Sexual selection and sex allocation

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CHAPTER 1

Introduction

The prevalence of conspicuous male characters that seemed detrimental to the survival of their bearers posed a major problem for Darwin's theory of evolution by natural selection. To overcome this, Darwin (1859) introduced the idea of sexual selection, which is the outcome of competition between members of one sex for matings with the opposite sex (Andersson, 1994). It is now widely acknowledged that this competition for matings can be a powerful selective force, leading to the evolution of many characters that continue to amaze students of evolution. For example, sexual selection is associated with the evolution of elaborate calls and songs (Searcy & Andersson, 1986), animal weapons such as horns and spines (Emlen, 2008), iridescent colors (Hill & McGraw, 2006), odors and pheromones (Johansson & Jones, 2007) and a range of other behaviours and morphologies that are difficult to explain from an evolutionary perspective that focuses on survival alone. Notwithstanding Darwin's (1871) exclusive focus on sexual selection in animals, sexual selection is now considered an important force in plants as well (Skogsmyr & Lankinen, 2002; Moore & Pannell, 2011) and more recently the process of sexual selection also has been found to occur in a number of fungi (Rogers & Greig, 2009; Nieuwenhuis *et al.*, 2011), showing that the process of sexual selection extends to many more taxa than Darwin (1871) had in mind.

1.1 The origin of sexual selection and the evolution of mate choice

The origin of sexual selection is widely considered to lie in the evolution of differential gamete investment or anisogamy (Trivers, 1972; Kokko *et al.*, 2006; Schärer *et al.*, 2012). Although not treated in-depth here, the evolutionary origins of anisogamy are interesting in their own right: anisogamy is commonly attributed to arise from disruptive selection due to the trade-off between gamete size and number (Bulmer & Parker, 2002), but alternative hypotheses on the evolution of anisogamy exist (see Lessells *et al.* 2009 for an insightful review). When anisogamy has evolved, smaller and more numerous sperm compete for fertilizations of larger and fewer eggs, giving rise to the competition over fertilizations that is the hallmark of sexual selection. It is easy to envisage the rapid evolution of male traits that result in a higher share of fertilizations at the cost of their competitors, and indeed genes that are directly related to male fertilization success show patterns of rapid evolutionary divergence (e.g., Swanson & Vacquier, 1998; Civetta & Singh, 1998; Van Doorn *et al.*, 2001).

Although anisogamy explains the evolution of male traits that increase a male's share of fertilizations through male-male competition (intrasexual selection, Darwin, 1859), it does not provide an explanation for the widespread evolution of characters involved in mate choice, such as mate assessment behaviours in lekking animals (Höglund & Alatalo, 1995), cryptic choice among mating partners through elaborate genital morphologies (Eberhard, 1996) or selective mating through mate avoidance behaviours (e.g., Arnqvist & Rowe, 1995). Mate choice traits are often more strongly expressed in females relative to males (Kokko & Monaghan, 2001) and understanding the evolution of female choice is essential to explain the widespread evolution of male ornaments or other display characters that increase male attractiveness to choosy females. Importantly, the evolution of female choice is still widely considered to be just a mirror image of the evolution of the male-male competition over fertilizations, based on the common observation that the sex which does not compete over matings (which is often the female) is the sex with the strongest degree of mate

choice (Emlen & Oring, 1977). However, female choice can evolve independently from male-male competition (Owens & Thompson, 1994; Kokko & Monaghan, 2001; Johnstone *et al.*, 1996), so that it is required to investigate which mechanisms can give rise to the evolution of female choice. Faced with a tremendous amount of interspecific and intraspecific variation in mate choice and coevolving display traits, one of the major goals of the study of sexual selection is therefore to understand the different mechanisms that underlie the benefits and costs of choice (Andersson, 1994; Kokko *et al.*, 2006; see chapter 2 of this thesis).

Why models of sexual selection are useful

Over the last century, students of sexual selection have proposed a variety of mechanisms to explain how mate choice could be beneficial. Initial hypotheses to explain the benefits and costs of choice have been formulated entirely in verbal terms. Fisher (1915) was the first to provide a hypothesis on the evolution of female choice by envisaging a mechanism in which a pre-existing mate preference character could indirectly assist in its own propagation: females expressing a mate preference are more likely to mate with those males bearing elaborate display characters, and their sons are thus likely to inherit both the alleles coding for the female preferences as well as alleles coding for the attractive display. Due to this attractive display, these sons, in turn, are preferred by females that bear the preference allele. As a result, these sons have a higher mating rate than sons from a mother that does not bear the preference allele, so that more grandoffspring will inherit the preference allele, leading to an increase in frequency of the preference allele. It was only 60 years later that, based on Fisher's insights, Zahavi (1975) devised the "handicap" hypothesis. This hypothesis states that females profit from being choosy if their preference is directed to those males that signal genes of high quality ("good-genes"). Zahavi argued that, in order to prevent low quality males from faking these signals, traits indicating quality should be costly to produce (a "handicap"), such that only high-quality males can afford to bear these costs.

Although the Fisher and good-genes processes are two very influential verbal hypotheses on the mechanisms underlying the benefits of mate choice, they have also given rise to considerable confusion. Debates on assumptions implicit in the Fisher process (e.g., Cameron *et al.*, 2003; Kokko *et al.*, 2006) and good-genes sexual selection (Maynard Smith, 1978; Bell, 1978; Grafen, 1990a; Kirkpatrick, 1992; Getty, 2006) have not been fully resolved. This continued confusion that surrounds these hypotheses shows that intuitive ideas and verbal arguments are a crucial first step in the development of any theory, but that processes like sexual selection are simply too intricate to fully understand their ramifications by verbal reasoning alone, at least for mere mortals not equipped with Fisher's brain power The subsequent implementation in mathematical models gives these ideas the necessary precision, exposes hidden assumptions, and clarifies the mechanisms at work. Moreover, when one knows the important components and assumptions of a model, it also becomes much easier to make predictions and to formulate new verbal ideas as extensions of the initial model.

Formal models that assess the plausibility of different hypotheses on the benefits of mate choice have been developed since the 1970s (see chapter 2 of this thesis for a review). For example, a substantial number of models have now been devised that

predict the conditions under which the Fisher and good-genes processes lead to the evolution of female choice and coevolving male display traits (e.g., Lande, 1981; Grafen, 1990b; Pomiankowski *et al.*, 1991; Iwasa *et al.*, 1991; Hall *et al.*, 2000; chapters 3, 7 and 8 of this thesis). In addition, models have assessed the plausibility of female choice being driven by other benefits of choice, such as male parental care (the "good parent" process) (Hoelzer, 1989; Price *et al.*, 1993; Iwasa & Pomiankowski, 1999), the evolution of female choice as a pleiotropic by-effect of natural selection (Kirkpatrick & Ryan, 1991; Arak & Enquist, 1993; Fuller *et al.*, 2005) or female choice as a means to avoid costly male matings (sexual conflict, Gavrilets *et al.*, 2001; Rowe *et al.*, 2005. The development of these models has had a major influence on empirical research, since it provided the field with predictions on the variety of factors that contribute to the costs and benefits of mate choice (Andersson, 1994; Andersson & Simmons, 2006), and how to experimentally disentangle them (e.g., Hettyey *et al.*, 2010). Moreover, the development of sexual selection theory has improved our understanding of other evolutionary processes as well, since sexual selection plays an important role in processes such as speciation (Ritchie, 2007) and the evolution of parental care (Kokko & Jennions, 2008).

Towards a robust theory of sexual selection

Aforementioned sexual selection models provide us with a set of testable predictions on the conditions that lead to the evolution of female choice and male display traits. For example, such models predict that the strength of selection on female choice involved in Fisher or good-genes processes is relatively weak, when compared to other forms of sexual selection, such as good-parent sexual selection or sexual conflict (Kirkpatrick & Barton, 1997; Cameron *et al.*, 2003). At the same time, it is clear that such predictions result from models which – understandably – make many simplifying assumptions, to facilitate analysis and interpretation. Such simplifying assumptions pervade at all levels of organismal complexity, from the genetic level where sexually selected traits are commonly assumed to be encoded by few loci that carry alleles with additive effects, to the population level, where stochastic demographical effects are typically considered absent, populations are infinite and spatial structure is generally considered to be unimportant.

To arrive at a testable theory of sexual selection, it is essential to assess if predictions from classical sexual selection models are robust to a relaxation of these assumptions. When the addition of slightly more mechanistic detail (say, allowing for non-additive genetic effects, Lehmann *et al.*, 2007; Puurtinen *et al.*, 2009) strongly changes the conditions under which female choice evolves, predictions based on classical models can thus be erroneous. In the case of non-additive genetic effects, it would imply that knowledge about the genetic architecture of sexually selected characters is essential when making predictions about the strength of sexual selection in a particular group of organisms.

Assessing the robustness of classical models of sexual selection through the addition of mechanistic detail is currently only in its infancy. Chapter 2 of this thesis highlights some of the most important implicit assumptions of classical models, in combination with more recent work in which the robustness of these assumptions is evaluated. One of the conclusions of the review in chapter 2 is that adding more mechanistic detail, such as the incorporation of more complicated genetic architectures

of sexually selected traits, or allowing sexually selected characters to coevolve with other life history traits, can substantially change conclusions derived from classical models. One of the aims of this thesis is, therefore, to relax a number of assumptions of classical models of sexual selection.

One assumption of almost all models on the evolution of mate choice and display traits is that individuals are gonochoric, meaning that the sexes reside in separate individuals (but see Morgan, 1994). However, hermaphroditic organisms, in which both sexes reside within one and the same individual, also have been shown to exhibit behaviours associated to mate choice or competition over fertilizations (Michiels, 1998; Anthes, 2010; Anthes *et al.*, 2010). Moreover, hermaphroditic organisms have a number of behaviours, such as sperm-trading, sperm digestion and reciprocal insemination, of which the role in sexual selection is currently poorly understood. As a first step to assess the coevolution of choice and display characters in hermaphroditic organisms, chapter 3 extends a model of the Fisher process to hermaphroditic organisms (see also Section 1.1.3 below).

Another assumption of classical models of sexual selection is that the evolution of mate choice is typically considered in isolation of other coevolutionary processes. Sex allocation, the amount of reproductive effort invested in sons versus daughters – or in the male versus female reproductive function in hermaphrodites – is one such coevolutionary process that has been repeatedly associated with the evolution of mate choice and sexual selection in general (Trivers & Willard, 1973). A well-known verbal hypothesis is that choosy females, which are likely to mate with attractive males, are selectively favored to overproduce sons, since these sons will inherit their father's attractiveness and will therefore sire more grandoffspring than sons from unattractive males (Burley, 1981, 1986a). However, the evolution of facultative sex ratios in the context of sexual selection has been given surprisingly little attention from a formal perspective. Although a number of models have been made that assess the evolution of facultative sex ratios given a fixed degree of sexual selection (Leimar, 1996; Wade *et al.*, 2003), the coevolution of sex allocation with the evolution of female choice and male display traits is still poorly understood (Pen & Weissing, 2000c). To fill this gap, chapters 7 and 8 provide two coevolutionary models in which the interaction between sex allocation and the evolution of mate choice is explored. In addition, chapters 4-6 assess some of the mechanisms through which facultative sex allocation can be achieved, which will be further discussed in Section 1.2.

Sexual selection and hermaphroditism

Hermaphroditism refers to cases in which individual organisms express both male and female sexual functions during their lifespan. Hermaphroditism is the dominant form of plant sexuality (Barrett, 2002) and occurs in 30% of all animal species outside of the extremely species-rich, but generally non-hermaphroditic insects (Jarne & Auld, 2006; Schärer, 2009) (except for a single case of hermaphroditism in insects, resulting from transovarial transmission of sperm producing tissue: Normark, 2009; Gardner & Ross, 2011). Both sequential and simultaneous forms of hermaphroditism exist: sequential hermaphrodites first attain one sexual form and then switch to the other sex function, whereas in simultaneously hermaphroditic organisms both sexes are expressed simultaneously. In the relevant parts of this thesis (chapters 3 and 9), the main focus is on sexual selection in simultaneous hermaphrodites (hereafter:

hermaphrodites).

The predominant hypothesis for the evolutionary maintenance of hermaphroditism is that the returns from investment in one or both sex functions are diminishing (Charnov *et al.*, 1976; Charnov, 1979a; Charlesworth, 1981). In the presence of such diminishing returns, individuals stand to gain a larger number of offspring by combining both sex functions as opposed to developing as a pure sex individual (see Box 1.1 for a formal argument). Diminishing returns on investment in the female function can occur when brooding space for embryos is limited or when there is restricted time available for oviposition (Heath, 1979), the occurrence of which has only seen relatively sparse attention from sex allocation studies (see Schärer, 2009, and references therein). More attention has been devoted to diminishing returns on investment in the male function (Charnov, 1979b; Arnold, 1994), which is associated with a variety of factors, such as low population densities (Ghiselin, 1969), low individual mobilities ('sluggishness', Altenburg, 1934; Puurtinen & Kaitala, 2002), males that remove sperm of their competitors (sperm displacement, Charnov, 1979b, 1996; Pen & Weissing, 1999) or in case of plants, due to the saturation of pollen vectors (Charnov, 1979b).

As predicted in a classical paper by Charnov (1979b), the scope for the evolution of sexually selected characters that increase male fertilization success is likely to be reduced in hermaphrodites relative to gonochorists, since investment in such a character is met with diminishing returns for hermaphrodites. Indeed, this argument has often been used to explain why the evolution of characters that increase male mating success is unlikely in hermaphrodites (Greeff & Michiels, 1999a). However, there are a substantial number of examples in which investment in the male function appears to be quite large. For example, gastropods and bivalves are known to make substantial investments into their male genitalia leading to bizarre exaggerations such as in *Limax corsicus,* a species in which male genitalia spans up to 10 times a male's body length (Gerhardt, 1933). In addition, hermaphrodites are well known to have a remarkable diversity of sperm traits (e.g., Schärer *et al.*, 2011) and love darts (Schilthuizen, 2005) which are associated with the male function (e.g., Landolfa *et al.*, 2001; Chase & Blanchard, 2006), but cannot easily be reconciled with the framework described in Box 1.1 which assumes that investment in these characters are met with rapidly decelerating returns.

To explain the evolution of characters that increase male mating success in simultaneous hermaphrodites, most studies invoke the presence of evolutionary constraints (Michiels *et al.*, 2009; Anthes, 2010). Hermaphroditic invertebrates with external fertilization (e.g., Ascidians, Porifera) typically have relatively simple male and female reproductive tissues, so that the evolution towards separate-sexed individuals may occur relatively easily. In contrast, other hermaphroditic organisms exhibit highly elaborate reproductive structures (Michiels *et al.*, 2009; Anthes, 2010) and more complex developmental pathways that code for sex-specific tissues (Schärer, 2009). As a result, evolution towards separate sexes requires overcoming a substantial amount of reproductive and developmental constraints, so that hermaphroditism in the presence of sexually selected traits might be maintained as an evolutionary one-way street (Bull & Charnov, 1985; Michiels *et al.*, 2009).

Box 1.1: The evolutionary maintenance of hermaphroditism

The evolutionary maintenance of hermaphroditism has been studied by several models, most notably that of Charnov *et al.* (1976). The model relies on arguments from evolutionary game theory (see Section 2.2.3 of this thesis), by tracking the fate of a rare mutant with sex allocation strategy s in an otherwise monomorphic population playing strategy $\bar{s}$. Investment in one sex function is assumed to trade off linearly with investment in the other sex function, so that s and $1-s$ represent the investment in the male and female reproductive functions respectively. I assume that the resulting female fertility $f(s)$ is a linear function of investment in the female function, $f(s) = 1 - s$. Male fertility $m(s)$ instead is assumed to be a nonlinear function of investment in the male function, $m(s) = s^{\alpha}$. In the absence of selfing, fitness of a rare mutant $W_{\mathrm{h}}(s,\bar{s})$ with sex allocation strategy s in a population with strategy $\bar{s}$ is then represented by

$$W_{\mathrm{h}}(s,\bar{s}) = \frac{1}{2W_{\mathrm{tot}}(\bar{s})}\left[f(s) + \frac{m(s)}{m(\bar{s})}f(\bar{s})\right]. \tag{1.1}$$

In other words, the mutant's fitness is proportional to the sum of returns through the female function $f(s)$ and the male function, which is a function of the mutant's male fertility relative to the fertility of all other males $m(s)/m(\bar{s})$ times the available number of eggs to fertilize $f(\bar{s})$. To ensure that the population is stable, a mutant's fitness is taken relative to total fitness of the rest of the population $W_{\mathrm{tot}}(\bar{s})$ (i.e., density dependence) (Mylius & Diekmann, 1995). The scalar $1/2$ avoids double counting of offspring (once via its mother, once via its father). By noting that $W_{\mathrm{tot}}(\bar{s}) = f(\bar{s}) = m(\bar{s})$ (total male and female fitness is necessarily equal, since each individual has one mother and one father), one obtains the classical Shaw-Mohler equation (Shaw & Mohler, 1953)

$$W_{\mathrm{h}}(s,\bar{s}) = \frac{1}{2}\left[\frac{f(s)}{f(\bar{s})} + \frac{m(s)}{m(\bar{s})}\right]. \tag{1.2}$$

The equilibrium sex allocation strategy $s^{\star}$ is then found by solving for $\mathrm{d}W_{\mathrm{h}}(s,\bar{s})/\mathrm{d}s|_{s=\bar{s}=s^{*}} = 0$, which yields

$$s^{\star} = \alpha/(1+\alpha) \tag{1.3}$$

To find out if the equilibrium $s^{\star}$ is indeed a stable endpoint of evolution, we can use stability concepts developed in the realm of adaptive dynamics (Geritz *et al.*, 1998) which describe the course of evolution near equilibrium. Figure B1.1A,C shows that when $\alpha > 1$, hermaphroditism is unstable and evolution will proceed towards a population with pure sexes. In contrast, Figure B1.1B,D shows that when $\alpha < 1$, hermaphroditism is evolutionarily maintained. *continued on the next page*

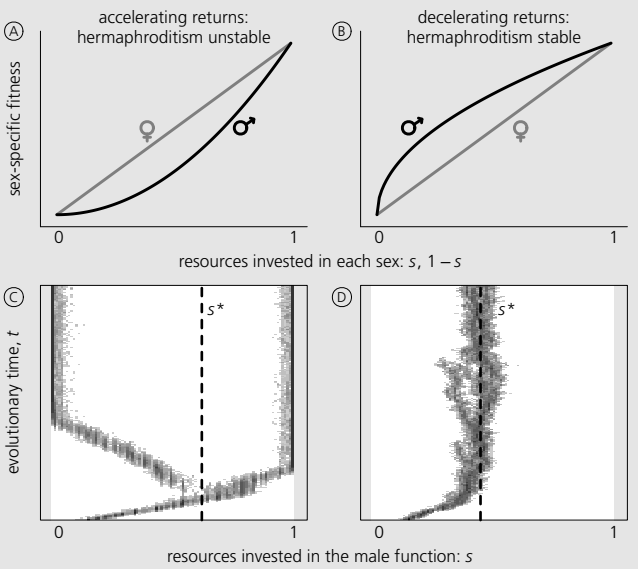

Figure B1.1: Sex allocation and the evolution of gonochorists and hermaphrodites. Whenever returns on investment in both sexes is accelerating ($\alpha > 1$), the evolution of separate sexes ensues (panels A,C). In contrast, when returns on one or both sexes is decelerating ($\alpha < 1$), evolution leads towards stable hermaphroditism (panels B,D). It can be easily shown that for both cases, populations with sex allocation strategies s will evolve towards the point s^*, which indicates that s^* is convergence stable. The condition for convergence stability of s^* is given by $(\partial/\partial s^*)(\partial W_h(s,\bar{s})/\partial s|_{s=\bar{s}=s^*}) < 0$, which holds regardless of the value of α. In addition, the equilibrium value s^* may be prone to invasion by mutants that have sex allocation strategies lower and higher than s^*. s^* is so-called evolutionarily stable when it is immune to invasion of such mutants (Geritz *et al.*, 1998), which is the case when $\partial^2 W_h(s,\bar{s})/\partial s^2|_{s=\bar{s}=s^*} < 0$. When s^* is both convergence and evolutionarily stable, stable hermaphroditism evolves (panels B,D). When s^* is convergence, but not evolutionarily stable (panels A,C), evolutionary branching of s occurs and evolution proceeds towards a population with separate sexes (panel C).

Assuming that such constraints on sex allocation are indeed present, chapter 3 of this thesis compares the evolution of mate choice characters and display traits between gonochorists and hermaphrodites, in the context of Fisherian sexual selection. Keeping sex allocation fixed, the model focuses on a number of other important differences between gonochorists and hermaphrodites: first, since hermaphroditic individuals express both sexes, any costs of display traits or preferences are incurred by all members of a population. In contrast, display traits and preferences in gonochorists are typically associated with male and female functions respectively, so that costs of expressing such traits are only incurred by members of the corresponding sex. Another important difference is that mutual choice is thought to be much more prevalent in hermaphrodites than in gonochorists (Charnov, 1979b; Vreys & Michiels, 1997; Michiels, 1998). The evolution of mutual choice in the context of the Fisher process has received some attention in gonochorists (Servedio & Lande, 2006; Servedio,

2007), but these models are yet to be extended to hermaphroditic organisms. Lastly, hermaphrodites also exhibit behaviours such as reciprocal insemination, which have not yet been considered in the context of sexual selection. Chapter 3 therefore aims to extend a model of Fisherian sexual selection to incorporate these aspects of certain hermaphrodites, and to assess if current insights on sexual selection are robust to hermaphroditic details.

1.2 Sexual selection and sex allocation

The previous section already briefly highlighted the importance of sex allocation theory in the maintenance of hermaphroditism, potentially limiting the evolution of sexually selected characters in hermaphrodites. Another link between sexual selection and sex allocation has been postulated in the context of condition-dependent sex allocation. Condition-dependent sex allocation refers to cases in which the investment in one sex versus the other depends on an environmental variable (Charnov & Bull, 1977; Bull, 1981b; Van Dooren & Leimar, 2003), such as temperature (Valenzuela & Lance, 2004) or population density (e.g., Aparici *et al.*, 1998). In a classical paper, Trivers & Willard (1973) postulated that in polygynous species where the most successful males achieve a disproportionate share of matings, a son in good condition can be expected to secure more mates and hence produce more offspring than a daughter in a similar condition. In case condition is passed on from mother to offspring, mothers should thus overproduce sons when in good condition, while producing daughters in poor condition (see Box 1.2). Ever since the formulation of the Trivers-Willard hypothesis, finding such facultative sex ratio adjustments has received a substantial amount of attention (see Hewison & Gaillard, 1999; Sheldon & West, 2004; West, 2009, for reviews)

Although the Trivers-Willard hypothesis was formulated in the context of sex allocation based on maternal condition in polygynous mammals, its logic applies more generally to other cases of condition-dependent sex allocation (reviewed in West, 2009, chapters 6,7), such as sex allocation based on host size in parasitoid wasps (Charnov *et al.*, 1981) or sex allocation based on environmental factors including temperature or population density (Charnov & Bull, 1977; Valenzuela & Lance, 2004). One influential version of the Trivers-Willard hypothesis that is further studied in this thesis is condition-dependent sex allocation based on mate attractiveness. Based on a classical experiment on sex ratio manipulation in zebra finches (*Taeniopygia guttata*) Burley (1981, 1986a) was the first to suggest that females mated to an attractive male should produce more sons in comparison to females mated to unattractive males. If male attractiveness is heritable, sons from these attractive fathers will be attractive themselves and thus secure a larger share of matings than sons from unattractive fathers. Hence, mothers would be selected to facultatively adjust the brood sex ratio, dependent on the attractiveness of their mate.

To demonstrate the existence of sex allocation based on male attractiveness, the experiment by Burley (1981) (see also Burley *et al.*, 1982; Burley, 1986a) involved manipulations of male attractiveness through colored leg bands, showing that females mated with the most attractive males produced more sons. Burley's studies sparked a great deal of attention and were quickly criticized (e.g., Immelmann *et al.*, 1982; Thissen & Martin, 1982, see Seguin & Forstmeier (2012) for a recent meta-analysis that criticizes the effect of leg band coloration on male attractiveness). Since then,

numerous studies have attempted to demonstrate the existence of sex allocation dependent on male attractiveness, focusing mainly on birds. Results from these studies are mixed (see Table 6.3 in West, 2009 and references therein), with some studies demonstrating sex ratio biases based on male attractiveness (e.g., Ellegren *et al.*, 1996; Sheldon *et al.*, 1999; Pike & Petrie, 2005), whereas others have failed to find any relationship between male attractiveness and the sex ratio (e.g., Saino *et al.*, 1999; Parker & Winker, 2005; Postma *et al.*, 2011). In general, studies of sex ratio adjustment in birds are rather notorious for their inconsistency, which is illustrated by a series of experiments that aimed to replicate Burley's study in Zebra Finches. Although some of these studies indeed found a pattern of sex allocation (Burley, 1986a), other studies found no sex ratio biases at all (Zann & Runciman, 2003), a sex ratio pattern opposite to that of Burley (1981) (Rutstein *et al.*, 2004) or only partial support of Burley's results (von Engelhardt *et al.*, 2004).

To make sense of these inconsistent results, it is essential to note that the predictions made by Burley (1981) were entirely formulated in verbal terms. As noted before, it is a challenging task to make sound verbal predictions in the context of sexual selection, since it is a multidimensional process that relies on a complex interdependence between male and female traits (Kokko *et al.*, 2006). Given that the evolution of sex allocation would add even more dimensions to this problem, any accurate prediction demands a formal assessment. Notwithstanding a number of analyses that focus on cases where sex allocation is dependent on maternal condition in polygynous animals (Leimar, 1996; Matessi & Saino, 2003; Wade *et al.*, 2003), only a single study has formally addressed the evolution of sex allocation based on paternal attractiveness (Pen & Weissing, 2000c). Using a reproductive value approach, Pen & Weissing (2000c) found that when selection is driven by a good-genes process, females mated to high quality males indeed bias their sex ratio towards sons, but this bias is relatively weak. In contrast, females mated to low quality males should exhibit a much larger sex ratio bias towards daughters, to avoid producing unattractive sons that will incur no fitness. In contrast, when female choice is driven by the Fisher process, biased sex allocation is predicted to be absent at equilibrium, since the increased mating success of attractive sons is exactly cancelled by their costs of bearing ornamentation.

The predictions by Pen & Weissing (2000c) show that sex allocation based on paternal attractiveness may be a more subtle process than previously anticipated, dependent on the particular type of sexual selection at work. However, the analysis by Pen & Weissing (2000c) itself also makes a number of simplifying assumptions that require further attention. First, their analysis only deals with two discrete types of male attractiveness, whereas in reality male attractiveness can be expected to attain a continuum of different values. Second, they assumed that female preferences are allowed to evolve in freedom of any costs, whereas it is well-known that even a slight cost of a female preference considerably alters the dynamics of the sexual selection process (Pomiankowski, 1987a; Bulmer, 1989, Section 2.3.3 of this thesis). Third, Pen & Weissing (2000c) concentrated on an analysis of evolutionary equilibria, whereas the course of evolution towards these equilibria is yet to be assessed.

To arrive at a comprehensive analysis incorporating these aforementioned aspects, chapter 7 uses individual-based simulations to assess the evolution of maternal sex-ratio manipulation in the presence of different forms of sexual selection. Chapter 8 takes the analysis of sex allocation in the context of sexual selection a step further, by focusing on the coevolutionary interaction between sex allocation and sexual selec-

tion. Although conventional studies often assess the evolution of sex allocation and the evolution of sexually selected characters in isolation, more inclusive coevolutionary analyses are essential to predict if both processes are indeed likely to co-occur. An important conclusion of chapter 8 is that allowing for this coevolutionary feedback gives rise to unexpected outcomes that defy well-established insights on sex allocation based on paternal attractiveness. Hence, this thesis shows that more inclusive models that incorporate sexual selection in combination with other important evolutionary processes (e.g., speciation, parental care, sex allocation) are likely to arrive at different conclusions, when compared to classical models that study sexual selection in isolation.

1.3 Condition-dependent sex allocation: mechanistic constraints

Next to complexities that arise from coevolutionary interactions between sexual selection and sex allocation, results on facultative sex ratios may be difficult to interpret for other reasons. One aspect that deserves further attention is constraints in the sex determining system that restrict the flexible adjustment of sex ratios. It is widely thought that facultative sex ratios are difficult to achieve in those species where the offspring's sex is determined by sex chromosomes (Williams, 1979; Bull & Charnov, 1988; Krackow, 2002, but see West *et al.*, 2005). In contrast, facultative sex ratios might be more easily achieved in the presence of arrhenotokous haplodiploidy, where egg fertilization determines offspring sex (with fertilized eggs developing as females, and unfertilized eggs as males). Given that females are able to fertilize eggs in a state dependent manner, flexible sex ratio adjustment may be a likely outcome. Alternatively, when the state variable of interest directly influences the molecular sex determining cascade, as has been demonstrated in studies of environment-dependent sex determination (ESD) (Sarre *et al.*, 2004; Quinn *et al.*, 2007, 2011), facultative sex ratios will be a logical outcome.

Although haplodiploidy and ESD may thus both facilitate condition dependent sex allocation, the evolution of these systems themselves is still far from understood (Shine, 1999; Normark, 2003). Part II of this thesis therefore explores a number of aspects that are likely to have played a role in the evolution of these mechanisms.

The evolution of haplodiploidy

The rules of genetic inheritance are not universal (Normark, 2003): the organisation of an organism's genetic material (i.e., genetic system) shows considerable variation among species (White, 1973; Normark, 2009). For example, the number and sex-specific inheritance of chromosomes varies between species, as does the the degree of sex-differences in ploidy (i.e., haplodiploidy versus diplodiploidy) or the presence of sexual versus asexual reproduction. The genetic system is a fundamental part of an the organism's genomic architecture (Lynch, 2007) and plays a role in aspects such as sex determination (Bull, 1983; Uller *et al.*, 2007) and the potential for asexual reproduction (Mogie, 1992; Judson & Normark, 1996). Since genetic systems exhibit a surprising amount of variation between closely related species (Normark, 2003), it has fueled the idea that genetic conflicts of interest (see Box 1.3) may have a played an important role in shaping variation in genetic systems (Haig, 1993; Hamilton, 1993; Burt & Trivers, 2006; Ross *et al.*, 2010).

Box 1.2: The evolution of condition-dependent sex allocation and the Trivers-Willard hypothesis

Düsing (1883, 1884) and Fisher (1930) were the first to show that negative frequency dependent selection favors parental investment in the rarer sex, explaining the common observation that parents invest roughly equal amounts of resources in sons versus daughters. However, the Düsing-Fisher model does not explain observations where sex allocation patterns are dependent on an individual's condition or the environment (see West, 2009 chapter 6 and references therein). To explain such cases of condition-dependent sex allocation, Trivers & Willard (1973) relaxed a crucial assumption implicit in the Fisher-Düsing model, namely that returns on investment should be invariant with respect to maternal state (Bull & Charnov, 1988). Instead, Trivers & Willard (1973) postulated that returns on investment in each sex may well vary with maternal condition in polygynous contexts. Given that mothers in a good condition produce offspring of higher quality than mothers in bad condition, sons stand to gain more from increased quality than daughters in polygynous contexts: their quality allows those sons to achieve a higher mating rate, whereas daughters of a similar quality experience only a limited increase in fecundity. Figure B1.2A shows a graph of such a supposed relationship between maternal condition and fitness accrued through sons and daughters respectively.

A reproductive value approach (Leimar, 1996; Taylor, 1996a) can be used to demonstrate that the verbal hypothesis by Trivers & Willard, 1973 indeed explains the evolution of condition dependent sex allocation (Charnov, 1979a; Bull, 1981b; Wild & West, 2007). Assume that mothers can be in two possible conditions (0 or 1) with respective probabilities $1-p$ and p. Maternal condition has no effect on the fitness of daughters, but sons born from 0-type and 1-type mothers have relative mating rates r and 1 respectively ($0 \leq r < 1$).

Assuming non-overlapping generations (see Charnov & Dawson, 1989; Schwanz *et al.*, 2006, for the case of overlapping generations), we can then find an expression for invasion fitness $W(\mathbf{s}, \bar{\mathbf{s}})$ of a rare mutant playing sex allocation strategy $\mathbf{s} = (s_0, s_1)$ in a resident population playing sex allocation strategy $\bar{\mathbf{s}} = (\bar{s}_0, \bar{s}_1)$ (see chapter 6). From this expression of invasion fitness, we can derive the evolutionarily stable sex allocation strategies. Figure B2.1B displays the main result from such a formal implementation of the Trivers-Willard hypothesis: mothers in a good condition will always produce a larger proportion of sons than mothers in a poor condition ($s_1 > s_0$) whenever a sons mating rate varies with maternal condition.

Figure B1.2A emphasizes a general point that underlies the Trivers-Willard hypothesis, namely that condition-dependent sex allocation is expected to evolve whenever returns on investment in males versus females varies reliably with a particular state variable. Chapters 7 and 8 in this thesis investigate if such a pattern of condition-dependent sex allocation is also expected to evolve whenever the state variable is not maternal condition, but the heritable attractiveness of a mother's mating partner.

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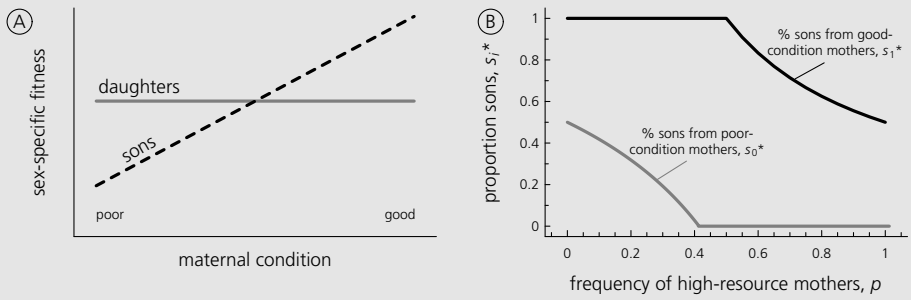

Figure B1.2: Panel A: the Trivers Willard hypothesis postulates that fitness accrued through sons is positively affected by improvements in maternal condition (black dashed lines), whereas fitness accrued through daughters is relatively insensitive to maternal condition (grey solid line). Panel B: A formal implementation of the Trivers-Willard hypothesis by Charnov & Bull (1977) (see also Charnov, 1979a; Bull, 1981b) indeed shows that poor condition mothers should overproduce daughters (grey lines), whereas mothers in good condition should overproduce sons (black lines). Only when variation in maternal state is absent ($p = 0$ or $p = 1$) do sex allocation strategies converge to equality as under the Düsing-Fisher model. Parameters: $r = 0.5$.

Haplodiploidy is one example of a genetic system, characterized by sperm that only transmit maternally inherited chromosomes. It has evolved independently approximately 20 times (Normark, 2003) and is well-known from insect groups such as Hymenoptera (bees, wasps and ants) and Thysanoptera (thrips). In these groups, most species exhibit a form of arrhenetokous haplodiploidy, in which fertilized, diploid eggs develop generally as females, whereas unfertilized, haploid eggs develop as males. However, other forms of haplodiploid have also been described, such as paternal genome elimination (PGE) that occurs, among others, in sciarid flies (Gutzeit *et al.*, 1985; Ribeiro & Perondini, 1991) and scale insects (Nur, 1980; Ross *et al.*, 2010). PGE or pseudoarrhenotoky is characterized by males that begin development as diploid zygotes, but do not pass on their paternally inherited chromosomes. It is often considered to be an ancestral form of arrhenotokous haplodiploidy (Cruickshank & Thomas, 1999; Ross *et al.*, 2010).

Several hypotheses have been set forth to explain the evolution of haplodiploidy and PGE (see chapter 4), such as the advantage to mothers that sons exclusively transmit their maternally inherited genes (Brown, 1964), the opportunity to purge deleterious recessive mutations through haploid males (Goldstein, 1994) or the possibility for mothers to control their sex ratio (Borgia, 1980; Sabelis *et al.*, 2002). However, most of these hypotheses would hold true in a wide range of contexts, which begs the question why haplodiploidy is not more commonly observed. To this end, a more recent meta-analysis on genetic systems in insects pointed out a more specific set of ecological factors that may facilitate the evolution of haplodiploidy (Normark, 2003). For example, most ancestors to haplodiploid clades are inferred to have been infected with endosymbionts, which are parasitic or mutualistic micro-organisms that reside in the cytoplasm and are often exclusively transmitted through the matriline. In addition, most haplodiploid organisms have gregarious broods which give rise to an

increased scope for kin competition. The presence of such endosymbionts often leads to genetic conflicts over the sex ratio (see Box 1.3): due to their matrilineal inheritance, endosymbionts have no interest in ending up in males and favor a strongly female-biased sex ratio, whereas genes residing in the nucleus often favor more even sex ratios (Werren & Beukeboom, 1998). More evidence is accumulating that such sex ratio conflicts shape the evolution of sex determination systems (see Cordaux *et al.*, 2011, and references therein)

The combined presence of endosymbionts and gregarious broods suggests the prevalence of a particular type of endosymbiont, so called male-killers (Hurst, 1991). Male-killing endosymbionts specifically rely on gregarious broods with competing kin to increase their inheritance through the matriline (Randerson *et al.*, 2000). This is because male-killing endosymbionts commit suicide when present in males, thereby killing their male hosts, which frees up resources that enhance the survival prospects of their sisters, which carry clonal copies of the endosymbiont. One efficient hypothetical mechanism with which male-killers may detect and kill their male hosts is by detecting and eliminating incoming Y-chromosomes: this guarantees detecting males only and the lack of the Y chromosome is likely to render males inviable. Subsequent coevolution between the endosymbiont and autosomal genes would then proceed in a number of steps: when the Y chromosome is eliminated from the population, strongly female biased sex ratios select for the evolution of a novel male-determining 'neo Y' chromosome (Charlesworth *et al.*, 2005), since rare males will have a high reproductive value. In turn, male-killers would then start to detect and eliminate this neo Y chromosome, following the invasion by another neo Y chromosome, until no chromosomes are left and males are rendered haploid.

A number of subsequent models have aimed to explore if this coevolution between host and male-killing endosymbionts could indeed lead to the evolution of haplodiploidy (Normark, 2004a; Engelstädter & Hurst, 2006; Ubeda & Normark, 2006). However, as we show in chapter 4, these studies did not properly take into account the coevolutionary dynamics between hosts and endosymbionts. For example, Engelstädter & Hurst (2006) assumed that male-killing endosymbionts are able to persist in a population, even when all males had evolved some means to survive haploidization (e.g., through enhanced dosage compensation). However, when all males survive, copies of the endosymbiont present in their sisters will not accrue any additional resources from their brothers, leading to endosymbiont extinction.

To fill this gap, chapter 4 provides a more thorough assessment of the coevolutionary dynamics of haploidizing, male-killing endosymbionts and their hosts. By using a demographical kin selection model (Taylor & Frank, 1996; Taylor *et al.*, 2007; Lion *et al.*, 2011) and individual-based simulations, we show that only those spatially structured populations that have a very low degree of dispersal and few individuals per deme allow for the successful maintenance of male-killing endosymbionts and viable haploidized males. This is because under these conditions, a female-biased sex ratio is evolutionarily favored due to local mate competition (Hamilton, 1967). Male-killing haploidizing endosymbionts in combination with partially viable males then provide a means of skewing the sex ratio towards daughters, while the deaths of superfluous sons maintain the male-killing endosymbiont. Alternatively, when male-killing endosymbionts are not entirely parasitic on their hosts, but provide some additional benefits to their host (Frank, 1997), for example by enabling their host to digest resources such as cellulose, haplodiploidy is maintained under a broader set

of conditions. These studies therefore provide a more specific theory for the evolution of haplodiploidy that takes account of the intricacies involved in the coevolution between endosymbionts and their hosts.

CSD can exist in both single and multilocus forms: sex determination in single-locus CSD (*sl*-CSD) is based on the homozygosity of a single sex determining locus and is considered to be the ancestral form of CSD (Van Wilgenburg *et al.*, 2006; Heimpel & De Boer, 2008). Multi-locus CSD (*ml*-CSD) is thought to have evolved from *sl*-CSD through gene duplication and substantially reduces the cost of inbreeding, since diploid male development only ensues when all sex loci are homozygous (Crozier, 1971). Little is known about the relative preponderance of *sl*-CSD and *ml*-CSD, whereas the presence of these different forms of sex determination may tell us a lot about the sensitivity of Hymenopteran taxa to inbreeding depression. This is particularly relevant for wasp populations that are used for biological control to reduce crop damage by butterfly host species, and which may endure population bottlenecks after introduction.

Sex determination in haplodiploid insects

The notion that arrhenotokous haplodiploid organisms produce daughters and sons from fertilized diploid and unfertilized haploid eggs respectively, begs the question how these differences in ploidy lead to a molecular signal that induces the molecular sex determining pathway to start female versus male sexual development. In Hymenoptera, two sex determining mechanisms have now been well characterized: the first system relies on a maternal effect to induce female development and has so far been found exclusively in the insect model system *Nasonia* (Beukeboom *et al.*, 2007; Verhulst *et al.*, 2010a). Another sex determining mechanism that seems to be more widespread across the Hymenoptera is complimentary sex determination (CSD) (Beye *et al.*, 2003; Van Wilgenburg *et al.*, 2006; Heimpel & De Boer, 2008), in which sex is determined by homozygosity at one or more sex loci: unfertilized, haploid eggs are hemizygous at all their sex loci and develop as males. Fertilized, diploid eggs are usually heterozygous for at least one sex locus and therefore develop as females. Only when diploid individuals are completely homozygous for all sex loci, which may occur under conditions of inbreeding, male development ensues. Often, such diploid males are inviable or sterile, representing a form of considerable inbreeding depression with potential negative consequences to population dynamics of taxa with CSD (Zayed & Packer, 2005). In chapter 5, we therefore use an inbreeding experiment to assess the occurrence of *sl*-CSD versus *ml*-CSD in a haplodiploid wasp, *Cotesia rubecula*. Given a successive number of inbred generations, homozygosity at the sex loci should increase, so that more and more diploid males should be produced. The exact increase in the production of diploid males with progressive generations of inbreeding depends, however, on the number of CSD-loci as well as the survival of diploid males. We estimate the number of CSD loci and male survival that best fit the data by running a series of individual-based simulations which mimic the experiment, but in which these two parameters are varied. In comparison to analytical models (e.g., Cook, 1993), individual-based simulations are more realistic, since they take account of a finite population size and also allow for demographic stochasticity during the experiment. The model presented in chapter 5 supports the prediction that *Cotesia rubecula* has *ml*-CSD consisting of two loci and relatively high values of diploid male

Box 1.3: Genetic conflicts

Genetic conflicts occur whenever different genetic elements within a genome have divergent evolutionary optima (Burt & Trivers, 2006). There are two basic types of genetic conflict (e.g., Rice, 1998 and Figure B1.3):

- **Intragenomic conflict** occurs when different gene loci (say A and B) residing within the same individual have divergent fitness optima (θ_A and θ_B respectively) (see Figure B1.3, panel A). A well known example is the conflict between cytoplasmic and nuclear genes over sex allocation, in which maternally inherited cytoplasmic genes have no interest in the production of males, in contrast to autosomal genes that typically favor investment in both males and females.

- **Intergenomic conflict** (Figure B1.3, panel B) occurs among genetic elements that are present in different individuals, where often a distinction is made between intralocus and interlocus conflicts:

  - *Intralocus conflict*: copies of the same allele (say A and A') that are present in different individuals experience divergent selective optima. Sexual antagonism is by far the best known example of an intralocus genomic conflict, in which an allele experiences divergent selective optima, dependent on the sex in which it is expressed (Van Doorn, 2009; Bonduriansky & Chenoweth, 2009).
  - *Interlocus genetic conflict*: conflicts occur among different gene loci (say A and B) expressed in different individuals, that contribute to a shared phenotype C. For example, selection on parental and offspring characters in the context of parent-offspring conflict can often be classified as an intergenomic conflict: gene loci in parents favor the equal distribution of care C over their offspring (reflected by gene locus A), whereas offspring are selectively favored to direct more care C' to themselves, at the expense of their siblings. As a result, offspring may express traits (reflected by gene locus B) such as begging (Wright & Leonard, 2002) or release hormones in the maternal bloodstream (Crespi & Semeniuk, 2004) that manipulate its parent to provide it with more care, closer to the offspring's optimum $\theta_{C'}$ (e.g., Trivers, 1974; Mock & Parker, 1997; Kuijper & Johnstone, 2012).

  Genetic conflicts are interesting from an evolutionary perspective, since they can give rise to coevolutionary arms races, where gene substitutions at antagonistically interacting loci lead to a series of adaptations and counteradaptions (Rice, 1998; Harris *et al.*, 2008; Werren, 2011). Consequently, the presence of genetic conflicts may cause closely related species or populations to diverge, so that genetic conflicts may both affect our understanding of biodiversity (e.g., Turelli & Hoffmann, 1991; Rice *et al.*, 2005; Martin & Hosken, 2004), but also be part of the explanation why so much heritable variation is maintained especially for those traits that have a close relationship to fitness (Harris *et al.*, 2008).

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Box 1.3 continued
The current thesis deals with genetic conflicts in both the context of sexual selection and sex allocation. Parts I and III of this thesis deal with intersexual selection, a process that starts from the premise that male and female characters are under divergent selection pressures, since females (or the female function in hermaphrodites, see chapter 3) are typically considered to be limited by resources to produce ova, whereas males are considered to be limited by the number of female mating partners (Bateman, 1948). As a result, male and female selective optima regarding mating traits often do not coincide (Parker, 1979; Rice, 1996; Arnqvist & Rowe, 2005). Divergent selective optima are particularly apparent in models on interlocus sexual conflict, in which harming and resistance behaviours coevolve (chapter 9). Next to that, part II of this thesis focuses on two genetic conflicts over sex allocation: in chapter 6, I focus on parent-offspring conflict over condition-dependent sex allocation, including an explicit coevolutionary scenario of interlocus parent-offspring conflict in which a novel genetic sex factor is selectively favored by offspring, leading to counteradaptations of condition-dependent sex allocation factors in parents. In chapter 4, I focus on a scenario of intragenomic conflict between cytoplasmic male-killing endosymbionts and autosomal genes.

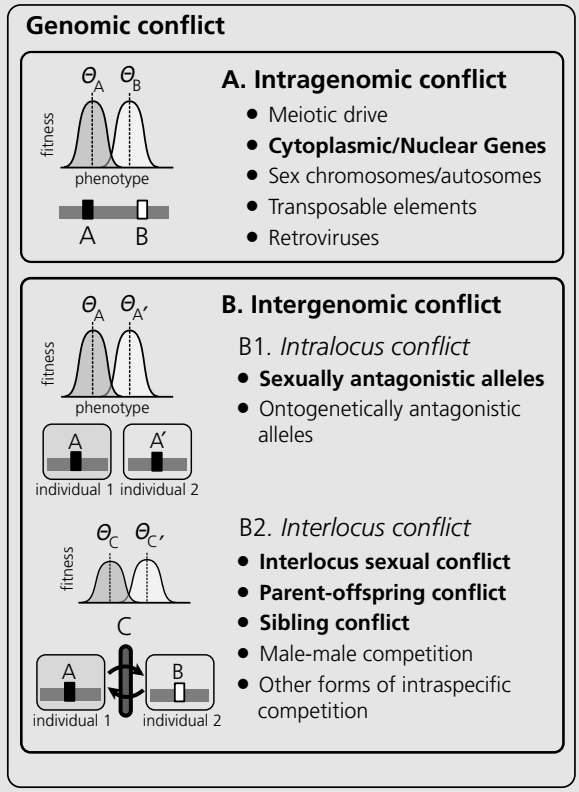

Figure B1.3 A schematic classification of some well studied forms of genetic conflict. Items in bold refer to cases which will be studied in this thesis.

survival. Inbreeding experiments such as these in combination with individual-based simulations are a straightforward approach to assess the presence of CSD in other haplodiploid taxa (e.g., Ma *et al.*, 2012, submitted manuscript).

Parent-offspring conflict over condition-dependent sex allocation

The previous sections already highlighted the role of genetic conflicts (see Box 1.3) in inducing transitions between different genetic systems. In addition, genetic conflicts of interest relating to the optimal sex ratio may also give rise to transitions in sex determination systems (Werren & Beukeboom, 1998; Uller *et al.*, 2007). Chapter 6 of this thesis aims to merge genetic conflicts with a model of condition-dependent sex allocation, by investigating a model of parent-offspring conflict over the sex ratio in the context of the Trivers-Willard hypothesis.

One scenario in which parent-offspring conflict over the sex ratio may occur is when one sex is more costly to produce than the other, so that under the equal allocation principle (Düsing, 1883; Fisher, 1930) mothers favor a sex ratio that is biased towards the cheaper sex. However, since offspring are more related to themselves than to their siblings, offspring will be selectively favored to develop themselves more often as a member rarer sex, since the rarer sex has a higher reproductive value (Werren & Hatcher, 2000; Werren *et al.*, 2002; Pen, 2006). Werren *et al.* (2002) and Kozielska (2008) have shown that when the optimal sex ratio differs between parents and offspring, rapid evolutionary transitions may occur between chromosomal sex determining systems, so that ZZ-ZW chromosome systems are replaced by XX-XY systems or vice versa. Such rapid switches in genetic systems resemble the rapid phylogenetic transitions in sex determining systems that are observed in certain insects (Dubendorfer *et al.*, 2002; Heimpel & De Boer, 2008) and vertebrates, such as teleost fish (Mank *et al.*, 2006) or tetrapods (Kraak & Pen, 2002).

However, phylogenetic studies of sex determining systems in vertebrates also show the preponderance of environmental sex determination (ESD), with numerous transitions from ESD to chromosomal sex determining systems (Janzen & Phillips, 2006; Pokorná & Kratochvíl, 2009). Chapter 6 shows that parent-offspring conflict can explain evolutionary transitions between ESD and chromosomal sex determination in a simple binary environment, but only under particular environmental conditions where one of both environments is much more common than the other. In other cases, ESD may in fact be robust to invasion by alternative sex determining mechanisms, either by resisting invasion altogether or by incorporating the invading genetic sex factors in a system of ESD. One important conclusion of Chapter 6 is, therefore, that different forms of environmental variation cannot be ignored when making inferences about phylogenetic transitions between different sex determining mechanisms.

1.4 Thesis overview

Mate choice is a highly intricate process, which has been the topic of a large number of models. **Chapter 2** of this thesis aims to review these intricacies involved in modeling sexually selected traits such as mate choice. One important general conclusion of this review is that well-established insights from sexual selection theory are generally not robust to the addition of more mechanistic detail, be it aspects like genetic details, the particular social context in which sexual selection occurs or the coevolution of sexually selected traits with other life history traits, such as sex allocation.

The sensitivity of the sexual selection process to mechanistic detail is an aspect that returns in **Chapter 3**. The well-established notion that sexual selection is weaker in hermaphrodites than in gonochorists is strongly contingent on the intricacies of the mate choice process: although unilateral choice is indeed weaker in hermaphrodites than in gonochorists, mutual choice leads to equally strong sexual selection in hermaphrodites and gonochorists alike.

A similar sensitivity to mechanistic details is the recurring theme in the second part of this thesis, which involves models on the evolution of sex allocation and sex determining systems. **Chapter 4** explores under which conditions coevolution between hosts and male-killing endosymbionts is likely to have led to the evolution of a haplodiploid genetic system. Genetic conflicts are commonly associated with rapid transitions between genetic systems, but the analysis shows that a transition from diplodiploidy to haplodiploidy is highly contingent on the type of symbiosis between host and symbiont: when endosymbionts are entirely parasitic and are not beneficial to their host, haplodiploidy only evolves when offspring compete only with their direct siblings. Haplodiploidy will only evolve under more relaxed ecological conditions when endosymbionts also provide certain beneficial functions to their host. Staying within the context of haplodiploidy, **Chapter 5** focuses on complementary sex determination, which is the sex determining mechanism that is often found in haplodiploid organisms. It shows that individual-based simulations are a versatile tool to assess the results of inbreeding experiments, aimed to unravel how many loci underly complimentary sex determination. Mechanistic constraints return in **Chapter 6**, when we focus on another genetic conflict, that between parent and offspring, in the context of facultative sex ratios. Since offspring favor different optimal sex ratios, this leaves a maternally controlled system of facultative sex ratio control prone to invasion by genetic sex determining systems. As a result, we can encounter situations in which the selective requirements for facultative sex ratios are met, but in which genetic conflicts bring up mechanistic constraints that refrain populations from adaptation.

In the last part of this thesis, two models are presented that combine insights on the evolution of mate choice with that of sex allocation. **Chapter 7** focuses on the evolution of facultative sex ratios based on partner attractiveness, when partner attractiveness is either the result of evolving mate preferences in the context of Fisherian or good-genes sexual selection. **Chapter 8** focuses on the coevolutionary feedback between maternal sex allocation based on partner quality.

CHAPTER 2

A guide to sexual selection theory

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Abstract

Mathematical models have played an important role in the development of sexual selection theory. These models come in different flavors and they differ in their assumptions, often in a subtle way. Similar questions can be addressed by modeling frameworks from population genetics, quantitative genetics, evolutionary game theory, adaptive dynamics or by individual- based simulations. Confronted with such diversity, non-specialists may have difficulties to judge the scope and limitations of the various approaches. Here we review the major modeling frameworks, highlighting their pros and cons when applied to different research questions. We also discuss recent developments, where classical models are enriched by including more detail regarding genetics, behavior, demography and population dynamics. It turns out that some seemingly well-established conclusions of sexual selection theory are less general than previously thought. Linking sexual selection to other processes like sex ratio evolution or speciation also reveals that enriching the theory can lead to surprising new insights.

2.1 Introduction

Sexual selection is the process by which individuals compete for access to mates and fertilization opportunities (Andersson, 1994; Jones & Ratterman, 2009). Darwin (1871) developed the concept of sexual selection to explain the evolution of exaggerated and flamboyant characters such as calls, odors, ornaments and conspicuous behaviors that are present in one sex only and cannot be easily explained as adaptations to the ecological conditions of a species. Darwin was well aware of the complex nature of sexual selection, "depending as it does, on the ardour of love, the courage, and the rivalry of the males, as well as on the powers of perception, the taste, and will of the female" (Darwin, 1871, pp. 296). Due to this interdependence of coevolving male and female traits, it is inherently difficult to capture the essential features of sexual selection in verbal theories. Yet it took more than a century after Darwin's seminal work before students of sexual selection started to develop mathematical models, allowing to capture the complexity of sexual selection in a rigorous fashion. Driven by these models, the empirical study of sexual selection has matured into one of the most active fields in evolutionary biology (Andersson & Simmons, 2006).

There are numerous reasons why sexual selection models tend to be more complicated than "standard" models of natural selection. First, whereas models of natural selection often make the simplifying assumption of asexual reproduction or random mating, sexual reproduction and non-random mating lie at the heart of sexual selection. Second, natural selection models tend to avoid the intricacies of multi-locus genetics. In contrast, sexual selection models are intrinsically multivariate since they reflect the coevolution of mating preferences, ornaments, and in the case of the "good-genes" process also variation in genetic quality. Moreover, the associations (linkage disequilibria) between traits or between preferences and traits are often crucial to understand the evolutionary outcome (Lande, 1981; Iwasa *et al.*, 1991). Third, natural selection models tend to be based on a single fitness component, while sexual selection reflects the interplay of viability selection (e.g., costs of ornaments, costs of choosiness), fecundity selection (e.g., trade-offs between parental care and mating opportunities) and selection on mating and fertilization rates. Fourth, sexual selection models have to address sex differences, such as the sex-limited expression of traits and differences in the strength and direction of selection between the sexes. Fifth, as a consequence of sex-differential selection, genetic details may play a more prominent role than in other evolutionary models. In particular, autosomal versus sex chromosomal inheritance may strongly affect the outcome of evolution. Sixth, the coevolution between the sexes often takes the form of an evolutionary arms race, resulting in ongoing oscillations or even more complex non-equilibrium dynamics. Accordingly, the analysis of sexual selection often necessitates more refined dynamical approaches than the classical equilibrium-oriented methods (Gavrilets & Hayashi, 2005; Van Doorn & Weissing, 2006). Seventh, sexual selection is intrinsically linked to other processes, like sex ratio evolution (Trivers & Willard, 1973), the evolution of parental care (Trivers, 1972) and speciation (Ritchie, 2007; Weissing *et al.*, 2011). It is becoming increasingly clear that robust conclusions on the outcome of sexual selection can only be obtained if such processes are explicitly included in the models (Kokko & Jennions, 2008; Fawcett *et al.*, 2011).

With all these intricacies in mind, it is no wonder that no single model is able to capture all relevant aspects of sexual selection in a fully satisfactory way. Hence,

it is understandable that a variety of modeling approaches has been developed over the years, all having specific strengths and weaknesses. In Section 2.2, we briefly review these approaches, thereby pointing out their scope and limitations. Section 2.3 provides an overview of the potential benefits (and costs) of choosiness. We discuss the relative importance of direct versus indirect benefits and some recent insights, such as the realization that already the classical Fisher model can exhibit ongoing oscillations of preferences and ornaments. In Section 2.4, we address the recent trend of adding mechanistic detail to the classical models of sexual selection. It turns out that some seemingly well-established results are less robust than anticipated by "standard" theory.

Finally, Section 2.5 discusses the implications of taking a more integrative approach and linking sexual selection to other evolutionary processes like the evolution of sex, sex ratio evolution, or speciation. Throughout this review, the focus is on those models that describe the evolution of female preferences for male ornaments, since this comprises the majority of work on the subject. Recent studies that focus on the evolution of male preferences are discussed in Section 2.4.2.

2.2 Modeling approaches

The evolutionary process that underlies sexual selection can be mathematically described in a variety of ways (chapter 2 in Dercole & Rinaldi, 2008). The current section discusses the pros and cons of four of the most widely used descriptions of evolutionary change used in the context of sexual selection: population genetics, quantitative genetics, invasion analysis and individual-based simulations. Box 2.1 on page 32 (and a more elaborate explanation in the Supplement on page 54) illustrates the application of the various modeling frameworks to the 'Fisher process[1]', thereby giving an idea of the congruences and differences between approaches.

Population genetics

Population genetics provides a description of evolution in terms of changes in genotype frequencies (see Box 2.1). In principle, a population genetics framework is the most comprehensive approach to sexual selection, since the evolutionary dynamics is directly modeled in terms of changing genotype frequencies. Whenever genetic processes are crucial for a proper understanding of sexual selection, a population genetics model will typically be the model of choice. A population genetics framework will often be indispensable when studying the implications of genetic architecture (such as sex linkage, recombination or epistatic gene interactions) on the course and outcome of sexual selection. Moreover, population genetic models are useful for delineating the scope and limitations of less comprehensive frameworks like quantitative genetics or adaptive dynamics.

[1] **Fisher process**: a process where selection on male ornaments exerted by a female preference indirectly leads to self-reinforcing selection on the preference itself, due to linkage disequilibrium between preference and ornament alleles.

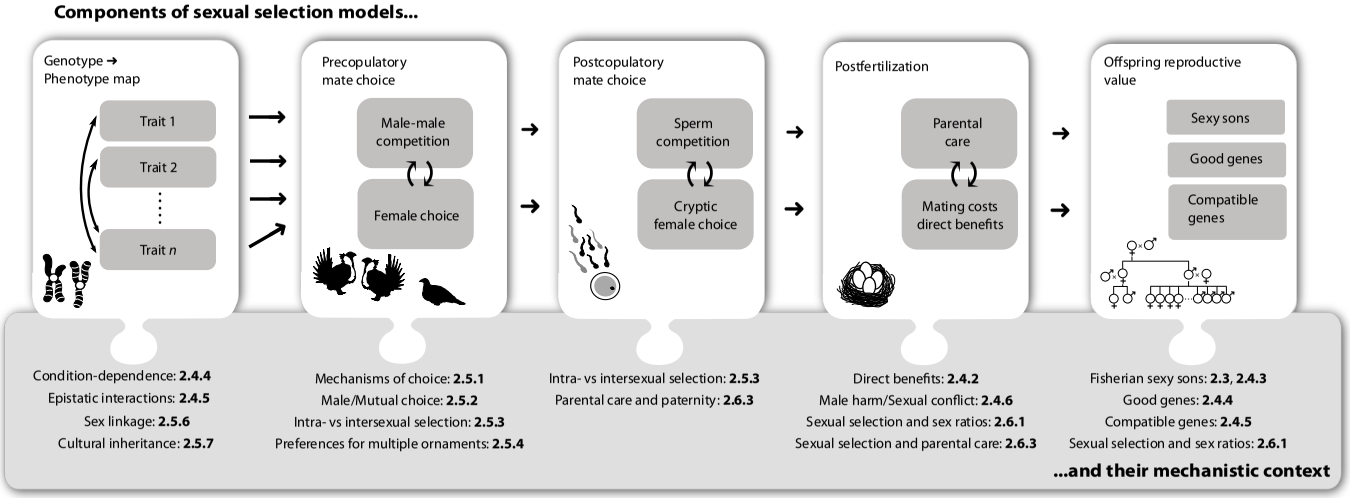

Figure 2.1: A global overview of the main components of sexual selection theory. Bold numbers refer to corresponding sections of this review.

Box 2.1: Four implementations of the Fisher process

In a seminal contribution to sexual selection theory, Fisher (1915) predicted that female preferences could evolve through a self-reinforcing 'runaway' process. Fisher argued that once a female preference for a certain ornament has gained foothold in a population (for whatever reason), both preference and ornament will be subject to positive selection. For the ornament, this is pretty obvious since ornamented males will have a mating advantage if sufficiently many females mate preferentially with such males. In case of the preference, the argument is more sophisticated, because selection on the preference is indirect. Since females with a strong preference will tend to mate with males with a pronounced ornament, preference and ornament alleles will often co-occur in the offspring of such matings, leading to a statistical association of these alleles. As a consequence, positive selection on the ornament will induce correlated positive selection on the preference. Hence preferences induce the evolution of ornaments and subsequently become selected themselves due to their association with the ornament. Fisher realized that this self-reinforcing process could explain the huge exaggeration of sexual ornaments observed in many organisms. Interestingly, Fisher's arguments apply to arbitrary ornaments. In other words, ornaments that evolved through the 'Fisher process' do not necessarily indicate any inherent 'quality' of their bearers. Notice that Fisher's argument on the statistical association between preference and ornament genes does not require the physical linkage of preference and ornament loci on the same chromosome.

For many decades, Fisher's ideas were greeted with skepticism. This is not surprising, since a verbal theory deriving far-reaching and counter-intuitive predictions from indirect processes and the emergence of statistical associations is in need of a sound quantitative underpinning. Yet, it was not before the 1980s that models from quantitative genetics (Lande, 1981) and population genetics (Kirkpatrick, 1982b) revealed that the Fisher process can indeed work. Here we discuss these models, along with an adaptive dynamics model for the Fisher process (Pen & Weissing, 2000c) and an individual-based simulation approach (Fawcett *et al.*, 2007). Our focus is on highlighting both the commonalities and the differences between the various approaches. A more detailed account is given in the Supplement, on page 54 of this thesis.

Population genetics

Central to the population genetics approach is that it tracks genotype frequencies. Kirkpatrick's (1982b) model of the Fisher process is a textbook example of a population genetics model of sexual selection. The model considers two haploid gene loci: the female preference locus P with alleles P_0 (no preference; random mating) and P_1 (preference for mating with males carrying an ornament trait), and the male trait locus T with alleles T_0 (no ornament) and T_1 (having a costly ornament). The relative allele frequencies of the preference and trait alleles are denoted by p and t, respectively, while the "linkage disequilibrium" parameter D describes the statistical association between alleles P_1 and T_1. *see next page.*

As shown in the Supplement (see also Bulmer, 1994), the change in allele frequencies and genetic association from one generation to the next is described by equations of the form:

$$\Delta t = \frac{1}{2}(1-t)A \tag{2.1a}$$

$$\Delta p = \frac{1}{2}DA \tag{2.1b}$$

$$\Delta D = \text{something complicated.} \tag{2.1c}$$

A is a function of p and t that describes the net effect of selection on the ornament (a balance between viability selection against the ornament and the mating advantage of ornamented males). The second equation shows that p will only change if $D \neq 0$, that is, if there is a statistical association between trait and preference alleles. A detailed analysis of eqns. (2.1a-2.1c) reveals that, starting at zero, D will indeed become positive. As shown in Figure 2.2A, the system converges either to loss ($t = 0$) or fixation ($t = 1$) of the ornament or to a line of internal equilibria (given by $A = 0$). The latter correspond to those combinations of t and p where the costs of carrying an ornament in terms of higher mortality are exactly balanced by the mating advantage of ornamented males. The whole set of equilibria is stable in the sense that selection prevents movements away from it, but each individual equilibrium is only 'neutrally stable', meaning that stochastic fluctuations can lead to shifts in p and t along the line of equilibria.

Quantitative genetics

Rather than tracking genotype frequencies, the quantitative genetics approach describes evolution in terms of changes of average phenotypic values. In the classical model of Lande (1981) the (phenotypic) values of a male ornament of size t and a female preference of intensity p are autosomally inherited, sex-limited, normally distributed traits with means $\bar{t}$ and $\bar{p}$. As shown in the Supplement (see also Mead & Arnold, 2004), the change in these means from one generation to the next can be described by the equations

$$\Delta \bar{t} = \frac{1}{2}G_t\beta_t \tag{2.2a}$$

$$\Delta \bar{p} = \frac{1}{2}G_{tp}\beta_t. \tag{2.2b}$$

Here β_t is the total force of directional selection acting on the ornament (which is given by a combination of natural selection against and sexual selection in favor of large ornament size; see eqn. (S2.8) in the Supplement). The system does not include a corresponding term for the preference, since $\beta_p = 0$ in the absence of direct costs and benefits of choosiness. G_t is the additive genetic variance of the ornament, and G_{tp} is the additive genetic covariance between trait and preference. The first equation in (2.2) describes the evolution of the male trait under direct selection, while the second equation describes the correlated evolution of the female preference, which is mediated by the (positive) covariance between trait and preference. *see next page.*

Like system (2.1), also system (2.2) has a line of equilibria. These equilibria correspond to the solutions of $\beta_t = 0$ (where natural and sexual selection are exactly balanced). Figure 2.2B shows the case that the line of equilibria is stable (which happens when the slope of the line of equilibria is larger than G_{tp}/G_t); if the covariance between trait and preference is very large, the line can also be unstable, leading to a never ending 'runaway' away from the line with ever-increasing speed.

Invasion analysis

Here we briefly highlight a model of the Fisher process by Pen & Weissing (2000c), which combines a reproductive value approach with methods from adaptive dynamics theory. The model considers a class-structured population consisting of females and two types of male: non-ornamented males ($♂_0$) and males expressing an ornament ($♂_1$) reducing their viability by a factor $1-s$. Evolvable traits are the females' preference p for mating with ornamented males and the tendency t of a male to develop the ornament (i.e, the tendency to become a type-1 male). The aim is to find the evolutionarily stable values p^* and t^* of preference and ornament, respectively. As shown in the Supplement, the fitness $W(t,p|t^*,p^*)$ of a rare mutant of type (t,p) in a resident population of type (t^*,p^*) can be derived systematically from life-history considerations. Evolutionarily stable strategies can be determined by inspecting the (total) derivatives of the fitness function W with respect to t and p at (t^*,p^*). These are of the form:

$$\mathrm{d}W/\mathrm{d}t = (1-s)\,v_{\mathrm{m1}} - v_{\mathrm{m0}} \tag{2.3a}$$

$$\mathrm{d}W/\mathrm{d}p = b_{pt}\cdot\left((1-s)v_{\mathrm{m1}} - v_{\mathrm{m0}}\right), \tag{2.3b}$$

where v_{m1} and v_{m0} are the reproductive values of ornamented and non-ornamented males in the resident population, while b_{pt} is the slope of the regression of the female preference on the male trait. This latter parameter, which describes the statistical association between trait and preference, is assumed to be positive. At an evolutionary equilibrium, both derivatives in (2.3) have to be zero. This is the case when $(1-s)v_{\mathrm{m1}} = v_{\mathrm{m0}}$, that is, when viability costs of the ornament are exactly balanced by the mating advantage provided by the ornament. As before, there is a whole line of equilibria (Figure 2.2C), and the approach to this line is governed by the canonical equation of adaptive dynamics.

Individual-based simulations

As indicated by Figure S8.2 in the Supplement, an individual-based simulation keeps track of a finite population of individuals, each of which with a set of properties (e.g., genotypes, sex, degree of preference, degree of ornamentation). Individuals interact and due to their properties and chance events they differ in survival, mating success and fecundity. During reproduction, individuals transmit (part of) their heritable properties to their offspring. Variation arises due to mutation. The simulation then tracks evolutionary change over the course of generations. Figure 2.2D shows the outcome of such a simulation in the implementation of the Fisher process by Fawcett and colleagues (2007). *see next page.*

In this simulation model, each individual harbors two loci (both with many alleles); one coding for an ornament of size t and one coding for a preference of intensity p. Making similar assumptions on the mortality costs of the ornament and the mating process as in Lande's (1981) model (see Supplement), the simulated population rapidly converges to a line of equilibria; subsequently drifting along this line. Interestingly, quite different behavior (ongoing oscillations) can occur in the same model for different parameter settings (see Section 2.3.3 and Figure 2.3D).

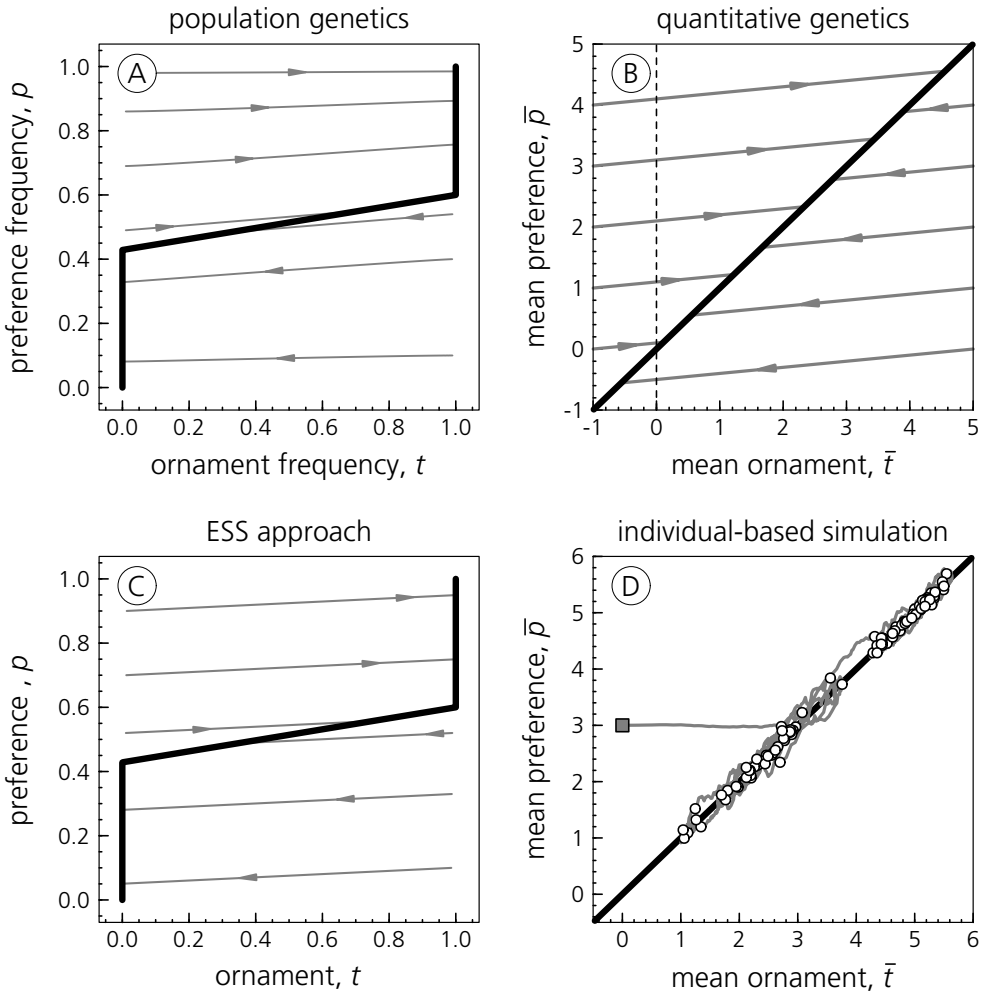

Figure 2.2: Four implementations of the Fisher process. Panel A: dynamics of Kirkpatrick's (1982b) population genetic model. Panel B: dynamics of Lande's (1981) quantitative genetic model. Panel C: Pen & Weissing's (2000c) adaptive dynamics model. Panel D: individual-based simulation based on the model of Fawcett et al. (2007). Black line segments: equilibria of the model (`line of equilibrium'). Grey lines: representative trajectories of the dynamical systems in panels A-C and a single simulation run in panel D.

While population genetic methods have been very important for theory development, they are less popular in practical applications, for at least two reasons. First, despite enormous advances in unraveling the genetic underpinning of sexual characters (Chenoweth & McGuigan, 2010) the genetic basis of traits and preferences is generally not known. Instead of making hypothetical assumptions on the underlying genetics, students of sexual selection tend to find quantitative genetics (Section 2.2.2) and phenotypic approaches (Section 2.2.3) more appealing, since these approaches do not specify the genetics and essentially treat it as a black box. Second, population genetics models quickly become mathematically intractable if several loci with genes of large effects are involved. Therefore in practice most population genetics models only address a small number of haploid loci, in a setting of discrete, non-overlapping generations. Perhaps more importantly, mathematical tractability necessitates to make highly simplifying assumptions concerning the phenotypic level, making it difficult to address the mechanisms of behavioral interactions or complex trade-offs between fitness components.

Due to the advent of the Quasi-Linkage-Equilibrium (QLE)[2] technique (Barton & Turelli, 1991; Kirkpatrick *et al.*, 2002, see Pomiankowski & Bridle, 2004 for a critical appraisal), the disadvantages of the population genetics approach can at least partly be overcome. Based on the assumption that selection is weak relative to the rate of recombination, the intricacies of multi-locus genetics can to a certain extent be avoided by approximating the (high-dimensional) dynamics of genotype frequencies by the dynamics of allele frequencies and lower-level genetic associations (e.g., Servedio, 2004; Kirkpatrick & Hall, 2004; Greenspoon & Otto, 2009). The conclusions obtained are often remarkably robust and not dependent on genetic detail or the mating system (Kirkpatrick & Barton, 1997). However, due to the inherent assumption of weak selection, it is not always clear whether, and to what extent, the predictions of the QLE approach extend to scenarios involving strong selection (e.g., Servedio, 2004).

Quantitative genetics

Quantitative genetics is a widely used technique to model sexual selection (Mead & Arnold, 2004). Quantitative genetics describes evolution at the phenotypic level but still takes account of genetics (to a certain extent) by making plausible assumptions on the transmission of phenotypic traits from parents to their offspring. The latter are encapsulated in the so-called 'G-matrix', the collection of additive genetic variances and co-variances of the phenotypic traits in question. As illustrated in Box 2.1 (and in more detail in the Supplement), the change of (mean) traits and preferences from one generation to the next is characterized by a relatively simple equation that inspires considerable insights into the dynamics of sexual selection. However, the simplicity and elegance of the quantitative genetics approach comes at a cost, since many assumptions have to be made to justify this approach. Moreover, these assumptions are often implicit and not easily testable. One key assumption of the quantitative genetics approach is that breeding values have a multivariate normal distribution, which has seen considerable debate (Barton & Turelli, 1991). Another often made

[2] **QLE**: a technique used to simplify population and quantitative genetics models by neglecting intricate interaction terms in a systematic and model-specific manner. The QLE technique is based on the assumption that selective and population dynamical processes are weak relative to recombination.

assumption is that viability costs are given by exponential functions (see the Supplement). Assumptions like these are mathematically convenient since they assure that the distribution of phenotypes at the mating stage remains Gaussian. However, they are not always realistic and potentially misleading. For example, the Fisher process has a stronger tendency to induce ongoing oscillations of traits and preferences if the costs of choosiness are related to the availability of the preferred males.

Quantitative genetic models often treat additive genetic variances and covariances as fixed parameters (e.g., Pomiankowski *et al.*, 1991). However, selection obviously affects the entries of the G-matrix (see Arnold *et al.*, 2008 for a recent review), due to the depletion of additive genetic variation (and also indirectly by shaping mutation rates) or the buildup of additive genetic covariation (i.e., linkage disequilibria) due to assortative mating. Under certain assumptions regarding mutation, recombination and the strength of selection, the evolution of the G-matrix can be studied within the quantitative genetic framework with the help of the aforementioned QLE approach (Barton & Turelli, 1991; Pomiankowski & Iwasa, 1993; Walsh & Lynch, 2012). In most cases, however, individual-based simulations provide a more convenient and versatile tool to assess the evolution of the G-matrix (e.g., Arnold *et al.*, 2008).

From the study of quantitative trait loci (QTL) it is known that quantitative traits are often affected by at least some major genes of large effect. If this is the case, a hybrid approach can be taken, in which one or more quantitative traits coevolve with large-effect modifiers (Lande, 1983). The same type of approach can be used when studying the interplay of sexual selection and segregation distortion or sex chromosome evolution.

Invasion analysis

While quantitative genetics is based on the assumption that a continuous distribution of phenotypes (and typically also genotypes) is available at all times, evolutionary game theory, adaptive dynamics and other 'phenotypic' approaches (Weissing, 1996) consider the opposite extreme of a monomorphic 'resident' population that is repeatedly challenged by the invasion attempts of rare mutants. The underlying idea is that evolution proceeds by a series of subsequent invasion and trait substitution events. The dynamics of this process can be described by the 'canonical equation[3]' of adaptive dynamics (Dieckmann & Law, 1996), where a 'mutational variance- covariance matrix' $\mathbf{M}$ (describing the distribution of invading mutants) plays a very similar role as the additive genetic variance-covariance matrix $\mathbf{G}$ does in quantitative genetics. Usually, the exact dynamics of how a mutant coexists with, and replaces the resident is not modeled explicitly. Instead, population dynamical considerations are used to derive an expression for the 'invasion fitness[4]' of rare mutants (Metz *et al.*, 1992). This fitness functions is then systematically analyzed in order to identify evolutionarily stable strategies and other potential end points of the evolutionary process

[3]**Canonical equation of adaptive dynamics**: the differential equation $\frac{\mathrm{d}}{\mathrm{d}\tau}\mathbf{x}^* = z\mathbf{M}\nabla W(\mathbf{x}|\mathbf{x}^*)$ gives a description of change in the vector of characters $\mathbf{x}^*$ over evolutionary time τ, through a successive series of invasion-substitution events. z reflects the variation in the rate of occurrence of mutations, whereas the mutational variance-covariance matrix $\mathbf{M}$ describes how a single mutation affects the different traits. The direction of evolution is given by the invasion fitness gradient $\nabla W(\mathbf{x}|\mathbf{x}^*)$ of a rare mutant $\mathbf{x}$ invading a population of $\mathbf{x}^*$ residents.

[4]**Invasion fitness**: the exponential growth rate of a rare mutant in an environment determined by the common resident strategy.

(Geritz *et al.*, 1998; McGill & Brown, 2007; Dercole & Rinaldi, 2008). In a life-history context, it is convenient to frame invasion fitness in terms of reproductive values[5] (Taylor, 1996a).

Since such phenotypic approaches neglect most genetic intricacies, they can address environmental feedbacks, frequency and density dependence, and age structure in more detail than other approaches. It is therefore not surprising that recent models investigating the interaction of sexual selection with parental care (Kokko & Jennions, 2008) or sex allocation (Fawcett *et al.*, 2011) rely on a phenotypic approach. Nonetheless, the shortcomings of phenotypic models should not be lost out of sight. One key assumption is that populations are nearly monomorphic. Since variation in male ornamentation is essential to the evolution of female choice, additional assumptions are thus necessary to maintain genetic variation (see the Supplement), but the amount of variation that is maintained can, alter the outcome in surprising ways (e.g., McNamara *et al.*, 2008). Relaxing the assumption that mutants only differ slightly from the resident can also strongly affect the evolutionary dynamics (e.g., Wolf *et al.*, 2008). Another disadvantage is that phenotypic models do not easily allow for a dynamical description of linkage disequilibria. Lastly, most of these models assume that evolution proceeds at a much slower timescale than the ecological dynamics, while it is well known that sexually selected characters may evolve rapidly (Van Doorn *et al.*, 2001; Swanson & Vacquier, 2002; Shirangi *et al.*, 2009).

Individual-based simulations

Individual-based simulations (see Box 2.1) provide a flexible and easily extendable way of modeling complicated scenarios with a high degree of realism (e.g., environmental and demographic stochasticity; spatial population structure, Fromhage *et al.*, 2009; complex genotype-phenotype maps, Ten Tusscher & Hogeweg, 2009; a concrete representation of the sensory system, Fuller, 2009). This is a clear advantage when compared to all the simplifying assumptions made by analytical models. For example, the presence of stochasticity in individual-based simulations allows for a straightforward assessment of the importance of drift (Uyeda *et al.*, 2009), which is much harder to assess in analytical models of sexual selection. On the downside, running complex simulations is often computationally demanding, limiting the number of parameter settings that can be investigated. If the model contains only 10 parameters (most have many more), then $3^{10} \approx 60,000$ simulations would have to be run in order to consider all combinations of only 3 values for each parameter. Moreover, replicates of each simulation have to be run, in order to cope with the stochasticity inherent in each simulation run. Since this is not always feasible, it is often not clear whether and to what extent a given set of simulations is representative. Nonetheless, we have the impression that the disadvantages of simulation models are often overemphasized (e.g., McElreath & Boyd, 2007, p. 8) in comparison to advantages as relative ease of implementation and applicability to all kinds of situations. Perhaps most importantly, individual-based simulations can nicely complement an analytical approach. In fact, the theoretical justification of analytical approaches is often restricted to a narrow domain (e.g., weak selection), and simulations are a useful tool to explore the robustness and general applicability of analytical predictions beyond this domain.

[5] **Reproductive value**: an individual's expected contribution to the population in the long term.

Table 2.1: Pros and cons of the different modeling approaches discussed in Section 2.2.

	Population Genetics	Quantitative Genetics	Invasion Approaches	Individual-based Simulations
Pros	most comprehensive description of sexual selection; explicit inclusion of genetical aspects (recombination, linkage disequilibrium); transparent model assumptions; provides justification of quantitative genetics approach	focus on (measurable) phenotypic variation; method applicable to systems with limited information on genetics; model parameters (additive genetic variances and covariances) can be estimated	minimal assumptions on genetics allow most comprehensive description of phenotypic level; based on transparent fitness concept; fitness function derived from first principles, allowing to consider demography, class structure, frequency and density dependence	most versatile approach; few restrictions on model structure, allowing for complicated genetic architectures and intricate selection scenarios; natural inclusion of demographic and environmental stochasticity
Cons	mainly suited for discrete variation; only tractable for highly simplified fitness scenarios; limited applicability since the genetic basis of most traits is unknown; analytical tractability limited to a small number of loci with few alleles;	assumptions (e.g. normal distribution of additive effects) often not met; G-matrix often assumed constant; difficult to include complex genetics, complex life histories, and complex fitness scenarios (age structure, trade-offs, etc.)	assumption of monomorphic population clearly unrealistic; only includes simplistic genetics while genetic associations may be crucial; multi-variate adaptive dynamics theory not well-developed	danger of cherry-picking since only small part of parameter space can be investigated; interpretation of results often subjective; difficult to judge the generality of conclusions; 'coarse' description of simulation programs
Solutions	in case of weak selection, powerful techniques (e.g., QLE) are becoming available to address complex multi-locus problems and fairly complex fitness scenarios	methods for studying the evolution of the G-matrix are becoming more broadly available; hybrid models combining a quantitative genetic approach with population genetics and adaptive dynamics	application of adaptive dynamics to genotypic level; combination of adaptive dynamics approach with genetic approaches	application of simulations to systems where analytical results are already available as a test case

A plea for pluralism

The famous quote that "each disadvantage has its advantage" (by the former Dutch football player Johan Cruijff) also applies to the various modeling frameworks considered thus far (Table 1). All approaches have their advantages and disadvantages, and the choice of approach should mainly depend on the research question to be tackled. But how should we deal with the often unrealistic assumptions made by virtually all approaches? Richard Levins' (1966, p. 423) statement that "our truth is the intersection of independent lies" provides an answer: if multiple model frameworks, varying in their underlying assumptions, arrive at a similar outcome, we can be confident that this conclusion is robust and not just a result of some limiting assumptions. Hence, the use of multiple modeling frameworks alongside each other helps us to delineate the scope and limitations of the predictions of sexual selection theory.

2.3 The costs and benefits of choice

Much debate in sexual selection theory was focused on the benefits driving the evolution of mating preferences. Females can benefit *directly*[6] from expressing a preference, if the resulting choice of mates gives them a higher viability or fecundity than they would have had without the preference (Møller & Jennions, 2001). Alternatively, the particular choice of mating partners may lead choosy females to have offspring with a higher reproductive value (Kokko *et al.*, 2002) for example through attractive sons (Fisher, 1915) or offspring of higher intrinsic quality (Zahavi, 1975), in which case benefits are said to be *indirect*[7]. In this context, offspring reproductive values often refer to offspring lifetime reproductive success, although there may be cases in which the reproductive success of grandoffspring or later generations should also be considered. Here, we highlight the major assumptions underlying direct and indirect benefits models of sexual selection.

No benefits: sensory by-products

Female preferences can evolve in the absence of any benefits related to mate choice, for example, as a pleiotropic byproduct of natural selection on the sensory system (Kirkpatrick, 1987b; Kirkpatrick & Ryan, 1991). This is confirmed by models employing evolving neural networks (mimicking a simple sensory system), which often lead to mating preferences (e.g., in favor of symmetric mates) as a by-product (reviewed in Enquist & Ghirlanda, 2005; Phelps, 2007). While early models based their conclusions on highly simplified network architectures (Arak & Enquist, 1993, see Dawkins & Guilford, 1995 for a critique), more recent models are tailored to the sensory system of particular organisms. For example, Fuller's (2009) model based on the sensory system of guppies (*Poecilia reticulata*) reveals that details of the sensory architecture, such as the number of output neurons, can strongly affect the evolution of sensory biases. It remains to be seen which types of sensory architecture are most conducive to the evolution of preferences as sensory byproducts, on which external

[6] **Direct benefits of choice**: choosy females sustain increased fecundity or survival, unrelated to the genotypes of her offspring.

[7] **Indirect benefits of choice**: an increase in reproductive value of a choosy female caused by the genotypes of her offspring.

conditions (i.e., the sensory environment) certainly has a large influence (Endler & Basolo, 1998). In addition, there are few predictions on the long term evolution of preferences that evolve as byproducts of natural selection. Will pleiotropy in preferences always decay over time (when pleiotropic preferences lead to suboptimal female mating decisions, Arnqvist, 2006), or can we find situations in which the co-evolutionary dynamics of both natural and sexual selection enhance the maintenance of pleiotropic preferences?

Direct benefits

There is direct selection on female choosiness whenever the degree of choosiness is related to a female's survival and fecundity. Often choosiness will be costly, for example when searching for mates exposes the female to predators, or when choosy females risk ending up unfertilized (Kokko & Mappes, 2005). As discussed below, even slight costs can override indirect benefits of choosiness, leading to the theoretical expectation that sexual selection driven only by indirect benefits of choosiness is rare in nature (Kirkpatrick & Barton, 1997; Cameron *et al.*, 2003).

The evolution of female preferences can most easily be explained if females gain direct benefits from being choosy. One likely mechanism is when females prefer males that advertise their quality to provide paternal care: illuminating models on this good-parent process have been made by Price *et al.* (1993) and Iwasa & Pomiankowski (1999). Direct-benefit models have received relatively little attention in the theoretical literature; presumably because the underlying mechanisms seem transparent and not too challenging (or sufficiently counter-intuitive) to modelers. However, a number of recent results indicate that direct benefits mechanisms are not as straightforward as one might think. For example, males may not only differ in parental ability but also in genetic quality, and these aspects of quality are not necessarily related. Hence, it matters what aspect of quality is being signaled and how to interpret male signals (Kokko, 1998; Alonzo, 2012). Moreover, mating with a male of high parental quality may not assure a high level of paternal care if such males tend to mate with many females and therefore have to distribute their care over many offspring (Cotar *et al.*, 2008; Tazzyman *et al.*, 2012). In addition, females can be expected to change their own care level dependent on their choice of mates (Ratikainen & Kokko, 2010). All this leads us to conclude that direct benefits sexual selection deserves more attention than currently is devoted to it.

Indirect benefits: the Fisher process

The Fisher process relates to the scenario in which female preferences are maintained due to self-reinforcing selection (see Box 2.1). The key benefit involved in the Fisher process is that choosy females have more grandoffspring through the production of attractive sons, which have a higher mating rate. These benefits are relatively small; it is a well-known result that the slightest costs of choosiness will lead to a breakdown of the line of equilibria and the disappearance of costly choosiness in models of the Fisher process (Kirkpatrick, 1982b; Pomiankowski, 1987a; Bulmer, 1989, see Figure 2.3A). However, the Fisher process can be rescued by including additional mechanisms in the model, such as a mutation bias (mutations that mainly have negative effects on male ornamentation; Pomiankowski *et al.*, 1991, Figure 2.3B) or migration bias (influx of migrant males with smaller ornaments; Day, 2000, Figure 2.3C).

