The Role of Pollen and Sperm Competition in Sex Chromosome Evolution

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To date, research on the evolution of sex chromosomes has focused on sexually antagonistic selection, which has been shown to be a potent driver of the strata and reduced recombination that characterize many sex chromosomes In this study, we expand our view of the forces driving sex chromosome evolution by considering also selection among haploids, which is likely to occur predominantly among male gametes in angiosperms and animals, i.e., during pollen or sperm competition. We find that suppressed recombination is favoured on the sex chromosomes, even without selective differences between male and female diploids. Reduced recombination is favoured because it creates a stronger association between haploid beneficial alleles and the male determining region (Y or Z), which experiences haploid selection most often. Similarly, reduced recombination creates linkage between alleles selected against in the haploid stage and the female determining region (X or W). In XY systems, these associations also result in biased sex ratios at birth. Overall, we predict that whether and how fast recombination suppression evolves on the sex chromosomes can depend on the degree of haploid competition, not just on selective differences between the diploid sexes. Based on our models, sex chromosomes should become enriched for genes that experience haploid selection, as is expected for genes that experience sexually antagonistic selection. Thus, we generate a number of promising predictions that can be evaluated in emerging sex chromosome systems.

recombination evolution | sex chromosomes | sperm competition | pollen competition | haploid selection | modifier model | evolutionary theory

Introduction

In organisms with diploid genetic sex determination, recombination is typically suppressed between the X and Y chromosomes or Z and W chromosomes. Suppressed recombination appears to begin near the sex-determining region (SDR) and then expand to include larger segments of each sex chromosome [1, 2, 3, 4, 5]. In the absence of recombination, the sex-limited chromosome (Y or W) accumulates deleterious mutations (including gene losses) within the non-recombining region and 'genetic degeneration' occurs [6, 7, 8, 9]. Thus, the selective forces driving reduced recombination on sex chromosomes are fundamental to our understanding of sex chromosome evolution.

Typically, selective differences between males and females have been evoked to explain the suppression of recombination around established sex-determining regions [10, 11, 12]. Charlesworth and Charlesworth [13] showed that loci where males and females differ in equilibrium allele frequency due to selection (for example, sexually antagonistic selection) should evolve complete linkage with the sex-determining locus via translocations or fusions. More recently, Lenormand [14] demonstrated that sex differences in allele frequencies at equilibrium are not required in order to favour reduced recombination with the sex-determining region. In fact, recombination suppression can evolve around the sex-determining region even if selection favours the same allele in both sexes as long as that allele is favoured more strongly in one sex than the other. In essence, these studies have demonstrated that suppressors of recombination can be favoured because they strengthen the association between the sex in which an allele is favoured and the chromosome that is present in that sex more often, e.g., between male beneficial alleles and the Y or Z and between female beneficial alleles and the X or W [15].

While differences in selection between the diploid sexes has attracted the most theoretical and empirical attention, haploid gametes/gametophytes produced by males and females typically differ in the potential for selection because competition among pollen and sperm is particularly intense [16, 17, 18]. To the extent that pollen and sperm success reflects differences in their haploid genotypes, selection among these gametes/gametophytes is qualitatively distinct from selection among diploid males. That is, diploids cannot be assigned fitness values that also account for the fitness of their haploid gametes [19]. In plants, selection among haploid male gametophytes is thought to be pervasive [20, 21, 22]; in *Arabidopsis*, 60-70% of all genes are expressed during the haploid phase [23], and pollen expressed genes exhibit stronger signatures of purifying selection and positive selection [24, 25]. For agricultural breeding, pollen has been exposed to a variety of selection pressures *in vivo* and *in vitro*, including temperature [26, 27], herbicides [28], metals [29], water stress [30], and pathogens [31], resulting in an increased frequency of resistant genotypes among the diploid sporophytic offspring. In animals, expression during the haploid sperm stage is traditionally thought to be suppressed [32], although recent evidence suggests that the extent and selective importance of postmeiotic gene expression may be underestimated [33, 18, 34, 35].

Significance

One feature that characterizes many sex chromosome systems is that, along most of the length, there is no recombination with the opposite sex chromosome. The typical explanation for this phenomenon entails differences in selection between the sexes. However, selection during the haploid phase of males and females can also be strikingly divergent, with male-derived pollen/sperm experiencing particularly intense competition. We show that competition between pollen/sperm can favour extreme suppression of recombination between sex chromosomes. Extremely suppressed recombination allows strong associations between pollen/sperm beneficial alleles and the male-determining region (Y or Z), which results in biased sex ratios in XY systems. Thus, we provide predictions about the rate of sex chromosome evolution and genomic location of pollen/sperm expressed genes.

