

# **Modelling the Evolution and Consequences of Mate Choice**

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*I, Samuel John Tazzyman, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.*



*for Katie, with my love*

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# Abstract

This thesis considers the evolution and the consequences of mate choice across a variety of taxa, using game theoretic, population genetic, and quantitative genetic modelling techniques.

Part I is about the evolution of mate choice. In chapter 2, a population genetic model shows that mate choice is even beneficial in self-fertilising species such as *Saccharomyces* yeast. In chapter 3, a game theoretic model shows that female choice will be strongly dependent upon whether the benefits are fixed, so that females receive the same fitness boost from a mating with a given male regardless of how many matings that male has, or dilutable, where the more females a male mates with, the lower the expected benefit to each. This leads to the prediction that mating skew should be higher in species in which the benefits of mate choice are hypothesised to be due to good genes.

Part II is about the consequences of mate choice. The theoretical prediction from chapter 3 is borne out by a literature review of studies of wild populations of birds in chapter 4. In chapter 5, a quantitative genetic model about poison-dart frogs suggests that sexual selection can speed up the effect of random genetic drift. This may be of more general importance, further widening the evolutionary impact of sexual selection. Finally, in chapter 6, a game theoretic model of sperm competition shows that pre-copulatory mate choice can also have evolutionary effects upon post-copulatory behaviour, affecting the optimal ejaculate expenditure of males.

Overall, mate choice is shown to be an important evolutionary force, with wide-ranging ramifications across diverse taxa, and effects so varied as to include the evolution of sex, the genetic variation in species, speciation, and post-copulatory behaviour, amongst others. These effects can be effectively explored using mathematical modelling.

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# Chapter 1

## Introduction

*“Sexual selection depends on the success of certain individuals over others of the same sex, in relation to the propagation of the species; whilst natural selection depends on the success of both sexes, at all ages, in relation to the general conditions of life.”*

Darwin (1871)

Sexual selection, defined so clearly by Darwin in the above quote, is the fundamental topic of this thesis. It affects an enormous variety of living organisms (Andersson, 1994), and is widely accepted as a hugely important evolutionary force (though this has not always been the case; Cronin, 1993). As the title suggests, in this work I will use mathematical models to theoretically examine why mate choice evolves, what type of choice we expect, and what further evolutionary consequences come from it.

To aim for universality in such a large field is to fall far short; this work is inevitably species-centred in some areas (e.g. chapters 2 and 5), while in others assumptions are made that may not hold true in all circumstances (e.g. the models in chapters 3 and 6). I have tried to be explicit about these shortcomings, and where possible to discuss how universal the conclusions drawn from this work might be if assumptions were relaxed, or findings extended to other species.

### 1.1 Sex

Sexual reproduction is widespread amongst eukaryotes, although the evolutionary advantages that led to its origin and contribute to its maintenance are still a subject of debate amongst evolutionary biologists (e.g. Williams, 1975; Maynard Smith, 1978; Bell, 1982; Otto and Lenormand, 2002; see also chapter 2 and references therein). Sexual reproduction is not as easy to define as might be thought. From our own

species we might be tempted to define it as the creation of offspring by combining gametes from a male and a female. We can recognise this process in many other invertebrates. However, in other species, things are not so straightforward (for a list of examples and references, see Hurst, 1995). Andersson (1994) calls it “the combination of genetic material from two parents in the progeny”, and notes that it therefore does not require two sexes. However, this also runs into difficulties - what of hermaphroditic organisms that are able to self-fertilise? What about facultative selfing, as seen in yeast, where outcrossing is very rare? It is perhaps best, then, to concentrate on the genetic level, and define sex as being the creation of offspring that are genetically different to the parent via the combination of two gametes. This can be contrasted to asexual reproduction, which is the creation of offspring that are genetically identical to the parent (apart from chance mutational events).

Even this definition of sex may turn out to be problematic, such is the wide variety of reproductive behaviour in the natural world. For example, bacteria (and even viruses) exchange genetic information, and this is often characterised as ‘sex’ (Redfield, 2001). I choose not to count this as sex here, as I wish to consider sex as an act of reproduction rather than simply of genetic information transfer.

Fortunately this semantic confusion can be generally ignored, since for most species it is clear what is meant by sexual reproduction. Indeed for many species this is the only form of reproduction possible, although even in higher organisms things are not always as they seem, as seen by the parthenogenesis recently reported in Komodo Dragons, *Varanus komodoensis* (Watts et al., 2006). In this thesis (apart from some elements of chapter 2), I concentrate on the evolutionary consequences of sexual reproduction, in the form of sexual selection, rather than the reasons for the origin and maintenance of sex. As Andersson (1994) pointed out, “how and why sexual reproduction arose are questions outside of sexual selection theory”.

## 1.2 Sexes

### 1.2.1 What is a sex?

The above definition of sexual reproduction requires that two gametes combine to form a new organism. In most species, these two gametes are of different types. This leads (as will be seen) to the evolution of the sexes. In much of the discussion that follows, therefore, the word ‘sex’ will be used where the somewhat clunkier term ‘gametic type’ would sometimes be more accurate. It is obvious in a two-sex species like humans that there is a large overlap between the two terms, with sperm and eggs at the gametic level, and males and females at the level of the individual organism. This simple correspondence breaks down, however, in species in which there exist hermaphrodites, since although individual organisms can be both

male and female, individual gametes cannot. Therefore the gametic level is the more fundamental one: the key difference between different sexes is in their gametes. What defines an individual organism's sex from a biological perspective is the different types of gametes it can produce. Thus a hermaphrodite is, by the definition here, both male (i.e. sperm-producing) and female (i.e. egg-producing).

### 1.2.2 The number of sexes

Evolution by sexual selection does not require that there are two sexes. There also exist species, for example the slime mould *Physarum polycephalum*, which have many sexes (Kawano et al., 1987). Indeed, the fact that most species have two sexes is perhaps surprising (Iwasa and Sasaki, 1987; Hurst, 1996), for the following theoretical reason. If there are many different sexes, then the frequency of matings that occur between any given two types can be defined as a function of the frequency of each type, giving a set of rules called "mating kinetics" (Iwasa and Sasaki, 1987). The simplest mating kinetics say that the frequency of matings between two sexes  $S_1$  and  $S_2$  is proportional to the frequencies of each sex, and that same-sex matings cannot occur. Under this scenario, a newly evolved rare sex  $S_3$  is at a great advantage, because nearly every meeting it has will be with either the  $S_1$  or the  $S_2$  sex, and so every encounter is a possible mating. For the common  $S_1$  and  $S_2$  sexes, however, a high proportion of their encounters will be same-sex ( $S_1$  meeting  $S_1$  or  $S_2$  meeting  $S_2$ ). This rare-sex advantage should result in the evolution of more and more sexes (Iwasa and Sasaki, 1987). Two sexes should in fact be the least-favoured number, since (assuming both are equally represented) it would mean only half of encounters could lead to reproduction, the lowest proportion possible (Hurst, 1996).

So why do we frequently only see two sexes? An explanation as to why two is the prevalent number of sexes is that this best allows for the inheritance of cytoplasmic DNA (such as mitochondria) to be divided: one sex always passes on these elements, the other never does (Hurst and Hamilton, 1992; Hurst, 1996). In this way the threat from selfish cytoplasmic DNA can be minimised. Evidence to support this hypothesis comes from looking at the sexual systems of species with more than two sexes (Hurst, 1995).

Regardless of the number of sexes a species has, sexual selection can have an effect, for example by promoting traits that make gametes more efficient at finding others with which they can fuse. This will be true of species with just one sex in addition to those with many. In this thesis, I consider only species with two sexes, since this is the norm.

### 1.2.3 Differences between males and females

In species with two sexes, what differences are there between males and females, other than that a gamete of each type is required to produce a zygote? Darwin (1871) defined the reproductive organs as the “primary sexual characters”, since these must be different in each sex (the reproductive organs of hermaphrodites can also be distinguished along these lines). Biological convention dictates that we define the sex with the smaller gametes as the male, and that with the larger as the female. This size discrepancy in gametes, called anisogamy, has an evolutionary explanation (Parker et al., 1972; Maynard Smith, 1982; Bulmer and Parker, 2002). Assume that there are two sexes, that the probability a gamete survives to mating and the probability a zygote survives to become an adult are both increasing functions of mass (a zygote’s initial mass is assumed to be the sum of the masses of the gametes that fused to form it), that the total mass adults have for gamete production has some upper limit, and that gamete size has some lower limit. We call the gametes from one sex ‘proto-sperm’ and those from the other ‘proto-eggs’. Then selection can favour either (1) isogamy, or (2) anisogamy for small proto-sperm from one sex and large proto-eggs from the other (leading to the evolution of small sperm and large eggs). Which of these outcomes occurs depends on the relationship between gamete size and gamete and zygote survival (Bulmer and Parker, 2002). For a plausible range of such relationships, disruptive selection favouring anisogamy will occur. Under these scenarios, the benefits of fusions between large proto-sperm and large proto-eggs will be offset by the rarity of such fusions due to the cost of production of large gametes. The frequency of fusions between small proto-sperm and small proto-eggs will be offset by the low survival rates of the resulting zygotes. Therefore there is an advantage to fusions between small and large gametes. This means that it pays the sex making proto-sperm to specialise in making small gametes, and the sex making proto-eggs to specialise in making large gametes, leading to the evolution of small sperm and large eggs (since we define the smaller of the two types of gamete as being the sperm, small eggs and large sperm is a contradiction in terms).

Note however that this simple dichotomy between small sperm and large eggs is not always the case. There are species (e.g. *Drosophila bifurca*; Pitnick et al., 1995) in which the sperm is very large compared to the egg. The reasons behind this are debated (Bressac et al., 1994; Pitnick et al., 1995); generally it is assumed that there must be some fertilising advantage to having large sperm, possibly due to sperm competition occurring within the reproductive tract of a polyandrous female (Parker, 1970; see section 1.3.1 and chapter 6).

In addition to gamete size and reproductive organs, there are also of course many other differences between males and females. Darwin (1871) called these the “secondary sexual characters”, though as he noted there is often no sharp distinction between these and the primary characters, but rather secondary



characters “of infinitely diversified kinds, graduate into those which are commonly ranked as primary, and in some cases can hardly be distinguished from them”. With the discovery of post-copulatory selection (see below) and the consequent fact that primary sexual characters can themselves be under sexual selection, finding a clear division between types of sexual character becomes even more problematic. Like many definitions, the idea is useful only in so far as it aids thinking about the subject; one shouldn’t be a slave to semantics.

Although in the strictest sense, males are defined as the sex that produces the smaller gametes, and females that which produces the larger, there are other characteristics that we think of as being typically ‘male’ or ‘female’. Typically males are seen to be the sex that competes for mating opportunities, and exhibits high levels of promiscuity, while females are the sex that is ‘choosy’ when it comes to copulation. As with many biological patterns, there are many examples of species that do not follow this rule; nevertheless it certainly seems true much of the time. The classic explanation for the pattern is due to Bateman (1948). He experimented with *Drosophila* and showed that males benefited from mating multiply, while females didn’t. He claimed this was fundamentally connected with anisogamy. In many species, the sperm of a male are so numerous that he could fertilise all of the eggs of every female he ever met (Williams, 1966). Because of this, what generally limits a male’s reproductive success is how many mates he can obtain, rather than the production of gametes. On the other hand, since eggs are larger, they cost more resources to produce, and this cost of production is what generally limits a female’s reproductive success. Therefore while males benefit by having multiple mates, females benefit by obtaining high quality mates and maximising the chances of survival of each offspring. This is called ‘Bateman’s principle’ (Bateman, 1948). It means that males have much higher variance in reproductive success than females, and gives ample opportunity for sexual selection.

Trivers (1972) expanded this argument to account for parental investment of all kinds, rather than just the investment per gamete. For a given zygote, the initial investment by the mother is greater than that by the father (as eggs are larger than sperm), but both sexes have a vested interest in raising an offspring. Both, however, would also rather that the other did most of the work. If one parent abandons the offspring, and it is still raised successfully by the other parent, then the abandoner will have done better, particularly if his/her resources are invested in mating with others rather than in parental investment. This sort of antagonism between the sexes can often be a powerful evolutionary force (see section 1.3.3 below). If evolution leads one sex to usually invest more in the offspring than the other, then the sex investing more will be selected to be choosy with regard to mates, so as to maximise the chances of offspring success, while the other sex will be selected to be promiscuous to maximise the total number of offspring. The initial imbalance in

size between sperm and egg means that in general it is the females who are choosy and the males who are promiscuous (Trivers, 1972).

Both Bateman's original work, and the principle itself, have been criticised by evolutionary biologists (Sutherland, 1985; Birkhead, 2000; Snyder and Gowaty, 2007), and certainly there are many examples of species in which females benefit by mating with multiple males (see review by Ridley, 1988). It is also true that while individual sperm are much less costly than eggs (although again here there are exceptions, e.g. *Drosophila bifurca*; Pitnick et al., 1995), each ejaculate may contain large quantities of sperm and thus may still require large quantities of energy to produce (Wedell et al., 2002). This is particularly true where there is sperm competition (Parker, 1970; see section 1.3.1 and chapter 6). However, in order to work as a general explanation for the described pattern, all that is required is a weaker version of Bateman's principle (Arnold and Duvall, 1994). The 'Bateman gradient' (Arnold and Duvall, 1994; Andersson and Iwasa, 1996) is the relationship between the number of mates an individual organism obtains and the expected number of offspring it obtains. If obtaining more mates results in lots more offspring, this equates to a steep Bateman gradient, and would be expected to lead to selection for obtaining more mates. If obtaining more mates results only in a few more offspring then the Bateman gradient will be more shallow and selection for quality of mate may be more important than selection for quantity of mates (Andersson and Iwasa, 1996). Recent thinking has stressed that the Bateman gradient will vary within individuals depending on the number of mates and the conditions, and that for both males and females there will likely be some number of mates after which number of organisms doesn't increase much as fitness becomes limited by other factors (Jones and Ratterman, 2009). Males, for example, cannot assign an infinitesimally small quantity of sperm to a mating; rather, there is a given minimum resource cost required to create one functioning sperm cell. Thus males can become sperm depleted after a given number of matings (e.g. in Soay sheep, *Ovis aries*; Preston et al., 2001), and thus their Bateman gradient will flatten out after this point.

An explanatory framework for the differences in males and females based on Bateman's principle is still possible: we require only that the Bateman gradient is usually larger for males than for females, so that males generally benefit more from having multiple mates than do females. We don't expect all females to carefully select a mate and never mate with any other male, nor do we expect all males to discard mates and search for new females as soon as copulation is over. The propensity for multiple mating by females is likely to be governed by complicated forces (Alonzo and Pizzari, 2010). In any given species, ecological and genetic factors will play a role in determining the Bateman gradients, and thus the sexual system (monogamy, polygyny, polyandry, or promiscuity), and the amount of parental investment by each sex (Trivers, 1972). This will in turn determine how and by how much sexual selection operates.

### 1.2.4 The sex ratio

Despite the differences in selective pressures on males and females, it is notable that in many species the sex ratio is approximately 1:1. Darwin famously considered finding the reasons behind this a problem ‘so intricate that it is safer to leave its solution for the future’ (Darwin, 1871), but Fisher came up with a simple verbal explanation (Fisher, 1930): the level of investment that parents put in to both male and female offspring should be equal, since the total expected fitness of males must equal that of females because each mating involves precisely one male and one female. To see that this is self-balancing, note that the expected reproductive success per individual of a given sex will be equal to the total reproductive success of that sex (which is equal for both sexes) divided by the number of individuals of that sex. Therefore if there is for some reason an excess of males, the expected reproductive success of sons will be lower than that of daughters. Thus parents investing more resources in daughters will benefit, and so the investment ratio will balance out across the population. This argument holds true even if males and females have different survival rates, or different variances in mating success.

Fisher’s argument falls down, however, in species where inbreeding is the rule, as under these circumstances mothers are better served producing only enough sons to fully fertilise all of their daughters (since there are unlikely to be any cases where sons outbreed with daughters from another mother). This was modelled by Hamilton (1967), along with other genetic drivers capable of offsetting Fisher’s arguments. There can also be external forces affecting the sex ratio. Arthropod species are often infected with inherited bacteria such as *Wolbachia*, which are transmitted vertically from mothers to offspring through the cytoplasm in the egg. These bacteria therefore have zero fitness in males, because they cannot pass to offspring via the sperm. This leads to selection pressure for a variety of sex ratio distorting phenotypes in *Wolbachia* and other inherited bacteria, such as feminisation of males, parthenogenesis induction, and male-killing (Stouthamer et al., 1999), all of which lead to female-biased populations.

In addition, although Fisher’s argument holds true in the main at the population level, so that overall parental investment across the population is likely to be equal in sons and daughters, it is possible that at an individual level there can be departures from this 1:1 ratio (Trivers and Willard, 1973). To see this, consider a species in which males invest little or no resources in offspring. In such species the variance in mating success is expected to be greater in males than in females, although the mean reproductive success of both must be equal. The sex ratio is determined by the (relative) investment that females put into male and female offspring. Suppose that the absolute level of investment in an offspring affects to a large degree its reproductive success in the future. This will mean that poor condition females, who can only afford low levels of investment, are likely to have offspring with lower reproductive success, while high condition

females, who can afford higher levels of investment, are likely to have offspring with higher reproductive success. Since reproductive success varies more in males than in females, it follows that good condition mothers should invest in sons, since the reproductive success of a high-quality male is higher than that of a high-quality female (since the mean reproductive success is equal in both sexes but variance is higher in males). By a similar argument, poor condition mothers should invest in daughters, since the reproductive success of a low-quality female is higher than that of a low-quality male. Such differential investment has been reported (for example in red deer, *Cervus elaphus*; Clutton-Brock et al., 1984; though see also Kruuk et al., 1999), although the means by which it occurs are often difficult to ascertain.

### **1.3 Sexual selection**

Let us now assume that the species in which we are interested have two sexes (that is, that there are two types of gamete, both of which are required to create offspring). While, as noted above, elements of sexual selection will apply to a broader range of species than those to which this assumption applies, by restricting ourselves in this way we can clarify the concepts which can then be applied elsewhere more accurately.

Sexual selection can be subdivided into two subcategories, which I here call selection by competition and by choice. These categories are not mutually exclusive; rather, the sexual selective forces acting on any given species are likely to be made up of a combination of elements of these types. Indeed, in many cases the two categories are not absolute, discrete concepts - some phenotypic adaptations may be useful both for fighting of other males and attracting females, for example. However, the categorisation forms a useful basis for the contemplation of evolution by sexual selection.

#### **1.3.1 Competition**

Selection by competition (often called intrasexual selection, despite the fact, as pointed out by Andersson and Iwasa, 1996, that all sexual selection is inherently selection within a single sex) is the “struggle... between the individuals of the same sex, generally the males, in order to drive away or kill their rivals, the females remaining passive” (Darwin, 1871). In this mode of selection, there is direct, potentially violent, competition between members of one sex, success in which leads to privileged access to the passive members of the other sex. It favours the evolution of phenotypes more likely to be victorious in same-sex combat, thus leading to armaments such as antlers, horns, and spurs, and frequently (at least in mammals) to large size dimorphism between the sexes, since larger individuals are more likely to be victorious in fights (sexual size dimorphism is reviewed in Chapter 10 of Andersson, 1994; for a model of increasing size under

competition dynamics, see Maynard Smith and Brown, 1986). Since the battling sex is usually male, this type of sexual selection is often referred to as ‘male-male competition’.

### **Male-male competition**

Because of Bateman’s principle, as stated in section 1.2.3 above, it is more common to see males fighting over control of (resources leading to access to) females than vice versa: fighting is usually more worthwhile to males than it is to females, because the multiple mating that may result from such behaviour is usually more beneficial, and so the risk more likely to be worth taking. Note however that in some species there is sex-role reversal, and females are the sex that competes for access to mates (e.g. the dung beetle, *Onthophagus sagittarius*; Watson and Simmons, 2010).

Male-male competition has generally been uncontroversial when compared to other aspects of sexual selection (Cronin, 1993), since there are many examples of males fighting for access to females. Frequently males will not engage in battles to the death (Maynard Smith and Price, 1973), but rather will sort themselves into a dominance hierarchy, with fighting occurring only in cases where the appropriate dominance relationship between two males is unclear. This reluctance to fight can be understood by considering the potential gains and losses resulting from potentially-fatal combat (Maynard Smith and Price, 1973; Maynard Smith, 1974; see also the Hawk-Dove game in section 1.6.3 below). In species where the males have little time or opportunity for mating the stakes are higher, and fights are more frequent (e.g. the Atlantic Salmon, *Salmo salar*, where certain male morphs fight to the death for mating opportunities, a result of them having only one breeding season in their lifetime; Fleming, 1996). This is also the case in species in which the victor gains much from fights, due to inheriting a harem (e.g. the Northern Elephant Seal, *Mirounga angustirostris*; Leboeuf, 1972), or a favoured breeding territory (e.g. lekking spots in Fallow Deer, *Dama dama*; Apollonio et al., 1990).

As can be seen from Darwin’s definition, females are supposedly passive in male-male competition. However there is an implicit assumption that they give tacit approval to the male dominance hierarchy by selectively mating in agreement with it (though in some species dominant males render female choice moot by actively breaking up matings between females and subdominant males, e.g. the Southern Elephant Seal, *Mirounga leonina*; McCann, 1981). The assumption is generally justified, since in many cases males fight for control of resources which the females want (resource defence polygyny; Alcock, 1998) rather than for the females themselves. Males cornering the most resources will therefore also obtain the most females (see chapter 3).

### **Sperm competition**

Male-male competition is not limited to pre-copulatory behaviour (Parker, 1970). In species in which females are polyandrous (that is, mate with multiple males), there is potential for ‘sperm competition’, defined as “competition between sperm of two or more males for fertilisation of an ovum” (Parker, 1970). This process favours the evolution of adaptations that increase the likelihood of fertilisation of a female by a male’s own sperm. Such adaptations can either improve a male’s own chances, often by leading to the ejaculation of more, faster, or larger sperm (chapter 6), or damage the chances of other males, for example by leading to the removal of other males’ sperm from a female’s reproductive tract during mating (e.g. the penis of the Damselfly, *Calopteryx maculata*; Waage, 1979).

### **1.3.2 Choice**

Selection by mate choice is often referred to as intersexual selection, a term I avoid here, because all sexual selection is in fact intrasexual (Andersson and Iwasa, 1996). In contrast to the battle between the members of one sex for access to mating with the opposite sex, selection by choice is “the struggle. . . likewise between the individuals of the same sex, in order to excite or charm those of the opposite sex, generally the females, which no longer remain passive, but select the more agreeable partners” (Darwin, 1871). In this mode of sexual selection, then, the members of one sex actively choose their mates, while members of the other sex attempt to convince them that they are the best choice. Choice selection favours the evolution of phenotypes that are attractive to the opposite sex, leading to ornaments such as the peacock’s tail (Petrie and Halliday, 1994), and behaviours such as the mating calls of tropical frogs (Ryan, 1980). Since it is generally the females that do the choosing, this is often referred to as ‘female choice’. It is with mate choice that this thesis is principally concerned, thus I say only a few words about it here, returning to the subject later in sections 1.4, 1.5 and 1.6.

### **Female choice**

The classical explanation as to why females are generally the choosy sex is the same as that given above for males being the more likely to engage in same-sex combat: anisogamy leading to Bateman’s principle, or at least the weaker version of it stated above, that males generally benefit from multiple matings more than do females. If members of either sex can increase their number of offspring more by a careful choice of a mate than by being promiscuous, then this behaviour will be evolutionarily favoured (Arnold and Duvall, 1994). Since females generally gain less from having multiple mates, it is therefore more likely that they will benefit by being choosy, and thus they are more frequently seen to be exercising choice.

The degree to which females are able to distinguish between males and select mates based on certain criteria has been historically controversial (Cronin, 1993), but there have been many empirical studies demonstrating the existence of female preferences for a variety of male characteristics in many different species (Andersson, 1994). There are several, not mutually exclusive theories as to why female choice is beneficial (section 1.4 below).

### **Male choice**

There are examples of species in which the males do most or all of the parental care (for examples see review by Ridley, 1978). In these species we would expect to see male choice. However we may also see it even in species in which sex roles are not reversed. If females vary in their fecundity, for example, and males can detect this variation, then there may be selection to make use of this information. Males might preferentially mate with females who will likely have more eggs (Bonduriansky, 2001), and/or invest more sperm when mating with such females (Gage and Barnard, 1996). Male choice is generally less frequently investigated than female choice (Andersson, 1994; Bonduriansky, 2001). It should be noted that since choice itself may be costly, it might not always be beneficial. If a male takes time to select and mate with the most fecund female, but the other males in the population use this time to achieve large numbers of matings and in so doing have more offspring than the focal male, then choice is not evolutionarily worthwhile. This logic applies equally to females but, as noted, they are likely to benefit less from having multiple mates.

### **Cryptic choice**

Just as with contests, choice does not necessarily end at copulation. Females may be able to favour fertilisation by preferred males (or disfavour it by non-preferred males) using many potential methods including ejecting sperm from less favoured males (e.g. in the Fowl, *Gallus gallus domesticus*; Pizzari and Birkhead, 2000), decreasing the rate or number of offspring resulting from the copulation (e.g. in the Scorpionfly, *Harpobittacus nigriceps*; Thornhill, 1983), preventing full copulation (e.g. in the Grasshopper, *Chorthippus curtipennis*; Hartmann and Loher, 1974), breaking off copulation early (e.g. in the Tiger Beetle, *Pseudoxychila tarsalis*; Eberhard, 1996), or by adjusting the internal storage or transport of sperm (e.g. in the Yellow Dungfly, *Scathophaga stercoraria*; Ward, 1993). Many other methods and examples exist (Eberhard, 1996). Because this form of choice often occurs within the female reproductive tract, after copulation, it is harder to see than pre-copulatory behaviour such as a female's response to a male mating display. For this reason post-copulatory female choice is often called "cryptic choice" (Thornhill, 1983; Eberhard, 1996).

### 1.3.3 Sexual conflict

In addition to the sexual selection modes above, there is also another evolutionary process that is frequently invoked when talking about sexual selection: sexual conflict. Although the work in this thesis doesn't explicitly involve sexual conflict I include mention of it here out of a sense of completeness. There have been several attempts to define what is meant by sexual conflict (Tregenza et al., 2006). Here I will follow Parker (1979) and broadly define sexual conflict as "differences in the evolutionary interests between males and females". This general definition can be divided into two key categories.

#### **Intralocus sexual conflict**

Intralocus sexual conflict, as defined by Bonduriansky and Chenoweth (2009), "occurs when selection on a shared trait in one sex displaces the other sex from its phenotypic optimum". A simple example is sexual size dimorphism, as mentioned above (section 1.3.1). In a species where selection favours larger males and smaller females, there will be conflict in the sense that genes for large body size are favoured when in males and disfavoured in females (and vice versa with genes for small size). This can lead to the mean phenotypes of one or both sexes being suboptimal (Lande, 1980b), with males smaller than ideal and females larger, due to the fact that neither 'large' nor 'small' genes will be eliminated by selection. In this example selection has a different sign in males and females, but intralocus sexual conflict can occur when this is not the case, for example if selection favours being larger in both sexes, but more so in one sex than in the other. Any difference in selection between sexes will cause sexual conflict and potentially slow down their evolutionary responses (Bonduriansky and Chenoweth, 2009).

Intralocus sexual conflict is predicted to favour the evolution of sex-linked genetic modifiers and sex chromosomes (Lande, 1980b; Rice, 1984). These resolve the conflict by confining the expression of alleles to the sex in which they are favourable. Alleles confined to being expressed in one sex are, however, ripe candidates for interlocus sexual conflict (Rice, 1984).

#### **Interlocus sexual conflict**

Interlocus sexual conflict, as defined by Chapman et al. (2003), occurs "when there is conflict over the outcome of male-female interactions, so that the optimal outcome is different for both sexes".

A classic example of such conflict is over mating rate. In many species of insect, for example, multiple mating decreases the lifespan of females (Arnqvist and Nilsson, 2000). For males, however, the more matings they get the more offspring they potentially have (see Bateman gradient discussion in section 1.2.3 above). Therefore selection for males is likely to favour adaptations that enable them to increase the mating



rate, while selection for females is likely to favour adaptations that decrease it (or at least to prevent males from increasing it). Support for this comes from experiments showing that when female resistance is experimentally constrained, mating rate increases (e.g. in the water strider, *Gerris incognitus*; Arnqvist and Rowe, 1995). This suggests that the optimal mating rate for males is higher than it is for females, and that females actively seek to lower the mating rate, while males seek to raise it.

In general, whatever the shared trait for which male and female optima differ, interlocus sexual conflict is predicted to result in sexually antagonistic coevolution, with males and females in a cycle of adaptation and counter-adaptation, each attempting to force the results of the interaction under conflict towards their own preferred outcome (see reviews by Chapman et al., 2003; Parker, 2006).

## **1.4 The reasons for mate choice**

As the title suggests, and as mentioned previously, the main topic of this thesis is mate choice: how and why organisms go about selecting their mates. Evolutionary theory suggests that such behaviour should be advantageous; that those organisms exhibiting choice should have (on average) more offspring surviving to maturity, meaning the spread of this behaviour throughout the population (Andersson, 1994). Since we see female choice in so many species, we are led to ask how it results in higher fitness? What benefit does choosing a mate provide to females so that they have more offspring than hypothetical females without this mating preference? There are several hypotheses for the nature of this benefit. They are generally divided into two categories: direct and indirect benefits. These are then subdivided further giving a variety of different potential hypotheses (Table 1.1).

The distinction between the two types of benefit is a simple one: does the benefit fall to the female herself, or to her offspring? In other words, will genes promoting choice be directly favoured themselves because females bearing those genes will have more offspring, or will they be indirectly favoured by being regularly associated with genes that are directly favoured, via mate choice; so that the offspring of females bearing genes for choice leave more offspring. This distinction is simple, but potentially important.

### **1.4.1 Direct benefits**

Female choice for direct benefits is the simplest to understand of the two types of benefit. Suppose there are genes that, when expressed in a females, cause her to have some phenotypic preference when it comes to choosing a mate. Suppose that males of her preferred phenotype provide her with some benefit when compared to males not of her preferred phenotype, and this benefit leads to the female having more offspring

Benefit type	Hypothesis	References
Direct	Phenotypic benefits	Alatolo et al., 1986; Hamilton, 1990; Arnqvist and Nilsson, 2000; Conner et al., 2000; Albo and Costa, 2010
Direct	Parental care	Norris, 1990; Knapp and Kovach, 1991
Direct	Fertility & fecundity benefits	Trivers, 1972; Bownes and Partridge, 1987; Butlin et al., 1987; Kalb et al., 1993; Sheldon, 1994; Chapman et al., 1995; Pizzari et al., 2004
Indirect	Species recognition	Fisher, 1930
Indirect	Fisher's runaway/'sexy son'	Fisher, 1930; Weatherhead and Robertson, 1979; Lande, 1981
Indirect	Good genes	Zahavi, 1975; Hamilton and Zuk, 1982; Grafen, 1990; Iwasa et al., 1991; Iwasa and Pomiankowski, 1995; Rowe and Houle, 1996; Kotiaho et al., 2001; Tomkins et al., 2004
Indirect	Compatible genes	Olsson et al., 1996; Zeh and Zeh, 1996; Tregenza and Wedell, 2000; Puurttinen et al., 2009

Table 1.1: List of hypotheses for mate choice benefits, together with references for relevant empirical or theoretical papers. The list of references is far from exhaustive and the number of references for each hypothesis should not be taken as a sign of its importance.

than females who do not have her preference. Then, since the focal female's offspring will likely inherit the preference genes, it is easy to see why such genes should spread in the population. The question, then, is what kinds of benefit can females obtain from males that give a direct effect in terms of increasing offspring number or survival? Many such benefits have been hypothesised, and they can be further divided into several categories.

### **Species recognition**

One way in which female choice could be of benefit is so clear as to be almost trivial. As pointed out by Fisher (1930), the ability to recognise a conspecific potential mate is likely to be under strong positive selection. An organism who consistently mates with individuals of another species will likely leave fewer offspring than one who can recognise mating partners that will result in viable offspring. This will of course be true in both males and females, but (for the reasons mentioned above, section 1.2.3), mate choice is more likely to be developed in females. This could be considered to be a special case of the compatible or good genes cases from the indirect benefits type below. However I choose to include it here to represent cases in which hybrid offspring are inviable, rather than being sterile (or just less fit). It's my contention that in these cases species recognition provides a direct benefit, by affecting the number of offspring a female has rather than the number her offspring have. In practice, everyone is likely to agree that species recognition is beneficial to females.

### **Phenotypic benefits**

Another easy-to-understand way in which females could benefit directly from choice of mate is via the male making it more likely that the female survives. This could come about due to the male providing benefits such as more food (e.g. nuptial gifts in many invertebrate species, such as the neotropical spider, *Paratrechalea ornata*; Albo and Costa, 2010), better territory (e.g. in many birds, such as the Pied Flycatcher, *Ficedula hypoleuca*; Alatolo et al., 1986), or protection from predators (e.g. the toxin supplied to females by males in the Scarlet-bodied Wasp Moth, *Cosmosoma myrodora*; Conner et al., 2000). However, it could also come about by avoidance of costs, so that by choosing wisely a female may make it less likely that she contracts a disease from her mate (Hamilton, 1990), or that he imposes direct costs on her by his mating behaviour (Arnqvist and Nilsson, 2000). The theoretical relationship of 'choice to obtain benefits' as opposed to 'choice to avoid costs' is generally assumed to be one of equivalence, although this has not been fully explored (Chapman et al., 2003). Of course, from the point of view of an organism, the two may be different: choosing to mate with the closest male to avoid the costs of travelling to find another male is

different from actively wishing to mate with a male because he has the best territory.

### **Parental care**

A wise choice of mate by a female could result in her getting more offspring not purely through the male lavishing care upon the female. Parental care also counts as a direct benefit, since it results in the female having more surviving offspring (e.g. in the Great Tit, *Parus major*; Norris, 1990). This will obviously frequently have an overlap with the benefits provided above, since good parental care can involve not spreading disease to the offspring, provisioning them with food, protecting them from predators, and even not cannibalising them (e.g. in the Bicolour Damselfish, *Stegastes partitus*; Knapp and Kovach, 1991). However, recall that parental care is something generally expected to be under sexual conflict (sections 1.2.3 and 1.3.3; Trivers, 1972), so this overlap will not always be the case. For example, in some species, females choose males based on ornament traits, with more favoured males actually providing worse paternal care than less favoured males (Møller and Jennions, 2001). Presumably in this case the females are getting some other benefit from mating with their chosen male which more than cancels out the cost of poor paternal care.

### **Fertility and fecundity benefits**

The final way in which males could directly provide females with more offspring concerns the number of offspring a female has. As Trivers (1972) noted, sperm depletion will often occur in males after several copulations (e.g. in Soay sheep, *Ovis aries*; Preston et al., 2001). Since there is (generally) no advantage to females mating with a male who cannot fertilise their eggs, females should exercise choice to avoid such males, instead choosing (where possible) males who have not recently mated.

Additionally, when postcopulatory selection is considered, it may be the case that different males are likely to invest differing amounts of resources in their ejaculate (chapter 6, and references therein). If there is consistent variation between males in their ability to fertilise females, then a female will benefit from being able to ascertain a male's fertilising ability and choose mates accordingly (Sheldon, 1994). By mating with males who generally fertilise more eggs, she will have more offspring per mating. This is often called the 'phenotype-linked fertility hypothesis' (Sheldon, 1994; Pizzari et al., 2004; see also chapter 3).

In some species males can increase female post-mating fecundity. This behaviour is beneficial for a male since it increases the amount of offspring that he has from a mating. There are two key ways in which this fecundity boost comes about, and they can cause different selective pressures. The first is by the transfer of nutrients with the ejaculate, generally in the form of a spermatophore (e.g. in the

grasshopper, *Chorthippus brunneus*; Butlin et al., 1987). In cases such as this, the nutrients provided by the spermatophore are used in egg production, increasing the female's fecundity. Females should be selected to search for males likely to provide large spermatophores (chapter 3), since they're getting extra fecundity for free. The second way in which males boost fecundity is less clearly beneficial. In some species (e.g. *Drosophila melanogaster*; Chapman et al., 1995), the male ejaculate contains proteins that boost a female's egg production, in addition to sperm. However, they do not do this by providing extra nutrients, since the nutrient content of the ejaculate is very low (Bownes and Partridge, 1987). Instead, it is believed that they act to chemically increase the egg-laying of the female (Kalb et al., 1993). However, in the longer term, these proteins seem to be harmful to the female (Chapman et al., 1995). The behaviour of these proteins is therefore evolving under sexual conflict: a male wants to maximise a female's egg-laying after he has mated with her. Whether the female will want to mate with males exhibiting such behaviour will depend on how harmful the proteins are. Even if they are detrimental to females overall, genes for these proteins could still spread in the population if they are sufficiently beneficial to males. Of course, if females can recognise males likely to exhibit behaviour that is detrimental to them and avoid it, then this recognition will be favoured (section 1.3.3).

### **Disentangling direct benefits**

It can be quite hard to disentangle these effects from one another and ascertain the evolutionary causes of observed behaviour. For example, we have seen that some males can cause females to increase egg-laying. However, this observation alone does not tell us the evolutionary value of this behaviour. Egg-laying rate could be under female control, and the increase could be cryptic female choice acting so as to favour some males. On the other hand, it could be that egg-laying rate is being controlled by seminal fluid peptides, and is thus under male control. Careful experiments are needed to distinguish in cases such as these.

### **1.4.2 Indirect benefits**

Benefits are indirect if they assist a female's offspring, rather than the female herself. Suppose the population differs with respect to some gene, and that this gene comes in 'good' or 'bad' varieties. Suppose further that females differ with respect to some other gene, which comes in 'preference' or 'no preference' varieties. Females bearing 'preference' are more likely to mate with males bearing 'good', and thus will have offspring bearing both 'good' and 'preference'. Females bearing 'no preference' will have offspring equally likely to have 'good' and 'bad' genes. Thus 'preference' will spread in the population through its association with 'good' (assuming 'good' will spread through the population by natural selection), driving

out ‘no preference’ which has no such association.

Indirect benefits can come in a variety of types, depending on the nature of the gain accrued by the offspring.

### **Fisher’s runaway process**

The first person to theorise about indirect benefits was Fisher (1930), who coined the concept of ‘Fisher’s runaway process’, using a simple verbal argument (later backed up by others’ mathematical models; e.g. Lande, 1981; see section 1.6.2). Suppose there is some phenotypic trait in males that provides an (arbitrarily small, natural selective) advantage over other males. Then there will be selective pressure on females to take note of this trait, since by so doing they will enhance the fitness of their sons. However, if enough females mate according to this trait, then males who bear it will gain a considerable sexual selective advantage in terms of numbers of mates they will get, in addition to the small initial natural selective advantage conferred by the trait. The total advantage of bearing this ‘sexy’ trait will thus be proportional to how intense the preference for the trait is in the population. The preference itself will also spread, because the sons of females with the preference will bear genes for both the preference and the trait, and will have lots of matings due to bearing the trait. Thus at every generation the preference and the trait become more extreme, a process described as ‘runaway’. Notably, the trait can be pushed by sexual selection past the natural selective optimum that caused the runaway to begin in the first place, as long as the benefits from obtaining more matings outweigh the costs of having a non-optimal trait.

This has also been called the ‘sexy son’ hypothesis (Weatherhead and Robertson, 1979), because the additional benefits accrued by the trait are due to the attractiveness of the sons. In Fisher’s original argument a runaway trait was posited to have an initial natural selective advantage, but in fact all that is necessary to start the runaway is that the trait be initially preferred by most females. Ways in which the preference could begin include it being an indicator of ‘good genes’ (see below) or of any type of direct benefit (section 1.4.1), or some sensory bias in females meaning they are inherently initially attracted to it (section 1.4.3). Once such a preference is established, the runaway process will begin, as long as males bearing the preferred trait have more offspring than those not bearing it.

### **Good genes**

There are several slightly different theories that sometimes get referred to as ‘good genes’ theories of female choice (Cronin, 1993). Indeed, since all indirect benefits are due to the inheritance by a female’s offspring of advantageous genes of some type or other, they could all be referred to as being choice for good genes

in some sense. The intuitive way of thinking about this is that females will gain indirect benefits by preferentially mating with males who bear genes that are likely to lead to ‘good’ offspring. In what sense ‘good’ is meant is usually a subject glossed over; it is generally understood to mean genes for ‘high viability’ or ‘good condition’, but can also (thanks to Fisher’s runaway process, described above) mean genes for attractive traits. We can define ‘good’ genes as being any genes likely to result in a female’s offspring themselves having more offspring - genes likely to result in the female having more grandoffspring, in essence.

However, this definition runs into problems, because of complementarity. Offspring inherit genes from the mother as well as the father, and the two sets of genes then have to coexist efficiently. A good gene for one female may not be good for another, for example due to dominance effects, or interactions between genes at different loci (Winter et al., 2002). So while there may be some genes that are good for all females, females will also likely benefit by taking into account compatible genes (see below).

### **Honest signals**

Suppose in some species there is an initial correlation between good genes and a phenotypic trait, for example tail length. If tail length is controlled by a few genes, these will quickly spread throughout the population, and result in all males having long tails, even those without the good genes that tail length was initially correlated with. Thus the signal would be dishonest, and preference for it would no longer be beneficial. So how can signals be kept honest?

The solution to this is called the handicap theory, verbally posited by Zahavi (1975), and the subject of extensive mathematical modelling (Kirkpatrick, 1986; Pomiankowski, 1987; Heywood, 1989; Grafen, 1990; Iwasa et al., 1991; Iwasa and Pomiankowski, 1994; Kirkpatrick, 1996; Houle and Kondrashov, 2002; Johnstone et al., 2009). It suggests that the traits females will evolve to prefer will be unbluffable signals of male quality because of the cost that bearing such traits imposes on the males. It seems likely that many male-only characteristics are held away from the natural selective optimum by sexual selection, since the female versions of such characteristics can be considered to be at the optimum (at least in cases where both sexes fill the same evolutionary niche). The classic example is the peacock’s tail - it seems difficult to believe that such a huge ornamental trait imposes no costs on peacocks, when peahens have much smaller tails. In fact, the very cost of such ornamentation is crucial to ensuring its honesty. If having a signal of a certain size is more costly on average to lower quality males than it is to higher quality males, then the signal can indeed evolve to be honest. The intuition is that by having a costly ornamental trait, males handicap themselves. Since females prefer males with larger traits, the cost of bearing a large trait is offset by obtaining more matings. Males will therefore evolve to the size of trait that maximises the difference

between the extra matings garnered and the cost born. Since larger ornaments are cheaper for higher-quality males, this optimal size is an increasing function of quality, and ornamental size becomes an accurate signal. Females therefore benefit from the preference.

Costly, honest signals are sometimes referred to as ‘indicator mechanisms’ rather than as handicaps, to highlight the fact that it is not the handicapping aspect that is important to females, but rather the fact that (because of their cost) such traits are honest indicators of a male’s quality (Andersson, 1994). Traits evolving due to Fisher’s runaway process, for example, can still be costly from a natural selective perspective, but need not be ‘indicators’ of any male quality other than their own attractiveness.

### **The Lek paradox**

It is generally expected that traits directly affecting an organism’s fitness will be under strong selection. Alleles likely to increase fitness will quickly spread to fixation in the population and thus genetic variation will be depleted (Fisher, 1930; Falconer, 1967). Therefore the heritability of fitness is likely to be low. In the field of sexual selection, this fact manifests itself in the ‘lek paradox’ (Kirkpatrick and Ryan, 1991; Andersson, 1994). If fitness has low heritability, then there is not much pressure for females to select mates based on good genes, since the differences in quality between males are unlikely to be heritable; on a genetic level, all males will be similarly good. Similarly, if females are selecting males based on certain traits, then the strong selection on those traits will quickly drive alleles coding for them to fixation, again meaning that such traits are not very heritable. Which of these two related paradoxes constitutes the lek paradox has been debated (Brookfield, 1996; Ritchie, 1996; Turner, 1996). The gist of both, however, is that female choice will impose strong selection, which should erode genetic variation and thus lead to choice becoming obsolete. In species where the female receives nothing but sperm from the male (i.e. no direct benefits), this is particularly a problem, since it is difficult to see what advantage choice could have other than by providing indirect, genetic benefits.

To address this issue, Pomiankowski and Møller (1995) conducted a study into the additive genetic variance of sexual and non-sexual characters, using data from many species. They found that, contrary to theoretical expectations, there was more additive genetic variance for sexual traits than for non-sexual traits. This suggests that the theoretical problems of the lek paradox are not in fact occurring in the real world. Pomiankowski and Møller’s explanation for this was that selection favours modifier alleles that increase genetic variance either by increasing the number of loci that affect a sexual trait, or by increasing the contribution of each locus to the trait. Such alleles will be selected for if the fitness functions relating to secondary sexual traits are strongly directional, and concave-up in their shape, as these conditions favour



an increase in developmental instability (Lande, 1980a; Shnol and Kondrashov, 1993).

Critics of this explanation claim that it is unlikely that secondary sexual characters are under such strong directional selection, since although sexual selection may favour larger ornamental traits, natural selection will often favour the converse. Because of this, Rowe and Houle (1996) claim that in general such traits are likely to be under concave-down “conflicting” selection rather than concave-up directional selection. If this is true then selection would in fact favour decreasing genetic variance with trait values clustering more and more tightly around the optimum.

The ‘genic capture hypothesis’ is an alternative explanation for the lack of a lek paradox (Rowe and Houle, 1996; Kotiaho et al., 2001; Tomkins et al., 2004). It stems from the honest signal concept above, in that it is based on the notion that secondary sexual ornaments are condition-dependent. The condition of an organism is generally held to be based on the combination of many environmental and genetic factors, with the whole genome potentially involved in determining an organism’s condition. Variation in condition is therefore likely to be affected both by mutations on any part of the genome, and by fluctuating selection caused by spatial or temporal variation in selection regime. Condition is therefore likely to have high genetic variance, and thus so is ornament size.

One example of a source of temporal variation in selection regime is due to parasites (Hamilton and Zuk, 1982). This fact was noted before the genic capture hypothesis, but can be seen as a special case of it. Organisms are involved in a perpetual evolutionary arms race against would-be parasites. This means that there is no single optimal genotype across evolutionary time: adaptations and counter-adaptations in both organism and parasites lead to shifting selection. This is known as the ‘Red Queen’ hypothesis, after the scene in Lewis Carroll’s *“Through the Looking-Glass”* (Carroll, 1897), in which the eponymous queen says that “it takes all the running you can do, to keep in the same place” (Figure 1.1). Costly secondary sexual ornamentation is difficult to maintain in an individual that is suffering from parasitic infection. Thus females selecting males with high quality ornamentation will benefit by gaining a mate with the current best in anti-parasitic genes (and also by not directly contracting parasites from their mate, see above), meaning preference genes can maintain positive correlation with the state of the art in adaptations to combat parasitic infection.

### **Compatible genes**

In diploid species, as mentioned above, dominance relationships between maternal and paternal alleles at each locus are likely to affect the fitness of offspring who share alleles from both parents. This fact means that females may often benefit by looking for ‘compatible’ genes rather than good genes (Tregenza and



Figure 1.1: Alice and the Red Queen: “here, you see, it takes all the running you can do, to keep in the same place”. Picture from Carroll, 1897.

Wedell, 2000). There has been some confusion in the field as to the precise distinction between compatible and good genes (Kempnaers, 2007), but it can be resolved (Lynch and Walsh, 1998; Puurtinen et al., 2009). If all the males and all the females in the population were crossed with one another and the reproductive success and offspring fitness recorded, the information could be used to derive breeding values for fitness for all males and females individually, and male-by-female interaction effects (Falconer, 1967; Lynch and Walsh, 1998; Puurtinen et al., 2009). The magnitude of good-genes benefits of female choice would then be estimated as the standard deviation of the male effects on fitness, while the magnitude of compatible-genes benefits for a given female would be the standard deviation of the interaction effect of her with all the males (Puurtinen et al., 2009). Thus while the former is the same for all females, the latter varies from female to female. This variation from female to female means that choice for compatible genes offers another way out of the lek paradox, since genetic variance would be kept higher as differing alleles are subject to varying selection pressures depending on compatibility.

Compatible genes benefits may also explain the evolution of polyandry (Zeh and Zeh, 1996, 1997; Tregenza and Wedell, 2000), particularly where there is cryptic female choice (Thornhill, 1983; Eberhard, 1996; see also section 1.3.2 above), because females may be able to favour sperm with more compatible genes (e.g. in Swedish sand lizards, *Lacerta agilis*; Olsson et al., 1996). It would therefore benefit females

to mate with multiple males in order to widen the chance of finding more compatible sperm.

### 1.4.3 Sensory bias

Earlier it was suggested that the widespread existence of female choice in nature implies that it must be in some way adaptive, leading to a raft of hypotheses for the benefits gained by females who exhibit it. However, there is an alternative explanation for the evolution of mate choice, which doesn't require any benefits to females. This is the sensory bias hypothesis (West-Eberhard, 1984; Basolo, 1990; Endler and Basolo, 1998; Ryan, 1998; Fuller et al., 2005). According to this explanation, natural selection drives the evolution of female sensory systems. Alleles that are favoured for non-sexually selected purposes then have pleiotropic effects that cause females to inherently prefer males with certain traits (Kirkpatrick and Ryan, 1991). Such males will therefore obtain more matings, and so if there is genetic variation in these traits, then secondary sexual characteristics could evolve. This is often referred to as the males 'exploiting' the sensory biases of the females. A key aspect of the sensory bias hypothesis is that female preference is not adaptive for sexually selective reasons; that is, that females do not obtain either direct or indirect benefits by preferentially mating with a certain type of male. Rather, female preference is a side-effect of some other process that is beneficial by natural (i.e. non-sexual) selection.

The problem with this hypothesis is that it is difficult to prove or disprove. Given a population in which the females are shown to have an affinity for a certain colour in both food and males, there are three different explanations possible (Fuller et al., 2005; see end of section 1.4.4 below). Either (as the sensory bias hypothesis postulates) natural selection for favouring this colour came first because females who had an affinity with it got better food. Then males that were closer to this colour were favoured, and so all males evolved to be this colour. Alternatively, sexual selection came first, since males that were this colour provided the largest benefits to females, favouring those who showed mate choice. Females who preferred this colour in mates then also preferred it in food. Finally, it could also be that choice for food and choice for mate colour are independent, and they just happened to have evolved to be the same.