Even in the absence of such mutation or migration biases, the exaggeration of sexually selected traits beyond the naturally selected optimum is possible if the costs of choosiness and ornamentation are sufficiently weak (Hall *et al.*, 2000). When this is the case, traits and preferences do not converge to equilibrium but oscillate forever on a limit cycle (chapter 3 of this thesis, Figure 2.3D). Similar cyclic dynamics have been described earlier in variants of the Fisher process with a curvilinear set of equilibria (Iwasa & Pomiankowski, 1995).

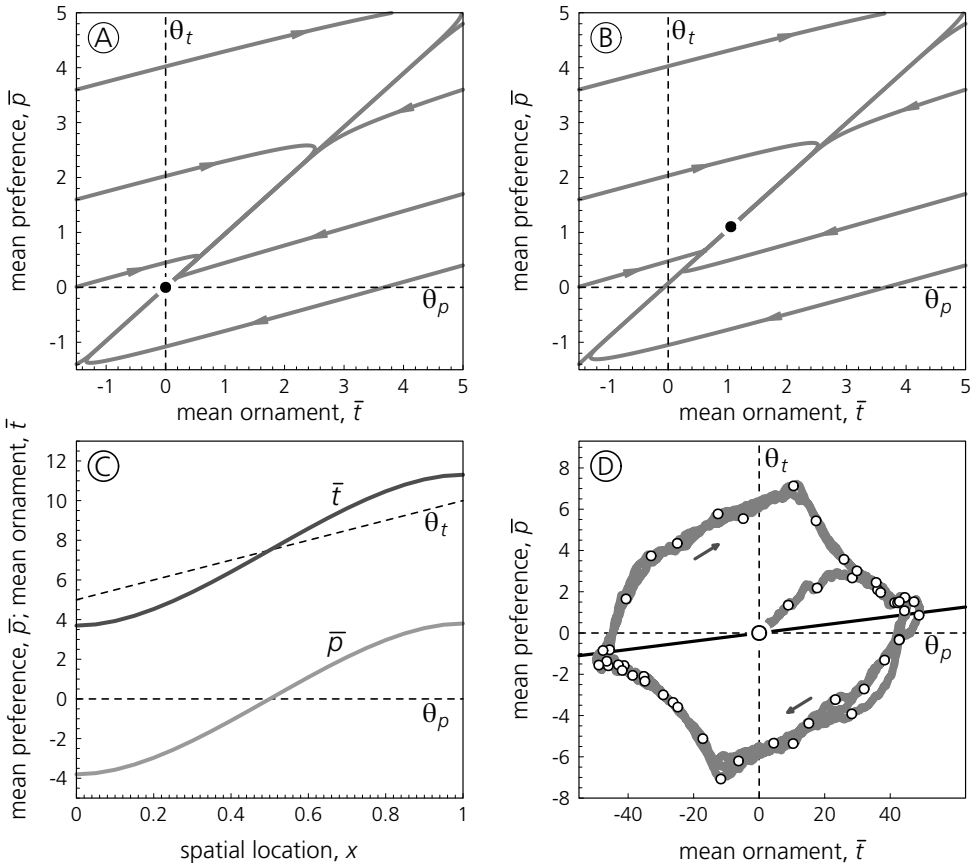

Figure 2.3: Costs of choice and the Fisher process. Panel A: In the presence of costly choice, the line of equilibria (see Figure 2.2B) breaks down to a single equilibrium point, coinciding with the naturally selected optimum of t and p (θ_t and θ_p), thus eliminating costly exaggeration of sexually selected traits (Pomiankowski, 1987a; Bulmer, 1989). Panel B: Biased mutations tending to reduce ornamentation can `rescue' sexual selection, giving rise to an equilibrium point away from the naturally selected optimum (Pomiankowski *et al.*, 1991). Panel C: Spatial variation and the influx of migrants with smaller ornaments provides another mechanism that leads to exaggeration of t (dark grey line) and p (light grey line) beyond their naturally selected optima (dotted lines), which now vary along a spatial gradient (x-axis) (Day, 2000). Panel D: When the viability costs of preferences and traits are small, the equilibrium in panel A becomes unstable and the system converges to a limit cycle, corresponding to indefinite cycling of traits and preferences around the naturally selected optimum (chapter 3 of this thesis).

Indirect benefits: good genes

In "good genes[8]" scenarios of sexual selection, female preferences evolve for male indicators of heritable quality. It is typically assumed that heritable "quality" is in terms of enhanced offspring survival, but it should actually be interpreted in terms of enhanced offspring reproductive value (Kokko, 2001; Kokko *et al.*, 2002). Even if offspring inherit genes intrinsically favoring their viability, their survival may actually be lower than that of lower- quality offspring (for example through intense signaling). This focus on reproductive values, in combination with the notion that all models of sexual selection, including the good-genes process, contain some element of the Fisher process has led some to conclude that differences between the Fisher process and good-genes sexual selection are small and superficial (Kokko, 2001; Kokko *et al.*, 2002). However, good-genes sexual selection is conceptually different from the Fisher process in several ways. First, the dynamics of both processes is different; while the Fisher process only requires coevolving preference and ornament genes, good-genes processes include at least one additional dimension (corresponding to heritable 'quality'). Second, the evolution of a genetic correlation between trait and preference is a crucial ingredient of the Fisher process (see Box 2.1). In contrast, good-genes sexual selection can still work (and in fact lead to a runaway process) if such a correlation cannot get established (e.g., when preferences inherit exclusively through the matriline, while ornaments inherit exclusively through the patriline). In cases like this, preferences can evolve through a genetic correlation with alleles related to heritable quality. Third, in the Fisher process ornaments and preferences are directly coupled; by definition, the ornament is the target of the preference. Discussions on whether the ornament can act as a 'reliable' indicator of genetic quality (here: male attractiveness) are irrelevant for the Fisher process, while they have played a dominant role in the context of good-genes processes. Hence, although it is important to point out the similarities between indirect-benefit models of sexual selection (Kokko *et al.*, 2002), the conceptual differences between the Fisher process and good-genes processes should not be neglected.

Another debate on good-genes models of sexual selection has centered on the question how females can reliably distinguish between males of different quality. According to Zahavi's (1975) handicap hypothesis, signals can only be reliable indicators of male heritable quality if costs are associated with these signals. However, not all costly signals are reliable indicators of quality (Getty, 2006). To separate the sheep from the goats, signals have been classified into different categories (Maynard Smith, 1985, see Van Doorn & Weissing, 2006 for formal definitions). Signals are said to be condition-dependent, when the expression of a given signal is less costly for males of higher quality. Alternatively, signals are designated as revealing if for a given level of resource allocation into the development of an ornament higher-quality males produce more pronounced ornaments than low-quality males. While condition-dependent and revealing signals can evolve as reliable indicators of heritable quality (Andersson, 1994), it has often been claimed that epistatic signals (which are neither condition-dependent nor revealing) are unreliable and therefore cannot lead to the evolution of costly female preferences. Several studies report the

[8] **Good-genes sexual selection**: the evolution of female preferences for male indicators of heritable quality, where quality influences the reproductive values of both sons and daughters and does not necessarily reflect the mating success of sons.

contrary. In a general model, Kirkpatrick & Barton (1997) show that – irrespective of the type of signal – the buildup of a correlation between male ornamentation and heritable quality can lead to indirect selection on the female preference (although this effect is weaker in case of epistatic signals; see also Siller, 1998). Van Doorn & Weissing (2006) show that female preferences for epistatic signals can evolve if the coevolution of ornaments and preferences does not lead to a stable equilibrium but to a limit cycle (as in Figure 2.3D).

Another problem is that female choice for high quality males can lead to the depletion of genetic variation in quality, commonly referred to as the 'lek paradox' (see Kotiaho *et al.* (2008) for a review). A potential resolution to the lek paradox that received by far the most interest is the genic capture hypothesis (Rowe & Houle, 1996). The central tenet of this hypothesis is that an individual's condition is determined by a large number of genes, providing a large mutational target so that always some genetic variation in condition exists. Indeed, sexual selection itself may cause the number of genes that underlie a signal to increase, giving rise to genic capture (Lorch *et al.*, 2003).

Indirect benefits: compatible genes

Whereas good genes models assume that "genetic quality" is an intrinsic property of the genetic make-up of an organism, reality may not be that simple (Hunt *et al.*, 2004; Puurtinen *et al.*, 2009). For example, males may signal local adaptation (Proulx, 2001; Reinhold, 2004) or adaptation in contexts of frequency-dependent, disruptive selection (Van Doorn *et al.*, 2009). In addition, offspring performance may not directly reflect the genetic quality of the parents if it is affected by epistatic interactions between paternally and maternally inherited genes. Epistatic interactions are a complicating factor for sexual selection theory, since different females may prefer different types of male, depending on their own genotype. The existence of sexually antagonistic variation (Albert & Otto, 2005; Arnqvist, 2011) or selection on heterozygosity and other forms of epistatic variation are examples of such "compatible allele" effects (Puurtinen *et al.*, 2009). In general, such effects weaken the selection on preference alleles. However, recent models show that directional preferences[9] can nevertheless evolve if certain conditions are met, such as biased mutations (Lehmann *et al.*, 2007) or spatial variation in finite populations (Fromhage *et al.*, 2009).

Avoiding male induced costs: sexual conflict

A large body of literature has revealed that females may have to endure mating-related costs, such as seminal toxins or damage by male genital spines (e.g., Rice, 1996; Arnqvist & Rowe, 2005). Resistance to harmful mating can be considered as a mating preference, since it can restrict the potential set of mating partners (Halliday, 1983). Importantly, selection on female resistance is direct, since any female that evolves higher levels of resistance reaps the benefits in terms of increased fecundity or survival (Kokko *et al.*, 2006).

The most influential coevolutionary models envisage female resistance as a threshold character, which causes females to only mate with those males that have harm

[9] **Directional preferences**: preferences where all females prefer similar male phenotypes, as opposed to preferences where the preferred male phenotype depends on the female's own genotype or state.

trait values, which surpass the female's threshold (Gavrilets *et al.*, 2001). Females that accept too many males will incur a fecundity cost on top of the mortality cost of developing a resistance trait. Depending on these costs, the course of evolution varies, but exaggeration of male harm and female resistance is a common outcome. The situation is substantially different when female resistance evolves in the form of insensitivity, making a female reluctant to mate with any male phenotype (Rowe *et al.*, 2005). In this case, male harm is effectively neutralized by female insensitivity, and an evolutionary standstill is a likely outcome, showing that mechanisms with which female resistance is realized can strongly change conclusions. Sexual conflict models have to face the further complication that females mating with the most persistent males may accrue indirect benefits, since they will tend to produce more persistent sons. Although these indirect benefits are unlikely to recoup the direct costs of harm for reasons stated above, they may nonetheless alter the coevolutionary dynamics substantially (Härdling & Karlsson, 2009).

Multiple costs and benefits

While the majority of models have focused on a single benefit or cost component, the co- occurrence of multiple costs and benefits has hardly been explored (but see Iwasa & Pomiankowski, 1999; Van Doorn & Weissing, 2004; Härdling & Karlsson, 2009). One particular aspect that deserves attention is the potential for trade-offs between direct and indirect benefits. For example, in the good-parent process, females may incur more direct benefits (i.e., more care) by mating with an unattractive males that has few mating opportunities and, hence, can focus its care on a small number of offspring (e.g., Cotar *et al.*, 2008; Tazzyman *et al.*, 2012). These direct benefits are associated with indirect costs, since the sons sired by these males will not be attractive and thus have a limited mating rate. Such trade-offs may lead to condition-dependent choice (Cotton *et al.*, 2006) in which some females prefer males that provide direct benefits and others males with indirect benefits.

2.4 Adding mechanistic detail to sexual selection models

Traditionally, sexual selection models incorporated the processes under study in an abstract and seemingly general way. More recent models tend to include more mechanistic detail, thereby making the models more specific but also more easily applicable to real systems. Here we highlight how seemingly general insights of traditional models can change when the mechanisms underlying sexual selection are explicitly incorporated.

The mechanisms of mate choice

Mate choice behaviors refer to any behavior that limits an individual's set of potential partners (Halliday, 1983). While any review on mate choice is eager to point out the behavioral and social complexities involved (Cotton *et al.*, 2006), little has been done to integrate these notions with formal models of sexual selection. In practice, most models assume that females sample from an effectively infinite pool of males, in which attractive males are more "apparent" to choosy females and are thus more easily encountered (e.g., Lande, 1981, 1980; Kirkpatrick, 1982b). Since slight changes to these assumptions can already dramatically affect the outcome (e.g., Seger, 1985),

it raises the question how females should optimally sample males in the face of costs and constraints. Optimal sampling models predict that females should evaluate males sequentially and stop sampling when they sample a male that matches a certain threshold (Real, 1990; Wiegmann *et al.*, 2010), where the value of this threshold may depend on the particular way females acquire information about the male phenotype distribution (e.g., Dombrovsky & Perrin, 1994; Luttbeg, 1996). Houle & Kondrashov (2002) show that sequential sampling in a good-genes model enhances sexual selection: whereas in classical models, costs of preferences increase with the level of exaggeration beyond an abstract survival optimum, sampling costs allow a female to have very strong preferences as long as she is likely to encounter a suitable male within a limited number of samples. In general, Houle & Kondrashov's result shows that there is much to say for a further integration of mechanistic aspects of mate choice with models of sexual selection.

Mutual choice and sex role reversal

The focus on female choice of many models denies the observed variation in sex differences in choosiness that exists across taxa, varying from female to male choice and instances of mutual choice (Clutton-Brock, 2007). It is generally understood that mutual choice may evolve when both sexes exhibit sufficient variation in quality, but is hampered by the possibility that choice may be more costly for the sex that competes most intensely for matings (Johnstone *et al.*, 1996). Moreover, the intensity of competition among members of one sex may itself be a function of the amount of choice exerted by the opposite sex, indicating that the evolution of sex differences in mate choice is the result of a complicated feedback between choice and competition (see Kokko & Johnstone, 2002; Kokko & Jennions, 2008). In order to disentangle this, recent models have taken a "self-consistent" approach where individuals of each sex are either breeding ("time out") or busy acquiring matings ("time in"). The time and mortality costs of each activity feeds back on the densities of individuals that breed or that compete for mates, which subsequently influences the evolution of mate choice in each sex (Kokko & Jennions, 2008). Using a version of this framework, Kokko & Johnstone (2002) found that mutual choice evolves only under restrictive conditions, since choosiness in one sex immediately reduces the mean mating rate of the other sex, which therefore becomes more competitive and is thus less likely to evolve choice (see also Servedio & Lande, 2006). Only when both the cost of breeding is high for both sexes and when mate-encounter rates are high for both sexes, it pays for both males and females to be choosy (Kokko & Johnstone, 2002).

Intrasexual versus intersexual selection

Apart from ornamentation that has evolved in the context of mate attraction, males may also evolve weapons or signals (i.e., badges of status) in the context of male-male competition. Another possibility is that male ornaments are used for both mate attraction and male-male competition, which has received little formal attention to date, despite considerable empirical support (Berglund *et al.*, 1996). A recent model by Veen (2008), however, considers the coevolution of female preferences for male signals that both signal dominance in male-male competition and quality to a female. Interestingly, whereas mate choice and male-male competition in isolation may only lead to the evolution of such signals under particular conditions (see Sections 2.2,

2.3), the interaction between both processes appears to be particularly conducive to the evolution of male signals.

When focusing on the postcopulatory stage, a sound body of theoretical predictions on intrasexual selection (i.e., sperm competition) exists (Parker & Pizzari, 2010). In addition, recent efforts have started to consider trade-offs between male investment in traits that increase a male's mating rate versus his success in sperm competition (e.g., Tazzyman *et al.*, 2009). However, the role of female choice in these contexts is only starting to be assessed (Ball & Parker, 2003; Fromhage *et al.*, 2008) and we know of no formal studies on the coevolution between female choice and heritable male traits that are directly related to ejaculate investment.

Preferences for multiple ornaments

Whereas the vast majority of sexual selection models typically focus on single, univariate display traits, sexual displays often involve many different components (for recent reviews, see Candolin, 2003; Bro-Jørgensen, 2010). Most models that formally investigated these hypotheses particularly focused on the redundant signal hypothesis (Møller & Pomiankowski, 1993), where preferences evolve for multiple indicator traits reflecting the same quality. The widely accepted view is that there is only a limited scope for preferences based on these backup signals, since females should always favor ornaments that have the greatest honesty and reliability; preferences for any additional ornament will only be tolerated when its costs are sufficiently low (Schluter & Price, 1993; Iwasa & Pomiankowski, 1994, Figure 2.4A versus 2.4B). Nonetheless, these analyses rely on equilibrium arguments, whereas a more dynamical analysis shows that females can easily maintain preferences for multiple redundant ornaments through conflicts between males and females over the honesty of signaling (Van Doorn & Weissing, 2006, Figure 2.4C). Moreover, preferences for multiple ornaments can also easily evolve when each ornament signals a different aspect of quality (multiple messages: Johnstone, 1995; Van Doorn & Weissing, 2004, Figure 2.4D). Hence, in contrast to more classical models (e.g., Schluter & Price, 1993), various studies point out that the evolution of multiple indicators of quality does not appear as restrictive as previously thought. Yet, these recent studies generate as many new questions as they resolve: for example, can external factors, such as context-dependence, also be responsible for the maintenance of multiple preferences? How much more likely is reproductive isolation due to drift in case of multiple ornaments (e.g., Pomiankowski & Iwasa, 1998)? These and other questions await further investigation.

Individual variation in ornamentation

Apart from the inheritance of sexually selected characters, another aspect that is poorly understood is the large developmental variation and plasticity observed in sexually selected characters (Pomiankowski & Møller, 1995). How sexual selection affects developmental variation has up to now only been investigated in the context of condition-dependent indicator traits. In particular, a number of theoretical studies have investigated how indicator traits develop over an individual's lifespan (Kokko, 1997; Rands *et al.*, 2011). Interestingly, these studies find that low quality males may express larger ornaments than high quality males, since their higher mortality rate induces them to make a terminal investment in order to achieve matings. It remains

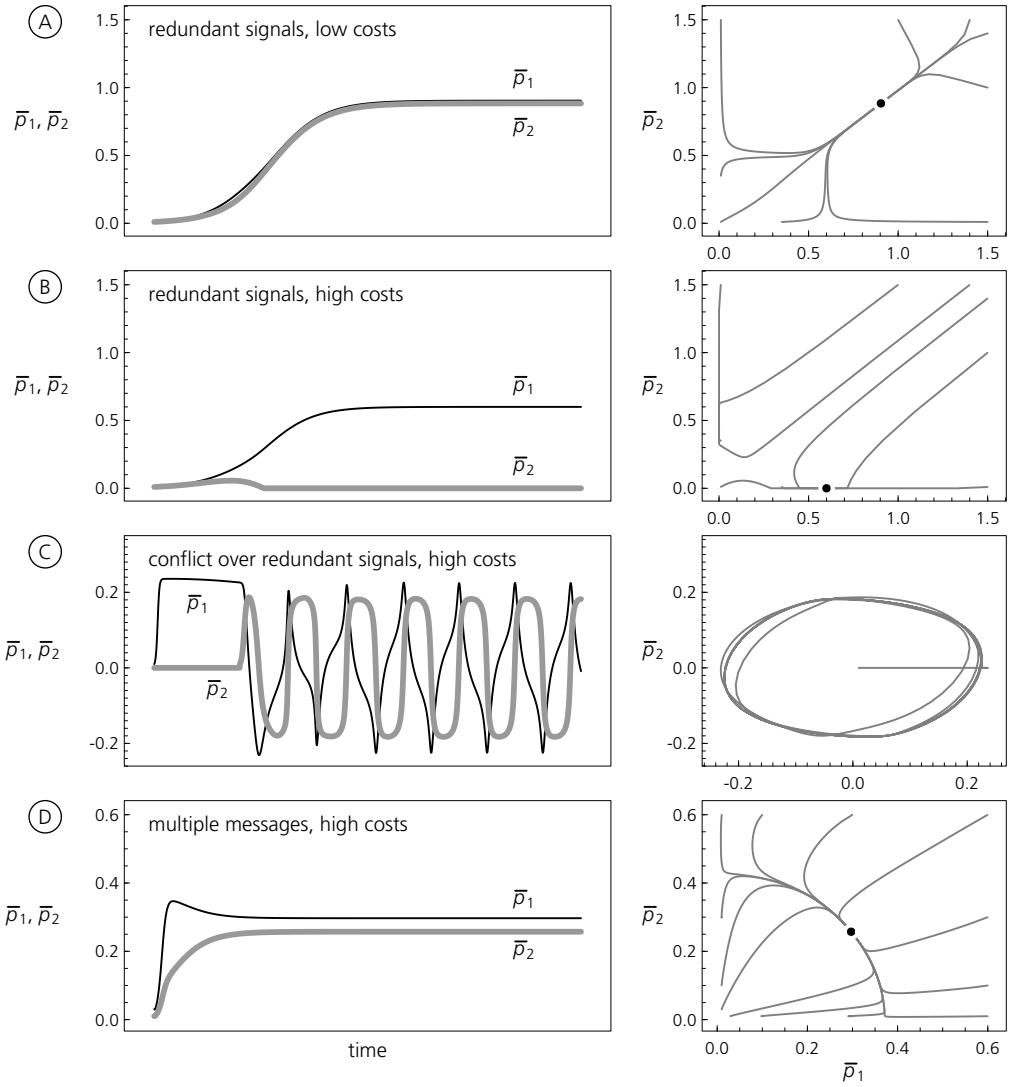

Figure 2.4: The evolution of female preferences for multiple indicators of male quality. Panel A: multiple female preferences for redundant signals only evolve to stable levels when the cost of expressing multiple preferences is sufficiently low (Iwasa & Pomiankowski, 1994). Panel B: In contrast, when the cost of expressing multiple preferences is higher, the cost of expressing the first preference ($\bar{p}_1 > 0$) blocks the evolution of any additional preferences ($\bar{p}_2 = 0$), and the evolution of a particular preference towards nonzero equilibrium levels depends on initial conditions. Panel C: Nonequilibrium dynamics of preferences and ornaments (for example due to sexual conflict over signaling) can lead to the establishment of multiple preferences for redundant indicators, even when high costs of female preferences would preclude the evolution multiple preferences at equilibrium (Van Doorn & Weissing, 2006). Panel D: multiple female preferences for indicators that each signal distinct components of male quality (``multiple messages'') can evolve to stable levels, even in the face of high costs (Van Doorn & Weissing, 2004).

to be seen if such "dishonest" signals are more widespread and extend to contexts in which individual variation is expressed in other aspects than variation in lifespan.

Genetic architecture: sex linkage

Sex-linked inheritance of sexually selected traits has received considerable interest (Reinhold, 1998; Reeve & Pfennig, 2003), which is based on the observation that groups with Z-W sex chromosomes (e.g., birds, butterflies) have more strongly exaggerated ornaments than groups with X-Y sex chromosomes (e.g., flies, mammals) (Hastings, 1994) and also on a growing number of concrete examples of sex-linked sexually selected traits (Qvarnström & Bailey, 2009). Although simple haploid inheritance models apply to cases of Y or W-linkage, other patterns of sex-linkage require a diploid locus in one sex (i.e., XX or ZZ), requiring a more complex model. Multilocus approximations such as QLE can considerably simplify such models. Using these techniques, Kirkpatrick & Hall (2004) confirmed that Z-W systems are more conducive to sexual selection in comparison to X-Y systems. For example, Z-linked preferences are favorable to Fisherian sexual selection, since it increases the time that preferences endure indirect selection when present in males (2/3 of the time), compared to no or negative selection when present in females (1/3 of the time). Z-W systems are also more conducive to sexual selection in case females also express a costly male ornament (i.e., due to a lack of sex-limited expression), representing a case of sexual antagonism (Albert & Otto, 2005). Z-linkage of the ornament ensures that an ornament endures net positive sexual selection (present in males 2/3 of the time), whereas viability selection in females is minimized (present in females 1/3 of the time). Beyond the sex-linkage of ornament and preferences, sex-linkage of other traits such as genetic quality can also affect sexual selection (Connallon, 2010), illustrating that the genetic architecture is important when making predictions about the strength of sexual selection.

Cultural imprinting

Cultural inheritance may also play an important role in the evolution of sexually selected characters. Examples are song imitation in passerine birds, or mate choice copying in guppies (Dugatkin, 1996). In principle, cultural evolution can be described by the frameworks from Section 2.2, with the modification that transmission may now occur horizontally and that genetic and culturally inherited traits may evolve independently at separate timescales (Lachlan & Feldman, 2003). A general result is that imitation often leads to positive frequency-dependence, since the most prevalent variant is also most likely to be imitated (Laland, 1994). As a result, the cultural variants and genotypes that are initially present in a population have a large impact on the eventual outcome, since any novel and rare variant is unlikely to invade in regimes of positive frequency dependence (e.g., Kirkpatrick & Dugatkin, 1994; Lachlan & Feldman, 2003). As a consequence, drift may play a crucial role in the eventual fixation of sexually selected characters, suggesting a large scope for population divergence when imitation is important (Lachlan & Servedio, 2004). In general, aspects such as learning and imitation as well as the social context in which this occurs (Vakirtzis, 2011) should play a more central role in sexual selection theory.

2.5 Sexual selection and other processes

Sexual selection and sex ratio evolution

Trivers & Willard (1973) predicted that females mated to attractive males should bias their sex allocation towards sons. Formal models indeed confirm this (Pen & Weissing, 2000c; Fawcett *et al.*, 2007) but also find that females mated to unattractive males overproduce daughters. Moreover, a more inclusive coevolutionary model in which sex allocation feeds back on the evolution of ornaments and preferences shows that sex allocation undermines sexual selection: by producing only daughters (which are certain to reproduce) but no unattractive sons, the fitness of females mated to unattractive males approaches that of females mated to attractive males. As a result, mating with attractive males ceases to yield any advantage to choosy females, eliminating sexual selection and sex allocation based upon it (Fawcett *et al.*, 2011). All in all, the latter example is a clear demonstration of how more inclusive coevolutionary models can change our insights. It now remains to be seen if there are any circumstances in which sex allocation based on male attractiveness can be maintained in the presence of mate choice, for example in case sex allocation is based on sexually antagonistic alleles (Alonzo & Sinervo, 2007; Blackburn *et al.*, 2010).

Sexual selection and parental care

Conventional sexual selection models assume females that provide care, whereas males compete over females. Although female parental care is indeed more widespread than male parental care, substantial taxonomic variation in sex biases in parental care exist, which is poorly explained by conventional theory (Kokko & Jennions, 2008). Modeling the evolution of parental care is complicated, since the decision of one parent to provide care versus to compete for matings depends not only on the behavior of its current partner, but also on the opportunity to gain future matings, which in turn, is a function of the population wide density of members of its sex that are competing for matings as opposed to caring (Houston *et al.*, 2005; Kokko & Jennions, 2008). To this end, more inclusive modeling approaches are required that take into account both the evolutionary dynamics (evolution of ornaments, preferences, care decisions) as well as the 'ecological' dynamics (acting at a much faster timescale) in which individual decisions feed back on the densities of caring and competing individuals.

One of the first models to take such a dynamical approach is one by (McNamara *et al.*, 2000). They point out that high population-wide levels of care select individuals to desert their brood more rapidly (since they are likely to have mated with a partner providing care) and pursue more matings. However, the more individuals desert, the more competition they face to attain any future matings, making it again less worthwhile to desert and to continue caring. They show that this can lead to alternations in patterns of care, where bouts of biparental care can be alternated by uniparental care or even no care at all. The feedback between deserting and competition also points out that one should be careful with attributing sex differences in care to biases in the operational sex ratio[10] (OSR): a typical example are male-biased OSRs, which are widely assumed to lead to increased male-male competition and hence less

[10] **Operational sex ratio (OSR)**: the ratio of male versus female individuals that are available for mating at any given time.

male care (Trivers, 1972). As reviewed by Kokko & Jennions (2008), a male-biased OSR itself would in fact select for increased parental care by males, since they now face increased competition over matings, increasing the relative value of parental care. Hence, the co-occurrence of male-biased OSRs and female biased patterns of parental care thus require more specific explanations, relating to the intricacies of the sexual selection process or species-specific differences in costs of competition.

Sexual selection and the evolution of sex

Despite recent theoretical progress on the evolution of sex along various lines, we still have a limited understanding of the various factors that can overcome the costs of sexual reproduction (Lehtonen *et al.*, 2012). Sexual selection, itself requiring sexual reproduction, may positively feedback on the maintenance of sex (for a recent review, see Whitlock & Agrawal, 2009). Specifically, if females choose those males that have the fewest mutations, this reduces the effective 'load' of deleterious mutations compared to asexual populations (Siller, 2001; Agrawal, 2001). In addition, the two-fold cost of sex is also overcome for those individuals that bear high-fitness offspring (e.g., attractive sons), which will result in a larger number of grandoffspring in comparison to asexual individuals (Hadany & Beker, 2007). It is obvious, however, that sexual selection may also work against the evolution of sex (Whitlock & Agrawal, 2009). For example, mean fitness can be reduced in sexual populations compared to asexual populations, due to the evolution of male harm or costly competition. Next to that, the increased variance in male reproductive success reduces the expected population size compared to asexual organisms, which may lead to an increased load of deleterious mutations due to drift. Lastly, the presence of divergent selection pressures in each sex (i.e., sexually antagonistic selection) has also been shown to disfavor sexual reproduction (Roze & Otto, 2012). To answer the question if sexual selection indeed defies the evolution of asexual reproduction, a more inclusive approach will be required, perhaps tailored to a specific model system, that takes this multitude of factors into account.

Sexual selection and speciation

Closely related species often differ most dramatically in the appearance of mating traits, suggesting that sexual selection plays an important role in speciation. Indeed sexual selection has been ascribed a prominent role in virtually all processes related to speciation. Already Lande (1981) noticed that the Fisher process can contribute to the divergence of mating preferences in geographically isolated populations and, hence, to the evolution of reproductive isolation mechanisms (Uyeda *et al.*, 2009). Such divergence can be strongly enhanced when the driving force is sexual conflict, leading to antagonistic coevolution of the two sexes (Hayashi *et al.*, 2007). Sexual selection can contribute to the low fitness of hybrids (and, hence, postzygotic reproductive isolation) if hybrids are less attractive as mates. Mating preferences can be a driving force of reinforcement (selection against the occurrence of deleterious hybridization) if they evolve due to the advantages of mating with genetically compatible partners (Servedio & Noor, 2003; Servedio, 2004, Section 2.3.5). In fact, reinforcement can give rise to particularly strong selection on female preferences for signals that indicate low degrees of hybrid incompatibility (Kirkpatrick & Servedio, 1999). Last but not least, sexual selection can lead to the evolution of prezygotic

isolation. This may even happen in sympatry, when two diverging Fisher processes co-occur in populations with a broad variation in female preferences (Higashi *et al.*, 1999; Van Doorn *et al.*, 2001; Van Doorn & Weissing, 2004). A more likely scenario is the evolution of preferences for indicators of local adaptation (Proulx, 2001; Reinhold, 2004, Section 2.3.5), which under parapatric conditions can strongly enhance disruptive natural selection (Van Doorn *et al.*, 2009). The role of sexual selection in speciation was recently reviewed by Ritchie (2007), Weissing *et al.* (2011) and the Marie Curie Speciation Network (2012).

Future directions

Despite the hundreds of models on sexual selection over the last four decades, we feel that we are only at the beginning of a more integrative theory of sexual selection. In particular, the following aspects should receive major attention in future models:

- Mating traits are typically modeled in a rather simplistic manner. In nature, preferences as well as traits will often be conditional strategies, depending on an individual's position on the mating market. There are virtually no models considering the possibility that individuals re-allocate resources from ornamentation to the provisioning of direct benefits like parental care (or vice versa) in a dynamic way. This may be of particular relevance for species with mutual mate choice. Also the evolution of preferences when different types of benefits are at stake has not received much theoretical attention. Current models (e.g., Kirkpatrick & Barton, 1997) lead to the clear-cut prediction that direct benefits should play a more prominent role than indirect benefits. Why then do many females engage in extra- pair copulations (yielding only indirect benefits), while this may endanger their social mate's investment in their joint clutch (hence risking the loss of direct benefits)? Questions like these have hardly been touched upon by sexual selection theory.

- The interplay between natural and sexual selection is still poorly understood: Are mating preferences and natural selection typically antagonistic (as often envisaged) or do they more often act in concert (Proulx, 2001; Van Doorn *et al.*, 2009)? How do ecological conditions affect the perception (and evolution) of sexually selected signals (Endler & Basolo, 1998)? Under which conditions do preferences evolve as a pleiotropic by-product of natural selection (Kirkpatrick & Ryan, 1991) and will such preferences be maintained in the long term? How do more explicit formulations of ecological interactions (predation, host-parasite interactions, Hamilton & Zuk, 1982) and environmental dynamics influence male quality and the benefits of choice?

- Inclusive models should take center stage in an integrative theory of sexual selection, in which ecological aspects (i.e., density-dependent feedbacks, resource dynamics) are modeled dynamically in combination with evolving preferences and ornaments. In addition, models are required that explicitly model the co-evolutionary interaction of female preferences, male ornaments and traits involved in other processes, such as parental care, sex allocation, and male-male competition.

- Most of the current predictions on the evolution of sexually selected traits are based on equilibrium situations, whereas several lines of evidence lead to the prediction that sexually selected traits exhibit rapid turnovers and strong inter-population divergence (Wiens, 2001; Bro-Jørgensen, 2010). More effort is needed to understand when non-equilibrium dynamics occur and how aspects such as the genetic architecture (e.g., Van Doorn & Weissing, 2006) or environmental dynamics (Bro-Jørgensen, 2010) affect the continuous evolution of sexually selected traits.

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S2 Supplement: Four implementations of the Fisher process

A population genetics model of the Fisher process

Genetic assumptions The model of Kirkpatrick (1982b) works with two haploid diallelic loci, of which P is the preference locus, with alleles P_0 (no preference) and P_1 (preference for ornamented males) and T the ornament locus, with alleles T_0 (no ornamentation) and T_1 (ornamentation). The evolutionary dynamics can be described by keeping track of the changes of the relative frequencies x_{ij} of the haploid genotypes ('haplotypes') P_iT_j. It is often more convenient to describe the dynamics in terms of allele frequencies, that is the frequencies $p = x_{10} + x_{11}$ of the preference allele P_1 and $t = x_{01} + x_{11}$ of the ornament allele T_1. For a complete description, one also has to keep track of linkage disequilibrium $D = x_{00}x_{11} - x_{01}x_{10}$. In fact, the four haplotype frequencies x_{ij} can be recovered from p, t and D. In statistical terms, linkage disequilibrium corresponds to the covariance of the alleles with identical subscripts: D is positive if P_1 and T_1 or P_0 and T_0 co-occur more often in the same individual than to be expected on basis of the allele frequencies p and t. The build-up of a positive association between P_1 and T_1 (a positive D) is *the* key ingredient of the Fisher process. To derive the evolutionary dynamics of p, t and D, we closely follow Bulmer (1989).

Viability selection The costs of expressing an ornament are assumed to be incurred during a period of viability selection, which precedes the mating stage. Ornamented males have a relative survival probability of $v_1 = 1 - s$ $(0 < s < 1)$ in comparison to the viability $v_0 = 1$ of males without ornamentation. Since ornaments are sex-limited, only males incur costs (see Seger & Trivers (1986); Albert & Otto (2005), where this assumption is relaxed). Costs of female preference are not considered in Kirkpatrick's (1982b) model. Viability selection changes the frequency of ornamented males from t to $t_m = t \cdot v_1/\bar{v}$, where $\bar{v} = (1-t)v_0 + tv_1 = 1 - st$ is the mean viability of males.

Mating stage We define U_{ij} as the probability that a female with allele P_i mates with a male carrying allele T_j. P_0 females mate at random, so that the probability of mating with T_0 and T_1 males is identical to their respective frequency in the population, $U_{00} = 1 - t_m$ and $U_{01} = t_m$. In contrast, for females carrying the P_1 allele the odds are $a > 1$ that she prefers a T_1 male over a T_0 male, so that $U_{11} : U_{10} = at_m : (1 - t_m)$. This way of exerting mate choice is known as 'fixed relative preferences'. It corresponds, for example, to a situation where females encounter males one at a time in a random sequence, until they accept a male for mating (Maynard Smith, 1985). Other mechanisms of mate choice can lead to very different outcomes (e.g., the 'best-of-N' model, Seger, 1985, or the 'absolute preference' model, Takahasi, 1997. Kirpatrick's model also assumes that all choosy females will eventually mate ($U_{11} + U_{10} = 1$). Together with the above condition on $U_{11} : U_{10}$ this yields $U_{10} = (1 - t_m)/(1 - t_m + at_m)$ and $U_{11} = at_m/(1 - t_m + at_m)$.

Evolutionary dynamics Once the frequency distribution of the various types of mating are known, the distribution of offspring genotypes can be derived in a standard way (taking into account recombination and Mendelian segregation). As shown

in Bulmer (1989), this leads to the following system of difference equations describing how the allele frequencies p and t and the linkage disequilibrium D change from one generation to the next:

$$\begin{aligned}\Delta t &= \frac{1}{2} t(1-t)A \\ \Delta p &= \frac{1}{2} DA \\ \Delta D &= D\left((1-r)A(\frac{1}{2}-t) - \frac{1}{4}A^2 t(1-t) - r\right) \\ &\quad + \frac{1}{2} rB\left(D^2 + p(1-p)t(1-t)\right),\end{aligned} \tag{S2.1}$$

where r is the recombination rate between the trait and the preference locus and A and B are defined as:

$$A = \frac{p\left(U_{11} - U_{01}\right) - \left(t - t_m\right)}{t(1-t)}, \quad B = \frac{U_{11} - U_{01}}{t(1-t)}. \tag{S2.2}$$

The factor $\frac{1}{2}$ in the first two equations of (S2.1) reflects the fact that preference and ornament are only expressed in one sex. The first equation of (S2.1) shows that there is direct selection on the ornament, which is characterized by the term A. This term includes two parts, corresponding to the mating advantage $p(U_{11} - U_{01})$ of ornamented males and the decline $t - t_m$ in ornament frequency in males due to natural selection. The second equation of (S2.1) shows that the preference allele changes in frequency as a correlated response to selection on the ornament allele: once there is a positive linkage disequilibrium D, p changes in the same direction as t. Hence, (S2.1) captures the essential features of the Fisher process.

Equilibria Solving for the equilibria by setting $\Delta t = 0$ and $\Delta p = 0$, one finds a set of boundary equilibria that constitute either loss of the ornament ($t = 0$), or fixation of the ornament ($t = 1$). Internal equilibria have to satisfy $A = 0$. A straightforward calculation yields:

$$A = pB - \frac{s}{1-st}, \quad B = \frac{1-s}{1-st} \cdot \frac{a-1}{1-t+a(1-s)t}. \tag{S2.3}$$

This implies that the equation $A = 0$ is equivalent to

$$p = s\frac{1 + (a(1-s) - 1)\, t}{(1-s)(a-1)} \tag{S2.4}$$

and that the internal equilibria constitute a line with a slope determined by the interplay of natural and sexual selection. For a given combination (p, t) on this line of equilibria, the equilibrium value of D can be obtained by solving $\Delta D = 0$, a quadratic equation in D. Since $A = 0$, this equation can be simplified considerably. It turns out that the solution of $\Delta D = 0$ does not depend on the recombination rate r. In other words, at equilibrium the statistical association between preference and ornament alleles does not depend on physical linkage. Figure 2.2A in the main text depicts the

line of equilibria and the dynamic behavior of Kirkpatrick's model. As predicted by Fisher (1915) the preference allele P_1 has to be sufficiently common initially to give rise to nonzero frequencies of the ornament allele T_1 (although this depends on the preference function, Takahasi, 1997). Once P_1 is sufficiently common it either gives rise to polymorphism in which both T_0 and T_1 alleles coexist or to fixation of the T_1 allele. Notice that the approach to the set of equilibria is roughly linear. The slope of the 'lines of approach' is affected by the 'genetic' parameter r. In contrast, the line of equilibria itself only depends on the 'fitness' parameters s and a.

Costly preferences As already noted by Kirkpatrick (1982b) and further investigated by Pomiankowski (1987a) and Bulmer (1989), the line of equilibria in Figure 2.2A is extremely sensitive to small deviations in the model assumptions. For example, the addition of the slightest costs of a female preference leads to a breakdown of the line of equilibria to a single equilibrium point in which mate choice is absent ($p = 0,\ t = 0$). Additional assumptions (like mutation bias) are then needed to explain the evolution of costly ornaments and preferences (see Figure 2.3 in the main text).

A quantitative genetics model of the Fisher process

Lande's (1981) quantitative genetics implementation of the Fisher process assumes that the female preference p and the male ornament t are continuous, normally distributed characters.

Viability selection As in the population genetics model considered above, the costs of expressing an ornament are assumed to be incurred during a period of viability selection. The survival probability $v(t)$ of a male carrying an ornament t is given by a Gaussian function

$$v(t) = \exp\left[-\frac{1}{2}\left(\frac{t-\theta}{\omega}\right)^2\right]. \tag{S2.5}$$

Hence selection is stabilizing, and each deviation from the optimal ornament value θ (with respect to viability selection) leads to a decrease in survival. The smaller the term ω the stronger deviations from θ are punished by natural selection. Hence $1/\omega$ reflects the strength of viability selection against exaggerated ornaments. Exponential fitness functions like the Gaussian above are popular in quantitative genetics models, since the distribution of traits (here: male ornaments) after selection is again normal. Like Kirkpatrick's model, Lande's model does not consider costs of female choosiness.

Mating stage The female "preference" is any character leading to non-random mating with respect to the male ornament. The tendency of a female with preference p to mate with an adult male with ornament value t is given by a preference function $\psi(t|p)$. Lande (1981) discusses three different preference functions, but here we focus on one of them: $\psi(t|p) = \exp(apt)$. In this 'psychophysical model', females with $p \neq 0$ always prefer the most extreme males most; the sign of p determines the di-

rection of the preference and the magnitude of p determines how strongly a female discriminates between males differing in ornamentation.

Evolutionary dynamics It is one of the basic insights of quantitative genetics theory that the evolution of the mean values of two sex-limited traits in a population with discrete, non-overlapping generations can be described by the 'multivariate breeder's equation' (Lande & Arnold, 1983). There are various version of this equation that differ in their description of selection and their assumptions on the multivariate distribution of traits (Walsh & Lynch, 2012). Here we follow the approach of Pomiankowski *et al.* (1991) that is relatively broadly applicable in case of weak selection:

$$\begin{bmatrix} \Delta\bar{t} \\ \Delta\bar{p} \end{bmatrix} = \frac{1}{2} \begin{bmatrix} G_t & G_{tp} \\ G_{tp} & G_p \end{bmatrix} \begin{bmatrix} \partial \ln\left(W_m\right)/\partial t \\ \partial \ln\left(W_f\right)/\partial p \end{bmatrix} \Bigg|_{t=\bar{t},p=\bar{p}} \tag{S2.6}$$

The matrix in (S2.6) is the so-called G-matrix, consisting of the additive genetic variances G_t and G_p of male traits and female preferences and the additive genetic covariance describing the (additive) genetic association between trait and preference. The vector to the right is the gradient vector of (relative) individual fitness (male fitness W_m and female fitness W_f) with respect to the individual trait values. The factor $\frac{1}{2}$ reflects the sex-limited expression of traits and preferences.

In Lande's model, the net effect of viability selection and mating preferences on the ornament trait is given by

$$\frac{\partial \ln\left(W_m\right)}{\partial t}\Bigg|_{t=\bar{t},p=\bar{p}} = a\bar{p} - \frac{\bar{t}-\theta}{\omega^2}. \tag{S2.7}$$

In the absence of direct selection on female preferences (no costs of choosiness), $\partial \ln(W_f)/\partial p = 0$, and (S2.6) can be written as

$$\begin{aligned} \Delta\bar{t} &= \frac{1}{2} G_t \left(a\bar{p} - \frac{\bar{t}-\theta}{\omega^2} \right) \\ \Delta\bar{p} &= \frac{1}{2} G_{tp} \left(a\bar{p} - \frac{\bar{t}-\theta}{\omega^2} \right). \end{aligned} \tag{S2.8}$$

Comparing (S2.8) with (S2.1) shows a close correspondence between Kirkpatrick's and Lande's models. If we assign the numerical values 0 and 1 to the alleles P_0 and P_1 and to T_0 and T_1 in Kirkpatrick's model, the allele frequencies p and t in this model correspond to the averages $\bar{p}$ and $\bar{t}$ of these numerical values; the term $t(1-t)$ in the first equation of (S2.1) corresponds to the variance (G_t) in t-values; and the linkage disequilibrium D corresponds to the covariance (G_{tp}) between p- and t-values. Finally, the term A in (A1) corresponds to $\partial \ln(W_m)/\partial t$ in Lande's model.

Equilibria The equilibria of Lande's model ($\Delta\bar{t} = \Delta\bar{p} = 0$) are given by

$$\bar{p} = \frac{\bar{t}-\theta}{a\omega^2}. \tag{S2.9}$$

Hence when plotting the mean preference $\bar{p}$ against the mean trait value $\bar{t}$ a line of equilibria results with slope $1/a\omega^2$ (see Figure 2.2B in the main text). If G_t and G_{tp} are constant, the evolutionary trajectories are straight lines with slope

$$b_{pt} = \frac{\Delta\bar{p}}{\Delta\bar{t}} = \frac{G_{pt}}{G_t}, \tag{S2.10}$$

corresponding to the (additive) genetic regression coefficient of the preference on the trait. If this regression is weak (more precisely: if the regression line is less steep than line of equilibria, or $G_{tp}/G_t < 1/a\omega^2$), then evolution will proceed towards the line of equilibria (as in Figure 2.2B of the main text). If the genetic regression is strong, any positive selection on t quickly increases the level of p as well. In this case a self-reinforcing runaway process occurs and evolution will proceed away from the line of equilibria.

Costly preferences Any costs of female preferences will strongly change the dynamics of Lande's model. The line of equilibria collapses to a single equilibrium point, located at the values maximizing male and female survival (i.e., $\bar{p} = \theta_p$, $\bar{t} = \theta_t$). As in the previous section, additional assumptions (like mutation bias) are needed to explain the evolution of costly ornaments and preferences (see Figure 2.3 in the main text).

An adaptive dynamics model of the Fisher process

Over the years several sexual selection models have been developed that make use of evolutionary game theory or adaptive dynamics (Pen & Weissing, 2000c; Kokko *et al.*, 2002; McNamara *et al.*, 2003; Fawcett *et al.*, 2011). All these models have in common that they consider the invasion prospects of a rare mutant phenotype in an otherwise monomorphic 'resident' population.

Here, we discuss Pen and Weissing's (2000c) model for the Fisher process. This model considers a class-structured population consisting of females and two types of males: non-ornamented males ($♂_0$) and males expressing an ornament ($♂_1$). Evolvable traits are the females' preference p for mating with ornamented males and the tendency t of a male to develop the ornament, that is, the probability to develop into a male of type 1. The aim is to find evolutionarily stable values p^* and t^* that cannot be invaded by any mutant phenotypes.

Viability selection The survival probability of ornamented males is reduced by a factor $1 - s$ when compared to non-ornamented males. Males that express an ornament survive with probability $1 - c$, whereas males without an ornament survive with probability 1. Costs of female preferences are not considered in Pen & Weissing's model but included in later extensions (Fawcett *et al.*, 2011).

Mating stage Females have a certain preference p for type 1 males, which translates into them giving a proportion α of their matings to type 1 males, $\alpha = \alpha(p)$ being an increasing function of p. For mutants and residents alike, the number of matings per type of male depends on the preference p^* of the females and on the relative frequency of the two types of (adult) males in the resident population. This follows from the

assumption that mutant females and males are rare. Let u_f^*, u_{m0}^* and u_{m1}^* denote the density of females, type 0 males and type 1 males in the resident population, respectively. Then the per capita number of matings per type of male, Q_0^* and Q_1^*, can be derived from the consistency requirements $Q_0^* u_{m0}^* = (1-\alpha^*)u_f^*$ and $Q_1^* u_{m1}^* = \alpha^* u_f^*$, where $\alpha^* = \alpha(p^*)$:

$$Q_0^* = \frac{(1-\alpha^*)u_f^*}{u_{m0}^*}, \quad Q_1^* = \frac{\alpha^* u_f^*}{u_{m1}^*}. \tag{S2.11}$$

Stage-transition matrix Assuming non-overlapping generations and a stationary resident population producing an even primary sex ratio (i.e., each female producing on average one male and one female offspring), the transitions between the different classes of mutant individuals from one generation to the next are summarized by the stage-transition matrix

$$\mathbf{A}\left(p,t|p^*,t^*\right) = \frac{1}{2}\cdot\begin{bmatrix} 1 & Q_0^* & Q_1^* \\ 1-t & (1-t)Q_0^* & (1-t)Q_1^* \\ (1-s)t & (1-s)tQ_0^* & (1-s)\,tQ_1^* \end{bmatrix}. \tag{S2.12}$$

This matrix should be interpreted as follows. The first column characterizes the per capita contribution of a mutant female to female mutants, type 0 male mutants and type 1 male mutants in the next generation, respectively. The first element is equal to 1, since a female produces on average one (surviving) female offspring. In addition, the female also produces on average one male offspring. With probability $1-t$ this male will be of type 0 (no ornamentation) and survive to adulthood. With probability t, the male will be of type 1 and survive with probability $1-s$ to adulthood. The other two columns correspond to the contributions of type 0 and type 1 mutant males to the various types of mutants in the next generation. These columns correspond to column 1 multiplied by the per capita number of matings (Q_0^* and Q_1^*) of the two types of male. The factor $\frac{1}{2}$ in (S2.12) reflects the fact that each individual has one father and one mother and prevents double counting of offspring. Notice that the matrix elements do not depend on p, and that the dependence on p^* and t^* is indirect (via Q_0^* and Q_1^*).

Invasion fitness The dominant eigenvalue $W(p,t|p^*,t^*)$ of the stage transition matrix $\mathbf{A}$ corresponds to the 'invasion fitness' of the rare mutant. For the resident population, the dominant eigenvalue is equal to one (reflecting the fact that this population is stationary). If $W>1$, the mutant will increase in relative frequency, while it will go extinct if $W<1$. To determine the properties of the invasion fitness function, we first have to investigate the stage transition matrix $\mathbf{A}_{res} = \mathbf{A}(p^*,t^*|p^*,t^*)$ of the resident population. The right eigenvector of the dominant eigenvalue 1 gives the stable distribution of the three types of individuals in the resident population (which via (S2.11) affect Q_0^* and Q_1^*):

$$u_f^* : u_{m0}^* : u_{m1}^* = 1 : (1-t^*) : (1-st^*). \tag{S2.13}$$

The left eigenvector of $\mathbf{A}_{res}$ corresponds to the reproductive values of the three types:

$$v_f^* : v_{m0}^* : v_{m1}^* = 1 : Q_0^* : Q_1^*. \tag{S2.14}$$

Using a standard result of life history theory (Taylor, 1996a), the dependence of $W(p,t|p^*,t^*)$ on p and t can now be determined without actually calculating invasion fitness (which can be quite tedious). In fact, the partial derivatives of W at (p^*,t^*) are of the form:

$$\frac{\partial W(p,t|p^*,t^*)}{\partial t} = \mathbf{v}^{*\mathrm{T}} \frac{\partial \mathbf{A}}{\partial t} \mathbf{u}^* / \mathbf{v}^{*\mathrm{T}} \mathbf{u}^*. \tag{S2.15}$$

Inserting (S2.13), (S2.14) and the partial derivatives of (S2.12) into this equation yields:

$$\left.\frac{\partial W}{\partial t}\right|_{t=t^*} = (1-s)Q_1^* - Q_0^*, \quad \left.\frac{\partial W}{\partial p}\right|_{p=p^*} = 0. \tag{S2.16}$$

The total selection differentials are then given by

$$\begin{aligned} \left.\frac{\mathrm{d}W}{\mathrm{d}t}\right|_{t=t^*} &= \left(\frac{\partial W}{\partial t} + b_{tp}\frac{\partial W}{\partial p}\right)_{t=t^*} = (1-s)Q_1^* - Q_0^* \\ \left.\frac{\mathrm{d}W}{\mathrm{d}p}\right|_{p=p^*} &= \left(\frac{\partial W}{\partial t} + b_{tp}\frac{\partial W}{\partial p}\right)_{t=t^*} = b_{pt}\left((1-s)Q_1^* - Q_0^*\right), \end{aligned} \tag{S2.17}$$

where b_{yx} is the statistical regression coefficient of y on x (Taylor & Frank, 1996), which is assumed to be a positive parameter.

Evolutionarily singular strategies At an internal 'equilibrium' both selection differentials in (S2.17) have to be zero, leading to the condition $(1-s)Q_1^* = Q_0^*$, which can be simplified to $t^* = \alpha(p^*)$. Once again, this condition describes a whole set of equilibria. For many preferences, this set is again a straight line. Assume, for example, that females have 'fixed relative preferences' as in Kirkpatrick's model with probability p^* while they mate at random with probability $1-p^*$. Then $\alpha(p^*)$ is given by

$$\alpha(p^*) = p^* \frac{a u_{m1}^*}{u_{m0}^* + a u_{m1}^*} + (1-p^*)\frac{u_{m1}^*}{u_{m0}^* + u_{m1}^*}. \tag{S2.18}$$

Equating this expression with t^* yields the same line of equilibria as in Kirkpatrick's model (see Figure 2.2C in the main text). The approach to equilibrium (via a sequence of gene substitution events), which is also indicated in Figure 2.2C, can be derived from the canonical equation of adaptive dynamics theory (Geritz *et al.*, 1998).

Individual-based simulation models of the Fisher process

Individual-based models of sexual selection are the most recent addition to the set of tools to analyze evolutionary models (Grimm & Railsback, 2005); a growing number

of sexual selection models makes use of this versatile technique (e.g., Lorch *et al.*, 2003; Gavrilets *et al.*, 2007; Kokko *et al.*, 2007; Fawcett *et al.*, 2007). Here, we discuss an individual-based model on Fisherian sexual selection that is used by Fawcett *et al.* (2007). The source code of this simulation can be found at `http://www.rug.nl/biol/theobio/fisher.cpp`. A flow diagram of the model is given in Figure S2.1.

Initialization An individual-based simulation starts with a definition of the individuals initially present and their properties. For sake of comparison to the previous analytical models, we assume that individuals are genetically characterized by their alleles at two haploid loci, one coding for a male ornament t and the other coding for a female preference p. However, we emphasize that individual-based simulations easily allow for the implementation of complex genetic architectures and genotype-phenotype maps (e.g., Ten Tusscher & Hogeweg, 2009). The population is initialized by generating a large collection of males and females (say, $n = 2000$ males and $n = 2000$ females) and randomly assigning ornament and preference alleles to each individual according to some pre-specified distribution (e.g. a bivariate normal distribution) (see Figure S2.1, step 1).

Viability selection Subsequently, the population of males enter a procedure that determines their survival (see Figure S2.1, step 2), whereas all females survive. The simulation assumes that a male's survival probability v is given by a Gaussian function $v(t) = \exp(-ct^2)$, where the parameter c determines the strength of viability selection. However, in contrast to the aforementioned deterministic models, survival is implemented in a stochastic fashion (i.e., by letting a chance process decide whether a given male will die or survive). In Figure S2.1 (step 2) males with larger ornaments are more likely to die, but individual males may, by chance, escape mortality.

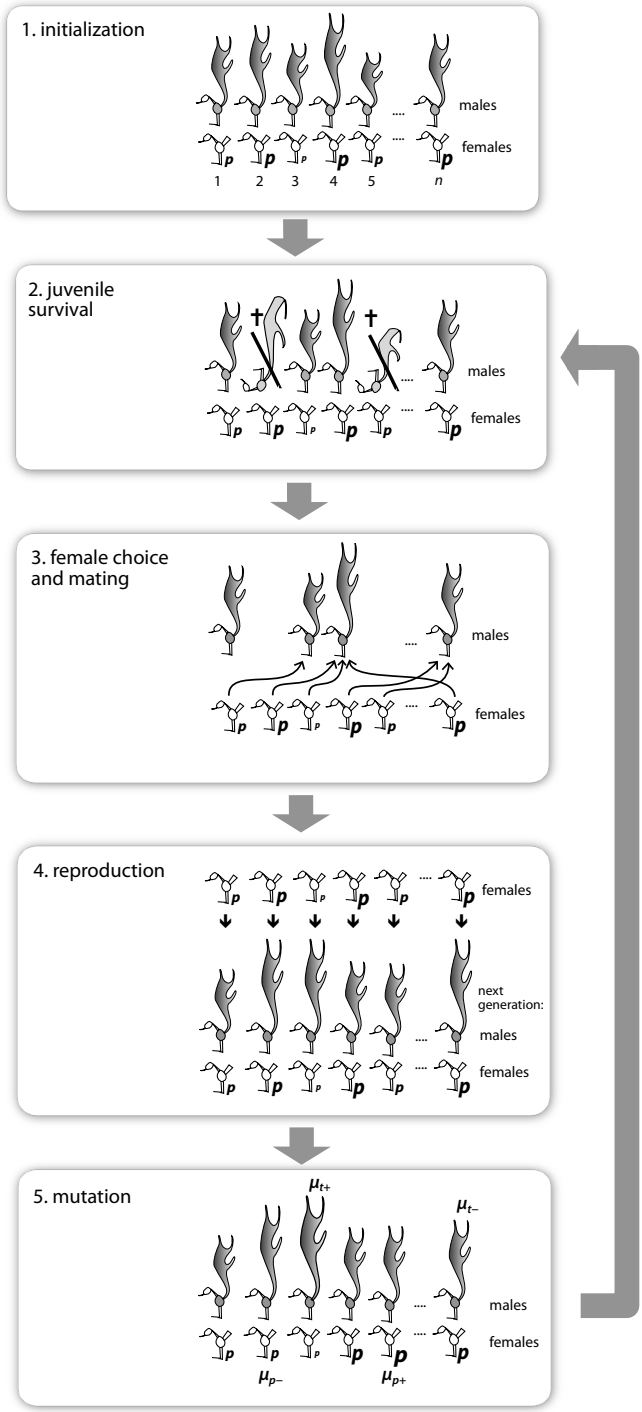
Mating stage The surviving males and all females then enter another procedure, in which females choose mates. As in the deterministic models, female mate choice can be implemented in various ways. For example, fixed relative preferences can be implemented by sequentially assigning randomly drawn males to each female, until one of these males is accepted by the female for mating. The probability of accepting any given male depends on both, the female's preference and the male's ornament. Alternatively, each female is confronted with a random sample of N males and subsequently mates with the male most closely fitting to her preference ('best-of-N' model). A third alternative (implemented by Fawcett *et al.*, 2007) combines the two approaches above: each female samples N males and subsequently mates with male i with a probability that is proportional to i's attractiveness value to the female. Figure S2.1 step 3 shows that large ornamented males are on average more often chosen by females with high values of the preference, but stochastic variation in mate sampling and mate choice reduces the strength of assortative mating to a certain extent.

Reproduction and mutation After having chosen a mate females reproduce, having their offspring sired by their mate of choice. In the simulation model considered here, all females contribute effectively two offspring to the next generation, at a 1:1 sex ratio (see Figure S2.1, step 4). As a consequence, the population size remains

Figure S2.1: (see next page) Flow diagram of an individual-based simulation, reflecting a population of Arnold's bird of paradise (Arnold, 1985) that experiences the Fisher process. Step 1: the population is initialized by generating a population of n males (that vary in tail length) and n females (that vary in their preferences for tail length, p). Step 2: male viability selection takes place, in which males with the smaller ornaments are most likely (but not certain) to survive. Step 3: female choices takes place based on ornaments and preferences (see Supplement, Section S2.4) and the eventual choices are indicated by black arrows. Males with large ornaments are likely (but not certain) to achieve a high mating rate. Step 4: after fertilization by their male of choice, females reproduce and give rise to the next generation of males and females. Step 5: mutation events can take place (indicated by μ) that can either increase or decrease values of ornaments and preferences (indicated by $+$ or $-$). After mutation, the next cycle of the program starts again at step 2.

constant over the generations. Upon reproduction, mutation takes place (see Figure S2.1, step 5). The current model assumes a continuum-of-alleles model of mutation, in which preference and ornament alleles present in an individual mutate with probabilities μ_p and μ_t respectively. When mutation takes place, a deviate from a normal distribution with mean 0 and standard deviation σ_μ is added to the current allelic value. The life cycle then repeats itself and males of the next generation enter the juvenile survival stage.

Evolutionary dynamics Figure 2.2D in the main text illustrates the course of evolution of an individual-based simulation. For similar parameter values as in Lande's model (Figure 2.2B), the system converges to Lande's line of equilibria and subsequently 'drifts' along this line. The irregularities in the evolutionary trajectory clearly illustrate the stochastic nature of an individual-based simulation.


1. initialization
males
females
1
2
3
4
5
n
2. juvenile survival
3. female choice and mating
4. reproduction
next generation:
5. mutation
μ_{t+}
μ_{t-}
μ_{p-}
μ_{p+}

CHAPTER 3

Fisherian sexual selection in simultaneous hermaphrodites

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Abstract

It has often been argued that sexual selection is inherently weaker in simultaneous hermaphrodites than in species with separate sexes, but this claim has received little formal investigation. Here, we compare the potential for evolution of costly preferences between gonochorists and simultaneous hermaphrodites in a scenario of Fisherian sexual selection. We compare female and mutual choice in separate-sexed organisms as well as unilateral and mutual choice in hermaphrodites. Additionally, we investigate the impact of reciprocal insemination in hermaphrodites. We confirm that hermaphrodites that exhibit unilateral choice are generally less likely to develop sexually exaggerated characters when compared to separate-sexed organisms that have female choice. In contrast, when choice is mutual, hermaphrodites and separate-sexed organisms do not differ in their propensities to develop exaggerated sexually selected characters. Due to increased assortative mating in scenarios of mutual choice, exaggeration under mutual choice is possible for a far wider range of parameters than for any of the other scenarios considered. In addition, we show that reciprocal transfer of gametes has no effect on the strength of sexual selection. As such, the nature of choice (mutual versus unilateral) may be an important determinant in understanding cases where sexual selection in hermaphrodites leads to exaggeration of sexually selected characters.

3.1 Introduction

The question why mate choice has evolved is a subject of a substantial body of both empirical and theoretical studies (see Andersson, 1994; Kokko *et al.*, 2006; Andersson & Simmons, 2006, chapter 2 of this thesis). Traditionally, studies have mainly concentrated on indirect benefits of mate choice, either through the production of attractive sons (the Fisherian sexy-sons mechanism: Fisher 1930) or offspring of higher genetic quality (good-genes: Zahavi 1975). Later developments stressed the importance of direct benefits of female choice, for example through male resources or nest protection (e.g., Price *et al.*, 1993; Iwasa & Pomiankowski, 1999), or interlocus sexual conflict between male persistence and female resistance traits (Gavrilets *et al.*, 2001; Rowe *et al.*, 2005). For all these different benefits of mate choice, theoretical models have been developed that have resulted in a set of testable predictions (e.g., Fuller *et al.*, 2005; Kokko *et al.*, 2006), followed by numerous studies in empirical settings (Andersson & Simmons, 2006).

Almost all of the effort mentioned above, however, concentrates on sexual selection in separate-sexed organisms (hereafter: gonochorists), whereas sexual selection in hermaphrodites has received considerably less attention (Arnqvist & Rowe, 2005; Anthes *et al.*, 2010). The fact that hermaphrodites unite both sex functions in a single organism has a number of interesting consequences that preclude a direct application of sexual selection theory developed for gonochorists. Typical coevolutionary models for gonochorists focus on the coevolution of costly male ornaments and costly female preferences, which are typically assumed to be expressed in a sex-limited fashion (Pomiankowski *et al.*, 1991; Hall *et al.*, 2000; Kuijper *et al.*, 2012, but see Lande & Arnold 1985). Here, ornaments typically refer to traits that increase a male's mating or fertilization success over that of other males and which are typically assumed to be costly in terms of survival (e.g., Cuervo *et al.*, 1996; Kotiaho *et al.*, 2001; Godin & McDonough, 2003; Hine *et al.*, 2011). Mating preferences generally refer to the female propensity of choosing certain male phenotypes more often than others (Heisler *et al.*, 1987) and such traits may impose costs in terms of search effort (Alatalo *et al.*, 1988; Gray, 1999; Alem & Greenfield, 2010). Due to the typical assumption of sex-limited expression in gonochorists, costs of ornaments and preferences are only borne by one particular sex. By contrast, since hermaphrodites express both sex functions, it will be more difficult (although not impossible, Abbott, 2011) to achieve sex limited expression, implying that a single individual may likely bear the costs of *both* ornaments and preferences during its lifetime (Morgan, 1994; Arnqvist & Rowe, 2005).

A second unique feature of hermaphrodites is that the expression of both sexes within one and the same individual can potentially lead to individual trade-offs between reproductive success through the male versus the female function: investment by a hermaphroditic individual in a trait that increases its reproductive success through the male role may be at the expense of reproductive success through the female role (Charnov, 1979b; Schärer, 2009; Anthes *et al.*, 2010), which is a trade-off that is not present in gonochorists. Additionally, the presence of both sexes in an individual has given rise to a number of interesting mating behaviours, such as a reciprocal exchange of sperm and/or eggs between members of a mating pair (Anthes *et al.*, 2006b; Anthes, 2010), or potential conflicts between members of a mating pair on which sex role to exhibit during mating (Charnov, 1979b; Michiels, 1998; Anthes *et al.*, 2010). Hence, it is currently far from clear if conclusions derived from conven-

tional sexual selection theory in gonochorists can be extrapolated to hermaphrodites.

In this spirit, a realization is growing that the unique features mentioned above as well as other aspects of hermaphroditic lifestyles deserve more attention from a theoretical perspective (Anthes *et al.*, 2010). This is not only because hermaphroditism occurs in nearly 30% of all animals apart from the order Insecta (Jarne & Auld, 2006), but also because it is the dominant reproductive mode in plants (Darwin, 1887; Renner & Ricklefs, 1995), which are increasingly studied in the context of sexual selection (Queller, 1983; Arnold, 1994; Delph & Ashman, 2006; Moore & Pannell, 2011). Another reason for increased interest in hermaphrodite sexual selection theory is that, in contrast to the long presumed absence of sexual selection in hermaphrodites (Darwin, 1871), recent research has exposed striking examples of extreme mating traits in hermaphrodites (Michiels, 1998; Baur, 1998; Michiels *et al.*, 2009; Anthes, 2010) and patterns of correlated evolution between male and female mating traits (e.g., Koene & Schulenburg, 2005; Beese *et al.*, 2006; Anthes *et al.*, 2008; Beese *et al.*, 2009; Garefalaki *et al.*, 2010; Schärer *et al.*, 2011).

A growing number of models are starting to assess the scope for sexually selected characters in hermaphrodites (e.g., Morgan, 1994; Greeff & Michiels, 1999b; Puurtinen & Kaitala, 2002; Michiels & Koene, 2006; Preece *et al.*, 2009; Michiels *et al.*, 2009; Schärer & Pen in press). A general conclusion drawn from these studies is that the evolution of sexually selected characters is generally more difficult in hermaphrodites than in gonochorists (except when sexual selection is based on mate-harming traits: Michiels & Koene 2006; Preece *et al.* 2009). This weaker evolutionary potential for sexually selected characters in hermaphrodites is partially caused by the aforementioned absence of sex-limited expression in hermaphrodites (Morgan, 1994), which causes costs of ornamentation to be incurred by all members of the population, instead of typically half the population as is the case for gonochorists. On the other hand, Morgan (1994) also showed that exaggeration of sexually selected characters is still possible in hermaphrodites when costs of expressing such characters are weak. To date, Morgan's analysis remains the only formal investigation in hermaphrodites that includes coevolution between preferences and ornaments, but at the cost of a number of limiting assumptions: for example, it assumes that preferences are cost-free, whereas costly preferences have been demonstrated to largely preclude the potential for the evolution of exaggerated sexually selected characters (Pomiankowski, 1987a; Bulmer, 1989), unless additional assumptions are taken into account, such as biased mutations (Pomiankowski *et al.*, 1991) or weak selection (Hall *et al.*, 2000; Uyeda *et al.*, 2009). Furthermore, in contrast to Lande (1981), Morgan (1994) did not consider the evolution of genetic variances and covariances. Given the increased amounts of costs that hermaphrodites suffer due to a simultaneous expression of ornaments and preferences, the magnitude of genetic variation in both traits is likely to be reduced when compared to gonochorists. The impact of this reduction in genetic variation has not been assessed in studies that considered the expression of ornaments and preferences in both sexes (Lande & Arnold, 1985), which is striking given the importance of genetic variation to the process of sexual selection.

Another study that hinted at a reduced potential for sexual selection in hermaphrodites has been made by Greeff & Michiels (1999a). It focused on the evolution of a pre-existing trait that increases the number of mating encounters of a sperm donor, while no coevolution with traits related to mate choice was included (cf. Morgan, 1994). The fitness effect of any mutant hermaphroditic mating trait that benefited

the male function needs to weighted, however, by the proportion of resources r_h that an hermaphroditic individual invests into the male function. In case hermaphroditism is evolutionarily stable, this proportion is typically $r_h \leq 1/2$ (e.g., Charnov, 1980), which led Greeff & Michiels (1999a) to conclude that sexual selection is at least twice as weak as in gonochorists, where male resource allocation to the male function is of course equal to one. However, Greeff & Michiels (1999a) overlooked that an autosomal mutant allele comprising a reproductive advantage to males in gonochorists is only under selection when it actually resides in a male body, which is typically half of the time. Hence, this conclusion about the strength of sexual selection is currently still ambiguous and needs further investigation.

Additionally, hermaphroditism is only expected to be evolutionarily stable when fitness returns on investment into one of the sex functions (often presumably the male function) is decelerating (Charnov, 1979b). Decelerating returns can limit the scope for investments into sexually selected characters that aim to increase fitness through the male function, in comparison to gonochorists where such returns are expected to be linear or even accelerating (Charnov, 1979b, 1982). How strongly sexual selection is diminished due to such decelerating returns is still an open question that has seen surprisingly little attention. Moreover, functions that yield decelerating returns on certain aspects of male investment are also often invoked in models dealing with gonochorists, for example in case of sperm displacement (reviewed in Parker & Pizzari, 2010) or certain forms of male-male precopulatory competition (Parker, 1983; Fawcett & Johnstone, 2003). Even in gonochorists, the coevolution between traits involved in precopulatory and postcopulatory sexual selection is only starting to be addressed (Fromhage *et al.*, 2008; Parker *et al.*, 2012, Kuijper, Engqvist & Pen, in preparation).

A last reason why hermaphroditism may be associated with weaker sexual selection is due to particular hermaphroditic behaviours such as sperm digestion, where a sperm recipient consumes a certain amount of a sperm donor's ejaculate, potentially reducing the siring success of the donor (Koene & Chase, 1998; Chase & Blanchard, 2006). Selection on sperm donors to overcome the effects of sperm digestion may lead to increased investment of resources into the male function (Greeff & Michiels, 1999b). In turn, such increased investment may lead to the male function becoming limited by resources, instead of being limited by the number of mating partners. Hence, Bateman's principle (Bateman, 1948), where the female function is limited by resources and the male function by mating partners, may not necessarily hold in hermaphrodites with sperm digestion, leading to a reduced potential of sexual selection (Greeff & Michiels, 1999b).

This brief review suggests that there appear to be good reasons to assume that sexual selection is indeed weaker in hermaphrodites. Nevertheless, it also clarifies that even the basic models, such as Fisherian or good-genes sexual selection, have not been properly worked out for hermaphrodites, especially compared to the solid body of theory in gonochorists, which has been instrumental to guide the empirical research agenda (Andersson, 1994; Andersson & Simmons, 2006; Kokko & Jennions, 2010). Here, we take a first step towards resolving this gap, by providing an analysis of Fisherian sexual selection in hermaphrodites. Although a complete model should ideally incorporate all of the aforementioned aspects associated with hermaphroditic lifestyles (e.g., evolvable and/or plastic sex allocation, sperm digestion, mate limitation, mutual choice and others), a first comparison should start with extending the

most elementary models of sexual selection in gonochorists, before more detail can be added.

In this study, we therefore aim to extend a standard, coevolutionary model of Fisherian sexual selection to hermaphrodites. In order to provide a framework that can be used for further studies, our systematic comparison primarily focuses on two key variables: first, we address whether the joint costs of ornaments and preferences may hamper the evolution of sexually selected characters in hermaphrodites in comparison to gonochorists. Second, we explore the variation in mating modes that exists in hermaphrodites, by modeling that inseminations can either take place unilaterally or reciprocally, as described for different hermaphroditic species (Anthes, 2010). Within the variation of mating modes, we also allow for different modes of choice, which can either involve unilateral or mutual choice. Although an increasing number of models has investigated the consequences of male and mutual mate choice in gonochorists (e.g., Johnstone *et al.*, 1996; Servedio & Lande, 2006; Härdling & Kokko, 2005; Nakahashi, 2008), these models have yet to be extended to hermaphrodites. While there is currently no clear empirical evidence (yet) for mutual choice in hermaphrodites (cf. Leonard, 2005; Milinski, 2006; Webster & Gower, 2006), observed precopulatory behaviours leading to size-assortative mating in flatworms (Vreys & Michiels, 1997) or alternating reciprocal exchange of sperm and eggs in seabasses indicates that reciprocal assessment of mating partners may indeed play an important role in hermaphrodites. To take into account of this observed variation in modes of mating and choice, our systematic comparison includes five different mating 'Scenarios' ranging from both female and mutual choice in gonochorists to mutual choice and mutual exchange of gametes in hermaphrodites.

3.2 The model

The first models of Fisherian sexual selection in gonochorists were able to successfully explain the exaggeration of male ornaments beyond their survival optima, but they did not include any direct selection acting on female preferences (Lande, 1981; Kirkpatrick, 1982b). Subsequent analyses showed that the addition of direct selection on female preferences prevents any exaggeration of both ornament and preference (i.e., no evolution beyond the survival optima of either trait), hence precluding evolution by Fisherian sexual selection (Kirkpatrick, 1985; Pomiankowski, 1987b; Bulmer, 1989). However, an analysis by Hall *et al.* (2000) demonstrated that even without additional factors, exaggeration of sexually selected traits is still possible, if direct selection acting on preferences is sufficiently weak (see also Kirkpatrick, 1987a and Figure 2.3 in chapter 2). Here, we follow the quantitative genetics model of Hall *et al.* (2000) and create a model of Fisherian sexual selection that incorporates weak natural and sexual selection on an ornament t and a preference p. Using this model, we compare the size of the parameter space where exaggeration of p and t beyond their naturally selected optima occurs between gonochorists and hermaphrodites. Crucial to the evolution of exaggerated ornaments and preferences are the sizes of the underlying additive genetic variances in t and p, as well as the additive genetic covariance between both traits. Whereas most previous models of sexual selection have assumed that the sizes of genetic covariances – or at least the genetic variances– are constant while varying the strength of natural and sexual selection (Pomiankowski *et al.*, 1991; Iwasa *et al.*, 1991; Pomiankowski & Iwasa, 1993; Hall *et al.*, 2000) we

derive expressions of the genetic variances and covariances as functions of sexual and natural selection acting on them. Moreover, we compare the robustness of our analytical estimates with individual-based simulations (a description of which can be found in the supplement).

Evolution of ornaments and preferences: quantitative genetics

We are interested in the evolution of two polygenic traits, an ornament t, which refers to any character that increases the mating rate of an individual in the light of a preference p for that trait, and a preference p that refers to any character that makes it more likely to mate with partners having particular phenotypes of t than with others. In accordance to standard assumptions, t and p are assumed to be normally distributed with means $\bar{t}$ and $\bar{p}$ and with additive genetic variances G_t and G_p and a covariance G_{tp}. An ornament of size zero indicates no exaggeration beyond an individual's survival optimum and a preference of zero indicates that sperm recipients accept sperm donors randomly. We follow the widely used convention that more extreme values of p favor more extreme values of t.

Our main interest lies in the stability conditions of the equilibrium where no exaggeration occurs and both traits are at their naturally selected optimum, i.e., $(\bar{t}, \bar{p}) = (0, 0)$. In order to derive such stability conditions, one needs to obtain the evolutionary recursion equations of character means and any higher order moments near $(\bar{t}, \bar{p}) = (0, 0)$, based on which one can assess stability using standard methods of linearization. For reasons of tractability, most previous analyses have focused only on the evolutionary recursions of $\bar{p}$ and $\bar{t}$, while the genetic variances and covariances were often assumed to be fixed parameters, or at least independent to the strength of survival selection acting on both traits (e.g., Pomiankowski *et al.*, 1991; Hall *et al.*, 2000). Here, we take those analyses a step further by also deriving recursion equations for the genetic variances and covariances, using an explicit model of mutation and recombination.

We do so by working out the changes in the joint normal distribution of ornaments and preferences $q(t, p)$ over a single generation. Changes in $q(t, p)$ can be attributed to four different events, which are assumed to act in subsequent stages: (i) a survival stage where costs of exaggerated characters are accounted for, (ii) a mating stage where mate choice takes place, (iii) a recombination stage during gametogenesis and (iv) a mutation stage acting in the gametes. Assuming weak selection, we can then obtain expressions of the first and second order moments (means, variances and covariances) of $q(t, p)$ during each stage of selection. While we provide more detail on each of the four stages below, full recursions and stability analyses are provided in the Supplement.

Survival Following previous analyses, survival selection against exaggerated ornaments and preferences is based on a Gaussian survival function with an optimal character size of θ, which can be scaled to any arbitrary value. Without loss of generality, we assume $\theta = 0$. For gonochorists with female choice only, the survival functions for males and females are given by $w^*_{\mathrm{m}}(t) = \exp[-ct^2]$ and $w^*_{\mathrm{f}}(p) = \exp[-bp^2]$ respectively, whereas for gonochorists with mutual choice and hermaphrodites, this is given by $w^*(t, p) = \exp[-ct^2 - bp^2]$, where c and b are parameters representing the

strength of selection acting against exaggerated ornaments and preferences respectively.

Mate choice After survival, mate choice takes place. We investigate five different scenarios of mating interactions, for which we derive a joint distribution of breeding pairs $q^{**}(t,p)$. This joint distribution informs us on the strength of assortative mating based on t and p and provides the total distribution of gametes produced, barring mutation and recombination. In the supplement, we provide more detail on the derivation of the distribution of breeding pairs $q^{**}(t,p)$ of each of the scenarios below. In all scenarios considered below, we consider that mating preferences p are open-ended, so that individuals that express a preference p favor ever larger values of ornamentation t in their mates, according to the function $\psi(t|p) = \exp(apt)$, in which a is a parameter that reflects the efficacy of mate choice. Such directional preferences have been found in a substantial number of studies (e.g., Ritchie, 1996; Groot *et al.*, 2006; Griggio *et al.*, 2009). We will also briefly consider the impact of different preference functions, such as absolute and relative preferences (Lande, 1981).

Scenario i – Gonochorists, female choice only: as modeled by the majority of previous models on the coevolution of ornaments and preferences by Fisherian sexual selection (e.g., Pomiankowski *et al.*, 1991; Hall *et al.*, 2000). Surviving females choose a partner out of a distribution of male candidates that spans the surviving population, according to her preference function $\psi(t|p)$ (Lande, 1981). After all females have chosen a mate, we obtain an expression of the joint distribution of breeding pairs $q^{**}(t,p)$ in terms of all possible combinations of t and p. This joint distribution informs us on the strength of assortative mating based on t and p and provides the total distribution of gametes produced, barring mutation and recombination (see Section S3.2.1)

Scenario ii – Gonochorists, mutual choice: Following previous analyses on mutual choice in the context of Fisherian sexual selection (Servedio & Lande, 2006; Nakahashi, 2008), we assume that males use a preference p_m to court females according to the size of their ornaments t_f, with preference function $\psi(t_f|p_m)$. This generates a distribution of courting males per female, and a female chooses (with her preference p_f) one of the males that are courting her, according to the size of the male's ornament t_m with preference function $\psi(t_m|p_f)$. We assume that male ornaments and preferences are expressed from the same loci as in females (i.e., $p_m = p_f, t_m = t_f$), resembling the 'pleiotropic' ornament expression scenario studied in Servedio & Lande (2006). Although we acknowledge that it is more likely that males and females express p and t from different, sex-specific loci, it serves here as a basis of comparison with hermaphrodites, where such pleiotropic ornaments and preferences are likely to be the norm due to the absence of sex-limited expression.

A fundamental aspect of mutual choice in the context of the Fisher process is the relationship between male preferences and the resulting potential number of female mating partners (Servedio & Lande, 2006). In case all males have an equal level of total courtship effort, larger absolute values of p_m indicate that males strongly bias their courtship towards the most ornamented females, while $p_m = 0$ reflects a random distribution of courtship effort across all females. As an alternative scenario, the level of courtship effort may vary among different males, so that males with larger absolute values of p_m may spend more effort on courtship relative to males with lower absolute

values of p_m. Both scenarios will be considered in the current study.

Scenario iii – Hermaphrodites, unilateral choice and insemination: (see Figure 3.1A). Every individual is assumed to successfully choose (using p) a single mate, based on that mate's ornament t. This mate always donates sperm, but there is no reciprocal transfer of sperm. With these assumptions, an individual will mate only once in the female role, but can mate numerous times in the male role, dependent on its attractiveness. Hence, the current scenario closely resembles the female choice scenario (i.e., Scenario i) in gonochorists. Moreover, under these assumptions, the joint distribution $q^{**}(t,p)$ after mate choice is identical to the distribution in gonochorists with female choice only, except for the fact that every individual hermaphrodite endures survival costs on both p and t.

Scenario iv – Hermaphrodites, unilateral choice and reciprocal insemination: (see Figure 3.1B). As in the previous scenario, each individual chooses with a preference p a certain sperm donor with ornament t that always accepts to donate sperm. Additionally, the choosing individual now also always donates sperm in return, unconditional on the preference of its partner. Hence, an individual receives one ejaculate when it actively chooses, and receives $M(t,p)$ ejaculates when it is chosen by others, where M is equal to an individual's number of mates. The individual that actually fertilizes the individual's eggs is determined through fair raffle sperm competition (Parker, 1990). This scenario allows us to investigate the effect of sperm transfer on the evolution of sexually selected characters.

Scenario v – Hermaphrodites, reciprocal choice and insemination: (see Figure 3.1C). First, every individual courts other individuals in the population, where some individuals may receive more courtship than others dependent on their attractiveness and the courting individual's preference. After all individuals have courted, we obtain a joint distribution of courting pairs $g^{**}(t,p)$ that is similar to the joint distribution of hermaphrodites with unilateral choice only. Then, every individual will choose a mate from among the individuals that courted it. This choice will lead to a mating pair and we can again derive the joint distribution of pairs $q^{**}(t,p)$ (see Section S3.2.5). Since we already know from Scenario iv that reciprocal insemination does not affect these results, we only have to assess how mutual choice affects the joint distribution of pairs $q^{**}(t,p)$, where the choice process is identical to the one for mutual choice in gonochorists (i.e., Scenario ii).

Recombination and mutation Assuming haploid inheritance, one can calculate all possible gamete combinations that result from the joint distribution of mating pairs $q^{**}(t,p)$. For example, given that mothers and fathers have character distributions $q(t_1,p_1)$ and $q(t_2,p_2)$ respectively, the probability of having a (t_1,p_2) zygote can be calculated from the marginal distribution of mating pairs $(\frac{1}{4})\iint q^{**}(t_1,p_1,t_2,p_2)dp_1t_2)$. Here, 1/4 indicates one of four possible combinations of gametes between a $(t_1,p_1)\times(t_2,p_2)$ mating. The last stage is mutation, where we assume a continuum-of-alleles model (although the model could be easily extended to allow for other models of mutation). Alleles mutate with rate μ after which an increment in their character value is drawn from a normal distribution with mean zero and variance ν (determining the mutation stepsize). For more details regarding the distribution of t and p after mutation and recombination, see Section S3.3.

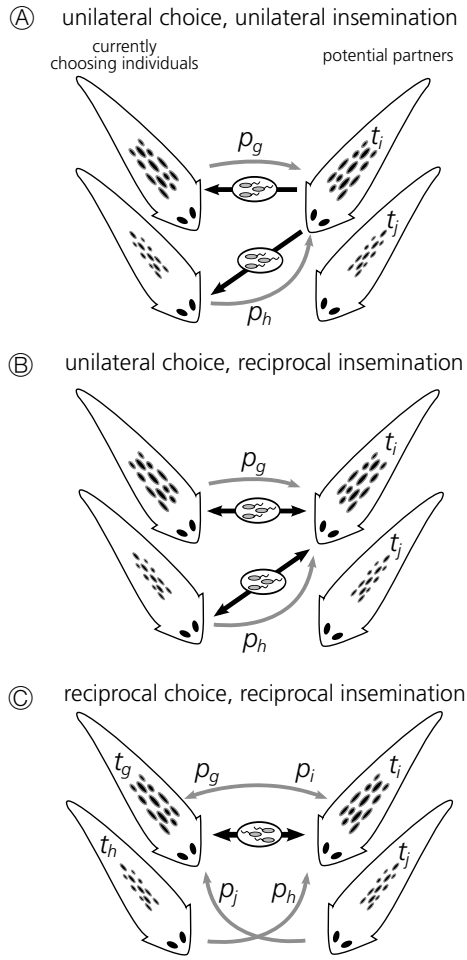

Figure 3.1: A schematic drawing of the mating scenarios used for hermaphrodites (i.e. Scenarios iii, iv, and v). Preferences p are indicated by grey arrows and the ornaments t are depicted as spots. Note that these do not resemble any currently known traits in hermaphrodites (possible actual preferences and traits are discussed in the main text). Subscripts indicate the traits of different individuals. Panel A represents unilateral choice and unilateral insemination (i.e., scenario iii): individuals g and h on the left choose (using their preferences p_g and p_h) a sperm donor based on variation in the sperm donor's ornaments t. In this case, both choosing individuals choose donor i with the largest ornament (t_i). In response, individual i donates sperm to both individuals g and h, but does itself not receive sperm from these individuals. Individual j is not chosen as a sperm donor, but will itself choose a sperm donor once during its lifetime (since we follow other models by assuming that female fitness is not related to mating success). Panel B represents unilateral choice and reciprocal insemination (i.e. scenario iv): again, individuals g and h on the left choose in the same fashion as in panel A, but upon receiving sperm from the preferred donor i, each choosing individual reciprocates the sperm donation (double arrows). This has the consequence that individual i donates two portions of sperm as a result of being chosen, but also receives two portions of sperm back, (while assuming that fertilizations are randomly distributed over its eggs). Panel C represents reciprocal choice and reciprocal insemination (i.e. scenario v): both individuals g and h preferentially court individual i. However, individual i itself prefers only individual g, leaving individuals h and j without sperm, until they are courted by other individuals at a later instance.

Stability conditions From previous quantitative genetics analyses, we know that without additional assumptions of mutation bias or spatial variation, there is only a single equilibrium at the naturally selected optimum, i.e., $(\bar{t},\bar{p}) = (0,0)$. Our task is to check when this equilibrium is unstable and hence a runaway will ensue towards exaggerated values of $\bar{t}$ and $\bar{p}$. Using linearizations of the recursion equations for $\bar{t}$, $\bar{p}$, G_t, G_p and G_{tp}, we have derived expressions for the stability of the point $(\bar{t},\bar{p}) = (0,0)$ for each of the scenarios (see Section S3.4 and Table S3.3). To arrive at analytical estimates of the stability conditions, we had to make the necessary assumptions that both natural and sexual selection are weak (i.e., $b \gg b^2, c \gg c^2, a \gg a^2$), that the covariance between ornament and preference remains relatively small ($G_t G_p > G_{tp}^2$) and as well that mutation rates μ and stepsizes ν are small.

3.3 Results

When does exaggeration in sexually selected characters occur?

Previous models of the Fisher process have expressed the parameter space in which exaggeration of sexually selected characters occurs in terms of the sizes of the additive genetic variances in ornaments, G_t, and preferences, G_p (e.g., Pomiankowski *et al.*, 1991; Hall *et al.*, 2000). Analogously, Table S3.2 derives the conditions for exaggeration in ornaments and preferences in terms of G_t, G_p and G_{tp} for each of the mating scenarios considered, from which already some insight about the differences between gonochorists and hermaphrodites can be obtained. However, conditions for exaggeration in terms of G_t, G_p and G_{tp} overlook the fact that the additive genetic (co)variances are themselves a function of natural and sexual selection, which may differ between hermaphrodites and gonochorists. To make a more insightful comparison of sexual selection between hermaphrodites and gonochorists, we therefore calculate the equilibrium values of G_t, G_p and G_{tp} in terms of the parameters a (efficacy of mate choice), b and c (survival costs of ornaments and preferences), and those that specify the mutation rate (see Table S3.3). Unsurprisingly, we find that in organisms which endure more natural selection on t and p due to the lack of sex-limited expression, G_t and G_p are smaller (see Figure S3.1). Subsequently, we then use these expressions for G_t and G_p to arrive at conditions for exaggeration that are completely expressed in terms of mutation, and natural and sexual selection. However, these conditions are long and not particularly informative, so we provide a graphical overview in Figures 3.2 and 3.3.

