

$$\lambda_{c,t} = N_{t+1}N_t^{\gamma-1}\quad (24.37)$$

If we then insert $\lambda_{c,t-1}$ and $\lambda_{c,t}$ as given by eqn 24.37 into the top equation of eqn 24.36 we obtain

$$N_{t+1}N_t^{\gamma-1} = N_tN_{t-1}^{\gamma-1-\gamma}e^\delta\quad (24.38)$$

This equation can be rearranged so that we can estimate γ and δ by the linear regression

$$\ln N_{t+1} + \ln N_{t-1} = (2 - \gamma) \ln N_t + \delta\quad (24.39)$$

From $\lambda_c^{**} = \lambda_c^{**}N^{**-\gamma}e^\delta$ (eqn 24.36) the density at the equilibrium is $N^{**} = e^{\delta/\gamma}$, which combined with $N^{**} = N^{**}\lambda_c^{**}N^{**-\gamma}$ (eqn 24.36) gives us the estimate $\lambda_c^{**} = e^\delta$.

Epilogue

Chapter 25

Summary

EMBEDDED in most of the traditional models in theoretical evolutionary ecology there is the assumption that we can explain natural patterns by disregarding the effects of interactions among competing individuals. Throughout this book I have tested this classical hypothesis against the contrasting hypothesis that many of the large scale patterns in nature are explained explicitly by the density dependent competitive interactions that exist among the individuals in populations. In this final chapter I will summarise my study and conclude upon the results obtained.

The major part of this book was on evolutionary biology. In relation to this subject the test on the importance of competitive interactions was conducted as a test between the classical theory of evolution, which disregards competitive interactions (reviewed by Roff, 1992; Stearns, 1992; Charnov, 1993; Bulmer, 1994; Charlesworth, 1994), and the theory of Malthusian relativity I developed from selection by density dependent competitive interactions. The test between these two theories of evolution was based on the concept of all-dimensional optima, in abbreviation, ADO (Section 9.2.2). This concept is the hypothesis that the predictions, or optima, of a successful theory of evolution need to be evolutionarily stable in all the phenotypic dimensions included explicitly in the theory. That is to say that when we explain the evolution of certain traits, then we need to make no assumptions with respect to the evolutionary determinants of the other traits.

In this chapter the tests between the proposed and the classical theory of evolution are summarised in the three sections 25.2 to 25.4. In Section 25.2 I deal with the evolution of basic traits like body mass, population abundance, and rate of reproduction, and I also consider the evolution of the body mass allometries. Then in Section 25.3 I focus on the evolutionary steady state that incorporates directionality into the evolutionary process. Having then explained that organisms increase in size, I deal in Section 25.4 with the

evolutionary transitions associated with the transition from a negligible to a relatively large organism. In Section 25.5 I change the topic in order to summarise the population dynamic consequences of selection by density dependent competitive interactions. But first, I deal with the effects of competitive interactions in the traditional framework of theoretical ecology that disregards evolutionary changes.

25.1 Traditional theoretical ecology

The traditional approach to theoretical ecology is based on the population dynamic theory that arose from Malthus' (1798) notion that all living organisms have the potential to increase geometrically in numbers. Malthus inferred that such increase is transient, occurring only up to the limit of the resource where lack of food will prevent further increase. In 1838 this regulation was formulated into the logistic equation by Verhulst to provide a description of the growth curve of a single population. In the early 1920s Lotka and Volterra extended the theory to incorporate interactions among competing species and between predators and their prey. By that time the classical theory had matured to the framework that provides the background for the description of ecology today.

This theory is generally based on the assumption that it is the exploitation of the resource that is essential for a proper description of the patterns in natural communities. In the most simple case, with a single population on a single resource, this idea is present in Lack's hypothesis that the population is resource regulated so that the equilibrium density is the carrying capacity, which is the number of individuals the resource can sustain. This idea is based on the simplifying assumption that the foraging individuals do not encounter one another. In the realistic case, the individuals do encounter one another, the population is both resource and interference regulated, and the equilibrium density is always lower than the carrying capacity.

25.1.1 Food chains

Lack's hypothesis of resource regulation by exploitative competition is contained implicitly in the predator-prey equations developed by Lotka (1925) and Volterra (1926) in order to explain the cyclic dynamics in natural populations. As it was pointed out by Arditi and Ginzburg (1989) the equilibria of these equations fail to explain the proportional relation that exists between the primary production and the biomass at all trophic levels in natural food chains. Instead, the equations predict a proportional response only at some trophic levels, whereas the biomass at other levels is inde-

pendent or even inversely related to the primary production. To avoid this paradox Arditi and Ginzburg introduced ratio-dependent models where the functional response of the predator depends upon the predator-prey ratio instead of being dependent only on the prey density as it is the case in the traditional equations. The rationale behind the ratio-dependent response is that it results from the fact that the time scale of foraging often is faster than the time scale of population dynamics, or that it results from interactions among predators that divide up the resource before they consume it. In Chapter 5 we saw that the proportional response is obtained also if we add the regulatory component of intra-population interference competition to the original Lotka-Volterra equations.