Another way of thinking about the flaws in the sensory bias hypothesis is to imagine what would happen if the best food shared its colour with diseased males. Then natural and sexual selection would be in opposite directions, and the result would presumably be either some resolution of preference, or a breaking down of the link between sexual and food preference.

Finally, it should be noted that, according to one way of thinking (Kirkpatrick, 1996), if the females obtain direct benefits from natural selection because of some sensory bias, and via pleiotropy this also leads to some mate preference, this could be categorised as a form of choice for direct benefits. I have chosen not

to categorise it as such here, since although the benefits in such a case would be for the female herself and not her offspring, they would not be due to sexual selection, but natural selection.

#### **1.4.4 Which kind of reason?**

With such a plethora of explanations for why mate choice might evolve, how do we work out which explanation is the correct one? Unpicking the differences between the different types of explanations is a job for theoretical models (Mead and Arnold, 2004; Fuller et al., 2005; see also section 1.6 below), which can provide insight into the fundamental aspects of each explanation and suggest tests by which they might be distinguished empirically. There is ongoing debate as to the differing levels of importance of direct and indirect benefits, with models often concluding that the effects of indirect benefits are likely to be weaker than those of direct benefits, and thus that preferences for indirect benefits are less likely to evolve (Kirkpatrick, 1996; Kirkpatrick and Barton, 1997; although not everyone agrees, Houle and Kondrashov, 2002). There is more empirical evidence supporting female choice for direct than for indirect benefits (reviewed by Kirkpatrick and Ryan, 1991; Johnstone, 1995; though see also Møller and Jennions, 2001). However, in many cases the different explanations are not mutually exclusive. It is entirely feasible that males might provide females with both direct and indirect benefits, for example. Indeed, it seems likely that Fisher's runaway process, at least, will occur alongside any other process causing the evolution of female preference, since being attractive will itself always be beneficial by sexual selection, regardless of whether there is an underlying reason why a certain phenotype is attractive (Kokko et al., 2003). The mix of forces causing the evolution of mate choice is likely to differ across species. Major tasks are to ascertain the relative importance of each explanation generally, and to work out how much the mix is likely to vary in different circumstances, as well as why, and how we can distinguish the different cases (Bradbury et al., 1987).

To illustrate the difficulty, I now consider the difficulty in determining which of the above explanations is (most) responsible for the evolution of mate choice in the guppy, *Poecilia reticulata*.

#### **Guppy, *Poecilia reticulata***

Guppies are promiscuous, internally-fertilising freshwater fish, which give birth to live young (Endler, 1983; Houde, 1987, 1997; Pitcher et al., 2007). They are sexually dimorphic, with the males displaying bright orange colour patches (Figure 1.2). Females from several populations have been empirically shown to prefer males bearing orange patches that have a more intense hue or saturation (Kodric-Brown, 1989; Houde and Torio, 1992; Grether, 2000). These characteristics are strongly related to the amount of carotenoid present in a male's body (Kodric-Brown, 1989; Grether, 2000).

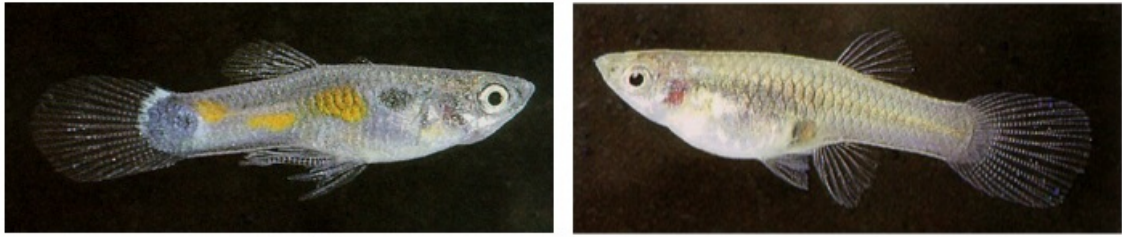


Figure 1.2: Male (left) and female (right) guppies, *Poecilia reticulata*. The species exhibits sexual dimorphism. In many populations, females are attracted to orange markings such as those on this male (see text).

Since animals cannot synthesise carotenoid (Goodwin, 1984), male guppies must obtain it via consumption of algae (Houde, 1997; Grether, 2000), which also form the main food source (Dussault and Kramer, 1981). Thus orange colouration has been hypothesised to honestly indicate to females the foraging ability of a male (Endler, 1980), something which is in turn expected to correlate with heritable fitness (Kodric-Brown and Brown, 1984). This is the genic capture hypothesis (see above): orange colouration stems from foraging ability, which in turn is likely to be determined by a large portion of the genome, and thus provides a large mutational target. In addition, both hue and saturation of the orange patches decrease with parasitic infection (Houde and Torio, 1992; as does male display rate, Kennedy et al., 1987). By preferentially mating with males bearing orange patches with a more intense hue and saturation, females will have offspring that inherit genes for traits that are likely to be closely related to fitness, such as foraging ability and parasite resistance.

However, things are not necessarily so clear. Grether et al. (2001) showed that the orange pigmentation is not caused by carotenoids alone; the patches also contain drosopterin, a type of red pteridine, a pigment that can be synthesised by animals (Hurst, 1980). If females prefer orange males, what is to prevent males from ‘cheating’, that is, from making themselves orange via these alternative pigments and thus giving the appearance of being good foragers regardless of their actual ability? If there is an optimal level of orange colouration for a given male, what prevents him from achieving this via endogenous drosopterin, rather than relying on exogenous carotenoids (Grether et al., 2001)? Looking at it from the other side, what maintains female preference for orange pigmentation if its value as an honest indicator is questionable due to the presence of drosopterin?

Grether et al. mention four mechanisms that have been proposed to enforce honesty in the use of carotenoids as a sexual signal in guppies and other species: that they are costly to obtain due to their scarcity in the environment (Endler, 1980), that parasitic infection affects male ability to use them (Houde and Torio, 1992), that they have an immune function and thus ‘wasting’ them on signalling is costly (Lozano, 1994),

and that they are in themselves toxic (Zahavi and Zahavi, 1997; though this has been questioned, e.g. by Olson and Owens, 1998). They note that the cost of carotenoids would be expected to favour the use of drosopterin as a substitute, rather than guaranteeing the honesty of orange colouration.

Their study, however, revealed that drosopterin does not seem to be used by male guppies to 'cheat'. If this were the case, one would expect that drosopterin and carotenoid concentrations would have negative relationships; the more carotenoid, the less need to use drosopterin, and vice versa. Instead, however, Grether et al. found that across natural populations from areas that differed in natural availability of carotenoids, there was a positive relationship between the two types of pigment. Populations from high-carotenoid environments had on average higher levels of both carotenoid and drosopterin pigmentation than those from low-carotenoid environments. The authors of the study discuss two main explanations for this: firstly that due to the slightly different spectral properties of drosopterin compared to carotenoids, a specific ratio of the two types of pigment is selected for because females have a preference for a specific hue of orange spot; and secondly that drosopterin is also costly, and a male's ability to meet this cost is similar to his ability to meet the cost of carotenoids, so that the two chemicals are positively correlated. There are difficulties with both of these explanations.

There are two difficulties with female preference being for a specific hue. The first is that previous experiments have reported preference as being apparently open-ended in nature (Kodric-Brown, 1989; Grether, 2000). The authors suggest that females may have a "sliding-ratio preference", so that they prefer more orange males, but require the carotenoid-to-drosopterin ratio to be above a certain value. This explanation has the added benefit of providing a way out of the second difficulty, which is to explain why females would evolve a preference for a specific hue if the presence of carotenoids is an honest signal of quality. Perhaps, suggest Grether et al., the preference for (at least) a certain ratio of carotenoid-to-drosopterin is what prevents cheating. Above this ratio, the more carotenoid the better. This does beg the question, however, as to why males use drosopterin at all if females are able to assess the ratio in this way? If no drosopterin was present then a male would automatically achieve the minimum ratio as long as he had some carotenoid pigmentation. As they note, empirical testing is needed to ascertain how attractive different ratios of carotenoid and drosopterin are to females.

The other explanation Grether et al. offer for their findings, that drosopterin and carotenoids have correlated costs, has the difficulty that it requires further explanation as to how such a correlation would come about. Grether et al. suggest that such a correlation could occur if the production of drosopterin is constrained by food intake (perhaps because the metabolic cost of producing drosopterin is high), since carotenoid availability is strongly related to food availability. Of course, if this is the case then drosopterin

is not in fact a way of cheating the honest signal but another, also costly, way of signalling foraging ability and fitness. This would raise the question of why there are two, somewhat overlapping signalling pathways, and whether they represent one combined signal or two separate signals (for a theoretical model of multiple sexual ornaments, see Iwasa and Pomiankowski, 1994).

I suggest an alternative explanation not mentioned by Grether et al. which could potentially account for their result. Their assumption that there is an optimal level of orange colouration for a given male (more accurately, a given male genotype) is likely to be incorrect. Rather, the optimal colouration for a given male genotype may vary with the environment, in particular with how easily carotenoids are obtained, and how much danger there is in being conspicuous. Bright orange colouration makes males more conspicuous to predators (Godin and McDonough, 2003), and is thus costly whether the pigmentation is due to carotenoids or drosopterin. The marginal benefits of increased hue or saturation may vary at different levels of pigmentation and in different environments, so that the eventual mean level of pigmentation in a population is the result of a complex coevolutionary process. This idea is supported by the large variation seen in colour patterns between *Poecilia reticulata* populations (Houde, 1997).

Whatever the reasons for male guppies not using drosopterin to cheat, the finding that they don't means that the study by Grether et al. (2001) doesn't contradict the genic capture hypothesis for the species. The orange colouration of a male guppy would still seem to correlate with foraging ability and parasite avoidance, and thus with "good genes". However, this is not the only possible explanation. Houde (1987) found that females prefer males with larger orange patches, and there is evidence that the area of orange colouration in male guppies is not based on the availability of carotenoids (Kodric-Brown, 1989), but rather is genetically determined by loci linked to the Y chromosome (Houde, 1992). A study by Pitcher et al. (2007) found that the area of orange colouration, but not its hue, saturation, or brightness, correlated positively with key sperm traits, so that males with larger orange patches had significantly longer and more motile sperm, as well as larger sperm loads, than males with smaller orange patches. Pitcher et al. (2007) suggested that females select males with larger orange patches so as to maximise their chances of fertility from a mating. This is the phenotype-linked fertility hypothesis (Sheldon, 1994; see section 1.4.1 above; also chapter 3). Of course, the orange patches could honestly advertise sperm traits via condition-dependence (Sheldon, 1994); if good foraging ability is correlated with high sperm quality then females will benefit from mating with orange males due to good genes and high fertility. But the sperm traits were not found to correlate with hue, brightness or saturation of orange colouration on males (Pitcher et al., 2007; though they also suggest possible experimental reasons for this), and these colour traits are the ones hypothesised to be condition-dependent (Kodric-Brown, 1989). Orange patch area, by contrast, is believed to be genetic

(Houde, 1992). If sperm traits are also genetically determined then they could correlate with area of orange colouration either through the same genes underlying both traits (pleiotropy) or through linkage disequilibrium between the genes for each trait (particularly if the sperm genes are also Y-linked, as seems likely). This suggests that females could benefit by choosing males with large orange patches and thus gaining fertility, or by choosing males with bright orange patches and thus gain by good genes.

Already, distinguishing between these two hypotheses for female choice will be difficult, particularly since there seems to be a large between-population variation in female preference (Endler and Houde, 1995; Grether, 2000). However, there is also a third alternative which further muddies the waters. Rodd et al. (2002) found that both female and male guppies were more responsive to orange-coloured objects than to objects of other colours, even in non-mating contexts. They also found that 94% of the between-population variation in female mating preferences could be explained by between-population variation in this attraction to orange-coloured objects. They claim these results suggest that female mating preferences in this species may have arisen via the ‘sensory bias’ hypothesis (see above): both male and female guppies may benefit from being attracted to orange objects because such an attraction allows them to detect rare food sources such as fruit. Female preference for orange males may then be simply a pleiotropic side-effect of this more general preference. In effect, males are therefore mimicking fruit by being orange. This rebuttal of the genic capture hypothesis is supported by the findings of Grether (2000). If orange colouration was an honest indicator, females should pay more attention to it in populations in which carotenoids are limited, since in these circumstances it will be a more reliable signal of foraging ability. However in two of three river drainage areas in the field that he looked at, this predicted change in preference strength was absent.

There is a counter-argument to the findings of Rodd et al. (2002) however. All that their findings show is that females that are more attracted to orange males are also more attracted to orange objects. This doesn’t tell us anything about whether this preference for orange originally came about through natural selection for females who were good at finding fruit or through sexual selection for females who were good at finding high-quality mates.

To sum up, we are left with three explanatory scenarios (Fuller et al., 2005). Firstly, selection on foraging preferences could have led to female preference for orange males as a side effect (sensory bias). Secondly, it could be the case that the male and female attraction to orange items arose as a side-effect of female mate choice. The genic capture or phenotype-linked fertility hypotheses could be the cause in this scenario. Finally, mating and foraging preferences may be completely or partially independent traits, each evolving to their own optima. Female perception and response is a complex trait, but elements of it (e.g. perception of the colour orange) are likely to be of importance in both foraging and mate choice.



These elements will therefore evolve by both natural and sexual selection (and random drift), with the precise direction of evolution at any given generation dependent on the effect of any change upon fitness as a whole (plus any stochastic effects due to population size). This could be affected by both natural and sexual selection simultaneously. This third explanation seems the most likely: in reality, the dependence relationship between mating and foraging preferences is unlikely to be as simple as the evolution of one occurring as a side-effect of the evolution of the other. Rather they will be interdependent in a complicated way, itself potentially a target for evolution as the covariance between elements of the preference trait changes.

## **1.5 Evolutionary effects of mate choice**

We have seen that sexual selection can be a powerful evolutionary force, causing phenotypic changes in both sexes. But what are the potential consequences of this force at a higher level? It has been suggested that sexual selection can be a force for speciation, and for extinction, as I will now briefly examine.

### **1.5.1 Speciation**

There are many different species concepts, and the decision as to the appropriate one often depends upon the nature of the scientific question one is attempting to answer (Coyne and Orr, 2004). Since we are concerned here with reproduction, it makes sense for us to use the “biological species concept” of Mayr (1942). It has been argued (Coyne and Orr, 2004) that the process of speciation is the process of accumulation of reproductive barriers impeding gene flow between two groups of organisms. Since sexual selection by its very nature affects which organisms mate with which (and, through post-copulatory selection, which matings are successful) it is clear that it could have a large role to play in speeding up speciation. The literature on modelling speciation is vast (see reviews by Turelli et al., 2001; Kirkpatrick and Ravigne, 2002; Gavrillets, 2003) as is that directly concerning sexual selection (see reviews by Panhuis et al., 2001; Ritchie, 2007). Since the field is not directly covered by this thesis I will only briefly outline the main ideas here.

The basic idea behind sexual selection and speciation is that prezygotic reproductive isolation (i.e. that which is due to organisms being unlikely or physically unable to mate) can come about due to sexual selection. If one population of organisms is divided by some geographical barrier into two populations, differences in sexual traits (such as female preferences, or male courtship behaviours or secondary sexual ornaments) between the two populations could evolve over time either through genetic drift, adaptation

to new ecological conditions, pleiotropy, or differences in sexual selection itself. In addition, if the two populations are brought back together (so-called “second contact”), the sexual traits could further evolve apart as individuals are selected to avoid heterospecific matings due to fitness costs of hybrids, a process called “reinforcement”. There is also the possibility that sexual selection itself can cause rapid change between populations, meaning it could have an indirect effect in speeding up speciation even if there is no direct effect. This could certainly be the case where there is sexual conflict (see above) which is expected to lead to rapid sexually antagonistic coevolution (Rice, 1998; Gavrilets, 2000). It has also been suggested (Panhuis et al., 2001) that sexual selection could speed up postzygotic isolation (i.e. that which is due to the failure of the gametes to create viable offspring), via the postcopulatory selective forces mentioned above. Again, sexual conflict has been mooted as potentially having a role to play.

This theory has been tested using comparative methods to see whether taxa with higher levels of sexual selection (usually determined by the levels of ornamentation in males, testes size, or sexual dimorphism) contain more species than those with lower levels (e.g. in passerine birds, Barraclough et al., 1995; in plants, Hodges and Arnold, 1995; in insects, Arnqvist et al., 2000; for more, see review by Panhuis et al., 2001). Although the results are usually in line with the theoretical predictions above, they show (at best) a correlation between sexual selection and speciation, rather than that the former causes the latter. In addition sexual selection may also have an effect on species extinction which would need to be taken into account in studies such as these (see section 1.5.2). Finally, there may be other confounding factors such as body size, life history, ecological or abiotic factors, and of course chance differences. Good studies will try to control for these factors (Owens et al., 1999).

It is also possible to look for population genetic ‘signatures’ of sympatric speciation by sexual selection (Panhuis et al., 2001; Via, 2001). Two that have been suggested are (1) that within species there will be variation in sexual traits which is likely to make matings between individuals from different populations less likely (e.g. in the guppy, *Poecilia reticulata*, section 1.4.4, or the Strawberry poison-dart frog, *Oophaga pumilio*, chapter 5), and (2) that closely related species will show large differences in sexual traits (see review by Panhuis et al., 2001).

In conclusion, there is strong theory to suggest a role for sexual selection in speciation, and some empirical support. More studies need to be done to rule out other explanations and confounding factors.

## **1.5.2 Extinction**

If sexual selection can have a role at the species level in making speciation more likely, then we must also consider whether it can affect the rates of extinction, another species-level event. That adaptations

that are maladaptive under natural selection may evolve due to sexual selection was recognised by Darwin (Darwin, 1871; Cronin, 1993; Morrow and Fricke, 2004). Thanks to female choice, males may be selected to grow secondary sexual ornaments much larger than the optimal size for natural selection. There is therefore a “load due to sexual selection” (Lande, 1980b) which may lead to a greater risk of extinction. An anecdotal example of this is the Irish Elk, *Megaloceros giganteus*, an extinct species which bore huge antlers, suggested to be due to sexual selection (Gould, 1974), and also suggested to have precipitated the extinction of the species at the end of the Ice Age by being an extra burden on a species trying to adapt to new environmental conditions. Experiments have shown that this sexual load really exists, since when sexual selection is artificially removed from a population the net reproductive rate (of females at least) increases (Holland and Rice, 1999). In addition, it has been suggested that sexual selection decreases effective population size and thus may lead more easily to fixation for deleterious alleles (Morrow and Fricke, 2004). However, this latter point is somewhat doubtful - if sexual selection is for ‘good genes’ then in fact it will likely remove deleterious alleles rather than increase their fixation probability (Agrawal, 2001; Siller, 2001; see also chapter 2). There is also the possibility that sexual selection may increase the rate at which populations evolutionarily adapt to new environments (Lorch et al., 2003).

Further adding to the confusion, there have been mixed results from comparative studies on this subject. In mammals, Brashares (2003) found that species exhibiting monogamy were more prone to extinction, while Morrow and Fricke (2004) found no correlation. On the other hand, Moore and Wilson (2002) found that polygynous species were more prone to parasitic infections, another potential cost of sexual selection (though theory suggests that parasite load should in fact be lowered by sexual selection; Hamilton and Zuk, 1982). In birds, upon which most of the comparative studies have been performed, the picture is less confused. Although Legendre et al. (1999) found that “monogamous mating led to a higher extinction risk than did polygynous mating” in passerine birds in New Zealand, other studies have found that male survival rates are lower in sexually dimorphic species than in those that are monomorphic (Promislow et al., 1992, 1994), that the rates of extinction are higher for sexually dimorphic species introduced to islands than for those that are monomorphic (McLain et al., 1995), and that extinction threat correlates with testis size, a measure of post-mating sexual selection (Morrow and Pitcher, 2003).

There is a need for more studies to be done, particularly in taxonomic groups other than birds, to see if the conclusion cautiously stated by Morrow and Pitcher (2003) with regard to birds is more widely true, “sexual selection may therefore be a double-edged process - promoting speciation on the one hand but promoting extinction on the other”.

## 1.6 Modelling mate choice

Since the title of this thesis is “Modelling the Evolution and Consequences of Mate Choice”, I will now address the modelling aspect. The research chapters all contain models that build upon mathematical modelling techniques that are standard in the literature: population genetics, quantitative genetics, and evolutionary game theory. I therefore give a brief introduction to each topic here.

For the purposes of this thesis, I refer to models based on small numbers of genes as ‘population genetics’; models of phenotypic traits controlled by large numbers of genes of small effect I refer to as ‘quantitative genetics’.

### 1.6.1 Population genetics

Evolution is often characterised in textbooks as being a generation-to-generation change in the frequency of alleles within the gene pool of a population (e.g. in Campbell and Reece, 2002). Population genetic models aim to track this evolution by formally analysing the expected effects of various genetic, evolutionary and ecological factors, such as selection, mutation, drift, recombination, and migration, on the genetic composition over time. They have a rich history, from Mendel, who used mathematics to explain the population structure seen in his breeding experiments, through Fisher, Haldane, and Wright, who reconciled Mendelian inheritance and Darwinian evolution by natural selection, forming the Modern Synthesis, all the way to the present day (Bürger, 2000; Campbell and Reece, 2002). Due to this huge quantity of literature I focus only on the basics here.

#### **Mendelian inheritance and Hardy-Weinberg equilibrium**

Darwin is often ascribed to have believed in blending inheritance, so that the offspring genotype would in some way be an average of that of its parents. He was aware, however, that this explanation had problems; the ‘averaging out’ process would, in modern parlance, halve the genetic variance at every generation, and lead inexorably to a uniform population of clones. It was Mendel who discovered the particulate nature of inheritance, and it is this that allows evolution by natural selection to occur. Population genetic models deal with Mendelian inheritance by keeping track of different alleles at each genetic locus. In the simplest case, if we have two alternative alleles, denoted  $A$  and  $a$ , segregating at a single locus, and are modelling a diploid organism, there are three possible genotypes,  $aa$  and  $AA$ , the homozygous genotypes, and  $Aa$ , the heterozygous genotype (we assume that it doesn’t matter which chromosome the alleles are on, so that  $Aa = aA$ ). Mendel’s laws are then as follows: 1) Law of Segregation: that each gamete contains exactly one of the two parental alleles, and 2) Law of Independent Assortment: that both parental alleles are equally

likely to be present in a given gamete, so that  $Aa$  individuals have half  $A$  gametes and half  $a$  gametes (Campbell and Reece, 2002).

From this formulation it is straightforward to see that Mendelian inheritance will not itself erode genetic variance, unlike blending inheritance. This can be seen at our single locus as follows. Suppose the proportion of  $A$  alleles in the gene pool is  $p$  (so that the proportion of  $a$  alleles is  $1 - p$ ) at some generation  $t$ . Then there will be  $p^2$   $AA$  individuals,  $(1 - p)^2$   $aa$  alleles, and  $2p(1 - p)$   $Aa$  individuals. These individuals will therefore produce gametes in the following frequencies: (1)  $p^2$   $A$  gametes, from  $AA$  individuals; (2)  $p(1 - p)$   $A$  gametes from  $Aa$  individuals; (3)  $p(1 - p)$   $a$  gametes from  $Aa$  individuals; (4)  $(1 - p)^2$   $a$  gametes from  $aa$  individuals. The frequency of  $A$  alleles in the gene pool in generation  $t + 1$  is then  $p^2 + p(1 - p) = p$ , and that of  $a$  alleles is  $1 - p$  by a similar calculation. Thus if the gametes mix randomly the same genotypic frequencies will result in the next generation. We can extend this result to show that given any number of possible alleles  $A_1, A_2, A_3, \dots$  with frequencies  $p_1, p_2, p_3, \dots$  the frequency  $p_{ij}$  of a genotype  $A_i A_j$  will simply be  $p_{ij} = p_i p_j$ . This state of affairs is called the Hardy-Weinberg equilibrium. It can be shown to also apply for cases in which there is random mating between organisms (as opposed to just randomly mixed gametes), and with the addition of more algebra it can be extended to cases where there are separate sexes, or X-linked loci (Bürger, 2000).

Though mathematically trivial, the Hardy-Weinberg equilibrium is conceptually important in evolutionary terms: it shows that in the absence of external forces (such as selection, mutation, drift, migration, non-random mating, etc) the frequencies of alleles in each generation will remain constant. Therefore Mendelian inheritance is itself evolutionarily neutral (unlike blending inheritance).

### **Selection**

There are many ways of incorporating selection into a population genetics model. Here we go through a simple one-locus diploid model with two alternative alleles, denoted  $A$  and  $a$ . This formulation of the model is adapted from that found in Britton (2003). As above, there are three possible genotypes,  $aa$ ,  $Aa$ , and  $AA$ . Though natural selection occurs at the phenotypic level, we assume that the population is large enough so that environmental effects on fitness can be averaged out, so that we can consider average fitness of each genotype as a constant based only on the alleles it has. We define this relative to the fitness of an  $AA$  individual for simplicity, so that relative fitnesses are as given in Table 1.2. The trajectories and results of evolution are then determined by the values of  $h$  and  $s$  that we use.

We assume that all genotypes have the same fertility and fecundity, and that gametes have the same survival regardless of their genotype. We also assume random mating. We want to see how the frequency

Genotype	Relative fitness
$AA$	1
$Aa$	$1 - hs$
$aa$	$1 - s$

Table 1.2: Typical single-locus population genetic fitness scheme. There are two alleles,  $A$  and  $a$ , giving three possible genotypes, with fitnesses as shown. The values of  $h$  and  $s$  affect the evolutionary outcome.

of alleles changes over time. We start at generation  $t$ , with a frequency  $p$  of  $A$  alleles. Then (since there are only  $A$  and  $a$  alleles possible), the frequency of  $a$  alleles will be  $(1 - p)$ . Because we are assuming random mating the frequencies of the three genotypes before selection will therefore be as in the Hardy-Weinberg case above, so that the ratio of genotypes  $AA : Aa : aa$  will be  $p^2 : 2p(1 - p) : (1 - p)^2$ . To find out what this ratio will become after selection we multiply the frequency of a genotype by its fitness to give  $p^2 : 2(1 - hs)p(1 - p) : (1 - s)(1 - p)^2$ . This gives a total of  $p^2 + 2(1 - hs)p(1 - p) + (1 - s)(1 - p)^2$ , so we divide by this to obtain the new frequencies of each genotype. Since we are assuming all individuals that survive this long will mate at random, we need only find out the allele frequency  $p'$  at this point to fully describe the next generation. This will be the frequency of  $A$  gametes, which will be equal to the frequency of  $AA$  individuals plus half the frequency of  $Aa$  individuals:

$$\begin{aligned}
 p' = f(p) &= \frac{p^2 + (1 - hs)p(1 - p)}{p^2 + 2(1 - hs)p(1 - p) + (1 - s)(1 - p)^2} \\
 &= p + p(1 - p) \frac{p(1 - (1 - hs)) + (1 - p)((1 - hs) - (1 - s))}{p^2 + 2(1 - hs)p(1 - p) + (1 - s)(1 - p)^2} \quad (1.1)
 \end{aligned}$$

The frequency of an allele at its next generation is completely determined by this equation, a version of the famous **Fisher-Haldane-Wright equation** (Britton, 2003). There are always fixed points at  $p^* = 0$  and  $p^* = 1$ , at which  $A$  and  $a$  are fixed in the population (note that this model does not include mutation). Whether there is a fixed point  $0 < p^* < 1$  will depend on the parameter values  $h$  and  $s$ . If  $s = 0$  then  $AA$  and  $aa$  genotypes have the same fitness, and from equation (1.1) we can see that  $p' = p$  for all values of  $p$ , so any frequency is stable. Let us assume, then, without loss of generality, that  $0 < s < 1$  so that the  $aa$  genotype is less fit than the  $AA$  genotype. The, since all fitnesses must be positive,  $1 - hs > 0$  and so  $h < 1/s$ . Under what conditions do we expect to retain the  $a$  allele in the population? We want to find out for what values of  $h$  there will be a value  $0 < p^* < 1$  such that  $p^* = p^*$ . Rearranging (1.1) gives us that this

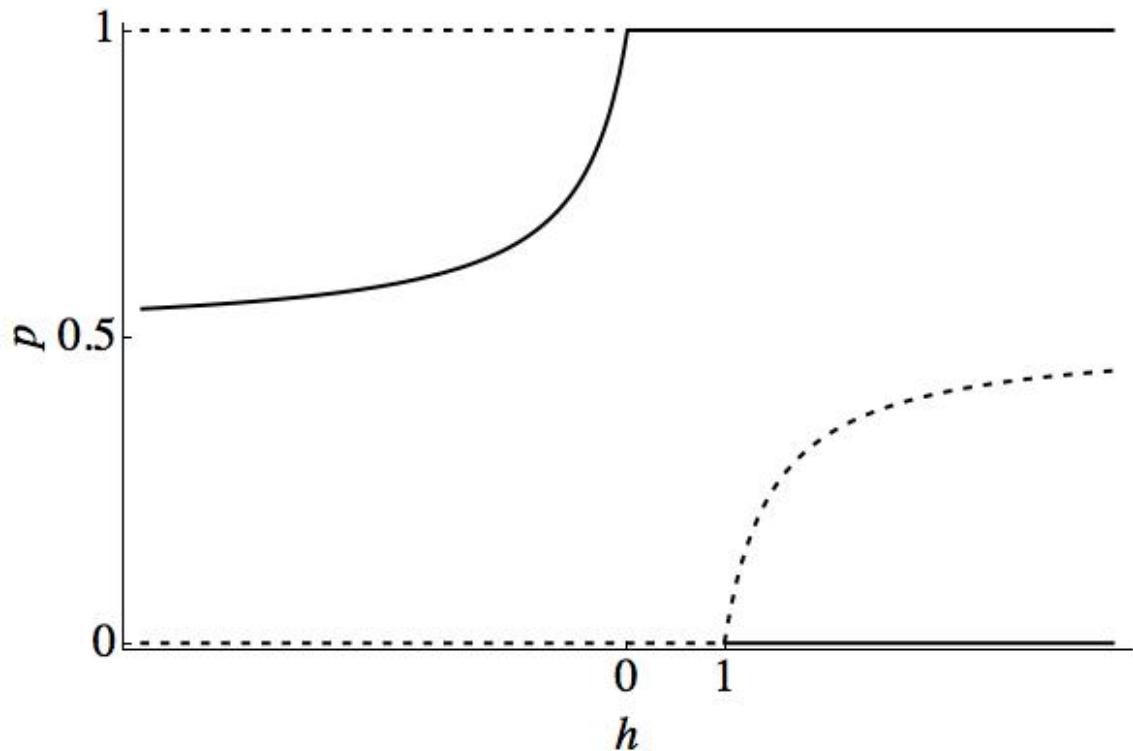


Figure 1.3: Equilibrium gene frequencies in one-locus, two-allele model under selection scheme where  $0 < s < 1$  for different  $h$ -values. The  $AA$  homozygote is always fitter than the  $aa$  homozygote. Solid black lines represent stable equilibria, dashed black lines represent unstable equilibria. For  $h < 0$ , the  $Aa$  heterozygote is fitter than both  $AA$  and  $aa$  homozygotes, and there is a stable polymorphic equilibrium at  $p^* = (h-1)/(2h-1)$  in addition to unstable equilibria at  $p^* = 0$  and  $p^* = 1$ . For  $0 < h < 1$ , the  $AA$  genotype is the fittest, followed by the  $Aa$  homozygote, followed by the  $aa$  homozygote. There is a stable equilibrium at  $p^* = 1$ , and an unstable equilibrium at  $p^* = 0$ . Finally, for  $h > 1$  the heterozygote  $Aa$  is the least fit of the three genotypes. There are two stable equilibria, at  $p^* = 0$  and  $p^* = 1$ , and an unstable equilibrium at  $p^* = (h-1)/(2h-1)$ .

will occur when

$$p^*(1 - (1 - hs)) + (1 - p^*)((1 - hs) - (1 - s)) = 0,$$

giving

$$p^* = \frac{h-1}{2h-1}. \quad (1.2)$$

This will give values  $0 < p^* < 1$  only when  $h < 0$  or  $h > 1$ . If we look at what this means, for  $h < 0$ , we have  $1 - hs > 1 > 1 - s$ , so the fitness of an  $Aa$  heterozygote is larger than the fitness of both homozygotes  $AA$  and  $aa$ . For  $h > 1$ , we have  $1 > 1 - s > 1 - hs$ , so the fitness of the heterozygote is smaller than the fitness of both homozygotes  $AA$  and  $aa$ . So we can have a balanced polymorphism either if there is a heterozygote fitness advantage (e.g. famously in sickle cell anemia in humans, Pauling et al., 1949), or if there is a heterozygote fitness disadvantage (e.g. in situations where hybrids are at a disadvantage).

Of course we are concerned not just with the location of the equilibria, but also with their stability. We can establish this by finding the derivative  $f'(p)$  with respect to  $p$  at each fixed point value, from equation (1.1). For  $p^* = 1$ ,  $f'(1) = 1 - hs$  and so a population in which there are only  $A$  alleles is stable if  $1 - hs < 1$ , i.e. if  $AA$  homozygotes are fitter than  $Aa$  heterozygotes. On the other hand, for  $p^* = 0$ ,  $f'(0) = (1 - hs)/(1 - s)$  and so a population in which there are only  $a$  alleles is stable if  $1 - hs < 1 - s$ . So populations with only one allele type (where  $p = 0$  or  $p = 1$ ) can be invaded only if the heterozygote fitness is higher than that of the homozygote of the most common allele. This makes sense because when a mutant allele first crops up it will be found most commonly in heterozygotes. If they are fitter the mutant will therefore spread in the population. This is that case for all  $h$ -values unless  $0 < h < 1$ . These are exactly the values for which there is no balanced polymorphic equilibrium. Finally, for  $p^* = (h - 1)/(2h - 1)$ , we get  $f'(p^*) < 1$  for  $h < 0$ , when heterozygotes are fitter than both homozygotes, and  $f'(p) > 1$  for  $h > 0$ , when heterozygotes are less fit than both homozygotes. Figure 1.3 shows the full picture of equilibrium points and their stability plotted against  $h$ .

Of course this model is a very simplistic way of representing evolution. In reality other effects such as mutation and migration, and random drift caused by finite populations, will also affect the picture. These have also been successfully incorporated into population genetics models. Examples are given in textbooks such as Bürger (2000) or Britton (2003). Similarly, selection will occur at multiple loci, which may not segregate independently (e.g. there is genetic hitch-hiking, Maynard Smith and Haigh, 1974).

### Limitations and drawbacks

One drawback to population genetics is its lack of information concerning the phenotype-genotype map. This is the mapping from the space of genotypes to the space of actually realised phenotypes in the population. It may be environmentally dependent. Population genetics models assign fitnesses to genotypes. These are taken to be constant parameters, the same in each generation. This will not in fact be the case for two reasons (Lewontin, 1974). Firstly, in many cases the population will not be large enough for environmental effects to be evenly distributed so that the mean effect is the same for all genotypes in each generation. Secondly, the fitness of genotypes is defined relative to the fitness of other genotypes in the population. But this will then change depending upon the genetic makeup of the population from one generation to the next. This could be handled using some sort of frequency-dependent fitness function, as in evolutionary game theory below.

In a plenary speech to a Cold Spring Harbor Symposium on Quantitative Genetics in 1959, Ernst Mayr famously referred to theoretical population genetics as being “beanbag genetics” (Mayr, 1959). This vivid



metaphor states that that gene-pool models are like sampling from a bag of coloured beans, the implication being that such an approach misses much of the important complexity of evolutionary processes, including linkage between alleles, epistasis, and dominance effects. J.B.S. Haldane responded by arguing wittily for the importance of mathematical theory in science in general, not just in evolutionary biology, because of the imprecision of verbal reasoning (Haldane, 1964). The merits of this argument have been debated (Borges, 2008; Ewens, 2008), and it has also been suggested that Mayr's initial complaint was due to a lack of mathematical understanding on his part (Provine, 2004; Borges, 2008), and that in some way the debate foreshadowed the later debates on the units of selection. Mathematical population genetics has become more advanced since this original debate took place (indeed, Haldane himself pointed out the mathematical simplicity of much of the work done by himself, Fisher, and Wright; Haldane, 1964). Many models now incorporate epistatic effects, hitch-hiking, and linkage disequilibrium, and the ubiquity of mathematical modelling in evolutionary biology has been extremely beneficial (not least to me in allowing me to study the subject). Any value Mayr's comments may have had in 1959 is now outdated (Crow, 2001, 2008).

## 1.6.2 Quantitative genetics

In this work we define quantitative genetics models as “models that describe the evolution of continuously distributed traits that are affected by many genes” (Mead and Arnold, 2004). These models have been extensively used to investigate the coevolution of female preferences and male ornaments, and are especially suited to such situations because it is generally believed that these phenotypic traits are affected by many genes. We briefly go through the background to the basic quantitative genetic model of sexual selection, before looking at some more general findings. Firstly, however, we look at the concept of the **G**-matrix.

### The **G**-matrix

Quantitative genetics models assume that the phenotypic traits under investigation (in the case of sexual selection models, typically ornament and preference) are continuously distributed and are governed by many genes, each of which has small effect. Unlike with the population genetics models above, then, no attempt is made to track the frequency of each allele at each locus. Rather the overall statistical properties of the population are what is modelled; the mean phenotype, and how it changes over time.

To capture the genetic inheritance of the traits from one generation to the next, a matrix known as the **G**-matrix is used. This is the genetic variance-covariance matrix for the traits. At its simplest, with just two

traits ( $x$  and  $y$ , say) being modelled, the  $\mathbf{G}$ -matrix is

$$\mathbf{G} = \begin{pmatrix} G_x & B \\ B & G_y \end{pmatrix},$$

where  $G_x$  and  $G_y$  are the additive genetic variances in  $x$  and  $y$  respectively, and  $B$  is the additive genetic covariance between the traits.  $B$  therefore represents the degree to which genes for large values of trait  $x$  are likely to be associated with genes for large values of  $y$ . If we take  $x$  to be male ornament size and  $y$  to be female preference, then females with large  $y$ -values will be more likely to mate with males with large  $x$ -values. Their offspring will therefore bear genes coding for large  $x$  and  $y$ , and so the genetic covariance between the two,  $B$ , will be positive. It is this fact that leads to Fisher's runaway, as will be seen below. With more traits, the  $\mathbf{G}$ -matrix will include the additive genetic variance of each, and the covariances between them. Needless to say this can become quite complex.

The  $\mathbf{G}$ -matrix is not simply a theoretical abstraction. If we imagine a population of organisms with traits  $x$  and  $y$ , we could mate all the males and all the females together, and measure the resultant offspring. The genetic value for  $x$  and  $y$  for each individual would be the average of their offspring's  $x$ - and  $y$ -values respectively (Falconer, 1967). We could then plot the genetic values of each individual in the population to get a 'cloud' of values, the spread and shape of which would be represented by the  $\mathbf{G}$ -matrix, representing as it does the additive genetic variance in  $x$ ,  $y$ , and the covariance between them.

Most models assume the  $\mathbf{G}$ -matrix to be constant over the timeframe of the model, with the variance eroded by selection being balanced by new variance generated by mutation and recombination. One reason for this assumption is simply that it is well known that running out of available genetic variance will limit evolution, and thus it is of more interest when modelling to find out what dynamics will be produced in the absence of this limiting factor. However, the assumption is also potentially biologically appropriate, at least over relatively short evolutionary timescales or for phenotypic changes that are not too great (Kirkpatrick and Barton, 1995; see further discussion below). There also exist models incorporating evolutionary changes in the  $\mathbf{G}$ -matrix (Kirkpatrick, 1996).

### **Derivation of a classic model**

The original quantitative genetic model for sexual selection was that of Lande (1981). It assumes there is no selection on female preference, generally seen as a weakness of the model, and replaced in many more recent models (reviewed in Mead and Arnold, 2004). Instead of going through that model, then, here we go through a model based on the formalism of Iwasa et al. (1991). This incorporates a cost to choice, and

can handle frequency-dependent selection. It also does not require the genetic values in the population to be normally distributed, but rather assumes weak selection, so that the individuals in the population do not differ a large amount in fitness.

Denote male ornament by  $z$  and female preference by  $p$ . Note that ‘ornament’ could in fact be any behavioural or physical trait involved in courtship, while ‘preference’ could be anything affecting the propensity of females to mate with males bearing the ornament. We assume both traits are autosomally inherited, and also that they are sex-linked, so that only males bear ornaments and only females bear preferences. The genes coding for the traits are present in both sexes, however.

Using the quantitative genetics techniques alluded to above, and detailed in work by Falconer (1967) and Bulmer (1980), we can decompose any trait  $i$  into an additive genetic value  $g_i$ , and an error value  $e_i$  (note that the error value is often referred to as the environmental component, but strictly speaking it could be a result of both environmental and nonadditive genetic factors) so that  $i = g_i + e_i$ , and the error terms have some distribution  $f(e_i)$  with mean 0. Natural selection acts upon phenotypes, so suppose we have an expression  $W[i|\bar{p}, \bar{z}]$  for the expected fitness of an individual with phenotype  $i$ . We want to derive from this an expression  $\tilde{W}[g_i|\bar{p}, \bar{z}]$  for the expected fitness of a given genotype  $g_i$  in a population with mean phenotypes  $\bar{p}, \bar{z}$ . Given the distribution of error terms  $f(e_i)$ , we have

$$\tilde{W}[g_i|\bar{p}, \bar{z}] = \int W[g_i + e_i|\bar{p}, \bar{z}]f(e_i)de_i,$$

which (since we assume weak selection) we can simplify by Taylor expanding around the mean error  $e_i = 0$  to get

$$\begin{aligned} \tilde{W}[g_i|\bar{p}, \bar{z}] &= \int \left( W[g_i|\bar{p}, \bar{z}] + e_i \frac{\partial W[g_i|\bar{p}, \bar{z}]}{\partial i} + \frac{1}{2} e_i^2 \frac{\partial^2 W[g_i|\bar{p}, \bar{z}]}{\partial i^2} + \text{higher order terms} \right) f(e_i)de_i \\ &= W[g_i|\bar{p}, \bar{z}] + \text{small terms.} \end{aligned}$$

Neglecting the small terms means we have  $\tilde{W}[g_i|\bar{p}, \bar{z}] \approx W[g_i|\bar{p}, \bar{z}]$  and we can therefore consider the expected fitness of genotypes rather than of phenotypes. For simplicity of notation we will from now on denote genotypes for preference and ornament as  $p$  and  $z$  respectively rather than as  $g_p$  and  $g_z$ .

Since there are genes for both ornament and preference, all organisms have a genotype of the form  $(p, z)$ . However, selection is different on males and on females. Therefore we split the fitness function  $W[p, z|\bar{p}, \bar{z}]$  into that for females,  $W_f[p, z|\bar{p}, \bar{z}]$ , and that for males,  $W_m[p, z|\bar{p}, \bar{z}]$ . We assume the population ratio to be equal so that a given genotype  $(p, z)$  has equal chance of being in a male or a female, and thus has expected

fitness

$$W[p, z|\bar{p}, \bar{z}] = \frac{1}{2} (W_f[p, z|\bar{p}, \bar{z}] + W_m[p, z|\bar{p}, \bar{z}]). \quad (1.3)$$

Now let us calculate the expected change in the mean preference trait  $\bar{p}$  from one generation to the next. Suppose we have a joint frequency distribution  $\phi(p, z)$  for genotypes  $(p, z)$ . Mean preference  $\bar{p}$  in a given generation will be

$$\bar{p} = \int_Z \int_P p \phi(p, z) dp dz,$$

(where  $P$  and  $Z$  are the domains from which our  $p$ - and  $z$ -values respectively come) since this is the  $p$ -value for each genotype multiplied by its frequency, integrated across all possible genotypes  $(p, z)$ . Since each genotype has a given fitness  $W[p, z|\bar{p}, \bar{z}]$ , we can calculate mean preference  $\bar{p}^*$  in the next generation by multiplying the  $p$ -value for each genotype by its frequency and its fitness, integrating across all genotypes, and then (to normalise) dividing by the mean fitness:

$$\bar{p}^* = \frac{\int_Z \int_P p W[p, z|\bar{p}, \bar{z}] \phi(p, z) dp dz}{\int_Z \int_P W[p, z|\bar{p}, \bar{z}] \phi(p, z) dp dz}. \quad (1.4)$$

Since we want to track the evolution of  $\bar{p}$  we are interested in  $\Delta\bar{p} = \bar{p}^* - \bar{p}$ , which from equation (1.4) can be given as

$$\begin{aligned} \Delta\bar{p} &= \frac{\int_Z \int_P p W[p, z|\bar{p}, \bar{z}] \phi(p, z) dp dz - \bar{p} \int_Z \int_P W[p, z|\bar{p}, \bar{z}] \phi(p, z) dp dz}{\int_Z \int_P W[p, z|\bar{p}, \bar{z}] \phi(p, z) dp dz} \\ &= \frac{E[pW[p, z|\bar{p}, \bar{z}]] - E[p]E[W[p, z|\bar{p}, \bar{z}]]}{\bar{W}} \\ &= \frac{\text{Cov}[p, W[p, z|\bar{p}, \bar{z}]]}{\bar{W}}, \end{aligned} \quad (1.5)$$

where  $E[a]$  is the expectation of a trait  $a$ ,  $\text{Cov}[a, b]$  is the covariance of two traits  $a$  and  $b$ , and  $\bar{W}$  is the population mean fitness. A similar equation to (1.5) can be derived in the same way for  $\Delta\bar{z}$ ,

$$\Delta\bar{z} = \frac{\text{Cov}[z, W[p, z|\bar{p}, \bar{z}]]}{\bar{W}}. \quad (1.6)$$

Because we assume weak selection we can Taylor expand our fitness function (equation (1.3)) around the

mean  $(\bar{p}, \bar{z})$  as

$$\begin{aligned}
 W[p, z|\bar{p}, \bar{z}] &= \frac{1}{2}W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}] + \frac{1}{2}W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}] + \frac{1}{2}(p - \bar{p})\frac{\partial W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}]}{\partial p} \\
 &+ \frac{1}{2}(p - \bar{p})\frac{\partial W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]}{\partial p} + \frac{1}{2}(z - \bar{z})\frac{\partial W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}]}{\partial z} \\
 &+ \frac{1}{2}(z - \bar{z})\frac{\partial W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]}{\partial z} + \text{higher order terms.} \tag{1.7}
 \end{aligned}$$

Generally, we want female fitness to depend only upon the preference the female bears and not on the unexpressed genes for ornament that she has. Similarly, male fitness should depend only upon upon the ornament the male bears and not on the unexpressed genes for preference that he has. Therefore  $\partial W_f[p, z|\bar{p}, \bar{z}]/\partial z = \partial W_m[p, z|\bar{p}, \bar{z}]/\partial p = 0$  for all genotypes  $(p, z)$ . This allows us to simplify equation (1.7) a bit further as we can disregard these terms.

Then we can approximate  $\text{Cov}[p, W[p, z|\bar{p}, \bar{z}]]$  as

$$\begin{aligned}
 \text{Cov}[p, W[p, z|\bar{p}, \bar{z}]] &= \frac{1}{2}\text{Cov}\left[p, W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}]\right] + \frac{1}{2}\text{Cov}\left[p, W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]\right] \\
 &+ \frac{1}{2}\text{Cov}\left[p, (p - \bar{p})\frac{\partial W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}]}{\partial p}\right] \\
 &+ \frac{1}{2}\text{Cov}\left[p, (z - \bar{z})\frac{\partial W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]}{\partial z}\right] \\
 &+ \dots \\
 &\approx \frac{1}{2}G_p \frac{\partial W_f[p, z|\bar{p}, \bar{z}]}{\partial p}\bigg|_{p=\bar{p}, z=\bar{z}} + \frac{1}{2}B \frac{\partial W_m[p, z|\bar{p}, \bar{z}]}{\partial z}\bigg|_{p=\bar{p}, z=\bar{z}}, \tag{1.8}
 \end{aligned}$$

where  $G_p$  is the additive genetic variance of  $p$ ,  $B$  is the covariance between  $p$  and  $z$ , and the partial derivatives (as noted) are calculated at the population mean values. Again, a similar expression can be derived for  $\text{Cov}[z, W[p, z|\bar{p}, \bar{z}]]$ .

We can also use equation (1.7) to get

$$\bar{W} \approx \frac{1}{2}\left(W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}] + W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]\right), \tag{1.9}$$

and we note that since mean fitness of males and females must be equal (because every offspring has one mother and one father),

$$W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}] \approx W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]. \tag{1.10}$$

We now plug equations (1.8) and (1.9) into equation (1.5), and then use equation (1.10) to give

$$\begin{aligned}\Delta\bar{p} &\approx \frac{G_p(\partial W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}]/\partial p)}{W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}] + W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]} + \frac{B(\partial W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]/\partial z)}{W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}] + W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]} \\ &\approx \frac{G_p(\partial W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}]/\partial p)}{2W_f[\bar{p}, \bar{z}|\bar{p}, \bar{z}]} + \frac{B(\partial W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]/\partial z)}{2W_m[\bar{p}, \bar{z}|\bar{p}, \bar{z}]} \\ &= \frac{1}{2}G_p \left. \frac{\partial \ln W_f[p, z|\bar{p}, \bar{z}]}{\partial p} \right|_{p=\bar{p}, z=\bar{z}} + \frac{1}{2}B \left. \frac{\partial \ln W_m[p, z|\bar{p}, \bar{z}]}{\partial z} \right|_{p=\bar{p}, z=\bar{z}}\end{aligned}$$

Since we can get a similar equation for  $\Delta\bar{z}$ , we can track the evolution of the mean genotype  $(\bar{p}, \bar{z})$  by using the matrix equation

$$\begin{pmatrix} \Delta\bar{p} \\ \Delta\bar{z} \end{pmatrix} = \frac{1}{2} \begin{pmatrix} G_p & B \\ B & G_z \end{pmatrix} \begin{pmatrix} \beta_p \\ \beta_z \end{pmatrix} = \frac{1}{2} \mathbf{G}\boldsymbol{\beta} \quad (1.11)$$

where  $\beta_p = \partial W_f/\partial p$  and  $\beta_z = \partial W_m/\partial z$ , both evaluated at  $p = \bar{p}$ ,  $z = \bar{z}$ ,  $\boldsymbol{\beta} = (\beta_p, \beta_z)^\top$ , and  $\mathbf{G}$  is of course the  $\mathbf{G}$ -matrix. The interesting thing to note is that changes in mean preference and mean ornament can occur due to covariances between the two, so that even in the absence of direct selection on one of these traits there can be change (for example if  $\beta_p = 0$ ,  $\beta_z > 0$ , and  $B > 0$ , then  $\Delta\bar{p} > 0$ , as could possibly initiate Fisher's runaway procedure).

