# Sexually antagonistic selection: a review of the theory and its implications

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**Abstract.** Sexually antagonistic selection arises when females and males have different fitness optima for traits with a shared genetic basis, so that the same alleles are favoured in one sex but disfavoured in the other. It has been implicated in a wide range of ecological and evolutionary processes, from the maintenance of a sex load to the evolution of sex chromosomes. Mathematical models have long been central to understanding sexually antagonistic selection and its consequences. Here, we review the theory of sexual antagonism across population genetic, adaptive dynamics, and quantitative genetic models. We highlight how these approaches give concordant and complementary descriptions of evolution in response to sex-specific selection and consider extensions incorporating genetic drift, non-random mating, demographic effects, jointly evolving multiple traits, sex-specific inheritance, and the evolution of genetic modifiers. We then discuss how these results bear on the sex load, the maintenance of genetic polymorphism, the evolution of sex linkage and sex chromosomes, and the origins of sexual dimorphism. By making assumptions and mechanisms explicit across approaches, we identify which conclusions are robust and which depend on specific modelling choices and finally outline open questions for future theoretical work.

**keywords:** sex-specific selection; sex load; balancing selection; dominance reversal; polymorphism; sex chromosomes; sex-specific gene expression; sexual dimorphism; antagonistic pleiotropy; evolutionary theory

# 1 Introduction

Males and females frequently differ in their reproductive roles, mating strategies, or ecological niche [1–3]. This generates divergent fitness landscapes such that fitness-maximising trait values differ between the sexes, and selection is sex-specific [4–7]. When such selection acts on traits with a shared genetic basis, different genotypes will be favoured in males and females, in which case the same allele can have opposite fitness effects across the sexes, a situation referred to as sexual antagonism or intra-locus sexual conflict (Box I) [1, 5, 8–11].

Sexual antagonism has been proposed to influence several key ecological and evolutionary processes. It can limit or redirect adaptive change across the sexes [4, 7, 12, 13], diminish average fitness (imposing a "sex load", see Box I [9, 14]), maintain genetic variation [15–18], as well as drive the evolution of sexual dimorphism, e.g., by favouring sex-specific gene expression [19–22]. It can also shape genomic architecture by affecting the distribution of antagonistic alleles across autosomes and sex chromosomes and by altering local recombination patterns [23–26]. Moreover, sexually antagonistic selection can have implications for demography through its effects on sex-specific vital rates [27–31], the evolution of the primary sex ratio [32, 33], transitions between sex determination systems [34, 35], and the emergence of separate sexes in hermaphrodites [36, 37].

Much of our understanding of sexual antagonism has derived from theoretical studies leveraging three main approaches (Fig. 1). Population genetic models describe allele-frequency change at one or several loci and identify conditions under which antagonistic polymorphism is maintained or lost across genomic regions experiencing sex-specific selection, mutation, and recombination [15, 19, 25, 30–32, 38–45]. A second class of models, in the adaptive dynamics tradition, explores the fate of many alternative alleles, assuming that mutations are rare and have small effect, using invasion analyses to study longer-term evolutionary change, including the gradual emergence of discrete polymorphisms [18, 28, 37, 46–49]. Finally, quantitative genetic models track the evolution of traits that are highly polygenic and investigate how a shared genetic basis, cross-sex covariances, and genetic constraints influence the evolutionary trajectories of sex-specific traits and the persistence of antagonism [4, 7, 12, 14, 22, 50, 51, 51–53]. Together, these approaches focus on complementary aspects of evolution in response to sexually antagonistic selection.

Here, we review these models and their insights regarding sexual antagonism. Using existing results, as well as a few new ones, we examine how different assumptions about genetic bases, fitness effects and demography affect modelling outcomes. Throughout, we emphasise the conditions under which sexually antagonistic selection imposes a sex load and when it maintains genetic variation, both in the

short term with specific alleles and in the long term when allowing new alleles to continuously arise. For those who wish to skip the theory, we end with a general discussion of the broader implications of sexually antagonistic selection for fitness, variation, genomic architecture, and sexual dimorphism (section 4).

## 2 Different theoretical approaches

### 2.1 Life cycle and trait architecture

Sexual antagonism is typically envisaged as arising when male and female fitness vary in opposite ways as a consequence of similar trait changes (e.g., [8, 9, 54]). A common starting point in theoretical studies is therefore to consider a continuous trait (e.g., weight, colouration, growth rate, gene expression), whose value we denote generally as $z$ and when necessary as $z_{\mathrm{m}}$ in males and $z_{\mathrm{f}}$ in females. Most models (e.g., [5, 15, 18, 55]) assume a large, density-regulated population with discrete generations where adult males and females mate randomly, produce many offspring, and then die.

Male and female fitness depend on expressed trait values, and are denoted $w_{\mathrm{m}}(z_{\mathrm{m}})$ for a male expressing $z_{\mathrm{m}}$ and $w_{\mathrm{f}}(z_{\mathrm{f}})$ for a female expressing $z_{\mathrm{f}}$ [†]. Sexual antagonism occurs when $w_{\mathrm{m}}(z_{\mathrm{m}})$ and $w_{\mathrm{f}}(z_{\mathrm{f}})$ vary in opposite ways in response to a genetic change with concordant effects on male and female trait value. This results in different alleles increasing fitness in each sex, the consequences of which have been extensively modelled. In the rest of this section, we review population genetic, adaptive dynamic, and quantitative genetic approaches and their key findings, focusing on the case where sexually antagonistic genetic variation lies on autosomes. In section 3, we then review a number of extensions to this baseline scenario, including non-autosomal inheritance.

### 2.2 Single-locus population genetic models

The earliest theoretical treatments of sexual antagonism analysed population genetic models [15, 19, 38, 56–58]. These studies describe the short-term response to sexually antagonistic selection at a single locus with two alternative alleles, $\mathrm{A}_1$ and $\mathrm{A}_2$, that have sex-specific effects on fitness (Figure 1A). Such analyses then characterise evolution by tracking changes in allelic frequency at the focal locus under different assumptions about the fitness effects of alleles, often focusing on the conditions for which

[†]Here, individual fitness refers to relative number of offspring produced, i.e., expected number of offspring produced before regulation, potentially incorporating effects of $z$ on survival, fertility, and mating success, measured relative to other individuals of the same sex. For the models considered in this paper, it is proportional to, but distinct from, absolute fitness, defined as the expected total number of recruited offspring.

sexual antagonism favours the maintenance of both alleles in the population.

### 2.2.1 Strong selection

When sexually antagonistic selection is strong, individuals with different genotypes can differ substantially in fitness across the sexes, leading allele frequencies to diverge between males and females. Let the frequency after selection of allele $A_2$ be $p_f$ in females and $p_m$ in males, and $z_{u,ij}$ be the trait value expressed by an individual of sex $u \in \{f, m\}$ carrying alleles $A_i$ and $A_j$ ($i, j \in \{1, 2\}$). The changes in allele frequencies in females and males over one generation can then be written as

$$\begin{aligned} \Delta p_f &= p_m p_f \frac{w_f(z_{f,22})}{\overline{w_f}} + \frac{1}{2}\left[p_m(1-p_f) + p_f(1-p_m)\right]\frac{w_f(z_{f,12})}{\overline{w_f}} - p_f \\ \Delta p_m &= p_m p_f \frac{w_m(z_{m,22})}{\overline{w_m}} + \frac{1}{2}\left[p_m(1-p_f) + p_f(1-p_m)\right]\frac{w_m(z_{m,12})}{\overline{w_m}} - p_m \end{aligned} \tag{1}$$

(eq. 1 in Kidwell et al. [15], see also Owen [56] p. 98 and Haldane [57] p. 1108, and [16, 19, 59, 60] for equivalent expressions for sex-linked alleles), which depends only on the relative fitnesses of the three genotypes in each sex. Here, $\overline{w_u}$ represents the average fitness in sex $u$, given by $(1-p_m)(1-p_f)\ w_u(z_{u,11}) + \left[p_m(1-p_f) + p_f(1-p_m)\right]\ w_u(z_{u,12}) + p_m p_f\ w_u(z_{u,22})$.

Starting with Kidwell et al. [15] (their eq. 3), it has been standard to write eq. (1) in terms of the sex-specific selection coefficients,

$$s_f = \frac{w_f(z_{f,11}) - w_f(z_{f,22})}{w_f(z_{f,11})} \quad \text{and} \quad s_m = \frac{w_m(z_{m,22}) - w_m(z_{m,11})}{w_m(z_{m,22})}, \tag{2a}$$

which measure the relative fitness costs to female and male homozygotes for expressing their least preferred allele, and sex-specific dominance coefficients,

$$h_f = \frac{w_f(z_{f,11}) - w_f(z_{f,12})}{w_f(z_{f,11}) - w_f(z_{f,22})} \quad \text{and} \quad h_m = \frac{w_m(z_{m,22}) - w_m(z_{m,12})}{w_m(z_{m,22}) - w_m(z_{m,11})}. \tag{2b}$$

The parameters $h_f$ and $h_m$ give the proportion of the fitness costs $s_m$ and $s_f$ experienced by heterozygotes (with $h_u = 1/2$ corresponding to fitness additivity in sex $u$). Because these coefficients are defined purely in terms of fitness, they make no assumption about the underlying basis of genotypic effects, such that equations (1)–(2) apply to a wide range of biological scenarios.

In particular, non-additive genetic effects on fitness ($h_f, h_m \neq 1/2$) can arise for two distinct reasons (Fig. 2A). First, they can reflect non-linear genotype–phenotype maps, where heterozygotes express trait values that are not exactly halfway between the homozygotes (i.e., phenotypic dominance Fig. 2A1

[47, 61]). Second, they can arise even when genetic effects on phenotypes are additive, if male or female fitness is a non-linear function of trait value (i.e., non-linear phenotype-fitness maps Fig. 2A2 [16, 17, 62]).

From the analysis of equation (1), there are two possible evolutionary outcomes for allele frequencies at equilibrium. Either one allele fixes, the identity of which depends on parameters and sometimes initial conditions, or the two alleles invade one another when rare and are maintained in a protected polymorphism (i.e., there exist equilibrium frequencies $0 < p_{\mathrm{f}}^{*} < 1$ and $0 < p_{\mathrm{m}}^{*} < 1$ such that $\Delta p_{\mathrm{f}} = \Delta p_{\mathrm{m}} = 0$), which occurs when

$$\frac{h_{\mathrm{m}}}{1 - h_{\mathrm{f}} + s_{\mathrm{m}} h_{\mathrm{m}}} < \frac{s_{\mathrm{f}}}{s_{\mathrm{m}}} < \frac{1 - h_{\mathrm{m}}}{h_{\mathrm{f}}(1 - s_{\mathrm{m}})}. \tag{3}$$

Such selection allowing alternate alleles to spread when rare, maintaining a polymorphism, is known as balancing selection.

Analysis of condition (3) has highlighted that two mechanisms can generate balancing selection (see eq. 7 in Kidwell et al. [15]). The first is adaptive dominance reversal (Fig. 2A), whereby alleles are more dominant when beneficial and more recessive when deleterious ($h_{\mathrm{m}}$ and $h_{\mathrm{f}}$ sufficiently low). This creates a sex-averaged heterozygote advantage in fitness and hence negative frequency-dependence at the allelic level, because rare alleles are more often expressed in heterozygotes. The second mechanism arises when sexually antagonistic selection is both strong and approximately balanced between the sexes (high $s_{\mathrm{m}} \approx s_{\mathrm{f}}$), such that intense competition within each sex generates negative frequency-dependence. When one allele becomes common, it increases the mean fitness of the sex in which is it is beneficial but reduces mean fitness of the other. A rare allele in that other sex is then favoured because it performs well relative to a poorly competitive background and can therefore invade more easily. This mechanism is analogous to soft selection in models of local adaptation (as highlighted in [38, 63]).

Eq. (1) does not generally admit a simple closed-form solution. Consequently, studies that need to track allele-frequency change explicitly (e.g., [42, 43, 64, 65]) or that incorporate additional ecological effects such as population structure (e.g., [44, 66]), typically make the simplifying assumption that selection is weak. Weak selection can be modelled using two different approaches (Figure 2B): one assumes that fitness varies little with trait values, while the second assumes that trait values vary little with genetic variation [67]. Both approaches result in small fitness differences but yield different insights, as we explore next.

### 2.2.2 Weak selection via mild fitness differences

The most common approach to weak selection in population genetic studies of sexual antagonism has been to allow alleles to have large effects on traits but only weak effects on fitness (the $w$-weak limit of [67]; top panel of Figure 2B). Formally, this corresponds to $s_\mathrm{m} \sim \mathcal{O}(\epsilon)$ and $s_\mathrm{f} \sim \mathcal{O}(\epsilon)$ in eq. (2), where $\epsilon \ll 1$ is a small parameter (e.g., [5, 17, 42, 43, 45, 64, 65]).

Under weak selection, allele-frequency differences between males and females remain small ($|p_\mathrm{f} - p_\mathrm{m}| \sim \mathcal{O}(\epsilon)$). Evolutionary dynamics can therefore be tracked using the sex-averaged allele frequency ($p = p_\mathrm{f}/2 + p_\mathrm{m}/2$, corresponding to reproductive-value weighted average allele-frequency change [68]). To leading order in $\epsilon$, frequency change is given by

$$\Delta p = \frac{p(1-p)}{2}\Big[s_\mathrm{m}\big(p + (1-h_\mathrm{m})(1-2p)\big) - s_\mathrm{f}\big(p + h_\mathrm{f}(1-2p)\big)\Big] + \mathcal{O}\left(\epsilon^2\right) \tag{4}$$

in the notation of Kidwell et al. [15], where the term between square brackets gives the direction of selection as a function of $p$ (e.g., eq. 1 in Connallon and Clark [43] and eq. 2 in Mullon et al. [42]).

A key result from eq. (4) is that $\Delta p$ has the same sign for all $p$, such that balanced polymorphism is impossible, unless

$$\frac{h_\mathrm{f}}{1-h_\mathrm{m}} < \frac{s_\mathrm{m}}{s_\mathrm{f}} < \frac{1-h_\mathrm{f}}{h_\mathrm{m}}, \tag{5}$$

which is the weak-selection equivalent of eq. (3). The frequency-dependent effects generated by strong sexual antagonism seen in eq. (3) and maintaining polymorphism have vanished (they appear only to order $\epsilon^2$). Thus, eq. (5) can never be satisfied if $h_\mathrm{m} + h_\mathrm{f} > 1$, so this equation formalises the requirement for adaptive dominance reversal for maintaining polymorphism ($h_\mathrm{m}, h_\mathrm{f}$ must both be sufficiently low).

Because alleles are often expected to have weak effects on fitness, this result has led many authors to argue that the prevalence of sexually antagonistic balancing selection is primarily limited by the scope for dominance reversal [15, 16, 38, 62, 69]. Although the prospect of widespread dominance-reversal was initially met with scepticism–with such dominance patterns dismissed as "too good to be true" [38, 69] (reviewed in [62, 70])–subsequent work demonstrated that they may arise readily for alleles when mutations have large additive effects on trait value and fitness in each sex is a concave function of trait value [16, 17] (bottom of Figure 2A).

### 2.2.3 Weak selection via small allelic effects

The second approach to implementing weak selection is to assume that allelic effects on phenotypes are small ($\delta$–weak limit, [67], bottom of Figure 2B), such that genotypes encode similar trait values (i.e., $\delta_u = |z_{u,11} - z_{u,22}| \sim \mathcal{O}(\epsilon)$; see [18, 37, 48, 49, 71, 72] for studies of sexually antagonistic selection assuming weak allelic effects). This assumption leads to an expression for allele-frequency change that is similar to eq. (4) (see Appendix A.1 for derivation and comparison).

Where alleles have additive effects on trait value that are the same in males and females (such that $z_{ij} = z_{\mathrm{m},ij} = z_{\mathrm{f},ij}$), allele-frequency change simplifies to

$$\Delta p = (z_{22} - z_{11})\, p(1-p) \underbrace{\left[\frac{S_\mathrm{m}(z_{11}) + S_\mathrm{f}(z_{11})}{4}\right]}_{=S(z_{11})} + \mathcal{O}\left(\epsilon^2\right), \tag{6}$$

where

$$S_\mathrm{f}(z) = \frac{w'_\mathrm{f}(z)}{w_\mathrm{f}(z)} \quad \text{and} \quad S_\mathrm{m}(z) = \frac{w'_\mathrm{m}(z)}{w_\mathrm{m}(z)} \tag{7}$$

are the linear selection gradients on female and male traits, respectively ([55, 73]; throughout a $'$ denotes a derivative). A positive value of $S_u(z)$ indicates that directional selection in sex $u$ favours an increase in trait value, whereas a negative value indicates selection favouring a decrease in $z$. Equation (6) shows that the direction of allele-frequency change is independent of allele frequency $p$ and is determined solely by the sex-averaged linear selection gradient, $S(z_{11})$. As a result, any allele that can successfully invade a population will also go to fixation, provided that there is directional selection (here provided that $S(z_{11}) \neq 0$).

Where alleles have non-additive effects on trait value that are sufficiently similar across the sexes (i.e., when allelic dominance for phenotype is similar in males and females), the direction of allele-frequency change is again entirely determined by $S(z_{11})$, which therefore also determines which allele goes to fixation (eq. (A-4) in Appendix A.1). By contrast, when non-additive effects on trait value differ across the sexes such that dominance reversal arises from the genotype–phenotype map, a balanced polymorphism can be maintained (eq. (A-2) in Appendix A.1). Here, the key difference with alleles that have weak fitness effects (Section 2.2.2 above) is that dominance effects on fitness can no longer arise through non-linearities in the mapping from phenotype to fitness when allelic effects on traits are small. This is because phenotypes are so similar that male and female fitness essentially change linearly with trait value over the phenotypic range spanned by the alleles (Fig. 2B2).

## 2.3 Models of gradual evolution

A second approach to study sexual antagonism uses invasion analysis to describe the gradual evolution of trait $z$ [74–78]. Now commonly referred to as adaptive dynamics [79–85], these models assume $z$ is determined by a single locus at which mutations are rare and introduce small changes in trait value following the continuum-of-alleles model [86] (Figure 1B). Although initially developed for haploid asexual populations [79, 80], this approach extends directly to sexual diploids (e.g., [87, 88]) and has since been used to study evolution under sexual antagonism, with particular emphasis on the emergence and maintenance of adaptive polymorphism (e.g., [18, 28, 37, 46–49]).

### 2.3.1 Invasion fitness and the nature of selection

The basis of invasion analysis is the geometric growth rate of a mutant lineage arising from a single initial copy in a large resident population at demographic equilibrium, which is commonly referred to as the mutant's invasion fitness (i.e., the leading eigenvalue in a stability analysis). The resident population is initially considered to be monomorphic (fixed) for an allele encoding trait value $z$ in both sexes. Under the scenario outlined in Section 2.1, the invasion fitness of a mutant allele encoding $z_\bullet$ (when homozygous in both sexes) in such a population is

$$W(z_\bullet, z) = \frac{1}{2}\left(\frac{w_\mathrm{f}\left(\frac{z_\bullet+z}{2}\right)}{w_\mathrm{f}(z)} + \frac{w_\mathrm{m}\left(\frac{z_\bullet+z}{2}\right)}{w_\mathrm{m}(z)}\right), \tag{8}$$

when allelic effects on phenotype are additive. Eq. (8) can be read as the expected number of heterozygote mutant offspring produced by a rare mutant heterozygote (eq. 1 in Van Dooren et al. [46], eq. 2 in, eq. B2 in Lesaffre et al. [49], eq. 2 in Flintham et al. [18], and eq. S10 in Siljestam et al. [47]). Equivalent expressions for additive sex-linked loci are given in Appendix D of [18].

A mutant allele can invade when rare if, and only if, $W(z_\bullet, z) > 1$. Otherwise the mutant allele goes extinct with certainty. When the mutant effect is small ($|z_\bullet - z| \sim \mathcal{O}(\epsilon)$, as in section 2.2.3), invasion fitness can be expressed as

$$W(z_\bullet, z) = 1 + (z_\bullet - z)S(z) + \frac{(z_\bullet - z)^2}{2}H(z) + \mathcal{O}\left(\epsilon^3\right) \tag{9}$$

where

$$S(z) = \left.\frac{\partial W(z_\bullet, z)}{\partial z_\bullet}\right|_{z_\bullet = z} = \frac{1}{2}\frac{S_\mathrm{f}(z) + S_\mathrm{m}(z)}{2}, \tag{10}$$

is the directional selection gradient on allelic value (obtained by averaging the sex-specific selection

gradients on trait value introduced in eq. 7). The second-order term

$$H(z) = \left.\frac{\partial^2 W(z_\bullet, z)}{\partial {z_\bullet}^2}\right|_{z_\bullet = z} = \frac{1}{4}\frac{H_\mathrm{f}(z) + H_\mathrm{m}(z)}{2} \tag{11}$$

is the quadratic selection gradient on allelic value, where

$$H_\mathrm{f}(z) = \frac{w''_\mathrm{f}(z)}{w_\mathrm{f}(z)} \quad \text{and} \quad H_\mathrm{m}(z) = \frac{w''_\mathrm{m}(z)}{w_\mathrm{m}(z)} \tag{12}$$

are the quadratic selection gradients on trait value in females and males. They measure the curvature of the sex-specific fitness functions at $z$ [55, 73]: fitness in sex $u$ is locally accelerating with trait value when $H_u(z) > 0$ and decelerating when $H_u(z) < 0$.

### 2.3.2 Mutation-limited evolution

According to eq. (9), as long as there is directional selection ($S(z) \neq 0$), the fate of a rare mutant depends on the sign of this selection. Mutant alleles that increase the trait value can invade when $S(z) > 0$, whereas alleles decreasing the trait can invade when $S(z) < 0$. Furthermore, because the phenotypic effect of an allele is assumed small, the direction of selection is independent of allele frequency (recall eq. (6)). Thus, any mutant that invades will fix and replace the resident allele (this is an instance of the *invasion-implies-substitution* principle, which is known to hold under a wide range of scenarios when allelic effects are weak [89]). Trait evolution can thus be represented as a sequence of substitutions, with the trait increasing when $S(z) > 0$ and decreasing when $S(z) < 0$.

Eventually, a sequence of substitutions may bring the population to a so-called singular trait value $z^*$ at which the directional selection gradient vanishes,

$$z^* \quad \text{such that} \quad S(z^*) = 0. \tag{13}$$

At this point, the male and female gradients balance exactly, $S_\mathrm{f}(z^*) = -S_\mathrm{m}(z^*)$ (recall eq. (10)). Any such singular value therefore corresponds to a compromise between the sexes. Convergence to $z^*$ occurs when $S'(z^*) < 0$, in which case $z^*$ is said to be convergence stable [74–77, 80].

Once the population has evolved to a convergence stable trait value $z^*$, the invasion fitness of nearby mutants is determined by $H(z^*)$ (eq. (8)). If $H(z^*) < 0$, selection is stabilising: $z^*$ is a local maximum of sex-averaged fitness, and any nearby mutant is selected against. By contrast, if $H(z^*) > 0$, selection is locally disruptive: $z^*$ is a local minimum of sex-averaged fitness. An allele encoding $z^*$ can then be invaded by a nearby mutant but is not replaced. Instead, a protected two-allele polymorphism is

established (see Appendix A.2 here; also eq. A1 in [80] or [90]). Subsequent mutations with increasingly divergent trait values may then invade and replace existing alleles, leading to the emergence of two progressively differentiated alleles in a process referred to as evolutionary branching [80].

Combining the requirements for convergence stability ($S'(z^*) < 0$) and disruptive selection ($H(z^*) > 0$), evolutionary branching driven by sexual antagonism occurs when

$$0 \; < \; H_{\mathrm{f}}(z^*) + H_{\mathrm{m}}(z^*) \; < \; S_{\mathrm{f}}(z^*)^2 + S_{\mathrm{m}}(z^*)^2. \tag{14}$$

(eq. 4 in Flintham et al. [18]). Satisfying eq. (14) in addition to the requirement that $z^*$ is a singular point (such that $S_{\mathrm{f}}(z^*) = -S_{\mathrm{m}}(z^*)$) requires strong within-sex selection in both sexes (i.e., large $S_{\mathrm{f}}(z^*)$ and $S_{\mathrm{m}}(z^*)$). This is because strong selection in both sexes prevents the selective interests of one sex from overwhelming those of the other. As a result, mutations that benefit the more poorly adapted sex are systematically favoured thereby driving a population towards the compromise $z^*$, in spite of it being a local minimum for sex-averaged fitness (as $H(z^*) > 0$).

Condition (14) for evolutionary branching is restrictive: not only does it require strong selection in both sexes, but it also requires accelerating fitness landscapes ($0 < H_{\mathrm{f}}(z^*) + H_{\mathrm{m}}(z^*)$). Only then will high fitness male genotypes and high fitness female genotypes emerge via branching, with low fitness intermediates, i.e., the opposite of the requirement for adaptive dominance reversal via the phenotype-fitness map. The requirement for accelerating fitness landscapes means that heterozygotes on average have low fitness close to $z^*$ which we refer to as "maladaptive dominance reversal" as it is opposite to the requirement for polymorphism with only two alleles (Eq. (5)). The restrictive nature of these conditions has led to the suggestion that long-term polymorphism is an unlikely outcome of sexual antagonism when alleles have similar effects on male and female traits [18, 47], except for traits that show unresolvable genetic constraints between the sexes such as the allocation to male versus female function in hermaphrodites (Box I [37]).

### 2.3.3 Adaptive dynamics under sex-specific genotype-phenotype maps

The above considers alleles acting in a sex-concordant manner: they encode the same trait value when homozygous in males and females and combine additively in heterozygotes of both sexes. However, homozygous allelic effects and dominance relationships in heterozygotes may be different in males and females. Such sex-specific genotype-phenotype maps can still be incorporated into adaptive dynamics models.

When homozygous allelic effects remain sex-concordant but dominance relationships do not–such that the phenotype expressed by male and female heterozygotes differ– the population still converges to the compromise trait value $z^*$ (where $S_f(z^*) = -S_m(z^*)$), provided dominance remains intermediate in each sex (ensuring that invasion implies substitution). However, sex-specific dominance can influence the conditions for evolutionary branching and so the scope for polymorphism. Siljestam et al. [47] show, for a specific genotype-phenotype map generating adaptive dominance reversal for phenotype, that this facilitates branching. The mechanism is that rare mutant alleles are primarily found in heterozygotes, and when these heterozygotes express phenotypes closer to the sex-specific optima, they have higher sex-averaged fitness than resident homozygotes, generating negative frequency dependence at the genetic level. We generalise this result in Appendix B to arbitrary sex-specific dominance, showing how genotype–phenotype mapping enters the branching condition. In particular, the lower bound ("0") in eq. (14) becomes negative when mutant alleles are more dominant in the sex in which they are beneficial (see eq. B-7b in Appendix B; Fig. 3C for example simulations).

The above assumes that homozygous allelic effects are constrained to be identical in males and females. When alleles can have different effects in male and female homozygotes, the population no longer converges towards a compromise trait value $z^*$. Instead, male and female trait values diverge ultimately reaching their respective optima, i.e., $z_f^*$ such that $S_f(z_f^*) = 0$ and $z_m^*$ such that $S_m(z_m^*) = 0$ [28, 46]. Nevertheless, the speed at which the sexes approach these optima depends on how strongly correlated mutational effects at the locus are across the sexes (Fig. 3D [46]).