25.1.2 Competitive coexistence

The hypothesis of resource regulation also underlies the traditional studies on the assembly of competing species. This field has been dominated by the Volterra-Gause principle that states that competitive exclusion inevitably takes place among species that occupy similar niches. Since it is virtually impossible to show that two species have truly overlapping niches the Volterra-Gause principle is basically impossible to falsify empirically. This is because a falsification would require that we found two coexisting species with completely overlapping niches. However, as it was shown by Schoener (1976) and as it is illustrated in Chapter 6, it is easy to show theoretically that the Volterra-Gause principle represents only a special case, because it is based on an implicit assumption of pure exploitative competition. When the regulatory effects of intra-specific interference is added to the equations it is no longer a problem to have many species that coexist in a single niche. Furthermore, it turns out that Hutchinson's rule, that two strongly competing species can coexist if their weight ratio is larger than two, follows from the regularities by which inter-specific interference partitions the resource among competing species.

25.2 Evolution of basic traits

Basic traits are traits that all organisms have, and these traits can be traced back to the self-replicating molecules at the origin of life. Among other things, basic traits include self-replication (reproduction), the population abundance, the size (body mass), and the energy metabolised by an individual. The evolutionary modulation of the quantitative values of these traits can generally be seen as the evolution of the balance by which the energy available per individual is allocated between numerical replication and traits like body mass and metabolic rate. In the following three subsec-

tions I describe this balance in respectively the dimensions of body mass, population abundance, and rate of reproduction. Then, in the fourth subsection I consider the body mass allometries that describe the across-species relationships between the different traits.

25.2.1 Body mass

According to the classical explanation of Roff (1981) a large body mass evolves from the within-species proportionality that often exists between reproduction and body mass. As shown in Chapter 10, this hypothesis is falsified by evidence because it is based on an assumption of constant relative fitnesses: Constant relative fitnesses imply that the fitness profile (i.e., the relative fitness of the i 'th variant in a population as a function of the mass of that variant) is similar to the selection profile (i.e., the relative fitness of the average variant as a function of the mass of that variant). This implies that if the classical hypothesis holds, then the selection pressure on body mass is given by the across-species relation between the intrinsic growth rate and body mass. Across natural species with large body masses the intrinsic growth rate is proportional to the $-1/4$ th power of body mass (Fenchel, 1974). Thus, the classical framework predicts the evolution of negligible body masses, and this is paradoxical because this prediction is estimated from the occurrence of organisms with large body masses. Due to this paradox the relative fitnesses are relative and not constant, as it is assumed in the classical theory.

From this result it is evident that the two major assumptions behind the classical theory on the evolution of body mass need some modifications before we can construct a model that is consistent with the patterns that exist both within and across species. First of all, the proportional relation between reproduction and body mass is inappropriate as an assumption, because the relation has evolved by natural selection, and thus it does not represent an evolutionary constraint. If instead, the proportional relation is exchanged with an inverse relation, then the relation between reproduction and body mass will reflect the evolutionary constraint defined by the physical constraint that the energy allocated to reproduction can be used only once. Then, by turning to the hypothesis of evolution by density dependent competitive interactions the second assumption of constant relative fitnesses is also avoided. As shown in Chapter 10, in this framework large body masses can evolve by the density dependence in the number of competitive interactions and the relation that large-bodied individuals can dominate small-bodied individuals during competitive encounters.

The body masses evolving by density dependent competitive interactions behave in resemblance to general observations. For example, they are inversely related to the rate of mortality, as it is observed in guppies (Reznick

et al., 1996). And they are positively related to the production in the resource, which is in agreement with the observations made by McNab (1971) and Geist (1986) in relation to Bergmann's rule. This rule was originally meant to cause thermal homeostasis, but now it is seen to reflect the duration of the productivity pulse in the resource. The predicted body masses also behave in resemblance to the island rule (Foster, 1964), which describes that large animals generally are smaller on islands than on mainlands, while small animals tend to be larger.

25.2.2 Population limitation

The classical approach with Fisherian selection and exploitative competition generally fails to explain the evolution of a population equilibrium that is limited so that the resource is not over-exploited. This is because Fisher's fundamental theorem of natural selection predicts that the equilibrium continues to increase while the resource becomes progressively more over-exploited.

This straightforward prediction is not in resemblance to empirical evidence where terrestrial herbivores often eat less than 10% of the edible plant biomass. To avoid this paradox Hairston et al. (1960) developed, and Hairston and Hairston (1993) elaborated, the influential argument that it is the inter-specific predator-prey interactions that maintain nature in a state of balance, where over-exploitation does not occur. Here, the essential argument is that the predators will eat the herbivores and with fewer herbivores the herb is no longer over-exploited. However, this argument holds only in the absence of evolutionary changes. With Fisherian selection the density of at least one species in the food chain is expected to increase in an uncontrolled manner. It is thus very understandable that Wynne-Edwards (1962, 1986, 1993) continues to argue that group selection is a necessity in order to explain the balance of nature, i.e., in order to explain the existence of balanced population densities.

Group selection is, though, not a necessity. As shown in Chapter 11, this is because balanced population equilibria are predicted by the type of individual selection that arises from density dependent competitive interactions. These equilibria are balanced in the sense that the equilibrium densities are the densities where the density dependent bias in the within-population access to resource is balanced against the intrinsic trade-off between the demographic traits and competitive quality. This theoretically deduced balance includes the well established empirical relations that the densities at all trophic levels are proportional to the primary production, that poikilotherm vertebrates are ≈ 30 times more abundant than homoiotherm vertebrates, that the population density is proportional to the $-3/4$ th power of body mass, and that the exploitation of the resource is body mass

invariant.