First, we compare exaggeration of ornaments and preferences between gonochorists and hermaphrodites between mating scenarios where choice is unilateral (i.e., female choice in gonochorists [Scenario i] and unilateral choice with or without reciprocal insemination in hermaphrodites [Scenarios iii,iv]). For the case where G_t, G_p and G_{tp} are considered parameters, Tables 3.1 (open-ended preferences) and S3.2 (relative, absolute preferences) show that costs of ornaments and preferences in unilaterally choosing hermaphrodites should at least be twice as small in comparison to gonochorists. Indeed, Figure 3.2 shows that for any preference function, exaggeration in hermaphrodites occurs for a much smaller range of costs of ornaments and preference than in gonochorists. Additionally, Table 3.1 also shows that reciprocal insemination has no effect on the exaggeration of sexually selected traits, so that the parameter space in which exaggeration occurs for hermaphrodites with reciprocal or unilateral insemination are identical in Figure 3.2.

Table 3.1: Approximations of the stability conditions of the equilibrium $\{\bar{t}, \bar{p}\} = \{0, 0\}$ for the different systems considered, while G_t, G_p and G_{pt} are considered parameters. Approximations are taken for first order terms around $\{a,\ b,\ c,\ \mu\nu,\ G_{tp}\} = 0$. See Table S3.2 for analogous conditions for relative and absolute preferences.

organism	choice scenario	insemination	stability condition
gonochorist	female choice	unilateral	$\frac{1}{2}aG_{tp} > bG_p + cG_t$
	mutual choice	unilateral	$\left(G_{tp}^2 - G_pG_t\right)\left(4bc - \frac{1}{4}a^2\right) > 0$
hermaphrodite	unilateral choice	unilateral	$\frac{1}{4}aG_{tp} > bG_p + cG_t$
	unilateral choice	reciprocal	$\frac{1}{4}aG_{tp} > bG_p + cG_t$
	mutual choice	reciprocal	$\left(G_{tp}^2 - G_pG_t\right)\left(4bc - \frac{1}{4}a^2\right) > 0$

Apart from the notion that exaggeration of sexually selected characters occurs for a smaller range of costs in unilaterally choosing hermaphrodites, Figure 3.2 corroborates previous studies (Hall *et al.*, 2000) by showing that open-ended preferences are the most conducive to exaggeration, whereas absolute preferences are, by far, the least favorable to exaggeration.

Whereas hermaphrodites are less prone to express sexually selected traits than gonochorists when choice is unilateral, the situation is different for scenarios involving mutual choice. Our analysis in the supplement (sections S3.2.2 and S3.2.5) shows that mutual choice leads to identical conditions for exaggeration in hermaphrodites and gonochorists (see Table 3.1). This is because both mutually choosing gonochorists and hermaphrodites express both ornaments and preferences in one and the same individual, so that hermaphrodites and gonochorists endure similar levels of costs acting against exaggerated characters. Additionally, reciprocal insemination in hermaphrodites also does not lead to differences between gonochorists and hermaphrodites (see supplement section S3.2.4), so that the exaggeration of ornaments and preferences is similar in mutually choosing hermaphrodites and gonochorists.

Exaggeration in mutually choosing gonochorists and hermaphrodites depends specifically on the preference function considered: open-ended preferences are conducive to exaggeration under mutual choice, whereas absolute or relative preferences prevent the evolution of exaggerated sexually selected traits. For the remainder, we therefore focus on mutual choice based on open-ended preferences only.

Importantly, exaggeration of sexually selected traits depends on the relationship between a donor's preference and the total level of courtship output, here reflected by the parameter z (see Table 3.1). When $z = 1$, all sperm donors have the same courtship output, and a donor's preference determines how courtship is distributed among the different sperm recipient phenotypes (i.e., $p = 0$: random distribution of courtship among recipients, $p \neq 0$: courtship skewed towards more ornamented sperm recipients). When $z = 1$, no exaggeration of ornaments and preferences occurs unless the unrealistic condition is met in which $G_{pt} > G_pG_t$ (i.e., the squared addi-

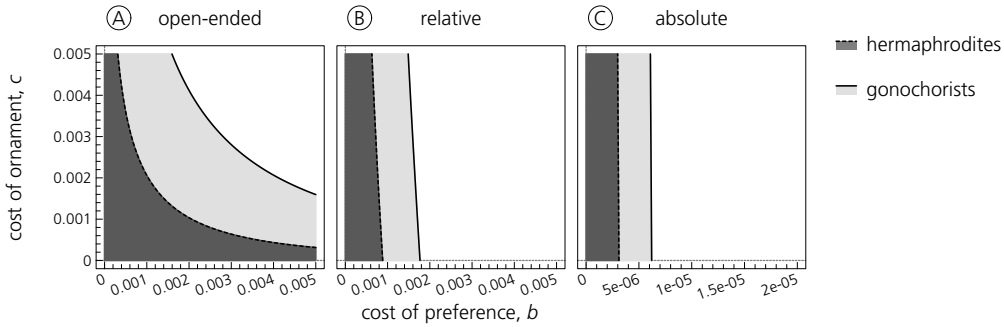

Figure 3.2: Exaggeration in case of female choice (gonochorists, light grey) or unilateral choice (hermaphrodites, dark grey). Contour plots show the regions where exaggeration away from the naturally selected optimum is expected. Parameters: $v = 0.0025$, μ=0.01, $a = 0.3$ for all panels.

tive genetic covariance that arises due to assortative mating should be larger than the product of both variances). However, G_{pt} is typically very small: Figure S3.1, Kirkpatrick & Barton, 1997), so this condition is unlikely to be met. The situation is drastically different in case donors with higher values of p court sperm recipients more often than donors with lower values of p, which occurs when $0 \leq z < 1$. Now mutual choice in both hermaphrodites and gonochorists is more conducive to exaggeration of ornaments and preferences (see Figure 3.3). Indeed, we find that even when courtship output only slightly deviates from a scenario in which all sperm donors have equal courtship output (i.e., z close to 1), exaggeration occurs for a range of costs that is far larger in comparison to the case of unilateral choice (compare Figures 3.2 and 3.3 when $z = 0.95$). Hence, whenever choosy sperm donors are able to court at higher rates than non-choosy sperm donors, mutual choice can be more favorable to exaggerated sexually selected traits, in both hermaphrodites and gonochorists alike.

Tracking the coevolutionary outcome using individual-based simulations

Although the stability analyses above inform us about the costs that allow for exaggeration away from the naturally selected equilibrium, such analyses do not provide us with much insight about the level of exaggeration that are reached and the eventual evolutionary dynamics (see Kokko *et al.*, 2003, for similar comments). To address this, we ran individual-based simulations for all mating scenarios considered (see Supplementary section S3.6 for a description). Figures 3.4 (gonochorists) and 3.5 (hermaphrodites) compare the analytically obtained region of exaggeration with results obtained from individual-based simulations. Importantly, the results from our quantitative genetics model are recovered in the simulations: the region of exaggeration is much larger for mutually choosing organisms than for the other mating scenarios considered here (see Figure 3.4B and 3.5C). Additionally, hermaphrodites with unilateral choice have a smaller region of exaggeration than their gonochorist counterparts with female choice (compare Figure 3.4A with 3.5A,B).

For the scenarios with unilateral choice (i.e., female choice in gonochorists, and hermaphrodites with unilateral choice with or without reciprocal insemination), values of exaggeration in Figures 3.4A and 3.5A,B vary strongly between adjacent pa-

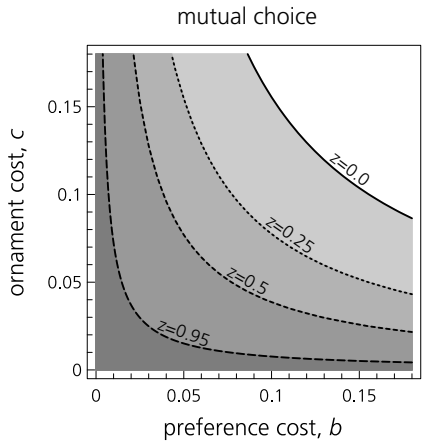

Figure 3.3: Exaggeration in gonochorists and hermaphrodites in case of mutual choice, when preferences are open-ended. Note that both gonochorists and hermaphrodites lack sex-limited expression in case of mutual choice, so that the region of exaggeration is similar in both hermaphrodites and gonochorists. Note that the range of exaggeration is several tenfolds larger in comparison to unilateral choice (Figure 3.2). When $z = 1$, courting one sperm recipient implies that other sperm recipients cannot be courted. When $z = 0$, courting one sperm recipient does not reduce the probability of courting other recipients. Even for values where z is close to 1 (say, 0.95), exaggeration occurs at a parameter space that is far larger than that for unilateral choice (cf. Figure 3.2). Parameters: $\nu = 0.0025$, $\mu = 0.01$, $a = 0.3$.

rameter values. Variation in exaggeration for these mating scenarios can be understood by inspecting the long-term evolution of $\bar{t}$ and $\bar{p}$ over time of the example runs in the right panels, which shows that the evolution of $\bar{t}$ and $\bar{p}$ is inherently cyclic. Near the naturally selected equilibrium, selection is extremely weak, allowing for a rapid runaway of $\bar{t}$ and $\bar{p}$ towards higher levels of exaggeration. Away from the naturally selected equilibrium, however, exaggeration becomes more costly, so that the benefit incurred by sperm donors (males) of achieving more matings is offset by the increased costs of exaggeration. Consequently, the benefits of giving birth to sexy sons also decreases for sperm recipients (females). Hence, levels of $\bar{p}$ decrease, followed by a decrease in $\bar{t}$. Once levels of $\bar{t}$ are sufficiently reduced, so that direct costs of a slightly higher level of ornamentation do not negate the benefits of an increased number of matings, a runaway of $\bar{t}$ and $\bar{p}$ may be initiated in the same or a different direction. Due to this inherent cyclicity, measuring the exaggeration of $\bar{t}$ at a particular point in time can thus yield very contrasting amounts of exaggeration among replicates, as they depend on a population's current position within the cycle of exaggeration (see also Uyeda *et al.*, 2009). To conclude, the default long-term evolutionary dynamic exhibited by unilaterally choosing organisms is a cyclic pattern of coevolution, where levels of $\bar{t}$ are typically far larger than those of $\bar{p}$.

For mutual choice however, long-term dynamics are different from the unilateral choice scenarios (see Figures 3.4B and 3.5C). Note that exaggeration under mutual choice only occurs whenever $z < 1$ (i.e., choosy sperm donors devote more energy to courtship than non-choosy sperm donors), which is what we have assumed in the simulations in Figures 3.4B and 3.5C ($z = 0$). Simulations show that both $\bar{t}$ and $\bar{p}$ do not exhibit any cyclic dynamics, but settle at a stable equilibrium. Additionally, the maximum level of exaggeration is smaller overall, in comparison to the maximum

levels of exaggeration observed when choice is unilateral.

3.4 Discussion

A long standing idea is that sexual selection is absent (Darwin, 1871) or at least very weak in hermaphrodites (Morgan, 1994; Greeff & Michiels, 1999a). Although a number of models have attempted to corroborate this idea, these studies contain certain weaknesses that have precluded a formal comparison between gonochorists and hermaphrodites (Arnqvist & Rowe, 2005). As outlined in the introduction, including the full complexity of hermaphrodite characteristics in a model of sexual selection is a complex task that should ideally be achieved by extending standard models in a stepwise fashion. Here, we made the first step by extending an established model of Fisherian sexual selection towards hermaphrodites, while exploring the variation in mating modes that exist in hermaphrodites.

Mutual choice The most important result of this study is that mutual choice is much more likely to lead to exaggeration of sexually selected characters than unilateral choice, and this applies equally to gonochorists and hermaphrodites. Previous studies on Fisherian sexual selection in gonochorists with mutual choice showed, however, that male or mutual choice in fact reduces the potential for exaggeration of sexually selected characters (Servedio & Lande, 2006; Nakahashi, 2008; Edward & Chapman, 2011). These studies assume that all males have the same courtship output, with choosy males devoting this courtship exclusively towards those females that are acceptable to them, whereas non-choosy males devote courtship to any female. As a consequence, the potential number of mates of a choosy male is reduced, in comparison to males which express no preferences (Servedio & Lande, 2006). Indeed, also in the current model exaggeration in ornaments and preferences does not occur whenever sperm donors (hermaphrodites) have the same courtship output (i.e., when $z = 1$). However, Servedio & Lande (2006) also showed that whenever choosy males have a larger courtship output in comparison to males that accept any mate, choosy males may achieve a higher number of mates than non-choosy males, resulting in ornaments and preferences evolving away from their naturally selected optima. In the current study, we show that even a slightly larger courtship output by choosy sperm donors relative to non-choosy sperm donors (e.g., $z = 0.95$ in Figure 3.3) can lead to stable exaggeration of ornaments and preferences for a much larger range of costs than in unilaterally choosing hermaphrodites, although the amount of exaggeration is typically modest relative to the maximum levels of exaggeration found in organisms with unilateral choice (see Figure Figures 3.4B2 and 3.5C2). Hence, Fisherian sexual selection may lead to modestly exaggerated sexually selected characters under a far larger range of costs in mutually choosing hermaphrodites than unilaterally choosing hermaphrodites, whenever choosy sperm donors devote more energy to courtship than randomly mating sperm donors. Moreover, our model shows that both the degree of exaggeration, as well as the range of costs under which exaggeration occurs, does not differ between mutually choosing gonochorists and hermaphrodites.

Although exaggerated sexually selected characters are thus expected to be equally prevalent in mutually choosing hermaphrodites and gonochorists, this is not the case for unilateral choice: exaggeration of sexually selected characters in unilaterally choosing hermaphrodites occurs for a substantially smaller range of costs of orna-

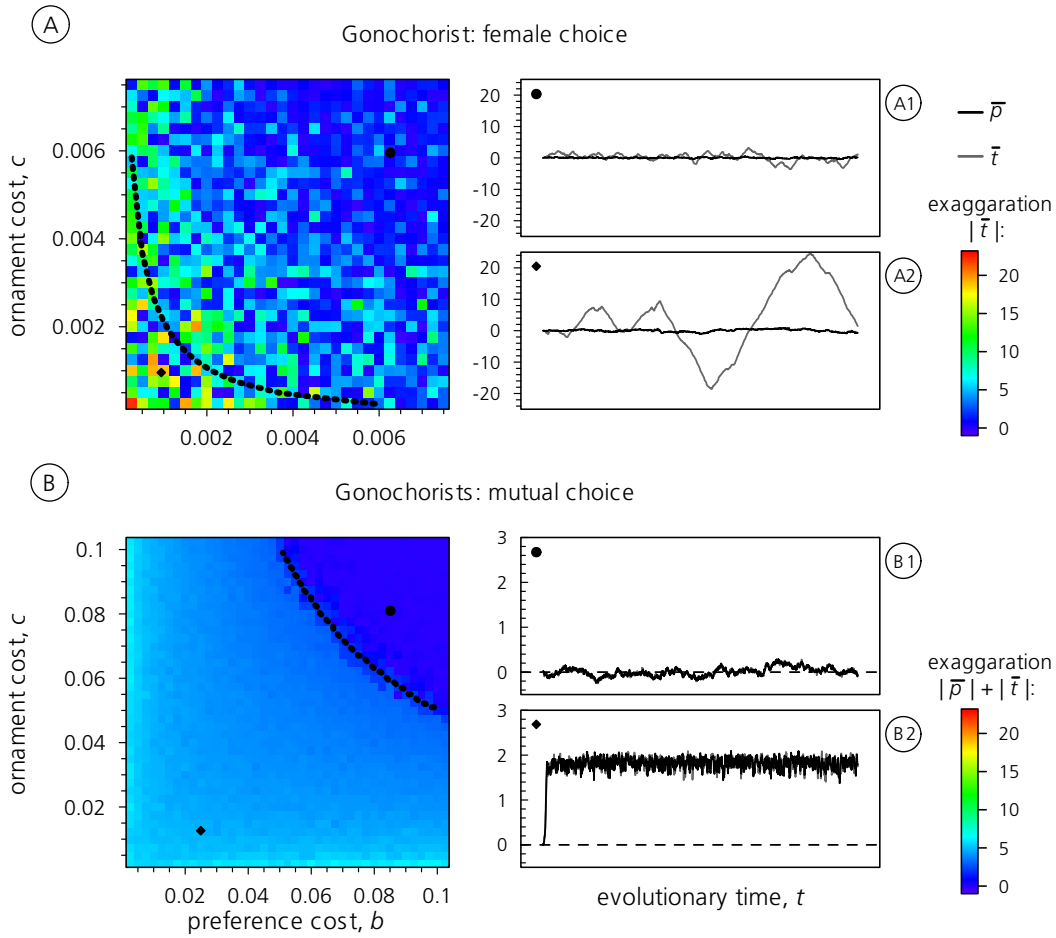

Figure 3.4: Comparing the analytically obtained region of exaggeration with the outcome of individual-based simulations for gonochorists. Panels on the left compare the values of $\bar{t}$ and $\bar{p}$ at the end of the simulation run across a range of parameter values. Panels on the right show individual simulation runs for conditions where the equilibrium was analytically shown to be stable or unstable, respectively. In the region left of each dotted black line, the naturally selected optimum is expected to be unstable. Each colored square represents the absolute exaggeration of $\bar{t}$ (panel A) or the sum of absolute exaggerations of $\bar{t}$ and $\bar{p}$ measured in a single simulation, averaged over generations 45 000 to 50 000. To correct for processes that may lead to exaggeration in character values other than sexual selection (e.g. drift), we subtracted from each measured character value, the character value that was measured in a replicate simulation where we imposed random mating (where $a = 0$). Parameters: $\mu_t = \mu_p = 0.01, \nu_t = \nu_p = 0.0025, a = 0.3, n = 10$. Total number of simulations is 3600.

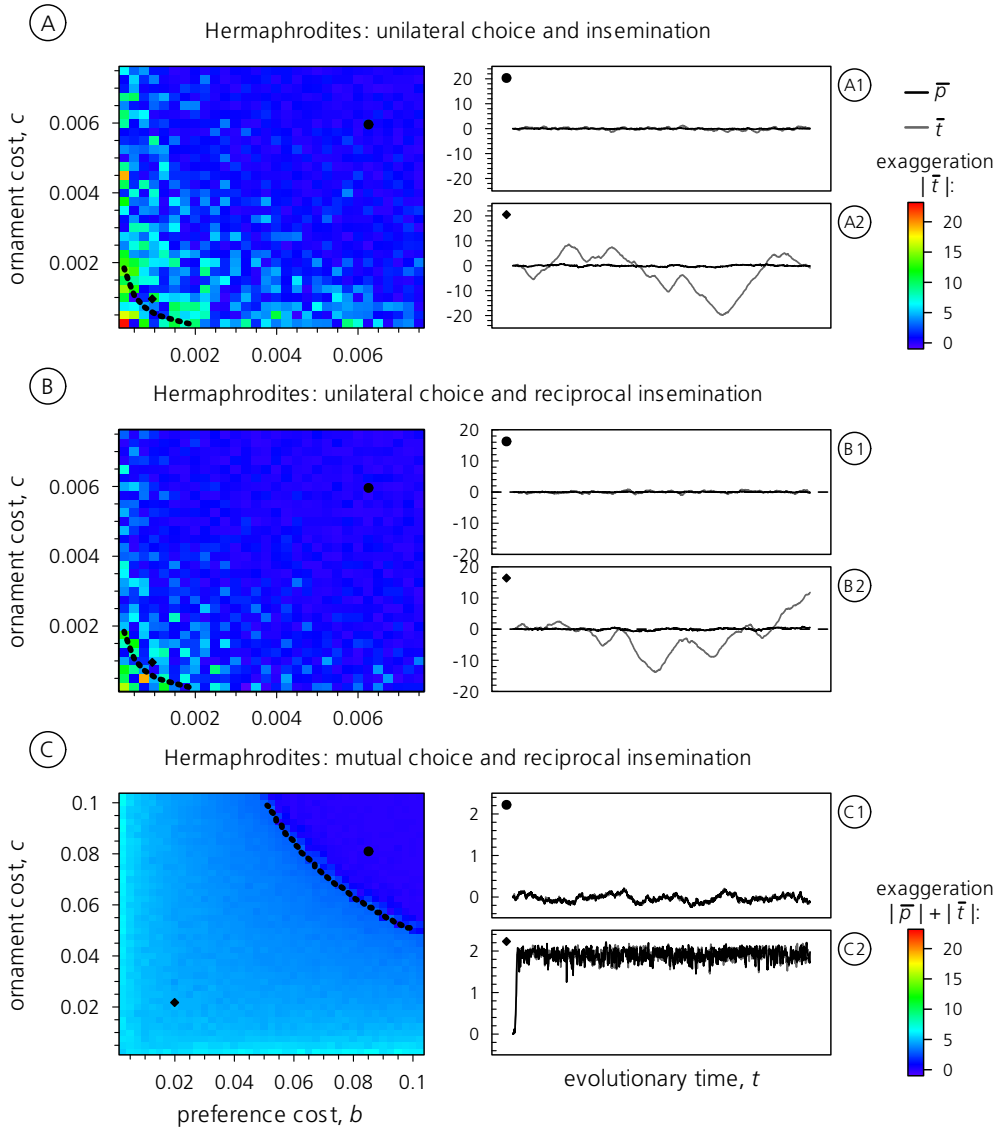

Figure 3.5: Comparing the analytically obtained region of exaggeration with the outcome of individual-based simulations for hermaphrodites, similar to Figure 3.4 for more information. Total number of simulations is 5400.

ments and preferences in comparison to gonochorists with female choice. This is because hermaphrodites simultaneously express a costly ornament and a costly preference, while a gonochorist individual only expresses one of both costly traits, and thus incurs a lower level of costs than a hermaphrodite. A similar reduction in the propensity for sexually selected characters to evolve away from the naturally selected optimum has been shown to occur in previous models that assumed a lack of sex-limited expression in ornamentation (e.g., Lande & Arnold, 1985; Morgan, 1994). Hence, exaggeration of sexually selected characters in hermaphrodites is limited to a smaller range of costs than in gonochorists. In mutually choosing gonochorists and hermaphrodites, this difference does not occur because each gonochorist individual now expresses ornaments and preferences simultaneously, so that the lack of sex-limited expression resembles that of hermaphroditic individuals considered here. Based on these results, we can conclude that unilaterally choosing hermaphrodites are considerably less likely to develop sexually selected traits than gonochorists with female choice. However, in these unilaterally choosing hermaphrodites, sexual selection is still possible if costs of sexually selected characters are relatively small. Hence, our model shows that even sexual selection based on unilateral choice is not exclusive to gonochorists and that a focus on measuring the actual costs that carriers of sexually selected characters incur (Kotiaho *et al.*, 2001) may shed more light on the likelihood for exaggerated characters in hermaphrodites. All in all, per unit of investment, ornaments and preferences should be cheaper to produce in unilaterally hermaphrodites with sexually selected traits than in gonochorists.

Assumptions A number of assumptions in our model merit further discussion: our model assumes, in accordance with Hall *et al.* (2000), that survival selection acting on t and p is weak. Given that weak stabilizing selection is regarded to be predominant form of selection in nature (Kingsolver *et al.*, 2001), we would predict that our model applies to most empirical situations: hence, the expected pattern is that sexual selection would occur in a cyclical fashion and that divergence among populations would be relatively common. But experiments that actually measure selection gradients with respect to survival on particular sexually selected traits are still rarely undertaken in gonochorists (Kotiaho *et al.*, 2001) (but see Hine *et al.*, 2011), let alone in hermaphrodites (Anthes *et al.*, 2010). Additionally, note that natural selection acting against ornamentation does not necessarily affect survival, as assumed in the current model, but could also affect other fitness components such as fecundity, ejaculate investment, or immunocompetence. Studies that measure the strength of natural selection on sexually selected characters are essential for making predictions regarding exaggeration of ornaments and preferences in hermaphrodites.

In contrast to the few studies focusing on the strength of natural selection on one or more characters, the strength of sexual selection (i.e., differential mating success) has been assessed by a larger number studies, at least in gonochorists (see Hunt *et al.* 2009 for a recent review of such studies). Even in hermaphrodites, a recent study by Pélissié *et al.* (2012) measured the strength of sexual selection on body size, showing that a single standard deviation increase in body weight increases an individual's fitness by 15-16%, which is close to the median strength of selection ($\beta_{\text{sex.sel.}} = 0.18$) found by Kingsolver *et al.* (2001) in a meta-analysis for traits under sexual selection in gonochorists. Taking unilateral choice in hermaphrodites as an example model,

our model assumes $\beta_{\text{sex.sel.}} \propto a$, and that $a^2 \ll a$, which is likely to be the case when $\beta_{\text{sex.sel.}} = 0.18$. However, Kingsolver (2001) also reviews a number of studies in which selection gradients have a much larger size: additional simulations for strong sexual selection in combination with a large mutational influx of genetic variation indicates that the coevolutionary dynamics for $\bar{t}$ and $\bar{p}$ become more complex, with rapidly fluctuating limit cycles and evolutionary branching in ornamentation (results not shown). Hence, we predict that extrapolating current models to cases with strong selection will yield more interesting dynamics than those that are represented in the current study.

Next to strong selection, another important omission of our model is sex allocation in simultaneous hermaphrodites, and the fact that there probably is a trade-off between allocation to the female versus the male function (Schärer *et al.*, 2005; Schärer, 2009). As noted by Charnov (1979b), simultaneous hermaphroditism is only stable whenever reproductive success through one sexual function, probably often the male function, shows saturating returns, leading to a reallocation of resources to the female function. In contrast, our model assumes that the male gain function accelerates in a linear or positively accelerating fashion, so that simultaneous hermaphroditism is unstable. Consequently, the current model applies mainly to those organisms where evolutionary transitions between hermaphroditism and dioecy are constrained. Especially those organisms that have internal fertilization and elaborated genital systems, such as flatworms, molluscs or oligochaetes appear to rarely show such transitions, which hints at the fact that constraints on evolutionary transitions may be present (Michiels *et al.*, 2009; Schärer, 2009).

To what extent our conclusions apply to organisms where the male gain function is decelerating as opposed to linear remains to be seen, although the same argument will have to be investigated for models of sexual selection in gonochorists: processes such as sperm displacement may lead to negatively accelerating gain curves in gonochorists as well and the effect of these assumptions to the sexual selection process is yet unknown, since sperm competition has yet to be formally incorporated in coevolutionary models of sexual selection (Kuijper, Engqvist & Pen, in preparation). To conclude, few formal predictions exist about the trade-offs involved in characters involved in mate choice versus sperm competition and their resulting coevolutionary dynamics (Alonzo, 2010).

The third assumption of our model is that the fitness of the female role does not affect the mate choice process. Stating this in terms of the Bateman gradient framework for hermaphrodites introduced by Anthes *et al.* (2010), our model assumes only the existence of a sexual selection gradient for the male function β_{mm}, while the cross-sex and the female sexual selection gradients are all assumed to be zero. Given the first results obtained using this framework, this assumption appears to be reasonable. Both Anthes *et al.* (2010) and Pélissié *et al.* (2012) found a steep male gradient, a weaker or absent female gradient, and no indications of cross-sex gradients. Also in gonochorists, there are only few models that make explicit assumptions about β_{ff} (Kokko & Mappes, 2005; Lessells, 2005). Taking the model by Kokko & Mappes (2005) as an example, they predict that female preferences are rapidly diminished whenever choosiness bears a cost of remaining unmated. Based on their results, we would expect that sperm limitation in hermaphrodites would undermine choosiness. However, we would predict that the relationship between preferences and sperm limitation is not merely negative: for example, choosiness by a sperm recipient may be

favored whenever sperm donors can be reliably assessed on their fertilizing ability. A promising extension of our model could focus on the evolution of preferences for multiple traits (Johnstone, 1995; Van Doorn & Weissing, 2004), in which one trait would reflect a sperm recipient's fecundity, while another trait would reflect a sperm donor's fertility.

How to measure Fisherian sexual selection in hermaphrodites? Anthes (2010) reviews existing empirical studies on mate choice in hermaphrodites and categorizes them according to three types of characters: i) mate choice based on body size, where larger individuals are generally preferred since they may constitute a higher fecundity for sperm donors (e.g., Vreys & Michiels, 1997; DeWitt, 1996). ii) mate choice for reduced sperm competition; in order to achieve a greater share of paternity, individuals may prefer unmated individuals or strategically adjust their mating effort based on the mating status of their partner (Haase & Karlsson, 2004; Anthes *et al.*, 2006a; Koene & Ter Maat, 2007). iii) mate choice based on relatedness, immune function or other aspects of genetic quality, where individuals may avoid certain levels of inbreeding (e.g., McCarthy & Sih, 2008) or prefer partners that are not infected with parasites (Webster & Gower, 2006). From the review by Anthes (2010), it can be noted that all the reviewed examples of mate choice are based on characters that are more easily associated to benefits of mate choice via good-genes (Iwasa *et al.*, 1991), good-parents (Hoelzer, 1989; Price *et al.*, 1993) or genetic compatibility (Puurtinen *et al.*, 2009; Fromhage *et al.*, 2009), as opposed to characters that constitute a heritable mating advantage to sperm donors. Despite this, it would be premature to argue that Fisherian sexual selection is absent from hermaphrodites all together. Importantly, all other models of mate choice, be it good genes, compatible genes, direct benefits or sexual conflict, necessarily include a Fisherian sexy-sons component (Kokko *et al.*, 2002, 2006): regardless of what the preferences and ornaments actually constitute, assortative mating generates a statistical association between both characters and the resulting feedback has the potential to drive evolution towards higher values of exaggeration of both traits, unless it is prohibited by direct costs. This has even led some to consider Fisherian sexual selection to be the null model for other models of mate choice (Prum, 2010), so that our analysis extrapolates this null model to include mate choice in hermaphroditic organisms as well. In relation to that, we argue that "pure" Fisherian sexual selection alone would be very difficult to disentangle from other indirect benefits of mate choice (Kokko *et al.*, 2002, 2006).

Disentangling between the various benefits of choice is a highly complicated task, even in gonochorists (Kokko *et al.*, 2002, 2003; Fuller *et al.*, 2005; Kokko *et al.*, 2006): different benefits of choice are often interrelated (for example, assortative mating typically gives rise to the build-up of genetic correlations, so that Fisherian sexual selection often arises as a side effect in most other models of sexual selection), benefits of choice can be confounded with differential maternal investment in offspring from different sperm donors (Ratikainen & Kokko, 2010) and, importantly, indirect benefits of choice (benefits due to Fisherian sexual selection, good-genes and compatible genes) typically require large experimental sample sizes, since their effects are often small (Kirkpatrick & Barton, 1997; Cameron *et al.*, 2003).

Although Fisherian sexual selection may be difficult to distinguish from good-genes sexual selection, both can be disentangled experimentally from other benefits

of mate choice. (Hettyey *et al.*, 2010) proposed an integrated experimental design to distinguish between the different benefits of mate choice, which can be considered to fall in three categories: i) additive genetic benefits resulting from Fisherian or good-genes sexual selection, ii) nonaddative benefits as a consequence of compatible genes (Puurtinen *et al.*, 2009) or iii) direct benefits of mate choice, for example due to male parental care or the avoidance of mating costs (e.g., Price *et al.*, 1993; Gavrilets *et al.*, 2001). The approach consists of two steps: first, a large range of mate choice experiments are carried out to identify characters that are involved in mate choice. In essence, such mate choice experiments are identical to experiments that employ the path analysis approach proposed by (Arnold, 1994) and (Arnold & Duvall, 1994) (see Anthes *et al.* (2010) for an extension to simultaneous hermaphrodites), where individual characters are related to mating success, reproductive success and lifetime fitness. However, the approach of (Hettyey *et al.*, 2010) acknowledges that lifetime fitness is not enough to correctly identify all benefits of mate choice. In order to identify the benefits of all possible combinations of mate choice characters, they propose as a second step a separate North Carolina II breeding design, where a large set of possible sires and dams are mated in order to assess the benefits of a large range of combinations of characters involved in mate choice. The breeding design does not only include the count of offspring numbers to establish direct benefits, but also the reproductive success of such offspring is measured in order to account for genetic or compatible genes benefits of mate choice. Although this integrated breeding design still contains some weaknesses (e.g., differential maternal allocation is poorly accounted for), only rigorous experiments such as these will provide us with a clue if indirect benefits mechanisms, such as Fisherian sexual selection, occur in hermaphrodites.

Identifying sexually selected traits in hermaphrodites

While understanding which type of sexual selection is acting in hermaphrodites is an important ultimate goal, an important initial aim will be to simply identify traits that are involved in sexual selection, and to determine whether they act at the stage of, for example, mate searching, mate encounter, mating success, sperm transfer success, fertilization success, or post-embryonic provisioning (Anthes *et al.*, 2010). In contrast to gonochorists, where the simple presence of sexual dimorphism in sexual traits could be and often is considered an indication for the action of sexual selection, the absence of sex-limited expression in hermaphrodites makes it much less clear which traits are involved. While we by now have clear indications that traits like, for example, testis size (Janicke & Schärer, 2009) and love dart shooting and morphology (Koene & Schulenburg, 2005; Chase & Blanchard, 2006) are important for siring success, it is much less clear which processes should be considered female preference traits.

Given the simultaneous presence of ornament and preference in the same individual, it may not always be easy to decide which is which. Take the example of mate choice in the planarian *Dugesia gonocephala*. These flatworms appear to have a preference for mating with a large partner, possibly due to direct fecundity benefits of having a large (and thus fecund) partner or a good genes scenario (with high quality individuals achieving a larger size). Before mating, they glide onto each other, and appear to flatten themselves as much as possible, allowing them to assess the part-

ners size, and similar sized pairs are more likely to mate (Vreys & Michiels, 1997). While the initial signal, body size, may honestly reveal mate quality, and the initial preference may be linked to detecting this signal, there is scope for a Fisherian ornament that allows to exaggerate that signal, possibly using a special set of muscles that only serves to allow greater flattening (even if they cause a cost to the bearer in the natural selection context). Given the general preference for large size, such a trait would be expected to spread through the population. However, measuring the size of the partner then also requires greater flattening, thus the preference may also evolve, possibly by enhancing the sensory acuity of the edge of the body (even if this causes a cost to the bearer in the natural selection context). Similar scenarios may occur in a number of hermaphrodites with size-assortative mating.

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S3 Supplement

Table S3.1: A summary of the main notation used in the text.

variable	description
p	preference
t	ornament
$\bar{x}$	mean of phenotypic character g before natural and sexual selection
$\bar{x}^*$	mean of phenotypic character g after natural selection
$\bar{g}^{**}$	mean of phenotypic character g after both natural and sexual selection
a	strength of sexual selection
b	survival costs of the preference
c	survival costs of the ornament
$\varepsilon = 1$	absolute female preferences
$\varepsilon = 0$	relative female preferences
G_i	genetic variance of character i
G_{ij}	genetic covariance between characters i and j
ρ_{ij}	phenotypic correlation between characters i and j before natural and sexual selection
ρ^*_{ij}	phenotypic correlation between characters i and j after natural selection
ρ^{**}_{ij}	phenotypic correlation between characters i and j after both natural and sexual selection

Survival selection

We calculate the means and variances of t and p after survival selection (indicated by a *) for each of the different scenarios of sex-limited expression.

Gonochorists, complete sex-limited expression of t and p This scenario applies to gonochorists which have female choice only and typically only express ornaments in males and preferences in females. We start with a bivariate normal distribution $q(t,p)$ and the corresponding marginal distributions $q(t) = \int q(t,p)\mathrm{d}t$ and $q(p) = \int q(t,p)\mathrm{d}p$. For weak selection, we can derive the following approximate recursion equations to calculate the means and variances of this bivariate distribution $q^*(t,p)$ after survival selection in males and females:

$$\bar{t}^*_m = \int t q^*_m(t)\,\mathrm{d}t = \int t \frac{w^*_m(t)q(t)}{\int w^*_m(t)q(t)\mathrm{d}t}\mathrm{d}t = \frac{\bar{t}}{1+2cG_t},$$

$$\bar{t}^*_f = \int t q^*_f(t)\,\mathrm{d}t = \int t \int \frac{w^*_f(p)q(t,p)}{\iint w^*_f(p)q(t,p)\mathrm{d}p\mathrm{d}t}\mathrm{d}p\mathrm{d}t = \frac{\bar{t}(1+2bG_p) - 2b\bar{p}G_{tp}}{1+2bG_p},$$

$$\bar{p}^*_m = \int p q^*_m(p)\,\mathrm{d}p = \int p \int \frac{w^*_m(t)q(p,t)}{\iint w^*_m(t)q(p,t)\mathrm{d}t\mathrm{d}p}\mathrm{d}t\mathrm{d}p = \frac{\bar{p}(1+2cG_t) - 2c\bar{t}G_{tp}}{1+2cG_t},$$

$$\bar{p}^*_f = \int p q^*_f(p)\,\mathrm{d}p = \int p \frac{w^*_f(p)q(p)}{\int w^*_f(p)q(p)\mathrm{d}p}\mathrm{d}p = \frac{\bar{p}}{1+2bG_p},$$

$$
\begin{aligned}
G^*_{t_m} &= \int t^2 q^*_m(t)\,\mathrm{d}t - \bar{t}^{*2}_m = \frac{G_t}{1+2cG_t},\\
G^*_{t_f} &= \frac{G_t(1+2bG_p) - 2bG^2_{tp}}{1+2bG_p},\\
G^*_{p_m} &= \frac{G_p(1+2cG_t) - 2cG^2_{tp}}{1+2cG_t},\\
G^*_{p_f} &= \frac{G_p}{1+2bG_p},\\
G^*_{tp,m} &= \int tpq^*_m(t,p)\mathrm{d}t\mathrm{d}p - \bar{t}^*_m\bar{p}^*_m = \frac{G_{tp}}{1+2cG_t},\\
G^*_{tp,f} &= \frac{G_{tp}}{1+2bG_p}.
\end{aligned}
\tag{S3.1}
$$

Gonochorists, absence of sex-limited expression of t and p In the case of mutual choice, males and females both express costly ornaments and preferences simultaneously. Means and variances are then given by

$$
\begin{aligned}
t^* &= \int tq^*(t)\,\mathrm{d}t = \int t \int \frac{w^*(t,p)q(t,p)}{\iint w^*(t,p)q(t,p)\mathrm{d}p\mathrm{d}t}\mathrm{d}p\mathrm{d}t\\
&= \frac{\bar{t}\left(1+2bG_p\right) - 2b\bar{p}G_{tp}}{\left(1-2cG_t\right)\left(1-2bG_p\right) - 4bcG^2_{tp}}\\
\bar{p}^* &= \frac{\bar{p}\left(1+2cG_t\right) - 2c\bar{t}G_{tp}}{\left(1-2cG_t\right)\left(1-2bG_p\right) - 4bcG^2_{tp}}\\
G^*_t &= \frac{G_t(1+2bG_p) - 2bG^2_{tp}}{(1+2bG_p)(1+2cG_t) - 4bcG^2_{tp}},\\
G^*_p &= \frac{G_p(1+2cG_t) - 2cG^2_{tp}}{(1+2bG_p)(1+2cG_t) - 4bcG^2_{tp}},\\
G^*_{tp,f} &= \frac{G_{tp}}{(1+2bG_p)(1+2cG_t) - 4bcG^2_{tp}}.
\end{aligned}
\tag{S3.2}
$$

Hermaphrodites As gonochorists with mutual choice, an individual hermaphrodite expresses t as well as p. Hence, recursions after survival selection are identical to the equations for gonochorists with mutual choice listed in (S3.2) with subscripts dropped.

Distribution of p and t after mate choice

Here, we calculate the joint distribution of mated pairs $q^{**}(t,p)$ of t and p after mate choice for each of the mating scenarios discussed in the main text. Based on the joint character distribution $q^{**}(t,p)$, we can then derive the character means and variances after recombination and mutation, which are calculated in section S3.3.

S3.2.1 Gonochorists with female choice

Assume that females with character values $\{p_f, t_f\}$ choose among males with values $\{p_m, t_m\}$. The joint distribution $q^{**}(p_f, t_f, p_m, t_m)$ of $\{p_f, t_f\} \times \{p_m, t_m\}$ pairs can then be derived from their underlying conditional distributions, which is given by

$$\begin{aligned} q^{**}(p_f, t_f, p_m, t_m) &= q^*(p_f) q^*(t_f|p_f) q^*(t_m|p_f, t_f) q^*(p_m|p_f, t_f, t_m), \\ &= q^*\left(p_f\right) q^*\left(t_f|p_f\right) q^*\left(t_m|p_f\right) q^*\left(p_m|p_f, t_f, t_m\right), \\ &= q^*\left(p_f\right) q^*\left(t_f|p_f\right) q^*\left(t_m|p_f\right) q^*\left(p_m|t_m\right), \\ &= q^*(t_f, p_f) \frac{\psi(t_m|p_f)\, q^*(t_m)}{\int \psi(t_m|p_f)\, q^*(t_m) \mathrm{d}t_m} \frac{q^*(p_m|t_m)}{q^*(t_m)}, \\ &= q^*(t_f, p_f) q^*(t_m, p_m) \exp\left(-\frac{1}{2} a^2 p_f^2 G^*_{t,m} + a p_f (t_m - \bar{t}^*_m)\right). \end{aligned} \quad \text{(S3.3)}$$

where we assume that all females mate a single time in accordance to previous models (Lande, 1981; Pomiankowski *et al.*, 1991) (i.e.,the joint distribution is independent of female mating success).

Using matrix notation The expression above can also be derived using matrix notation which allows us to obtain means and variances of the pair distribution in a relatively simple fashion (see the note in section S3.5). Let $\mathbf{x}$ be a vector of the $\mathbf{x} = \begin{bmatrix} p_f & t_f & p_m & t_m \end{bmatrix}^{\mathrm{T}}$ and the variance-covariance matrix for sex i, $\mathbf{G}_i = \begin{bmatrix} G_{p,i} & G_{tp,i} \\ G_{tp,i} & G_{t,i} \end{bmatrix}$, where T denotes transposition. This allows us to write the product of the male and female joint trait distributions after natural selection as

$$q^*(t_f, p_f) q^*(t_m, p_m) = \frac{1}{4\pi^2 |\mathbf{G}^*|} \exp\left(-\mathbf{x}^{\mathrm{T}} \mathbf{A}^* \mathbf{x} + \mathbf{s}^{*\mathrm{T}} \mathbf{x} - \bar{\mathbf{x}}^{*\mathrm{T}} \mathbf{A}^* \bar{\mathbf{x}}^*\right), \quad \text{(S3.4)}$$

where $|\mathbf{G}^*|$ is the determinant of $\mathbf{G}^*$ and

$$\mathbf{A}^* = \frac{1}{2} \begin{bmatrix} \mathbf{G}_f^{*-1} & 0 \\ 0 & \mathbf{G}_m^{*-1} \end{bmatrix}, \quad \mathbf{s}^* = \begin{bmatrix} \mathbf{G}_f^{*-1} \begin{bmatrix} \bar{p}^*_f & \bar{t}^*_f \end{bmatrix}^{\mathrm{T}} \\ \mathbf{G}_m^{*-1} \begin{bmatrix} \bar{p}^*_m & \bar{t}^*_m \end{bmatrix}^{\mathrm{T}} \end{bmatrix}. \quad \text{(S3.5)}$$

Using eqns. (S3.4, S3.5), (S3.3) can now be written as

$$q^{**}(p_f, t_f, p_m, t_m) = \frac{1}{4\pi^2 |\mathbf{G}^*|} \exp\left(-\mathbf{x}^{\mathrm{T}} \mathbf{A}^*_1 \mathbf{x} + \mathbf{s}^{*\mathrm{T}}_1 \mathbf{x} - \frac{1}{2} \bar{\mathbf{x}}^{*\mathrm{T}} \mathbf{A}^* \bar{\mathbf{x}}^*\right), \quad \text{(S3.6)}$$

where

$$\mathbf{A}^*_1 = \frac{1}{2} \begin{bmatrix} G^*_{t,f}/|\mathbf{G}^*_f| + a^2 G^*_{t,m} & -G^*_{tp,f}/|\mathbf{G}^*_f| & 0 & -a \\ -G^*_{tp,f}/|\mathbf{G}^*_f| & G^*_{p,f}/|\mathbf{G}^*_f| & 0 & 0 \\ 0 & 0 & G^*_{t,m}/|\mathbf{G}^*_m| & -G^*_{tp,m}/|\mathbf{G}^*_m| \\ -a & 0 & -G_{tp,m}/|\mathbf{G}^*_m| & G^*_{p,m}/|\mathbf{G}^*_m| \end{bmatrix}, \quad \text{(S3.7)}$$

$$\mathbf{s}_1^{*\mathrm{T}} = \begin{bmatrix} \mathbf{G}_f^{*-1}\begin{bmatrix}\bar{p}_f^* & \bar{t}_f^*\end{bmatrix}^{\mathrm{T}} - \begin{bmatrix}a\bar{t}_m^* & 0\end{bmatrix}^{\mathrm{T}} \\ \mathbf{G}_m^{*-1}\begin{bmatrix}\bar{p}_m^* & \bar{t}_m^*\end{bmatrix}^{\mathrm{T}} \end{bmatrix}, \tag{S3.8}$$

Both $\mathbf{A}_1^*$ and $\mathbf{s}_1^*$ are obtained by respectively summing $\mathbf{A}^*$ and $\mathbf{s}^*$ with the corresponding matrix elements that are present in the exponential in eq. (S3.3). Using the note in section S3.5, we calculate the preference and ornament means $\bar{p}^{**}$, $\bar{t}^{**}$ after sexual selection:

$$\begin{aligned}\bar{p}^{**} =& \frac{\int \frac{1}{2}(p_f + p_m)q^{**}(p_f, t_f, p_m, t_m)\mathrm{d}\mathbf{x}}{\int q^{**}(p_f, t_f, p_m, t_m)\mathrm{d}\mathbf{x}}, \\ =& \frac{\mathbf{u}^{\mathrm{T}}\frac{1}{2}\mathbf{A}_1^{*-1}\mathbf{s}_1^* \exp\left(\frac{1}{4}\mathbf{s}_1^{*\mathrm{T}}\mathbf{A}_1^{*-1}\mathbf{s}_1^*\right)}{\exp\left(\frac{1}{4}\mathbf{s}_1^{*\mathrm{T}}\mathbf{A}_1^{*-1}\mathbf{s}_1^*\right)} = \frac{1}{2}\mathbf{u}^{\mathrm{T}}\mathbf{A}_1^{*-1}\mathbf{s}_1^*, \\ =& \frac{1}{2}\left(\bar{p}_f^*\left[1 + aG_{tp,m}^*\right] + \bar{p}_m^*\right),\end{aligned} \tag{S3.9}$$

where $\mathbf{u}^{\mathrm{T}} = \begin{bmatrix}\frac{1}{2} & 0 & \frac{1}{2} & 0\end{bmatrix}$. Similarly,

$$\begin{aligned}\bar{t}^{**} =& \frac{\int \frac{1}{2}(t_f + t_m)q^{**}(p_f, t_f, p_m, t_m)\mathrm{d}\mathbf{x}}{\int q^{**}(p_f, t_f, p_m, t_m)\mathrm{d}\mathbf{x}}, \\ =& \frac{1}{2}\left(\bar{t}_m^* + \bar{t}_f^* + a\bar{p}_f^* G_{t,m}^*\right).\end{aligned}$$

Similarly, we can use the general expressions in section S3.5 to work out the variances of p and t after sexual selection:

$$\begin{aligned}G_p^{**} =& \frac{1}{2}\left[\frac{1}{2}\left(\mathbf{A}_1^*\right)_{1,1} + \frac{1}{2}\left(\mathbf{A}_1^*\right)_{3,3}\right], \\ =& \frac{1}{2}G_{p,m}^* + \frac{1}{2}G_{p,f}^*\left(1 + a^2 G_{tp,m}^{*2}\right), \\ G_t^{**} =& \frac{1}{2}\left[\frac{1}{2}\left(\mathbf{A}_1^*\right)_{2,2} + \frac{1}{2}\left(\mathbf{A}_1^*\right)_{4,4}\right], \\ =& \frac{1}{2}G_{t,f}^* + \frac{1}{2}G_{t,m}^*\left(1 + a^2 G_{t,m}^* G_{t,f}^*\right). \\ G_{tp}^{**} =& \frac{1}{4}\left[\frac{1}{2}\left(\mathbf{A}_1^*\right)_{1,2} + \frac{1}{2}\left(\mathbf{A}_1^*\right)_{1,4} + \frac{1}{2}\left(\mathbf{A}_1^*\right)_{3,2} + \frac{1}{2}\left(\mathbf{A}_1^*\right)_{3,4}\right] \\ =& \frac{1}{4}\left[G_{tp,m}^*\left(1 + a\left[G_{tp,f}^* + aG_{t,m}^* G_{t,f}^*\right]\right) + G_{tp,f}^* + aG_{p,f}^* G_{t,m}^*\right].\end{aligned}$$

Since our exponential mate choice functions $\psi(t|p)$ maintain a Gaussian distribution after mate choice, the distributions of p and t after survival selection and mate choice are completely described by these means and variances.

S3.2.2 Gonochorists, mutual choice

We focus on a $(p_\mathrm{f}, t_\mathrm{f})$ female, who is courted by $(p_\mathrm{m}, t_\mathrm{m})$ males. The probability that a p_m male courts a t_f female, relative to all other surviving females is then proportional

to (e.g., Nakahashi, 2008, eq. 5):

$$\psi^*\left(t_\mathrm{f}|p_\mathrm{m}\right) = \frac{\psi\left(t_\mathrm{f}|p_\mathrm{m}\right)}{\bar{\psi}_z\left(p_\mathrm{m}\right)},$$
$$\bar{\psi}_z\left(p_\mathrm{m}\right) = \int \psi\left(t_\mathrm{f}|p_\mathrm{m}\right) q^*\left(t_\mathrm{f}\right) \mathrm{d}t_\mathrm{f} = \exp\left[z \cdot \frac{1}{2} a p_\mathrm{m}\left(a p_\mathrm{m} G^*_{t,\mathrm{f}} + 2\bar{t}^*_\mathrm{f}\right)\right]$$

where z $(0 \leq z \leq 1)$ is a parameter that reflects how increased courtship towards one female comes at the expense of courtship to other females. If $z = 1$, courtship effort towards one particular female comes at the expense of the courtship effort devoted to other females (Servedio & Lande, 2006). In other words, all males devote the same total courtship effort: males with no preference ($p_\mathrm{m} = 0$) randomly distribute their courtship effort over the female distribution. Males with preference $p_\mathrm{m} \neq 0$ distribute their courtship effort non-randomly across the female population, and spend most effort at those females for which $\psi\left(t_\mathrm{f}|p_\mathrm{m}\right)$ yields larger values.

Instead, if $z = 0$, we have $\psi^*\left(t_\mathrm{f}|p_\mathrm{m}\right) = \psi\left(t_\mathrm{f}|p_\mathrm{m}\right)$. In this case, the total courtship effort differs between males and is dependent on the value of the male preference p_m. Males with preference $p_\mathrm{m} = 0$ invest no effort in courtship at all, whereas males with ever larger values of p_m will devote ever more effort in courting females. As a result of ever larger values of p_m, these males become more apparent to all females alike. The plausibility of both scenarios is discussed in Servedio & Lande (2006) and will be highlighted in the discussion in the main text.

The distribution $\hat{g}^{**}_{p_\mathrm{m} t_\mathrm{m}}$ of $(p_\mathrm{m}, t_\mathrm{m})$ males courting a $(p_\mathrm{f}, t_\mathrm{f})$ female is then the distribution of surviving male phenotypes $q^*\left(p_\mathrm{m}, t_\mathrm{m}\right)$, multiplied by the probability that a particular p_m male courts a t_f female, relative to all other surviving males in the population

$$\hat{g}^{**}_{p_\mathrm{m} t_\mathrm{m}} = q^*\left(p_\mathrm{m}, t_\mathrm{m}\right) \frac{\psi^*\left(t_\mathrm{f}|p_\mathrm{m}\right)}{\int \psi^*\left(t_\mathrm{f}|p_\mathrm{m}\right) q^*\left(p_\mathrm{m}\right) \mathrm{d}p_\mathrm{m}}.$$

From this distribution $\hat{g}^{**}_{p_\mathrm{m} t_\mathrm{m}}$ of suitors, females make a choice dependent on their preference p_f. Similar to previous models, we assume a polygynous system where every female mates a single time and receives enough sperm to fertilize her eggs, while males may vary in their mating success. Female mating success is thus not affected by her attractiveness. The distribution of pairs after a female has chosen one of her suitors is

$$\begin{aligned} q^{**}\left(t_\mathrm{f}, p_\mathrm{f}, t_\mathrm{m}, p_\mathrm{m}\right) &= q^*\left(p_\mathrm{f}, t_\mathrm{f}\right) \hat{g}^{**}_{p_\mathrm{m} t_\mathrm{m}} \frac{\psi\left(t_\mathrm{m}|p_\mathrm{f}\right)}{\iint \psi\left(t_\mathrm{m}|p_\mathrm{f}\right) \hat{g}^{**}_{p_\mathrm{m} t_\mathrm{m}} \mathrm{d}t_\mathrm{m} \mathrm{d}p_\mathrm{m}} \\ &= q^*\left(p_\mathrm{f}, t_\mathrm{f}\right) q^*\left(p_\mathrm{m}, t_\mathrm{m}\right) \frac{\psi^*\left(t_\mathrm{f}|p_\mathrm{m}\right) \psi\left(t_\mathrm{m}|p_\mathrm{f}\right)}{\iint \psi\left(t_\mathrm{m}|p_\mathrm{f}\right) \psi^*\left(t_\mathrm{f}|p_\mathrm{m}\right) q^*\left(p_\mathrm{m}, t_\mathrm{m}\right) \mathrm{d}p_\mathrm{m} \mathrm{d}t_\mathrm{m}} \end{aligned}$$

Again, this is a Gaussian. When $z = 0$ (i.e., male courtship devoted to one female does not affect the degree of male courtship to other females), the quotient

in $q^{**}\left(t_{\mathrm{f}}, p_{\mathrm{f}}, t_{\mathrm{m}}, p_{\mathrm{m}}\right)$ equals

$$\exp\left[-a\left(\frac{1}{2}ap_{\mathrm{f}}^2G_{t,\mathrm{m}}^*+at_{\mathrm{f}}p_{\mathrm{f}}G_{tp,\mathrm{m}}^*+\frac{1}{2}at_f^2G_{p,m}^*-t_fp_m-t_mp_f+\bar{t}_m^*p_f+\bar{p}_m^*t_f\right)\right]. \tag{S3.10}$$

As in section S3.2.1, coefficients in the above exponent can now be added to the matrices $\mathbf{A}^*$ and $\mathbf{s}^*$ (S3.5) obtaining (up to a constant)

$$q^{**}(t_{\mathrm{f}}, p_{\mathrm{f}}, t_{\mathrm{m}}, p_{\mathrm{m}}) = \exp\left(-\frac{1}{2}\mathbf{x}^{\mathrm{T}}\mathbf{A}_2^*\mathbf{x}+\mathbf{s}_2^{*\mathrm{T}}\mathbf{x}\right) \tag{S3.11}$$

When $z > 0$ (i.e., a male's courtship devoted to one female comes at the expense of courtship to other females) an equivalent expression for eq. (S3.10) is long and tedious, but it can be shown that the matrices $\mathbf{A}_2^*$ and $\mathbf{s}_2^*$ are given by

$$\mathbf{A}_2^* = \begin{bmatrix} \frac{G_{t,\mathrm{f}}^*}{|\mathbf{G}_{\mathrm{f}}^*|}+\frac{a^2\left(G_{t,\mathrm{m}}^*+za^2G_{t,\mathrm{f}}^*|\mathbf{G}_{\mathrm{m}}^*|\right)}{1+za^2G_{t,\mathrm{f}}^*G_{p,\mathrm{m}}^*} & -\frac{G_{tp,\mathrm{f}}^*}{|\mathbf{G}_{\mathrm{f}}^*|}+\frac{a^2G_{tp,\mathrm{m}}^*}{1+za^2G_{t,\mathrm{f}}^*G_{p,\mathrm{m}}^*} & 0 & -a \\ -\frac{G_{tp,\mathrm{f}}^*}{|\mathbf{G}_{\mathrm{f}}^*|}+\frac{a^2G_{tp,\mathrm{m}}^*}{1+za^2G_{t,\mathrm{f}}^*G_{p,\mathrm{m}}^*} & \frac{G_{p,\mathrm{f}}^*}{|\mathbf{G}_{\mathrm{f}}^*|}+\frac{a^2G_{p,\mathrm{m}}^*}{1+za^2G_{t,\mathrm{f}}^*G_{p,\mathrm{m}}^*} & -a & 0 \\ 0 & -a & \frac{G_{t,\mathrm{m}}^*}{|\mathbf{G}_{\mathrm{m}}^*|}+za^2G_{t,\mathrm{f}}^* & -\frac{G_{tp,\mathrm{m}}^*}{|\mathbf{G}_{\mathrm{m}}^*|} \\ -a & 0 & -\frac{G_{tp,\mathrm{m}}^*}{|\mathbf{G}_{\mathrm{m}}^*|} & \frac{G_{p,\mathrm{m}}^*}{|\mathbf{G}_{\mathrm{m}}^*|} \end{bmatrix},$$

$$\mathbf{s}_2^* = \begin{bmatrix} \mathbf{G}_{\mathrm{f}}^{*-1}\begin{bmatrix}\bar{p}_{\mathrm{f}}^* & \bar{t}_{\mathrm{f}}^*\end{bmatrix}^{\mathrm{T}} \\ \mathbf{G}_{\mathrm{m}}^{*-1}\begin{bmatrix}\bar{p}_{\mathrm{m}}^* & \bar{t}_{\mathrm{m}}^*\end{bmatrix}^{\mathrm{T}} \end{bmatrix}$$

$$-\frac{a}{1+za^2G_{t,\mathrm{f}}^*G_{p,\mathrm{m}}^*}\begin{bmatrix} \bar{t}_{\mathrm{m}}^*\left(1+za^2G_{t,\mathrm{f}}^*G_{p,\mathrm{m}}^*\right)-z\left(aG_{tp,\mathrm{m}}^*\bar{t}_{\mathrm{f}}^*+a^2G_{tp,\mathrm{m}}^*G_{t,\mathrm{f}}^*\bar{p}_{\mathrm{m}}^*\right) \\ \bar{p}_{\mathrm{m}}^*-zaG_{p,\mathrm{m}}^*\bar{t}_{\mathrm{f}}^* \\ z\bar{p}_{\mathrm{m}}^*\left(1+za^2G_{t,\mathrm{f}}^*G_{p,\mathrm{m}}^*\right) \\ 0 \end{bmatrix}. \tag{S3.12}$$

For mutually choosing gonochorists, the overall means and variances after sexual selection are then:

$$\begin{aligned}
\bar{p}^{**} &= \frac{1}{2}a\left(\bar{p}_f^*G_{tp,m}^*+\bar{t}_f^*G_{p,m}^*\right)+\frac{1}{2}\bar{p}_m^*+\frac{1}{2}\bar{p}_f^*, \\
\bar{t}^{**} &= \frac{1}{2}a\left(\bar{t}_f^*G_{tp,m}^*+\bar{t}_f^*G_{p,m}^*\right)+\frac{1}{2}\bar{t}_m^*+\frac{1}{2}\bar{t}_f^*, \\
G_p^{**} &= \frac{1}{2}a^2\left(G_{tp,m}^{*2}G_{p,f}^*+2G_{tp,f}^*G_{tp,m}^*G_{p,m}^*+G_{p,m}^{*2}G_{t,f}^*\right)+\frac{1}{2}G_{p,f}^*+\frac{1}{2}G_{p,m}^*, \\
G_t^{**} &= \frac{1}{2}a^2\left(G_{tp,m}^{*2}G_{t,f}^*+2G_{tp,f}^*G_{tp,m}^*G_{t,m}^*+G_{t,m}^{*2}G_{p,f}\right)+\frac{1}{2}G_{t,f}^*+\frac{1}{2}G_{t,m}^*, \\
G_{tp}^{**} &= \frac{1}{4}a\left(G_{p,f}^*G_{t,m}^*+G_{p,m}^*G_{t,f}^*\right)\left(1+\frac{1}{2}aG_{tp,m}^*\right)+\frac{1}{4}G_{tp,f}^*\left(1+a^2G_{t,m}^*G_{p,m}^*\right) \\
&\quad +\frac{1}{2}G_{tp,m}^*\left(1+aG_{tp,f}^*\left[1+\frac{1}{2}aG_{tp,m}^*\right]\right).
\end{aligned}$$

Again, note that the recursions for the variances are independent of the mean trait values $\bar{t}^*$ and $\bar{p}^*$.

S3.2.3 Hermaphrodites, unilateral choice & insemination

Analogous to the default assumptions in models of Fisherian sexual selection in gonochorists, we assume that every individual always mates a single time in the female role, so that every individual receives enough sperm to fertilize its eggs (e.g., (Lande, 1981)). Similarly, we assume that every individual also can be chosen as a sperm donor multiple times, without suffering from sperm-limitation (polygyny).

Assume that an individual mating in the female role with character values $\{p_1, t_1\}$ chooses among sperm donors with values $\{p_2, t_2\}$. The joint distribution of all $\{p_1, t_1\}\times\{p_2, t_2\}$ pairs is then given by:

$$q^{**}(t_1, p_1, t_2, p_2) = q^*(t_1, p_1)q^*(t_2, p_2)\frac{\psi(t_2|p_1)}{\int \psi(t|p_1)\, q^*(t)\mathrm{d}t},$$

leading to similar arguments to work out each of the conditional distributions as in section S3.3. Hence, the distribution of mating pairs is not any different as for gonochorists and leads to the similar expressions for the moments after sexual selection as functions of the moments after natural selection (where the moments after natural selection are given by . Calculating the moments according to the steps in section S3.2.1, we get results for $\mathbf{A}_1^*$ and $\mathbf{s}_1^*$ that are similar to the expressions for gonochorists (eq. [S3.7]), where one omits the sex-specific indices of t and p.

$$\begin{aligned}
\mathbf{A}_1^* &= \frac{1}{2|\mathbf{G}^*|}\begin{bmatrix} G_t^* + a^2 G_t^*|\mathbf{G}^*| & -G_{tp}^* & 0 & -a|\mathbf{G}^*| \\ -G_{tp}^* & G_p^* & 0 & 0 \\ 0 & 0 & G_t^* & -G_{tp}^* \\ -a|\mathbf{G}^*| & 0 & -G_{tp}^* & G_p^* \end{bmatrix} \\
\mathbf{s}_1^{*\mathrm{T}} &= \begin{bmatrix} \mathbf{G}^{*-1}\bar{\mathbf{x}}^* - [a\bar{t}^* \quad 0]^{\mathrm{T}} \\ \mathbf{G}^{*-1}\bar{\mathbf{x}}^* \end{bmatrix}.
\end{aligned} \tag{S3.13}$$

Hence, executing the same procedures as in section S3.2.1, moments after sexual selection are:

$$\begin{aligned}
\bar{p}^{**} &= \frac{1}{2}\bar{p}^*\left[2 + aG_{tp}^*\right], \\
\bar{t}^{**} &= \frac{1}{2}\bar{t}^*\left[2 + a\bar{p}^* G_t^*\right], \\
G_p^{**} &= \frac{1}{2}G_p^*\left(2 + a^2 G_{tp,m}^{*2}\right), \\
G_t^{**} &= \frac{1}{2}G_t^*\left(2 + a^2 G_p^* G_t^*\right), \\
G_{tp}^{**} &= \frac{1}{4}G_{tp}^*\left(2 + aG_{tp}^* + a^2 G_t^* G_p^*\right) + \frac{1}{4}aG_t^* G_p^*.
\end{aligned} \tag{S3.14}$$

S3.2.4 Hermaphrodites, unilateral choice and reciprocal insemination

We maintain the assumption from the previous scenarios that each individual only chooses a mate once and subsequently receives an ejaculate from that mate, which is referred to as the primary donor. However, now the choosing individual will subsequently also donate sperm in return to the primary donor. An individual that is chosen multiple times as a primary donor will thus also receive multiple ejaculates back, allowing for sperm competition.

In order to arrive at a joint distribution of pairs, we focus on the population of individuals after mate choice and transfer of ejaculates, right before syngamy. As we have seen before, the probability density of $\{p_2, t_2\}$ individuals being chosen (based on t_2) by $\{p_1, t_1\}$ individuals (through p_1) is

$$q^{**}(t_2, p_2|t_1, p_1) = q^*(t_2, p_2)\frac{\psi(t_2|p_1)}{\int \psi(t|p_1)\, q^*(t)\mathrm{d}t}.$$

But there is also a chance that the $\{p_1, t_1\}$ individual gets chosen (based on t_2) by any $\{p_2, t_2\}$ individual (through p_2) and the probability that this occurs is proportional to

$$q^*(t_2, p_2)\frac{\psi(t_1|p_2)}{\int \psi(t|p_2)\, q^*(p_2)\mathrm{d}t}.$$

Now we have obtained the distributions of $\{p_1, t_1\}$ gametes that achieve syngamy (either through eggs or sperm). The total distribution of pairs is now

$$\begin{aligned}
q^{**}(t_1, p_1, t_2, p_2) &= q^{**}(t_1, p_1)q^{**}(t_2, p_2)\left[\frac{\psi(t_2|p_1)}{\int \psi(t|p_1)q^*(t)\mathrm{d}t} + \frac{\psi(t_2|p_1)}{\int \psi(t|p_1)q^*(t)\mathrm{d}t}\right], \\
&= q^{**}(t_1, p_1)q^{**}(t_2, p_2)\left[\exp\left(-\frac{1}{2}a^2 G_t^* p_1^2 + a p_1 t_2 - a p_1 \bar{t}^*\right)\right. \\
&\left. + \exp\left(-\frac{1}{2}a^2 G_t^* p_2^2 + a p_2 t_1 - a p_2 \bar{t}^*\right)\right], \\
&\equiv \tilde{q}^{**}(t_1, p_1, t_2, p_2) + \hat{q}^{**}(t_1, p_1, t_2, p_2).
\end{aligned}$$

Note that this joint distribution is not properly normalized yet (does not integrate to one), which will be fixed when computing the first and second order moments. Also note that a mixture of two normal distributions is not normal (can even be bimodal in extreme cases, when the means are far apart). Given that selection is rather weak, we assume that this is close enough to reflect a normal distribution. We again use matrix notation to calculate the moments, and we have

$$\begin{aligned}
\tilde{q}^{**}(t_1, p_1, t_2, p_2) &= \frac{1}{4\pi|\mathbf{G}^*|}\exp\left(-\mathbf{x}^{\mathrm{T}}\mathbf{A}_1\mathbf{x} + \mathbf{s}_1^{\mathrm{T}} - \frac{1}{2}\bar{\mathbf{x}}^{\mathrm{T}}\mathbf{A}\bar{\mathbf{x}}\right) \\
\hat{q}^{**}(t_1, p_1, t_2, p_2) &= \frac{1}{4\pi|\mathbf{G}^*|}\exp\left(-\mathbf{x}^{\mathrm{T}}\mathbf{A}_2\mathbf{x} + \mathbf{s}_2^{\mathrm{T}} - \frac{1}{2}\bar{\mathbf{x}}^{\mathrm{T}}\mathbf{A}\bar{\mathbf{x}}\right)
\end{aligned}$$

where $\mathbf{A}_1$ and $\mathbf{s}_1$ are given in eq. (S3.13) and

$$\mathbf{A}_2^* = \frac{1}{2|\mathbf{G}^*|}\begin{bmatrix} G_t^* & -G_{tp}^* & 0 & -a|\mathbf{G}^*| \\ -G_{tp}^* & G_p^* & 0 & 0 \\ 0 & 0 & G_t^* + a^2 G_t^* |\mathbf{G}^*| & -G_{tp}^* \\ -a|\mathbf{G}^*| & 0 & -G_{tp}^* & G_p^* \end{bmatrix}$$
$$\mathbf{s}_2^{*\mathrm{T}} = \begin{bmatrix} \mathbf{G}^{*-1}\bar{\mathbf{x}}^* \\ \mathbf{G}^{*-1}\bar{\mathbf{x}}^* - [a\bar{t}^* \quad 0]^{\mathrm{T}} \end{bmatrix}. \tag{S3.15}$$

Analogous to the procedures in section S3.2.1, the moments can now be calculated as follows

$$\begin{aligned}\bar{p}^{**} &= \frac{\int \frac{1}{2}(p_1+p_2)\tilde{q}^{**}(t_1,p_1,t_2,p_2)\mathrm{d}\mathbf{x} + \int \frac{1}{2}(p_1+p_2)\hat{q}^{**}(t_1,p_1,t_2,p_2)\mathrm{d}\mathbf{x}}{\int \left[\tilde{q}^{**}(t_1,p_1,t_2,p_2)+\hat{q}^{**}(t_1,p_1,t_2,p_2)\right]\mathrm{d}\mathbf{x}}, \\ &= \frac{\mathbf{u}^{\mathrm{T}}\frac{1}{2}\mathbf{A}_1^{*-1}\mathbf{s}_1^* \exp\left(\frac{1}{4}\mathbf{s}_1^{*\mathrm{T}}\mathbf{A}_1^{*-1}\mathbf{s}_1^*\right)/|\mathbf{A}_1^*| + \mathbf{u}^{\mathrm{T}}\frac{1}{2}\mathbf{A}_2^{*-1}\mathbf{s}_2^* \exp\left(\frac{1}{4}\mathbf{s}_2^{*\mathrm{T}}\mathbf{A}_2^{*-1}\mathbf{s}_2^*\right)/|\mathbf{A}_2^*|}{\exp\left(\frac{1}{4}\mathbf{s}_1^{*\mathrm{T}}\mathbf{A}_1^{*-1}\mathbf{s}_1^*\right)/|\mathbf{A}_1^*| + \exp\left(\frac{1}{4}\mathbf{s}_2^{*\mathrm{T}}\mathbf{A}_2^{*-1}\mathbf{s}_2^*\right)/|\mathbf{A}_2^*|},\end{aligned}$$

where $\mathbf{u}^{\mathrm{T}} = \begin{bmatrix}\frac{1}{2} & 0 & \frac{1}{2} & 0\end{bmatrix}$ as in section S3.2.1. Conveniently, it turns out that $|\mathbf{A}_1^*| = |\mathbf{A}_2^*|$ and $\mathbf{s}_1^{*\mathrm{T}}\mathbf{A}_1^{*-1}\mathbf{s}_1^* = \mathbf{s}_2^{*\mathrm{T}}\mathbf{A}_2^{*-1}\mathbf{s}_2^*$, hence the exponentials cancel and we are left with

$$\bar{p}^{**} = \frac{1}{4}\mathbf{u}^{\mathrm{T}}\left(\mathbf{A}_1^{*-1}\mathbf{s}_1^* + \mathbf{A}_2^{*-1}\mathbf{s}_2^*\right) = \frac{1}{2}\bar{p}\left(2 + a\mathbf{G}_{tp}^*\right).$$

Interestingly, this is exactly the same as for unilateral choice in (S3.14), which also holds for the other moments. Hence, there is no difference between unilateral and reciprocal insemination, except for the assumption that the reciprocal mixed distribution is normal, which is a given for the unilateral case.

S3.2.5 Hermaphrodites, mutual choice and reciprocal insemination

Knowing that reciprocal insemination in hermaphrodites does not alter the distribution of mated pairs (see previous section), we only need to focus on the pair distribution resulting from reciprocal mate choice, which we already obtained for gonochorists. Analogous to the procedure in section S3.2.2, we focus on a (p_1, t_1) sperm recipient, who is courted by (p_2, t_2) sperm donors. The intensity of courtship of a sperm donor with preference p_2 towards a sperm recipient with ornament t_1 is given by one of the three preference functions $\psi(t_1|p_2)$ discussed in the main text. The probability that a p_2 sperm donor courts a t_1 sperm recipient, relative to all other sperm recipients is then proportional to (e.g., Nakahashi, 2008, eq. 5):

$$\begin{aligned}\psi^*(t_1|p_2) &= \frac{\psi(t_1|p_2)}{\bar{\psi}_z(p_2)}, \\ \bar{\psi}_z(p_2) &= \int \psi(t_1|p_2)\, q^*(t_1)\,\mathrm{d}t_1 = \exp\left[z \cdot \frac{1}{2} a p_2 \left(a p_2 G_{t,1}^* + 2\bar{t}_1^*\right)\right].\end{aligned}$$

Similar to mutual choice in gonochorists, the parameter z ($0 \leq z \leq 1$) reflects how increased courtship towards one sperm recipient comes at the expense of courtship to other sperm recipients.

The distribution $\hat{g}^{**}_{p_2 t_2}$ of (p_2, t_2) donors courting a (p_1, t_1) recipient is then the distribution of surviving sperm donor phenotypes $q^*(p_2, t_2)$, multiplied by the probability that a particular p_2 donor courts a t_1 recipient, relative to all other surviving sperm donors in the population

$$\hat{g}^{**}_{p_2 t_2} = q^*(p_2, t_2) \frac{\psi^*(t_1|p_2)}{\int \psi^*(t_1|p_2)\, q^*(p_2)\, \mathrm{d}p_2}.$$

From this distribution $\hat{g}^{**}_{p_2 t_2}$ of suitors, sperm recipients make a choice dependent on their preference p_1. Also here, we assume a polygynous system where every individual mates in the female role a single time and receives enough sperm to fertilize her eggs, while an individual. Hence, mating success through the female role is thus not affected by an individual's attractiveness. The distribution of pairs after a recipient has chosen one of her suitors is

$$\begin{aligned} q^{**}(t_1, p_1, t_2, p_2) &= q^*(p_1, t_1)\, \hat{g}^{**}_{p_2 t_2} \frac{\psi(t_2|p_1)}{\iint \psi(t_2|p_1)\, \hat{g}^{**}_{p_2 t_2} \mathrm{d}t_2 \mathrm{d}p_2} \\ &= q^*(p_1, t_1)\, q^*(p_2, t_2) \frac{\psi^*(t_1|p_2)\, \psi(t_2|p_1)}{\iint \psi(t_2|p_1)\, \psi^*(t_1|p_2)\, q^*(p_2, t_2)\, \mathrm{d}p_2 \mathrm{d}t_2} \end{aligned}$$

When $z = 0$ (i.e., a donor's courtship devoted to one recipient does not affect the degree of that donor's courtship to other recipients), the quotient in $q^{**}(t_1, p_1, t_2, p_2)$ equals

$$\exp\left[-a\left(\frac{1}{2}a p_1^2 G^*_{t,2} + a t_1 p_1 G^*_{tp,2} + \frac{1}{2}a t_1^2 G^*_{p,2} - t_1 p_2 - t_2 p_1 + \bar{t}^*_2 p_1 + \bar{p}^*_2 t_f\right)\right]. \quad \text{(S3.16)}$$

which is analogous to the exponential function in eq. (S3.10) for mutual choice in gonochorists. We can reformulate the above expression using matrices $\mathbf{A}^*$ and $\mathbf{s}^*$ so that we obtain (up to a constant)

$$q^{**}(t_1, p_1, t_2, p_2) = \exp\left(-\frac{1}{2}\mathbf{x}^{\mathrm{T}} \mathbf{A}^*_2 \mathbf{x} + \mathbf{s}^{*\mathrm{T}}_2 \mathbf{x}\right) \quad \text{(S3.17)}$$

When $z > 0$ (i.e., a donor's courtship devoted to one recipient comes at the expense of courtship to other sperm recipients) matrices $\mathbf{A}^*_2$ and $\mathbf{s}^*_2$ are similar to those in equation (S3.12) for mutual choice in gonochorists, where the subscripts $_f$ and $_m$ are replaced by $_1$ and $_2$ respectively. Hence, for mutually choosing hermaphrodites, the overall means and variances after sexual selection are then:

$$\begin{aligned} \bar{p}^{**} &= \frac{1}{2}a\left(\bar{p}^*_f G^*_{tp,m} + \bar{t}^*_f G^*_{p,m}\right) + \frac{1}{2}\bar{p}^*_m + \frac{1}{2}\bar{p}^*_f, \\ \bar{t}^{**} &= \frac{1}{2}a\left(\bar{t}^*_f G^*_{tp,m} + \bar{t}^*_f G^*_{p,m}\right) + \frac{1}{2}\bar{t}^*_m + \frac{1}{2}\bar{t}^*_f, \\ G^{**}_p &= \frac{1}{2}a^2\left(G^{*2}_{tp,m} G^*_{p,f} + 2 G^*_{tp,f} G^*_{tp,m} G^*_{p,m} + G^{*2}_{p,m} G^*_{t,f}\right) + \frac{1}{2}G^*_{p,f} + \frac{1}{2}G^*_{p,m}, \end{aligned}$$

$$G_t^{**} = \frac{1}{2}a^2\left(G_{tp,m}^{*2}G_{t,f}^* + 2G_{tp,f}^*G_{tp,m}^*G_{t,m}^* + G_{t,m}^{*2}G_{p,f}\right) + \frac{1}{2}G_{t,f}^* + \frac{1}{2}G_{t,m}^*,$$
$$G_{tp}^{**} = \frac{1}{4}a\left(G_{p,f}^*G_{t,m}^* + G_{p,m}^*G_{t,f}^*\right)\left(1 + \frac{1}{2}aG_{tp,m}^*\right) + \frac{1}{4}G_{tp,f}^*\left(1 + a^2G_{t,m}^*G_{p,m}^*\right)$$
$$+ \frac{1}{2}G_{tp,m}^*\left(1 + aG_{tp,f}^*\left[1 + \frac{1}{2}aG_{tp,m}^*\right]\right).$$

Again, note that the recursions for the variances are independent of the mean trait values $\bar{t}^*$ and $\bar{p}^*$.

Recombination and mutation

Let $q_r(p_i t_j)$ be the density of offspring where parent $i \in \{1,2\}$ donates the p-locus and parent $j \in \{1,2\}$ the t-locus, where 1 indicates inheritance through eggs and 2 indicates inheritance through sperm. After recombination, the density of $\{p,t\}$ offspring is given by $\frac{1}{4}\sum_{i,j} q_r(p_i,t_j)$, where

$$\begin{aligned}
q_r(t_1,p_1) &= q^*(t_1,p_1),\\
q_r(t_2,p_1) &= \iint q^{**}(t_1,p_1,t_2,t_2)\mathrm{d}t_1\mathrm{d}p_2,\\
q_r(t_1,p_2) &= \iint q^{**}(t_1,p_1,t_2,t_2)\mathrm{d}t_2\mathrm{d}p_1,\\
q_r(t_2,p_2) &= \iint q^{**}(t_1,p_1,t_2,t_2)\mathrm{d}t_1\mathrm{d}p_1.
\end{aligned} \tag{S3.18}$$

In the first row of (S3.18), the joint distribution $q_r(t_1,p_1)$ is equal to the distribution after natural selection, since we assume that all eggs are successfully fertilized by a single ejaculate, and are hence independent of mating success. Hence, inheritance of both t_1 and p_1 through eggs is independent of a mother's mating success. This is different for traits inherited through sperm, t_2 and p_2, which are dependent on a sire's mating success. Hence, for the distributions involving any traits of the sire, the distribution after recombination is a function of the sire's mating success, incorporated in the joint distribution of pairs, $q^{**}(t_1,p_1,t_2,t_2)\mathrm{d}t_1\mathrm{d}p_1$.

We incorporate mutation as a continuum of alleles. After mutation, the joint distribution $q_\mu(t,p)$ can be approximated as follows (e.g., see Kimura (1965) eq. [6]), with a mean mutational effect of zero, a mutation rate μ and mutational variance ν:

$$q_\mu(t,p) \approx \frac{1}{4}\left(\sum_{i,j=\{1,2\}}\left[q_r(t_i,p_j) + \frac{1}{2}\mu\nu\left(\frac{\partial^2 q_r(t_i,p_j)}{\partial t_i^2} + \frac{\partial^2 q_r(t_i,p_j)}{\partial p_j^2}\right)\right]\right)\Bigg|_{t_i=t_j=t,p_i=p_j=p}. \tag{S3.19}$$

This is straightforward to implement using a computer algebra package like Mathematica or Maple. Note that mutation only affects the variances and not the means, since the mean mutational effect is assumed to be zero here. Hence, the recursions for all mating scenarios are then simply

$$\bar{p}_\mu \approx \bar{p}^{**}$$

$$\begin{aligned}\bar{t}_{\mu} &\approx \bar{t}^{**}\\ G_{p,\mu} &\approx G_p^{**} + \mu\nu,\\ G_{t,\mu} &\approx G_t^{**} + \mu\nu,\\ G_{tp,\mu} &\approx G_{tp}^{**}.\end{aligned} \tag{S3.20}$$

Equilibrium variances and stability analyses

Having obtained full recursions over the stages natural selection, sexual selection, recombination and mutation, we can then solve for the equilibrium variances and covariances near $\{\bar{t}, \bar{p}\} = \{0, 0\}$, assuming weak selection ($a \gg a^2,\ b \gg b^2,\ c \gg c^2$), and G_{tp} as well as $\mu\nu$ being small, so that second order terms are negligible. For all cases considered, we list the equilibrium variances and covariances in Table S3.3. The table shows that three factors affect differences in variances and covariances: (i) absence or presence of sex-limited expression of t and p, (ii) the different preference functions considered and (iii) absence or presence of mutual choice.

Under the assumptions used in the model, sex-limited expression of t and p occurs in the scenario where gonochorists exhibit female choice. In this case, an autosomal female preference or male ornament allele endures selection only when expressed in its corresponding sex, which happens only half of the time. In all the other scenarios, t and p are expressed in every individual, because of the lack of sex-limited expression (hermaphrodites), or because both sexes choose using their preference p and display their ornament t (gonochorists with mutual choice). Hence, the decrease in additive genetic (co)variation with increasing selection is less pronounced for gonochorists with female choice than for the other scenarios.

Stability of the trivial equilibrium For the system of recursion equations in (S3.20), we can calculate the stability matrix $\mathbf{J}$ evaluated at $\bar{t} = 0$ and $\bar{p} = 0$. In all cases considered in this study, the evaluation of the Jacobian conveniently results in a block diagonal matrix

$$\mathbf{J} = \begin{bmatrix} \mathbf{j} & \mathbf{0} \\ \mathbf{0} & \mathbf{g} \end{bmatrix},$$

where $\mathbf{j}$ is the 2×2 Jacobian for $\bar{t}$ and $\bar{p}$ and $\mathbf{g}$ the 3×3 Jacobian for the genetic variances.

Stability conditions for all scenarios are listed in Tables 3.1,S3.2 and graphically displayed in Figures 3.2,3.3 in the main text.

Table S3.2: Approximations of the stability conditions of the equilibrium $\{\bar{t}, \bar{p}\} = \{0, 0\}$ for absolute and relative preferences. Approximations are taken for first order terms around $\{a,\ b,\ c,\ \mu\nu,\ G_{tp}\} = 0$.

organism	choice scenario	insemination	stability condition
gonochorist	female choice	unilateral	$a(G_{tp} - \varepsilon G_t) > bG_p + cG_t$
	mutual choice	unilateral	$\frac{1}{2}a([1-\varepsilon]G_{tp} - G_p - \varepsilon G_t) > bG_p + cG_t$
hermaphrodite	unilateral choice	unilateral	$\frac{1}{2}a(G_{tp} - \varepsilon G_t) > bG_p + cG_t$
	unilateral choice	reciprocal	$\frac{1}{2}a(G_{tp} - \varepsilon G_t) > bG_p + cG_t$
	mutual choice	reciprocal	$\frac{1}{2}a([1-\varepsilon]G_{tp} - G_p - \varepsilon G_t) > bG_p + cG_t$

A brief note about Gaussian integrals

To understand the derivations in this supplement, the following facts about Gaussian integrals are helpful. We often encounter Gaussian functions of the form

$$q(\mathbf{x}) = \exp\left(-\mathbf{x}^{\mathrm{T}}\mathbf{A}\mathbf{x} + \mathbf{s}^{\mathrm{T}}\mathbf{x} + \mathbf{c}\right)$$

where $\mathbf{x}$, $\mathbf{s}$ and $\mathbf{c}$ are n-vectors and $\mathbf{A}$ is a symmetrical n-by-n matrix. Integrating over $\mathbf{R}^n$, we have

$$\int_{\mathbf{R}^n} q(\mathbf{x})\mathrm{d}\mathbf{x} = \frac{\left(\sqrt{\pi}\right)^n}{\sqrt{|\mathbf{A}|}} \exp\left(\frac{1}{4}\mathbf{s}^{\mathrm{T}}\mathbf{A}^{-1}\mathbf{s} + \mathbf{c}\right) \equiv K$$

$$\int_{\mathbf{R}^n} \mathbf{u}^{\mathrm{T}}\mathbf{x}\, q(\mathbf{x})\mathrm{d}\mathbf{x} = \frac{1}{2}\mathbf{u}^{\mathrm{T}}\mathbf{A}^{-1}\mathbf{s}K$$

$$\int_{\mathbf{R}^n} \mathbf{x}^{\mathrm{T}}\mathbf{D}\mathbf{x}\, q(\mathbf{x})\mathrm{d}\mathbf{x} = \left(\frac{1}{4}\mathbf{s}^{\mathrm{T}}\mathbf{A}^{-1}\mathbf{D}\mathbf{A}^{-1}\mathbf{s} + \frac{1}{2}\mathrm{Trace}\left[\mathbf{D}\mathbf{A}^{-1}\right]\right)K$$

Calculation of moments when $q(\mathbf{x})$ is a non-normalized density:

$$\mathrm{E}(x_i) = \frac{\int x_i\, q(\mathbf{x})\mathrm{d}\mathbf{x}}{\int q(\mathbf{x})\mathrm{d}\mathbf{x}} = \left[\frac{1}{2}\mathbf{A}^{-1}\mathbf{s}\right]_i$$

$$\mathrm{var}(x_i) = \frac{\int \left(x_i - \mathrm{E}(x_i)\right)^2 q(\mathbf{x})\mathrm{d}\mathbf{x}}{\int q(\mathbf{x})\mathrm{d}\mathbf{x}} = \frac{1}{2}\left[\mathbf{A}^{-1}\right]_{ii}$$

$$\mathrm{cov}(x_i, x_j) = \frac{\int \left(x_i - \mathrm{E}(x_i)\right)\left(x_j - \mathrm{E}(x_j)\right) q(\mathbf{x})\mathrm{d}\mathbf{x}}{\int q(\mathbf{x})\mathrm{d}\mathbf{x}} = \frac{1}{2}\left[\mathbf{A}^{-1}\right]_{ij}$$

Individual-based simulations

The results of the analytical model were checked by running individual-based simulations. We simulated a Fisherian process of sexual selection in a population of 5000 individuals and allowed two diploid loci, t and p, to evolve. In accordance to the an-

alytical model, we assumed a continuum-of-alleles model, in which the probability of mutation per allele is given by μ_t and μ_p for ornament and preference alleles respectively. Upon mutation, allelic values are updated with a value drawn from a normal distribution with mean 0 and variances $\nu_t = \nu_p = 0.0025$ respectively. Ornaments and preference values could take any real number, both positive and negative.

At the start of each generation, survival occurs stochastically by comparing a value drawn from a uniform distribution between 0 and 1 to the respective survival functions (see section "Survival" in the main text) for each individual. The pool of surviving individuals subsequently enter the mating stage, where they are allowed to choose a partner by assessing $n = 10$ randomly picked other survivors. The probability that a given partner in the sample of n will be chosen is proportional to the preference functions $\psi(t|p)$ listed in the paragraph "Preference functions", whereas it is weighed by the product of both preference functions $\psi(t_1|p_2) \times \psi(t_2|p_1)$ for both mutual choice in gonochorists and hermaphrodites. Multiple inseminations can occur in reciprocally inseminating hermaphrodites, where competition between multiple ejaculates is given by fair raffle sperm competition. After mating and fertilization, each individual produces 4 offspring, from which a new generation consisting of 5000 individuals was randomly selected. All simulations ran for 50 000 generations. The simulation code is written in C and is available upon request.

