

## 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.

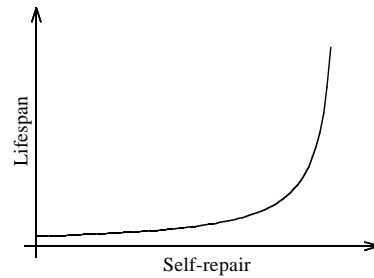
#### 17.3.1 Trade-off between self-repair and senescence

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  $\omega$  is a constant of senescence that will change with changes in the degree of self-repair.

In order to model the selective modification of  $\omega$  let us define  $\omega$  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  $\omega$  will take a minimum value which we can denote  $\omega_{min}$ . 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  $\omega$  and  $s$  can be defined as  $\omega = \zeta/s$ , where  $\zeta$  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  $\omega$ . As in this chapter we are modelling the evolutionary changes in  $\omega$  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  $\epsilon$

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  $\lambda_i^* = p\zeta\epsilon e^{\psi\iota^{*e}(\ln s_i - \ln s)} / ws_i B$ , where  $\iota^{*e}$  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  $\lambda_i^*$  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 ( $\epsilon$ ) during the transition from uniformly distributed individuals ( $z = 0$ ) to completely formed groups ( $z = 1$ ) can be approximated by the linear function

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

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  $\epsilon_i$  is given by eqn 18.3 and  $\iota^{*e}$  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

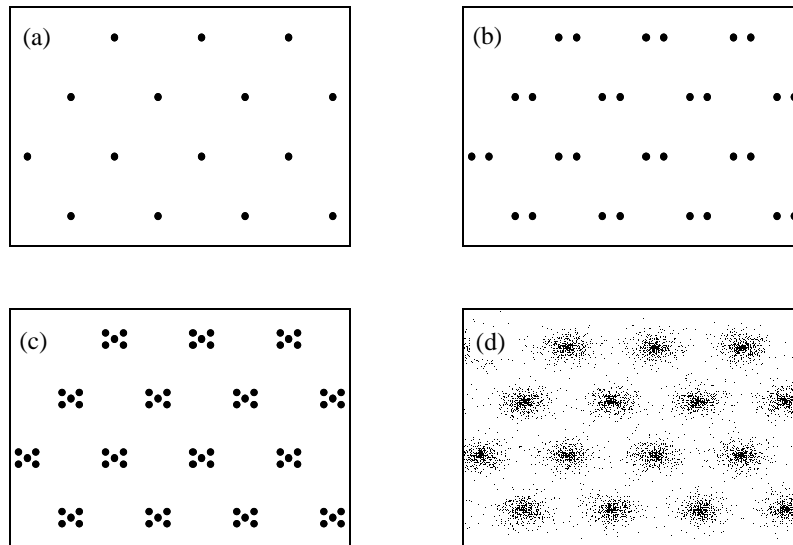
$$n^{**} = v(\psi\iota^{*e} + 1) \quad (18.8)$$

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  $\iota^{*e}$ , 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 ( $\iota^{*e}$ ) 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

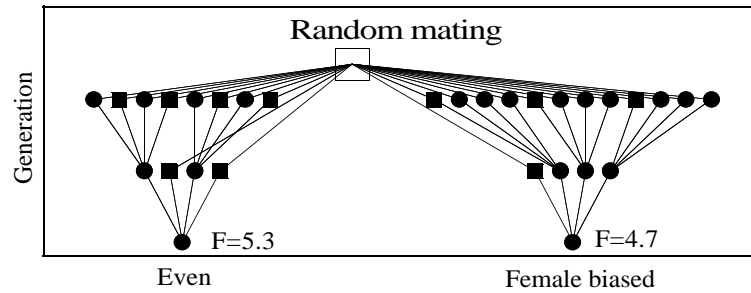
# Fisherian sex ratios

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  $\theta$  be the proportion of females in the population, let  $\phi = 1 - \theta$  be the proportion of males, let  $\theta_i$  and  $\phi_i$  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  $\phi_i$  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 ( $w_\theta$ ) and a male ( $w_\phi$ ) offspring may differ, and that a female has a given amount of resource ( $\epsilon$ ) 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

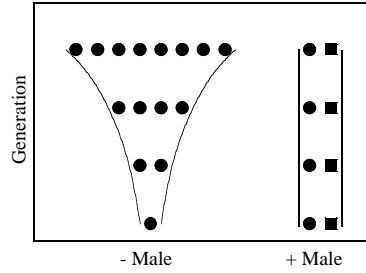
and Levin, 1988; Bulmer, 1994). But this holds only at the limit where the investment sex ratio is completely female biased, and males and females still exist. Such a strong bias can arise only when the numerical sex ratio is extremely female biased, or when the male individual is extremely small compared 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