These balanced equilibria behave distinctively different from the equilibria in the classical theory of population dynamics. Population limitation in the classical theory is based on the concept that a limiting factor is any factor that when changed causes a change in either reproduction, survival, and/or density regulation. Among other things, this implies that an environmentally induced change in reproduction and/or survival is converted directly into a change in the population equilibrium. For example, if we begin to remove individuals at a constant rate from the population the equilibrium density will decline. This is not the case under the hypothesis of density dependent competitive interactions where, as a first approximation, the evolutionarily determined population equilibrium is invariant with respect to environmentally induced changes in reproduction and/or survival. This means that if we begin to remove individuals at a constant rate from the population, then the equilibrium density will remain the same. The reason for this is that, after the evolutionary equilibration has occurred, the energy taken from the population by the removal of individuals is taken from the body mass and the metabolic rate instead of being taken from the equilibrium density, as held in classical theory.

25.2.3 Reproduction

The classical theory on the evolution of the reproductive rate is based on Lack's clutch size, which is the idea that the optimal rate of reproduction is the rate where most offspring survive. According to this hypothesis the reproductive rate is a derived trait that evolves from a more fundamental trade-off that exists between reproduction and offspring, or parent, survival. This hypothesis has been confirmed on a local scale where demographic models are fitted to the empirical evidence within species.

In Chapter 12 it was shown that Lack's clutch size fails as a theory on the evolution of a limited rate of reproduction. This is because the fundamental theorem of natural selection predicts a continuous increase in the intrinsic growth rate, and because the evident solution to this problem is a continuous increase in the rate of reproduction. More specifically, the continuous increase in reproduction occurs because the trade-offs between reproduction and survival are part of the phenotype so that selection operates not only on the rate of reproduction, but also upon the trade-offs. When this is the case, it follows from the classical framework that the absolute values of the trade-offs continue to decline generating a continuous increase in the reproductive rate.

This classical prediction of unlimited reproduction is in contrast to the prediction from the hypothesis of selection by density dependent competitive interactions. In this latter situation a limited rate of reproduction will evolve

because fitness no longer is equivalent to the intrinsic growth rate. Instead, when the level of interference competition is high, a large variant with low intrinsic reproduction may easily out-reproduce a small variant with high intrinsic reproduction, and this is because the large variant has access to more resource than the small variant. If instead, the level of interference is low, then the two variants have access to approximately the same amount of resource and it is the small variant with the highest intrinsic growth rate that will out-reproduce the large variant.

In general, selection by density dependent competitive interactions implies that reproduction is balanced in accordance with the ecological constraints that determine the within-species bias in the access to resource. This balance implies that poikilotherm vertebrates reproduce at a higher rate than homoiotherm vertebrates, that the intrinsic rate of reproduction is positively related to the primary production, that it is negatively related to the size of the resource quanta for which the individuals compete, and that it is balanced against the extrinsic level of mortality. These predictions are generally confirmed by empirical evidence.

When a limited growth rate has evolved by density dependent competitive interactions it follows that the selection procedure underlying Lack's clutch size is consistent with the new prediction. This consistency is intuitively sound because, irrespective of the evolutionary optimum to the intrinsic growth rate, we always expect that selection will optimise reproduction against mortality. Only in relation to the original proposal of Lack, that it is the reproductive rate that evolves from the trade-off between reproduction and survival, the causality is changed around so that it is the absolute value of the trade-off that evolves from the optimal growth rate given by ecological constraints. Also, the new prediction is evolutionarily stable while Lack's prediction is evolutionarily unstable.

25.2.4 Body mass allometries

During this century the major life-histories across natural species have been empirically established as allometric relations describing the various traits as power functions of body mass. These relations are now reviewed in books by Calder (1984), Peters (1983) and Reiss (1989), and it is generally agreed that the rounded exponents of the allometric relations are $3/4$ for the metabolic rate, $1/4$ for lifespan, $1/4$ for the level of social behaviour, $-1/4$ for the intrinsic growth rate, $-3/4$ for the population density, 1 for the home-range area, 0 for the resource consumed by a population, and 0 for maximal lifetime reproduction. While a number of explanations have been proposed for these relations it is generally agreed that no single one is convincing.

However, in Chapter 13 I showed that the empirically estimated exponents are explained by the ecological constraints associated with a foraging process that is optimised by natural selection. The essential constraints are the number of competitive encounters per individual and foraging self-inhibition, i.e., the degree to which the exploitation of an individual inhibits the foraging of that individual. The regulation of the foraging efficiency by these two processes are counteractive so that there is an intermediate optimum from which it is possible to deduce the allometric exponents. According to this deduction the 1/4th and 3/4th exponents apply to organisms foraging in two dimensions, whereas the expected exponents are 1/6 and 5/6 among organisms that forage in three dimensions. The two-dimensional exponents are confirmed by terrestrial organisms while the three-dimensional exponents generally apply to pelagic organisms.

25.3 Evolutionary steady state

These results suggest that selection by density dependent competitive interactions is essential, but they do not suggest that evolution is directional. This conclusion depends on the assumption that the per individual exploitation efficiency is fixed, an assumption that does not hold because an individual will generally do better if it has more resource, or energy, available.

In Chapter 14 I showed that when selection operates on the exploitation efficiency, then the evolutionary process in a stable environment will equilibrate at an evolutionary steady state where both the exploitation efficiency and the body mass increase exponentially, while the other phenotypic traits evolve in concordance with the exponents of the body mass allometries. Among other things, this implies that Fisherian fitness continues to decline, a result that is diametrically opposite to Fisher's fundamental theorem that predicts a steady increase.