Table S3.3: Approximations for the additive genetic variances and covariances at equilibrium near the point $\{\bar{t}, \bar{p}\} = \{0, 0\}$. See Figure S3.1 for a graphical overview. Approximations are taken for first order terms around $\{a,\ b,\ c,\ \mu\nu,\ G_{tp}\} = 0$.

organism	choice scenario	insemination	preference function	$\hat{G}_t$	$\hat{G}_p$	$\hat{G}_{tp}$
gonochorist	female choice	unilateral	open-ended	$\sqrt{\frac{\mu\nu}{c}}$	$\sqrt{\frac{\mu\nu}{b}}$	$\frac{1}{2}\frac{a\mu\nu}{\sqrt{bc}}$
			absolute / relative	$\sqrt{\frac{\mu\nu}{a+c}}$	$\sqrt{\frac{\mu\nu}{b}}$	$\frac{a\mu\nu}{\sqrt{b(a+c)}}$
gonochorist	mutual choice	unilateral	open-ended	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{c}}$	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{b}}$	$\frac{1}{2}\frac{a\mu\nu}{\sqrt{bc}}$
			absolute / relative	$\sqrt{\frac{\mu\nu}{a+2c}}$	$\sqrt{\frac{\mu\nu}{a+2b}}$	$2\frac{a\mu\nu}{\sqrt{a+2b}\sqrt{a+2c}}$
hermaphrodite	unilateral choice	unilateral	open-ended	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{c}}$	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{b}}$	$\frac{1}{4}\frac{a\mu\nu}{\sqrt{bc}}$
			absolute / relative	$\sqrt{\frac{\mu\nu}{a+2c}}$	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{b}}$	$\frac{1}{2}a\mu\nu\sqrt{\frac{2}{b(a+2c)}}$
		reciprocal	open-ended	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{c}}$	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{b}}$	$\frac{1}{4}\frac{a\mu\nu}{\sqrt{bc}}$
			absolute / relative	$\sqrt{\frac{\mu\nu}{a+2c}}$	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{b}}$	$\frac{1}{2}a\mu\nu\sqrt{\frac{2}{b(a+2c)}}$
	mutual choice	mutual	open-ended	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{c}}$	$\frac{1}{2}\sqrt{\frac{2\mu\nu}{b}}$	$\frac{1}{2}\frac{a\mu\nu}{\sqrt{bc}}$
			absolute / relative	$\sqrt{\frac{\mu\nu}{a+2c}}$	$\sqrt{\frac{\mu\nu}{a+2b}}$	$2\frac{a\mu\nu}{\sqrt{a+2b}\sqrt{a+2c}}$

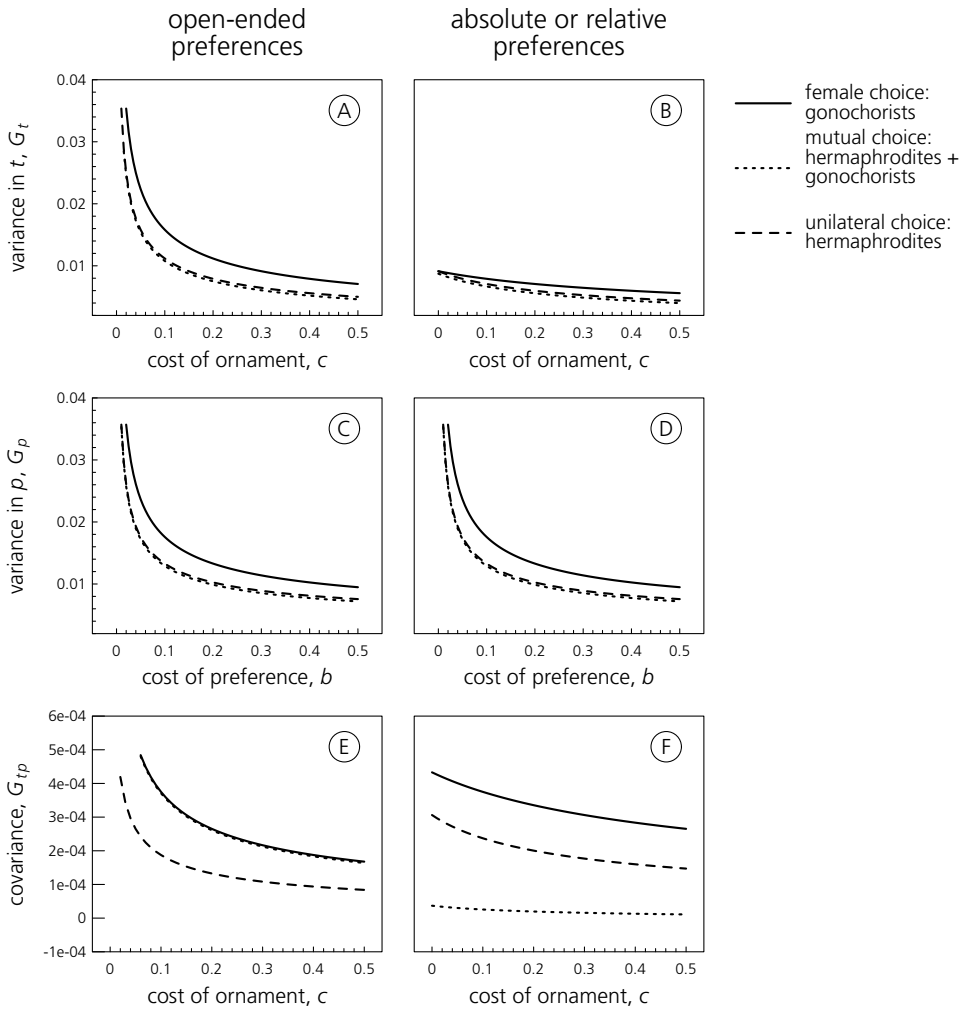

Figure S3.1: A graphical depiction of the analytical estimates of G_t, G_p and G_{tp} from Table S3.3 versus the strength of survival selection on ornaments c and preferences b. As expected, all genetic variances and covariances decrease under stronger survival selection. Nonetheless, this pattern is less pronounced for G_t and G_{tp} in case of absolute / relative preferences. Parameters: $\mu = 0.01$, $\nu = 0.0025$, $a = 0.3$.

CHAPTER 4

The evolution of haplodiploidy by male-killing endosymbionts: importance of population structure and endosymbiont mutualisms

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Abstract

Haplodiploid inheritance systems, characterized by male transmission of only their maternally inherited genomic elements, have evolved more than 20 times within the animal kingdom. A number of theoretical studies have argued that infection with certain male-killing endosymbionts can potentially lead to the evolution of haplodiploidy. By explicitly investigating the coevolutionary dynamics between host and endosymbiont, we show that the assumptions of current models cannot explain the evolution of haplodiploidy very well, since the endosymbiont will often go extinct in the long-term. Here, we provide two additional mechanisms that can explain the stable evolution of haplodiploidy by male-killing endosymbionts. First of all, a spatially structured population can facilitate the long-term persistence of haplodiploidy, but this applies only when levels of inbreeding are very high. In contrast, endosymbionts that are mutualistic with their hosts provide a much more general and promising route to the stable evolution of haplodiploidy. This model is the first to provide a formal explanation of the supposed association between the evolution of haplodiploidy and the highly inbred lifestyles of some ancestors, while it also provides a hypothesis for the evolution of haplodiploidy in more outbred ancestors.

4.1 Introduction

Haplodiploidy is a genetic system in which males transmit exclusively maternally inherited genes to the next generation. Arrhenotokous haplodiploidy (defined as the development of unfertilized haploid eggs into males and fertilized diploid eggs into females) is well known from groups such as the Hymenoptera and Thysanoptera. Haplodiploidy also comprises paternal genome elimination (PGE), in which the paternally inherited genome is eliminated from diploid male eggs. PGE is a common mode of inheritance in groups such as the scale insects (Iceryini, Neococcoidea) and sciarid flies (Sciaridae) (Hughes-Schrader, 1948; Haig, 1993). Haplodiploidy in both forms has evolved at least 20 independent times, 10 of which in insects (Otto & Jarne, 2001; Normark, 2003, 2004a). It is currently poorly understood why haplodiploidy has evolved only in some groups and not in others. A number of different hypotheses have been postulated on the adaptive significance of haplodiploidy:

1. *maternal transmission advantage*: all of the genes transmitted by haploid sons are of maternal origin, thereby partially circumventing the two-fold cost of sex (Brown, 1963, 1964). As noted by Normark (2004a), this advantage of haplodiploidy over diplodiploidy always holds true from the maternal perspective. However, it fails to explain why haplodiploidy is only found in specific groups of organisms.

2. *deleterious mutation clearance*: deleterious mutations can be purged more efficiently when there is an extensive haploid male phase (Goldstein, 1994). Again, clearance of deleterious mutations can be regarded as an inherent advantage to haplodiploidy and therefore fails to explain its particular phylogenetic distribution. For example, haplodiploidy as an adaptation to mutagenic or exposed environments does not match current ecological data on haplodiploid ancestors (Bell, 1982; Normark, 2004a). Furthermore, this hypothesis would not apply to certain forms of PGE in which the paternally inherited genome is still expressed in many tissues (as is the case in *Sciara* (Goday & Esteban, 2001) or in the lecanoid and diaspidid PGE systems in scale insects (Herrick & Seger, 1999)).

3. *maternal sex-ratio control*: arrhenotokous haplodiploidy facilitates maternal control of the sex-ratio, and it may thus be favored when such maternally controlled sex ratios are selectively favored, which can be the case under certain conditions of inbreeding (Hamilton, 1967; Borgia, 1980; Charnov *et al.*, 1981). Although this hypothesis matches with the inbred life histories of many haplodiploid ancestors, it fails to provide a mechanistic explanation for the transition between diplodiploidy and haplodiploidy. Also note that this hypothesis only applies to systems exhibiting PGE when some maternal control on the elimination of paternally inherited genome can be assumed.

4. *endosymbiont-induced haplodiploidy*: a more mechanistic extension of the previous hypothesis on sex-ratio control was provided by W.D. Hamilton, based on the observation that inbred haplodiploid groups such as bark beetles and mites are infected by endosymbiotic bacteria. Hamilton postulated that endosymbionts present in heterogametic males (XY) of a diplodiploid ancestor

may have eliminated or disabled the paternally inherited chromosome set, allowing only the transmission of the maternal, X-bearing chromosome set to offspring, which results in an overproduction of daughters as offspring. Especially under conditions of inbreeding, such higher proportions of daughters would be strongly favored by both the endosymbiont and the maternal host (Hamilton, 1978, 1993). In contrast to the previous two hypotheses, this hypothesis appears to focus more on the evolution of PGE than on arrhenotokous haplodiploid systems, since it still assumes fertilization of an egg by the paternal genome, after which elimination takes place.

Recently, Hamilton's idea about the role of endosymbionts in the evolution of haplodiploidy saw renewed interest after a meta-analysis indicated that other haplodiploid ancestors are also associated with maternally inherited endosymbionts (Normark, 2003). Moreover, almost all of the haplodiploid ancestors appear to have an increased scope for kin competition, since broods are gregarious and interact in a confined space such as crevices or bark galleries. Based on the supposed preponderance of both endosymbionts and gregarious broods, Normark (2004a) extended Hamilton's hypothesis by assuming an endosymbiont with a male-killing phenotype that would haploidize males. Male-killing endosymbionts are associated with gregarious broods, since these endosymbionts can only persist when enough resources are reallocated from killed males to their infected female sibs (Werren, 1987; Hurst, 1991; Freeland & McCabe, 1997).

The hypothetical endosymbiont in Normark's model achieves male-killing by elimination of the incoming paternal genome upon fertilization, when this genome carries a male-determining element (i.e., a Y chromosome). This renders male zygotes haploid and therefore inviable. Central to Normark's hypothesis is that some haploidized males survive this haploidization and may eventually evolve towards normal levels of survival. Normark showed that haplodiploidy according to this hypothesis would evolve in a relatively wide range of values of both resource reallocation efficiency and haploidized male viability (Normark, 2004a). More sophisticated analyses by Engelstädter & Hurst (2006) and Ubeda & Normark (2006), which also took into account sex-ratio selection, showed that the endosymbiont is able to persist whenever the product of endosymbiont transmission rate a and the average offspring survival R is larger than 1. However, neither of these studies investigated whether coevolution between host and endosymbiont can lead to long-term persistence of haploidizing endosymbionts. In this study, we demonstrate that current models in fact do not allow for a long-term persistence of haplodiploidy by male haploidizing endosymbionts and therefore do not provide a satisfactory explanation for the evolution of haplodiploidy. We show that additional ecological features of haplodiploid ancestors, such as spatial population structure and direct mutualistic benefits provided by the endosymbiont, are required for the long-term persistence of haplodiploidy.

Spatial population structure can have important consequences for the evolutionary dynamics of endosymbiont-induced haplodiploidy. On the one hand it may promote the coexistence of endosymbionts and their hosts, since female-biased sex-ratios caused by a sex-ratio-distorting endosymbiont may benefit the host under conditions of local mate competition (Vala *et al.*, 2003). Furthermore, local extinction-recolonization dynamics can stabilize coexistence between hosts and sex-ratio-distorters, such as male killing endosymbionts (Hatcher *et al.*, 2000; Groenenboom &

Hogeweg, 2002). On the other hand, spatial population structure may also hamper the coexistence of endosymbionts and their hosts, because inbreeding can devalue the maternal benefit of having haploidized sons, since inbreeding reduces the relative increase in relatedness of a mother to her haploid over her diploid sons (Smith, 2000). Given these opposing selection pressures, it is difficult to predict without a formal analysis whether or not spatial population structure facilitates the evolution of haplodiploidy.

In addition, the type of symbiosis between host and endosymbiont may have been an important factor affecting the coevolution of a haplodiploid ancestor with its endosymbiont. Maternally inherited endosymbionts can vary from being entirely parasitic manipulators of their host's reproductive systems, to having more mutualistic relationships in which the host accrues certain benefits from being infected (e.g. through provision of nutrients or protection against stress), extending even to relationships that are fully obligate and in which hosts are unable to reproduce without endosymbiont infection (Moran *et al.*, 2008). The previous models on the evolution of haplodiploidy considered male-killing endosymbionts that did not confer any direct benefits upon their host. Here we generalize these models by allowing for varying degrees of such direct benefits and investigate the effect of this on the long-term persistence of haplodiploidy.

In this paper, we use individual-based simulations in combination with an analytical kin selection model to examine the significance of spatial population structure and endosymbiont mutualisms for the evolution of haplodiploidy. In contrast to previous models that rely on invasion analyses, we used individual-based simulations in addition to an analytical reproductive value approach to examine the full coevolutionary dynamics between the host and the endosymbiont.

4.2 The model

The main goal of our model is to investigate the invasion prospects as well as the long-term persistence of an endosymbiont with a male haploidizing phenotype, in an initially diplodiploid population. The endosymbiont is transmitted vertically by a maternal host to her offspring with transmission probability a. Initially, the offspring have a 1:1 sex-ratio. Males that are infected by the endosymbiont are haploidized during early development. A proportion s of haploidized males are assumed to survive. A specific scenario would be that such surviving males have mutations in a pre-existing dosage compensation complex, so that it upregulates expression in a haploidized male to match expression levels of a diploid male. If a male does not survive haploidization, his resources are reallocated to the remaining members of the brood with efficiency factor b. This means that a proportion b of the resources allocated to such males will become available to their surviving sibs. Specifically, the relative amount of resources available to survivors is given by:

$$R = 1 + \frac{ba(1-s)}{2-a(1-s)}. \tag{4.1}$$

This equation shows that offspring of uninfected females have a baseline amount of resources of 1, while offspring of infected females receive an additional $ba(1-s)$ units of resources from brothers that did not survive haploidization, equally shared over the $2-a(1-s)$ surviving sibs.

Table 4.1: A summary of the main notation used in the current chapter.

Variable	Description
a	endosymbiont transmission probability of mutant foundress
a^*	endosymbiont transmission probability of resident foundress
k	number of males and females produced per foundress
s	survival probability of a focal haploidized male
$\bar{s}$	survival probability of a haploidized male sharing a brood with the focal male
$\tilde{s}$	survival probability of a haploidized male sharing a patch with the focal male
s^*	survival probability of a resident haploidized male
b	efficiency of resource redistribution from sons to siblings
$n_f^{i,u}$	number of infected i or uninfected u females
$n_m^{i,u}$	number of infected i (haploidized) or uninfected u (diploid) males
$\mathrm{u}_m^{i,u}$	equilibrium class frequency of infected i or uninfected u males
$\mathrm{u}_f^{i,u}$	equilibrium class frequency of infected i or uninfected u females
$\mathrm{v}_m^{i,u}$	reproductive value of infected i or uninfected u males
$\mathrm{v}_f^{i,u}$	reproductive value of infected i or uninfected u females
p_1^*	average numbers of mates of an uninfected male
p_2^*	average numbers of mates of an infected male
m	direct host survival benefit of possessing endosymbiont
N	number of foundresses per patch

Nonspatial model

Invasion condition for haplodiploidy We first describe our analytical framework by formulating a nonspatial version of the model, which is based on previous models by Engelstädter & Hurst (2006) and Ubeda & Normark (2006), but uses a reproductive value approach (Taylor, 1996a; Pen & Weissing, 2002). In A 4.1, we generalize previous models by adding the possibility that the endosymbiont can also provide direct benefits to its host. Specifically, an infected host has $1+m$ times the amount of resources of an uninfected host. Thus, we obtain a more general condition for the persistence of a male haploidizing endosymbiont:

$$(1+m)aR > 1 \tag{4.2}$$

Mutant invasion dynamics Following Ubeda & Normark (2006), control of a and s was given to either the host or the endosymbiont, resulting in four different coevolutionary scenarios (see Appendix A 4.2 on page 120 and Figure 4.1). In this analysis, we did not assume any direct benefits of being infected with the haploidizing endosymbiont. The results are summarized in Figure 4.1: if the endosymbiont is in control of the survival of haploidized males s, selection favors maximal resource reallocation to daughters, leading to complete mortality of infected males and either extinction of the population as a whole or the complete loss of the endosymbiont from the population (Figure 4.1, panel b,d). Therefore, invasion and successful short-term persistence of the endosymbiont in a population of hosts is only possible when the

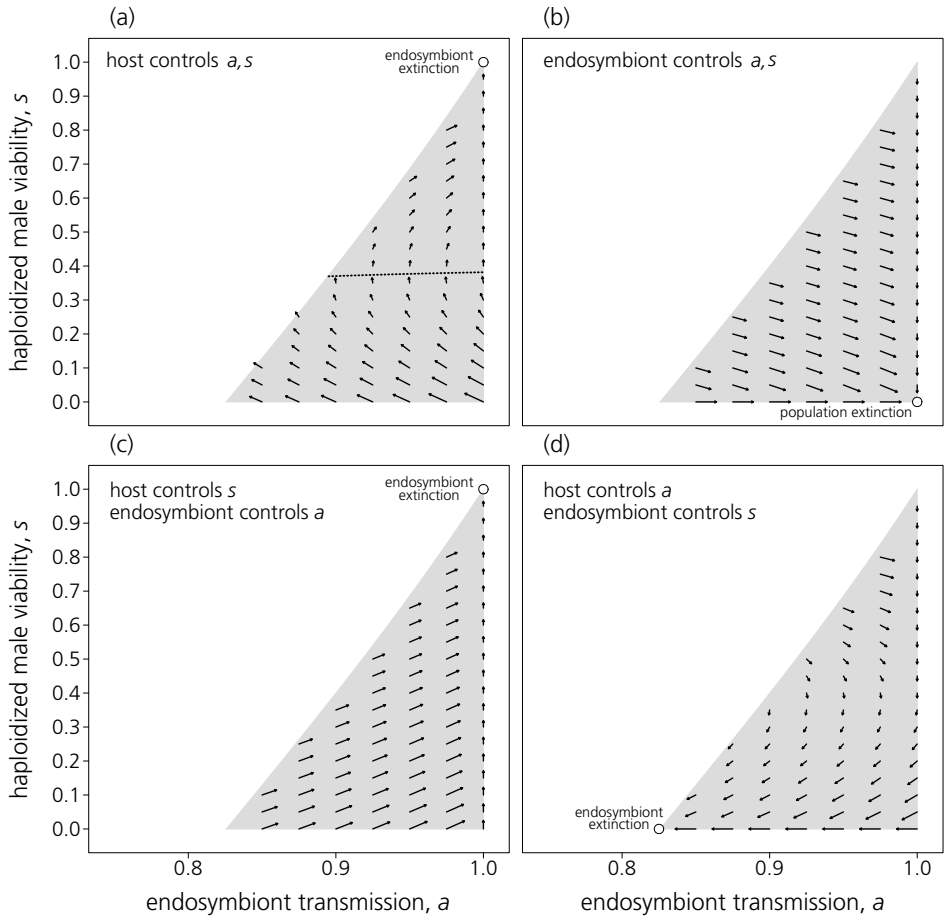

Figure 4.1: Coevolutionary dynamics of endosymbiont transmission a and haploidized male viability s in the non-spatial model, under different scenarios of host and endosymbiont control. The grey area depicts the parameter space in which the condition (4.2) is met, which allows persistence of the endosymbiont. In all four cases, evolution between host and endosymbiont leads to extinction of the endosymbiont. Panel (a): host control of both a and s. When s is still too low, females suffer from being infected with the endosymbiont and are selected for lower levels of a. This can lead to evolution outside the grey area and thus extinction of the endosymbiont. The dotted black line depicts the minimal value of s beyond which the host is selected to favor higher endosymbiont transmission rates, since at that point the maternal transmission advantage of the s viable haploidized sons outweighs the deaths of the $1-s$ remaining males. Beyond that line, a and s both evolve towards 1. At the point $\{a,s\} = \{1,1\}$, no resources are reallocated to the endosymbiont, and the endosymbiont goes extinct due to drift. Panel (b): endosymbiont control of both a and s. s is selected towards zero, and transmission rates a evolve towards 1, after which the population goes extinct due to lack of males. Panel (c): endosymbiont control of a, host control of s. The endosymbiont always will be selected to increase transmission a and the host is selected to increase s as well. Nevertheless, at the point $\{a,s\} = \{1,1\}$ the endosymbiont again goes extinct, since resource reallocation to infected hosts ceases. Panel (d): host control of a, endosymbiont control of s. s and a are both always selected against, leading to extinction of the endosymbiont. Parameters: $b = 0.3$, $m = 0$.

host is in control of s. In that case, the maternal host may benefit from the endosymbiont because viable haploidized sons are more efficient vehicles for her genes compared to diploid sons. Selection will then favor ever-increasing haploidized male viability s as well as endosymbiont transmission rates, regardless of who controls

the latter (Figure 4.1, panels a,c). This will continue until both s and a reach their maximal values of unity. These results were also obtained by Ubeda & Normark (2006). However, these authors did not point out that long-term persistence of the haploidizing endosymbiont is not possible in this equilibrium. The reason is that maximal survival of haploidized males implies that no reallocation of resources to daughters occurs ($R = 1$), in which case inequality (4.2) is no longer satisfied and the endosymbiont will drift to extinction. Additional mechanisms are thus needed to allow long-term persistence of the haploidizing endosymbiont in the population. Below, we investigate the role of spatial population structure as well as the role of direct benefits conferred by the endosymbiont upon its host.

Spatial model

Life cycle A population consists of 4000 initially diploid individuals and is subdivided into $2000/N$ identical patches, in which N is the number of foundresses per patch. To investigate the effect of inbreeding, N was varied: $N = \{2, 5, 10, 20, 50\}$. Each foundress produces 20 eggs and the sex of each egg is randomly assigned. If the foundress carries male haploidizing endosymbionts, each egg has a chance a of being infected, where a is determined by a single locus with many potential alleles and is either under maternal (diploid) or endosymbiont (haploid) control. At the start of each simulation the population was monomorphic for a specific transmission rate $a_{t=0}$ which was varied between simulations in the range from 0 to 1. Initially, 10% of the population was infected by the endosymbiont. After male-killing took place in a brood, resources were reallocated from killed males to their siblings according to the formula for R given in equation (4.1). Subsequently, both sexes undergo resource-based survival, in which their survival probability is given by $\frac{1}{2}R$.

Following resource-based survival, females mate in their natal patch with a random male (allowing for sib mating). If no fertilization opportunities are present, because no males survived the male-killing or the resource-based survival phase, the patch goes extinct. All fertilized females are added to a dispersal pool, from which the foundresses of the new generation are randomly selected to found a random patch. The mutation rates for a and s were fixed at 0.01 and the mutation steps were taken from a normal distribution with mean of 0 and standard deviation of 0.01. We discuss only the results when the host was in control of s, since simulations of our spatial model when the endosymbiont was in control of s were similar to the nonspatial version and therefore showed no long-term endosymbiont persistence. This agrees with the result of (Groenenboom & Hogeweg, 2002), who found endosymbiont persistence to occur only in spatially explicit models and not in spatially implicit models having random dispersal, which is similar to the approach we used.

Results In a scenario in which either the host or the endosymbiont has control of endosymbiont transmission, stable persistence of the haploidizing endosymbiont is generally not possible. Figure 4.2 depicts the persistence of the endosymbiont when the host is in control of its transmission: endosymbionts are generally unable to be maintained in the population for a period that is longer than 20 000 generations. Supplementary Figs. S4.1 and S4.2 show 10 replicate runs of such simulations over time, in the case of, respectively, host or endosymbiont control of a. The latter figures illustrate that the initial endosymbiont invasion is followed by ever-increasing numbers

of viable haploidized males, after which the endosymbiont goes extinct again

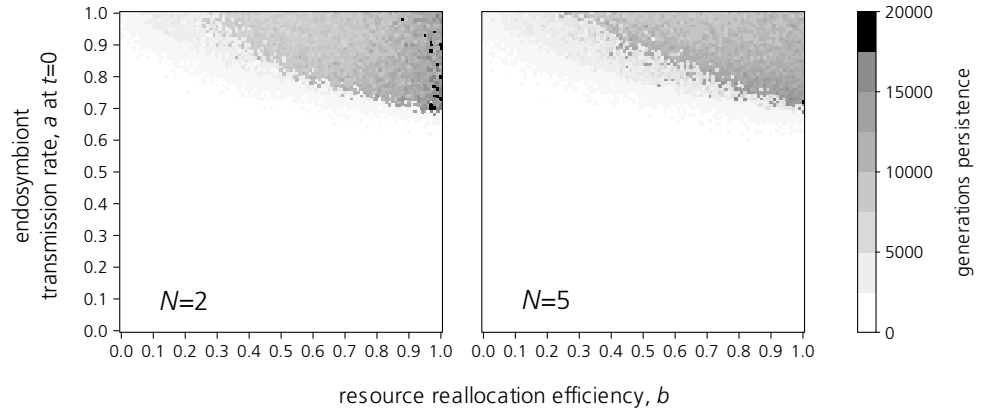

Figure 4.2: No stable persistence possible of the male haploidizing endosymbiont in the spatial model under female-biased dispersal, when the host is in control of both endosymbiont transmission probability a and haploidized male viability s. The color of each cell represents the persistence (in generations) of the haploidizing endosymbiont during a single simulation run (see Figure S4.1 for example simulations). Each run is characterized by an initial transmission probability $a_{t=0}$ (y-axis) and resource reallocation efficiency b (x-axis) for two different patch sizes $N = 2$ and $N = 5$. Results for $N = \{10, 20, 50\}$ resemble $N = 5$ and are therefore not shown. The male haploidizing endosymbiont will only persist for 10 000 generations or less. Only when inbreeding is very common, endosymbiont persistence and the presence of haploidized males is continued for longer than 20 000 generations (left panel), but also in these cases the endosymbiont will eventually go extinct (results not shown). $N = 2$ represents a boundary case of long-term persistence of the male haploidizing endosymbiont, when relatedness is very high (see S 4.1). In this figure, the host did not accrue any additional benefits from the endosymbiont, $m = 0$.

due to lack of resource reallocation, thereby restoring the initial diplodiploid population. The purging of the endosymbiont and the resulting loss of haploidized males for almost all values of $a_{t=0}$ and b in our simulations confirm our previous results from the nonspatial model that, although transient coexistence of host and endosymbiont may be possible in the short term, persistence of the endosymbiont and haploidized males is not possible in the long term.

Figure 4.2, however, also shows that in case of $N = 2$, the endosymbiont is sometimes able to persist for longer than 20 000 generations. Although extended simulations show that also in these cases, the endosymbiont will eventually go extinct before generation 30 000, a kin selection model in the S 4.1 shows that $N = 2$ represents a boundary case of a region of very high local relatedness in which long-term endosymbiont persistence is possible. When relatedness is high, local mate competition will select haploidized males to forgo mating themselves. Instead, a certain percentage of males now allow themselves to be killed, so that their resources can be reallocated to their dispersing sisters. In this scenario, optimal levels of s will thus be below 1, in which case $R > 1$ and therefore condition (4.2) remains satisfied. Thus, a spatial population structure can in principle allow for the stable persistence of haplodiploidy, but only under very high levels of inbreeding (see also Supplementary Figure S4.3).

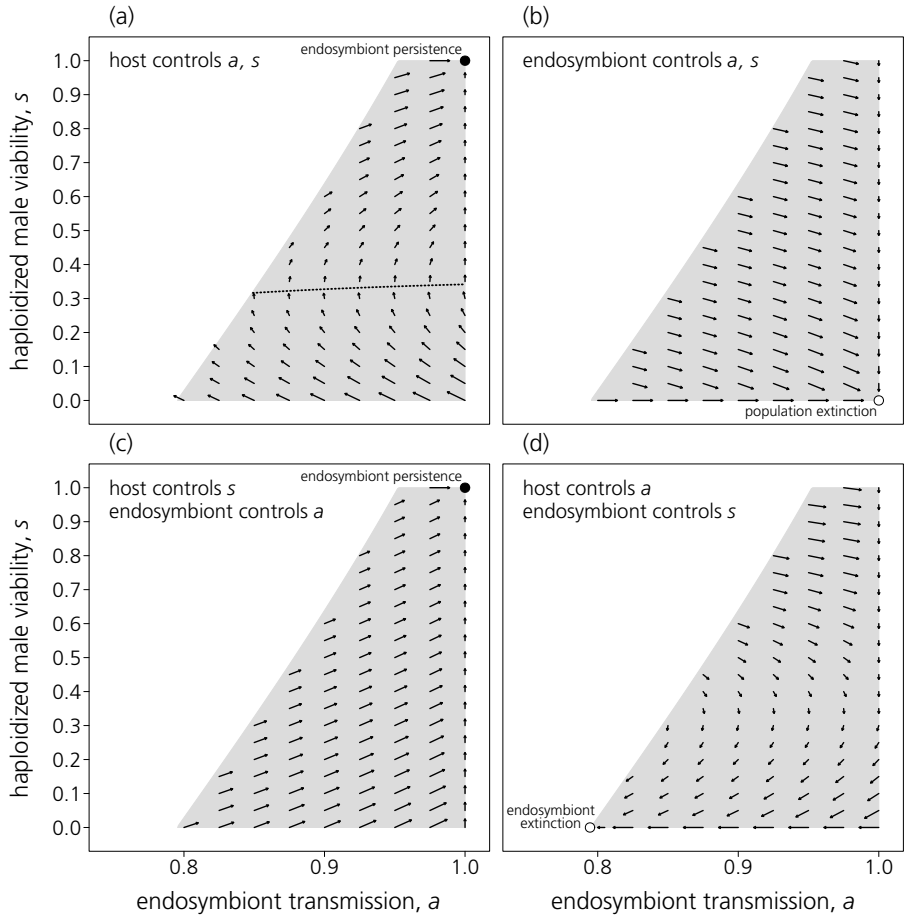

Figure 4.3: Coevolutionary dynamics of endosymbiont transmission a and haploidized male viability s in the nonspatial model, when the male haploidizing endosymbiont confers direct benefits ($m = 0.05$) upon its host. Direct benefits now allow for persistence of the endosymbiont, when the host is in control of s (panels a,c): a larger region now exists in which condition 4.2 is satisfied, which is especially important when coevolution has reached point $\{a, s\} = \{1, 1\}$. Direct benefits now maintain the endosymbiont's advantage in comparison to uninfected hosts, despite the lack of resource reallocation. Direct benefits do not alter the conclusions when the endosymbiont is in control of s: either extinction of both the host and endosymbiont occurs (panel b) or only that of the endosymbiont (panel d): the endosymbiont still favors $s = 0$, leading to extinction of either the host population or only the endosymbiont. Parameters: $b = 0.3, m = 0.05$.

Endosymbiont mutualisms

We can conclude from the previous sections that stable persistence of both the haploidizing endosymbiont and viable haploidized males is virtually impossible, unless inbreeding is extreme. However, the previous analysis only took into account a purely parasitic relationship of the endosymbiont with its host, whereas many cases exist in which an endosymbiont provides a competitive advantage to its host through means other than resource reallocation of killed males. An increasing number of examples are reported of endosymbiont infections in which the endosymbiont provides certain nutritional functions to its host (Moran *et al.*, 2008).

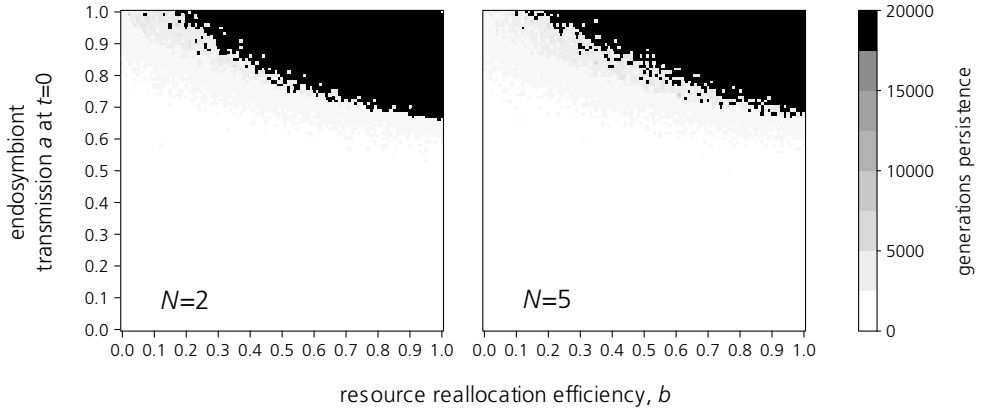

Figure 4.4: Small direct viability benefits from carrying the male haploidizing endosymbiont leads to stable persistence of haplodiploidy. As in Figure 4.2, the host is in control of both haploidized male viability s and the endosymbiont transmission rate a, but now the host has a direct viability advantage of 5% of carrying the endosymbiont ($m = 0.05$). Results for $N = \{10, 20, 50\}$ resemble $N = 5$ and are therefore not shown. For details, see Figure 4.2.

We incorporated a scenario of endosymbiont mutualism in the nonspatial model by allowing for a slight benefit of endosymbiont infection: $m = 0.05$ (see Figure 4.3). A possible scenario corresponding to such a value of m would be that the male haploidizing endosymbiont provides a certain nutrient to its host, but the host enjoys only a slight advantage from this (our model could consider obligate relationships between hosts and symbionts as well, when $m \to \infty$). When the host is in control of s, Figure 4.3 shows that $m > 0$ precludes extinction of the endosymbiont when the equilibrium $\{s, a\} = \{1, 1\}$ is attained: direct benefits always provide infected hosts with a competitive advantage over uninfected hosts, which allows the endosymbiont to be maintained in the long-term, even if resource reallocation from dead haploidized males ceases when s attains 1. We can thus conclude that even slight amounts of direct benefits of endosymbiont infection assure long-term persistence of the male haploidizing endosymbiont.

To check if these conclusions also apply to a spatial context, we ran simulations of our spatial model for three different values of endosymbiont survival benefits: $m = \{0.05, 0.11, 0.25\}$. Again, Figs. 4.4 and 4.5 show that the incorporation of small survival benefits drastically alleviates the restrictive conditions under which the haploidizing endosymbiont can stably persist; the endosymbiont is already maintained under modest values of $a_{t=0}$ and b and although the degree of local relatedness may increase the likelihood of endosymbiont persistence even further, it is not a necessary requirement for long-term stability. From both a spatial and a nonspatial version of our model, it can be concluded that even small degrees of endosymbiotic benefits can stabilize persistence of the male haploidizing endosymbiont.

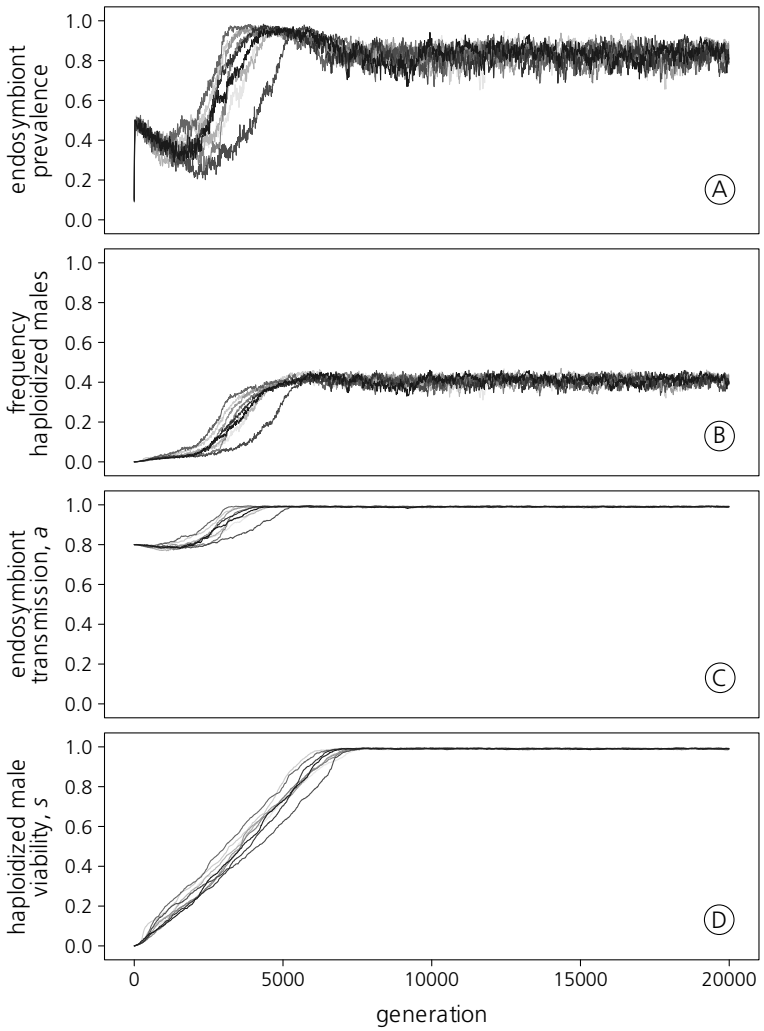

Figure 4.5: Ten replicate simulations in which male haploidizing endosymbionts are able to persist, since they constitute a direct survival benefit to their hosts. Panel (a) shows the total frequency of the endosymbiont in the population, panel (b) the frequency of haploidized males in the population, panel (c) the average endosymbiont transmission rate a under host control and panel (d) the average survival probability s under host control. Parameters: $b = 0.8$, $a_{t=0} = 0.8$, $m = 0.05$, $N = 20$.

4.3 Discussion

Three recent models explored the conditions under which endosymbionts with a male haploidizing phenotype could lead to the transition from diplodiploidy to a haplodiploid genetic system (Normark, 2004a; Engelstädter & Hurst, 2006; Ubeda & Normark, 2006). The general conclusion of these models was that endosymbiont-induced haplodiploidy (EIH) could in principle evolve, but only under rather restrictive conditions including high endosymbiont transmission and high levels of resource reallocation. Our analysis shows that achieving permanent haplodiploidy under the assumptions used in previous models is in fact not possible: scenarios that allow for

the invasion of the haploidizing endosymbionts and viable haploidized males will eventually also select for maximal levels of haploid male viability. As soon as all males survive from haploidization, resource reallocation to infected hosts ceases, thereby eliminating any fitness benefits for hosts infected with the endosymbiont and making the endosymbiont very likely to be purged due to drift effects. In order to achieve a permanent maintenance of EIH, additional mechanisms have to be assumed that maintain a permanent fitness advantage of infected over infected hosts.

Both this study and previous studies have not addressed scenarios in which control of haploidized male viability or endosymbiont transmission is 'shared' in some fashion between host and endosymbiont. The simplest scenarios of such joint control would be when gene products of host and endosymbiont interact either additively (e.g., phenotype is determined by the total amount of gene products present) or multiplicatively (e.g., gene products of the endosymbiont directly eliminate gene products of the host). In the case of additive control of haploidized male viability (s) one can easily imagine stable coexistence of host and endosymbiont: the optimal endosymbiont's viability level is $s_e = 0$, whereas the host's optimum is $s_h = 1$, leading to an average survival probability of $\bar{s} = 0.5$ in a haploidized male individual, which would lead to long-term coexistence (see eq. (4.2)). Since this scenario is optimal neither for the host nor the endosymbiont, it is likely to be prone to invasion by a modifier which either bypasses the currently used pathway, leading to full control of one party and eventually resulting in a scenario described in this and previous studies. In a simple multiplicative scenario, evolution of both loci would also lead to one party winning the conflict, since now $\bar{s} = s_e \cdot s_h = 0 \cdot 1 = 0$. To conclude, an important question left for future studies is to what extent more complex scenarios of interaction (i.e., multiple loci or specific genetic constraints) are able to prevent one party winning the conflict or at least prolong intermediate coexistence for a considerable time.

In this study, we investigated two different, but not mutually exclusive, routes that may lead to a situation in which a competitive advantage of infected hosts over uninfected hosts is maintained. First of all, by assuming a spatially substructured population with female-biased dispersal in which mothers produce a 1:1 sex-ratio, we showed that high levels of relatedness between random males and females within a deme may be sufficient to achieve stable haplodiploidy. As soon as the offspring of less than two foundresses compete on a patch, it can be worthwhile for a male to allow itself to be killed by the endosymbiont, to reallocate his resources to the sisters in his brood that disperse. When relatedness or resource reallocation efficiency is too low, a male is better off pursuing matings with other females on the patch and will be selected to maximize his survival probability. If the mother is capable of producing a female-biased sex-ratio without the action of the endosymbiont, this would preclude the evolution of haplodiploidy through inbreeding, since the very few males that are produced comprise insufficient resources for the endosymbiont to reallocate. However, strongly female-biased sex-ratios in diploid species are generally rare and would require additional assumptions such as gamete selection (Reiss, 1987; Pen & Weissing, 2002). The strong dependence of the evolution of haplodiploidy on the level of local relatedness closely matches Hamilton's predicted association of inbreeding and female-biased sex-ratios with haplodiploid ancestors (Hamilton, 1967). Our study is the first formal model that explicitly links the presence of endosymbionts and the inbred lifestyle of many of these haplodiploid ancestors with the actual evolution of

haplodiploidy.

A second route to haplodiploidy is when the competitive advantage of infected over uninfected hosts is realized by means other than resource reallocation (Hurst *et al.*, 1997). Endosymbionts can provide important functions for the host's nutrition, as demonstrated by numerous cases of endosymbionts that are involved in nutrition (Dale & Moran, 2006; Moran *et al.*, 2008; Janson *et al.*, 2008), or play a role in the host's reproduction (Peleg & Norris, 1972; Starr & Cline, 2002; Zchori-Fein *et al.*, 2006). By giving infected hosts a small survival advantage, we demonstrated that male haploidizing endosymbionts can persist across a much larger range of parameters, since their hosts always have a competitive edge over uninfected hosts, irrespective of potential resource reallocation. This also reduces the dependence of the male haploidizing endosymbiont on high levels of local relatedness: cessation of resource reallocation due to the complete rescue of haploidized males when $N \geq 2$ may reduce some part of the competitive advantage, but the direct survival benefit m maintains the haploidizing endosymbiont in the population.

To conclude, endosymbiont-induced haplodiploidy through the mechanism investigated in this study is likely to evolve through two different routes. The first route requires that four conditions are met, namely high transmission fidelity of the haploidizing endosymbionts, high levels of resource reallocation, extremely high relatedness and a sex chromosome system that prevents mothers from producing female-biased sex-ratios autonomously. The second route to haplodiploidy appears to be more general: it requires that the endosymbiont bestows direct benefits on its host, accompanied by minimally modest levels of endosymbiont transmission fidelity and efficiency of resource reallocation. We will now briefly address the empirical evidence on whether these conditions are likely to be met.

First, a key assumption of the EIH is the putative mechanism of male haploidization: that the endosymbiont detects the incoming male genome that carries a Y, and eliminates it before zygote development is fully initiated. Investigations into the molecular basis of male detection showed that male-killing *Spiroplasma* that infect *Drosophila* detect maleness based on specific proteins of the male dosage compensation complex (Veneti *et al.*, 2005). Killed males have intact germline formation and only somatic cells are affected, which is not in line with the EIH hypothesis. Recently, a different male-killing mechanism that acts at a much earlier stage of development has been found in the haplodiploid wasp *Nasonia*. Here, *Arsenophonus* bacteria blocked centrosome formation, thereby deregulating the first nuclear division of males (Ferree *et al.*, 2008). Nevertheless, this cytologically appealing mechanism of male killing is still confined to haplodiploids, in which ploidy differences between the sexes make cytological detection of males versus females potentially much more straightforward than in any diplodiploid ancestor. Our hypothesis would require a male-killing endosymbiont that i. could detect maleness before germline differentiation based on sex chromosome content or other sex-specific cytological cues, and ii. is able to eliminate the paternally inherited genome copy as a whole, potentially by targeting the formation of the paternally inherited centrosome. To what extent such a mechanism is plausible can only be investigated by assessing the cytological mechanisms that are used by other known male-killing endosymbionts, for example the different types of bacteria that are present in ladybirds (Hurst *et al.*, 1997). More information on potential idiosyncrasies in the cytogenetic machinery of haplodiploid ancestors may reveal why inheritance systems in certain clades appear to be much

more vulnerable to endosymbiont action than in others.

Related to the previous point on the detection of diploid males by the endosymbiont is the subsequent assumption of EIH that haploidized individuals are always transformed into males (Ubeda & Normark, 2006). This is especially problematic, since the genome containing a genetic element that is always associated with males (i.e., containing the Y chromosome) is assumed to be eliminated in our model. Moreover, in the insect model system *Drosophila*, haploid mutants develop as females and not as males. However, an important thing to note from the sex-determining cascades of *Drosophila* and other insects like *Musca* is that female development requires that the main protein on top of the sex-determining cascade successfully achieves a self-regulatory feedback loop (e.g., *sxlPe* in *Drosophila*), whereas male development starts when this feedback loop cannot be instantiated (Cline & Meyer, 1996; Burghardt *et al.*, 2005). When expression levels of these proteins on top of the cascade are thus disrupted during early development (e.g., expression levels are reduced due to haploidization), one may expect male instead of female development. In fact, the reason why haploid mutants do not develop as male in *Drosophila* is that an additional cell division leads to a longer time of sensitivity to *sxlPe* which thus initiates female development in haploid embryos, despite the lower levels of *sxlPe* expression from the haploid genome (Erickson & Quintero, 2007). To what extent haploid mutants of other insects may lack such additional embryonal characteristics and therefore could be prone to male development remains an open question. In any case, explicit modeling of the underlying sex-determining cascade in models that investigate the evolution of haplodiploidy may be necessary to assess which developmental mechanisms increase the likelihood of haploid individuals to develop as males.

Furthermore, making explicit assumptions about the mechanism of sex determination is important in the light of the maternal sex allocation decision. If the mother is not constrained by a chromosomal sex determining system to produce even sex-ratios, female-biased sex ratios under local mate competition would make it less beneficial for sons to allow male killing. However, it is an important question to what extent genetic sex determination allows the production of such biased sex-ratios, without assuming additional mechanisms such as gamete selection (Krackow, 2002) or, as is our focus here, male killing in combination with intermediate levels of viability as a first step towards the evolution of haplodiploidy.

Although our model aims to provide an explanation for the elimination of the paternally inherited genomes in males (PGE), it does not yet give a full account for the evolution of arrhenotokous haplodiploidy, which involves the development of viable haploid males from unfertilized eggs. We follow the conventional viewpoint that PGE can be considered a precursor to arrhenotoky (Cruickshank & Thomas, 1999; Normark, 2004a) and that our mechanism on the evolution of PGE may be followed by other adaptations regarding facultative fertilization of eggs, resulting in arrhenotoky. We note, however, that it is currently debated to what extent PGE can be considered to be a primitive form of arrhenotokous haplodiploidy or if both instances of haplodiploidy have evolved independently or even that PGE may be a derived form of arrhenotoky (Burt & Trivers, 2006; Normark, 2009). In that case, it remains to be seen if our model on EIH applies also to the ancestral form of arrhenotokous haplodiploidy.

The second condition regards the type of relationship that endosymbionts have with their hosts (parasitic or mutualistic). Cases of infection with endosymbionts are

present in all the insect haplodiploid ancestral groups, varying from endosymbionts that are strictly obligate to their host to presumably more transient and parasitic interactions involving endosymbionts such as *Wolbachia* (Normark (2004a) and references therein, see (Graham *et al.*, 2008) for evidence of *Wolbachia* in sawflies, the only ancestral group for which previously no endosymbionts have been reported). We showed that haplodiploidy can be achieved in two different ways: either when the condition of high local relatedness is met, or when the condition of direct endosymbiont benefits to their hosts is met. The first condition does not involve any assumption about the type of relationship the host has with its endosymbiont and both parasitic or mutualistic haploidizing bacteria could have induced haplodiploidy on their hosts. The second condition requires a strictly mutualistic relationship between host and endosymbiont. Normark (2003) noted that 4 out of 10 haplodiploid insect clades showed clear signs of inbreeding ('regular brother-sister mating'): the Hymenoptera, Thysanoptera and two bark beetle clades (Curculionidae: Scolytinae)). According to our hypothesis, we would predict that haplodiploidy in those inbred groups is caused by endosymbionts that could either be parasitic or mutualistic. Nevertheless, if our hypothesis would work, haplodiploidy in the other six ancestral insect groups should all involve infection with endosymbionts that provide certain benefits to their hosts. A clear sign of such mutualistic relationships are intricate host structures that interact and/or contain the endosymbionts, such as bacteriomes. Normark's analysis shows that such bacteriomes so far have only been observed in the non-inbred clades (Normark, 2004b), which is roughly in line with our hypothesis that outbred haplodiploid ancestors always contain mutualistic bacteria. However, more information on the incidence of parasitic or mutualistic bacteria in haplodiploid ancestors is necessary to make a proper quantification of the importance of mutualistic host-endosymbiont relationships to the evolution of haplodiploidy.

The third condition for the evolution of endosymbiont-induced haplodiploidy relates to the combination of high levels of endosymbiont transmission and high efficiencies of resource reallocation from killed males to infected females. These two conditions are important if haplodiploidy is to evolve via the condition of high local relatedness, but less so when the pathway to haplodiploidy is mediated by endosymbionts that are beneficial to the host. It is agreed that infection rates of male-killing endosymbionts appear to be generally high in nature (Hurst *et al.*, 2001; Jiggins *et al.*, 2002; Dyer & Jaenike, 2004; Charlat *et al.*, 2009), but the likelihood of high resource reallocation efficiencies has been debated (Engelstädter *et al.*, 2006; Ubeda & Normark, 2006). Fitness advantages for female offspring infected by male-killing endosymbionts due to reduced kin competition (Jaenike *et al.*, 2003) or cannibalism of killed males (Hurst *et al.*, 1993; Nakamura *et al.*, 2006) have been investigated in a number of organisms, but there is only a single study from which levels of b can be inferred (Dyer & Jaenike, 2004). Since this study on male-killing endosymbionts in *Drosophila innubila* reports the fitness benefit of infected versus infected females ($R \approx 1.04 - 1.05$), the survival rate of infected males ($s \approx 0 - 0.03$) and the transmission rate of the endosymbiont ($a \approx 0.97$), one can solve equation 4.1 for b while assuming no direct fitness effects of the endosymbiont ($m = 0$). Inferred values of b are between 0.045 and 0.055. If such low levels of resource reallocation efficiencies are the norm in insects infected with male-killing bacteria, any increased levels of male viability despite infection would quickly reduce aR to levels equal or below 1, unless inbreeding is extremely high ($N < 1.023 - 1.028$ for the above values of b, see

equation S5). Given such low levels of resource reallocation, we can therefore expect that it is much more likely that endosymbionts achieve long-term persistence if they provide some additional benefits to their host.

Another assumption that only applies to our spatial model is female-biased dispersal. The combination of female-biased dispersal and inbreeding is observed in a number of haplodiploid groups, of which the two bark-beetle clades Scolytinae and Xyleborini are the foremost examples. To a lesser extent, female-biased dispersal is also present in sawflies (Hymenoptera) and Thysanoptera, although the ancestral groups of the latter order are currently unresolved (Mound & Morris, 2007), making a characterization of ancestral traits difficult. To what extent inbreeding and female-biased dispersal have also played an important role in other haplodiploid groups is currently difficult to assess, due to the lack of well-resolved phylogenies, comparative data on the amount of inbreeding and information on dispersal asymmetries between the sexes. A systematic assessment of ancestral groups and their levels of inbreeding, dispersal asymmetries and prevalence of beneficial or parasitic endosymbionts may shed more light on the origins of haplodiploidy.

4.4 Acknowledgements

We thank Daniel Bopp for sharing ideas on the transition of an insect sex determining cascade from diplodiploidy to haplodiploidy, the ICT services of the University of Groningen for providing computational resources to run the simulations and Tim Fawcett for comments.

A 4 Appendix

Population dynamics in the nonspatial model

The number of uninfected (superscript u) and infected (superscript i) females (subscript f) and males (subscript m) can be described by the following recursion equations:

$$\begin{aligned} n_f^u(t+1) &= n_f^u(t) + n_f^i(t)(1-a)R \\ n_f^i(t+1) &= n_f^i(t)(1+m)\,aR \\ n_m^u(t+1) &= n_f^u(t) + n_f^i(t)(1-a)R \\ n_m^i(t+1) &= n_f^i(t)(1+m)\,aRs \end{aligned} \tag{A 4.1}$$

Uninfected mothers produce equal numbers of uninfected daughters and uninfected diploid sons. Infected mothers $n_f^i(t)$ obtain m additional resources compared to uninfected mothers due to direct benefits of possessing the endosymbiont. Furthermore, offspring from uninfected mothers receive R additional resources from their brothers who did not survive haploidization. Offspring of infected mothers are infected with probability a. If a son is infected, it will survive haploidization with probability s.

We can write the recursion equations above in matrix form $\mathbf{n}_{t+1} = \mathbf{A}\mathbf{n}_t$:

$$\begin{bmatrix} n_f^u \\ n_f^i \\ n_m^u \\ n_m^i \end{bmatrix}_{t+1} = \begin{bmatrix} 1 & (1-a)R & 0 & 0 \\ 0 & (1+m)\,aR & 0 & 0 \\ 1 & (1-a)R & 0 & 0 \\ 0 & (1+m)\,aRs & 0 & 0 \end{bmatrix} \begin{bmatrix} n_f^u \\ n_f^i \\ n_m^u \\ n_m^i \end{bmatrix}_t . \tag{A 4.2}$$

The transition matrix $\mathbf{A}$ has leading eigenvalues $\lambda_1 = 1$ and $\lambda_2 = (1+m)\,aR$, with corresponding right eigenvectors $\mathbf{x}_1 = [1,0,1,0]$ and $\mathbf{x}_2 = [z,1,z,s]$, where $z = (1-a)R/\left(\lambda_2 - 1\right)$. Clearly, only if $\lambda_2 > \lambda_1$ can the endosymbiont persist, which results in inequality (4.2). In that case, the stable distribution of infected and uninfected females and males is given by the right eigenvector $\mathbf{x}_2$.

Mutant invasion dynamics in the nonspatial model

To investigate if a resident population with strategy $\{a^*, s^*\}$ is stable against the invasion of mutant strategies, recursion equations also need to include the contribution of males to the next generation, since mutant strategies can arise in either males or females. In that case, the state-transition matrix $\mathbf{B}^*$ of the resident population is given by:

$$\mathbf{B}^* = \begin{bmatrix} \frac{1}{2} & \frac{1}{2}(1-a^*)R^* & \frac{1}{2}\left(p_1^* + p_2^*(1-a^*)R^*\right) & p_1^* + p_2^*(1-a^*)R^* \\ 0 & \frac{1}{2}(1+m)a^*R^* & \frac{1}{2}p_2^*(1+m)a^*R^* & p_2^*(1+m)a^*R^* \\ \frac{1}{2} & \frac{1}{2}(1-a^*)R^* & \frac{1}{2}\left(p_1^* + p_2^*(1-a^*)R^*\right) & p_1^* + p_2^*(1-a^*)R^* \\ 0 & \frac{1}{2}(1+m)a^*R^*s^* & 0 & 0 \end{bmatrix} \tag{A 4.3}$$

where $R^* = R(a^*, s^*)$ and $\{p_1, p_2\}$ are the expected numbers of mates per male with uninfected and infected females respectively, in which x_j^k are the corresponding values

from the leading right eigenvector $\mathbf{x}_2$:

$$p_1 = \frac{\mathrm{u}_f^u}{\mathrm{u}_m^u + \mathrm{u}_m^i} \qquad p_2 = \frac{\mathrm{u}_f^i}{\mathrm{u}_m^u + \mathrm{u}_m^i}.$$

The reproductive values of uninfected and infected females and males is given by the leading left eigenvector v:

$$\mathrm{v} = \left[1, 2(1-a^*)R^* + 2s^*(2\lambda_2 - 1), 2\lambda_2 - 1, 4\lambda_2 - 2\right]. \tag{A 4.4}$$

In the rest of this appendix, we will use these reproductive values to derive selection gradients for s and a for four different combinations of host and endosymbiont control.

Host control of a The invasion prospects of a rare mutant with strategy a in a population of residents with strategy a^* is governed by the transition matrix **B**. Following Taylor (1996a), the fitness gradient for the mutant strategy a can be calculated as follows:

$$\left.\frac{\partial W}{\partial a}\right|_{a=a^*} = \sum_{i,j} \mathrm{v}_i \mathrm{u}_j \left.\frac{\partial b_{ij}}{\partial a}\right|_{a=a^*} \tag{A 4.5}$$

which involves only taking into account the elements of matrix **B** (second column) that are dependent on the mutant strategy a:

$$\left.\frac{\partial W}{\partial a}\right|_{a=a^*} = (\mathrm{v}_f^u + \mathrm{v}_m^u)(-R^* + (1-a^*)R_a^*) + (\mathrm{v}_f^i + \mathrm{v}_m^i s)(R^* + a^* R_a^*)(1+m) \tag{A 4.6}$$

where R_a^* is $\partial R/\partial a|_{a=a^*}$.

Host control of s If the maternal host is in control of haploidized male viability, the invasion prospects of a rare mutant with strategy s is governed by the transition matrix **B**, where R in the second column is replaced by:

$$R = 1 + \frac{\frac{1}{2}ba^*((1-s)+(1-s^*))}{2 - \frac{1}{2}a^*((1-s)+(1-s^*))} \tag{A 4.7}$$

since half of the haploidized male offspring produced by a heterozygous mutant mother will carry the mutant allele. The fitness gradient then becomes:

$$\left.\frac{\partial W}{\partial s}\right|_{s=s^*} = \left(\mathrm{v}_f^u + \mathrm{v}_m^u\right)(1-a^*)R_s^* + \left(\mathrm{v}_f^i + \mathrm{v}_m^i s^*\right)(1+m)a^* R_s^* + \mathrm{v}_m^i(1+m)a^* R^* \tag{A 4.8}$$

Endosymbiont control of a Since from the viewpoint of a haploidizing endosymbiont, uninfected daughters and sons have zero reproductive value, selection will maximize the number of infected daughters produced by infected mothers, given by

$(1+m)aR$. The selection gradient is therefore given by:

$$\left.\frac{\partial W}{\partial a}\right|_{a=a^*} = (1+m)R^* + a^*(1+m)R_a^*. \tag{A 4.9}$$

where $R_a = \partial R/\partial a|_{a=a^*} > 0$. The selection gradient is always positive, hence the endosymbiont always favors increasing its own transmission rate.

Endosymbiont control of s By the same reasoning as in Appendix B.3, selection on endosymbiont control of s maximizes $(1+m)aR$, hence the selection gradient is given by:

$$\left.\frac{\partial W}{\partial s}\right|_{s=s^*} = a^*(1+m)R_s^* \tag{A 4.10}$$

where $R_s^* = \partial R/\partial s|_{s=s^*} < 0$. Thus selection favors zero survival of haploidized males.

S 4 Supplementary information

Extreme inbreeding facilitates haplodiploidy

In order to investigate the result from the individual-based simulations in which the male haploidizing endosymbiont persisted for a longer span of generations under conditions of high local relatedness, we analyzed local interactions on a patch by using an analytical model based on a direct fitness approach (Taylor & Frank, 1996). The individual-based simulations showed that evolution towards maximal levels of s preclude the stable persistence of the haploidizing endosymbiont, since this nullifies resource reallocation to infected females. When the haploidizing endosymbiont does not provide any direct survival benefits to the host, stable coexistence of the haploidizing endosymbiont and viable haploidized males is only possible if s does not evolve towards 1, but maintains intermediate levels (i.e., $0 < s < 1$). In this analysis, we investigate the conditions under which such intermediate values of s can be achieved.

We assume that endosymbiont transmission fidelity is maximized ($a = 1$), since this makes it feasible to keep track of the different phenotypes that are interacting in a local population. Our simulations warrant this assumption, since a always evolved towards 1 if the endosymbiont successfully persisted in the population (e.g., see Figure S4.1c).

Fitness equations Total fitness over male and female components can be expressed as the sum of male and female fitnesses, multiplied by the class reproductive values for males and females, c_m and c_f, respectively:

$$W = c_m W_m + c_f W_f$$

For $a = 1$, diploid infected females and haploidized males represent the different classes. Their fitnesses are:

$$W_f = R(\bar{s})$$

$$W_m = sR(\bar{s})\frac{1}{\tilde{s}} \tag{S 4.1}$$

in which s is the focal individual's survival probability as a haploidized male, whereas $\bar{s}, \tilde{s}$ are the average survival probabilities of the focal individual's brood

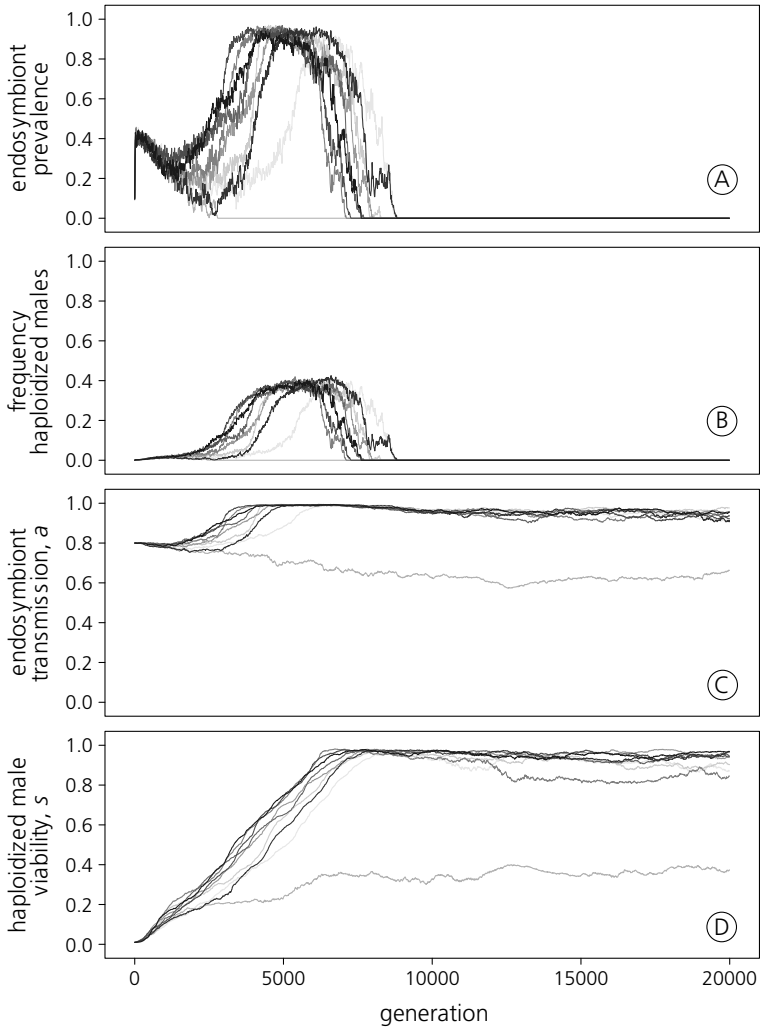

Figure S4.1: Ten replicate simulations showing the rise and fall of the male haploidizing endosymbiont and the transient presence of haploidized males in the population. Panel (a) shows the total frequency of the endosymbiont in the population, panel (b) the frequency of haploidized males in the population, panel (c) the average endosymbiont transmission rate a under host control and panel (d) the average survival probability s under host control. In 1 out of 10 replicates, the endosymbiont is purged from the population before generation 5000 (see text). If the endosymbiont manages to persist, hosts are selected that have ever higher levels of s (panels (b) and (d)) and more viable haploidized males will be present in the population. Subsequently, when most males are able to survive haploidization, resource reallocation from dead males to infected sisters will be diminished. Since this annihilates the competitive advantage of infected over uninfected females, this causes the decline of endosymbiont prevalence in the population until extinction. Once the endosymbiont goes extinct, both a and s are selectively neutral traits. Parameters: $b = 0.8$, $a_{t=0} = 0.8$, $m = 0$, $N = 20$.

and the average survival probabilities of haploidized males in the rest of the patch, respectively. The fitness gradient of haploidized male viability from the perspective of a haploidized male then becomes:

$$\left.\frac{\mathrm{d}W}{\mathrm{d}s}\right|_{s=\bar{s}=\tilde{s}} = c_f \frac{\partial W_f}{\partial \bar{s}} r_{\bar{f},m} + c_m \frac{\partial W_m}{\partial s} \cdot r_{m,m} + c_m \frac{\partial W_m}{\partial \bar{s}} r_{\bar{m},m} + c_m \frac{\partial W_m}{\partial \tilde{s}} r_{\tilde{m},m} \tag{S 4.2}$$

Relatedness coefficients between the focal male as an actor and other males and females in the brood or males in the patch as recipients can be calculated according to:

$$r^z_{xy} = \frac{f_{zy}}{f_{xy}} \tag{S 4.3}$$

in which r^z_{xy} represents the relatedness of individual y to x from the viewpoint of the actor z (Michod & Hamilton, 1980). f_{zy} is the coefficient of consanguinity between the actor z and another individual y, whereas f_{zx} is the coefficient of consanguinity between an individual and himself (or herself). In haploidized males $f_{zx} = 1$. Relatedness from the viewpoint of a haploidized male actor in control of its haploid survival strategy s to the relevant other patch members is given by:

$$\begin{aligned}
r_{m,m} &= 1 \\
r_{\tilde{m},m} &= \frac{1}{2} + \frac{1}{2}F \\
r_{\tilde{m},m} &= \frac{1}{N}\left(\frac{1}{2} + \frac{1}{2}F\right) \\
r_{\bar{f},m} &= \frac{1}{2}\left(\frac{1}{2}\cdot 1 + \frac{1}{2}F\right) + \frac{1}{2}F = \frac{1}{4} + \frac{3}{4}F
\end{aligned}$$

in which F is defined as the coefficient of inbreeding of the female part of a fertilized mother. F can be found by solving the following recursion equation:

$$F(t+1) = \frac{1}{N}\left[\frac{1}{2}\left(\frac{1}{2} + \frac{1}{2}F(t)\right) + \frac{1}{2}F(t)\right]$$

yielding:

$$F = \frac{1}{4N-3}$$

The class reproductive values correspond to a normal haplodiploid system if action of the endosymbiont is perfect (Taylor, 1988):

$$\{c_f, c_m\} = \{2, 1\}$$

Based on equation S 4.2, the fitness gradient therefore becomes:

$$\begin{aligned}
\frac{\mathrm{d}W}{\mathrm{d}s} = (1+m)\Bigg[& 2\frac{\mathrm{d}R(\bar{s})}{\mathrm{d}\bar{s}}\left(\frac{1}{4} + \frac{3}{4}F\right) + 1\cdot\frac{R(s)}{s} \\
& + 1\cdot\frac{\mathrm{d}R(s)}{\mathrm{d}\bar{s}}\left(\frac{1}{2} + \frac{1}{2}F\right) - 1\cdot\frac{R(s)}{s}\frac{1}{N}\left(\frac{1}{2} + \frac{1}{2}F\right)\Bigg]
\end{aligned} \tag{S 4.4}$$

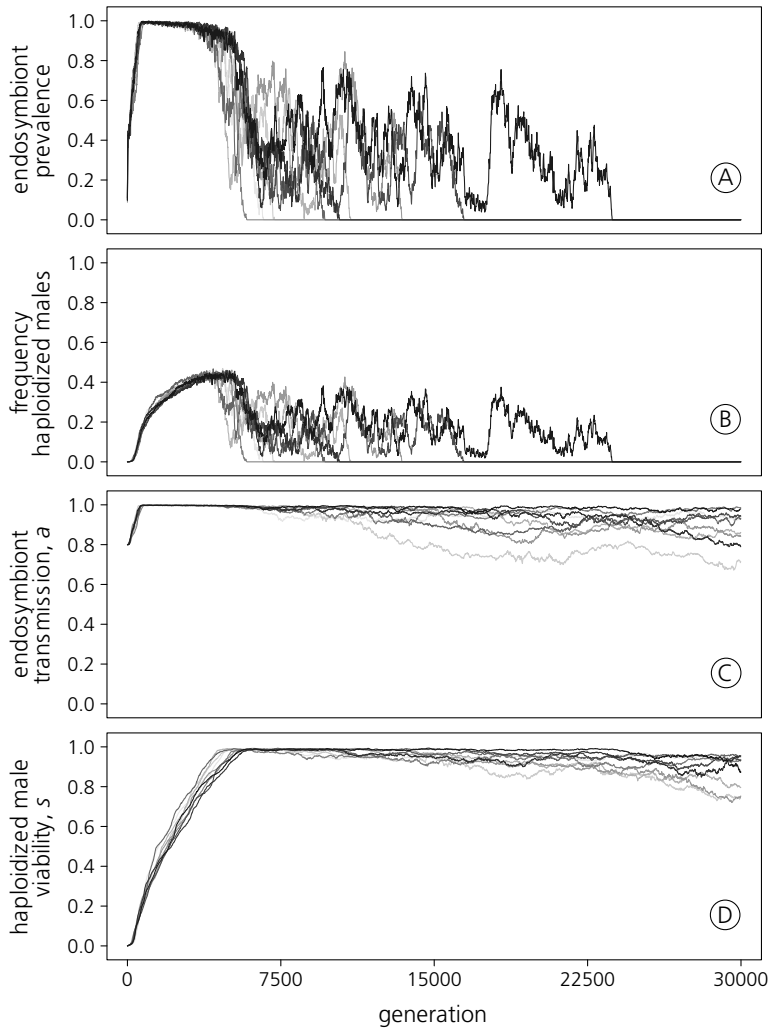

Figure S4.2: Ten replicate simulations showing the rise and subsequent decline of the endosymbiont, when it is in control of its own transmission rate, while the host controls s. Once viability has reached maximal levels ($s = 1$), resource reallocation ceases and infected hosts accrue no net fitness benefit compared to uninfected hosts. Drift subsequently leads to extinction of the endosymbiont. Parameters: $b = 0.8$, $a_{t=0} = 0.8$, $m = 0$, $N = 20$.

Evaluating the fitness gradient at $s = 0$ shows that values of $s > 0$ will always be selected for, since it will always pay off for a haploidized male to have a nonzero chance of survival. When evaluating the gradient at $s = 1$, negative values indicate that selection will lead to intermediate rescue of haploidized males and thus to stable endosymbiont persistence. Investigating the sign of the selection gradient at $s^* = 1$ yields the circumstances under which intermediate haploidized male survival probabilities are selectively favored

$$\left.\frac{\mathrm{d}W}{\mathrm{d}s}\right|_{s^*=\bar{s}=\tilde{s}=1} = \frac{(1+m)\left(2-4N^2(b-2)-N(10-b)\right)}{2N(4N-3)} \tag{S 4.5}$$

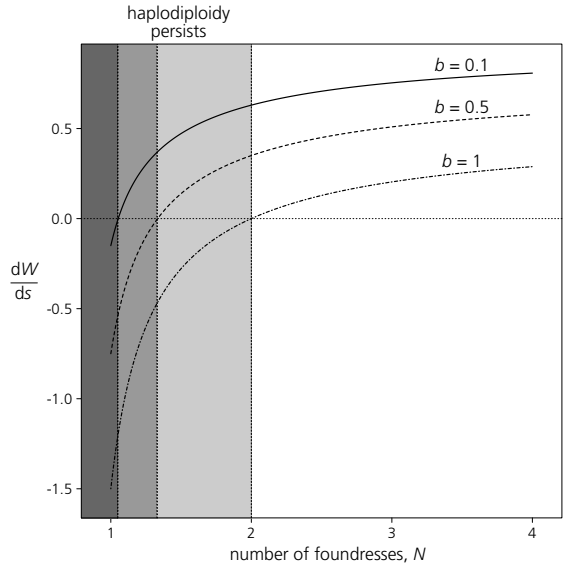

Figure S4.3: Stable persistence of haplodiploidy is only possible under very high local relatedness. The fitness gradient $(\mathrm{d}W/\mathrm{d}s)|_{s^*=\tilde{s}=\tilde{s}=1}$ is shown for different values of resource reallocation efficiency, b. Corresponding to each value of b, the grey areas indicate the range of local relatedness (expressed in number of foundresses, N) in which the selection gradient evaluated at $s = 1$ is negative, implying selection towards lower levels of s. Since $1-s$ males of each brood are still killed in these grey areas, resource reallocation is maintained, assuring the stable coexistence of the haploidizing endosymbiont and viable haploidized males.

In order to have stable persistence of both the haploidizing endosymbiont and haploidized males, the following condition must hold:

$$4N^2(b-2)+N(10-b)>2. \tag{S 4.6}$$

Figure S4.3 illustrates the latter condition by plotting the sign of the fitness gradient against N. This shows that only for values of $N < 2$ selection leads to intermediate values of s and thus stable persistence of the haploidizing endosymbiont. The requirement of high local relatedness in order to obtain stable persistence of haplodiploidy makes sense, since males will only allow themselves to be killed to a certain degree when reallocation of resources to their sisters outweighs the possibility of surviving haploidization in order to fertilize sisters and other females on the patch. The efficiency with which resources are reallocated, b, is crucial in determining the minimally required amount of local relatedness: if reallocation is maximal ($b = 1$), $N < 2$ suffices, but lower efficiencies also require more extreme degrees of local relatedness in order for males to be selected to sacrifice themselves to the good of their sisters. We can conclude that in the case where the host does not accrue any direct benefits from the haploidizing endosymbiont, only extreme levels of inbreeding allow for stable persistence of the haploidizing endosymbiont.

CHAPTER 5

Sex determination meltdown upon biological control introduction of the parasitoid *Cotesia Rubecula*?

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Abstract

Natural enemies may go through genetic bottlenecks during the process of biological control introductions. Such bottlenecks are expected to be particularly detrimental in parasitoid Hymenoptera that exhibit complementary sex determination (CSD). CSD is associated with a severe form of inbreeding depression because homozygosity at one or multiple sex loci leads to the production of diploid males that are typically inviable or sterile. We observed that diploid males occur at a relatively high rate (8-13% of diploid adults) in a field population of *Cotesia rubecula* in Minnesota, USA, where this parasitoid was introduced for biological control of the cabbage white *Pieris rapae*. However, our laboratory crosses suggest two-locus CSD in a native Dutch population of *C. rubecula* and moderately high survival of diploid males (approximately 70%), a scenario expected to produce low proportions of diploid males. We also show that courtship behavior of diploid males is similar to that of haploid males, but females mated to diploid males produce only very few daughters that are triploid. We use our laboratory data to estimate sex allele diversity in the field population of *C. rubecula* and discuss the possibility of a sex determination meltdown from two-locus CSD to effective single-locus CSD during or after introduction.

5.1 Introduction

Natural enemies used for importation biological control are at risk of going through population bottlenecks during collection, culturing or establishment. These bottlenecks may be associated with reduced genetic variation and are expected to impair biological control efficacy (Hopper *et al.*, 1993; Hufbauer & Roderick, 2005). Reduced genetic variation is especially problematic in parasitoids in the insect order Hymenoptera because of the production of diploid males (Stouthamer *et al.*, 1992). Males are normally haploid in sexually reproducing Hymenoptera as they develop from unfertilized eggs. Fertilized eggs usually develop as diploid females. Diploid males are the result of homozygosity at one or multiple sex loci under complementary sex determination (CSD) while female development is initiated by heterozygosity at these loci (Whiting, 1943; Beye *et al.*, 2003; De Boer *et al.*, 2008). Diploid males are thus produced instead of females, and because they are generally inviable or (effectively) sterile, they represent a severe form of inbreeding depression (Heimpel & De Boer, 2008). Under inbreeding or reduced allelic diversity at the sex locus, as many as 50% of diploid offspring develop as males. This may lead to male-biased sex ratios and can reduce population growth rates and rates of establishment (Stouthamer *et al.*, 1992; Heimpel & Lundgren, 2000, but see Hein *et al.*, 2009). Diploid male production under CSD may even theoretically lead to extinction through a so-called diploid male vortex: decreased population size leads to a reduction in sex allele diversity, leading to an increase in the production of diploid males, which in turn leads to further decreases in population size and so on (Zayed & Packer, 2005). Hence, mechanisms such as CSD may cause a sex determination meltdown in populations that endure inbreeding. However, surprisingly little information is available on the occurrence of diploid males in field populations of parasitoid wasps and on the consequences of CSD for biological control (Antolin *et al.*, 2003; Stouthamer *et al.*, 1992; Heimpel & Lundgren, 2000).

Despite its fundamental role in the development of animals, sex determination appears to be a rapidly evolving trait throughout the animal kingdom. Indeed, the CSD phenotype is present in many – but not all – hymenopteran species (Van Wilgenburg *et al.*, 2006). Mechanisms of sex determination in non-CSD species are poorly understood although recent evidence indicates a role for imprinting at the basis of the sex determination pathway in the parasitoid *Nasonia vitripennis* (Chalcidoidae) (Verhulst *et al.*, 2010b; Dobson & Tanouye, 1998). Phylogenetic analyses support CSD as the ancestral mechanism for haplodiploidy in the Hymenoptera, but it is currently not possible to determine whether one or more loci were responsible in the ancestral form of CSD (Asplen *et al.*, 2009). It is commonly assumed that *single-locus* CSD (*sl*-CSD) is ancestral and that evolution to other modes of sex determination without the production of diploid males has occurred, for example, in habitually inbreeding species (Van Wilgenburg *et al.*, 2006; Heimpel & De Boer, 2008). *Multiple-locus* CSD (*ml*-CSD) could have evolved from *sl*-CSD by one or more duplications of the sex locus. This significantly reduces the fitness costs of inbreeding because homozygosity at each sex locus is required for diploid male development (Crozier, 1971; De Boer *et al.*, 2008). We expect that the negative consequences of biological control introductions will also be reduced in species with *ml*-CSD compared to species with *sl*-CSD.

In this article, we investigated the sex determination mechanism of *Cotesia rubecula* Marshall, which has been introduced for biological control of the cabbage white

Pieris rapae L. in North America (Van Driesche, 2008). Based on the presence of CSD in other *Cotesia* species and previous unpublished results, we expected *C. rubecula* to exhibit CSD (Stouthamer *et al.*, 1992; Zhou *et al.*, 2006; De Boer *et al.*, 2007a). Our specific objectives were (i) to investigate diploid male occurrence and sex allele diversity in an introduced population of *C. rubecula*, (ii) to assess the number of CSD loci and diploid male survivorship, using a native population from the Netherlands, and (iii) to study diploid male courtship behavior and reproductive success. We aim at evaluating the results in the context of biological control introductions and discuss the potential for a sex determination meltdown in *C. rubecula*.

5.2 Material and methods

Insects

Parasitoid wasps are insects with free-living adults and parasitic larvae. Female parasitoids lay their eggs in or on a host insect on which their larvae develop. As the parasitoid larvae mature, the host insect is killed (Godfray, 1994). *Cotesia rubecula* (Hymenoptera: Braconidae) is a specialist parasitoid on caterpillars of the cabbage white *P. rapae*. It is a solitary parasitoid, producing a single offspring per host caterpillar. This species is native to Eurasia and has been introduced into N. America at various localities beginning in 1963 (Van Driesche, 2008), with releases in Minnesota, USA, in 1992 (Wold-Burkness *et al.*, 2005). Recent evidence suggests that it is displacing *Cotesia glomerata*, which had been released in N. America earlier (Wold-Burkness *et al.*, 2005; Van Driesche, 2008), and this displacement may have impacts not only on pests but also on native butterfly species through indirect pathways (Harvey *et al.*, 2010). *Cotesia rubecula* has also recently become established on *P. rapae* in New Zealand, where *P. rapae* has emerged as a serious threat to an endemic endangered plant (Cameron & Walker, 2002; Hasenbank *et al.*, 2011).

Collection of field material

In the summers of 2005 (July 6–August 8) and 2006 (July 5–August 16), *C. rubecula* cocoons and *P. rapae* caterpillars were collected from an organically maintained cabbage crop at the St. Paul campus of the University of Minnesota, USA. Collected hosts were reared in the laboratory on cabbage leaves in plastic containers to allow development of parasitoids. Upon emergence, *C. rubecula* wasps were sexed and frozen for flow cytometric analysis of ploidy level (see below).

Laboratory experiments

To start a laboratory culture, *C. rubecula* was collected in the summer of 2009 from *P. rapae* in cabbage fields around Wageningen University, the Netherlands, where it occurs natively. The wasps were reared on *P. rapae* on Brussels sprout plants (*Brassica oleracea* L. var. gemmifera cv. Cyrus) in a climatized room at the Laboratory of Entomology (20–22˚C, 50–70% RH, 16L:8D). All experiments were conducted at the Laboratory of Entomology in a climate-controlled room at 25 ± 1˚C, 60% RH and 16L:8D.

Mother-son crosses We first performed mother-son crosses (parental generation) to determine whether *C. rubecula* indeed has CSD. Under any form of CSD (single

or multiple loci), mother-son crosses should lead to the production of diploid male offspring because the number of alleles per putative sex locus is reduced to two and thus any mother-son cross is matched in terms of sex alleles. Because homozygosity is required at all sex loci to produce diploid males, however, the proportion of diploid males (DMP) decreases with the number of sex loci – even in mother-son crosses (Crozier, 1971; Heimpel & De Boer, 2008). Virgin *C. rubecula* females were first allowed to produce haploid sons by exposing individual newly emerged wasps overnight to approximately 12 first-to-second instar *P. rapae* larvae on a piece of cabbage leaf in Petri dishes (9 cm diameter). Females were then kept in a clean Petri dish with several drops of honey and moist cotton in an incubator at 20°C until their sons had developed, which took approximately 16 days. Parasitized hosts were reared on cabbage in transparent plastic 500-mL cups until cocoons appeared. The plastic cups had two ventilation holes (1.5 cm diameter) in the side covered with fine mesh. Fresh cabbage leaves were added regularly and dead hosts removed. When haploid males emerged, one male was paired with its mother in a plastic 50-mL tube with a piece of host-infested cabbage leaf and a droplet of honey for 24 h to allow mating. Males were subsequently frozen and females were exposed to approximately 30 first-to-second instar *P. rapae* in a Petri dish for 24 h. Females were exposed to a maximum of three such sets of hosts on three consecutive days, and parasitized hosts were reared as described above. The numbers of cocoons, host pupae and dead hosts were recorded for each replicate. Cocoons were separated in vials to provide virgin males and females to start the F1 generation (see below). We counted the number of males, females and non-emerged cocoons. Males were frozen at −25°C for flow cytometric analyses of ploidy level (see below). Of the 28 females that were allowed to mate with one of their own sons, two did not produce any offspring and 15 produced only males (presumably haploid as a result of no mating or unsuccessful mating). This left 11 successful mother-son matings for further experimental analysis.

Diploid male survival, behavior and fertility Offspring of mother-son crosses were used to set up the next series of crosses with the purpose of assessing diploid male survival and fertility and to gain insight into the number of loci underlying CSD, as well as comparing copulation behavior of haploid and diploid males. We made 52 brother-sister crosses with males of unknown ploidy (generation F1). Ploidy level was determined afterward by flow cytometry and crosses were categorized as brother-sister crosses with haploid males (33) and diploid males (16); ploidy level remained unknown in three cases. 27 control crosses were made by combining a female with a son from a different mother. Ploidy of these fathers was tested afterward with flow cytometry (see below), and we report the results of 23 crosses with haploid fathers (ploidy of two fathers remained unknown; two fathers were diploid and did not produce any offspring).

Copulation behavior was observed in all replicates of the three types of crosses by placing a virgin male and female together in a plastic 50-mL tube with a piece of host-damaged cabbage and a droplet of honey. We recorded the occurrence of wing fanning, which is an important component of copulation behavior in parasitoid wasps (Field & Keller, 1993b), the occurrence and location of mounting and time until mounting. Observations lasted until mounting was observed or for a maximum of 10 min. Pairs were subsequently left together for another 24-72 h. Males were then

frozen for analysis of ploidy level and females were exposed to two sets of approximately 30 hosts on two subsequent days as described above for mother-son crosses. Hosts were then reared to allow development of parasitoids. We counted the number of cocoons, dead hosts and *P. rapae* pupae as well as the number of females, males and non-emerged *C. rubecula* cocoons (generation F2). Offspring were frozen for analysis of ploidy level.

Flow cytometric analyses of ploidy level

Ploidy level was analyzed with flow cytometry following methods described previously (De Boer *et al.*, 2007b). In short, the head of an individual wasp was pulverized in 0.5 mL of Galbraith buffer (Galbraith *et al.*, 1983) and stained with propidium iodide (25 μg per sample). Analyses were done on a FACSCalibur flow cytometer (Becton Dickinson Immunocytometry, San Jose, CA, USA) for wasps collected in the field and on an Epics® XL^{TM} flow cytometer (Beckman Coulter, Brea, CA, USA) for laboratory crosses. DNA content of 2500 nuclei from head tissue was measured per wasp and the DNA histogram compared to that of known haploid males and diploid females to classify it as haploid, diploid or unknown. We analyzed ploidy level of 148 males and 60 females collected in the field, and ploidy level of all male offspring of 11 mother-son crosses, 23 brother-sister crosses with haploid fathers and 13 control crosses. Ploidy level was also analyzed for daughters from two brother-sister crosses with diploid fathers and for 10 males in the same two replicates. Ploidy level of 18 out of 208 field-collected wasps and 6 out of 556 males from laboratory crosses remained unknown after flow cytometry.

Data analyses

Data selection In the analyses of sex ratio and diploid male proportions of laboratory crosses, we included those replicates with at least seven diploid offspring because when seven diploid offspring (males and females) are produced, the probability that at least one of them is a diploid male under the null hypothesis of sl-CSD with full survival of diploid males is more than 99%. This led to the exclusion of three replicates of mother-son crosses, five replicates of brother-sister crosses with haploid fathers and four replicates of control crosses. In addition, two brother-sister crosses with haploid fathers were discarded because the mother-son cross from which they originated was excluded. In the analyses of DMP and diploid family size, we included only those control crosses for which we determined ploidy level of all male offspring (i.e. $N = 10$ after removing replicates with < 7 diploid offspring). Compared to analyses of the complete dataset (including replicates with < 7 diploid offspring), data selection does not influence our conclusions.

Simulations to assess the number of CSD loci We used a simulation model to statistically compare our results of diploid male production to predictions of CSD while varying two parameters: the number of putative CSD-loci, n_{loci} (1, 2 or 3), and the survival probability s of diploid males (between 0 and 1). This model was designed to simulate our experiment exactly in terms of female wasps used in both generations and number of diploid offspring per female. Individual males and females were represented by one (haploid) or two (diploid) binary strings, each of length n_{loci}. While

diploid family size was equal to the observed values, the number of surviving diploid males varied according to n_{loci} and s. We assumed no linkage between the putative CSD loci for $n_{\text{loci}} > 1$. Increasing linkage would result in outcomes intermediate to the single and two-locus results presented in Figure 5.1A, B. Details of the simulation model are presented in the Supplementary Information on page 143.

We compared our data on diploid male production by *C. rubecula* to predictions of the simulation model with a likelihood ratio test. Instead of using binomial and multinomial density functions as our likelihood functions (De Boer *et al.*, 2008), the likelihood functions were directly obtained from our simulations, following a procedure presented in the Supplementary Information (see also Figures S5.1–S5.3 on pages 143-145). For each n_{loci} (1, 2 or 3), we used the log likelihood curves (Figure S5.2) to assess the value of survival s which maximized the likelihood. This resulted in the following three-parameter combinations used in subsequent statistical analyses: ($n_{\text{loci}} = 1;\ s = 0.19$), ($n_{\text{loci}} = 2;\ s = 0.69$) and ($n_{\text{loci}} = 3;\ s = 0.92$). We then calculated the likelihood ratio $\mathrm{L}\left(x|\mathbf{v}_i\right)/\mathrm{L}\left(x|\mathbf{v}_0\right)$ to assess the relative fit of the data given an alternative parameter combination $\mathbf{v}_i$ (e.g. $n_{\text{loci}} = 2;\ s = 0.69$) compared to the parameter values assumed under a null hypothesis $\mathbf{v}_0$ (e.g. $n_{\text{loci}} = 1;\ s = 0.19$) (Figure S5.3):

$$\frac{\mathrm{L}\left(x|\mathbf{v}_i\right)}{\mathrm{L}\left(x|\mathbf{v}_0\right)} = \sum_{k}^{m} \left(\ln f_k\left(x_k|\mathbf{v}_i\right) - \ln f_k\left(x_k|\mathbf{v}_0\right)\right).$$

To obtain significance values, we generated a distribution of likelihood ratios taking one of the three parameter combinations as the null hypothesis. In contrast to conventional likelihood ratio tests (LRTs), our likelihood ratio test is non-nested, since the alternative hypothesis is not a special case of the null hypothesis. Therefore, we used each of the three parameter combinations as the null hypothesis and tested it against the two remaining parameter combinations as the alternative hypotheses, following a procedure described in Lewis *et al.* (2011) to perform LRTs for non-nested model comparisons (see Supplementary Information).