### Added complexity

The simplicity of equation (1.11) (and that of other, similar, models) means that there is lots of scope for adaption to take into account a variety of scenarios. Female preference can be modelled as being open-ended, absolute, or relative. Open-ended preference is directional, so that the larger (or smaller) the ornament the more preferred the male (e.g. Iwasa et al., 1991; Pomiankowski et al., 1991; Pomiankowski and Iwasa, 1993; Kirkpatrick, 1996). Absolute preference is when females are most likely to mate with males who bear a particular ornament value (e.g. Kirkpatrick, 1985; see also chapter 5). Relative preference is when females prefer males with ornament value a certain size relative to the population mean (e.g. Houle and Kondrashov, 2002). These three preference schemes were first characterised by Lande (1981).

In addition to this layer of complexity, different natural selection schemes can be incorporated affecting either ornaments and/or preferences. Most commonly these are stabilising, but occasionally directional (e.g. Hall et al., 2000), or cubic 'plateau' schemes have been used (e.g. Iwasa and Pomiankowski, 1995).

Finally, these models can be used to model many of the reasons behind female choice detailed above, such as sexy son (e.g. Pomiankowski and Iwasa, 1993), good genes (e.g. Iwasa et al., 1991; Iwasa and Pomiankowski, 1994; Kirkpatrick, 1996; Houle and Kondrashov, 2002), good parent (e.g. Kirkpatrick,

1985; Iwasa and Pomiankowski, 1999), and even sexual conflict (e.g. Gavrilets, 2000; Gavrilets et al., 2001).

### **Possible results**

We plot the expected evolutionary trajectories of quantitative genetics models as phase plots (Figure 1.4). They generally produce one of a few possible results (Mead and Arnold, 2004). The classic model by Lande (1981) gave two possibilities: evolutionary trajectories either ‘walk towards’ a line of equilibria (Figure 1.4a), or ‘run away’ from it (Figure 1.4b). In the former case evolution may proceed by drift along the line (see also chapter 5). In the latter case evolution will tend towards larger and larger ornaments and preferences, as Fisher’s verbal model suggested (Fisher, 1930). With the addition of a cost to female choice the line of equilibria collapses to a point (Figure 1.4c), as it does in other models (see examples given by Mead and Arnold, 2004). Depending upon the details such a point could be stable or unstable, and may correspond to an enlargement of preference and ornament size past the natural selection optima or not. Finally, under some circumstances, the model results in evolution driving trajectories to a stable limit cycle (Figure 1.4d; e.g. Iwasa and Pomiankowski, 1995; chapter 11 of Otto and Day, 2007), where periods of exaggeration in ornament and preference will alternate with periods of no exaggeration (or even of diminution) as the cycle is followed. The diagrams given in Figure 1.4 are sketches only; for details of the exact models see the citations given. For an analysis of which models produce which results see the review by Mead and Arnold (2004).

### **Limitations and drawbacks**

One criticism of quantitative genetics models of the kind described here is that there is much evidence that the **G**-matrix is not in reality constant, but rather evolves (reviewed by Steppan et al., 2002; Arnold et al., 2008). How much this matters depends upon the timescale over which the model applies, and the type of evolution undergone by the **G**-matrix. Theoretical approaches to the latter question as a general problem are intractable unless potentially unrealistic assumptions are made (Turelli, 1988) and so empirical and computer simulation techniques have been used (reviewed by Steppan et al., 2002; Arnold et al., 2008). These suggest that the **G**-matrix is relatively stable under many circumstances but that this stability can “be prone to erratic wobbling” (Mead and Arnold, 2004) in other circumstances (Jones et al., 2003). Research into this is ongoing, but at this stage we can tentatively suggest that assuming the **G**-matrix to be constant is generally biologically wrong, but as a simplifying assumption is often fine from a modelling perspective, depending on the details of what is being modelled.

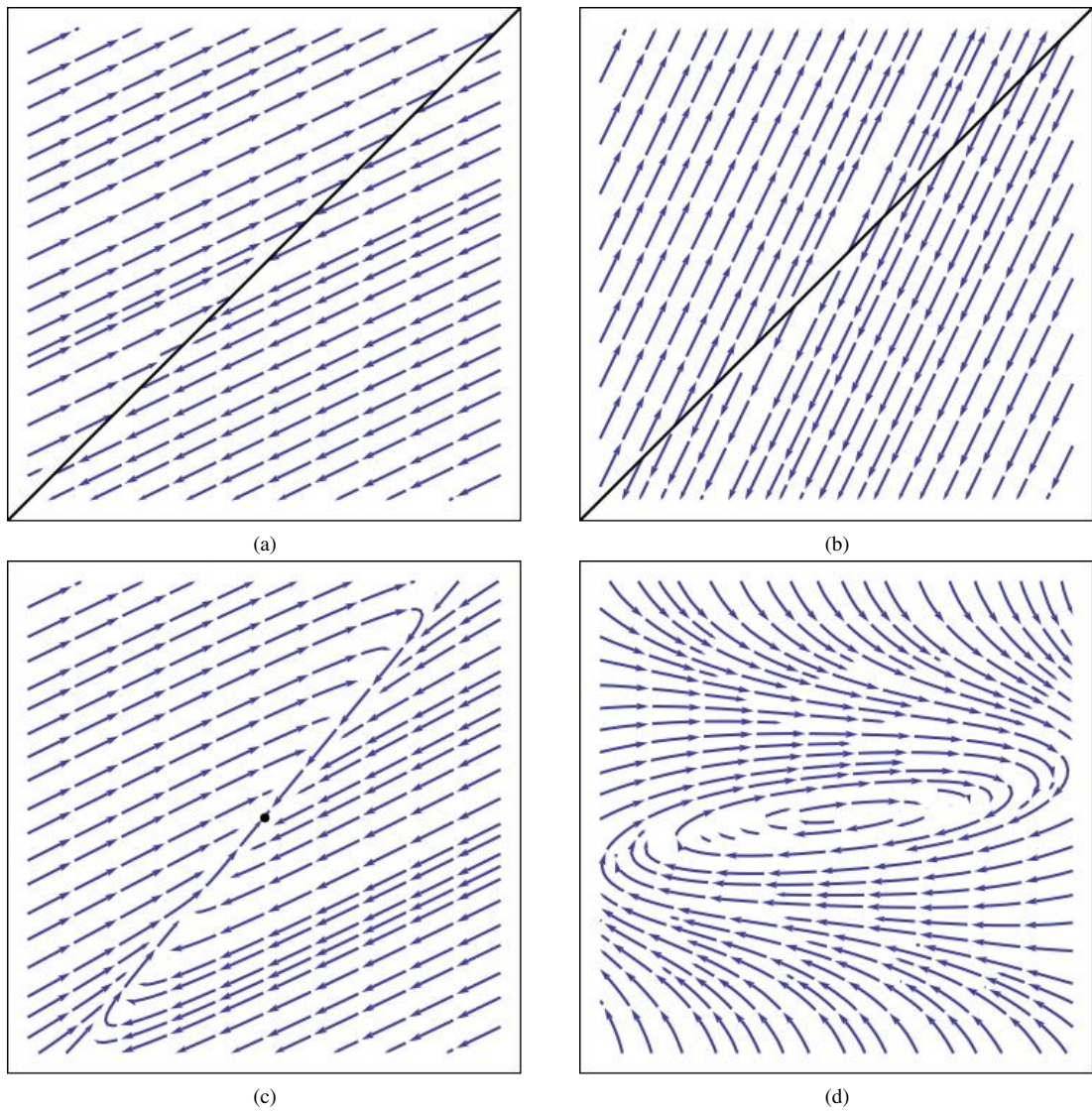


Figure 1.4: Examples of solution phase portraits from quantitative genetics models. (a) ‘walk-towards’ line of equilibria; (b) ‘runaway’ from line of equilibria; (c) stable equilibrium point; (d) attracting limit cycle. Either axis could represent preference or ornament size. The lines of equilibria in (a) and (b) are marked by a black line. The equilibrium point in (c) is marked with a point. There is a limit cycle in (d) but it has not been marked.



The infinitesimal model upon which quantitative genetics models are based assumes that phenotypic characters of interest are controlled by an effectively infinite number of genes of infinitesimally small effect. However, theory suggests that much adaptation occurs as a result of a small number of mutations of large effect (Orr, 2003), an idea supported by empirical studies that show an L-shaped distribution of effect sizes for Quantitative Trait Loci (Bost et al., 2001; and references therein). Although there may be many thousands of genes contributing to a given character, they do not all have small effect; rather genes of large effect do exist and are evolutionarily important, particularly where adaptive mutation is concerned (Bell, 2010). Therefore it has been suggested (Bell, 2010) that the infinitesimal model of evolution be replaced with an oligogenic model, whereby evolution proceeds by strong selection favouring mutations of large effect. There are problems with this model too, however, such as the high heritability of traits (even those closely related to fitness). If selection is strong, and adaptation takes place through mutations of large effect, these mutations should quickly become fixed, meaning low heritability, but instead many traits seem to be highly heritable (Pomiankowski and Møller, 1995; Weigensberg and Roff, 1996). Quantitative genetics models will have to develop to take into account information provided by modern molecular biology, but they remain the best way of understanding and interpreting the results (Walsh, 2001), whether the underlying assumptions are those of an infinitesimal or an oligogenic model.

### **1.6.3 Evolutionary game theory models**

The field of evolutionary game theory is generally held to have begun with the paper by Maynard Smith and Price (1973) which introduced the concept of an evolutionarily stable strategy (ESS), although arguments of a game-theoretic type had already been used by Fisher (1930) and by Hamilton (1967) (Maynard Smith, 1982; Hammerstein and Selten, 1994). The mathematical field of game theory was formalised long before this (Von Neumann and Morgenstern, 1944), but it carried the assumption that the players were rational, self-interested agents. This may have been fine for humans (although even here there are doubts, e.g. see Gigerenzer and Selten, 2001) but could not be accepted for biological needs. Thus the foundations of evolutionary game theory are slightly different from those of its economic cousin.

#### **Biological background**

We assume that the strategies played in the game under consideration are programs that completely control the behaviour of an individual organism, and are inherited. It is the frequency of these strategies in the population that we consider, rather than the fate of any individual organism. The population of individuals under consideration is assumed to be infinite, reproduction is assumed to be asexual, and either all contests

are assumed to be pairwise so that each contest takes place between two players (Maynard Smith, 1982), or contests are assumed to take place between a focal individual (more precisely, his/her strategy) and all members of the population (called “playing the field” games; Hammerstein and Selten, 1994; we shan’t consider such population games here).

The organisms being modelled thus do not choose their strategy according to rational self-interest, but rather inherit their strategy from their parent. The relative merits of a strategy are determined by the pay-off function. It is by this that we are able to analyse the changes in frequency of each strategy in the population over time. Whereas in economic game theory the pay-off functions are in terms of ‘utility’, in evolutionary game theory they are in terms of ‘fitness’, generally described as reproductive success. Since the pay-off to a player will depend on what strategy he/she plays, and what is played by his/her opponent, game theory describes frequency-dependent selection; that is, the merits of each strategy depend upon the frequency of strategies being played by others in the population.

In general it is not assumed that at the outset of the game every possible strategy is being played in the population. Rather, we assume that by mutation (or, in models based on learning rather than strict genetic inheritance, by innovation) new strategies can and do arise during the game. Since they have arisen by chance mutation, novel strategies are assumed to initially appear in the population at very low frequencies. This is important, and has a bearing on the equilibrium concept that we will introduce.

At every generation the individuals in the population randomly pair up and play the game according to their inherited strategies. We assume there is no spatial structure inherent in the population, so that strategies encounter one another based on their frequencies alone. Strategies that do well have more offspring in the next generation. In this way the frequencies of those strategies increase, while the frequencies of strategies that don’t do well decrease. This is the process of natural selection. If one strategy comes to be played by the whole population, then we can ask whether this state of affairs is ‘stable’. Suppose a new, mutant strategy arises in this otherwise uniform population. Since it will initially be at a very low frequency, most of the interactions the mutant has will be with the resident strategy. If the mutant does better against the resident than the resident does against itself, then individuals using the mutant strategy will have more offspring on average than individuals using the resident strategy. Thus the mutant strategy will increase in frequency, and so the resident strategy is not ‘evolutionarily stable’. If, on the other hand, the mutant does worse against the resident than the resident does against itself, then individuals using the mutant strategy will have less offspring on average than individuals using the resident strategy. Thus the mutant strategy will decrease in frequency until it goes extinct, and so the resident is resistant to invasion by this particular mutant. If the resident strategy is resistant to invasion by all possible mutants, then it is called an ‘evolutionarily stable

strategy' (ESS, see Criterion 1, Figure 1.5). The intuitive character of this concept means that there is more than one way it can be formalised, a flexibility often seen as an advantage by biologists "since they feel that the great variety of naturally occurring selection regimes require an openness with respect to formalization" (Hammerstein and Selten, 1994).

### Mathematical formalism

We have to introduce some terminology to proceed. Firstly, we have the notion of a **pure strategy**, denoted  $s$ , which is a specific strategic move in the game: "play  $s$ ". We can then define the **set of pure strategies**, denoted  $S$ , which is therefore the set of all possible moves available to a player. As an example, when tossing a coin you can either guess "heads" or "tails". The set of all possible pure strategies is therefore  $S = \{\text{heads}, \text{tails}\}$ . However, on any given coin toss you can also decide randomly whether to call heads or tails. For example you might decide to call heads  $2/3$  of the time, and tails the remaining  $1/3$  of the time. This is an example of a **mixed strategy**, denoted  $p$ , and defined formally as a probability distribution over the set of pure strategies  $S$ , so that for each  $s \in S$ , we have  $p(s)$ , the probability assigned to playing  $s$  (if  $S$  is an infinite set then  $p$  is a probability density function on  $S$ ). The **set of all mixed strategies** is denoted  $P$ , and if  $|S| = N$  is finite (i.e. there are a finite number  $N$  of pure strategies) then  $P$  is an  $(N - 1)$ -simplex. In our coin-tossing example, we have  $|S| = |\{\text{heads}, \text{tails}\}| = 2$  and thus the space  $P$  is fully defined by the 1-simplex, which represents "probability the player says heads". We can represent any possible mixed strategy by some point on this line.

In addition to strategies, we also require a **pay-off function**, denoted  $E$ , which represents the pay-off an individual gets from a game, given the strategy he/she plays and that adopted by his/her opponent. Given a pair of pure strategies  $s, t \in P$ ,  $E$  assigns a real value  $E[s|t]$ , which we interpret as the pay-off to an individual playing pure strategy  $s$  against an individual playing strategy  $t$ . We can then extend this pay-off function to account for mixed strategies by taking it to be an average of the different pay-offs weighted by the probabilities of each pair  $(s, t)$  occurring, which will depend upon the mixed strategies being played. To return to our simple coin-tossing example, supposing that a £1 coin is tossed, and you have to guess whether it comes down heads or tails. If you guess correctly you get the £1, if you guess incorrectly you have to give away £1. We can capture this using a pay-off function  $E[s|t]$  where  $s$  corresponds to your guess and  $t$  to what the coin comes down as. Therefore  $E[\text{heads}|\text{heads}] = £1$ ,  $E[\text{heads}|\text{tails}] = -£1$ ,  $E[\text{tails}|\text{heads}] = -£1$ , and  $E[\text{tails}|\text{tails}] = £1$ . Obviously in this instance we have an economic example rather than a fitness one, but the principle is the same. We can then extend this to the mixed strategy space. If we denote the probability that you say heads by  $p$ , and the probability that the coin lands heads by  $q$  (note that we need not assume

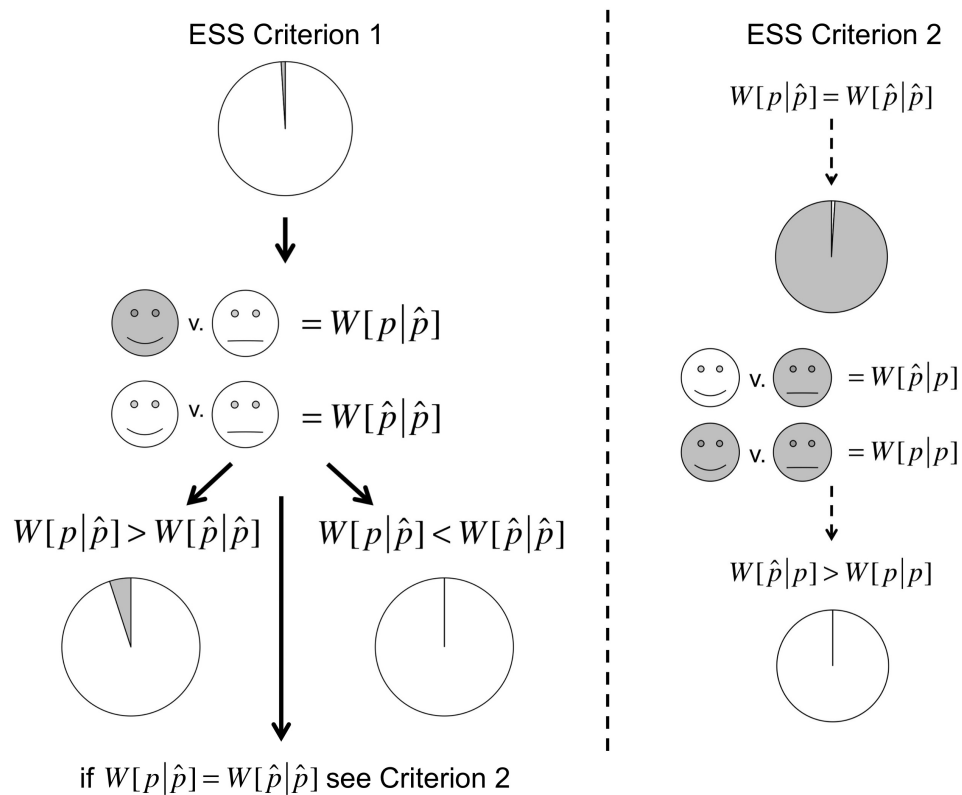


Figure 1.5: ESS criteria.

The population initially has all resident strategies, represented here by white, and denoted  $\hat{p}$ . A small number of mutants appear, represented by the grey, and denoted  $p$ .

*Criterion 1.* There are two key values, the fitness of the mutant in contests with the resident, denoted  $W[p|\hat{p}]$  and the fitness of the resident in contests with itself, denoted  $W[\hat{p}|\hat{p}]$ . If the mutant outperforms the resident in contests against the resident (i.e.  $W[p|\hat{p}] > W[\hat{p}|\hat{p}]$ ) then the frequency of the mutant strategy will increase, so the resident strategy is not stable. If, however, the resident outperforms the mutant in contests against the resident (i.e.  $W[p|\hat{p}] < W[\hat{p}|\hat{p}]$ ) then the frequency of the mutant strategy will decrease. The mutant will therefore go extinct, and the resident is stable against this mutant. If the resident is stable against all possible mutants then we say it is an evolutionarily stable strategy (ESS).

*Criterion 2.* If the fitness of the mutant strategy against the resident equals the fitness of the resident against itself (i.e.  $W[p|\hat{p}] = W[\hat{p}|\hat{p}]$ ), then the frequency of the mutant strategy could fluctuate randomly. If it doesn't go extinct, eventually there will come a time when the mutant is in the majority. At this stage the roles are in effect reversed, with the old resident strategy now in the minority. There are then two new key values, the fitness of the old resident in contest with the mutant, denoted  $W[\hat{p}|p]$ , and the fitness of the mutant in contest with itself, denoted  $W[p|p]$ . If the old resident does better against the mutant than the mutant does against itself (i.e.  $W[\hat{p}|p] > W[p|p]$ ), then the frequency of the mutant will again be driven down. Thus when the mutant is rare its frequency will fluctuate randomly, but when it's common it will decrease; eventually it will go extinct in the population just through chance fluctuations, since it cannot achieve a full takeover. Therefore the old resident is said to be stable against the mutant. If we have  $W[\hat{p}|p] \leq W[p|p]$ , this is not the case.

Our two ESS conditions are therefore as given in the text.

the coin is fair, i.e. that  $q = 0.5$ ), then the probability of getting (heads, heads) is  $pq$ , the probability of (heads, tails) is  $p(1 - q)$ , the probability of (tails, heads) is  $(1 - p)q$ , and the probability of (tails, tails) is  $(1 - p)(1 - q)$ . Therefore the pay-off function is

$$\begin{aligned} E[p|q] &= pqE[\text{heads}|\text{heads}] + p(1 - q)E[\text{heads}|\text{tails}] + (1 - p)qE[\text{tails}|\text{heads}] + (1 - p)(1 - q)E[\text{tails}|\text{tails}] \\ &= \pounds(pq - p(1 - q) - (1 - p)q + (1 - p)(1 - q)) \end{aligned}$$

We can then ask how we can maximise your pay-off. We define a **best reply** to a mixed strategy  $q$  as being a strategy  $p$  such that if the opponent is playing  $q$  you can do no better than play  $p$ . That is,  $E[p|q] \geq E[r|q]$  for all  $p, r \in Q$ . If we have a pair of strategies  $(p, q)$  that are each best replies to each other then we call this an **equilibrium point**, since neither player can benefit by changing strategy.

Our game  $G$  is then defined as  $G = (S, E)$ . To make this more biologically relevant, we often add to the pay-off function some baseline fitness  $F$  to gain a **fitness function**  $W$ , so that  $W[p|q] = F + E[p|q]$ . Adding a constant value to the pay-off function doesn't change the dynamics, and from hereon we will only mention fitness functions, thus defining our game as  $G = (S, W)$ .

### Evolutionarily Stable Strategies

We are now in a position to formalise the intuitions about evolutionary stability presented above. As mentioned previously, there is more than one way in which this can be done (van Damme, 1991; Binmore and Samuelson, 1992), and things are more complicated still when  $S$  is not finite (Vickers and Cannings, 1987; Bomze and Pötscher, 1989). Here we will concern ourselves with the usually-accepted definition of an ESS: that due to Maynard Smith and Price (1973). The following derivation is adapted from that of Hammerstein and Selten (1994).

Imagine a population of organisms all playing strategy  $p$ . Since  $p$  is a mixed strategy, note that this could be interpreted in two (mathematically equivalent) ways. Firstly, it could be that all individuals in the population literally play strategy  $p$ , so that they choose which pure strategy  $s$  to play in each contest according to the probability given by  $p(s)$ . Secondly, it could be that the individuals in the population are a polymorphic mix of strategies (pure or mixed) so that the mean strategy is  $p$ . In our coin-tossing game, if the population strategy was “say heads half the time”, it could be that all organisms in the population are saying heads half the time, or it could be that half the population says heads in every contest while the other half says tails in every contest (or some other mix of strategies which led to heads being played overall half the time). Now imagine there is some mutant strategy  $r \neq p$ . As mentioned above, the intuition is that this

strategy is initially present at time  $t$  in some small proportion  $\varepsilon_t > 0$ . Thus the population mean strategy  $q_t$  at time  $t$  is  $q_t = (1 - \varepsilon_t)p + \varepsilon_t r$ . For simplicity we assume that generations are non-overlapping, and that the fitness of individuals in the population is defined by the outcome of the contest for that individual in that generation, which will be based on the payoff function  $W$  (defined so that this is always strictly positive). So the average fitness of organisms playing the mutant strategy  $r$  is  $W[r|q_t]$ , while the average fitness of organisms playing the majority strategy  $p$  is  $W[p|q_t]$ . We assume that the reproductive success of individuals is proportional to their fitness relative to the average in the population. If a strategy is equally as fit as the average member of the population, then the number of organisms playing it will be the same in the next generation. In the same way, strategies fitter than the average will be more represented in the next generation, and strategies less fit will be less represented. The number of individuals playing a given strategy in the next generation must also be proportional to the number playing that strategy in this generation, since there are only a limited number of potential parents to have offspring playing any given strategy. The proportion  $\varepsilon_{t+1}$  of individuals playing mutant strategy  $r$  in the next generation can then be expressed as

$$\varepsilon_{t+1} = \frac{W[r|q_t]}{W[q_t|q_t]} \varepsilon_t. \quad (1.12)$$

We want to know the conditions under which  $\varepsilon_t$  tends to 0 as  $t \rightarrow \infty$ . We look at the difference in frequency  $\varepsilon_{t+1} - \varepsilon_t$ ,

$$\varepsilon_{t+1} - \varepsilon_t = \frac{W[r|q_t] - W[q_t|q_t]}{W[q_t|q_t]} \varepsilon_t. \quad (1.13)$$

Therefore  $\varepsilon_{t+1} < \varepsilon_t$  if and only if  $W[r|q_t] < W[q_t|q_t]$ . Since

$$W[q_t|q_t] = W[(1 - \varepsilon_t)p + \varepsilon_t r|q_t] = (1 - \varepsilon_t)W[p|q_t] + \varepsilon_t W[r|q_t],$$

we get  $W[r|q_t] < W[q_t|q_t]$  if and only if  $W[r|q_t] < W[p|q_t]$ . This is exactly in line with our biological intuition that the mutant strategy shouldn't invade if its fitness is worse than the original strategy  $p$  in the new population with mean strategy  $q_t$ . We can then expand this further, to give

$$\begin{aligned} W[r|q_t] &< W[p|q_t] \\ \varepsilon_t W[r|r] + (1 - \varepsilon_t)W[r|p] &< \varepsilon_t W[p|r] + (1 - \varepsilon_t)W[p|p] \\ 0 &< (1 - \varepsilon_t)(W[p|p] - W[r|p]) + \varepsilon_t(W[p|r] - W[r|r]). \end{aligned} \quad (1.14)$$

We can see that if  $W[r|p] > W[p|p]$  then for  $\varepsilon_t$  small enough, the right-hand side of equation (1.14) will be negative and thus  $r$  will not be driven out of the population by  $p$  once it exists. Therefore  $p$  is not stable

against invasions by  $r$ . If, on the other hand,  $W[r|p] < W[p|p]$ , then there will be some  $\varepsilon > 0$  small enough such that for all values  $\varepsilon_t < \varepsilon$ , the right-hand side of equation (1.14) will be positive and so the frequency of  $r$  in the population will decline (and thus  $r$  will go extinct). Thus tiny initial frequencies of  $r$ -players will be unable to get a foothold, and  $p$  is stable against invasion by  $r$ . Finally, if  $W[r|p] = W[p|p]$ , the right-hand side of equation (1.14) will be positive if and only if  $W[p|r] > W[r|r]$ . Thus only if this holds will  $p$  be stable against  $r$ . In order to be an ESS we require that  $p$  is stable against all possible other strategies  $r$  (Figure 1.5):

**Definition 1.** A strategy  $p^*$  is an **evolutionarily stable strategy (ESS)** if for all mixed strategies  $r \in Q$  such that  $r \neq p^*$ , either

1.  $W[p^*|p^*] > W[r|p^*]$  ( $p^*$  is a strict best reply to itself), or
2.  $W[p^*|p^*] = W[r|p^*]$ , and  $W[p^*|r] > W[r|r]$ .

There are other derivations of these conditions which don't rely upon the dynamics used above (Maynard Smith, 1974; Vickers and Cunnings, 1987; Bomze and Pötscher, 1989; Hofbauer and Sigmund, 1998).

### Hawk-Dove game

We now consider a classic example in evolutionary game theory, the Hawk-Dove game, first introduced in Maynard Smith and Price (1973) and Maynard Smith (1974). There are of course many examples of evolutionary game theory models but since this the first to have been explicitly set out as a problem under this formalism it is worthy of inclusion. The problem as set out seeks to find a reason why many males competing for access to females often engage in ritual fights rather than battles to the death; a fact the more remarkable given the weapons males often bear (see section 1.3.1 above).

We model idealised organisms that fight one another. There are two pure strategies: “conventional” tactics, denoted  $C$ , that are unlikely to cause serious injury to an opponent, and “dangerous” tactics, denoted  $D$ , which represent escalating the conflict so as to severely damage the opponent. Conventional tactics represent the ritualised combat frequently seen, while dangerous tactics would represent the use of the weapons the organisms bear to injure the opponent. The pay-off function is worked out as follows (Table 1.3).  $V$  is the benefit of winning a contest, and  $U$  is the cost of an injury. If the row player plays dangerous tactics  $D$ , against a column player also playing  $D$ , then the probability the row player wins is  $1/2$  (we assume row and column players are identical as this is a symmetric game), and if this occurs he gets a pay-off of  $V$ . The probability that the row player loses is also  $1/2$ , and if this occurs he is injured by the column player's dangerous tactics and so gets a score of  $-U$ . His total expected score is thus  $(V - U)/2$ .

		column player	
		D	C
row player	D	$(V - U)/2$	$V$
	C	$0$	$V/2$

Table 1.3: Hawk-Dove pay-off table. The upper left score in each box is the pay-off to the row player, the lower right the pay-off to the column player. To convert these payoffs into fitnesses we add a baseline fitness value of  $F$  to ensure that they are greater than zero. See text for more details.

The same logic gives the same expected pay-off to the column player in this instance. When an individual playing  $D$  comes up against an individual playing  $C$ , the  $C$ -player is assumed to back down and run away, obtaining a pay-off of 0, and leaving the pay-off  $V$  to the  $D$  player. Finally, when two  $C$  players meet, they are again assumed to be equally likely to win, and so they gain a (winning) pay-off  $V$  with probability  $1/2$ , and a (losing) pay-off of 0 with probability  $1/2$ , for a total expected pay-off of  $V/2$ . The complete pay-off matrix is thus as shown in Table 1.3, where the upper left entry in each box is the expected pay-off to the row player, and the lower right the expected pay-off to the column player. Since we require all the fitnesses to be positive we can add a baseline fitness value  $F$  to the pay-off values given in order to obtain the fitness (see above; Hammerstein and Selten, 1994).

From this we can define our mixed strategies as being  $p$ , the probability of playing  $D$  in a random contest. In this game, the organisms have no conception of whether they are “row” or “column” players, and the pay-off matrix is the same (e.g. a  $D$  player gets  $V$  against a  $C$  player regardless of whether the players are row or column players). This is called a symmetric game. There are also asymmetric games, in which the pay-offs depend on whether a player is a row or column player, and players have access to this information. In symmetric games like this, however, the ESS must be the same for row and column players, and so our job is simplified. We can calculate the expected fitness  $W[p|q]$  of a player playing  $p$  against an opponent playing  $q$  as

$$W[p|q] = F + p \left( q \frac{V - U}{2} + (1 - q)V \right) + (1 - p) \left( (1 - q) \frac{V}{2} \right),$$

where  $F$  is the baseline fitness. Then, since we know the frequency of all strategies in the population, we



can extend this to being the expected fitness of a player playing  $p$  in a population with mean strategy  $\hat{p}$ ,  $W[p|\hat{p}]$ . We want to find the ESS value  $p^*$ . We do this by calculating the value of  $p$  for which

$$\left. \frac{\partial W}{\partial p} [p|\hat{p}] \right|_{\hat{p}=p} = 0$$

In this case the solution is  $p^* = V/U$ . Thus if the population mean strategy is  $\hat{p} = p^*$  no individual can benefit by changing strategy. This result gives us an insight into the relative rarity of escalated combat between males - if the cost of injury  $U$  is high relative to the benefits of success  $V$  then  $p^*$  will be very small. Vicious combat is more likely in species where the cost of damage is small compared to the benefits of victory, for example in the Atlantic Salmon, *Salmo salar*, where adult males die after only one breeding season anyway (Fleming, 1996) and so it matters little if they get injured fighting for access to females.

The formulation of the Hawk-Dove game here is based on it being a ‘one-shot’ game; that is, that strategies are defined for just a single iteration of the game. Of course it is easy to imagine evolutionary situations in which the same organism will play a game repeatedly over the course of its lifespan, either a specified number of times (in which case the repeated game can be seen in a sense as a complicated one-shot game) or an unspecified number of times dependent upon some probability of each game being the last. These are more complicated scenarios and exposition of them has been left out of this thesis. Evolutionary game theory is a large subject and the aim here is only to give a very brief outline of it. Note, though, that repetitions of games provide the opportunity for conditional strategies; i.e. those in which a player’s move depends upon the move his opponent made in a previous round (e.g. the “Bully”, “Retaliator”, and “Prober-Retaliator” strategies described by Maynard Smith and Price, 1973; amongst many other examples).

### Replicator Dynamics

Evolutionary biologists are not only interested in the equilibrium states of a particular model, but also in what evolutionary trajectories it might produce. In order to establish this we require a formal system of dynamics to govern how much each strategy will increase over time given the composition of the population. There are several alternatives (see Weibull, 1995, and Hofbauer and Sigmund, 1998, for some examples); here we concentrate on the most commonly used: the replicator dynamics. The derivation that follows is again based on that in Hammerstein and Selten (1994).

Recall equation (1.12) above, and the difference equation (1.13) that it led to. Given a population mean strategy  $q_t$  at time  $t$  we can denote the frequency with which a pure strategy  $s$  is played as  $q_t(s)$ . We can then use similar reasoning to that used above to extend equation (1.13) to account for the change in this

frequency  $q_{t+1} - q_t$  from generation  $t$  to generation  $t + 1$  as

$$q_{t+1}(s) - q_t(s) = \frac{W[s|q_t] - W[q_t|q_t]}{W[q_t|q_t]} q_t(s).$$

Assuming the changes from one generation to the next are small we can approximate this difference equation with the differential equation,

$$\dot{q}(s) = \frac{W[s|q] - W[q|q]}{W[q|q]} q(s),$$

where  $\dot{q}(s)$  denotes the derivative of  $q(s)$  with respect to time. Since the denominator  $W[q|q]$  will be the same for all pure strategies we can ignore it, as it will change the speed of the evolutionary trajectories but not their direction. Therefore we are left with the following equations, called the **replicator dynamics**:

$$\dot{q}(s) = q(s) (W[s|q] - W[q|q]). \quad (1.15)$$

This formulation has an important feature: suppose we have that a strategy  $p$  that is an ESS in the two-person symmetric game  $G = (S, W)$ . Then the population state  $p$  is asymptotically stable under the replicator dynamics.

### Limitations and drawbacks

A major criticism of the use of evolutionary game theory is that it does not involve any genetic details, unlike the population genetics and quantitative genetics approaches described above. In the case of population genetics, there are in fact methods by which this and evolutionary game theory can be combined (for examples see Maynard Smith, 1982; Hofbauer and Sigmund, 1998). However, through the **G**-matrix, quantitative genetics incorporates explicit details of genetic variances and covariances that are neglected by game theoretic approaches. These could be crucial because the **G**-matrix can affect evolutionary trajectories (Steppan et al., 2002). In addition (and possibly more importantly) genetic covariance between traits can constrain the extent to which phenotypes can be optimised (e.g. see chapter 5). This is just one case of a more general criticism of optimisation and game theory models in evolution: that developmental and genetic constraints can prevent natural selection from optimising traits, and that this fact is ignored by the ‘adaptationist programme’ (Gould and Lewontin, 1979). Much of this criticism is based on a misunderstanding of the purpose of optimisation models (Maynard Smith, 1982), of which game theoretical models are an important part. Such models seek to show what outcome we would expect under natural selection based on simplifying assumptions. Assuming the analysis of the model is correct, then if the predicted out-

come differs from what is actually seen then the assumptions must be invalid (Parker and Maynard Smith, 1990). In particular, great care must be taken when defining the set of possible strategies available in a game (Maynard Smith, 1982). This is especially the case when a model is of a particular organism and a real-life scenario. Too broad a set of strategies will likely ignore developmental and genetic constraints. This can in itself be revealing, if the results suggest that a species has not optimised, and thus imply the existence of constraints. Too narrow a set of strategies may constrain evolution too severely. Davies (1979) pointed out that an ESS is only stable against stated alternatives, and that it is always possible to invent some other strategy that would be able to invade - “a butterfly with a machine gun” (Davies, 1979). But these criticisms are unsound. All models must rely on some intuition as to what is or is not biologically possible (Dawkins, 1980). One advantage of game theory is that it explicitly features the competition between individuals of the same species, and can easily handle frequency-dependent effects. This is such an important feature that it has since been incorporated into quantitative genetics models (see the basic model in section 1.6.2 above; also chapter 5). That genetic and developmental constraints are ignored for the sake of simplicity of analysis is one of the prices that must be paid when using a game theoretical approach. If this price is too high, e.g. because specific modes of inheritance are strange and important (e.g. see chapter 2), or because developmental constraints are crucial, then other modelling techniques should be used. As a tool for understanding the evolution of behaviour in organisms that compete with one another, evolutionary game theory is extremely useful.

Another criticism of evolutionary game theory is that it tends to focus on the ESS points rather than on trajectories; that is, the focus is usually on equilibria rather than on change. This is in fact true of population and quantitative genetics models as well to some extent (Maynard Smith, 1982). It is understandable from a mathematical perspective to focus on equilibria because they are easier to analyse, and also because in the absence of empirical knowledge about the starting position of a population they give information as to where evolution could be expected to lead. However, in some situations, where there is no ESS but rather evolutionary limit cycles (e.g. the side-blotched lizard, *Uma stansburiana*; Sinervo and Lively, 1996), trajectories could be important, and in these scenarios the replicator dynamics described above may not be enough to capture the situation. Population genetics details can then be incorporated.

## 1.7 Thesis outline

The main body of the thesis is split into two parts. Part I comprises chapters 2 and 3, and is concerned with the evolution of mate choice, while part II comprises chapters 5, 6, and 4, and is concerned with the consequences of mate choice.

Chapter 2 is an investigation into the evolution and maintenance of sex and mate choice in *Saccharomyces* yeast. A game-theoretical model is constructed to investigate the optimal behaviour of a parent yeast cell with regard to reproduction. Conclusions are drawn as to the benefits of mate choice for largely asexual yeast. The mathematical analysis was done with the assistance of Rob Seymour.

Chapter 3 concerns the evolutionary benefits females derive from choice. A novel way of categorising such benefits is proposed, distinct from the traditional direct/indirect distinction. A simple model shows that this new categorisation has consequences for the form of female preference. The mathematical analysis was done with the assistance of Rob Seymour.

Chapter 4 details a literature survey undertaken to assess the theoretical claims of chapter 3 by analysing whether the expected consequences of the types of mate choice predicted do indeed occur. The findings broadly support the theory, although they do not rule out other possible explanations.

Chapter 5 considers *Oophaga pumilio*, a brightly-coloured, poisonous tropical frog, the colouration of which is polymorphic across an archipelago in Panama. It is hypothesised that the polymorphism may be caused by sexual selection interacting with genetic drift. A quantitative genetic model is constructed to test the feasibility of this hypothesis. As a consequence, the potential importance of an evolutionary process, named “coupled drift” here, is highlighted. The mathematical analysis was done with the assistance of Yoh Iwasa. The chapter is adapted from the published paper by Tazzyman and Iwasa (2010).

Chapter 6 is about the effect of female choice on sperm competition. It is suggested that pre-copulatory factors such as the ease with which a male obtains a mate, or the quantity of resources that he has, may affect his post-copulatory ejaculate investment strategy. A game-theoretical model is constructed to investigate these connections, and their consequences are considered. The mathematical analysis was done with the assistance of Rob Seymour. The chapter is adapted from the published paper by Tazzyman et al. (2009).

Finally, in Chapter 7, the work is summarised, conclusions are drawn about the evolution and consequences of mate choice, and possible directions of future work are described.

## **Part I**

# **The evolution of mate choice**

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The following two chapters detail research into the evolution of mate choice. We consider both its initial occurrence, and the forms it is likely to take.

The first chapter is an investigation into *Saccharomyces* yeast, and shows the surprising fact that sexual selection can be adaptive even in the absence of outbreeding. Thus we can make the predictions that empirical investigation will find this form of mate choice to be widespread among self-fertilising species, and that sexual selection may have evolved before sex (in the sense of full outbreeding).

The second chapter concerns the form that female mating preferences are likely to take. As mentioned in the introduction, benefits from mate choice are often considered to be either direct or indirect. We introduce another distinction, between benefits that are fixed, so that females mating with a single male each have the same expected benefit regardless of their number, and benefits that are dilutable, so that the benefit a female expects from a male declines as the number of females choosing to mate with him increases. We show that this distinction leads to interesting predictions that the optimal form of mating preference will be different in each case, in turn leading to potentially interesting consequences.

## Chapter 2

# Suppression of mutational load in

## *Saccharomyces* yeast

Inbreeding is generally considered to be deleterious for organisms, and organisms that self-fertilise are usually thought to do so as a last resort when they lack the opportunity for outbreeding. However, in some species, inbreeding is the rule and very little outbreeding occurs. For example, meiosis in *Saccharomyces* yeast produces four haploid gametes which usually fuse back together again in an extreme form of self-fertilisation known as automixis or intratetrad mating. We construct a mathematical model that shows intratetrad mating 1) improves the long-term fitness of a yeast colony, and 2) lowers its mutational load. Both of these outcomes are enhanced by sexual selection at the haploid stage. We therefore suggest both intratetrad mating and intratetrad sexual selection evolved and are maintained to eliminate deleterious mutations, something *Saccharomyces* is therefore able to do without having to bear the costs and risks of outcrossing. This is likely to be the case for other species. We conclude 1) that inbreeding with recombination can be adaptive by lowering genetic load, 2) that (gametic) sexual selection is likely to have evolved in many species that self-fertilise, 3) that the evolution of outcrossing cannot be explained solely by the need to avoid mutational load because this can be achieved by inbreeding, and 4) that recombination and outcrossing should be considered as separate theoretical concepts, with potentially distinct evolutionary benefits.

## 2.1 Background

Sexual reproduction breaks up beneficial combinations of genes, reducing the heritability of adaptation. Despite its apparent disadvantage compared to asexual reproduction, it is almost universal in eukaryotes, and therefore requires an explanation (Maynard Smith, 1978; Rice, 2002; Otto, 2009). Many have been suggested (reviewed in Otto, 2009); in particular, it has been suggested that sex is favoured as it allows organisms to escape the build-up of deleterious mutations that occurs if a species never undergoes sex and recombination (Kondrashov, 1993; Rice, 2002). Clonally-reproducing asexual species are likely to accumulate deleterious mutations in their genome via the Mutational Deterministic process (Kondrashov, 1993; Rice, 2002) or Müller's Ratchet (Müller, 1964; Rice, 2002) (or both). So, according to this interpretation, sex is beneficial in allowing organisms to reduce their mutational load.

But what of species that undergo recombination without outcrossing? Reproduction by the fusion of gametes from the same individual occurs in several taxa (e.g. insects, Normark, 2003; plants, Stebbins, 1950; diatoms, Mann and Stickle, 1989; and yeast, Zakharov, 2005). In many species, selfing is prevented via a variety of mechanisms (reviewed by Jarne and Charlesworth, 1993), so as to avoid inbreeding depression. There are a number of hypotheses as to why selfing is retained in other species (again reviewed by Jarne and Charlesworth, 1993). These can be broadly split into two types: the reduction or avoidance of the cost or risk of attempting to outcross, and the preservation of successful genotypes. The relative importance of these two explanations may differ across species.

We focus on the genus *Saccharomyces*, which includes the Baker's yeast *S. cerevisiae* (Figure 2.1) and its undomesticated relative *S. paradoxus*. Although little is known about the ecology of wild *Saccharomyces*, it is believed individuals usually reproduce asexually as diploids. However, under starvation conditions, diploid cells undergo meiosis, each forming a tetrad of haploid spores within an ascus (Greig and Leu, 2009). When renewed growth conditions occur, the spores germinate and mate with others from the same tetrad forming two diploid offspring cells (hereafter referred to as "intratetrad mating"). This is a form of selfing. Due to segregation and recombination between loci during meiosis, the resulting offspring cells are genetically distinct from the mother cell (and each other). Outcrossing (the mating of spores from different parent cells) is known to be very rare in natural populations (Ruderfer et al., 2006; Zeyl and Otto, 2007; Tsai et al., 2008). Here we explain the benefits to yeast of this particular form of inbreeding.

Several previous explanations have suggested that the benefit of intratetrad mating lies in its promotion of heterozygosity. In this way it could serve to mask deleterious recessive alleles (Antonovics and Abrams, 2004; Johnson et al., 2005; Zakharov, 2005; Knop, 2006). Diploid yeast cells result from fusion of haploid cells of different mating types. Mating type is specified by one of two alleles (*MAT $\alpha$*  and *MAT $a$* ) at the *MAT*-



locus, so diploids are always *MATa/MAT $\alpha$*  heterozygotes, spores are always hemizygous *MATa* or *MAT $\alpha$* , and tetrads always consist of two spores of one mating-type and two of the other. Any heterozygous loci linked to *MAT* will thus tend to be heterozygous in diploid offspring, unless separated by a recombination event during meiosis. Because *MAT* is weakly linked to the chromosome III centromere ( $\sim 30\text{cM}$ , Cherry et al., 1997), loci linked to centromeres on other chromosomes will be likely to co-segregate with one of the *MAT* alleles. This tends to preserve heterozygosity in the resulting diploid offspring in loci close to the centromeres on all yeast chromosomes. However the total proportion of the yeast genome within 50cM of *MAT* on chromosome III or within 20cM of the centromere on other chromosomes (i.e. the proportion linked to the *MAT* locus) is only  $\sim 15\%$  (Cherry et al., 1997), so this process is likely to have limited effect.

The promotion of heterozygosity at unlinked loci is less obvious. Zakharov (2005) has shown that selfing, where two gametes (spores) from the same parent but different meioses are randomly mated, generates less heterozygosity than intratetrad mating, where two gametes from the same meiosis are randomly mated. However, this theory seems incomplete as an explanation of intratetrad mating. If the preservation of heterozygosity is the chief aim, it would be better served if the *MAT*-locus were more tightly linked to the centromere on chromosome III, increasing the *MAT*-linked proportion of the genome, and thus also the genome-wide heterozygosity. In addition, regardless of the position of the *MAT*-locus, yeast cells could preserve heterozygosity much more efficiently by clonal reproduction rather than by sex. In fact clonal reproduction by budding is the usual form of reproduction in yeast, and so by switching to sexual reproduction, a yeast cell will in fact ensure its offspring become less heterozygous. Furthermore, preservation of heterozygosity at a locus is only beneficial until the non-deleterious wildtype allele suffers a mutation. Preserving a locus in a heterozygous state in the short term dooms it to deleterious homozygosity in the long term.

In addition, the heterozygosity promotion hypothesis does not incorporate the possibility of non-random mating within the tetrad. When spores germinate they release a pheromone indicating their mating type (**a** or  **$\alpha$** , Figure 2.1). This signal is attractive to potential mates of the other mating-type, and stronger signallers are preferred over weaker signallers (Jackson and Hartwell, 1990; Rogers and Greig, 2009). It has been proposed that signal strength indicates genetic quality (Pagel, 1993), a hypothesis that now has strong experimental support (Smith and Greig, 2010). Haploid cells of each mating type could therefore preferentially fuse with stronger signalling haploid cells of the other mating type. This form of gamete choice could have important effects on offspring fitness. In this chapter we explore the hypothesis that intratetrad mating is favoured because it allows yeast cells to purge their genomes of deleterious alleles. We show how intratetrad mating alters the mutational load of offspring of the yeast, and how this process

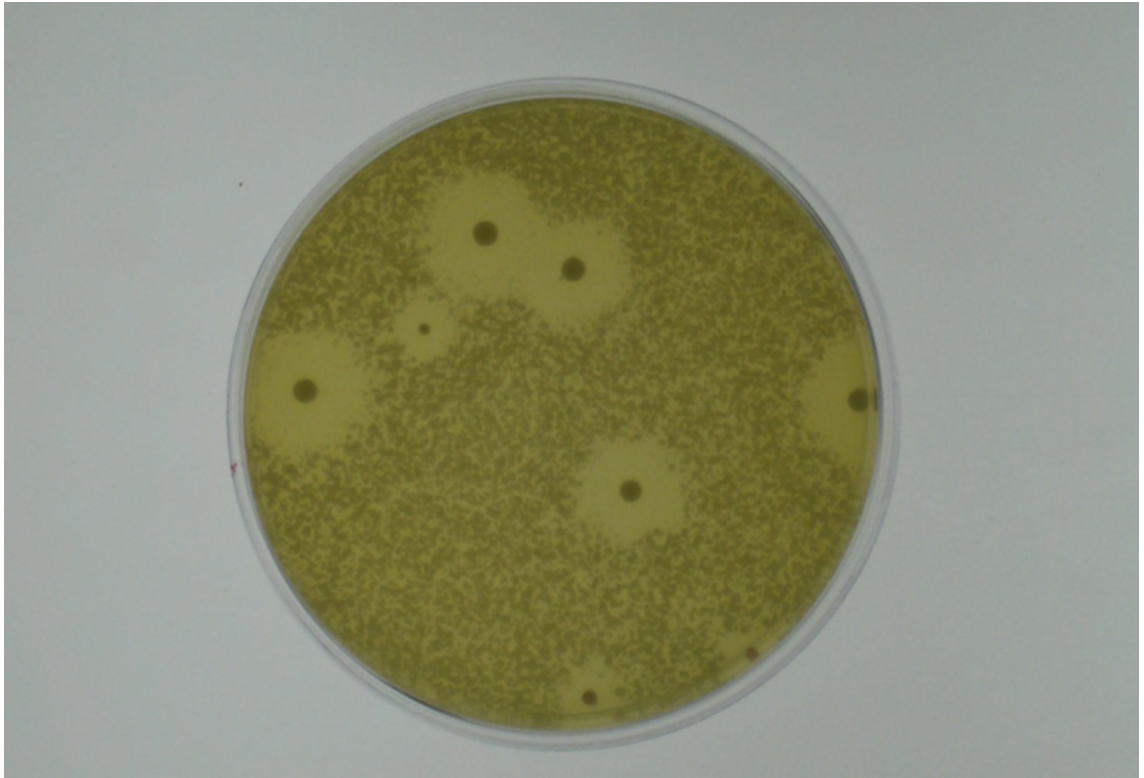


Figure 2.1: Haploid *Saccharomyces cerevisiae* cells in a petri dish. The large black blobs are colonies of mating type  $\alpha$  haploid cells. The dish is sprayed with a tester strain of haploid cells supersensitive to the  $\alpha$  mating pheromone. The 'halo' of space around each colony therefore indicates the presence of the mating pheromone.

is enhanced by intratetrad sexual selection. This gives an important insight into the evolutionary effect of sexual signalling in the life cycle of yeast, and allows us to make predictions about other sexual species, and the evolution of outcrossing sex more generally.