Reserved for Publication Footnotes

The maintenance of polymorphism at loci that experience sex specific selection in both haploid and diploid phases was considered by Immler et al. [19], demonstrating that polymorphisms can be maintained by sexually antagonistic selection or overdominance as well as by conflicting selection pressures in haploids and diploids (haploid-diploid conflict or ploidally antagonistic selection) or a combination of these selective regimes. In this study, we include selection during the male haploid phase (sperm or pollen competition) in models for the evolution of recombination with the sex-determining region (XY or ZW).

Model Background

Recombination evolution on sex chromosomes is usually modelled by considering a locus under selection, the sex-determining region, and another locus that modifies the recombination rate between them, where modifiers include inversions, fusions, hotspot changes, and changes to genes involved in double strand breaks and recombination repair. Thus, a general model includes three loci and the recombination rates between them, which is typically too complex to interpret without further simplifying assumptions [36]. Lenormand [14] assumed that the recombination rates between these loci are large relative to selection, such that the linkage disequilibrium between loci equilibrates on a faster timescale than changes in allele frequencies (a 'quasi-linkage equilibrium' approximation). This analysis is most appropriate for selected loci that are far from the sex-determining region on sex chromosomes and when modifiers of recombination are weak and loosely linked (e.g., autosomal modifiers of recombination machinery). Secondly, Charlesworth and Charlesworth [13] assumed that the selected locus is initially autosomal and then considered fusions with (or translocations to) the sex-determining region, where their analysis assumed these rearrangements became closely linked to the selected locus. Their model also corresponds to modifications on sex chromosomes (e.g., inversions) that change the recombination rate with the sex-determining region from a very high to a very low level. Finally, Otto [37] considered modifiers of recombination between the sex-determining region and selected loci when the linkage between them is initially very tight.

Here, we study recombination evolution in a manner akin to Charlesworth and Charlesworth [13] and Otto [37] except that we include a period of selection among haploid male gametes/gametophytes. The model of Lenormand [14] is very general and allows a period of haploid selection (assuming weak linkage); he recognizes but does not discuss the potential of such sex-specific haploid selection to favour suppressed recombination on sex chromosomes. Here, our goal is to complete the set of recombination evolution analyses that include a period of haploid pollen/sperm competition and explicitly describe why loci that experience haploid selection can drive the evolution of reduced recombination near sex-determining regions. Models where haploid selected loci and the sex-determining region can become tightly linked are particularly significant because sex in an XY sex determination system is determined by the chromosome carried by the successful pollen/sperm (after haploid selection). Thus, strong associations between haploid selected alleles and the sex-determining region (that can build up when linkage is tight) will cause diploid sex ratios to become biased, figure 1.

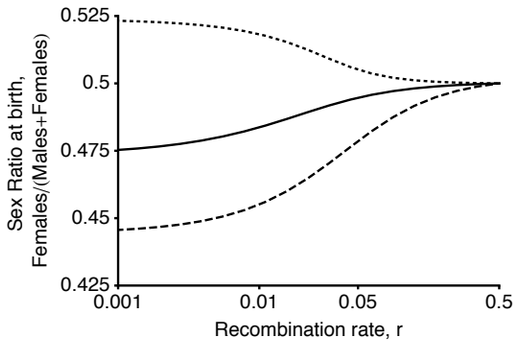

Fig. 1. The sex ratio at birth is biased by linkage between an XY sex-determining region (SDR) and a locus that experiences haploid selection during pollen/sperm competition (A). Here, we assume that the population is fixed for a particular modifier of recombination such that all individuals have the same recombination rate, r. We then allow the **A** locus to reach an equilibrium frequency and calculate the birth sex ratio. Alleles with high fitness during pollen/sperm competition typically become associated with the Y, causing sex ratios to become male-biased (solid and dashed lines). However, female biased sex ratios can arise if the haploid-beneficial allele is also strongly female-beneficial, causing it to become associated with the X (dotted line). The parameters used in this plot are: solid line ($w^m_{ij} = w^f_{ij} = w_{ij}$, $w_{aa} = 1$, $w_{Aa} = 0.97$, $w_{AA} = 0.91$, $w_a = 0.9$, $w_A = 1$) dashed line ($w^m_{ij} = w^f_{ij} = w_{ij}$, $w_{aa} = 1$, $w_{Aa} = 1.12$, $w_{AA} = 1.24$, $w_a = 0.8$, $w_A = 1$), dotted line ($w^m_{aa} = 1$, $w^m_{Aa} = 0.94$, $w^m_{AA} = 0.8$, $w^f_{aa} = 1$, $w^f_{Aa} = 1.14$, $w^f_{AA} = 1.2$, $w_a = 0.9$, $w_A = 1$).