On empirical grounds the continuous increase in body mass is confirmed by Cope's law, which suggests that the majority of phylogenetic lineages tend to increase in size during their history in the fossil record. Another observation, explained by the steady state, is the within-population proportionality between reproduction and body mass. You will recall that it was this relation that was used together with Fisherian selection to explain the evolution of a large body mass in the classical theory. Under the hypothesis of selection by competitive interactions, it is instead the relation between reproduction and body mass that evolves from the evolutionary setting of the selection gradient on body mass, a gradient that is given by the ecological constraints at the evolutionary steady state. As both the ability to

metabolise energy into competitive interactions and the ability to perform complex behavioural interactions are crucial components for the outcome of a competitive encounter both of these traits are expected to increase exponentially at the steady state, just like the body mass. In relation to this general increase in competitive quality it is possible to estimate the evolutionary changes in the metabolic rate by examining the evolutionary trajectory in body mass. This is possible because the shape of the trajectory in body mass reflects the time scaling between the evolutionary process and astronomical time, and because this time scaling depends upon the generation time that is inversely related to the metabolic rate. In Chapter 15 I applied this framework to MacFadden's data on fossil horses and found that, when the metabolic rate is corrected for the allometric relation to body mass, then the metabolic rate of horses is ≈ 10 times larger today than it was 57 million years ago. This estimate implies also that the 25 kg horse 57 million years ago had a lifespan of ≈ 90 years, given that the lifespan of the 500 kg horse of today is ≈ 20 years.

The prediction of a steady increase in competitive quality depends upon the assumptions that the abiotic and biotic factors extrinsic to the population are constant, and that there is no immediate limit to the exploitation efficiency. When instead the exploitation efficiency reaches its upper limit, the body mass will cease to increase, and this levelling-off will occur at lower masses on smaller resources. Hence, we may expect a variety of body masses among natural organisms. Also, if the resource evolves to be less edible, or if competitive interactions with competitively superior individuals from other species causes a decline in the access to resource, then the increase in body mass might even reverse to a decline. Inter-specific interactions and environmental variation may then explain why there always seems to be some organisms that decline in size.

The increase in competitive quality also depends on a sufficiently high influx of energy to the overall biological system. As shown in Chapter 16, when the influx of energy begins to decline there can be a mass extinction that will eliminate predominantly the larger species, while the remaining species will tend to dwarf in size. From the fossil record we know that widespread dwarfing tends to co-occur with events of mass extinction, and that mass extinctions tend to eliminate predominantly the larger species. Finally, if the influx of energy continues to decline and the physical conditions remain suitable for life then most, if not all, species are expected to dwarf until they reach the molecular level. So, in conclusion, the evolutionary process is expected to be reversible with the particular direction being defined by environmental conditions.

25.4 Evolution of derived traits

From the predictions of the evolutionary steady state we expect that a self-replicating molecule in a stable environment with a large resource automatically evolves toward a large-bodied organism with life-histories in accordance with known body mass allometries. Then, from empirical evidence we know that the transition from a negligible to a large body mass is associated with the evolution of a set of derived traits that, among other things, include senescence, males, sexual reproduction, and a diploid genome. The crucial thing is then to show theoretically that the transition to a large body mass is associated with the evolutionary emergence of these derived traits.

As the derived traits tend to be associated with an increase in competitive quality their evolution can be explained by selection due to density dependent competitive interactions. Under this hypothesis, the fitness gained through interference competition is proportional to the number of competitive encounters per individual per unit time and, thus, the evolutionary development of the derived traits is positively related to the level of intra-population interference. This implies that it is essential to show theoretically that the level of intra-population interference associated with a negligible body mass is so low that the derived traits do not evolve in these organisms, and to show theoretically that the level of interference associated with a large body mass is exactly so high that the derived traits will evolve to the level of expression known from large-bodied organisms. In the following three subsections I will summarise how this is possible in relation to respectively the evolution of senescence, the evolution of males and sex ratios, and the evolution of sexual reproduction and a diploid or haplodiploid genome. In the fourth subsection I will turn to the evolution of eusocial colonies and their associated traits, and summarise how these systems can evolve from a body mass that is evolutionarily constrained relative to the exploitation efficiency.

25.4.1 Senescence and soma

To explain the evolution of senescence and soma in mobile organisms we need to explain both why senescence and soma are absent in negligibly sized organisms, like prokaryotes and viruses, and why they are present in large-bodied organisms, like the higher eukaryotes.

The theory on the evolution of senescence reaches back to Weismann (1889), and it was mainly Medawar (1952) and Williams (1957) that developed the traditional view that senescence is a derived trait that evolves from a somatic tissue from which no part is passed on in either sexual or asexual reproduction. This hypothesis coincides with the presence of senescence in the large-bodied eukaryotes that have a soma, and its absence in

the small-bodied prokaryotes that have no soma. This theory does, though, fail on a global scale because it does not explain why the higher eukaryotes have evolved a soma, when prokaryotes have not.

The alternative hypothesis, I developed in Chapter 17, is that the soma is the trait through which senescence is expressed. This hypothesis is based on the notion that senescence cannot evolve without a clear separation between a senescing somatic tissue and a non-senescing reproductive tissue. Senescence and soma can then evolve when it is beneficial to allocate energy from self-repair in the non-reproductive tissue to other processes that can enhance early reproduction and/or survival.