The conclusions of this section rely on mutation-limited evolution with weak phenotypic effects. They differ from models in which rare mutations can have larger effects on trait values. In Fisher's geometric models of sex-specific adaptation, mutations with large additive effect can generate transient balanced polymorphisms because heterozygotes may have higher sex-averaged fitness than either homozygote, owing to the curvature of the male and female fitness landscapes (e.g. satisfying eq. 5, [5, 17]). Such polymorphisms can arise during adaptation toward a compromise trait value $z^*$ or toward sex-specific optima $z_f^*$ and $z_m^*$, but they are not generally expected to persist indefinitely when additional alleles can arise. They may instead be displaced by mutations with higher sex-averaged fitness, or lost through drift, while still contributing to standing genetic variation during adaptation [17].

### 2.4 Quantitative genetic models

The third approach to study the evolutionary genetics of sexual antagonism is quantitative genetics. These models typically assume that traits are influenced by many autosomal loci of small effect such

that the joint distribution of additive allelic effects expressed in females and males is approximately Gaussian [4, 7, 14, 50, 51] (Figure 1C). Sexual antagonism arises because allelic effects expressed in one sex can be correlated with the effects of the same alleles when expressed in the other sex, generating cross-sex genetic covariances. Studies using this approach are often interested in how such shared genetic architecture constrains the response of males and females to sex-specific selection and limits the rate at which sexual dimorphism can evolve. Because additive genetic effects are assumed to be multivariate normal, the evolution of males and females can be described by the joint evolution of sex-specific trait means and their associated genetic (co)variances.

### 2.4.1 Changes in mean trait values in males and females

One of the first treatments was by Lande [4] who, assuming constant additive genetic (co)variances, showed that the changes in the mean female and male phenotypes, $\overline{z}_\mathrm{f}$ and $\overline{z}_\mathrm{m}$, satisfy

$$\begin{aligned} \Delta\overline{z}_\mathrm{f} &= \sigma^2_\mathrm{f} S_\mathrm{f}(\overline{z}_\mathrm{f}) + \sigma_\mathrm{fm} S_\mathrm{m}(\overline{z}_\mathrm{m}), \\ \Delta\overline{z}_\mathrm{m} &= \sigma^2_\mathrm{m} S_\mathrm{m}(\overline{z}_\mathrm{m}) + \sigma_\mathrm{fm} S_\mathrm{f}(\overline{z}_\mathrm{f}), \end{aligned} \tag{15}$$

where $\sigma^2_\mathrm{f}$ and $\sigma^2_\mathrm{m}$ are the additive genetic variances for the trait in females and males, respectively, and $\sigma_\mathrm{fm}$ is the additive genetic covariance between the sexes, which is typically assumed to be positive (see also e.g., eq. (1) in [50], eq. (1) in [7]).

Eq. (15) makes explicit that each sex responds both to selection acting directly on that sex (first terms) and to selection acting on the other sex through a correlated response (second terms). The direct responses are proportional to the sex-specific additive genetic variances $\sigma^2_\mathrm{f}$ and $\sigma^2_\mathrm{m}$, while the cross-sex terms are proportional to the between-sex additive genetic covariance $\sigma_\mathrm{fm}$. Sexually antagonistic selection arises when the selection gradients $S_\mathrm{f}(\overline{z}_\mathrm{f})$ and $S_\mathrm{m}(\overline{z}_\mathrm{m})$ have opposite signs but a shared genetic architecture imposes $\sigma_\mathrm{fm} > 0$. As a result, the correlated-response terms oppose the direct responses, impeding adaptation in each sex.

In the special case where allelic effects are identical in males and females so that the between-sex correlation is one ($\sigma_\mathrm{fm} = \sigma^2_\mathrm{f} = \sigma^2_\mathrm{m}$) and $\overline{z}_\mathrm{f} = \overline{z}_\mathrm{m}$, eq. (15) collapses to the response of a single shared phenotype to selection. Evolution is then predicted to approach a singular *compromise* trait value $z^*$ at which directional selection vanishes (eq. (13)), consistent with the weak allelic effect models from population genetics (eq. 6) and adaptive dynamics (eq. (10)).

By contrast, when the between-sex genetic correlation is less than one and there is additive genetic variance in each sex ($\sigma^2_\mathrm{f} > 0$ and $\sigma^2_\mathrm{m} > 0$), divergent male and female selection gradients can drive

female ($\overline{z}_\mathrm{f}$) and male ($\overline{z}_\mathrm{m}$) traits in different directions, with the rate of divergence depending on the magnitude of $\sigma_\mathrm{fm}$ relative to $\sigma^2_\mathrm{f}$ and $\sigma^2_\mathrm{m}$ (Fig. 4). Thus, although a positive but incomplete between-sex correlation ($0 < \sigma_\mathrm{fm}/(\sigma_\mathrm{f}\sigma_\mathrm{m}) < 1$) slows the response to sex differences in selection, it does not in itself prevent adaptation towards the optimum for each sex (see chap. 13 in [91] for more general treatment).

During this process of adaptation towards the sex-specific optima, a transient sex load arises while trait means are displaced from their optima, an effect quantified by Matthews et al. [14]. Moreover, if male and female optima shift sufficiently frequently, the sexes spend little time at their optima ((Fig. 4)C), in which case a depression in sex-averaged fitness persists as a form of lag load ([13], and see [12] for explicit genetic simulations).

### 2.4.2 Changes in the genetic variance and covariance

Genetic variances within traits and covariances among traits can themselves change through the combined effects of selection, mutation, and drift (e.g., [45, 55, 91–95]). Existing theoretical work in the context of sex-specific adaptation has focused on changes in $\sigma^2_\mathrm{f}$, $\sigma^2_\mathrm{m}$, and $\sigma_\mathrm{fm}$ induced by selection within a generation (i.e., after selection and before segregation and recombination). Barker et al. [51] analysed the effects of selection on these (co)variances in the absence of directional selection (i.e., when $S_\mathrm{f}(\overline{z}_\mathrm{f}) = S_\mathrm{m}(\overline{z}_\mathrm{m}) = 0$), an approach later extended to include directional selection by McGlothlin et al. [7]. Together, these studies show that within-generation changes due to selection can be expressed as

$$\begin{aligned}
\left[\Delta\sigma^2_\mathrm{f}\right]_\mathrm{sel} &= -\frac{1}{4}\left(\Delta\overline{z}_\mathrm{f}\right)^2 + \frac{1}{2}\left[\sigma^4_\mathrm{f} H_\mathrm{f}(\overline{z}_\mathrm{f}) + \sigma^2_\mathrm{fm} H_\mathrm{m}(\overline{z}_\mathrm{m})\right] \\
\left[\Delta\sigma^2_\mathrm{m}\right]_\mathrm{sel} &= -\frac{1}{4}\left(\Delta\overline{z}_\mathrm{m}\right)^2 + \frac{1}{2}\left[\sigma^4_\mathrm{m} H_\mathrm{m}(\overline{z}_\mathrm{m}) + \sigma^2_\mathrm{fm} H_\mathrm{f}(\overline{z}_\mathrm{f})\right] \\
\left[\Delta\sigma_\mathrm{fm}\right]_\mathrm{sel} &= -\frac{1}{4}\left(\Delta\overline{z}_\mathrm{f}\right)\left(\Delta\overline{z}_\mathrm{m}\right) + \frac{1}{2}\sigma_\mathrm{fm}\left[\sigma^2_\mathrm{f} H_\mathrm{f}(\overline{z}_\mathrm{f}) + \sigma^2_\mathrm{m} H_\mathrm{m}(\overline{z}_\mathrm{m})\right]
\end{aligned} \tag{16}$$

(eq. 4 in [51], eq. 12 in [7]).

Eq. (16) reveals that changes in the within-sex genetic variances, $\sigma^2_\mathrm{f}$ and $\sigma^2_\mathrm{m}$, are determined by three effects: (i) directional selection, through the terms $-(\Delta\overline{z}_\mathrm{f})^2$ and $-(\Delta\overline{z}_\mathrm{m})^2$, which depletes variance; (ii) direct quadratic selection within a sex, captured by $H_\mathrm{f}(\overline{z}_\mathrm{f})$ and $H_\mathrm{m}(\overline{z}_\mathrm{m})$, which increases variance under disruptive selection ($H_u > 0$) and diminishes it under stabilising selection ($H_u < 0$); and (iii) indirect quadratic selection acting through the other sex, captured by $\sigma^2_\mathrm{fm} H_\mathrm{m}(\overline{z}_\mathrm{m})$ and $\sigma^2_\mathrm{fm} H_\mathrm{f}(\overline{z}_\mathrm{f})$, which can reinforce or counteract the direct within-sex effect (note that when $\sigma_\mathrm{fm} = \sigma^2_\mathrm{f} = \sigma^2_\mathrm{m}$ and $\overline{z}_\mathrm{f} = \overline{z}_\mathrm{m} = z^*$ are such that $\Delta\overline{z}_\mathrm{f} = \Delta\overline{z}_\mathrm{f} = 0$, the condition for selection to favour an increase in genetic variance according to eq. (16) reduces to $H(z^*) > 0$, in line with adaptive dynamics models). Eq. (16) further shows that directional and quadratic selection also act to change the between-sex genetic covariance $\sigma_\mathrm{fm}$.

Directional selection (the $\Delta\overline{z}_{\mathrm{f}}\Delta\overline{z}_{\mathrm{m}}$ term) decreases the covariance when selection is antagonistic [7]. Quadratic selection, via $\sigma_{\mathrm{fm}}\left(\sigma_{\mathrm{f}}^2 H_{\mathrm{f}}(\overline{z}_{\mathrm{f}}) + \sigma_{\mathrm{m}}^2 H_{\mathrm{m}}(\overline{z}_{\mathrm{m}})\right)$, also reduces covariance under stabilising selection but increases it under disruptive selection [51].

These within-generation changes due to selection are, however, largely undone by segregation and recombination, so that the genetic variances and covariances remain roughly constant in the short term [94, 96]. Longer-term dynamics do depend on how allele frequencies and disequilibria are reshaped by selection, mutation, drift, segregation and recombination, so that their behaviour generally depends on genetic architecture itself [45, 94, 95, 97, 98].

### 2.5 The concordance among all three approaches

Each of the three approaches makes different assumptions and is based on different quantities: allele-frequency change, $\Delta p$ (eqs. 4 and 6); invasion fitness, $W(z_\bullet, z)$ (eq. 9); and changes in the moments of allelic effects, e.g., trait means $\Delta\overline{z}_{\mathrm{f}}$ and $\Delta\overline{z}_{\mathrm{m}}$ (eq. 15). They have also been used to tackle different questions: population genetics and adaptive dynamics have considered the short- and long-term maintenance of genetic variation, while quantitative genetic studies have focused on constraints on sex-specific adaptation and the sex load. Nevertheless, these frameworks agree whenever they address the same biological question under comparable assumptions (e.g., allowing multiple alleles); in that sense, they give concordant descriptions of sex-specific adaptation. In particular, they agree that sexually antagonistic selection depletes genetic variation when it generates directional or stabilising selection on traits but can maintain genetic variation under disruptive selection (Appendix C.1 for details).

This concordance between approaches means that for many questions, the choice of framework is largely one of preference. However, some biological scenarios lend themselves more naturally to a particular framework. Adaptive dynamics is typically the most direct route when the aim is to characterise the long-term outcome of continuous trait evolution, including the conditions for disruptive selection. Population genetic models are better suited when evolution involves large and/or strongly non-additive sex-specific effects on trait value (eqs. (1) and (4) and Appendix A). Both situations can in principle be formalised with the other approaches but the underlying assumptions may become restrictive: in adaptive dynamics, large-effect alleles need not satisfy the invasion-implies-substitution principle, and in quantitative genetics, breeding values need not remain approximately normally distributed.

## 2.6 What about complex traits?

One specific case that is difficult to analyse within any of the approaches, but is of special biological relevance [91, 99, 100], arises when continuous traits have a complex basis, i.e., are controlled by a large but finite number of loci with arbitrary phenotypic effects and linkage. In this setting, it is not immediately clear whether predictions from adaptive dynamics or quantitative genetics should continue to hold because their usual assumptions are violated. Multilocus population genetic models that explicitly track dynamics across loci without assuming weak selection are in principle possible, but become prohibitively complicated once more than a handful of loci are considered ([39–41, 101] for two-locus models that assume independent and fixed fitness effects, which could for example arise if each locus underlies a different monogenic trait).

To bridge this gap, computer simulations have increasingly been used. In simulations of complex traits with sex-specific optima and additive genotype–phenotype maps, the evolutionary trajectories of male and female trait values and the direction of changes in genetic variation are remarkably well-predicted by adaptive dynamics and quantitative genetic models. This close match has been observed across simulations considering a wide range of genetic architectures, including different numbers of loci, patterns of linkage, and skew in the distributions of allelic effects amongst loci [12, 18, 22, 102] (see also [45] and [103–105] for examples outside of sexual antagonism; and also [102, 106] for a summary of quantitative discrepancies between simulations and theory).

Comparatively less is known about the evolutionary dynamics of complex traits that may show sex-specific phenotypic dominance (although see [22] for example study, and also [107] for equivalent analyses with seasonally fluctuating selection). In Fig. 5 and Appendix C.2 we present simulation results for traits encoded by multiple loci at which new alleles may differ in their homozygous and heterozygous contributions to phenotype in each sex (i.e., sex-specific allelic and dominance effects, respectively). These simulations show that when constraints on sex-specific allelic effects are weak or moderate, such that mutations may readily exhibit different homozygous phenotypic contributions in males and females, the consequences of sex-specific dominance are largely imperceptible. Here, the evolution of sexual dimorphism proceeds in a similar fashion to simulations with additive gene action, and is associated with low levels of genetic variation (Fig. 5 and Supp. Fig. S1).

When constraints on sex-specific allelic effects are strong, meanwhile, divergence of male and female trait values is slower and can be associated with a transient period of inflated levels of genetic variation, especially if constraints on sex-specific dominance are much weaker than those on allelic value. This elevated variation is produced by the appearance of adaptive dominance-reversal for phenotype

at individual loci, leading to balanced polymorphisms (see Appendix C.2 for details). These polymorphisms also generate sexual dimorphism as heterozygote loci make sex-specific contributions to trait value [22]. These simulations are thus in agreement with adaptive dynamics (section 2.3.3 and Appendix B) and single locus population genetic models (Appendix A.1.1) that sex-specific dominance may offer a source of elevated genetic variation, but only when tight constraints exist on the potential for alleles with sex-specific effects.

The results reviewed in this section concern a baseline setting with autosomal inheritance, random mating, large populations, and a single evolving trait. In the next section, we review work that departs from this baseline by incorporating additional biological features, including finite population size, non-random mating, demographic structure, multiple traits, sex-biased inheritance, and genetic modifiers, and consider how these features alter evolutionary outcomes in the presence of sexually antagonistic selection.

# 3 Extensions

## 3.1 Models with genetic drift

In small populations, allele frequencies experience random fluctuations due to sampling effects, and several studies have examined how this stochasticity interacts with genetic variation expected under sexual antagonism [17, 42–44, 65] (see also [108] for a quantitative genetics approach to stochastic sex-specific evolution). Assuming weak selection and a fixed number $N_\mathrm{f}$ of females and $N_\mathrm{m}$ of males, this is done by approximating the Wright–Fisher process by a diffusion for average allele frequency $p$, whose expected change is given by eq. (4) and whose variance in change is

$$\mathrm{Var}[\Delta p] = \frac{p(1-p)}{2N_\mathrm{e}}, \tag{17}$$

where

$$N_\mathrm{e} = \frac{4N_\mathrm{f}N_\mathrm{m}}{N_\mathrm{f}+N_\mathrm{m}} \tag{18}$$

is the effective population size at an autosomal locus (assuming random mating, p. 124 in [109]). Standard diffusion methods then allow computation of both the probability and the expected time to fixation (or loss) of a mutation under sexually antagonistic selection [42]. With recurrent mutation between alleles, the diffusion approximation also allows for the calculation of the stationary distribution $\hat{\phi}(p)$ of allele frequencies across replicate realisations (or across loci with identical parameters) [43].

This distribution characterises mutation–selection–drift balance and can be used to compute the expected heterozygosity, $\int_0^1 2p(1-p)\hat{\phi}(p)\mathrm{d}p$, which quantifies the amount of standing genetic variation expected at a sexually antagonistic locus.

These diffusion analyses have generated three particularly useful insights. First, in the weak-selection regime assumed by the diffusion approximation with two trait-alleles, balancing selection under sexual antagonism can only arise through dominance reversal (section 2.2.2). This generates a sex-averaged heterozygote advantage but the resulting balancing selection is typically weaker than classical overdominance [17]. As a consequence, substantial increases in heterozygosity relative to neutrality require large $N_\mathrm{e}$ and/or high recurrent mutational input [17, 43].

Second, even when sexual antagonism generates balancing selection, it does not necessarily extend the time alleles spend segregating compared to neutrality. Prolonged segregation occurs mainly when balancing selection favours an intermediate equilibrium allele frequency $p^*$ sufficiently far from 0 and 1 (roughly $0.2 < p^* < 0.8$), which in turn requires that selection in females and males is of broadly comparable strength [42]. When this condition is not met and the equilibrium $p^*$ lies close to 0 or 1, drift can rapidly drive loss or fixation, so that sexually antagonistic alleles can be lost faster than neutral ones [42]. For the same reason, sexually antagonistic selection does not necessarily maintain higher heterozygosity than at neutral loci: when antagonism favours allele frequencies close to 0 or 1, alleles spend little time segregating and standing variation is on average low, with heterozygosity largely determined by recurrent mutation rather than by balancing selection [43].

Third, when sexual antagonism does not generate balancing selection but instead favours the fixation of one allele or the other, opposing selection in females and males can, all else equal, slow allele-frequency change relative to sex-concordant directional selection (i.e., when one allele is favoured in both sexes), thereby prolonging segregation and increasing standing variation relative to sex-concordant loci (though not relative to neutrality) [17, 43].

An additional consequence of finite population size arises because offspring number is finite and thus subject to stochastic variation. Selection then acts not only to increase mean offspring number but also to reduce an individual's variance in offspring number and its covariance with the offspring number of other individuals [110, 111]. These effects are likely to be relevant for sex-specific evolution because males and females can differ widely in reproductive variance depending on the mating system [112–114]. However, they remain little explored in models of sexual antagonism, with few studies explicitly incorporating sex-specific reproductive variance [115]. Because selection on reproductive variance is inversely proportional to population size, its effects are expected to be weak unless the population is subdivided into local groups [116–118]. Extending theory to incorporate sex-specific variance in

interconnected mating groups would therefore be of interest.

### 3.2 Non-random mating and interactions among relatives

Several studies have relaxed the common assumption in models of sexual antagonism that mating and interactions are random, with some models further considering selfing in hermaphrodites where sexual antagonism can arise between the male and female functions of an individual [65]. Two effects stand out from this work. The first concerns non-random mating, which changes the genotype frequencies on which sexually antagonistic selection acts. When non-random mating increases inbreeding, e.g., through self-fertilisation [65, 119, 120], assortative mating by genotype [121], or limited dispersal [44], it increases homozygosity and reduces the frequency of heterozygotes. This generally disfavours polymorphism, because balancing selection driven by dominance reversal requires heterozygotes to be sufficiently common ([44, 65, 120, 121], see [122] for more general effects of selfing on overdominance). In finite populations, inbreeding further reduces the efficacy of sexually antagonistic balancing selection relative to drift [44, 65], especially under partial selfing which simultaneously decreases $N_e$ [65, 122]. Selfing in hermaphrodites can also reduce sexually antagonistic polymorphism by reducing the fraction of offspring exposed to antagonistic selection (because selfing aligns male and female fitness functions, leaving sexual antagonism only among outcrossed individuals [119]). By contrast, assortative mating by fitness can have the opposite effect: under sexual antagonism, high-fitness males and high-fitness females tend to carry different genotypes at the antagonistic locus, so fitness-based assortment leads to disassortative mating by genotype, increasing heterozygosity [121, 123].

The second class of effects concerns non-random interactions among relatives, independent of mating. By increasing relatedness among interacting individuals, limited dispersal generates kin competition, whereby an individual's reproductive success tends to come at the expense of genetic relatives [68]. This weakens the effective strength of selection on competitive traits. When dispersal or demography (i.e., the number of males and females competing within groups) differs between the sexes, sexually antagonistic selection is biased towards alleles that benefit the sex experiencing weaker kin competition, typically the more dispersive or more numerous sex [44].

When selection is weak, the effects of genetic structure within individuals (i.e., excess homozygosity) [71] and between individuals (i.e., relatedness) [44] can be studied by expressing allele-frequency change in an inclusive-fitness form [68]. This perspective is useful for understanding how genetic transmission, relatedness, and the scale of competition jointly determine how antagonistic allelic effects expressed in males and females translate into evolutionary change [71]. For instance, it can help

explain how asymmetric transmission and inbreeding in haplodiploids generally promote female-beneficial alleles under arrhenotoky and paternal genome elimination, while sex-specific scales of competition can reinforce or counteract this bias [124] (see [125] for a complementary dissection of the genetic asymmetries underlying sexual antagonism in haplodiploids).

### 3.3 Demographic effects and feedbacks

When sexual antagonism arises in traits that affect vital rates, such as survival, maturation, or fecundity, it may influence population size and population structure, e.g., the age distribution or adult sex ratio [8, 14, 28, 126]. Sexual antagonism can thus have demographic consequences. The approaches of section 2 readily incorporate such effects if sex-specific selection is independent of population size and structure, so that demography depends on the evolving trait distribution but selection does not depend on demography (e.g., chapter 1 in [127]). In this case, evolutionary change is formalised by eq. (1), (8) or (15), and the demographic fallout of sexual antagonism can be characterised by evaluating the population size and structure at the resulting evolutionary outcome.

Kokko and Brooks [29] used this logic to examine how a sexually antagonistic locus affects population size. They showed that, under hard selection–that is, when sex-specific differences in survival or fecundity translate directly into differences in recruitment and population growth [128], and when female fitness is demographically limiting [129], sexually antagonistic selection can reduce population size whenever it lowers female survival or fecundity. This occurs most readily when selection acting through both sexes is strong, so that the shared trait is displaced away from the female optimum, leading to a significant reduction in female fitness. In extreme cases, high female costs combined with strong selection in males can even lead to the fixation of alleles that drive population extinction [28–30].

Evolutionarily driven demographic change may also feed back on selection [130]. Such feedbacks will be relevant to sexual antagonism when evolutionary change alters the population in a way that modifies the strength or direction of selection acting in females versus males, e.g., through sex-specific density dependence or changes in age-specific sex ratio [131–133]. Few models have examined such feedbacks explicitly for sexually antagonistic loci, nevertheless, they are in principle straightforward to incorporate using a separation of timescales between evolutionary and ecological changes assuming weak selection, allowing demography to be treated at an equilibrium induced by the resident trait value [79, 82, 89, 134]. This equilibrium can then be substituted into the allele frequency dynamics (eq. (1)) or the fitness function (eq. (8)) before analysing selection in the usual way (although charac-

terising the demographic equilibrium may itself be non-trivial). Such a strategy has been used more extensively in models of sexual selection and sexual conflict in which the fitness effects of expressing traits with demographic impacts are dependent on population state, for instance when male harm, mate limitation, or mating rates depend on population density or sex ratio [46, 135–138]. A separation of timescale can also be used in spatially structured populations, where demography varies across space: spatial demography, through its effects on migration, can alter where sexual antagonism occurs across a species' range, for instance concentrating antagonism near the range center [139]. By altering the absolute fitness of males and females, sexual antagonism could also modify metapopulation dynamics, generating feedback between local adaptation, migration, and demography and potentially producing sex-specific sources and sinks.

When selection is strong, allele-frequency change and demography can occur on comparable timescales. In this regime, if alleles have a large impact on demography while simultaneously showing fitness effects that are highly sensitive to population structure, then genetic and demographic dynamics must be tracked jointly, as in stage-structured eco-evolutionary models of sexually antagonistic loci [27, 30]. This is because demographic feedbacks could conceivably alter evolutionary outcomes, e.g., an initially net-beneficial allele may become net-deleterious as it increases in frequency, if it sufficiently affects population size or structure in a manner that alters the outcome of opposing selection between the sexes. Such interactions may provide another route towards balancing selection. Eco-evolutionary dynamics here could be complex, depending on population structure and the demographic components under selection, and likely demand numerical analyses.

### 3.4 Multiple traits

Selection in males and females typically acts simultaneously on several phenotypes [1, 3, 8, 9, 140], so sexual antagonism need not be confined to a single trait. When multiple traits share a genetic basis, the sexually antagonistic effect of an allele may arise through pleiotropic effects expressed in each sex. For all three modelling approaches reviewed in section 2, this can be formalised by writing sex-specific fitness as $w_{\mathrm{f}}(\boldsymbol{z})$ and $w_{\mathrm{m}}(\boldsymbol{z})$ where $\boldsymbol{z} = (z_1, \ldots, z_n)$ is the vector of $n$ trait values expressed by an individual.

Multivariate responses to selection are particularly well developed in quantitative genetics, which emphasises that responses to sex-specific selection depend on the structure of additive genetic variances and covariances within and between sexes ([141, 142], see chap. 13 in [91]; [50] for review). This is captured by the multivariate equivalent of eq. (15), which gives the joint change in female and male mean

trait vectors $\overline{\boldsymbol{z}}_{\mathrm{f}}$ and $\overline{\boldsymbol{z}}_{\mathrm{m}}$ :

$$\begin{pmatrix} \Delta\overline{\boldsymbol{z}}_{\mathrm{f}} \\ \Delta\overline{\boldsymbol{z}}_{\mathrm{m}} \end{pmatrix} = \frac{1}{2}\begin{pmatrix} \mathbf{G}_{\mathrm{f}} & \mathbf{B} \\ \mathbf{B} & \mathbf{G}_{\mathrm{m}} \end{pmatrix}\begin{pmatrix} \boldsymbol{S}_{\mathrm{f}}(\overline{\boldsymbol{z}}_{\mathrm{f}}) \\ \boldsymbol{S}_{\mathrm{m}}(\overline{\boldsymbol{z}}_{\mathrm{m}}) \end{pmatrix}, \tag{19}$$

where $\mathbf{G}_{\mathrm{f}}$ and $\mathbf{G}_{\mathrm{m}}$ are $n \times n$ matrices collecting the additive genetic variances for each trait and covariances among traits in females and males, respectively; the $n \times n$-matrix $\mathbf{B}$ collects the additive genetic covariances between female and male traits (i.e., its $ij$-entry is the covariance between trait $i$ in females and trait $j$ in males); and $\boldsymbol{S}_{\mathrm{f}}(\overline{\boldsymbol{z}}_{\mathrm{f}})$ and $\boldsymbol{S}_{\mathrm{m}}(\overline{\boldsymbol{z}}_{\mathrm{m}})$ are vectors of length $n$ whose $i$-th entry give the selection gradient acting on trait $i$ in females and males (eq. (19) is equivalent to eq. 1 in Lande [4]). Eq. (19) shows that the trajectory of sexual dimorphism need not align with the direction of sex-specific selection, with dimorphism potentially arising as an indirect response mediated by genetic covariances among traits and between sexes [52]. In other words, while positive cross-sex covariance for the same trait impedes sex-specific adaptation in that trait (when $\sigma_{\mathrm{fm}} > 0$ in eq. (15)), cross-sex covariances between different traits (the off-diagonal entries of $\mathbf{B}$) can instead facilitate divergence as selection on one trait in one sex generates correlated responses in other traits in the other sex (e.g., eq. 10 in [50]; see also [52]).