## 2.2 Model

### 2.2.1 The yeast life cycle

We model a simplified version of the *Saccharomyces* life cycle as follows (Figure 2.2). We assume the initial diploid parent cell is experiencing starvation conditions. It has a choice. It can either simply wait until nutrition is restored, or it can undergo meiosis and form a tetrad of four haploid spores, two of which are **a**-type and two of which are  $\alpha$ -type (Herskowitz, 1988; Spencer and Spencer, 1997). When favourable growth conditions return, a waiting diploid cell can simply resume asexual growth by budding, producing two identical diploids. Alternatively, in the tetrad formed by a parent that underwent meiosis, the **a** and  $\alpha$

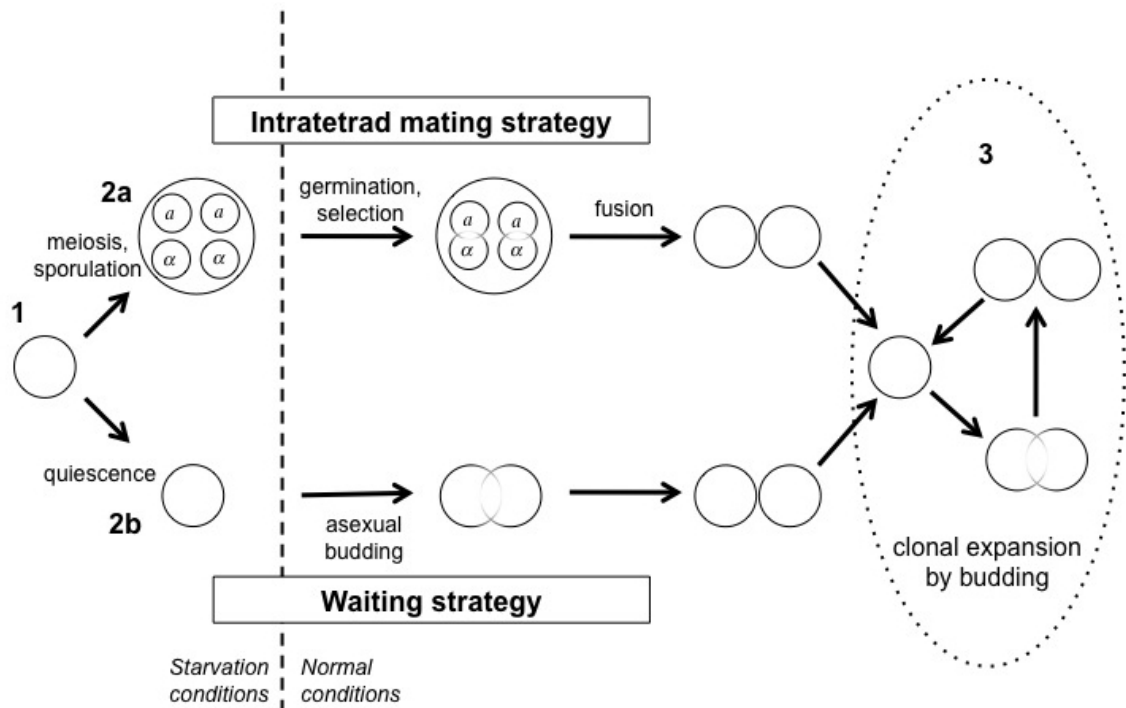


Figure 2.2: Idealised *Saccharomyces* yeast life cycle. (1) The diploid parent cell, under starvation conditions. It can either follow the Intratetrad mating strategy, or the Waiting strategy. (2a) The Intratetrad mating strategy begins with meiosis and sporulation. When starvation conditions cease (dashed vertical line) the spores germinate, forming four haploid cells, which signal their mating type via a pheromone. Due to this pheromone signalling, the haploid cells can fuse selectively, or can do so at random. Either way the cells fuse to form two diploid cells which are (in general) genetically distinct from the parent cell and each other. (2b) The Waiting strategy begins with the parent cell becoming quiescent. It then waits until the end of starvation conditions (dashed vertical line), at which point it begins asexual budding, forming two diploid offspring cells that are genetically identical to the parent cell and each other. (3) Both the Intratetrad mating strategy and the Waiting strategy result in the formation of two diploid cells, which then undergo clonal expansion via asexual budding, resulting in a colony.

haploid spores will germinate and mate, forming two non-identical diploids. Mating can only occur between **a** and **α** haploids. The haploids can either fuse randomly, or fuse selectively according to the strength of the pheromone signal they produce. We assume that haploid spores indicate their genetic quality, as the strength of pheromone production correlates with the number of deleterious alleles carried in the haploid genome (Smith and Greig, 2010). Whatever the mating strategy, asexual or sexual, with or without mate selection, two diploid offspring are produced. These then reproduce by asexual budding, producing a colony of cells whose number depends on the initial reproductive strategy.

In the interests of simplicity, the model does not include the possibility of outcrossing (whereby haploid cells from two different yeast parent cells mate together), since these events are believed to be rare (Ruderfer et al., 2006; Zeyl and Otto, 2007; Tsai et al., 2008). It also neglects the difference in the time

Ascus type	<b>a</b> genotypes	$\alpha$ genotypes	Probability	Possible offspring genotypes
1	(+) (+)	( <i>m</i> ) ( <i>m</i> )	1/6	(+ <i>m</i> )
2	(+) ( <i>m</i> )	(+) ( <i>m</i> )	2/3	(++), (+ <i>m</i> ), ( <i>mm</i> )
3	( <i>m</i> ) ( <i>m</i> )	(+) (+)	1/6	(+ <i>m</i> )

Table 2.1: Ascus types, the probabilities that they occur, and the possible resultant offspring genotypes.

taken for the spores in a tetrad to germinate and fuse into two diploids compared to the time for a “waiting” asexual diploid to bud into two diploids, the possibility of the haploid cells themselves undergoing clonal expansion, and the possibility that meiosis fails to form four viable spores (see Discussion, section 2.4, for consideration of these assumptions).

Since we are interested in the suppression of deleterious mutations, we consider cases where the parent yeast cell is heterozygous for deleterious alleles at a number of loci in its genome, and assume that these loci are not linked to the *MAT* locus. We start with the one-locus case, and then proceed to consider higher numbers of loci.

### 2.2.2 One locus

Denote the wildtype allele as +, and the deleterious mutant as *m*. Then the parent yeast cell’s genotype is denoted (+*m*). If it undergoes asexual budding, both offspring cells will also have genotype (+*m*) (for simplicity we assume there is no further mutation at the locus in question). If it undergoes intratetrad mating, however, the possible offspring genotypes are (++) , (+*m*), and (*mm*). When the parent cell undergoes meiosis, there are three different possible types of ascus (after accounting for symmetry) with probabilities as seen in Table 2.1.

We assume that a haploid signals the number of deleterious mutations it has in its genome through the amount of  $\alpha$  and **a** pheromone it releases. Throughout this chapter we refer to the haploid of each mating type with the least number of deleterious alleles as the strongest, and call the other haploid the weakest. For example, in the one-locus model, in a type-2 ascus (Table 2.1), the **a**-haploid with genotype (+) is the strongest, whereas the **a**-haploid with genotype (*m*) is the weakest. Obviously this terminology only applies where there are genetic differences between haploids, but sexual selection can only occur where this is the case (ascus type 2 in Table 2.1, but not types 1 and 3).

Strategy	Probability of genotype		
	(++)	(+m)	(mm)
Waiting	0	1	0
Intratetrad mating with probability $p$	$p/3$	$1 - 2p/3$	$p/3$
Random intratetrad mating ( $p = 1/2$ )	1/6	2/3	1/6
Selective intratetrad mating ( $p = 1$ )	1/3	1/3	1/3

Table 2.2: Offspring genotype probabilities depending on strategy

Denote the probability that the strongest **a**-haploid fuses with the strongest  $\alpha$ -haploid (and thus also the weakest **a**-haploid fuses with the weakest  $\alpha$ -haploid) by  $p$ . We suppose that  $p$  is genetically determined, and we investigate the effect on the future colony that differing values of  $p$  would have. Two  $p$ -values of particular interest are:  $p = 1$  representing always fusion of the strongest **a**-haploid with the strongest  $\alpha$ -haploid (and thus also weakest-to-weakest), and  $p = 1/2$  representing random fusion (i.e. no haploid selection). We ignore strategies with  $p < 1/2$  representing preferential fusion of the strongest **a**-haploid with the weakest  $\alpha$ -haploid and vice versa, as these seem implausible. For a parent yeast cell with strategy  $p$ , the expected proportion of offspring cells of each of the possible genotypes is seen in Table 2.2.

We are therefore interested in three alternative strategies for the parent cell: waiting followed by asexual mitosis to produce two diploid clonal cells (hereafter called the “waiting” strategy); meiosis of diploid cells to produce four haploid spores followed by germination and random intratetrad mating (with  $p = 1/2$ ) to produce two diploid non-clonal offspring (the “random intratetrad mating” strategy); or meiosis of diploid cells to produce four haploid spores followed by germination and intratetrad mating with haploid selection (with  $p = 1$ ) to produce two diploid non-clonal offspring (the “selective intratetrad mating” strategy). We assume all strategies take the same time to produce two diploid offspring.

After the starvation conditions end, and the alternative strategies have produced two diploid offspring cells, these continue to reproduce by asexual budding. We assume that the (++) genotype is the fittest, and therefore one discrete timestep is defined as the expected time it takes for a cell with genotype (++) to complete an asexual budding event and become two (++) cells. This amount of time is insufficient for the other genotypes to completely divide. We parameterise this by defining values  $0 < h < 1$ ,  $0 < s < 1$ , such that in one timestep each (+m) cell will partially split to become  $(1 + (1 - hs))$  (+m) cells, and each (mm)

Genotype	Offspring after $k$ timesteps
(++)	$2^k$
(+m)	$(1 + (1 - hs))^k$
(++)	$(1 + (1 - s))^k$

Table 2.3: Number of offspring of a given genotype after  $k$  generations

cell will partially split to become  $(1 + (1 - s))$  ( $mm$ ) cells. Thus  $s$  is the measure of homozygous fitness loss and  $h$  the dominance coefficient.

Starting at timestep 0 with a single diploid offspring yeast cell following a given strategy, after  $k$  timesteps, the number of offspring expected is as seen in Table 2.3. Therefore after  $k$  timesteps of budding the expected number  $W_i[p|k, h, s]$  of cells in a colony descended from a parent cell that underwent intratetrad mating with strategy  $p$  is

$$W_i[p|k, h, s] = \frac{p}{3}2^k + \left(1 - \frac{2p}{3}\right)(1 + (1 - hs))^k + \frac{p}{3}(1 + (1 - s))^k, \quad (2.1)$$

which is the probability of occurrence of each genotype after mating multiplied by the number of that type expected after  $k$  timesteps. The expected number  $W_a[k, h, s]$  of cells in a colony descended from a parent cell that underwent asexual doubling instead of intratetrad mating, however, will be

$$W_a[k, h, s] = (1 + (1 - hs))^k, \quad (2.2)$$

as this colony will contain only (+m) individuals.

### 2.2.3 Multiple loci

We also consider the case where the parent cell is heterozygous for a deleterious recessive allele at multiple loci. For simplicity we assume that each locus is unlinked to the *MAT*-locus or to the other loci of interest. This assumption becomes problematic as  $n$  becomes large, since there are only 16 chromosomes (Spencer and Spencer, 1997), but as will be shown later the qualitative findings still hold. With  $n$  loci the number of possible results of meiosis is  $3^n$ , as is the number of possible genotypes that the offspring can take.

We assume that fitnesses are multiplicative, and that all deleterious mutations have the same effect upon

fitness, so that a diploid yeast cell with a genotype that is heterozygous ( $+m$ ) at  $x$  loci, homozygous ( $mm$ ) at  $y$  loci, and homozygous ( $++$ ) at  $n - x - y$  loci will produce  $1 + (1 - hs)^x(1 - s)^y$  offspring per timestep by budding.

## 2.3 Results

### 2.3.1 One locus

We can now analyse the effect of the strategy followed by the parent yeast cell. Firstly, the decision to undergo intratetrad mating compared to asexual reproduction. Using equations (2.1) and (2.2) we see that a colony whose parent cell underwent intratetrad mating is larger than one whose parent cell underwent asexual doubling if the scaled difference  $V[k, h, s]$ ,

$$V[k, h, s] = 1 - 2 \left( 1 - \frac{hs}{2} \right)^k + \left( 1 - \frac{s}{2} \right)^k, \quad (2.3)$$

is positive. Given values of  $h$  and  $s$  we can find a number of timesteps  $z$  satisfying  $V[z, h, s] = 0$  (Appendix, section 2.5.1). For all time after this point,  $V[z, h, s] > 0$ , and the intratetrad mating strategy wins. Although we can't derive an explicit formula for  $z$  in terms of  $h$  and  $s$ , we can show that higher values of  $h$  and  $s$  give lower values of  $z$  (Appendix, section 2.5.1). In particular, for  $h \geq 1/2$ ,  $V[z, h, s] > 0$  for all  $z \geq 1$ , and so the intratetrad mating strategy wins immediately. However, this is not the case for  $h < 1/2$ , so for these values we can plot  $z$  as a function of  $h$  for a given fixed  $s$  (Figure 2.3).

We can also ask what the optimal choice of  $p$  is. In the one locus model, it follows from equation (2.3) that the number of timesteps  $z$  before the intratetrad mating strategy wins is independent of  $p$ . We can see from equation (2.1), however, that after this point the larger the value of  $p$  the better, and thus the strategy  $p = 1$ , of mating the strongest  $\mathbf{a}$ -haploid to the strongest  $\alpha$ -haploid, will be the best. So if the number of timesteps the asexual budding period is expected to last is greater than  $z$ , then intratetrad mating is worthwhile. If intratetrad mating is worthwhile, then it is also worthwhile haploids selecting their mates for genetic quality.

We can also calculate a measure of the mutational load of the colony at the locus of interest after  $k$  timesteps. This will be the proportion  $\Lambda$  of deleterious alleles at the  $+ / m$  locus across the colony of descendant cells, depending upon the strategy chosen. If the parent cell underwent asexual reproduction instead of intratetrad mating then all offspring cells have the genotype ( $+m$ ) and the mutational load is  $1/2$ . If, however, the parent cell underwent intratetrad mating then we can calculate its mutational load at the

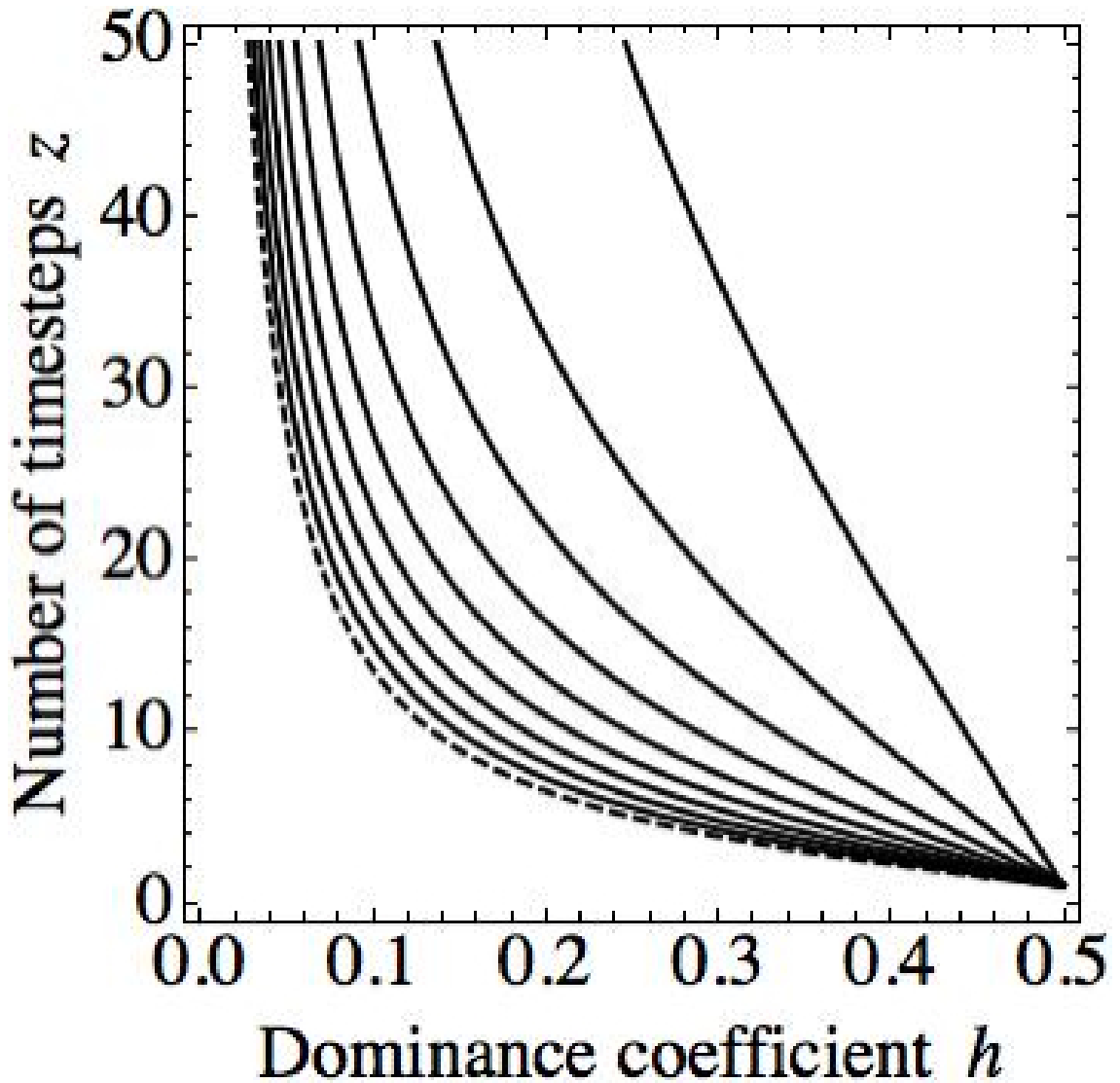


Figure 2.3: Number of timesteps  $z$  after which the intratetrad mating strategy outcompetes the asexual budding strategy, plotted against  $h$ . The ten curves are for  $s = 1, 0.9, 0.8, 0.7, 0.6, 0.5, 0.4, 0.3, 0.2,$  and  $0.1$ , proceeding in order from  $s = 1$  which is the dashed curve that takes the lowest value for any  $h$ . As  $s$  and  $h$  increase, the intratetrad mating strategy requires fewer timesteps before becoming victorious.



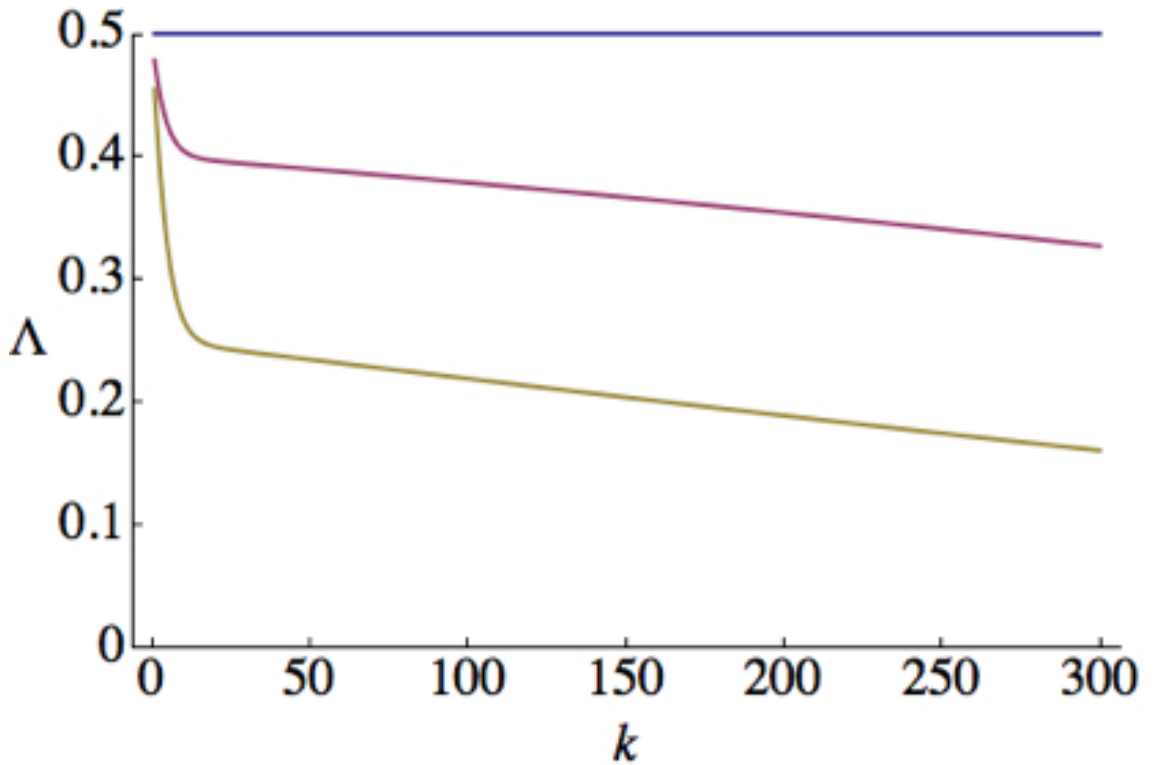


Figure 2.4: Proportion  $\Lambda$  of the loci in the population that have the deleterious mutant allele  $m$  at the locus of interest after  $k$  timesteps for three different strategies: waiting (blue, for which the mutation load is fixed at 0.5), random intratetrad mating (pink,  $p = 1/2$ ), and selective intratetrad mating (yellow,  $p = 1$ ). Parameter values are  $h = 0.01$  and  $s = 0.5$ . The functions have been plotted to  $k = 300$  since the “strongest-to-strongest”  $p = 1$  strategy is outperforming the others by this stage.

locus of interest as a function of its strategy  $p$ ,

$$\Lambda[p|k, h, s] = \frac{(1/2 - p/3)(1 + (1 - hs))^k + p/3(1 + (1 - s))^k}{W_i[p|k, h, s]}.$$

This is the number of each genotype multiplied by the proportion of that genotype that is deleterious, divided by the total number of offspring. We can compare the three strategies of interest (asexual reproduction, intratetrad mating with  $p = 1/2$ , intratetrad mating with  $p = 1$ ) (Figure 2.4). We see that both intratetrad mating strategies suppress mutational load, but that it is suppressed more when  $p = 1$  than when  $p = 1/2$ . We can show that  $\Lambda[p|k, h, s]$  is a decreasing function of  $p$  for all appropriate  $k, h, s$  (Appendix, section 2.5.2).

### 2.3.2 Multiple loci

Rather than analytically working the number of timesteps  $z$  after which the intratetrad mating strategy wins for every possible number of deleterious mutations  $n$ , we instead calculated the results numerically (see Appendix, section 2.5.3 for details) for values of  $n$  from 2 to 10. Since the results for all values were qualitatively similar, we have only displayed those from  $n = 2, 5$ , and 10 (Figure 2.5), with  $h = 0.01$  and  $s = 0.5$ . In all three cases, the intratetrad mating strategy outperforms the asexual reproduction strategy over the long term and selective intratetrad mating outperforms asexual reproduction faster than random intratetrad mating. With larger numbers of fitness loci, both types of intratetrad mating suffer greater short-term fitness loss. Selective intratetrad mating outperforms asexual reproduction faster as the number of loci heterozygous for deleterious alleles increases, but this is not the case for random intratetrad mating (Figure 2.5). So with a larger number of fitness loci, selective intratetrad mating shows a larger advantage over random intratetrad mating (Figure 2.5). In all cases, selective intratetrad mating is the best long-term strategy.

We also modelled the mutational load at  $n$  loci (for  $n = 2$  to 10), and found that as expected, intratetrad mating suppressed mutational load by exposing the deleterious loci to selection, and that this process was enhanced by selective intratetrad mating. The findings were qualitatively similar to those found with a single locus (Figure 2.4).

## 2.4 Discussion

Species that reproduce asexually are expected to build up genetic load over time, due to the effects of the Mutational Deterministic process (Kondrashov, 1993; Rice, 2002) and/or Müller's Ratchet (Müller, 1964; Rice, 2002). This loss of fitness is one of the reasons offered as to why sexual reproduction is favoured and why sexual organisms are so prevalent (Maynard Smith, 1978; Otto, 2009). However, we have shown that outcrossing is not necessary to avoid this problem. Rather, it can be circumvented through recombination via selfing alone. This process is enhanced by the evolution of (gametic) sexual selection (Pagel, 1993), which makes the purging of deleterious recessive alleles much more efficient. Thus both selfing and sexual selection may evolve and be maintained in order to purge the genome of deleterious alleles in species that reproduce exclusively or predominantly in an asexual manner. The evolution of outcrossing must therefore be considered a separate question to the purging of deleterious mutations.

In this work we have modelled a simplified version of the yeast reproductive cycle, in order to investigate the benefits *Saccharomyces* yeast derives from intratetrad mating and sexual selection. We consider a single

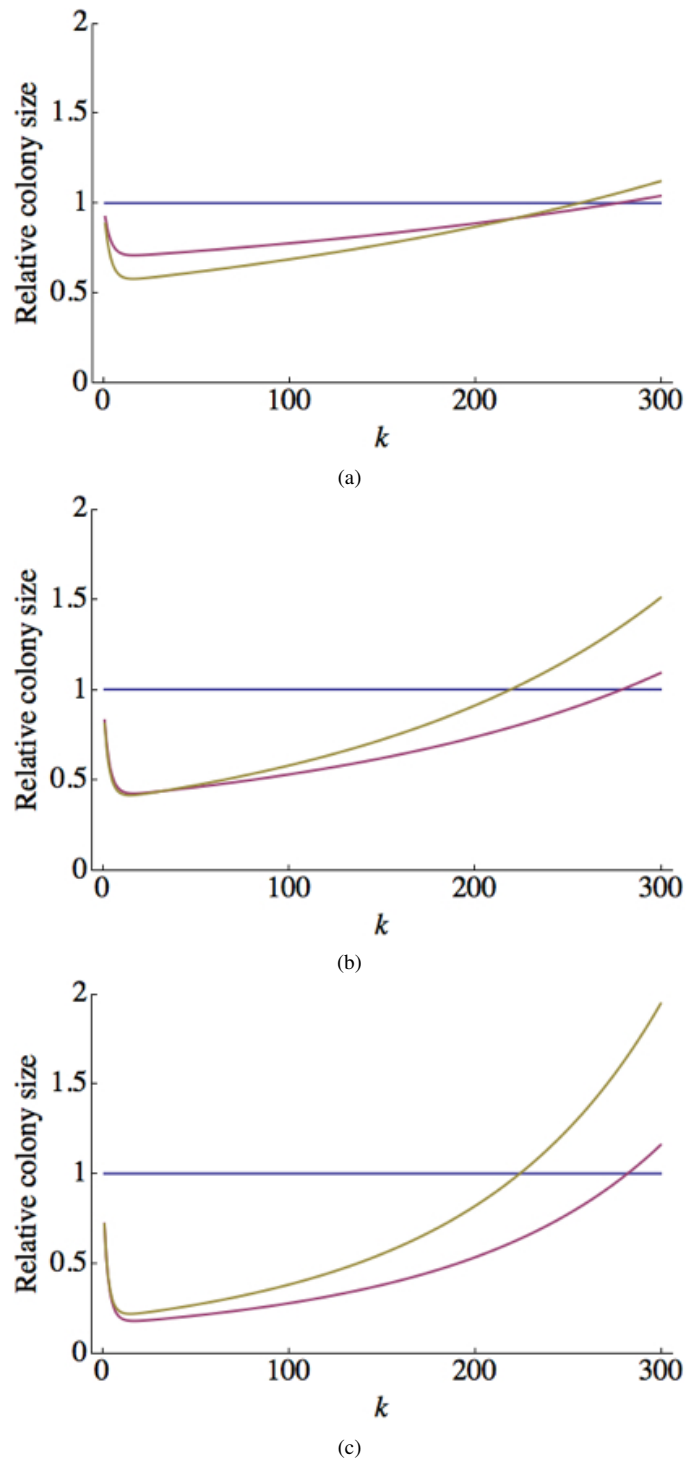


Figure 2.5: Sizes of colonies that underwent asexual budding (blue), random intratetrad mating (pink) and selective intratetrad mating (yellow) relative to the size of a colony that underwent asexual budding, after  $k$  timesteps, with selection parameters  $h = 0.01$ ,  $s = 0.5$ . The asexual budding strategy is unity throughout, and is eventually surpassed by both intratetrad mating strategies, with the selective strategy eventually being victorious in all cases. The number of loci  $n$  for which the parent cell was heterozygous for a deleterious mutation is (a) 2, (b) 5, and (c) 10. All three cases are qualitatively similar.

meiosis followed by intratetrad mating and a period of clonal expansion by asexual budding, and compare this strategy with simple asexual mitosis (also followed by clonal expansion). The immediate impact of intratetrad mating is to increase the frequency of homozygous ( $++$ ) and ( $mm$ ) loci in offspring.

An example of this process is seen in the one-locus model, in which a heterozygous parent generates equal frequencies of offspring with genotypes ( $++$ ), ( $+m$ ) and ( $mm$ ) through selective intratetrad mating. In the short-term this reduces fitness if deleterious  $m$  alleles are recessive as the fitness loss due to homozygous ( $mm$ ) genotypes outweighs the fitness benefit due to homozygous ( $++$ ) genotypes, compared to parents with asexual reproduction that only have ( $+m$ ) offspring. However, in the mid-to-long term, because the growth of the colony is exponential, the offspring stemming from ( $++$ ) zygotes have a reproductive advantage and will eventually outnumber those from ( $+m$ ) zygotes. So the intratetrad mating strategy can outcompete asexual reproduction if there is sufficient time for the advantage to be felt. How long this takes depends on heterozygote fitness (Figure 2.3). If selection is weak ( $s \sim 0$ ) and/or deleterious mutants are nearly completely recessive ( $h \sim 0$ ), heterozygotes have fitness approximately equal to that of the wildtype homozygote (i.e.  $hs \sim 0$ ), and the expected number of timesteps of asexual budding needed for the intratetrad mating strategy to outcompete the asexual reproduction becomes very large. However even with quite small loss of heterozygote fitness (i.e.  $hs \sim 0.01-0.03$ ), the number of timesteps falls significantly and intratetrad mating becomes the favoured strategy in 30 to 100 timesteps (Figure 2.3).

Intratetrad mating also causes a lowering of the proportion of deleterious alleles in a colony of related yeast cells (Figure 2.3). This effect occurs when there is random mating between haploids in the tetrad (i.e.  $p = 0.5$ ), but is more marked if haploids preferentially fuse according to their genetic quality ( $p = 1$ , Figure 2.3, Appendix section 2.5.2). These results suggest that one of the main functions of intratetrad mating is to enable yeast to escape the build-up of deleterious mutations that would otherwise occur if reproduction was exclusively asexual (Müller, 1964; Kondrashov, 1993; Rice, 2002).

In addition to suppressing deleterious mutations more thoroughly, haploid selection also has a fitness advantage over random intratetrad mating. This is particularly the case for higher numbers of deleterious loci, as the intratetrad mating with haploid selection strategy outcompetes both other strategies more rapidly when there are more deleterious alleles (Figure 2.5). The direct estimate of the genome-wide deleterious mutation rate for haploid yeast cells by Lynch et al. (2008) showed it to be high (approximately 0.32; although other estimates have suggested it is lower; Wloch et al., 2001; Zeyl and DeVisser, 2001). Given that meiosis and intratetrad mating are assumed to occur intermittently in our model, at the end of periods of clonal expansion, there is the potential for large numbers of deleterious mutations to build up between recombination events. This would suggest that once intratetrad mating is established, there will be selection

for haploid signalling and mate choice to enhance the production of diploid offspring with relatively fewer deleterious alleles.

Our model greatly simplifies the *Saccharomyces* mating system. Firstly, we ignore the possibilities of haploid clonal expansion by mitosis (after germination, but before fusion to form diploid cells). Should this occur, it is reasonable to suppose that the haploid cells with the least deleterious mutations (the strongest cells according to our terminology) would be able to reproduce faster. The net effect (ignoring spatial structure) would be that the probability of fusing strongest-to-strongest would be higher even in the absence of signalling and sexual selection. If this phenomenon is confirmed, it would be interesting to incorporate it into the model. Related to this is mating-type switching that can occur when haploid cells divide by mitosis and then switch from **a** to **α**, or vice versa (Haber, 1998). This enables haploid cells to mate with their own clones. Another situation potentially promoting haploid mitosis is when the number of viable haploid cells in the ascus is less than four, with the result that any haploid cells left without partners within the tetrad may undergo clonal expansion and mating-type switching. However, spore number control appears to have evolved to minimise the intertetrad mating, haploid mitosis, and mating-type switching expected from asci with fewer than 4 spores (Taxis et al., 2005). We would expect the selective value of mating-type switching and intra-clone mating to be heavily dependent on the number and severity of deleterious mutations, since the resultant offspring would be a totally homozygous diploid cell (apart from at the *MAT* locus and linked loci). It may be that this therefore only occurs when a haploid cell has no alternative with which to fuse. Our model also ignores outcrossing, the fusion of haploids derived from two different diploid parent cells. This can occur if asci are neighbours, or if haploids from different asci are mixed in the guts of insect dispersal vectors (Reuter et al., 2007), and could be important in the masking of deleterious alleles or the generation of novel combinations of advantageous epistasis. In general, these processes are believed to be rare (Zeyl and Otto, 2007; Tsai et al., 2008; but see Murphy and Zeyl, 2010). However, since little is known about the reproductive biology of *S. cerevisiae* under natural conditions (Greig and Leu, 2009), it is difficult to gauge their evolutionary importance. In the future, this model could be extended to investigate how these different outcomes impact on the advantage of intratetrad mating and signalling (see section 7.3.1, chapter 7).

We also neglect the possibility of deleterious loci being linked either with one another or with the *MAT* locus. That intratetrad mating causes retention of heterozygosity at sites linked to the *MAT* locus has been remarked upon elsewhere (Kirby, 1984; Antonovics and Abrams, 2004; Zakharov, 2005; Knop, 2006); since gametic signalling and selective fusion cannot affect this we chose instead to focus upon the larger portion of the genome that is unlinked to the *MAT* locus (~ 85%, Cherry et al., 1997). The possibility of the deleterious alleles being linked to one another must increasingly be taken into account as the number of loci

at which such alleles appear goes up. To see what effect this has on our results, consider what happens to two loci with deleterious alleles that are on the same chromosome. The higher the degree of linkage the less likely a meiotic crossover event will split them up, and so the more likely that a haploid cell will contain both. Therefore the effective number of segregating deleterious mutations is reduced (although since we are taking fitnesses to be multiplicative, the mean severity of the mutations is effectively increased). This means that  $n$  is effectively smaller than in reality. Since our conclusions are qualitatively similar, however, the overall effect will be similar.

Intratetrad mating takes considerably longer than asexual budding (Miller and Hoffmann, 1964; Herskowitz, 1988). Yeast colonies undergo fast expansion on new food, and are under selection to reproduce quickly because they are likely to be in competition with other colonisers. Thus, under conditions of growth, the opportunity cost of intratetrad mating is probably prohibitive because it means foregoing the chance for several generations of mitotic expansion. However, our model starts with the parent cell under starvation conditions. Under these conditions clonal expansion is not possible, so the opportunity cost of meiosis and sporulation is likely to be much lower: we therefore have neglected this cost. Despite this, we could easily incorporate costs of this sort into the model by allowing the asexual doubling strategy to start with more than two diploid offspring. Our qualitative conclusions would be unchanged, it would just take the intratetrad mating strategy more timesteps to achieve victory.

Our work here focuses on *Saccharomyces* yeast, but our findings should also apply to other species. However, care is needed in interpretation; intratetrad mating differs from the usual concept of selfing in that it involves the fusion of gametes from the same meiotic division, in addition to being from the same parent individual. This affects the genetics. In addition there may be important distinctions between unicellular organisms, and multicellular organisms. Despite these caveats, if we ignore the potential effects of gamete selection, selfing in which the gametes come from different meiotic divisions will in fact produce more homozygotes than intratetrad mating (Zakharov, 2005), and so it is likely to have the same beneficial effects when compared to clonal reproduction, particularly if it occurs when conditions are such that the opportunity cost of not reproducing clonally is low. Any diploid organism which reproduces exclusively by asexual reproduction will have the problem of the accumulation of deleterious alleles. We have shown that a way round this is meiosis and inbreeding, and that this process is considerably improved with the addition of sexual selection. We therefore expect to see (gametic) sexual selection evolving in many species that self-fertilise.

Even in systems without gametic sexual selection our model has relevance, as can be seen for example in the evolution of palindromes (inverted repeats) on the Y chromosomes of higher primates (Rozen et al.,

2003; Skaletsky et al., 2003; Hughes et al., 2010). Palindromes will naturally accumulate mutations and so diverge, but can make themselves identical once more by gene conversion (Rozen et al., 2003). This is equivalent to transforming a  $(+m)$  heterozygote into a  $(++)$  homozygote (half the time) or a  $(mm)$  homozygote (the other half of the time), except in this case the two alleles are different genetic loci on the same chromosome. If gene conversion generates a  $(mm)$  homozygote, it will disappear due its selective disadvantage. On the other hand, if gene conversion generates a  $(++)$  homozygote, it will be favoured by selection. Since the Y chromosome has no recombination, these mutations would otherwise build up, leading in general to the erosion of genetic activity on Y chromosomes (Charlesworth and Charlesworth, 2000). This is similar to yeast, and to other asexually reproducing individuals. It suggests that the suppression of mutational load is an important factor in the evolution and maintenance of recombination in *Saccharomyces* yeast in particular, and more generally wherever reproduction is asexual.

## 2.5 Appendix

### 2.5.1 Calculation of number of timesteps before selective intratetrad mating strategy becomes the best

We want to find out when  $V[k, h, s] > 0$  (equation (2.3)). To determine when this is, let  $x = s/2$ , and consider the function

$$f(h) = 1 + (1 - x)^z - 2(1 - hx)^z, \quad (2.4)$$

where  $0 < h < 1$ ,  $0 < x < 1/2$  and  $z \geq 1$ . It is clear that  $f(h) > 0$  if and only if  $V[k, h, s] > 0$ . Note that we do not assume  $z$  is an integer.

We first show that  $f(h) \geq 0$  for  $0 < x < 1/2$ ,  $z \geq 1$ , and  $h \geq 1/2$ , with equality only when  $z = 1$  and  $h = 1/2$ . From equation (2.4), when  $z = 1$  we have

$$f(h)|_{z=1} = 2x(h - 1/2),$$

and thus that  $f(1/2)|_{z=1} = 0$ , and  $f(h)|_{z=1} > 0$  for  $h > 1/2$ .

Now assume that  $z > 1$ . Differentiating equation (2.4) with respect to  $h$  gives  $f'(h) = 2xz(1 - hx)^{z-1} > 0$ , so  $f(h)$  is a monotonically increasing function of  $h$  for fixed  $x$  and  $z$ , and hence  $f(h) \geq f(1/2)$  for  $h \geq 1/2$ .

Now,

$$\frac{\partial f(1/2)}{\partial x} = z \left( (1 - x/2)^{z-1} - (1 - x)^{z-1} \right)$$

so  $f(1/2)$  is monotonically increasing in  $x$ , since  $z > 1$  and  $1 - x/2 > 1 - x$ . Therefore  $f(h) \geq f(1/2) > f(1/2)|_{x=0} = 0$  for all  $h \geq 1/2$  and  $0 < x < 1/2$ , and we have proved our result.

We now consider the range  $0 < h < 1/2$ . Given a fixed  $x$  and  $z$ , we can solve the inequality explicitly to get a condition on  $h$ ,

$$h \geq \frac{1}{x} \left( 1 - \left( \frac{1 + (1-x)^z}{2} \right)^{\frac{1}{z}} \right). \quad (2.5)$$

Define the function  $h(z)$  by equality in (2.5). Then  $h(z)$  is a monotonically decreasing function of  $z$  (see Figure 2.3). It follows that the equation  $h = h(z)$  can be solved uniquely to obtain  $z$  as a monotonically decreasing function  $z(h)$  of  $h$  for fixed  $x$ . This function will give the value  $z$  for which  $1 + (1-x)^z - 2(1-hx)^z = 0$ , and thus for all integers  $k > z(h)$ ,  $V[k, h, s] > 0$ .

## 2.5.2 The mutational load of a colony decreases as $p$ increases

We can write  $\Lambda[p|k, h, s]$  as

$$\Lambda[p|k, h, s] = \frac{\frac{1}{2} (1 + (1 - hs))^k + \frac{p}{3} \left( (1 + (1 - s))^k - (1 + (1 - hs))^k \right)}{\frac{p}{3} 2^k + \left( 1 - \frac{2p}{3} \right) (1 + (1 - hs))^k + \frac{p}{3} (1 + (1 - s))^k}$$

and differentiate with respect to  $p$  to get

$$\frac{d\Lambda}{dp} = \frac{3 \left( (2 - s)^k - 2^k \right) (2 - hs)^k}{2 \left( (2p - 3) (2 - hs)^k - p \left( 2^k + (2 - s)^k \right) \right)^2}.$$

This is negative since  $(2 - s)^k - 2^k < 0$ , and  $(2 - hs)^k > 0$ .

## 2.5.3 Details of numerical modelling

We used the open source programming environment Processing 1.09 ([www.processing.org](http://www.processing.org)) to provide the genotypic probabilities for number of loci  $n$ , from 2 to 10, then Mathematica 7 (Wolfram, 2007) to numerically calculate the number of cells in colonies following the three different strategies after  $k$  generations, from  $k = 1$  to 300. Since the growth of the colonies is exponential, we represented the number of cells relative to the number in a colony that followed the asexual budding strategy. The code is available on request.



## Chapter 3

# Fixed and dilutable benefits of mate choice

Benefits accruing to females who exercise mate choice have been defined to be either “direct” or “indirect”. We suggest an alternative distinction: benefits should be considered “fixed”, meaning the benefit obtained by a female from mating with a given male is independent of the number of mates he has (for example good genes benefits); or “dilutable”, meaning they are shared between females mating with the same male, so that the more mates a male has the lower the average benefit to each (for example fertility benefits or many forms of direct benefit). Using a simple model we show that this distinction has a major effect on the form of female preference. We predict that mating skew will be far greater in species where the benefits are fixed compared to those where the benefits are dilutable.

### 3.1 Background

The existence and purpose of female mating preferences for male ornaments has been a subject of debate since Darwin’s time (Darwin, 1871; Cronin, 1993; Andersson, 1994). Many hypotheses have been put forward which suggest the types of benefit accruing to a female from carefully selecting her mate (Andersson, 1994). These benefits are generally categorised as being either “direct” or “indirect” in nature (Andersson and Simmons, 2006). Examples of direct benefits include getting more or higher quality food or territory from the male, a larger nuptial gift when mating, good parenting skills (for review of these, see Andersson, 1994), or high fertilising efficiency (Trivers, 1972; Sheldon, 1994). Indirect benefits, on the other hand, are those that give an advantage to the female’s offspring rather than directly to her. Examples are having

offspring with high genetic viability due to “good genes” (Zahavi, 1975) or “compatible genes” (Tregenza and Wedell, 2000), avoiding the cost of inbreeding (Madsen et al., 1992; Parker, 1992), or having attractive offspring (sometimes called the “sexy son” hypothesis, originally proposed by Fisher, 1930; see also Weatherhead and Robertson, 1979). Since the different benefits to females are not mutually exclusive, neither are the hypotheses.

One way in which potential benefits differ that has been overlooked in previous discussions is whether or not benefits can be shared. Consider the difference in this regard between the “good genes” hypothesis (Iwasa et al., 1991; Houle and Kondrashov, 2002), and the “phenotype linked fertility” hypothesis (Sheldon, 1994). The “good genes” hypothesis postulates that females select their mates based on genetic quality, so as to have high viability offspring. A male passes on an equal number of high-viability genes (on average) every time he mates, and so the expected benefit a female gets from mating with a given male is not dependent on the number of copulations he has with other females. A high genetic quality male will pass on the same high-quality genes whether it is his first mating or his thousandth. We call this type of benefit “fixed”.

Now consider the “phenotype-linked fertility” hypothesis. This postulates that females select males who have a higher fertilising ability (Sheldon, 1994), to minimise the probability of unfertilised eggs. Fertilising ability may depend on the number of mates a male has obtained: if males are sperm-limited, those that are preferred and thus mate many times will have a decreased expected ejaculate investment per mating, due to depletion (Danielsson, 2001; Preston et al., 2001). A highly-fertile male may ejaculate a large number of sperm the first time he mates, but this number will decrease as he has more matings (Trivers, 1972). Thus the expected fertility benefit a female gets from a given male depends on the number of previous copulations he has had with other females. As the number of other females he mates with increases, the expected benefit to the focal female decreases. This effect will be exacerbated on an evolutionary timescale because there will also be selection for more attractive males to invest fewer sperm per mating (chapter 6). In this case, the benefits a male confers are “dilutable”.

The advantage of making this distinction in types of female benefit is that it will have a bearing upon female behaviour, as we aim to show using a simple model. We consider optimal female preference in two idealised circumstances. In the first, females are selecting for good genes, and thus the benefits to be gained from higher quality males are fixed. In the second, females are selecting for high sperm count in ejaculates. Males are assumed to be sperm limited, and so the benefits to be gained from higher quality males are dilutable. By contrasting these two cases, we show that there is potential for female behaviour to provide evidence of which benefits inform female preferences.

## 3.2 Model

We model an idealised species. All females are identical in terms of expected number of matings. The differences in fitness come from the female's preference function. By selecting a mate appropriately, a female can acquire benefits, either having more offspring (perhaps because her mate provides fertility benefits), or having more offspring survive to breeding age (perhaps because her mate provides more resources for the offspring, or has good or compatible genes). We assume that females cannot directly assess the benefits they wish to maximise when selecting a mate, but rather make decisions based on some male trait, characterised by a real variable  $z$ . This may be a single male trait, such as tail length, or it may be a combination of several traits, in which case  $z$  can be taken to be some suitable weighted average. We assume that there are lower and upper bounds on  $z$ , which we denote  $a$  and  $b$  respectively, so that for every male,  $a \leq z \leq b$ .

For simplicity, we assume that choice is cost-free, so females can examine all possible males before making their decision. We also assume that there is a large density of available males of every trait value, and the duration of copulation is short relative to its frequency, so that copulation with a single male of trait value  $z$  does not significantly decrease the number of  $z$ -males in the mating pool. Females exhibit preferences based on  $z$ . We model female preference as a function  $p(z)$ , defined for  $a \leq z \leq b$ , and satisfying  $p(z) \geq 0$ . Because we are interested in female preference as a relative measure (i.e. the attractiveness of a trait value  $z_1$  compared to that of another trait value  $z_2$ ), we scale the preference function such that

$$\int_a^b p(z) dz = 1. \quad (3.1)$$

Thus the preference function can be regarded as a probability density function describing the probability that a female will mate with a male whose trait value falls within a given interval. We now define the expected benefit that accrues to a female depending on her preference function. The form this takes will differ depending upon the nature of the benefit at stake.

### 3.2.1 Fixed benefits

We firstly model the situation where females obtain fixed benefits from their mates. To make it easier to follow we explicitly consider the case where females select a mate so as to maximise the genetic quality of their offspring. In this way they improve the chances of their offspring surviving to breeding age. This is assumed to be dependent on the genetic quality of the male; it is the "good genes" case. We note that the logic used here will apply to any fixed benefit case, however, not only to good genes.

We define the continuous benefit function,  $s(z)$ . This is the expected benefit accruing to females should

they mate with a male of trait value  $z$ . Note that because  $s(z)$  is the expected benefit it is not necessary that all males of trait value  $z$  give the same genetic benefits - there may be environmental effects that inform a male's trait value in addition to the genetic effects.

A female with preference function  $p(z)$  will then obtain expected benefit

$$W[p] = \int_a^b p(z)s(z)dz. \quad (3.2)$$

The preference function  $p$  that maximises equation (3.2) will therefore be the function that maximises female fitness  $W[p]$  in the “good genes” case.

### 3.2.2 Dilutable benefits

Secondly we model the situation where the benefits females obtain from their mates are dilutable. Again, to make it easier to follow we explicitly state the nature of the benefit, but note that the logic will apply to any form of dilutable benefit. In this situation, females attempt to maximise fertilisation of their eggs when they select a mate. This is in turn assumed to be dependent on the number of sperm that the male transfers during the mating. We assume that males suffer sperm depletion, so that the mean number of sperm transferred per mating decreases as the number of matings obtained by a given male increases. Thus the expected benefit a female will get from a mating with a male will depend on how attractive that male is considered to be by other females in the population, as well as on his trait value  $z$ . Denote the average female preference function by  $\hat{p}$ , so that the average preference for trait value  $z$  is  $\hat{p}(z)$ . We will call this the “popularity” of trait value  $z$ , and refer to  $\hat{p}$  as the popularity function. We assume that the higher the popularity of a trait value, the more matings males bearing that trait value are likely to get, and thus the fewer sperm they will transfer per mating on average (for a fuller justification, see Appendix, section 3.5.1). Therefore the expected benefit conferred on a female by a given male is a function of his trait value  $z$  and his popularity  $q = \hat{p}(z)$ . We denote this  $s(z, q)$ , and assume that it is continuous in both  $z$  and  $q$ , and greater than zero for all pairs  $(z, q)$ . Then a focal female with preference function  $p$  in a population with popularity function  $\hat{p}$  will obtain expected benefit

$$W[p|\hat{p}] = \int_a^b p(z)s(z, \hat{p}(z))dz. \quad (3.3)$$

This benefit function is linear in  $p$  but not (in general) linear in  $\hat{p}(z)$ . We want the benefit function  $s(z, q)$  to obey two conditions. Firstly, we assume that the more matings a male gets the fewer sperm he ejaculates per mating (on average), so we require

*Condition (i):* Given trait value  $z$ , denote popularity  $\hat{p}(z) = q$ , so that the benefit function is  $s(z, q)$ . Then  $s(z, q)$  is differentiable in  $q$  and

$$\frac{\partial s}{\partial q}(z, q) < 0,$$

for all  $q > 0$ , i.e. that the benefit function is monotonically decreasing in  $q$ , so that for fixed  $z$  the benefit function  $s(z, q)$  declines as the popularity  $q$  of  $z$ -males increases. This is because as a male's popularity increases, he will mate more often, and thus the benefits that he confers will be shared between more females, meaning a lower expected share to each. Note that as  $q \rightarrow \infty$  we also want  $s(z, q) \rightarrow 0$ , so that if female preference for a male of trait value  $z$  increases indefinitely, then the reward to any particular female from mating with such a male diminishes to insignificance. Secondly, for simplicity, we also want to assume that males with larger trait values give larger benefits (i.e. more sperm) all else being equal (the phenotype-linked fertility hypothesis; Sheldon, 1994). So the benefit function must also satisfy

*Condition (ii):*

$$\frac{\partial s}{\partial z}(z, q) > 0,$$

i.e. that the benefit function is monotonically increasing in  $z$  for fixed  $q$ , so that if there are two males of equal popularity, the male with the larger trait value will confer higher benefits.