Under the hypothesis of selection by density dependent competitive interactions, the energy that is not used for self-repair can be used to enhance the competitive quality. When this is the case it turns out that the theoretically deduced level of interference in negligibly sized organisms is so low that senescence and a soma are unlikely to evolve, and that the deduced level of interference in large-bodied organisms is so high that senescence and a soma evolve.

25.4.2 Males and sex ratios

To explain the evolution of males and sex ratios we need, among other things, to explain why males are absent in negligibly sized organisms like prokaryotes and viruses, and why there often is one male per female in large-bodied mobile organisms like the mobile higher eukaryotes.

It was Fisher (1930) who developed the classical hypothesis that an even sex ratio is a derived trait that evolves because an average individual of the rare sex is expected to leave more offspring than an average individual of the common sex. This result depends on the assumptions of sexual reproduction between males and females, a diploid or a haplodiploid genome, and random mating at the population level. Hamilton (1967) extended the theory, and showed that the sex ratio will be female biased if mating is local, in the sense that the sons of a single, or related, female/s compete/s with one another for a limited number of matings.

This classical, or Fisherian, sex ratio theory makes sense on a local scale, but it fails on an evolutionary scale because the cost to sexual reproduction implies that the classical sex ratio equilibria are evolutionarily unstable in the assumptions that underlie those equilibria. Traditionally, it has been assumed that sexual reproduction is two-fold costly, but this holds only in a few special cases. More generally, sexual reproduction between a male and a female is four-fold costly, and this is because it is both the male individual and the meiotic division of the genome into haploid gametes that are two-fold costly. The problems with the Fisherian sex ratios then are, (*i*) that the two-fold cost of the male selects for an extreme degree of local mating

and a sex ratio that is biased to the limit where males do not exist, and (ii) that the two-fold cost of meiosis selects for a haploid genome, asexual reproduction, and the absence of males, at least if hermaphrodites are not allowed. If instead, hermaphrodites are allowed, then there may be selection for hermaphrodites, and this is because they have the potential to bypass the four-fold cost of sex. This is possible because hermaphrodites contain no male individuals and because they contribute to reproduction through both the male and the female function. But, in either case, the Fisherian sex ratio theory fails to explain the evolutionary maintenance of sex ratios in mobile organisms with males and females.

If we disregard the classical sex ratio theory, then, under the hypothesis of evolution by density dependent competitive interactions, males can easily evolve by individual selection despite their two-fold cost. This is because the male can invest both energy and time in competitive quality, instead of using it on physiological self-replication as it is done by the female. In this sense the male individual gains fitness not only through sexual reproduction, but also from interference competition where he can prevent that the asexual variant has access to the resource. Hereby the sexual variant can out-reproduce the asexual variant, despite the fact that the sexual variant has the lowest intrinsic growth rate.

In this scenario the number of males per female will depend upon the level of interference in the population. If the level of interference is high, it pays to invest in the competitive quality of the male so that it is optimal to be in relatively large groups containing many males and only a few females. If, on the other hand, the level of interference is low, competitive quality tends to be wasted so that it pays to invest in the female component of numerical replication, generating a female biased sex ratio. The essential point is then to show that the theoretically deduced level of interference will explain the sex ratio patterns that exist among natural species.

From the theoretical deductions in relation to the evolution of body mass, it turns out that the level of interference in negligibly sized organisms, like prokaryotes and viruses, is so low that males cannot evolve. This is in contrast to the deduced level of interference at the evolutionary equilibrium in large-bodied organisms that is exactly so high that the optimal number of males per female is one. Finally, at the evolutionary steady state the deduced number of males per female is approximately two. As shown in Chapter 22, this latter prediction depends upon the assumption that the interfering individuals are males. If, on the other hand, they are allowed also to be the offspring of the female, then the optimal solution is cooperative breeding between a pair and a single offspring. This form of reproduction is widespread in birds and mammals.

Unlike the Fisherian sex ratios, the sex ratios that are predicted by

competitive interactions do not depend upon the presence of sexual reproduction and, therefore, they are also independent of the ploidy level of the genome and of the degree of local mating. However, if we assume sexual reproduction and a diploid, or a haplodiploid, genome, then the two sex ratio theories can be integrated with one another, and it can be shown that the original equilibrium relations will evolve, including the relationship between the degree of female bias and the degree of local mating. The essential differences between the new and the original form of this prediction is (*i*) that the original prediction is evolutionarily unstable while the new prediction is evolutionarily stable, and (*ii*) that the evolutionary causality is turned upside down. This suggests that it is the degree of local mating that evolves from the sex ratio, and not the other way around as it originally was proposed by Hamilton.

In many cases males differ from females, e.g., in the size or by the presence of male characters that may include physiological ornaments or a colourful plumage. Traditionally, these differences between females and males have been explained by sexual selection where females choose the large or the more extravagant males. These differences between the two sexes may, however, evolve also by interference competition for other resources than females. According to this latter hypothesis there is a general trend where males tend to be larger than females in species with male biased sex ratios, while the reverse is expected when the sex ratio is female biased. This trend is widespread among natural species where birds and mammals tend to have male biased sex ratios and males that are larger than females, while the reverse often is the case in lower organisms, like insects and other invertebrates.