Model

We consider a modifier model in which the recombination rate between a locus under selection (selected locus, **A**, with alleles A and a) and the sex-determining region (SDR) depends on the genotype at the modifier locus (**M**, with alleles M and m). In our model, male haploid gametes/gametophytes experience selection according to their genotype at the **A** locus (see table S.1) before random mating with female gametes/gametophytes. The resulting zygotes develop as males or females depending on their genotype at the sex-determining region. Diploid genetic sex determination systems are either male heterogametic (females XX and males XY) or female heterogametic (females ZW and males ZZ). There are therefore two asymmetries in the model, the sex in which haploid selection occurs and the sex which is heterogametic. For simplicity, we primarily describe XY sex determination with male gametophytic selection (pollen/sperm competition), although we also present results for ZW sex determination and male gametophytic selection.

After a period of selection among diploid males and females (table S.1), meiosis with recombination occurs to produce haploid gamete/gametophytes. Because females are homozygous at the SDR (with XY sex determination), the only recombination event of consequence in females is between the **A** and **M** locus, which occurs at rate R_f. In males, recombination similarly occurs between the selected locus **A** and the modifier locus **M** at rate R_m. Recombination can also occur between the SDR and the **A** locus in males, this recombination rate is controlled by the modifier locus and is given by r_{ij}, where ij is the genotype at the **M** locus (MM, Mm, or mm), allowing this recombination rate to evolve. Double recombination events in males occur at rate χ_{ij}, such that any ordering of the loci or type of modifier (genic, inversion, fusion) can be modelled with appropriate choices of χ_{ij}, r_{ij}, and R_m. We track the frequencies of MA, Ma, mA and ma genotypes among female eggs/ovules, male X-bearing sperm/pollen, and male Y-bearing sperm/pollen separately to allow sex-specific allele frequencies and disequilibria. The recursion equations describ-

ing the change in genotype frequencies after a single generation of this life cycle are provided in the Sup. Mat.

In our first analysis, we assume that selection is weak relative to the initial recombination rate (r_{MM}), such that allele frequency differences between males and females are small. We then evaluate the spread of modifiers of recombination (m) that cause recombination rates to become very small (assuming r_{Mm}, χ_{Mm}, and R_m are all small). These modifiers could be translocations or fusions from autosomes to sex chromosomes or, if the selected locus (**A**) begins on the sex chromosome, inversions or expansions of the non-recombining region. We assume that chromosomes are still able to disjoin regularly from their homologs during meiosis.

In our second analysis, following Otto [37], we assume that the **A** locus begins at equilibrium and in tight linkage with the SDR (r_{MM} and χ are on the order of a small term, ϵ). We then consider whether any modifiers can invade that increase this recombination rate slightly (where the change in recombination rate, $r_{Mm} - r_{MM}$, is on the order of ϵ). The recombination rate between the modifier locus and these sex chromosome loci (R_f and R_m) is not constrained. This analysis focuses on the final stages of sex chromosome evolution, asking when complete recombination is favoured or not.

Results

Considering a population originally fixed for the M allele at the modifier locus, the frequency of the A allele among X-bearing eggs/ovules, X-bearing sperm/pollen, and Y-bearing sperm/pollen is given by p_{Xf}, p_{Xm}, and p_{Ym} respectively. The spread of rare mutants that change the recombination rate can be evaluated using the leading eigenvalue, λ, of the system described by equations (A1c), (A1d), (A2c), (A2d), (A3c), and (A3d).

Complete suppressors of recombination ($r_{Mm} = 0$) that are closely linked to the **A** locus ($R_f = R_m = \chi = 0$) experience the strongest selective force. These modifiers can bring either the A or the a allele into tight linkage with either the X or Y chromosome. Thus, the invasion of these mutants can be evaluated separately and is given by λ_{ij}, where ij is the haplotype at the newly linked SDR and **A** loci.