Additional analyses of diploid male production and diploid male survival In subsequent analyses, we compared the results of brother-sister crosses with those of control crosses, because these crosses were performed at the same time under exactly the same conditions, while mother-son crosses were performed earlier and mothers were older because their haploid sons had to develop first. The DMP and sex ratio (proportion males) were compared with a generalized linear model with a quasibinomial error distribution and logit link function. Diploid male survival cannot be directly estimated from our data because it is difficult to measure developmental mortality of parasitoid larvae that develop inside their host since we cannot see whether the parasitoid larva is alive. It is expected that when a parasitoid larva dies, the host dies as well, but hosts may also die for reasons unrelated to parasitism. We therefore used diploid family size and the proportion of dead hosts as proxies for developmental survival and compared them between brother-sister crosses and control crosses with a GLM, using a quasipoisson error distribution and log link function for diploid family size and a quasibinomial error distribution and logit link function for the proportion of dead hosts. Our expectations were that if diploid male survival equals that of fe-

males (i.e. $s = 1$), brother-sister crosses produce equal diploid family sizes (diploid males + females) and equal proportions of dead hosts as control crosses, in which diploid males are not produced. Moreover, among inbred families, we would expect a positive relationship between the ratio of diploid males to females, $q_{\mathrm{dip},♂}/q_{♀}$, and diploid family size when diploid male survivorship is significantly lower than that of females (s (1), and a negative relationship between $q_{\mathrm{dip},♂}/q_{♀}$ and the number of dead hosts. To test this statistically, we calculated Pearson's correlation coefficient between $q_{\mathrm{dip},♂}/q_{♀}$ and (1) diploid family size, and (2) number of dead hosts for both types of inbred crosses combined (mother-son crosses and brother-sister crosses with haploid males).

Analyses of behavior In our behavioral observations, we were interested in two effects: (i) haploid males versus diploid males in brother-sister pairs and (ii) inbred versus control matings in pairs with haploid males. We therefore statistically compared the probability to mate and the time until mounting between brother-sister crosses with haploid and diploid males and between brother-sister crosses with haploid males and control crosses separately. We used a Bonferroni correction because the data of brother-sister crosses with haploid males were used in both comparisons. We used Fisher's exact test to compare the probability to mount and a Wilcoxon rank test to compare time until mounting between crosses. All statistical analyses were performed in R 2.12.0 (R Development Core Team, 2011).

5.3 Results

Diploid males in an introduced population of *Cotesia Rubecula* in Minnesota

A total of 307 *C. rubecula* cocoons were directly collected from an organic cabbage crop in Minnesota in 2005 and 2006, and 253 cocoons from *P. rapae* caterpillars collected in the same field developed in the laboratory (Table 5.1). The sex ratio of emerged *C. rubecula* adults was 46% males in 2005 and 41% males in 2006. Flow cytometric analysis showed that 23 males were diploid, and the DMP among diploid offspring was 0.13 in 2005 and 0.08 in 2006. When all data are combined, DMP was 0.11, while 15% of all males were diploid, suggesting that diploid male survivorship is relatively high and/or that the population harbors few sex alleles (see below).

To obtain an estimate of the effective number of sex loci and diploid male survival, we did a series of laboratory crosses using a native Dutch population of *C. rubecula*. Diploid male production in the laboratory ranged from zero to more than half of all diploid offspring (mean ± SE, DMP = 0.22 ± 0.07; weighted by diploid family size)

Table 5.1: Fate of *Cotesia rubecula* cocoons collected in Minnesota in 2005 and 2006.

Year	Cocoons	Females	Haploid males	Diploid males	Unknown males	Hyper-parasitoids	Not emerged
2005	424	119	76	17	9	54	149
2006	136	70*	34	6	8	8	10
Total	560	189	110	23	17	62	159

*60 females collected in 2006 were also analyzed for ploidy level: 57 females were diploid while ploidy of three females remained unknown.

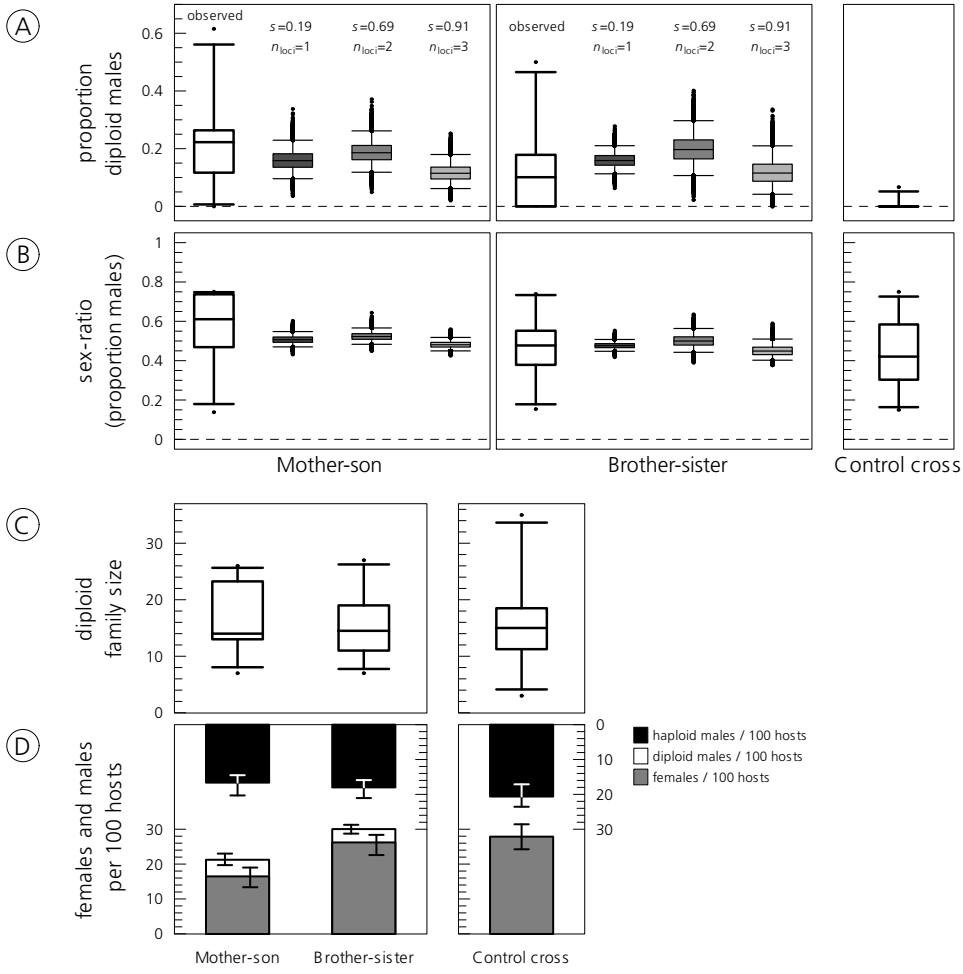

Figure 5.1: Box plots of diploid male proportion (A), sex ratio (B), diploid family size (C) and number of females, haploid and diploid males normalized per 100 hosts (D) in three types of crosses: mother-son crosses (left), brother-sister crosses (middle) and control crosses (right). Panels (A) and (B) include boxplots of 50 000 simulations for three combinations of parameter values of the number of complementary sex determination (CSD) loci (n_{loci}) and the probability of diploid male survival (s) that gave the highest likelihood for single-locus CSD, two-locus CSD and three-locus CSD (Figure S5.2). In panels (A), (B) and (C), boxes represent 0.25 and 0.75 quantiles and median value, whiskers represent 0.025 and 0.975 quantiles and outliers are shown as black bullets. In panel (D), error bars represent standard errors.

in eight mother-son crosses that resulted in the production of at least seven diploid offspring (Figure 5.1A and Table 5.2). The secondary sex ratio (proportion males among adult offspring) produced by mother-son crosses was 0.58 ± 0.08. Diploid male production (0.12 ± 0.04) and sex ratio (0.47 ± 0.05) were slightly lower in the following generation of brother-sister mating (with haploid brothers; $N = 16$) (Figure 5.1A,B). In comparison, DMP was significantly lower in control crosses (0.01 ± 0.01; $N = 10$; GLM: $\chi^2_{\text{df}=1,n=26} = 30.46$, $P = 0.001$). Sex ratio was female-biased in control crosses (0.38 ± 0.04; $N = 17$) but did not differ significantly from sex ratio in brother-sister crosses (GLM: $\chi^2_{1,33} = 6.44$, $P = 0.14$). Other parameter values for all types of crosses are presented in Table 5.2.

The LRTs with the three-parameter combinations for which maximum likelihood was obtained (from Figure S5.2) are provided in Table 5.3. A comparison between ($n_{\text{loci}} = 2$; $s = 0.69$) and ($n_{\text{loci}} = 1$; $s = 0.19$) shows that the two-locus model as null hypothesis is non-significant ($P = 0.27$), whereas it is highly significant as an alternative model ($P < 0.001$) (Figure S5.3). Hence, according to the procedure described in Lewis *et al.* (2011), this makes ($n_{\text{loci}} = 2$; $s = 0.69$) a more plausible model to describe the data than ($n_{\text{loci}} = 1$; $s = 0.19$). A similar pattern is found when comparing the other multilocus model ($n_{\text{loci}} = 3$; $s = 0.92$) with the single-locus model ($n_{\text{loci}} = 1$; $s = 0.19$). Finally, when both multilocus models are compared, we find that ($n_{\text{loci}} = 2$; $s = 0.69$) as alternative hypothesis is highly significant ($P = 0.0042$), whereas the reverse model with ($n_{\text{loci}} = 3$; $s = 0.92$) as alternative hypothesis is not ($P = 0.34$). Again, the parameter combination ($n_{\text{loci}} = 2$; $s = 0.69$) provides a more plausible explanation of the data than ($n_{\text{loci}} = 3$; $s = 0.92$). All in all, the parameters $n_{\text{loci}} = 2$; $s = 0.69$ give the best fit to the current dataset. This fit is also visualized in Figure S5.4 on page 146 which shows the predicted DMP per generation for the three different models and the proportions observed in our experimental crosses. There is a clear difference in fit between the single-locus model ($n_{\text{loci}} = 1$; $s = 0.19$) and the multilocus models, where the multilocus models explain the increased variation in numbers of diploid males in the brother-sister matings better than the single-locus model.

Because *C. rubecula* is an endoparasitoid, its developmental survival cannot be directly measured. We therefore used diploid family size and the proportion of dead hosts as proxies for diploid male survival. Under any form of CSD, diploid males are produced instead of females, and low diploid male survival should thus affect diploid family size. However, we found no indication of a significant difference between diploid family sizes of brother-sister and control crosses (Figure 5.1C,D; GLM, $\chi^2_{1,26} = 5.93$, $P = 0.18$). Moreover, when *C. rubecula* diploid males die during development, their *P. rapae* hosts are expected to die as well, and low diploid male survival should thus result in high proportions of dead hosts in brother-sister crosses but not in control crosses. However, the proportion of dead hosts was not statistically different between these two types of crosses (Table 5.2; GLM $\chi^2_{1,33} = 18.36$, $P = 0.12$; average weighted by the number of hosts offered was 0.31 ± 0.05 for brother-sister crosses and 0.23 ± 0.04 for control crosses). Finally, within inbred families (data of mother-son and brother-sister crosses combined), we found no indication for a correlation between the DMP and diploid family size (Pearson's correlation, $r = -0.06$, $P = 0.77$, $N = 24$; Figure S5.5A on page 147), or the number of dead hosts (Pearson's correlation, $r = 0.16$, $P = 0.45$, $N = 24$; Figure S5.5B). Since these analyses indicate that a low probability of diploid male survival is unlikely in *C. rubecula*, they indirectly support

Table 5.2: Overview of parameter values for the different types of crosses.

Type of cross	Mean ± SE Hosts exposed	Fate of exposed host			Fate of cocoons			Total numbers	
		Pieris rapae	Dead	*Cotesia* cocoon	Female	Male	Not emerged	Haploid male	Diploid male
Mother-son ($N = 8$)	81.38±6.35	11.50±2.78	33.88±6.76	34.00±5.27	13.00±2.28	18.13±4.09	2.89±1.08	112	30
Brother-sister with haploid father ($N = 16$)	54.19±2.45	7.69±1.36	16.63±2.69	29.00±1.90	13.75±1.28	12.00±1.29	3.25±0.54	159	31
Brother-sister with diploid father ($N = 14$)	55.93±3.34	6.43±1.12	16.21±2.86	32.71±3.06	0.14±0.10	30.57±3.11	2.00±0.54	20	0
Control ($N = 17$)	53.53±2.11	5.65±0.89	11.65±2.15	34.94±2.38	19.53±1.89	12.18±1.45	3.24±0.52	126*	1

* Ploidy was analyzed for male offspring of 10 out of 17 control crosses (unknown for 80 males from seven crosses).

a multilocus model because the single-locus CSD model only explains our data if diploid male survival is low ($s = 0.19$, Figure S5.2).

Reproductive behavior and success of diploid males

Almost all observations of mating behavior resulted in mounting within 10 min: out of a total of 72 observations, no mounting was observed in three brother-sister crosses with a haploid male, two brother-sister crosses with a diploid male and four control crosses. Probability of mating was thus not affected by male ploidy level in brother-sister crosses (haploid versus diploid, Fisher's exact test, $P = 1$) or by the type of cross (haploid males in control crosses versus brother-sister crosses, Fisher's exact test, $P = 0.96$). Haploid males (2.07 $\pm$ 0.27 min, $N = 30$) mounted significantly faster than diploid males (4.54 $\pm$ 0.73 min, $N = 14$) in brother-sister crosses (Wilcoxon rank test, $W = 329.5$, $P = 0.004$) but not faster than haploid males in control crosses (3.26 $\pm$ 0.64 min, $N = 19$, Wilcoxon rank test, $W = 224.5$, $P = 0.42$).

Although females readily accepted mating attempts by diploid males, only two diploid males ($N = 14$) produced daughters; they each sired a single triploid female offspring. We also analyzed 10 sons of each of these two families and they were all haploid. The remaining crosses with diploid fathers produced only males and we did not determine their ploidy level. In contrast, reproductive success (the proportion of males that produced at least one daughter) of haploid males was high in brother-sister crosses (23 out of 27) and control crosses (21 out of 21).

Table 5.3: Results of likelihood ratio tests, comparing the parameter sets that were shown to have the largest log-likelihood in Figure S5.2.

Null	Alternative	p	LR	LR distribution when null correct		
				min	mean	max
$n_{\text{loci}} = 1; s = 0.19$	$n_{\text{loci}} = 2; s = 0.69^*$	0.0000	10.71961	−17.869468	−8.791265	6.613286
$n_{\text{loci}} = 2; s = 0.69^*$	$n_{\text{loci}} = 1; s = 0.19$	0.2688	−10.71961	−37.503763	−14.3479	5.084059
$n_{\text{loci}} = 1; s = 0.19$	$n_{\text{loci}} = 3; s = 0.92^*$	0.0000	6.587093	−23.482181	−12.36556	4.810596
$n_{\text{loci}} = 3; s = 0.92^*$	$n_{\text{loci}} = 1; s = 0.19$	0.0620	−6.587093	−36.739005	−15.71077	4.428497
$n_{\text{loci}} = 3; s = 0.92$	$n_{\text{loci}} = 2; s = 0.69^*$	0.0042	4.132517	−11.856699	−3.211836	8.363512
$n_{\text{loci}} = 2; s = 0.69^*$	$n_{\text{loci}} = 3; s = 0.92$	0.3352	−4.132517	−16.160327	−5.472591	6.415336

*Models that are significantly preferred over the other model in the non-nested comparison of two models

Sex allele diversity in the field population of *Cotesia rubecula*

The DMP can be used to assess sex allele diversity making assumptions on the number of sex loci and diploid male survival (Adams *et al.*, 1977). Here, we use the estimates of n_{loci} and diploid male survival s obtained from our laboratory experiments with the native Dutch *C. rubecula* population to assess sex allele diversity in the introduced field population in Minnesota. Using the parameter combination that gave maximum likelihood in our simulations and the best fit to our data (i.e. $n_{\text{loci}} = 2, s = 0.69$, Figure S5.2, Table 5.3), 11% diploid males over the 2 years of sampling could be explained by the presence of two to four sex alleles at each locus within the Minnesota population. Single-locus CSD with low probability of diploid male survival ($s = 0.19$,

as estimated from Figure S5.2) can explain the DMP observed in Minnesota when the population harbors only two sex alleles. Yet another scenario could be that the founders of the population that we sampled in Minnesota had two-locus CSD ($n_{\text{loci}} = 2, s = 0.69$) but one of these two sex loci has become fixed (i.e. homozygous) upon introduction or establishment, and thus two-locus CSD has collapsed to single-locus CSD in the population that wë sampled from (Engelstädter *et al.*, 2011; Asplen *et al.*, 2009). If we assume such a scenario, which is effectively the same as single-locus CSD ($n_{\text{loci}} = 1, s = 0.69$), the population in Minnesota would have harbored five to nine sex alleles at the remaining polymorphic locus.

5.4 Discussion

Our study is among the first to report the presence of diploid males in a field population of parasitoid wasps, with approximately 11% of diploid offspring developing as males in *C. rubecula* in Minnesota. Diploid males are the result of homozygosity at one or multiple sex loci in most hymenopteran insects, and their production represents a severe form of inbreeding depression because diploid males are generally inviable or sterile (Heimpel & De Boer, 2008). Hymenopteran species with CSD are expected to have evolved a variety of mechanisms that reduce the sex determination load, for example, behavioral mechanisms such as pre-mating dispersal and kin recognition (Gu & Dorn, 2003; Ode *et al.*, 1995), or multiple sex loci (De Boer *et al.*, 2008) (reviewed in Van Wilgenburg *et al.*, 2006). Natural populations are also expected to harbor a large number of sex alleles that are maintained in the population through negative frequency-dependent selection (Ross *et al.*, 1993). However, when population bottlenecks occur during the process of biological control introductions or invasions, sex allele diversity may become reduced and the inbreeding depression associated with CSD may be exacerbated (Stouthamer *et al.*, 1992; Zayed *et al.*, 2007). We believe that it is therefore particularly important to investigate CSD and diploid male production in parasitoid wasps used for biological control.

Complementary sex determination is likely based on two loci in *Cotesia rubecula*

To allow estimates of sex allele diversity, assumptions on the number of CSD loci and diploid male survival must be made (Adams *et al.*, 1977), so we investigated these 'CSD characteristics' of *C. rubecula* in the laboratory using a native Dutch population. A CSD model with two loci and high probability of diploid male survival (approximately 70%) best explains our data, although it remains difficult to obtain exact estimates of diploid male survival. This is because developmental survival cannot be measured directly in endoparasitoids, and, in addition to our simulation analyses, we used diploid family size and the proportion of dead hosts as proxies instead. We found no indications for statistical differences in these parameters between inbred and control crosses, indeed suggesting relatively high diploid male survival. However, we note that statistical power for these analyses was low: since the average diploid family sizes (Figure 5.1C) and the average proportions of dead hosts were similar in these two types of crosses (31% in brother-sister versus 23% in control crosses), the resulting small effect sizes of these tests would require enormous sample sizes to achieve sufficient statistical power. Nevertheless, we frequently observed diploid males in the field as well as in our laboratory experiments, suggesting that diploid male survival is certainly not low. Only when diploid male survival is low

(approximately 20%; Figure S5.2), could our data be best explained by CSD with a single-sex locus, whereas even slightly higher survival rates give more support to multilocus CSD in *C. rubecula*.

Our results corroborate previous findings of a multilocus CSD phenotype in a sister species *Cotesia vestalis* (De Boer *et al.*, 2008). Two-locus CSD may in principle evolve from *sl*-CSD by duplication of the CSD locus and reduces the production of diploid males significantly because homozygosity at both sex loci is required for diploid male development (Crozier, 1971). Duplications of sex determination genes are known from other hymenopterans with the CSD phenotype. In honeybees, the *csd*-gene arose from a duplication of the *feminizer* gene (*fem*) (Hasselmann *et al.*, 2008). While heterozygous *csd* is required to initiate female development, *fem* activity maintains the female pathway throughout development (Gempe *et al.*, 2009). *Fem* is structurally as well as functionally similar to transformer in other insect species (Gempe & Beye, 2011; Verhulst *et al.*, 2010b). Interestingly, while *fem* also occurs in lineages related to honeybees, such as bumblebees and stingless bees, *csd* occurs only in Apis, suggesting a recent duplication in this clade despite the presence of CSD phenotype in related lineages (Hasselmann *et al.*, 2008). The genome of the fire ant *Solenopsis invicta*, another species with *sl*-CSD (Ross & Fletcher, 1986), also contains two linked sequences with similarity to transformer/feminizer genes from honeybees and other insects, but their functions have not been fully characterized yet (Würm *et al.*, 2011). Phylogenetic analysis of transformer-like gene sequences in honeybees and ants confirmed that duplication events occurred independently in these lineages. Gempe & Beye (2011) suggest that small-scale changes in regulatory and coding regions of existing or duplicated genes may lead to the observed variety of sex determination mechanisms across insects. Importantly, to explain an *ml*-CSD phenotype, i.e., lower proportions of diploid males as we found in *C. rubecula*, gene duplicates must segregate independently instead of being linked as found for honeybees and *S. invicta*. In addition, alleles from separate loci must not interact in ways that would produce haploid females. Mapping and molecular genetic studies in *Cotesia* are required to gain insight into the number of loci and genes involved in sex determination in these parasitoids, and we are currently taking these steps in *C. vestalis*.

Sex allele diversity in field populations of parasitoid Hymenoptera

While our laboratory studies suggest that CSD in *C. rubecula* is based on two loci – a scenario that should reduce diploid male production — 8-13% of diploid adults were male in a field population in Minnesota. Diploid males have been detected in field populations of parasitoids only two times before as far as we are aware. Natural populations of *Bracon* (=*Habrobracon*) *hebetor* and *Diadromus pulchellus* were estimated to harbor more than 10 different sex alleles at a single-sex locus (Heimpel *et al.*, 1999; Antolin *et al.*, 2003; Periquet *et al.*, 1993) although *Bracon hebetor* allelic diversity was estimated to be lower in laboratory crosses between wasps from different locations (Heimpel *et al.*, 1999). Although directly extrapolating our laboratory data to an introduced population with a different origin remains speculative, our laboratory estimates ($n_{\text{loci}} = 2$ and $s = 0.69$) suggest that the sampled population of *C. rubecula* in Minnesota harbors two to four sex alleles at each of the two independent sex loci (Adams *et al.*, 1977).

Alternatively, one of the sex loci might have become fixed in allele composition,

and *ml*-CSD may have collapsed to *sl*-CSD in the population that we sampled from (Engelstädter *et al.*, 2011; Asplen *et al.*, 2009). While frequency-dependent selection should impede fixation of a single-sex locus through the advantage of rare sex alleles on lowering the production of diploid males, the strength of frequency-dependent selection on a given locus may be much weaker under *ml*-CSD. High allelic diversity at other sex loci may reduce the production of diploid males sufficiently to offset the advantage of rare sex alleles at a locus with low allelic diversity that is at risk of becoming fixed (Asplen *et al.*, 2009). (Engelstädter *et al.*, 2011) predicted that *ml*-CSD may degrade to effective *sl*-CSD in < 100 generations during the spread of parthenogenesis through a population of parasitoid wasps due to the loss of genetic variation at all but one of the sex loci. We suggest that such a sex determination meltdown may not be unlikely in an introduced population that has been founded by few individuals or has experienced population bottlenecks during establishment. *Cotesia rubecula* is native to Eurasia and has been introduced (both intentionally and accidentally) multiple times in North America (Biever, 1992; Van Driesche, 2008). In Minnesota, *C. rubecula* was recorded in cabbage fields from the year 2000 onward (Wold-Burkness *et al.*, 2005), perhaps established from small numbers released locally in 1992: 12 and 59 adults from China and Yugoslavia, respectively. The population of wasps that we sampled likely originated from a small founding population, and this may explain the low allelic diversity at the sex loci or the collapse from two-locus CSD to effective *sl*-CSD. We expect diploid male production to be lower in the native range of *C. rubecula*. Although such information is not yet available, proportions of diploid males were much lower in a native Taiwanese population of *C. vestalis* (J. G. de Boer, unpublished data). A comparison of diploid male production in the fire ant *S. invicta* in its native and introduced range also demonstrated a significantly lower diversity of sex alleles in the introduced range (Ross *et al.*, 1993).

Reproductive behavior and success of diploid males

Our finding that 15% of *C. rubecula* males were diploid in the field underlines the significance of investigating behavior and fertility of diploid males (Heimpel & De Boer, 2008). We observed no effect of male ploidy on courtship behavior, and females readily accepted a diploid male as a mate although time until mounting was significantly longer for diploid males. Yet, mating with a diploid male was costly to a female because their reproductive success is very low: only two out of 14 females mated to a diploid male produced one daughter each while the other females produced only sons. Moreover, daughters produced by diploid males were triploid and we expect them to be sterile (De Boer *et al.*, 2007b). In contrast, females mated to haploid males had high reproductive success and produced many daughters. In our laboratory setup, females were confined with a single male (haploid or diploid) and thus could not choose their mate. A next important step will be to test whether females can discriminate between haploid and diploid males, and what the competitive abilities of diploid males are under field conditions, especially considering they took longer to mount a female than haploid males in our laboratory test. Competition among males of *C. rubecula* can be intense and males may 'steal' females from other males without courting or may display female mimicry to distract rivals (Field & Keller, 1993a). Although *C. rubecula* females normally mate once, remating does occur (Field & Keller, 1993a), and it will be interesting to investigate whether females

are more likely to remate when their first mate is diploid.

Conclusions and implications for biological control

In conclusion, we demonstrated CSD in *C. rubecula*, and our laboratory data suggest that it is based on two loci. While CSD with surviving and effectively sterile diploid males, as we found in *C. rubecula*, is expected to be most disadvantageous to fitness and population growth (Stouthamer *et al.*, 1992; Zayed & Packer, 2005; Heimpel & De Boer, 2008), the presence of two loci should lessen population-level consequences in native populations. However, our observation of 8–13% diploid males in a field population of *C. rubecula* that was introduced for biological control purposes suggests that allelic diversity at the sex loci may be reduced or that two-locus CSD may degrade to effective *sl*-CSD locally. Despite these considerations, *C. rubecula* appears to establish readily when introduced and is capable of impressive levels of pest control (Cameron & Walker, 2002; Van Driesche, 2008). However, levels of parasitism would presumably be higher if diploid males were not produced. The local loss of sex allele diversity may be the result of a genetic bottleneck that occurred during biological control introduction. Unfortunately, biological control introductions are rarely accompanied by population genetic studies. A population genetic comparison of native and introduced populations of the parasitoid *Aphidius ervi* showed that a mild bottleneck indeed occurred despite the release of more than 1000 parasitoid wasps (Hufbauer *et al.*, 2004). Yet, whether reduced genetic variation is associated with low fitness and poor performance of biological control agents remains to be established (Hufbauer & Roderick, 2005). We suggest that biological control introductions of parasitoids with CSD represent excellent study systems to investigate the relationship between neutral and non-neutral genetic variation and biological control efficacy.

Acknowledgements We thank Christine Kulhanek and Laura Stone for their help in collecting and rearing of field material in Minnesota, André Gidding of the Laboratory of Entomology, Wageningen University, for maintaining colonies of *C. rubecula* and *P. rapae*, and Unifarm for supplying cabbage plants. We also thank Greg Veltri of the Flow Cytometry Core Lab at the University of Minnesota Cancer Center and Willem van de Poll of Marine Biology of the University of Groningen for their help in flow cytometry analyses, and we thank Tim Fawcett for comments on the simulation model and statistical analyses. JGdB acknowledges financial support from the Netherlands Organisation for Scientific Research (ALW grant nr. 863.07.010).

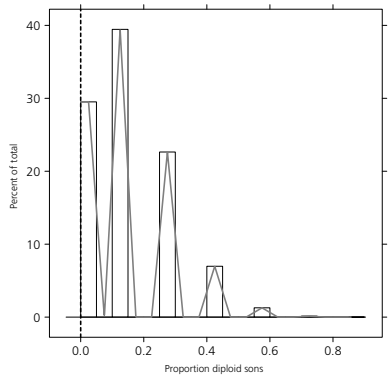

Figure S5.1: A histogram of the 50,000 simulated values for the proportion of diploid males for a single female with id=M-7 using parameters $n_{\text{loci}} = 1; s = 0.19$. The grey line depicts the smoothened density function $f_{\text{M-7}}$ obtained by `approxfun()`. The actual data point of the proportion of diploid males for female M-7 is 0 out of 7 diploid offspring (dashed vertical line). The likelihood for the current parameters is $f_{\text{M-7}}(0|1, 0.19) = 0.295$.

S5 Supplement

Simulation model

The simulation was initiated by generating eight diploid virgin females with n_{loci} unlinked CSD loci that are all heterozygous. Each virgin female produced a single haploid genome through meiosis to obtain a son for the mother-son mating. Subsequently, diploid offspring were produced by combining the son's genome and one of both maternal genome copies (randomly sampled for each offspring). As in the experiment, all diploid offspring produced by a mother were sired by the same son. Diploid males are produced if both genome copies in a newly produced diploid offspring are identical. Diploid male survival, s, was implemented by comparing a random number, drawn from a uniform distribution, against s. We continued to generate adult diploid offspring from a single replicate until we matched the number of diploid offspring that was produced for a particular replicate in the actual experiment. Hence, while diploid family size was equal to the observed values, the number of surviving diploid males varied according to n_{loci} and s.

For each mother in the mother-son generation, we then generated the same number of brother-sister matings as in the experiment, unless a mother had produced only diploid sons, which is a realistic consequence of the stochasticity resulting from CSD-allele segregation with a limited number of CSD loci. In that case, no brother-sister matings were performed for that particular mother. This happened only rarely in our simulations: in the most likely case of having 100% male broods ($n_{\text{loci}} = 1$, $s = 1$), this occurred in 386 simulations out of 50,000 (0.72% of all replicates). Brother-sister matings were generated by randomly sampling a daughter from the mother's female offspring, and by generating a haploid son from that same mother. Again, a mated daughter produced the same number of adult diploid offspring as in the actual experiment.

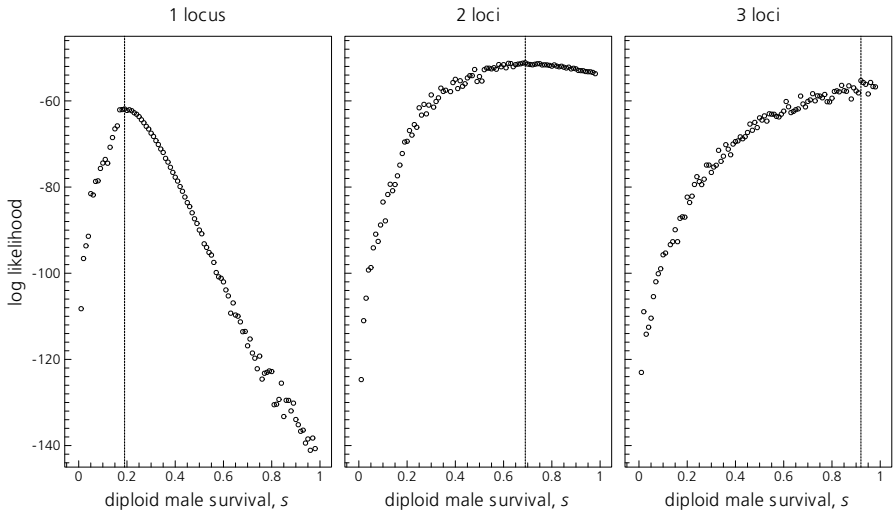

Figure S5.2: Total likelihood value for different values of diploid male survival s and the number of CSD loci, n_{loci}. Likelihood is maximized for $s = 0.69$ and $n_{\text{loci}} = 2$.

Likelihood functions

We denoted the proportion of diploid males x produced by a particular mother k by x_k. We ran 50,000 replicate simulations of the inbreeding experiment, resulting in 50,000 simulated deviates of each data point, $\hat{x}_k$, for each set of model parameters $\mathbf{v} = \{n_{\text{loci}}, s\}$. From a histogram of these simulated deviates $\hat{x}_k$, we obtained a simulated density function $f_k(\hat{x}_k|\mathbf{v})$ that informs us of the probability of the actual data point x_k given the current parameters. For each data point x_k, the density function f_k was obtained from the frequency histogram of the simulated deviates, which was smoothened using R's `approxfun()` method (R version 2.12.1, R Development Core Team, 2011). Figure S5.1 shows an example of the density function f_k. The function is discrete since a female's fecundity value can only consist of integers, but nonetheless provides us with a likelihood value that reflects the simulated outcome. Hence, the likelihood function for an individual data point x_k is $f_k(x_k|\mathbf{v})$, and the total likelihood for the vector x of all data points resulting from the experiment is $\mathrm{L}(\mathbf{x}|\mathbf{v}) = \prod_k^m f_k(x_k|\mathbf{v}_i)$. The overall likelihood (taking logs and summing) is shown in Figure S5.2, the values of $\mathrm{L}(\mathbf{x}|\mathbf{v})$ are shown for varying s and $n_{\text{loci}} = \{1; 2; 3\}$.

Comparisons between different models were carried out with likelihood-ratio tests (LRTs). LRTs re conventionally used to compare nested models (i.e., situations where one of the models is a special version of the other, having additional parameters), with the null hypothesis that the data are drawn from the simpler of the two models. However, LRTs can also be applied to models that are non-nested (i.e., where one model does not have additional parameters compared to the other), as is the case in our study. To do this, we used the following approach (for details see Lewis *et al.*, 2011): First, when comparing two non-nested models (say, model A and model B), one cannot simply assign one of both models as a null model (unless prior information is available). Instead, two reciprocal model comparisons are necessary, so that

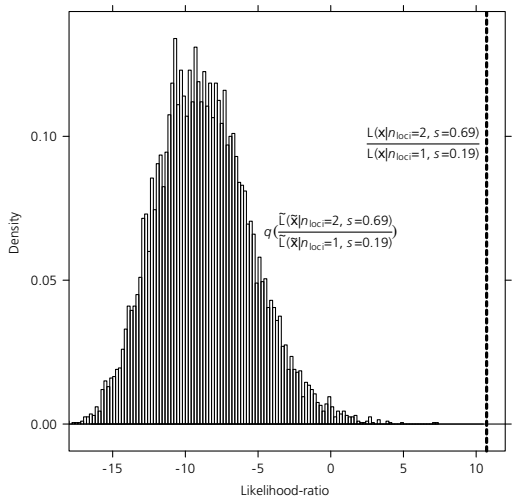

Figure S5.3: The histogram depicts the distribution q of 5,000 likelihood ratio test values for simulated data $\tilde{\mathbf{x}}$ when the null hypothesis $n_{\text{loci}} = 1, s = 0.19$ holds true. This is compared to the actual likelihood ratio for the experimentally observed dataset $\mathbf{x}$, which amounts to a value beyond the distribution q. As such, the likelihood ratio test in Table 5.3 in the main text on page 138 results in a significance value $P = 0.000$.

both models A and B are considered as a null model. The observed value of the likelihood ratio test statistic $\mathrm{L}(\mathbf{x}|\mathbf{v}_1)/\mathrm{L}(\mathbf{x}|\mathbf{v}_0)$ (see main text) falls into one of the following categories (Lewis *et al.*, 2011):

1. An LRT with A as the null model is non-significant, but an LRT with model B is significant. Model A is therefore preferred over model B.

2. An LRT with B as the null model is non-significant, but an LRT with model A is significant. Model B is therefore preferred over model A.

3. Both LRTs (A as a null model, B as a null model) are significant: neither model can be considered appropriate.

4. Neither of the LRTs (A as a null model, B as a null model) are significant: no discrimination between the models is possible.

In case of a comparison between non-nested models, significance of the likelihood ratio test statistic cannot be calculated from the chi-squared distribution. Instead, we generated the appropriate test distribution from the simulations of the experiment, assuming that the null hypothesis is true. To generate the test distribution for a null hypothesis (which assumes the particular parameter values $\mathbf{v}_0$), a set of 5,000 replicates was randomly sampled from the full set of 50,000 replicate simulations for the parameter combination $\mathbf{v}_0$. Every single data point, $\tilde{x}_k$ within each of these sampled replicates is now used as a data point to calculate a likelihood ratio using the density function mentioned above, i.e.,

$$\tilde{L}(\tilde{x}_k|\mathbf{v}_1)/\tilde{L}(\tilde{x}_k|\mathbf{v}_0) = \sum_k^m \left(\ln f_k\left(\tilde{x}_k|\mathbf{v}_i\right) - \ln f_k\left(\tilde{x}_k|\mathbf{v}_0\right)\right).$$

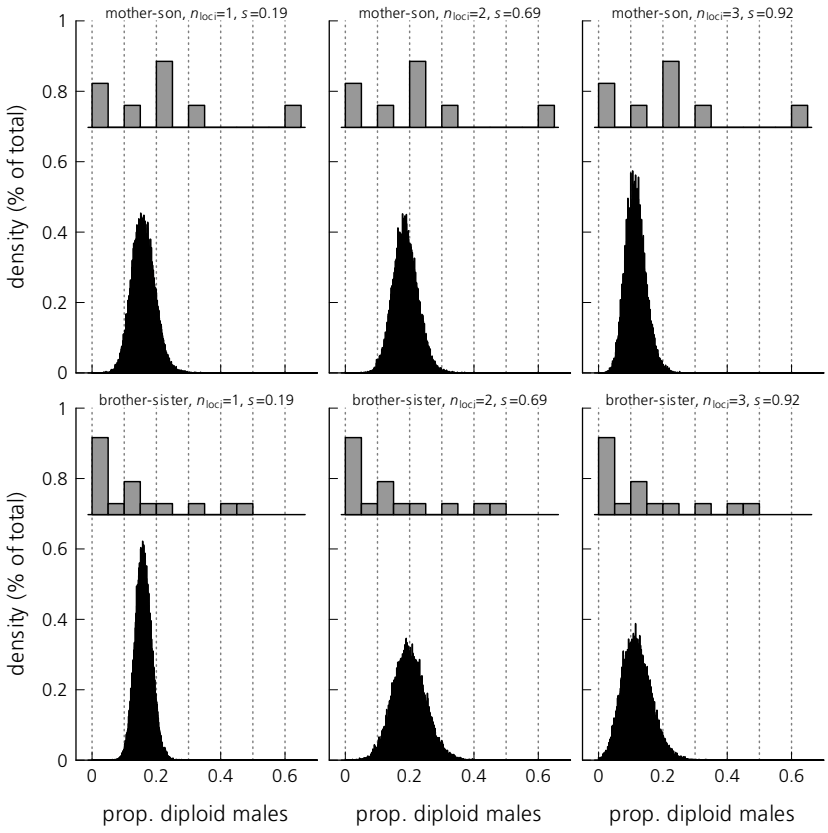

Figure S5.4: Predicted (black histogram) and observed (grey histogram) proportions of diploid males per generation for each of the three models that had the highest likelihoods in Figure S5.2. Distributions with multiple loci are predicted to be wider in generation two, which matches the data. In contrast, the single locus distribution becomes narrower, as the proportion of diploid males is always 0.5 for each individual mother, whereas the proportion of diploid males is more variable in a multilocus model due to more possibilities of segregation of different CSD alleles in different mothers during the BS generation. MS: mother-son (generation 1), BS: brother-sister (generation 2).

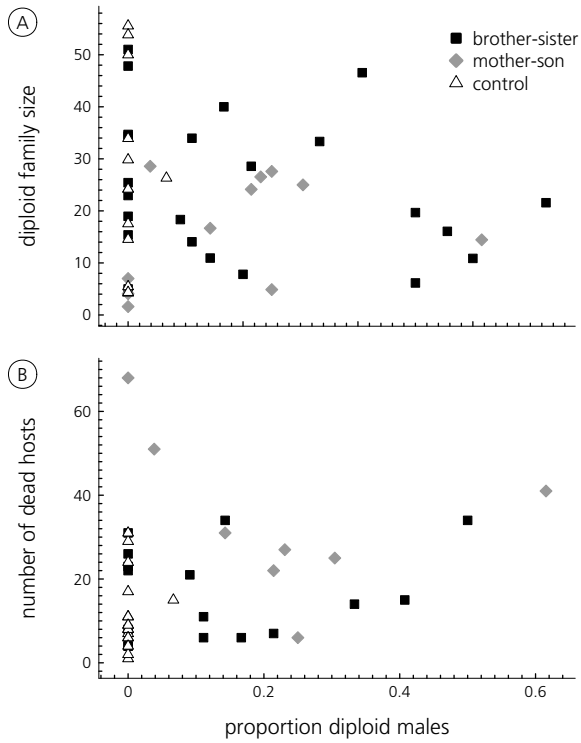

Figure S5.5: Scatter plots showing the absence of a relationship between the proportion of diploid males and (A) diploid family size, and (B) the number of dead hosts.

This step was repeated for all 5,000 sampled simulations, resulting in a distribution of 5,000 likelihood ratio test values that were then used for null hypothesis testing, summarized in Table 5.3. An example of a distribution $q\left(\tilde{L}(\tilde{x}_k|\mathbf{v}_1)/\tilde{L}\left(\tilde{x}_k|\mathbf{v}_0\right)\right)$ of likelihood ratio test values, in comparison to the actual likelihood ratio is given in Figure S5.3. Although significance values are not corrected for multiple comparisons, a Bonferroni correction by multiplying significance values by $1/n = 1/6$ does not alter our conclusions.

CHAPTER 6

Parent-offspring conflict over condition-dependent sex allocation

Bram Kuijper & Ido Pen

Abstract

Recent models suggest that evolutionary conflicts of interest can drive rapid turnovers between sex determining mechanisms, but these models apply only to cases in which sex determination is invariant with respect to environment or condition. Here, we model parent-offspring conflict in the presence of condition-dependent sex allocation. Offspring are assumed to develop in one of two different environments with sex-specific fitness consequences. In addition, one sex is assumed to be more costly to produce than the other, so that offspring generally favor a sex allocation strategy that is more biased towards the rarer, more costly sex than favored by their parents. The extent of parent-offspring conflict is, however, dependent on the relative frequency of both environments: when one environments is not extremely common relative to the other, parent-offspring conflict can be reduced or even entirely absent, despite a biased population sex ratio. The model explores when parent- offspring conflict makes populations prone to invasions of condition-independent sex factors (e.g., sex chromosomes), which result in loss of condition-dependent sex allocation, or lead to mixtures of condition-dependent and condition-independent sex factors, corresponding to observations in which sex chromosomes are present in organisms with environment-dependent sex determination. Alternatively, parent-offspring conflict may favor traits that lead to errors in assessing the environment, which can result in loss of condition-dependent sex allocation without genetic changes in the sex determining cascade.

6.1 Introduction

Condition-dependent sex allocation –where investment in one sex versus the other is dependent on the environment– provides an adaptation to environments that have different fitness consequences for males and females (Trivers & Willard, 1973; Charnov & Bull, 1977). Indeed, studies on a variety of taxa have shown that environments that are more beneficial to males than females lead to the overproduction of sons, whereas the reverse condition leads to the overproduction of daughters (see West, 2009, and references therein). Prominent examples are the sensitivity of sex-specific fitness to developmental temperature in lizards, by means of temperature dependent sex determination (Warner & Shine, 2008; Pen *et al.*, 2010) or facultative sex ratios based on host size in parasitoid wasps (Charnov *et al.*, 1981) and maternal condition in polygynous ungulates (Clutton-Brock & Iason, 1986; Hewison & Gaillard, 1999). However, results are not always that straightforward, with facultative sex ratios being strikingly absent in other taxa, despite clear indications that male and female fitness differentially depend on the environment (e.g., Hewison & Gaillard, 1999; Rutstein *et al.*, 2005; Uller & Olsson, 2006). Hence, the factors that underlie the evolutionary maintenance of condition-dependent sex allocation are still poorly understood (West, 2009).

A simple mechanism to achieve condition-dependent sex allocation is when the sex determining mechanism depends directly on the environmental variable of interest. Such environment-dependent sex determining mechanisms (ESD) have been observed in shrimp (Adams *et al.*, 1987), nematodes (Blackmore & Charnov, 1989), many reptiles (Valenzuela & Lance, 2004) and some fish (Conover & Heins, 1987). An interesting observation arising from phylogenetic studies is that transitions between ESD and genetic sex determining systems (GSD) are relatively rapid (but see Pokorná & Kratochvíl, 2009), in which closely related species (Mank *et al.*, 2006; Janzen & Phillips, 2006; Sarre *et al.*, 2011; Quinn *et al.*, 2011) and sometimes even different local populations of the same species (Pen *et al.*, 2010) have diverged in their mode of sex determination.

Most models about the evolutionary transitions between condition-independent and condition-dependent sex determining mechanisms have focused on the role of environmental variation. Bull (1981a) confirmed insights from Charnov and Bull's (1977) seminal model, by showing that transitions from male heterogamety (XX-XY) to ESD are possible when sex-specific fitness varies with a predictable environmental variable, although the loss of heteromorphic sex chromosomes may constrain the evolution of ESD (Bull, 1983; Bull & Charnov, 1985). Bulmer & Bull (1982) showed that ESD may be replaced by GSD in fluctuating environments, in which case ESD can give rise to extreme fluctuations of the population sex ratio. However, less 'precise' forms of ESD that have a certain amount of developmental noise may be more robust against such fluctuations (Van Dooren & Leimar, 2003). More recently, models have focused on the evolution of temperature dependent sex determination (TSD) in particular, where abiotic factors such as climatic variation may drive transitions between GSD and ESD (Pen *et al.*, 2010; Grossen *et al.*, 2011). Apart from environmental variation, however, less attention has been given to other mechanisms that could induce evolutionary transitions between GSD and ESD (but see Feldmeyer, 2009).

Genetic conflict, where different genes have different selective optima, has often been associated with rapid evolutionary transitions between genetic sex determining

mechanisms (reviewed in Werren & Beukeboom, 1998; Pen, 2006; Uller *et al.*, 2007; Cordaux *et al.*, 2011). For example, intralocus sexual conflict (Van Doorn & Kirkpatrick, 2007), conflicts between hosts and endosymbionts (Rigaud & Juchault, 1993; Cordaux *et al.*, 2011) and conflicts between parents and offspring (Werren & Hatcher, 2000; Werren *et al.*, 2002; Kozielska, 2008) have all been associated with transitions in genetic means of sex determination. Little work has been done, however, on the role of genetic conflicts in the context of condition-dependent sex allocation. Since genetic conflicts have been widely known to lead to maladaptive evolutionary outcomes (Burt & Trivers, 2006) and can lead to rapid evolutionary change, they may be part of the explanation why condition-dependent sex allocation is absent, even when selective conditions for it are being met and why switches occur so rapidly between ESD and GSD.

Here we therefore focus on a model of parent-offspring conflict (or more specifically, mother-offspring conflict) over condition-dependent sex allocation. Evolutionary interests between mothers and offspring over sex allocation can diverge, since mothers are equally related to all their offspring and therefore maximize their fitness by producing a sex ratio that maximizes the total reproductive value of her current and future broods. In contrast, individual offspring are more related to themselves than to their siblings, and may therefore prefer to develop as the rarer sex with a higher reproductive value, at the expense of their siblings (Trivers, 1974). Such conflicts between parents and offspring over sex allocation have already been associated with transitions in female and male heterogamety in the context of GSD (Werren *et al.*, 2002; Pen, 2006; Kozielska, 2008), but whether parent-offspring conflict can also play a role in transitions between condition-dependent and condition-independent sex determining mechanisms is currently unclear.

To model the interaction between parent-offspring conflict and condition-dependent sex allocation, we extend the classical Charnov-Bull model (e.g., Charnov & Bull, 1977; Schwanz *et al.*, 2006), which assumes that mothers encounter one of two environmental conditions, one of which reduces the fitness (here juvenile survival) of one of both sexes. For example, mothers could breed in different environments, one of which benefits male development more than female development. In case of condition-independent sex determination, individuals would then often develop as females in the 'wrong' environment, whereas condition-dependent sex determination limits the production of that sex in the environment to which it is maladapted (e.g., Trivers & Willard, 1973; Charnov & Bull, 1977; Bull, 1981b; Bull & Bulmer, 1989). Here, we analyze equilibrium sex allocation strategies when condition-dependent sex allocation is either controlled by the mother or by the offspring. Using an analytical reproductive value approach (Leimar, 1996; Taylor, 1996a) in combination with individual-based simulations, we then assess the conditions in which maternal and offspring sex allocation strategies diverge (i.e., the conflict 'battleground', Godfray, 1995).

Subsequently, we study a number of evolutionary resolutions to the conflict. Models in which sex determination is independent of condition have shown that parent-offspring conflict can pave the way for the invasion of genetic sex modifiers, that allow parents to achieve brood sex ratios closer to their optimal sex allocation, or allows offspring to develop more often as the sex with the higher reproductive value (Werren *et al.*, 2002; Pen, 2006; Kozielska, 2008). Here, we investigate if condition-independent genetic masculinizers or feminizers are able to invade in populations

with condition-dependent sex allocation. In addition, we assess if mothers and zygotes can also evolve modifiers that change the reliability of sensing environmental cues, on the basis of which sex is determined, to achieve patterns of sex allocation that are closer to their respective optima.

The results of our study provide a number of hypotheses on the role of parent-offspring conflict in driving transitions from condition-dependent to condition-independent sex determination which could be tested from empirical data: for example, our study shows that turnovers in sex determining mechanisms only occur when some environments are much more common than others, whereas for more 'even' environments in which different conditions occur at similar frequencies, condition-dependent sex allocation is remarkably robust to parent-offspring conflict, which may explain the maintenance of condition-dependent sex allocation in some groups, but not in others.

6.2 The model

In the model below, we refer to parent-offspring conflict as a conflict between a mother and her offspring. We consider a panmictic, monogamous sexual population with discrete generations. Due to environmental variation (say, nutrition), a mother will breed in one of two possible environments that differ in their fitness consequences to each sex. With constant probability p, the mother breeds in the so-called poor environment (environment 1), which is detrimental to newborn daughters (which have survival v) relative to sons. With probability $1-p$, the mother breeds in a good environment (environment 2), in which juvenile survival is not sex-specific.

Additionally, we assume that the production of a son requires$c$ units of maternal resources, per unit invested in daughters, where all mothers have accumulated an identical amount of resources. Sex ratios in the poor and good environments respectively are given by the strategy (s_1, s_2). (s_1, s_2) can either be under maternal (subscript 'm') or offspring (subscript 'o') control. To summarize, the life cycle is as follows: (i) birth and sex determination of an offspring in natal environment i, (ii) environment and sex-specific juvenile survival to adulthood, (iii) random mating and (iv) random settlement of mothers in one of two environments followed by the production of offspring.

We implement a model for the evolution of condition-sex determination using a reproductive value approach where we assume that the population consists of three classes of individuals. First, there are n_{f1} females that breed in environment 1, n_{f2} females that breed in environment two and n_{m} males. We analyze a population dynamical model $[n_{\mathrm{f1}}, n_{\mathrm{f2}}, n_{\mathrm{m}}]^{\mathrm{T}}_{t+1} = \mathbf{A}[n_{\mathrm{f1}}, n_{\mathrm{f2}}, n_{\mathrm{m}}]^{\mathrm{T}}_{t}$ (T denoting transposition), where $\mathbf{A}$ is a matrix that governs transitions between the three different classes:

$$\mathbf{A} = \frac{1}{2}\begin{bmatrix} pvf_1(s_1) & pf_2(s_2) & p\left[\frac{y_{\mathrm{f1}}}{y_{\mathrm{m}}}vf_1(s_1) + \frac{y_{\mathrm{f2}}}{y_{\mathrm{m}}}f_2(s_2)\right] \\ (1-p)vf_1(s_1) & (1-p)f_2(s_2) & (1-p)\left[\frac{y_{\mathrm{f1}}}{y_{\mathrm{m}}}vf_1(s_1) + \frac{y_{\mathrm{f2}}}{y_{\mathrm{m}}}f_2(s_2)\right] \\ m_1(s_1) & m_2(s_2) & \frac{y_{\mathrm{f1}}}{y_{\mathrm{m}}}m_1(s_1) + \frac{y_{\mathrm{f2}}}{y_{\mathrm{m}}}m_2(s_2) \end{bmatrix}. \quad (6.1)$$

Note that the transition matrix $\mathbf{A}$ is multiplied by $\frac{1}{2}$, reflecting the genetic share of each parent in its offspring. $f_i(s_i)$ and $m_i(s_i)$ represent the number of females and males produced by a mother that breeds in environment i, using resident strategy

(s_1, s_2). For the moment, we assume that mothers and offspring are able to correctly perceive the maternal condition or environment, but we relax this assumption later (see Section S6.3 in the Supplementary Information). We assume that a son is c times more costly to produce than a daughter, so that the average cost of producing a brood in environment i is proportional to $s_i c + 1 - s_i$. Since the total number of offspring is inversely proportional to the average cost of the brood, we obtain the following expressions for the number of $f_i(s_i)$ daughters and $m_i(s_i)$ sons that are produced in environment i:

$$f_i(s_i) = \frac{1 - s_i}{s_i c + 1 - s_i},$$
$$m_i(s_i) = \frac{s_i}{s_i c + 1 - s_i}.$$

We briefly explain the different entries of the transition matrix $\mathbf{A}$ in order to clarify the setup of our model. The top-left entry $\mathbf{A}_{11}$ describes the contribution of females that breed in environment 1 at time t to females breeding in environment 1 at time $t+1$. Adult females in environment 1 produce a proportion of daughters given by $f_1(s_1)$. Since these females are born in environment 1, they have a reduced juvenile survival rate $\nu < 1$, relative to males born in environment 1 and all offspring born in environment 2. A surviving female subsequently has a probability p of breeding in environment 1 as an adult. The middle entry in the top row, $\mathbf{A}_{12}$, describes the contribution of females breeding in environment 2 at time t to females breeding in environment 1 at time $t+1$. Since daughters grow up in environment 2, their survival probability is equal to 1, after which they settle with probability p in environment 1. The right entry in the top row, $\mathbf{A}_{13}$, describes the contribution of males at time t to females breeding in environment 1 at time $t+1$. Males are assumed to mate randomly and subsequently settle in a random environment. As such, the mating rate of a male with a female that breeds in environment 1 is given by the stable class frequency y_{f1} of females breeding in environment 1 divided by the stable class frequency of males y_{m}. Subsequently, any daughters are produced with probability $f_1(s_1)$ and will survive in environment 1 with probability ν. Otherwise, a male may mate with a female breeding in environment 2 with rate $y_{\mathrm{f1}}/y_{\mathrm{m}}$. Here, any daughters are produced with probability $f_2(s_2)$ and they have a relative survival probability of 1. Subsequently, daughters sired by a male in environment 1 or 2 will encounter environment 1 as a breeding female with probability p. The entries in the other two rows can be derived in a similar fashion.

Given these population dynamics, we are interested in the determining optimal sex allocation strategy $(s_1^\star, s_2^\star)$. We do so by describing the population dynamics of a rare mutant, with a slightly deviating sex allocation strategy $(\hat{s}_1, \hat{s}_2)$, amidst a resident population that has sex allocation strategy (s_1, s_2). In the Appendix, we work out scenarios where either parents (A6.1.1) or offspring (A6.1.2) are in control over sex allocation. The class transitions of this rare mutant are given by the mutant transition matrix $\mathbf{B}$ (eqns. A6.1, A6.8). Based on this mutant population dynamic, selection differentials acting on a particular trait s_x are obtained using a standard result (e.g.,

Taylor, 1996a),

$$\left.\frac{\partial W}{\partial \hat{s}_x}\right|_{\hat{s}_x=s_x} = \mathbf{z}^{\mathrm{T}}\frac{\partial \mathbf{B}}{\partial \hat{s}_x}\mathbf{y}/\mathbf{z}^{\mathrm{T}}\mathbf{y}, \tag{6.2}$$

where $\mathbf{y}$ is a vector containing the stable class frequencies of the resident population (a dominant right eigenvector of matrix $\mathbf{B}$ evaluated at the resident behavior (s_1, s_2)), while $\mathbf{z}$ are the class reproductive values (equal to a dominant left eigenvector of matrix $\mathbf{B}$ evaluated at the resident behavior (s_1, s_2)). Selection differentials for s_1 and s_2 for maternal control are given in eq. (A6.5) in the Appendix. In case of offspring control over sex allocation, a mutant offspring's fitness is affected both by its own sex allocation strategy $(\hat{s}_1, \hat{s}_2)$ and the sex allocation strategy $(\tilde{s}_1, \tilde{s}_2)$ of its siblings. We assume that females mate only once, so that relatedness R of the focal mutant with any random offspring in the brood is approximately $1/2$ when broods are large. Using a direct fitness approach, (Taylor & Frank, 1996; Pen & Weissing, 2002; Taylor *et al.*, 2007) expressions for the selection differentials on offspring sex allocation traits are given by

$$\frac{\mathrm{d}W}{\mathrm{d}\hat{s}_j} = \left.\frac{\partial W}{\partial \hat{s}_j}\right|_{\hat{s}_j=\tilde{s}_j=s_j} + R\left.\frac{\partial W}{\partial \tilde{s}_j}\right|_{\hat{s}_j=\tilde{s}_j=s_j},$$

of which explicit expressions are derived in the Appendix.

We subsequently investigate if there are optimal sex allocation strategies where the selection differentials vanish. We find no equilibria where both s_1 and s_2 are mixed, so we assess the optimal sex allocation strategies by focusing on boundary conditions.

Individual-based simulations

In addition to the analytical model, we also ran stochastic individual-based simulations to corroborate analytical results and to explore some of the coevolutionary dynamics. We modeled a population of 5000 individuals, each with diploid, autosomal genetic values for s_1 and s_2. Mutation in the unlinked sex allocation loci occurs following a continuum of alleles model, where each individual allele has a mutation rate $\mu = 0.01$. If an allele mutates, its value is incremented with a deviate from a normal distribution with mean 0 and variance $\sigma_\mu^2 = 2.5 \times 10^{-5}$. If the new allelic value lies outside the range $(0, 1)$ it is set to its nearest value within that range (i.e., 0 or 1).

The life cycle mimics that of the analytical model: during each generation, females are randomly assigned to one of two environments with probability p. Subsequently, each female is assigned a mating partner that is randomly selected from the pool of males and offspring are produced. We assume that each female has a total amount of reproductive resources r that is equal to the cost of fifty sons. Sex of each offspring is determined randomly for each individual offspring, based on the sex allocation locus that corresponds to the environment perceived by the individual controlling sex allocation (mother versus offspring). As in the analytical model, an individual incorrectly perceives the environmental state to be different from the actual state with probability ε). With the production of each offspring, maternal resources are

region	I	II	III
maternal control			
boundaries	$0 < p < \frac{1}{2}$	$\frac{1}{2} < p < \frac{1}{1+v}$	$\frac{1}{1+v} < p < 1$
s_{1m}	1	1	$\frac{1-p(1-v)}{1-p(1-v)-c(1-p)+cpv}$
s_{2m}	$\frac{1-2p}{1-2p+c}$	0	0
population SR (% males)	$\frac{1}{1+c}$	$\frac{p}{c+p(1-c)}$	$\frac{1}{1+cv}$
offspring control			
boundaries	$0 < p < \frac{2c}{1+3c}$	$\frac{2c}{1+3c} < p < \frac{1+c}{1+c+2v}$	$\frac{1+c}{1+c+2v} < p < 1$
s_{1o}	1	1	$\frac{3(1-c(1-p)-p)+4vp-\sqrt{K_2}}{2(1-c)((1-c)(1-p)+2pv)}$
s_{2o}	$\frac{p(2+c(3-c))-4c+c\sqrt{K_1}}{2(1-c)(p-c(2-p))}$	0	0
population SR (% males)	$\frac{p(2+c(3-c))-4c+c\sqrt{K_1}}{2(1-c)(p-c(2-3p))}$	$\frac{p}{c+p(1-c)}$	$\frac{4p}{5p-1-c(p-1)+\sqrt{K_2}}$
derivatives	$\frac{dW}{d\hat{s}_1} > 0,\ \frac{dW}{d\hat{s}_2} = 0$	$\frac{dW}{d\hat{s}_1} > 0,\ \frac{dW}{d\hat{s}_2} < 0$	$\frac{dW}{d\hat{s}_1} = 0,\ \frac{dW}{d\hat{s}_2} < 0$

Table 6.1: Evolutionary outcomes when environmental assessment is perfect ($\varepsilon = 0$) for maternal or offspring control of condition-dependent sex allocation. Top half: maternal control over sex allocation, bottom half: offspring control over sex allocation. For reasons of space, the contents of the square roots of the sex allocation strategies under offspring control are given here: $K_1 = 16c - 8(1+3c)p + (3+c)^2p^2$, $K_2 = (1-c)^2(1-p)^2 + 8c(1-c)(1-p)pv + 16cp^2v^2$,

depleted with an amount z_i that is dependent on the sex of the offspring (son: $z = c$, daughter: $z = 1$) and offspring production is ceased when resources are equal to 0. In case the level of resources r is larger than zero, but less than the amount z that is required for the production of the next offspring, this offspring will only produced in case a deviate drawn from a uniform distribution is smaller than r/z. Subsequently, offspring survive dependent on their maternal environment and their sex, after which adults that make up the next generation are randomly selected from the surviving pool of offspring. Simulations were coded in C++ and are available on request.

6.3 Results

Divergence of maternal and offspring sex allocation optima

In Table 6.1, we compare the evolutionary outcomes when condition dependent sex allocation is controlled by the mother versus the offspring. An example comparison of optimal maternal and offspring sex ratios is given in Figure 6.1. Qualitative outcomes of our model are similar to previous analyses of the classical Charnov-Bull model (e.g., compare Figure 6.1A to Figure 1A in Wild & West (2007)). For both parents and

offspring, one of both sex ratios must always be at a boundary (0 or 1, see Appendix), leading to three qualitatively different regions (denoted by I, II and III in Figure 6.1 and Table 6.1). Without loss of generality, we discuss the case depicted in Figure 6.1, where sons are twice as costly as daughters (i.e., $c = 2$) and where environment 1 (the "poor environment") is disadvantageous to females, since they suffer a reduced survival probability v.

- In region I, the poor environment 1 is relatively rare. This selectively favors individuals to exclusively produce males in the poor environment, while individuals in the good environment overproduce females. Compared to their mothers, offspring always favor a more male-biased sex ratio, since sons are the more costly (and hence rarer) sex, having a higher reproductive value (Trivers, 1974). Hence, $s_{2o} > s_{2m}$.

- Region II: when the poor environment 1 becomes more prevalent, individuals in the poor environment eventually exclusively produce males, whereas individuals in the good environment exclusively produce daughters (a "bang-bang" sex allocation strategy: $s_1 = 1$, $s_2 = 0$). However, parents and offspring have a different range of frequencies p in which they selectively favor a bang-bang sex allocation strategy. Since sons are the more costly sex, they will remain the rarer sex unless the poor environment 1 (in which only sons are produced) becomes very common (see Figure 6.1B). As a result, offspring are still selectively favored to develop as males with a certain probability ($s_{2o} > 0$), whereas mothers are selected to produce exclusively daughters ($s_{2m} = 0$). For higher values of p, however, offspring are also selectively favored to follow a "bang-bang" strategy, similar to their mothers. Hence, for a limited range of frequencies of the poor environment p, parents and offspring agree about the sex ratios produced (grey region in Figure 6.1). Hence, condition-dependent sex allocation sometimes allows for the cessation of parent-offspring conflict, despite the presence of sex-specific production costs, $c \neq 1$.

- Region III: when the poor environment 1 becomes even more common, the exclusive production of males in that environment would lead to very strong male-biased sex ratios at the population level, so that the production of some females in the poor environment 1 becomes selectively advantageous ($0 < s_1 < 1, s_2 = 0$), despite the survival disadvantage of these females. However, offspring favor a more male-biased sex ratio in environment 1 than their mothers. That offspring favor a more male-biased sex ratio is perhaps surprising, as the population sex ratio in region III under maternal control is already at equality for the parameters considered in Figure 6.1 and could even be male-biased for different values of c or v. Hence, one would naively expect the reproductive value of any offspring that develop as males to be reduced, reducing parent-offspring conflict. The reduction in male reproductive values due to a male-biased population sex ratio is checked, however, by a larger reduction in female reproductive values, because of the predominance of the poor environment 1. Hence, the higher production costs of sons versus daughters allow parent-offspring conflict to persist despite male-biased sex ratios.

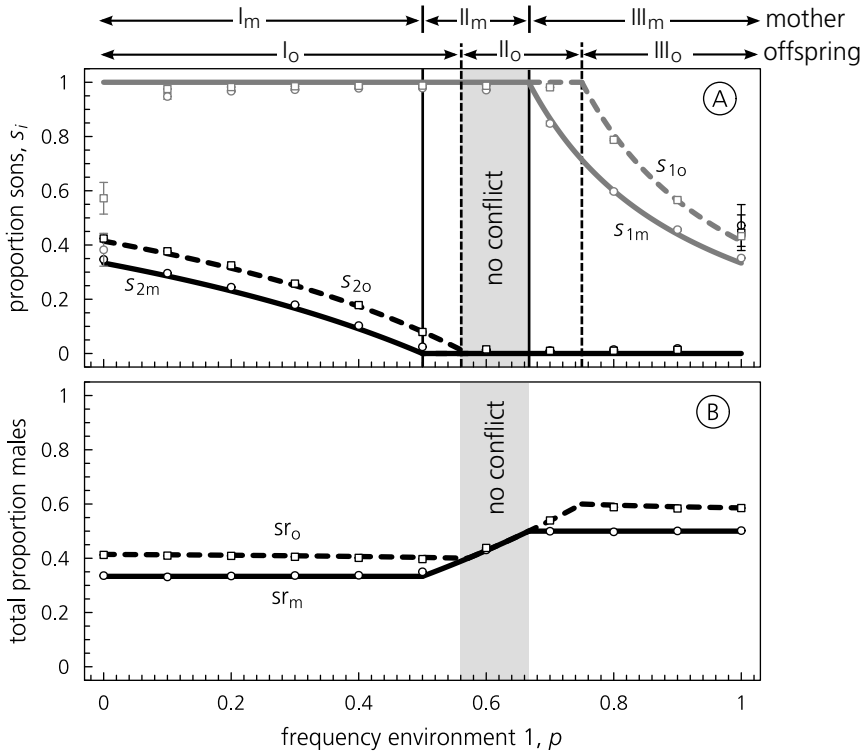

Figure 6.1: A comparison of maternal (s_{1m}, s_{2m}) (solid lines) and offspring (s_{1o}, s_{2o}) (dotted lines) sex allocation strategies when sons are twice as costly to produce than daughters (panel A) and the resulting population sex ratios (panel B). The different regions I, II, III from Table 6.1 for maternal and offspring control are depicted by the arrows on top of panel A. Parent-offspring conflict exists in the white regions, where offspring sex allocation is more biased towards the costly and hence rarer sex. Interests of parents and offspring converge in the middle grey region. The mean sex allocation strategies ($\bar{s}_1, \bar{s}_2$) averaged over 10 replicate individual based simulations after 70 000 generations are indicated by circles (maternal control) and squares (offspring control), with bars indicating standard errors (which are generally small). Parameters: $v = 0.5$, $\varepsilon = 0$.

In the absence of condition-dependent sex allocation, parent-offspring conflict over sex allocation arises as soon as there are sex-specific production costs ($c \neq 1$) (Trivers, 1974; Werren *et al.*, 2002). When sex allocation is condition-dependent, however, we find that environmental variation modulates the extent of parent-offspring conflict. Specifically, whenever the poor environment is modestly prevalent (grey regions in Figure 6.1) parent-offspring conflict is absent, despite the presence of sex-specific production costs. Figure 6.2 explores in greater detail how parent-offspring conflict over condition-dependent sex allocation is affected by different values of sex-specific production costs c (Figure 6.2A-C), as well as by different female survival probabilities v in the poor environment (Figure 6.2D-F).

Varying the sex-specific production costs c affects the direction of the sex-ratio conflict: when females are the more costly sex, offspring favor more female-biased sex ratios than their mothers (Figure 6.2A), and vice versa when males are the more costly sex (Figure 6.2C). Figure 6.2B shows that when production costs are similar for both sexes ($c = 1$), parent-offspring conflict is absent: the sex ratio of a brood is now independent of the resources distributed among siblings and is only a function

of the global environmental frequencies. Sex ratio selection due to environmental variation acts however similarly on all members of a brood, hence it does not lead to parent-offspring conflict.

Interestingly, decreasing the female survival probability v in the poor environment 1 reduces the overall likelihood of parent-offspring conflict, which is shown by the increasing grey regions (no conflict) from Figure 6.2F to 6.2D. When v is low, both parents and offspring are selected to avoid producing females in environment 1 for a broad range of frequencies p. The resulting exclusive production of males in environment 1 generates strong sex-ratio selection to exclusively produce females in environment 2, which overwhelms any selection on offspring to develop more often as the more costly sex (males). As a result, maternal and offspring sex allocation strategies coincide across both environments, leading to a cessation of parent-offspring conflict for a considerable range of p, despite the occurrence of sex-specific production costs.

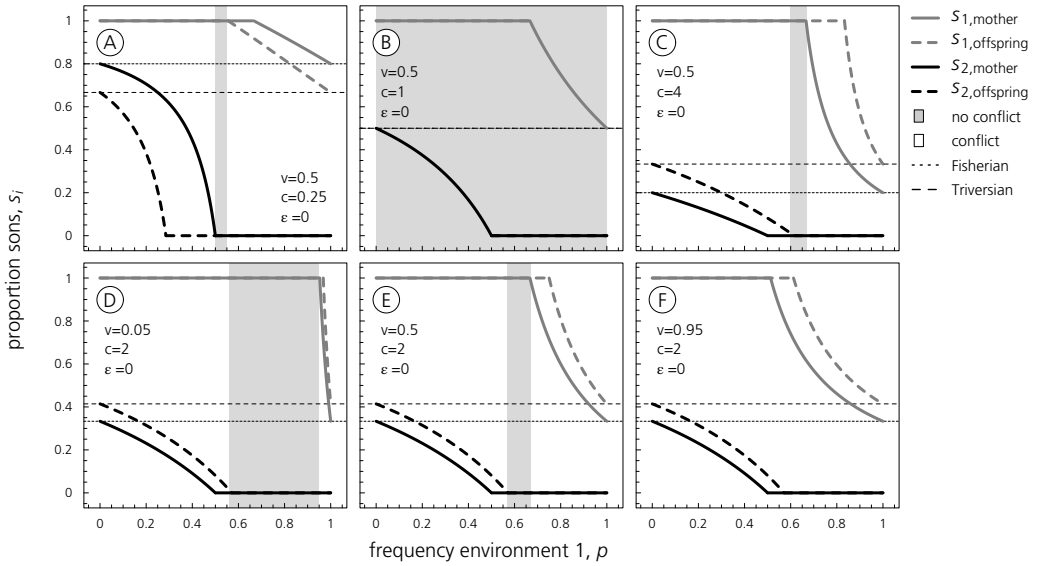

Figure 6.2: A comparison of maternal (s_{1m}, s_{2m}) (thick solid lines) and offspring (s_{1o}, s_{2o}) (thick dashed lines) sex allocation strategies when varying survival of daughters in environment 1, v (panels A-C), the cost of producing sons versus daughters, c (panels D-F) and the assessment error ε (panels G-I). White regions: parent-offspring conflict over sex allocation in one or both environments. Grey regions: no parent-offspring conflict over sex allocation. Thin dotted lines depict the Fisherian sex ratio $1/(1+c)$ where sex allocation is under maternal control and environmental variation is absent. Similarly, Triversian sex ratios $1/(1+\sqrt{c})$ are given by the thin dashed lines.

Can unconditional sex determination invade?

Following previous models which showed that parent-offspring conflict can lead to the invasion and establishment of novel genetic sex factors (Werren *et al.*, 2002; Kozielska, 2008), we now analyze the invasion of unconditional sex modifiers, such as sex chromosomes or maternal feminizers/masculinizers, in the context of condition-dependent sex allocation (see Supplement, Sections S6.1 and S6.2).

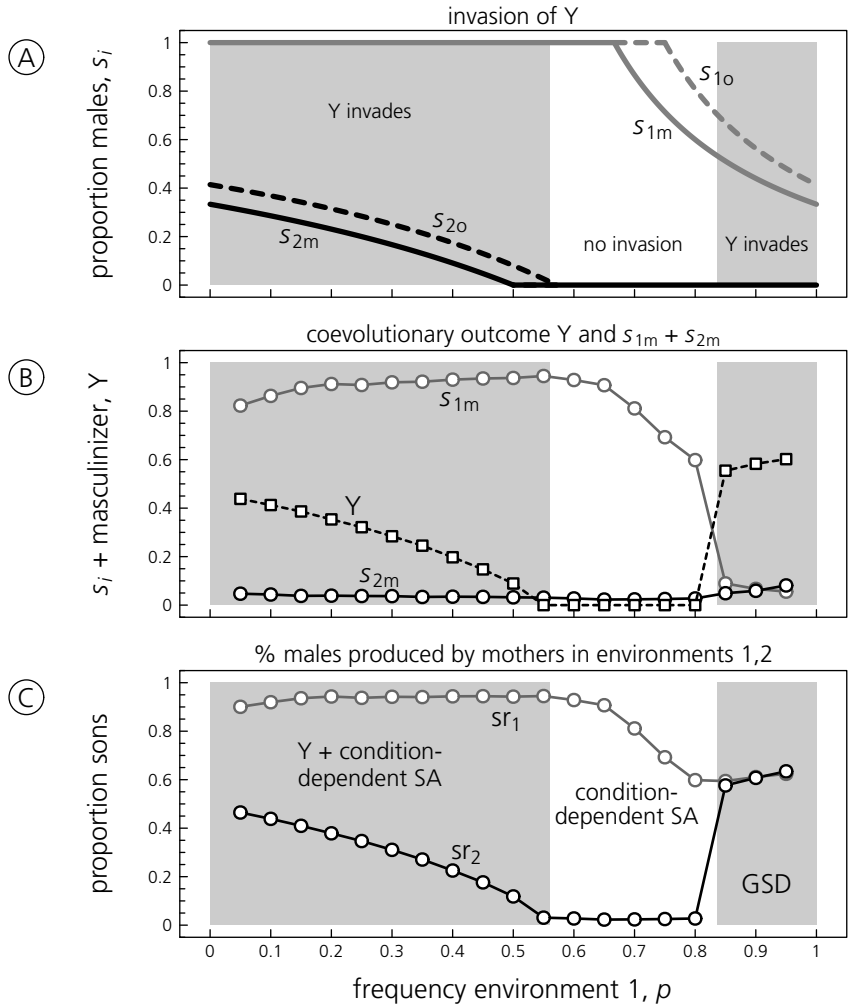

Figure 6.3: Invasion of a dominant masculinizer (Y) under offspring control, when condition-dependent sex allocation loci are maternally controlled (s_{1m}, s_{2m}) and when sons are twice as costly as daughters ($c = 2$). Panel A: the invasion of Y (grey areas, see Table S6.1) occurs when sex allocation optima diverge between parents and offspring, but under certain conditions (right part of middle white area) divergent sex ratio optima between parent and offspring do not lead to the invasion of Y. Panel B: upon successful invasion, the stable frequency of Y depends on the particular environmental configuration. As a coevolutionary response to the offspring's Y, maternal sex allocation strategies become more extreme, leading either to $(s_{1m}, s_{2m}) \approx (1, 0)$ or $(s_{1m}, s_{2m}) \approx (0, 0)$. Panel C: despite the invasion of Y, the proportion of males produced still depends strongly on the maternal environment for a large range of p, although resulting sex ratios are now closer to offspring than to parental optima. Only when the poor environment is highly prevalent (i.e., $p > 0.83$) does the invasion of Y lead to a replacement of ESD by GSD. Parameters: $v = 0.5$.

Invasion of unconditional sex factors under offspring control We focus here on the invasion of a dominant masculinizing locus (hereafter Y) under offspring control, which leads to male development regardless of the environment (i.e., GSD), thereby overriding condition-dependent sex allocation under maternal control. The analogous results for the invasion of a dominant feminizing locus W under offspring control are given in the Supplement.

Unsurprisingly, invasion of Y is typically possible when sons are more costly than daughters ($c > 1$, see Table S6.1), since this causes offspring to prefer a more male-biased sex ratio than their mothers. Additionally, the invasion of Y is strongly dependent on the frequency p of the poor environment. In particular, Figure 6.3 shows that for certain values of p, condition-dependent sex allocation under maternal control is robust to the invasion of Y, despite offspring favoring a more male-biased sex ratio than their mothers in the poor environment (i.e., see white region in Figure 6.3A where $0.66 < p < 0.83$). In this particular region, Y would facilitate offspring in the poor environment by generating the desired more male-biased sex ratio. However, it also results in the undesired production of males in the good environment, where offspring favor to develop exclusively as females, $s_{2o} = 0$. For those cases where $s_{2o} = 0$, invasion of Y will therefore only ensue when environment 2 becomes sufficiently rare (Figure 6.3A).

When Y is able to invade, coevolution between Y and maternal sex allocation loci s_{1m} and s_{2m} results in two qualitatively different outcomes: when the poor environment predominates (right side in Figure 6.3), the invasion of Y is selectively favored by offspring in the poor environment. Mothers can only counter the male-biased sex ratios resulting from the invading Y by producing more daughters in the poor environment ($s_{1m} \to 0$), which in turn selects for ever higher frequencies of Y. Coevolution between Y and s_{1m}, s_{2m} eventually leads to an equilibrium where $(s_{1m}, s_{2m}) = (0, 0)$ with high frequencies of Y, so that sex ratios are male-biased and independent of the environment (Figure 6.3C, right grey area). Hence, when the poor environment predominates, parent-offspring conflict can lead to the replacement of condition-dependent sex allocation by GSD (i.e., male heterogamety), despite the fitness disadvantage caused by the production of daughters in the predominant poor environment.

When the poor environment is less common ($p \leq 0.5$), the invasion of Y is selectively favored by offspring in the good environment, whereas both parents and offspring in the poor environment 1 favor the exclusive production of sons (and hence are not affected by the invasion of Y). Upon invasion of Y, mothers in environment 2 are selected to produce ever more daughters from those offspring that did not receive Y ($s_{2m} \to 0$). Coevolution between Y and s_{2m} eventually halts when all non-Y individuals are maternally induced as daughters ($s_{2m} = 0$) and Y reaches frequencies close to the offspring sex allocation optimum for environment 2 (in fact, sex ratios are marginally higher than s_{2o} due to the highly discrete nature of Y, see Figure S6.1A). Instead of a replacement of condition-dependent sex allocation by GSD, the invasion of Y now results in a transition from condition-dependent sex allocation under maternal control to condition-dependent sex allocation under offspring control (i.e., ESD). Interestingly, the offspring ESD phenotype is the combined result of the offspring genetic factor Y and environment-dependent sex determining factors under maternal control.

Invasion of genetic sex factors under maternal control Figure 6.4 shows the invasion of an unconditional feminizer F_m under maternal control, which leads to complete female development of a brood regardless of the environment, overriding condition-dependent sex allocation under offspring control (see Supplementary Figure S6.3 for the invasion of a maternal masculinizer M_m). Maternal production of all female broods (i.e., monogeny) has been observed in a variety of arthropod taxa (e.g., White, 1973; Ullerich, 1984; Tabadkani *et al.*, 2011).

Successful invasion of F_m typically requires that sons are more costly than daughters ($c > 1$), since this causes mothers to favor a more female-biased sex ratio than their offspring (see Table S6.2). Similar to the invasion of Y, evolutionary outcomes can be separated in three different regions: when p is low, condition-dependent sex allocation under offspring control (i.e., ESD) is replaced by condition-independent sex allocation under maternal control, in which mothers carrying F_m produce all female broods and females without F_m produce all male broods (monogeny). For intermediate frequencies of the poor environment, condition-dependent sex allocation is robust to invasion and is maintained, even when sex allocation optima in environment 2 diverge between mothers and zygotes. For high frequencies of p, condition-dependent sex allocation is maintained as well, but condition-dependent sex allocation is now the result of interaction between maternally controlled genetic elements and (s_{1o}, s_{2o}) expressed in the offspring.

Imperfect environmental assessment

So far, we have assumed that mothers always correctly perceive the state of the environment or their own condition. In Section S6.3 of the Supplement, we relax this assumption by allowing for errors in perception of the environment: with probability ε ($0 \leq \varepsilon \leq 0.5$), individuals perceive the environment be in a state that is opposite to its actual state. Unsurprisingly, Figure 6.5 shows that nonzero levels of ε reduce sex ratio biases in each environment, until sex ratios for both parent and offspring finally converge towards their condition-independent equilibria $1/(1+c)$ and $1/(1+\sqrt{c})$ when $\varepsilon = 0.5$. In the previous sections, we showed that whenever mothers and offspring both favor a bang-bang sex allocation strategy $(s_1, s_2) = (1, 0)$, parent-offspring conflict is absent. However, since ε reduces the parameter space in which a bang-bang strategy is achieved, increased assessment errors also increase the parameter space in which parent-offspring conflict occurs (compare Figure 6.5B with Figure 6.1A).

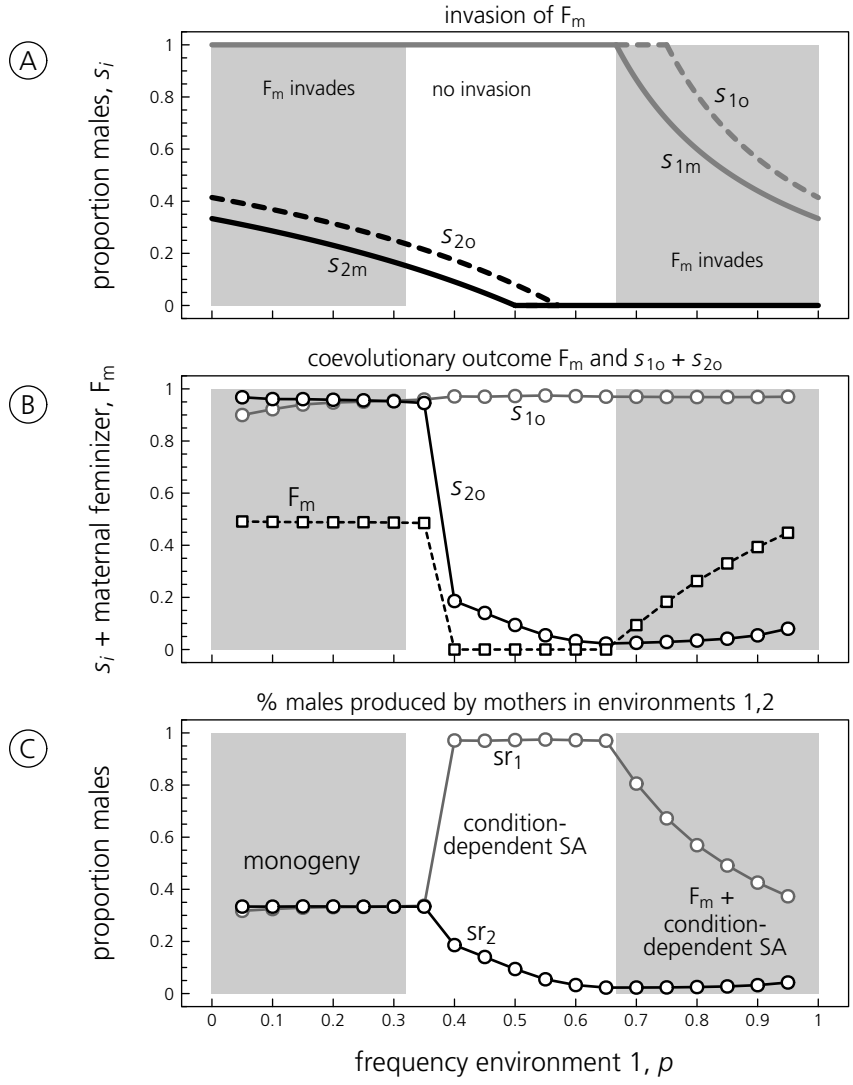

Figure 6.4: Invasion of a dominant feminizer (F_m) under maternal control, when condition-dependent sex allocation loci are controlled by the offspring (s_{1o}, s_{2o}) and when sons are twice as costly as daughters ($c = 2$). Panel A: grey regions depict the parameter space in which invasion of F_m is possible according to the analytical model. Panel B: upon successful invasion, the stable frequency of F_m depends on the frequency p of the poor environment. As a coevolutionary response to F_m, offspring sex allocation becomes more male biased, leading either to $(s_{1o}, s_{2o}) \approx (1, 0)$ or $(s_{1o}, s_{2o}) \approx (1, 1)$. Note that due to the stochastic nature of the individual-based simulations, F_m invades in a slightly wider range of conditions than predicted from the analytical invasion conditions. Panel C: despite the invasion of F_m, condition-dependent sex allocation is maintained for a wide range of p. Only when the poor environment is relatively rare (i.e., $p < 0.35$), does the invasion of F_m lead to a replacement of condition-dependent sex allocation with monogeny. Parameters: $v = 0.5$.

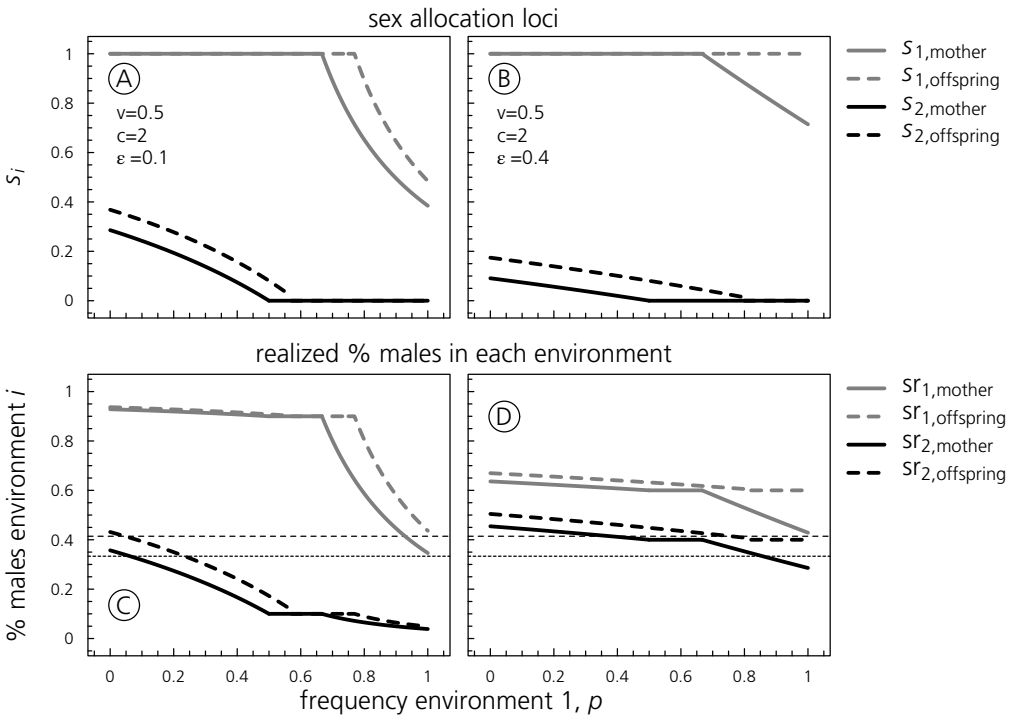

Figure 6.5: Errors in the assessment of the current environment reduce condition-dependent sex ratio biases, and increase parent-offspring conflict. Panels A,B: expression from maternal and offspring sex allocation loci s_1 and s_2 becomes more extreme to counter the effect of environmental errors. Panels C,D: unsurprisingly, with increasing error resulting sex ratios produced in each environment (e.g., $\text{sr}_1 = (1-\varepsilon)s_1 + \varepsilon s_2$) become less biased and will eventually converge to Fisherian (maternal control) and Triversian sex ratio optima in the absence of condition-dependent sex allocation (when $\varepsilon = 0.5$).

Coevolution between ε and sex allocation Since sex ratios become less biased with increasing levels of perception errors ε, this also begs the question if nonzero values of ε may be selectively favored by either parents or offspring, to achieve sex ratios closer to their respective optima. Figures 6.6 and S6.4 shows that parent-offspring conflict indeed leads to the evolution of nonzero values of ε. For example, Figure 6.6B shows that maternal errors ε_m invade in a scenario where sex allocation is controlled by the offspring whenever the good environment is relatively common. Under these conditions, mothers in environment 1 favor a more equal sex ratio than their offspring, and reducing the reliability of information present to offspring then results in a less biased sex ratio. However, ε_m only invades whenever environment 2 is relatively scarce, so that the benefits of producing a less biased sex ratio in environment 1 outweigh the negative effects of diverging from the optimum sex allocation optimum in environment 2, $s_{2\text{o}} = s_{2\text{m}} = 0$. The coevolutionary outcome is either a weaker form of condition-dependent sex allocation when perception errors evolve in the range $0 < \varepsilon_\text{m} < 0.5$, or effectively condition-independent sex allocation when $\varepsilon \approx 0.5$.