With respect to male characters there is a difference in the expected pattern depending upon whether they evolve by interference competition for resources or by female choice and sexual selection. If they evolve by interference competition for resources, then, as it was shown in Chapter 20, we expect that they will evolve in many instances when there is overlap between the breeding site and the area in which the organism forages, but only in few instances when there is no overlap between the breeding site and the foraging area. This is in contrast to the situation with female choice where we expect no such differentiation. Bird species generally conform to the situation with interference competition for resources suggesting that female choice for extravagant males is a secondary trait that evolves because it is these males that can supply the females with the largest amount of resource. In other words, it seems that it is female choice and sexual selection that evolve from the presence of male characters, and not the other way around as it traditionally has been thought ever since Darwin introduced the concept of sexual selection.

According to the hypothesis of density dependent competitive interactions this change is not the only major change in the role played by sexual selection. According to the original proposal by Darwin, and all subsequent treatments, sexual selection is seen as the link explaining the non-sexual differences between females and males from the assumption of sexual reproduction. However, as illustrated in the following section, under the hypothesis of competitive interactions sexual selection is the link that explains the evolution of sexual reproduction from the functional differences between males and females. Once again, it seems that the dependent and the independent variables need to be changed around.

25.4.3 Sexual reproduction and ploidy level

To explain the evolution of sexual reproduction we need to explain why sexual reproduction is absent in negligibly sized organisms, like prokaryotes and viruses, why it generally exists among hermaphrodites in sessile organisms, and why it generally exists between males and females in large-bodied mobile organisms. Also, we need to explain why the optimal solution to sexual reproduction is a diploid, or haplodiploid, genome with a meiotic division into haploid gametes.

The problem with sexual reproduction is that the two-fold cost of meiosis selects for a haploid genome and asexual reproduction when sexual reproduction occurs between males and females. In contrast, there is no real paradox associated with the maintenance of sexual reproduction in hermaphrodites, and this is because these species have the potential to avoid both the two-fold cost of the male and the two-fold cost of meiosis. In other words, the difficult task is to explain why we have sexual reproduction between males and females in the large-bodied mobile organisms.

Although at least twenty different theories have been proposed to account for the evolution of sexual reproduction, the case with sexual reproduction in the large-bodied mobile organisms has remained probably the largest paradox in evolutionary biology. A common feature to nearly all the proposed models is that they are based on the idea that genetic diversity is beneficial to the organism per se and, thus, a diploid organism with sexual reproduction is expected to be more fit than a haploid organism with no sex, simply because the diploid organism is genetically more diverse. These hypotheses will, however, not explain the evolution of the male individual at the cost of hermaphrodites, and this is because, even in the presence of genetic variation, the male is still four-fold costly when he is compared with the hermaphrodite. Also, the traditional models will generally not explain why the natural solution to the problem of genetic diversity should be exactly a diploid, or haplodiploid, genome with haploid gametes. In Chapter 21 I was aiming at a more complete solution that could explain

the general patterns associated with sexual reproduction, i.e., a model that could explain why sexual reproduction in natural species takes the particular form with a diploid, or a haplodiploid, genome with haploid gametes, why it generally occurs only in large-bodied organisms and, finally, why in these organisms it generally occurs between males and females when the organism is mobile, while it generally occurs among hermaphrodites when the organism is sessile.

In order to obtain this goal I focused on the hypothesis of evolution by density dependent competitive interactions, where the existence of males and females has been explained independently of the existence of sexual reproduction. In this scenario sexual reproduction can evolve because a pair that contains a female and a male has a higher competitive quality than a lonely female and a pair that contains two females. This implies that it is favourable for the female to attract an unrelated male, and she can do this by sexual reproduction where she can increase the male's fitness by transferring some fraction of his genome to her offspring.

Generally, we expect that the females will choose the males that are competitively superior to other males, and that the competitively superior males will dominate the inferior males in order to choose the females that transfer the largest fraction of their genome on to the offspring. Under this set-up it turns out that, if the body mass is negligible, then the theoretically deduced level of interference is so low that sexual reproduction will not evolve. If instead, the body mass is large and in evolutionary equilibrium, then the deduced level of interference is exactly so high that the female's genome will replicate at the fastest rate when the offspring receives half of its genes from the mother and the other half from a single father. An obvious solution to this mode of sexual inheritance is the form known from diploid organisms, where the diploid offspring receives one set of chromosomes from its mother and an other set from its father, or the form known from haplodiploid organisms, where the diploid female transfers haploid gametes to both daughters and sons, while the haploid male transfers a haploid gamete only to daughters.

The model behind these results is valid only in mobile organisms because it depends upon the implicit assumption that the male can help the female during competitive encounters. This is not the case in sessile organisms where the sessility implies that if a male competes for a female, then he competes also against her. This implies that sessile males are energetically costly, and that the evolutionary optimum to sexual reproduction is hermaphroditic individuals. Moreover, as males generally are absent in sessile organisms there is no male choice to prevent that asexual reproduction and self-fertilization evolve in order to enhance the intrinsic growth rate of the hermaphrodite. Not surprisingly, among sexual organisms, asexual

reproduction and self-fertilization are common in sessile organisms while these traits are rare to absent among mobile organisms.

25.4.4 Eusocial colonies

A eusocial colony is characterised by a large worker caste, and eusociality is known mainly from social insects, where it occurs in one form in ants and bees, and in another form in termites. These two forms of eusociality are characterised by two different sets of co-occurring traits: In ants and bees the queen forms the colony on her own after she has mated with a sexual male, the genome is haplodiploid, the workers are the sterile daughters of the queen, and there are typically three queens per sexual male. In termites the queen forms the colony together with the king, the genome is diploid, the workers are the sterile daughters and sons of the queen, and there typically is one king per queen. Hence, when we explain the evolution of eusociality, it is essential to explain also the evolution of these two patterns of co-occurring traits.