When fitness depends on multiple traits and their interaction, selection acts not only on each trait but also on combinations of traits. This can be captured by the cross-derivatives $\partial^2 w_u/\partial z_i \partial z_j$ in sex $u$, which measure correlational selection, that is, whether selection favours positive ($\partial^2 w_u/\partial z_i \partial z_j > 0$) or negative ($\partial^2 w_u/\partial z_i \partial z_j < 0$) associations among traits $i$ and $j$ within individuals of sex $u$ [73]. Barker et al. [51] and McGlothlin et al. [7] provide multivariate equivalents of eq. (16) for within-generation changes in additive genetic (co)variances that depend on correlational selection. However, because these results concern only within-generation changes due to selection, population genetic and adaptive dynamics are more informative about the long-term effect of selection on genetic variation, and remarkably few such models have been formalised specifically for multiple traits under sexually antagonistic selection [28].

From $w_{\mathrm{f}}(\boldsymbol{z})$ and $w_{\mathrm{m}}(\boldsymbol{z})$, one can readily derive the sex-specific selection and dominance coefficients ($s_{\mathrm{f}}$, $s_{\mathrm{m}}$, $h_{\mathrm{f}}$ and $h_{\mathrm{m}}$) acting on two specific alleles with pleiotropic effects on multiple traits, which then determine allele-frequency change at one locus (eqs. (1) and (4); Appendix D). The effect of having multiple traits depends both on the genotype-phenotype map and on how the resulting trait combinations affect fitness. In particular, if two specific alleles at one locus affect two traits in the same direction and correlational selection is negative (i.e., $\partial^2 w_u/\partial z_1 \partial z_2 < 0$ in sex $u$), deleterious alleles become more recessive for fitness (Appendix D.2.1). This is because the joint fitness cost of changing both traits increases more than additively with allelic effect, so that the deleterious homozygote pays

a disproportionately larger cost than the heterozygote. If this occurs in both sexes it promotes dominance reversal for fitness and can thereby increase the scope for maintaining sexually antagonistic polymorphism in models with a limited set of alleles. By contrast, when different traits are encoded by different sexually antagonistic loci, the same negative correlational selection can generate negative epistasis between loci (Appendix D.2.2), thereby narrowing the parameter range over which multilocus polymorphism is maintained [41].

In adaptive dynamics, when selection acts on multiple traits and mutations do not affect all traits in a perfectly correlated way, evolution proceeds under directional selection until the population reaches a combination of trait values at which the selection gradient on each trait is zero (eq. (10) defined for each trait [143, 144]; Appendix D). Correlational selection therefore does not alter the singular point but it can affect evolution once the population is close to that point. In particular, it can generate disruptive selection even when selection on each trait is stabilising in isolation [144, 145]. This occurs when selection on trait combinations is sufficiently strong to favour divergence along particular directions in trait space. But for this to result in evolutionary branching and the maintenance of genetic variation, the underlying genetic architecture must allow selection to act on such trait combinations. This requires either pleiotropic loci or strong linkage disequilibrium arising from very low recombination rates (e.g. [37]).

### 3.5 Sex linkage and sex-biased inheritance

Some loci are associated with sex and therefore experience sex-biased inheritance. In species with genetic sex determination, this mainly concerns regions of the sex chromosomes (which may be in strong or weak linkage with the sex-determining region), but can also include uniparentally inherited elements such as mitochondrial genomes. Sex-biased inheritance has important consequences for evolution under sexually antagonistic selection at the affected loci. These consequences have been analysed using population genetic, adaptive dynamics, and quantitative genetic models, focusing on genomic regions that differ in their degree of association with sex. Here, we review the results obtained by these different modelling approaches for different forms of sex-biased inheritance. Throughout, we assume male heterogamety (XY sex determination) for clarity, but similar results apply to female heterogamety (ZW) systems by interchanging the sexes and sex chromosomes (m $\leftrightarrow$ f, X$\leftrightarrow$Z, Y$\leftrightarrow$W).

### 3.5.1 X-linkage

The most frequently considered case is a locus in the non-recombining region of the X chromosome, with no functional homologue on the Y. Here, males are hemizygous so allelic effects on the X are expressed directly in males. A central question has been whether complete X-linkage facilitates the maintenance of sexually antagonistic polymorphism relative to autosomal inheritance. Population genetic models show that a protected polymorphism between two alleles is maintained when

$$\frac{1-h_{\mathrm{f}}s_{\mathrm{f}}}{2(1-h_{\mathrm{f}})} < \frac{s_{\mathrm{f}}}{s_{\mathrm{m}}} < \frac{1+h_{\mathrm{f}}s_{\mathrm{f}}}{2h_{\mathrm{f}}} \tag{20}$$

[16, 19, 146] (using Kidwell's notation [15]; e.g. using eq. (2) above and assuming complete dosage compensation in males such that carrying a single copy of allele $\mathrm{A}_i$ leads to expression of trait value $z_{\mathrm{m},ii}$). Comparing eq. (20) with the autosomal condition eq. (3) under sex-concordant dominance ($h_{\mathrm{f}} = 1 - h_{\mathrm{m}}$) led Rice [19] to argue that the X should be especially favourable to sexually antagonistic polymorphism. However, Fry [16] pointed out that this conclusion depends on dominance being sex-concordant, and that autosomes can be more permissive when they show adaptive dominance reversal; specifically when $h_{\mathrm{m}} < 1/(2 - s_{\mathrm{m}})$ [16, 147]. Thus, complete X-linkage is not intrinsically more conducive to polymorphism; its effect depends on dominance and the strength of selection in the two sexes. A similar conclusion applies to fixation: whether male- or female-beneficial alleles are more prone to fix on the X compared to autosomes depends on prior assumptions regarding dominance and selection coefficients [148]. Two-allele population genetic models therefore do not predict a general enrichment of sexually antagonistic polymorphism, or a male- or female-beneficial bias, on the X.

Predictions concerning polymorphism at X-linked loci are clearer when considering long-term evolution with recurrent mutation: adaptive dynamics models show that evolutionary branching, and hence polymorphism, is typically more restrictive under complete X-linkage than under autosomal inheritance (see Appendix D of [18]). This reflects the fact that X chromosomes do not remain associated with the sex in which alleles are beneficial, i.e., X chromosomes carrying male-beneficial alleles in fathers are transmitted to daughters, while sons inherit X chromosomes from mothers. This breaks the association between allelic effects and the sex in which they are favoured, limiting the scope for branching.

Quantitative genetic models have also addressed X-linked evolution. When the loci underlying a polygenic trait are X-linked, the equations for the change in mean male and female trait values retain the same structure as in the autosomal case (eq. 15), but with different weights on the genetic vari-

ance–covariance terms because of asymmetric transmission (eq. A7 in [4]). This does not change the evolutionary equilibrium: if sex-specific genetic variation is available, the population evolves toward male and female optima where sex-specific selection gradients vanish. However, the trajectory can differ from the autosomal case because X-linkage changes the partitioning of additive genetic variance in the two sexes [4, 12, 149, 150]. Without dosage compensation in males, X-linked variance is larger in females, so adaptation in females can proceed more quickly than in males. With complete dosage compensation, this bias is reduced, making the response more similar to the autosomal response.

### 3.5.2 Pseudo-autosomal region of sex-chromosomes

A different situation arises in the recombining or "pseudoautosomal" region (PAR) of sex-chromosomes, where linkage between loci and the sex-determining region is incomplete. Population genetic models show that PAR loci tend to maintain protected polymorphism under broader conditions than autosomal loci because linkage to the sex-determining region allows associations to build between alleles and the sex in which they are favoured [25, 151–153]. These associations broaden the conditions for polymorphism, but only when recombination is sufficiently weak relative to selection [25, 153] (see exact conditions in Appendix E). In the limit of tight linkage, a male-beneficial allele can invade whenever it increases male fitness because it becomes associated with Y-bearing haplotypes and is transmitted disproportionately from fathers to sons. Alleles beneficial in females also gain from association with X-bearing haplotypes, but less completely because the X passes through both sexes, so invasion still requires that selection in females outweighs costs in males (see eq. (E-10) in Appendix E). More generally, strong selection combined with tight linkage is expected to generate substantial allele-frequency differences between X- and Y-linked copies [26, 60, 152]. Where loci are fully linked to the sex-determining region, stable polymorphism can arise with the male-beneficial allele fixed on the Y and the female-beneficial allele fixed or polymorphic on the X, depending on the relative strength of selection [59].

Adaptive dynamics models extend these results to long-term evolution with recurrent mutation. Evolutionary branching is more readily obtained at PAR loci than at autosomal loci when recombination with the sex-determining region is sufficiently rare relative to the strength of sex-specific selection [18]. The reason is the same as in two-allele models: linkage to the SDR allows alleles with male-beneficial effects to remain associated with Y-bearing haplotypes and alleles with female-beneficial effects to remain associated with X-bearing haplotypes. The PAR is therefore predicted to be especially conducive to the gradual emergence and maintenance of sexually antagonistic polymorphism, provided recombination is low enough for these associations to persist.

One might think that the occurrence of one polymorphism in or near the non-recombining region of the sex chromosome might facilitate the maintenance of polymorphism at loci further along the sex chromosome, causing differentiation to snowball down the chromosome. Models show that this effect is modest, at best [101]. Under weak selection, polymorphism at one locus does not affect the conditions for maintaining polymorphism at another non-epistatic locus, regardless of their positions. Invasion conditions broaden only slightly when selection is strong and the loci are tightly linked to each other but at intermediate distance to the sex-determining region.

### 3.5.3 Regions outside of sex-chromosomes and processes other than selection

Other genomic regions than sex chromosomes can show sex-biased inheritance and thus be disproportionately affected by sexual antagonism. The clearest case is the mitochondrial genome, which is usually maternally inherited. Under strict maternal inheritance, selection on mitochondrial variants is determined by their effects on female fitness, so mutations that are beneficial in females spread regardless of their effects on male fitness [154]. Population genetic models show that this "mother's curse" effect can be weakened when male fitness contributes to the transmission success of mitochondrial lineages, for example through inbreeding or kin selection, which link male performance to the reproductive output of females carrying the same cytotype, or through paternal leakage, which allows mitochondrial variants to be transmitted partly through males [154–156]. Models of cyto-nuclear co-evolution further show that nuclear modifiers can compensate for male-harming mitochondrial mutations, generating a transient male load before compensation evolves [157].

The conflict between the transmission interests of cytoplasmic and nuclear genes is particularly acute in hermaphrodites, which can act as both maternal and paternal parents. In these species, selection favours cytotypes causing male sterility whenever they increase the number of offspring produced through female function. The spread of such cytotypes strongly favours nuclear alleles restoring male fertility, leading to an evolutionary arms race between cytoplasmic and nuclear genomes (e.g., [158–162]).

Models of mitochondrial inheritance and mito-nuclear linkage also show that the mode of inheritance itself can evolve under sexually antagonistic selection. Males may favour paternal leakage or recombination that improves mito-nuclear matching in males, whereas females often favour stricter maternal inheritance when this preserves female-beneficial associations between mitochondrial and nuclear genotypes [163, 164].

Finally, sex linkage can influence evolution under sexual antagonism via other evolutionary processes

than selection. First, sex-linked regions can differ in mutational input when male and female germline mutation rates differ. For example, under male-biased mutation, the X receives fewer new mutations per chromosome than autosomes, which can affect heterozygosity at sexually antagonistic loci at mutation-selection–drift balance [43]. Second, genetic drift can have a disproportionate impact on the persistence and fixation of sex-linked mutations because sex-linked regions often have lower effective population sizes than autosomes [42, 43]. For example, in randomly mating populations with $N_\mathrm{m}$ breeding males and $N_\mathrm{f}$ breeding females, $N_\mathrm{e}^\mathrm{Y} = N_\mathrm{m}/2$ and $N_\mathrm{e}^\mathrm{X} = 9N_\mathrm{m}N_\mathrm{f}/(4N_\mathrm{m} + 2N_\mathrm{f})$, to be compared with the autosomal case shown in eq. (18) [165, 166]. These differences are modulated by sex-specific reproductive variance, e.g. higher male reproductive variance, as in polygynous mating systems, reduces $N_\mathrm{e}^\mathrm{Y}$ more strongly than $N_\mathrm{e}^\mathrm{X}$ relative to autosomes [167]. When both drift and selection are accounted for, substitution rates for sexually antagonistic alleles can be higher or lower on the X than on autosomes depending on dominance: X-linked substitution is favoured for recessive male-beneficial alleles and dominant female-beneficial alleles [168].

## 3.6 Evolution of genetic modifiers

A modifier is a locus at which alleles alter how genetic information is expressed, transmitted, or reorganised across generations, e.g., by modifying mutation rate, recombination, dominance, or expression [169]. A modifier may or may not have a direct fitness effect; being selected only through genetic associations with selected "target" loci in the latter case (e.g., [170, 171]). In the context of sexual antagonism, modifier models typically consider one or more target loci under sexually antagonistic selection and another *modifying* locus for which alleles change some property of those target loci.

We distinguish two main classes of modifiers (Table 1 for list). The first alters the genotype–fitness map, changing how sexually antagonistic variation is expressed at target loci. The second alters genetic transmission, for example through recombination or sex linkage, and evolves through associations with sexually antagonistic alleles. Below, we highlight how modifier evolution affects the maintenance of polymorphism, both in the short- and long-term, and the magnitude of the sex load (Table 1 for summary).

### 3.6.1 Modification of the genotype-fitness map

**Dominance modification.** Dominance was one of the earliest features considered in modifier theory, although initially not in the context of sexual antagonism [172]. Such modifiers are impactful only when there is appreciable standing variation at the target locus or loci, because dominance influences

heterozygote (but not homozygote) trait values [173]. When sexual antagonism maintains polymorphism at the target locus, Spencer and Priest [61] showed that selection favours modifiers that increase the dominance of the allele beneficial in each sex thus producing adaptive dominance-reversal. This broadens the conditions for maintaining a protected polymorphism (in the sense of eq. 3) and reduces the sex load. It does not remove the load completely though, because homozygotes carrying the least fit allele in each sex can still be produced. Spencer and Priest [61]'s model is formulated directly in terms of genotype–fitness relationships (i.e., on $h_{\mathrm{f}}$ and $h_{\mathrm{m}}$ in eq. 2) and is thus largely agnostic to the biological basis of dominance modification.

Several models have focused on specific mechanisms that change dominance by modifying the genotype-phenotype map. One route is sex-specific allelic expression in heterozygotes, whereby modifiers control the relative contribution of target alleles to phenotype in a sex-specific manner ([22, 47, 70]; e.g. see eq. (C-1) in Appendix here). In this case, the allele coding for the adaptive phenotype in homozygotes of a given sex evolves to contribute more strongly to the trait in heterozygotes of that sex. This produces better adapted heterozygotes with sexually dimorphic phenotypes. Such dominance evolution can facilitate the long-term maintenance of polymorphism (Appendix B), as well as the maintenance of polygenic variation [22], provided that homozygous allelic effects homozygote phenotypes are constrained to remain similar in males and females.

Another route that can lead to sex-specific heterozygote phenotypes is genetic imprinting, which evolves when it increases the expression of alleles inherited from the parent of the same sex [174]. This is because these alleles are (after selection) more likely to be beneficial in that sex. This effect, however, is expected to be weak because it relies on allele frequency differences between male and female parents, which are typically very small for autosomal loci unless selection at the focal locus is extremely strong [121].

Finally, dominance may also evolve as a byproduct of selection for robustness. If selection favours reduced sensitivity to molecular or environmental perturbations, the genotype–fitness map can become buffered, for example through redundant pathways or improved physiological homeostasis [175, 176]. Such buffering can reduce the phenotypic effect of carrying a single perturbed allele, and thereby generate dominance. To our knowledge, this mechanism has not been modelled for traits experiencing sex-specific selection, leaving its consequences for the maintenance of sexually antagonistic variation and sexual dimorphism unclear.

**Expression modification.** Another class of modifiers alters the genotype–phenotype map by changing the overall expression of a target locus in a sex-specific, and sometimes allele-specific, manner.

Unlike dominance modifiers, these effects apply in heterozygotes and homozygotes alike. The general logic, anticipated verbally by Fisher [177], is that sex-limited or sex-biased expression allows male and female phenotypes to diverge toward their respective optima. Rhen [21] formalised this idea by analysing selection among three alleles at a single autosomal locus: one causing expression only in males, one only in females, and one in both sexes. Under sexually antagonistic selection, sex-limited alleles are favoured because they retain the benefit of expression in the sex in which they are advantageous while avoiding the cost in the other sex. In this model, sex-specific expression is encoded directly by alleles at the focal locus, rather than by a separate modifier acting on a target locus.

More explicit modifier models separate these components by allowing regulatory alleles to alter the expression of linked target loci. Such modifiers are favoured when they alter expression in a way that moves the phenotype of one of both sexes closer to their optimum. For example, an expression modifier can change the contribution of a target locus (which may be polymorphic, but need not be) to the trait in a sex-specific manner, increasing it in one sex while reducing it in the other [22].

Because expression modifiers act in both heterozygotes and homozygotes, they can in principle allow both sexes to reach their optima while the population is homozygous across target loci, which fully alleviates the sex load. As a result, these modifiers tend to eliminate sexually antagonistic polymorphism, even when such polymorphism was previously maintained by dominance reversal [22] (see also Appendix C.2 here).

**Gene-copy number.** A related route is to change copy number rather than the expression of a single copy. Duplication of a sexually antagonistic coding sequence can, after paralogue divergence, create a form of fixed heterozygosity in which male- and female-beneficial variants are retained in different copies, but duplicate invasion is most readily favoured when antagonistic variation is already segregating, and is constrained when the duplicate is initially redundant or dosage is costly [178]. This complements models of sex-specific gene expression, where cis-regulatory changes can resolve antagonism by restricting expression to the sex or tissue in which an allele is favoured, where pleiotropy, sex linkage and recombination can make this a sequential or epistatic process [179].

### 3.6.2 Modification of genetic transmission

**Recombination modifiers.** There has been considerable interest in the evolution of recombination modifiers, which alter genetic transmission by changing how tightly sexually antagonistic alleles remain associated, either with one another or with the sex-determining region. Nei [180] provided an

early model of a recombination modifier acting between two loci with sexually antagonistic effects, which he framed as loci jointly determining sexual development. He showed that reduced recombination among these loci is favoured because recombination generates maladapted intermediate genotypes, in which alleles promoting male development are expressed in genetic backgrounds favouring female development, and vice versa.

Lenormand [24] generalised this result using separation of time scales arguments (quasi-linkage equilibrium, QLE), which assumes recombination is frequent relative to selection. In addition to multilocus systems, he considered recombination between a target locus and a sex-determining locus. In this setting, modifiers reducing recombination between the two loci are favoured whenever selection at the target locus is sexually antagonistic, because recombination breaks the positive association between alleles and the sex in which they are beneficial that is present at quasi-linkage equilibrium. This extends earlier arguments for the evolution of recombination suppression around sex-determining regions, including via structural changes such as inversions or chromosomal fusions [181]. Later models have shown that this selection can be strong, including outside of the quasi-linkage equilibrium regime [182], and especially when the target locus has reached its long-term equilibrium under sexually antagonistic selection [48]. Nevertheless, complete suppression of recombination with the sex determining region is not always favoured: recombination can be maintained when fathers benefit from passing alleles currently linked to their Y to their daughters, which can occur when there is heterozygous advantage in males [26].

When tighter linkage to the sex-determining region evolves, alleles rise to higher frequency in the sex in which they are beneficial. This broadens the conditions for maintaining polymorphism (section 3.5) and reduces the frequency of maladapted genotypes, thereby lowering the sex load. However, sex linkage alone does not eliminate this load, because alleles favoured on one chromosomal background can still be expressed in the opposite sex (e.g. X-linked male-deleterious alleles in males), unless this sex exhibits overdominance [48, 183].

**Sex ratio and sex-determination modifiers.** Selection can also favour alleles that bias transmission toward the sex in which they are beneficial. For example, sex-ratio distorters can be selected when individuals carrying male-beneficial alleles produce more sons, and those carrying female-beneficial alleles produce more daughters [33].

Several models have extended this same logic to modifiers of sex determination. Here, the modifier changes the probability that its bearer develops as male or female, and is favoured when it becomes associated with alleles beneficial in the sex it tends to produce. It has been used to consider several

types of transitions among sex-determining systems.

This idea was first developed for transitions from polygenic to monogenic sex determination. Rice [184] considered the fate of a dominant major sex-determining allele introduced in a population initially balanced by polygenic sex determination. This allele can spread when it is tightly linked to a sexually antagonistic allele beneficial in the sex it produces. Its spread biases the sex ratio, which then favours compensatory changes in the polygenic background and can ultimately replace polygenic sex determination by a single locus system.

Related models consider turnover between genetic sex-determining systems. Van Doorn [185] modelled the invasion of a new dominant masculinising mutation at an autosomal locus in a population with an ancestral sex-determining locus, and considered the influence of sexually antagonistic loci linked either to the ancestral or the new sex determiner. The new allele can invade when linkage to a sexually antagonistic allele gives it an advantage in the sex it produces. Van Doorn and Kirkpatrick [34] extended this model to transitions between XY and ZW systems, with two sex-determining loci and multiple sexually antagonistic loci linked to one or both systems.

Finally, other models looked at transitions between environmental and genetic sex determination. Building on earlier threshold models of environmental sex determination [186], Muralidhar and Veller [35] considered a system with two types of loci: threshold loci determining the probability of developing as male or female depending on the environment, and sexually antagonistic loci. A haplotype combining a sex-biasing threshold allele with a sexually antagonistic allele can invade when linkage is sufficiently tight. As it spreads, sex-ratio selection favours compensatory changes leading to the progressive build-up of a non-recombining, sex-specific haplotype that functions as a new sex-determining region, thereby converting environmental into genetic sex determination. However, when fitness varies in a sex-specific way with the environment (e.g., at some temperatures, developing as female gives higher fitness than developing as male) shifting the threshold can cause some individuals to develop as the less-fit sex. This can result in mixed sex-determining systems in which both threshold variation and major-effect alleles contribute to sex [187].

**Mating preference modifiers.** Mating preferences can modify the transmission of sexually antagonistic alleles by changing which males contribute to offspring. The idea is that there is a trait locus expressed in both sexes with sexually antagonistic fitness effects, and a preference locus expressed in females that biases mating towards males carrying one of the trait alleles. Albert and Otto [32] analysed such a model and showed that when the trait is X-linked in an XY system, preferences evolve for males carrying the female-beneficial allele, because fathers transmit their X chromosome to daughters

but not to sons. When the trait is Z-linked in a ZW system, preferences can instead evolve for males carrying the male-beneficial allele, but only when the preference locus is also Z-linked; in that case, the preference spreads through its association with fitter sons.

Albert and Otto [32] also considered the case where both the trait and preference loci are autosomal. Here, preference evolution depends more strongly on physical linkage. If the trait and preference loci are tightly linked, and heterozygotes at the sexually antagonistic locus have high sex-averaged fitness, for example under adaptive dominance reversal, any new preference allele can invade because it generates linkage disequilibrium with the trait allele it favours, causing preferred matings to produce more heterozygous offspring with high mean fitness.

Muralidhar [188] analysed a complementary case: the sexually antagonistic trait is autosomal, but the mating preference is sex-linked. The model shows that in a ZW system, a W-linked preference evolution causes females to prefer males carrying an autosomal allele that increases female viability but reduces male viability. Because the W is transmitted only through daughters, selection on the preference depends on daughter fitness and is insensitive to the fitness costs paid by sons or by the preferred males. The preference can therefore fix and, if sufficiently strong, drive the male-costly trait to high frequency even when that trait is strongly deleterious in males.

Overall, these models show that mating-preference modifier evolution need not stabilise sexually antagonistic polymorphism. By giving one trait allele a mating advantage, they can drive it towards fixation, thereby resolving the antagonism at the genetic level, while potentially maintaining or even increasing the sex load.

### 3.6.3 The importance of standing genetic variation for modifier evolution

Selection on many types of modifiers depends on standing variation at the loci being modified. For dominance, recombination, sex-ratio and mating-preference modifiers, selection arises because the modifier becomes associated with segregating sexually antagonistic alleles. These associations can reduce the sex load by increasing the expression, transmission, or production of alleles in the sex in which they are beneficial (Table 1). However, this also means that many modifier models rely on the prior existence of sexually antagonistic polymorphism.

This reliance may be restrictive when such variation can only be maintained by balancing selection as stable autosomal polymorphism is often difficult to obtain when multiple alleles or multiple loci are possible [18, 45]. The requirement is much less restrictive for recombination modifiers near sex-determining regions, where sex linkage itself broadens the conditions for polymorphism at sexually

antagonistic loci (section 3.5). In the absence of balancing selection, mutation-selection-drift balance may still generate enough transient heterozygosity for modifiers to evolve [22, 47]. However, this entails substantial mutational input, either through high mutation rates or through a highly polygenic architecture in which modifiers similarly influence many downstream sexually antagonistic target loci and/or sexually antagonistic loci are similarly and simultaneously altered by many upstream modifiers.

By contrast, this constraint does not apply in the same way to expression modifiers, which can spread as ordinary beneficial mutations when they move male and female phenotypes closer to their optima, irrespective of levels of standing variation at target loci.

# 4 Discussion

The theory reviewed above suggests that sexual antagonism can influence several major aspects of the biology of sexual species. In the following section, we synthesise the main implications of this theory for the emergence, persistence, and consequences of sexual antagonism, focusing in particular on its relationship with fitness and genetic variation, sex determination, and sexual dimorphism. In doing so, we highlight gaps in current knowledge that could be fruitful avenues for future theoretical work.

## 4.1 Sex load, fitness variation, demographic and co-evolutionary outcomes

By impeding sex-specific adaptation, sexual antagonism can reduce mean fitness below the value attainable if males and females evolved independently (Box I [8, 9, 14, 55]). This additional load (the sex load) is expected to be most pronounced when populations are already close to their sex-specific optima (i.e., are well-adapted). Here, male and female selection gradients are more likely to point in opposite directions, and positive cross-sex genetic correlations become a major constraint on adaptation [5, 13, 14, 55]. By contrast, after a substantial environmental change, males and females are likely to be displaced from their optima in the same direction. Selection is then largely concordant across the sexes, and positive cross-sex correlations are not a major source of maladaptation (Fig. 4A; [13, 14]).

This implies that the sex load should usually be transient in stable environments. If genetic variation with sex-specific effects is available, males and females are expected to evolve towards their respective optima, dissipating both antagonism and the associated load (Fig. 4B; [8, 22, 54, 55]). A persistent sex load is therefore most likely when ecological conditions change often enough that dimorphism cannot catch up, but not so strongly that male and female selection gradients become aligned (Fig. 4C

[12, 13]). This may occur under temporally or spatially varying sex-specific selection [139], as generated by abiotic environmental changes or potentially by recurrent biotic change due to co-evolutionary dynamics (e.g., between males and females via mate choice or sexual conflict, or through sex-specific interactions with parasites, predators, competitors or mutualists), although the latter remains little explored theoretically [133].