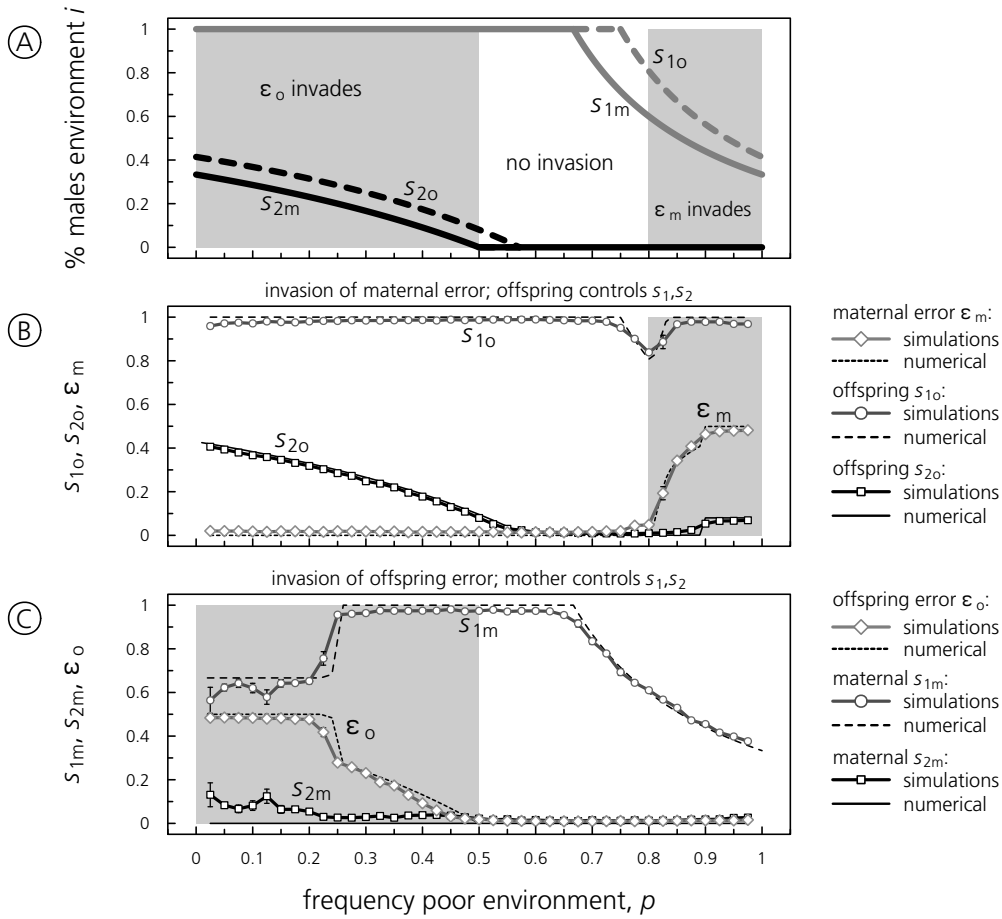

Figure 6.6: The invasion of environmental perception errors ε under maternal or offspring control. Panel A: $\varepsilon > 0$ under either maternal (ε_m) or offspring control (ε_o) invades (grey areas) whenever optimal sex allocation in one environment is closer to 0.5 than the actual sex allocation. Panel B: invasion of a maternal factor that increases offspring perception errors ε_m and subsequent coevolution of the offspring sex allocation loci s_{1o}, s_{2o}. Panel C: invasion of an offspring factor that increases maternal perception errors ε_o and subsequent coevolution of the maternal sex allocation loci s_{1m}, s_{2m}. Parameters: $c = 2$, $\nu = 0.5$.

A similar pattern is observed when mothers control sex allocation and offspring control ε. This reflects a putative scenario in which mothers sense the environment and adjust sex of each offspring accordingly, but where each zygote is able to secrete hormones that influence maternal perception when determining the offspring's sex. A genetic modifier coding for such hormone levels ε_o invades whenever maternal sex ratios are more biased away from equality than offspring sex ratios (see Figure 6.6C). Again, ε_o either evolves to intermediate levels, weakening condition-dependent sex allocation, or towards $\varepsilon_o = 0.5$, replacing condition-dependent sex allocation by condition-independent sex allocation. Although the effective replacement of condition-dependent sex allocation by condition-independent sex allocation through invading ε_m or ε_o occurs only in a limited region of parameter space (see Figure S6.4), it shows that genetic conflicts over sex determination can also be resolved

by behavioral or hormonal factors, that do not directly involve modifiers within the sex determining cascade.

6.4 Discussion

Although the role of genetic conflicts in the evolution of sex determining mechanisms is increasingly appreciated (Werren & Beukeboom, 1998; Burt & Trivers, 2006), existing predictions mainly focus on conflicts in the context of genetic sex determination (GSD) (e.g., Rigaud & Juchault, 1993; Werren *et al.*, 2002; Van Doorn & Kirkpatrick, 2007; Kozielska *et al.*, 2009), while environmental or conditional influences on sex determination have seen sparse attention in this context. The current study shows, however, that genetic conflicts may also affect the evolutionary maintenance of condition-dependent sex determining systems, such as temperature-dependent sex allocation (Valenzuela & Lance, 2004) or maternal control of sex allocation based on condition (Trivers & Willard, 1973) or host size (Charnov *et al.*, 1981).

Our model suggests that conflicts between maternally expressed and zygotically expressed genes can select for the invasion of sex factors that are independent of condition. Upon successful invasion, the subsequent coevolution between these unconditional sex factors and condition-dependent sex allocation gives rise to a variety of coevolutionary outcomes. Importantly, what outcome occurs depends to a large degree on the actual environmental variation. Typically, transitions from condition-dependent to condition-independent sex determining mechanisms (e.g., from ESD to maternally controlled, condition-independent sex determination) only occur when one environment is far more common than the other (see Figures 6.3, 6.4, S6.2, S6.3), since benefits of the invading unconditional sex factor in one environment have to outweigh the costs of producing suboptimal sex ratios in the other environment. The requirement of such 'uneven' environments for the successful invasion of sex factors could also be relevant in the context of environmental change (e.g., global warming): populations in which parent-offspring conflict occurs, but which are normally robust to the invasion of sex factors may endure environmental change, where one environmental condition starts to predominate. In turn, this could initiate the invasion of condition-independent sex factors, which typically occur on relatively fast timescales, see Figure S6.1. Although environmental change is widely implicated in the loss of condition-dependent sex determination (Ospina-Ãlvarez & Piferrer, 2008; Pen *et al.*, 2010; Grossen *et al.*, 2011), our model shows that environmental perturbations may do so in surprising ways, via parent-offspring conflict.

When transitions between condition-dependent and independent sex determining mechanisms indeed occur, they can lead to a variety of sex determining systems, such as male heterogamety (XX-XY), female heterogamety (ZZ-ZW), or monogeny, in which sex is under exclusive control of the mother. Except for the occurrence of monogeny, transitions between condition-dependent sex determining mechanisms, such as temperature-dependent sex determination, and all these genetic sex determining mechanisms have been observed in lizards, which have a striking diversity of sex determining systems (Janzen & Phillips, 2006; Ezaz *et al.*, 2009; Sarre *et al.*, 2011) and teleost fishes (Mank *et al.*, 2006).

Alternatively, cases where one environment is more common than the other may also give rise to a different outcome, in which condition-dependent sex allocation is evolutionarily maintained together with invading unconditional sex factors, such as

sex chromosomes. Unconditional sex factors and condition-dependent sex allocation loci coevolve to produce the sex ratio that is optimal in one environment, whereas the unconditional sex factor is selectively neutral in the other environment, because the condition-dependent sex determining mechanism already causes similar sex ratio biases as the invading sex factor in that environment (e.g., see Figure 6.3). In such cases, the invading sex factor is effectively integrated in the condition-dependent sex determining cascade. Our study thus suggests that parent-offspring conflict could explain observations in which sex chromosomes are stably maintained in species that have ESD as a form of condition-dependent sex allocation (Lagomarsino & Conover, 1993; Shine *et al.*, 2002; Quinn *et al.*, 2007; Radder *et al.*, 2008; Baroiller *et al.*, 2009; Alho *et al.*, 2010). Additionally, it also provides an evolutionary explanation for the finding that both maternal and offspring factors have been shown to contribute to condition-dependent sex determination in a number of reptiles (Radder *et al.*, 2009; Warner, 2011).

The current model also predicts that whenever different environments are encountered with roughly similar frequencies, condition-dependent sex allocation can be surprisingly robust against invasion of unconditional sex factors. Condition-dependent sex determining mechanisms are so robust, since although unconditional sex factors lead to more favorable sex ratios in one environment, they also give rise to undesirable brood sex ratios in the other environment. Future models are necessary to investigate how this prediction holds in case environmental variation is given by more continuous distributions. Previous models have shown that more continuous patterns of environmental variation lead to a larger predominance of "bang-bang" sex allocation strategies (e.g., Charnov, 1982; Frank, 1987; Charnov & Bull, 1989; Van Dooren & Leimar, 2003), in which individuals produce exclusively sons or daughters (but not both). As shown in our model, "bang-bang" sex allocation patterns reduce the scope for parent-offspring conflict, leading to the hypothesis that condition-dependent sex allocation would be even more robust to the invasion of unconditional sex factors than suggested by the current model. However, conflicts may still persist over the particular point at which individual sex allocation changes from the exclusive production of sons to that of daughters, so formal efforts are required to assess the scope for parent-offspring conflict over sex allocation in more complex environmental settings.

Apart from the invasion of unconditional sex factors, genetic conflicts over condition-dependent sex allocation may also be resolved at the perceptual level (see Figure 6.6). Our model shows that both parents and offspring may evolve perceptual errors in order to reduce the reliability of information available to the other party, thereby bringing the sex ratio closer to their own optimum. Currently, we can only speculate about the traits that could affect perception of the environment to either mother or offspring. A promising candidate behavior is maternal basking behavior, which has recently been associated with temperature-dependent sex determination in viviparous lizards (Wapstra *et al.*, 2004): in case of offspring control over condition-dependent sex determination, mothers could, for example, change their basking behavior, so that variation in temperatures experienced by the offspring is out of touch with actual temperature variation. When mothers are in control over condition-dependent sex determination, offspring may manipulate mothers by releasing hormones in the maternal bloodstream, which could putatively alter maternal perception of the environment (e.g., perception of temperature or population density). In many reptiles, embryos release a variety of hormones already early in

development (Xavier *et al.*, 1988; Guillette, 1989); although most of these factors are postulated to be involved in parent-offspring conflict over maternal nutrition (Crespi & Semeniuk, 2004), the actual function of these hormones is yet awaiting further exploration. Analogously, in oviparous species with offspring control over condition-dependent sex determination, mothers might influence the reliability of information to offspring by changing the structure of the egg or the structure of the nest (e.g., Shine & Harlow, 1996; Weisrock & Janzen, 1999; Morjan, 2003), which affects heat exchange and potentially could reduce offspring sensitivity to different temperatures. In oviparous species, however, there is a reduced scope for offspring traits that manipulate maternal perception, with expression of such traits being restricted to early developmental stages before eggshell formation prevents the release of offspring hormones. Albeit these aforementioned mechanisms are merely based on speculation, our model highlights that conflicts over condition-dependent sex allocation do not exclusively lead to evolutionary changes within the sex determining cascade itself: next to genetic sex factors, hormones or behaviors that alter environmental information could potentially play an important role as well in driving transitions between sex determining mechanisms.

By showing that invading genetic sex factors can be incorporated in sex determining systems that are sensitive to the environment or condition, our model corroborates recent work that environment-dependent and genetic sex determining systems should be considered as ends of a continuum (Sarre *et al.*, 2004; Crews & Bull, 2009; Pen *et al.*, 2010; Uller & Helanterä, 2011), as opposed to two disparate mechanisms of sex determination (Valenzuela *et al.*, 2003). Moreover, our study also supports the more recent view that transitions from condition-dependent to condition-independent sex determination are unlikely to be exclusively captured by changes in thermal sensitivity of the most upstream elements alone (see Uller & Helanterä, 2011, and references therein). Although the invasion of a thermally insensitive upstream element (e.g., a genetic sex factor) is obviously crucial to a transition from condition-dependent to condition-independent sex determination in the absence of perception errors, the eventual transition also requires a coevolutionary response of the downstream elements (in this case s_1 and s_2). In Figures 6.3B and 6.4B they lead to environmental insensitivity of s_1 and s_2 as well, with $s_1 \approx s_2$ being a consistent coevolutionary outcome (see also Figures S6.2B and S6.3B). In general, indications for the role of parent-offspring conflict in driving transitions from condition-dependent to condition-independent sex determination may be found from cases where both maternal and offspring sex factors should contribute to sexual development, for which there is now some initial evidence (Saillant *et al.*, 2003; Navarro-Martín *et al.*, 2011). Specifically, our model predicts that when the upstream environment-independent sex factor is controlled by the zygote (as is likely to be the case in taxa with GSD, where male or female heterogamety is likely to have replaced environment-dependent sex determination under maternal control), we predict that downstream elements in the sex determining cascade should be controlled by the mother. Moreover, these maternally controlled downstream elements should exhibit a temperature insensitive gene expression pattern in taxa with GSD, whereas their homologous counterparts in closely related taxa with environment-dependent sex determination under maternal control should be highly sensitive to temperature. Predictions such as these could be tested in the foreseeable future, given the increasing molecular knowledge about sex determination in phylogenetic groups that con-

tain taxa where the environment (by means of temperature, condition or population density) affects sex determination (Quinn *et al.*, 2007; Marshall Graves, 2008; Quinn *et al.*, 2011; Sarre *et al.*, 2011).

An aspect left unexplained by our model on parent-offspring are transitions from condition-independent to condition-dependent sex determination, as opposed to transitions in the reverse direction studied here. The evolutionary mechanisms that lead to transitions from condition-independent to condition-dependent sex determination have been relatively well studied (Bull, 1981a; Van Dooren & Leimar, 2003; Feldmeyer, 2009; Pen *et al.*, 2010), but these studies have not considered if genetic conflicts may contribute to any such transitions. Our model shows that parent-offspring conflict can lead to the maladaptive outcome where condition-independent sex determination evolves, despite condition-dependent sex determination being a more adaptive solution to both parents and offspring. It is difficult to envisage similar cases where parent-offspring conflict results in transitions from condition-independent sex determination to condition-dependent sex determination, despite condition-dependent sex determination being maladaptive to both parents and offspring. One highly hypothetical way in which such transitions may occur is when genetic conflicts eliminate the upstream environment-insensitive sex factor, thereby exposing downstream elements that may be sensitive to environment or condition. However, current theory on parent-offspring conflict would rather predict the addition of novel upstream elements as opposed to any removal of such elements (Werren *et al.*, 2002; Kozielska, 2008). Alternatively, condition-dependent sex determination may be an adaptive outcome after all, but only for either the offspring or parents, but not for both simultaneously. For example, when phenotypic plasticity that is inherent to condition-dependent sex determination comes with certain costs (Auld *et al.*, 2010) that affect a mother's fecundity, each individual offspring may potentially favor a larger investment in phenotypic plasticity than what is selectively favored by the mother. Potentially, condition-dependent sex determination under offspring control may then invade and replace condition-independent sex determination under maternal control. Future models should assess if mothers and zygotes indeed selectively favor divergent levels of phenotypic plasticity.

Finally, different costs of producing sons versus daughters is the essential aspect that drives parent-offspring conflict in the current model. Currently, there is surprisingly little information available about sex-specific production costs in organisms that have condition-dependent sex allocation. A study in the lizard *Bassiana duperreyi* has shown that females typically develop from larger eggs, indicative of sex specific reproduction costs (Radder *et al.*, 2009). Moreover, the addition of additional resources (e.g., yolk) to freshly laid eggs of this species even has a direct affect on sex determination under certain temperatures, increasing the likelihood of female development (Radder *et al.*, 2009). In a haplodiploid mite species with facultative sex ratios, larger eggs are more likely to get fertilized (initiating female development) than smaller eggs, which is also indicative of a sex-specific production costs (Macke *et al.*, 2011). If sex-specific production costs are indeed a general phenomenon remains to be assessed by future experiments, since such costs are currently only starting to be explored in taxa with and without condition-dependent sex allocation (see Rutkowska *et al.* 2011 and references therein). In the absence of sex-specific production costs, however, other factors such as local interactions among offspring may also lead to parent-offspring conflict over condition-dependent sex allocation (Werren &

Hatcher, 2000; Pen, 2006; Wild & West, 2007). It would be interesting to assess if our conclusions on parent-offspring conflict in taxa with condition-dependent sex allocation change due to the influence of local interactions, such as local mate competition (Hamilton, 1967) or local resource competition (Clark, 1978). For example, Wild & West (2007) have shown that local resource competition may lead to a reduced scope for "bang-bang" sex allocation patterns in the context of condition-dependent sex allocation, which could result in an increased scope for parent-offspring conflict as shown by the current model. Many opportunities thus remain to improve our understanding of role of genetic conflicts in the evolution of condition-dependent sex allocation.

Acknowledgements We thank the Groningen High Performance Cluster for computing time.

A6.1 Appendix

Maternal control over sex allocation

The matrix $\mathbf{B}\left(\hat{s}_1,\hat{s}_2|s_1,s_2\right)$ (hereafter $\mathbf{B}$) represents the invasion dynamics of a mutant mother with strategy $(\hat{s}_1,\hat{s}_2)$ in a resident population with sex allocation strategy (s_1,s_2):

$$\mathbf{B}=\frac{1}{2\lambda}\begin{bmatrix} pvf_1(\hat{s}_1) & pf_2(\hat{s}_2) & p\left[\frac{y_{f1}}{y_m}vf_1(s_1)+\frac{y_{f2}}{y_m}f_2(s_2)\right] \\ (1-p)vf_1(\hat{s}_1) & (1-p)f_2(\hat{s}_2) & (1-p)\left[\frac{y_{f1}}{y_m}vf_1(s_1)+\frac{y_{f2}}{y_m}f_2(s_2)\right] \\ m_1(\hat{s}_1) & m_2(\hat{s}_2) & \frac{y_{f1}}{y_m}m_1(s_1)+\frac{y_{f2}}{y_m}m_2(s_2) \end{bmatrix}. \quad \text{(A6.1)}$$

Mutant strategies appear only in the first and second columns of $\mathbf{B}$, since we consider maternal control of the sex ratio, so any mutant sex ratio strategy will only be expressed in females. All entries in the matrix are multiplied by the constant $1/2$, which considers the probability that a mutant sex ratio gene copy has a probability of $\frac{1}{2}$ of being passed on from a parent to its offspring. Additionally, the model considers the growth rate of a mutant individual relative to the resident (Mylius & Diekmann, 1995), hence we scale the matrix $\mathbf{B}$ by $1/\lambda$, where λ is the dominant eigenvalue of the resident transition matrix $\mathbf{A}$ in eq. (6.1).

Relative frequencies and reproductive values For consistency, it is required that all females (having relative frequencies y_{f1} and y_{f2} when breeding in environments 1 and 2 respectively) have the same reproductive output as all males (y_m):

$$\mathbf{a}_1y_{f1}+\mathbf{a}_2y_{f2}=\mathbf{a}_3y_m, \quad \text{(A6.2)}$$

where $\mathbf{a}_i$ is the ith column of the resident transition matrix $\mathbf{A}$. Using this equation, we can easily find the dominant eigenvalue λ of $\mathbf{A}$. Let $\mathbf{y}=(y_{f1},y_{f2},y_m)^T$ be the dominant right eigenvector of $\mathbf{A}$, containing the stable relative class frequencies (T denotes transposition). This eigenvector is given by $\mathbf{Ay}=\lambda\mathbf{y}$, or $\mathbf{a}_1y_{f1}+\mathbf{a}_2y_{f2}+\mathbf{a}_3y_m=\lambda\mathbf{y}$. Substituting (A6.2) into this equation, we get $\lambda\mathbf{y}=2(\mathbf{a}_1y_{f1}+\mathbf{a}_2y_{f2})$, so that

$$\lambda=(1-p)f_2(s_2)+pvf_1(s_1). \quad \text{(A6.3)}$$

We can then solve $\mathbf{Ay}=\lambda\mathbf{y}$ for the vector of stable relative class frequencies $\mathbf{y}$ of the resident population, which is

$$\mathbf{y}^T=\left[y_{f1},y_{f2},y_m\right]=\left[\ p,\quad 1-p,\quad \frac{pm_1(s_1)+(1-p)m_2(s_2)}{(1-p)f_2(s_2)+pvf_1(s_1)}\ \right]. \quad \text{(A6.4)}$$

Similarly, we can solve $\mathbf{zA}=\lambda\mathbf{z}$ to obtain the elements of the dominant left eigenvector $\mathbf{z}=(z_{f1},z_{f2},z_m)$ of $\mathbf{A}$, containing the reproductive values. These values are quite long and not particularly informative, so we will not show them here.

Selection differentials We calculate the selection differentials on $(\hat{s}_1,\hat{s}_2)$ using eq. (6.2) in the main text. In the results below, the positive, constant $\mathbf{y}^T\mathbf{z}$ in the denominator is not included, since it does not play a role in the calculation of equilibria.

The selection differentials for the mutant sex allocation strategy are given by

$$\left.\frac{\partial W}{\partial \hat{s}_1}\right|_{\hat{s}_1=s_1} = \frac{y_{f1}\gamma_1^2}{2\lambda}\left[z_m - vc\left(z_{f1}p + z_{f2}(1-p)\right)\right],$$
$$\left.\frac{\partial W}{\partial \hat{s}_2}\right|_{\hat{s}_2=s_2} = \frac{y_{f2}\gamma_2^2}{2\lambda}\left[z_m - c\left(z_{f1}p + z_{f2}(1-p)\right)\right], \tag{A6.5}$$

where $\gamma_i = s_i c + 1 - s_i$. Solving both selection differentials in (A6.5) simultaneously requires that $c\left(z_{f1}p + z_{f2}(1-p)\right) = vc\left(z_{f1}p + z_{f2}(1-p)\right)$, which only holds in the trivial case whenever $v = 1$. Therefore, the equilibria for one or both sex allocation strategies (s_1, s_2) should reside at the boundaries $s_i = 0$ or $s_i = 1$, which is a well-known feature of the Charnov-Bull model (e.g., Schwanz *et al.*, 2006; Wild & West, 2007). For a boundary strategy $(s_k = 0, s_j = 1)$ $k \neq j$ to be is stable, the following should hold:

$$\left.\frac{\partial W}{\partial \hat{s}_k}\right|_{\hat{s}_k=s_k=0} < 0$$
$$\left.\frac{\partial W}{\partial \hat{s}_j}\right|_{\hat{s}_j=s_j=1} > 0. \tag{A6.6}$$

In case one of both sex allocation traits is at its boundary, but the other trait s_j is mixed, the following condition applies for the stability of the equilibrium strategy:

$$\left.\frac{\partial W}{\partial \hat{s}_k}\right|_{\hat{s}_k=s_k} \begin{cases} < 0 & s_k = 0 \\ > 0 & s_k = 1 \end{cases}, \tag{A6.7}$$

and when

$$\left.\frac{\partial^2 W}{\partial \hat{s}_k^2}\right|_{\hat{s}_j=s_j} < 0.$$

Stability of boundary equilibria results in three qualitatively different regions, whose boundaries are represented in Table 6.1 in the main text.

Offspring control over sex allocation

The matrix $\mathbf{B}_o\left(\hat{s}_1, \hat{s}_2, \tilde{s}_1, \tilde{s}_2 | s_1, s_2\right)$ (hereafter $\mathbf{B}_o$) represents the invasion dynamics of a mutant offspring with sex allocation strategy $(\hat{s}_1, \hat{s}_2)$ in a brood with siblings having sex allocation strategies $(\tilde{s}_1, \tilde{s}_2)$, while the population sex allocation strategy is given by (s_1, s_2):

$$\mathbf{B}_o = \frac{1}{2\lambda}\begin{bmatrix} pvf_1(\hat{s}_1,\tilde{s}_1) & pf_2(\hat{s}_2,\tilde{s}_2) & p\left(\frac{y_{f1}}{y_m}vf_1(\hat{s}_1,\tilde{s}_1) + \frac{y_{f2}}{y_m}f_2(\hat{s}_2,\tilde{s}_2)\right) \\ (1-p)vf_1(\hat{s}_1,\tilde{s}_1) & (1-p)f_2(\hat{s}_2,\tilde{s}_2) & (1-p)\left(\frac{y_{f1}}{y_m}vf_1(\hat{s}_1,\tilde{s}_1) + \frac{y_{f2}}{y_m}f_2(\hat{s}_2,\tilde{s}_2)\right) \\ m_1(\hat{s}_1,\tilde{s}_1) & m_2(\hat{s}_2,\tilde{s}_2) & \frac{y_{f1}}{y_m}m_1(\hat{s}_1,\tilde{s}_1) + \frac{y_{f2}}{y_m}m_2(\hat{s}_2,\tilde{s}_2) \end{bmatrix}. \tag{A6.8}$$

The numbers of males and females resulting from a brood with a mutant offspring are now dependent on both the mutant and the sibling allocation strategies

$$f_i(\hat{s}_i,\tilde{s}_i)=\frac{1-\hat{s}_i}{\tilde{s}_i c+1-\tilde{s}_i},$$
$$m_i(\hat{s}_i,\tilde{s}_i)=\frac{\hat{s}_i}{\tilde{s}_i c+1-\tilde{s}_i}.$$

Using a direct fitness approach (e.g., see Taylor *et al.* (2007)), the selection differential acting on the mutant sex allocation locus $\hat{s}_j$ is given by

$$\left.\frac{\mathrm{d}W}{\mathrm{d}\hat{s}_j}\right|_{\hat{s}_1=\tilde{s}_1=s_1}=\frac{\partial W}{\partial \hat{s}_j}+R\frac{\partial W}{\partial \tilde{s}_j},$$

with R being the coefficient of relatedness among siblings, which is $\frac{1}{2}$ when females mate only once. Selection differentials under offspring control are

$$\begin{aligned}\left.\frac{\mathrm{d}W}{\mathrm{d}\hat{s}_1}\right|_{\hat{s}_1=\tilde{s}_1=s_1}&=\frac{\gamma_1 y_{\mathrm{f}1}}{\lambda}\Bigg(z_{\mathrm{m}}-\nu\left(z_{\mathrm{f}1}p+z_{\mathrm{f}2}(1-p)\right)\\&\quad+\frac{\gamma_1}{2}(1-c)\left[z_{\mathrm{m}}s_1-\nu\left(1-s_1\right)\left(z_{\mathrm{f}1}p+z_{\mathrm{f}2}(1-p)\right)\right]\Bigg),\\\left.\frac{\mathrm{d}W}{\mathrm{d}\hat{s}_2}\right|_{\hat{s}_2=\tilde{s}_2=s_2}&=\frac{\gamma_2 y_2}{\lambda}\Bigg(z_{\mathrm{m}}-\left(z_{\mathrm{f}1}p+z_{\mathrm{f}2}(1-p)\right)\\&\quad+\frac{\gamma_2}{2}(1-c)\left[z_{\mathrm{m}}s_2-\left(1-s_2\right)\left(z_{\mathrm{f}1}p+z_{\mathrm{f}2}(1-p)\right)\right]\Bigg).\end{aligned}$$

Again, stability of boundary equilibria results in three qualitatively different regions, whose boundaries are represented in Table 6.1 in the main text.

S6 Supplementary Information

The invasion of offspring genetic sex modifiers

Here, track the invasion of condition-independent and dominant offspring sex determining modifiers in a population having condition-dependent sex allocation under maternal control. To this end, we extend a population genetical approach by Werren *et al.* (2002), that was used to track the invasion of sex determining modifiers under parent-offspring conflict in the absence of condition-dependent sex allocation.

S6.1.1 Invasion of a dominant offspring masculinizer

Consider two diallelic loci: the first locus codes for maternally controlled condition-dependent sex allocation. Here, aa is the common genotype and such females produce a proportion of males s_i in environment i. The second locus is expressed in the zygote; individuals having the bb genotype defer control over sex allocation to the mother, and thus become male with probability s_i or $\hat{s}_i$ dependent on the maternal genotype. In contrast, individuals having the mutant Bb genotype always become male, independent of their maternal genotype and the environment. Since Bb males will only mate with bb females, the genotype BB does not exist. Let x_1, x_2, x_3 be the

frequencies of $aabb$ females, $aabb$ males and $aaBb$ males respectively, so that we have the following system of recursions

$$\begin{aligned}
\bar{w}x_{1,t+1} &= x_{1,t}\sum_i p_i v_i\left[x_{2,t}h_{bb}\left(s_i\right)\left(1-s_i\right)+\frac{1}{2}x_{3,t}h_{Bb}\left(s_i\right)\left(1-s_i\right)\right],\\
\bar{w}x_{2,t+1} &= \sum_i p_i\left[x_{2,t}x_{1,t}h_{bb}\left(s_i\right)s_i+\frac{1}{2}x_{3,t}x_{1,t}h_{Bb}\left(s_i\right)s_i\right],\\
\bar{w}x_{3,t+1} &= \frac{1}{2}x_{3,t}\sum_i p_i x_{1,t}h_{Bb}\left(s_i\right),
\end{aligned} \tag{S6.1}$$

where

$$h_{Bb}\left(s_i\right)=\frac{1}{\frac{1}{2}\left(1-s_i\right)+\frac{1}{2}\left(1+s_i\right)c},\quad h_{bb}\left(s_i\right)=\frac{1}{1-s_i+s_ic},$$

being the number of newborns produced by mothers mated with Bb and bb males respectively. Lastly, mean fitness is given by

$$\begin{aligned}
\bar{w}=\sum_i p_i\Bigg(&x_{1,t}v_i\left[x_{2,t}h_{bb}\left(s_i\right)\left(1-s_i\right)+\frac{1}{2}x_{3,t}h_{Bb}\left(s_i\right)\left(1-s_i\right)\right]+x_{2,t}x_{1,t}h_{bb}\left(s_i\right)s_i\\
&+\frac{1}{2}x_{3,t}x_{1,t}h_{Bb}\left(s_i\right)s_i+\frac{1}{2}x_{3,t}x_{1,t}h_{Bb}\left(s_i\right)\Bigg).
\end{aligned}$$

We now calculate if a rare B allele can invade in a population that entirely consists of bb individuals. To this end, we note that $x_1+x_2+x_3=1$, allowing us to reduce the system (S6.1) to two dimensions. We then calculate the eigenvalues of the 2×2 Jacobian evaluated at the point where $x_1=1-\mathrm{sr}_m,\ x_2=\mathrm{sr}_m,\ x_3=0$, for each of the three regions in Table 6.1. The invasion conditions are summarized in Table S6.1 and are discussed below.

S6.1.2 Invasion of a dominant offspring feminizer

Again, individuals having the bb genotype defer control over sex allocation to the mother, and thus become male with probability s_i or $\hat{s}_i$ dependent on the maternal genotype. In contrast, individuals having the mutant Bb genotype always become female, independent of their maternal genotype and the environment. Since Bb females will only mate with bb males, the genotype BB does not exist. Let x_1, x_2, x_3 be the frequencies of $aabb$ females, $aabb$ males and $aaBb$ females respectively, so that we have the following system of recursions

$$\begin{aligned}
\bar{w}x_{1,t+1} &= \sum_i p_i v_i\left[x_{1,t}x_{2,t}g_{bb}\left(s_i\right)\left(1-s_i\right)+\frac{1}{2}x_{3,t}x_{2,t}g_{Bb}\left(s_i\right)\left(1-s_i\right)\right],\\
\bar{w}x_{2,t+1} &= \sum_i p_i\left[x_{2,t}x_{1,t}g_{bb}\left(s_i\right)s_i+\frac{1}{2}x_{3,t}x_{2,t}g_{Bb}\left(s_i\right)s_i\right],\\
\bar{w}x_{3,t+1} &= \frac{1}{2}x_{3,t}\sum_i p_i v_i x_{2,t}g_{Bb}\left(s_i\right),
\end{aligned} \tag{S6.2}$$

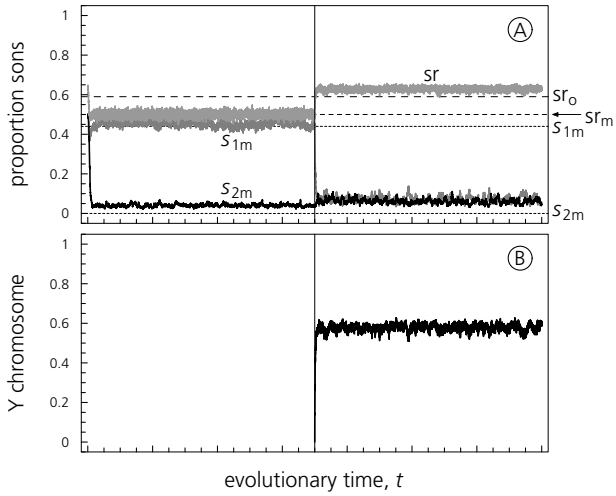

Figure S6.1: Example simulation showing the successful invasion of a dominant masculinizer Y under offspring control. The black vertical line indicates the introduction of the Y chromosome in the population. Panel A: before the invasion of Y, maternal condition-dependent sex allocation loci s_{1m} and s_{2m} achieve their analytically predicted optima (dotted lines). The subsequent invasion of Y (panel B) leads to the overproduction of males, which selects mothers to induce those offspring who did not receive the Y chromosome to develop exclusively as daughters ($s_{1m} \to 0, s_{2m} \to 0$). As a result, sex ratios produced become independent of the environment and determined by the presence of Y only (GSD). Note that the resulting sex ratio is slightly higher than the optimal sex ratio under offspring control sr_o. This is because mutation-selection balance in s_{1m} and s_{2m} leads to the occasional production of sons. Parameters: $c = 2,\ p = 0.9,\ v = 0.5$.

where

$$g_{Bb}(s_i) = \frac{1}{\frac{1}{2}(2 - s_i + s_i c)}, \quad g_{bb}(s_i) = \frac{1}{1 - s_i + s_i c},$$

being the number of newborns produced by mothers having genotypes Bb and bb respectively. Lastly, mean fitness is given by

$$\bar{w} = \sum_i p_i \left(v_i \left[x_{1,t} x_{2,t} g_{bb}(s_i)(1 - s_i) + \frac{1}{2} x_{3,t} x_{2,t} g_{Bb}(s_i)(1 - s_i) \right] + x_{2,t} x_{1,t} g_{bb}(s_i) s_i \right.$$
$$\left. + \frac{1}{2} x_{3,t} x_{2,t} g_{Bb}(s_i) s_i + \frac{1}{2} v_i x_{3,t} x_{2,t} g_{Bb}(s_i) \right).$$

Invasion conditions are calculated in a similar fashion as for dominant offspring masculinizers and are listed in Table S6.1.

S6.1.3 Invasion conditions of offspring genetic modifiers: summary

It is obvious from Table 6.1 that the invasion of dominant offspring masculinizers typically occurs whenever males are more costly to produce than females, and the reverse applies for the invasion of the dominant offspring feminizer. This is particularly clear in regions I_m (masculinizer always invades when $c > 1$) and III_m (feminizer always invades when $c < 1$). For other combinations of the SA region and masculin-

Table S6.1: Invasion conditions for dominant masculinizers (e.g., Y-chromosomes) and feminizers (e.g., W-chromosomes) under offspring control (assuming $c > 0$). Regions I_m, II_m and III_m refer to the different sex allocation regions depicted in Figure 6.1 under maternal control. †: second order approximation near $v = 0$. ‡: when $v < \frac{1}{2}$ the additional condition $p(1+v) < \frac{1}{2}$ also applies.

SA region (maternal control):	I_m	II_m	III_m
boundaries:	$0 < p < \frac{1}{2}$	$\frac{1}{2} < p < \frac{1}{1+v}$	$\frac{1}{1+v} < p < 1$
dominant offspring masculinizer:	$c > 1$	$c > \frac{p}{2-3p}$	$c > 1 + \frac{4pv(1-p(1-2v))}{(1-p)^2}$ †
dominant offspring feminizer:	$c < \frac{(1+c)(1-2p^2-c)}{4p^2(1+c)-2p(1+3c)} <$ $v < 1$, $c < 1-2p^2$	$c < \frac{p(1+2v)-1}{1-p}$ ‡	$c < 1$

izer or feminizer loci, invasion conditions are more complicated and depend on the frequency of the poor environment p and the survival probability of females v in the poor environment. To provide more insight, the invasion conditions of a dominant offspring masculinizer are graphically represented for the case $c = 2$ and $v = 0.5$ in Figure S6.1. Similarly, Figure S6.2 shows the invasion of a dominant offspring feminizer for the case $c = 0.5$ and $v = 0.5$.

The invasion of maternal genetic sex modifiers

We now investigate the invasion of maternal genetic sex modifiers which override condition-dependent sex allocation under offspring control.

S6.2.1 Invasion of a dominant maternal masculinizer

Again, aa is the common genotype, which specifies that offspring develop as a male with probability s_i in environment i. The second locus is expressed in the mother; mothers having the mm genotype defer control over sex allocation to their offspring. In contrast, mothers having the mutant Mm genotype (in the main text denoted as M_m) give birth to broods consisting only of males, independent of the genotypes of their offspring. Let x_1, x_2, x_3 be the frequencies of $aamm$ females, $aamm$ males and $aaMm$ females respectively, so that we have the following system of recursions

$$\begin{aligned}
\bar{w}x_{1,t+1} &= \sum_i p_i v_i x_{1,t} x_{2,t} z_{mm}(s_i)(1-s_i), \\
\bar{w}x_{2,t+1} &= \sum_i p_i x_{2,t} x_{1,t} z_{mm}(s_i) s_i + \frac{1}{2} x_{3,t} x_{2,t} z_{Mm}, \\
\bar{w}x_{3,t+1} &= \frac{1}{2} x_{3,t} x_{2,t} z_{Mm},
\end{aligned} \tag{S6.3}$$

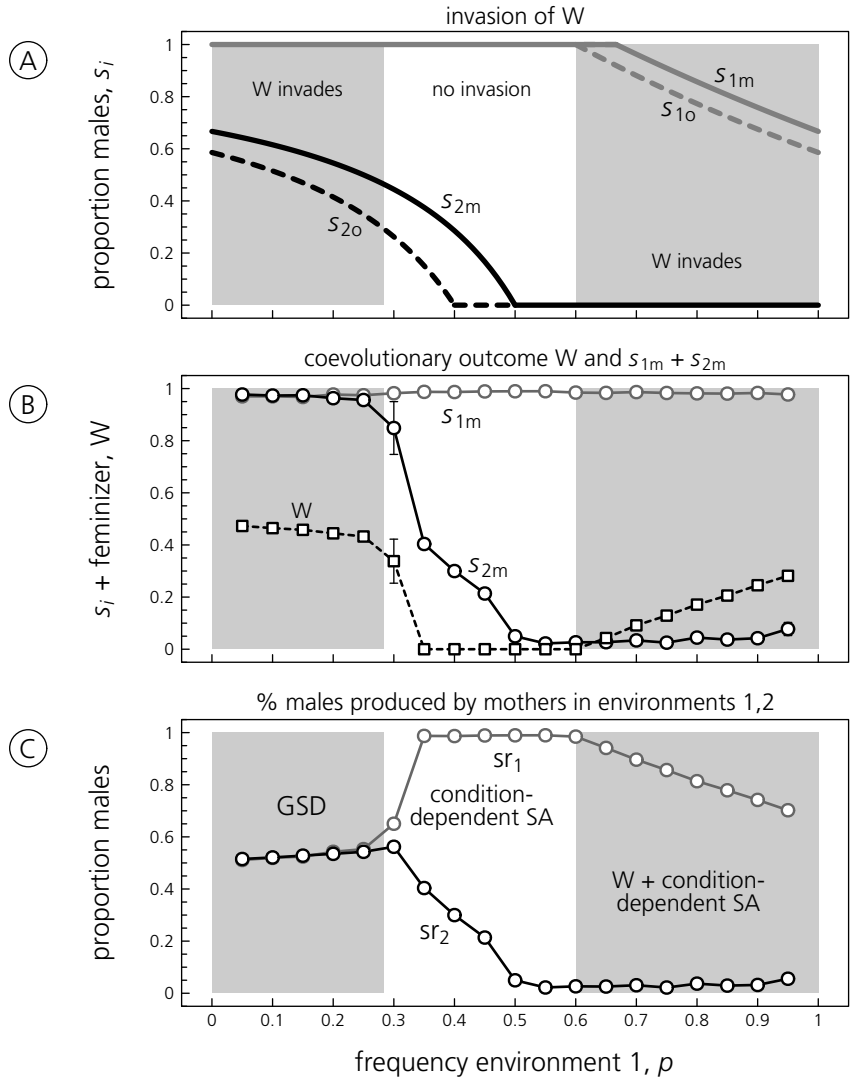

Figure S6.2: Invasion of a dominant feminizer (W) under offspring control, when condition-dependent sex allocation is controlled by the mother (s_{1m}, s_{2m}) and when daughters are twice as costly as sons ($c = 0.5$). Panel A: the invasion of W (grey areas, see Table S6.1) occurs when sex allocation optima diverge between parents and offspring, but under certain conditions (left part of middle white area) sex ratio selection prevents the invasion of W despite divergent maternal and offspring sex ratio optima. Panel B: upon successful invasion, the stable frequency of W depends on the frequency p of the poor environment. As a coevolutionary response to the offspring's W, maternal sex allocation strategies become more extreme, often leading either to $(s_{1m}, s_{2m}) \approx (1, 1)$ or $(s_{1m}, s_{2m}) \approx (1, 0)$. Panel C: despite the invasion of W, the proportion of males produced still depends strongly on the environment for a substantial range of p, although resulting sex ratios are now closer to offspring than to parental optima. Only when the poor environment is relatively rare (i.e., $r < 0.28$) does the invasion of W lead to a replacement of condition-dependent sex allocation with GSD. Parameters: $v = 0.5$.

where

$$z_{Mm} = \frac{1}{c}, \quad z_{mm}(s_i) = \frac{1}{1 - s_i + s_i c},$$

being the number of newborns produced by mothers with Mm and mm genotypes respectively. Mean fitness is given by

$$\bar{w} = \sum_i p_i \left[v_i x_{1,t} x_{2,t} z_{mm}(s_i)(1 - s_i) + x_{2,t} x_{1,t} z_{mm}(s_i) s_i \right] + \frac{1}{2} x_{3,t} x_{2,t} z_{Mm} + \frac{1}{2} x_{3,t} x_{2,t} z_{Mm}$$

Invasion conditions are calculated as described in section S6.1.1 and are listed in Table S6.2.

S6.2.2 Invasion of a dominant maternal feminizer

Mothers having the ff genotype defer control over sex allocation to their offspring. In contrast, mothers having the mutant Ff genotype (in the main text denoted as F_m) give birth to broods consisting only of females, independent of the genotypes of their offspring. Let x_1, x_2, x_3 be the frequencies of $aaff$ females, $aaff$ males and $aaFf$ females respectively, so that we have the following system of recursions

$$\begin{aligned}
\bar{w} x_{1,t+1} &= \sum_i p_i v_i \left[x_{1,t} x_{2,t} z_{ff}(s_i)(1 - s_i) + \frac{1}{2} x_{3,t} x_{2,t} z_{Ff} \right], \\
\bar{w} x_{2,t+1} &= \sum_i p_i x_{2,t} x_{1,t} z_{ff}(s_i) s_i, \\
\bar{w} x_{3,t+1} &= \frac{1}{2} x_{3,t} \sum_i p_i v_i x_{2,t} z_{Ff},
\end{aligned} \tag{S6.4}$$

where

$$z_{Ff} = 1, \quad z_{ff}(s_i) = \frac{1}{1 - s_i + s_i c},$$

being the number of newborns produced by mothers with Ff and ff genotypes respectively. Mean fitness is given by

$$\bar{w} = \sum_i p_i \left(v_i \left[x_{1,t} x_{2,t} z_{ff}(s_i)(1 - s_i) + \frac{1}{2} x_{3,t} x_{2,t} z_{Ff} \right] + x_{2,t} x_{1,t} z_{ff}(s_i) s_i + \frac{1}{2} x_{3,t} v_i x_{2,t} z_{Ff} \right).$$

Invasion conditions are calculated as described in section S6.1.1 and are listed in Table S6.2. In region I_o, invasion conditions for the maternal feminizer F_m are rather complicated and cannot be expressed in insightful terms, but feminizers typically invade whenever sons are more costly than daughters ($c > 1$); we refer the reader to Figure 6.4A for a more informative graphical example of these invasion conditions. Regarding region II_o, a maternal feminizer invades for those frequencies p of the poor environment for which the offspring region II_o $(s_{1o}, s_{2o}) = (1, 0)$ and III_m $(s_{1m}, s_{2m}) =$

Table S6.2: Invasion conditions for maternally controlled masculinizers M_m and feminizers F_m when condition-dependent sex allocation is under offspring control (assuming $c > 0$). Regions I_o, II_o and III_o refer to the different sex allocation regions depicted in Figure 6.1 under offspring control. K_1 and K_2 are given in the caption of Table 6.1.

SA region (offspring control):	I_o	II_o	III_o
boundaries:	$0 < p < \frac{2c}{1+3c}$	$\frac{2c}{1+3c} < p < \frac{1+c}{1+c+2v}$	$\frac{1+c}{1+c+2v} < p < 1$
dominant maternal masculinizer M_m:	$c < 1$	no invasion	$\frac{(1-c)(1-p)-4cpv-\sqrt{K_2}}{8cp(p(1-v)-1)}$
dominant maternal feminizer F_m:	$\frac{2(1-c)(1-p(1-v))}{4-(3+c)p-\sqrt{K_1}} > 1$	$\frac{1}{1+v} < p < \frac{1+c}{1+c+2v}$	$c > 1$

$(0 < s_{1m} < 1, 0)$ overlap, which only occurs when $c > 1$. In region III_o, invasion of the feminizer occurs for all frequencies p whenever $c > 1$. An illustration of the invasion conditions for a dominant maternal feminizer for the case $c = 2,\ v = 0.5$ is given in the main text in Figure 6.4A.

Errors in environmental perception

We alter the model in the main text by assuming that mothers accurately assess the state of the environment or their condition with probability $1-\varepsilon$, whereas with probability ε, mothers incorrectly perceive the opposite environment. For a mother with sex allocation strategy (s_i, s_j), the functions f_i and m_i in eq. (A6.1) that give the number of her sons and daughters are now given by

$$\begin{aligned} f_i\left(s_i,s_j\right) =& (1-\varepsilon)\frac{1-s_i}{s_i c+1-s_i} + \varepsilon\frac{1-s_j}{s_j c+1-s_j}, \\ m_i\left(s_i,s_j\right) =& (1-\varepsilon)\frac{s_i}{s_i c+1-s_i} + \varepsilon\frac{s_j}{s_j c+1-s_j}. \end{aligned} \tag{S6.5}$$

Regarding offspring control over environmental perception, we assume that each offspring is able to perceive the particular environment for itself. As a result, $1-\varepsilon$ of all offspring correctly perceive the environment, whereas ε of all offspring perceive the wrong environment, leading to the following functions f_i and m_i:

$$\begin{aligned} f_i\left(s_i,s_j\right) =& \frac{(1-\varepsilon)\left(1-s_i\right)+\varepsilon\left(1-s_j\right)}{(1-\varepsilon)\left[1-s_i+s_i c\right]+\varepsilon\left[1-s_j+s_j c\right]}, \\ m_i\left(s_i,s_j\right) =& \frac{(1-\varepsilon)\left(1-s_i\right)+\varepsilon\left(1-s_j\right)}{(1-\varepsilon)\left[1-s_i+s_i c\right]+\varepsilon\left[1-s_j+s_j c\right]}. \end{aligned}$$

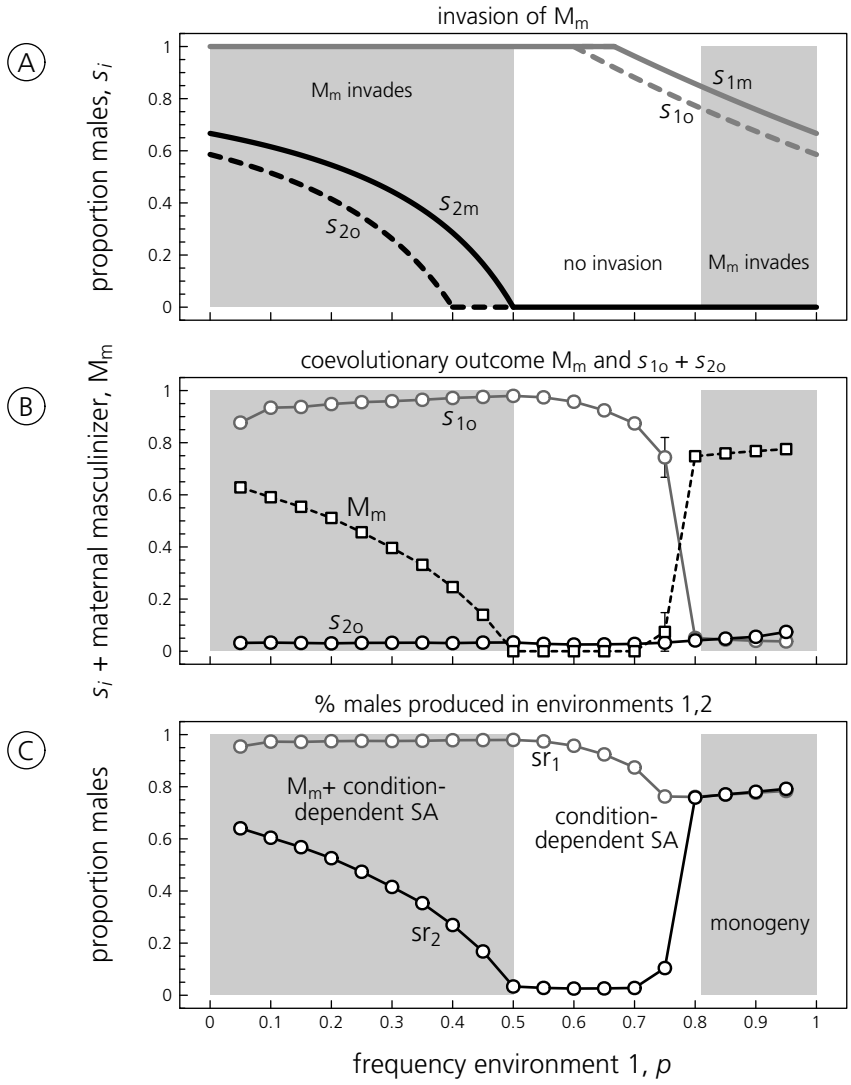

Figure S6.3: Invasion of a dominant masculinizer under maternal control M_m, when condition-dependent sex allocation is controlled by the offspring (s_{1o}, s_{2o}) and when daughters are twice as costly as sons ($c = 0.5$). Panel A: the invasion of M_m (grey areas, see Table S6.2) occurs when sex allocation optima diverge between parents and offspring, but under certain conditions (right part of middle white area) sex ratio selection prevents the invasion of M_m despite divergent maternal and offspring sex ratio optima. Panel B: upon successful invasion, the stable frequency of M_m depends on the frequency p of the poor environment. As a coevolutionary response to maternal M_m, offspring sex allocation strategies become more extreme, often leading either to $(s_{1o}, s_{2o}) \approx (1, 0)$ or $(s_{1o}, s_{2o}) \approx (0, 0)$. Panel C: despite the invasion of M_m, condition-dependent sex allocation persists over a substantial range of p, although resulting sex ratios are now closer to parental than to offspring optima. Only when the poor environment is very common (i.e., $p > 0.8$) does the invasion of M_m lead to a replacement of condition-dependent sex allocation with condition-independent monogeny. Parameters: $v = 0.5$.

In the presence of perception errors, selection differentials on s_1 and s_2 under maternal control are given by

$$\left.\frac{\mathrm{d}W}{\mathrm{d}\hat{s}_1}\right|_{\hat{s}_1=s_1} = \frac{\gamma_1^2}{2\lambda}\left[\left((1-\varepsilon)y_{\mathrm{f1}}+\varepsilon y_{\mathrm{f2}}\right)z_{\mathrm{m}} - c\left(y_{\mathrm{f2}}\varepsilon + y_{\mathrm{f1}}(1-\varepsilon)v\right)\left(z_{\mathrm{f1}}p + z_{\mathrm{f2}}(1-p)\right)\right],$$
$$\left.\frac{\mathrm{d}W}{\mathrm{d}\hat{s}_2}\right|_{\hat{s}_2=s_2} = \frac{\gamma_2^2}{2\lambda}\left[\left((1-\varepsilon)y_{\mathrm{f1}}+\varepsilon y_{\mathrm{f2}}\right)z_{\mathrm{m}} - c\left(y_{\mathrm{f2}}\varepsilon + y_{\mathrm{f1}}(1-\varepsilon)v\right)\left(z_{\mathrm{f1}}p + z_{\mathrm{f2}}(1-p)\right)\right], \tag{S6.6}$$

and similar selection gradients can be derived for both cases of offspring control. Results are more tedious in comparison to the error-free scenario, so we focus on a numerical analysis, corroborated with individual-based simulations (see main text and Figure S6.4).

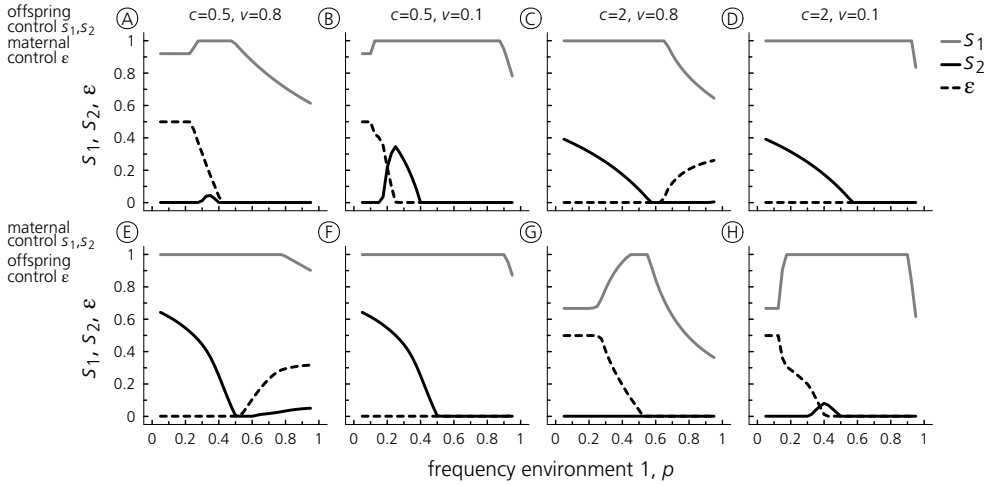

Figure S6.4: Coevolution between perception errors and sex allocation loci. Panels A-C: offspring control sex allocation, whereas mothers control perception errors. Panels D-F: mothers control sex allocation, whereas offspring are in control over perception errors (e.g., by expressing hormones that may affect maternal behaviour).

CHAPTER 7

Should attractive males have more sons?

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Abstract

It is often argued that females with attractive partners should produce more sons because these sons will inherit their father's attractiveness. Numerous field and laboratory studies have addressed this hypothesis, with inconsistent results, but there is surprisingly little theoretical work on the topic. Here, we present an extensive investigation of the link between male attractiveness and offspring sex ratios, using evolutionary, individual/based computer simulations. In situations where sexual selection leads to the stable exaggeration of a costly male trait and a costly female preference, we find that females with attractive partners produce more sons than females with unattractive partners. This same qualitative pattern is seen for a wide range of different models, with discrete or continuous variation in the male trait, under Fisherian or good-genes sexual selection and for abrupt or gradual sex ratio adjustment. However, in all simulations, it takes a huge number of generations to evolve, suggesting that selection acting on sex ratio adjustment is weak. Our models ignore many potential costs and constraints associated with manipulation, which implies that selection may be weaker still in natural populations. These results may explain why published evidence for sex ratio bias in relation to male attractiveness is mixed.

7.1 Introduction

Whether animals adaptively adjust the sex ratio (Trivers & Willard, 1973) of their offspring is currently one of the hottest topics in behavioral ecology. Sex-allocation theory has met with considerable success when applied to haplodiploid insects (Godfray & Werren, 1996), but the evidence in vertebrates with chromosomal sex determination is controversial and has attracted a number of recent, high-profile reviews and meta-analyses (e.g., Komdeur & Pen (2002); West & Sheldon (2002); Ewen *et al.* (2004); Sheldon & West (2004); Cassey *et al.* (2006)). For mammals and birds, in particular, very little is understood about the possible mechanisms of sex ratio adjustment (Krackow, 1995; Pike & Petrie, 2003), and this fact is compounded by often weak and inconsistent patterns reported in the literature (Ewen *et al.*, 2004; Sheldon & West, 2004; Cassey *et al.*, 2006).

One influential idea in this research area is that females mated to attractive males should produce more sons because these sons will inherit their father's attractiveness and enjoy high mating success, thereby yielding greater fitness returns than daughters. This verbal argument was originally developed by Burley (1981, 1986a) in her classic work on zebra finches (*Taeniopygia guttata*) and has been restated in numerous subsequent papers by others (e.g., Ellegren *et al.* (1996); Radford & Blakey (2000); Edvardsson & Arnqvist (2005); Dreiss *et al.* (2006)). It has commonly been investigated in birds, in which the males often show obvious sexually selected traits (Darwin, 1871; Andersson, 1994) and females, as the heterogametic sex, potentially have control over offspring sex (Krackow, 1995). In mammals, in contrast, many of which live in highly structured societies, patterns of sex ratio variation have been related more to maternal condition or dominance rank (Cockburn *et al.*, 2002; Sheldon & West, 2004).

Despite considerable research effort, the evidence for a link between offspring sex ratios and paternal attractiveness is mixed. For example, Sheldon *et al.* (1999) reported experimental evidence that brood sex ratios in wild blue tits (*Parus caeruleus*) were biased in response to the ultraviolet reflectance of the male's crown feathers, a known sexually selected trait. Griffith *et al.* (2003) found correlative evidence in support of this from the same population, but Dreiss *et al.* (2006) found no association between male plumage color and offspring sex ratios in a different population, and Korsten *et al.* (2006), in a faithful replication of the study of Sheldon *et al.* (1999), found an association in only one out of two years. Results from other species have been similarly variable, some studies (e.g., Pike & Petrie (2005)) finding strong support for the hypothesis, some (e.g., Parker & Winker (2005)) casting doubt on previously published evidence, and others suggesting that patterns of adjustment are not consistent across populations (Rosivall *et al.*, 2004) or years (Radford & Blakey, 2000). Even the findings of Burley (1981, 1986a) in zebra finches, which initiated this research area, have not been replicated in other studies (Zann & Runciman, 2003; Rutstein *et al.*, 2004, 2005), including one following the same experimental design (von Engelhardt *et al.*, 2004).

Given the prominence of this topic and the inconsistency in results, it is surprising that so little theoretical work exists to complement the published experiments. Several authors (e.g., Leimar (1996); Wade *et al.* (2003)) have investigated the effect of maternal condition on offspring sex ratios, revealing some interesting caveats to the hypothesis of Trivers & Willard (1973), but only one study (Pen & Weissing,

2000c) has addressed the link between offspring sex ratios and paternal attractiveness. The evolutionarily stable strategy (ESS) approach of Pen & Weissing (2000c) suggested that, when subjected to a formal theoretical analysis, the verbal reasoning that attractive males should have more sons might not be so straightforward. First, an association between male attractiveness and offspring sex ratios depends on whether the female preference is for an arbitrary "Fisherian" trait or an indicator of "good genes". Consistent sex ratio biases are expected in a good-genes model but not in a pure Fisherian model with no cost to female choice and unbiased mutations on the male trait. Second, the pattern of sex ratio adjustment seen in the good-genes model differs from that in the common verbal argument: females with attractive partners should in fact produce a roughly even sex ratio, whereas those with unattractive partners should overwhelmingly produce daughters. However, although the analysis of Pen and Weissing provided some important insights, their simplified approach considered only 2 types of males, "attractive" males and "unattractive" males. In reality, male attractiveness is likely to vary along a continuum (Andersson, 1994; Grant & Grant, 1997).

Here, we provide an extensive theoretical investigation of the link between male attractiveness and offspring sex ratios, using evolutionary, individual/based computer simulations. These simulations are complementary to the ESS analysis of Pen & Weissing (2000c) and also allow us to model complex situations that are beyond the reach of analytical techniques. Our general approach involves first simulating the evolution of a conspicuous male trait and female preference according to standard models of sexual selection then incorporating into this model the possibility for females to determine the sex of their offspring.

We present two main models, differing in the form of the male trait. Model 1 considers discrete variation, with males either possessing or lacking the trait (as in Pen & Weissing (2000c)). This is based on the 2-locus genetic model of Kirkpatrick (1982b) and the 3-locus models of Maynard Smith (1985), Kirkpatrick (1986) and Pomiankowski (1988). Model 2 considers a continuously variable male trait and is based on the quantitative genetic models of Iwasa *et al.* (1991) and Pomiankowski *et al.* (1991). For each model, we consider the two classic forms of sexual selection (Andersson, 1994): Fisherian "runaway" selection, in which males vary in attractiveness but not in heritable viability, and good-genes selection, in which variation in heritable viability is included.

7.2 Model 1: Discrete variation in the male trait

We consider a sexually reproducing population of males and females, each with five genetic loci of interest: a trait locus T (with alleles t_0 and t_1), a preference locus P (alleles p_0 and p_1), a viability locus V (alleles v_0 and v_1), and two sex ratio loci S_0 and S_1 (many possible alleles). Males with allele t_1 express a conspicuous trait, for example, elongated tail feathers, and pay an associated survival cost relative to males with allele t_0, who do not express the trait. Females with allele p_1 prefer to mate with trait-bearing males and pay a survival cost for being choosy, whereas those with allele p_0 have no preference and mate randomly. Males and females with allele v_0 have reduced premating survival compared with those with allele v_1 (for the results we present, the survival reduction was 1/6). The sex ratio loci S_0 and S_1 are expressed in females and determine the probability of producing a son when mated

to a trait-bearing male (S_1) or a traitless male (S_0). We consider a large number of alleles at these two loci, coding for any probability between 0 (exclusively daughters) and 1 (exclusively sons) in steps of 1/250.

Each mating produces a single offspring, which inherits alleles from its parents in a simple Mendelian fashion. We assume a small frequency of mutation at the sex ratio loci S_0 and S_1, causing their value to change up or down by 1/250. Mate search and reproduction continue until the number of female offspring produced is equal to the number of females in the adult population. When this point is reached, all adult individuals die and are replaced by the offspring generation (i.e., generations are nonoverlapping).

In the Fisherian version of our model, we eliminate all variation in heritable viability (i.e., at the V locus) and introduce recurrent deleterious mutations at the T locus, such that a small frequency of inherited t_1 alleles mutate into t_0. Recurrent deleterious mutations are a standard ingredient of genetic models of sexual selection (Maynard Smith, 1991) and prevent t_1 from going to fixation, thereby maintaining variability among males (Charlesworth, 1987). For direct comparison with the analysis of Pen & Weissing (2000c), we also consider a "pure" Fisherian model without such mutations. In the good-genes version of our model, we allow variation in V and assume recurrent deleterious mutations at this locus, rather than at T. In line with previous good-genes models (e.g., Maynard Smith (1985); Pomiankowski (1988); Andersson (1994)), we investigate different forms of the trait: if it is a "pure epistatic indicator", all males with allele t_1 express the trait; if it is a "conditional indicator", only those with both t_1 and v_1 express it (for further details, see Andersson, 1994). A "revealing indicator", in which all males with t_1 express the trait but only those that also have v_1 are preferred by choosy females, yielded virtually identical results to the conditional indicator, so we do not discuss this further.

Our simulations were implemented in Pascal and C++ programming languages. Copies of the code are available from the authors on request.

Results

We present the results for a population of 4000 females and a variable number of males (initially also 4000). Data are given as mean values ± standard error from 10 replicate simulation runs.

7.2.1.1 Coevolution of male trait and female preference

Male trait and female preference coevolve in the standard way predicted by analytical models of sexual selection (summarized by Maynard Smith, 1991; Andersson, 1994). Above some threshold frequency of the female preference (p_1), the trait allele t_1 spreads rapidly at first: within 50 generations, the frequency of trait-bearing males rises from 1% to over 80%. Under Fisherian sexual selection, this spread is only stable when there are recurrent deleterious mutations on the trait (frequency of t_1 after 500 generations $= 0.83 \pm 0.003$); otherwise, any cost to female choice causes both trait and preference to go to extinction. Deleterious mutations prevent t_1 from reaching a frequency where trait-bearing and traitless males are equally fit and thereby maintain the benefit of female choice. Under good-genes sexual selection, costly female choice maintains the male trait if it is a conditional indicator (t_1 fixated within 500

generations) but not if it is a pure epistatic indicator (t_1 extinguished within 500 generations). This set of results is well known to researchers working on sexual selection (Andersson, 1986, 1994; Pomiankowski, 1988; Maynard Smith, 1991).

7.2.1.2 Sex ratio adjustment

We consider the evolution of sex ratio adjustment in all cases where the male trait allele t_1 persists (pure Fisherian trait; Fisherian trait plus mutation bias; condition-dependent indicator). Change at the sex-allocation loci S_0 and S_1 is summarized in Figure 7.1. In a pure Fisherian model (no mutations at T and no cost to female choice), sex ratio adjustment does not evolve, as predicted by Pen & Weissing (2000c). Trait-bearing males have no net fitness advantage in this scenario (because their greater mating success is counterbalanced by reduced survival), so this result is not surprising. For a condition-dependent indicator or a Fisherian trait subject to recurrent deleterious mutations, however, sex ratio adjustment based on male attractiveness gradually develops (Figure 7.1, panels B and C). In a twist to the common verbal argument, females mated to trait-bearing (attractive) males do not overproduce sons; rather, those mated to traitless (unattractive) males overproduce daughters. This fits nicely with the analytical work of Pen & Weissing (2000c), who predicted that a bias in the sex ratio related to male attractiveness would lie primarily with the females with unattractive partners, not those with attractive partners. The explanation for the pattern is that the vast majority of males possess the trait, and so a strong sex ratio bias in their offspring is likely to be counteracted by selection pressures favoring the rarer sex, which tend to preserve a roughly even population sex ratio (Fisher, 1930). In contrast, sex ratio bias in the few females with traitless partners has a limited effect on the population sex ratio. These females are selected to overproduce daughters because sons will lack the trait and therefore suffer a mating disadvantage. However, this pattern of sex ratio adjustment takes tens of thousands of generations to develop. Compared with selection on the conspicuous male trait, which reaches a stable frequency within 300 generations, selection on sex ratio adjustment appears to be rather weak.

7.3 Model 2: Continuous variation in the male trait

In Model 2, the conspicuous male trait is not binary (e.g., elongated tail either present or absent) but can take a continuous range of values (e.g., precise length of tail). Heritable viability, female preference, and the traits determining sex allocation also show continuous variation. To model this in an individual-based simulation, we give each individual a "genetic value" for each trait, which can be any real number within a certain range. Offspring values are calculated as the average of the maternal and paternal values, though in a small frequency of offspring these change up or down through mutation. A mechanistic interpretation of this would be that each trait is coded by a single locus, each with an infinite number of alleles. However, we expect that the patterns we observe would be no different if each trait was in fact influenced by many different loci (i.e., polygenic), each with a small effect and interacting in an additive fashion. Our predictions can therefore be compared with those from quantitative genetic models of sexual selection (Mead & Arnold, 2004).

We based our simulations on the classic quantitative genetic models of Iwasa *et al.*

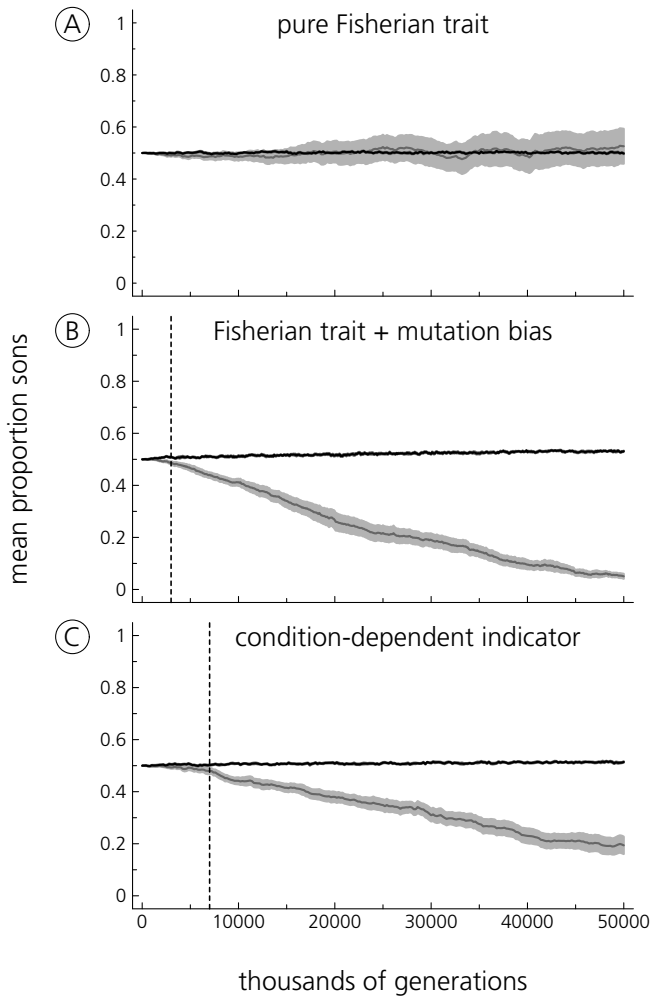

Figure 7.1: Evolution of sex ratio adjustment for three different forms of a conspicuous male trait showing discrete variation (Model 1). Plots show the average proportion of sons produced by females mated to trait-bearing males (locus S_1, black line) and traitless males (locus S_0, gray line), given as mean (solid line) $\pm$ standard error (surrounding intervals) from 10 replicate simulation runs. Vertical dotted lines indicate points beyond which the 2 sex ratios are significantly different (paired t tests, $P < 0.05$).

(1991) and Pomiankowski *et al.* (1991). As for Model 1, we present two main scenarios, one implementing Fisherian sexual selection (in accordance with Pomiankowski *et al.* (1991)) and the other implementing good-genes sexual selection (in accordance with Iwasa *et al.* (1991)). In each case, we first consider the evolution of the male trait and female preference in the absence of variation in offspring sex ratios to assess the extent to which our simulations support the predictions of the analytical models of Iwasa *et al.* (1991) and Pomiankowski *et al.* (1991). We then incorporate the possibility of sex ratio adjustment and investigate whether this becomes associated with male trait expression.

Trait values and heritable viability

We consider a sexually reproducing population of N individuals, each with genetic values t (male trait), p (female preference), and v (heritable viability). Males express the trait but not the preference, whereas females express the preference but not the trait, although both sexes carry genes for both trait and preference. Heritable viability v influences survival to maturity in both sexes and can take any value between 0 (minimum viability) and 1 (maximum viability). In the Fisherian version of our model, we eliminate variation in heritable viability by giving all individuals the same value of v and setting the mutation rate on this trait to zero. In the good-genes version, in contrast, we allow v to vary and assume a certain frequency of mutations, drawn from a negative exponential probability distribution (after Iwasa *et al.* (1991)). These mutations keep the mean value of$v$ below its maximum, maintaining variation in heritable viability between males.

Male trait and survival to maturity

The genetic value t determines a male's potential trait expression, but how this translates into realized (phenotypic) trait expression depends on the type of trait we are considering. In all cases, we use x to denote the trait phenotype, with this directly influencing survival and mating success. For a Fisherian trait and a pure epistatic indicator, $x = t$, such that all males with the same value of t express the trait to exactly the same extent (e.g., have the same tail length), regardless of any differences in heritable viability. For a conditional indicator, in contrast, both t and v determine the phenotype of the trait according to the function $x = tv$. Thus, males of higher genetic viability express the trait more strongly for a given value of t. For example, if tail length is a conditional indicator, males of higher viability will grow longer tails than males with the same potential trait value (t) but lower viability. (The same results were obtained for a revealing indicator, so we do not discuss this further.)

After Pomiankowski *et al.* (1991), we set the survival optimum for the trait phenotype at a value of $x = 0$. In terms of our example, $x = 0$ represents the tail length that optimizes flight performance, escape from predators, and so on. Male survival decreases either side of this optimum (i.e., for both longer and shorter tails) according to the function $\exp(-cx^2)$, where c is a positive constant. However, female choice (described below) may drive the male trait away from the survival optimum in either direction, leading to positive or negative trait values. The likelihood that a male will reach reproductive maturity therefore depends on both heritable viability and expression of the trait and is calculated as $v{\cdot}\exp(-cx^2)$.

Female preference and survival to maturity

A female's preference is determined by her genetic value for p. A value of $p < 0$ indicates preference for lower phenotypic trait values, whereas $p > 0$ indicates preference for higher values; a female with $p = 0$ mates at random (Pomiankowski *et al.*, 1991). Because choosiness over mates is likely to entail costs, we assume a decrease in female survival either side of $p = 0$, according to the function $\exp(-2bp^2)$ where b is a positive constant scaling the cost of choice. Female survival is also influenced by heritable viability v and is calculated as $v{\cdot}\exp(-bp^2)$.

For each mating event, a female is drawn from the population at random with

a chance proportional to her survival probability. To select a father for each of her offspring, the female randomly samples n males and chooses one of them on the basis of his trait expression. (We present the results for $n = 10$, but changing this to $n = 5$ or $n = 20$ did not affect the outcome.) The probability a given male in the sample will be chosen is weighted by the function $\exp(apx)$, where a is a positive constant scaling the importance of the male trait to female choice. Thus, if the female in question has a preference of $p < 0$, such that she favors males with below-average trait expression, those males with more negative values of x (e.g., shorter tails) will be more likely to mate. Conversely, if she favors males with above-average trait expression ($p > 0$), those males with more positive values of x (e.g., longer tails) will be more likely to mate. If the female has no preference ($p = 0$), each male in the sample has an equal chance of being selected.

Each mating produces a single offspring. Reproduction continues until the number of offspring equals the size of the adult population (N), at which point all the adults die and are replaced by the offspring generation.

Sex allocation

Building on these standard models of sexual selection, we then allow mothers to influence the sex ratio of their offspring. We explore two possibilities for this, one a "bang-bang" strategy with an abrupt switch between two sex ratios and the other permitting more gradual adjustment. In the former case, we incorporate two sex-allocation traits s_+ and s_-, which can take any real value between 0 and 1. For any given mating, offspring sex is determined by the mother's values of s_+ and s_-; the father's sex-allocation genes are assumed to have no influence. The s_+ specifies the probability of producing a son when mated to a male whose trait expression (x) is above average, whereas s_- specifies the probability of producing a son when mated to a male whose trait expression is below average. This requires that females have some way of knowing the average male phenotype $\bar{x}$, a point we will return to in the Discussion.

The alternative, more gradual, form of adjustment also involves two sex-allocation traits, this time α and β, which can take any real value (positive or negative). The probability that the offspring will be male is given by the logistic function $(1 + \exp[-(\alpha + \beta)])^{-1}$, which implies that sex allocation will be related to the father's trait phenotype provided $\beta \neq 0$. Three examples of the form of this function are depicted in Figure 7.2. Higher values of β give a steeper relationship between x and the sex ratio, with $\beta = \pm\infty$ implying an extreme type of bang-bang strategy in which the probability of a son switches suddenly between 0 and 1. The a determines the value of x for which a 50:50 sex ratio is produced.

Compared with bang-bang adjustment, the logistic function allows greater flexibility in the sex-allocation strategy and a more subtle response to the male trait. On the other hand, the former is more straightforward to analyze and allows the two sex-allocation traits to evolve independently of each other.

Mutation

For each trait (except for v in the Fisherian version of the model), we assume that mutations occur in small proportion of offspring, changing their genetic value for that trait. Changes can occur in either direction but may have a downward bias, such that

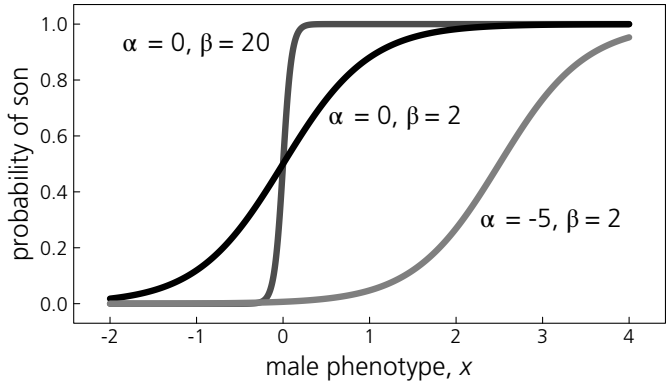

Figure 7.2: Three possible strategies of maternal sex allocation in relation to male phenotype (x) and their associated values of the sex-allocation traits α and β. The probability of producing a son is given by the logistic function $(1+\exp[-(\alpha+\beta)])^{-1}$, where the genetic values of α and β are free to evolve through mutation and selection. Negative values of β (not shown) are also possible, leading to a decrease in the probability of a son with male phenotype x. All simulations started with $\alpha = \beta = 0.0$, which gives a flat line of height 0.5 (even sex allocation, regardless of male phenotype).

mutations are more likely to reduce the genetic value than increase it. Where we have implemented a downward mutation bias, this is in line with standard models of sexual selection (Andersson, 1994) and, like the recurrent deleterious mutations in Model 1, is important for maintaining fitness differences between males (Charlesworth, 1987). Moreover, it has been argued that the fitness consequences of random mutations in a complex trait, such as viability or a conspicuous male ornament, are much more likely to be detrimental than beneficial (Pomiankowski *et al.*, 1991).

Mutations in p and t occur in proportions μ_P and μ_T of offspring, respectively, and cause the genetic value to change by an amount drawn from a uniform probability distribution (up to a certain maximum amount). For p, upward and downward changes are equally likely (i.e., mutations are unbiased), and hence their average effect in an individual offspring is zero. For t, we represent the average downward effect of mutations by the parameter u. In the Fisherian version of the model, these may be biased ($u > 0$), whereas in the good-genes model, they are always unbiased ($u = 0$). In the good- genes model, mutations also occur in v in a proportion μ_V of offspring and change the genetic value by an amount drawn from a negative exponential distribution (as in Iwasa *et al.*, 1991). These mutations have a net downward effect, with the average decrease in v represented by the parameter w ($w > 0$). In both Fisherian and good-genes models, mutations in the sex-allocation traits appear at a frequency of μ_S in the offspring and are unbiased.

7.4 Results

We present the results for a fixed population size of $N = 5000$ individuals, initially with equal numbers of males and females. Simulations were run for 50000 generations, over which we monitored the changing mean values for potential trait expression t, preference p, viability v, and the sex-allocation traits (s_+ and s_- or α and β, depending on the version of the model). The patterns were similar regardless of the initial conditions, but for the purpose of comparison, we present here the results of

simulations with starting values $t = 0$, $\bar{p} = 3$ and, for the the good-genes versions, $\bar{v} = 0.01$. All mothers in the first generation produced sons and daughters with equal probability ($\alpha = \beta = 0$ or $s_+ = s_- = 0$). Values for parameters not mentioned below were as follows: $a = 1.0$, $c = 0.5$, $\mu_P = \mu_T = \mu_V = 0.05$. For the simulations allowing sex ratio adjustment, we changed the mutation rate in the sex-allocation traits from $\mu_S = 0$ to $\mu_S = 0.05$. Despite small fluctuations, the population sex ratio never became more skewed than 42.4% males (2881 females and 2119 males) or 56.3% males (2187 females and 2813 males).

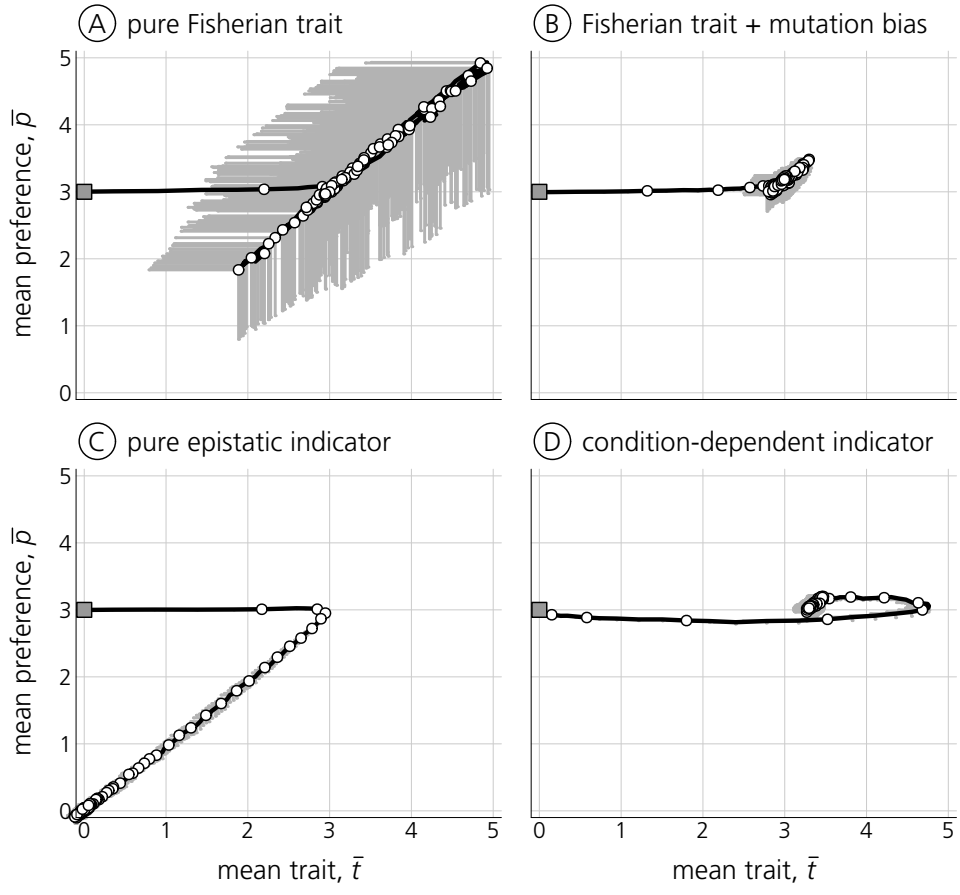

Figure 7.3: Coevolution of a female preference and a conspicuous male trait showing continuous variation (Model 2) for four different forms of the male trait. Plots show population mean values for the female preference ($\bar{p}$) and male trait ($\bar{t}$) over 5000 generations of selection, with sex ratio adjustment able to evolve ($\mu_S = 0.05$). The starting point of the simulations is indicated by a gray square; open circles mark intervals of 50 generations. Data are given as mean (solid line) ± standard error (stippling) from 10 replicate simulation runs. Parameter values are given in the main text.

Coevolution of male trait and female preference The simulation results fit well with the analytical predictions of the quantitative genetic approach of Iwasa *et al.* (1991) and Pomiankowski *et al.* (1991). In a pure Fisherian model with no cost to female choice ($b = 0$) and no mutation bias on the male trait ($u = 0$), trait and preference values evolve toward a line of equilibria where the degree of trait exaggeration balances the associated survival cost with the mating advantage (Figure 7.3, panel A). The equilibria on this line are unstable: incorporating even a small cost of choice ($b = 0.001$) drives trait and preference to their survival optima (not shown). A downward mutation bias ($u = 0.008$) rescues this process and allows stable exaggeration of the trait, this time with a single equilibrium point rather than a line of equilibria (Figure 7.3, panel B).

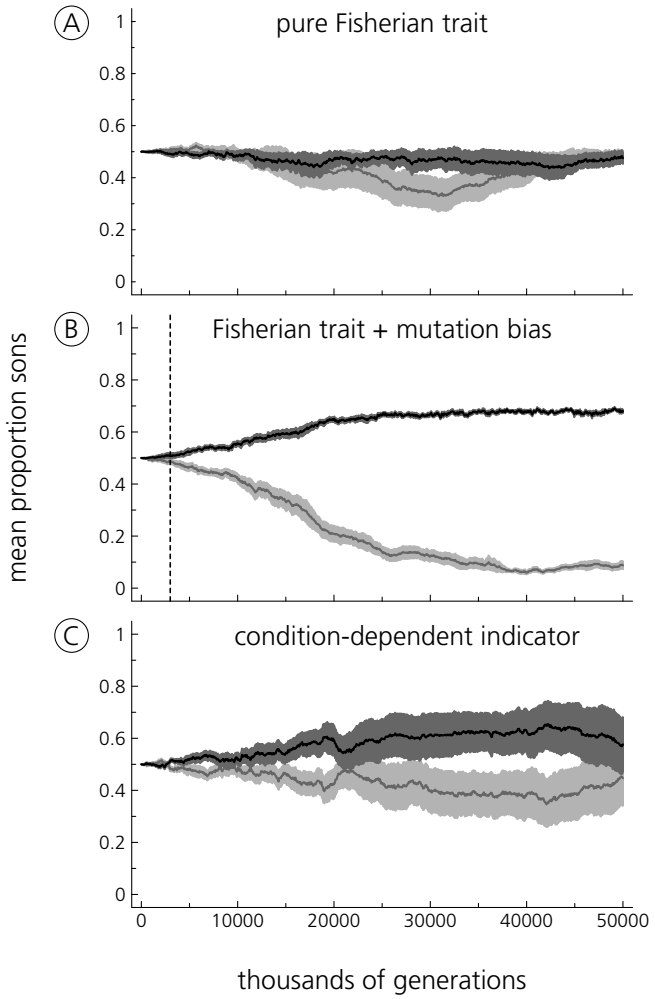

Figure 7.4: Evolution of sex ratio adjustment for three different forms of a conspicuous male trait showing continuous variation (Model 2). Plots show the average proportion of sons produced by females mated to males with above-average (s_+, black line) and below-average (s_-, gray line) trait expression, given as mean (solid line) ± standard error (stippling) from 10 replicate simulation runs. Vertical dotted lines indicate points beyond which the two sex ratios are significantly different (paired t tests, $P < 0.05$). Parameter values are given in the main text.

In the good-genes model, stable exaggeration of a costly male trait and costly female preference ($b = 0.0025$) is only possible if there is a downward mutation pressure on heritable viability ($w = 0.02$). This keeps $\bar{v}$ just below its maximum value, thereby ensuring that a female who chooses at random will, on average, obtain a mate of lower viability than one who chooses on the basis of a sufficiently reliable indicator trait. Even with this effect of mutation, stable exaggeration will occur only for a conditional indicator, not for a pure epistatic indicator (Figure 7.3, compare panels C and D). For this reason, we do not consider sex ratio adjustment for a pure epistatic indicator.

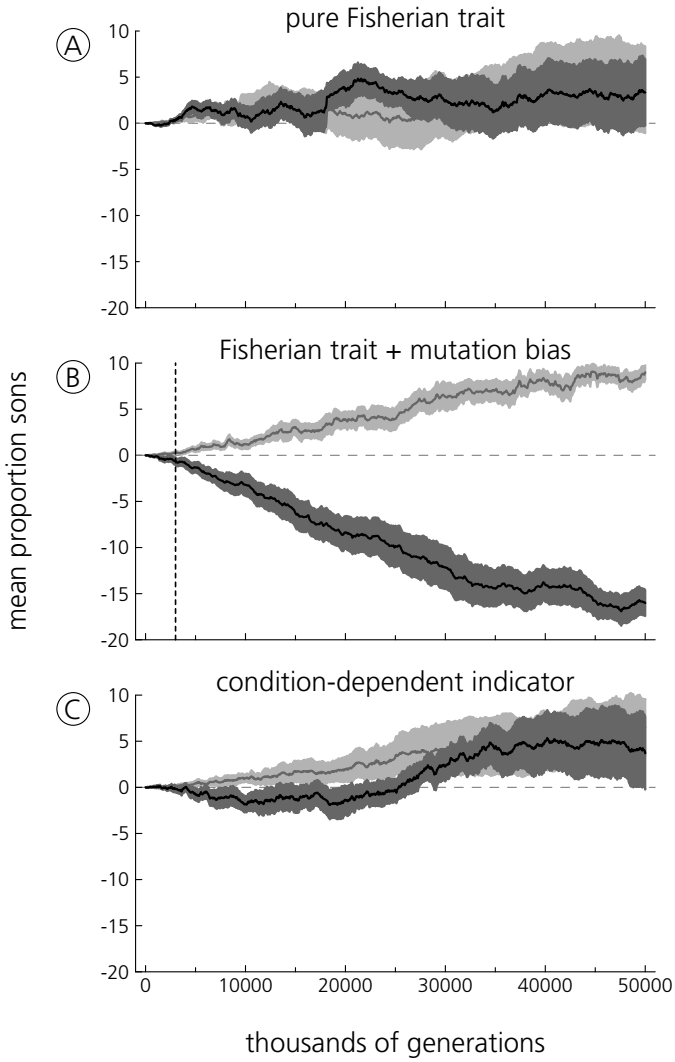

Figure 7.5: Evolution of sex ratio adjustment for three different forms of a conspicuous male trait showing continuous variation (Model 2). Plots show population mean values for sex-allocation traits α (black line) and β (gray line), given as mean (solid line) $\pm$ standard error (stippling) from 10 replicate simulation runs. Vertical dotted lines indicate points beyond which the trait value is significantly different from zero (1-sample t tests, $P < 0.05$). Parameter values are given in the main text.