According to the traditional view introduced by Hamilton (1964, 1972), eusocial colonies evolve by kin selection where the sterile workers gain inclusive fitness through the sexual reproduction of their queen. This hypothesis depends on the assumption that the workers are constrained to be the offspring of the queen. If instead, the workers were allowed also be the sexual partners of the queen, then the degree of kin selection is reflected by the proportion of the workers that are offspring workers. In this case eusocial colonies can evolve independently of kin selection when there are no offspring workers. Hence, in order to explain that kin selection is operating in eusocial species, it is essential to relax the assumption of kin selection and show that selection in eusocial species favours offspring workers at the cost of sexual workers.

From Chapter 22 we have that when the assumption of kin selection is relaxed and the workers are allowed to be both offspring and sexual males, then it turns out that the optimal worker caste contains only offspring workers. This is because it is at this limit that each gene in each individual in the colony has the highest probability of being copied to a sexual offspring of the queen. Hence, it seems that it is kin selection that evolves from, and/or is maintained by, eusociality, and not the other way around as it was originally proposed by Hamilton.

As kin selection is not the ultimate cause of the evolution of eusocial colonies they must evolve by other means. According to the hypothesis of selection by density dependent competitive interactions eusocial colonies will evolve when the body mass is upward constrained and there is plenty of resource. This is because, then the energy that cannot be allocated to body mass is selected into reproduction instead, and this results in increased

interference and selection for large groups that can dominate small groups. These groups reach their evolutionary optimum when they contain many interfering individuals (workers), and only a few or a single reproductive females (queens). This result may explain why eusociality is relatively common in insects, where the ecto-skeleton might set an upper limit to the body mass, and why eusociality is almost absent in vertebrates, where the body mass seems to be evolutionarily more flexible.

In relation to the sex ratio in the sexuals produced by a eusocial colony it is likely that it is controlled by the workers because they are numerically far superior to the queen. It was Trivers and Hare (1976) who combined this observation with the genomic selection of the Fisherian sex ratio theory in order to develop the classical explanation of the evolution of the sex ratio in eusocial species. According to this explanation the sex ratio is the derived trait that evolves from the relatedness between the workers and the sexual offspring produced by the queen. This implies that the predicted sex ratio of three queens per sexual male in ants and bees depends upon the assumptions that the genome is haplodiploid and that the workers are the daughters of the queen. Likewise, the prediction of an even sex ratio in termites is the result of the diploid genome.

If we disregard this classical causality on the evolution of sex ratios, then it is possible to relax the traditional assumptions and let both the ploidy level of the genome and the sex ratio in the workers evolve by selection, just like the sex ratio in the sexuals. This is possible because individual selection on the sex ratio in the sexuals dominates over the Fisherian form of genomic selection. This implies that if, as it is the case in ants and bees, the queen establishes the colony on her own, then the two-fold cost of the male selects for a female biased sex ratio. When this prediction is combined with an extended model on Fisherian sex ratios, it turns out that the individual selection on the sex ratio in the sexuals imposes selection on both the ploidy level of the genome and the sex ratio in the worker caste. As shown in Chapter 22, the evolutionary equilibrium of this system is a haplodiploid genome and a worker caste containing only female offspring, i.e., an equilibrium that coincides with the pattern in ants and bees. In this system, the sex ratio in the sexuals is three queens per male at the limit where the force of individual selection is completely diluted by the spread of genes in the population. If instead, the population is substructured and individual selection operates more independently of genomic selection, then the sex ratio in the sexuals will be more female biased.

In the case of termites the situation is different because the sexual male stays with the queen. This implies a constraint of pair formation that induces individual selection for an even sex ratio in the sexuals. When this result is coupled to genomic selection on the ploidy level of the genome, and

to genomic selection on the sex ratio in the worker caste, then it turns out that the evolutionary equilibrium is a diploid genome and a worker caste containing both female and male offspring. In this latter case the sex ratio in the sexuals is always even because an even sex ratio is the equilibrium at the level of both genomic and individual selection.

25.5 Evolutionary population dynamics

The predictions in the previous sections are based on the assumption that the population is in dynamic equilibrium. When this is not the case the population shows dynamic behaviour, and this tends to be inherently cyclic in natural populations. This is unlike the classical theory where population dynamics is inherently non-cyclic, unless there is some sort of delayed regulation caused by over-compensation or by interactions with other species.

Although over-compensatory dynamics have been found in animals as diverse as ungulates and insects, it is now generally agreed that over-compensation cannot explain the general tendency for cyclic dynamics. This is because the single species models, which can over-compensate due to the action of direct density dependence, generally do not explain the observed dynamics, and because the models that may explain the dynamics are based on delayed density regulation, lacking a firm mechanism that can explain why the delay occurs.

At first, these limitations to the single species models in the classical theory suggests that most population cycles are caused by predator-prey interactions in their broadest definition, which includes herb-herbivore and host-parasitoid interactions. However, this hypothesis has always been associated with the problem that many populations apparently continue to cycle even in the absence of the essential interactions. This appears to be the case with *Daphnia*, lemmings, and snow-shoe hares, and with the larch budmoth that has the best documented cycle in forest insects. Due to these limitations it remains questionable whether the mechanisms in the classical theory of population dynamics are sufficient in order to explain the periodic dynamics in natural species.

