

# Softness of selection and mating system interact to shape trait evolution under sexual conflict

Xiang-Yi Li Richter<sup>1,2</sup>  and Brian Hollis<sup>3</sup> 

<sup>1</sup>*Institute of Biology, University of Neuchâtel, Neuchâtel CH-2000, Switzerland*

<sup>2</sup>*E-mail: li@evolbio.mpg.de*

<sup>3</sup>*Department of Biological Sciences, University of South Carolina, Columbia, South Carolina 29208*

Received March 19, 2021

Accepted July 9, 2021

Sexual selection and sexual conflict play central roles in driving the evolution of male and female traits. Experimental evolution provides a powerful approach to study the operation of these forces under controlled environmental and demographic conditions, thereby allowing direct comparisons of evolutionary trajectories under different treatments such as mating systems. Despite the rapid progress of experimental and statistical techniques that support experimental evolution studies, we still lack clear theoretical predictions on the effects of different mating systems beyond what intuition suggests. For example, polygamy (several males and females in a mating group) and polyandry (one single female and multiple males in a mating group) have each been used as treatments that elevate sexual selection on males and sexual conflict relative to monogamy. However, polygamy and polyandry manipulations sometimes produce different evolutionary outcomes, and the precise reasons why remain elusive. In addition, the softness of selection (i.e., scale of competition within each sex) is known to affect trait evolution, and is an important factor to consider in experimental design. To date, no model has specifically investigated how the softness of selection interacts with different mating systems. Here, we try to fill these gaps by generating clear and readily testable predictions. Our set of models were designed to capture the most important life cycle events in typical experimental evolution studies, and we use simulated changes of sex-specific gene expression profiles (i.e., feminization or masculinization) to quantify trait evolution under different selection schemes. We show that interactions between the softness of selection and the mating system can produce results that have been identified as counterintuitive in previous empirical work such as polyandry producing stronger feminization than monogamy. We conclude by encouraging a stronger integration of modelling in future experimental evolution studies and pointing out remaining knowledge gaps for future theoretical work.

**KEY WORDS:** experimental evolution, gene expression, sexual conflict, sexual dimorphism, soft/hard selection, theory.

## Introduction

The importance of sexual selection in the evolution of male and female reproductive traits has been evident since Charles Darwin (1871), but the role of sexual conflict has taken much longer to be recognized (Trivers 1972; Parker 1979). For decades, empirical work has focused on demonstrating the mere existence of sexual conflict, which has been challenging due to various difficulties, including the measurement of the many entangled costs and benefits of traits involved in different stages of sexual inter-

actions, and the continual coevolutionary arms race between the sexes, which often cause sexual conflict to be hidden from our eyes (Arnqvist and Rowe 2005). Research on the roles of sexual conflict and its inevitable interactions with sexual selection in driving trait evolution has been revolutionized through experimental evolution, which allows direct comparison of independent evolutionary trajectories under controlled environmental and demographic conditions. For example, numerous studies have compared evolutionary trajectories under enforced monogamy, which prevents male competition and disfavours harassment of females, and contrasted this with polygamy and polyandry treatments, where sexual selection and conflict can operate (see Supporting

[Correction added on 25 APRIL 2022, after first online publication: CSAL funding statement has been added.]

information Appendix Table SA1 and references therein). Despite the rapid growth of knowledge gained from (many idiosyncratic) experimental evolution studies, it remains unclear how sex-specific trait optima shift under sexual selection and sexual conflict due to a combination of factors, including the different species or populations used across studies, subtle but important differences in the details of experimental design, as well as a lack of easily testable theoretical predictions.

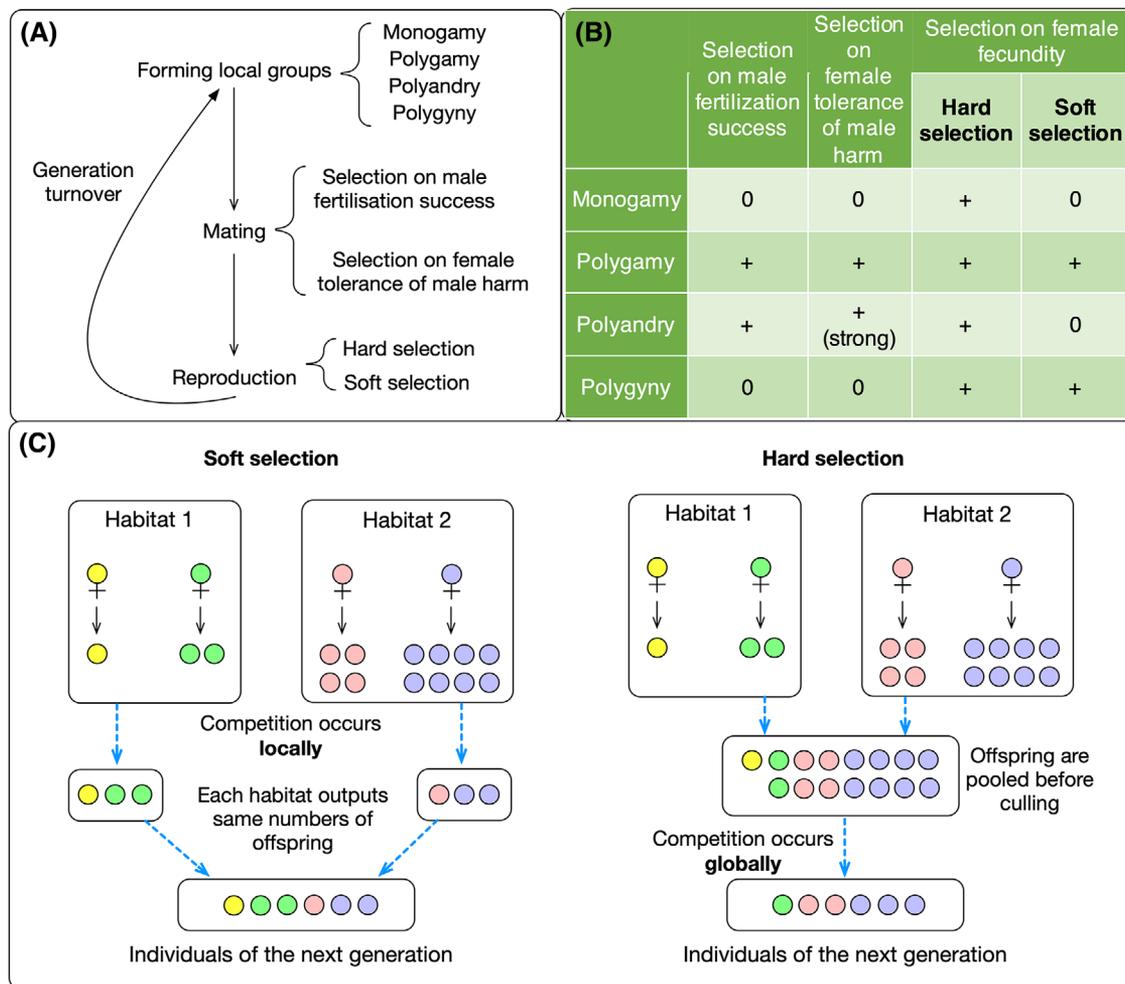
Among the existing experimental evolution studies that investigated trait evolution under sexual selection and sexual conflict, the mating system is probably the most often manipulated factor, and the comparison between enforced monogamy and polygamy/polyandry is probably the most frequently implemented experimental design. After all, sexual selection is generally considered to be stronger in males than in females in most species (Bateman 1948; Clutton-Brock and Parker 1992; Andersson 1994; Arnqvist and Rowe 2005; Janicke et al. 2016; Singh and Punzalan 2018). Therefore, removing sexual selection by enforced monogamy has been hypothesized to cause a shift of male trait mean towards the female optimum, when there is a genetic correlation between male and female traits. This general hypothesis, however, does not necessarily enable comparisons between studies that test it, because the studies often differ in many other aspects including the scale of competition in each sex.