## 3.3 Results

### 3.3.1 Fixed benefits

Any preference function  $s(z)$  must attain a maximum value  $\tilde{s}$  at at least one point which we denote  $\tilde{z}$ , so that  $\tilde{s} = s(\tilde{z})$ . We define  $q_{\tilde{z}}$  to be a preference function that is also a delta function,  $q_{\tilde{z}}(z) = \delta(z - \tilde{z})$ , so that  $q_{\tilde{z}} = 0$  for all  $z \neq \tilde{z}$ . In other words, females with preference function  $q_{\tilde{z}}$  will only mate with males of trait value  $\tilde{z}$ .

The fitness of this preference function is

$$W[q_{\tilde{z}}] = \int_a^b q_{\tilde{z}}(z)s(z)dz = s(\tilde{z}) = \tilde{s}.$$

Since  $\tilde{s}$  is the maximum value attained by  $s(z)$  on  $[a, b]$ ,  $q_{\tilde{z}}$  is at least jointly optimal, since for any preference function  $p$ ,

$$\int_a^b p(z)s(z)dz \leq \tilde{s} \int_a^b p(z)dz = \tilde{s}, \quad (3.4)$$

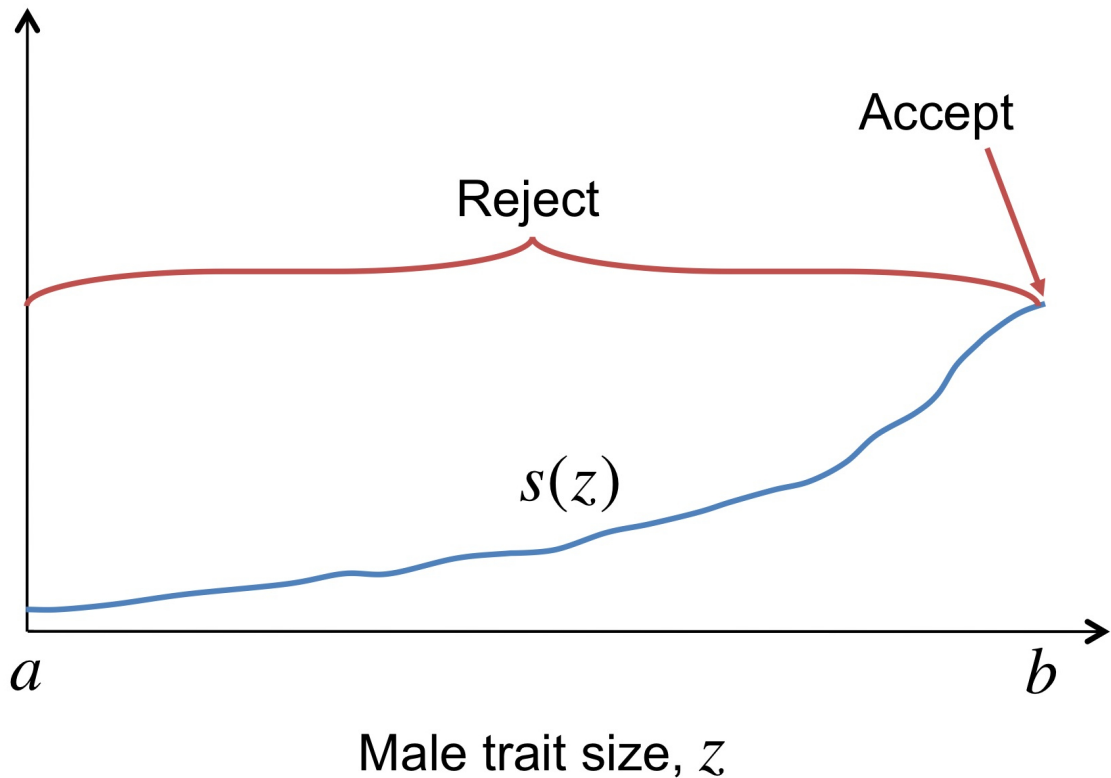


Figure 3.1: An example of female preference for fixed benefits. The blue line represents the fixed benefit  $s(z)$  plotted against trait size  $z$ . It has a unique maximum at  $z = b$ . The optimal female preference function  $q_b(z)$  is zero for all  $z \neq b$ , and thus females should reject any male with a trait value in this range. The only trait value for which the preference function is non-zero is  $z = b$ , and so females should accept only males with this trait value.

using equation (3.1). The two sides of equation (3.4) will be equal (and thus  $p$  optimal) if and only if  $p(z) = 0$  almost everywhere on the (open) set of points  $z$  for which  $s(z) \neq \tilde{s}$ .

The optimal preference functions will therefore split males into two groups: the best, and the rest. Only males with the best genes will gain (consensual) matings; all others will be rejected by females. All preference functions that reject all males giving less than the maximum benefit will be equally optimal. Mathematically, this corresponds any preference function with  $p(z) = 0$  for all  $z$  with  $s(z) < \tilde{s}$ . If there is only a unique, optimal male trait value  $z^*$  that maximises  $s$ , there will be a unique optimum preference function, which will be a delta function  $q_{z^*} = \delta(z^* - x)$  (Figure 3.1). More generally, there is no fitness consequence to having a preference between all those male trait values  $z$  for which  $s(z) = \tilde{s}$  (Figure 3.2).

In the natural world, then, if preference is for good genes, all females should choose to mate with the same optimal male type (or possibly with males from a set of types conferring approximately equal benefits), who will therefore monopolise all (consensual) matings. Males with genes worse than the optimum (as

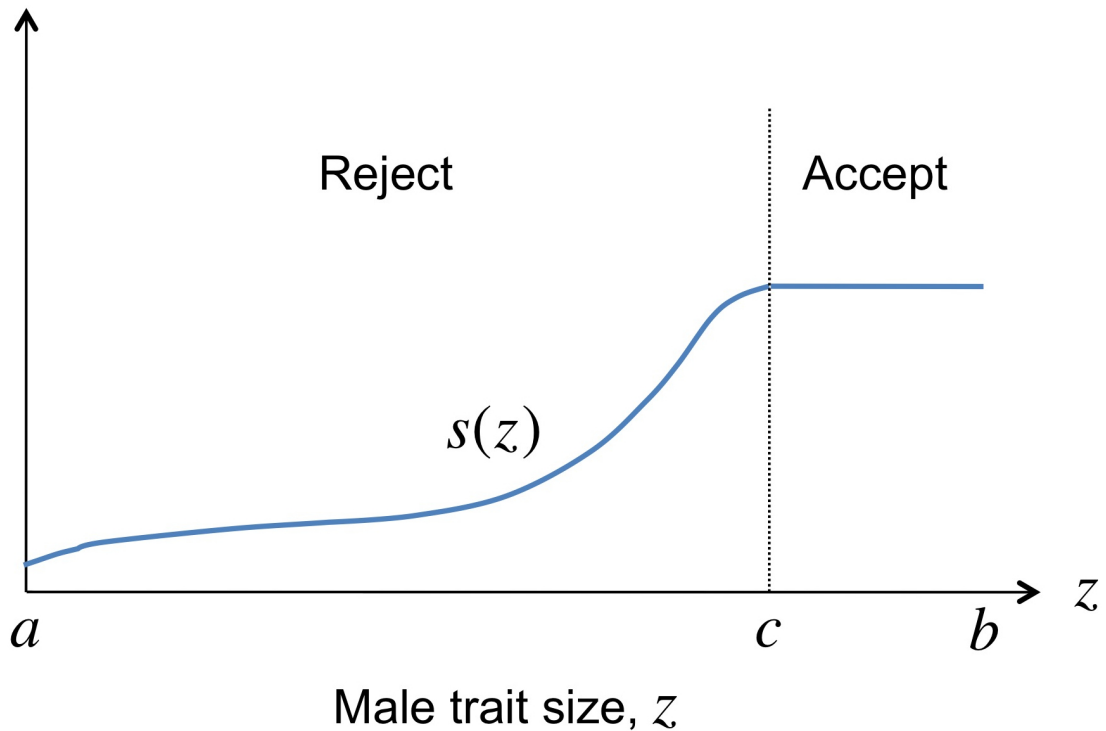


Figure 3.2: Another example of female preference for fixed benefits. The blue line represents the fixed benefit  $s(z)$  plotted against trait size  $z$ . It attains its maximum for trait sizes  $z$  such that  $c \leq z \leq b$ . Optimal female preference functions  $p^*(z)$  will be zero for all  $z < c$ , and thus females should reject any male with a trait value in this range. Optimal preference functions will be non-zero for some trait values  $z \geq c$ , and so females should accept males with trait values in this range. There is no fitness consequence to distinguishing between males with trait values  $z \geq c$ ; all preference functions that reject males with  $z < c$  will be equally optimal. Females are thus likely to be on average indifferent if given a choice between males with trait sizes  $z \geq c$ .

signalled by their trait values) will be rejected. Given a choice between two males, females should always choose the male with the better genes.

### 3.3.2 Dilutable benefits

The idea behind the concept of an evolutionarily stable strategy (ESS) is that a population, all of whose members use the same “equilibrium strategy”, denoted  $p^*$ , should be resistant to invasion by a sufficiently small influx of mutants using a different strategy, denoted  $p$  (see Introduction, section 1.6.3). This notion has been extensively developed in the context of finite strategy games (Maynard Smith, 1982). The situation for games with infinite strategy spaces, such as those in the dilutable benefits case, is somewhat more problematic, the more so when payoff functions are non-linear. Here we shall require that an ESS be a

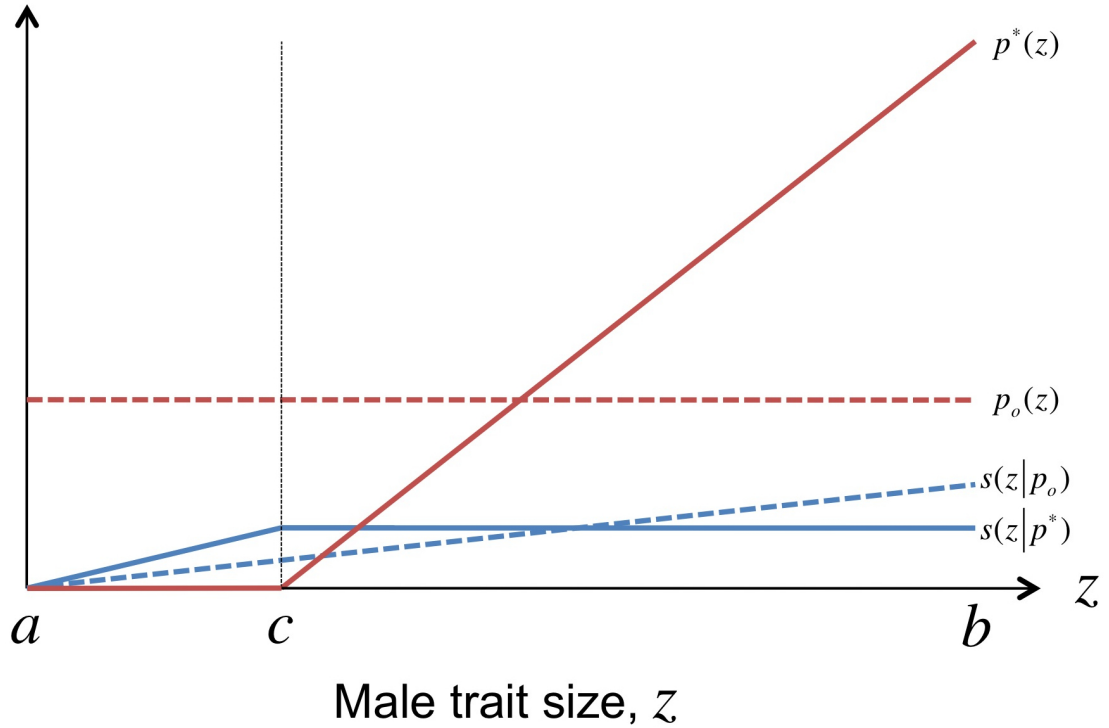


Figure 3.3: An example of female preference for dilutable benefits. The dashed pink line represents the preference function where females consider all males equally attractive,  $p_o(z) = 1/(b - a)$ . The corresponding benefit function  $s(z, p_o(z))$  is shown by the dashed blue line. Note that it is increasing in  $z$ , as per Condition (ii), section 3.2.2. The optimal preference function  $p^*(z)$  is shown by the solid pink line. Females will reject all males with trait values  $z < c$ . Males with trait values  $z \geq c$  will be accepted with increasing likelihood as their trait size increases. The corresponding benefit function  $s(z, p^*(z))$  is shown by the solid blue line. Males with trait values  $z \geq c$  will all give the same benefit, while those with trait values  $z < c$  will give average benefits less than this (and hence are not worth mating with).

*totally uninvadable strategy* (Vickers and Cannings, 1987; Bomze and Pötscher, 1989), so that

$$W[p^*(1 - \varepsilon)p^* + \varepsilon p] > W[p(1 - \varepsilon)p^* + \varepsilon p] \quad (3.5)$$

for all  $0 < \varepsilon \leq 1$ , and all alternative strategies  $p \neq p^*$ . This means that if we begin with an initial population of females who all use the strategy  $p^*$ , and replace a non-zero fraction of size  $\varepsilon$  of this population with a subpopulation of females who all use an alternative strategy  $p \neq p^*$ , then the  $p^*$ -females have higher fitness in this mixed population than do the mutant  $p$ -females.

Firstly, under this criterion, there are no pure strategy equilibria such as the delta function solution given in the fixed benefit case. To see this, suppose that females use a pure strategy; that is, all females in the population mate only with males who have a particular trait value  $z^*$ . In this case, any such male will be



totally depleted, and hence the expected payoff to each female will be zero (since from above,  $s(z^*, q) \rightarrow 0$  as  $q \rightarrow \infty$ ). However, a mutant female who will mate with a male of trait value  $z \neq z^*$  will obtain the positive payoff  $s(z, 0)$ . This shows that mating only with  $z^*$ -males cannot be an equilibrium strategy.

Secondly, there is a unique equilibrium female preference function  $p^*(z)$ , which is an ESS in the above sense (Appendix, section 3.5.2). This function will be continuous on  $[a, b]$ , and will have a threshold trait value  $c$  with  $a \leq c < b$ , such that  $s(z, p^*(z)) = w^*$  (a non-zero constant) for  $c \leq z \leq b$ , while  $p^*(z) = 0$  and  $s(z, p^*(z)) = s(z, 0) < w^*$  for  $z < c$  (applicable only if  $c > a$ ). Finally, for all  $z \in [c, b]$ ,  $p^*(z)$  is monotonically increasing (Figure 3.3).

In nature, then, this means that males with larger ornaments, who would give larger fertility benefits if female preference were uniform, will get more matings than those with smaller ornaments, but they will not have a monopoly on matings. Instead, expected number of matings will decline as ornament size decreases. It may be that all males achieve some matings (so that the threshold value  $c = a$ ), or it may be that some threshold is reached, below which males will receive no (consensual) matings at all (so that the threshold value  $c > a$ ). Interestingly, given a choice between a male with a higher and a male with a lower trait value, females will not always choose to mate with the bearer of the higher trait. Rather there will be some probability of choosing each, with more females choosing the bearer of the higher trait value, who will therefore obtain more matings overall, but some choosing the bearer of the lower trait value, who will receive fewer matings overall. This is because the higher trait value male will obtain more matings and hence will be subject to greater sperm depletion relative to the lower trait value male. In effect the female is negotiating a trade off between a more attractive, but more depleted male, against a less attractive, but also less depleted male. The equilibrium female preferences would be expected to result in the expected benefit from a mating with each male being identical (Figure 3.3).

An intuitive explanation for this result follows; for a mathematical proof see Appendix, section 3.5.2. Suppose the popularity function  $\hat{p}(z)$  gave a resulting benefit function  $s(z, \hat{p}(z))$  so that some trait value  $\tilde{z}$  gave a higher benefit than any other. Then females that prioritised matings with males of this trait value would gain greater benefits, and so any mutant preference function  $p$  that lead to more matings with  $\tilde{z}$ -males would be able to invade. The only preference function that cannot be invaded in this way is the one that results in all males giving equal benefit to females who mate with them.

### 3.4 Discussion

The evolution of female mate choice has been broadly characterised as being due either to “direct” or “indirect” benefits (Andersson and Simmons, 2006). We propose another way to characterise the benefits

females obtain from mate choice, in terms of whether they are “dilutable” or “fixed”. As we have shown in the model above, this distinction will affect female preference. If females are selecting for fixed benefits, they should only mate with males who provide them with the largest payoff, rejecting all others. On the other hand, if females are selecting for dilutable benefits, their mating pattern will follow a probability distribution so that the best males (those with the highest trait values) get more matings, but do not have a monopoly.

We have modelled explicitly for the cases of good genes (fixed) and fertility benefits (dilutable), but our model could equally apply to other hypotheses for female mate choice, as we now consider. Most types of indirect benefits will be fixed, since characteristics inherited genetically will benefit offspring from all matings equally. This logic applies both for benefits to viability and attractiveness. Where genetic benefits arise from genetic compatibility, they are contingent on complementarity between the parents and so will not flow equally to all females. But they are not dilutable, as the gain of one female is independent of others’ choices (in this case each female’s optimal preference function would be different, but the shape would correspond to that for a fixed benefit).

Generally direct benefits will be dilutable. Parenting ability, for example, seems likely to decrease in most cases as the number of offspring with different mothers increases (see the polygyny threshold model; Orians, 1969; Searcy and Yasukawa, 1989 ; although there can be benefits to polygyny too; Altmann et al., 1977). However there are many exceptions where direct benefits are likely to be fixed. A possible example could be where females choose mates for good parenting, and form socially monogamous pairs. Then paternal care is not diluted, as the male only provides care for his social partner’s offspring. Even if the father sires offspring through extra-pair copulations, the benefits he provides are likely to be fixed to first order, unless the time he spends seeking extra-pair matings impedes his provision of parental care (Magrath and Komdeur, 2003). Another example is the case of prey items acting as nuptial gifts. Then the benefit is direct, but it is not dilutable, since it is consumed entirely by a single female. Nuptial gifts such as spermatophores may be somewhat dilutable, however, if a male’s ability to produce a large spermatophore is affected by frequent copulation.

The different potential benefits to female choice are not mutually exclusive, so that there may be combinations of dilutable and fixed benefits. A hypothetical example is a species where females select males for their size and strength, because large, strong males can defend better territory. In this instance the benefit to females is direct, since they gain better territory, and dilutable since sharing the male with other females means each female gets a smaller share of the rewards from the territory. However, if the male’s size and strength are genetic, a female will also gain fixed, indirect benefits through her offspring inheriting

their father's genetic quality. This model shows that female mate choice can itself offer some clues as to the relative strength of the forces underlying its evolution.

A simple way of combining these two scenarios would be to define the benefit function as a sum of dilutable and fixed benefits, so that the benefit from mating with a male of trait size  $z$  and popularity  $q = \hat{p}(z)$  is  $s(z, q) = as_f(z) + bs_d(z, q)$ , where  $a$  and  $b$  are constants,  $s_f(z)$  represents the fixed benefits a female obtains from a mating with a male of trait value  $z$ , and  $s_d(z, q)$  those that are dilutable. Since the overall benefit function  $s(z, q)$  is dilutable, female preference will evolve so that the (overall) benefit from any male is the same (with the exclusion of males that fall below a quality threshold). The relative importance of dilutable to fixed benefits could be estimated by looking at the skew in the number of mates that males obtain; since choice for fixed benefits will lead to only optimal males achieve any matings, we would expect it to produce larger reproductive skew than choice for dilutable benefits. Thus species in which female choice is for good genes would be expected to have high reproductive skew (chapter 4).

Of course our model makes a number of simplifying assumptions, and in reality female choice is likely to be a much more complicated affair. For example, we assume that females are able to assess and compare all potential mates, having no time constraints or costs of mate choice. This is unlikely to be the case. Even if females are not time-constrained, comparing a large number of potential mates may be beyond the cognitive abilities of many species. In reality, females may make errors in assessment (Johnstone, 1994), only be able to assess and choose between a smaller subset of available males, or to accept males of certain trait sizes with conditional probability based on time and cost constraints (Real, 1990; Gibson and Langen, 1996). It would be interesting to see what changes to our conclusions would result from the incorporation of these more realistic elements into our model. We think the qualitative conclusions would be largely unchanged.

If we accept that benefits accruing to a female from a given male can depend on the number of other females he mates with, there is the possibility that the more mates a male has, the higher the expected benefit to each. In some species it is thought that increased polygyny may in fact benefit females. For example, in birds, breeding in the same territory as other females may decrease the chances of nest predation (Searcy and Yasukawa, 1989). This could be modelled by considering a benefit function  $s(z, q)$  that is increasing with increasing popularity  $q$ , rather than decreasing as we have assumed above. Although we have chosen not to consider this possibility, note that under these circumstances, the ESS strategy could be one where all females will choose males of the same trait value  $x$ , if  $s(z, q_z(z)) < s(x, q_x(x))$  for all  $z \neq x$  and delta functions  $q_z$  and  $q_x$ . However, the exact solution is likely to be highly dependent upon the way  $s(z, q)$  varies with  $z$ , giving several possibilities.

Under the “sexy son” hypothesis, by mating with a male bearing a trait value that other females in the population have a preference for, a female gains benefits by maximising the chances of having attractive male offspring (Fisher, 1930; Weatherhead and Robertson, 1979). This could be covered by the last framework, with the benefit accruing to a female for mating with a male of a given trait value increasing if more females in the population have a preference for that value. It is therefore interesting to note the similarity of the result in this case to Fisher’s runaway process. It is also notable that female choice for dilutable benefits in our formulation gives a similar result to the polygyny threshold model (Orians, 1969; Searcy and Yasukawa, 1989; Andersson, 1994), with females balancing the trade-off between attractiveness and sperm depletion, so as to make the expected benefit from each (acceptable) male identical. If the benefit function  $s(z, q)$  declines linearly with increasing  $q$ , the resulting preference function will resemble an ideal free distribution, with males’ popularity being proportional to  $s(z, 0)$ , which could be seen as the total amount of resources they have to give to females. Thus although our formalism is simple and intuitive, it encompasses a wide spectrum of possible patterns of female choice, and uncovers previously unrealised relationships between them.

## 3.5 Appendix

### 3.5.1 Justification of dilutable benefit function formulation

Since the benefit females obtain from males is related to the number of sperm in the male’s ejaculate, and since males are assumed to suffer sperm depletion as they mate more frequently, we can define a second male trait which we call ‘condition’ and denote by  $x$ . Whatever a male’s trait value  $z$ , the lower his condition  $x$  is, the lower the benefit he confers on a female with whom he mates. Suppose the number of matings a male has had when a female mates with him is  $n$ . We assume that a male’s condition is determined by the number of matings he has had: the more matings, the lower the condition. Without loss of generality, then, we take  $x$  to be some increasing function of  $1/n$ , and express the benefit conferred to a female in a given mating by a male with trait value  $z$  who has mated  $n$  times as  $s_0(z, 1/n)$ . Note that this will be a decreasing function of  $n$  for fixed  $z$ . If the average female preference (referred to hereafter as “popularity”) for a given trait value is  $\hat{p}(z) = q$ , then we can assume that males bearing that trait value get  $n$  matings with Poisson probability  $q^n e^{-q}/n!$ . Thus the expected benefit to a female from a mating with a (random) male can be expressed as a function both of his trait value  $z$ , and the popularity  $q = \hat{p}(z)$  for males with that trait value,

$$s(z, q) = \sum_{n \geq 0} s_0 \left( z, \frac{1}{n} \right) \frac{q^n e^{-q}}{n!}.$$

Then  $s(z, q)$  is decreasing in  $q$  (for fixed  $z$ ) since

$$\begin{aligned} \frac{\partial s(z, q)}{\partial q} &= -\sum_{n \geq 0} s_0\left(z, \frac{1}{n}\right) \frac{q^n e^{-q}}{n!} + \sum_{n \geq 0} s_0\left(z, \frac{1}{n}\right) n \frac{q^{n-1} e^{-q}}{n!} \\ &= -\sum_{n \geq 0} s_0\left(z, \frac{1}{n}\right) \frac{q^n e^{-q}}{n!} + \sum_{n \geq 1} s_0\left(z, \frac{1}{n}\right) \frac{q^{n-1} e^{-q}}{(n-1)!} \\ &= \sum_{n \geq 0} \frac{q^n e^{-q}}{n!} \left( s_0\left(z, \frac{1}{n+1}\right) - s_0\left(z, \frac{1}{n}\right) \right) \end{aligned}$$

which is negative since  $s_0(z, c)$  is decreasing in  $c$ .

Defined like this, the benefit function  $s(z, q)$  considered in the main text satisfies conditions (i) and (ii) (provided the function  $s_0(z, x)$  is increasing in  $z$  for fixed  $x$ ). Note that we do not in fact require the conditional benefit function  $s(z, q)$  to take this exact form; only that it obey conditions (i) and (ii) above. The above construction serves as an illustration of why these conditions are reasonable.

### 3.5.2 Proof of results for dilutable case

#### Existence and uniqueness of $p^*$

From conditions (i) and (ii) and surrounding discussion in the main text, we assume that there is a continuous, 2-variable benefit function  $s(z, q)$ , defined for  $a \leq z \leq b$ ,  $q \geq 0$  and satisfying:

$$s(z, q) > 0 \text{ for all } z \text{ and } q, \quad (3.6)$$

$$s(z, q) \text{ is monotonically increasing in } z \text{ for fixed } q, \quad (3.7)$$

$$s(z, q) \text{ is monotonically decreasing in } q \text{ for fixed } z, \quad (3.8)$$

$$s(z, q) \rightarrow 0 \text{ as } q \rightarrow \infty, \text{ for each } z. \quad (3.9)$$

It follows from (3.8) that, for each  $z$ ,  $s(z, q) < s(z, 0)$  for all  $q > 0$ . Further, from (3.7),  $s(a, 0) < s(z, 0) < s(b, 0)$  for all  $z \in [a, b]$ .

Let  $\alpha$  be a constant, with  $0 < \alpha < s(a, 0)$ . Then, by (3.8) and (3.9), for each  $z \in [a, b]$  there is a unique popularity value  $p_\alpha(z) > 0$  such that

$$s(z, p_\alpha(z)) = \alpha \quad (3.10)$$

See Figure 3.4 for the intuition behind this. It follows from the continuity of  $s(z, q)$  that  $p_\alpha(z)$  is continuous in  $z$  and  $\alpha$ . Further, from (3.7),  $p_\alpha(z)$  is monotonically increasing in  $z$  for fixed  $\alpha$ , and from (3.8),  $p_\alpha(z)$  is monotonically decreasing in  $\alpha$  for fixed  $z$ . It also follows from (3.9) that  $p_\alpha(z) \rightarrow \infty$  as  $\alpha \rightarrow 0$  for each  $z$ .

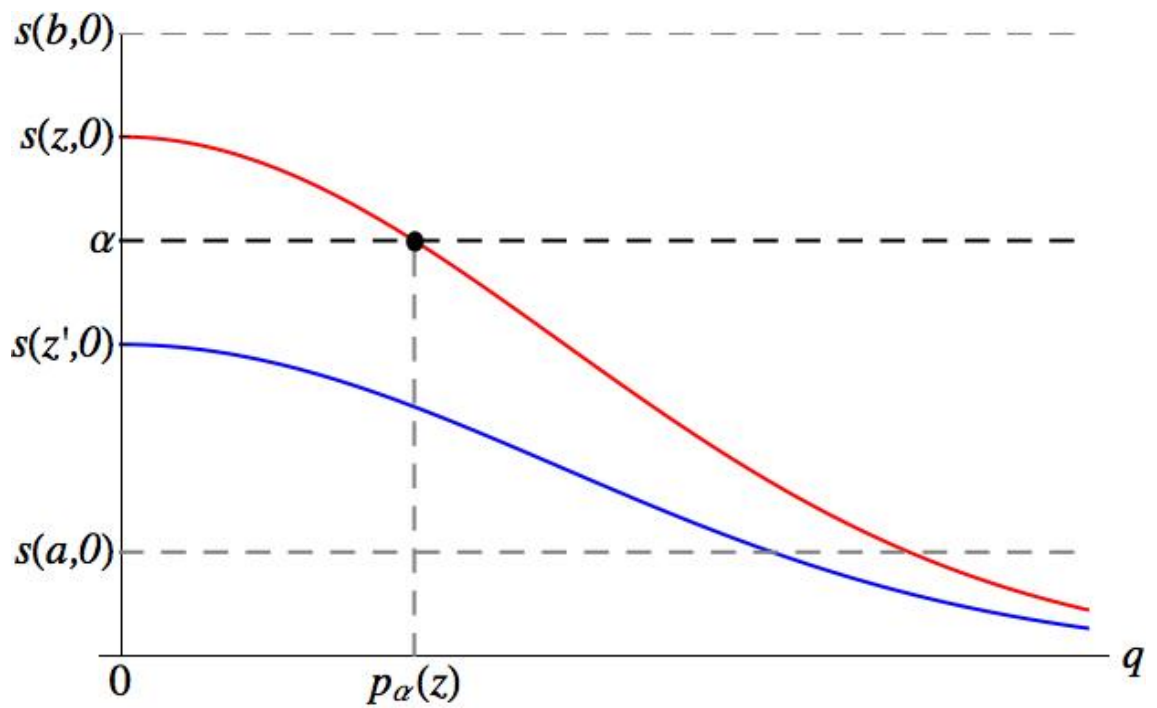


Figure 3.4: Construction of the function  $p_\alpha(z)$ . The red curve is the function  $s(z, q)$  as a function of  $q$  for fixed  $z$ . If  $z$  is such that  $s(z, 0) > \alpha$ , then  $p_\alpha(z)$  exists, as shown. For any  $z' < z$  with  $s(z', 0) < \alpha$ , then the curve  $s(z', p)$ , (blue curve) lies below  $\alpha$  for all  $p \geq 0$ . In this case we set  $p_\alpha(z')$ . Thus,  $p_\alpha(z)$  is defined for all  $z$  in the range  $a \leq z \leq b$ , and all  $0 < \alpha \leq s(b, 0)$ .

Now suppose that  $s(a, 0) \leq \alpha < s(b, 0)$ . Then it follows from (3.7) that there exists a unique  $z_0(\alpha) \in [a, b]$  such that

$$s(z_0(\alpha), 0) = \alpha. \quad (3.11)$$

Further, it follows from (3.7) that  $z_0(\alpha)$  is monotonically increasing in  $\alpha$ . Also,  $s(z, q) < \alpha$  for all  $a \leq z < z_0(\alpha)$  and  $q \geq 0$ . On the other hand, if  $z_0(\alpha) < z \leq b$ , then  $s(z, 0) > \alpha$ , and it follows from (3.8) and (3.9) that there is a unique value  $p_\alpha(z) > 0$  such that (3.10) holds (see Figure 3.4). We also have  $s(z, q) > \alpha$  for  $q < p_\alpha(z)$  and  $s(z, q) < \alpha$  for  $q > p_\alpha(z)$ .

Clearly, by construction,  $p_\alpha(z) \rightarrow 0$  as  $z \rightarrow z_0(\alpha)$ . We therefore define  $p_\alpha(z) = 0$  for  $a \leq z \leq z_0(\alpha)$ . With this definition, it follows that  $p_\alpha(z) \geq 0$  for all  $a \leq z \leq b$ , and from the continuity of  $s(z, q)$ , that  $p_\alpha(z)$  is continuous in both  $z$  and  $\alpha$ . Further, from (3.7), (3.8) and (3.11), it follows that  $p_\alpha(z)$  is monotonically increasing in  $z \geq z_0(\alpha)$  for fixed  $\alpha$ . Also, for fixed  $z > z_0(\alpha)$ ,  $p_\alpha(z)$  decreases as  $\alpha$  increases.

Now suppose that, for  $0 < \alpha < s(b, 0)$ , we define the function  $k(\alpha)$  as

$$k(\alpha) = \int_a^b p_\alpha(z) dz. \quad (3.12)$$

Then it follows from the construction of  $p_\alpha(z)$  that  $k(\alpha)$  is a continuous, monotonically decreasing function of  $\alpha$ . Clearly, if  $\alpha = s(b, 0)$ , then  $z_0(\alpha) = b$ , and  $p_\alpha(z) = 0$  for all  $z$ . Hence,  $k(s(b, 0)) = 0$ .

At the other extreme, since  $p_\alpha(z) \rightarrow \infty$  as  $\alpha \rightarrow 0$ , then given any constant  $\ell > 0$  we may find an  $\alpha_0 > 0$  (depending on  $\ell$ ), such that  $p_\alpha(z) \geq p_{\alpha_0}(z) \geq p_{\alpha_0}(a) \geq \ell$  for all  $a \leq z \leq b$  and  $\alpha < \alpha_0$ . Thus, for such an  $\alpha$ , we have

$$k(\alpha) = \int_a^b p_\alpha(z) dz \geq \ell(b - a).$$

Since  $\ell$  is arbitrary, this shows that  $k(\alpha) \rightarrow \infty$  as  $\alpha \rightarrow 0$ .

As  $k(\alpha)$  increases monotonically from 0 to  $\infty$  as  $\alpha$  decreases from  $s(b, 0)$  to 0, it now follows that there is a *unique*  $\alpha^*$ , with  $0 < \alpha^* < s(b, 0)$ , such that  $k(\alpha^*) = 1$ . Then, setting  $c = z_0(\alpha^*)$  if  $\alpha^* \geq s(a, 0)$ , and  $c = a$  if  $0 < \alpha^* < s(a, 0)$ , we define  $p^*(z) = p_{\alpha^*}(z)$ . We therefore have that

$$\int_a^b p^*(z) dz = \int_c^b p^*(z) dz = 1. \quad (3.13)$$

This shows that  $p^*(z)$  is a probability density function on  $[a, b]$  satisfying: (i)  $p^*(z)$  is continuous; (ii)  $p^*(z) = 0$  for  $z < c$  (if  $c > a$ ); (iii)  $p^*(z)$  is monotonically increasing for  $c \leq z < b$ . It completes the construction of the unique candidate ESS equilibrium  $p^*$ .

**Proof that  $p^*$  is an ESS**

We have constructed the probability density function  $p^*(z)$  so that

$$p^*(z) \text{ is continuous on } [a, b], \quad (3.14)$$

$$p^*(z) = 0 \text{ for } z \in [a, c) \text{ (applicable only if } c > a), \quad (3.15)$$

$$p^*(z) \text{ is monotonically increasing for } z \in [c, b], \quad (3.16)$$

$$s(z, p^*(z)) = w^*, \text{ a constant, for } z \in [c, b], \quad (3.17)$$

$$s(z, p^*(z)) = s(z, 0) < w^* \text{ for } z \in [a, c). \quad (3.18)$$

We need to prove that

$$W[p^*|(1-\varepsilon)p^* + \varepsilon p] > W[p|(1-\varepsilon)p^* + \varepsilon p] \quad (3.19)$$

for all  $0 < \varepsilon \leq 1$  and  $L_2$ -probability densities  $p \neq p^*$ .

To prove this result, we need to make a stronger assumption than (3.8), namely that

$$s(z, q) \text{ is differentiable in } q \text{ for each } z, \text{ and } \frac{\partial s}{\partial q}(z, q) < 0 \text{ for all } q > 0. \quad (3.20)$$

For any  $L_2$ -probability density function  $p(z)$  defined for  $z \in [a, b]$ , we let  $\int_a^c p(z)dz = m$ , and  $\int_c^b p(z)dz = 1 - m$ , with  $0 \leq m \leq 1$ . Let  $v = p^* - p$ . Then

$$\int_a^c v(z)dz = - \int_a^c p(z)dz = -m, \text{ and} \quad (3.21)$$

$$\int_c^b v(z)dz = m. \quad (3.22)$$

Thus, from (3.15), (3.17), (3.18), (3.21), and (3.22), we have

$$\begin{aligned} \int_a^b v(z)s(z, p^*(z))dz &= \int_a^c v(z)s(z, 0)dz + w^* \int_c^b v(z)dz \\ &= - \int_a^c p(z)s(z, 0)dz + w^* \int_c^b v(z)dz \\ &\geq -w^* \int_a^c p(z)dz + w^* m \\ &= -w^* m + w^* m \\ &= 0. \end{aligned}$$



That is

$$\int_a^b v(z)s(z, p^*(z))dz \geq 0, \quad (3.23)$$

with equality if and only if  $m = 0$ ; i.e. if and only if  $v(z) = -p(z) = 0$  almost everywhere on  $[a, c)$ .

Now observe that  $(1 - \varepsilon)p^* + \varepsilon p = p^* - \varepsilon v$ . Using the assumption (3.20), we apply the Mean Value Theorem in the form

$$\begin{aligned} s(z, p^*(z) - \varepsilon v(z)) - s(z, p^*(z)) &= - \int_{p^*(z) - \varepsilon v(z)}^{p^*(z)} \frac{\partial s}{\partial q}(z, q) dq \\ &= -\varepsilon v(z) \int_0^1 \frac{\partial s}{\partial q}(z, p^*(z) - t\varepsilon v(z)) dt. \end{aligned} \quad (3.24)$$

Then,  $W[p^*(1 - \varepsilon)p^* + \varepsilon p] - W[p(1 - \varepsilon)p^* + \varepsilon p] = W[v|p^* - \varepsilon v]$ , and by (3.24) and (3.23),

$$\begin{aligned} W[v|p^* - \varepsilon v] &= \int_a^b v(z)s(z, p^*(z) - \varepsilon v(z))dz \\ &= \int_a^b v(z)s(z, p^*(z))dz - \varepsilon \int_a^b v(z)^2 \left\{ \int_0^1 \frac{\partial s}{\partial q}(z, p^*(z) - t\varepsilon v(z)) dt \right\} dz \\ &\geq -\varepsilon \int_a^b v(z)^2 \left\{ \int_0^1 \frac{\partial s}{\partial q}(z, p^*(z) - t\varepsilon v(z)) dt \right\} dz \\ &> 0 \end{aligned}$$

for any  $0 < \varepsilon \leq 1$ , since  $\partial s / \partial q < 0$  by assumption (3.20), and  $p \neq p^*$  implies that  $v^2 > 0$  almost everywhere on  $[a, b]$ . This shows that (3.19) holds, and hence that  $p^*$  is an ESS.

## **Part II**

# **The consequences of mate choice**

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The following three chapters detail research into the consequences of mate choice.

Chapter 4 reviews existing literature in order to ascertain whether or not the consequences of the second chapter's predicted forms of mate choice are found in reality. 38 published studies of wild populations of bird species are analysed. From each we assess the mating skew using two different measures, and show that lekking species have significantly higher mating skew than non-lekking species. This result is in line with the prediction of chapter 3, since in leks females are more likely to be concerned with fixed benefits as they receive only sperm from their mates.

Chapter 5 is an investigation into the Strawberry poison-dart frog, *Oophaga pumilio*. A wide variety of colours is seen between populations of this frog in the Bocas del Toro archipelago in Panama, especially when compared to other sympatric poison-frog species that share the same ecology. We use a quantitative genetic approach to investigate the hypothesis that this polymorphism is caused by female choice, and discover that in principle this could occur.

Chapter 6 shows that male post-copulatory strategy could be affected by pre-copulatory female choice. A game theoretical model shows that males that pay a smaller resource cost to obtain matings will be selected to invest less ejaculate in each mating. If males all pay the same resource cost to obtain matings they will all invest the same amount of ejaculate in each.

## Chapter 4

# Comparative study of mating skew in bird populations

Dividing mate choice into that for fixed and that for dilutable benefits, as suggested in the previous chapter, leads to the prediction that choice for good genes should result in high reproductive skew. One example of a situation in which choice is predicted to be for good genes is in lekking bird species. Taking data from 38 published studies into wild populations of both lekking and non-lekking species of birds, we found that the lekking species did indeed show significantly higher mating skew than non-lekking species for two different measures of skew. This is in line with the hypothesis that choice for good genes gives higher reproductive skew; therefore the hypothesis is not falsified.

### 4.1 Background

The theoretical model in chapter 3 made the prediction that species in which the females obtain fixed benefits from choice of mate should show a higher reproductive skew than those in which the females obtain dilutable benefits from choice of mate. In order to test this hypothesis, we conducted a comparative study of species of birds. The decision to concentrate on a single class was made in order to minimise as far as possible ecological differences, and also to focus the study. Birds were chosen because of the large number of available studies with data on wild populations.

Many bird species mate in leks. A lek is a group of males that accumulate, often at traditional sites, during the breeding season. Females visit the lek site, and choose their mates. Leks are of great theoretical interest because of the lek paradox (see Introduction, section 1.4.2), and also because they allow biologists

an excellent opportunity to understand female choice, since in lekking species males appear to provide only sperm to the female (Andersson, 1994); they offer neither paternal care, nor territory. Therefore they are often suggested to be an example of female choice for good genes. Good genes benefits are fixed by the categorisation of chapter 3, and therefore in leks we would expect to see high mating skew. In non-lekking species, on the other hand, males are frequently described as providing territory or paternal care in addition to sperm, and thus female choice is more likely to be governed by dilutable benefits. Therefore we would expect to see low mating skew. To test this assertion, we examined data on mating distributions in wild populations of both lekking and non-lekking birds, taken from a wide variety of sources in the literature. We then attributed two mating skew values to each population, and compared these scores between populations, and between lekking and non-lekking species.

## 4.2 Methods

### 4.2.1 Data retrieval

We obtained data from 38 published studies, all of which gave data on the distributions of matings or of paternity achieved by males in at least one wild population. The studies were found from references in chapter 6 of Andersson, 1994; in Kokko et al., 1998; and in Griffith et al., 2002; with the addition of the more recent study by Balenger et al. (2009), which was suggested by Ian Owens (personal communication). Of these references, we used only those that concerned field studies of wild populations, with no experimental manipulation. Often these populations were control groups as part of a larger experiment. Some of the studies gave mating distribution (number of copulations each male was observed to have), while others gave a measure of paternity distribution using molecular techniques. We treated the two types of study equally in order to have as large a dataset as possible.

Some studies (Lenington, 1980; Simmons, 1988a,b; Lightbody and Weatherhead, 1988; Kraaijeveld et al., 2004) failed to record the number of males that achieved no matings at all. For these we calculated the skew measurements  $\lambda$ ,  $\lambda_{\min}$ ,  $\lambda_{\max}$ , and  $I_{\delta}$  with the data as given, and also with the data plus 100 males that achieved no matings. For  $\lambda$ ,  $\lambda_{\min}$ , and  $\lambda_{\max}$ , in all populations the measurements were broadly similar with and without the addition of the extra 100 males (all  $\lambda_{\min}$  and  $\lambda_{\max}$  values within 0.03 of the value for the original data;  $\lambda$  values all within 0.06 of the original), and so we have kept to the original data. For  $I_{\delta}$ , however, in all populations the addition of 100 males led to enormous variation in skew, and therefore for the purposes of our analysis using this measure we left these studies out.

CHAPTER 4. COMPARATIVE STUDY OF MATING SKEW IN BIRD POPULATIONS

Species	Lek?	Number of populations	References
<i>Acrocephalus arundinaceus</i> ; Great reed warbler	No	1	Catchpole, 1986
<i>Agelaius phoeniceus</i> ; Redwinged blackbird	No	6	Searcy, 1979; Lenington, 1980; Weatherhead and Boag, 1995
<i>Calamospiza melanocorys</i> ; Lark bunting	No	1	Pleszczynska, 1978
<i>Circus cyaneus</i> ; Northern harrier	No	2	Simmons, 1988a,b
<i>Cygnus atratus</i> ; Black swan	No	1	Kraaijeveld et al., 2004
<i>Dendroica petechia</i> ; Yellow warbler	No	1	Yezerinac and Weatherhead, 1997
<i>Dolichonyx oryzivorus</i> ; Bobolink	No	1	Bollinger and Gavin, 1991
<i>Euplectes progne</i> ; Long-tailed weaverbird	No	1	Andersson, 1982
<i>Hirundo rustica</i> ; Barn swallow	No	1	Saino et al., 1997
<i>Junco hyemalis</i> ; Dark-eyed junco	No	1	Ketterson et al., 1998

*Continued on next page*

CHAPTER 4. COMPARATIVE STUDY OF MATING SKEW IN BIRD POPULATIONS

Species	Lek?	Number of populations	References
<i>Luscinia svecica</i> ; Bluethroat	No	1	Johnsen et al., 2001
<i>Setophaga ruticilla</i> ; American redstart	No	1	Perreault et al., 1997
<i>Sialia currocoides</i> ; Mountain bluebird	No	1	Balenger et al., 2009
<i>Spiza americana</i> ; Dickcissel	No	1	Zimmerman, 1966
<i>Telmatoodytes palustris</i> ; Long-billed marsh wren	No	1	Verner and Engelsens, 1970
<i>Wilsonia citrina</i> ; Hooded warbler	No	1	Stutchbury et al., 1997
<i>Xanthocephalus xanthocephalus</i> ; Yellow-headed blackbird	No	1	Lightbody and Weatherhead, 1988
<i>Centrocercus urophasianus</i> ; Sage grouse	Yes	1	Gibson and Bradbury, 1985
<i>Euplectes jacksoni</i> ; Jackson's widowbird	Yes	1	Andersson, 1989
<i>Gallinago media</i> ; Great snipe	Yes	3	Höglund and Lundberg, 1987; Höglund and Robertson, 1990

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CHAPTER 4. COMPARATIVE STUDY OF MATING SKEW IN BIRD POPULATIONS

Species	Lek?	Number of populations	References
<i>Molothrus ater</i> ; Cowbird	Yes	2	West et al., 1981
<i>Paradisaea minor</i> ; Lesser bird of paradise	Yes	1	Beehler, 1983
<i>Parotia lawesii</i> ; Lawes' parotia	Yes	1	Pruett-Jones and Pruett-Jones, 1990
<i>Pavo cristatus</i> ; Peafowl	Yes	2	Petrie et al., 1991; Yasmin and Yahya, 1996
<i>Perissocephalus tricolor</i> ; Capuchinbird	Yes	1	Trail, 1990
<i>Philomachus pugnax</i> ; Ruff	Yes	4	Hill, 1991
<i>Ptilonorhynchus violaceus</i> ; Satin bowerbird	Yes	3	Borgia, 1985; Borgia and Collis, 1989
<i>Rupicola rupicola</i> ; Cock-of-the-rock	Yes	1	Trail, 1990
<i>Tetrao tetrix</i> ; Black grouse	Yes	11	Alatolo et al., 1992
<i>Tryngites subruficollis</i> ; Buff-breasted sandpiper	Yes	3	Pruett-Jones, 1988; Lanctot et al., 1997

*Continued on next page*



Species	Lek?	Number of populations	References
<i>Tympanuchus cupido pinnatus</i> ; Greater Prairie Chicken	Yes	2	Robel, 1966
<i>Tympanuchus phasianellus</i> ; Sharp-tailed grouse	Yes	3	Gratson et al., 1991

Table 4.1: Data used for the comparative analysis.

The combined data covers 32 different species, and a total of 62 different populations. Of these, 15 species (39 populations) were described as lekking, while the remaining 17 species (23 populations) were not. For details of the studies used and species involved, see Table 4.1 above.

#### 4.2.2 Mating skew measurement

There are several different metrics for mating skew (Morisita, 1962; Keller and Reeve, 1994; Pamilo and Crozier, 1996; Kokko and Linstrom, 1997; Tsuji and Tsuji, 1998; Nonacs, 2000), and an ongoing debate in the literature as to which is the most appropriate for different situations (Kokko et al., 1999; Tsuji and Kasuya, 2001; Nonacs, 2003). To get around this we have used two mating skew measures here so as to guard against our results being an artefact of the choice of skew measure. The two metrics we chose are the  $\lambda$  of Kokko and Linstrom (1997) and Kokko et al. (1998), and the  $I_\delta$  of Morisita (1962) and Tsuji and Tsuji (1998). Details of how they are calculated are given below.  $\lambda$  was initially developed for measuring skew in lekking populations given the mating distribution for a population, so was a natural choice.  $I_\delta$  was chosen as, unlike many other measures, it is not directly dependent on average reproductive success (Tsuji and Tsuji, 1998; Tsuji and Kasuya, 2001), ideal for us because the wide variety of populations we measure come with a wide variety of average reproductive success.

##### Calculation of $\lambda$

We rank a hypothetical set of  $N$  males in order of attractiveness, so that the top-ranked male is first, the second-ranked next, and so on. With skew  $\lambda$ , and total number of males  $N$ , we define the probability that a

given mating goes to the  $i$ -ranked male, denoted  $E[p_i]$ , as

$$E[p_i] = \frac{\lambda(1-\lambda)^{i-1}}{1-(1-\lambda)^N}. \quad (4.1)$$

The maximum skew value is  $\lambda = 1$ , for which the top-ranked male is expected to get all of the matings. The minimum skew is  $\lambda = 0$ , for which all males have an expected number of matings equal to  $1/N$ . Using the probability distribution defined by equation (4.1), and a given total number of matings  $n$ , we use Mathematica 7.0 (Wolfram, 2007) to produce 1000 sample mating distributions for all  $\lambda$ -values from  $\lambda = 0.01$  to  $\lambda = 0.99$  in steps of 0.01. Each of these sample distributions has a sample variance  $t^2$ , so we have a set of 1000 sample variances  $t^2$  for each  $\lambda$ -value. We denote the median  $t^2$ -value of this set for a given  $\lambda$  by  $s_\lambda^2$ .

Given a distribution of  $n$  matings across  $N$  real males observed in a wild population, we denote the observed variance in number of matings per male by  $s_{\text{obs}}^2$ . Our mating skew measure  $\lambda$  for the real population is then defined as that which gives the lowest possible value  $|s_\lambda^2 - s_{\text{obs}}^2|$  (the difference between median sample variance and observed variance). In some cases,  $\lambda$  cannot be uniquely determined, because several values fulfil the above criterion equally. This problem was not explicitly mentioned by Kokko and Linstrom (1997) (though it was noted by Tsuji and Kasuya, 2001). We have solved it by taking the median of the possible values as our  $\lambda$ , since in all cases the possible  $\lambda$ -values formed an interval.