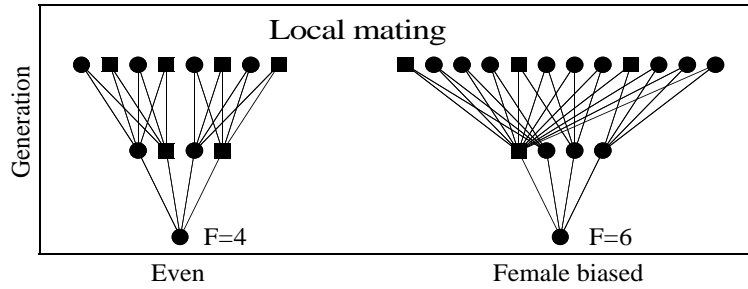
**Fig. 19.2** An illustration of the two-fold cost of the male when females (circles) produce two offspring per lifetime. The lineage to the left reproduces asexually without males, whereas the sexual lineage to the right requires males (squares) for reproduction.

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

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

Hence, the selection gradient on the mating structure is negative

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

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  $\epsilon_i$  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  $\iota^{*e}$  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  $\epsilon_i$ , 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  $\epsilon_i$ . This means that we can model the discrete growth rate of the  $i$ th variant as  $\lambda_i^* = p\epsilon e^{\psi\iota^{*e}(\ln \epsilon_{i,i} - \ln \epsilon_i)} / wB$ , where  $\epsilon$  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

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

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

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

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  $\lim_{\epsilon_{i,i} \rightarrow \epsilon} \partial x_i^* / \partial \epsilon_{i,i} \geq 0$  for  $\epsilon_i = \dot{\epsilon}$ . By exchanging  $\epsilon_i$  in eqn 20.3 with  $\dot{\epsilon}$ , setting  $\lim_{\epsilon_{i,i} \rightarrow \epsilon} \partial x_i^* / \partial \epsilon_{i,i} \geq 0$ , 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

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

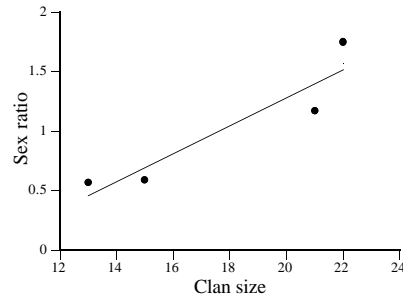
where the  $\ln \phi$  terms in the exponent indicate that it is the group containing most males that will dominate the other groups during the competitive encounters. The selection gradient on the proportion of males is then

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

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

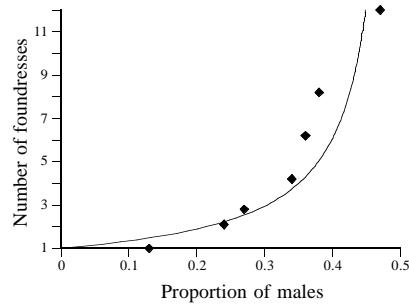
$$s^{**} = \frac{\psi \iota^{*e} + 1}{1 - \psi \iota^{*e}} \quad (20.9)$$

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  $\epsilon_l$ , 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  $\epsilon_l$  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 ( $w_\theta$ ), plus the mass of the male ( $w_\phi$ ) times the number of males per female ( $\phi/\theta$ ), 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 ( $\epsilon$ ) 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  $\epsilon_i$  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 have 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  $R_0^* = pe^*/wB$  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 ( $n_\theta$ ), 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  $\iota^{*e}$  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  $\theta_w$  and  $\phi_w$  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 ( $\theta$ ) will be situated at the Fisherian optimum ( $\theta^{*F}$ ), 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 ( $\theta_w$ ). By eqn 22.10 this implies that  $\partial\lambda/\partial\theta_w = 0$  for all  $\theta_w$  because  $\partial\theta^{*F}/\partial\theta_w = 0$  for all  $\theta_w$ . 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  $\rho$ , 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  $\rho$  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  $\rho$  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  $\phi/\theta$ . 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  $\theta/\phi$ . 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

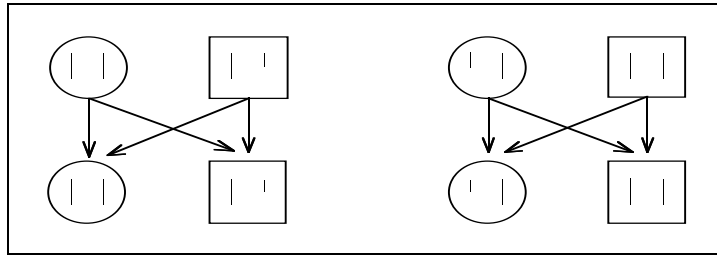
by an even sex ratio in the sexuals and a worker caste containing only male offspring. This prediction does not correspond to the situation in ants and bees, and the current model suggests that this is because pair formation generally does not occur between the sexuals in these two taxa.