The scale of competition, also called the softness of selection (Christiansen 1975; Wallace 1975; Gardner and West 2004; Débarre and Gandon 2011; De Lisle and Svensson 2017; McDonald et al. 2019), is fundamental in studying natural and sexual selection in metapopulations. Soft selection and hard selection refer to situations where offspring production of the local population is independent of, or directly proportional to, the competitiveness of its members, respectively. Under soft selection, only the *relative* competitiveness of individuals in the local population matters in determining the genetic composition of the next generation. In a sexual selection context, if males in the metapopulation are under soft selection, the fittest males in each subdivided local group will have comparable numbers of offspring that survive to the next generation, even if absolute fitnesses vary markedly. To illustrate soft selection, Wallace (1975) drew an analogy to selecting a dean of faculty at a university: “The position exists and needs filling; the creation of a dean does not require, however, the existence of faculty members with dean-like qualities because the position will be filled in any case.” In experimental evolution studies, each independent replicate of a selection regime can be considered as a metapopulation, where individuals (either as fertilized eggs, juveniles, or adults) are separated into smaller units of interactions (e.g., when matings take place in groups of a few individuals) and then mixed into larger ones (e.g., when offspring across those groups are pooled for logistical reasons) multiple times at different stages of a life cycle. If, how, and when individuals are

subdivided and combined, as well as how population size is regulated (i.e. the culling of adults and offspring) can vary greatly between different studies. Therefore, to help better understand and compare the results of previous studies and to inform the design of future experimental evolution work, we aim to study the overlooked effect of the softness of selection, and to make predictions on how the softness of selection interacts with mating systems to affect trait evolution under sexual selection and sexual conflict.

To this end, we built a set of individual-based simulations under a factorial design with four different mating systems (monogamy, polygamy, polyandry, and polygyny) and either soft or hard selection on females. Under monogamy and polygyny, there is only one male in each mating group, and under polygamy and polyandry, several males compete for fertilization opportunities within a local group. Therefore, selection on males is always soft (in the sense that males in different groups across the metapopulation never compete with each other), whereas in females, the softness of competition can vary depending on how offspring are pooled before culling. In the simulations, we vary the intensity of selection on male competition for fertilization opportunities, female competition for offspring production, and female tolerance of male harassment. The three traits are assumed to be condition-dependent. The condition of an individual is determined by its expression levels of male- and female-biased genes. Male- and female-biased genes have higher baseline expression levels in males and females, respectively. Upregulation of female-biased genes and/or downregulation of male-biased genes cause genome feminization, while upregulation of male-biased genes and/or downregulation of female-biased genes cause genome masculinization. A feminized gene expression profile implies evolution toward female trait optima, and is assumed to be beneficial for females but detrimental to males; a masculinized gene expression profile has the opposite effect, in agreement with empirical findings (Pointer et al. 2013; Dean et al. 2018; Rayner et al. 2019). The evolution of the gene expression profile occurs through the evolution of a number of upstream regulator genes. We studied the effect of two different mechanisms for how the regulator genes might function. Using the change of gene expression profiles to quantify the evolution of sex-specific trait optima allows us to directly compare our simulation results with that of several previous experimental evolution studies (Hollis et al. 2014; Immonen et al. 2014; Veltsos et al. 2017).

Our models reveal that the interaction between mating systems and the softness of selection on females can lead to differences in the relative conditions of males and females, as measured by the degree of genome feminization/masculinization, across selection regimes. Some of our results can help explain previously counterintuitive empirical findings. For example, we show that the magnitude of genome feminization can be stronger under monogamy than polygyny when selection on females is hard,



**Figure 1.** (A) Schematic illustration of the life cycle in the individual-based simulation model. (B) The presence and absence of different forces of selection under each mating system. (C) An illustration of the difference between soft and hard selection. Filled circles of each colour represent offspring produced by the female of the same colour. Under soft selection, the competition (population size regulation) of offspring occurs within each local habitat; under hard selection, offspring produced across habitats are pooled before population size regulation occurs.

but the outcome is the opposite when selection is soft. We also found conditions where the evolutionary outcomes of polygamy and polyandry can be identical or fundamentally different. For example, when female mortality due to male harassment is substantial, selection under polyandry can lead to stronger genome feminization (implying trait evolution shifts toward female optima) than under monogamy, especially when selection is soft, while polygamy always led to genome masculinization, possibly explaining the apparent contradictory results from two previous experimental evolution studies (Hollis et al. 2014; Veltsos et al. 2017). We discuss the broader implications of our results in helping design future experimental evolution studies and explaining existing results, and show as a proof-of-principle that the lament of “experimental evolution studies are useful in defining what may happen, but not necessarily why something did not happen” (Snook et al. 2010) can possibly be remedied by theoretical work

that incorporates the necessary biological factors and processes that drive phenotypic evolution.

## Models

We model eight distinct life cycles, with four different mating systems (monogamy, polygamy, polyandry, and polygyny) in combination with either soft or hard selection on female fecundity (Fig. 1). Males compete over fertilization opportunities in the same local group under polygamy and polyandry (i.e. sexual selection on males is always soft). Females experience selection on their tolerance of male harm (i.e. probability of female mortality before reproduction) and on their fecundity. Under hard selection, offspring produced in all local groups are directly pooled before population size regulation, and therefore, the scale of female fecundity competition is across the entire metapopulation.

Under soft selection, each local group contributes the same numbers of offspring of each sex to the next generation, and therefore, females only compete with each other within the same mating group (see Fig. 1C for an illustration of the differences between hard and soft selection). Therefore, selection on female fecundity is essentially absent under monogamy and polyandry, when selection is soft. In these cases, all females that survived to reproduction contribute the same numbers of offspring of each sex to the next generation.

### SEXUAL SELECTION ON MALES

Under polyandry and polygamy, males compete within each mating group for fertilization opportunities, and the probability of a male  $i$  to win a contest (i.e. being the sire of an offspring) is  $p_i = \theta_i^\beta / \sum_{j=1}^{n_c} \theta_j^\beta$ , where  $\theta_i$  denotes the physiological condition (hereafter “condition” for brevity) of the male,  $n_c$  is the number of competing males (i.e. 5 under polygamy and polyandry), and  $\beta$  adjusts the intensity of competition. When  $\beta = 0$ , all males have equal probability to win a contest; when  $\beta = 1$ , the probability of a male to win a contest is proportional to his condition; when  $\beta > 1$ , high-condition males are disproportionately favoured as sires. Under monogamy and polygyny, the condition of a male is irrelevant to his fitness, which instead depends solely on the fecundity of the female(s) in the same mating group.

### FECUNDITY SELECTION ON FEMALES

Mating competition is absent for females under all treatments (assuming that males are not limited by sperm production even under polygyny). However, females are subject to either soft or hard selection on their fecundity. When selection is soft, offspring population size regulation first happens at the local level, so that the number of eggs each mating group (if at least one female survives) contributes to the next generation is the same. When selection is hard, offspring population regulation happens only globally, namely, all eggs produced in the entire population are pooled before population size regulation. In either case, the fecundity of a female  $i$  (relative to females in the same mating group under soft selection, and relative to all females under hard selection) is proportional to  $\theta_i^\alpha / \sum_{j=1}^{\tilde{n}} \theta_j^\alpha$ , where  $\tilde{n}$  is the number of competing females depending on the mating system and hardness of fecundity selection, and  $\alpha$  adjusts the intensity of female intra-sexual competition. Note that in this simplified model we do not consider offspring viability selection during development, essentially assuming that all offspring will develop into adults if they survive the condition-independent culling of population size.