The spread of modifiers that create tight linkage between the Y and A allele is given by

$$\lambda_{YA} = \bar{w}^m_{YA}/\bar{w}^m \qquad [\mathbf{1}]$$

where $\bar{w}^m_{YA}$ is the marginal fitness of YA haplotypes and $\bar{w}^m$ is the mean fitness of males, see table S.2. Such modifiers will spread if $\lambda_{YA} > 1$, which is true when $\bar{w}^m_{YA} > \bar{w}^m$.

Invasion of modifiers that create a strong linkage between the X and a allele is determined by the largest solution to the characteristic polynomial **S.4**. For such modifiers, the leading eigenvalue λ_{Xa} is greater than one if

$$\bar{w}^{mat,f}_{Xa}/\bar{w}^f + (\bar{w}^{mat,m}_{Xa}/\bar{w}^m)(\bar{w}^{pat,f}_{Xa}/\bar{w}^f) > 2 \qquad [\mathbf{2}]$$

where $\bar{w}^f$ is the mean fitness of females and $\bar{w}^{i,j}_{Xa}$ indicates the marginal fitness of Xa haplotypes when inherited from the mother ($i = mat$) or father ($i = pat$) and found in offspring of sex j This condition demonstrates that the newly formed sex chromosome is able to invade if its marginal fitness is higher than average (once appropriately weighted across carriers of maternal and paternal copies). However, we have not yet considered constraints on the initial frequency of the A allele.

Next, we will consider the case where the **A** locus is initially at an intermediate frequency maintained by selection. Polymorphisms can be maintained by a combination of sexually antagonistic selection, ploidally antagonistic selection, and/or overdominance [19]. We then write λ_{YA} in terms of the difference in fitness between haploid genotypes ($\delta_H = w_A - w_a$) and the difference in equilibrium allele frequency between Y-bearing pollen/sperm and ovules/eggs ($\delta = \hat{p}_{Ym} - \hat{p}_{Xf}$) where the caret indicates an equilibrium frequency. We can then write equation **1**, for the invasion of modifiers that bring the A allele into tight linkage with the Y chromosome, as

$$\lambda_{YA} = 1 + \frac{r_{MM} w^m_{Aa}}{\hat{p}_{Ym}\bar{w}^m}\left(\delta + V_m \delta_H/\bar{w}_H\right) \qquad [\mathbf{3}]$$

where $V_m = \hat{p}_{Ym}(1 - \hat{p}_{Ym})$ is the variance among Y-bearing pollen/sperm and $\bar{w}_H = (\hat{p}_{Ym}w_A + (1 - \hat{p}_{Ym})w_a)$ is the mean fitness of haploid male gametes/gametophytes. If there is no selection among haploid genotypes ($w_A = w_a$), equation **3** is equivalent to equation (A3) in Charlesworth and Charlesworth [13], in which case these tightly linked YA haplotypes invade if the A allele is more common in males than females ($\hat{p}_m - \hat{p}_{Xf} > 0$), as expected if the A allele is beneficial in males with sexually antagonistic selection. Here we also find an additional term, demonstrating that tight linkge is also favoured when the A allele is beneficial during haploid selection ($w_A > w_a$), even in the absence of frequency differences between males and females ($\hat{p}_{Ym} = \hat{p}_{Xf}$), i.e., even when there is no difference in selection between diploid males and females.

Here, in order to solve **S.4** for λ_{Xa}, we will assume that linkage is initially loose between the SDR and **A** locus ($r_{MM} = 1/2$), such that segregation in males is random and $\hat{p}_{Xm} = \hat{p}_{Ym} = \hat{p}_m$. In the Sup. Mat we present equivalent results for cases where we do not assume that recombination is initially free ($r_{MM} < 1/2$). We will further assume that selection is weak, such that the difference in frequency between A alleles in males and females ($\delta = \hat{p}_m - \hat{p}_{Xf}$) and the difference in fitness between haploid genotypes ($\delta_H = w_A - w_a$) are small (δ and δ_H of order ϵ^2). Ignoring terms of order ϵ^3 and higher

$$\lambda_{Xa} = 1 + \frac{1}{3}\frac{w^m_{Aa}}{2(1 - \hat{p}_m)\bar{w}^m}\left(\delta + V_m\delta_H\right). \qquad [\mathbf{4}]$$