By slowing the evolution of traits towards their (male and female) optima, sexual antagonism can affect sex-specific variance in fitness and population demography. Strong cross-sex genetic constraints result in populations experiencing sex-specific selection spending more time in regions of the fitness landscape where phenotypic differences translate into large fitness differences [4, 18]. Consequently, sexual antagonism may be associated with high variance in individual fitness even when it does not maintain high levels of genetic polymorphism at underlying loci [18, 42, 43]. The demographic impacts of sexual antagonism, meanwhile, are likely to be more circumscribed. Although displacing the sexes from their optima can in principle reduce population growth by lowering sex-specific fitness components, especially in females, substantial demographic effects are expected only when the ensuing fitness costs translate directly into large effects on recruitment [14, 29, 30, 129, 189]. Conditions for this appear restrictive [14], suggesting that sexual antagonism may have evolutionary consequences without causing large changes in population size.

Because in most cases male and female trait values reach their optima eventually, sexual antagonism is generally thought to weakly impact trait values over evolutionary timescales. However, exceptions are possible in the presence of co-evolutionary dynamics, in which case sexual antagonism can in fact affect long-term trait evolution. For example, cross-sex genetic correlations in sexually selected traits can dampen escalation during co-evolutionary runaways or arms races between females and males (because changes favoured in one sex induce correlated costs in the other), and this alters the ultimate phenotypic outcomes of these dynamics [190]. With ecological traits, meanwhile, sexual antagonism can constrain the evolution of sex-specific niche use. This is because intense competition for resources can favour the sexes to diverge ecologically [191], but this divergence only happens if cross-sex correlations are weak enough; else if they are strong, ecological divergence between the sexes is impeded and instead ecological speciation may be favoured [46, 192]. These examples therefore suggest sexual antagonism could play an underappreciated role in the long-term response to selection from male-female interactions and thus in directing sex-specific ecology [193].

## 4.2 The effect of sexual antagonism on genetic polymorphism

Much of the theory on sexually antagonistic selection has been concerned with the potential to *maintain* polymorphism (e.g., [15–19, 22, 25, 36, 41–45, 47, 56, 57, 59, 60, 69, 71, 101, 119, 123, 151, 152, 183]). That is, for more genetic variation to be present under sexually antagonistic selection than under neutrality, which requires balancing selection [42, 43]. As highlighted in section 2, when examining the simplest case, namely variation at a single large-effect locus acting in isolation, this question is best separated into two. First, whether a fixed set of alleles can be maintained in the short term, and second, whether polymorphism persists over longer timescales when new alleles can arise.

Considering autosomes, population genetic models have identified two main routes for short-term polymorphism owing to sexual antagonism at such a locus: (1) strong and opposing selection in the two sexes (large $s_\mathrm{f}$ and $s_\mathrm{m}$ in eq. 3, [15]); and/or (2) dominance reversal for fitness, whereby alleles tend to be more dominant in the sex in which they are favoured (low $h_\mathrm{f}$ and $h_\mathrm{m}$ in eq. 3, [15]). This reversal may be due to sex-specific properties of either the genotype-phenotype map (dominance reversal for phenotype, Fig. 2A1) or the phenotype-fitness map ([22, 47], Fig. 2A2).

For polymorphism to be maintained over longer evolutionary timescales here, two additional requirements must be met. First, sexual antagonism must persist, which requires constraints on sex-specific gene expression. Second, the polymorphism must also be robust to invasion by alternative alleles. This makes the criteria for long-term balancing selection more restrictive: either selection on the trait is disruptive and thus disfavours intermediate trait values (eq. 14 [18]); or the genotype-phenotype map is such that dominance reversal occurs at the level of the phenotype, i.e. such that any allele that is better adapted in one sex in homozygous form has a dominant effect on trait expression in that sex [47] (section 2.3.3).

The same conditions also apply to small-effect loci underlying polygenic traits [18, 45]. Here, balancing selection across loci contributing to the trait is expected in the short- or long-term only if either the trait itself is under disruptive selection, or if individual loci show dominance reversal for phenotype, so that heterozygotes express trait values closer to the sex-specific optimum in each sex (Appendix C). If a polygenic trait is instead under stabilising selection for a compromise trait value $z^*$, dominance reversal for fitness may still arise, but only at a single locus (out of all those encoding $z$) whose homozygotes straddle $z^*$ [18, 45].

Owing to the unusual fitness landscapes required for disruptive selection in dioecious populations [18], long-term or polygenic balancing selection appears to rely primarily on dominance reversal for phenotype. Such patterns of non-additive gene action may arise following the spread of sex-specific

dominance modifiers [22, 47]. Selection on these modifiers is, however, proportional to heterozygosity at target loci [61, 172, 173]. This creates a chicken-and-egg problem: sex-specific dominance can evolve to stabilise a polymorphism only if the polymorphism is there in the first place, requiring either prior balancing selection or very high mutational input at these loci (section 3.6.3; [22, 47]). Moreover, the maintenance of dominance modifiers entails constraints on alternative routes to resolving antagonism more completely, such as sex-specific gene expression [22], which would otherwise displace polymorphism. Given these challenges, the scope for sexual antagonism to generate long-term or polygenic balancing selection likely hinges on how often new mutations are dominance-reversed when they first appear, as well on aspects of population structure and reproductive ecology that otherwise contribute to higher levels of heterozygosity [44, 119, 121, 123].

Outside of autosomes, similar outcomes are also observed for sex-linked loci in non-recombining sex chromosomes (X or Z chromosomes). Here, large effect loci can more readily exhibit balancing selection when alleles have non-additive fitness effects in the homogametic sex [16, 19]. However, long-term or polygenic balancing selection again requires disruptive selection, and in fact the conditions for this are more restrictive than for autosomal loci [18]. By contrast, sexual antagonism can more readily generate long-term or polygenic polymorphism in the recombining or pseudo-autosomal regions of sex chromosomes [25, 151, 152]. This is because sex-linkage in these regions allows antagonistic alleles to become associated with the sex they benefit, creating more permissive conditions for disruptive selection and polymorphism [18, 48].

What can we glean from all of this? Overall, while sexual antagonism is adept at producing balancing selection at large-effect loci when there are few potential alleles, theory suggests that long-term or polygenic polymorphism should be uncommon outside of certain sex-linked regions, unless traits experience unusual patterns of selection [18] – as may occur for sex allocation loci [49] (Box I) – or unless loci exhibit particular forms of sex-specific trait expression; with sex-specific effects in heterozygotes but not in homozygotes. Uncommon does not mean impossible. Several empirical examples of large-effect and/or phenotypically dominance-reversed sexually antagonistic loci are known [70, 194–197]. The theoretical expectation is simply that such cases should be exceptions rather than the general outcome of sex-specific selection on shared traits.

Even if sex-specific selection on shared traits rarely elevates standing genetic variation relative to neutrality, it may still result in more variation (although still at sub-neutral levels) than would be seen under sex-concordant selection, all else being equal [42–44] (see also [198] for an analogous result under fluctuating selection). This is because opposing selection in females and males reduces net directional selection relative to sex-concordant selection, leading to weaker erosion of variance at

mutation-selection-drift balance (section 3.1). Moreover, because sexually antagonistic traits should on average underlie greater fitness variance than sex-concordant ones for a given level of trait variation (section 4.1), antagonistic alleles that do segregate may disproportionately contribute to fitness differences. Sexual antagonism may therefore generate substantial genetic variance for fitness even while eroding genetic variation itself.

### 4.3 The evolution of sex chromosomes, sex-determining and sexual systems

Because the fitness effects of sexually antagonistic variation depend on whether it is carried by males or females, sexually antagonistic alleles have particularly strong evolutionary implications for processes that influence their likelihood of being found in the sex they are beneficial to, including the evolution of sex chromosomes and sex determination mechanisms.

One of the most well-established effects of sexual antagonism is that it readily drives recombination suppression along sex chromosomes. This is because sexually antagonistic variation is relatively easily maintained at loci in pseudo-autosomal regions of sex-chromosomes [18, 48, 59, 65, 151–153] (see section 3.5). Such polymorphism in turn favours recombination suppression between these loci and the sex-determining region [23, 24, 26, 48, 180–182, 199] (see section 3.6), which can eventually result in the evolution of non-recombining sex chromosomes [200]. After recombination arrest, however, selective interference (e.g., Muller's ratchet) can lead to the accumulation of deleterious alleles and gene loss [201, 202]. This degeneration can favour mutations that restore recombination, such as the reversion of inversions [203], or the movement of sex determining genes to other genomic regions [204, 205], leading to sex-chromosome turnover.

The presence of sexually antagonistic variation can influence this turnover in multiple ways. On one hand, sexually antagonistic alleles segregating elsewhere in the genome can promote turnover when new sex-determining factors arise in linkage with alleles beneficial to the sex they produce (section 3.6; [34, 184, 185, 206]). On the other hand, once antagonistic alleles have become associated with a sex chromosome, recombination restoration can become costly because it breaks associations between alleles and the sex in which they are favoured. This stabilising effect may be reinforced after recombination has been suppressed: degeneration of the heterogametic chromosome reduces expression in that sex, and compensatory upregulation can generate further sexual antagonism when regulatory effects are shared across males and females [207]. As dosage compensation and other sex-specific regulatory differences accumulate, recombination restoration and sex-chromosome turnover become increasingly costly because these events would disrupt the regulatory context in which linked genes

have evolved (i.e., the accumulation of regulatory differences between the sexes eventually generates sexual antagonism) [203, 208].

Sexual antagonism may also contribute to the evolution of sex-determining cascades – the regulatory hierarchies linking the initial sex-determining signal to downstream genes involved in sexual differentiation. For instance, it has been proposed that *Drosophila*'s cascade evolved through successive recruitment of upstream regulators when mutations sharpened male–female developmental divergence, with sexually antagonistic mutations acting as transient steps in this process [209]. More generally, the lability of sex-determining pathways across taxa suggests that such cascades can evolve rapidly, although the role of sexual antagonism in driving this lability remains unclear. A model of hierarchical gene regulation with shared expression across sexes shows that when sexually antagonistic selection maintains polymorphism in gene expression, selection can favour the displacement of this variation upstream in the cascade, as this reduces the fitness cost of intermediate expression at downstream targets [210].

Finally, sexual antagonism can also reshape sexual systems more fundamentally by favouring the evolution of separate sexes from hermaphroditism. Because allocation to male versus female function draws on a shared resource budget, sex allocation itself is intrinsically antagonistic in hermaphrodites (Box I). While many ecological mechanisms favour the maintenance of hermaphroditism in these species, others have been proposed to favour specialisation into male and female functions [49, 211, 212]. Additional sexually antagonistic variation can facilitate this transition, either by favouring sterility mutations linked to alleles beneficial to the sex they produce [36] or by allowing individuals specialising into male or female function to express phenotypes more beneficial to their preferred function leading to the simultaneous emergence of separate sexes and sexual dimorphism through correlation selection [37].

### 4.4 The evolution of sexual dimorphism

While sexual dimorphism can arise through sex-concordant selection when males and females differ in their evolutionary response to the same selective pressure (Fig. 4D [52, 55]), and even transiently simply through genetic drift [213], the more pronounced and persistent cases of sexual dimorphism observed in many taxa are generally thought to result from sex-specific selection [1–3]. When male and female traits have different optima but share a common genetic basis, such selection generates sexual antagonism. If constraints on male and female trait expression are not immutable (see Box I), evolution can proceed through several routes that decouple male and female phenotypes, at least in

stable environments, allowing each sex to move closer to its optimum and thereby reducing sexual antagonism and the associated sex load [5, 9, 54, 55, 214].

Some of these routes involve the modification of aspects of the genotype-fitness map, such as sex-specific dominance, or changes to genetic transmission, such as sex linkage (section 3.6; Table 1 for summary). But the impact of these mechanisms on sexual dimorphism may be limited, as most of them act only when there is genetic variation at target loci and provide only a partial resolution of sexual antagonism. For example, the evolution of sex-specific dominance, which requires substantial heterozygosity at target loci, can improve heterozygote fitness while still leaving maladapted homozygotes in each sex.

The complete resolution of sexual antagonism, where females and males evolve to their respective optima and the sex load is completely eliminated, is likely to instead require evolutionary responses at loci with direct sex-specific effects on phenotype. Quantitative genetic models predict the divergence of traits in males and females as long as sex-specific genetic variation is present, that is when the between-sex genetic correlation is less than one (section 2.4, [12, 102, 215].

Perhaps because sex-specific trait expression provides an obvious route to resolving sexual antagonism, it has received comparatively little theoretical scrutiny (e.g., [21, 22]). However, how and when such expression evolves in practice, and what genetic signatures it should generate, remain important gaps in our understanding of sex-specific adaptation. Future work could leverage tools such as Gene Regulatory Network models (GRNs, [216, 217]), models of modifiers of mutational effects (so-called **M** matrices [218]), and theoretical approaches that integrate sex-specific development (e.g., [28]).

## 5 Concluding remarks

Sexual antagonism may be an inevitable consequence of the sexes sharing most of their genome. For more than 70 years, mathematical and computational models have been used to ask when this antagonism should arise, what form it should take, and what consequences it should have for adaptation, polymorphism, and the evolution of sex chromosomes and sexual dimorphism. One aim of this review has been to clarify how these questions have been addressed across three main modelling traditions: population genetics, invasion analysis, and quantitative genetics. At times these approaches can appear to give different answers, but this largely reflects differences in assumptions and in the quantities they track. For comparable scenarios, they provide consistent and complementary views of the same process, from short-term allele frequency change, to long-term evolutionary trajectories, to the re-

sponse of highly polygenic traits constrained by cross-sex genetic covariance.

A large part of this theory has focused on whether sexual antagonism can maintain genetic polymorphism. That focus has been productive, leading to clear predictions about when balancing selection should be expected. Outside certain sex-linked regions, sexual antagonism does not seem especially effective at maintaining variation either across long time periods or across multiple loci unless traits show very specific patterns of sexual dimorphism in heterozygote but not homozygote genotypes. Whilst observed in a handful of systems, it remains unclear how common such patterns of sex-specific dominance are in nature or how they can be connected with an underlying molecular mechanism. The restrictive nature of polymorphism criteria does not imply that sexual antagonism is unimportant for genetic variation: it can still contribute to substantial genome-wide sex-specific genetic variance in fitness even without maintaining balanced polymorphism at underlying loci. Rather than asking whether sexual antagonism acts to maintain elevated genetic variation, it may be more fruitful to ask when the genetic architecture of traits makes conflict-resolution slow enough for antagonistic variation to persist. Answering this will require more explicit representations of how traits are built, for example through gene regulatory networks or other mechanistic descriptions of how allelic effects are expressed across sexes.

The emphasis on genetic polymorphism has perhaps left the ecological side of sexual antagonism less fully developed. This matters because sexual antagonism is likely to be most persistent when selection on males and females changes through time, such that sexual dimorphism does not simply catch up and resolve the conflict. Much of the existing theory has treated this in terms of fluctuating external environments. Yet the selective environment of each sex is also shaped by biotic interactions, and this remains less well explored. What counts as a fit male or a fit female may change continuously owing to co-evolution between the sexes, for example through mate choice or sexual conflict, and owing to sex-specific interactions with co-evolving parasites, predators, preys or mutualists. Sex-specific selection may also depend on how sexual traits and preferences are acquired, including through non-genetic inheritance such as cultural transmission, yet such gene-culture co-evolutionary dynamics remain largely absent from existing models of sexual antagonism.

In addition to co-evolutionary change, sex-specific selection can vary over shorter timescales, for example across developmental or life-history stages and with changing demographic conditions. What is favoured in males and females may depend on when in the life cycle a trait is expressed, on the condition or age of the individual, and on the demographic setting in which mating and competition occur. How traits respond to such fluctuations in selection will also depend on developmental constraints that limit when, where, or how far trait expression can diverge between the sexes. These processes are

still only weakly integrated into most models, yet they are likely to be important because they affect mating opportunities, the strength of sexual selection, and the relative importance of survival, fecundity, and mating success. Extending theory in these directions should help place sexual antagonism within a more explicit genetic and ecological setting.

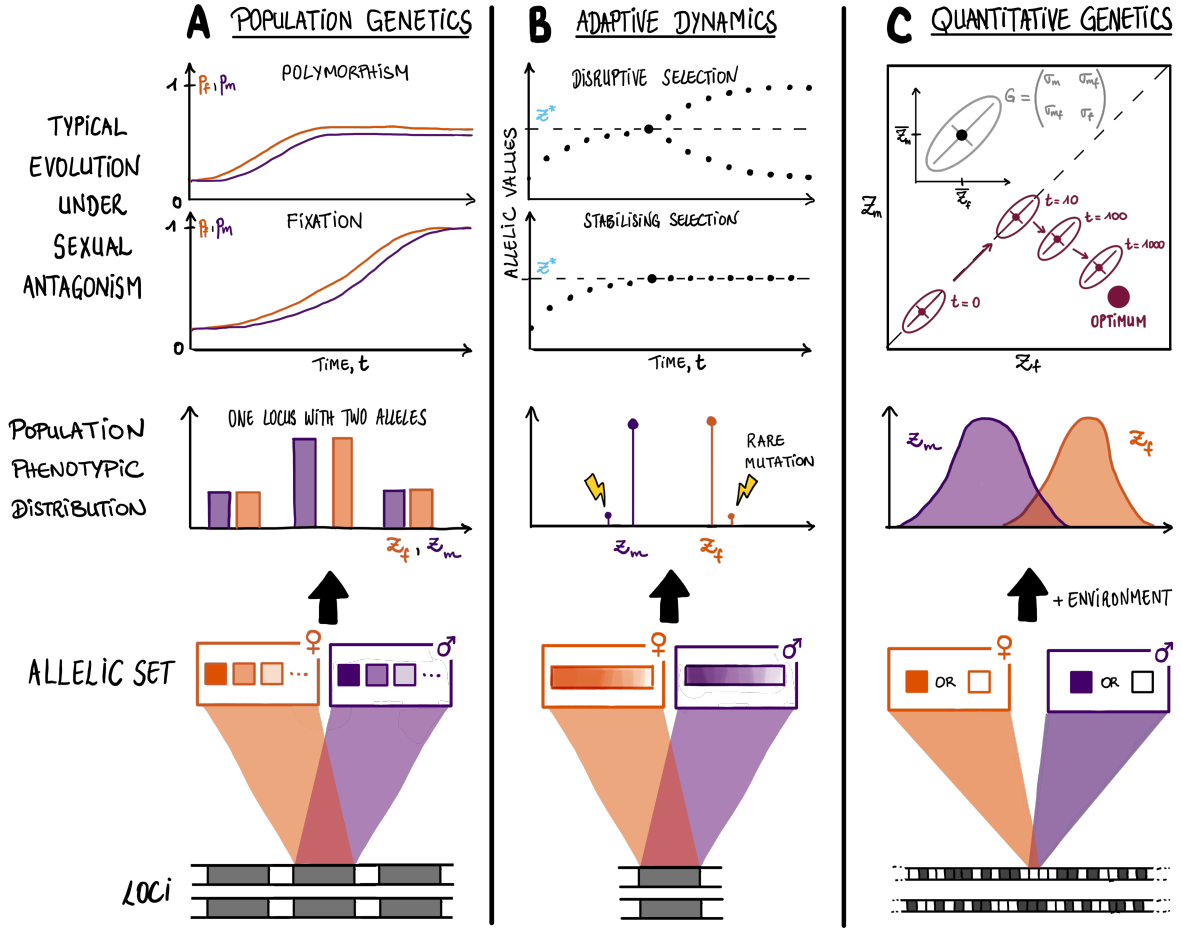


**Figure 1: Genetic architectures and resulting evolutionary dynamics under the three main theoretical approaches that have been used to investigate evolution under sex-specific selection**. **A** Population genetic models of sexual antagonism consider evolution at one or a handful of loci with a finite, fixed set of alleles that may have sex-specific properties (e.g., different dominance coefficients in males and females), resulting in a discrete distribution of male and female phenotypes. This approach studies the change in allele frequencies at these loci to answer questions such as when we should expect them to remain polymorphic given a fixed set of alleles, therefore focusing on short term evolution (Section 2.2 for details). **B** The adaptive dynamics approach is primarily concerned with how selection shapes phenotypic evolution in the long-term. To investigate this, it assumes that traits are encoded by a single locus at which alleles encoding any (potentially sex-specific) trait value may arise through mutation. Mutations are assumed to be rare and to have small effects so that evolution proceeds gradually, potentially leading to the emergence of divergent alleles maintained in a long-term polymorphism (see Section 2.3 for details). **C** Quantitative genetic models of sexual antagonism assume that male and female traits are affected by an infinite number of loci that may each carry either trait-increasing or trait-decreasing alleles with potentially sex-specific effects. Under this assumption, the phenotypic distributions of males and females in the population can be described statistically. The quantitative genetic approach describes how the moments of these distributions (their means and (co)variances under normal closure) are changed by selection, segregation and mutation to answer questions such as how genetic correlations between the sexes delay sex-specific adaptation (Section 2.4 for details).

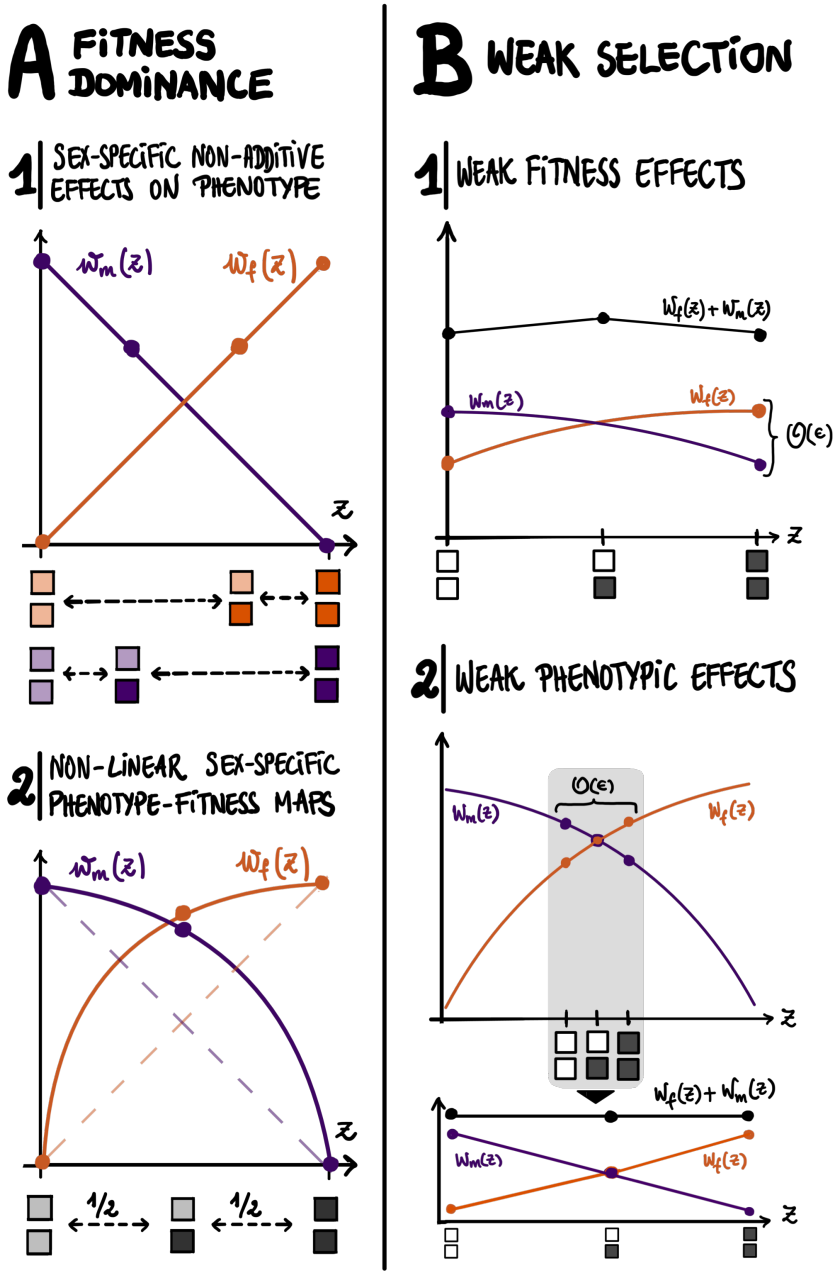


**Figure 2: Sources of dominance and their relationship to different notions of weak selection**. **A** Fitness dominance at a di-allelic locus under sex-specific selection may be produced in two ways. The first occurs when the locus shows phenotypic dominance (top panel), such that heterozygotes express trait values that do not sit halfway between homozygotes. This leads heterozygotes to have sex-specific fitnesses that deviate from the average of the two homozygotes (i.e., to fitness dominance). The second way to produce fitness dominance is for the phenotype-to-fitness map to be non-linear (bottom panel), which causes heterozygote fitnesses to deviate from the mean homozygote even when alleles combine additively at the phenotypic level. **B** Weak selection can be incorporated in different ways. In population genetics, it is common to assume that the fitness cost of carrying the least-preferred allele in each sex is small (i.e., $s_m$ and $s_f$ small, top panel). This preserves dominance relationships between alleles irrespective of whether they stem from phenotypic dominance or non-linear phenotype-to-fitness maps. In contrast, many other models assume that weak selection stems from the fact that alleles encode similar trait values (bottom panel). This causes fitness to vary linearly with trait value over the small phenotypic range spanned by the alleles, which negates dominance effects coming from non-linear phenotype-to-fitness maps. Fitness dominance at a locus with small effects on phenotype must then stem from phenotypic dominance.