### SELECTION ON FEMALE TOLERANCE OF MALE HARASSMENT

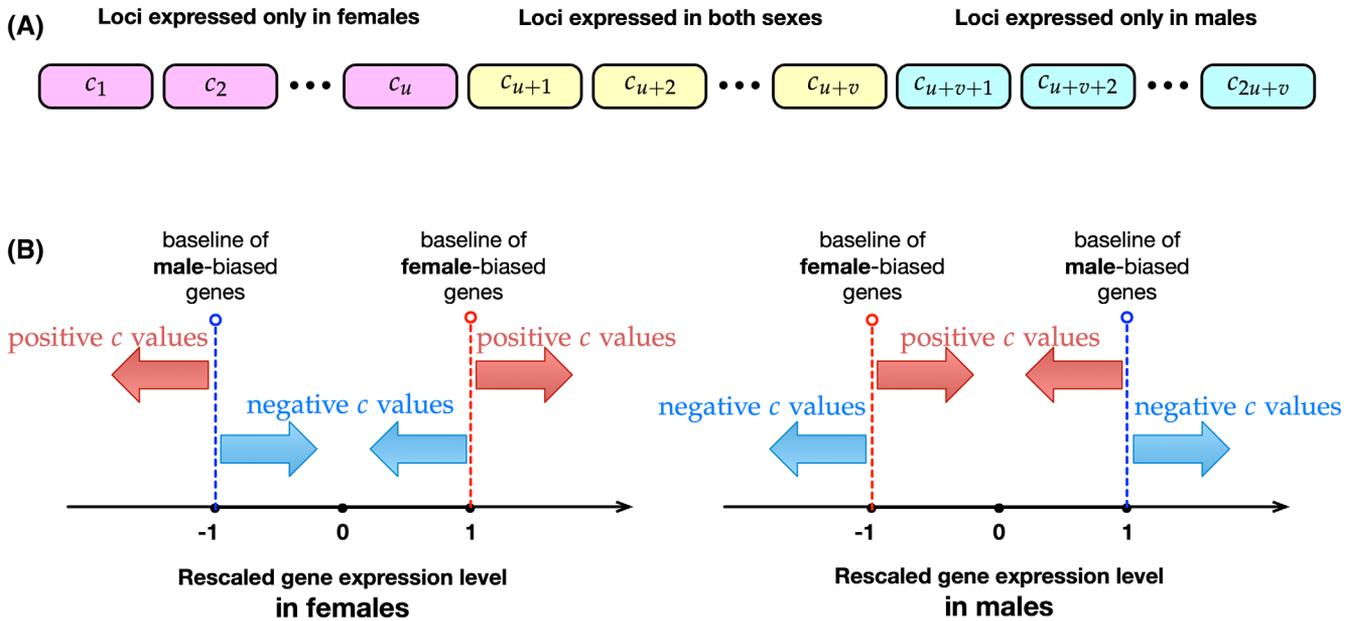
Females experience selection not only on their fecundity, but also on their tolerance of male harassment. We assume that females

may die because of male harassment before egg laying, and the mortality rate is proportional to the absolute number of males and the male:female ratio in a mating group. Unless otherwise specified, we set the proportion of females that die from male harassment under monogamy to  $m_0 = 0.05$ , and that under other mating systems to  $m_0 \sqrt{n_m \cdot \lambda}$ , where  $n_m$  is the number of males in a mating group, and  $\lambda$  is the sex ratio (the number of males divided by the number of females), so that the death rate is the highest under polyandry and the lowest under polygyny. Given the death rate under each mating system, the probability that a female survives male harassment depends on her condition relative to other females, so that the survival rate of female  $i$  is proportional to  $\theta_i^\gamma / \sum_{j=1}^n \theta_j^\gamma$ , where  $\theta_i$  is the condition of the female, and  $\gamma$  adjusts how strongly survival depends on condition. In the boundary case where  $\gamma = 0$ , all females have the same chance to survive independent of their conditions, while at larger  $\gamma$  values, females of higher conditions are more likely to survive than those with poorer conditions.

### CONDITIONS DETERMINED BY SEX-BIASED GENE EXPRESSION

The conditions of individuals are determined by the levels of sex-biased gene expression. For females, the condition is  $\theta^F = \varphi_f^F - \varphi_m^F$ , where  $\varphi_f^F$  is the cumulative expression level of female-biased genes, and  $\varphi_m^F$  is the cumulative expression level of male-biased genes. Correspondingly, the condition of males is  $\theta^M = \varphi_m^M - \varphi_f^M$ . Our formulation implies that high expression of female-biased genes is beneficial to females but detrimental to males, and vice versa for male-biased genes, in agreement with theoretical expectations and empirical findings in a number of species (Mank 2009; Pointer et al. 2013; Dean et al. 2018; Rayner et al. 2019).

The expression levels of sex-biased genes are regulated by a number of upstream regulator genes or “controller genes” (hereafter “controllers” for brevity) that each has a small effect and interacts additively in adjusting the expression levels of the sex-biased genes (Limousin et al. 2012; Randall et al. 2013; Veltsos et al. 2015; Kim et al. 2017). We model two different ways in which the controllers might function. In the first case (Mechanism I), the controllers have pleiotropic effects on the expression levels of sex-biased genes, so that upregulating female-biased genes automatically causes downregulation of male-biased genes, resembling the effect of hormonal regulators such as testosterone (Cox et al. 2017). In the second case (Mechanism II), the effect of the controllers is sex-specific so that varying the expression levels of female-biased genes has no influence on the expression of male-biased genes and vice versa. We expect the situation in most species would probably be a combination of both mechanisms.



**Figure 2.** (A) Mechanism I of how controllers determine the expression level of female- and male-biased genes. Some of the controllers are expressed only in females (represented by pink capsules) or only in males (cyan capsules), the rest are expressed in both sexes (yellow capsules). (B) Positive values at the controller loci increase the expression of female-biased genes and at the same time decrease the expression of male-biased genes. Negative values at the controller loci have the opposite effect.

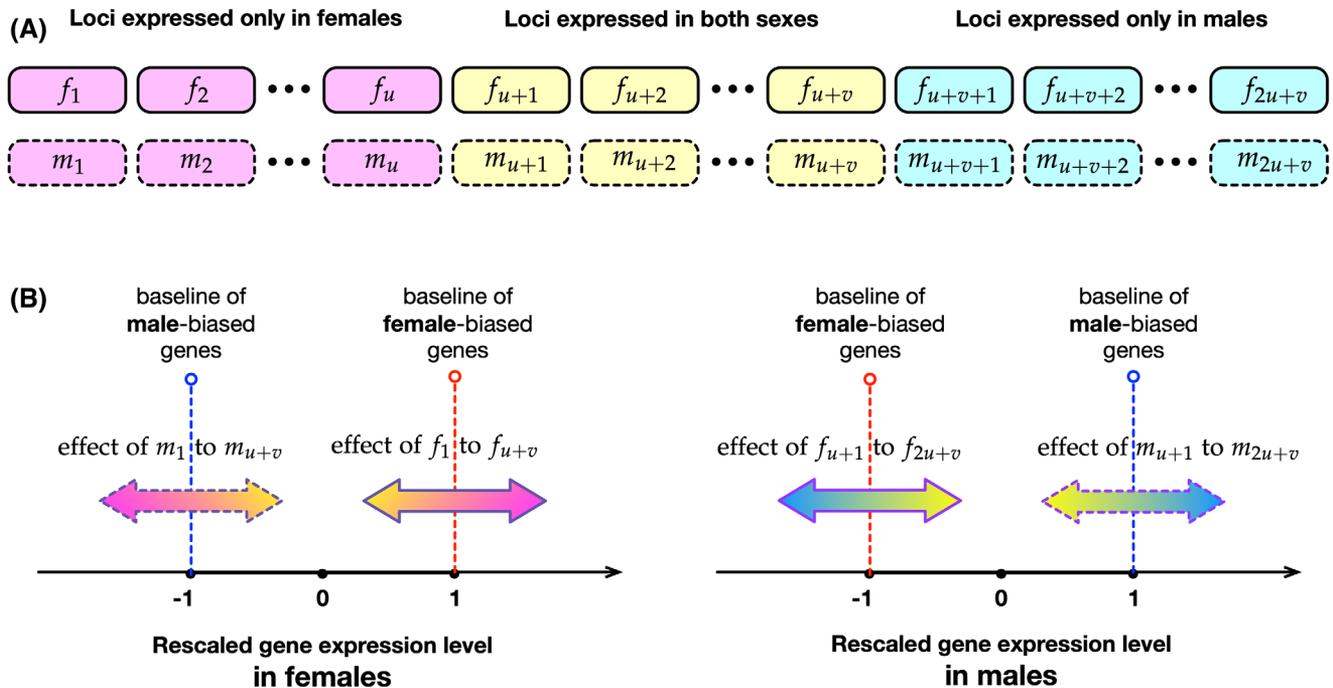
Under Mechanism I, we model the controllers with  $N_c$  unlinked diploid loci, each with a continuous allelic value  $c_i$  ranging from  $-1$  to  $1$ . As illustrated in Figure 2A,  $u$  of the loci are expressed exclusively in females or males each, and  $v$  of the loci are expressed in both sexes. The proportion of shared loci  $\delta = v/(2u + v) = v/N_c$ . The controllers regulate the expression levels of male- and female-biased genes additively, so that positive values of  $c$  upregulate female-biased genes and simultaneously downregulate male-biased genes from the baselines; negative values of  $c$  have the opposite effect. Eventually, in a female individual, the expression level of female-biased genes ( $\varphi_f^F$ ) and male-biased genes ( $\varphi_m^F$ ) are  $\varphi_f^F = 1 + \frac{1}{u+v} \sum_{i=1}^{u+v} c_i$  and  $\varphi_m^F = -1 - \frac{1}{u+v} \sum_{i=1}^{u+v} c_i$ , respectively. Similarly, the expression levels of female- and male-biased genes in a male are  $\varphi_f^M = -1 + \frac{1}{u+v} \sum_{i=u+1}^{2u+v} c_i$  and  $\varphi_m^M = 1 - \frac{1}{u+v} \sum_{i=u+1}^{2u+v} c_i$ , respectively.