Thus, the same conditions that favour linkage between the Y and the A allele, favour linkage between the X and the a allele. Specifically, when the a allele is more common in females ($\delta > 0$, e.g., a is a female beneficial allele) and when the A allele is favoured during haploid competition ($\delta_H > 0$). In the special case where there is no difference in selection between male and female diploids ($w^m_{ij} = w^f_{ij} = w_{ij}$), we can find an exact expression for λ_{XA} by solving for $\hat{p}_m$ and $\hat{p}_{Xf}$ without assuming that selection is weak, which confirms the expectation from **4** that linkage between the X chromosome and alleles deleterious in haploid pollen/sperm is favoured by selection, see Sup. Mat.

It may not be intuitively obvious why an association with the allele that is less fit during haploid selection should be favoured. This result comes from the fact that the a allele is initially maintained at an equilibrium frequency when loosely linked to the SDR. At equilibrium, selection against a in haploid male gametes/gametophytes must be balanced by selection in favour of A in female and/or male diploids. However, the X chromosome is found in males less often than an autosomal or loosely linked locus and therefore experiences haploid selection less frequently. Thus linkage between the a locus and the X is favoured because it allows the a allele to experience haploid selection less often. Similarly, equation **3** indicates that linkage between the Y, which experiences haploid selection most often, and a haploid beneficial allele is favoured.

As with previous analyses [13, 38, 14], we find that the strength of selection in favour of recombination modifiers is

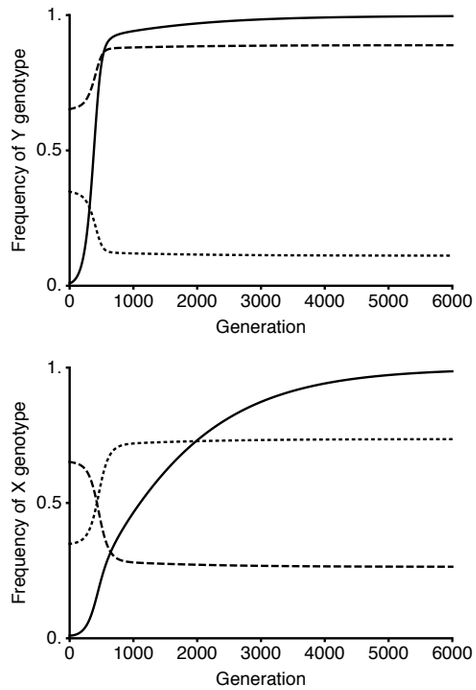

Fig. 2. A modifier that reduces the recombination rate between the A locus and the SDR can spread to fixation despite causing sex ratios to become biased. Here, we iterate the recursion equations S.1, S.2, S.3 to track the change of genotype frequencies among X-bearing female haploids (X_i^f), X-bearing male haploids (X_i^m), and Y-bearing male haploids (Y_i^m), respectively. Across this plot, X-bearing haploids in males and females have very similar haplotype frequencies so we plot X_i^m only. We assume that the population initially has loose linkage between the **A** locus and the SDR ($r_{MM} = 0.5$, where M is initially fixed) and allow allele frequencies to reach a polymorphic equilibrium. We then introduce a modifier allele m that reduces the recombination rate between **A** locus and the SDR ($r_{Mm} = r_{mm} = 0.01$); in generation 0, m is at frequency 0.01 and in linkage equilibrium with M. We assume that the **M** locus lies between the **A** locus and the SDR such that $\chi_{ij} = (r_{ij} - R_m)/(1-2R_m)$, where $R_m = R_f = 0.005$. Fitness parameters are as in the solid line in figure . That is, there are no differences in selection between diploid sexes and selection is ploidally antagonistic with A favoured by haploid selection, thus $\hat{p}_{Xf} = \hat{p}_{Xm} = \hat{p}_{Ym}$ initially, see Sup. Mat. Lines show the frequencies of the A allele (dashed), the a allele (dotted) the recombination suppression mutant, m (solid). Due to continuing recombination between the **A** locus, **M** locus, and the SDR, a particular haplotype does not fix on the Y chromosome, as is the case when $r_{ij} = 0$ (see Sup. Mat.). However, after recombination has evolved to a lower level, the haploid beneficial allele (A, dashed lines) becomes more common on the Y and less common on the X.

strongest on Y chromosomes because these are always found in only one sex whereas the X will sometimes be found in males and sometimes in females. In particular, **3** and **4** differ by a factor of 1/3 once we account for the difference between the probability of linkage arising with the A allele, p_m, or the a allele, $(1 - p_m)$. However, mutations causing linkage with the Y (e.g., fusions) should also arise at a lower rate because there are three times as many X chromosomes as Y chromosomes in the population, such that the overall establishment rate of recombination modifiers is the same on the X and Y [39].