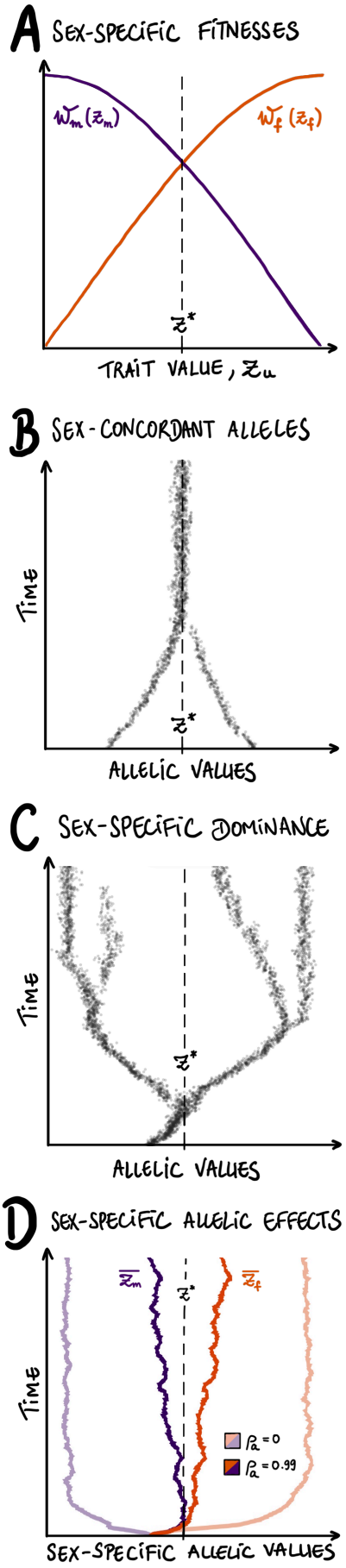


**Figure 3: Gradual evolution at a locus under different genetic architectures**. **A** Sex-specific phenotype-fitness maps, $w_u(z_u) = \exp\left(-\omega_u(z_u-\theta_u)^2\right)$, $u \in \{\mathrm{m},\mathrm{f}\}$, where $\theta_u$ gives the optimal trait value and $\omega_u > 0$ measures the strength of selection in sex $u$. Here, $\theta_\mathrm{f} = -\theta_\mathrm{m}$ and $\omega_\mathrm{f} = \omega_\mathrm{m}$ and such that larger trait values are favoured in females than males, but selection is stabilising for a compromise trait value $z^*$. **B-C** Simulated evolution at a trait locus mutating according to the continuum-of-allele model. **B** Sex-concordant allelic values and dominance. Each point is an allele, with its position giving its allelic value and time since the start of the simulation. The population is initialised with two highly diverged alleles but stabilising selection favours more intermediate values. The two lineages gradually merge into a one that encodes the compromise trait value $z^*$. **C** Sex-specific dominance with sex-concordant allelic values. Alleles with value $a$ are expressed in proportion to their sex-specific dominance level $b_u(a)$ in heterozygotes in sex $u$, leading to adaptive dominance reversal is produced, with alleles increasing trait value being dominant in females and recessive in males (i.e., $b_\mathrm{f}(a)$ and $b_\mathrm{m}(a)$ increase and decrease with $a$, respectively). Alleles first evolve to encode the compromise trait value $z^*$ and then undergo recurrent evolutionary branching as adaptive dominance reversal for phenotype generates heterozygote advantage as in [47] (Appendix B for details). **D** Sex-specific dominance and allelic values. Here, each allele can also encode sex-specific values such that male and female traits (purple and orange lines, respectively) can diverge in response to directional selection in each sex. How readily this divergence occurs depends on the degree to which mutations affect male- and female-allelic values in a correlated way. In the absence of correlation ($\rho_a = 0$, lighter lines), male and female traits evolve independently and rapidly to their respective optima. With a strong correlation ($\rho_a = 0.99$, darker lines), male and female trait values first converge towards the compromise $z^*$ before slowly diverging.

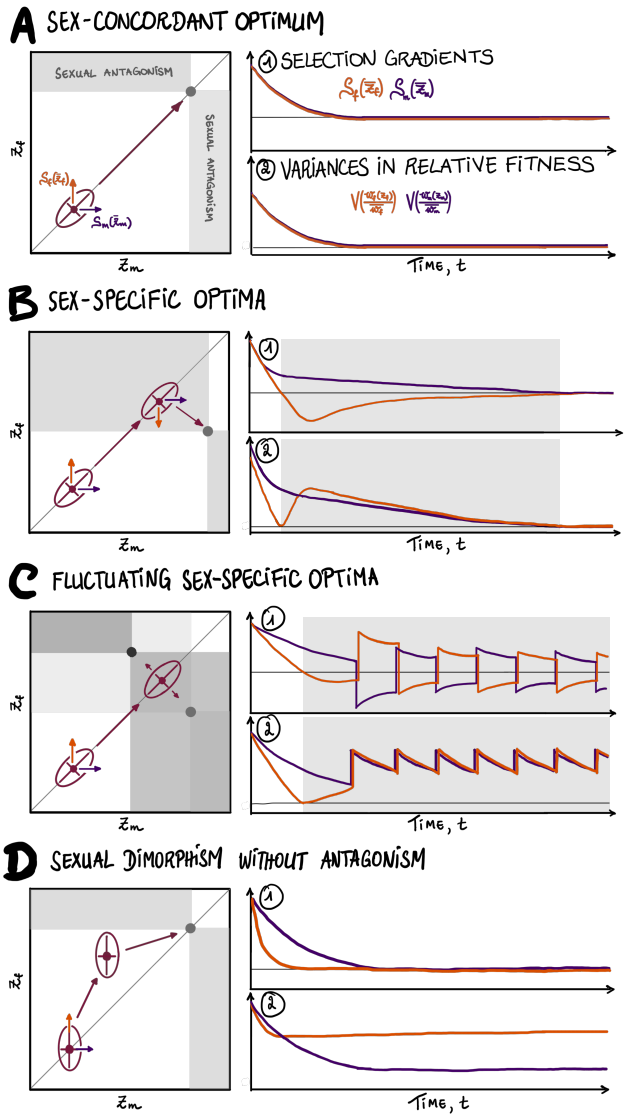


**Figure 4: Evolutionary trajectories and associated sex-specific fitness variances in a polygenic trait.** For each case, the left panel illustrates the expected trajectory of the polygenic trait in phenotypic space, with optimum shown as gray circle and population represented as ellipse. Grey areas indicate regions where sexual antagonism occurs under positive cross-sex genetic correlation (i.e. where male and female traits are selected in opposite directions); and the right panel shows sex-specific selection gradients (top) and sex-specific variances in relative fitness (bottom) along the expected evolutionary trajectory. **A** Evolution towards a sex-concordant optimum. **B** Sex-specific optima and transient sexual antagonism. The population first evolves sex-concordantly before overshooting the female optimum, resulting in sexual antagonism, and then slowly converges to optimum. Sex-specific fitness variances thus remain elevated for longer than in A. **C** Fluctuating sex-specific optima. The population evolves to sit between the male and female optima that periodically switch position, preventing the resolution of sexual antagonism, resulting in the persistence of substantial genetic variance for fitness in males and females. **D** Sex-concordant optimum with and large differences in sex-specific variance and no covariance across sexes. Because females have much more heritable variation for selection to act on, their trait evolves towards the sex-concordant optimum much more rapidly than the male trait does. This results in transient sexual dimorphism in the absence of sexual antagonism.

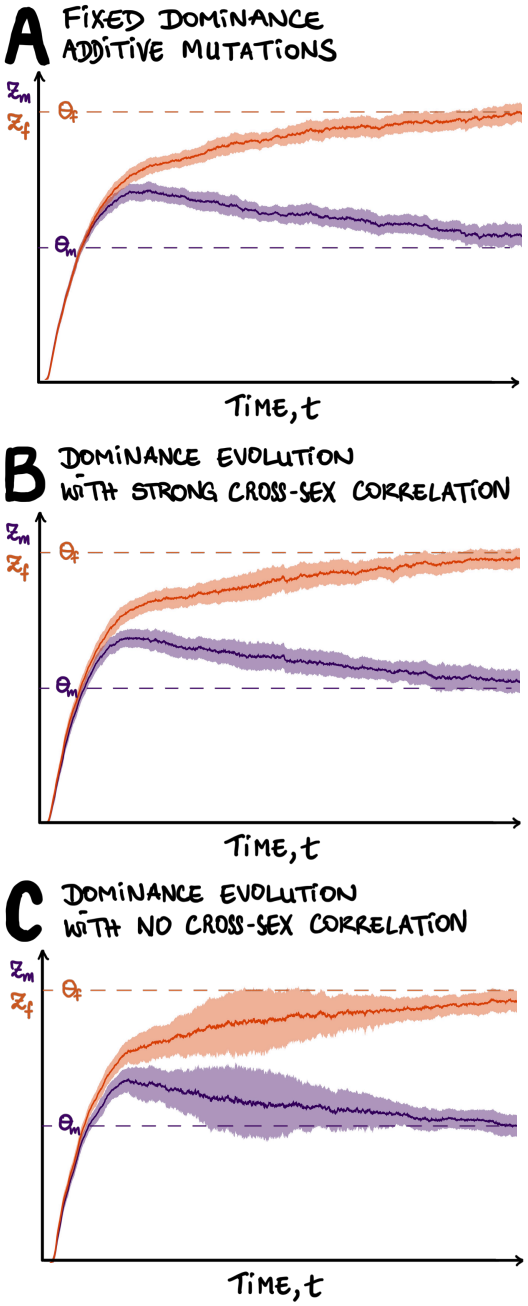


**Figure 5: Effect of the evolution of sex-specific dominance on dynamics of a polygenic trait.** This shows the results of individual based simulations where a sexually antagonistic trait evolves via joint changes at multiple loci potentially leading to the evolution of sex-specific dominance on phenotype (Appendix C.2.1 for details). All panels consider the case when mutations have largely sex-concordant effects on allelic values since otherwise sexual dimorphism evolves very rapidly (Fig. S1). Solid lines give the mean trait value in females (orange) and males (purple). Shaded areas around each line correspond to the two-standard deviations interval around mean trait values, as an indicator of the phenotypic variance in each sex. **A** Baseline scenario with fixed additive effects. Male and female trait values gradually diverge with limited variance in each sex. **B** Dominance can evolve, but with a high correlation across sexes ($\rho_b = 0.9$), making sex-specific dominance harder to evolve. Trait divergence is associated with slightly greater levels of genetic variance to the additive case in both sexes (larger shaded area). **C** Dominance can evolve in males and females independently ($\rho_b = 0$). Variance is substantially higher in both sexes as male and female traits begin to diverge, but eventually decreases to a level similar to the additive case as the sexes reach their respective optima.

Box I. Sexual antagonism, sex load, and constraints

In this review, we follow the common definition of sexual antagonism as a genetic trade-off arising when male and female fitness are maximised by different genotypes [5, 17, 219], such that different alleles are favoured in each sex at a given locus (hence *intralocus* sexual conflict [9]). Under this definition, sexual antagonism may arise in dioecious and hermaphroditic species alike: the trade-off is between allelic effects on male versus female individual fitness in dioecious species [220], whereas in hermaphrodites it concerns male and female fitness components of the same individual [221] (i.e., allocation to sperm versus eggs).

For different genotypes to have sex-specific fitness effects, there must be constraints on trait expression that prevent male and female trait values from evolving independently. We distinguish two types. The first type, which can arise in all species, consists of **genetic constraints**. These occur male and female fitness could in principle be simultaneously maximised, but available genetic variation does not allow trait expression to be decoupled across the sexes. For example, if male and female optima for body size diverge in a dioecious species (e.g., following a change in sex-specific ecology), sexual antagonism can arise if alleles underlying body size initially have similar effects in males and females, as expected if previous selection had been largely sex-concordant. Such antagonism is ultimately **resolvable** by the appearance of alleles with sex-specific effects that alleviate genetic constraints on male and female trait values and thereby facilitate the evolution of sexual dimorphism. However, depending on how readily such variants arise, this resolution may take a long time and impose long-lasting restrictions on sex-specific adaptation [13, 55].

A second type of constraint arises specifically in hermaphroditic species, when male and female fitness components cannot be simultaneously maximised. This occurs when the enhancement of male and female functions draws on a shared resource budget (e.g., energy, time or space), and is therefore limited by **physiological constraints**. For example, if different body sizes are optimal for the two sexual functions in a hermaphrodite, the trade-off will cannot be resolved while the species remains hermaphroditic. Sexual antagonism will therefore persist for as long as the ecological source of sex-specific selection does. The only way for such constraints to be lifted is through the evolution of separate sexes (dioecy), allowing individuals to specialise in only one sexual function (e.g., through subsequent sex-specific gene expression). Such transitions may often be opposed by ecological factors such as limited dispersal or reproductive assurance, which can make hermaphroditism a stable strategy [211]. In many cases, sexual antagonism in hermaphrodites may therefore be, for all intents and purposes, **unresolvable**.

As a consequence of these trade-offs, mean fitness is reduced below the value attainable if male and female fitness components could be maximised independently [9]. We refer to this reduction as the "sex load" (also sometimes known as the "gender load" [222, 223]). Operationally, the sex load can be defined as the reduction in mean fitness relative to a counterfactual population exposed to the same environment in which male and female trait values can evolve independently (i.e., without cross-sex genetic correlations). The sex load can be a fixed load when the population is monomorphic, but it includes a segregation load if polymorphic and a recombination load if alleles are segregating at multiple loci. This fitness deficit is an inherent consequence of ongoing sexual antagonism [4, 5, 9, 12], and it persists so long as genetic constraints prevent the evolution of well-adapted sex-specific phenotypes [8, 224]. The sex load is thus expected to be a common feature of many, if not all, sexual species.

| Modified Trait | Synopsis | Short-term polymorphism | Long-term polymorphism | Sex load | Ref. |
|---|---|---|---|---|---|
| | **Modifiers of the Genotype-Fitness map** | | | | |
| Dominance | Modifiers spread if they increase the average fitness of heterozygotes, with long-term effects on polymorphism depending on the mechanism and sex-specificity. | + | +/− | − | [61] |
| Genomic Imprinting | Imprinting modifiers evolve to upregulate the expression of trait alleles in same sex offspring. | + | + | − | [174] |
| Gene Expression | Cis-acting modifiers that cause an allele to be more sex-limited in expression are favored when those effects coincide with the direction of selection in that sex. | + | + | − | [22] |
| Gene Copy Number | Duplication can be seen as a modifier of gene copy number, which can be favoured when there is an SA polymorphism. Gene duplication allows fixed heterozygosity of male-beneficial and female-beneficial alleles. | − | − | − | [178] |
| | **Modifiers of genetic transmission** | | | | |
| Recombination | Modifier alleles that reduce recombination between polymorphic SA loci and the sex-determining region generally spread (but see [225]). They can include structural variants such as chromosomal fusions [181] or inversions [208]. | + | + | − | [24] |
| Sex Ratio | Modifiers that adjust offspring sex ratio in response to SA can increase the chance that offspring of a sex inherit the allele beneficial in that sex († When sex-linked, generates sex ratio distortion that can, however, drive allele fixation). | $+^{\dagger}$ | $+^{\dagger}$ | $-^{\dagger}$ | [33] |
| Sex determination | A gene modifying sex determination can spread when linked with an autosomal SA locus, driving transitions between sex chromosomes. | + | + | − | [34] |
| Mating Preference | When SA and preference loci are sex-linked, preferences evolve to favor mates carrying alleles beneficial to daughters with XY sex determination but to favor mates carrying alleles beneficial to sons in ZW systems. | − | − | + | [32] |

**Table 1: Models exploring the influence of sexually antagonistic (SA) selection on other features.** Columns 3-5 describe the typical outcome following modifier evolution on the short-term maintenance of a polymorphism with a fixed set of alleles, say $A_1$ and $A_2$ ("Short-term polymorphism"), long-term maintenance via branching where new alleles can emerge ("Long-term polymorphism"), and the sex load ("Sex load"). References provide only an entry point into the topic.

## Supplementary Figures

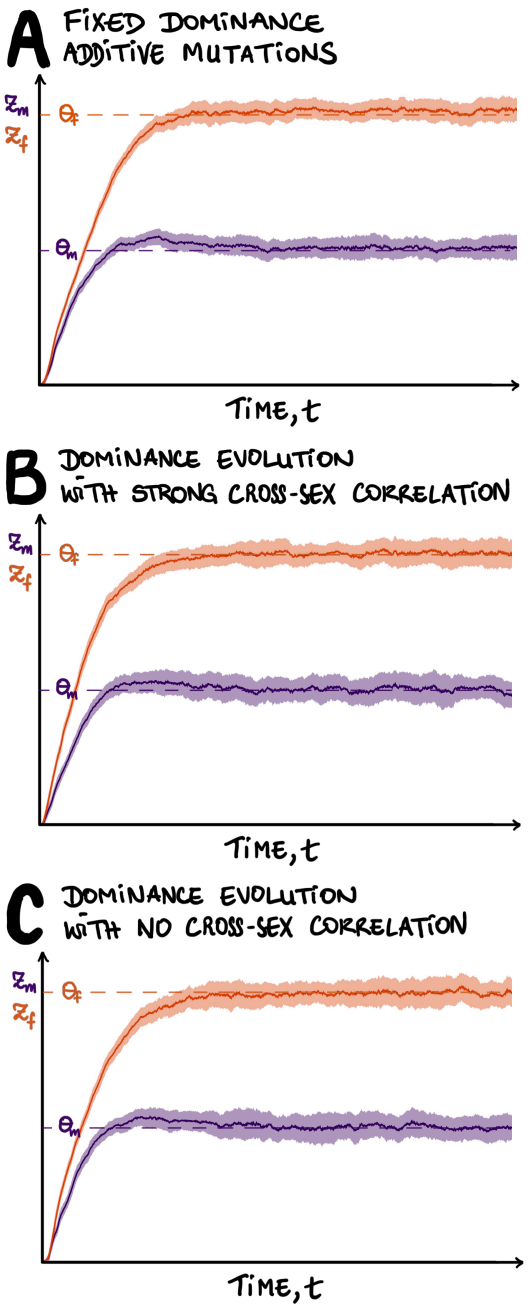


**Figure S1:** Effect of dominance on the evolution of a polygenic trait under sexually antagonistic selection, when mutations frequently have sex-specific effects on allelic values ($\rho_a = 0.4$; Appendix C.2.1 for details). Solid lines give the mean trait value as a function of time in females (orange) and males (purple). Shaded areas around each line indicate the two standard deviations interval around mean trait values. Dominance evolution has little to no impact on the evolution of sex-specific trait means and variances when mutations can often be favoured in the two sexes simultaneously.

# Appendix to

# "Sexually antagonistic selection: a review of the theory and its implications"

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# A Population genetics of a single locus under sex-specific selection

## A.1 Evolution at a diallelic locus

In this section, we consider evolution at a locus with two alleles, $A_1$ and $A_2$, when these have small, sex-specific effects on trait $z$ (i.e., generalising eq. (6) of the main text). $A_1A_1$ homozygotes express trait value $z_{u,11}$ while $A_2A_2$ homozygotes express $z_{u,11}+\delta_u$ in sex $u \in \{\mathrm{m},\mathrm{f}\}$ , where $\delta_u \sim \mathcal{O}(\epsilon)$ is a small sex-specific deviation. Female heterozygotes express trait value $z_{\mathrm{f},11}+\eta_\mathrm{f}\delta_\mathrm{f}$ and male heterozygotes express $z_{\mathrm{m},11}+(1-\eta_\mathrm{m})\delta_\mathrm{m}$ such that $\eta_\mathrm{f} \in [0,1]$ is the phenotypic dominance coefficient of allele $A_2$ in females and $\eta_\mathrm{m} \in [0,1]$ is the phenotypic dominance coefficient of allele $A_1$ in males. Phenotypic dominance is the same in males and females when $\eta_\mathrm{f} = 1-\eta_\mathrm{m}$ and eq. (6) of the main text considers the case when $\eta_\mathrm{f} = \eta_\mathrm{m} = 1/2$.

With $p_\mathrm{f}$ and $p_\mathrm{m}$ as the frequency of allele $A_2$ in female and male gametes, the change in allelic frequencies can be expressed as main text eq. (1). Under weak selection, we can track the genetic state of the population by the reproductive-value weighted $p = \alpha_\mathrm{f}p_\mathrm{f} + \alpha_\mathrm{m}p_\mathrm{m}$, where $\alpha_u$ denotes the class-reproductive value of sex $u \in \{\mathrm{m},\mathrm{f}\}$ [68]. By definition, reproductive values can be obtained from $\Delta p = \alpha\Delta p_\mathrm{f} + (1-\alpha)\Delta p_\mathrm{m}$, which must be equal to zero under neutrality. Plugging eq. (1) into this condition and setting $w_\mathrm{f}(z) = w_\mathrm{m}(z) = 1$ for all $z$, we obtain $\alpha = 1/2$, as expected.

### A.1.1 Consequences of sex-specific phenotypic effects and dominance reversal

Using eq. (1), we obtain the change in weighted frequency $\Delta p = \Delta p_\mathrm{f}/2 + \Delta p_\mathrm{m}/2$ is

$$\Delta p = \frac{p(1-p)}{2}\left[\delta_\mathrm{f}\Big(p+\eta_\mathrm{f}(1-2p)\Big)S_\mathrm{f}(z_{\mathrm{f},11}) + \delta_\mathrm{m}\Big(p+(1-\eta_\mathrm{m})(1-2p)\Big)S_\mathrm{m}(z_{\mathrm{m},11})\right] + \mathcal{O}\left(\epsilon^2\right). \tag{A-1}$$

Eq. (A-1) has the same structure as the change in allele frequency obtained assuming small effects on fitness (rather than phenotype) given in eq. (4) of the main text, with two important differences. First, the sex-specific selection coefficients $s_\mathrm{f}$ and $s_\mathrm{m}$ in eq. (4) have been replaced by the sex-specific selection gradients on trait $z$, $S_\mathrm{f}(z_{\mathrm{f},11})$ and $S_\mathrm{m}(z_{\mathrm{m},11})$ (defined in eq. 7 of main text). This reflects the fact that sex-specific fitnesses change approximately linearly with trait value when alleles only cause small phenotypic changes (Fig. 2). Second, sex-specific dominance coefficients $h_\mathrm{f}$ and $h_\mathrm{m}$ in eq. (4), which are agnostic to whether dominance relationships between alleles result from dominance effects at the phenotypic level or from non-linear fitness landscapes, have been replaced by phenotypic dominance coefficients $\eta_\mathrm{f}$ and $\eta_\mathrm{m}$. Dominance effects can no longer stem from the shape of sex-specific

fitness landscapes here (because these landscapes are approximately linear over the phenotypic range spanned by alleles $A_1$ and $A_2$, Fig. 2).

From eq. (A-1), the condition for the locus to remain polymorphic is

$$\frac{\eta_f}{1-\eta_m} < -\frac{\delta_m}{\delta_f}\frac{S_m(z_{m,11})}{S_f(z_{f,11})} < \frac{1-\eta_f}{\eta_m}, \tag{A-2}$$

which can only be satisfied if $\eta_m + \eta_f < 1$, that is, if alleles are on average more dominant in the sex they are most beneficial to, leading to male and female heterozygotes trait values that are shifted towards the male and female optima respectively. Heterozygotes are thus sexually dimorphic. In other words, when alleles have weak phenotypic effects, polymorphism is only possible if the locus shows adaptive dominance reversal for phenotype.

By contrast, when alleles show the same dominance across the sexes ($\eta_f = 1-\eta_m$) or maladaptive dominance reversal ($\eta_f + \eta_m > 1$), polymorphism is impossible. With $\eta_f = 1-\eta_m$, eq. (A-1) becomes

$$\Delta p = \delta \frac{p(1-p)}{2}\big(\eta + p(1-2\eta)\big)\big[\delta_f S_f(z) + \delta_m S_m(z)\big] + \mathcal{O}\left(\epsilon^2\right), \tag{A-3}$$

and allele frequency $p$ no longer affects the sign of $\Delta p$ (because $\eta + p(1-2\eta) > 0$). Either $A_1$ or $A_2$ to go to fixation, depending on $\delta_m$ and $\delta_f$ and on directional selection in each sex. In particular, when selection is sexually antagonistic ($S_f(z)$ and $S_m(z)$ are of opposing signs), eq. (A-3) shows that any allele that takes the sexes towards their preferred direction will go to fixation, e.g. if selection favours a larger trait in females and a smaller trait in males (so $S_f(z) > 0$ and $S_m(z) < 0$) then an allele increasing the trait in females ($\delta_f > 0$) and decreasing it in males ($\delta_m < 0$) is always favoured (the term between square brackets in eq. (A-3) is strictly positive). The repeated fixation of such alleles appearing through recurrent mutation would eventually allows the sexes to reach their optima (such that $S_f(z) = S_m(z) = 0$).

### A.1.2 Condition for polymorphism with sex-concordant effects

When allelic effects are always identical in both sexes (i.e., when $z_{f,11} = z_{m,11} = z$, $\eta_f = 1-\eta_m = \eta$ and $\delta_f = \delta_m$), eq. (A-1) becomes

$$\Delta p = \delta \frac{p(1-p)}{2}\big[\eta + p(1-2\eta)\big]\big(S_f(z) + S_m(z)\big) + \mathcal{O}\left(\epsilon^2\right), \tag{A-4}$$

which with $\eta = 1/2$ yields eq. (6) in the main text. Eq. (A-4) shows that no polymorphism can be maintained here. However, if the resident population is distributed close to a compromise trait value $z = z^*$ such that such that $S_f(z^*) = -S_m(z^*)$ (see main text section 2.3.2), leading order effects vanish (i.e.,

$\Delta p = 0 + \mathcal{O}\left(\epsilon^2\right)$) and second-order effects of selection come into play (terms of order $\mathcal{O}\left(\epsilon^2\right)$).

At $z = z^*$, a second-order Taylor expansion of $\Delta p$ around $\epsilon = 0$ using eq. (1) gives us

$$\Delta p = \delta^2 \frac{p(1-p)}{4} \Big[ \left(\eta^2 + p\left(1-2\eta^2\right)\right)\left(H_{\mathrm{f}}(z^*) + H_{\mathrm{m}}(z^*)\right) - 2p\big(2\eta + p(1-2\eta)\big)\big(\eta + p(1-2\eta)\big)\left(S_{\mathrm{m}}(z^*)^2 + S_{\mathrm{f}}(z^*)^2\right)\Big] + \mathcal{O}\left(\epsilon^3\right), \tag{A-5}$$

which depends on the quadratic selection gradients in each sex (eq. (12)) as well as the phenotypic dominance coefficient $\eta$. For eq. (A-5), the conditions for polymorphism to be maintained are

$$0 \;<\; H_{\mathrm{f}}(z^*) + H_{\mathrm{m}}(z^*) \;<\; 2\frac{1}{1+\eta}\left(S_{\mathrm{m}}(z^*)^2 + S_{\mathrm{f}}(z^*)^2\right). \tag{A-6}$$

This shows that $H(z^*) > 0$ is required for polymorphism to be maintained when alleles have sex-concordant effects. The condition for polymorphism obtained here strongly resembles the branching condition given in the main text (eq. (14)), but is slightly more permissive as polymorphism only constitutes a first step in the process of evolutionary branching, as we elaborate in the next section.

### A.2 The population-genetic process of evolutionary branching

In this section, we detail the onset of evolutionary branching using population genetics. Readers familiar with this process may wish to skip this section, although we develop it here in detail for sex-structured diploid populations, a setting that has received little explicit treatment.

Trait $z$ is determined by a single locus at which alleles may encode any trait value, combine additively in heterozygotes (so $\eta = 1/2$) and are subject to rare mutations with small effects. Under this assumption, the population will converge to a singular trait value $z^*$ such that $S_{\mathrm{f}}(z^*) = -S_{\mathrm{m}}(z^*)$ if

$$\left.\frac{\mathrm{d}S(z)}{\mathrm{d}z}\right|_{z=z^*} = H(z^*) + I(z^*) < 0, \tag{A-7}$$

where $H(z^*)$ is given by eq. (11) in the main text and

$$I(z^*) = \left.\frac{\partial^2 W(z_\bullet, z)}{\partial z_\bullet \partial z}\right|_{z_\bullet = z = z^*} = H(z^*) - \frac{S_{\mathrm{f}}(z^*)^2 + S_{\mathrm{m}}(z^*)^2}{4} \tag{A-8}$$

can be thought of as capturing the strength and direction of frequency-dependent selection [90] (with $W(z_\bullet, z)$ invasion fitness as given by eq. (8) in the main text). Once the population expresses $z^*$, the

population will undergo evolutionary branching if $H(z^*) + I(z^*) < 0$ and $H(z^*) > 0$, i.e., if

$$0 < H(z^*) < -I(z^*), \tag{A-9}$$

which is equivalent to condition (14) in the main text.