Under Mechanism II, we model the controllers with  $N_f$  diploid loci that only affect the expression level of female-biased genes, and  $N_m$  diploid loci that only affect the expression level of male-biased genes, so that  $N_f = N_m = N_c/2$ . Among the  $N_f$  controllers that only affect the expression levels of female-biased genes (represented by the first row of capsules in Fig. 3A),  $u$  of them are expressed exclusively in females or males each, and  $v$  of them are expressed in both sexes. It is similar for the  $N_m$  controllers that only affect the expression levels of male-biased genes (second row of capsules in Fig. 3A). For both types of controllers, a proportion of  $\delta = v/(2u + v) = v/N_f = v/N_m$

are expressed in both sexes. Each of the controllers ( $f_i$  or  $m_i$ ) has a continuous allelic value ranging from  $-1$  to  $1$ . They interact additively to move the expression of sex-biased genes up or down from their baseline levels. Eventually, in a female individual, the expression levels of female- and male-biased gene are  $\varphi_f^F = 1 + \frac{1}{u+v} \sum_{i=1}^{u+v} f_i$  (ranging from  $0$  to  $2$ ) and  $\varphi_m^F = -1 + \frac{1}{u+v} \sum_{i=1}^{u+v} m_i$  (ranging from  $-2$  to  $0$ ), respectively. Similarly, in a male individual, the expression levels of female- and male-biased genes are  $\varphi_f^M = -1 + \frac{1}{u+v} \sum_{i=u+1}^{2u+v} f_i$  (ranging from  $-2$  to  $0$ ) and  $\varphi_m^M = 1 + \frac{1}{u+v} \sum_{i=u+1}^{2u+v} m_i$  (ranging from  $0$  to  $2$ ), respectively.

**POPULATION INITIALIZATION AND RUNNING OF THE MODEL**

We used diploid individuals in the population, with either 5000 females (for showing results under the evolutionary equilibrium) or 300 females (for showing results under a population size that is more realistic for experimental evolution studies) at the beginning of each generation in the metapopulation under each selection regime. The expression levels of sex-biased genes are controlled either by 40 general controller loci (mechanism I), or 20 female-specific loci and 20 male-specific loci (mechanism II). All loci are subject to Mendelian inheritance without linkage. The initial allelic values at each locus were drawn from a continuous uniform distribution between  $-1$  and  $1$  to generate standing genetic variations in the founder population. In simulations with 5000 females in each population, the mutation rate at each



**Figure 3.** (A) Mechanism II of how controllers determine the expression levels of female- and male-biased genes. Capsules with solid/dashed boundaries represent controllers that only affect the expression levels of female-/male-biased genes. The pink/blue colored capsules represent controllers that are expressed exclusively in females/males, and the yellow collared capsules represent controllers expressed in both sexes. (B) Positive/negative values at the controllers  $f_i$  increases/decreases the expression levels of female-biased genes in both males and females from the baselines when expressed. Analogously, positive/negative values at the controllers  $m_i$ , increases/decreases the expression levels of male-biased genes in both male and female individuals when expressed.

locus was set to 0.01 to accelerate the simulations to reach evolutionary equilibrium. The numbers of generations required for the simulations to reach evolutionary equilibrium differ between selection regimes and, therefore, the corresponding information is provided in the captions of each figure. The allelic value after mutation is also drawn from a uniformly distributed random number between  $-1$  and  $1$ , implying that mutations of large or small effects are equally likely to occur. In simulations with 300 females in each population, mutations were turned off, and the simulations were run for 20, 50, or 100 generations, corresponding to typical time scales of experimental evolution studies. The Python codes for simulations and the Mathematica notebooks for data visualization are provided in the Supporting information.

## Results

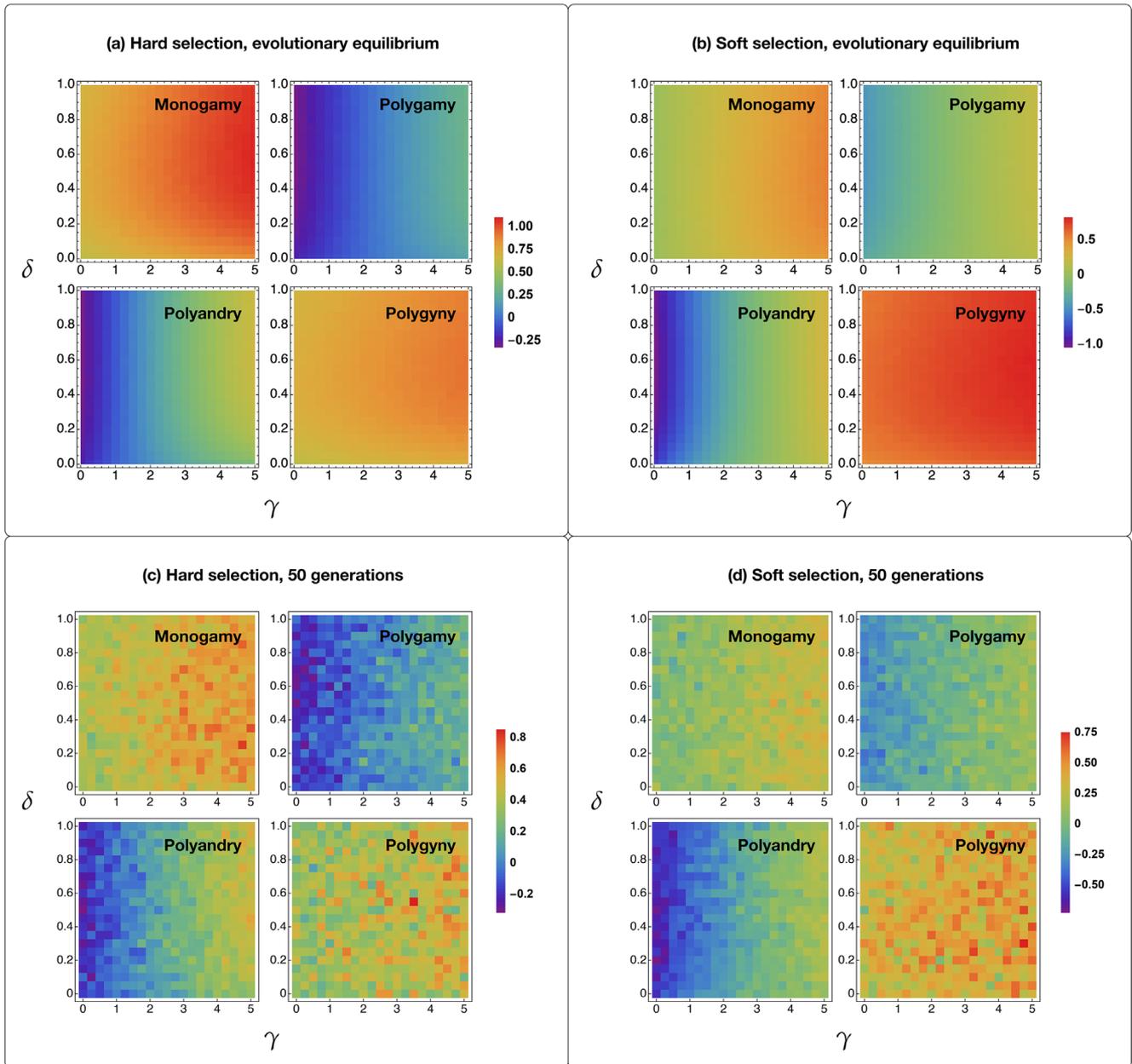
### EFFECTS OF SELECTION ON FEMALE TOLERANCE OF MALE HARM ( $\gamma$ ) AND THE PROPORTION OF CONTROLLER GENES EXPRESSED IN BOTH SEXES ( $\delta$ )

We use the difference between the population median conditions of females and males at evolutionary equilibrium to represent the magnitude of sex-biased gene expression at the whole genome

level. The larger the difference ( $\theta_f - \theta_m$ ), the stronger the degree of genome feminization. As shown in Figure 4, under all treatments, the magnitude of genome feminization increases with the intensity of selection on condition-dependent female tolerance of male harassment (large  $\gamma$ ), and when a large proportion of the controller genes are expressed in both sexes (large  $\delta$ ). When  $\delta = 0$ , all controllers are expressed sex specifically and both sexes, thus, have the greatest potential to reach a high condition and their sex-specific trait optima. Consequently, the difference between male and female conditions at evolutionary equilibrium is the smallest. The effect of  $\delta$ , however, is generally weaker than the effect of the intensity of selection on female tolerance of male harassment ( $\gamma$ ) and only makes a difference when  $\delta$  is relatively small.