Bang-bang sex ratio adjustment (traits s_+ and s_-) As for the discrete-trait model, evolution of sex ratio adjustment proceeds slowly. For a pure Fisherian model, females whose partners have below-average trait expression initially develop a slight (non significant) bias toward daughters, but this pattern subsequently disappears (Figure 7.4, panel A). With costly choice and a mutation bias on the male trait, however, a clear pattern emerges: females mated to attractive males overproduce sons, whereas those mated to unattractive males overproduce daughters (Figure 7.4, panel B). These sex ratio biases take 40000 generations to reach their full extent. When the male trait is a conditional indicator of good genes, the sex ratio biases that evolve are slight and show some inconsistency between simulation runs. Females mated to attractive partners (above-average x) produce slightly more sons than those mated to unattractive partners (below-average x), but this difference is not significant (Figure 7.4, panel C).

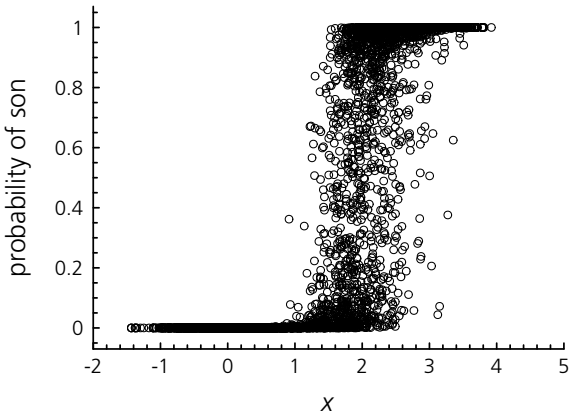

Figure 7.6: An example of the relationship between male attractiveness and offspring sex ratios, for a continuously variable, Fisherian male trait with biased mutations (Model 2) and a gradual form of sex ratio adjustment (traits a and b, explained in main text). The male's trait phenotype (x) and the probability of producing a son are shown for all mated pairs in the 50 000th generation of one simulation run. Each point represents a separate mating. Parameter values are given in the main text.

Gradual sex ratio adjustment (traits α and β) Similar results emerge for the alternative form of sex ratio adjustment. The sex-allocation traits α and β show significant divergence from their starting values of 0 only in the case of a Fisherian trait with costly choice and biased mutations, with α gradually decreasing and β gradually increasing (Figure 7.5, panel B). The positive value of β indicates that, on average, more sons are produced for higher values of the father's trait phenotype x. The precise pattern of sex ratio adjustment differs slightly between simulation runs; the data from one simulation are shown as an example in Figure 7.6. Here, contrary to the results from Model 1, offspring sex ratios are biased to a similar extent by females with attractive and unattractive partners. Sex ratio adjustment for a conditional indicator is less clear and shows high variability between simulation runs (Figure 7.5, panel C; note the large standard errors). Attractive males do have slightly more sons on average: considering only those matings involving the top 10% and bottom 10% of male trait phenotypes (x), the proportion of sons in the 50000th generation is, re-

spectively, 0.68 ± 0.082 and 0.32 ± 0.091. However, the change in the sex-allocation traits is not significant. Selection on these traits is evidently quite weak.

7.5 Discussion

Our simulations confirm that sex ratio adjustment based on male attractiveness can evolve, such that females with attractive partners produce more sons than those with unattractive partners. However, it appears that the selection pressures responsible for this pattern are rather weak. This suggests that in many animal populations, we may be unlikely to detect any clear relationship between male attractiveness and the sex ratio.

Pattern of sex ratio adjustment

As predicted by quantitative genetic models (e.g., Iwasa *et al.* (1991); Pomiankowski *et al.* (1991)), sexual selection can only maintain a costly male trait and a costly female preference in an exaggerated state, away from their survival optima, when the male trait is a Fisherian trait under mutation bias or a conditional (or revealing) indicator. In both of these scenarios, the same qualitative pattern of sex ratio adjustment subsequently evolves: females with attractive mates produce more sons than those with unattractive mates. This fits with widespread verbal arguments and makes good sense because sons inherit their father's attractiveness. The fact that the same pattern appears for both discrete and continuous variation in the male trait, in both Fisherian and good-genes models, and for both abrupt (bang-bang) and gradual sex ratio adjustment suggests that this result is widely applicable.

The quantitative pattern of sex allocation, however, depends on the type of model we use. For discrete variation (Model 1), where males either express the trait to its full extent or do not express it at all, the major sex ratio bias is seen in the offspring of unattractive males. Females with attractive, trait-bearing partners hardly deviate from a 50:50 sex ratio, whereas those with unattractive, traitless partners greatly overproduce daughters. This was predicted from an earlier model of evolutionarily stable sex-allocation strategies by Pen and Weissing (2000). Though at first sight it seems unrealistic to model the male phenotype as having only two possible states, some conspicuous traits may in fact fit this caricature well. Some striking plumage features in birds are governed by a small number of genes (Grant & Grant (1997); Theron *et al.* (2001)), and work on *Drosophila* has shown that genetic differences at a single locus can dramatically affect male attractiveness (e.g., Ringo *et al.* (1992); Singh & Sisodia (1999)). It is possible, then, that a single mutation could create a novel male trait, dividing the male population into two distinct types: those that possess the novel trait and those that lack it. If females are more attracted to the novel phenotype, the trait will spread through sexual selection to most of the males. According to the results of our simulations, there should then be a selection pressure for females to overproduce daughters if they happen to end up with an unattractive, traitless partner.

Although this simple scenario of all-or-nothing expression may apply in certain cases, most sexually selected traits are likely to show a continuous range of expressed values (Andersson (1994); Grant & Grant (1997)). In such cases, encapsulated by Model 2, we predict that offspring sex ratios should be biased not only by females with unattractive partners, who have more daughters, but also by those with attractive

partners, who have more sons. Thus, the conclusion of Pen & Weissing (2000c) that sex ratio biases should be largely restricted to females with unattractive partners seems to be a consequence of considering only two male types.

The grouping of males into two types also explains why, under the bang-bang form of adjustment (traits s_+ and s_-) in the Fisherian version of Model 2, the sex ratio bias for unattractive males is stronger than that for attractive males (Figure 7.4, panel B). Due to their greater attractiveness, males with above-average trait expression are responsible for most of the matings, and the sex ratio bias is therefore tempered strongly by selection pressures favoring the rarer sex. Males with below-average expression are less well represented in the mating population, and so stronger sex ratio biases are possible. By categorizing males into two types according to their trait expression, the s_1 and s_+ sex-allocation traits bear some similarity to the S_1 and S_0 loci of Model 1. For the gradual form of adjustment (traits α and β), in contrast, the degree of bias for females mated to the most attractive and the least attractive males is very similar (Figure 7.6). In answer to the question posed in the title of this paper, attractive males should indeed have more sons, just as unattractive males should have more daughters.

The clearest pattern of sex ratio adjustment was seen for a Fisherian trait subject to biased mutations (panel B of Figures 7.1, 7.4, and 7.5), in which males varied in heritable attractiveness but not in heritable viability. In this situation, only sons inherit paternal attractiveness, which is why females mated to highly attractive partners are selected to produce a more male-biased offspring sex ratio than those with unattractive partners. In the good-genes version (condition-dependent indicator), however, daughters as well as sons profit from having a father who is attractive because his attractiveness indicates high heritable viability for both sexes. It is perhaps not surprising, then, that under this scenario, sex ratio biases were less marked and showed inconsistency between simulation runs. In line with Burley (1986a), we predict that strong biases are most likely to be seen in species where the heritable benefits of mating with an attractive male are largely sex limited.

Strength of selection on sex ratio adjustment

Although our results confirm that sex ratio adjustment with respect to male attractiveness can evolve, a consistent feature of our simulations is that this process takes a great deal of time. Only very slight sex ratio biases were seen after 5000 generations, and for all models, it took at least 40000 generations for the full extent of the bias to emerge. For the conditional handicap in Model 2, the offspring sex ratios for attractive and unattractive males were barely different from 0.5 even after 50000 generations of evolution.

It is important to point out here that the "generations" in our simulations have no direct correspondence to the generations of a real organism. To make such a link, we would need to know the precise details of the genetic system controlling the traits involved and have reliable estimates of the frequency and effects of mutations in these traits. Furthermore, our simplifying assumption of nonoverlapping generations does not hold for many real populations. For these reasons, we cannot say whether a pattern found after 50000 generations of our simulation would appear more quickly or more slowly in a natural system.

Nonetheless, by comparing the evolution of sex ratio adjustment with that of the

male trait and female preference, it is clear that selection on sex ratio adjustment is far weaker. The male trait reaches an exaggerated state within 500 generations, and often much sooner than that, whereas the sex-allocation traits show only slight changes after several thousand generations. This is not due to insufficient variation; new mutations occur just as often in the sex-allocation traits as in the male trait and female preference, and even with a mutation rate 4 times higher ($\mu_s = 0.2$), there is still very little sex ratio bias after 5000 generations (unpublished results). Overall, the slow rate of evolution implies that selection on sex ratio adjustment with respect to male attractiveness is weak. This probably reflects that, in general, selection on the variable part of a conditional trait (e.g., how the sex ratio varies with attractiveness) is weak compared with selection on the mean value of that trait (e.g., the mean sex ratio) or on a non-conditional trait. We should therefore not be surprised if, in natural populations, attractive male traits show little relationship with offspring sex ratio, particularly if those traits have evolved relatively recently.

Constraints on sex ratio adjustment

To investigate the adaptive basis of sex ratio adjustment, we omitted from our models many possible factors that might constrain the evolution of such strategies in real animals. First, through the evolution of the sex-allocation traits, we allowed females to have full control over the sex of their offspring, free from any mechanistic constraints or costs. In animals with chromosomal sex determination, it may be that Mendelian segregation of the sex chromosomes is not amenable to manipulation by parents (Williams (1979); Krackow (2002); though, for an alternative viewpoint, see West & Sheldon (2002) and West *et al.* (2005)). Manipulation may be possible after meiosis, for example, in birds through the selective resorption, selective ovulation, or selective fertilization of Z- and W-bearing ova (Pike & Petrie, 2003), but this is likely to entail some energetic costs, which were not included in our models. Costs of sex ratio control are expected to weaken any bias (Pen *et al.*, 1999). In general, very little is known about how sex ratio biases might be achieved, and in the absence of clear evidence for a suitable mechanism, we must consider the possibility that such manipulation is beyond maternal control or too costly to be worthwhile.

Second, we assumed that females had perfect information about a male's genetic quality (whether in terms of heritable attractiveness or heritable viability). In real systems, however, there will always be some "noise" associated with both signaling and receiving; males will not advertise their quality perfectly accurately, and females will not perceive male phenotypes perfectly accurately. This error means that females will be uncertain about the true quality of their mate and therefore might be expected to hedge their bets somewhat by reducing the degree of any sex ratio bias (Charnov *et al.* (1981); but see Pen I, Lessells CM, Weissing FJ, and Colegrave N, in preparation). The upshot is that sex ratios might be less strongly biased than those predicted by our models. In support of this, uncertainty regarding the consequences of sex ratio adjustment seems to have such an effect in parasitic wasps (West & Sheldon, 2002).

For the bang-bang form of sex allocation, we made a further assumption about the information available to females, namely, that they were aware of the average trait phenotype for all males in the population. We feel it is not unreasonable to assume that a female would have some estimate of this average through her mate-sampling behavior and encounters with males in other situations, but it is highly unlikely that

she would know the true average value. For the same reasons discussed above, the error in her estimate might reduce the extent of sex ratio bias.

The aim of our models was to determine whether it makes sense, adaptively, for mothers to manipulate the sex of their offspring in relation to their partner's attractiveness. We have shown that there is indeed an adaptive reason for such manipulation when it is free from constraints or costs but that even in this unrealistically conducive situation it appears to be weak. What our models do not show is that this kind of manipulation will necessarily take place in nature. With constraints and costs acting against sex ratio adjustment, we expect patterns of bias to be even weaker than those found in our simulations.

Advantages of the simulation approach

The individual-based simulations we used in this study, though lacking some of the insight of analytical methods, have some important strengths. First, they clearly illustrate the dynamics of selection in a system where stochastic processes such as mutation and genetic drift are part and parcel of the evolutionary process. Second, in contrast to most quantitative genetic models, we do not need to assume particular values for the variances and covariances of the traits of interest but rather allow these to develop in response to selection. Third, and for the present study most crucially, it is sometimes possible to build simulation models for situations where an analytical approach is difficult and perhaps unfeasible. Taking the quantitative genetic model of Pomiankowski *et al.* (1991) as an example, it is not obvious how one would calculate the selection gradient for a conditional sex-allocation strategy in this framework. It is also unclear what kind of values, or even their signs, would be realistic for the genetic variances and covariances between the sex-allocation traits and the male trait and female preference. Finally, the quantitative genetic approach focuses on mean values, whereas for our research question, it is the variance in traits that is of central importance. A simulation study avoids all these problems by directly monitoring the genetic values for each individual in the population.

Possible extensions

Our models can easily be modified to take account of additional complications that might be present in some biological systems. For example, evidence suggests that many conspicuous male traits and female preferences in birds and butterflies are coded by genes located on the sex chromosomes and therefore show sex-linked inheritance (Reeve & Pfennig, 2003; Kirkpatrick & Hall, 2004). The models presented above assume autosomal inheritance of trait and preference, but we are currently investigating how patterns of sex ratio bias are affected by different kinds of sex linkage. Traits with sex-linked inheritance may often show sex-limited expression, in which case we would expect stronger selection for sex ratio adjustment. Similarly, Fisherian sexual selection is most pronounced when female preferences are Z-linked (Kirkpatrick & Hall, 2004), and so we might expect to see greater sex ratio biases under this form of inheritance.

Another interesting complication is that some genes may have sexually antagonistic effects, in that they increase the fitness of one sex but decrease the fitness of the other (Chippindale *et al.*, 2001). In the simulations presented above, the genes coding for male trait expression may enhance the direct fitness of sons (via increased

attractiveness), but they are neutral with respect to the direct fitness of daughters (who do not express the trait). We could alter this situation by reducing the survival of daughters carrying genes for strong trait expression. This would magnify the difference in fitness returns from sons and daughters for females mated to highly ornamented males and is therefore likely to strengthen the degree of sex ratio bias.

Concluding remarks

Overall, we can confirm the intuition that attractive males should have more sons, but we argue that selection on this is weak and may be counteracted by constraints and costs of manipulation. Viewed in this light, the mixed evidence for adaptive sex allocation in birds (Clutton-Brock & Iason, 1986; Sheldon, 1998; Komdeur & Pen, 2002; Krackow, 2002; West & Sheldon, 2002; West *et al.*, 2002; Ewen *et al.*, 2004; Cassey *et al.*, 2006) is not so surprising.

7.6 Acknowledgements

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CHAPTER 8

Sex-ratio control erodes sexual selection, revealing a feedback from adaptive plasticity

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[1] TWF and BK contributed equally to this work

Abstract

Female choice is a powerful selective force, driving the elaboration of conspicuous male ornaments. This process of sexual selection has profound implications for many life-history decisions, including sex allocation. For example, females with attractive partners should produce more sons, because these sons will inherit their father's attractiveness and enjoy high mating success, thereby yielding greater fitness returns than daughters. However, previous research has overlooked the fact that there is a reciprocal feedback from life-history strategies to sexual selection. Here, using a simple mathematical model, we show that if mothers adaptively control offspring sex in relation to their partner's attractiveness, sexual selection is weakened and male ornamentation declines. This weakening occurs because the ability to determine offspring sex reduces the fitness difference between females with attractive and unattractive partners. We use individual-based, evolutionary simulations to show that this result holds under more biologically realistic conditions. Sexual selection and sex allocation thus interact in a dynamic fashion: The evolution of conspicuous male ornaments favors sex-ratio adjustment, but this conditional strategy then undermines the very same process that generated it, eroding sexual selection. We predict that, all else being equal, the most elaborate sexual displays should be seen in species with little or no control over offspring sex. The feedback process we have described points to a more general evolutionary principle, in which a conditional strategy weakens directional selection on another trait by reducing fitness differences.

8.1 Introduction

Conspicuous male ornaments such as brightly colored or elongated feathers, loud vocalizations and complex courtship dances are the hallmark of sexual selection, maintained despite their obvious costs because females find them attractive (Andersson, 1994). This has profound implications for many life-history decisions, including which sex of offspring to produce and how to invest in them (Trivers & Willard, 1973; Burley, 1981; West, 2009). Provided the heritable benefits of ornamentation are to some degree sex-limited, selection favors a conditional strategy of sex allocation: females mated to attractive, highly ornamented males should overproduce sons, whereas those mated to unattractive males should overproduce daughters (Trivers & Willard, 1973). This pattern of sex allocation has been supported by a number of theoretical (Pen & Weissing, 2000c; Blackburn *et al.*, 2010, Chapter 7) and empirical (Ellegren *et al.*, 1996; Sheldon *et al.*, 1999; Pike & Petrie, 2005) studies, but no one has considered how it might feed back to alter sexual selection. Here we investigate the dynamic interplay between sexual selection and sex-ratio adjustment. We first develop a simple mathematical model in which male ornamentation, female preference and the sex-allocation strategy can coevolve, and use this to determine the direction of selection acting on all of these traits. We then extend our analysis to more biologically realistic conditions using a series of individual-based, evolutionary simulations, incorporating continuous variation in ornamentation and preference, a finite population size and stochastic factors such as genetic drift. This dual approach allows us to uncover the evolutionary forces linking sexual selection and sex allocation. After analysing this coevolutionary feedback process in depth, we show how the same principle extends to a wide range of other contexts in which selection favors phenotypic plasticity in response to a directionally selected trait.

8.2 Model

Basic Scenario For the sake of tractability we consider just two types of males, which differ in their ornamentation (Pen & Weissing, 2000c; Kirkpatrick, 1982b; Kokko *et al.*, 2002): those of type 0 lack ornamentation, while those of type 1 are ornamented to a degree given by the evolvable trait t ($t > 0$). Ornamentation is costly in that it reduces survival to adulthood, with the relative survival of type-1 (compared to type-0) males given by ν_{m1} ($\nu_{\mathrm{m1}} \leq 1$). This reflects the energy or resources invested in the development of secondary sexual traits, or an associated predation risk of being conspicuous (Andersson, 1994).

Females are of one type only and have an evolvable preference p ($p \geq 0$), which is costly and lowers their survival to ν_{f} ($\nu_{\mathrm{f}} \leq 1$). This cost may arise because the female has to invest in sensory apparatus for assessing males, or because she incurs a higher predation risk while choosing a mate (Pomiankowski, 1987a). Her preference makes her more inclined to mate with an ornamented than a non-ornamented male, resulting in a proportion α of females that mate with the former type (note that α is not fixed but depends on p, t and the relative frequencies of type-0 and type-1 males). Consequently, the expected number of mates is q_1 for an ornamented male and q_0 for a non-ornamented male, with $q_1 \geq q_0$.

Crucially, females can adjust offspring sex ratios in relation to their partner's ornamentation: sex allocation is determined by the evolvable traits s_0 and s_1, where s_0 is the proportion of sons produced when mated to a non-ornamented male and s_1 the

proportion when mated to an ornamented male. Ornamentation is heritable from father to son, except when mutations occur: with probability μ_0 the son of a non-ornamented male is ornamented and with probability μ_1 the son of an ornamented male is non-ornamented. In common with standard models of sexual selection (Bulmer, 1989; Pomiankowski *et al.*, 1991) we assume that mutations are biased towards the loss of ornamentation, i.e., that $\mu_1 > \mu_0$. This prevents fixation of the male ornament and thereby preserves the benefit of female choice (Pomiankowski *et al.*, 1991). Individuals are assumed to die before their offspring become reproductively mature, so that generations are non-overlapping. Figure 8.1 summarizes the sequence of events in our model. Table 1 lists the variables and parameters with their associated symbols. Table 8.1 lists the variables and parameters with their associated symbols.

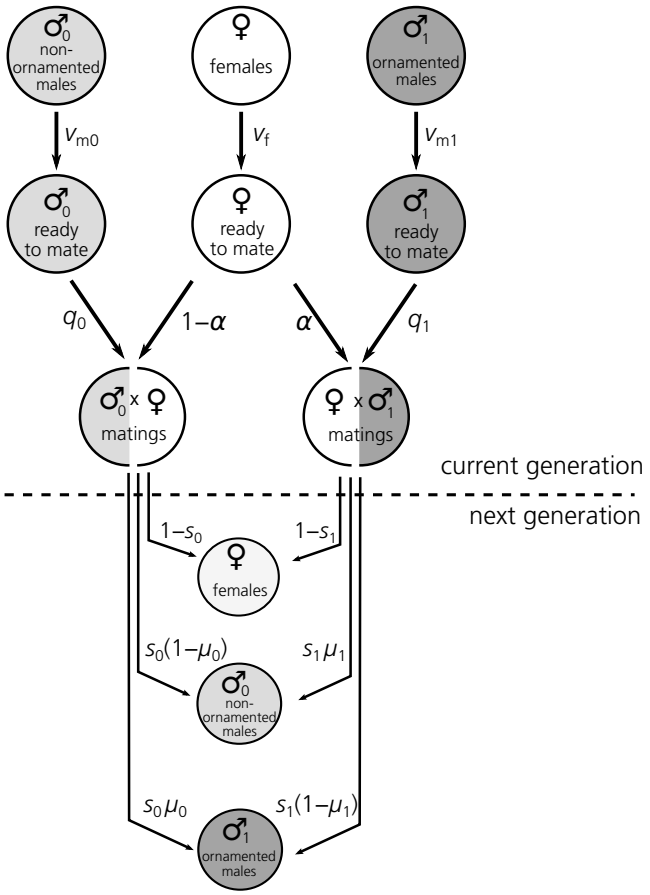

Figure 8.1: Summary of the sequence of events in each generation of our model. The survival of females (ν_f) and ornamented males (ν_{m1}) to reproduction is reduced by the cost of their preference and ornament, respectively (non-ornamented males do not pay a cost; $\nu_{m0} = 0$). Reproducing females give a proportion α of their matings to ornamented males, resulting in an average number of mates q_1 for ornamented males compared to q_0 mates for non-ornamented males ($q_1 \geq q_0$). For each offspring produced, the probability that it is a son is s_0 for females with non-ornamented partners and s_1 for those with ornamented partners. With mutation probability μ_0 the son of a non-ornamented male is ornamented, while with mutation probability μ_1 the son of an ornamented male is non-ornamented ($\mu_1 > \mu_0$, i.e., mutations are biased towards the loss of ornamentation).

Table 8.1: Variables and parameters used in the model.

Symbol	Meaning
y_{f}	Relative frequency of females
$y_{\mathrm{m}i}$	Relative frequency of type-i males ($i = 0, 1$)
z_{f}	Class-specific individual reproductive value of females
$z_{\mathrm{m}i}$	Class-specific individual reproductive value of type-i males
v_{f}	Viability of females
$v_{\mathrm{m}i}$	Viability of type-i males
α	Probability that a female mates with a type-1 male
q_i	Average number of mates per type-i male
s_i	Proportion of sons produced when mated to a type-i male
μ_i	Probability that a son of a type-i male mutates into the alternative type

Overview of the Method Given this set-up, we can use a reproductive value approach (Taylor, 1996b; Pen & Weissing, 2000a,b) to obtain selection differentials for male ornamentation, female preference and conditional sex allocation, which together describe how the system evolves. This involves three basic steps: (i) determine the dynamics of a resident population with trait value x for a given trait of interest; (ii) determine the invasion fitness of a rare mutant with alternative trait value $\hat{x}$ within this resident population; (iii) determine how mutant fitness w depends on x and $\hat{x}$. Below we outline these steps in detail.

Dynamics of the Resident Population We first consider the dynamics of a resident population with ornamentation level t, preference p and sex-allocation traits s_0 and s_1. The numbers of females, type-0 males and type-1 males change from one generation to the next according to the transition matrix

$$\mathbf{A} = \frac{1}{2}k \begin{bmatrix} \left[(1-\alpha)(1-s_0)+\alpha(1-s_1)\right] v_{\mathrm{f}} & q_0(1-s_0)v_{\mathrm{f}} & q_1(1-s_1)v_{\mathrm{f}} \\ \left[(1-\alpha)s_0(1-\mu_0)+\alpha s_1\mu_1\right] v_{\mathrm{m0}} & q_0 s_0(1-\mu_0)v_{\mathrm{m0}} & q_1 s_1\mu_1 v_{\mathrm{m0}} \\ \left[(1-\alpha)s_0\mu_0+\alpha s_1(1-\mu_1)\right] v_{\mathrm{m1}} & q_0 s_0\mu_0 v_{\mathrm{m1}} & q_1 s_1(1-\mu_1)v_{\mathrm{m1}} \end{bmatrix}. \tag{8.1}$$

The factor $1/2$ is a formality to prevent offspring being counted twice (once via its mother and once via its father), while the constant k is a scaling factor (equivalent to the average clutch size) to ensure that the population is stable (in technical terms, to ensure that the dominant eigenvalue is 1; see Mylius & Diekmann, 1995; Pen & Weissing, 2000b). The leftmost column of $\mathbf{A}$ represents the per-capita reproductive output of females, the middle column that of type-0 males and the rightmost column that of type-1 males. The three rows represent, from top to bottom, the result of this reproductive output in terms of surviving females, type-0 males and type-1 males in the next generation.

The entries in the matrix are derived from the basic assumptions of our model. To give an example, take the leftmost entry in the middle row, which represents the reproductive contribution of mothers to type-0 males in the next adult generation. There are two scenarios in which a female gives birth to a type-0 son: either she

mates with a type-0 male (probability $1-\alpha$) and produces a son (probability s_0) who is unaffected by mutation (probability $1-\mu_0$), or she mates with a type-1 male (probability α) and produces a son (probability s_1) who mutates to a non-ornamented state (probability μ_1). In either case, the survival of that son to reproductive age is v_{m0}. The other entries in matrix $\mathbf{A}$ are derived using similar logic, detailed in the supporting information (SI) at the end of this chapter.

The relative frequencies of females (y_{f}), type-0 males (y_{m0}) and type-1 males (y_{m1}) change from one generation to the next according to the dynamic equation $\mathbf{y}_{t+1}=\mathbf{A}\mathbf{y}_t$, where $\mathbf{y}$ is the column vector $(y_{\mathrm{f}}, y_{\mathrm{m0}}, y_{\mathrm{m1}})^{\mathrm{T}}$ representing the relative frequencies in the current generation ($\mathbf{y}_t$) and the next generation ($\mathbf{y}_{t+1}$), respectively (note that the superscript T indicates transposition). Explicit equations for y_{f} and y_{m1} are given in the Supplement.

Invasion Fitness of a Rare Mutant Now we ask whether rare mutants with different values for the traits of interest can invade the resident population. The dynamics of mutants are governed by a matrix similar to $\mathbf{A}$, with the appropriate parameters replaced by their mutant counterparts:

$$\mathbf{B}=\frac{1}{2}k\begin{bmatrix} \left[(1-\hat{\alpha})(1-\hat{s}_0)+\hat{\alpha}(1-\hat{s}_1)\right]\hat{v}_{\mathrm{f}} & q_0(1-s_0)\hat{v}_{\mathrm{f}} & \hat{q}_1(1-s_1)\hat{v}_{\mathrm{f}} \\ \left[(1-\hat{\alpha})\hat{s}_0(1-\mu_0)+\hat{\alpha}\hat{s}_1\mu_1\right]v_{\mathrm{m0}} & q_0s_0(1-\mu_0)v_{\mathrm{m0}} & \hat{q}_1s_1\mu_1v_{\mathrm{m0}} \\ \left[(1-\hat{\alpha})\hat{s}_0\mu_0+\hat{\alpha}\hat{s}_1(1-\mu_1)\right]\hat{v}_{\mathrm{m1}} & q_0s_0\mu_0\hat{v}_{\mathrm{m1}} & \hat{q}_1s_1(1-\mu_1)\hat{v}_{\mathrm{m1}} \end{bmatrix}. \tag{8.2}$$

Mutant phenotypes are equipped with a hat ($\wedge$) to distinguish them from resident phenotypes. Note that the probability $\hat{\alpha}$ that a mutant female mates with a type-1 male is distinct from the corresponding resident probability α, because it is determined by the female's mutated preference $\hat{p}$. Her viability $\hat{v}_{\mathrm{f}}$ also depends on $\hat{p}$, while that of mutant males $\hat{v}_{\mathrm{m1}}$ depends on their mutant level of ornamentation $\hat{t}$. In contrast, since type-0 males lack ornamentation altogether, their viability v_{m0} and per-capita number of mates q_0 are the same as for resident type-0 males and so are left without a hat. The sex-allocation traits s_0 and s_1 receive hats in the first column, representing the reproductive output of mutant females, but not in the second and third columns since mutant males are assumed to mate with resident females only (due to the rarity of mutant females). Note that when the mutant's trait values are the same as those of the resident ($\hat{x}=x$), matrices $\mathbf{A}$ and $\mathbf{B}$ are identical.

8.3 Results

Analytical results The ability of mutant individuals to invade the resident population is given by their fitness w, which is the dominant eigenvalue of matrix $\mathbf{B}$. Assuming mutations of small effect, the selection differential $\partial W/\partial\hat{x}$ expresses how w depends on $\hat{x}$, the mutant value for the trait of interest. According to a standard result (Otto & Day, 2007) from evolutionary invasion analysis, this is

$$\frac{\partial w}{\partial\hat{x}}=\mathbf{z}^{\mathrm{T}}\frac{\partial\mathbf{B}}{\partial\hat{x}}\mathbf{y}/\mathbf{z}^{\mathrm{T}}\mathbf{y}, \tag{8.3}$$

where $\mathbf{y}$ represents the relative frequencies of females, type-0 males and type-1 males in the resident population (technically, a dominant right eigenvector of $\mathbf{A}$), $\mathbf{z}=$

$(z_f, z_{m0}, z_{m1})^T$ represents their reproductive values (technically, a dominant left eigenvector of **A**) and the derivatives are evaluated at the resident trait values. Using the approach (Pen & Weissing, 2000c; Kokko *et al.*, 2002) outlined in the SI, we can use eq. (8.3) to obtain the following selection differentials (Taylor, 1996b) for the traits p, t, s_0 and s_1, evaluated at the resident trait values (i.e., where $\hat{p} = p$, $\hat{t} = t$, $\hat{s}_0 = s_0$ and $\hat{s}_1 = s_1$):

$$\frac{\partial w}{\partial \hat{p}} = \alpha' \left[\frac{z_{m1}}{q_1} - \frac{z_{m0}}{q_0} \right] y_f + \frac{v_f'}{v_f} z_f y_f, \tag{8.4}$$

$$\frac{\partial w}{\partial \hat{t}} = \frac{q_1'}{q_1} z_{m1} y_{m1} + \frac{v_{m1}'}{v_{m1}} z_{m1} y_{m1}, \tag{8.5}$$

$$\frac{\partial w}{\partial \hat{s}_0} = \frac{1-\alpha}{s_0} \left[\frac{z_{m0}}{q_0} - \frac{1}{2(1-\bar{s})} \right] y_f, \tag{8.6}$$

$$\frac{\partial w}{\partial \hat{s}_1} = \frac{\alpha}{s_1} \left[\frac{z_{m1}}{q_1} - \frac{1}{2(1-\bar{s})} \right] y_f, \tag{8.7}$$

where $\bar{s} = (1-\alpha)s_0 + \alpha s_1$ is the average offspring sex ratio. Primes ($'$) denote differentiation with respect to the trait under consideration.

At the equilibrium for the sex-allocation traits s_0 and s_1, the selection differentials given by eq. (8.6) and eq. (8.7) must be zero (Otto & Day, 2007), and $z_{m0}/q_0 = 1/[2(1-\bar{s})]$ and $z_{m1}/q_1 = 1/[2(1-\bar{s})]$. Thus we have $z_{m0}/q_0 = z_{m1}/q_1$, which implies that the first term on the right of eq. (8.4) vanishes as well. Assuming that $v_f' = \mathrm{d}v_f/\mathrm{d}p$ is negative, i.e., that female choice is costly (Pomiankowski, 1987a), it follows that the selection differential for p is negative. Hence, at the sex-allocation equilibrium, selection cannot sustain a costly female preference. Sex-ratio adjustment dependent on male ornamentation erodes the female preference to zero, and as a result male ornamentation will evolve to zero as well.

Numerical results A numerical implementation of this analytical model, illustrated in Figure 8.2, shows how conditional sex-ratio adjustment erodes sexual selection (see SI on page 216 for full details of the calculation). Initially we fix the sex-allocation traits at $s_0 = s_1 = 0.5$, such that offspring sex ratios are unbiased (to the left of the vertical dashed lines in Figure 8.2). Under these conditions, male ornamentation and female preference evolve away from their survival optima (at zero elaboration and zero preference, respectively) to a stable, exaggerated level (Figure 8.2A), following predictions from standard models of sexual selection (Lande, 1981; Bulmer, 1989; Pomiankowski *et al.*, 1991; Iwasa *et al.*, 1991). Then, from the point indicated by the dashed lines, we allow the sex-allocation traits s_0 and s_1 to evolve. Conditional sex-ratio adjustment evolves as predicted by theory (Pen & Weissing, 2000c; Fawcett *et al.*, 2007): females mated to highly ornamented males have more sons than those mated to less-ornamented males (Figure 8.2B, to the right of the dashed line). [Note that s_1 is prevented from deviating too far from 0.5 due to counter-selection to restore an even population sex ratio, since type-1 males vastly outnumber type-0 males (Fawcett *et al.*, 2007).] As biased sex allocation develops, however, this strategy weakens sexual selection, leading to a gradual decline in male ornamentation and female preference (Figure 8.2A, to the right of the dashed line).

Thus, sexual selection favors conditional sex allocation, but this then erodes sex-

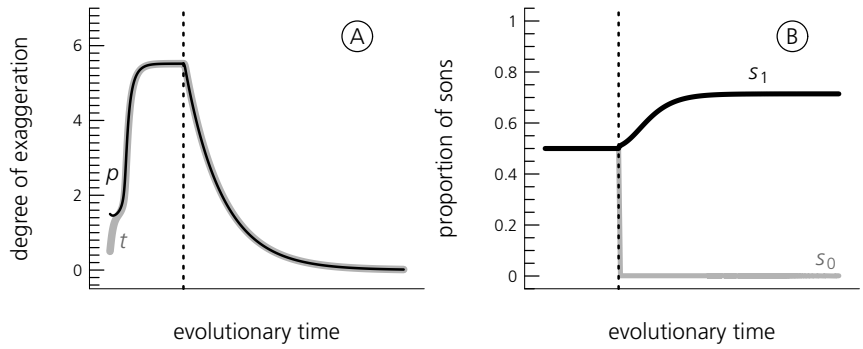

Figure 8.2: Sex-ratio adjustment erodes sexual selection (numerical results). Panel (A) shows the level of male ornamentation (t, grey) and female preference (p, black; note that this partly obscures the grey line), while panel (B) shows the proportion of sons produced by females mated to non-ornamented (s_0, grey) and ornamented (s_1, black) males. Offspring sex ratios are initially unbiased ($s_0 = s_1 = 0.5$), but are allowed to evolve from the point indicated by the vertical dashed lines. Male ornamentation and female preference reach a stable level of exaggeration in the absence of sex-ratio bias, then decline to zero as conditional sex allocation develops [subject to counter-selection on s_1 to restore an even population sex ratio, see Chapter 7] and s_0 and s_1 reach their optima. For the example shown, $c_p = 0.2$, $c_\mathrm{f} = 0.001$, $c_\mathrm{m} = 0.1$, $\mu_0 = 0.02$ and $\mu_1 = 0.3$; the starting values for ornamentation and preference were $t = 0.5$ and $p = 1.5$.

ual selection. Two main processes are responsible for this erosion. First, sex-ratio adjustment allows females with unattractive partners to mitigate the fitness disadvantage of low male ornamentation. In simple terms, ending up with an unattractive male is not so disastrous if a female can skew offspring production towards daughters. This reduces the fitness benefit of female choosiness, which is selected against due to its costs. Second, since choosier females tend to mate with more ornamented males and therefore produce mainly sons, their strong preference genes will rarely be expressed by their offspring. This lowers the average female preference in subsequent generations and thereby reduces the fitness benefit of male ornamentation. In effect, the conditional strategy of sex allocation reduces the heritability of both low attractiveness and strong preferences, undermining selection to invest in costly ornamentation.

Individual-based simulations Using individual-based computer simulations, we can extend this analysis to a more realistic situation where male ornamentation and female preference vary continuously and the evolutionary dynamics are subject to stochastic demographic factors. We simulated a finite population in which a costly male ornament and a costly female preference could change over time through selection and mutation (see section "Materials and Methods" on page 214 for full details). As in the earlier numerical results, evolutionary change in the male ornament and female preference follows predictions from standard analytical models of sexual selection (Lande, 1981; Bulmer, 1989; Pomiankowski *et al.*, 1991; Iwasa *et al.*, 1991) with both traits quickly evolving to a stable, exaggerated level (Figure 8.3A, left of dashed line). Similar patterns are seen regardless of whether male ornamentation is an arbitrary Fisherian trait (Figure 8.3) or is a condition-dependent indicator of "good genes" (see additional simulation results in the Supplement on page 216 and

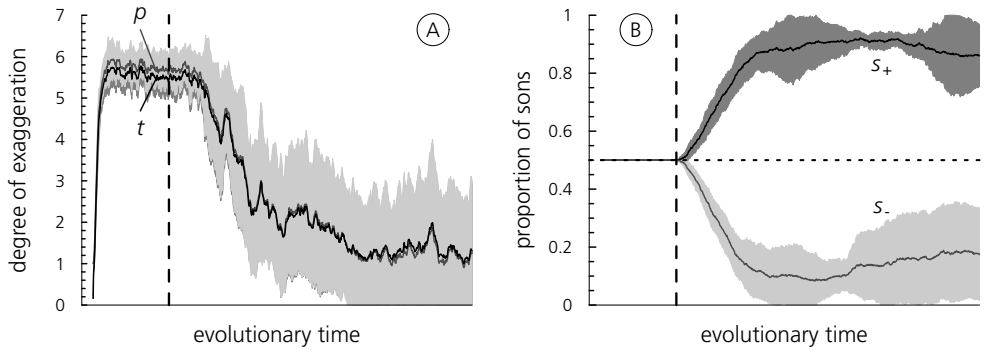

Figure 8.3: Coevolutionary dynamics of sexual selection and sex allocation (individual-based simulations). A shows the level of male ornamentation (t, black) and female preference (p, light grey) under Fisherian sexual selection, whereas B shows the female sex-allocation strategy in the same set of simulations based on traits s_+ and s_-, where s_+ (dark grey) is the probability of producing a son when her partner has above-average ornamentation and s_- (light grey) is the probability of producing a son when he has below-average ornamentation. Offspring sex ratios are initially unbiased ($s_+ = s_- = 0.5$), but they are allowed to evolve from the point indicated by the vertical dashed lines. All values are shown as the mean (solid line) ± SD (shading) from 20 replicate simulation runs. For parameter values, see Materials and Methods.

Figure S8.1 on page 221).

We then allowed a conditional strategy of sex-ratio adjustment to evolve by incorporating two additional traits, s_- and s_+ (Fawcett *et al.*, 2007). These traits determine a female's sex-allocation strategy, with s_- ($0 \leq s_- \leq 1$) being the chance of producing a son when mated to a male with below-average ornamentation and s_+ ($0 \leq s_+ \leq 1$) that when mated to a male with above-average ornamentation. Starting from a situation in which offspring sex ratios are unbiased ($s_- = s_+ = 0.5$), conditional sex-ratio adjustment gradually develops as predicted by theory (Pen & Weissing, 2000c; Fawcett *et al.*, 2007): Females mated to highly ornamented males overproduce sons, whereas those mated to less-ornamented males overproduce daughters (Figure 8.3B). [Note that with continuous variation in male ornamentation, s_- and s_+ become biased to a similar extent (Fawcett *et al.*, 2007).] This strategy then weakens sexual selection, leading to a gradual decline in male ornamentation and female preference (Figure 8.3A, right of the dashed line).

When male ornamentation is a condition-dependent indicator of good genes (Iwasa *et al.*, 1991), sexual selection is weakened to a lesser extent than when it is a purely Fisherian trait (see Supplement). In the former case, the heritable benefits for a female who mates with an attractive male are not entirely sex-limited; although only her sons can profit from their father's ornamentation genes, both her daughters and her sons will inherit his genes for viability. Thus, even when females exert a great degree of control over the sex of their offspring, it still pays to mate with more ornamented males. This difference notwithstanding, for both Fisherian and good genes models of sexual selection, ornamentation and preference are substantially reduced as conditional sex allocation develops.

To check that sex-ratio adjustment is directly responsible for this decline, we ran another set of simulations in which strategies with varying degrees of sex-ratio bias were introduced partway through (Figure 8.4). Initially, with the sex ratio fixed at

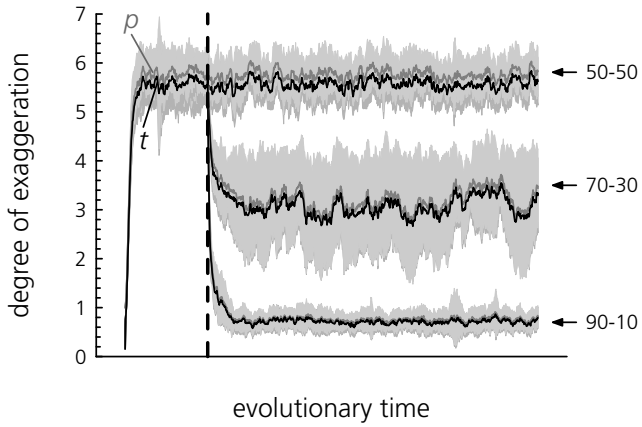

Figure 8.4: Stronger degrees of sex-ratio bias have stronger eroding effects on sexual selection. The plots show the level of male ornamentation (t, black) and female preference (p, grey). Offspring sex ratios are initially unbiased ($s_+ = s_- = 0.5$), but from the point indicated by the vertical dashed line, females use a fixed sex-allocation strategy conditional on the male's ornamentation. Three different degrees of sex-ratio bias are shown: $s_+ = s_- = 0.5$ (50–50, i.e., no bias); $s_+ = 0.7, s_- = 0.3$ (70–30); and $s_+ = 0.9, s_- = 0.1$ (90–10). All values are shown as the mean (solid line) ± SD (shading) from 20 replicate simulation runs. For parameter values, see Materials and Methods.

0.5, the male ornament and female preference quickly evolve to a stable, exaggerated level as before. We then introduced a biased sex-allocation strategy for all females, causing them to produce more sons when mated to an attractive partner and more daughters when mated to an unattractive partner. The effect on sexual selection is dramatic. For a moderate degree of bias, $s_+ = 0.7$ and $s_- = 0.3$, very rapidly the ornament and preference drop to approximately one-half of their original level of expression. Adjustment strategies involving weaker biases result in a smaller drop, whereas with stronger biases the decline in ornamentation is even sharper (Figure 8.4).

Our simulation results confirm that the equilibrium levels of female preference and male ornamentation are substantially lower when sex-ratio adjustment is possible. In effect, sexual selection undermines itself by favoring a conditional strategy of sex-ratio adjustment based on male attractiveness.

8.4 Discussion

Previous theory (Pen & Weissing, 2000c; Fawcett *et al.*, 2007) has confirmed the empirical suggestion (Burley, 1981) that variation in male sexual displays favors conditional sex allocation by females. Here, we have shown an unexpected consequence of this process: that by reducing the fitness difference between females with attractive and unattractive partners, this sex-allocation strategy undermines the same selective force that created it, causing male ornamentation to decline. Moreover, because choosier females tend to mate with more highly ornamented males and, therefore, produce sons, their stronger preference genes are likely to be masked in the next generation, weakening sexual selection still further. Our evolutionary simulations predict a lower level of sexual display than in cases where facultative sex-ratio adjustment is not possible. This finding implies that, all else being equal, the most exaggerated secondary sexual traits should be seen in species with little or no con-

trol over offspring sex. For instance, we might expect that species with genotypic sex determination will have more exaggerated sexual ornamentation than closely related species with temperature-dependent sex determination, assuming that the latter mechanism affords parents greater control over the sex of their offspring.

It is known that the evolution of phenotypic plasticity in a quantitative trait can alter the evolution of the average phenotype for that trait (Kirkpatrick, 1982a; Frank & Swingland, 1988; Pál & Miklós, 1999; Lande, 2009). Here, we have shown a related effect: that plasticity in one trait (sex-ratio bias) can alter the evolution of another trait (ornamentation) on which it is conditional. We propose that this phenomenon is not restricted to sex allocation, but is an example of a more general principle. Whenever heritable variation in fitness is maintained for a given trait, selection should favor any conditional strategy that improves the fitness prospects of the least successful phenotypes, but in doing so, it erodes selection on the trait.

To illustrate the general nature of our argument, we give examples from a range of contexts that do not involve sex allocation. The first concerns kleptoparasitism (Brockmann & Barnard, 1979), in which one animal steals food that a conspecific has caught before the latter can eat it. Selection for good hunting skills is expected to be strong in any predatory species, but there may still be substantial variation in hunting success because of mutations in polygenic traits affecting the development of motor skills. If poor hunters adopt kleptoparasitic behavior, however, this conditional strategy will reduce fitness differences based on hunting success and, thereby, weaken selection on hunting ability. Combined with the costs incurred by parasitized hunters, the weakened selection may lead to a decline in hunting skills that, in turn, will reduce the benefits of stealing. Thus, selection on hunting ability and kleptoparasitism interact in a highly dynamic fashion.

The second example involves polygynous mating systems in which access to females is determined by male dominance relations. In such systems, there will be strong selection for male characteristics related to dominance, such as large body size. Slight differences between males in these characteristics early in life may largely determine their relative positions in the dominance hierarchy, leading to substantial differences in lifetime reproductive success. If small males adopt a “sneaker” tactic (Gross, 1996), however, allowing them to achieve significant reproductive success by subversive means, this conditional strategy will reduce fitness differences between males of high and low dominance rank and, thereby, weaken selection on body size. This weakening of selection, in turn, will alter the selection-mutation balance, allowing greater levels of genetic variation for body size to persist in the population.

Our final example deals with costly dispersal. In many plant and animal species, dispersal away from the natal habitat may be favored despite the energetic cost or mortality risk associated with this movement. The benefits of dispersal will typically be frequency-dependent, with the greatest pressure to disperse occurring when most individuals stay at home. However, if individuals that forgo dispersal can adapt better to overcrowding, for example, through niche construction, then the strength of selection on dispersal will be weakened. Selection favors a strategy that mitigates the fitness disadvantage of staying in the natal habitat, and this plasticity erodes directional selection on the ability to disperse away from that habitat. These diverse examples show that our model applies to a broad range of contexts. The evolutionary feedback process we have described is likely to be a widespread and important force maintaining phenotypic variation in the face of directional selection.

8.5 Materials and Methods

Details of the Individual-Based Simulations The individual-based simulations were similar to those described in an earlier paper (Fawcett *et al.*, 2007). In the main text, we focus on Fisherian sexual selection, whereas the simulations for good genes sexual selection are presented in the Supplement. We modeled a population of 5,000 individuals, each with diploid, autosomal genetic values for the following traits: p, coding for preference (expressed only by females); t, coding for ornamentation (expressed only by males); and two sex-allocation traits, s_- and s_+ (expressed only by females). The value for p can take any real number, whereas t is limited to positive values and s_- and s_+ are limited between 0 and 1. We chose to restrict t to positive values because this range might better represent certain forms of male display (Kokko *et al.*, 2006), for example, the height of a plumage crest, but we obtain similar results when male ornamentation can also take negative values (see additional simulation results in the Supplement and Figure S8.2 on page 222). Female preference and male ornamentation are both assumed to be costly; survival to maturity is maximized for $p = 0$ and $t = 0$ and declines away from these optima as specified by the functions $\exp(-c_f p^2)$ and $\exp(-c_m t^2)$, where c_f and c_m are positive constants.

For reproduction, females are drawn from the population with a chance proportional to their survival probability. Each surviving female then samples 10 males, again weighted by survival probability, and chooses one of them on the basis of his ornamentation. The chance that she picks a given male is proportional to $\exp(c_p p t)$, where c_p is a positive constant scaling the importance of ornamentation to female choice. Thus, females with a positive preference ($p > 0$) prefer more ornamented males, those females with a negative preference ($p < 0$) prefer less ornamented males and those females with $p = 0$ mate randomly. To facilitate sexual selection, we started the simulations with a positive preference (Lande, 1981; Kokko *et al.*, 2006); the same process occurs when starting from a situation of random choice, but it takes longer.

Each mating produces a single offspring, whose genetic values are determined by standard Mendelian inheritance. We assume that there is no genetic dominance and that the loci are unlinked. Offspring sex is determined by the father's ornamentation and the mother's sex-allocation strategy: the probability of producing a son is s_+ when the father's ornamentation level is above average and s_- when it is below average. For each trait, we assume that mutations occur in a small fraction of offspring (with probability μ_p for p, μ_t for t, and μ_s for s_- and s_+), causing the genetic value to change upward or downward by an amount drawn from a uniform probability distribution (up to a certain maximum amount). Upward and downward mutations are equally likely except in the ornamentation trait t, for which we assume that a downward mutation bias reduces ornamentation by an average amount g (Fawcett *et al.*, 2007; Pomiankowski *et al.*, 1991). Reproduction continues until a total of 5,000 offspring have been produced, at which point all of the adults die and are replaced by the offspring generation. The same cycle of events was repeated for 100,000 generations, which is the timespan depicted in our figures. Computer code for the simulations is available from the authors upon request.

For the results shown in the main text, the parameter values were $c_p = 1.0$, $c_f = 0.001$, $c_m = 0.5$, $\mu_p = \mu_t = \mu_s = 0.05$ and $g = 0.02$, with the average genetic values in the initial population set at $\bar{p} = 1, \bar{t} = 0$ and $\bar{s}_+ = \bar{s}_- = 0.5$. However, the eroding effect of sex-ratio adjustment is seen for a wide range of parameter values, whenever

sexual selection leads to exaggerated male ornamentation.

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S8 Supplementary Information

Supplementary Information on the model

Full Derivation of the Resident Transition Matrix A Here, we explain how we derived the entries of the transition matrix **A** (Eq. 8.1), which governs the dynamics of a resident population with ornamentation level t, preference p, and sex-allocation traits s_0 and s_1. We reproduce the matrix here for clarity:

$$\mathbf{A} = \frac{1}{2}k \begin{bmatrix} \left[(1-\alpha)(1-s_0)+\alpha(1-s_1)\right]\nu_{\mathrm{f}} & q_0(1-s_0)\nu_{\mathrm{f}} & q_1(1-s_1)\nu_{\mathrm{f}} \\ \left[(1-\alpha)s_0(1-\mu_0)+\alpha s_1\mu_1\right]\nu_{\mathrm{m0}} & q_0 s_0(1-\mu_0)\nu_{\mathrm{m0}} & q_1 s_1\mu_1\nu_{\mathrm{m0}} \\ \left[(1-\alpha)s_0\mu_0+\alpha s_1(1-\mu_1)\right]\nu_{\mathrm{m1}} & q_0 s_0\mu_0\nu_{\mathrm{m1}} & q_1 s_1(1-\mu_1)\nu_{\mathrm{m1}} \end{bmatrix}. \tag{S8.1}$$

To recap: The first column of **A** ($\mathbf{a}_1$) represents the per-capita reproductive output of females, the second column ($\mathbf{a}_2$) that of type-0 males, and the third ($\mathbf{a}_3$) that of type-1 males. The three rows represent, from top to bottom, the result of this reproductive output in terms of surviving females, type-0 males, and type-1 males in the next generation. We will use the notation a_{nm} to represent the element occupying the nth row and mth column of matrix **A**.

The top-left entry (a_{11}) represents the per-capita reproductive contribution of mothers in the current generation to mothers in the next generation. A proportion $1-\alpha$ of their matings are with nonornamented males, with whom a proportion $1-s_0$ of the offspring they produce are daughters. The remaining α matings are with ornamented males, with whom a proportion $1-s_1$ of the offspring they produce are daughters. In both cases, these daughters survive to reproduce with probability ν_{f}.

The middle-left entry (a_{21}) represents the per-capita reproductive contribution of mothers in the current generation to nonornamented fathers in the next generation. A proportion $1-\alpha$ of these mothers' matings are with nonornamented males, with whom a proportion s_0 of the offspring they produce are sons; with probability $1-\mu_0$, these sons are unaffected by mutation and, therefore, inherit their father's lack of ornamentation. The remaining α matings are with ornamented males, with whom a proportion s_1 of the offspring they produce are sons; with probability μ_1, these sons lose their father's ornamentation through mutation. In both cases, these nonornamented sons survive to reproduce with probability ν_{m0}.

The bottom-left entry (a_{31}) represents the per-capita reproductive contribution of mothers in the current generation to ornamented fathers in the next generation. A proportion $1-\alpha$ of these mothers' matings are with nonornamented males, with whom a proportion s_0 of the offspring they produce are sons; with probability μ_0, these sons mutate into the ornamented state. The remaining α matings are with ornamented males, with whom a proportion s_1 of the offspring they produce are sons; with probability $1-\mu_1$, these sons are unaffected by mutation and, therefore, inherit their father's ornamentation. In both cases, these ornamented sons survive to reproduce with probability ν_{m1}.

The top-center entry (a_{12}) represents the per-capita reproductive contribution of nonornamented fathers in the current generation to mothers in the next generation. The expected number of mates for these fathers is q_0. A proportion $1-s_0$ of their offspring are daughters, who survive to reproduce with probability ν_{f}.

The middle-center entry (a_{22}) represents the per-capita reproductive contribution

of nonornamented fathers in the current generation to nonornamented fathers in the next generation. The expected number of mates for these fathers is q_0. A proportion s_0 of their offspring are sons, who inherit their father's lack of ornamentation with probability $1-\mu_0$ and then survive to reproduce with probability $\nu_{\mathrm{m}0}$.

The bottom-center entry (a_{32}) represents the per-capita reproductive contribution of nonornamented fathers in the current generation to ornamented fathers in the next generation. The expected number of mates for nonornamented fathers is q_0. A proportion s_0 of their offspring are sons, who mutate into the ornamented state with probability μ_0 and then survive to reproduce with probability $\nu_{\mathrm{m}1}$.

The top-right entry (a_{13}) represents the per-capita reproductive contribution of ornamented fathers in the current generation to mothers in the next generation. The expected number of mates for these fathers is q_1. A proportion $1-s_1$ of their offspring are daughters, who survive to reproduce with probability ν_{f}.

The middle-right entry (a_{23}) represents the per-capita reproductive contribution of ornamented fathers in the current generation to nonornamented fathers in the next generation. The expected number of mates for ornamented fathers is q_1. A proportion s_1 of their offspring are sons, who mutate into the nonornamented state with probability μ_1 and then survive to reproduce with probability $\nu_{\mathrm{m}0}$.

Finally, the bottom-right entry (a_{33}) represents the per-capita reproductive contribution of ornamented fathers in the current generation to ornamented fathers in the next generation. The expected number of mates for these fathers is q_1. A proportion s_1 of their offspring are sons, who inherit their father's ornamentation with probability $1-\mu_1$ and then survive to reproduce with probability $\nu_{\mathrm{m}1}$.

Finding the relative frequencies For consistency, it is required that all females (relative frequency y_{f}) have the same reproductive output as all males ($y_{\mathrm{m}0}+y_{\mathrm{m}1}$), in other words that

$$\mathbf{a}_1 y_{\mathrm{f}} = \mathbf{a}_2 y_{\mathrm{m}0} + \mathbf{a}_3 y_{\mathrm{m}1}. \tag{S8.2}$$

This equation is helpful in finding the dominant eigenvalue λ of $\mathbf{A}$, which is the long-term growth rate of the resident population. Let $\mathbf{y} = (y_{\mathrm{f}}, y_{\mathrm{m}0}, y_{\mathrm{m}1})^{\mathrm{T}}$ be the dominant right eigenvector of $\mathbf{A}$, containing the stable relative class frequencies; this eigenvector is given by $\mathbf{A}\mathbf{y} = \lambda\mathbf{y}$, or, in terms of the columns of $\mathbf{A}$, $\mathbf{a}_1 y_{\mathrm{f}} + \mathbf{a}_2 y_{\mathrm{m}0} + \mathbf{a}_3 y_{\mathrm{m}1} = \lambda\mathbf{y}$. Substituting (S8.2) into this equation, we get $\lambda\mathbf{y} = 2\mathbf{a}_1 y_{\mathrm{f}} \Rightarrow \lambda = 2a_{11} = k(1-\bar{s})\nu_{\mathrm{f}}$, where $\bar{s} = (1-\alpha)s_0 + \alpha s_1$ is the average offspring sex ratio. The long-term growth rate is therefore equal to the per-capita number of surviving daughters. Note that k gets rescaled by density dependence so that in a stable population $\lambda = 1$, i.e., $k = 1/(1-\bar{s})\nu_{\mathrm{f}}$ (see Pen & Weissing (2000b) and Mylius & Diekmann (1995)). For the rest of our analysis, we do not need an explicit solution for the stable class distribution, but it will prove useful to have explicit equations for y_{f} and $y_{\mathrm{m}1}$:

$$2\lambda y_{\mathrm{f}} = (1-\bar{s})\,\nu_{\mathrm{f}} y_{\mathrm{f}} + q_0\left(1-s_0\right)\nu_{\mathrm{f}} y_{\mathrm{m}0} + q_1\left(1-s_1\right)\nu_{\mathrm{f}} y_{\mathrm{m}1}, \tag{S8.3}$$

$$\begin{aligned} 2\lambda y_{\mathrm{m}1} = {} & \left[(1-\alpha)s_0\mu_0 + \alpha s_1\left(1-\mu_1\right)\right]\nu_{\mathrm{m}1} y_{\mathrm{f}} + q_0 s_0 \mu_0 \nu_{\mathrm{m}1} y_{\mathrm{m}0} \\ & + q_1 s_1\left(1-\mu_1\right)\nu_{\mathrm{m}1} y_{\mathrm{m}1}. \end{aligned} \tag{S8.4}$$

Calculating the selection differentials for a rare mutant The transition matrix **B**, as given in Eq. (8.2) is as follows

$$\mathbf{B} = \frac{1}{2}k \begin{bmatrix} \left[(1-\hat{\alpha})(1-\hat{s}_0)+\hat{\alpha}(1-\hat{s}_1)\right]\hat{v}_{\mathrm{f}} & q_0(1-s_0)\hat{v}_{\mathrm{f}} & \hat{q}_1(1-s_1)\hat{v}_{\mathrm{f}} \\ \left[(1-\hat{\alpha})\hat{s}_0(1-\mu_0)+\hat{\alpha}\hat{s}_1\mu_1\right]v_{\mathrm{m0}} & q_0 s_0(1-\mu_0)v_{\mathrm{m0}} & \hat{q}_1 s_1\mu_1 v_{\mathrm{m0}} \\ \left[(1-\hat{\alpha})\hat{s}_0\mu_0+\hat{\alpha}\hat{s}_1(1-\mu_1)\right]\hat{v}_{\mathrm{m1}} & q_0 s_0\mu_0\hat{v}_{\mathrm{m1}} & \hat{q}_1 s_1(1-\mu_1)\hat{v}_{\mathrm{m1}} \end{bmatrix}, \tag{S8.5}$$

where mutant phenotypes have hats (ˆ) to distinguish them from resident phenotypes. This matrix describes the dynamics of rare mutant individuals in the resident population.

To quantify the invasion prospects of mutants, we investigate the sensitivity of **B**'s dominant eigenvalue w with respect to small changes in the mutant trait values. If **z** and **y** are left and right eigenvectors of **A**, then according to a standard result (Otto & Day, 2007),

$$\frac{\partial w}{\partial \hat{x}} = \mathbf{z}^{\mathrm{T}}\frac{\partial \mathbf{B}}{\partial \hat{x}}\mathbf{y}/\mathbf{z}^{\mathrm{T}}\mathbf{y}, \tag{S8.6}$$

where $\hat{x}$ is the mutant value for the trait of interest and the derivatives are evaluated at the resident trait values. Because we are mainly interested in the direction of selection, we will ignore the denominator of the right-hand side (which is always positive). The vectors **y** and **z** correspond to the stable class distribution and class reproductive values for the resident population (Otto & Day, 2007). As with **y**, it is not necessary to calculate the reproductive values **z** explicitly, but we do need the following equations for the reproductive values of type-0 and type-1 males:

$$2\lambda z_{\mathrm{m0}} = q_0\left[(1-s_0)\,v_{\mathrm{f}}z_{\mathrm{f}} + s_0(1-\mu_0)\,v_{\mathrm{m0}}z_{\mathrm{m0}} + s_0\mu_0 v_{\mathrm{m1}}z_{\mathrm{m1}}\right], \tag{S8.7}$$

$$2\lambda z_{\mathrm{m1}} = q_1\left[(1-s_0)\,v_{\mathrm{f}}z_{\mathrm{f}} + s_1\mu_1 v_{\mathrm{m0}}z_{\mathrm{m0}} + s_1(1-\mu_1)\,v_{\mathrm{m1}}z_{\mathrm{m1}}\right]. \tag{S8.8}$$

Now we can work out the selection differential for p, using the numerator of the right-hand side of Eq. (S8.6). Writing $v'_{\mathrm{f}} = \mathrm{d}v_{\mathrm{f}}/\mathrm{d}p$ and $\alpha' = \mathrm{d}\alpha/\mathrm{d}p$, we get

$$\frac{\partial w}{\partial \hat{p}} = \frac{\mathbf{z}^{\mathrm{T}}}{2\lambda}\begin{bmatrix} \alpha'(s_0-s_1)\,v_{\mathrm{f}} + v'_{\mathrm{f}}(1-\bar{s}) & q_0(1-s_0)v'_{\mathrm{f}} & q_1(1-s_1)v'_{\mathrm{f}} \\ \alpha'\left[s_1\mu_1 - s_0(1-\mu_0)\right] & 0 & 0 \\ \alpha'\left[s_1(1-\mu_1) - s_0\mu_0\right] & 0 & 0 \end{bmatrix}\mathbf{y} \tag{S8.9}$$

$$= \frac{\mathbf{z}^{\mathrm{T}}}{2\lambda}\begin{bmatrix} \alpha'(s_0-s_1)\,v_{\mathrm{f}} + v'_{\mathrm{f}}/v_{\mathrm{f}}[(1-\bar{s})\,v_{\mathrm{f}}y_{\mathrm{f}} \\ \quad + q_0(1-s_0)\,v_{\mathrm{f}}y_{\mathrm{m0}} + q_1(1-s_1)\,v_{\mathrm{f}}y_{\mathrm{m1}}] \\ \alpha'\left[s_1\mu_1 - s_0(1-\mu_0)\right]y_{\mathrm{f}} \\ \alpha'\left[s_1(1-\mu_1) - s_0\mu_0\right]y_{\mathrm{f}} \end{bmatrix} \tag{S8.10}$$

$$\overset{(S8.3)}{=} \frac{\mathbf{z}^{\mathrm{T}}}{2\lambda}\begin{bmatrix} \alpha'(s_0-s_1)\,v_{\mathrm{f}} + \left(v'_{\mathrm{f}}/v_{\mathrm{f}}\right)2\lambda y_{\mathrm{f}} \\ \alpha'\left[s_1\mu_1 - s_0(1-\mu_0)\right]y_{\mathrm{f}} \\ \alpha'\left[s_1(1-\mu_1) - s_0\mu_0\right]y_{\mathrm{f}} \end{bmatrix} \tag{S8.11}$$

$$\overset{(S8.7,S8.8)}{=} \alpha'\left[\frac{z_{\mathrm{m1}}}{q_1} - \frac{z_{\mathrm{m0}}}{q_0}\right]y_{\mathrm{f}} + \frac{v'_{\mathrm{f}}}{v_{\mathrm{f}}}z_{\mathrm{f}}y_{\mathrm{f}}. \tag{S8.12}$$

Likewise, the selection differential for t, with primes denoting differentiation with

respect to t, is

$$\frac{\partial w}{\partial \hat{t}} = \frac{\mathbf{z}^{\mathrm{T}}}{2\lambda} \begin{bmatrix} 0 & 0 & a_{13} q_1'/q_1 \\ 0 & 0 & 0 \\ a_{31} v_{\mathrm{m}1}'/v_{\mathrm{m}1} & a_{32} v_{\mathrm{m}1}'/v_{\mathrm{m}1} & a_{33} \left(v_{\mathrm{m}1}'/v_{\mathrm{m}1} + q_1'/q_1 \right) \end{bmatrix} \mathbf{y} \tag{S8.13}$$

$$= \frac{\mathbf{z}^{\mathrm{T}}}{2\lambda} \begin{bmatrix} a_{13} \left(q_1'/q_1 \right) y_{\mathrm{m}1} \\ a_{23} \left(q_1'/q_1 \right) y_{\mathrm{m}1} \\ \left(a_{31} y_{\mathrm{f}} + a_{32} y_{\mathrm{m}0} + a_{33} y_{\mathrm{m}1} \right) v_{\mathrm{m}1}'/v_{\mathrm{m}1} + a_{33} \left(q_1'/q_1 \right) y_{\mathrm{m}1} \end{bmatrix} \tag{S8.14}$$

$$\overset{(S8.4)}{=} \frac{\mathbf{z}^{\mathrm{T}}}{2\lambda} \begin{bmatrix} a_{13} \left(q_1'/q_1 \right) y_{\mathrm{m}1} \\ a_{23} \left(q_1'/q_1 \right) y_{\mathrm{m}1} \\ 2\lambda \left(v_{\mathrm{m}1}'/v_{\mathrm{m}1} \right) y_{\mathrm{m}1} + a_{33} \left(q_1'/q_1 \right) y_{\mathrm{m}1} \end{bmatrix} \tag{S8.15}$$

$$= \frac{1}{2\lambda} \left(q_1'/q_1 \right) \left(a_{13} z_{\mathrm{f}} + a_{23} z_{\mathrm{m}0} + a_{33} z_{\mathrm{m}1} \right) y_{\mathrm{m}1} + \left(v_{\mathrm{m}1}'/v_{\mathrm{m}1} \right) z_{\mathrm{m}1} y_{\mathrm{m}1} \tag{S8.16}$$

$$\overset{(S8.8)}{=} \left(q_1'/q_1 \right) z_{\mathrm{m}1} y_{\mathrm{m}1} + \left(v_{\mathrm{m}1}'/v_{\mathrm{m}1} \right) z_{\mathrm{m}1} y_{\mathrm{m}1}. \tag{S8.17}$$

Next we turn to the sex-allocation traits. The selection differential for s_0 is

$$\frac{\partial w}{\partial \hat{s}_0} = \frac{1-\alpha}{2(1-\bar{s})\, v_{\mathrm{f}}} \left[-v_{\mathrm{f}} + (1-\mu_0)\, v_{\mathrm{m}0} z_{\mathrm{m}0} + \mu_0 v_{\mathrm{m}1} z_{\mathrm{m}1} \right] y_{\mathrm{f}} \tag{S8.18}$$

$$= \frac{1-\alpha}{2(1-\bar{s})\, v_{\mathrm{f}}} \left[-\frac{v_{\mathrm{f}} z_{\mathrm{f}}}{s_0} + \frac{1-s_0}{s_0} v_{\mathrm{f}} z_{\mathrm{f}} + (1-\mu_0)\, v_{\mathrm{m}0} z_{\mathrm{m}0} + \mu_0 v_{\mathrm{m}1} z_{\mathrm{m}1} \right] y_{\mathrm{f}} \tag{S8.19}$$

$$= \frac{1-\alpha}{2(1-\bar{s})\, v_{\mathrm{f}}} \left[-\frac{v_{\mathrm{f}} z_{\mathrm{f}}}{s_0} + \frac{2(1-\bar{s})\, v_{\mathrm{f}}}{q_0 s_0} z_{\mathrm{m}0} \right] y_{\mathrm{f}} \tag{S8.20}$$

$$= \frac{1-\alpha}{s_0} \left[\frac{z_{\mathrm{m}0}}{q_0} - \frac{1}{2(1-\bar{s})} \right] y_{\mathrm{f}}, \tag{S8.21}$$

whereas that for s_1, calculated in a similar way, is

$$\frac{\partial w}{\partial \hat{s}_0} = \frac{\alpha}{s_1} \left[\frac{z_{\mathrm{m}1}}{q_1} - \frac{1}{2(1-\bar{s})} \right] y_{\mathrm{f}}. \tag{S8.22}$$

The selection differentials (S8.12, S8.17, S8.21, S8.22) form equations (8.4-8.7) in the main text.

Numerical Simulations For numerical simulations, we need to make some additional specific assumptions. Let $r = \exp\left(c_p p t\right)$ be the odds that a resident female with preference p chooses a type-1 male with ornamentation level t over a type-0 (non-ornamented) male, where c_p is a positive constant. Then the probability α that such a female will mate with a type-1 male is given by

$$\alpha = \frac{r y_{\mathrm{m}1}}{y_{\mathrm{m}0} + r y_{\mathrm{m}1}}. \tag{S8.23}$$

Eq. (S8.23) is actually an implicit equation for α, because the class frequencies $y_{\mathrm{m}i}$ will depend on α. However, for a mutant female, the class frequencies are constant, giving $\alpha' = \alpha(1-\alpha)(\mathrm{d}r/\mathrm{d}p)/r$. The odds that a type-1 male with mutant ornamenta-

tion level $\hat{t}$ is chosen by a resident female is $\hat{r} = \exp(c_p p \hat{t})$ which makes his expected number of mates

$$\hat{q}_1 = y_{\mathrm{f}} \frac{\hat{r}}{y_{\mathrm{m0}} + r y_{\mathrm{m1}}}, \tag{S8.24}$$

and, therefore, $q_1'/q_1 = (\mathrm{d}r/\mathrm{d}t)/r$. Likewise, $\hat{q}_0 = y_{\mathrm{f}}/(y_{\mathrm{m0}} + r y_{\mathrm{m1}}) = q_0$ (the expected mating success of mutant and resident type-0 males is identical because neither expresses an ornament). Finally, we assume (as in the individual-based simulations) that viability decreases according to a Gaussian function with p and t:

$$v_{\mathrm{f}} = \exp\left(-c_{\mathrm{f}} p^2\right), \tag{S8.25}$$

$$v_{\mathrm{m1}} = \exp\left(-c_{\mathrm{m}} t^2\right), \tag{S8.26}$$

and $v_{\mathrm{m0}} = 1$.

Evolutionary dynamics can be modeled by using the dynamic equation $\dot{x} = \partial w/\partial x|_{x=\hat{x}}$ for the traits p, t, s_0 and s_1. An example is shown in Figure 8.2.

Supplementary results

Good genes sexual selection For the good genes simulations, we included an extra trait, v, to model genetic variation in viability, which affects survival to adulthood. Female survival now depends on her viability and her expressed preference, as specified by the function $v \cdot \exp(-c_{\mathrm{f}} p^2)$, whereas male survival is proportional to $v \cdot \exp(-c_{\mathrm{m}} \gamma^2)$, where γ denotes his expressed ornamentation. This ornamentation is assumed to be condition-dependent, with $\gamma = tv$, which means that males of higher viability are more ornamented for a given value of t. Thus, male ornamentation acts as a conditional indicator of genetic viability (Iwasa *et al.*, 1991). Females choose on the basis of this condition-dependent ornamentation; so now the chance that a given male is picked from the sample of 10 males is proportional to $\exp(c_p p \gamma)$. Likewise, sex-ratio adjustment by females is based on the expressed ornamentation of her chosen partner, so it depends on γ rather than t. Mutations occur in both t (with probability μ_t) and v (with probability μ_v), but in contrast to the Fisherian simulations, those mutations in t are unbiased ($g = 0$). Instead, biased mutations are assumed to affect the viability trait v, reducing its value by an average amount h ($h > 0$) and, thereby, maintaining genetic variation between males (Iwasa *et al.*, 1991; Fawcett *et al.*, 2007). Figure S8.1 shows the results of these good genes simulations with parameter values $c_p = 1.0$, $c_{\mathrm{f}} = 0.0025$, $c_{\mathrm{m}} = 0.5$, $\mu_p = \mu_t = \mu_s = \mu_v = 0.05$, and $h = 0.02$ and average genetic values starting at $\bar{p} = 1$, $\bar{t} = 0$, $\bar{v} = 0.01$, and $\bar{s}_+ = \bar{s}_- = 0.5$. As in the Fisherian simulations, male ornamentation and female preference coevolve to exaggerated levels under sexual selection, but then decline as sex-ratio adjustment develops.

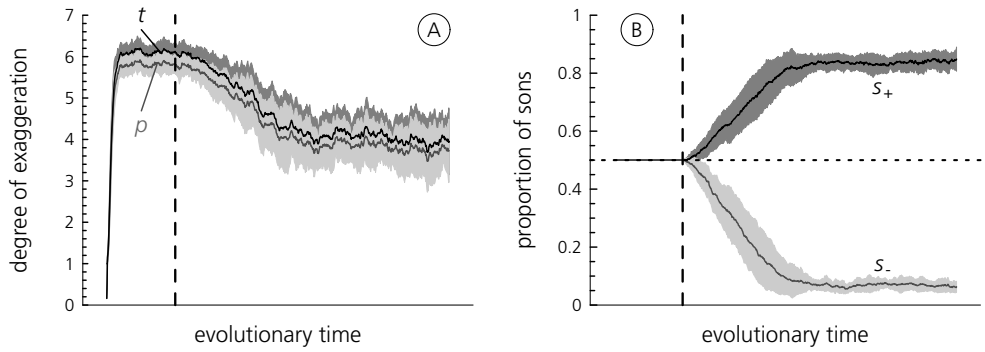

Figure S8.1: Coevolutionary dynamics of good genes sexual selection and sex allocation (individual-based simulations). A shows the level of male ornamentation (t, black) and female preference (p, grey) under good genes sexual selection, whereas B shows the female sex-allocation strategy in the same set of simulations based on traits s_+ and s_-, where s_+ (dark grey) is the probability of producing a son when her partner has above-average ornamentation and s_- (light grey) is the probability of producing a son when he has below-average ornamentation. Offspring sex ratios are initially unbiased ($s_+ = s_- = 0.5$) but are allowed to evolve from the point indicated by the vertical dashed lines. All values are shown as the mean (solid line) ± SD (stippling) from 20 replicate simulation runs.

When ornamentation can take negative values We ran additional simulations in which the genetic value for ornamentation could take any real number, as in some previous models of sexual selection (Iwasa *et al.*, 1991; Pomiankowski *et al.*, 1991). Otherwise, the details of the simulations were kept the same, with male survival maximized for $t = 0$ and declining away from this optimum according to the function $\exp(-c_{\mathrm{m}} t^2)$, where c_{m} is a positive constant.

Figure S8.2 shows the results of these simulations for Fisherian sexual selection with parameter values $c_p = 1.0$, $c_{\mathrm{f}} = 0.001$, $c_{\mathrm{m}} = 0.5$, $\mu_p = \mu_t = \mu_s = 0.05$, and $g = 0.02$ and average genetic values starting at $\bar{p} = 1$, $\bar{t} = 0$. The sex-allocation traits were initially fixed at $\bar{s}_+ = \bar{s}_- = 0.5$, but partway through the simulations, we allowed these traits to evolve. As before, male ornamentation and female preference coevolve to exaggerated levels under sexual selection, but then decline as sex-ratio adjustment develops.

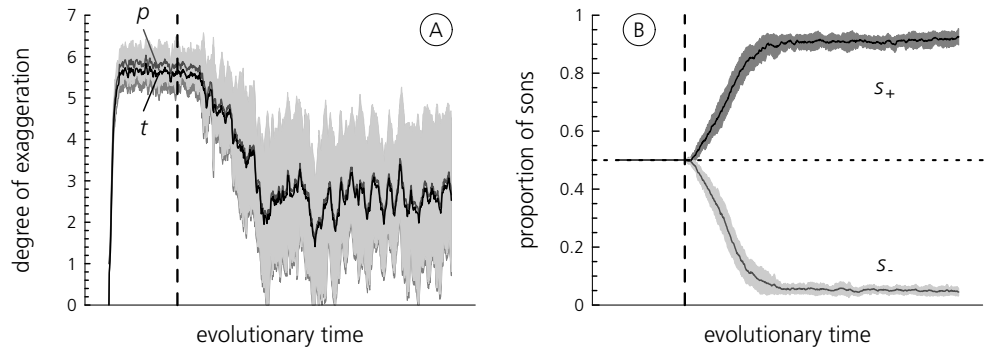

Figure S8.2: Coevolutionary dynamics of sexual selection and sex allocation when ornamentation can evolve either above (positive) or below (negative) its survival optimum, as in some previous models of sexual selection. A shows the level of male ornamentation (t, black) and female preference (p, grey) under Fisherian sexual selection, whereas B shows the female sex-allocation strategy in the same set of simulations based on traits s_+ and s_-, where s_+ (dark grey) is the probability of producing a son when her partner has above-average ornamentation and s_- (light grey) is the probability of producing a son when he has below-average ornamentation. Offspring sex ratios are initially unbiased ($s_+ = s_- = 0.5$) but are allowed to evolve from the point indicated by the vertical dashed lines. All values are shown as the mean (solid line) ± SD (stippling) from 20 replicate simulation runs.

CHAPTER 9

Synthesis and aftherthoughts

How many resources should a mother devote to offspring of a particular sex, in case the heritable quality of her partner will have a larger effect on the mating success of her sons than that of her daughters? One of the main goals of this thesis is to understand the evolution of sex allocation within the context of mate choice, and to predict how sex allocation and sexually selected traits evolve in response to each other. Working towards this goal, the first two parts of this thesis focus on the evolution of sexually selected traits and sex allocation in isolation of each other, whereas the last part of this thesis combines the evolution of sexually selected characters and sex allocation in a single coevolutionary model. Based on the conclusions from this coevolutionary model, in this final chapter I discuss a number of further aspects that surround the interaction between sex allocation and the evolution of sexually selected characters.

One of the main findings of this thesis is that when facultative sex ratios based on partner quality are allowed to coevolve with sexually selected traits that determine parter quality, a negative feedback develops: initially, facultative sex ratios indeed evolve in the context of sexual selection, and females mated to high quality males overproduce sons, to profit from the reproductive value of these high quality sons. However, facultative sex ratios are also useful to females mated to low quality males, who overproduce daughters to avoid the fitness costs of producing low quality sons. Since daughters will be very likely of achieving at least some reproductive success in a polygynous system, fitness differences between females mated to high versus low quality males are largely eliminated, which in turn substantially weakens the benefits of female choice. As a result, the model presented in chapter 8 leads to the prediction that species with the most elaborate sexually selected displays are likely to be associated with sex determining mechanisms that do not allow for facultative sex ratios. Moreover, it leads to the insight that facultative sex ratios based on mate attractiveness may undermine their own evolutionary cause: Indeed, Figure 8.2B on page 210 shows that the evolution of facultative sex ratios eventually eliminates female choice and the resulting variation in male attractiveness.

The elimination of female choice by facultative sex ratios is an example of a mechanism that could apply to a broader range of contexts, in which heritable variation is present within any given trait, but where a conditional strategy increases the fitness prospects of the least successful phenotypes, thereby reducing selection on the trait itself. To illustrate the generality, it is straightforward to think of examples outside the context of mate choice, such as the evolution of parasite resistance versus tolerance (e.g., Roy & Kirchner, 2000; Restif & Koella, 2004). While individuals that are resistant to parasites have a higher survival, infected individuals may overcome this fitness gap by evolving ways to tolerate parasite infections (which is a conditional strategy, since it is not expressed in parasite-free, resistant individuals). Consequently, the evolution of parasite tolerance by infected individuals reduces any fitness differences between resistant and susceptible individuals, thereby weakening selection on parasite resistance. All in all, the complicated relationship between facultative sex ratios and partner quality is thus part of a more general mechanism.

A question left largely unaddressed in the previous chapters is how sensitive this negative feedback between facultative sex ratios and sexual selection is to particular assumptions. The sections below therefore discusses a number of issues that surround the interplay between sex allocation and sexual selection, such as the role of physiological or perceptual constraints on biasing the brood sex ratio, the notion that current

analyses have mainly focused on equilibrium conditions, or the role of sexually antagonistic variation in driving facultative sex ratios. The last part of this chapter explores a different aspect of the interplay between sex allocation and sexual selection, in the context of simultaneous hermaphroditism. As already highlighted in Box 1.1 on page 13, sexual selection may affect the returns on investment in the male or female reproductive functions, which in turn may affect the stability of hermaphroditism. To this end, I briefly explore a model on sexually selected harming traits in hermaphrodites, and the consequences to hermaphrodite sex allocation of the coevolution between such harming traits and different forms of resistance against harm. I finish this chapter by discussing future areas for theoretical research into the interplay between sex allocation and sexual selection in hermaphrodites.

9.1 Facultative sex ratios based on male attractiveness: constraints

Chapter 8 focused on the evolution of facultative sex ratios based on partner attractiveness, while assuming that mothers experience no costs or limitations in biasing the sex ratio of their brood. It is, however, likely that mothers are not always able to precisely achieve the desired sex ratio, for example due to the presence of constraints in the form of chromosomal sex determination (Williams, 1979; Reiss, 1987; Krackow, 2002). In this case, secondary mechanisms such as gamete selection or selective abortion are required to facultatively adjust brood sex ratios dependent on mate quality (Pike & Petrie, 2003; Alonso-Alvarez, 2006). However, these secondary mechanisms may only evolve under rather restrictive circumstances (e.g., Reiss, 1987; Krackow, 2002) and can be highly sensitive to particular costs (Pen *et al.*, 1999).

Alternatively, constraints may arise at the perceptual level (e.g., Schwanz & Proulx, 2008), with mothers being unable to accurately determine the average level of attractiveness of their mates. For example, when mothers adjust the brood sex ratio based on their partner's attractiveness relative to the average attractiveness (s_+ and s_- in model 1 of chapter 7 on page 186), sex ratio adjustment evolves in an asymmetric fashion: females mated to unattractive males produce a sex ratio strongly biased towards daughters, whereas females mated to attractive males only slightly overproduce sons (see also Pen & Weissing, 2000c). In contrast, when the probability of producing a son is given by a sigmoidal function that is only dependent on a male's absolute level of attractiveness – so that females are unable to estimate the male attractiveness relative to the mean attractiveness – (model 2 of chapter 7 on page 188), brood sex ratios are biased to a similar extent by females with attractive (towards sons) and unattractive partners (towards daughters). Both mechanisms of facultative sex allocation lead to a weakening of sexual selection, measured in terms of reduced exaggeration of sexually selected traits. However, the pattern is somewhat less clearcut in case of the sigmoidal function, with only a subset of all simulation runs that exhibit a substantial weakening of sexual selection. Hence, when mothers only have limited information about a male's relative level of attractiveness, more variability in evolutionary outcomes is expected, showing that perceptual constraints deserve a more prominent role in future analyses.

Whatever the nature of these sex ratio constraints, one would expect that levels of exaggeration of female choice and male ornamentation will be higher when costs limit the degree of sex ratio adjustment. As noted by Booksmythe, Schwanz and Kokko (submitted manuscript), measurements on the costs and benefits of sexually

selected characters alone (Kotiaho, 2001) are thus not enough to explain levels of exaggeration of sexually selected traits. In addition, information about the relationship between brood sex ratios and partner quality is necessary to make predictions on the circumstances that favor particular levels of exaggeration of sexually selected traits.

Meta-analyses of over more than 40 studies on sex ratios in birds show that substantial between species variation in the tendency to skew the brood sex ratio exists (West & Sheldon, 2002; Ewen *et al.*, 2004; Cassey *et al.*, 2006; West, 2009). In particular, the dataset presented in Ewen *et al.* (2004) and Cassey *et al.* (2006) provides a promising starting point future studies that aim to link the degree of exaggeration of sexually selected traits to the existence of facultative sex ratios. An initial analysis shows, however, anything but a negative relationship between the effect size of facultative sex ratio adjustment and the degree of ornament exaggeration (see Figure 9.1). Reptiles would provide another suitable group in which sex ratio reaction norms may be linked to partner quality. Substantial between and even within-species variation exists in the tendency to facultatively adjust the sex ratio in reptiles (e.g., Uller *et al.*, 2006; Uller & Olsson, 2006; Warner & Shine, 2008; Pen *et al.*, 2010). However, the majority of studies have aimed to relate these patterns to abiotic conditions (e.g., sex ratios in response to temperature, Valenzuela & Lance, 2004 or population-wide sex ratio biases, Olsson & Shine, 2001), whereas aspects such as partner quality have yet to be considered at a broader scale in reptiles (but see Cox & Calsbeek, 2010; Cox *et al.*, 2011). Recently, a number of datasets have been published that aimed to link sexual dimorphism (a widely used proxy for the exaggeration of sexually selected traits) to several life history traits in reptiles (e.g., Cox *et al.*, 2003), but facultative reptilian sex ratios have not yet been related to the degree of sexual dimorphism in reptiles, providing interesting opportunities for future comparative analyses.

Maternal or offspring control over facultative sex allocation

Apart from specific costs or constraints that may hamper facultative sex allocation, control over sex allocation may also influence the evolutionary outcome. In chapters 7 and 8 only maternal control over sex allocation is considered, whereas offspring control over its own sexual development may be an alternative scenario. The plausibility of offspring control over sex allocation, when sex allocation is based on partner quality, depends on the capability of offspring to "sense" the quality of their father. For example, fathers of a high quality might be strongly correlated with an environmental variable that can be sensed by the offspring (e.g., good territories with high levels of nutrition, which changes the maternal hormonal milieu). Additionally, paternal quality may be inferred from gene expression patterns at early zygotic stages, before sex determination is initiated. For example, zygotic gene expression of the major histocompatibility complex (MHC) (which is often associated to aspects of paternal quality, Wedekind *et al.*, 1995; Wedekind & Penn, 2000; Kempenaers, 2007) starts even before the first zygotic cell division (Sprinks *et al.*, 1993; Doyle *et al.*, 2009), well before sex determination starts in most vertebrates (Wilhelm *et al.*, 2007; Sekido & Lovell-Badge, 2009)

In general, the mechanisms that underlie facultative sex ratios are now starting to be unraveled (Rutkowska & Badyaev, 2008; Uller & Badyaev, 2009). However, we know only little about if either the mother or the offspring is in control over sex determination in those taxa where facultative sex ratios based on partner quality

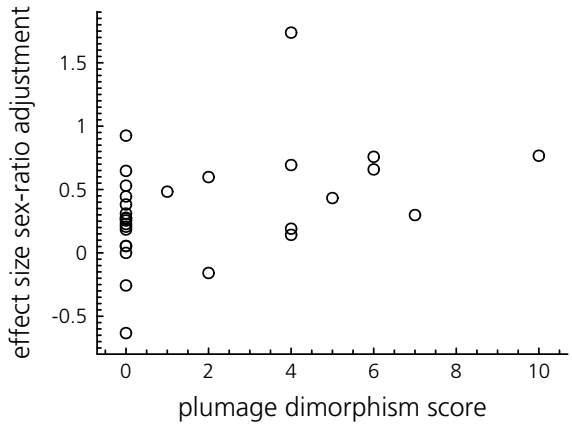

Figure 9.1: A preliminary analysis of the relationship between plumage-color dimorphism and sex-ratio adjustment in birds (Tim Fawcett, unpublished data). Plumage-color dimorphism has been measured according to the method in Owens & Bennett (1994) and Owens & Hartley (1998), in which each of five body regions obtain a dimorphism score between 0 (no dimorphism) and 2 (strong plumage-color dimorphism), and the overall score is then the sum of scores over the five body regions. Sex ratio adjustment is taken as the maximal effect size of sex-ratio adjustment from the dataset of Cassey *et al.* (2006). In contrast to the expected negative relationship between dimorphism and sex ratio adjustment, we find a positive correlation coefficient $r = 0.32$, with a 95% confidence interval obtained by bootstrapping 2000 times given by $r_{0.025} = 0.06$ and $r_{0.975} = 0.64$ (subscripts indicating quantiles of the distribution of correlation coefficients).

have been observed. Therefore, the model discussed below aims to assess if offspring control over sex allocation matters to the coevolution of sex allocation and sexual selection.

It can be shown that, when everything else is equal, incorporating offspring control over sex allocation does not change the equilibrium conditions in eqns. (8.4-8.7) on page 209. This is unsurprising, as parent-offspring over sex allocation has only been shown to occur when individuals interact locally (e.g., Werren & Hatcher, 2000; Pen, 2006) or in the context of sex-specific production costs (see Werren *et al.* (2002), chapter 6 of this thesis). Here, we extend the model by Fawcett *et al.* (2011) (chapter 8 of this thesis) to incorporate sex-specific production costs (see Appendix A9 on page 243), where the successful production of a son costs γ resources relative to producing a daughter. The numbers of sons $m(s_i)$ and daughters $f(s_i)$ produced by a mother that mated with a type i male are then proportional to

$$m\left(s_i\right) = \frac{s_i}{s_i\gamma + 1 - s_i},\ f\left(s_i\right) = \frac{1 - s_i}{s_i\gamma + 1 - s_i},$$

in which the denominator reflects the cost of offspring, averaged over all members of the brood, making the total number of offspring inversely proportional to this denominator.