## 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

$$\begin{aligned}
 p_{d,m} &= h/(1 + h) & p_{s,m} &= 1/2 & p_{d,d} &= (2h^2 + h + 1)/2(1 + h)^2 \\
 p_{d,f} &= 1/(1 + h) & p_{s,f} &= 1/2 & p_{d,s} &= (1 + 3h)/2(1 + h)^2 \\
 p_{m,d} &= h/(1 + h) & p_{f,d} &= 1/2 & p_{s,s} &= (3 + h)/4(1 + h) \\
 p_{m,s} &= 1/(1 + h) & p_{f,s} &= 1/2 & p_{s,d} &= (1 + 3h)/4(1 + h) \\
 v_s &= 2/(3 + h) & & & v_d &= (1 + h)/(3 + h) & (22.19)
 \end{aligned}$$

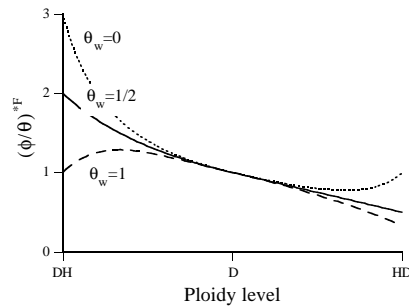
when the genome is diploid to diplohaploid. If we insert these values into eqns 22.4 and 22.6, and solve for the Fisherian equilibrium, we find that

$$\theta^{*F} = \frac{2\theta_w(1 - m) + 1 + 3m}{2(m + 1)(\theta_w[1 - m] + m + 1)} \quad (22.20)$$

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 ( $\theta_w$ ) 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 ( $\theta_w$ ) 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

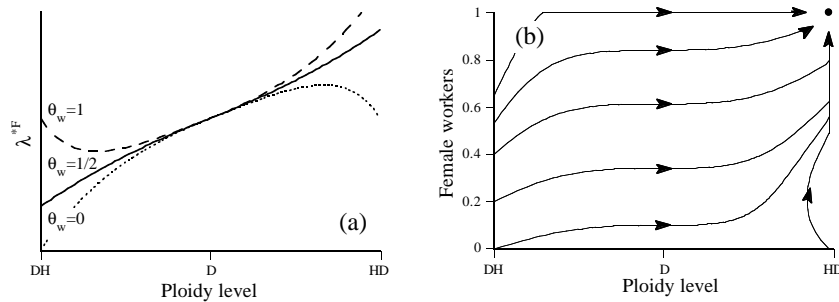
$$\begin{aligned}\frac{\partial \lambda}{\partial h} &= \frac{\partial \lambda^{*F}}{\partial \theta^{*F}} \frac{\partial \theta^{*F}}{\partial h} \quad \text{for } m = 1 \\ \frac{\partial \lambda}{\partial m} &= \frac{\partial \lambda^{*F}}{\partial \theta^{*F}} \frac{\partial \theta^{*F}}{\partial m} \quad \text{for } h = 1\end{aligned}\quad (22.22)$$

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



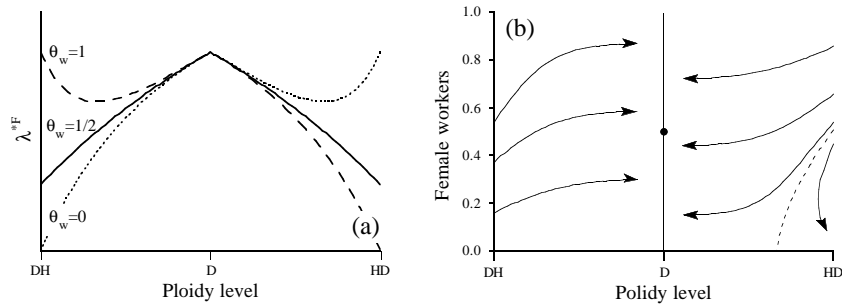
**Fig. 22.3** The ant- and bee-like case where individual selection on the sex ratio in the sexuals is defined by the two-fold cost of the sexual male. **(a)** Fitness profiles on the ploidy level of the genome, given that the proportion of female workers ( $\theta_w$ ) is respectively zero, one half, 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 ( $\theta_w$ ) 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.