When selection on female fecundity is hard (Fig. 4A and C), genome feminization is the strongest under monogamy and polygyny. Under both mating systems, all females compete for reproduction across the entire metapopulation, while selection on male condition is absent. The magnitude of genome feminization under the two mating systems is identical when selection on female tolerance of male harassment is absent ( $\gamma = 0$ ), but as  $\gamma$  increases, genome feminization becomes stronger under monogamy. This is because female mortality due to male

Difference between the median condition of females and males at equilibrium ( $\theta_f - \theta_m$ )



**Figure 4.** The magnitude of genome feminization as represented by the difference between the median conditions of females and males. Warmer colours represent stronger feminization and colder colours represent stronger masculinization. The expression levels of male- and female-biased genes are controlled by  $N_c = 40$  general controller loci. The selection intensities on female fecundity competition and male fertilization competition are  $\alpha = 1$ ,  $\beta = 2$ . Panels (a) and (b) represent the results at evolutionary equilibrium under hard and soft selection on female fecundity, respectively. The simulations were run with  $n = 5000$  females in each population for 5000 generations with a mutation rate of 0.01 at each locus. Panels (c) and (d) show the results under more realistic conditions for experimental evolution studies. The simulations were run with  $n = 300$  females in each population for 50 generations, in the absence of mutation.

harassment is higher under monogamy than under polygyny. In contrast, under both polygamy and polyandry, the presence of male competition selects for genome masculinization. Since the sex-specific softness of selection is identical under both mating systems (females compete across the entire metapopulation

while males compete within the local group), the magnitude of genome masculinization at equilibrium is the same under both mating systems when selection on female tolerance of male harm is absent ( $\gamma = 0$ ). As  $\gamma$  increases, genome masculinization becomes weaker (feminization becomes stronger) under polyandry

than under polygamy, because female mortality due to male harassment is much higher under polyandry.

When selection on female fecundity is soft (Fig. 4B and D), the degrees of genome feminization are reduced under all four mating systems (note that the colour scales are different across panels). Under monogamy, soft selection of female fecundity creates a “middle-class neighbourhood” selection scheme (Moorad and Hall 2009) when  $\gamma = 0$ , where selection on the condition of either sex is absent. As expected, gene expression evolves neither toward feminization nor toward masculinization ( $\theta_f - \theta_m = 0$ ). Now the magnitude of genome feminization is stronger under polygyny than under monogamy, because females experience competition over fecundity within the local group under polygyny, while selection on female fecundity is absent under monogamy. Similarly, the within-group female fecundity competition under polygamy (in contrast to no competition on female fecundity under polyandry) now drives stronger genome feminization (weaker genome masculinization) under polygamy than under polyandry when selection on female tolerance of male harassment is not particularly strong (small  $\gamma$ ).

The above results are robust despite the two different ways that the controller genes might function (Supporting information Fig. SC1). The general pattern also holds under different intensities of within-sex competition over reproduction in both sexes (Supporting information Fig. SC2–C5). Simulation results at the end of 20 or 100 generations in relatively small populations in the absence of mutation are qualitatively similar to those in Figure 4C and D, while shorter durations of selection produced more stochastic results (Supporting information Fig. SI-G1).

#### EFFECTS OF THE INTENSITIES OF SELECTION ON FEMALE FECUNDITY ( $\alpha$ ) AND MALE FERTILIZATION COMPETITION ( $\beta$ )

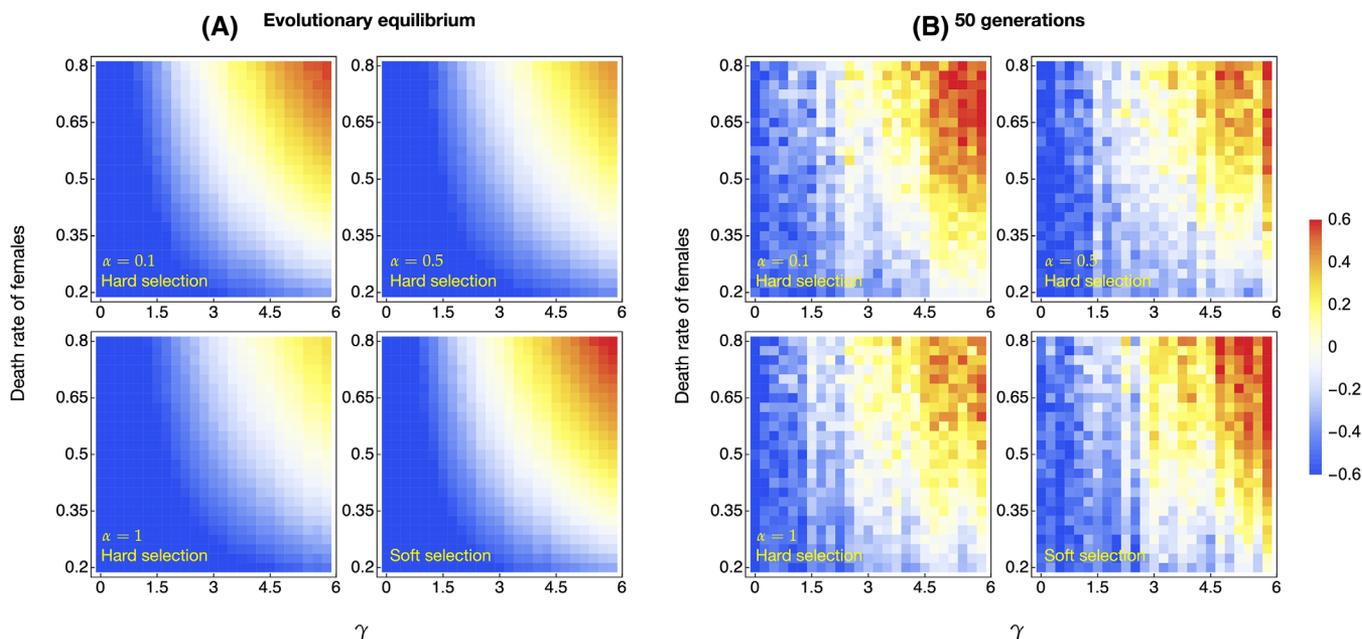
The effects of varying the intensities of female reproductive competition and male mating competition on the shift of male and female trait optima are relatively intuitive to understand: when fecundity selection is hard, increasing  $\alpha$  always leads to stronger feminization; when fecundity selection is soft, increasing  $\alpha$  leads to stronger feminization only under polygamy and polygyny (where several females compete in each local group) but not under monogamy or polyandry (where there is a single female in each mating group). Similarly, increasing  $\beta$  leads to stronger masculinization under polygamy and polyandry (where several males compete for fertilization within a mating group) but not under monogamy and polygyny (where there is a single male in each mating group). See Supporting information sections D and E for figures and more details on the effect of varying  $\alpha$  and  $\beta$ , respectively.

#### CONDITIONS FOR GREATER GENOME FEMINIZATION UNDER POLYANDRY THAN UNDER MONOGAMY

From the previous results we can see that the effects of polygamy and polyandry do differ considerably, but both produce genome masculinization relative to monogamy. However, the results can change when selection on female viability against male harassment becomes stronger.