We will show that, when condition (A-9) holds, a population initially fixed for an allele encoding $z^*$ can be invaded by a nearby mutant, say $z^* + \delta$, leading to a two-allele polymorphism. This polymorphism is then susceptible to invasion by a second mutant on the opposite side of $z^*$, say $z^* - \delta$. The latter displaces the allele encoding $z^*$, resulting in a stable polymorphism in which two alleles are maintained on either side of the singular value ($z^* - \delta$ and $z^* + \delta$).

To do so, let us consider a situation where the locus encoding $z$ has three alleles, $\mathrm{A}_1$, $\mathrm{A}_2$ and $\mathrm{A}_3$. In homozygotes, allele $\mathrm{A}_1$ encodes a singular trait value $z^*$ such that $S_\mathrm{f}(z^*) = -S_\mathrm{m}(z^*)$. Allele $\mathrm{A}_2$ and $\mathrm{A}_3$, meanwhile encode trait values $z^* + \delta$ and $z^* - \delta$, respectively, with $\delta > 0$. For brevity, we write as $z_{ij}$ the trait value expressed by an individual carrying alleles $\mathrm{A}_i$ and $\mathrm{A}_j$. Under this genetic model and using these notations, the change in frequency of allele $\mathrm{A}_i$ ($i \in \{1,2,3\}$) over one generation can be expressed as

$$\Delta p_{i,u} = p_{i,\mathrm{m}} p_{i,\mathrm{f}} \frac{w_u(z_{ii})}{\overline{w}_u} + \frac{1}{2} \sum_{j \neq i} \Big( p_{i,\mathrm{m}} p_{j,\mathrm{f}} + p_{j,\mathrm{m}} p_{i,\mathrm{f}} \Big) \frac{w_u(z_{ij})}{\overline{w}_u} - p_{i,u}, \tag{A-10}$$

where

$$\overline{w}_u = \sum_{i=1}^{3} \sum_{j=1}^{3} p_{i,\mathrm{m}} p_{j,\mathrm{f}} w_u(z_{ij}) \tag{A-11}$$

is the mean fitness in sex $u$. We leverage again the fact that under weak selection, we can describe the genetic state of the population in terms of the reproductive value weighted average allele frequencies of $\mathrm{A}_1$ and $\mathrm{A}_2$, $p_1$ and $p_2$ (with $p_3 = 1 - p_1 - p_2$), with $p_i = (p_{i,\mathrm{f}} + p_{i,\mathrm{m}})/2$, [68]. Using this, the change in allele frequencies can be written as

$$\Delta p_1 = -\delta^2 p_1 \left[ \frac{H(z^*)}{2} (1 - p_1) + I(z^*)(p_2 - p_3)^2 \right] + \mathcal{O}\left(\epsilon^3\right), \tag{A-12a}$$

and

$$\Delta p_2 = \delta^2 p_2 \left[ \frac{H(z^*)}{2} p_1 + I(z^*)(p_2 - p_3)\big(1 - (p_2 - p_3)\big) \right] + \mathcal{O}\left(\epsilon^3\right), \tag{A-12b}$$

where $p_3 = 1 - p_1 - p_2$.

From eq. (A-12), there are six possible equilibria

$$p_1^*, p_2^* \quad \text{such that} \quad \Delta p_1(p_1^*, p_2^*) = \Delta p_2(p_1^*, p_2^*) = 0 \tag{A-13}$$

for this three-allele system. Three of these equilibria correspond to the fixation of one of the three alleles (i.e., $p_i^* = 1$ and $p_j^* = 0$ for all $j \neq i$, $i, j \in \{1,2,3\}$); and three polymorphic where two of the three alleles coexist as a polymorphism (we refrain from giving these explicitly as they are unsightly but they can readily be obtained using an algebraic computer program). The three alleles can never coexist at equilibrium.

The stability of these equilibria is assessed from the Jacobian matrix $\mathbf{J}(p_1^*, p_2^*)$, defined as

$$\mathbf{J}(p_1^*, p_2^*) = \begin{pmatrix} \left.\frac{\partial \Delta p_1(p_1,p_2)}{\partial p_1}\right|_{\substack{p_1=p_1^* \\ p_2=p_2^*}} & \left.\frac{\partial \Delta p_1(p_1,p_2)}{\partial p_2}\right|_{\substack{p_1=p_1^* \\ p_2=p_2^*}} \\ \left.\frac{\partial \Delta p_2(p_1,p_2)}{\partial p_1}\right|_{\substack{p_1=p_1^* \\ p_2=p_2^*}} & \left.\frac{\partial \Delta p_2(p_1,p_2)}{\partial p_2}\right|_{\substack{p_1=p_1^* \\ p_2=p_2^*}} \end{pmatrix}, \tag{A-14}$$

with stability requiring the real parts of both eigenvalues of $\mathbf{J}(p_1^*, p_2^*)$, $\lambda_1(p_1^*, p_2^*)$ and $\lambda_2(p_1^*, p_2^*)$, to be negative.

Let us consider the stability of all the equilibria. We have

$$\lambda_1(1,0) = \lambda_2(1,0) = \frac{\delta^2}{2} H(z^*), \tag{A-15}$$

and

$$\lambda_1(0,1) = \lambda_1(0,0) = -2\delta^2 I(z^*) \quad \text{and} \quad \lambda_2(0,1) = \lambda_2(0,0) = -\delta^2 \left( \frac{H(z^*)}{2} + I(z^*) \right). \tag{A-16}$$

Hence, when the branching condition (eq. A-9) holds, none of the equilibria with fixed $A_1$, $A_2$ or $A_3$ is stable (i.e., all eigenvalues are positive). Similarly, since

$$\lambda_1(p_1^*, 1) = (p_1^*, 0) = \delta^2 H(z^*), \tag{A-17}$$

where $0 < p_1^* < 1$, the polymorphic equilibria involving the coexistence of $A_1$ and allele $A_2$ or $A_3$ are also unstable when the branching condition (eq. A-9) holds. In contrast, we obtain

$$\lambda_1(0, p_2^*) = -\delta^2 \frac{H(z^*)}{2} \quad \text{and} \quad \lambda_2(0, p_2^*) = \delta^2 I(z^*), \tag{A-18}$$

where $0 < p_2^* < 1$. This shows that the equilibrium involving the coexistence of the two alleles $A_2$ and $A_3$ sitting on either side of $z^*$ is the only stable equilibrium of the three allele system when the branching condition (eq. A-9) holds, as required.

Once such a polymorphism is established, evolutionary branching proceeds through the recurrent invasion of mutations affecting each allele $A_2$ and $A_3$ leading to their divergence. To model this, we may

consider a three-allele system where two alleles encode trait values on either side of $z^*$, i.e., $A_2$ encoding $z^* + \delta$ and $A_3$ encoding $z^* - \delta$, as before, and the third allele $A_1$ now encodes a trait value $z^* + k\delta$, where $k > 1$ so that it sits further way from $z^*$ than the trait-increasing allele $A_2$. In this case, eq. (A-12) is still sufficient to describe the genetic dynamics of the population, so that we may employ the same approach as above. Running through all possible equilibria, we find that the only stable equilibrium is one where the allele encoding $z^* + k\delta$ coexists with allele encoding $z^* - \delta$, as required.

# B Adaptive dynamics with sex-specific genotype-phenotype maps

In this appendix, we use an adaptive dynamics approach to investigate the evolution of trait $z$ under sex-specific genotype-phenotype maps allowing for dominance-reversal on phenotype. We assume that the trait is influenced by a single locus evolving under the continuum-of-alleles model with rare, small-effect mutations, but in contrast to the main text, the effects encoded at these alleles translate into trait values through continuous sex-specific genotype-phenotype maps that can produce sex-specific phenotypic dominance relationships. The analysis is similar in spirit to that of Siljestam et al. [47] who approached this problem with a pre-specified model of dominance-reversal.

## B.1 A continuous genotype-phenotype map and the dominance relationships it generates

Alleles are characterised by their allelic value $a \in \mathbb{R}$ i.e., by the trait value they encode when homozygous, which is assumed to be the same in both sexes (so homozygotes express the same phenotype in males and females; an identical interpretation of allelic effects is implicit in Siljestam et al. [47], see their eq. A-1). The phenotype expressed by an individual of sex $u \in \{\mathrm{m},\mathrm{f}\}$ carrying alleles with effects $a_1$ and $a_2$ as its maternally and paternally copies is given by the function $z_u(a_1,a_2)$, which we assume to be continuous and differentiable with respect to both its arguments. These functions define the genotype-phenotype map in males and females. We describe these in more detail in this section and also derive some of their properties that will be useful for later analyses.

We assume that there is no parent-of-origin effect (e.g., due to imprinting) on the phenotype, so that $z_u(a_1,a_2) = z_u(a_2,a_1)$. This entails that

$$\left.\frac{\partial^n z_u(a_1,a_2)}{\partial a_1^n}\right|_{a_1=a_2=a} = \left.\frac{\partial^n z_u(a_1,a_2)}{\partial a_2^n}\right|_{a_1=a_2=a} \tag{B-1a}$$

for any $n \in \mathbb{N}$.

From the definition above of allelic value, the genotype-phenotype maps are such that $z_\mathrm{f}(a,a) = z_\mathrm{m}(a,a) = a$ for all allelic value $a$ that can arise by mutation at the evolving locus (recall: the allelic value of an allele is the trait value expressed by a homozygote for that allele). This entails that the equality

$$z_u(a+\epsilon, a+\epsilon) = a+\epsilon, \tag{B-1b}$$

must hold for all $\epsilon$ and thus at all orders of $\epsilon$. In particular, to first order

$$\underbrace{z_u(a,a)}_{=a} + 2\epsilon \left.\frac{\partial z_u(a_1,a_2)}{\partial a_1}\right|_{a_1=a_2=a} = a+\epsilon \tag{B-1c}$$

must hold (where we used eq. B-1a), which implies that

$$\left.\frac{\partial z_u(a_1,a_2)}{\partial a_1}\right|_{a_1=a_2=a} = \left.\frac{\partial z_u(a_1,a_2)}{\partial a_2}\right|_{a_1=a_2=a} = \frac{1}{2}. \tag{B-1d}$$

Although homozygotes of both sexes show the same phenotype, heterozygotes can differ between males and females. The phenotypic dominance coefficient of an allele with allelic value $a+\epsilon$ relative to an allele with value $a$ in sex $u$ is given

$$\begin{aligned}\eta_u(a+\epsilon,a) &= \frac{z_u(a+\epsilon,a)-z_u(a,a)}{z_u(a+\epsilon,a+\epsilon)-z_u(a,a)} \\ &= \frac{1}{2} - \frac{\epsilon}{2}\frac{\left.\dfrac{\partial^2 z_u(a_1,a_2)}{\partial a_1 \partial a_2}\right|_{a_1=a_2=a}}{2\left.\dfrac{\partial z_u(a_1,a_2)}{\partial a_1}\right|_{a_1=a_2=a}} + \mathcal{O}\left(\epsilon^2\right) \\ &= \frac{1}{2} - \frac{\epsilon}{2}\left.\frac{\partial^2 z_u(a_1,a_2)}{\partial a_1 \partial a_2}\right|_{a_1=a_2=a} + \mathcal{O}\left(\epsilon^2\right).\end{aligned} \tag{B-1e}$$

where we used eq. (B-1d) to go from the second to third equality. This expression shows that, to leading order, alleles have additive effects ($\eta_u(a,a) = 1/2$), and that deviations from additivity are determined by the cross-derivative of the genotype–phenotype map. This term measures how changes in one allele modify the effect of changes in the other on phenotype. When the cross-derivative is negative, jointly increasing both allelic values by the same amount $\epsilon$ has a smaller effect on the trait than increasing the value of only one allele by twice this amount ($2\epsilon$). As a result, alleles increasing trait value are phenotypically dominant (and decreasing alleles recessive). When it is positive, the opposite holds, and trait-increasing alleles are recessive.

Dominance relationships between two alleles therefore emerge directly from their allelic value through the genotype-phenotype map. This contrasts models where dominance is specified independently of allelic effects, such as population genetic analyses where these quantities are treated as uncorrelated parameters [68] (e.g., in Appendix A where $\eta$ and $\delta$ are independent parameters), or models of dominance evolution (e.g., [226]) where an allele's dominance effect has a separate evolving basis to its allelic value.

## B.2 Invasion analysis

Under the life-cycle described in the main text and the above genetic effects, the invasion fitness of an allele with value $a_\bullet$ in a population otherwise fixed for another allele with value $a$, is given by

$$W(a_\bullet, a) = \frac{1}{2}\left(\frac{w_\mathrm{f}(z_\mathrm{f}(a_\bullet, a))}{w_\mathrm{f}(z_\mathrm{f}(a, a))} + \frac{w_\mathrm{m}(z_\mathrm{m}(a_\bullet, a))}{w_\mathrm{m}(z_\mathrm{m}(a, a))}\right). \tag{B-2}$$

This lays the basis of usual analysis of selection under the assumption of rare mutations with weak effects [80].

### B.2.1 Directional selection

The selection gradient, which determines the direction favoured by selection, is

$$S(a) = \left.\frac{\partial W(a_\bullet, a)}{\partial a_\bullet}\right|_{a_\bullet = a} = \frac{1}{2}\frac{S_\mathrm{f}(z_\mathrm{f}(a, a)) + S_\mathrm{m}(z_\mathrm{m}(a, a))}{2} \tag{B-3}$$

where $S_\mathrm{f}(z_\mathrm{f}(a, a))$ and $S_\mathrm{m}(z_\mathrm{m}(a, a))$ are given in eq. (7) in the main text. Equation (B-3) tells us that the genotype-phenotype maps as defined above do not affect evolutionary dynamics under directional selection (this is because alleles are additive to leading order here–recall eq. B-1e–as in [47]).

Accordingly, a singular allelic value $a^*$ is defined by

$$a^* \quad \text{such that} \quad S(a^*) = 0 \quad \Leftrightarrow \quad S_\mathrm{f}(z_\mathrm{f}(a^*, a^*)) = -S_\mathrm{m}(z_\mathrm{m}(a^*, a^*)), \tag{B-4}$$

and is locally attracting to evolutionary dynamics (i.e., convergence stable) when $\mathrm{d}S(a)/(\mathrm{d}a) < 0$ at $a = a^*$. Using the properties of sex-specific genotype-phenotype maps (eq. B-1), convergence stability requires

$$\left.\frac{\mathrm{d}S(a)}{\mathrm{d}a}\right|_{a=a^*} = \frac{H_\mathrm{f}(z_\mathrm{f}(a^*, a^*)) + H_\mathrm{m}(z_\mathrm{f}(a^*, a^*))}{4} - \frac{S_\mathrm{f}(z_\mathrm{f}(a^*, a^*))^2 + S_\mathrm{m}(z_\mathrm{f}(a^*, a^*))^2}{4} < 0, \tag{B-5}$$

or equivalently,

$$H_\mathrm{f}(z_\mathrm{f}(a^*, a^*)) + H_\mathrm{m}(z_\mathrm{m}(a^*, a^*)) < S_\mathrm{f}(z_\mathrm{f}(a^*, a^*))^2 + S_\mathrm{m}(z_\mathrm{m}(a^*, a^*))^2. \tag{B-6}$$

### B.2.2 Disruptive/stabilising selection

Once the population has reached a convergence stable $a^*$, it experience either stabilising selection when $H(a^*) < 0$ or disruptive selection when $H(a^*) > 0$ where $H(a^*) = \partial^2 W(a_\bullet, a)/(\partial {a_\bullet}^2)$ with $a_\bullet = a = a^*$. For our model, $H(a^*)$ can be written as

$$H(a^*) = \frac{1}{4}\left[\frac{H_\mathrm{f}(z_\mathrm{f}(a^*,a^*)) + H_\mathrm{m}(z_\mathrm{m}(a^*,a^*))}{2} + H_\mathrm{GP}(a^*)\right] \tag{B-7a}$$

where

$$H_\mathrm{GP}(a^*) = \left[S_\mathrm{f}(z_\mathrm{f}(a^*,a^*)) - S_\mathrm{m}(z_\mathrm{m}(a^*,a^*))\right]\left[\left.\frac{\partial^2 z_\mathrm{m}(a_1,a_2)}{\partial a_1 \partial a_2}\right|_{a_1=a_2=a^*} - \left.\frac{\partial^2 z_\mathrm{f}(a_1,a_2)}{\partial a_1 \partial a_2}\right|_{a_1=a_2=a^*}\right]. \tag{B-7b}$$

Eq. (B-7a) has two components. The first corresponds to the disruptive selection in the absence of sex-specific genotype-phenotype maps (same as eq. (11) in the main text). The second, $H_\mathrm{GP}(a^*)/4$, is the contribution of sex-specific genotype-phenotype maps through the cross-derivatives with respect to the maternally and paternally inherited allelic copies, which recall capture sex-specific dominance relationships between alleles (eq. B-1e).

Eq. (B-7) thus reveals two routes for disruptive selection. First, disruptive selection can be driven by positively curved fitness landscapes ($H_\mathrm{f}(a^*) + H_\mathrm{m}(a^*) > 0$), which is the only route without sex-specific genotype-phenotype maps (such that $H_\mathrm{GP}(a^*) = 0$). Second, it can arise from sex-specific genotype-phenotype maps when mutations are more dominant in the sex in which they are beneficial and more recessive in the sex in which they are deleterious, that is, under adaptive dominance reversal. To see this, suppose females are under positive selection and males under negative selection such that $S_\mathrm{f}(z_\mathrm{f}(a^*,a^*)) - S_\mathrm{m}(z_\mathrm{m}(a^*,a^*)) > 0$ holds. Then, $H_\mathrm{GP}(a^*)$ is positive when a mutation increasing trait value is more dominant in females than in males (i.e., when the cross-derivative of $z_\mathrm{f}(a_1,a_2)$ is smaller than the cross-derivative of $z_\mathrm{m}(a_1,a_2)$, see eq. (B-7b)). This corresponds to adaptive dominance reversal on phenotype among the sexes. Conversely, maladaptive dominance-reversal has the opposite effect and favours stabilising selection (i.e., makes $H_\mathrm{GP}(a^*) < 0$).

### B.2.3 Evolutionary branching

Combining the requirements for convergence to $a^*$ (eq. B-6) and disruptive selection (eq. (B-7a) positive), evolutionary branching and thus the emergence of polymorphism occurs when

$$-2H_\mathrm{GP}(a^*) \;<\; H_\mathrm{f}(z_\mathrm{f}(a^*,a^*)) + H_\mathrm{m}(z_\mathrm{m}(a^*,a^*)) \;<\; S_\mathrm{f}(z_\mathrm{f}(a^*,a^*))^2 + S_\mathrm{m}(z_\mathrm{m}(a^*,a^*))^2. \tag{B-8}$$

This shows that non-additive maps can alter the conditions for evolutionary branching only if they show sex-specificity (i.e., $H_{\mathrm{GP}}(a^*) \neq 0$, otherwise eq. B-8 collapses to main text eq. (14)). This widens or narrows the scope for branching according to whether these genotype-phenotype maps generate adaptive ($H_{\mathrm{GP}}(a^*) > 0$) or maladaptive ($H_{\mathrm{GP}}(a^*) < 0$) dominance reversal among the sexes.

Comparing eq. (B-8) with main text eq. (14) reveals that evolutionary branching on allelic value and thus evolutionary branching may derive from two pathways.

The first pathway is negative frequency-dependent disruptive selection [227] acting directly on phenotype through sex-specific phenotype–fitness maps (whenever condition (14) is satisfied). Evolutionary branching in this case would occur even if individuals were haploids. It is driven by the fitness consequences of competitive interactions within males and within females, favouring morphs specialising toward male and female functions. To see how this arises under sexual antagonism, consider a rare mutant allele that shifts trait expression closer to the male optimum and further from the female optimum. When rare, its carriers compete against residents expressing $a^*$. If $0 < H_{\mathrm{f}}(z_{\mathrm{f}}(a^*, a^*)) + H_{\mathrm{m}}(z_{\mathrm{m}}(a^*, a^*))$, this deviation from $a^*$ yields a net fitness gain: the benefit in males of moving toward their optimum outweighs the cost in females of moving away from theirs, owing to the curvature of the phenotype–fitness map. However, as the mutant increases in frequency, the average population trait value shifts toward the male optimum. As a result, the marginal benefit in males declines, and under sufficiently strong sex-specific selection (i.e., when $H_{\mathrm{f}}(z_{\mathrm{f}}(a^*, a^*)) + H_{\mathrm{m}}(z_{\mathrm{m}}(a^*, a^*)) < S_{\mathrm{f}}(z_{\mathrm{f}}(a^*, a^*))^2 + S_{\mathrm{m}}(z_{\mathrm{m}}(a^*, a^*))^2$ with $S_{\mathrm{f}}(z_{\mathrm{f}}(a^*, a^*)) = -S_{\mathrm{m}}(z_{\mathrm{m}}(a^*, a^*))$), the net fitness gain becomes negative, preventing fixation. Thus the mutant is favoured when rare but not when common, generating negative frequency dependence. Because the same reasoning applies symmetrically to mutations on either side of $a^*$, selection is also disruptive and favours divergence into male- and female-specialised alleles.

The second pathway is negative frequency-dependent disruptive selection acting on allelic value through sex-specific genotype–phenotype maps. Here, disruptive selection is generated by interactions between allelic copies within individuals, as captured by $H_{\mathrm{GP}}(a^*)$. Evolutionary branching can therefore occur even when selection on the phenotype itself is stabilising (i.e., when $H_{\mathrm{f}}(z_{\mathrm{f}}(a^*, a^*)) + H_{\mathrm{m}}(z_{\mathrm{m}}(a^*, a^*)) < 0$). To see how this arises, consider again a rare mutant allele that shifts trait expression closer to the male optimum and further from the female's. When rare, it is mostly found in heterozygotes. If the genotype–phenotype map generates adaptive dominance reversal among the sexes, the fitness gain in males outweighs the loss in females and the mutant can invade; this can occur even when phenotype–fitness maps are linear (i.e., when $H_{\mathrm{f}}(z_{\mathrm{f}}(a^*, a^*)) = H_{\mathrm{m}}(z_{\mathrm{m}}(a^*, a^*)) = 0$). As the allele increases in frequency, it is more often found in homozygotes, in which both allelic copies contribute equally to phenotype, so its deleterious effect in females become more apparent. Its advan-

tage therefore declines with frequency, generating negative frequency dependence at the allelic level. In the context of sexual antagonism, this mechanism substantially expands the scope for the gradual evolution of polymorphism, because it does not require strong disruptive selection on the phenotype itself [18, 47]. In particular, eq. B-8 can be satisfied even when $H_{\mathrm{m}}(a^*) + H_{\mathrm{f}}(a^*)$ is small or negative, relaxing the requirement that $S_{\mathrm{f}}(a^*)^2 + S_{\mathrm{m}}(a^*)^2$ be relatively large.

# C Evolution of continuous traits under sexually antagonistic selection: connections and extensions to complex traits

In this Appendix, we detail the alignment between the responses to sex-specific selection predicted by each theoretical approach (Appendix C.1) and discuss how these connect with results from individual-based simulations of complex traits (Appendix C.2).

## C.1 Predictions of all theoretical approaches converge under weak gene action

Reassuringly, population genetics, adaptive dynamics, and quantitative genetic approaches provide a coherent description of evolution under sexually antagonistic selection when alleles have weak effects on phenotype. This is easiest to demonstrate when allelic effects are also additive and fully sex-concordant (i.e., when $\sigma_{\mathrm{fm}} = \sigma_{\mathrm{f}}^2 = \sigma_{\mathrm{m}}^2$ in eq. 15 for quantitative genetics). Under this assumption, initial evolutionary change in all three approaches is determined by the selection gradient $S(z)$ (eq. 7). This gradient governs the behaviour of allele frequency $\Delta p$ (eq. 6), the sign of invasion fitness around one $W(z_\bullet, z) - 1$ (eq. 8), and the responses of mean trait values $\Delta\overline{z}_{\mathrm{f}} = \Delta\overline{z}_{\mathrm{m}}$ (eq. 15). There is therefore agreement that selection is initially directional, favouring either a net increase or decrease in trait value $z$. The population thus progressively evolves in this direction. All models predict that directional selection reduces genetic variation during this evolutionary phase (e.g., $\Delta p \neq 0$ until $p = 1$ or $p = 0$ in eq. 6, and $\Delta\sigma_{\mathrm{f}}^2$ and $\Delta\sigma_{\mathrm{m}}^2$ decrease with $S_{\mathrm{f}}(z)$ and $S_{\mathrm{m}}(z)$ in eq. 16). However, multilocus population genetic models suggest that when traits show a highly polygenic genetic architecture (as in quantitative genetics models), the effects of selection on genetic variance are often dominated by those of segregation, recombination, and mutation and so may be too weak to be perceptible [45, 94, 95, 97, 98].

In any case, without sex-specific effects in gene action, all three approaches show that the population is expected to converge to a singular compromise trait value $z^*$ at which $S(z^*) = 0$. Once the population is close to $z^*$, selection is determined by $H(z^*)$, which measures the sex-average curvature of the fitness landscape (eq. 12; in the population-genetic recursion this appears by retaining terms of order $\epsilon^2$ in $\Delta p$, see eq. A-5). All three approaches therefore show that sexual antagonism promotes an increase in genetic variation when $H(z^*) > 0$ (corresponding to disruptive selection), and a decrease when $H(z^*) < 0$ (corresponding to stabilising selection) (see eq. A-5 for population genetics, and eq. 16 for quantitative genetics–though again the effects of selection on genetic variation may be undetectable for very highly polygenic traits [98, 228]).

The consistency in outcomes amongst approaches is still visible when assumptions of additivity and

sex-concordance allelic effects are relaxed. Population genetic and adaptive dynamics results have revealed that the above description of evolutionary dynamics also applies to traits encoded by alleles with non-additive – although still sex-concordant – effects. That is, allele frequency change (see eq. A-3) and the relevant components of invasion fitness (eqs. B-3-B-5) are determined by the linear and quadratic selection gradients within sexes in the same way as for the additive case (quantitative genetic models for such non-additive traits evolving under sex-specific selection have yet to be developed but this suggests they would predict the same evolutionary trajectory as under additivity).

Relaxing the assumption that allelic effects (i.e., homozygous phenotypic effects) are sex-concordant, all modelling approaches agree that female $z_f$ and male $z_m$ trait values experiencing opposing selection will diverge (e.g. when allowing $\sigma_{fm} \neq \sigma_f^2 \neq \sigma_m^2$ in eq. 15 for quantitive genetics). Here, directional selection in each sex, captured by the male and female linear gradients $S_f(z_f)$ and $S_m(z_m)$, favours the fixation of alleles with adaptive sex-specific effects until females and males reach their respective optima $z_f^*$ and $z_m^*$ such that $S_f(z_f^*) = S_m(z_m^*) = 0$, with genetic variation diminished throughout this process (this is predicted by population genetic eqs. 15-16; quantitative genetic eq. A-3 with $\delta_f \neq \delta_m$ here; by adaptive dynamics eqs. 5-6 in Van Dooren et al. [46]).