We also calculate lower and upper bounds  $\lambda_{\min}, \lambda_{\max}$  for each population. Our method here is a slight departure from that used in Kokko and Linstrom (1997). We define the bounds as being the minimum and maximum values of  $\lambda$  for which the observed data would pass a chi-squared goodness-of-fit test with expected number of matings for male  $i$  given by  $nE[p_i]$  (Chapter 17, Sokal and Rohlf, 1995). Populations which pass chi-squared goodness of fit tests for  $\lambda = 0.01$  are also tested against the  $E[p_i] = 1/N$  (all males equal) distribution corresponding to  $\lambda = 0$ . Populations which pass chi-squared goodness of fit tests for  $\lambda = 0.99$  were assigned  $\lambda_{\max} = 1$  if only a single male achieved any matings.

The Mathematica code implementing this is available on request.

### Calculation of $I_\delta$

We have  $n$  matings distributed across  $N$  males in the population, as above. Label each male  $1, 2, \dots, N$ , and denote the mating success of the  $i$ th male by  $x_i$ . The mating skew measure  $I_\delta$  is then

$$I_\delta = N \frac{\sum_{i=1}^N x_i^2 - \sum_{i=1}^N x_i}{\left(\sum_{i=1}^N x_i\right)^2 - \sum_{i=1}^N x_i}. \quad (4.2)$$

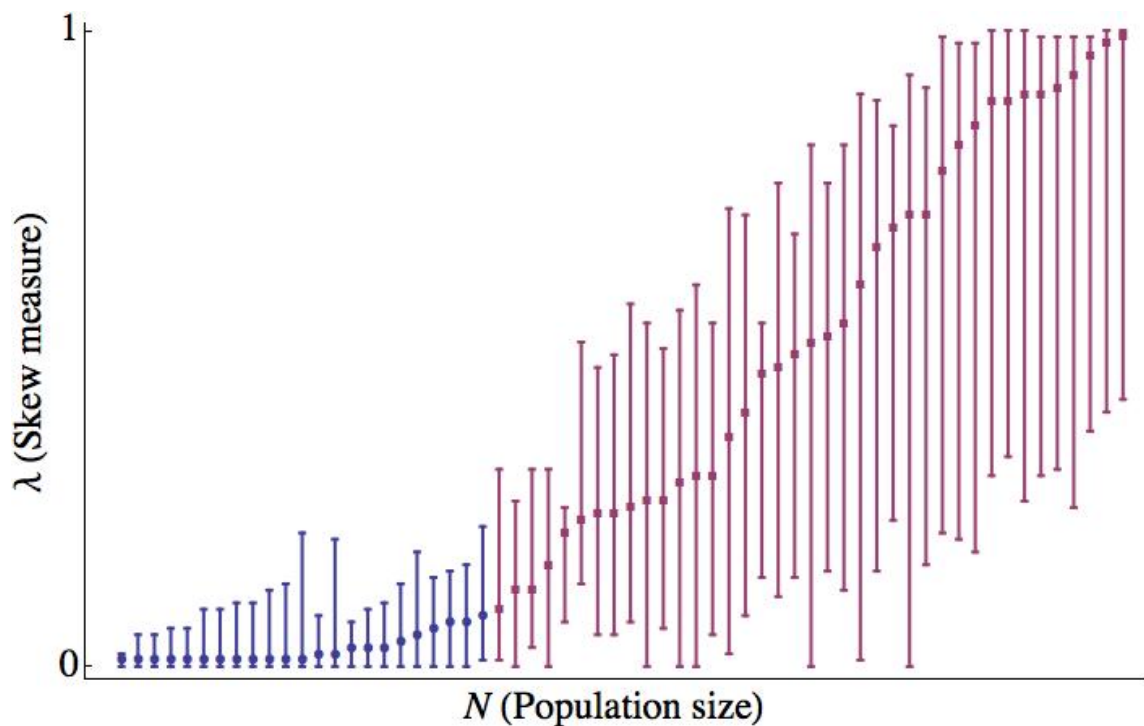


Figure 4.1: Mating skew in sixty-two populations of birds. The populations are displayed in order of increasing skew measure  $\lambda$ , from left to right. Lekking populations, in which females receive only sperm from males, are displayed in pink, while non-lekking populations are displayed in blue. Every lekking population has a higher skew than every non-lekking population, in line with the prediction. See Appendix, section 4.5 for details of the identity of each population.

$I_\delta$  varies from 0 to  $N$ . As can be seen from equation (4.2) it is directly dependent on group size, unlike  $\lambda$ .

## 4.3 Results

### 4.3.1 Comparison of $\lambda$ -values

Every lekking population we looked at had a higher  $\lambda$ -value than every non-lekking population (Figure 4.1, Appendix section 4.5). These results therefore strongly suggest that lekking species generally have higher mating skew than non-lekking species. To confirm this we performed a statistical analysis on the two groups of data.

In order to get around the fact that many of the data points come from the same species and thus can't be considered to be independent, we took only one  $\lambda$ -value per species. To make our analysis as conservative as possible (in the sense of making lekking and non-lekking groups as similar as possible and then seeing whether there is still a statistically significant difference between them) we took the lowest  $\lambda$ -value given when there were multiple values for a lekking species, and the highest  $\lambda$ -value when there were multiple

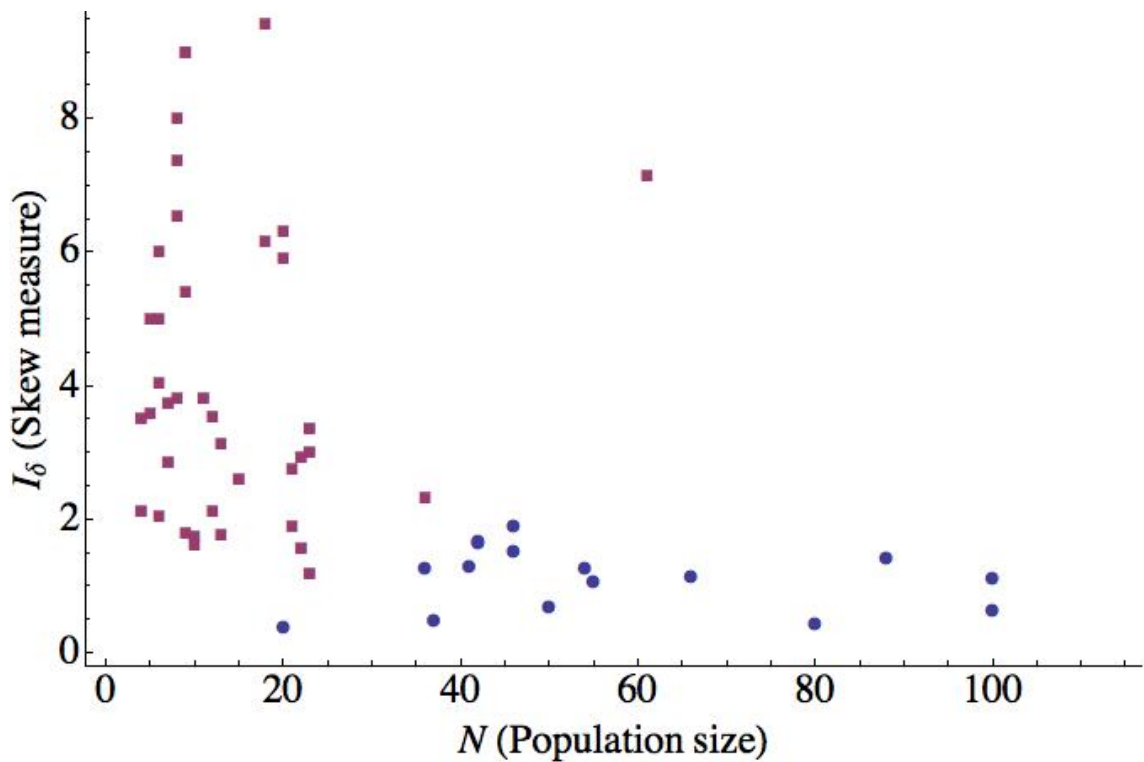


Figure 4.2: Mating skew  $I_\delta$  against population size in fifty-six populations of birds. As in Figure 4.1, lekking populations are marked by pink squares, while non-lekking populations are marked by blue circles. Population sizes are measured as number of males. Even though  $I_\delta$  is sensitive to population size (having maximum value  $N$ ), the generally smaller lekking populations tended to have higher mating skew than the generally larger non-lekking populations.

values for a non-lekking species. Excluded populations are marked with an asterisk in table 4.2 in the Appendix, section 4.5. After these exclusions, the mean  $\lambda$ -values were  $\bar{\lambda}_n = 0.025$  for non-lekking species, and  $\bar{\lambda}_l = 0.41$  for lekking species.

We employed the Mann-Whitney  $U$ -test for two samples, which showed a highly significant difference between the two groups ( $U = 255, P < 0.001$ ; Rohlf and Sokal, 1969; Sokal and Rohlf, 1995), unsurprisingly given the fact they don't overlap at all. So mating skew as measured by  $\lambda$  is significantly higher in lekking species than in non-lekking species.

### 4.3.2 Comparison of $I_\delta$ -values

As mentioned previously, we left out populations in which the number of males without any mates at all was unrecorded (Lenington, 1980; Simmons, 1988a,b; Lightbody and Weatherhead, 1988; Kraaijeveld et al., 2004; marked with two asterisks in table 4.2 in the Appendix, section 4.5).

There was still a clear difference in skew between lekking and non-lekking populations when using  $I_\delta$

as the measure (Figure 4.2), despite the effect population size  $N$  has on  $I_\delta$  (Tsuji and Kasuya, 2001). The mean  $I_\delta$ -values were  $\bar{I}_{\delta n} = 1.07$  for non-lekking species, and  $\bar{I}_{\delta l} = 4.18$  for lekking species.

As before we employed the Mann Whitney  $U$ -test, which once more showed a highly significant difference between the two groups ( $U = 645, P < 0.001$ ; Rohlf and Sokal, 1969; Sokal and Rohlf, 1995), again indicating higher skew in lekking species than in non-lekking species.

## 4.4 Discussion

Using a meta-analysis we have shown that mating skew is greater in lekking species than in non-lekking species. How much does this confirm the theoretical work in chapter 3?

In lekking species, the females are generally considered to only receive sperm from the males (Andersson, 1994). Of the variety of benefits discussed above in the Introduction (section 1.4), the only ones that will apply in such an instance are the good genes, and fertility benefits. By our characterisation in chapter 3, good genes benefits are considered fixed, while fertility benefits are dilutable.

In non-lekking species, many studies suggested that the males provide territory or paternal care. By our characterisation in chapter 3, these would be dilutable benefits. Thus our prediction before the review was that lekking species would show higher mating skew than non-lekking species, because females in lekking species are more concerned with fixed benefits than are females in non-lekking species. Our prediction is therefore in line with these results.

However, it is difficult to say how much this study supports our theory. All we have shown is that lekking species show higher mating skew. We can say nothing from this data as to causation. It could be that species in which females are looking for fixed good genes benefits exhibit mate choice where they pick only the best male, as predicted by our theory. This then could lead to high mating skew, in turn leading to the evolution of leks. Alternatively, it could be that leks themselves lead to higher mating skew, for example by somehow constraining the choice of all females to the same small subset of males.

One way in which we could test this would be by focussing on the lekking species in which mating skew was relatively low, for example the Sage grouse, *Centrocercus urophasianus* ( $\lambda = 0.09, I_\delta = 1.18$ ; Gibson and Bradbury, 1985) and the Satin bowerbird, *Ptilorhynchus violaceus* ( $\lambda = 0.12, I_\delta = 1.57$ ; Borgia and Collis, 1989). If our hypothesis is correct then in these species females might be getting something other than fixed mating benefits, for example fertility benefits. We were unable to ascertain this information from the literature.

There is already a large body of theoretical work on the evolution of reproductive skew (see reviews by Keller and Reeve, 1994; Johnstone, 2000; Buston et al., 2007), much of which focusses on the evolution of

skew in stable groups. These models tend to focus on how the matings are shared out between males (as an aside, some of these models have also been applied to eusocial species where the females are those with high mating skew), treating the females as a passive resource that needs to be shared out. Our predictions, by contrast, assume that females are in control of mating skew, with males passive. In reality the mating skew is likely to be governed by a complicated coevolutionary process dependent on both female choice and male-male competition. We don't claim that the mate choice patterns predicted by our theory are the only force governing skew, as there will be other factors predicted by skew theory that will also have a bearing.

Evidence exists showing that mating skew generally decreases as lek size increases (Widemo and Owens, 1995; Kokko et al., 1998). This could potentially provide an alternative explanation for our results, since there is a significant difference in number of males between lekking and non-lekking populations in our study, with lekking populations generally much smaller (Mann-Whitney  $U$ -test,  $U = 868.5$ ,  $P < 0.001$ ). Therefore it could be that larger groups cause a lower mating skew in general, and that whether a species is defined as 'lekking' or 'non-lekking' is a function of group size more than anything. Perhaps above a certain size of group, skew becomes so low that field biologists no longer define the accumulation of males as a 'lek', and therefore our study's finding that leks exhibit higher skew is tautological. This seems unlikely, however, as the definition of a lek involves males providing only sperm to the females, rather than explicitly mentioning high skew (Andersson, 1994). Another possibility is that leks themselves promote high mating skew, but that above a certain group size lekking breaks down and mating skew decreases. This could work because in small lekking groups females can assess all males, while in larger non-lekking groups they can assess only a subset of males. Therefore even if they are following the "pick the best male" fixed benefit-type strategy from chapter 3, mating skew will not be as high as it would be if they were able to assess all males.

Finally, there is a problem with lack of independence of the data points in our analysis. This is twofold. First, many of the datapoints are from different populations of the same species, or even from the same population at different times. Therefore they are clearly not independent. Second, even given those datapoints that are not from the same species, there is likely to be some shared evolutionary history between closely-related species, meaning that these are not truly independent either. To account for this we would have needed to do a proper phylogenetic comparative study (Bennett and Owens, 2002), but this would have required a full phylogeny.

To sum up, then, there are many other factors that could affect mating skew other than whether the benefits females receive are fixed or dilutable. Many of these elements also covary with whether species are lekking or non-lekking, and so there is the possibility that one of these elements is the real causal factor. A

full study would therefore have taken a great deal of time, and much more data, in order to try to tease apart these different factors, but may be worth it in the future (see 7.3.3 in the Conclusion). The purpose of this study, however, was to do a preliminary test to see whether skew could be shown to be higher in lekking species. Although there are these problems with our study, it is certain that our prediction in chapter 3 is at the very least not falsified by these results, as it would have been if we had found that lekking species did not have a greater mating skew than non-lekking species. As it is, the results we have found do not contradict our prediction that female choice for fixed benefits leads to higher mating skew.

## 4.5 Appendix

The full results are as follows:

Study	Species	$\lambda_{\min}$	$\lambda$	$\lambda_{\max}$	$I_{\delta}$	Lek?
Bollinger and Gavin, 1991	<i>Dolichonyx oryzivorus</i>	0	0.01	0.02	0.43	No
Stutchbury et al., 1997	<i>Wilsonia citrina</i>	0	0.01	0.05	1.12	No
Verner and Engelsens, 1970	<i>Telmatodytes palustris</i>	0	0.01	0.05	0.43	No
Simmons, 1988b <sup>*,**</sup>	<i>Circus cyaneus</i>	0	0.01	0.06	0.70	No
Zimmerman, 1966	<i>Spiza americana</i>	0	0.01	0.06	0.63	No
Balenger et al., 2009	<i>Sialia currocooides</i>	0	0.01	0.09	1.05	No
Lightbody and Weatherhead, 1988 <sup>**</sup>	<i>Xanthocephalus xanthocephalus</i>	0	0.01	0.09	0.81	No
Ketterson et al., 1998	<i>Junco hyemalis</i>	0	0.01	0.1	0.67	No
Simmons, 1988a <sup>**</sup>	<i>Circus cyaneus</i>	0	0.01	0.1	0.47	No
Lenington, 1980 <sup>*,**</sup>	<i>Agelaius phoeniceus</i>	0	0.01	0.12	0.65	No
Catchpole, 1986	<i>Acrocephalus arundinaceus</i>	0	0.01	0.13	0.47	No
Pleszczyńska, 1978	<i>Calamospiza melanocorys</i>	0	0.01	0.21	0.38	No
Yezerinac and Weatherhead, 1997	<i>Dendroica petechia</i>	0	0.02	0.08	1.14	No
Lenington, 1980 <sup>*,**</sup>	<i>Agelaius phoeniceus</i>	0	0.02	0.2	1.02	No
Weatherhead and Boag, 1995 <sup>*</sup>	<i>Agelaius phoeniceus</i>	0	0.03	0.07	1.42	No
Kraaijeveld et al., 2004 <sup>**</sup>	<i>Cygnus atratus</i>	0	0.03	0.09	1.21	No
Saino et al., 1997	<i>Hirundo rustica</i>	0	0.03	0.1	1.25	No
Johnsen et al., 2001	<i>Luscinia svecica</i>	0	0.04	0.13	1.28	No
Andersson, 1982	<i>Euplectes progne</i>	0	0.05	0.18	1.26	No
Searcy, 1979 <sup>*</sup>	<i>Agelaius phoeniceus</i>	0	0.06	0.14	1.52	No

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Study	Species	$\lambda_{\min}$	$\lambda$	$\lambda_{\max}$	$I_{\delta}$	Lek?
Searcy, 1979*	<i>Agelaius phoeniceus</i>	0	0.07	0.15	1.64	No
Searcy, 1979	<i>Agelaius phoeniceus</i>	0	0.07	0.16	1.67	No
Perreault et al., 1997	<i>Setophaga ruticilla</i>	0.01	0.08	0.22	1.90	No
Gibson and Bradbury, 1985	<i>Centrocercus urophasianus</i>	0.01	0.09	0.31	1.18	Yes
Borgia and Collis, 1989	<i>Ptilorhynchus violaceus</i>	0	0.12	0.26	1.57	Yes
Andersson, 1989	<i>Euplectes jacksoni</i>	0.03	0.12	0.31	2.31	Yes
Borgia, 1985*	<i>Ptilorhynchus violaceus</i>	0	0.16	0.31	1.88	Yes
Trail, 1990	<i>Rubicola rubicola</i>	0.07	0.21	0.25	7.15	Yes
Höglund and Lundberg, 1987	<i>Gallinago media</i>	0.13	0.23	0.51	1.76	Yes
Pruett-Jones, 1988	<i>Tryngites subruficollis</i>	0.05	0.24	0.47	2.74	Yes
Alatolo et al., 1992	<i>Tetrao tetrrix</i>	0.05	0.24	0.49	3.01	Yes
Borgia, 1985*	<i>Ptilorhynchus violaceus</i>	0.07	0.25	0.57	2.93	Yes
Yasmin and Yahya, 1996	<i>Pavo cristatus</i>	0	0.26	0.54	1.61	Yes
Pruett-Jones and Pruett-Jones, 1990	<i>Parotia lawesii</i>	0.06	0.26	0.5	3.36	Yes
Petrie et al., 1991*	<i>Pavo cristatus</i>	0	0.29	0.56	1.75	Yes
Höglund and Lundberg, 1987*	<i>Gallinago media</i>	0	0.3	0.6	2.12	Yes
Gratson et al., 1991	<i>Tympanuchus phasianellus</i>	0.05	0.3	0.54	2.60	Yes
Gratson et al., 1991*	<i>Tympanuchus phasianellus</i>	0.02	0.36	0.72	1.80	Yes
Alatolo et al., 1992*	<i>Tetrao tetrrix</i>	0.08	0.4	0.71	3.14	Yes
Alatolo et al., 1992*	<i>Tetrao tetrrix</i>	0.14	0.46	0.54	5.90	Yes
Gratson et al., 1991*	<i>Tympanuchus phasianellus</i>	0.11	0.47	0.76	3.54	Yes
Alatolo et al., 1992*	<i>Tetrao tetrrix</i>	0.14	0.49	0.68	6.32	Yes
West et al., 1981	<i>Molothrus ater</i>	0	0.51	0.82	2.05	Yes
Alatolo et al., 1992*	<i>Tetrao tetrrix</i>	0.15	0.52	0.76	6.15	Yes
Alatolo et al., 1992*	<i>Tetrao tetrrix</i>	0.12	0.54	0.82	3.81	Yes
Höglund and Robertson, 1990*	<i>Gallinago media</i>	0.01	0.6	0.9	2.86	Yes
Alatolo et al., 1992*	<i>Tetrao tetrrix</i>	0.15	0.66	0.89	3.81	Yes
Alatolo et al., 1992*	<i>Tetrao tetrrix</i>	0.23	0.69	0.85	9.41	Yes
Hill, 1991	<i>Philomachus pugnax</i>	0	0.71	0.93	2.11	Yes
Hill, 1991*	<i>Philomachus pugnax</i>	0.16	0.71	0.91	3.73	Yes

Continued on next page



Study	Species	$\lambda_{\min}$	$\lambda$	$\lambda_{\max}$	$I_{\delta}$	Lek?
Lanctot et al., 1997*	<i>Tryngites subruficollis</i>	0.21	0.78	0.99	5.40	Yes
Hill, 1991*	<i>Philomachus pugnax</i>	0.2	0.82	0.98	4.04	Yes
Hill, 1991*	<i>Philomachus pugnax</i>	0.18	0.85	0.98	3.59	Yes
Alatolo et al., 1992*	<i>Tetrao tetrax</i>	0.3	0.89	1	6.00	Yes
Alatolo et al., 1992*	<i>Tetrao tetrax</i>	0.33	0.89	1	9.00	Yes
Lanctot et al., 1997*	<i>Tryngites subruficollis</i>	0.26	0.9	1	5.00	Yes
Alatolo et al., 1992*	<i>Tetrao tetrax</i>	0.3	0.9	0.99	6.55	Yes
Robel, 1966	<i>Tympanuchus cupido pinnatus</i>	0.31	0.91	0.99	5.00	Yes
Robel, 1966*	<i>Tympanuchus cupido pinnatus</i>	0.25	0.93	0.99	3.50	Yes
Beehler, 1983	<i>Paradisaea minor</i>	0.37	0.96	0.99	7.38	Yes
West et al., 1981*	<i>Molothrus ater</i>	0.4	0.98	1	9.00	Yes
Trail, 1990	<i>Perissocephalus tricolor</i>	0.42	0.99	1	8.00	Yes

Table 4.2: Mating skew data. Populations are ordered as they are in Figure 4.1, first by  $\lambda$ , then (in case of a tie) by  $\lambda_{\min}$ , then (in case of a continued tie) by  $\lambda_{\max}$ . The  $I_{\delta}$ -value is also given. Populations excluded from analysis of means of  $\lambda$  values are marked with a single asterisk (\*), populations excluded from analysis of means of  $I_{\delta}$  values are marked with a double asterisk (\*\*) (see section 4.2.1 for details).

## Chapter 5

# Colour polymorphism in *Oophaga pumilio*

The variation in colour pattern between populations of the poison-dart frog *Oophaga pumilio* across the Bocas del Toro archipelago in Panama is suggested to be due to female mate choice, as two other Dendrobatid species that share the same ecology do not exhibit this variation, and females of these species do not show mate choice. To create a proof of principle of this hypothesis, we use a quantitative genetic sexual selection model incorporating aposematic colouration and random drift. We find that sexual selection could indeed cause the observed variation via a novel process we call coupled drift. Our model shows that, for certain areas of parameter space, sexual selection forces frog colour to closely follow the evolution of female preference. If genetic drift causes variation between populations for preference, this variation is passed on to colour. If drift strongly affects female preference in *O. pumilio*, but not colour in the nonsexually selecting Dendrobatid species, coupled drift will cause increased between-population phenotypic variation in the former species compared to the latter. However, in other areas of parameter space, coupled drift will result in between-population variation in colour being suppressed by sexual selection, or in little or no effect. We suggest that coupled drift is a novel theoretical process that could link sexual selection with speciation both in *O. pumilio*, and more widely.

### 5.1 Background

The Strawberry poison-dart frog, *Oophaga pumilio* (Grant et al., 2006), is a brightly coloured diurnal amphibian found in the aquatic lowlands of the Caribbean coasts of Nicaragua, Panama, and Costa Rica. Its



Figure 5.1: *Oophaga pumilio* individuals from different populations. Clockwise from bottom left, the individuals pictured are from populations from the mainland, Aguacate, Popa South and Bastimentos Central (thanks to Martine Maan for the pictures).

colouration is monomorphic for most of this range (Savage, 1968), but in the Bocas del Toro archipelago region in northwestern Panama there are approximately 15 different phenotypes (Daly and Myers, 1967; Summers et al., 2003), bearing a wide variety of colour patterns, some of which can be seen in Figure 5.1.

The archipelago is estimated to have been formed less than 10,000 years ago (Anderson and Handley, 2002). Because this is a relatively short time from an evolutionary standpoint, it is likely that at the time of formation, *O. pumilio* was already widespread in the area. Therefore the frogs on the islands in the archipelago are descended from pre-existing populations that were cut off by the rising sea level when the archipelago formed, rather than being descended from small founder populations. It is possible that in 10,000 years genetic drift has contributed to the divergence of colour patterns. However, the archipelago

is also inhabited by two other Dendrobatid species (*Phyllobates lugubris* and *Minyobates sp.*, hereafter referred to as the control species), which live in very similar habitats to *O. pumilio*, but show very little phenotypic variation between populations (Summers et al., 1997). If either drift or natural selection (possibly due to differing predation rates or different types of predator on different islands) were the main cause of polymorphism in *O. pumilio*, they would be also expected to have caused the control species to exhibit phenotypic polymorphism.

It has been suggested (Summers et al., 1997) that the key difference between the control species and *O. pumilio* is the breeding system. *Oophaga pumilio* mothers perform most of the parental care, each one having to carry young tadpoles on her back to various water-filled leaf axils high up in the trees, and then feeding the young with unfertilised eggs for about 7 weeks until they metamorphose into fully grown adults (Weygoldt, 1980, 1987; Brust, 1993). By contrast, *O. pumilio* fathers have only to watch the eggs for a week between initial laying and fertilisation and hatching into tadpoles. The control species have uniparental male care (Weygoldt, 1987; Summers et al., 1997). The high level of investment in young by female *O. pumilio* individuals would be expected to lead to them exhibiting mate choice, and indeed courtship in this species is a protracted affair (Summers et al., 1999). *O. pumilio* frogs can perceive their bright colouration (Siddiqi et al., 2004), and there is evidence that female frogs prefer to associate with conspecifics that bear the same colouration (and possibly spotting pattern; Summers et al., 1999; Reynolds and Fitzpatrick, 2007; though there is less consensus on this; Maan and Cummings, 2008). Experiments have also shown that on occasion, alternative phenotypes can be equally or more acceptable (Maan and Cummings, 2008), possible evidence for variation in preference. Based on these empirical findings, it is hypothesised that the phenotypic polymorphism exhibited in *O. pumilio* is a result of sexual selection, coupled with aposematism and genetic drift (Summers et al., 1999; Reynolds and Fitzpatrick, 2007; Maan and Cummings, 2008). Here we attempt for the first time to theoretically validate this hypothesis by showing that in principle it could occur. To ascertain whether sexual selection could increase phenotypic variation between *O. pumilio* populations, we assume for simplicity that *O. pumilio* and the control species are identical in all other aspects (population sizes, genetic architecture, mutation rates, natural selection scheme, etc.) apart from the presence of sexual selection. Thus, we model two idealised species: “*O. pumilio*,” which has sexual selection, and the “control species,” which does not. The two species are identical in every other regard. We are ignorant as to how similar these aspects are in reality between *O. pumilio* and the control species, but the fact that differences in these other elements could lead to drift affecting each species differently is already well known. By making these other elements identical we isolate the effect of sexual selection on drift and phenotypic variation, and by so doing can discover whether this too could have a role to play.

The colour and spot patterns on *O. pumilio* individuals that form the differences between populations are extremely complex and wide-ranging. meaning that a full model of the differences between phenotypes would be necessarily very complex. Empirical studies use spectrometers to come up with specific reflectance spectra for both dorsal and ventral surfaces of the frogs (Summers et al., 1999, 2003, 2004; Siddiqi et al., 2004; Maan and Cummings, 2008). These reflectance spectra typically have a peak wavelength at which most light is reflected. We use this peak as our focal trait, and refer to it as the frogs' colour. Since evidence exists showing that dorsal colour is the most important factor, both in predator-prey relations (Saporito et al., 2007; Noonan and Comeault, 2009) and in conspecific sexual attraction (Maan and Cummings, 2008), we model this alone, neglecting ventral colour and spotting pattern. In reality, the many different elements of the frog phenotype will coevolve together, in a process likely to be extremely complex, and requiring data on the covariances of the different traits as well as their variances. This complexity could be incorporated into a future model; for now, however, we keep it simple.

We model frog colour as a real number. In this context, the real number line is a simplified representation of the visible light spectrum. Although in reality the visible light portion of the spectrum is bounded, our trait, as a real number, is unbounded. This is also for the sake of simplicity: although we could incorporate the endpoints of the spectrum into the model, they are not required to answer our fundamental question, and so can be safely neglected.

Although the genetics underlying frog colour have not yet been fully explored, there is evidence that the trait is polygenic (Summers et al., 2004), so we model it as a quantitative trait controlled by many genes. We assume this is true for both *O. pumilio* and the control species. We also assume that female preference is also a quantitative trait, in line with standard quantitative genetics models of sexual selection (reviewed by Mead and Arnold, 2004). We then model the evolution of mean colour in allopatric populations of the control species (for which there is no sexual selection), and of *O. pumilio* (for which there is). Our primary interest is the variance in mean colour between populations; we wish to ascertain whether sexual selection increases this. If it does, then the mating system could in principle be the cause of the polymorphism seen in *O. pumilio* across the Bocas del Toro archipelago.

## 5.2 Model

### 5.2.1 *O. pumilio* model

We denote frog colour by  $x$  and female preference by  $y$ . The population mean values of  $x$  and  $y$  are respectively denoted  $\bar{x}$  and  $\bar{y}$ , and the phenotype of an *O. pumilio* individual is denoted  $(x, y)^T$  (where  $^T$

$x$	Colour
$y$	Female preference
$\mathbf{G}$	Variance-covariance matrix
$G_x, G_y$	Additive genetic variances of $x$ and $y$
$B$	Additive genetic covariance of $x$ and $y$
$N$	Population size
$\bar{x}, \bar{y}$	Population means of $x$ and $y$
$\bar{x}_t, \bar{y}_t$	Population means of $x$ and $y$ at generation $t$
$x_0$	Initial value of $\bar{x}$ and $\bar{y}$
$\Delta\bar{x}, \Delta\bar{y}$	Per-generation change in $\bar{x}$ and $\bar{y}$
$\Delta_s\bar{x}, \Delta_s\bar{y}$	Per-generation change in $\bar{x}$ and $\bar{y}$ due to selection
$\Delta_d\bar{x}, \Delta_d\bar{y}$	Per-generation change in $\bar{x}$ and $\bar{y}$ due to drift
$W_m[x \bar{x}, \bar{y}]; W_f[x, y \bar{x}]$	Relative fitness functions of males and females respectively
$a$	Efficacy of male signalling
$b$	Cost of differing from aposematic optimum
$c$	Cost of female mate preference
$(X_i, Y_i)^\top$	Phenotype of sampled individual
$\bar{X}, \bar{Y}$	Sample means for $X_i$ and $Y_i$
$\Delta X_i, \Delta Y_i$	Differences between sampled individual's trait and population means
$\Omega_A[t], \Omega_B[t], \Omega[t]$	Independent stochastic variables with mean 0 and variance 1
$z_t$	$\bar{y}_t - \bar{x}_t$
$D$	$aG_x + cG_y - (a + c)B$
$\eta[t]$	$\frac{1}{\sqrt{N}}(-1, 1)\mathbf{G}^{1/2}(\Omega_A[t], \Omega_B[t])^\top$
$L$	$aG_x - cB$
$\xi[t]$	$\frac{1}{\sqrt{N}}(1, 0)\mathbf{G}^{1/2}(\Omega_A[t], \Omega_B[t])^\top$
$K_u$	$(L/D)(1 - (1 - D)^{u-1})$
$\text{var}[\bar{x}_t]_{O.p}$	Approximation to between-population variance of mean colour in <i>O. pumilio</i>
$\text{var}[\bar{x}_t]_{c.s}$	Between-population variance of mean colour in control species
$R$	Ratio of variances, $\text{var}[\bar{x}_t]_{O.p}/\text{var}[\bar{x}_t]_{c.s}$
$\alpha_i^p, \alpha_i^m$	Additive genetic values for paternal and maternal genome for $x$ in individual $i$
$\beta_i^p, \beta_i^m$	Additive genetic values for paternal and maternal genome for $y$ in individual $i$
$e_{x,i}, e_{y,i}$	Environmental values for traits $x$ and $y$ in individual $i$

Table 5.1: List of symbols

stands for transpose).

The original population is defined as having mean phenotype  $(x_0, x_0)^\top$ , with mean colour and mean preference initially equal. We build on classic quantitative genetics sexual selection models (Lande, 1981; Barton and Turelli, 1991; Iwasa et al., 1991; Pomiankowski et al., 1991; Pomiankowski and Iwasa, 1993; Iwasa and Pomiankowski, 1995; see also section 1.6.2 in chapter 1). The additive genetic variances of  $x$  and  $y$  are respectively denoted by  $G_x$  and  $G_y$ , and the genetic covariance between them by  $B$ . We treat them as constants here, although in fact they are determined by selection, mutation, and random drift (Lande, 1981; Iwasa et al., 1991; Pomiankowski et al., 1991). Since mate choice will inevitably lead to association between colour and preference,  $B > 0$  (in fact an approximation to  $B$  is calculated in the Appendix, section 5.5.1, based on work by Barton and Turelli, 1991). The variance-covariance matrix  $\mathbf{G}$  is then defined in the usual way as

$$\mathbf{G} = \begin{pmatrix} G_x & B \\ B & G_y \end{pmatrix}.$$

We assume weak selection, which means that selection parameters are all small (of the order of magnitude of some small number  $\epsilon$ , denoted  $O(\epsilon)$ ), and large population ( $1/N$  is also  $O(\epsilon)$ ). We also assume that  $G_x$  and  $G_y$  are not small, but  $B$  is ( $G_x$  and  $G_y$  are  $O(1)$ ,  $B$  is  $O(\epsilon)$ ). This last assumption is justified by section 5.5.1 in the Appendix.

Because of these assumptions, the changes of the mean traits in one generation are small, so we can express them as a sum of the changes caused by selection ( $\Delta_s$ ) and the stochastic changes caused by random drift ( $\Delta_d$ ),

$$\begin{pmatrix} \Delta \bar{x} \\ \Delta \bar{y} \end{pmatrix} = \begin{pmatrix} \Delta_s \bar{x} \\ \Delta_s \bar{y} \end{pmatrix} + \begin{pmatrix} \Delta_d \bar{x} \\ \Delta_d \bar{y} \end{pmatrix}. \quad (5.1)$$

Mutation contributes to maintain variances  $G_x$  and  $G_y$ ; we assume that it is unbiased, and so it does not appear in equation (5.1).

### Sexual and natural selection

We model the fitness functions for males and females separately, because preference is only expressed in females (since the genes for it are assumed to be autosomal, they will be carried but not expressed in males). The fitness functions for males and females respectively are

$$W_m[x|\bar{x}, \bar{y}] = \exp\{-a(x - \bar{y})^2 - b(x - \bar{x})^2\}, \quad (5.2)$$

$$W_f[x, y|\bar{x}] = \exp\{-b(x - \bar{x})^2 - c(y - \bar{x})^2\}, \quad (5.3)$$

The first term in the exponent on the right-hand side of the male fitness function (equation (5.2)) indicates the mating success of a male of colour  $x$ . We model female preference as being absolute (Lande, 1981), so the highest average mating success goes to male frogs whose colour  $x$  matches the mean female preference  $\bar{y}$ , and mating success declines as  $x$  moves away from this point. The efficacy of signalling strength and the intensity of female mate preference is indicated by parameter  $a$ .

The second term in the exponent on the right-hand side for males, and the first term on the right-hand side for females, indicates the survival chance due to aposematic colouration. The relation between aposematism, toxicity, and predator avoidance is the subject of much research (Mallet and Joron, 1999; Darst and Cummings, 2006; Darst et al., 2006; Blount et al., 2009). For the purposes of our model, we simply assume that predators learn to avoid frogs as prey by sampling the population. When they catch a frog, they dislike the taste and thus tend to avoid frogs near that phenotype in future. The total effect of this averaged across all predators means a frog is less likely to suffer predation the closer its colour  $x$  is to the mean colour  $\bar{x}$ . The intensity of this effect is represented by parameter  $b$ . Both male and female fitness equations have this same factor because this aposematic effect works equally in both sexes.

The second term in the exponent on the right-hand side for females represents the cost of having an extreme preference. The closer a female's preference  $y$  is to the population mean  $\bar{x}$ , the more easily and quickly she is likely to be able to find a mate. Thus fitness tails off as preference  $y$  deviates from this optimum because females require more time and effort to find a suitable male. The intensity of this effect is indicated by parameter  $c$ . As an aside, note that it is likely that  $c \ll a$ , that is, that an extreme preference is much less costly than an extreme colour.

Finally, note that the parameters included in the fitness function equations (5.2) and (5.3) could be estimated by regression of logarithmic fitness components (mating success, survivorship, etc.) as a linear or quadratic regression of the quantitative traits.

Following the formalism of Iwasa and Pomiankowski (Iwasa et al., 1991; Pomiankowski et al., 1991; Pomiankowski and Iwasa, 1993; Iwasa and Pomiankowski, 1995; see also section 1.6.2 of chapter 1), we derive the dynamics of the quantitative traits to be



$$\begin{aligned}
 \begin{pmatrix} \Delta_s \bar{x} \\ \Delta_s \bar{y} \end{pmatrix} &= \frac{1}{2} \begin{pmatrix} G_x & B \\ B & G_y \end{pmatrix} \begin{pmatrix} \frac{\partial}{\partial x} \{\ln W_m + \ln W_f\} \\ \frac{\partial}{\partial y} \ln W_f \end{pmatrix} \\
 &= \mathbf{G} \begin{pmatrix} -a \\ c \end{pmatrix} (\bar{x} - \bar{y}), \tag{5.4}
 \end{aligned}$$

where the partial derivatives are evaluated at  $x = \bar{x}$ ,  $y = \bar{y}$ . Notice that the parameter  $b$  controlling the intensity of selection due to predation does not appear in the dynamics, because at each generation the optimal colour with regard to avoiding predation is the mean colour, and so there is no net directional selection upon this trait.

### Random Drift

We model random drift as a stochastic shift of quantitative traits caused by the random sampling of a finite number of individuals (Lande, 1976). The sampling occurs every generation, and samples in different generations are assumed to be independent of one another. Because we assume that the traits are centered on the population means we can focus on the shift in the mean traits only. Specifically, in a given generation, if  $(\bar{x}, \bar{y})^\top$  is the population mean phenotype, we consider a distribution with that as the mean and variance-covariance given by  $\mathbf{G}$ .

Let  $(X_i, Y_i)^\top$  ( $i = 1, 2, \dots, N$ ) be  $N$  independent samples from this distribution. These represent the breeding individuals in the population. Note that  $X_i$  and  $Y_i$  are correlated, but  $X_i$  and  $Y_j$  in different individuals ( $i \neq j$ ) are independent. The sample means are then

$$\bar{X} = \frac{1}{N} \sum_{i=1}^N X_i,$$

$$\bar{Y} = \frac{1}{N} \sum_{i=1}^N Y_i.$$

We would like to know the differences  $\Delta_d \bar{x} = \bar{X} - \bar{x}$  and  $\Delta_d \bar{y} = \bar{Y} - \bar{y}$  between the sample means and the current population means  $\bar{x}$  and  $\bar{y}$  because these differences tell us the change in the mean of each trait at the next generation due to drift. Because the sampling is stochastic, the variables  $\Delta_d \bar{x}$  and  $\Delta_d \bar{y}$  are stochastic variables (we assume that one-generation changes are small, which is justified by our weak selection and large population assumptions, though we do not need to assume that one-generation changes are normally distributed). Then  $E[\Delta_d \bar{x}] = E[\Delta_d \bar{y}] = 0$ ,  $\text{var}[\Delta_d \bar{x}] = G_x/N$ ,  $\text{var}[\Delta_d \bar{y}] = G_y/N$ , and  $\text{cov}[\Delta_d \bar{x}, \Delta_d \bar{y}] = B/N$

(see section 5.5.2 in the Appendix for derivations).

Now consider two stochastic variables,  $\Omega_A[t]$  and  $\Omega_B[t]$ , defined to be independent of each other, and to have the property that  $\Omega_A[s]$  and  $\Omega_B[s]$  are independent of  $\Omega_A[t]$  and  $\Omega_B[t]$  for all  $s \neq t$ . Both variables have mean 0 and variance 1. We can then express the distribution at time  $t$  for the one-generation change in  $(\bar{x}_t, \bar{y}_t)^\top$  due to drift as

$$\begin{pmatrix} \Delta_d \bar{x}_t \\ \Delta_d \bar{y}_t \end{pmatrix} = \frac{1}{\sqrt{N}} \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix}, \quad (5.5)$$

where  $\mathbf{G}^{1/2}$  is the square root of  $\mathbf{G}$ , which satisfies  $\mathbf{G}^{1/2} \mathbf{G}^{1/2} = \mathbf{G}$ . Justification of equation (5.5) is in section 5.5.3 of the Appendix.

### Selection and drift together

Substituting equation (5.4) and equation (5.5) into equation (5.1) gives

$$\begin{pmatrix} \Delta \bar{x}_t \\ \Delta \bar{y}_t \end{pmatrix} = \mathbf{G} \begin{pmatrix} -a \\ c \end{pmatrix} (\bar{x}_t - \bar{y}_t) + \frac{1}{\sqrt{N}} \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix}. \quad (5.6)$$

The first part on the right-hand side of equation (5.6) is the change due to selection. It is dependent upon the difference between  $\bar{x}_t$  and  $\bar{y}_t$ , and so to help we define  $z_t = \bar{y}_t - \bar{x}_t = (-1, 1)(\bar{x}_t, \bar{y}_t)^\top$ . We can then express the per-generation change in  $z_t$  and  $\bar{x}_t$  as

$$\Delta z_t = -D z_t + \eta[t], \quad (5.7)$$

$$\Delta \bar{x}_t = L z_t + \xi[t], \quad (5.8)$$

(section 5.5.4 of the Appendix) where

$$D = (-1, 1) \mathbf{G} \begin{pmatrix} -a \\ c \end{pmatrix} = aG_x + cG_y - (a+c)B \approx aG_x + cG_y, \quad (5.9)$$

$$L = (-1, 0) \mathbf{G} \begin{pmatrix} -a \\ c \end{pmatrix} = aG_x - cB \approx aG_x, \quad (5.10)$$

$$\eta[t] = \frac{1}{\sqrt{N}} (-1, 1) \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix}, \quad (5.11)$$

$$\xi[t] = \frac{1}{\sqrt{N}}(-1, 0)\mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix}. \quad (5.12)$$

$D$  and  $L$  are positive constants. From section 5.5.1 in the Appendix (due to weak selection assumptions),  $B$  is small compared to  $G_x$  and  $G_y$ , and so  $D$  and  $L$  can be approximated as in equations (5.9) and (5.10) above. The other values,  $\eta[t]$  and  $\xi[t]$ , are stochastic variables calculated from random drift in the current generation. They have mean zero.

Equation (5.7) describes the dynamics of the difference  $z_t$  between mean female preference and mean body colour. The first term on the right-hand side of equation (5.7) indicates that it tends to return to zero. This is because mean colour evolves towards mean preference due to sexual selection, and mean preference evolves towards mean colour due to cost of female preference. The speed constant for this is  $D$ , given by equation (5.9), which is the sum of the speed of sexual selection  $aG_x$  and the speed of the cost of female preference  $cG_y$ . The second term on the right-hand side of equation (5.7) is the random drift in the two traits. If there is no random genetic drift,  $z_t$  will converge to zero. With the initial condition  $z_0 = 0$ , we can solve the difference equation (5.7) as

$$z_t = \sum_{s=0}^{t-1} (1-D)^{t-s-1} \eta[s], \quad (5.13)$$

meaning the magnitude of the difference between mean female preference and mean colour is a weighted sum of random drift terms from previous generations.

Equation (5.8) is the difference equation for mean colour. The first term on the right-hand side is the effect of sexual selection, with the magnitude proportional to the difference  $z_t$  between mean female preference and mean colour. Because we know by equation (5.13) that  $z_t$  is a weighted sum of past random drift, the first term of equation (5.8) is also a weighted sum of past random drift. The second term on the right-hand side of equation (5.8) is the current random drift. With the initial condition  $\bar{x}_0 = x_0$  we can solve equation (5.8) (see section 5.5.4 of the Appendix for details) to give

$$\bar{x}_t = x_0 + \sum_{u=1}^t \frac{1}{\sqrt{N}} (1 - K_u, K_u) \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t-u] \\ \Omega_B[t-u] \end{pmatrix}, \quad (5.14)$$

where  $K_u$  is defined as

$$K_u = \frac{L}{D} (1 - (1-D)^{u-1}). \quad (5.15)$$

The  $(\Omega_A[t-u], \Omega_B[t-u])^\top$  part of equation (5.14) is a white noise term, so equation (5.14) shows that  $\bar{x}_t$

is a weighted sum of white noise terms representing drift and selection at each generation since  $x_0$ . It is a stochastic variable, with expected value the same as the initial condition

$$E[\bar{x}_t] = x_0 + \sum_{u=1}^t \frac{1}{\sqrt{N}} (1 - K_u, K_u) \mathbf{G}^{1/2} \mathbb{E} \left[ \begin{pmatrix} \Omega_A[t-u] \\ \Omega_B[t-u] \end{pmatrix} \right] = x_0. \quad (5.16)$$

For sufficiently large  $t$  we can approximate the variance of  $\bar{x}_t$  (see section 5.5.5 of the Appendix), denoted  $\text{var}[\bar{x}_t]_{O,p}$  as it represents the variance in colour between populations of *O. pumilio*, as

$$\text{var}[\bar{x}_t]_{O,p} \approx \frac{t}{N} \left[ \left(1 - \frac{L}{D}\right)^2 G_x + \left(\frac{L}{D}\right)^2 G_y \right]. \quad (5.17)$$

### 5.2.2 Control species model

In the control species there is no sexual selection, and so no female preference trait, only frog colour  $x$ . The population mean colour is denoted  $\bar{x}$  and the phenotype of an individual is simply denoted  $x$ . Without sexual selection, male and female fitness functions are identical,

$$W[x|\bar{x}] = \exp \left\{ -b(x - \bar{x})^2 \right\}. \quad (5.18)$$

The only term in the exponent on the right-hand side is the selection due to predation, which is modelled in the same way as that for *O. pumilio* above, with the same coefficient of intensity  $b$ . Since there is no selection acting on mean colour, we have single-trait neutral drift, and

$$\Delta \bar{x}_t = \Delta_d \bar{x}_t = \frac{1}{\sqrt{N}} G_x^{1/2} \Omega[t], \quad (5.19)$$

where  $\Omega[t]$  is a stochastic variable with the same properties as  $\Omega_A[t]$  and  $\Omega_B[t]$  above. In this case, then,

$$\bar{x}_t = x_0 + \sum_{u=1}^t \frac{1}{\sqrt{N}} G_x^{1/2} \Omega[u]. \quad (5.20)$$

As in the *O. pumilio* case,  $\bar{x}_t$  is a sum of white noise terms, but this time there is no effect of selection, only of drift. The expected value  $\bar{x}_t$  is simply the initial condition,  $E[\bar{x}_t] = x_0$ , just as in the sexual selection case above (equation (5.16)). The variance, denoted  $\text{var}[\bar{x}_t]_{c,s}$  as it represents the control species, is different, however:

$$\text{var}[\bar{x}_t]_{c,s} = \frac{1}{N} \sum_{u=1}^t G_x = \frac{t}{N} G_x. \quad (5.21)$$

## 5.3 Results

### 5.3.1 Comparison of variances

Equations (5.9) and (5.10) give

$$\frac{L}{D} \approx \frac{aG_x}{aG_x + cG_y}, \quad (5.22)$$

so that  $0 < L/D < 1$ . We can then get an approximation  $R$  of the ratio of the two variances given by equations (5.17) and (5.21) as

$$\frac{\text{var}[\bar{x}_t]_{O,p}}{\text{var}[\bar{x}_t]_{c.s.}} \approx \frac{G_y}{G_x} \frac{1 + \frac{c^2}{a^2} \frac{G_y}{G_x}}{\left(1 + \frac{cG_y}{aG_x}\right)^2} = R. \quad (5.23)$$

Equation (5.23) gives an approximation  $R$  of the ratio of the between-population variation in colour in *O. pumilio* compared to that in the control species. If  $R > 1$ , the between-population variation is increased by sexual selection, if  $R < 1$  it is decreased, and if  $R = 1$  there is no difference. As shown by equation (5.23),  $R$  depends on the ratio  $G_y/G_x$  of the additive genetic variances and the ratio  $c/a$  of the parameters. In Figure 5.2  $R$  is plotted against  $\ln(G_y/G_x)$  and  $\ln(c/a)$  to investigate this relationship. The regions in which the between-population variance in colour is amplified ( $R > 2$ ), suppressed ( $R < 1/2$ ), and not much affected ( $1/2 < R < 2$ ) by sexual selection are indicated by contour lines. The parameter space has been divided into two regions to simplify analysis. The division is along the line  $aG_x = cG_y$ , the dashed line of slope  $-1$  passing through  $(0, 0)$ , the point in the centre of Figure 5.2. To the right and above this line,  $cG_y \gg aG_x$ , and  $R$  is generally close to 1, so in this region sexual selection does not affect the variance very much.

To the left and below the dashed line,  $aG_x \gg cG_y$ . Here sexual selection greatly influences the between-population variance in colour, and  $R$  is generally either larger or smaller than 1. Whether the variance is amplified or suppressed is determined by the sign of  $\ln(G_y/G_x)$  (itself of course determined by whether  $G_y < G_x$  or  $G_y > G_x$ ). In the region with positive  $\ln(G_y/G_x)$ , the additive genetic variance of female preference is greater than that of body colour, and the between-population variation in colour is greater with sexual selection than without ( $R > 1$ ). In contrast, in the region with negative  $\ln(G_y/G_x)$ , the additive genetic variance of female preference is less than that of body colour, and the between-population variation in colour is reduced by sexual selection ( $R < 1$ ).