Maternal control Under maternal control, with sex specific production costs, we obtain a system of selection differentials (see Section A9.1) that is very similar to

eqns. (8.4-8.7):

$$\frac{\partial w}{\partial \hat{p}} = \alpha' \left[\frac{z_{\mathrm{m}1}}{q_1} - \frac{z_{\mathrm{m}0}}{q_0} \right] y_{\mathrm{f}} + \frac{v'_{\mathrm{f}}}{v_{\mathrm{f}}} y_{\mathrm{f}}, \tag{9.1}$$

$$\frac{\partial w}{\partial \hat{t}} = z_{\mathrm{m}1} y_{\mathrm{m}1} \left[\frac{q'_1}{q_1} + \frac{v'_{\mathrm{m}1}}{v_{\mathrm{m}1}} \right], \tag{9.2}$$

$$\frac{\partial w}{\partial \hat{s}_0} = \frac{1-\alpha}{\left(s_0\gamma + 1 - s_0\right)^2 m\left(s_0\right)} \left[\frac{z_{\mathrm{m}0}}{q_0} - \frac{1}{2\bar{f}} \right] y_f, \tag{9.3}$$

$$\frac{\partial w}{\partial \hat{s}_1} = \frac{\alpha}{\left(s_1\gamma + 1 - s_1\right)^2 m\left(s_1\right)} \left[\frac{z_{\mathrm{m}1}}{q_1} - \frac{1}{2\bar{f}} \right] y_f. \tag{9.4}$$

where $\bar{f}$ is the average number of daughters produced by a mother, i.e., $\bar{f} = (1-\alpha)f(s_0) + \alpha f(s_1)$. Regardless of these sex-specific production costs, the outcome is identical as in Fawcett *et al.* (2011): at the sex ratio equilibria $\partial w/\partial \hat{s}_1 = 0$, $\partial w/\partial \hat{s}_2 = 0$, we obtain the condition $z_{\mathrm{m}0}/q_0 = z_{\mathrm{m}1}/q_1$. In turn, this eliminates any positive selection acting on p in (9.1), and leaves only the negative term $(v'_{\mathrm{f}}/v_{\mathrm{f}})y_{\mathrm{f}}$ which involves selection on p towards the naturally selected equilibrium $p = 0$. Hence, the main result by Fawcett *et al.* (2011), that sex allocation based on partner quality will undermine selection on female choice, is robust to sex-specific production costs, at least when sex allocation is under maternal control.

Offspring control over sex allocation While p and t are still controlled by the offspring's mother and father respectively, I now assume that s_0 and s_1 are controlled by the offspring. The selection differentials on p and t in (9.1,9.2) remain thus the same, but the selection differentials on s_0 and s_1 now have a different form (see Appendix A9.2):

$$\frac{\partial w}{\partial \hat{s}_0} = \frac{m\left(s_0\right)}{s_0^2} \left(1 - \alpha + q_0 y_{\mathrm{m}0}\right) \left(\frac{z_{\mathrm{m}0}}{q_0} \left[1 + \frac{1}{2} s_0 \left(1-\gamma\right) \right] - \frac{1}{2\bar{f}} \right), \tag{9.5}$$

$$\frac{\partial w}{\partial \hat{s}_1} = \frac{m\left(s_1\right)}{s_1^2} \left(\alpha + q_1 y_{\mathrm{m}1}\right) \left(\frac{z_{\mathrm{m}1}}{q_1} \left[1 + \frac{1}{2} s_1 \left(1-\gamma\right) \right] - \frac{1}{2\bar{f}} \right). \tag{9.6}$$

Interestingly, at the sex ratio equilibrium under offspring control, $(z_{\mathrm{m}0}/q_0)[1 + (1/2)s_0(1-\gamma)] = (z_{\mathrm{m}1}/q_1)[1 + (1/2)s_1(1-\gamma)]$ holds. This is a different identity than $z_{\mathrm{m}0}/q_0 = z_{\mathrm{m}1}/q_1$ found under maternal control, which led to the elimination of positive, sexual selection on the preference p in eq. (9.3). It can thus be concluded that offspring control and differential costs of producing sons versus daughters ($\gamma \neq 1$, $s_0 \neq s_1$) could potentially maintain positive sexual selection on the preference, leading to nonzero values of p in certain cases.

Numerical simulations in Figure 9.2 illustrate how offspring versus maternal control over sex allocation can lead to a slightly different evolutionary outcome in terms of sexual selection. Focusing on a scenario in which sons are cheaper to produce than daughters, sexual selection is eliminated at the sex ratio equilibria when sex allocation is controlled by the mother (Figure 9.2A,C). In contrast, when sex allocation is controlled by the offspring, exaggerated ornaments and preferences are maintained (albeit at slightly lower levels than in the absence of facultative sex ratios) when s_0

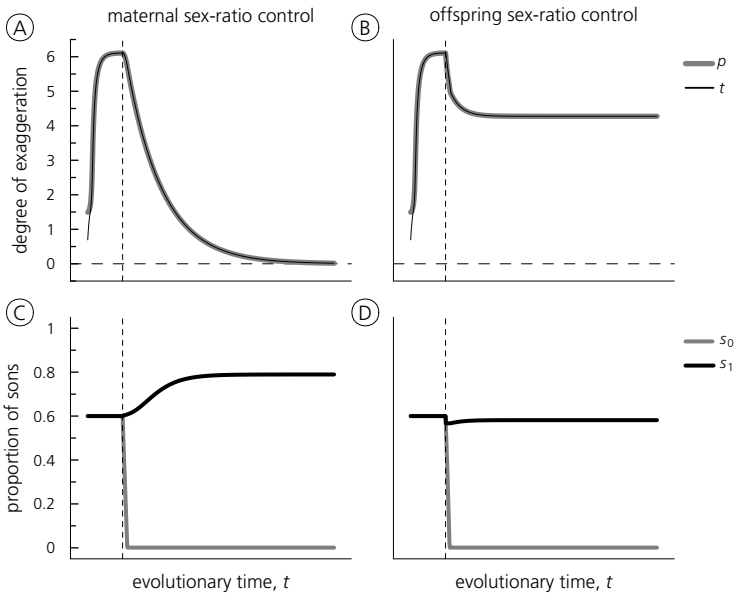

Figure 9.2: Facultative sex allocation based on male attractiveness for maternal (A,C) and offspring control (B,D) over sex allocation, when daughters are 1.5 times more costly to produce than sons ($\gamma = 1/1.5$). s_0 and s_1 are initially at their Fisherian equilibria ($s_0 = s_1 = 0.6$) under the equal allocation principle (Fisher, 1930), but are allowed to evolve from the point indicated by the vertical dotted lines. Male ornaments t and female preferences p first reach stable levels in the absence of sex ratio biases. When flexible sex allocation is allowed to evolve, t and p eventually decline to zero, when sex allocation is controlled by the mother (panels A,C). When sex allocation is controlled by the offspring, t and p still decline slightly, but are eventually maintained at nonzero levels. Parameters as in Figure 8.2.

and s_1 attain their equilibria (Figure 9.2B,D). Interestingly, under offspring control s_0 still evolves towards zero: offspring sired by unattractive fathers are selected to always develop as females, avoiding the cost of becoming an unattractive male. s_1 is still male biased, but attains slightly lower values than when sexual selection is absent. Naively, one might have predicted that whenever sons are the cheaper sex, s_1 should become more strongly male-biased. However, a well-known result is that offspring are selectively favored to develop more often into the more rarer, costly sex, since it has a higher reproductive value (Trivers, 1974). Offspring therefore prefer a substantially lower male bias than their mothers, resulting in a lower number of attractive sons that are produced. However, the lower number of sons that are produced prevents the complete elimination of indirect benefits to their choosy mothers: any attractive male will face less competition from other males (and hence sires more grandoffspring) when compared to the scenario in which mothers control the sex ratio. Because of this larger number of grandoffspring by mothers mated to attractive males, mothers mated to unattractive males cannot entirely recoup their fitness loss by producing only female offspring. To conclude, although offspring control over facultative sex ratios still weakens sexual selection, it prevents its complete elimination.

Parental care biased towards sons versus daughters & the complex interplay between parental care and male attractiveness

Most studies that investigate sex allocation based on paternal quality assess the presence of facultative sex ratios, since these are relatively straightforward to measure. However, when faced with constraints in biasing the sex ratio, mothers can also skew the amount of parental care given to offspring of a particular sex, based on the attractiveness of their father (Maynard Smith, 1980; Charnov, 1982). Such sex-biases in maternal care have now been assessed in a number of empirical studies (e.g., Limbourg *et al.*, 2004; Ligon & Hill, 2010; Pariser *et al.*, 2012). Similar to previous results on facultative sex ratios however (see West, 2009, and references therein), results are generally mixed with some studies finding biased maternal investment towards sons sired by attractive males (Ligon & Hill, 2010), whereas other studies do not find any sex-biases in maternal investment at all (Limbourg *et al.*, 2004).

Such mixed results are symptomatic of the complexities that surround research in the broader context of parental care based on mate attractiveness. The question if mothers should adjust their parental care based on offspring sex is a subset of a broader area of research that focuses on differences in maternal investment to offspring sired by fathers of different qualities. Such patterns of "differential allocation" (Burley, 1986b) are receiving a growing amount of attention (reviewed in Sheldon, 2000; Qvarnström & Price, 2001; Ratikainen & Kokko, 2010). Empirical studies have demonstrated that patterns of maternal investment based on paternal attractiveness vary considerably between taxa. For example, a substantial number of studies have found that mothers increase investment to those offspring sired by attractive males (e.g., Cunningham & Russell, 2000; Kolm, 2001; Kotiaho *et al.*, 2003; Limbourg *et al.*, 2004; Gilbert *et al.*, 2012). However, other studies have found a reverse pattern, in which mothers increase investment to those offspring sired by unattractive males instead (e.g., Saino *et al.*, 2002; Navara *et al.*, 2006; Bolund *et al.*, 2009). What factors underlie this variation in parental investment based on paternal attractiveness is currently poorly understood.

Only recently, the evolution of facultative patterns of maternal care based on male quality has been formally modeled by Harris & Uller (2009). This model shows that mothers (who mate multiply) typically invest more in offspring sired by high quality fathers, than those sired by low quality fathers. This is because offspring that inherit their father's high quality genes have a higher reproductive value than offspring with low quality genes, so that maternal care is biased towards those offspring with the highest reproductive value (Trivers & Willard, 1973). Only when the baseline level of offspring survival is high anyway, Harris & Uller (2009) find the reverse pattern, in which offspring from low quality fathers receive more resources than offspring from high quality fathers. In this case, offspring reproductive values are not substantially affected by paternal quality, so that the slightly lower reproductive value of offspring from low quality fathers can easily be compensated through additional maternal investment.

Although the model by Harris & Uller (2009) is an important first step, it leaves a number of crucial aspects unaddressed (see also Ratikainen & Kokko, 2010). Here, I highlight two aspects that are particularly important to understand the evolution of parental care based on male attractiveness. First, Harris & Uller (2009) acknowledge that their model only focuses on the evolution of facultative maternal care, whereas

the coevolution of mate choice and genetic quality (or male attractiveness) has not been considered. It will be particularly interesting to investigate the interaction between maternal care and indirect benefits associated with Fisherian or good-genes processes. Although other coevolutionary studies have shown that maternal effects could potentially enhance the benefits of female choice by increasing the phenotypic correlation between male quality and female preference (Wolf *et al.*, 1997, 1999), it remains to be seen how robust this is to facultative maternal care. Indirect benefits are typically small, and if maternal effects are able to compensate for a loss in heritable male quality, facultative maternal care may undermine the benefits of female choice in a manner reminiscent to the pattern found in chapter 8 of this thesis.

Even when parental effects (such as facultative maternal care) would not eliminate female choice, the resulting coevolutionary dynamics deserve a systematic analysis: it is well-established that parental effects can lead to complicated evolutionary dynamics, such as limit cycles (Lande & Kirkpatrick, 1990; Inchausti & Ginzburg, 2009) and rapid evolutionary change at the ecological timescale (Räsänen & Kruuk, 2007; Badyaev, 2008), which might be a crucial in understanding the observed variation in sexually selected traits (Pomiankowski & Møller, 1995; Miller & Moore, 2007).

The second aspect omitted in the model by Harris & Uller (2009) is the possibility that both parents provide care. As suggested by Burley (1986b), allocation of maternal care may also facultatively depend on male quality, when high quality males are more prone to desert their partners (i.e., due to a higher prospective mating rate) than males of a poor genetic quality. In order to compensate for the lack of paternal care, females mated to high quality males may then be forced to provide more care than females mated to low quality males. Alternatively, the reverse scenario in which mothers provide more care to offspring from low quality males could occur when females choose their mates based on the quality of paternal care (the good-parent process: Hoelzer, 1989; Price *et al.*, 1993): in this case, high quality males provide a lot of care, so that their female partners may reduce their own levels of care accordingly. In contrast, low quality males provide less care, forcing their partners to increase their level of care. Again, coevolutionary models are essential to assess these verbal predictions, in which characters involved in mate choice coevolve with traits that determine maternal and paternal care. Such an approach is necessarily complex and multifaceted, since assumptions are also required about how parents bargain over the level of care (e.g., Houston & Davies, 1985; McNamara *et al.*, 2003; Lessells & McNamara, 2011), how paternal care is distributed over the various mating partners of a particular male (Tazzyman *et al.*, 2012) and the potential of synergistic interactions between parents when caring together (e.g., Kokko & Johnstone, 2002).

Coevolutionary aspects and the consequences of uniparental versus biparental care are thus important to understand the variation in parental care to broods sired by fathers of different quality. But what about biases in maternal care towards offspring of a particular sex? Naively extrapolating the model described in chapter 8 would lead us to predict that mothers mated to high quality fathers should invest more in sons than in daughters, whereas mothers mated to poor quality fathers should invest more in daughters than sons. However, also here complications arise due to the transgenerational consequences of parental effects: when only females care for the brood, reproductive values of daughters are likely to increase faster with increased care than the reproductive value of sons (Leimar, 1996). This is because daughters that received more maternal care are likely to give more maternal care to

their offspring as well, so that increased maternal care to daughters results in more grandoffspring than increased maternal care to sons. However, it remains to be seen if such sex-biases in care have consequences to the evolution of mate choice, since females still reap the (indirect) benefits of mating with attractive males that produce attractive sons, even when these sons would not benefit from any care at all.

This section only scratched the surface of the many complexities involved in the interaction between parental care and the sexual selection process (see Kokko & Jennions, 2008; Ratikainen & Kokko, 2010 for in-depth reviews). Nonetheless, it is clear that the strong empirical interest in this field of research is not balanced by the availability of clearcut formal predictions. Models that focus on the coevolution of parental care and sexually selected will therefore be one of the more fruitful applications of sexual selection theory in the near future.

Facultative sex ratios based on partner choice: nonequilibrium dynamics

The analysis of facultative sex ratios and sexually selected characters so far only considered conditions at equilibrium, in which display traits or preferences remain at a stable level of exaggeration for prolonged periods of evolutionary time. However, sexual selection is often associated with rapid turnovers in display traits (Wiens, 2001) and striking patterns of divergence between closely related populations (Pomiankowski & Møller, 1995). As highlighted by previous analyses (e.g., Iwasa & Pomiankowski, 1995; Gavrilets *et al.*, 2001; Houle & Kondrashov, 2002; Van Doorn & Weissing, 2006, chapter 3 of this thesis), continuous and cyclical coevolution of preferences and display traits is not unlikely, in particular when there is weak survival selection against display or harming traits and when female preferences or resistance traits are characterized by substantial levels of genetic variation. The impact of nonequilibrium on the evolution of facultative sex ratios has, however, not been assessed so far.

Individual-based simulations provide a straightforward approach to analyze the role of nonequilibrium dynamics on facultative sex ratios (e.g., Figure 2.3D on page 42). Figure 9.3 provides an example run from an individual based simulation model of Fisherian sexual selection (see Chapter 7 for a description) with weak costs of ornamentation. Display traits t and preferences p are allowed to attain both positive and negative values, while also allowing for the evolution of two sex allocation loci s_+ and s_-. s_+ is the proportion of sons produced by mothers mated with attractive males (i.e., males with ornaments larger than the average ornament when $p \geq 0$ and males with ornaments smaller than the average ornament when $p < 0$), while s_- is the proportion of sons produced by mothers mated with unattractive males.

As illustrated by Figure 9.3, the coevolutionary dynamics of t, p and both sex ratio loci are generally complicated and demand a systematic analysis that is beyond the scope of this chapter. The most important aspect to notice from the example simulations in Figure 9.3A is that the maximum levels of exaggeration of t and p are not reduced when coevolving with s_- and s_+ (compare light and dark lines), which contrasts with the equilibrium predictions made in chapter 8. The degree of sex ratio bias (measured by s_- and s_+) fluctuates over time, but in general females mated to attractive males bias their brood sex ratio towards sons ($s_+ > 0.5$), whereas females mated to unattractive males bias their sex ratio towards daughters ($s_- < 0.5$) (see Figure 9.3C). Fluctuations in facultative sex ratios based on male attractiveness (by

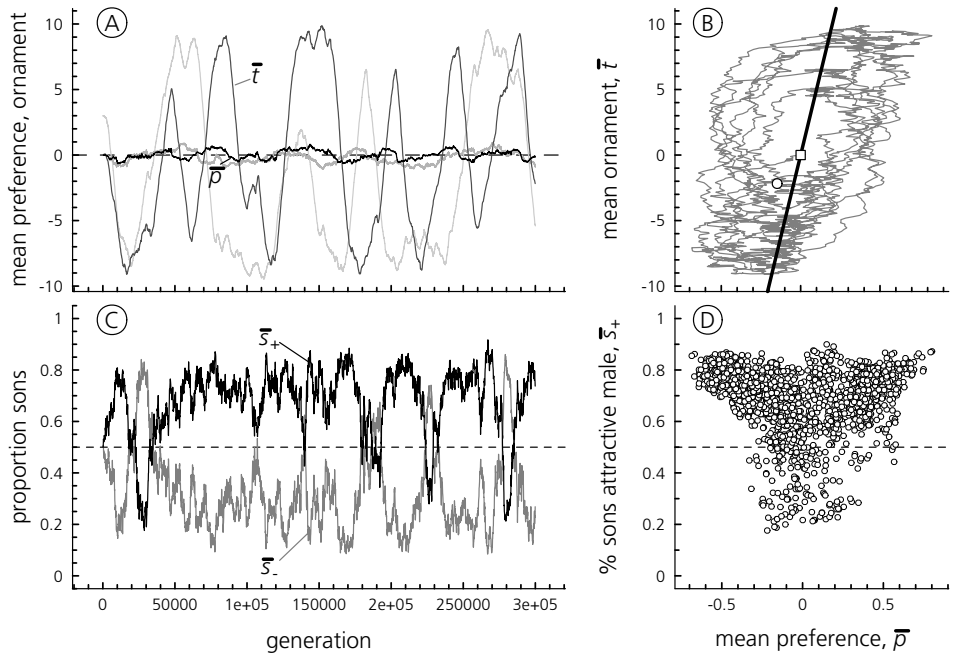

Figure 9.3: The evolution of sex ratio adjustment when ornaments and preferences coevolve in a continuous fashion. Panel A: light grey lines reflect the course of evolution of t (ornaments) and p (preferences) in the absence of facultative sex ratios, whereas darker lines reflect values of t and p in the presence of facultative sex ratios. Panel B: a phaseplot of t and p in the presence of facultative sex ratios. Panel C: facultative sex ratio loci s_+ and s_-. Panel D: relationship between the mean value of p and values of the sex ratio loci s_+. Each dot represents the value of $(\bar{p}, \bar{s}_+)$ for each generation. When mean preference $\bar{p}$ is weak, $\bar{s}_+$ is highly variable. When $\bar{p}$ is relative strong however, $\bar{s}_+$ is consistently male biased. Parameters: $a = 1,\ b = 0.0025,\ c = 0.01,\ \mu_t = 0.05,\ \mu_p = 0.05$.

means of s_- and s_+) occur because male attractiveness fluctuates as well over time. Only whenever t reaches its most extreme level of either positive or negative exaggeration, do s_+ and s_- appear to attain their equilibria, thereby eliminating indirect sexual selection on p, and reducing the levels of s_- and s_+. Female preferences (and hence male ornamentation) then decrease towards their naturally selected optima at 0. Because of weak selection, however, p may overshoot the naturally selected optimum, leading to exaggeration in the opposite direction, reinstating selection for increased sex ratio biases based on male attractiveness.

The resulting fluctuations in s_- and s_+ can even lead to transient scenarios in which females mated to attractive males overproduce daughters. However, the latter scenario only occurs when preferences are very weak (Figure 9.3D) so a strong association between paternal attractiveness and offspring sex ratios is expected to be absent in such cases. Temporary fluctuations in s_- and s_+ might nonetheless be an explanation for the observed variety of sex ratio results (see West, 2009), where different studies on the same species found that females mated to attractive males either overproduce sons, show no sex ratio bias at all, or overproduce daughters.

9.2 Sexual selection and sex allocation in hermaphrodites

The vast majority of studies that investigate the evolution of sexually selected characters are strikingly biased towards organisms with separate sexes (gonochorists). As shown in chapter 3 of this thesis, models of sexual selection based on gonochorists cannot be directly extrapolated to hermaphroditic organisms: first, costs of sexually selected characters are typically associated to a particular sex in gonochorists, with females often being the choosy sex and males the sex that express display traits (but see Clutton-Brock, 2009). As a result, costs of choice or costs of ornamentation are typically only paid by one sex. Since hermaphrodites unite both sexes in one individual, such costs are likely to lose their sex-specific association, leading to situations in which individuals incur both costs during their lifespan. Second, since hermaphrodites combine both sexes within one individual, they are more likely to exhibit mating behaviours like mutual choice (e.g., Vreys & Michiels, 1997; Michiels, 1998), which – as shown in chapter 3 – can sustain exaggeration of sexually selected characters under far larger levels of costs when compared to scenarios in which choice is unilateral.

One important assumption of the model in chapter 3 is that investment in male versus female reproductive functions is fixed, whereas a more realistic models should include the relative investment in both sex functions. As highlighted in Box 1.1, however, hermaphroditism is only evolutionarily stable when returns on investment in at least one sex function are decelerating (i.e., decelerating fitness gain curves) (Charnov *et al.*, 1976). Moreover, such decelerating fitness gain curves are typically associated with the male function, since this i) leads to outcomes where a surplus of resources is invested in the female function, resulting in a larger intrinsic population growth in comparison to gonochoristic populations (Charnov, 1979a; Arnold, 1994) and ii) from the limited number of studies that measured returns on investment in the female function, evidence for limiting returns on female investment is relatively sparse (Schärer, 2009). Scenarios that lead to decelerating returns through the male function are, for example, a low density of mates (Ghiselin, 1969) or poor mate searching capabilities (Puurtinen & Kaitala, 2002). Such decelerating returns on the male function limit the evolutionary scope for pre or postcopulatory characters that achieve an increasing number of fertilizations in the male role (Charnov, 1979b; Michiels *et al.*, 2009).

The theoretical prediction that hermaphrodites have limited evolutionary potential for characters affecting mating success of the male role is, however, at odds with the observation that hermaphrodites are well known for a striking variety of sperm traits (e.g., Schärer *et al.*, 2011), elaborate genital morphologies (e.g., Gerhardt, 1933; Hoch, 2009), love darts (Schilthuizen, 2005; Koene & Schulenburg, 2005; Chase & Blanchard, 2006) or complicated mate assessment behaviours (Vreys & Michiels, 1997; Lüscher & Wedekind, 2002). A number of mechanisms have therefore been postulated that could explain the occurrence of characters that increase male mating success, while at the same time warranting the stability of hermaphroditism. For example, a recent model by Schärer & Pen (in press) focused on the coevolution of investment in sperm relative to eggs, combined with investment in pre or postcopulatory traits. Their model shows that stochastic fertilization biases can lead to female-biased sex allocation (due to local sperm competition, Charnov, 1980; Schärer, 2009) and stable hermaphroditism, even when when substantial investments are made into

male postcopulatory traits (e.g., penises or male genital appendages that increase fertilization success). However, the same model by Schärer & Pen also shows that precopulatory traits such as display traits are still strongly limited by any decelerating returns through the male function. The evolution of these precopulatory mating traits in hermaphrodites (e.g., chapter 3) is therefore most likely to occur when sex allocation is constrained from evolving towards pure sexes, although it remains to be seen if this conclusion extends to all forms of precopulatory mating behaviours and when a broader range of trade-offs are assumed (see section "future models" below).

Another hypothesis on the evolution of elaborate postcopulatory characters in hermaphrodites relies on the evolution of collateral harming traits in hermaphrodites (Michiels & Koene, 2006; Preece *et al.*, 2009). Harm traits increase fertilization success of a sperm donor by reducing a sperm recipient's survival, and hence the probability that a sperm recipient engages in rematings with other sperm donors. Examples of harm traits are harassment (e.g., Shine *et al.*, 2005; Gay *et al.*, 2009), traumatic insemination (e.g., Smolensky *et al.*, 2009) or seminal fluids that contain toxins (Chapman *et al.*, 1995; Kuijper *et al.*, 2006). Often, a distinction is made between collateral harm and adaptive harm (Johnstone & Keller, 2000; Morrow *et al.*, 2003), where collateral harm evolves as a side effect of traits involved in conflicts between sperm donors (e.g., seminal toxins that are not only detrimental to competing sperm, but also to the sperm recipient). Adaptive harm refers to the evolution of traits that are selectively favored to reduce a sperm recipient's survival, resulting in a fertilization advantage of the sperm donor due to a reduced remating rate of the sperm recipient. Whereas adaptive harm appears to evolve only in restrictive circumstances (Johnstone & Keller, 2000; Michiels & Koene, 2006, but see Lessells, 2006), collateral harm is predicted to evolve rapidly in hermaphrodites (Michiels & Koene, 2006). Moreover, when adaptive harm is associated with characters involved in sperm displacement, hermaphroditism appears to be stable even when sperm donors engage in a considerable number of matings (Preece *et al.*, 2009).

Current studies on the evolution of these harming traits have exclusively focused on the coevolution of harm characters and sex allocation (Preece *et al.*, 2009). From work on gonochorists, however, it is well known that harm imposed by sperm donors may also select for resistance traits that reduce the effects of harm (e.g., Rice, 1996; Wigby & Chapman, 2004; Rönn *et al.*, 2007). Such resistance traits are predicted to substantially alter the evolutionary outcome of harm traits (Gavrilets *et al.*, 2001; Rowe *et al.*, 2005), either leading to substantial exaggeration of harm traits to overcome resistance, or to an evolutionary standstill in which females are insensitive to harm. The coevolution of resistance and harm traits, and the potential effects on sex allocation, has however not been explored in hermaphrodites. The following section therefore explores a model on harm in hermaphrodites.

The evolution of harm and resistance in hermaphrodites

Here we extend a previous model by Preece *et al.* (2009) on the evolution of a harmful mating tactic that promotes sperm precedence, by allowing for the evolution of a resistance trait that reduces the effect of harm. A central component of the model by Preece *et al.* (2009) is the sperm displacement function $\phi(s,h,a)$, which describes the total fraction of sperm that a focal male displaces from the sperm stores of a sperm recipient (see also Charnov, 1996; Pen & Weissing, 1999; Greeff & Michiels,

1999b), which is a function of the proportion of recourses invested in sperm s by the focal male, the level of harm h expressed by the focal male and the level of resistance a expressed by the sperm recipient. Here, I use Charnov's (1996) so-called 'Case 2' function, that describes a scenario in which sperm from previous males, currently stored by the recipient, is mixed with incoming sperm from a new mating, after which a fair sample of old and new sperm is stored again. We then have

$$\phi(s,h,a) = \frac{s\,(\delta + hz(a))}{s(\delta + hz(a)) + 1},$$

where δ is the ratio of sperm transferred by the donor, divided by the total sperm stored in the recipient (Charnov, 1996) and h is the harm trait which causes an additive increase in the amount of sperm displaced from other sperm donors, but also collaterally harms the sperm recipient (e.g., Parker, 1979; Morrow *et al.*, 2003). Lastly, $z(a)$ is the effect of the resistance trait a on sperm displacement, where I use the function $z(a) = \mathrm{e}^{-ka}$ to describe how much effect the harm trait of the sperm donor has on sperm displacement, for a given investment in the resistance trait a. The parameter $k = (0,1)$ allows for the incorporation of different resistance phenotypes: for example, sperm recipients may possibly release antibodies that bind and neutralize toxic seminal fluid proteins, thereby both eliminating the effect of toxins on sperm displacement and reducing harm to the sperm recipient. For such cases, $k = 1$. Alternatively, resistance may simply reduce the effect of harm on the recipient's survival without affecting sperm displacement ($k = 0$). For example, sperm recipients may produce a thicker vaginal wall that reduces the negative effects of seminal fluid toxins to the sperm recipient, while toxins still have a role in sperm displacement.

Survival of a particular sperm recipient is given by

$$\sigma(h,a) = \left[\frac{1}{1 + hy(a)}\right]^{m},$$

where the level of harm h by a particular sperm donor is inversely related to the survival probability σ of the recipient. m is a parameter that scales the severity of harm on survival and $y(a)$ the mitigating effect of resistance on the level of harm, which is given by the function $y(a) = \mathrm{e}^{-a}$.

Following the model by Preece *et al.* (2009), it can be shown that the fitness w_{f} of a rare, mutant sperm recipient with character values $\hat{a}$, $\hat{s}$ and $\hat{h}$ in a resident population with character values $a,\ s,\ h$ is given by

$$w_{\mathrm{f}}\left(\hat{a},\hat{s},\hat{h},a,s,h\right) = R\left(1 - \hat{s} - c_a\left(\hat{a}\right) - c_h\left(\hat{h}\right)\right)\sum_{i=1}^{n}\sigma\left(h,\hat{a}\right)^{i}. \tag{9.7}$$

The sum on the right hand side in eq. (9.7) reflects the survival of the recipient over the number of n matings in which she is engaged, which is multiplied by the fecundity of the sperm recipient. A sperm recipient's fecundity is given by the proportion of the reproductive budget R that is neither invested in sperm $\hat{s}$ nor in harm or resistance traits, which have respective cost functions $c_a(\hat{a}) = 1 - \mathrm{e}^{-\gamma_1\hat{a}}$ and $c_h(\hat{h}) = 1 - \mathrm{e}^{-\gamma_2\hat{h}}$. Whereas Preece *et al.* (2009) make the assumption that harm traits are cost-free, the current model makes the more realistic assumption that both harm and resistance traits impose certain costs to their bearers (see also Gavrilets *et al.*, 2001; Rowe *et al.*,

2005; Poiani, 2006).

Similarly, fitness through the male role of a mutant $(\hat{a},\hat{s},\hat{h})$ individual can be derived as (see (Greeff & Michiels, 1999b))

$$w_{\mathrm{m}}\left(\hat{a},\hat{s},\hat{h},a,s,h\right) = R\left(1-s-c_a\left(a\right)-c_h\left(h\right)\right)\phi\left(\hat{s},\hat{h},a\right)\sigma\left(\hat{h},a\right) \times \sum_{i=1}^{n}\left[\sigma\left(h,a\right)^{i-1}\sum_{j=0}^{n-i}\sigma\left(h,a\right)^{j}\left(1-\phi\left(s,h,a\right)\right)^{j}\right], \tag{9.8}$$

where the first sum is taken over the mutant's mating partners that have mated $i-1$ times with other, resident sperm donors before receiving sperm from the mutant. The second sum in (9.8) reflects the $j=n-i$ subsequent matings with other, resident sperm donors after the recipient has mated with the mutant sperm donor. By calculating $w = w_{\mathrm{f}} + w_{\mathrm{m}}$ and solving for the following system of equations we can obtain the equilibrium values of $a,\ s$ and h

$$\left.\frac{\mathrm{d}w}{\mathrm{d}a}\right|_{\mathbf{x}=\hat{\mathbf{x}}} = 0,\ \left.\frac{\mathrm{d}w}{\mathrm{d}s}\right|_{\mathbf{x}=\hat{\mathbf{x}}} = 0,\ \left.\frac{\mathrm{d}w}{\mathrm{d}h}\right|_{\mathbf{x}=\hat{\mathbf{x}}} = 0,$$

where $\mathbf{x}$ is the character vector $\mathbf{x} = [a,s,h]^{\mathrm{T}}$ with T denoting transposition.

The coevolution of harm and resistance: results Figures 9.4A-C depict the evolution of harm h and the proportion of resources invested in sperm s, when resistance a is absent. Harm is particularly likely to evolve when both δ and m are modest (Figure 9.4A), whereas in other regions of parameter space hermaphroditism is either unstable (i.e., $s=0$) or nonzero levels of harm do not evolve ($s>0,\ h=0$). By means of example, Figures 9.4B,C depict a single numerical iteration of the evolution of both harm and sex allocation towards nonzero levels.

In the presence of resistance (Figures 9.4D-I), the coevolutionary outcome strongly depends on the type of resistance that evolves. If resistance eliminates the effect of harm on survival, but not on sperm displacement (Figures 9.4D-F), coevolution between both harm and resistance can lead to exaggeration of both characters (panel E), with harm being expressed to substantially higher levels than when resistance is absent. The evolution of resistance increases a sperm recipient's survival, leading to more rematings and hence more competition among sperm from different males. As a result, it pays for sperm donors to invest more in the harm trait to displace more rival sperm. The effects on sex allocation are, however, negligible, despite a decrease of the overall reproductive budget with $\approx 20\%$ due costs of developing harm and resistance traits. To summarize, when resistance only reduces the detrimental effect of harm on survival without affecting the effect of harm on sperm displacement, harm and resistance coevolve to substantial levels of exaggeration.

The situation is different, however, when resistance not only eliminates the deleterious effects of harm on survival, but also the effects of harm on sperm displacement (panels G-I). In a situation in which a pre-existing harm trait is invaded by a resistance trait, either resistance and harm coexist for high levels of δ. The levels of harm are substantially lower, however, than in the absence of the resistance trait. Moreover, when δ is low, the evolution of resistance leads to the complete elimination of harm (panel H). For low values of the sperm displacement coefficient δ, however,

hermaphroditism ($0 < s < 1$) is not stable in the absence of harm when the number of matings is limited (see Figure 2 in Greeff & Michiels, 1999b). Hence, the invasion of resistance ultimately leads to the elimination of hermaphroditism altogether.

Although the current model only scratches the surface on the coevolution of harm and resistance in hermaphrodites, the model demonstrates that counterevolutionary responses can change predictions from previous models in which harm is allowed to evolve unchecked (Michiels & Koene, 2006; Preece *et al.*, 2009). Moreover, the current analysis shows that the particular mechanism of resistance (see also Lessells, 2006) cannot be ignored when considering the stability of hermaphroditism. In particular, stable hermaphroditism is most likely for those resistance traits that only affect survival, while maintaining harmful effects on sperm displacement. The current model makes the testable prediction that palliative traits, such as thicker walls of reproductive organs, are more likely to be present in hermaphrodites than counteradaptations that directly target the harm trait itself, such as profound structural changes in reproductive morphology, or antibodies that eliminate toxins. To conclude, coevolutionary models such as these are timely and necessary to understand the evolution of sexually selected traits, and hermaphrodites are no exception in this context.

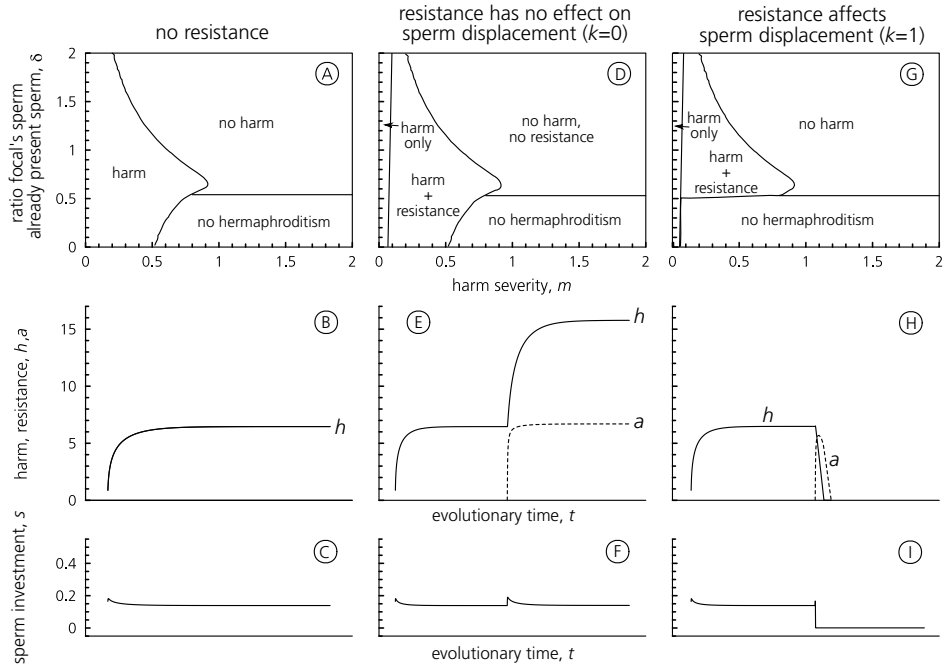

Figure 9.4: The coevolution of harm, resistance and sex allocation in hermaphrodites. Panels A-C: only harm h and sex allocation s evolve. Panels D-F: harm, resistance a and sex allocation coevolve. Resistance only eliminates the effect of harm on survival, but does not effect of harm on sperm displacement ($k = 0$). Panels G-I: harm, resistance a and sex allocation coevolve. Resistance both eliminates the effect of harm on survival and the effect of harm on sperm displacement ($k = 1$). Parameters panels A,D,G: $\gamma_1 = \gamma_2 = 0.01$, $n = 4$. Additional parameters for the numerical iterations: $\delta = 0.3$, $m = 0.4$.

9.3 Sexual selection in hermaphrodites: future steps

The analysis of coevolving harm and resistance in the previous section shows how the evolutionary stability of hermaphroditism depends on the particular mechanisms with which postcopulatory traits expressed in sperm donors and recipients interact. Hence, coevolutionary models, in which both sex allocation as well as sexually selected characters evolve are elementary to understand the evolution of sexually selected traits in hermaphrodites. In that respect, a more elaborate theory of sexual selection for hermaphrodites would be desirable. Here I briefly set out a number of future models to arrive at such a theory, in order to improve our understanding of the aforementioned variety of sexually selected traits in hermaphrodites.

Trade-offs between pre- and postcopulatory investment

As demonstrated by the model of Schärer & Pen (in press), the evolution of precopulatory traits in hermaphrodites that increase the number of mates of a particular sperm donor is met with considerable difficulty: investment in precopulatory display traits (while trading off with postcopulatory investment) is only favored when it gives a sperm donor a very strong increase in the number of matings relative to sperm donors lacking the trait. Moreover, the same analysis shows that investment in a precopulatory mating character often causes male-biased investments, which makes hermaphroditic populations vulnerable to the invasion of pure females (which have a higher intrinsic growth rate).

However, there are some unresolved issues regarding the evolution of precopulatory traits in hermaphrodites: as mentioned by Schärer & Pen (in press), the same strong increase in the number of matings is required for the evolution of precopulatory display traits in gonochorists, at least when investment in precopulatory display trades off with postcopulatory investment (i.e., sperm or accessory characters), as opposed to the usually assumed trade-off between precopulatory display and survival (e.g., chapter 2 of this thesis). Indeed, more explicit models based on the ESS framework presented in chapter 8 in which both preferences and ornaments evolve (and trade off with postcopulatory investment) in gonochorists show that investment in precopulatory traits rapidly decays with even slight levels of polyandry (Kuijper, Pen and Engqvist, manuscript in preparation, see also Collet *et al.* 2012). To summarize, the difficulties surrounding evolution of precopulatory display traits is not unique to hermaphrodites, so that a more systematic comparison between hermaphrodites and gonochorists is required.

As a first step, models are required in which precopulatory investment either trades off with postcopulatory investment or trades of with survival, in both gonochorists and hermaphrodites. For example, a proportion z of the costs of a precopulatory character is paid in terms of juvenile survival costs, while the remainder $1-z$ of the precopulatory trait's costs are subtracted from the postcopulatory budget. An intermediate value of z ($0 < z < 1$) would then reflect a scenario in which the early morphological development of a precopulatory trait involves survival costs during the juvenile stage, whereas the effort spent on behaviorally displaying of a precopulatory trait may reduce investment in seminal fluids or other postcopulatory traits. It is straightforward to predict that when precopulatory traits trade off more strongly with survival than postcopulatory investment, the more likely are precopulatory traits to evolve in polyandrous contexts. However, the exact relationship between pre- and

postcopulatory investment for different levels of z will be more difficult to predict, since this relationship is not linear, as precopulatory investment affects the number of matings a male undertakes and hence its level of sperm competition (e.g., Williams *et al.*, 2005; Fromhage *et al.*, 2008; Tazzyman *et al.*, 2009). Subsequently, one can then compare the relationship between pre and postcopulatory investment between gonochoristic versus hermaphroditic lifestyles (i.e., linear/accelerating versus decelerating male fitness gain curves, see Box 1.1). Here we would predict that gonochorists are less sensitive to any costs of precopulatory trait that affect the postcopulatory budget, simply because hermaphrodites not only pay investment in sperm or accessory traits, but also investment in eggs from this budget. One would thus predict that hermaphrodites are thus less likely to develop precopulatory traits than gonochorists, but a quantification of this statement is essential to understand the scope for sexually selected traits in hermaphrodites. It also remains to be seen for what values of z, sex allocation in hermaphrodites tends to be male-biased when investing in precopulatory characters (Schärer & Pen, in press), which affects the stability of hermaphroditism against the invasion of pure-sex individuals.

Mate choice: behavioral assumptions

Chapter 3 of this thesis shows that the particular nature of mate choice behaviours, such as unilateral versus mutual choice, can lead to different evolutionary outcomes: in comparison to unilateral choice, mutual choice leads to the evolution of preferences for display characters over a broader range of costs, but typically leads to smaller degrees of exaggeration. Since simultaneously hermaphroditic individuals contain both sexes, mutual choice may be more likely to occur in hermaphrodites than gonochorists (Michiels, 1998), so behaviours such as mutual choice should be considered when modeling the evolution of precopulatory behaviours in hermaphrodites. A central assumption of the model in chapter 3, is however, that mutual choice is based on the Fisher process, in which fecundity of the female function is assumed to be invariant. Models on gonochorists show, however, that mutual choice is more likely to evolve when females vary in fecundity: although mutual choice reduces a male's potential mating rate (males will only prefer to mate with a subset of females), this is compensated by the higher fecundity of his mating partners (Parker, 1983; Kokko & Johnstone, 2002; Servedio & Lande, 2006). It would be interesting to see how interindividual variation in the number of unfertilized eggs affects the evolution of mutual choice in hermaphrodites. Mutual choice based on fecundity may, for example, lead to assortative mating based on fecundity-related proxies such as size (Johnstone *et al.*, 1996), which has been observed in a number of hermaphrodites (Vreys & Michiels, 1997; Lüscher & Wedekind, 2002). As a result, mutual choice based on fecundity may possibly restrict the number of potential matings of sperm donors, since it limits the number of partners to those that have a similar size. As a result, male mating success may be limited by mutual choice, which could potentially lead to the evolution of stable hermaphroditism (see Box 1.1). Mutual choice should thus be considered in future models on sexual selection in hermaphrodites.

Another point to make about behavioral mechanisms are the behaviours that are often understood to be associated to precopulatory mating traits. Precopulatory traits that have been currently considered lead to an increase in the number of fertilizations (e.g., Puurtinen & Kaitala 2002, Schärer & Pen in press, chapter 3), whereas

characters that maintain a given number of fertilizations, through monopolization of mating partners have not been considered. Examples of such monopolization traits expressed in sperm donors may be nuptial gifts or mate guarding behaviours, such as badges of status (Veen, 2008) or armaments. Returns from investment in such monopolization traits are likely to be characterized by decelerating functions, since the number of fertilizations will be limited by the number of sperm recipients that can be monopolized by a single male. As a result, stable hermaphroditism might be more likely in the context of mate guarding characters, when compared to other precopulatory traits.

Mate choice at the postcopulatory level

Hermaphrodites are well known for their substantial variation in postcopulatory traits (Michiels, 1998; Anthes, 2010), such as a large variety of sperm traits (e.g., Michiels 1998; Schärer *et al.* 2011) and complex genital morphologies (Michiels, 1998). Quite a number of models have analyzed the evolution of postcopulatory characters in hermaphrodites, either related to sperm displacement (Charnov, 1996; Pen & Weissing, 1999; Greeff & Michiels, 1999b; Preece *et al.*, 2009), accessory characters such as penises that improve fertilization success (Michiels *et al.* 2009, Schärer & Pen in press), or characters involved in cryptic female choice, where characters expressed in sperm recipients bias the paternity among sperm donors (van Velzen *et al.*, 2009). It is increasingly understood that postcopulatory traits expressed in the male function often evolve in a correlated fashion with characters associated to the female function. For example, the number of sperm transferred has been shown to be correlated to the size of the spermatheca across different populations of the helicid land snail *Arianta arbustorum* (Beese *et al.*, 2006), while in Ophistobranches, seminal fluid producing glands correlate with the size of the bursa copulatrix, which is associated to sperm digestion (Anthes *et al.*, 2008). How the coevolution of male and female postcopulatory traits affects conclusions from aforementioned models in which such traits are often considered to evolve in isolation is only starting to be considered (Greeff & Michiels 1999b; van Velzen *et al.* 2009, see previous section). Models on the evolution of cryptic female choice, for example, focused on the coevolution between investment in sperm by sperm donors and the quantity of sperm from undesirable donors that is removed by sperm recipients (van Velzen *et al.*, 2009), showing that this could lead to interesting coevolutionary phenomena such as cyclic evolution of cryptic choice and sperm investment, which may explain the substantial degree of interpopulation variation in postcopulatory characters (e.g., Beese *et al.*, 2006). Nonetheless, existing models on cryptic female choice assume that sperm donors only differ in a non-heritable character that make them either 'desirable' or 'undesirable', on the basis of which cryptic female choice evolves (Ball & Parker, 2003; van Velzen *et al.*, 2009). It remains to be seen how heritable male attractiveness changes conclusions from previous models, since it allows investment in sperm to coevolve with characters that relate to the attractiveness of a sperm donor. An attractive sperm donor may be likely to invest fewer resources in sperm than unattractive sperm donors, which could potentially lead to polymorphisms in ejaculate investment and thus alternative reproductive tactics in which competing sperm donors use divergent strategies to achieve mating success (Gross, 1996; Oliveira *et al.*, 2008). All in all, a more integrative approach is required in models of cryptic female choice, in which not

only heritable male attractiveness is incorporated, but also the trade-offs between investment in sperm versus eggs, sperm versus precopulatory traits or sperm versus survival.

A9 Appendix: maternal versus offspring control over sex ratios based on partner quality

Maternal control

First, we generalize the model by Fawcett *et al.* (2011) (see chapter 8 of this thesis) to include differential costs of producing males versus females. Let $f_m(s_i)$ and $m_m(s_i)$ be the allocation to females and males when mothers have mated with a type i male and when sex allocation is under maternal control,

$$f_m(s_i) = \frac{1-s_i}{1-s_i+s_i\gamma}, \quad m_m(s_i) = \frac{s_i}{1-s_i+s_i\gamma},$$

here, γ is the relative cost of producing sons versus daughters. The transition matrix of a rare mutant when sex ratio is under maternal control is then given by (consult Table 8.1 on page 207 for notation):

$$\mathbf{B} = \frac{1}{2k}\begin{bmatrix} \left[(1-\hat{\alpha})f_m(\hat{s}_0)+\hat{\alpha}f_m(\hat{s}_1)\right]\hat{v}_{\mathrm{f}} & & \\ \left[(1-\hat{\alpha})m_m(\hat{s}_0)(1-\mu_0)+\hat{\alpha}m_m(\hat{s}_1)\mu_1\right]\hat{v}_{\mathrm{m0}} & & \\ \left[(1-\hat{\alpha})m_m(\hat{s}_0)\mu_0+\hat{\alpha}m_m(\hat{s}_1)(1-\mu_1)\right]\hat{v}_{\mathrm{m1}} & & \\ q_0 f_m(s_0)\hat{v}_{\mathrm{f}} & q_1 f_m(s_1)\hat{v}_{\mathrm{f}} & \\ q_0 m_m(s_0)(1-\mu_0)\hat{v}_{\mathrm{m0}} & q_1 m_m(s_1)\mu_1\hat{v}_{\mathrm{m0}} & \\ q_0 m_m(s_0)\mu_0\hat{v}_{\mathrm{m1}} & q_1 m_m(s_1)(1-\mu_1)\hat{v}_{\mathrm{m1}} & \end{bmatrix}.$$

From chapter 8 of this thesis, the resident eigenvalue is $\lambda = 2a_{11}$ or $k\bar{f}_m v_{\mathrm{f}}$ (where $\bar{f}_m = (1-\alpha)f_m(s_0)+\alpha f_m(s_1)$) and we scale the matrices by density dependence so that the population is stable, i.e., $\lambda = 1$ or $k = 1/\bar{f}_m v_{\mathrm{f}}$.

Obviously, the selection gradients of p and t for sex allocation under maternal control are unaltered when compared to eqns (8.4) and (8.5) in chapter 8 of this thesis. The sex-specific production cost γ is accounted for in the reproductive values and in the stable class distribution (not given here), and does not affect equilibrium conditions. To obtain intuitive expressions for the selection gradients on $\hat{s}_0$ and $\hat{s}_1$, the following equations for the reproductive values is useful (scaling the female reproductive value z_{f} arbitrarily to 1):

$$2\lambda z_{\mathrm{m0}} = q_0\left[f_m(s_0)v_{\mathrm{f}} + m_m(s_0)(1-\mu_0)v_{\mathrm{m0}}z_{\mathrm{m0}} + m_m(s_0)\mu_0 v_{\mathrm{m1}}z_{\mathrm{m1}}\right],$$

so that

$$\begin{aligned}
\frac{\partial w}{\partial s_0} &= \frac{1-\alpha}{2\bar{f}_m v_{\mathrm{f}}}\left[v_{\mathrm{f}}f_m'(s_0) + (1-\mu_0)v_{\mathrm{m0}}m_m'(s_0)z_{\mathrm{m0}} + \mu_0 v_{\mathrm{m1}}m_m'(s_0)z_{\mathrm{m1}}\right]y_f, \\
&= \frac{1-\alpha}{2\bar{f}_m v_{\mathrm{f}}}\frac{1}{c\left(s_0\right)^2}\left[-v_{\mathrm{f}}\gamma - \frac{f_m(s_0)v_{\mathrm{f}}}{m_m(s_0)} + \frac{f_m(s_0)v_{\mathrm{f}}}{m_m(s_0)} + (1-\mu_0)v_{\mathrm{m0}}z_{\mathrm{m0}},\right. \\
&\left. + \mu_0 v_{\mathrm{m1}}z_{\mathrm{m1}}\right]y_f, \\
&= \frac{1-\alpha}{2\bar{f}_m v_{\mathrm{f}}}\frac{1}{c\left(s_0\right)^2}\left[-\frac{v_{\mathrm{f}}}{m_m(s_0)} + \frac{2(1-\bar{s})v_{\mathrm{f}}z_{\mathrm{m0}}}{q_0 m_m(s_0)}\right]y_f,
\end{aligned}$$

$$= \frac{1-\alpha}{c\left(s_0\right)^2 m_m\left(s_0\right)}\left[\frac{z_{\mathrm{m}0}}{q_0} - \frac{1}{2\bar{f}_m}\right] y_f,$$

where $c\left(s_0\right) = s_0\gamma + 1 - s_0$. Similarly for s_1, one obtains

$$\frac{\partial w}{\partial s_1} = \frac{\alpha}{c\left(s_1\right)^2 m_m\left(s_1\right)}\left[\frac{z_{\mathrm{m}1}}{q_1} - \frac{1}{2\bar{f}_m}\right] y_f.$$

Offspring control

In case sex allocation is under offspring control, these sex allocation functions are

$$f_o(\tilde{s}_i, \bar{s}_i) = \frac{1-\tilde{s}_i}{1-\bar{s}_i+\bar{s}_i\gamma}, \quad m_o(\tilde{s}_i, \bar{s}_i) = \frac{\tilde{s}_i}{1-\bar{s}_i+\bar{s}_i\gamma},$$

where $\tilde{s}_i$ is the sex allocation locus expressed by a focal, mutant offspring, which reflects its probability to develop into a male. $\bar{s}_i$ is the probability of developing into a male averaged over all offspring (including the focal offspring) in the brood. The mutant sex allocation matrix $\mathbf{B}_o$ under offspring sex allocation control is then given by

$$\mathbf{B}_o = \frac{1}{2k}\begin{bmatrix} \left[(1-\hat{\alpha})f_o(\tilde{s}_0,\bar{s}_0) + \hat{\alpha}f_o(\tilde{s}_1,\bar{s}_1)\right]\hat{v}_{\mathrm{f}} & q_0 f_o(\tilde{s}_0,\bar{s}_0)\hat{v}_{\mathrm{f}} & q_1 f_o(\tilde{s}_1,\bar{s}_1)\hat{v}_{\mathrm{f}} \\ \left[(1-\hat{\alpha})m_o(\tilde{s}_0,\bar{s}_0)(1-\mu_0) + \hat{\alpha}m_o(\tilde{s}_1,\bar{s}_1)\mu_1\right]\hat{v}_{\mathrm{m}0} & q_0 m_o(\tilde{s}_0,\bar{s}_0)(1-\mu_0)\hat{v}_{\mathrm{m}0} & q_1 m_o(\tilde{s}_1,\bar{s}_1)\mu_1\hat{v}_{\mathrm{m}0} \\ \left[(1-\hat{\alpha})m_o(\tilde{s}_0,\bar{s}_0)\mu_0 + \hat{\alpha}m_o(\tilde{s}_1,\bar{s}_1)(1-\mu_1)\right]\hat{v}_{\mathrm{m}1} & q_0 m_o(\tilde{s}_0,\bar{s}_0)\mu_0\hat{v}_{\mathrm{m}1} & q_1 m_o(\tilde{s}_1,\bar{s}_1)(1-\mu_1)\hat{v}_{\mathrm{m}1} \end{bmatrix},$$

We calculate the selection gradients using a direct fitness approach (Taylor & Frank, 1996; Taylor *et al.*, 2007):

$$\frac{\partial w}{\partial \tilde{s}_i} = \left.\frac{\partial w}{\partial \tilde{s}_i}\right|_{\tilde{s}_i=\bar{s}_i=\bar{s}_i} + r_{\mathrm{focal}\to\mathrm{sibling}}\left.\frac{\partial w}{\partial \bar{s}_i}\right|_{\tilde{s}_i=\bar{s}_i=\bar{s}_i}$$

where $r_{\mathrm{focal}\to\mathrm{sibling}}$ is the relatedness between a focal offspring and its siblings which is $1/2$ in monogamous taxa. For example, the derivation of $\partial w/\partial \hat{s}_0$ is as follows:

$$\begin{aligned}
\frac{\partial w}{\partial s_0} &= \frac{1}{2kc(s_0)}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\left[\left(-v_{\mathrm{f}} + (1-\mu_0)v_{\mathrm{m}0}z_{\mathrm{m}0} + \mu_0 v_{\mathrm{m}1}z_{\mathrm{m}1}\right)\right. \\
&\quad \left. + \frac{1}{2}\frac{1-\gamma}{2k}\frac{1}{c\left(s_0\right)^2}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\left(v_{\mathrm{f}}\left(1-s_0\right) + v_{\mathrm{m}0}z_{\mathrm{m}0}s_0 + v_{\mathrm{m}1}z_{\mathrm{m}1}s_0\right),\right. \\
&= \frac{1}{2kc(s_0)}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\left(\frac{2kz_{\mathrm{m}0}}{q_0 m\left(s_0\right)} - \frac{v_{\mathrm{f}}}{s_0}\right) \\
&\quad + \frac{1}{2}\frac{1-\gamma}{2k}\frac{1}{c\left(s_0\right)^2}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\left[\frac{2kz_{\mathrm{m}0}s_0}{q_0 m\left(s_0\right)} + v_{\mathrm{f}}(1-s_0) - \frac{v_{\mathrm{f}} f\left(s_0\right)s_0}{m(s_0)}\right],
\end{aligned}$$

$$
\begin{aligned}
&= \frac{1}{c(s_0)}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\left(\frac{z_{\mathrm{m}0}}{q_0 m\left(s_0\right)}-\frac{1}{2\bar{f}s_0}\right)\\
&+\frac{1}{2}\frac{1-\gamma}{c\left(s_0\right)^2}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\frac{z_{\mathrm{m}0}s_0}{q_0 m\left(s_0\right)},\\
&= \frac{1}{c(s_0)}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\left(\frac{z_{\mathrm{m}0}}{q_0 m\left(s_0\right)}\left[1+\frac{1}{2}\left(1-\gamma\right)m(s_0)\right]-\frac{1}{2\bar{f}s_0}\right)\\
&= \frac{1}{c(s_0)s_0}\left[1-\alpha+q_0 y_{\mathrm{m}0}\right]\left(\frac{z_{\mathrm{m}0}}{q_0}\left[1-\frac{1}{2}(1-\gamma)s_0\right]-\frac{1}{2\bar{f}}\right).
\end{aligned}
$$

$\partial w/\partial \hat{s}_1$ can be derived in a similar fashion, leading to the selection differentials listed in the main text in eqns (9.5,9.6).

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Nederlandse Samenvatting

Seksuele selectie

Veel dieren, maar ook planten en zelfs lagere organismen zoals schimmels hebben opvallende en schijnbaar nutteloze phenotypische kenmerken, waarvoor een evolutionaire verklaring die volledig gebaseerd is op overlevingssucces onwaarschijnlijk lijkt. Extreme voorbeelden zijn de indrukwekkende staart van een pauw, of het enorme gewei van het (uitgestorven) reuzenhert, maar er zijn ook voorbeelden dichter bij huis, zoals bloemvormen in planten, het zingen van vogels en voor mensen een avondje dansen in de disco. Voor Darwin vormden zulke kenmerken in eerste instantie een probleem, gezien ze de overlevingskansen van een individu eerder leken te schaden dan te baten. Als verklaring voor deze kenmerken bedacht Darwin daarom de theorie van seksuele selectie, waarbij kenmerken die het overlevingssucces schijnbaar lijken te verlagen toch kunnen evolueren, doordat ze de hoeveelheid paringsmogelijkheden met het andere geslacht vergroten, ten koste van een verlaagd paringssuccess voor andere individuen van hetzelfde geslacht.

Alhoewel de ideeën van Darwin over seksuele selectie in eerste instantie sceptisch werden ontvangen door zijn vakgenoten, is seksuele selectie nu één van de belangrijkste aspecten binnen de huidige evolutietheorie. Niet alleen hebben veel studies aangetoond dat seksuele selectie de meest plausibele verklaring is voor talrijke opvallende gedragingen en eigenschappen in planten en dieren, het helpt ons ook te begrijpen waarom er zoveel verschillen zijn tussen de geslachten (denk bijvoorbeeld aan verschillen tussen mannetjes en vrouwtjes in veel diersoorten in de zorg voor nakomelingen). Bovendien wordt seksuele selectie ook bij fundamentelere vraagstukken beschouwd als een belangrijke factor: gezien seksuele selectie kan leiden tot reproductieve isolatie, speelt het bijvoorbeeld een rol bij soortsvorming en biodiversiteit. Ook speelt seksuele selectie waarschijnlijk een rol in onopgeloste vraagstukken zoals de evolutionaire handhaving van seksuele voortplanting en recombinatie.

Seksuele selectie en theorie

Gezien de rol die seksuele selectie lijkt te spelen in bovengenoemde evolutionaire processen, doen veel evolutionair biologen experimenten en observaties om de werking en gevolgen van seksuele selectie beter te begrijpen. Essentieel voor het doen van zulk werk is dat men beschikt over een robuust raamwerk van duidelijke en testbare voorspellingen. Seksuele selectie is echter een gecompliceerd proces, waarin de evolutie van kenmerken in het ene geslacht wederzijds afhankelijk is van de evolutie van bepaalde kenmerken in het andere geslacht. Door deze wederzijdse evolutionaire afhankelijkheid blijkt het lastig om verbale voorspellingen te maken over de

coevolutie van mannelijke en vrouwelijke eigenschappen, zonder verborgen verbanden over het hoofd te zien. Daarentegen zijn formele beschrijvingen, gebruikmakend van wiskundige analyses of simulatiemodellen, beter in staat om dit gecompliceerde proces te bevatten. Vanaf de jaren 70 van de vorige eeuw, toen het gebruik van formele, evolutionaire modellen populair werd, zijn er dan ook honderden modellen gepubliceerd over seksuele selectie.

De vraag is echter of al deze modellen inderdaad hebben geleid tot een robuust raamwerk van duidelijke en testbare voorspellingen. Voor mensen die niet direct betrokken zijn bij theoretisch werk lijkt het er eerder op dat de vele modellen, met al hun verschillende aannames, meer verwarring scheppen dan duidelijkheid. Met het doel om deze theoretische literatuur daarom inzichtelijker te maken, geeft **hoofdstuk 2** van dit proefschrift een overzicht van de meest belangrijke theoretische resultaten. Ook bespreekt hoofdstuk 2 de verschillen tussen de meest gebruikte technieken om evolutionaire processen te modelleren. Een belangrijke conclusie is bijvoorbeeld dat verschillende modellen met zeer uiteenlopende aannames (bijvoorbeeld met betrekking tot de hoeveelheid genetische variatie, of de aanwezigheid van kansprocessen) soms toch tot dezelfde conclusies kunnen leiden. Wat de kans groot maakt dat dergelijke voorspellingen algemeen zijn, en minder gebonden aan bepaalde soorts- of omgevingsspecifieke eigenschappen. Dit zou bijvoorbeeld het geval kunnen zijn voor de voorspelling dat verschillende subpopulaties van eenzelfde soort sterk zouden moeten verschillen in mannelijke baltskenmerken en vrouwelijke voorkeuren (zie Figuur 2.2 op pagina 35). Aan de andere kant zijn er ook evenzoveel voorbeelden te geven waarbij een dergelijke robuustheid niet geldt, en waarbij uitkomsten zeer afhankelijk zijn of men bijvoorbeeld een speltheoretische of een kwantitatief genetisch modeltype gebruikt. Wij stellen daarom een pluralistische aanpak voor waarbij studies altijd meer dan één modelleertechniek gebruiken, om zeker te weten dat theoretische voorspellingen niet gebonden zijn aan de specifieke aannames die nu eenmaal inherent zijn aan het gebruik van een bepaalde techniek.

Over het algemeen maken de meeste modellen ook vaak zeer abstracte aannames met betrekking tot de biologische mechanismes die ten grondslag liggen aan partnerkeuze. Er is bijvoorbeeld veel theoretisch werk gedaan aan de kosten en baten van vrouwelijke voorkeuren voor mannelijke aantrekkelijkheid. Terwijl de veelgeciteerde modellen concluderen dat zelfs kleine kosten van zulke voorkeuren sommige vormen van seksuele selectie vrijwel onmogelijk maken, schetsen recentere, dynamischere modellen een positiever beeld: deze modellen laten bijvoorbeeld zien dat het veel uitmaakt of de kosten voor het hebben van een sterke voorkeur in één keer worden betaald (wellicht als de neuronale aanleg van een sterke paringsvoorkeur veel energie kost tijdens de groei), of verdeeld worden over de levensduur (bijvoorbeeld als er bij de evaluatie van iedere kandidaat-partner telkens een predatierisico optreedt). Ook kunnen kostbare paringsvoorkeuren evolueren wanneer mannelijke aantrekkelijkheid nooit een vast punt bereikt, maar altijd blijft evolueren (zie bijvoorbeeld Figuur 2.3 op pagina 42). Hoofdstuk 2 concludeert daarom dat het toevoegen van dergelijke mechanistische aannames een belangrijke stap is om aan zo'n robuust raamwerk van voorspellingen te komen: als het toevoegen van meer mechanismen geen effect heeft eerdere voorspellingen, laat het zien dat bepaalde processen kunnen worden verwacht onafhankelijk van de soort of omgeving in kwestie. In het interessantere geval dat de uitkomst afhankelijk blijkt te zijn van bepaalde details (zoals bijvoorbeeld de aanwezigheid van bepaalde sex chromosomen), leidt dit juist

tot specifieke en testbare voorspellingen: een vergelijkende studie zou dan bijvoorbeeld de werking van seksuele selectie kunnen vergelijken tussen nauw verwante soorten met verschillende soorten sex chromosomen (zoals het geval is in vissen of reptielen).

Hermafrodieten

Een deel van dit proefschrift (hoofdstukken 3, 7, 8 en 9) concentreert zich daarom op het toevoegen van meer biologische details in bestaande modellen van seksuele selectie. Bijvoorbeeld in **hoofdstuk 3**: terwijl vrijwel alle modellen over seksuele selectie gelden voor organismen met aparte geslachten (gonochoristen), heeft seksuele selectie in hermafrodiete organismen (in hermafrodieten zijn beide geslachten aanwezig zijn in hetzelfde individu) slechts weinig aandacht gekregen. Hermafrodieten worden vaak als een uitzonderlijke en ook zeldzame groep organismen beschouwd, maar dit is niet correct: als we de insecten achterwege laten, zijn ongeveer 30% van alle diersoorten hermafrodiet. In planten is het bovendien de meest algemene seksuele vorm. In hermafrodiete dieren vinden we bovendien ook een aantal unieke seksuele gedragingen, zoals reciproke inseminatie (waar beide partners gelijktijdig elkaars eieren bevruchten) en lijkt het erop dat paringskeuze vaak wederzijds plaatsvindt in hermafrodieten, waarbij beide partners elkaar kiezen, in plaats van één partner de andere.

Het model over seksuele selectie in hermafrodieten dat ik bestudeer is gebaseerd op het zogenaamde 'Fisher proces' (zie Box 2.1 op pagina 32), waarbij voorkeuren voor de meest aantrekkelijke partners coevolueren met deze aantrekkelijkheid, louter en alleen omdat nakomelingen van dergelijke aantrekkelijke partners ook zelf weer aantrekkelijk zijn. Dit zichzelf versterkende proces is een onderliggende factor in vrijwel ieder model van seksuele selectie. In hoofdstuk 3 laten we zien dat, in vergelijking met gonochoristen, dergelijke voorkeuren voor aantrekkelijkheid over het algemeen moeilijker van de grond komen in hermafrodieten: de kosten van deze voorkeuren moeten immers door ieder individu worden opgebracht in een populatie van hermafrodieten, terwijl in een populatie van gonochoristen enkel de vrouwtjes de kosten van dergelijke voorkeuren voelen. Ook vind ik dat reciproke inseminatie geen invloed heeft op de uitkomst van seksuele selectie. Het meest interessante resultaat is echter dat wederzijdse partnerkeuze in zowel hermafrodieten als gonochoristen vaak veel makkelijker van de grond kan komen, ook wanneer de kosten voor het hebben van partnervoorkeuren zeer hoog zijn. Hoofdstuk 3 laat dus zien dat sommige mechanismen helemaal geen effect lijken te hebben op bestaande conclusies (reciproke inseminatie bijvoorbeeld), terwijl andere mechanismen juist een groot effect hebben (wederzijdse partnerkeuze bijvoorbeeld). Al met al zijn hermafrodieten een zeer interessante groep organismen om dergelijke voorspellingen omtrent wederzijdse partnerkeuze te testen, gezien er redelijk wat variatie blijkt te bestaan in partnerkeuzemechanismen tussen verwante soorten.

Seksallocatie

Een ander aspect waar het toevoegen van meer biologische details in bestaande modellen van seksuele selectie een ingrijpende rol blijkt te hebben is de co-evolutie van seksuele selectie met seksallocatie. In organismen met seksuele voortplanting kan een

eenheid van ouderlijke investering twee routes volgen: een mannelijke en een vrouwelijke. De verdeling van een reproductieve investering in mannelijke of vrouwelijke nakomelingen (of in zaad- versus eicellen in hermafrodieten) wordt *seksallocatie* genoemd. In de praktijk is seksallocatie vaak hetzelfde als de sekse ratio, de numerieke verhouding van het aantal zonen en dochters, alhoewel dit niet opgaat als de energie die benodigd is voor het maken van een zoon verschilt van de energie benodigd voor een dochter. Er is lang gedacht dat ouders in de meeste diersoorten altijd gelijke hoeveelheden investeerden in zonen en dochters, en dat een investering in zonen versus dochters onafhankelijk was van de conditie van de ouders, of de toestand van de omgeving. Mede door onderzoek naar seksuele selectie in dierpopulaties weten we nu echter dat seksratios wel degelijk kunnen verschillen afhankelijk van conditie of de omgeving.

Seksallocatie en seksuele selectie

Het algemeen geaccepteerde idee dat seksratios onafhankelijk zijn van omgevingsfactoren of van de conditie van de ouders veranderde mede door een invloedrijke studie door Robert Trivers en Dan Willard in 1973. Gebaseerd op observaties in rendieren suggereerden zij dat in sommige gevallen een moeder beter het geslacht van haar nakomelingen kan aanpassen aan haar fysieke gesteldheid (zie Box 1.2 op pagina 18)). In een aantal diersoorten hangt het reproductieve succes van zonen namelijk sterk af van de conditie van hun moeder (deze bepaalt namelijk de lichaamsgrootte van haar zonen, en die lichaamsgrootte bepaalt weer of een zoon later een harem kan verdedigen). Het reproductief succes van dochters daarentegen, is vaak veel minder afhankelijk van de conditie van haar moeder. In dergelijke gevallen kan het voor moeders dus voordelig zijn om in zwakke conditie meer dochters te produceren en in goede conditie juist meer zoons. Inderdaad zijn in redelijk wat diersoorten zulke *conditie-afhankelijke seksratios*, afhankelijk van fysieke gesteldheid van ouders of van omgevingsinvloeden zoals temperatuur of dichtheid, naderhand ook aangetroffen.

In een latere studie over seksratios in Zebravinken (*Taeniopygia guttata*) paste Nancy Burley de hypothese van Trivers en Willard toe in de context van seksuele selectie. Net zoals bij veel andere vogelsoorten hebben zebravinken een polygyn paringssysteem, waar aantrekkelijke mannetjes met veel vrouwtjes kunnen paren, terwijl het reproductieve succes van vrouwtjes minder afhankelijk is van haar aantrekkelijkheid. Burley voorspelde dat een vrouwtje die gepaard is met een aantrekkelijk mannetje het beste meer zonen dan dochters zou kunnen produceren: wanneer deze zonen de aantrekkelijkheid van hun vader erven, is het waarschijnlijk dat deze aantrekkelijke zonen met veel vrouwtjes paren en dus vele legsels bevruchten. Als er in plaats van aantrekkelijke mannetjes, dochters zouden worden geproduceerd, beperkt zich de hoeveelheid nakomelingen slechts tot het eigen legsel van deze dochters. Burley voorspelde dus dat vrouwtjes die met aantrekkelijke mannetjes paren het beste meer zonen dan dochters zouden produceren.

De verbale hypothese van Burley kreeg direct veel aandacht, maar de vele experimenten en observaties over de afgelopen dertig jaar laten een zeer variabel beeld zien (zie West (2009) voor een overzicht van eerdere studies): sommige studies vinden een sterk positief verband tussen aantrekkelijkheid van een partner en de hoeveelheid zoons die worden geproduceerd, terwijl evenzoveel studies helemaal geen verband vinden, en een aantal onderzoeken laten juist het tegenovergestelde zien, waarbij

aantrekkelijke vaders meer dochters produceren. Om deze patronen beter te begrijpen moeten we Burley's hypothese wellicht wat beter onder de loep nemen: immers, Burley presenteerde enkel een verbaal model, en gezien de eerder genoemde complexiteit van seksuele selectie is de kans groot is dat bepaalde processen of aannames over het hoofd zijn gezien. We weten bijvoorbeeld niet of seksratios gebaseerd op de aantrekkelijkheid van een partner altijd tot zeer extreme, of juist zeer zwakke seksratioverschillen zullen leiden. Ook maakt Burley geen voorspelling over de seksratios van vrouwtjes die met onaantrekkelijke, in plaats van aantrekkelijke, mannetjes gepaard zijn. Als laatste is het de vraag hoe de evolutie van de belangrijkste eigenschap – partnervoorkeur voor aantrekkelijke mannetjes – eigenlijk beïnvloed wordt door de aanwezigheid van flexibele seksratios.

Om daarom een beter inzicht te krijgen in de coevolutionaire interacties tussen seksuele selectie en seksallocatie, presenteren **hoofdstukken 7 en 8** van dit proefschrift modellen waarin zowel een vrouwelijke paringsvoorkeur, mannelijke aantrekkelijkheid en seksallocatie kunnen evolueren. **Hoofdstuk 7** concentreert zich vooral op de evolutie van seksallocatie: in overeenkomst met Burley's voorspellingen vinden we inderdaad dat vrouwtjes gepaard met aantrekkelijke mannetjes meer zonen produceren, terwijl vrouwtjes gepaard met onaantrekkelijke mannetjes juist meer dochters produceren. Dit resultaat is desalniettemin zeer gevoelig voor het onderliggende mechanisme van seksuele selectie: als vrouwelijke voorkeuren bijvoorbeeld gebaseerd zijn op mannelijke eigenschappen die geassocieerd zijn met erfelijke kwaliteit (zogeheten 'good-genes' modellen), zijn deze seksratio patronen erg zwak vergeleken met modellen waarbij erfelijke kwaliteit geen rol speelt (zoals bepaalde vormen van het Fisher proces). Ook is selectie voor de overproductie van zonen of dochters vaak zeer zwak: terwijl vrouwelijke voorkeuren en mannelijke baltskenmerken snel evolueren, duurt het vaak duizenden generaties voordat er significantie seksratio verschillen optreden tussen vrouwtjes die gepaard zijn met aantrekkelijke of onaantrekkelijke partners. Al met al lijken duidelijke seksratio afwijkingen gebaseerd op mannelijke aantrekkelijkheid daarom eerder uitzondering dan regel te zijn.

In **Hoofdstuk 8** gaan we een stap verder door ook te kijken naar de coevolutie van seksallocatie met vrouwelijke paringsvoorkeuren voor aantrekkelijke mannetjes. We vinden net zoals in Hoofdstuk 7 dat flexibele seksallocatie gebaseerd op aantrekkelijkheid van de partner inderdaad kan evolueren, maar dat dit vervolgens een ingrijpend effect heeft op de evolutie van de paringsvoorkeur: in de aanwezigheid van flexibele seksallocatie verdwijnt de paringsvoorkeur voor aantrekkelijke mannetjes namelijk uit de populatie, of is tenminste aanzienlijk verminderd. Omdat hiermee ook mannelijke aantrekkelijkheid verdwijnt uit de populatie, ondermijnt flexibele seksallocatie dus juist het doel waarvoor het evolueerde. Kortgezegd komt dit omdat vrouwtjes gepaard met onaantrekkelijke mannetjes (type 0 vrouwtjes) veel meer profiteren van flexibele seksratios dan vrouwtjes gepaard met aantrekkelijke mannetjes (type 1 vrouwtjes). Terwijl deze type 0 vrouwtjes in het geval van vaste seksratios een grote hoeveelheid onaantrekkelijke zoons produceerden die weinig kans hebben op nageslacht, produceren deze vrouwtjes nu vrijwel alleen dochters, die allen verzekerd zijn van reproductief succes. Hierdoor wordt het fitnessverschil tussen vrouwtjes die paren met onaantrekkelijke versus aantrekkelijke mannetjes juist eerder kleiner dan groter. En daarmee zijn ook de voordelen van het hebben van een voorkeur voor aantrekkelijke mannetjes kleiner. In tegenstelling tot de verbale voorspellingen van Burley leiden voorspellingen gebaseerd op wiskundige modellen dus tot hele andere

conclusies, waaruit blijkt dat flexibele sekseallocatie gebaseerd op de aantrekkelijkheid van een partner seksuele selectie ondermijnt .

Om huidige studies waar sekseallocatie toch gebaseerd lijkt te zijn op partnerkwaliteit beter te begrijpen, moeten we dus op zoek naar specifiekere hypotheses: vrouwelijke voorkeuren zouden bijvoorbeeld behouden kunnen blijven als de flexibiliteit van seksratios slechts beperkt is. Ook zijn er verklaringen mogelijk die deels onafhankelijk zijn van seksuele selectie: recente modellen laten bijvoorbeeld zien dat de aanwezigheid van seksueel antagonistische variatie ook kan leiden tot seksratio verschillen, gebaseerd op het phenotype van de partner (zie bijvoorbeeld Blackburn *et al.* 2010).

Sekseallocatie: mechanismen

Terwijl de voorafgaande modellen zich uitsluitend concentreerden op de interactie van seksallocatie met seksuele selectie, hebben we de mechanismen van sekseallocatie tot nu toe nog grotendeels buiten beschouwing gelaten. Als het zo is dat de conditie van de moeder, de aantrekkelijkheid van de partner of de omgeving van invloed is op sekseallocatie, wat zijn dan voor de hand liggende mechanismen om het geslacht te bepalen? Een genetisch systeem dat vaak geassocieerd wordt met flexibele geslachtsbepaling is bijvoorbeeld haplodiploïdie, wat voorkomt in bijen, wespen en mieren, maar ook in schildluizen en tripsen.

Haplodiploïdie Terwijl beide geslachten in diplodiploïde organismen (het genetisch systeem van veel hogere organismen) twee sets chromosomen hebben, geldt dat in haplodiploïde organismen slechts enkel voor vrouwtjes. Mannetjes hebben daarentegen slechts één set chromosomen. Deze mannetjes worden meestal asexueel geproduceerd en komen voort uit onbevruchte eicellen, terwijl vrouwtjes voortkomen uit bevruchte eicellen. Ook is het in veel soorten zo dat de moeder kan bepalen welke eicellen wel of niet bevrucht worden, waarmee ze dus de seksratio van haar nakomelingen kan veranderen. Dit is vooral onderzocht in parasitoïde wespen, die eieren leggen in gastheren (bijvoorbeeld rupsen of vliegenpoppen), waarbij de seksratio wordt aangepast aan de grootte van de gastheer. Ook dit is dus weer een voorbeeld van het Trivers-Willard principe, waarbij conditie-afhankelijke seksratios worden geproduceerd.

Analyses laten zien dat haplodiploïdie herhaaldelijk is geëvolueerd uit diplodiploïdie: recente studies schatten dat haplodiploïdie maximaal 20 keer geëvolueerd is. Omdat het er niet op lijkt dat er een adaptieve verklaring te vinden is voor de evolutie van haplodiploïdie in deze gevallen, wordt de verklaring vaak gezocht in de hoek van de *genetische conflicten* (zie Box 1.3 op pagina 22). Genetische conflicten ontstaan wanneer genetische elementen aanwezig in hetzelfde genoom of individu verschillende selectieve optima hebben. Dit is bijvoorbeeld het geval bij endosymbionten: intracellulaire bacteriën die worden aangetroffen in de meeste insecten. Dergelijke endosymbionten worden alleen via eicellen overgedragen (ze passen niet in een zaadcel) en hebben daarom geen belang bij de productie van mannetjes. Dit in tegenstelling tot genen in de celkern, die juist belang hebben bij de productie van zowel mannetjes als vrouwtjes. Deze verschillen in optimale seksratios tussen endosymbionten en genen in de celkern kunnen daarom dus leiden tot een evolutionaire wapenwedloop.

In **hoofdstuk 4** onderzoek ik of zo'n wapenwedloop in principe tot de evolutie van haplodiploidie kan leiden. Ik concentreer me op zogeheten 'male-killers', endosymbionten die zelfmoord plegen en daarmee hun gastheer doden, wanneer ze aanwezig zijn in mannetjes. De vraag is echter hoe zo'n endosymbiont het geslacht van zijn gastheer herkent. In organismen met XX-XY geslachtsbepaling kan zo'n endosymbiont het geslacht al herkennen tijdens de bevruchting: brengt de zaadcel een Y chromosoom mee (zodat de zygote in kwestie een mannetje wordt), dan kan de endosymbiont efficiënt zijn gastheer doden door deze zaadcel te vernietigen (haploïdisatie). Als gevolg van dergelijke male-killers ontstaat er dus al gauw een vrouwenoverschot. Op zijn beurt zorgen dergelijke seksratios ook weer voor selectie op de gastheer: een mannetje dat toch een kleine kans ziet om te overleven heeft immers een enorm voordeel, omdat hij vanwege het vrouwenoverschot nu met vele vrouwtjes kan paren en daardoor veel nakomelingen krijgt. Door deze wapenwedloop tussen selectie voor grotere overlevingskansen van gehaploïdiseerde mannetjes en de male-killers, kan haplodiploïdie in principe evolueren. Het blijkt echter vaak ook dat de endosymbiont ook weer uitsterft, waardoor diplodiploïdie uiteindelijk gehandhaafd blijft. Als de endosymbiont echter bepaalde voordelen met zich meebrengt (zoals bijvoorbeeld het verteren van cellulose), is uitsterven van de endosymbiont echter onwaarschijnlijk, zodat de evolutie van haplodiploïdie veel makkelijker teweeg is te brengen.

Binnen de context van haplodiploïdie, bestudeert **hoofstuk 5** het daadwerkelijke geslachtsbepalingssysteem dat wordt gebruikt door haplodiploïde organismen: wat maakt individuen met slechts één set chromosomen bijvoorbeeld tot mannetje? In veel soorten wordt dit geregeld door genloci die behoren tot het Complementair Sekse Determinatiesysteem (CSD). Wanneer minimaal één van deze CSD genloci heterozygoot is, volgt vrouwelijke ontwikkeling. Wanneer alle CSD loci homozygoot (of in geval van haploïde individuen hemizygoot) zijn, volgt daarentegen ontwikkeling als mannetje. Daarmee zijn organismen met weinig CSD loci zeer gevoelig voor inteelt, gezien dit leidt tot meer homozygotie en daardoor een grote kans op het produceren van diploïde mannetjes, welke vaak onvruchtbaar zijn. In hoofdstuk 5 bepalen we de hoeveelheid CSD loci in de wesp *Cotesia glomerata* om erachter te komen wat de inteeltgevoeligheid van deze soort is. Op zijn beurt is deze inteeltgevoeligheid van nut voor de landbouw, gezien *Cotesia glomerata* wordt gebruikt om rupsen van het koolwitje *Pieris rapae* te bestrijden. We maken gebruik van computersimulaties om een inteeltexperiment over twee generaties na te bootsen voor verschillende hoeveelheden CSD loci. Gebaseerd op de gevonden seksratios, concluderen we dat dat gevonden data het best verklaard worden door twee onderliggende CSD loci. Daarmee is *Cotesia glomerata* bijvoorbeeld al een stuk robuuster tegen inteelt dan vele andere soorten wespen, die slechts één CSD locus hebben.

Genetische conflicten over conditie-afhankelijke seksratios In hoofdstuk 4 wordt onderzocht of evolutie van haplodiploïdie kan worden veroorzaakt door een genetisch conflict over de optimale seksratio tussen de gastheer en intracellulaire endosymbionten. Een ander belangrijk genetisch conflict dat geassocieerd wordt met de evolutie van geslachtsbepalingssystemen is het conflict tussen ouders en hun nakomelingen. Voor de ouder wordt de voortebrachte hoeveelheid nakomelingen vaak gemaximaliseerd wanneer de ouder de sekseallocatie optimaliseert over alle nako-

melingen als één geheel. Vanuit het oogpunt van een nakomeling is het daarentegen voordeliger als het zich ontwikkelt tot het geslacht met de hoogste 'reproductieve waarde' (reproductieve waarde is vakjargon voor de bijdrage van dat individu aan de populatie in de verre toekomst), ten koste van de reproductieve waarde van zijn broertjes en zusjes (en dus ook ten koste van de reproductieve waarde van zijn moeder), bijvoorbeeld omdat zijn keuze voor een bepaald geslacht meer grondstoffen kost dan de keuze van zijn moeder.

In de context van conditie-onafhankelijke geslachtsbepalingssystemen hebben eerdere studies al laten zien dat een dergelijk genetisch conflict tussen ouders en nakomelingen kan leiden tot een coevolutionaire wapenwedloop, waarbij uiteenlopende chromosomale geslachtsbepalingssystemen zoals ZZ - ZW, XX - XY of maternale monogenie (iedere moeder produceert slechts één geslacht) kunnen evolueren. Deze modellen concentreren zich echter alleen op conditie-afhankelijke geslachtsbepalingssystemen, terwijl voorspellingen ontbreken over de rol van genetische conflicten in conditie-afhankelijke geslachtsbepalingssystemen (zoals temperatuur-afhankelijke geslachtsbepaling in veel reptielen en vissen).

De gevolgen van conflicten tussen ouders en nakomelingen op de evolutionaire handhaving van conditie-afhankelijke seksratios wordt bestudeerd in **hoofdstuk 6**. Allereerst stellen we vast dat variatie in conditie op zichzelf onvoldoende is om seksratio conflicten te creëren. Er zijn dus andere factoren nodig, zoals verschillen in productiekosten tussen beide seksen, waardoor het 'duurdere' geslacht zeldzamer is en dus een hogere reproductieve waarde heeft dan het andere geslacht. Dergelijke verschillen in productiekosten leiden altijd tot conflict tussen ouders en nakomelingen wanneer geslachtsbepaling conditie-onafhankelijk is, maar verrassend genoeg vinden we dat wanneer geslachtsbepaling wel afhankelijk is van conditie, zulke conflicten soms afwezig kunnen zijn. In het geval er wel conflicten optreden tussen ouders en nakomelingen over conditie-afhankelijke sekseallocatie, kan dit leiden tot de invasie van een conditie-onafhankelijk geslachtsbepalingssysteem (bijvoorbeeld geslachtsbepaling door middel van sekschromosomen). Desalniettemin hangt de succesvolle invasie sterk af van de variatie in conditie die aanwezig is in de populatie. Bovendien leidt een successvolle invasie van een dergelijk conditie-onafhankelijk geslachtsbepalingssysteem lang niet altijd tot een complete vervanging van conditie-afhankelijke geslachtsbepaling door conditie-onafhankelijke geslachtsbepaling. Er kunnen bijvoorbeeld ook situaties ontstaan waarin conditie-onafhankelijke en afhankelijke geslachtsbepalingsfactoren samen stabiel blijven voortbestaan. Dergelijke 'gemengde' geslachtsbepalingssystemen zijn recentelijk ook aangetroffen in reptielen. Al met al leiden coëvolutionaire wapenwedlopen voortgebracht door genetische conflicten tot een aantal verschillende uitkomsten, afhankelijk van de aanwezige variatie in conditie.

De belangrijkste bevindingen van dit proefschrift

- Het maken van voorspellingen gebaseerd op slechts één modelleertechniek is vragen om problemen. Een pluralistische aanpak waarbij meerdere technieken met elkaar worden vergeleken geeft de meest robuuste voorspellingen met betrekking tot evolutionaire processen (**hoofdstuk 2**).
- Nieuwe theoretische modellen zijn nodig om te onderzoeken of conclusies van bestaande modellen over seksuele inderdaad zo algemeen geldend zijn als wordt

gedacht: hierbij moet vooral worden gedacht aan mechanistische processen met betrekking tot paringskeuze, overerving en interacties met andere evolutionaire processen (zorg voor nakomelingen, seksealloca tie) (**hoofdstuk 2**).

- Seksuele selectie in hermafrodieten (beide seksen aanwezig in hetzelfde individu) komt over het algemeen moeilijker van de grond dan in gonochoristen (seksen in aparte individuen). Dit geldt echter niet wanneer beide partners elkaar kiezen (in plaats van één partner de ander, welke dan altijd instemt): dan is seksuele selectie even plausibel in gonochoristen als in hermafrodieten (**hoofdstuk 3**).

- De evolutie van haplodiploïdie door toedoen van 'male-killers' lijkt veel specifieker te zijn dan eerder werd voorspeld (bijvoorbeeld Normark 2004a). Haplodiploïdie komt daarom alleen onder specifieke omstandigheden van de grond, bijvoorbeeld wanneer 'male-killers' mutualistisch zijn of wanneer populaties gekenmerkt worden door een hoge mate van inteelt (**hoofdstuk 4**).

- Twee of meerdere CSD loci liggen waarschijnlijk ten grondslag aan de geslachtsbepaling van de wesp *Cotesia glomerata*, waardoor het één van de weinige bekende soorten binnen de Hymenoptera is met multi-locus CSD (**hoofdstuk 5**).

- Conflict tussen ouders en nakomelingen over seksealloca tie kan leiden tot een stabiele mix van verschillende seksedeterminatiesystemen. Een dergelijk conflict kan bijvoorbeeld verklaren waarom sex chromosomen aanwezig zijn in sommige soorten met temperatuurafhankelijke geslachtsbepaling (**hoofdstuk 6**).

- Flexibele seksratios gebaseerd op de aantrekkelijkheid van de partner evolueren vaak naar een patroon waarbij vrouwtjes gepaard met aantrekkelijke mannetjes zonen produceren, en vrouwtjes met onaantrekkelijke mannetjes dochters. Desalniettemin is selectie voor flexibele seksratios gebaseerd op de aantrekkelijkheid van de partner vaak zwak en afhankelijk van het mechanisme dat seksuele selectie veroorzaakt (**hoofdstuk 7**).

- De evolutie van seksealloca tie gebaseerd op aantrekkelijkheid van partners èn samen met de evolutie van een vrouwelijke voorkeur voor aantrekkelijke mannetjes leidt uiteindelijk tot het verlies van deze voorkeur. Daarmee gaat ook mannelijke aantrekkelijkheid verloren, waarmee seksealloca tie zichzelf uiteindelijk ondermijnt (**hoofdstuk 8**).

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