Thus far, we have kept the mortality rate of females due to male harm fixed under each mating system (0.05 under monogamy, 0.25 under polyandry). We next allow the mortality rate of females under polyandry to vary, while keeping that under monogamy fixed at the original value. As shown in Figure 5 (note that the y-axis in each panel now reflects the proportion of females that die from male harassment under polyandry), when female mortality rates are high and condition-dependent selection on female tolerance against male harassment is strong, genome feminization can be stronger under polyandry than monogamy. When selection on female fecundity is hard, the size of the parameter region where polyandry produces stronger genome feminization decreases in size as the intensity of selection on condition-dependent female fecundity,  $\alpha$ , increases. This is because increasing  $\alpha$  promotes genome feminization more strongly under monogamy (where male competition is absent) than under polyandry (where males also compete) when females are under hard selection (see Supporting information Fig. SI-F1). When females are under soft selection, because there is only one female in each mating group under both polyandry and monogamy, fecundity competition between females is absent. Therefore, the evolution of genome feminization is driven solely by condition-dependent female tolerance of male harassment (the value of  $\alpha$  is no longer relevant). Consequently, the size of the parameter space where feminization is stronger under polyandry than under monogamy is always larger when females are under soft selection than under hard selection as long as  $\alpha > 0$ . Simulations in smaller populations in the absence of mutation for 50 generations produced qualitatively similar but more stochastic results (Fig. 5B, for results at the end of 20 and 100 generations see Supporting information Fig. SI-G2).

To summarize, genome feminization can evolve to be stronger under polyandry than under monogamy when condition-dependent mortality rate of females due to male harassment is much higher under polyandry than under monogamy, especially when female fecundity is under soft selection or of low selection intensity under hard selection. This is a feature that clearly distinguishes polyandry from polygamy, where an even sex ratio reduces the scope of male harm. The result is robust despite the different genetic architectures of the controller genes (Supporting information Fig. SI-F2). The size of the parameter region where feminization is stronger under polyandry than monogamy increases with  $\delta$ , the proportion of controllers expressed in both



**Figure 5.** The difference of the equilibrium magnitude of genome feminization between polyandry and monogamy. Warm colours represent regions where feminization is stronger under polyandry than under monogamy; cold colours represent the regions where feminization is stronger under monogamy than under polyandry. Note that the darkest blue was used to represent all data equal or smaller than  $-0.6$ . The expression levels of male- and female-biased genes are controlled by  $N_c = 40$  general controller loci. The intensity of selection on male fertilization competition was set to  $\beta = 2$ , and the proportion of controller genes expressed in both sexes was  $\delta = 0.5$ . The simulations in panel (A) were run with  $n = 5000$  females in each population for 2000 generations with a mutation rate of 0.01 at each locus. The simulations in panel (B) were run with  $n = 300$  females in each population for 50 generations, in the absence of mutation.

sexes (compare Fig. 5A with Supporting information Figs. SI-F3, SI-F4, SI-F5, and SI-F6 for results under the assumption of general controller genes; compare Supporting information Figs. SI-F2, SI-F7, SI-F8, SI-F9, and SI-F10 for results under the assumption of sex-specific controller genes).

## Discussion

In this work, we performed simulations under eight different life cycles that capture the interactions between four different mating systems and soft/hard selection on females. We varied the intensities of selection on female fecundity ( $\alpha$ ), male competitiveness in fertilization ( $\beta$ ), and female tolerance of male harassment ( $\gamma$ ), under varying proportions of controller genes that are expressed in both sexes ( $\delta$ ). We used changes in sex-specific gene expression profiles as an objective and easily quantifiable measurement of the evolutionary response under the combined effect of sexual selection and sexual conflict. Genome feminization corresponds to an evolutionary shift of antagonistic male and female sexual traits toward female optima, while genome masculinization corresponds to an evolutionary shift of those traits toward male optima. Our results showed clearly that trait evolution under different mating systems needs to be interpreted with the softness of

selection in mind. Also, the intensity of selection on female tolerance of male harassment ( $\gamma$ ) plays a crucial role on the evolutionary outcome. When females are under hard selection, the selection regimes of polygamy and polyandry are equivalent, and the same for monogamy and polygyny, only if selection on female tolerance against harassment is absent ( $\gamma = 0$ ); but when selection on female tolerance of male harassment is strong ( $\gamma$  is large), the degree of genome feminization generally follows the decreasing order of monogamy > polygyny > polyandry > polygamy. When selection on female fecundity is soft, feminization was always stronger under polygyny than under monogamy.

We also found an interesting result that genome feminization can evolve to be stronger under polyandry than under monogamy, when female tolerance of male harassment is strongly condition-dependent and the mortality rate is much higher under polyandry than under monogamy. In contrast, the degree of feminization was always higher under monogamy than under polygamy. These predictions correspond to the findings of a pair of experimental evolution studies in *Drosophila* (Hollis et al. 2014; Veltsos et al. 2017). The Hollis et al. (2014) work implemented enforced monogamy in contrast to polygamy (five male and five female *Drosophila melanogaster* in each local group) and found that the genomes of populations evolving under enforced monogamy

were feminized relative to polygamous populations. The Veltsos et al. (2017) work, however, implemented enforced monogamy in contrast to polyandry (one female and six male *D. pseudoobscura* in each local group), and found that polyandrous populations were feminized relative to monogamous populations. The hypothesis of Veltsos et al. (2017) was that polyandry imposes strong mating competition on males, and thus, should lead to genome masculinization, similar to the effect of polygamy in Hollis et al. (2014), and, therefore, the opposite experimental result — feminized flies under polyandry — was surprising. In light of our model results, however, this difference in outcomes could be explained by high female mortality under polyandry. In *Drosophila*, females suffer increased mortality from exposure to multiple males (Partridge et al. 1987; Fowler and Partridge 1989; Partridge and Fowler 1990), in part mediated by the transfer of seminal fluid proteins (Chapman et al. 1995), and multiple mating reduces female lifetime fecundity (Orteiza et al. 2005). Female mating rates under a male-biased regime like polyandry are greatly elevated relative to unbiased regimes (Wigby and Chapman 2004), amplifying harm as well as the importance of female tolerance. In addition, the importance of female tolerance of male harm over the course of experimental evolution in the populations used in Veltsos et al. (2017) was further elevated by an experimental detail: each generation, offspring were only retained from the most productive 40 females (Crudginton et al. 2005), which is equivalent to imposing 38% mortality. If the less productive females that were culled were also of lower condition, following the logic of our model, we would expect such a high effective mortality rate of females to lead to feminization of the transcriptome as populations adapt to increased mating rates and concomitant increases in male harm. Indeed, Wigby and Chapman (2004) and Crudginton et al. (2005) both found that females evolving under male-biased sex ratios evolved increased tolerance to male harm.

Aside from helping to better explain and compare the results of existing empirical work, our model has the potential to inform the design of future experimental evolution studies. But caution must be exercised when applying theoretical results in the context of more complex experimental systems. In the current model, we implemented soft selection on males and a simple dichotomy of soft/hard selection on females. In experimental evolution studies, hard and soft selection can form a continuum, where either sex can be placed between the two extremes, for example, by changing the size of each mating group relative to the entire population, or the timing of density regulation relative to pooling of individuals at different stages. For example, selection on female fecundity competition in Veltsos et al. (2017) was softer than that in Hollis et al. (2014) since females were not pooled during egg laying, but not as soft as the soft selection treatment in our model, because offspring produced in each enclosure were still pooled before random culling to form the next generation. Similarly, although the

selection on female fecundity in Hollis et al. (2014) was relatively hard because females were pooled into two groups before egg laying, it was not as hard as the hard selection treatment in our model, where all females lay eggs in a global pool. In addition, the “intensity” and “softness” of selection can in principle vary independently, and it is convenient to do so in simulations. But experimental manipulations often involve a change of both, and the effect can be different for different types of selection. To illustrate this, we use the work of Yun et al. (2018) as an example.