Consequently, there is a consensus that when mutations have weak effects and alleles show similar dominance across the sexes, sexual antagonism can only promote genetic variation if it generates disruptive selection on trait values. This requires strong correlations in male and female allelic values, such that the population behaves effectively as evolving a shared trait and is driven toward a compromise value, which must also be a local fitness minimum. Condition (14) provides a common criterion for this to occur when these genetic correlations approach 1.

## C.2 Theory is informative for polygenic responses to sexual antagonism across a broad range of genetic architectures

Simulations have shown that the evolutionary dynamics of complex traits (i.e., those underlaid by multiple, but not infinite, loci) are extremely well-described by theory assuming much simpler architectures, at least when allelic effects are additive or show sex-concordant phenotypic dominance [12, 18, 45, 102]. The correspondence between theoretical predictions and the dynamics of polygenic traits is less clear when phenotypic dominance is sex-specific. In this section, we explore this case with individual based simulations. We simulate the evolution of a polygenic trait under sex-specific selection, allowing alleles to differ in their homozygous effects and in their dominance relationships between the sexes. We compare these simulation outcomes to the predictions of the theoretical ap-

proaches reviewed above.

### C.2.1 A simulation model

We consider a large, density-regulated population with discrete generations that follows the same life cycle as in the main text (i.e. Wright Fisher process; section 2.1 in main text). The evolving trait is encoded by $L$ loci at which multiple alleles may segregate. Each allele $k$ at locus $\ell \in \{1,\dots,L\}$ is characterised by four parameters, corresponding to its effects in males and females. Specifically, in each sex $u \in \{\mathrm{m},\mathrm{f}\}$, the allele has (i) an allelic value $a^u_{k,\ell} \in \mathbb{R}$, defined as its contribution to the trait when homozygous, and (ii) a dominance level $b^u_{k,\ell} \in \mathbb{R}^*_+$, which determines its contribution in heterozygotes. The trait value $z_u$ expressed by an individual of sex $u \in \{\mathrm{m},\mathrm{f}\}$ is then assumed to be given by

$$z_u = \sum_{\ell=1}^{L} a^u_{1,\ell} \frac{b^u_{1,\ell}}{b^u_{1,\ell} + b^u_{2,\ell}} + a^u_{2,\ell} \frac{b^u_{2,\ell}}{b^u_{1,\ell} + b^u_{2,\ell}}. \tag{C-1}$$

Here, alleles at a locus combine additively within the locus if they have the same dominance level, i.e., $b^u_{1,\ell} = b^u_{2,\ell}$. Otherwise, the allele with the highest dominance level contributes more to trait value as per the "affinity" model of Van Dooren [226].

With probabilities $\mu_a$ and $\mu_b$, mutations affect the allelic value and dominance level of each allele, respectively. When a mutation affects allelic value, the sex-specific effects of the mutated copy are drawn from a bivariate Gaussian distribution centred on ancestral values with variance-covariance matrix $\mathbf{\Sigma}_a$ with a small variance. This corresponds to a continuum-of-alleles model [86]. By contrast, mutations affecting dominance level are drawn from a distribution with variance-covariance matrix $\mathbf{\Sigma}_b$ centred on $(1,1)$ with a larger variance, corresponding to house-of-cards model [229] (the distribution is truncated to only allow positive values). This choice generates larger mutational variation in dominance than in allelic value, allowing deviations from additivity to arise readily.

The off-diagonal elements of matrices $\mathbf{\Sigma}_a$ and $\mathbf{\Sigma}_b$ are determined by the cross-sex correlations of mutational effects, $\rho_a$ and $\rho_b$, respectively, which quantify the extent to which mutations are sex-specific. For example, when $\rho_a = 1$, mutations in allelic value have identical effects in both sexes whereas when $\rho_a = 0$, their effects are uncorrelated.

During meiosis, recombination occurs between adjacent loci at rate $r = 0.1$. Although not shown, we ran simulations with varying values for $r$ and found no qualitative effect of recombination rate unless $r$ was extremely small, in which case linked loci behaved effectively as a single locus, consistent with previous simulations (without dominance reversal for phenotype, [18, 104]).

### C.2.2 Sex-specific dominance can generate transient polymorphism in polygenic evolution

In agreement with previous simulation work with sex-concordant dominance [12, 102],we find that when mutations can generate sex-specific allelic values ($\rho_a < 1$), male and female trait means eventually approach their respective optima under directional selection within each sex (Fig. A1, top row). This is consistent with predictions from population genetic, adaptive dynamics, and quantitative genetic models [4, 5, 46]. When mutational effects on allelic value are weakly correlated between the sexes (low $\rho_a$), this divergence occurs rapidly and with little genetic variation, regardless of the sex-specificity of dominance mutations (i.e., regardless of $\rho_b$, Fig. A1 left-hand column).

When mutational effects on allelic value are strongly correlated between the sexes (high $\rho_a$) divergence toward the sex-specific optima is slower (Fig. A1 middle column; [12, 13, 55]). If $\rho_a$ is close to one, the population first approaches the compromise trait value $z^*$ for a shared trait (eq. 13), before male and female trait values eventually diverge as sex-specific allelic effects accumulate (Fig. A1, middle and right-hand columns). The amount of genetic variation observed as this unfolds depends primarily on the correlation between new mutations' dominance levels in males and females ($\rho_b$). When $\rho_b$ is high, sex-specific dominance reversal is rare and genetic variation remains low. When $\rho_b$ is low, dominance reversals arise more readily and can generate a transient increase in genetic variation.

The increase in genetic variance owing to dominance reversals occurs mainly once the population is close to $z^*$ (Fig. A1 middle column). Prior to this, selection favours alleles that move both sexes toward the compromise phenotype (captured by $S(z)$, eq. 7). This directional selection overwhelms any advantage provided by dominance-reversals, preventing balanced polymorphisms. Near $z^*$, directional selection on alleles with sex-concordant effects is weak, so sex-specific dominance reversal can maintain balanced polymorphisms. However these polymorphisms are transient. This is because they are eventually displaced by alleles with sex-specific homozygous effects that move male and female trait toward their separate optima (Fig. A1 middle column).

When sex-specific allelic effects are especially strongly constrained, balanced polymorphisms can arise even when dominance effects are also highly correlated among the sexes (e.g., $\rho_a = \rho_b = 0.99$ Fig. A1 right-hand panels). This is likely because the population remains close to $z^*$ for long enough for dominance-reversed alleles to arise, a possibility facilitated by modelling dominance mutations with a house-of-cards process and allelic-value mutations with a continuum-of-alleles process. In this case, sex-specific dominance can generate adaptive divergence in male and female trait values (compare $\rho_b = 0, \rho_b = 0.99$, and $\rho_b = 1$ in Fig. A1 right-hand panel). However, simulations in which sex-specific dominance arises readily show the slowest final approach to the sex-specific optima (Fig. A1 right-hand

panel). This suggests that established dominance-reversed polymorphisms can inhibit the invasion of alleles with adaptive sex-specific homozygous effects, thereby prolonging sexual antagonism.

Overall, these findings suggest that sex-specific dominance reversal can maintain elevated genetic variation at loci underlying a complex trait, but mainly under restrictive conditions. Mutational effects on allelic value must be strongly correlated between males and females, so that sex-specific allelic effects arise slowly, while mutations affecting dominance must be sufficiently sex-specific and large to generate adaptive dominance reversal. Even then, the resulting polymorphisms are ultimately transient, because they are eventually displaced by alleles with sex-specific homozygous effects that move males and females toward their respective optima.

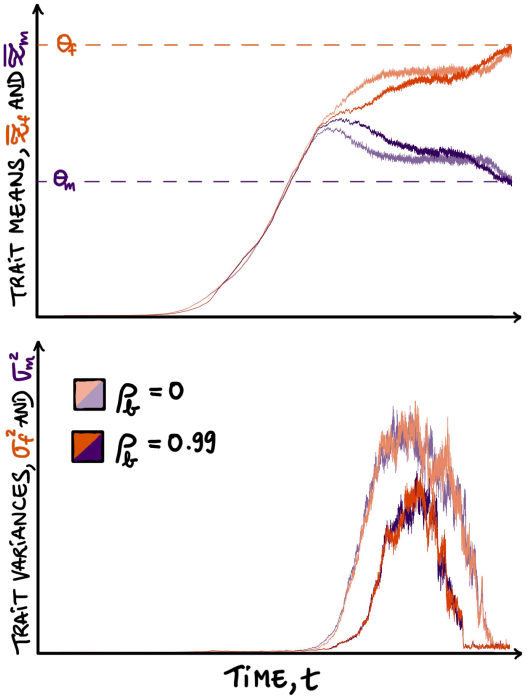


**Figure A1:** Effect of dominance evolution on the divergence of male and female trait values under sexually antagonistic selection, when mutations almost always have sex-concordant effects on allelic values ($\rho_a = 0.99$). The top panel shows mean male (purple) and female (orange) trait values as a function of time (on a log-scale). Lighter shades show the case where the dominance of mutations is independent in males and females ($\rho_b = 0$), and darker shades show the case where dominance is as strongly constrained as allelic values ($\rho_b = \rho_a = 0.99$). The bottom panel shows how sex-specific variances in trait value change over the same time-frame.

# D Sexual antagonism on multiple traits

## D.1 Adaptive dynamics of two traits encoded by a pleiotropic locus

In this section, we investigate the joint evolution of two traits $z_1 \in \mathbb{R}$ and $z_2 \in \mathbb{R}$ under sex-specific selection using adaptive dynamics (see [143, 230] for more general considerations on adaptive dynamics in multiple dimensions). The two traits affect individual fitness in a sex-specific way, with the fitness of females and males expressing trait values $\boldsymbol{z} = (z_1, z_2)$ given by $w_\mathrm{f}(\boldsymbol{z})$ and $w_\mathrm{m}(\boldsymbol{z})$. They are encoded by a single pleiotropic locus with additive effects, so that the the invasion fitness of a rare mutant allele $\boldsymbol{z}_\bullet = (z_{1,\bullet}, z_{2,\bullet})$ arising as a single copy in a resident population otherwise fixed for allele $\boldsymbol{z} = (z_1, z_2)$ is given by

$$W(\boldsymbol{z}_\bullet, \boldsymbol{z}) = \frac{1}{2}\left( \frac{w_\mathrm{f}\left(\frac{\boldsymbol{z}_\bullet + \boldsymbol{z}}{2}\right)}{w_\mathrm{f}(\boldsymbol{z})} + \frac{w_\mathrm{m}\left(\frac{\boldsymbol{z}_\bullet + \boldsymbol{z}}{2}\right)}{w_\mathrm{m}(\boldsymbol{z})} \right). \tag{D-1}$$

Equation (D-1) is the two-trait analogue of eq. (8) in the main text.

Similar to the single trait case, the evolutionary dynamics of the population can be decomposed in two phases. First, it evolves under directional selection while remaining largely monomorphic. The direction of selection during this phase is given by the two-dimensional selection gradient $\mathbf{S}(\boldsymbol{z}) = \big(S_1(\boldsymbol{z}), S_2(\boldsymbol{z})\big)$, the $i^{\text{th}}$ element of which is the linear selection gradient on each trait, obtained as

$$S_i(\boldsymbol{z}) = \left.\frac{\partial W(\boldsymbol{z}_\bullet, \boldsymbol{z})}{\partial z_{i,\bullet}}\right|_{\boldsymbol{z}_\bullet = \boldsymbol{z}} = \frac{S_{\mathrm{f},i}(\boldsymbol{z}) + S_{\mathrm{m},i}(\boldsymbol{z})}{4} \tag{D-2a}$$

for trait $i$, where

$$S_{u,i}(\boldsymbol{z}) = \frac{1}{w_u(\boldsymbol{z})}\frac{\partial w_u(\boldsymbol{z})}{\partial z_i} \tag{D-2b}$$

denotes the linear selection gradient on trait $i$ in sex $u \in \{\mathrm{f}, \mathrm{m}\}$.

Through the recurrent fixation of mutations taking the population in the direction given by $\mathbf{S}(\boldsymbol{z})$, the population may eventually reach a singular phenotype $\boldsymbol{z}^*$ at which directional selection vanishes on both traits, i.e.,

$$\boldsymbol{z}^* \quad \text{such that} \quad \mathbf{S}(\boldsymbol{z}^*) = (0, 0). \tag{D-3}$$

Convergence to such a phenotype requires that all eigenvalues of the Jacobian matrix $\mathbf{J}(\boldsymbol{z}^*)$, the $(p, q)$-entry of which is given by

$$J_{pq}(\boldsymbol{z}^*) = \left.\frac{\partial S_p(\boldsymbol{z}^*)}{\partial z_q}\right|_{\boldsymbol{z}=\boldsymbol{z}^*} = \frac{1}{2}\left[ \frac{H_{\mathrm{f},pq}(\boldsymbol{z}^*) + H_{\mathrm{m},pq}(\boldsymbol{z}^*)}{2} - \frac{S_{\mathrm{f},p}(\boldsymbol{z}^*)S_{\mathrm{f},q}(\boldsymbol{z}^*) + S_{\mathrm{m},p}(\boldsymbol{z}^*)S_{\mathrm{m},q}(\boldsymbol{z}^*)}{2} \right], \tag{D-4}$$

have negative real parts, where

$$H_{u,pq}(\boldsymbol{z}^*) = \frac{1}{w_u(\boldsymbol{z}^*)} \left. \frac{\partial^2 w_u(\boldsymbol{z})}{\partial z_p \partial z_q} \right|_{\boldsymbol{z}=\boldsymbol{z}^*}, \tag{D-5}$$

with $p, q \in \{1,2\}$, capture second-order effects of selection. For $p = q$, $H_{u,pp}(\boldsymbol{z}^*) > 0$ (resp. $H_{u,pp}(\boldsymbol{z}^*) < 0$) indicates that fitness accelerates (resp. decelerates) with a change in trait $p$ in sex $u$, i.e. it is informative on whether selection on trait $p$ via sex $u$ is disruptive or stabilising. For $p \neq q$, $H_{u,pq}(\boldsymbol{z}^*)$ gives the correlational selection coefficient between traits $p$ and $q$ in sex $u$; when $H_{u,pq}(\boldsymbol{z}^*) > 0$ (resp. $H_{u,pq}(\boldsymbol{z}^*) < 0$), selection favours a positive (resp. negative) correlation between the two traits in sex $u$, i.e., because a joint change in both traits has a positively (resp. negatively) synergistic effect on fitness.

Once the population has converged to a singular phenotype $\boldsymbol{z}^*$, selection may either be stabilising around $\boldsymbol{z}^*$ or disruptive, favouring the emergence of polymorphism. Which of these two outcomes unfolds depends on the Hessian matrix $\mathbf{H}(\boldsymbol{z}^*)$, the $(p,q)$-entry of which $H_{pq}(\boldsymbol{z}^*)$ is given by

$$H_{pq}(\boldsymbol{z}^*) = \left. \frac{\partial^2 W(\boldsymbol{z}_\bullet, \boldsymbol{z})}{\partial z_{p,\bullet} \partial z_{q,\bullet}} \right|_{\boldsymbol{z}_\bullet = \boldsymbol{z} = \boldsymbol{z}^*} = \frac{H_{\mathrm{f},pq}(\boldsymbol{z}^*) + H_{\mathrm{m},pq}(\boldsymbol{z}^*)}{8}, \tag{D-6}$$

with disruptive selection requiring that the leading eigenvalue of $\mathbf{H}(\boldsymbol{z}^*)$ is positive. There are two ways for this to occur. The first is for one of the diagonal elements of $\mathbf{H}(\boldsymbol{z}^*)$, $H_{11}(\boldsymbol{z}^*)$ and $H_{22}(\boldsymbol{z}^*)$ – the quadratic selection gradients on each trait – to be positive. In this case selection favours evolutionary branching for that trait as in the single-trait case, which may in turn favour branching in the other trait if selection favours a correlation between the two ($H_{12}(\boldsymbol{z}^*) \neq 0$). Alternatively, when quadratic selection is stabilising on each trait individually ($H_{11}(\boldsymbol{z}^*) < 0$ and $H_{22}(\boldsymbol{z}^*) < 0$), such that they would remain monomorphic when evolving in isolation, evolutionary branching may yet be favoured if correlational selection between the two traits is sufficiently strong to overcome stabilising selection acting on each trait individually, which occurs when

$$H_{12}(\boldsymbol{z}^*)^2 > H_{11}(\boldsymbol{z}^*) H_{22}(\boldsymbol{z}^*). \tag{D-7}$$

The joint evolution of several traits thus creates additional routes for polymorphism to emerge, i.e., through correlational selection [144]. Condition (D-7) is satisfied most readily when correlational selection acts in the same direction in both sexes (i.e., when $H_{\mathrm{m},pq}(\boldsymbol{z}^*)$ and $H_{\mathrm{f},pq}(\boldsymbol{z}^*)$ are the same sign).

With two sexually antagonistic traits, correlational selection can thus drive evolutionary branching when, in both sexes, the fitness benefit of expressing the sex-beneficial value of one trait is increased by also expressing the sex-beneficial value of the other (positive fitness synergy between traits: $H_{\mathrm{m},pq}(\boldsymbol{z}^*) > 0$ and $H_{\mathrm{f},pq}(\boldsymbol{z}^*) > 0$). A possible example is sex-specific resource use, where successful

exploitation of each resource requires a coordinated combination of morphological and physiological traits. If the male-beneficial resource is best exploited by high values of both traits, whereas the female-beneficial resource is best exploited by low values of both traits, selection favours a positive association between the traits within each sex. Alleles producing the male-beneficial trait combination will then tend to be costly in females, whereas alleles producing the female-beneficial combination will tend to be costly in males.

## D.2 Population-genetic consequences of selection on multiple traits

In this section, we connect the selection terms that emerged in previous section (eqs. D-2b and D-5) with selection coefficients that are typically used in population genetics modelling. This will help us link sexual antagonism over multiple traits with notions of fitness dominance and fitness epistasis.

### D.2.1 The effect of pleiotropy on patterns of sex-specific dominance

To connect with dominance, let us first consider a single pleiotropic locus that affects both traits simultaneously and in the same way. We assume that two alleles exist at this locus, a and A, that encode phenotypes $\boldsymbol{z}_{\mathrm{a}} = (z_1, z_2)$ and $\boldsymbol{z}_{\mathrm{A}} = (z_1 + \delta, z_2 + \delta)$, respectively, where $\delta > 0$ is a positive constant. Throughout, we assume that larger trait values are female-beneficial and smaller trait values male-beneficial, so that allele a is favoured in males and A in females. Following the convention established by Kidwell et al. [15], relative genotypic fitnesses in each sex can be written in terms of sex-specific selection and dominance coefficients of the least-preferred allele in each sex, $s_u$ and $h_u$, $u \in \{\mathrm{m}, \mathrm{f}\}$ as laid out in Table 2.

| | aa | Aa | AA |
|---|---|---|---|
| Relative female fitness | $1 - s_{\mathrm{f}}$ | $1 - h_{\mathrm{f}} s_{\mathrm{f}}$ | 1 |
| Relative female fitness | 1 | $1 - h_{\mathrm{m}} s_{\mathrm{m}}$ | $1 - s_{\mathrm{m}}$ |

**Table 2:** Genotypic relative fitnesses in the notation of Kidwell et al. [15].

The selection coefficients against allele a in females and allele A in males, $s_{\mathrm{f}}$ and $s_{\mathrm{m}}$, can therefore respectively be expressed as

$$s_{\mathrm{f}} = \frac{w_{\mathrm{f}}(\boldsymbol{z}_{\mathrm{A}}) - w_{\mathrm{f}}(\boldsymbol{z}_{\mathrm{a}})}{w_{\mathrm{f}}(\boldsymbol{z}_{\mathrm{A}})} \quad \text{and} \quad s_{\mathrm{m}} = \frac{w_{\mathrm{m}}(\boldsymbol{z}_{\mathrm{a}}) - w_{\mathrm{f}}(\boldsymbol{z}_{\mathrm{A}})}{w_{\mathrm{f}}(\boldsymbol{z})}, \tag{D-8a}$$

which to leading order in $\delta$ yields

$$s_{\mathrm{f}} = \delta\big(S_{\mathrm{f},1}(z_{\mathrm{a}}) + S_{\mathrm{f},2}(z_{\mathrm{a}})\big) + \mathscr{O}\left(\delta^2\right) \quad \text{and } s_{\mathrm{m}} = -\delta\big(S_{\mathrm{m},1}(z_{\mathrm{a}}) + S_{\mathrm{m},2}(z_{\mathrm{a}})\big) + \mathscr{O}\left(\delta^2\right), \tag{D-8b}$$

where $S_{u,i}(z)$ is defined in eq. (D-2b). Note that under the Kidwell et al. [15] notational convention, selection coefficients $s_u$, $u \in \{\mathrm{m},\mathrm{f}\}$ are positive because they quantify the strength of selection against the least-preferred allele in each sex.

Meanwhile, sex-specific dominance coefficients $h_{\mathrm{m}}$ and $h_{\mathrm{f}}$ can be obtained using Table 2 as

$$h_{\mathrm{f}} = \frac{w_{\mathrm{f}}(z_{\mathrm{A}}) - w_{\mathrm{f}}(\frac{1}{2}z_{\mathrm{A}} + \frac{1}{2}z_{\mathrm{a}})}{w_{\mathrm{f}}(z_{\mathrm{A}}) - w_{\mathrm{f}}(z_{\mathrm{a}})} \quad \text{and} \quad h_{\mathrm{m}} = \frac{w_{\mathrm{m}}(z_{\mathrm{a}}) - w_{\mathrm{m}}(\frac{1}{2}z_{\mathrm{A}} + \frac{1}{2}z_{\mathrm{a}})}{w_{\mathrm{m}}(z_{\mathrm{a}}) - w_{\mathrm{m}}(z_{\mathrm{A}})}, \tag{D-9a}$$

which yields

$$h_{\mathrm{f}} = \frac{1}{2}\left[1 + \frac{\delta}{4}\frac{H_{\mathrm{f},11}(z_{\mathrm{a}}) + 2H_{\mathrm{f},12}(z_{\mathrm{a}}) + H_{\mathrm{f},22}(z_{\mathrm{a}})}{S_{\mathrm{f},1}(z_{\mathrm{a}}) + S_{\mathrm{f},2}(z_{\mathrm{a}})}\right] + \mathscr{O}\left(\delta^2\right) \tag{D-9b}$$

in females and

$$h_{\mathrm{m}} = \frac{1}{2}\left[1 + \frac{\delta}{4}\frac{H_{\mathrm{m},11}(z_{\mathrm{a}}) + 2H_{\mathrm{m},12}(z_{\mathrm{a}}) + H_{\mathrm{m},22}(z_{\mathrm{a}})}{-\big(S_{\mathrm{m},1}(z_{\mathrm{a}}) + S_{\mathrm{m},2}(z_{\mathrm{a}})\big)}\right] + \mathscr{O}\left(\delta^2\right) \tag{D-9c}$$

in males, where the $H_{u,pq}(z_{\mathrm{a}})$ coefficients ($p, q \in \{1,2\}$) capture second-order effects of selection (eq. D-5). Because $S_{\mathrm{f},1}(z_{\mathrm{a}}) + S_{\mathrm{f},2}(z_{\mathrm{a}}) > 0$ and $-\big(S_{\mathrm{m},1}(z_{\mathrm{a}}) + S_{\mathrm{m},2}(z_{\mathrm{a}})\big) > 0$ must hold as per eq. (D-8b), the effect of fitness interactions between the two traits are revealed by the sign of the $H_{u,12}(z_{\mathrm{a}})$ coefficients, which give the strength and direction of correlational selection in each sex. From these, it is clear that negative correlational selection between $z_1$ and $z_2$ in sex $u$ ($H_{u,12}(z_{\mathrm{a}}) < 0$) tends to make the least-preferred allele more recessive in that sex. Intuitively, this is because when $H_{u,12}(z_{\mathrm{a}}) < 0$, increasing $z_1$ reduces the fitness effect of increasing $z_2$ (and vice-versa), causing fitness effects to saturate with allelic value and so resulting in a lower $h_u$.

### D.2.2 Fitness interactions between loci affecting separate sexually antagonistic traits

To connect with notions of epistasis, we now consider the case where each trait is affected by a separate di-allelic locus. The first locus, which we refer to as locus A, is assumed to have alleles a and A that affect trait $z_1$ with allelic values $z_1$ and $z_1 + \delta$, respectively. The second locus, which we call locus B, has alleles b and B affecting trait $z_2$, with allelic values $z_2$ and $z_2 + \delta$.

To compute the effect of fitness interactions between the two loci, we assume that they affect sex-specific fitness multiplicatively with an additional additive-by-additive epistasis term between least-preferred alleles at each locus, as shown in Table 3. Using this notation, the selection coefficients

against the least-preferred allele at each locus can be expressed to leading order in $\delta$ as

$$s_{\mathrm{f}}^{\mathrm{A}} = \delta S_{\mathrm{f},1}(\boldsymbol{z}) + \mathscr{O}\left(\delta^2\right) \quad \text{and} \quad s_{\mathrm{f}}^{\mathrm{B}} = \delta S_{\mathrm{f},2}(\boldsymbol{z}) + \mathscr{O}\left(\delta^2\right) \tag{D-10a}$$

in females, and

$$s_{\mathrm{m}}^{\mathrm{A}} = -\delta S_{\mathrm{m},1}(\boldsymbol{z}) + \mathscr{O}\left(\delta^2\right) \quad \text{and} \quad s_{\mathrm{m}}^{\mathrm{B}} = -\delta S_{\mathrm{m},2}(\boldsymbol{z}) + \mathscr{O}\left(\delta^2\right) \tag{D-10b}$$

in males, where $S_{u,i}(\boldsymbol{z})$ is defined in eq. D-2b. The dominance coefficients of these alleles, meanwhile, are given by

$$h_{\mathrm{f}}^{\mathrm{A}} = \frac{1}{2}\left(1 + \frac{\delta}{4}\frac{H_{\mathrm{f},11}(\boldsymbol{z})}{S_{\mathrm{f},1}(\boldsymbol{z})}\right) + \mathscr{O}\left(\delta^2\right) \quad \text{and} \quad h_{\mathrm{f}}^{\mathrm{B}} = \frac{1}{2}\left(1 + \frac{\delta}{4}\frac{H_{\mathrm{f},22}(\boldsymbol{z})}{S_{\mathrm{f},2}(\boldsymbol{z})}\right) + \mathscr{O}\left(\delta^2\right) \tag{D-10c}$$

in females and

$$h_{\mathrm{m}}^{\mathrm{A}} = \frac{1}{2}\left(1 + \frac{\delta}{4}\frac{H_{\mathrm{m},11}(\boldsymbol{z})}{-\left(S_{\mathrm{m},1}(\boldsymbol{z})\right)}\right) + \mathscr{O}\left(\delta^2\right) \quad \text{and} \quad h_{\mathrm{m}}^{\mathrm{B}} = \frac{1}{2}\left(1 + \frac{\delta}{4}\frac{H_{\mathrm{m},22}(\boldsymbol{z})}{-\left(S_{\mathrm{m},2}(\boldsymbol{z})\right)}\right) + \mathscr{O}\left(\delta^2\right) \tag{D-10d}$$

in males, where $H_{u,pq}$ is defined in eq. (D-5). Note that since owing to the definitions given in Table 3, the sex-specific selection coefficients at each locus must be positive, i.e., $S_{\mathrm{f},i}(\boldsymbol{z}) > 0$ and $S_{\mathrm{m},i}(\boldsymbol{z}) < 0$ for each trait. As a result, whether the least-preferred allele is dominant or recessive at each locus and in each sex is entirely determined by the quadratic selection gradient on the corresponding trait (eq. D-5).