### 5.3.2 Coupled Drift

Sexual selection drives the population mean values of colour and preference together, coupling the two traits. Because both traits are likely to take near identical values in a given population, the differences in mean colour between any two allopatric sexually selecting populations will be near identical to the

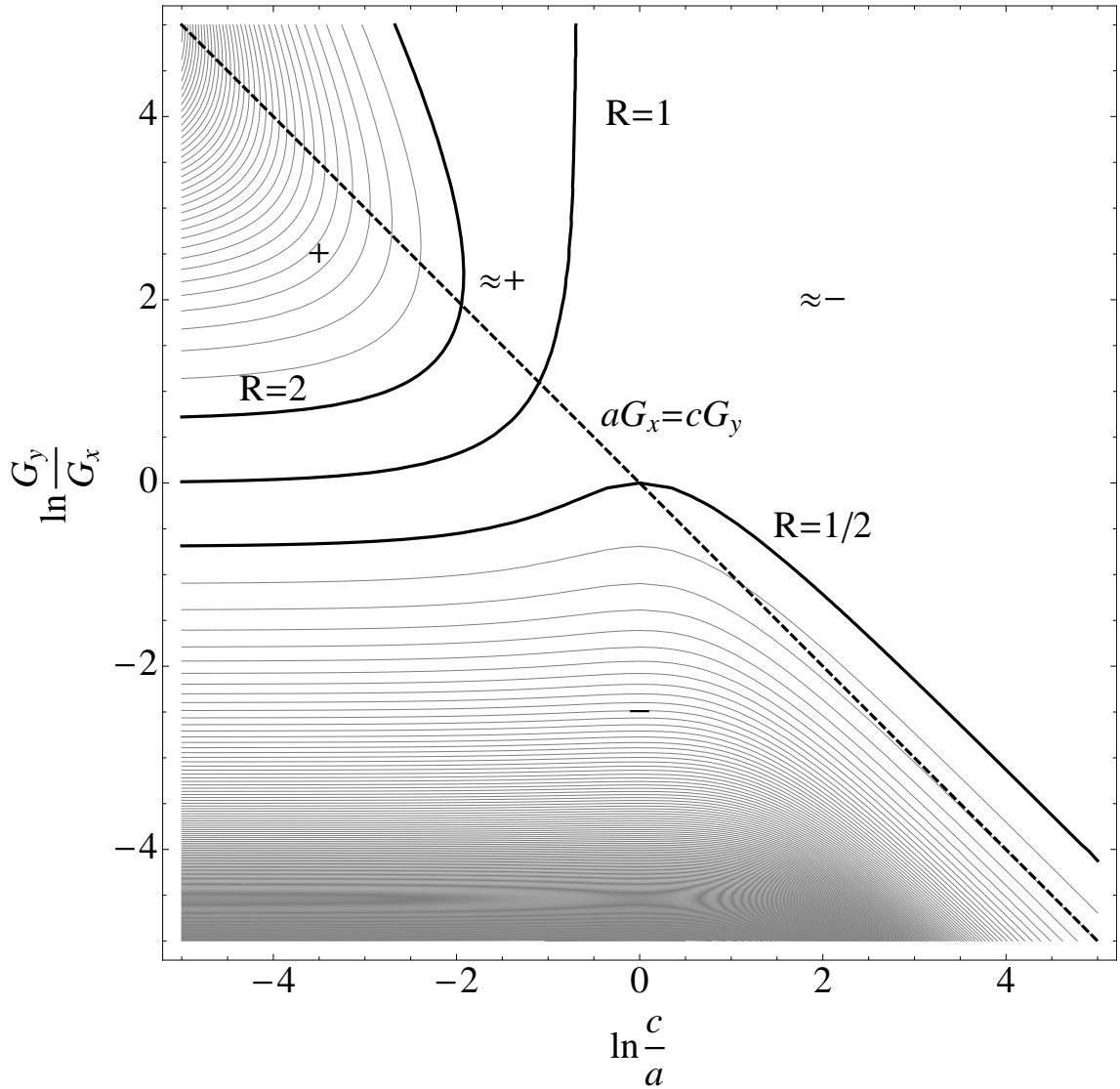


Figure 5.2: The approximation  $R$  to the ratio of the variance in colour between populations of *O. pumilio* against that of the control species, plotted against logs of the key parameter ratios,  $G_y/G_x$  and  $c/a$ . The contours mark out regions of similar  $R$ -values. The thick black contour lines marked show where the two variances are equal ( $R = 1$ ), where the focal variance is twice that of the control ( $R = 2$ ), and where the control variance is twice that of the focal ( $R = 1/2$ ). The other grey contour lines mark  $R = 3, 4, 5, \dots$  and  $1/3, 1/4, 1/5, \dots$  as they move further into the areas marked + or -. The dashed line with gradient  $-1$  across the center marks out  $aG_x = cG_y$ , splitting the plane into two regions, that where  $aG_x < cG_y$  (above and to the right of the dashed line) and sexual selection has less effect on variance ( $R \approx 1$  in general) and that where  $aG_x > cG_y$  and sexual selection has more effect on variance (either  $R \ll 1$  or  $R \gg 1$  in general).

differences in mean preference, and so between-population variance for both traits is approximately equal. Equation (5.17) shows that this resultant variance can be approximated as a linear combination of the single-trait neutral drift values of both traits.

Depending on the parameter values, selection can affect one of the coupled pair of traits more than the other. To see this, recall from above that the difference between the population mean traits is  $z = \bar{y} - \bar{x}$ , and consider equation (5.4), which deals with selection. We can now rewrite it

$$\begin{pmatrix} \Delta_s \bar{x} \\ \Delta_s \bar{y} \end{pmatrix} = -\mathbf{G} \begin{pmatrix} -a \\ c \end{pmatrix} z = \begin{pmatrix} aG_x - cB \\ aB - cG_y \end{pmatrix} z.$$

Because of our weak selection assumptions,  $B$  can be neglected (see section 5.5.1 in Appendix), so the main thing which determines which of  $\bar{x}$  and  $\bar{y}$  is more affected by selection is the relationship between  $aG_x$  and  $cG_y$ .

If  $cG_y \gg aG_x$ , selection forces mean preference to match mean colour. Of the two traits, colour is then the ‘leader’, with preference forced to follow it as it evolves. Since colour is not strongly affected by selection, its evolution is controlled by random drift. This drift in colour is then passed on to preference. Any drift in preference itself is cancelled out by selection forcing it to track colour. With these parameters, equation (5.22) gives  $L/D \approx 0$ , and so by equation (5.17),  $\text{var}[\bar{x}_t]_{O,p} \approx (t/N)G_x = \text{var}[\bar{x}_t]_{C,S}$ , the between-population variance in colour due to random drift (equation (5.21)). Here, then, sexual selection has no effect on between-population variation in colour because mean preference is forced to follow mean colour, and thus has very little effect on its evolution.

If, on the other hand,  $aG_x \gg cG_y$ , selection forces mean colour to match mean preference. In this situation preference is the leader, and colour follows it as it evolves. Since preference is the leader trait, and is not strongly affected by selection, its evolution is largely controlled by random drift, which is then passed on to colour. From equation (5.22) we have  $L/D \approx 1$ , and so  $\text{var}[\bar{x}_t]_{O,p} \approx (t/N)G_y$  by equation (5.17). Therefore under these conditions, sexual selection strongly affects the between-population variation in colour, so that the colour of a given population of frogs simply follows the evolution by drift of the preference trait. Thus we say colour is ‘evolving by coupled drift.’

This region of parameter space can then be further split into three smaller areas. In all three areas, mean colour evolves by coupled drift, but this can lead to amplification or suppression of between-population variance in mean colour, or in it being approximately unchanged compared to the control species. Which of these phenomena occurs depends on the relationship between the variance supplied by coupled drift and that which would be supplied by neutral drift if colour were not coupled.

If  $G_y < G_x$ , coupled drift in colour is less than the neutral drift in colour in the control species. To see why this is, note that for this region of parameter space, preference is evolutionarily quite static compared to colour. Thus when selection couples colour to preference it actually suppresses some of the variation that would occur were colour free to evolve alone. Between-population variance in colour is therefore less where there is sexual selection than where it is absent. If  $G_y \approx G_x$ , then coupled drift in colour is more or less equal to neutral drift, since for this region of parameter space preference is just as evolutionarily static as uncoupled colour. Finally, if  $G_y > G_x$  then preference is less evolutionarily static than colour. Thus by being coupled to mean preference, mean colour varies more than it would from its own neutral drift, so sexual selection amplifies between-population variance in colour.

For regions in which  $aG_x$  and  $cG_y$  are of a similar order, selection pushes  $\bar{x}$  and  $\bar{y}$  together approximately equally, so neither colour nor preference is the leader trait. Under these circumstances, sexual selection could suppress or amplify between-population variance in mean colour (or have no effect), depending on the relative values of the variances  $G_x$  and  $G_y$  (and possibly the covariance  $B$ , see section 5.5.5 in the Appendix).

## 5.4 Discussion

We have investigated whether the large phenotypic variation seen between populations of *O. pumilio* in the Bocas del Toro archipelago compared to two sympatric nonsexually selecting Dendrobatid species could be caused by sexual selection (Summers et al., 1997). We have used stochastic difference equations in modelling the interaction between selection and drift, which we believe to be a novel approach in this field. It is a powerful method because it demonstrates clearly the relationship between the two evolutionary forces.

This investigation has also highlighted the potential importance of an evolutionary process we call ‘coupled drift.’ When selection pushes two traits into having matching phenotypic values, the between-population variation in the mean value of both traits will also be equal. Selection can act asymmetrically on the two traits, meaning one trait (the follower) is forced to track the evolution of the other (the leader). The leader trait then evolves mainly by drift, because there is little selective pressure on it. Thus drift in the leader trait mainly determines the evolution of the follower trait because of the coupling caused by selection. We call this evolution by coupled drift.

In the case of *O. pumilio*, our two traits are colour and female preference. For coupled drift via sexual selection to be the cause of the polymorphism seen in *O. pumilio* across the Bocas del Toro archipelago, we require first that female preference is the leader trait, and colour the follower. Since the cost to females of



having a preference far from the mean colour is likely to be very small when compared to the cost to a male of having a colour far from the mean preference (i.e.  $c \ll a$  in our model) this seems likely (because under these circumstances it is more likely that  $aG_x \gg cG_y$ ). We then require that additive genetic variance in female preference is larger than additive genetic variance in colour (i.e.,  $G_y > G_x$ , although not by so much that  $aG_x < cG_y$ ). Under these circumstances, within each population, mean colour follows mean female preference, which evolves by drift. Thus, if preference is more strongly affected by drift than colour would be in the absence of sexual selection, coupled drift causes more variation in colour than neutral drift, and so sexually selecting species vary more than nonsexually selecting ones, leading to polymorphism, potentially as seen in *O. pumilio*. This basic process was predicted by Lande (1981), when he pointed out that “the interaction of random genetic drift with natural and sexual selection” can cause rapid evolution in sexually selecting species due to the effect of “random genetic drift in female mating preferences.”

Nichols and Butlin (1989) suggested that in finite populations the genetic correlations between alleles affecting a sexual character and alleles affecting preference would break down and be overwhelmed by drift. As a result of coupled drift, however, even in the absence of any correlation assortative mating can still have a strong effect ( $B$  is  $O(\epsilon)$  in our model where  $G_x$  and  $G_y$  are  $O(1)$ , as seen in section 5.5.1 in the Appendix, and for much of the parameter space  $B = 0$  would give qualitatively identical conclusions). It is notable, however, that in our model there is no fixed optimal trait value; rather the optimal colour is the mean colour at each generation. If the evolution of the leader trait is dependent on natural selective forces as well as random drift then these effects too will be felt by the follower trait. Of course it is already well known that differences in natural selective forces between populations can cause speciation (Coyne and Orr, 2004).

We have modelled *O. pumilio* and the control species as being identical except for the presence of sexual selection in the former. This, of course, is an extreme simplification. It is more likely that there are many differences between these two groups, even though they inhabit the same environments. For example, both generation times and population sizes could differ between species, and affect genetic drift. In particular, it is generally assumed that sexual selection decreases effective population size due to the fact that a subset of males monopolise the available matings. This will increase the effect of genetic drift on *O. pumilio* relative to the control species. Estimates of effective population size and generation time for each of the species should therefore be obtained to confirm that it is not this alone that is responsible for the creation of numerous phenotypes of *O. pumilio*. On a related note, additive genetic variance  $G_x$  of colour could potentially be different in *O. pumilio* and the control species, which would affect drift. Note, though, that there is evidence that the control species have the evolutionary potential to vary in colour (Summers et al., 1997). Finally, because males provide the majority of the care in the control species, they could

plausibly have evolved male mate choice and thus might also be subject to sexual selection. However, the fact that there may be other factors additionally involved in causing the large variation in *O. pumilio* does not invalidate our conclusions. We sought only to prove in principle that sexual selection could be a factor, and to uncover the mechanism by which this could occur.

In line with many quantitative genetic sexual selection models (reviewed by Mead and Arnold (2004); see also section 1.6.2 in the Introduction), our results suggest selection drives the system to a line of equilibria. Because along this line mean preference and mean colour take the same values, our model predicts assortative mating as an evolutionary outcome. Several experiments have suggested such assortative preferences do indeed exist in *O. pumilio* females (Summers et al., 1999; Reynolds and Fitzpatrick, 2007; Maan and Cummings, 2008). Interestingly, Maan and Cummings (2008) also found that some females preferred males bearing a colour different from their own. This could be interpreted as evidence for large within-population variance in female preference compared to colour. There is also evidence from studies of other amphibians that when preference functions and male trait values are unimodal in nature, the curvature of the former is weaker than that of the latter (Gerhardt and Huber, 2002; Uyeda et al., 2009). This is not conclusive, however, and we suggest that before coupled drift can be judged to be the cause of the numerous phenotypes of *O. pumilio*, more work needs to be done to establish the relative sizes of the additive genetic variances in colour and female preference in this species.

We assume that brightness of frog colouration remains constant as populations evolve, and that no one colour is inherently better for aposematic purposes than any other. In actual fact such equality of colour seems intuitively unlikely to exist in the natural world because both the inherent sensory biases of predators and the background colours of the environments in which the frogs live could lead to certain colours being more memorable or noticeable than others. In our theoretical framework, change per generation is modelled as being the sum of change due to selection and (stochastic) change due to drift. To incorporate inherent fitness benefits of certain colours we would need to alter the fitness function so that certain values of colour  $x$  provide fitness benefits regardless of the position of the mean colour  $\bar{x}$ . The resulting nonlinear stochastic difference equations could then be simulated or, in some cases, analysed. However, because there is a lack of any data on the subject in *O. pumilio*, and for the purposes of simplicity, we have left such considerations out of the model. The inherent superiority of some colours for aposematic purposes is unlikely in practice to be a very strong evolutionary force acting on *O. pumilio*, given the wide variety of colouration seen in phenotypic variants of the species in reality. Other theoretical work (Broom et al., 2006; Ruxton et al., 2007) suggests that as long as the frogs are sufficiently toxic to cause would-be predators to remember to avoid them, any colour pattern can act as an aposematic signal, as long as it is above a certain threshold of

conspicuousness (i.e., so long as predators can easily spot and remember the frogs). The population could thus conceivably evolve along a ridge of higher fitness in the phenotypic landscape (Gavrilets, 1997), with colour changing, but conspicuousness remaining above a threshold value.

Aposematic selection is an important aspect of our model, because it means that the optimal phenotype for colour is the population mean. It is thus a form of frequency-dependent selection. Because our model is based on that of Iwasa, Pomiankowski, and Nee (Iwasa et al., 1991; Pomiankowski et al., 1991), it is able to handle this type of selection. If it is to be unconstrained, evolution by coupled drift requires that the leader trait evolves by drift alone, and the follower trait is under selection only to track the leader. Frequency-dependent selection lends itself to this situation more easily. Whether other selective regimes could lead to this situation is worth further investigation.

Aposematic selection also justifies our use of a one-dimensional colour line to model the evolution of frog colour. Because we do not know the underlying genetic architecture of the colouration, we cannot rule out the possibility that a single mutation could cause a large jump in colour, for example, transforming a red frog into a blue frog. However, aposematic selection would rapidly eliminate such extreme mutants from the population, and thus only mutations of small effect will be retained. Therefore the mean colour value  $\bar{x}$  will likely change only very slightly at each generation, justifying our use of a one-dimensional line as the topology over which evolution of colour occurs.

Other, nonfrequency-dependent selective regimes could be modelled using alternative approaches, for example that of Lande (1981). The relative unimportance of the additive genetic covariance  $B$  differentiates our model from these more traditional Fisherian runaway approaches (reviewed by Mead and Arnold, 2004). In these models, it is this covariance that leads to runaway evolution, potentially checked by natural selection. In contrast, in our model the evolution of colour (the sexually selected trait) is determined (depending upon the underlying genetic and selective parameters) by random drift in female preference, and the covariance between this and colouration is not important (section 5.5.1 in the Appendix). Therefore this is not evolution by Fisherian runaway, but by coupled drift.

The concept of variation due to coupled drift could have wider ramifications. In the wild, salt-water barriers largely separate the populations of different *O. pumilio* phenotypes. There is also evidence that female *O. pumilio* individuals assortatively mate (Summers et al., 1999; Reynolds and Fitzpatrick, 2007; Maan and Cummings, 2008) when given a choice between males of their own phenotype and those from different islands. Thus, even where the different phenotypes can mix (for example due to historical translocation by humans; Rudh et al., 2007) there may be a prezygotic barrier to gene flow between them. In addition, in cases where two phenotypes are sufficiently different in colour, there may also be postzygotic

selection against gene flow. For example, the offspring of a red and green frog may bear a colour unlikely to afford it protection from predators due to aposematism (Summers et al., 2004). Thus, frog colour may be a “magic trait,” subject to both assortative mating and disruptive selection (Gavrilets, 2004), and, via coupled drift, could lead to speciation of *O. pumilio* phenotypes in the future. Coyne and Orr (2004) suggested that reproductive isolation would evolve very slowly by drift, but simulations (Nei et al., 1983; Wu, 1985; Uyeda et al., 2009) have suggested that under some circumstances its evolution can be hastened by sexual selection. To fully investigate this possibility, we would need to incorporate a model of reproductive isolation into our model (Mead and Arnold, 2004; Uyeda et al., 2009).

There is the intriguing possibility that more than two traits could be coupled together by selection. This could occur if several follower traits are constrained by selection to track the evolution of a single leader trait, which itself evolves by drift. Alternatively, it could potentially involve much more complicated leader-follower relationships, with selection and drift interacting to govern the evolution of a suite of traits. Either way, multitrait coupled drift could speed up speciation by causing allopatric populations to diverge in several traits simultaneously.

There is also a possibility that coupled drift could affect traits between species, because there is no need for follower traits to be expressed in the same species as leader traits. For this to occur, the leader trait in one species would need to evolve by neutral drift, while selection forces the follower trait in another species to match it. A potential example of this could be in cases of Batesian mimicry, with the colour of a toxic aposematic species changing by drift and the colour of a Batesian mimic species being forced to evolve with it. How neutral the evolution of colour pattern in the toxic species is likely to be is perhaps questionable, however. There are examples of mimicry rings in *Heliconius* butterflies, but whether these count as examples of between-species coupled drift is open to debate, especially because many changes in *Heliconius* colour patterns would seem to be major-gene switches rather than gradual change by drift (Mallet and Joron, 1999).

We conclude that coupled drift could be important in explaining the variation in *O. pumilio* in the Bocas del Toro archipelago. Furthermore, it could have a bearing on the ongoing debate about random drift, sexual selection, and speciation (Coyne and Orr, 2004; Gavrilets, 2004). We suggest that it could cause or speed up speciation in *O. pumilio* and in other species, by causing two (or perhaps more) traits to diverge between populations via drift, potentially much faster than the same traits could were they uncoupled. It is our belief that further theoretical and empirical work should be done to ascertain how general and powerful an evolutionary force it is, and to model other areas in which it could have an effect (see section 7.3.4 in the Conclusion).

## 5.5 Appendix

### 5.5.1 Approximation of $B$

We follow the argument laid out by Barton and Turelli (1991) and by Pomiankowski and Iwasa (1993). The contribution to the next generation of a mating between a male with colour  $x_m$  and a female with colour  $x_f$  and preference  $y$  is

$$W[x_m, x_f, y] = \frac{\exp\{-a(x_m - y)^2 - b(x_m - \bar{x})^2\} \exp\{-b(x_f - \bar{x})^2 - c(y - x_m)^2\}}{\langle \exp\{-a(x_m - y)^2 - b(x_m - \bar{x})^2\} \rangle_{x_m} \langle \exp\{-b(x_f - \bar{x})^2 - c(y - x_m)^2\} \rangle_{x_f, y}}$$

where  $\langle \rangle_{x_m}$  means average with respect to  $x_m$  but still a function of  $x_f$  and  $y$ , and  $\langle \rangle_{x_f, y}$  means average with respect to  $x_f$  and  $y$  but still a function of  $x_m$ . Taylor expanding this expression around  $\bar{x}$  (for both  $x_m$  and  $x_f$ ) and  $\bar{y}$  and dividing by average fitness gives

$$\begin{aligned} \frac{W[x_m, x_f, y]}{\bar{W}[x_m, x_f, y]} &= 1 + 2(a + c)(\bar{y} - \bar{x})(x_m - \bar{x}) - 2(a + c)(\bar{y} - \bar{x})(y - \bar{y}) \\ &\quad - (a + b + c) \left[ (x_m - \bar{x})^2 - G_x \right] - b \left[ (x_f - \bar{x})^2 - G_x \right] \\ &\quad - (a + c) \left[ (y - \bar{y})^2 - G_y \right] + 2(a + c) \left[ (x_m - \bar{x})(y - \bar{y}) - B \right] \\ &\quad + (\text{higher order terms}) \end{aligned}$$

The second term on the right hand side indicates the intensity of direct selection on  $x_m$  due to female mate preference, the third term the intensity of direct selection on female mate preference due to predation rate, and the fourth, fifth and sixth terms stabilising selection with respect to male and female colour and female preference. The seventh term represents the association between colour and preference.

Following Barton and Turelli (1991) and Pomiankowski and Iwasa (1993), we can get an equation for the per-generation change in genetic covariance between  $x$  and  $y$ . We assume that  $x$  is controlled by the same alleles in both males and females, that both traits are polygenic and the breeding values of the alleles have a multivariate normal distribution. We further assume that there are no genes that affect both  $x$  and  $y$ . Our equation for the per-generation change in genetic covariance  $B$  is then

$$\Delta B = -\frac{1}{2}B + \frac{-a - 2b - c}{2}BG_x + \frac{a + c}{2}(G_x G_y + B^2)$$

Using this expression and a weak selection argument we can get an approximation to  $B$  as

$$B = (a + c)G_x G_y.$$

Since  $a$  is  $O(\epsilon)$ , so is  $B$ .

### 5.5.2 Derivations of $\text{var} [\Delta_d \bar{x}]$ , $\text{var} [\Delta_d \bar{y}]$ , $\text{cov} [\Delta_d \bar{x}, \Delta_d \bar{y}]$

We consider the traits to be controlled by a diploid genome. When the environmental variance is independent of the genotypic value, we have

$$\begin{aligned} X_i &= \bar{x} + \alpha_i^p + \alpha_i^m + e_{x,i}; \\ Y_i &= \bar{y} + \beta_i^p + \beta_i^m + e_{y,i}, \end{aligned}$$

where  $\alpha_i^p$  and  $\alpha_i^m$  are the additive genetic contributions to colour from the paternal and maternal genomes respectively,  $e_{x,i}$  is the value of the environmental contribution to colour,  $\beta_i^p$  and  $\beta_i^m$  are the additive genetic contributions to preference from the paternal and maternal genomes respectively, and  $e_{y,i}$  is the value of the environmental contribution to preference. For the sake of simplicity we focus on the case in which all genetic values are additive and environmental value is uncorrelated with genetic value. Mean values of  $\alpha_i^p$ ,  $\alpha_i^m$ ,  $e_{x,i}$ ,  $\beta_i^p$ ,  $\beta_i^m$  and  $e_{y,i}$  are zero. The additive genetic variances and covariance are then:

$$\begin{aligned} G_x &= \text{E} \left[ (\alpha_i^p + \alpha_i^m)^2 \right], \\ G_y &= \text{E} \left[ (\beta_i^p + \beta_i^m)^2 \right], \\ B &= \text{E} \left[ (\alpha_i^p + \alpha_i^m) (\beta_i^p + \beta_i^m) \right], \end{aligned}$$

Now we calculate the one-generation change in the two quantitative traits by random drift:

$$\begin{aligned} \Delta_d \bar{x} &= \frac{1}{N} \sum_{i=1}^N X_i - \bar{x} = \frac{1}{N} \sum_{i=1}^N (\alpha_i^p + \alpha_i^m + e_{x,i}); \\ \Delta_d \bar{y} &= \frac{1}{N} \sum_{i=1}^N Y_i - \bar{y} = \frac{1}{N} \sum_{i=1}^N (\beta_i^p + \beta_i^m + e_{y,i}). \end{aligned}$$

Hence we have  $\text{E} [\Delta_d \bar{x}] = \text{E} [\Delta_d \bar{y}] = 0$ . The variance is

$$\text{var} [\Delta_d \bar{x}] = \text{E} \left[ (\Delta_d \bar{x})^2 \right] = \frac{1}{N^2} \sum_{i=1}^N \sum_{j=1}^N \text{E} \left[ (\alpha_i^p + \alpha_i^m + e_{x,i}) (\alpha_j^p + \alpha_j^m + e_{x,j}) \right]$$

Noting that different individuals are sampled independently, the factors with different suffix  $i \neq j$  are uncorrelated. Hence

$$\text{var} [\Delta_d \bar{x}] = \frac{1}{N^2} \sum_{i=1}^N \text{E} \left[ (\alpha_i^p + \alpha_i^m + e_{x,i})^2 \right] = \frac{1}{N^2} N G_x = \frac{G_x}{N},$$

since environmental and genetic values are uncorrelated. In a similar manner we have  $\text{var} [\Delta_d \bar{y}] = G_y/N$ , and also

$$\begin{aligned} \text{cov} [\Delta_d \bar{x}, \Delta_d \bar{y}] &= \text{E} [(\Delta_d \bar{x})(\Delta_d \bar{y})] \\ &= \frac{1}{N^2} \sum_{i=1}^N \sum_{j=1}^N \text{E} [(\alpha_i^p + \alpha_i^m + e_{x,i})(\beta_j^p + \beta_j^m + e_{y,j})] \\ &= \frac{1}{N^2} \sum_{i=1}^N \text{E} [(\alpha_i^p + \alpha_i^m + e_{x,i})(\beta_i^p + \beta_i^m + e_{y,i})] \\ &= \frac{B}{N}. \end{aligned}$$

### 5.5.3 Use of $\Omega_A[t]$ and $\Omega_B[t]$

Firstly, note that since  $\mathbf{G}$  is a positive and symmetric matrix, there exists a matrix  $\mathbf{G}^{1/2}$  which is also positive and symmetric (though note there will in general be other matrices that square to give  $\mathbf{G}$  that are not positive and symmetric).  $\mathbf{G}^{1/2}$  also has an inverse. Now, consider the following vector:

$$\begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix} = \sqrt{N} (\mathbf{G}^{1/2})^{-1} \begin{pmatrix} \Delta_d \bar{x}_t \\ \Delta_d \bar{y}_t \end{pmatrix}.$$

This has mean

$$\text{E} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix} = \sqrt{N} (\mathbf{G}^{1/2})^{-1} \text{E} \begin{pmatrix} \Delta_d \bar{x}_t \\ \Delta_d \bar{y}_t \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix},$$

and variance-covariance matrix

$$\begin{aligned} \text{E} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix} (\Omega_A[t], \Omega_B[t]) &= \sqrt{N} (\mathbf{G}^{1/2})^{-1} \text{E} \begin{pmatrix} \Delta_d \bar{x}_t \\ \Delta_d \bar{y}_t \end{pmatrix} (\Delta_d \bar{x}_t, \Delta_d \bar{y}_t) (\mathbf{G}^{1/2})^{-1} \sqrt{N} \\ &= N (\mathbf{G}^{1/2})^{-1} \frac{\mathbf{G}}{N} (\mathbf{G}^{1/2})^{-1} \\ &= \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}. \end{aligned}$$

Thus equation (5.5) is justified.

#### 5.5.4 Recursion solution for $\bar{x}_t$

We start by multiplying equation (5.6) by  $(-1, 1)$  to give  $\Delta z_t = \Delta \bar{y}_t - \Delta \bar{x}_t$ , the one-generation change in the difference between  $\bar{x}$  and  $\bar{y}$  at time  $t$ ,

$$\Delta z_t = -(-1, 1)\mathbf{G} \begin{pmatrix} -a \\ c \end{pmatrix} z_t + \frac{1}{\sqrt{N}}(-1, 1)\mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix}.$$

This can be rewritten as equation (5.7) using the definitions of  $D$  and  $\eta[t]$  given by equations (5.9) and (5.11) respectively. Therefore  $z_{t+1} = (1 - D)z_t + \eta[t]$ . From the weak selection assumption, we have  $D > 0$ , and the initial condition is  $z_0 = 0$ . Hence we can solve the recursion to give equation (5.13).

By left-multiplying equation (5.6) by the row vector  $(1, 0)$ , and using equations (5.10) and (5.12), we get the difference equation for  $\bar{x}_t$  given by equation (5.8). Using equation (5.13), equation (5.8) becomes

$$\Delta \bar{x}_t = Lz_t + \xi[t] = L \sum_{s=0}^{t-1} (1 - D)^{t-s-1} \eta[s] + \xi[t].$$

Note that  $L > 0$ . With the initial condition  $\bar{x}_0 = x_0$ , we have

$$\begin{aligned} \bar{x}_t - x_0 &= \sum_{t'=1}^{t-1} L \sum_{s=0}^{t'-1} (1 - D)^{t'-s-1} \eta[s] + \sum_{t'=1}^{t-1} \xi[t'] \\ &= \sum_{s=0}^{t-2} L \sum_{t'=s+1}^{t-1} (1 - D)^{t'-s-1} \eta[s] + \sum_{s=1}^{t-1} \xi[s], \end{aligned}$$

where we changed the order of the two sums (with respect to  $s$  and  $t'$ ) in the first term on the right-hand side. This is rewritten as

$$\begin{aligned} \bar{x}_t - x_0 &= \sum_{s=0}^{t-2} \left\{ L \left( \sum_{t'=s+1}^{t-1} (1 - D)^{t'-s-1} \right) \eta[s] + \xi[s] \right\} + \xi[t-1] \\ &= \sum_{s=0}^{t-2} \left\{ L \frac{1 - (1 - D)^{t-s-1}}{D} \eta[s] + \xi[s] \right\} + \xi[t-1] \\ &= \sum_{s=0}^{t-2} \left\{ \frac{1}{\sqrt{N}} \left[ \frac{L}{D} (1 - (1 - D)^{t-s-1}) (-1, 1) + (1, 0) \right] \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t] \\ \Omega_B[t] \end{pmatrix} \right\} + \xi[t-1], \end{aligned}$$

using the definitions of  $\eta[t]$  and  $\xi[t]$  given by equations (5.11) and (5.12) respectively. We can then use the



definition of  $K_u$  given in equation (5.15) to give us equation (5.14).

### 5.5.5 Calculation of $\text{var}[\bar{x}_t]$

The variance of  $\bar{x}_t$  is

$$\begin{aligned} \text{var}[\bar{x}_t] &= \text{E} \left[ \left( \sum_{u=1}^t \frac{1}{\sqrt{N}} (1 - K_u, K_u) \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t-u] \\ \Omega_B[t-u] \end{pmatrix} \right)^2 \right] \\ &= \text{E} \left[ \left( \sum_{u=1}^t \frac{1}{\sqrt{N}} (1 - K_u, K_u) \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t-u] \\ \Omega_B[t-u] \end{pmatrix} \right) \left( \sum_{u'=1}^t \frac{1}{\sqrt{N}} (1 - K_{u'}, K_{u'}) \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t-u'] \\ \Omega_B[t-u'] \end{pmatrix} \right) \right] \\ &= \text{E} \left[ \frac{1}{N} \left( \sum_{u=1}^t (1 - K_u, K_u) \mathbf{G}^{1/2} \begin{pmatrix} \Omega_A[t-u] \\ \Omega_B[t-u] \end{pmatrix} \right) \begin{pmatrix} \Omega_A[t-u] & \Omega_B[t-u] \end{pmatrix} \mathbf{G}^{1/2} \begin{pmatrix} 1 - K_u \\ K_u \end{pmatrix} \right) \right] \end{aligned}$$

since the expectation of the product of two stochastic factors at different times  $u \neq u'$  is zero. Because the covariance of the two stochastic factors is also zero, and their variance is one, this can then be rewritten as

$$\begin{aligned} \text{var}[\bar{x}_t] &= \frac{1}{N} \left( \sum_{u=1}^t (1 - K_u, K_u) \mathbf{G}^{1/2} \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} \mathbf{G}^{1/2} \begin{pmatrix} 1 - K_u \\ K_u \end{pmatrix} \right) \\ &= \frac{1}{N} \sum_{u=1}^t (1 - K_u, K_u) \mathbf{G} \begin{pmatrix} 1 - K_u \\ K_u \end{pmatrix} \\ &= \frac{1}{N} \sum_{u=1}^t \left[ (1 - K_u)^2 G_x + 2K_u(1 - K_u)B + K_u^2 G_y \right]. \end{aligned} \quad (5.24)$$

The complex looking formula of equation (5.24) comes from the fact that the initial variance of  $z$  is zero, and will take some time to increase to a steady state. After a sufficiently long time we can regard  $K_u \approx L/D$  (see equation (5.15)), and equation (5.24) becomes approximately

$$\text{var}[\bar{x}_t] = \frac{t}{N} \left[ \left(1 - \frac{L}{D}\right)^2 G_x + 2\frac{L}{D} \left(1 - \frac{L}{D}\right) B + \left(\frac{L}{D}\right)^2 G_y \right]. \quad (5.25)$$

Because of our weak selection assumptions, we can neglect  $B$  to result in equation (5.17).

## Chapter 6

# Differential ejaculate expenditure

Sperm competition theory has focussed largely on the evolution of ejaculate expenditure strategies across different species or populations or across discrete mating roles on which sperm competition operates in different ways. Few studies have considered how male ejaculate expenditure is influenced by continuous change in male phenotype within a population. Here we model how optimal ejaculate expenditure responds to two sources of continuous variation: (1) the quantity of resources allocated by a male to mating within a breeding season, and (2) the resource cost of obtaining a mate. We find that variation in the amount of resources available for mating does not alone produce selection for differing ejaculate investment strategies. However, when there is variation in the cost of obtaining a mate, males with a lower cost will be selected to invest fewer sperm per mating than males whose cost is higher. Any parameter decreasing this cost will also select for decreased ejaculate investment per mating. These results provide a novel insight into the evolution of male ejaculate expenditure strategies, revealing that individual constraints on the ability to secure matings can lead to variation in ejaculate expenditure even when the risk of sperm competition is the same for all males.

### 6.1 Background

In many species, females mate multiply and the ejaculates of different males compete to fertilise a set of eggs in a process called sperm competition (Parker, 1970). A key determinant of the outcome of sperm competition is the relative number of sperm delivered by competing ejaculates (Martin et al., 1974; Wedell et al., 2002; Gage and Morrow, 2003). This generates post-insemination sexual selection on male investment in the number of sperm in an ejaculate (ejaculate expenditure). Sperm competition theory has traditionally

modelled strategies of ejaculate expenditure in a species or population as the response to the average degree of sperm competition faced by an ejaculate (Parker, 1990a, 1998; Parker et al., 1996; Wedell et al., 2002). This view has been challenged by recent studies (Parker and Ball, 2005; Williams et al., 2005) that propose a more dynamic alternative in which optimal ejaculate expenditure and the degree of sperm competition coevolve within a population, each having an effect on the other. This interdependence arises because as the optimal ejaculate expenditure increases, the expected number of matings per male decreases. Since each mating takes place between a single male and a single female, the expected number of matings per female will also decrease, and with it the degree of sperm competition that an ejaculate is expected to face. This feedback drives the coevolutionary process.

Models of sperm competition have generally assumed that differences between males within a species or population are negligible compared to those between species or populations. Where variation within species or populations has been investigated, the male population has been split into discrete reproductive strategies or roles. These discrete categories can be associated with different levels of sperm competition, possess different information on the level of sperm competition associated with a population, and/or face 'loaded' raffles affected by their role in a given population (Ball and Parker, 2000; Mesterton-Gibbons, 1999a,b; Parker, 1990b,a). These studies predict that each male reproductive strategy, or role, will have a corresponding discrete optimal ejaculate expenditure strategy. However, much phenotypic variation among males is likely to be continuous, for example because of variable access to resources, female mate choice, environmental or genetic factors, and differences in age or phenotype. Despite its biological generality, the potential effect of continuous, within-population variation on ejaculate expenditure has not been explicitly modelled. In this chapter, we provide a starting point for an analysis of such effects.

We adapt a non-classical theoretical model for ejaculate expenditure (Williams et al., 2005) to include within-population variation, and we consider two candidate continuously varying traits. First, the quantity of resources allocated to reproduction ( $R$ ) may vary continuously across a population of males. Some males may be able to obtain more resources overall and thus have proportionately more for reproduction. Alternatively, males may allocate resources differently in each breeding season throughout their lifetime (e.g. yearlings vs. mature adults; Pianka and Parker, 1975). In either case, the quantity of resources available for reproductive effort in a given breeding season will differ from male to male, and we expect this to vary in a continuous manner.

Second, males may differ in the resource cost ( $c$ ), that is, the time and energy they spend seeking out and courting a female in order to obtain a mating. One mechanism that could result in such variation is female mate choice. For example, if females display a preference toward certain male phenotypic traits, a

male will pay progressively less to mate as his phenotype approaches that preferred by females (Andersson, 1994; Andersson and Iwasa, 1996). Similarly, males may vary continuously in their competitive ability to gain access to females. In this case, a male with a higher  $c$  value must work harder to secure a mating than one with a lower  $c$  value. Again, we expect that this cost will vary continuously across a population. Note that the amount of resources a male puts into the growth and maintenance of a secondary sexual ornament or a position in a social hierarchy in order to attract females is a cost distinct from that being modelled here. For the purposes of this chapter, the cost a male pays to attract a female is simply a measure of the mean amount of effort he has to expend to attract each mate in a given breeding season. This may be related to a male's investment in attractiveness or competitive ability, but the cost of this initial investment is assumed to have been paid before the breeding season begins. Thus, all males arrive in our model at the start of the breeding season with their  $c$  and  $R$  values already in place. We do, however, allow for the possibility that these parameters are not independent.

## 6.2 Model

Let  $R$  be the quantity of resources allocated by a male to reproduction in a given mating season. Suppose that it costs a male  $c$  units of resource to obtain a mate and that for every mating a further  $s$  units of resource are used for the production of sperm. For simplicity, we assume that a male's ejaculate expenditure is the same for every mating in a breeding season (the validity of this assumption is considered in section 6.4 below). The total cost per mating is therefore  $c + s$ . A male playing a strategy  $s$  then has an expected number of matings  $n[s]$ , where

$$n[s] = \frac{R}{c + s}.$$

In order to calculate the relative fitness  $W[s, \hat{s}]$  of a mutant strategy  $s$  against a population of males all playing strategy  $\hat{s}$ , we calculate its expected reproductive success  $v[s, \hat{s}]$  per mating. Then the expected reproductive success is

$$W[s, \hat{s}] = n[s]v[s, \hat{s}], \tag{6.1}$$

the product of the expected number of matings and the expected success per mating (Parker, 1998; see also Mesterton-Gibbons, 1999a,b; Ball and Parker, 2000, for discussions of alternative additive models).

To calculate  $v[s, \hat{s}]$ , we assume that sperm competition is a fair raffle (Parker, 1982, 1990a,b, 1998) and that the "fertilising power" of the sperm produced by a male is linearly proportional to the resource expenditure  $s$  that the male invests in producing the sperm. Thus, putting twice as many resources into an ejaculate is assumed to double its competitive efficiency, perhaps because there are twice as many sperm

in the ejaculate (we consider the generality of this assumption in section 6.4). The expected reproductive success of a mutant male playing strategy  $s$  from a mating with a female who also mates with  $k$  wild-type males playing strategy  $\hat{s}$  will simply be the proportion of his sperm inseminated in the female,  $s/(s + k\hat{s})$ .

We now need to know the probability  $p_k$  that a female who mates with a focal male will have mated with  $k$  additional males in the breeding season. In a large population with unitary sex ratio, random mating, and a negligible chance that any male will mate with the same female twice, this probability is given by the Poisson distribution

$$p_k = \frac{\hat{n}^k e^{-\hat{n}}}{k!}, \quad (6.2)$$

where  $\hat{n}$  is the expected number of matings achieved by a male playing strategy  $\hat{s}$ <sup>1</sup>. Therefore, the expected reproductive success  $v[s, \hat{s}]$  of a male playing strategy  $s$  against a population playing strategy  $\hat{s}$  is

$$v[s, \hat{s}] = \sum_{k=0}^{\infty} p_k \frac{s}{s + k\hat{s}}. \quad (6.3)$$

We now adopt an evolutionarily stable strategy (ESS) approach (Maynard Smith, 1974, 1982; see also section 1.6.3 in the Introduction). We look for an ESS  $s^*$  that is a strict best reply to itself. In other words, against a population of males playing strategy  $s^*$  any mutant male playing an alternative strategy  $s \neq s^*$  will have strictly lower fitness than the wild type playing  $s^*$ . Mathematically, this states that  $W[s^*, s^*] > W[s, s^*]$  for any  $s \neq s^*$ . Note that, usually, an ESS  $s^*$  requires the weaker condition that  $W[s^*, s^*] \geq W[s, s^*]$  for any  $s \neq s^*$  and that for any mutant strategy  $s$  such that  $W[s^*, s^*] = W[s, s^*]$  we also have  $W[s^*, s] > W[s, s]$ . However, here there will be no alternative best replies, since we show in the Appendix, section 6.5.1, that  $(\partial^2 W / \partial s^2)|_{s=s^*} < 0$ , and so the ESS is a nondegenerate maximum (Williams et al., 2005).

### 6.2.1 Variation in Resources and Costs

We assume a fixed distribution of resource allocation  $R$  and cost  $c$  over the population of males. A male with resources  $R$  and cost  $c$  (referred to as an  $(R, c)$ -male) follows ejaculate investment strategy  $s[R, c]$  and has a corresponding expected number of matings  $n[R, c]$ :

$$n[R, c] = \frac{R}{c + s[R, c]}. \quad (6.4)$$

We denote the population mean values for ejaculate investment and expected number of matings by  $\bar{s}$  and  $\bar{n}$ , respectively. If an  $(R, c)$ -male plays strategy  $s$  against a population of males with mean strategy  $\bar{s}$  and

<sup>1</sup>Note that Williams et al. (2005) use the probability  $q_k$  that a male mates with a female who mates with  $k - 1$  other males, for  $k = 1, 2, 3, \dots$ . Thus, their  $q_k$  is the same as my  $p_{k-1}$ .

mean number of matings  $\bar{n}$ , his fitness is then calculated by substituting equations (6.2), (6.3), and (6.4) into equation (6.1), giving

$$W[R, c, s|\bar{s}, \bar{n}] = \frac{R}{c + s} \sum_{k=0}^{\infty} \frac{\bar{n}^k e^{-\bar{n}}}{k!} \frac{s}{s + k\bar{s}}. \quad (6.5)$$

We wish to find the ESS ejaculate expenditure  $s^*$  for the population as a function of  $R$  and  $c$ . This is the strategy  $s^*[R, c]$ , such that for any pair  $R$  and  $c$  and any  $s \neq s^*[R, c]$ ,

$$W[R, c, s|\bar{s}^*, \bar{n}^*] < W[R, c, s^*[R, c]|\bar{s}^*, \bar{n}^*],$$

so an  $(R, c)$ -male in a heterogeneous male population whose ejaculate expenditure is determined by the function  $s^*$  can only lower his fitness by using a strategy other than  $s^*[R, c]$  (see Appendix, section 6.5.1, for proof that the ESS is a nondegenerate maximum and thus that there can be no alternative best reply).

## 6.2.2 Risk and Intensity of Competition

In classical sperm competition models (e.g. Parker, 1998), the ejaculate expenditure strategy of a male varies with the risk or intensity of sperm competition. Here we follow Williams et al. (2005) and define the risk of competition for a focal ejaculate as the probability that sperm competition occurs ( $1 - e^{-\bar{n}^*}$  at the ESS, since  $e^{-\bar{n}^*}$  is the probability that a female receives no other matings) and the intensity of competition as the number of ejaculates expected to compete for paternity of a given female's offspring ( $\bar{n}^*$  at the ESS). By these definitions, all males face the same risk and intensity of sperm competition. Any differences in optimal ejaculate expenditure strategy will therefore not be due to the risk or intensity differences outlined in classical models.

## 6.3 Results

### 6.3.1 Analysis

An ESS for an  $(R, c)$ -male with quantity of resources  $R$  and cost of obtaining a mate  $c$  will be a solution of the equation

$$\frac{\partial W}{\partial s}[R, c, s|\bar{s}^*, \bar{n}^*] = 0.$$

Assuming that such an ESS exists<sup>2</sup>, we differentiate equation (6.5) with respect to  $s$  at  $\bar{s} = \bar{s}^*$ ,  $\bar{n} = \bar{n}^*$ , to obtain

$$\frac{\partial W}{\partial s}[R, c, s|\bar{s}^*, \bar{n}^*] = \frac{Rc}{(c+s)^2} \sum_{k=0}^{\infty} \frac{(\bar{n}^*)^k e^{-\bar{n}^*}}{k!} \frac{1}{s+k\bar{s}^*} - \frac{Rs}{(c+s)} \sum_{k=0}^{\infty} \frac{(\bar{n}^*)^k e^{-\bar{n}^*}}{k!} \frac{1}{(s+k\bar{s}^*)^2}.$$

At ESS this equals 0, which implies that

$$\frac{c}{c+s^*} \sum_{k=0}^{\infty} \frac{(\bar{n}^*)^k e^{-\bar{n}^*}}{k!} \frac{1}{s^*+k\bar{s}^*} = s^* \sum_{k=0}^{\infty} \frac{(\bar{n}^*)^k e^{-\bar{n}^*}}{k!} \frac{1}{(s^*+k\bar{s}^*)^2}. \quad (6.6)$$

This final rearrangement no longer includes  $R$ , and so the ESS ejaculate investment strategy will not depend on  $R$  (unless the cost  $c$  depends on  $R$ ). Equation (6.6) can be solved to obtain  $c$  as a function of  $s^*$ ,  $\bar{s}^*$ , and  $\bar{n}^*$ :

$$c[s^*|\bar{s}^*, \bar{n}^*] = \frac{\sum_{k=0}^{\infty} [(\bar{n}^*)^k e^{-\bar{n}^*}/k!] [s^*/(s^*+k\bar{s}^*)]^2}{\sum_{k=0}^{\infty} [(\bar{n}^*)^k e^{-\bar{n}^*}/k!] [k\bar{s}^*/(s^*+k\bar{s}^*)]^2} \quad (6.7)$$

Now  $c[s^*|\bar{s}^*, \bar{n}^*] > 0$  for all  $s \geq 0$ , and  $c[s^*]$  is monotonically increasing with  $s^*$  (because each summand in the numerator is monotonically increasing and each summand in the denominator is monotonically decreasing). Therefore, there is a unique inverse function  $s^*[c|\bar{s}^*, \bar{n}^*]$ , defined for  $c \geq 0$ , that is also monotonically increasing. This function is the ESS  $s^*$ .

Thus, the ESS ejaculate investment strategy  $s^*$  is monotonically increasing in  $c$  and depends on  $R$  only insofar as  $c$  depends on  $R$  (e.g. if  $c$  is a decreasing function of  $R$ , then  $s^*$  will also be a decreasing function of  $R$ ). Biologically, this means that a male's optimal ejaculate expenditure will not vary with respect to the quantity of resources allocated to reproduction unless the cost that males pay to obtain a mating also varies with respect to quantity of resources. On the other hand, as a male's expected cost of obtaining a mating increases, his optimal ejaculate expenditure per mating also increases. This makes sense intuitively: males that have to pay a higher cost in resource expenditure in order to obtain a mating (high- $c$  males) will value that mating more highly and thus will invest more sperm. Such males expect to obtain fewer matings, and therefore in this way they exploit the small number of opportunities they get to maximise their overall reproductive success. In contrast, males that have to pay a relatively low cost to obtain a mating (low- $c$  males) will not value each mating so highly and will benefit by investing fewer sperm per mating. By doing this they exploit the large number of matings they gain, more than making up for the lower expected share of paternity per mating.

This can be illustrated using the marginal-value theorem (Charnov, 1976). Males want to maximise

<sup>2</sup>It can be shown analytically that such an ESS exists under weak assumptions on the joint distribution of  $R$  and  $c$  over the male population (R. M. Seymour, personal communication). The analysis can be extended more generally to cover distributions over any set of male characteristics that influence cost, specified by parameters  $g_1, \dots, g_i, \dots$ , with  $g_1 = R$  and  $c = c[g_1, \dots, g_i, \dots]$ , a function of these parameters. However, here we instead construct illustrative examples of the ESS by using an iterative numerical procedure; see Figures 2, 3.