There are three different selection regimes in Yun et al. (2018): enforced monogamy, polygamy in a simple environment, and polygamy in a complex environment. We focus on the comparison between the two polygamy treatments. Under both treatments, there are 35 male and 35 female *D. melanogaster* flies in each enclosure, and therefore, the softness of selection for males and females seems to be equal under the two treatments. A closer look, however, reveals subtle differences. In the complex environment, the flies were provided with five separate food sources and plenty of places for hiding. Therefore, the competition for female tolerance against male harassment is not only less intense but also softer than in the simple environment, as each female in the complex environment might interact with only a few other individuals throughout the whole mating period. Interestingly, the selection on female fecundity took place at a different scale for both treatments, because the females were pooled, randomly culled, and then the rest were separated into standard fly vials of 15 individuals for 1 day of egg laying. The softness of female fecundity competition was, therefore, equal for both treatments, probably intermediate between the softness of female viability competition in the simple and complex environments. This example illustrates the practical difficulties that would be encountered in experimental evolution studies that attempt to control for the intensity and softness of selection independently. The results of our current work provide conceptual help, but cannot provide precise quantitative predictions unless many experimental parameters are carefully controlled. Since computer simulations can be done much easier and faster than real experiments, we encourage an integration of both approaches in future experimental evolution studies. The simulation models can then include species-specific life history features and experimental details including the necessary steps of population pooling and subdivision. For species where we have sufficient knowledge of genome architecture, the underlying genetic elements and their expected interactions can also be included or tested in the model.

Our results also revealed several new questions and knowledge gaps for theoretical investigation. First, what happens if the intensity and/or softness of selection change over the course of evolution under sexual selection and conflict? The question is important in the context of environmental and climate change, and also relevant because empirical work has shown that females

can evolve to better tolerate male harassment (Wigby and Chapman 2004; Maklakov et al. 2006; Harano 2015), which might cause the selection intensity on female tolerance against male harassment to decrease over time. Second, how does the softness of selection interact with male harassment that is targeted at high-condition females — a phenomenon found in at least some *D. melanogaster* populations (Long et al. 2009; Yun et al. 2017; MacPherson et al. 2018)? Correspondingly, it is reasonable to expect males of higher condition may cause more severe harm to females. How does such condition-dependent male harm interact with sexual selection to affect the sex-specific trait evolution under different selection schemes? Also, does the softness of selection play a significant role in affecting trait evolution in hermaphroditic animals and plants? Since hermaphrodites, such as *Caenorhabditis elegans* (Carvalho et al. 2014; Palopoli et al. 2015) and the plant *Collinsia heterophylla* (Lankinen et al. 2017; Lankinen and Strandh 2019), have been widely used to study the effect of sexual selection and sexual conflict in experimental evolution studies, we need to develop theoretical predictions for these systems.

In this work, we showed that the interaction between the softness of selection and mating system can affect trait evolution under sexual selection and sexual conflict in nonintuitive ways. Our main conclusions are likely to generalize to sexually dimorphic phenotypes other than gene expression, as long as these phenotypes are rooted in a similar genetic architecture. In *D. melanogaster*, quantitative traits like development time and body size, as well as relative fitness, are known to involve some combination of sexually-concordant and sexually-antagonistic genetic effects (Chippindale et al. 2001; Prasad et al. 2007; Hollis et al. 2017) and there is no obvious reason to expect otherwise for sexually-dimorphic traits in general. Based on our results, the design of future experimental evolution work — whether focused on the evolution of gene expression or other traits — should carefully consider the scale of sex-specific competition and how this might qualitatively change predicted evolutionary outcomes. The results of the current work represent a “proof of principle”; we are now in a position to design better theory-informed experimental studies and to tackle the remaining theoretical challenges, bringing us closer to a more comprehensive understanding of how sexual selection and sexual conflict interact to drive phenotypic evolution.

#### CONFLICT OF INTEREST

The authors declare no conflict of interest.

#### AUTHOR CONTRIBUTIONS

X.L. and B.H. conceived the study and designed the model; X.L. constructed the model, produced the results, and wrote the manuscript with inputs from B.H.

#### ACKNOWLEDGMENTS

We thank the three anonymous reviewers for their comments and suggestion, and the Swiss National Science Foundation for financial support (PZ00P3\_180145 to X.L. and PZ00P3\_161430 to B.H.).

Open Access Funding provided by Universite de Neuchatel.

#### LITERATURE CITED

- Andersson, M. 1994. Sexual selection. Princeton Univ. Press, Princeton.
- Arnqvist, G., and L. Rowe. 2005. Sexual conflict. Princeton Univ. Press, Princeton.
- Bateman, A. J. 1948. Intra-sexual selection in *Drosophila*. *Heredity* (Edinb) 2:349–368.
- Carvalho, S., P. C. Phillips, and H. Teotónio. 2014. Hermaphrodite life history and the maintenance of partial selfing in experimental populations of *Caenorhabditis elegans*. *BMC Evol. Biol.* 14:117.
- Chapman, T., L. F. Liddle, J. M. Kalb, M. F. Wolfner, and L. Partridge. 1995. Cost of mating in *Drosophila melanogaster* females is mediated by male accessory gland products. *Nature* 373:241–244.
- Chippindale, A. K., J. R. Gibson, and W. R. Rice. 2001. Negative genetic correlation for adult fitness between sexes reveals ontogenetic conflict in *Drosophila*. *Proc. Natl. Acad. Sci.* 98:1671–1675.
- Christiansen, F. B. 1975. Hard and soft selection in a subdivided population. *Am. Nat.* 109:11–16.
- Clutton-Brock, T. H., and G. A. Parker. 1992. Potential reproductive rates and the operation of sexual selection. *Q. Rev. Biol.* 67:437–456.
- Cox, R. M., C. L. Cox, J. W. McGlothlin, D. C. Card, A. L. Andrew, and T. A. Castoe. 2017. Hormonally mediated increases in sex-biased gene expression accompany the breakdown of between-sex genetic correlations in a sexually dimorphic lizard. *Am. Nat.* 189:315–332.
- Crudgington, H. S., A. P. Beckerman, L. Brüstle, K. Green, and R. R. Snook. 2005. Experimental removal and elevation of sexual selection: Does sexual selection generate manipulative males and resistant females? *Am. Nat.* 165:S72–S87.
- Darwin, C. 1871. The descent of man, and selection in relation to sex. John Murray, Albemarle Street, London.
- De Lisle, S. P., and E. I. Svensson. 2017. On the standardization of fitness and traits in comparative studies of phenotypic selection. *Evolution* (N. Y.) 71:2313–2326.
- Dean, R., C. Hammer, V. Higham, and D. K. Dowling. 2018. Masculinization of gene expression is associated with male quality in *Drosophila melanogaster*. *Evolution* (N. Y.) 72:2736–2748.
- Débarre, F., and S. Gandon. 2011. Evolution in heterogeneous environments: Between soft and hard selection. *Am. Nat.* 177:E84–E97.
- Fowler, K., and L. Partridge. 1989. A cost of mating in female fruitflies. *Nature* 338:760–761.
- Gardner, A., and S. A. West. 2004. Spite and the scale of competition. *J. Evol. Biol.* 17:1195–1203.
- Harano, T. 2015. Receptive females mitigate costs of sexual conflict. *J. Evol. Biol.* 28:320–327.
- Hollis, B., D. Houle, Z. Yan, T. J. Kawecki, and L. Keller. 2014. Evolution under monogamy feminizes gene expression in *Drosophila melanogaster*. *Nat. Commun.* 5:3482.
- Hollis, B., L. Keller, and T. J. Kawecki. 2017. Sexual selection shapes development and maturation rates in *Drosophila*. *Evolution* (N. Y.) 71:304–314.
- Immonen, E., R. R. Snook, and M. G. Ritchie. 2014. Mating system variation drives rapid evolution of the female transcriptome in *Drosophila pseudoobscura*. *Ecol. Evol.* 4:2186–2201.
- Janicke, T., I. K. Häderer, M. J. Lajeunesse, and N. Anthes. 2016. Darwinian sex roles confirmed across the animal kingdom. *Sci. Adv.* 2:e1500983.