Sex-specific epistasis coefficients, $\varepsilon_u$, $u \in \{\mathrm{m},\mathrm{f}\}$ in Table 3, are computed from sex-specific fitnesses $w_{\mathrm{f}}(z_1, z_2)$ and $w_{\mathrm{m}}(z_1, z_2)$ from :

$$\varepsilon_{\mathrm{f}} = \frac{w_{\mathrm{f}}\left(z_1 + \frac{\delta}{2}, z_2 + \frac{\delta}{2}\right) w_{\mathrm{f}}\left(z_1 + \delta, z_2 + \delta\right)}{w_{\mathrm{f}}\left(z_1 + \frac{\delta}{2}, z_2 + \delta\right) w_{\mathrm{f}}\left(z_1 + \delta, z_2 + \frac{\delta}{2}\right)} - 1 = \frac{\delta^2}{4}\left[H_{\mathrm{f},12}(\boldsymbol{z}) - S_{\mathrm{f},1}(\boldsymbol{z})S_{\mathrm{f},2}(\boldsymbol{z})\right] + \mathscr{O}\left(\delta^3\right) \tag{D-11a}$$

in females and

$$\varepsilon_{\mathrm{m}} = \frac{w_{\mathrm{m}}\left(z_1 + \frac{\delta}{2}, z_2 + \frac{\delta}{2}\right) w_{\mathrm{m}}\left(z_1, z_2\right)}{w_{\mathrm{m}}\left(z_1 + \frac{\delta}{2}, z_2\right) w_{\mathrm{m}}\left(z_1, z_2 + \frac{\delta}{2}\right)} - 1 = \frac{\delta^2}{4}\left[H_{\mathrm{m},12}(\boldsymbol{z}) - S_{\mathrm{m},1}(\boldsymbol{z})S_{\mathrm{m},2}(\boldsymbol{z})\right] + \mathscr{O}\left(\delta^3\right) \tag{D-11b}$$

in males. Eq. (D-11) shows that directional selection in each sex, as captured by linear gradients $S_{u,i}(\boldsymbol{z})$ generates negative epistasis between the two loci. This is because the effect of the loci on genotypic fitnesses are defined on a multiplicative scale, such that increasing both traits by the same amount produces a less than linear increase in log-fitness. Correlational selection also affects epistatic interactions between loci, either making them more negative or more positive depending on the sign of correlational selection gradients $H_{u,12}(\boldsymbol{z})$. In particular, negative correlational selection ($H_{u,12}(\boldsymbol{z}) < 0$) makes epistasis more negative. Intuitively, this is because any change in one trait in a given direction

reduces the fitness effect of a change in the other trait in the same direction when $H_{u,12}(\boldsymbol{z}) < 0$, which leads to diminishing returns to joint trait changes. This reduces the genotypic fitness of combinations of alleles changing the two traits in the same direction relative to other combinations, which amounts to negative epistasis. A symmetrical reasoning holds for positive correlational selection.

### D.2.3 A note on two-locus population genetic models of sexual antagonism

The results obtained in this section help contextualise two-locus population genetics models analysed in the literature.

The first such model was analysed by Patten et al. [39] and Úbeda et al. [40], who considered two sexually antagonistic loci with multiplicative effects on fitness and no epistasis (on log-fitness, $\varepsilon_{\mathrm{f}} = \varepsilon_{\mathrm{m}} = 0$ in the fitness scheme shown in Table 3). Under this assumption, they found that loci evolved largely independently under weak selection (small selection coefficients $s_u^{\mathrm{X}}$, $u \in \{\mathrm{m},\mathrm{f}\}$ and $\mathrm{X} \in \{\mathrm{A},\mathrm{B}\}$), whereas strong selection (large $s_u^{\mathrm{X}}$) and very low recombination rates could allow linkage disequilibrium between the two loci to build up, leading to the formation of male- and female-beneficial haplotypes and expanding the scope for balancing selection at both loci. This result emerges from the fact that the effects amongst the two loci, while not producing epistasis on a multiplicative scale, do in fact lead to a fitness interaction on the linear scale; specifically to a positive fitness synergy that increases with the size of the selection coefficients $s_u^1$ and $s_u^2$ (here, $H_{u,12}(\boldsymbol{z}) = S_{u,1}(\boldsymbol{z})S_{u,2}(\boldsymbol{z}) > 0$ for both $u \in \{\mathrm{m},\mathrm{f}\}$ in eq. D-11). Consequently, when selection coefficients are large, there is additional selection within sexes favouring the carriage of beneficial alleles at both loci. However, for such correlational selection to drive an evolutionary response requires very tight linkage between the loci [37], else selection cannot generate linkage disequilibrium between co-adapted alleles within each sex.

Importantly, one can see from eq. (D-11) that epistatic interactions amongst loci are inevitably generated by explicit genotype-phenotype-fitness maps, even when loci affect different traits and even under weak selection. The analyses of Patten et al. [39] and Úbeda et al. [40] are therefore not obviously informative about the ability of sexual antagonism to maintain multilocus polymorphism more generally, especially that potentially arising from selection acting across loci in polygenic traits. A model incorporating additive-by-additive epistasis of the kind calculated in this section was later analysed with simulations by Arnqvist et al. [41]. They found that the effect of this type of epistasis on polymorphism conditions is not straightforward and depends on selection and dominance coefficients at the loci in each sex, though strong negative additive-by-additive epistasis (which would, for example, be an interaction arising under stabilising selection on an additive trait) appeared to result in decreased

| | Females | Males |
|---|---|---|
| aabb | $(1-s_{\mathrm{f}}^{\mathrm{A}})(1-s_{\mathrm{f}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{f}})^4$ | 1 |
| Aabb | $(1-s_{\mathrm{f}}^{\mathrm{A}}h_{\mathrm{f}}^{\mathrm{A}})(1-s_{\mathrm{f}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{f}})^2$ | $1-s_{\mathrm{m}}^{\mathrm{A}}h_{\mathrm{m}}^{\mathrm{A}}$ |
| AAbb | $1-s_{\mathrm{f}}^{\mathrm{B}}$ | $1-s_{\mathrm{m}}^{\mathrm{A}}$ |
| aaBb | $(1-s_{\mathrm{f}}^{\mathrm{A}})(1-s_{\mathrm{f}}^{\mathrm{B}}h_{\mathrm{f}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{f}})^2$ | $1-s_{\mathrm{m}}^{\mathrm{B}}h_{\mathrm{m}}^{\mathrm{B}}$ |
| AaBb | $(1-s_{\mathrm{f}}^{\mathrm{A}}h_{\mathrm{f}}^{\mathrm{A}})(1-s_{\mathrm{f}}^{\mathrm{B}}h_{\mathrm{f}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{f}})$ | $(1-s_{\mathrm{m}}^{\mathrm{A}}h_{\mathrm{m}}^{\mathrm{A}})(1-s_{\mathrm{m}}^{\mathrm{B}}h_{\mathrm{m}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{m}})$ |
| AABb | $1-s_{\mathrm{f}}^{\mathrm{B}}h_{\mathrm{f}}^{\mathrm{B}}$ | $(1-s_{\mathrm{m}}^{\mathrm{A}})(1-s_{\mathrm{m}}^{\mathrm{B}}h_{\mathrm{m}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{m}})^2$ |
| aaBB | $1-s_{\mathrm{f}}^{\mathrm{A}}$ | $1-s_{\mathrm{m}}^{\mathrm{B}}$ |
| AaBB | $1-s_{\mathrm{f}}^{\mathrm{A}}h_{\mathrm{f}}^{\mathrm{A}}$ | $(1-s_{\mathrm{m}}^{\mathrm{A}}h_{\mathrm{m}}^{\mathrm{A}})(1-s_{\mathrm{m}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{m}})^2$ |
| AABB | 1 | $(1-s_{\mathrm{m}}^{\mathrm{A}})(1-s_{\mathrm{m}}^{\mathrm{B}})(1+\varepsilon_{\mathrm{m}})^4$ |

**Table 3:** Genotypic fitnesses at two loci under sex-specific selection with epistasis

scope for multilocus polymorphism, which is consistent with results from polygenic selection models [18, 45, 104]. However, any genotype-phenotype-fitness map that produces such strong additive-by-additive epistasis would also generate other types of epistasis, such as dominance-by-additive and dominance-by-dominance. These other types can be ignored under weak selection (as they are of order $\delta^3$ at most) but become important when stronger fitness effects are considered.

# E Conditions for a protected polymorphism in the pseudo-autosomal region of sex chromosomes

In this section, we examine the conditions for polymorphism to be maintained at a diallelic locus under sexually antagonistic selection when it is located on the pseudo-autosomal region of sex chromosomes, building on the seminal analysis by Bull [151] (see also [25, 153] for numerical analyses of this problem). We consider a locus at which alleles $A_1$ and $A_2$ have sex-specific effects on fitness such that allele $A_1$ is favoured in males and $A_2$ is favoured in females. We assume that this locus recombines with an XY sex-determining region at a rate $r < 1/2$, so that it shows partial sex-linkage.

By analysing the conditions for the mutual invasibility of $A_1$ and $A_2$ given in Bull [151], we derive the exact values for the critical recombination rate $r$ allowing the spread of a male-beneficial and female-beneficial allele, respectively.

## E.1 Invasibility conditions

For the sake of completeness, we first re-derive the invasibility condition found in Bull [151] (p. 265) using our notation.

Individuals follow the same life cycle as described in Section 2.1 of the main text: adult males and females mate randomly, produce many offspring and then die. Evolutionary dynamics at the sexually antagonistic locus can therefore be described by tracking the frequency of allele $A_2$ amongst gametes. However, we must now distinguish between three types of gametes owing to linkage with the SDR: Y-bearing male gametes, X-bearing male gametes and (X-bearing) female gametes, in which allele $A_2$ is found at frequency $p_{\mathrm{m}}^{\mathrm{Y}}(t)$, $p_{\mathrm{m}}^{\mathrm{X}}(t)$ and $p_{\mathrm{f}}^{\mathrm{X}}(t)$ at time $t$, respectively.

The frequency of allele $A_2$ in Y-bearing male gametes in the next generation $p_{\mathrm{m}}^{\mathrm{Y}}(t+1)$ can be written as a function of frequencies at time $t$, and is given by

$$p_{\mathrm{m}}^{\mathrm{Y}}(t+1) = p_{\mathrm{m}}^{\mathrm{Y}}(t)\left[p_{\mathrm{f}}^{\mathrm{X}}(t)\frac{w_{\mathrm{m}}(z_{\mathrm{m},22})}{\overline{w_{\mathrm{m}}}(t)} + \left(1-p_{\mathrm{f}}^{\mathrm{X}}(t)\right)\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{\overline{w_{\mathrm{m}}}(t)}(1-r)\right] + \left(1-p_{\mathrm{m}}^{\mathrm{Y}}(t)\right)p_{\mathrm{f}}^{\mathrm{X}}(t)\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{\overline{w_{\mathrm{m}}}(t)}r, \tag{E-1a}$$

where

$$\overline{w_{\mathrm{m}}}(t) = p_{\mathrm{m}}^{\mathrm{Y}}(t)\left[p_{\mathrm{f}}^{\mathrm{X}}(t)\,w_{\mathrm{m}}(z_{\mathrm{m},22}) + \left(1-p_{\mathrm{f}}^{\mathrm{X}}(t)\right)w_{\mathrm{m}}(z_{\mathrm{m},12})\right] + \left(1-p_{\mathrm{m}}^{\mathrm{Y}}(t)\right)\left[p_{\mathrm{f}}^{\mathrm{X}}(t)\,w_{\mathrm{m}}(z_{\mathrm{m},12}) + \left(1-p_{\mathrm{f}}^{\mathrm{X}}(t)\right)w_{\mathrm{m}}(z_{\mathrm{m},11})\right] \tag{E-1b}$$

is mean male fecundity.

Equation (E-1a) can be understood as follows. Following gamete fusion, males carrying allele $A_2$ on their Y chromosome are in frequency $p_{\mathrm{m}}^{\mathrm{Y}}(t)$. These males can either carry another $A_2$ allele on their X chromosome, which occurs with probability $p_{\mathrm{f}}^{\mathrm{X}}(t)$ (as the X carried by males necessarily comes from their mother), in which case their relative contribution to the pool of Y-bearing male gametes in the next generation is given by $w_{\mathrm{m}}(z_{\mathrm{m},22})/\overline{w_{\mathrm{m}}}(t)$ and all their contributed gametes carry allele $A_2$; or they carry an $A_1$ allele with probability $1-p_{\mathrm{f}}^{\mathrm{X}}(t)$, make a relative contribution $w_{\mathrm{m}}(z_{\mathrm{m},12})/\overline{w_{\mathrm{m}}}$ to the Y-bearing male gamete pool, a proportion $1-r$ of which carries allele $A_2$. Males carrying allele $A_1$ on their Y chromosome, meanwhile, are in frequency $1-p_{\mathrm{m}}^{\mathrm{Y}}(t)$ following gamete fusion and can only transmit Y-bearing gametes with an $A_2$ if they carry this allele on their X, which occurs with probability $p_{\mathrm{f}}^{\mathrm{X}}(t)$. Such heterozygous males make a relative contribution to the Y-bearing male gamete pool $w_{\mathrm{m}}(z_{\mathrm{m},12})/\overline{w_{\mathrm{m}}}$, and a fraction $r$ of their contributed gametes carry allele $A_2$.

Following a similar logic for X-bearing male gametes and female gametes, we find that the frequency of allele $A_2$ in these gamete types in the next generation are given by

$$p_{\mathrm{m}}^{\mathrm{X}}(t+1)=p_{\mathrm{m}}^{\mathrm{Y}}(t)\left[p_{\mathrm{f}}^{\mathrm{X}}(t)\frac{w_{\mathrm{m}}(z_{\mathrm{m},22})}{\overline{w_{\mathrm{m}}}(t)}+\left(1-p_{\mathrm{f}}^{\mathrm{X}}(t)\right)\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{\overline{w_{\mathrm{m}}}(t)}r\right]+\left(1-p_{\mathrm{m}}^{\mathrm{Y}}(t)\right)p_{\mathrm{f}}^{\mathrm{X}}(t)\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{\overline{w_{\mathrm{m}}}(t)}(1-r) \tag{E-1c}$$

for X-bearing male gametes, and

$$p_{\mathrm{f}}^{\mathrm{X}}(t+1)=p_{\mathrm{m}}^{\mathrm{X}}(t)\left[p_{\mathrm{f}}^{\mathrm{X}}(t)\frac{w_{\mathrm{f}}(z_{\mathrm{f},22})}{\overline{w_{\mathrm{f}}}(t)}+\left(1-p_{\mathrm{f}}^{\mathrm{X}}(t)\right)\frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{\overline{w_{\mathrm{f}}}(t)}\frac{1}{2}\right]+\left(1-p_{\mathrm{m}}^{\mathrm{X}}(t)\right)p_{\mathrm{f}}^{\mathrm{X}}(t)\frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{\overline{w_{\mathrm{f}}}(t)}\frac{1}{2} \tag{E-1d}$$

in female gametes, where

$$\overline{w_{\mathrm{f}}}(t)=p_{\mathrm{m}}^{\mathrm{X}}(t)\left[p_{\mathrm{f}}^{\mathrm{X}}(t)w_{\mathrm{f}}(z_{\mathrm{f},22})+\left(1-p_{\mathrm{f}}^{\mathrm{X}}(t)\right)w_{\mathrm{f}}(z_{\mathrm{f},12})\right]+\left(1-p_{\mathrm{m}}^{\mathrm{X}}(t)\right)\left[p_{\mathrm{f}}^{\mathrm{X}}(t)w_{\mathrm{f}}(z_{\mathrm{f},12})+\left(1-p_{\mathrm{f}}^{\mathrm{X}}(t)\right)w_{\mathrm{f}}(z_{\mathrm{f},11})\right] \tag{E-1e}$$

denotes mean female fecundity. Jordan and Charlesworth [25] iterated numerically eqs. E-1 for a set of parameter values to infer on the evolutionary outcomes.

More generally, for a protected polymorphism to exist at the locus, alleles $A_1$ and $A_2$ must be able to reciprocally invade, i.e., to increase in frequency in a population otherwise fixed for the other allele. Whether this is the case can be determined by assessing the stability of the equilibria where either allele is fixed, that is where $p_{\mathrm{m}}^{\mathrm{Y}}=p_{\mathrm{m}}^{\mathrm{X}}=p_{\mathrm{f}}^{\mathrm{X}}=0$ and $p_{\mathrm{m}}^{\mathrm{Y}}=p_{\mathrm{m}}^{\mathrm{X}}=p_{\mathrm{f}}^{\mathrm{X}}=1$. This is done by computing the

Jacobian matrix for the dynamical system described in eq. (E-1), which is given by

$$\mathbf{J}(p_{\mathrm{m}}^{\mathrm{Y}}, p_{\mathrm{m}}^{\mathrm{X}}, p_{\mathrm{f}}^{\mathrm{X}}) = \begin{pmatrix} \dfrac{\partial p_{\mathrm{m}}^{\mathrm{Y}}(t+1)}{\partial p_{\mathrm{m}}^{\mathrm{Y}}(t)} & \dfrac{\partial p_{\mathrm{m}}^{\mathrm{Y}}(t+1)}{\partial p_{\mathrm{m}}^{\mathrm{X}}(t)} & \dfrac{\partial p_{\mathrm{m}}^{\mathrm{Y}}(t+1)}{\partial p_{\mathrm{f}}^{\mathrm{X}}(t)} \\ \dfrac{\partial p_{\mathrm{m}}^{\mathrm{X}}(t+1)}{\partial p_{\mathrm{m}}^{\mathrm{Y}}(t)} & \dfrac{\partial p_{\mathrm{m}}^{\mathrm{X}}(t+1)}{\partial p_{\mathrm{m}}^{\mathrm{X}}(t)} & \dfrac{\partial p_{\mathrm{m}}^{\mathrm{X}}(t+1)}{\partial p_{\mathrm{f}}^{\mathrm{X}}(t)} \\ \dfrac{\partial p_{\mathrm{f}}^{\mathrm{X}}(t+1)}{\partial p_{\mathrm{m}}^{\mathrm{Y}}(t)} & \dfrac{\partial p_{\mathrm{f}}^{\mathrm{X}}(t+1)}{\partial p_{\mathrm{m}}^{\mathrm{X}}(t)} & \dfrac{\partial p_{\mathrm{f}}^{\mathrm{X}}(t+1)}{\partial p_{\mathrm{f}}^{\mathrm{X}}(t)} \end{pmatrix}. \tag{E-2}$$

Specifically, an equilibrium is considered unstable if at least one eigenvalue of the Jacobian matrix $\mathbf{J}(p_{\mathrm{m}}^{\mathrm{Y}}, p_{\mathrm{m}}^{\mathrm{X}}, p_{\mathrm{f}}^{\mathrm{X}})$ evaluated at this equilibrium is greater than one. The eigenvalues of the Jacobian matrix at $p_{\mathrm{m}}^{\mathrm{Y}} = p_{\mathrm{m}}^{\mathrm{X}} = p_{\mathrm{f}}^{\mathrm{X}} = 0$ and $p_{\mathrm{m}}^{\mathrm{Y}} = p_{\mathrm{m}}^{\mathrm{X}} = p_{\mathrm{f}}^{\mathrm{X}} = 1$ are complicated, so that it is difficult to obtain explicit mathematical results from their analysis. However, more insight can be obtained by looking at the characteristic polynomial of $\mathbf{J}(p_{\mathrm{m}}^{\mathrm{Y}}, p_{\mathrm{m}}^{\mathrm{X}}, p_{\mathrm{f}}^{\mathrm{X}})$, the roots of which correspond to the eigenvalues of the matrix. This polynomial is given by

$$P_i(\lambda) = -\lambda^3 + \frac{1}{2}\left(\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},ii})} + \frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},ii})}\right)\lambda^2 + \frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},ii})}\left(\frac{1}{2} - r\right)\left(\lambda^2 - \frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},ii})}\frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},ii})}\right), \tag{E-3}$$

where $i = 1$ at the equilibrium where allele $\mathrm{A}_1$ is fixed ($p_{\mathrm{m}}^{\mathrm{Y}} = p_{\mathrm{m}}^{\mathrm{X}} = p_{\mathrm{f}}^{\mathrm{X}} = 0$) and $i = 2$ when allele $\mathrm{A}_2$ is fixed ($p_{\mathrm{m}}^{\mathrm{Y}} = p_{\mathrm{m}}^{\mathrm{X}} = p_{\mathrm{f}}^{\mathrm{X}} = 1$). For a protected polymorphism to exist, it must be the case that $P_i(\lambda)$ poses at least one real root greater than one when $i = 1$ and when $i = 2$. Eq. (E-3) is equivalent to Bull [151]'s conditions (p. 266).

## E.2 Analyses

We now use eq. (E-3) to determine the conditions allowing the spread of the male-beneficial and female-beneficial allele.

### E.2.1 Autosomal case

As a baseline, let us first consider the case where $r = 1/2$. In this case, the sexually antagonistic locus recombines freely with the SDR, so that it effectively behaves like an autosomal locus. The characteristic polynomial $P_i(\lambda)$ then reduces to

$$P_i(\lambda) = \lambda^2\left(\frac{1}{2}\left(\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},ii})} + \frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},ii})}\right) - \lambda\right), \tag{E-4}$$

which has a double root at $\lambda = 0$, and a third root at

$$\lambda = \frac{1}{2}\left(\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},ii})} + \frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},ii})}\right). \tag{E-5}$$

This third root must exceed one at both monomorphic equilibria for a protected polymorphism to be favoured, which occurs when the sex-averaged relative fecundity of heterozygotes exceeds that of both homozygotes when rare, or in other words, when invasion by heterozygotes improves mean fecundity in the population, as expected from invasion analyses on autosomes (e.g., eq. 8 in main text).

### E.2.2 Partial linkage to the SDR

When the sexually antagonistic locus does not recombine freely with the SDR ($r < 1/2$), partial sex-linkage affects the conditions for a protected polymorphism, but showing exactly how it does so requires a more involved analysis.

The discriminant of $P_i(\lambda)$ is strictly positive when $0 < r < 1/2$, which implies that it always has exactly three real roots. Furthermore, because the cubic coefficient of $P_i(\lambda)$ (i.e., the constant multiplying $\lambda^3$) is negative, we know that: $P_i(\lambda)$ is positive at $-\infty$ ($\lim_{\lambda\to-\infty} P_i(\lambda) = +\infty$), goes through a negative local minimum and a local positive maximum, and is negative at $+\infty$ ($\lim_{\lambda\to+\infty} P_i(\lambda) = -\infty$). In addition, one can show that the polynomial has a local minimum at $\lambda = 0$ since we have

$$P_i(0) = -\left(\frac{1}{2} - r\right)\frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},11})}\left(\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},11})}\right)^2 < 0 \quad \text{and} \quad \left.\frac{\mathrm{d}P_i(\lambda)}{\mathrm{d}\lambda}\right|_{\lambda=0} = 0. \tag{E-6}$$

Thus, there are two ways for $P_i(\lambda)$ to have at least one root greater than one. Either it is positive when $\lambda = 1$ ($P_i(1) \geqslant 0$), in which case it has exactly one root greater than one; or it is negative and increasing at $\lambda = 1$, i.e.,

$$P_i(1) < 0 \quad \text{and} \quad \left.\frac{\mathrm{d}P_i(\lambda)}{\mathrm{d}\lambda}\right|_{\lambda=1} > 0, \tag{E-7}$$

in which case it has exactly two roots greater than one.

When genotypic fitnesses are such that a protected polymorphism would be established in the autosomal case, i.e., when

$$\frac{1}{2}\left(\frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},ii})} + \frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},ii})}\right) > 1 \tag{E-8}$$

for $i = 1$ and $i = 2$, it can be shown that one of these two requirements is always fulfilled. Biologically, this means that a locus at which polymorphism would be maintained on an autosome will always be

polymorphic on the PAR as well.

When Condition (E-8) is not met, such that the locus would not be polymorphic if it were located on an autosome, the male-beneficial allele $A_1$ will be able to invade a population fixed for $A_2$ provided that

$$r < \frac{w_{\mathrm{m}}(z_{\mathrm{m},12}) - w_{\mathrm{m}}(z_{\mathrm{m},22})}{w_{\mathrm{m}}(z_{\mathrm{m},12})\left(1 - \dfrac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},22})}\dfrac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},22})}\right)}\left[1 - \frac{1}{2}\frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},22})}\left(1 + \frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},22})}\right)\right]. \tag{E-9}$$

This threshold recombination rate is strictly positive under the assumptions that the allele would not be able to invade on an autosome (i.e. when condition (E-8) holds), is beneficial in males ($w_{\mathrm{m}}(z_{\mathrm{m},12})/w_{\mathrm{m}}(z_{\mathrm{m},22}) > 1$) and detrimental in females ($w_{\mathrm{f}}(z_{\mathrm{f},12})/w_{\mathrm{f}}(z_{\mathrm{f},22}) < 1$). This shows that sufficiently tight linkage with the SDR can aid invasion by the male-beneficial allele.

Meanwhile, the female-beneficial allele $A_2$ will be able to invade into a population fixed for $A_1$ if

$$r < \frac{w_{\mathrm{m}}(z_{\mathrm{m},12}) - w_{\mathrm{m}}(z_{\mathrm{m},11})}{w_{\mathrm{m}}(z_{\mathrm{m},12})\left(1 - \dfrac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},11})}\dfrac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},11})}\right)}\left[1 - \frac{1}{2}\frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},11})}\left(1 + \frac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},11})}\right)\right], \tag{E-10}$$

which can only happen when selection is sufficiently strong in females relative to males, specifically when

$$\frac{w_{\mathrm{f}}(z_{\mathrm{f},12})}{w_{\mathrm{f}}(z_{\mathrm{f},11})} > \frac{2}{1 + \dfrac{w_{\mathrm{m}}(z_{\mathrm{m},12})}{w_{\mathrm{m}}(z_{\mathrm{m},11})}}. \tag{E-11}$$

This is because stronger selection in females allows genetic associations to build between the X and the female-beneficial allele $A_2$, despite the fact that the X chromosome spends 1/3 of the time in males.

Altogether, these results show that linkage to the sex-determining region allows for polymorphism to be maintained between alleles $A_1$ and $A_2$ in the PAR even in the absence of dominance reversal because of the stronger genetic associations that develop between alleles of high fitness in a sex and presence in that sex. When selection is weak, this requires very tight linkage as the threshold recombination rates required for polymorphism are small (proportional to the strength of selection). With strong selection, these conditions can be met even at loci distant from the SDR, especially for male-beneficial alleles. Similar qualitative conclusions were drawn by Charlesworth et al. [153] who approximated the cubic.

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