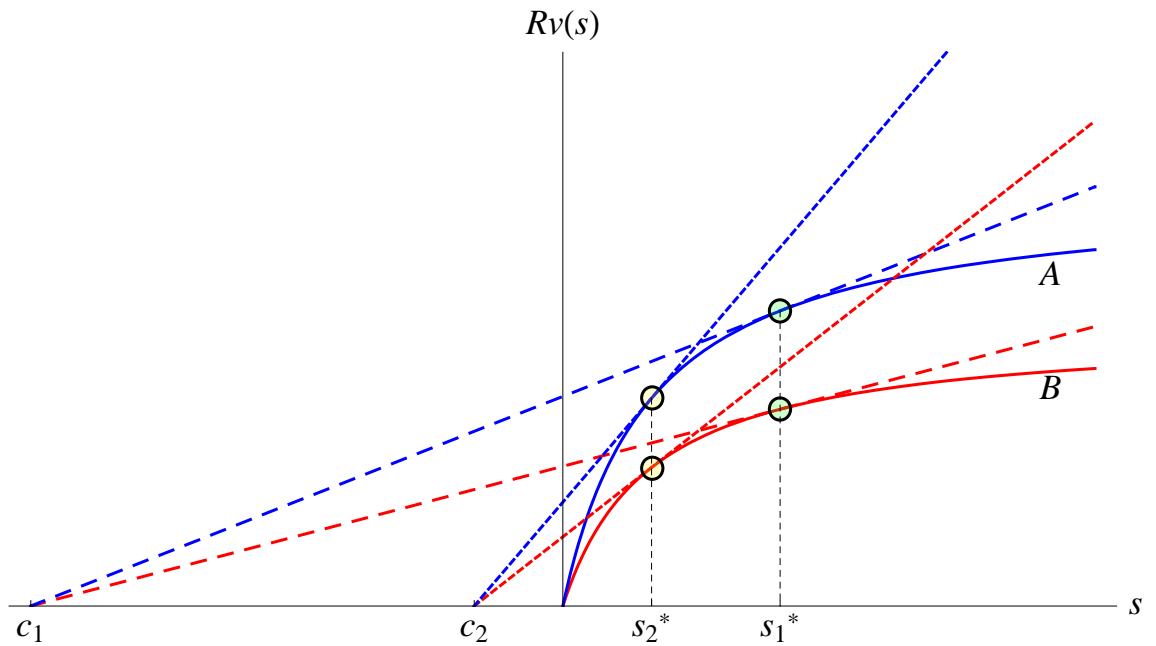


Figure 6.1: Illustration that the optimal ejaculate investment strategy  $s^*$  increases with resource cost ( $c$ ) but is independent of resources ( $R$ ). The solid lines represent the expected reproductive success per mating multiplied by the quantity of resources for two males,  $A$  and  $B$ , plotted against sperm strategy  $s$ . Both males are competing in the same population and thus against the same population mean ejaculate investment strategy  $\bar{s}$  and population mean number of matings  $\bar{n}$ . Male  $A$  has more resources than male  $B$ . Both males attempt to optimise their fitness, which is equal to  $Rv/(c + s)$ , which amounts to maximising the gradients of the dashed lines. If the two males have the same cost (i.e., both have cost  $c_1$  or  $c_2$ ), then their optimal ejaculate investment strategy is the same ( $s_1^*$  if they are both cost  $c_1$ ,  $s_2^*$  if they are both cost  $c_2$ ), despite having differing amounts of resources. If they have differing costs (one has cost  $c_1$  and the other cost  $c_2$ ), then they will have differing optimal ejaculate investment strategies ( $s_1^*$  or  $s_2^*$ , corresponding to cost  $c_1$  and  $c_2$ , respectively).

their overall fitness, which is  $v[s]R/(c + s)$ . Since  $v[s]$  is a sum of increasing concave functions, it is itself increasing and concave (see Appendix, section 6.5.1, for further details). The optimal ejaculate expenditure strategy will increase with  $c$  but will be independent of  $R$  (Figure 6.1). In summary, the harder it is for a male to obtain a mating, the more sperm he will invest in that mating.

### 6.3.2 Numerical Simulations

We used an iterative method that converges on the optimal strategy (see Appendix, section 6.5.2, for details) to numerically calculate some explicit examples of evolutionarily stable ejaculate investment strategies. For clarity of presentation, we broke the problem down into two discrete cases, one in which all males have the same  $c$  value but vary in  $R$  and one in which all males have the same  $R$  value but vary in  $c$ .



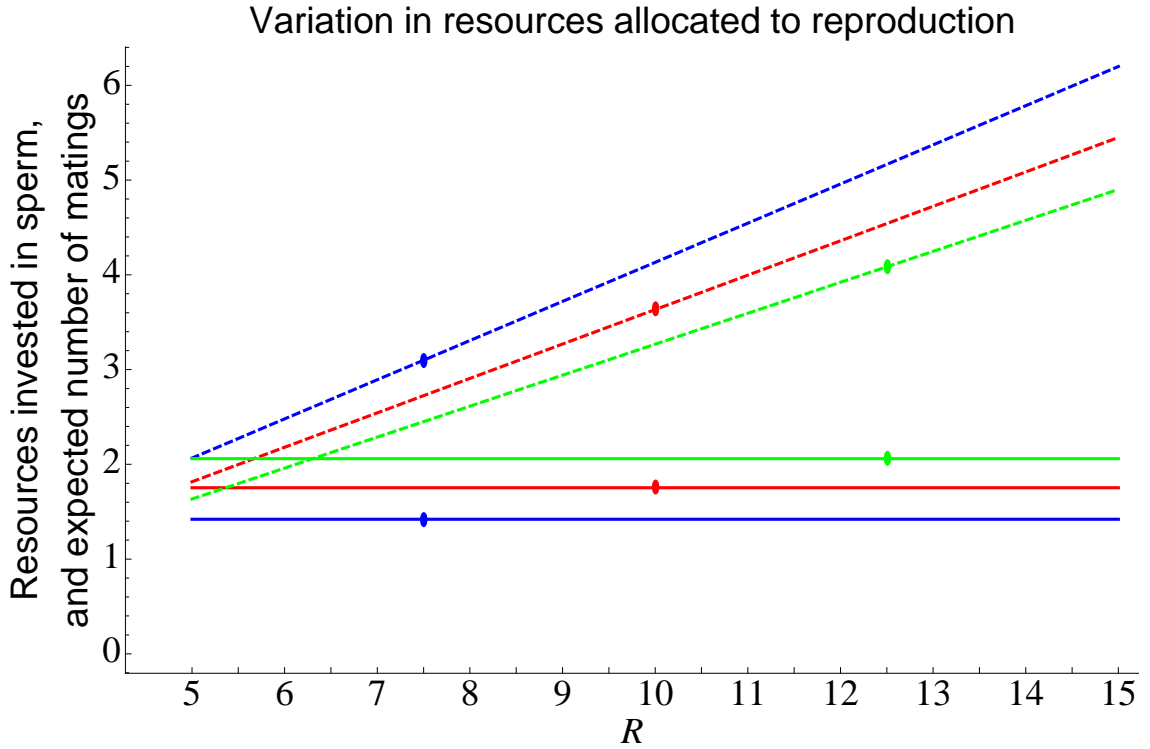


Figure 6.2: Evolutionarily stable optimal ejaculate expenditure strategies  $s^*[R]$  (*solid lines*) and corresponding expected numbers of matings  $n^*[R]$  (*dashed lines*) plotted against total quantity of resources allocated to reproduction ( $R$ ). The position of the mean male is marked on each curve. For each curve,  $R$  values are normally distributed (truncated between 5 and 15) across the population, with standard deviation of 1.5 and means of 7.5 (*blue*), 10 (*red*), and 12.5 (*green*). The cost of obtaining a mate is  $c = 1$  for all males. The functions were obtained with the numerical iterative method described in the Appendix, section 6.5.2.

### Variation in $R$

For a fixed value of  $c$  common to all males, we considered a distribution of  $R$ -values across the male population specified by a truncated normal distribution. Three example ESSs are shown in Figure 6.2. As expected, for all three sets of parameters the evolutionarily stable ejaculate expenditure strategy  $s^*$  does not vary with  $R$ . Since  $n[R] = R/(c + s^*)$ , it follows that the expected number of matings for a male increases linearly with the quantity of resources he allocates to mating. Thus, males with more resources obtain more matings because they are able to stay the course longer.

With all males following the same ejaculate expenditure strategy  $s = \bar{s}^*$ , the fitness function (equation (6.5)) becomes

$$W[R, c, \bar{s}^* | \bar{s}^*, \bar{n}^*] = R \frac{1 - e^{-\bar{n}^*}}{\bar{n}^*(c + \bar{s}^*)},$$

and a male's fitness is proportional to the resources he allocates to breeding. Therefore, lower- $R$  males can never achieve a fitness as high as that of higher- $R$  males.

Using this method, we also investigated how changing the population mean quantity of resources allocated to reproduction  $\bar{R}$  affects the solution to the model when the minimum and maximum  $R$  values are fixed (Figure 6.2). We found that the optimal ejaculate expenditure for all males increases (decreases) with increasing (decreasing)  $\bar{R}$ . Thus, although a male's individual quantity of resources does not affect his optimal ejaculate expenditure, the population mean quantity of resources does. To see mathematically why this occurs, see the Appendix, section 6.5.3. Biologically, this makes sense because if all males on average have more resources for mating and the cost of mating remains the same, then they profit by investing the extra resources in ejaculate expenditure.

### Variation in $c$

For a fixed value of  $R$  common to all males, we considered a distribution of  $c$  values across the male population specified by a truncated normal distribution. Three example ESSs are shown in Figure 6.3. As expected, for all three sets of parameters, the ESS  $s^*[c]$  is an increasing function of  $c$ .

We also note that if  $R$  is fixed for all males, then a higher- $c$  male can never have a fitness higher than that of a lower- $c$  male. To see this, consider a population playing ESS mean ejaculate expenditure strategy  $\bar{s}^*$ , with a corresponding mean number of matings  $\bar{n}^*$ . Suppose we have two males, with costs of obtaining a mating  $c_1$  and  $c_2$ , where  $c_1 < c_2$ . By definition, both males will achieve their maximum fitness only by playing the ESS strategies  $s^*[c_1]$  and  $s^*[c_2]$ . Thus, since expression (6.5) decreases as  $c$  increases for fixed  $s$ , it follows that

$$\begin{aligned} W[R, c_2, s^*[c_2]|\bar{s}^*, \bar{n}^*] &< W[R, c_1, s^*[c_2]|\bar{s}^*, \bar{n}^*] \\ &< W[R, c_1, s^*[c_1]|\bar{s}^*, \bar{n}^*], \end{aligned}$$

and so fitness decreases as cost  $c$  increases.

We also investigated the effects of changing the population's mean cost of obtaining a mate when the minimum and maximum  $c$  values were fixed (Figure 6.3). As illustrated, the ejaculate expenditure strategy for a focal male decreases as the population mean cost  $\bar{c}$  increases. Thus, when males are in a population in which, they find it harder on average to obtain a mate (represented by a higher mean cost  $\bar{c}$ ), the optimal ejaculate expenditure strategy drops. To see why, imagine a focal male with some fixed cost of mating  $c_0$ , and consider the status of this male in two different populations with mean cost values  $\bar{c}_1$  and  $\bar{c}_2$ , where  $\bar{c}_1 < \bar{c}_2$ . Our focal male, regardless of the value of  $c_0$ , will be relatively more attractive in the  $\bar{c}_2$  population, where the average cost per mating is higher, than in the  $\bar{c}_1$  population, where the average cost per mating is

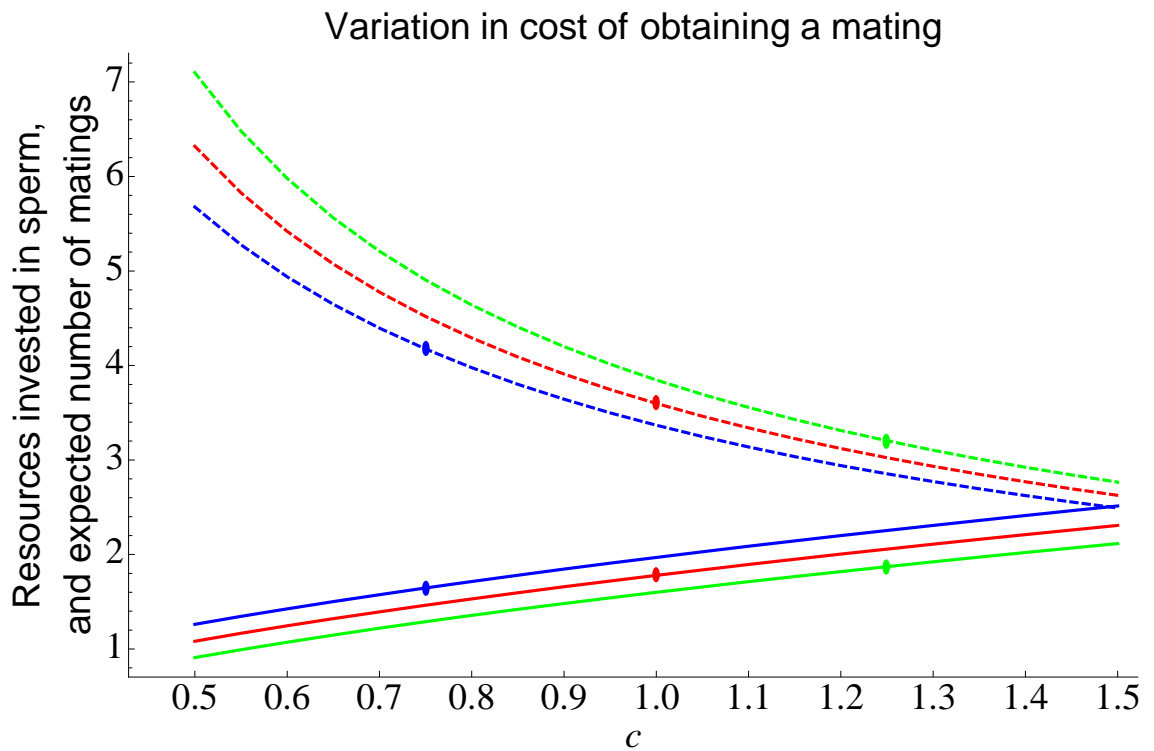


Figure 6.3: Evolutionarily stable optimal ejaculate expenditure strategies  $s^*[c]$  (solid lines) and corresponding expected numbers of matings  $n^*[c]$  (dashed lines) plotted against cost of obtaining a mating ( $c$ ). The position of the mean male is marked on each curve. For each curve,  $c$  values are normally distributed across the population (truncated between 0.5 and 1.5), with standard deviation of  $1/6$  and means of 0.75 (blue), 1 (red), and 1.25 (green). The quantity of resources allocated to reproduction is  $R = 10$  for all males. The functions were obtained with the numerical iterative procedure described in the Appendix, section 6.5.2.

lower, and therefore will invest fewer resources in ejaculate expenditure in the former case than the latter. Since this is true for all values of  $c_0$ , it will be true for all males, and thus it explains why the optimal ejaculate expenditure strategy decreases as the mean cost value  $\bar{c}$  rises.

## 6.4 Discussion

This study investigates how ejaculate expenditure strategies evolve across a continuously varying phenotypic gradient within a single population of males, under the assumptions that sperm competition occurs as a fair raffle with no perceptible differences among females (e.g. degree of polyandry, fecundity). The first general conclusion we draw is that differences in ejaculate expenditure strategies can evolve even within populations of males playing the same reproductive strategy. Sperm competition theory has focussed on ejaculate expenditure strategies in relation to sperm competition risk or intensity (Parker, 1998). While these are obviously important in shaping ejaculate expenditure across populations, our model shows that within a population there can be variation in optimal ejaculate expenditure even when all males face the same risk and intensity of competition in every mating.

To investigate this new theoretical framework, we considered two potential sources of continuous variation: the cost of obtaining a mate ( $c$ ) and the total resources allocated to reproduction ( $R$ ). Our second general conclusion is that variation in the cost of obtaining a mating alone is sufficient to cause differences in optimal ejaculate expenditure strategy, whereas variation in resources allocated to reproduction is not. This can be illustrated by the marginal-value theorem (Figure 6.1; Charnov, 1976). When a male obtains matings more easily than his rivals, he will value each mating less and thus will invest fewer sperm per mating. However, when males all pay the same cost to achieve matings, they all have the same evolutionarily stable ejaculate expenditure strategy, irrespective of the resources at their disposal.

We also investigated the effects of changing the population means of the two candidate traits (i.e.  $R$  and  $c$ ) on the optimal ejaculate expenditure strategy (Figures 6.2 and 6.3). We found that, in the case of varying mating resources  $R$ , the optimal ejaculate expenditure increases with the amount of resources that the population, on average, allocates to reproduction. This is because if, on average, males have more resources available for reproduction and pay the same cost to obtain a mating, they invest the extra resources in ejaculate expenditure (Gage and Cook, 1994). By contrast, in the case of varying mating cost  $c$ , optimal ejaculate expenditure for a given male decreases as the population mean cost of obtaining a mate increases. This is because a given male with a fixed cost of obtaining a mating will be relatively more attractive in a population with a higher mean cost than in one with a lower mean cost.

Our numerical modelling highlights the fact that less attractive males (higher  $c$ ) cannot achieve a fitness

as high as that of more attractive males (lower  $c$ ) by changing their ejaculate expenditure strategy alone, if they have the same quantity of resources. Similarly, in the varying-resources ( $R$ ) model, at the ESS, males with higher  $R$  values have more resources to spend on mating overall and thus end up with higher fitness, because a male's expected fitness is proportional to his  $R$  value if all males have the same cost  $c$ . Thus, in both cases, the least successful males cannot catch up with their more successful rivals through differential ejaculate expenditure alone. If they are to compete successfully, these males will need to adopt some other tactics. In this way, selection could favor the evolution of alternative reproductive strategies (Shuster and Wade, 2003) or other adaptations used by males to defend their paternity, for example, nonfertilising sperm (Cook and Wedell, 1999; Pizzari and Foster, 2008), nonsperm ejaculate compounds such as seminal fluid peptides (Ram and Wolfner, 2007), nonspermic copulations (Løvlie et al., 2005), mate guarding (Alcock, 1994), the imposition of fitness costs associated with mating (Clutton-Brock and Parker, 1995; Johnstone and Keller, 2000; Lessells, 2005), and mating plugs (Simmons, 2001; Moreira et al., 2007).

The value of the cost of obtaining a mating could arise from female preference, with low- $c$  males being those with whom females prefer to mate. However, this is likely to engender a dynamic scenario because low- $c$  males may impose fitness costs on their partners. First, our model suggests that males more likely to be chosen will invest less per ejaculate. Thus, the most attractive males may not be able to fertilise all the eggs of the females that they are able to attract (e.g. in the bluehead wrasse, *Thalassoma bifasciatum*; Warner et al., 1995), generating intersexual conflict over remating rates (Alonzo and Warner, 1999; Pizzari and Snook, 2004). Second, mating with low- $c$  males may also be associated with a higher risk of contracting sexually transmitted diseases since these males will get more mates (Graves and Duvall, 1995). Together, these mating costs generate selection for females to avoid - rather than mate with - attractive males. When the intensity of such selection exceeds the direct or indirect benefits promoting female mating with these males, female preference might cease to favour attractive males. Therefore, continuously varying ejaculate expenditure strategies within a population could have important consequences for the evolution of female preference (see section 7.3.2 in the Conclusion).

In turn, female behavior may drive the evolution of ejaculate expenditure strategies. The model investigated here assumes that there is no relationship between a male's  $c$  value and the promiscuity of his mate, since all males mate randomly with females whose promiscuity is distributed according to the Poisson distribution. In reality, there may be some feedback in this system. Females mated to attractive, low- $c$  males might have a lower propensity to mate again, compared to females mated to unattractive, high- $c$  males. An example of such a mechanism in socially monogamous birds is when females paired with a poor-quality social partner "trade up" through extra-pair copulations with males of more attractive phenotypes or more

compatible genotypes (Jennions and Petrie, 2000). The effect of this on the model would be for the optimal ejaculate expenditure of low- $c$  males to decrease yet further, as they would be less likely to face sperm competition. Conversely, the optimal ejaculate expenditure of high- $c$  males would increase, as they would be more likely to face greater sperm competition. This change would be in line with classical ideas about sperm competition risk (Wedell et al., 2002; Gage and Morrow, 2003). However, the relationship between female promiscuity and  $c$  value may go in the opposite direction, for example, if females mate more often to reduce infertility costs associated with attractive males. In this case, females might be directly selected to remate, and the increased risk of sperm competition faced by low- $c$  males would counterbalance the reduced costs of obtaining mates. It would be interesting to evaluate the relative strength of these selective forces. Clearly, the evolution of ejaculate expenditure strategies must be considered within a dynamic context in which competing male mating strategies coevolve with each other and with female responses (Moore and Pizzari, 2005; Parker and Ball, 2005; Gavrillets and Hayashi, 2006).

In addition, for simplicity our model assumes that a male's ejaculate expenditure is the same for every mating in a given breeding season. This assumption has biological relevance and is consistent with classic sperm competition theory; constant ejaculate expenditure tailored to the average level of sperm competition in the population is expected to arise whenever information on the level of sperm competition associated with individual mating opportunities is unavailable to males (Parker, 1998). Even under loaded-affle dynamics, the ejaculate expenditure of a male is not expected to vary across matings if males have no information on individual mating roles and have similar probabilities of mating in different roles at each mating (Parker, 1998). However, it is possible that differential rates of sperm depletion may, in principle, affect ejaculate expenditure, which would expose low- $c$  males to high remating rates and a higher risk of sperm depletion (see above).

Finally, our numerical modelling considers the cost of obtaining a mate and the quantity of resources allocated to mating as distinct, uncorrelated categories of continuous variation. In the real world, these two parameters could be linked. For example, males who invest heavily in secondary sexual ornaments and thus pay a low cost to obtain a mate may have fewer resources available for breeding. Under these circumstances, we would expect low  $c$  values to correspond to low  $R$  values. On the other hand, in some species, male attractiveness to females (and therefore  $c$ ; see above) may depend directly on male resource levels (or some proxy). This would result in a coupling of low  $c$  values with high  $R$  values. Such associations are also to be expected when  $c$  and  $R$  values are dependent on some other trait(s), such as a male's quality. For example, higher-quality males may be able to invest more in courtship display and/or the maintenance of a sexual ornament or position in a social hierarchy (reducing  $c$  values) as well as in resources for reproduction

(increasing  $R$  values). All of these possibilities can be encompassed by our analysis if we express  $c$  as a function of other parameters, so that  $c = c[g_1, g_2, \dots]$ , one of which could be  $R$ . Then for any  $g_i$ , if  $c$  increases (decreases) with  $g_i$ ,  $s^*$  also increases (decreases) with  $g_i$ . Thus, any factor that makes it easier for a male to obtain a mate would also cause selection to decrease his optimal ejaculate expenditure.

There is some empirical evidence consistent with the model's predictions. For example, in domestic fowl, *Gallus gallus domesticus* (Froman et al., 2002; Cornwallis and Birkhead, 2007; Pizzari et al., 2007); Arctic char, *Salvelinus alpinus* (Rudolfson et al., 2006); and Alpine whitesh, *Coregonus zugensis* (Rudolfson et al., 2008); males with privileged access to females (i.e. low  $c$ ) produce ejaculates of lower fertilising quality (controlling for number of sperm inseminated) than do subordinate males. These findings fit our predictions if sperm quality reflects investment. However, care is required to establish whether reduced ejaculate expenditure in low- $c$  males is a strategic investment by the male or arises coincidentally through sperm depletion due to higher remating rates. To test our ideas, we also need to identify species in which males do not vary greatly in the amount of effort they have to expend in order to obtain a mating. This may plausibly apply to externally fertilising sessile organisms that broadcast gametes or to systems where males have to expend little effort to obtain matings because of female promiscuity. Under these circumstances, the model predicts that all males should invest the same amount of sperm in each mating. More empirical work is needed before strong conclusions can be drawn. Our theoretical work provides a framework for considering individual variation in ejaculate expenditure strategies.

## 6.5 Appendix

### 6.5.1 Proof that any ESS is a nondegenerate maximum

We rewrite the fitness function (equation (6.5)) in the form

$$W[R, c, s|\bar{s}, \bar{n}] = \frac{R}{c+s} \sum_{k=0}^{\infty} \frac{\bar{n}^k e^{-\bar{n}}}{k!} \frac{s}{s+k\bar{s}} = R \frac{v[s|\bar{s}, \bar{n}]}{c+s}, \quad (6.8)$$

where

$$v[s|\bar{s}, \bar{n}] = e^{-\bar{n}} \sum_{k=0}^{\infty} \frac{\bar{n}^k}{k!} \frac{s}{s+k\bar{s}}.$$

For fixed  $\bar{s}$  and  $\bar{n}$ ,  $v[s|\bar{s}, \bar{n}]$  is positive, increasing, and concave, because it is a sum of positive, increasing and concave functions of  $s$ . Equation (6.8) then gives

$$\frac{\partial W}{\partial s}[R, c, s|\bar{s}, \bar{n}] = R \frac{(c+s)v'[s|\bar{s}, \bar{n}] - v[s|\bar{s}, \bar{n}]}{(c+s)^2}, \quad (6.9)$$

where  $v'[s|\bar{s}, \bar{n}]$  is the derivative of  $v[s|\bar{s}, \bar{n}]$  with respect to  $s$ . Equation (6.9) is equal to 0 when

$$v'[s|\bar{s}, \bar{n}] = \frac{v[s|\bar{s}, \bar{n}]}{c + s}. \quad (6.10)$$

The marginal-value theorem illustrated in Figure 6.1 is based on equation (6.10). At any value of  $s$  for which equation (6.10) holds, we have

$$\begin{aligned} \frac{\partial^2 W}{\partial s^2}[R, c, s|\bar{s}, \bar{n}] &= R \frac{(c + s)^3 v''[s|\bar{s}, \bar{n}] - 2(c + s)((c + s)v'[s|\bar{s}, \bar{n}] - v[s|\bar{s}, \bar{n}])}{(c + s)^4} \\ &= \frac{R}{c + s} v''[s|\bar{s}, \bar{n}] \\ &< 0, \end{aligned}$$

where  $v''[s|\bar{s}, \bar{n}]$  is the second derivative of  $v$  with respect to  $s$ . This is true because  $v[s|\bar{s}, \bar{n}]$  is concave. Then any solution of equation (6.10) is a nondegenerate maximum of  $W[R, c, s|\bar{s}, \bar{n}]$ . An ESS  $s = s^*$  is a maximum of  $W[R, c, s|\bar{s}, \bar{n}]$  satisfying  $\bar{s} = \bar{s}^*$  and  $\bar{n} = \bar{n}^*$  and hence must be a nondegenerate maximum of  $W[R, c, s|\bar{s}^*, \bar{n}^*]$ .

## 6.5.2 Details of numerical iterative method

Wolfram Mathematica 6 (Wolfram, 2007) was used to implement the following iterative numerical procedure. Here we describe the procedure for the cost-varying case; an analogous procedure was used for the resource-varying case.

First, the initial parameter value  $R$  of resources each male has for mating is specified. We then suppose that the distribution of  $c$  values over males is specified by a probability density function  $f[c]$ , typically taken to be a truncated normal distribution between fixed values  $c_{\min}$  and  $c_{\max}$ .

To begin the procedure, an initial sperm strategy function  $s_0[c] = 1$  is chosen. The corresponding function  $n_0[c]$  is

$$n_0[c] = \frac{R}{c + s_0[c]} = \frac{R}{c + 1}.$$

Next, Mathematica is used to numerically integrate (using a Gauss-Kronrod quadrature method with a global adaptive strategy; Malcolm and Simpson, 1975) over the interval  $[c_{\min}, c_{\max}]$  to obtain  $\bar{n}_0$

$$\bar{n}_0 = \int_{c_{\min}}^{c_{\max}} \frac{R}{c + 1} f[c] dc.$$

The best reply function  $s_1[c]$  for a male of cost  $c$  in a population with  $\bar{s}_0 = 1$  and  $\bar{n}_0$ , as above, is then the



value that maximises the fitness  $W[R, c, s|\bar{s}, \bar{n}]$ , as defined in equation (6.5), given  $c$  and the fixed  $R$  of the population. The corresponding expected number of matings  $n_1[c] = R/(c + s_1[c])$  can then be obtained. Numerical integration is then used to calculate the means  $\bar{s}_1$  and  $\bar{n}_1$ :

$$\begin{aligned}\bar{s}_1 &= \int_{c_{\min}}^{c_{\max}} s_1[c]f[c]dc, \\ \bar{n}_1 &= \int_{c_{\min}}^{c_{\max}} \frac{R}{s_1[c] + c} f[c]dc.\end{aligned}$$

The procedure is then iterated until solutions cease to vary by more than a specified tolerance. In our implementation, the procedure was repeated until  $|\bar{s}_i - \bar{s}_{i-1}| < 10^{-4}$ . The ESS was then taken to be  $s_i[c]$ , with corresponding expected number of matings  $n_i[c] = R/(c + s_i[c])$ .

### 6.5.3 Proof that increasing $\bar{R}$ increases $\bar{s}^*$

Assume that the distribution of  $R$  over the male population is specified by a probability density function  $g[R]$ . Consider the equilibrium equation

$$\frac{c}{c + s^*} \sum_{k=0}^{\infty} \frac{(\bar{n}^*)^k e^{-\bar{n}^*}}{k!} \frac{1}{s^* + k\bar{s}^*} = s^* \sum_{k=0}^{\infty} \frac{(\bar{n}^*)^k e^{-\bar{n}^*}}{k!} \frac{1}{(s^* + k\bar{s}^*)^2}. \quad (6.11)$$

where  $s^* = \bar{s}^*$  is independent of  $R$  and

$$\bar{n}^* = \int_0^{\infty} \frac{R}{c + s^*[R]} g[R]dR = \frac{\bar{R}}{c + \bar{s}^*}. \quad (6.12)$$

To simplify notation, write  $s^* = \bar{s}^* = \bar{s}$  and  $\bar{n}^* = \bar{n}$ . Then, equations (6.11) and (6.12) give two equilibrium equations:

$$\bar{s} \sum_{k=0}^{\infty} \frac{\bar{n}^k}{(k+1)(k+1)!} = c \sum_{k=0}^{\infty} \frac{\bar{n}^k}{(k+1)!} \left(1 - \frac{1}{k+1}\right), \quad (6.13)$$

$$\bar{R} = \bar{n}(c + \bar{s}). \quad (6.14)$$

Note that equation (6.13) defines  $\bar{s}$  as a function of  $\bar{n}$ , and hence equation (6.14) defines  $\bar{R}$  as a function of  $\bar{n}$ . Multiplying equation (6.13) through by  $\bar{n}$ , we can write

$$\bar{s} = c \left( \frac{\sum_{k=0}^{\infty} \bar{n}^{k+1}/(k+1)!}{\sum_{k=0}^{\infty} \bar{n}^{k+1}/(k+1)(k+1)!} - 1 \right). \quad (6.15)$$

The expression in the numerator,  $N[\bar{n}]$ , in equation (6.15) is

$$N[\bar{n}] = e^{\bar{n}} - 1, \quad (6.16)$$

and the expression in the denominator,  $D[\bar{n}]$ , can be written as

$$D[\bar{n}] = \sum_{k=0}^{\infty} \bar{n}^{k+1} \frac{1}{(k+1)(k+1)!} = \int_0^{\bar{n}} \frac{1}{u} (e^u - 1) du. \quad (6.17)$$

Thus, from equations (6.15)-(6.17) we obtain  $\bar{s}$  as a function of  $\bar{n}$ :

$$\bar{s}[\bar{n}] = c \left( \frac{N[\bar{n}]}{D[\bar{n}]} - 1 \right) = c \left( \frac{e^{\bar{n}} - 1}{\int_0^{\bar{n}} (1/u)(e^u - 1) du} - 1 \right). \quad (6.18)$$

We show below that  $\bar{s}[\bar{n}]$  is monotonically increasing in  $\bar{n}$ . Note that  $N[\bar{n}]/D[\bar{n}] \rightarrow 1$  as  $\bar{n} \rightarrow 0$  (by l'Hôpital's rule), and hence  $\bar{s}[0] = 0$ .

It follows from equation (6.14) that, if  $\bar{s}[\bar{n}]$  is a monotonically increasing function of  $\bar{n}$ , then so is  $\bar{R}[\bar{n}]$ . Thus,  $d\bar{s}/d\bar{R} = (d\bar{s}/d\bar{n})/(d\bar{R}/d\bar{n})$  is positive. This gives the required result that  $\bar{s}$  increases as  $\bar{R}$  increases.

It remains to show that  $\bar{s}[\bar{n}]$  is a monotonically increasing function of  $\bar{n}$ . From equation (6.18), we are required to show that  $N[\bar{n}]/D[\bar{n}]$  is increasing. Since  $N[\bar{n}]$  and  $D[\bar{n}]$  are both nonnegative, this is the case if

$$\frac{N'[\bar{n}]}{N[\bar{n}]} \geq \frac{D'[\bar{n}]}{D[\bar{n}]},$$

with equality only if  $\bar{n} = 0$ . That is, using equations (6.16) and (6.17),

$$D[\bar{n}] \geq \frac{1}{\bar{n}} (e^{\bar{n}} + e^{-\bar{n}} - 2), \quad (6.19)$$

with equality only if  $\bar{n} = 0$ .

To verify equation (6.19), consider the function  $F[\bar{n}] = D[\bar{n}] - (1/\bar{n})(e^{\bar{n}} + e^{-\bar{n}} - 2)$ . Then,  $F[0] = 0$ , and

$$\begin{aligned} F'[\bar{n}] &= \frac{1}{\bar{n}} (e^{\bar{n}} - 1) - \frac{1}{\bar{n}} (e^{\bar{n}} - e^{-\bar{n}}) + \frac{1}{\bar{n}^2} (e^{\bar{n}} + e^{-\bar{n}} - 2) \\ &= \frac{1}{\bar{n}^2} (1 - e^{-\bar{n}}) (e^{\bar{n}} - (1 + \bar{n})) \\ &\geq 0, \end{aligned}$$

with equality only when  $\bar{n} = 0$ . Thus  $F[\bar{n}]$  is monotonically increasing for  $\bar{n} > 0$ , which proves equation (6.19) and hence gives the required result.

## Chapter 7

# Summary, conclusions and further work

### 7.1 Summary

The aim of this thesis was to investigate the evolutionary causes and consequences of mate choice using mathematical modelling techniques. Sexual selection as an evolutionary force is caused by mate choice, since it is this non-random mating that affects the evolution of a species. Mate choice is therefore of potentially huge evolutionary importance, and modelling it of great interest.

Since mate choice is such an enormous field of research, it is impossible for any single piece of work to cover it in its entirety. Therefore I have necessarily had to brush over some aspects. I have split the work into two halves. The first covered the evolution of mate choice, while the second considered its consequences.

#### 7.1.1 Evolution of mate choice

The evolution of mate choice was considered in two aspects. First we looked at *Saccharomyces* yeast, a single-celled organism that usually reproduces via asexual budding. Under starvation conditions, however, yeast cells undergo meiosis and form an ascus of four haploid spores which germinate once conditions improve, and then mate to form two diploid offspring. Of the four haploids, two are of mating type  $\alpha$ , and two are of mating type **a** (Herskowitz, 1988; Spencer and Spencer, 1997). Mating occurs only between spores of different mating types, so  $\alpha$  haploids mate with **a** haploids. If the parent yeast cell is heterozygous for deleterious mutant alleles at some loci, one of the  $\alpha$  cells will in general inherit fewer deleterious

mutant alleles than the other (and similarly, one of the **a** cells will inherit fewer deleterious mutant alleles than the other). We call the haploid of each mating type with the fewer deleterious mutant alleles the ‘stronger’ haploid (and the other, by contrast, the ‘weaker’). Because there is this difference in genetics in the haploids, there is the possibility for selective mating across the ascus. This is equivalent to gametic choice of the diploid parent, and is therefore a form of mate choice (Pagel, 1993). We used a population genetics model to show that if the stronger  $\alpha$  haploid and the stronger **a** haploid mate together (leaving the weaker of each mating type to mate together) then the yeast colony resulting from the two diploid offspring will have higher fitness in the long term than either a colony descended from an ascus in which mating was at random, or a colony descended from two diploids identical to the original parent (as though the parent had not undergone meiosis and sporulation at all but had rather reproduced by asexual budding). The mate choice strategy will also lead to a colony with lower mutational load than the other two strategies (in the sense of having a smaller proportion of deleterious mutants segregating in the population). Therefore mate choice is beneficial even in the absence of outbreeding, and so we conclude that it is likely to be even more widespread in the natural world than previously thought.

The second aspect of the evolution of mate choice that we considered was to see what kind of mate choice we expect to evolve. We assumed that females get some sort of benefit from a wise choice, but that this benefit could be one of two kinds, fixed or dilutable. The difference can be seen as follows. Take a given male. Every female who mates with him gains a benefit of some sort. We can characterise the average benefit he provides to his mates as the expected benefit from mating with that male. Fixed benefits are those in which the expected benefit accruing to each female that mates with a given male is independent of the number of mates he has. An example of this is a good genes benefit, since a male will pass on the same (expected) quality of genetic material to each of his mating partners. Dilutable benefits, by contrast, are those in which the expected benefit accruing to each female mating with a given male will decrease as the number of mates he has increases. An example of this is paternal care, which is likely on average to decrease as the number of mates a male has increases (for example the polygyny threshold model, Searcy, 1979). This distinction in types of benefit is different to the usual distinction drawn between direct and indirect benefits (see Introduction, section 1.4). We demonstrated that it is a useful distinction by using a game theoretic model to show that it leads to different types of mate choice. In the fixed benefits case, the best strategy for females is to mate with the male giving the highest benefit. Since it doesn’t matter how many other females mate with the same male, they will therefore all mate with the top male, leading to high mating skew. In the dilutable benefits case, on the other hand, female choice will be more complicated. Imagine if all males got the same number of mates. It may be that some males would then give higher

expected benefits than others. We call these males the ‘better’ males in the following argument, while those that give lower expected benefits are called ‘worse’. The evolutionary stable mate choice strategy for females turns out to be to mate with males according to some probability distribution, favouring better males, but not necessarily excluding worse males entirely. Better males then end up with more mates than worse males on average, but unlike in the fixed benefit case the best male will not necessarily monopolise all matings. The net effect of this ESS female choice strategy is that the group of females is spread out across the group of males so that all males who get mates give the same expected benefit. In some cases there may be some subset of males too poor to be worth any females mating with them at all, but this will not always happen. In the fixed benefit case, on the other hand, all males but the very best will be excluded. One consequence of this finding is that mating skew is expected to be much lower in species in which females obtain dilutable benefits from their mates than in species in which they obtain only fixed benefits.

### **7.1.2 Consequences of mate choice**

The second part of this thesis, on the consequences of mate choice, was broken down into three chapters. The final chapter of the first part, with its theoretical prediction of the consequences of mate choice on mating skew, led us nicely into the first chapter of the second part. In this chapter we performed a review of 38 studies into wild populations of birds and analysed the mating skew using two different measures (Kokko and Linstrom, 1997; Tsuji and Tsuji, 1998). We found that, by both measures, lekking populations had a significantly higher mating skew than non-lekking populations. This could be a consequence of the fact that in lekking populations females receive only sperm from the males, and therefore may be getting good genes benefits from mate choice. Good genes benefits are fixed in the above sense, and thus we would expect high mating skew. On the other hand, in non-lekking populations, females often receive territory or paternal care benefits from the males, which are expected to be dilutable in the above sense. So the data are in line with our theoretical predictions, although we cannot conclude with certainty whether fixed benefits effects are truly the root cause of the high mating skew, or whether lekking, high mating skew, and fixed benefits are all correlated for some other reason.

The rest of the second part concerned consequences of mate choice in areas other than just mating skew. In chapter 5 we used quantitative genetics techniques to model the effect of mate choice on colour in reproductively isolated populations of *Oophaga pumilio*, the Strawberry Poison-dart Frog. We considered the evolution of the population means of two traits: colour, and female preference. Our approach was novel in using stochastic difference equations, splitting the change per generation of the mean traits into two parts; that due to selection, and that due to drift. That due to selection was dealt with using standard

quantitative genetics, while that due to drift was given as a stochastic function, independent from one generation to the next. We showed that sexual selection will drive mean colour and mean female preference together. However, small changes due to drift in each generation will cause slight differences between the two means, so that evolution does not cease once the two mean trait values become equal. Rather, drift is constantly moving the two traits apart, and selection has to constantly work to push them back together. In this way the population travels around the trait space. In pushing mean traits together, selection could either (a) largely push preference to match colour, (b) push both traits together approximately equally, or (c) largely push colour to match preference. For parameter values in which (c) is the case, we showed that sexual selection could increase the variation in mean colour between populations, when compared to the variation in colour between populations evolving by drift alone. This is because in case (c), sexual selection forces colour to match female preference as it evolves by drift. Thus if preference is particularly prone to evolution by drift it will lead to large variation in colour. We call this “evolution by coupled drift”, and suggest it could be a widespread evolutionary force. However, for other parameter values, sexual selection could actually decrease the variation in mean colour between populations. For example, if we are in case (c), but preference is largely unaffected by drift and is thus evolutionarily very stable, then colour will not be able to drift far, because sexual selection will constantly push it back to take the same value as preference. In addition, some parameter values will lead to sexual selection having little or no effect on drift, for example if colour and preference have very similar propensities to evolve by drift, since then it doesn’t matter whether they are coupled or uncoupled. The full analysis of parameter space therefore shows a wide range of possibilities, and doesn’t prove that the evolution of the wide-ranging array of colour morphs in *O. pumilio* is due to sexual selection via coupled drift. However, our work does serve as a proof of principle, showing that the hypothesis that it is sexual selection that caused *O. pumilio* populations to vary so much when compared to other sympatric poison-dart frogs (Summers et al., 1999) is theoretically plausible.

The final chapter in the thesis showed that pre-copulatory mate choice can also affect post-copulatory strategy, in the form of sperm competition. We used a game theoretic model to assess how this could occur. Males had two traits, their quantity of resources for attracting mates and allocating sperm ( $R$ ), and the cost they had to pay to obtain a mate ( $c$ ). Unlike in many sperm competition models (e.g. Parker, 1990a; Mesterton-Gibbons, 1999b), in which the males in the population take up discrete morphs or mating roles, in our model the variation between males was continuous, so that all males had some values ( $R, c$ ), and the distribution of males across this space was governed by some function. Males could choose their ejaculate investment strategy  $s$ , which was the mean amount of resources they would allocate to sperm per mating.

The higher a male's strategy  $s$ , the higher the reproductive success he would get from each mating, but (as males are limited by their total quantity of resources  $R$ ) the fewer matings he would be able to afford (since each mating would cost him  $s + c$  resources, as  $c$  is the resource cost of obtaining a mate). In addition, the situation was further complicated by the fact that reproductive success would also depend upon how much sperm the average male was investing per mating,  $\bar{s}$ , and how many matings each female was expected to have,  $\bar{n}$  (which was equal to the mean number of matings a male was expected to have since the operational sex ratio was assumed to be unity). Therefore our model captured a complex evolutionary process in which a male's optimal strategy depended upon his own trait values ( $R, c$ ) and also upon the behaviour of other males. Our first finding was that, perhaps surprisingly, the quantity of resources a male had (his  $R$ -value) had no effect on his ESS for ejaculate investment. Therefore the important trait was the cost  $c$  a male paid to obtain a mating, which could be seen as a measure of his attractiveness, with unattractive males having to pay more to get mates than attractive males. Along the continuum of attractiveness, the ESS ejaculate investment strategy declines as attractiveness increases (i.e. as the cost to obtain a mate declines). Thus, the easier it is for a male to obtain a mate, the fewer sperm he is expected to invest per mating. A male who paid a high cost  $c$  to obtain a mate would do best by also having a high  $s$ , since in this way he could maximise his paternity from the few matings he could afford. A male who paid a low cost  $c$  to obtain a mate would do best by having a low  $s$ , since in this way he would be able to maximise the number of matings he had overall and thus gain a higher fitness. We used numerical simulations to show the ESS and corresponding expected number of matings in populations in two scenarios; (1) with fixed  $c$ -values but  $R$ -values normally distributed with given means and standard deviations, and (2) with fixed  $R$ -values but  $c$ -values normally distributed with given means and standard deviations.

## 7.2 Conclusion

This thesis has covered a wide range of topics, all linked through their association with mate choice. We have gone from the minutiae of genetic details in single-celled *Saccharomyces* yeast to the effects of mate choice on events possibly leading to speciation in *Oophaga pumilio*, and dealt with the evolution of different strategies in pre- and post-copulatory situations.

We have seen that mate choice can be expected to evolve in an extraordinary variety of species. We already know that sex is widespread amongst living organisms (Maynard Smith, 1978), but chapter 2 showed that even in the absence of outbreeding, the evolution of a form of mate choice between gametes can be favoured, meaning the set of species that could be affected by the evolution of mate choice is even larger than previously thought.

The consequences of mate choice have also been shown to be enormously varied. Females gaining fixed benefits from males are likely to all choose to mate with the same male (or small subset of males) leading to high mating skew, which in turn could lead to lower genetic variation in the population, possibly slowing the response to selection of a population and making extinction more likely. Alternatively, high mating skew could aid in suppression of deleterious mutations (Agrawal, 2001; Siller, 2001), or in selection of the most advantageous genes (Iwasa et al., 1991), meaning the population responds to selection rapidly. Females gaining dilutable benefits from males will spread matings around the population of males so that all mate-worthy males give the same expected benefits, but meaning males will be under selection pressure to maximise the benefits they give to females. Conflict or adaptation enabling males to obtain better resources will then be favoured. We have also seen that mate choice can, under some circumstances, cause allopatric populations to vary to a greater degree because of genetic drift than they otherwise would. This, coupled with the fact that in such cases assortative mating can also occur, could cause pre-zygotic barriers to mating and speed up speciation (Coyne and Orr, 2004). But it has also been shown that for other parameter values sexual selection could in fact suppress variation between populations, and thus act to slow speciation down. Finally, we have seen that pre-copulatory mate choice can have an effect on post-copulatory strategy in the form of ejaculate investment strategy. This could have intriguing knock-on effects. If females obtain very little sperm from males that they prefer, then there is a possibility that preference will switch to other types of male who will provide them with more fertility (Sheldon, 1994). This could lead to a fascinating coevolutionary system. If disfavoured males are unable to make up for their disadvantage through sperm strategy, the evolution of alternative mating tactics may be selected for, leading to a wide variety of behaviours and adaptations, each with further potential evolutionary consequences.

The overall conclusion of this thesis, then, is that sexual selection is likely to be extremely widespread and powerful an evolutionary force, and could affect evolutionary events that seem to have no obvious link to mating behaviour. Mathematical modelling is uniquely placed to be able to ascertain whether or not sexual selection could affect evolution across such a wide variety of contexts, and can also assess the likely consequences of this effect.

### **7.3 Further work**

Some of the work in this thesis throws up intriguing possibilities for follow-up studies to investigate the conclusions drawn. I will give four examples here of lines of investigation that build on the work already done.



### **7.3.1 The evolution of mating in yeast**

The work in chapter 2 contained an idealised model of the yeast life cycle. In reality, of course, things are more complicated, particularly with regard to intratetrad mating. Haploid spores can themselves reproduce by budding, and frequently do so, mating-type switching can occur, and even in the absence of these effects, asci do not always result in two diploid offspring (C. Smith, personal communication). In addition, levels of outbreeding in yeast may in fact be higher than previously thought, and may depend on environmental conditions (Murphy and Zeyl, 2010; C. Smith, personal communication). There is a need for mathematical modelling to assess how adaptive much of this behaviour is likely to be and to make predictions about when it is most likely to be seen. This is especially the case since so little is known about the ecology of yeast in the wild.

### **7.3.2 The phenotype-linked fertility hypothesis**

The work in chapter 6 suggests that males favoured by females will be selected to invest less sperm per mating than those disfavoured. The phenotype-linked fertility hypothesis, however, suggests that females choose their mates based upon the fertility benefits they are likely to obtain (Introduction, section 1.4.1; Chapter 3, section 3.1; Trivers, 1972; Sheldon, 1994). Therefore there is likely to be coevolutionary feedback between females choosing males with strategies favouring large ejaculate investment, and consequent selection on preferred males to lower their ejaculate investment. Against this background, theoretical modelling is required to assess whether the phenotype-linked fertility hypothesis can be evolutionarily stable or not. Chapter 3 may give a framework in which to ground the work, as it features a model dealing with female choice. This will be particularly helpful as fertility is likely to be a dilutable benefit. Also, the model assumes that females cannot directly assess the benefits they obtain, but rather some proxy trait. However, we want to consider the effects of selection to lower ejaculate investment on an evolutionary timescale, rather than the effect of sperm depletion. This is likely to result in the relationship between the trait that females are able to observe and the expected benefit (given a certain popularity) changing over evolutionary time. We therefore wish to build a model to find out whether female choice for certain traits that correlate with fertility benefits can remain stable over evolutionary time under the pressures described, and under what circumstances this can occur.

### **7.3.3 Phylogenetic study of mating skew in birds**

Chapter 4 was a preliminary study of mating skew in lekking and non-lekking species of birds, and showed that mating skew is higher in the former mating system than the latter. However, what we really want

to know is whether female choice for fixed benefits leads to higher mating skew than that for dilutable benefits. We used lekking and non-lekking as proxies for fixed and dilutable benefits respectively. A full study, however, would have to feature more data than was used in chapter 4, and involve characterising the benefits believed to be obtained by females for each species of bird. These could then be labelled as being fixed or dilutable. In addition, a phylogeny would have to be used to account for the shared evolutionary history between bird species which means that they are not truly independent data points (Bennett and Owens, 2002). Such a study would obviously be of interest as an empirical test of the theoretical findings of chapter 3, and would also be of use to those studying mating skew and those studying mate choice in birds.

### **7.3.4 Coupled drift and the evolution of genitalia**

The analysis of chapter 5 throws up the potential widespread importance of evolution by coupled drift, whereby one trait is forced by selection to match another, which in turn evolves by random drift. It would be interesting to find other situations in which this evolutionary effect could occur in order to assess how general it is. One situation in which it may occur is in the evolution of genitalia. There is a huge variety in shape and size of male intromittent genitalia across a wide range of animal taxa, such that genitalia often vary much more than other traits, and can often be used to distinguish between closely related species (Arnqvist, 1998; Eberhard, 2010). Three major possible explanations for this phenomenon have been put forward: the lock-and-key hypothesis (Shapiro and Porter, 1989), whereby females benefit by having different genital shapes to those of closely related species so as to avoid costly hybridisation events; sexual conflict (Arnqvist, 1998), whereby male genital structures evolve so as to exert control over females in some damaging way, and female genital structures evolve to prevent this occurring (see section 1.3.3 in the Introduction); and cryptic female choice (Eberhard, 2010), whereby female genitalia evolve to allow for active selection of sperm from preferred males so that only these gain access to eggs, and male genitalia act as courtship devices to encourage females to choose the sperm of their bearer. Evidence for all of these three hypotheses is unclear, with differing conclusions being drawn in different studies (Arnqvist, 1998; Eberhard, 2010). Coupled drift could provide an alternative explanation: that female genitalia is under little selection, and evolves largely by drift. Selection then forces male genitalia to coevolve to fit the shape of female genitalia, causing the between-species divergence seen. A model of this using the techniques of chapter 5 might be able to shed light on how plausible an explanation it is, providing predictions that could be tested by empirical or comparative studies, and compared with other possible explanations.

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