One obvious limitation to the mechanisms in the classical theory of population dynamics is that they are based on the assumption of no evolutionary changes. I relaxed this assumption in Chapter 24, where I developed a logistic equation based on selection by density dependent competitive interactions. The major result was that the density dependent selection extended the classical theory so that the dynamics of a single species become inherently cyclic, taking a period that coincides with the periods in the forest insects with cyclic dynamics. In other words, we do no longer have to incorporate interactions with other species in order to explain the

cyclic dynamics that occur in natural populations.

According to the action of selection by density dependent competitive interactions the cyclic changes in the population densities are associated with a cycle in the phenotype that, among other things, includes the intrinsic growth rate, the body mass, and the sex ratio. Cyclic changes in body mass have been observed among the *Daphnia* and lemmings that show cyclic dynamics, and evidence suggests that the phenotypic cycle in lemmings also includes the sex ratio and the intrinsic growth rate.

25.6 Conclusion

We have now gone through the major dimensions of the organism, and in nearly all instances obtained the result that the equilibria or predictions in the classical theory are evolutionarily unstable in their phenotypic assumptions. Moreover, we have established a new theory based on a single ADO model that contains nearly all the classical equilibria, only in a new form. The major differences between the new and the classical versions of these equilibria are: (i) that the new versions are evolutionarily stable because they generally do not depend upon phenotypic components that are evolutionarily unstable, and (ii) that the propagation of the selection pressure through the population generally is in opposite directions in the two theories. In the classical theory the propagation is “inside-out” in the sense that it generally is the intrinsic constraints of the genomic and phenotypic systems that determine the selection pressure at higher phenotypic levels. In the proposed theory the propagation is “outside-in” in the sense that it generally is the selection pressure of interference competition that can explain the evolutionary optimum of the major phenotypic dimensions. It is this overall evolutionary optimum together with its behaviour in relation to its position on the major evolutionary trajectory that is the general theory of evolution I propose.

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Symbols

S	Trait	Relations
w	Body mass	
B	Metabolic rate per unit mass	
ω	Senescence parameter	
T	Lifespan	$T = \omega/B$
q	Energetic and competitive quality	$q = wTB$
θ	Proportion of females	
ϕ	Proportion of males	$\phi = 1 - \theta$
ϕ/θ	Sex ratio	
H	Home-range	
N	Population density	$N^* = \sqrt[3]{\lambda_m}$
ι	Intra-population interference, also I	$I = e^\iota$
γ_α	Density regulation by exploitation	$-\gamma_\alpha \ln N$
γ_ι	Density dependence in interference	$\iota = \gamma_\iota \ln N$
μ	Regulation by interference	$-\mu\iota, \cdot g(I)$
γ	Total density regulation	$\gamma = \gamma_\alpha + \mu\gamma_\iota$
f	Regulation by foraging self-inhibition	$\cdot f$
α	Exploitation efficiency	
κ	Realised foraging efficiency	$\kappa = fg\alpha$
r_e	Rate of increase in the resource	
γ_e	Density regulation in the resource	
E	Resource density	$E^* = (r_e - \kappa N^*)/\gamma_e$
ϵ	Energy used on reproduction	$\epsilon = \kappa E^*$
R	Lifetime reproduction	$R = T\epsilon/q = \epsilon/wB$
p	Survival scalar	
R_0	Net lifetime reproduction	$R_0 = pR$
λ	Discrete Malthusian parameter	$\lambda = R_0$
r	Intrinsic Malthusian parameter	$r^* = \ln(p\alpha E^*/wB)$
x	Extrinsic Malthusian parameter	$x^* = \ln(p\kappa E^*/wB)$
d	Spatial dimensionality of behaviour	
$\sigma_{\ln q}^2$	Genetic variance in \ln quality	
ψ	Within population slope in μ	
Q	Fitness, or resource, quanta	$\psi \propto Q$

The most important symbols (S). The superscript star indicates the population equilibrium, and the subscript m maximal values

During evolution on Earth there has been a directional change where self-replicating molecules have evolved into large multi-cellular organisms with high metabolic rates and complex behavioural interactions. Associated with this increase there has been a transition from an asomatic, non-senescing, haploid, and asexually reproducing organism to a somatic, senescing, diploid, and sexually reproducing organism. In a few cases there has been an additional transition to eusocial communities.

Despite this directional increase in complexity evolutionists since Darwin have agreed that evolution by natural selection is historical and non-directional. However, in this book the author develops a new theory suggesting for the first time that evolution by natural selection is inherently directional in the mentioned directions. The theory developed also provides the first explanation for the body mass allometries that describe the major life-histories across mobile organisms, and it provides a single-species mechanism for the population cycles that have fascinated ecologists for decades.

The proposed theory is based on selection by density dependent competitive interactions and on a new concept of evolutionary stability in all phenotypic dimensions. This concept invalidates the classical theories because their equilibria are evolutionarily unstable in their phenotypic assumptions. Based on a new causality the author reestablishes the classical equilibria, but in an improved form that is evolutionarily stable.

This book will interest all biologists wishing to understand evolutionary and population dynamic processes, and written as a text book it is useful to both professionals and students who are familiar with basic calculus.



“Reading these claims ... I formed the belief that the book was either crazy, or very important. ... So, what do I make of the book? It is certainly not ‘crazy’”

-- John Maynard Smith

“a fascinating protracted argument which may well be correct.”

-- Anonymous reviewer