- Kim, K.-W., C. Bennison, N. Hemmings, L. Brookes, L. L. Hurley, S. C. Griffith, T. Burke, T. R. Birkhead, and J. Slate. 2017. A sex-linked supergene controls sperm morphology and swimming speed in a songbird. *Nat. Ecol. Evol.* 1:1168–1176.
- Lankinen, Å., S. Hydbom, and M. Strandh. 2017. Sexually antagonistic evolution caused by male–male competition in the pistil. *Evolution (N. Y.)* 71:2359–2369.
- Lankinen, Å., and M. Strandh. 2019. Can sexual selection cause divergence in mating system-related floral traits? *Int. J. Plant Sci.* 180:996–1003.
- Limousin, D., R. Streiff, B. Courtois, V. Dupuy, S. Alem, and M. D. Greenfield. 2012. Genetic architecture of sexual selection: QTL mapping of male song and female receiver traits in an acoustic moth. *PLoS One* 7:e44554.
- Long, T. A. F., A. Pischedda, A. D. Stewart, and W. R. Rice. 2009. A cost of sexual attractiveness to high-fitness females. *PLoS Biol.* 7:e1000254.
- MacPherson, A., L. Yun, T. S. Barrera, A. F. Agrawal, and H. D. Rundle. 2018. The effects of male harm vary with female quality and environmental complexity in *Drosophila melanogaster*. *Biol. Lett.* 14:20180443.
- Maklakov, A. A., N. Kremer, and G. Arnqvist. 2006. Ageing and the evolution of female resistance to remating in seed beetles. *Biol. Lett.* 2:62–64.
- Mank, J. E. 2009. Sex chromosomes and the evolution of sexual dimorphism: Lessons from the genome. *Am. Nat.* 173:141–150.
- McDonald, G. C., A. Gardner, and T. Pizzari. 2019. Sexual selection in complex communities: Integrating interspecific reproductive interference in structured populations. *Evolution (N. Y.)* 73:1025–1036.
- Moorad, J. A., and D. W. Hall. 2009. Mutation accumulation, soft selection and the middle-class neighborhood. *Genetics* 182:1387–1389.
- Orteiza, N., J. E. Linder, and W. R. Rice. 2005. Sexy sons from remating do not recoup the direct costs of harmful male interactions in the *Drosophila melanogaster* laboratory model system. *J. Evol. Biol.* 18:1315–1323.
- Palopoli, M. F., C. Peden, C. Woo, K. Akiha, M. Ary, L. Cruze, J. L. Anderson, and P. C. Phillips. 2015. Natural and experimental evolution of sexual conflict within *Caenorhabditis* nematodes. *BMC Evol. Biol.* 15:93.
- Parker, G. A. 1979. Sexual selection and sexual conflict. Pp. 123–166 in M. S. Blum and N. A. Blum, eds. *Sexual selection and reproductive competition in insects*. Academic Press, New York.
- Partridge, L., and K. Fowler. 1990. Non-mating costs of exposure to males in female *Drosophila melanogaster*. *J. Insect Physiol.* 36:419–425.
- Partridge, L., A. Green, and K. Fowler. 1987. Effects of egg-production and of exposure to males on female survival in *Drosophila melanogaster*. *J. Insect Physiol.* 33:745–749.
- Pointer, M. A., P. W. Harrison, A. E. Wright, and J. E. Mank. 2013. Masculinization of gene expression is associated with exaggeration of male sexual dimorphism. *PLoS Genet.* 9:e1003697.
- Prasad, N. G., S. Bedhomme, T. Day, and A. K. Chippindale. 2007. An evolutionary cost of separate genders revealed by male-limited evolution. *Am. Nat.* 169:29–37.
- Randall, J. C., T. W. Winkler, Z. Kutalik, S. I. Berndt, A. U. Jackson, K. L. Monda, T. O. Kilpeläinen, T. Esko, R. Mägi, S. Li, et al. 2013. Sex-stratified genome-wide association studies including 270,000 individuals show sexual dimorphism in genetic loci for anthropometric traits. *PLoS Genet.* 9:e1003500.
- Rayner, J. G., S. Pascoal, and N. W. Bailey. 2019. Release from intralocus sexual conflict? Evolved loss of a male sexual trait demasculinizes female gene expression. *Proc. R. Soc. B* 286:20190497.
- Singh, A., and D. Punzalan. 2018. The strength of sex-specific selection in the wild. *Evolution (N. Y.)* 72:2818–2824.
- Snook, R. R., L. D. Bacigalupe, and A. J. Moore. 2010. The quantitative genetics and coevolution of male and female reproductive traits. *Evolution (N. Y.)* 64:1926–1934.
- Trivers, R. L. 1972. Parental investment and sexual selection. Pp. 136–179 in *Sexual selection & the descent of man*. Aldine Publishing Company, Illinois.
- Veltsos, P., Y. Fang, A. R. Cossins, R. R. Snook, and M. G. Ritchie. 2017. Mating system manipulation and the evolution of sex-biased gene expression in *Drosophila*. *Nat. Commun.* 8:2072.
- Veltsos, P., E. Gregson, B. Morrissey, J. Slate, A. Hoikkala, R. K. Butlin, and M. G. Ritchie. 2015. The genetic architecture of sexually selected traits in two natural populations of *Drosophila montana*. *Heredity (Edinb)* 115:565–572.
- Wallace, B. 1975. Hard and soft selection revisited. *Evolution (N. Y.)* 29:465–473.
- Wigby, S., and T. Chapman. 2004. Female resistance to male harm evolves in response to manipulation of sexual conflict. *Evolution (N. Y.)* 58:1028–1037.
- Yun, L., P. J. Chen, K. E. Kwok, C. S. Angell, H. D. Rundle, and A. F. Agrawal. 2018. Competition for mates and the improvement of non-sexual fitness. *Proc. Natl. Acad. Sci.* 115:6762–6767.
- Yun, L., P. J. Chen, A. Singh, A. F. Agrawal, and H. D. Rundle. 2017. The physical environment mediates male harm and its effect on selection in females. *Proc. R. Soc. B* 284:20170424.

Associate Editor: N. G. Prasad  
 Handling Editor: A. G. McAdam

## Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Supplementary information

Supplementary information

Supplementary information

Table 1. A non-exhaustive collection of experimental evolution studies on the role of sexual selection and/or sexual conflict that compared regimes of enforced monogamy and either polyandry or polygamy in populations of *Drosophila* and other organisms, showing the vast diversity of experimental design.

Figure SI-A1. Evolutionary trajectories under different genetic architectures of the controller genes.

Figure SI-B1. Histograms of the numbers of offspring produced by individuals of each sex, under different mating systems and either hard or soft selection on female fecundity.

Figure C1. A comparison of simulation results showing the magnitude of genome feminization at evolutionary equilibrium under  $N_C = 40$  general controllers (left panel) and those simulated under  $N_f = N_m = 20$  sex-specific controllers (right panel).

Figure C2. A comparison of simulation results showing the magnitude of genome feminization at evolutionary equilibrium under  $N_C = 40$  general controllers (left panel) and those simulated under  $N_f = N_m = 20$  sex-specific controllers (right panel).

Figure C3. A comparison of simulation results showing the magnitude of genome feminization at evolutionary equilibrium under  $N_C = 40$  general controllers (left panel) and those simulated under  $N_f = N_m = 20$  sex-specific controllers (right panel).

Figure C4. A comparison of simulation results showing the magnitude of genome feminization at evolutionary equilibrium under  $N_C = 40$  general controllers (left panel) and those simulated under  $N_f = N_m = 20$  sex-specific controllers (right panel).

Figure C5. A comparison of simulation results showing the magnitude of genome feminization at evolutionary equilibrium under  $N_C = 40$  general controllers (left panel) and those simulated under  $N_f = N_m = 20$  sex-specific controllers (right panel).

Figure SI-D1. The magnitude of genome feminization as the intensity of female fecundity competition ( $\alpha$ ) increases under different mating systems.

Figure SI-D2. The figure presents the same information as in Figure SI-D1.

Figure SI-E1. The magnitude of genome feminization as the intensity of male mating competition ( $\beta$ ) increases under different mating systems.

Figure SI-E2. The figure presents the same information as in Figure SI-E1.

Figure SI-F1. (a) Magnitude of genome feminization ( $\theta_f - \theta_m$ ) under polyandry at different combinations of female death rate and the intensity of condition-dependent selection for female tolerance of male harassment,  $\gamma$ .

Figure SI-F2. The figure presents the same information as in Figure 5a.

Figure SI-F3. The figure presents the same information as in Figure 5a.

Figure SI-F4. The figure presents the same information as in Figure 5a.

Figure SI-F5. The figure presents the same information as in Figure 5a.

Figure SI-F6. The figure presents the same information as in Figure 5a.

Figure SI-F7. The figure presents the same information as in Figure SI-F3.

Figure SI-F8. The figure presents the same information as in Figure SI-F4.

Figure SI-F9. The figure presents the same information as in Figure SI-F5.

Figure SI-F10. The figure presents the same information as in Figure SI-F6.

Figure SI-G1. The figure represents the same information as in figure 4c–d, where panels (a) and (c) correspond to figure 4c, showing the results under hard selection at the end of 20 generations, or 100 generations.

Figure SI-G2. The figure represents the same information as in figure 5b.