The tight linkage case considered above is the best case scenario for generating selection in favour of recombination suppressors. For a few parameters, Charlesworth and Charlesworth [13] find numerically that recombination suppressors spread, but at lower rates, if R_m and R_f are larger. Here, we find analytical results by assuming the recombination rates between the **A** locus, the **M** locus, and the SDR are small (χ, R_m and R_f or order ϵ^3). Neglecting terms of order ϵ^4 and higher, the growth rate of such mutants ($\lambda_{\tilde{ij}}$) is.

$$\lambda_{\tilde{YA}} \approx \lambda_{YA} - \frac{(1-\hat{p}_m) w^m_{Aa} R_m}{\bar{w}^m} - \chi_{Mm} \qquad [\mathbf{5a}]$$

$$\lambda_{\tilde{Xa}} \approx \lambda_{Xa} - \frac{\hat{p}_m}{3}\left(\frac{2w^f_{Aa}R_f}{\bar{w}^f} + \frac{w^m_{Aa}R_m}{\bar{w}^m}\right) - \frac{\chi_{Mm}}{3} \qquad [\mathbf{5b}]$$

where λ_{ij} corresponds to the tight linkage results **3** and **4**. The additional terms in **5** illustrate that the spread of linked haplotypes is slowed when the alternative **A** allele recombines onto the modifier and SDR background (recombination rate R_m or R_f) or when the modifier recombines onto the opposite sex chromosome (which occurs at rate χ_{Mm} in males). In figure , we track the spread of a recombination modifier where $R_m, R_f, \chi, r_{Mm} \neq 0$, such that both **M** alleles and both **A** alleles can recombine onto both sex chromosomes. As predicted from equation **5**, a recombination suppressor increases in frequency and the X and Y chromosomes become associated with the a and A alleles, respectively.

We derive equivalent results for ZW sex chromosome systems (where males are ZZ and females are ZW) with a period of haploid selection among male gametes/gametophytes. We again consider invasion of a modifier that creates tight linkage between the **A** locus and the **M** locus (r_{Mm}, χ, R_m and R_f of order ϵ^3) in a population in which linkage is initially loose between the SDR and **A** locus ($r_{MM} = 1/2$). Here, we present λ_{Wa} and λ_{ZA} under the same assumptions as **5**, where the difference in frequency of the A allele between males and females and the difference in fitness between haploid genotypes are small ($\delta = \hat{p}_{Zm} - \hat{p}_{Wf}$ where δ and δ_H are of order ϵ^2), yielding

$$\lambda_{\tilde{Wa}} \approx 1 + \frac{r_{MM} w^f_{Aa}}{(1-\hat{p}_f)\bar{w}^f}(\delta + V_f \delta_H) - \frac{\hat{p}_f w^f_{Aa} R_f}{\bar{w}^f} - \chi_{Mm} \qquad [\mathbf{6a}]$$

$$\lambda_{\tilde{ZA}} \approx 1 + \frac{1}{3}\frac{w^f_{Aa}}{2\hat{p}_f\bar{w}^f}(\delta + V_f\delta_H) - \frac{(1-\hat{p}_f)}{3}\left(\frac{2w^m_{Aa}R_m}{\bar{w}^m} + \frac{w^f_{Aa}R_f}{\bar{w}^f}\right) - \frac{\chi_{Mm}}{3} \qquad [\mathbf{6b}]$$

where we discard terms of $O(\epsilon^4)$. λ_{ZA} and λ_{Wa} show that, when the A allele is more common in males ($\delta > 0$), linkage between the male Z chromosome and the A allele and linkage between the female specific W chromosome and the a allele are both favoured. In addition, linkage is favoured between the Z and the allele favoured during haploid selection (A if $\delta_H > 0$) and between the female specific W chromosome and the allele with low haploid fitness (a if $\delta_H > 0$).

Finally, we evaluate the evolution of recombination during the final stages of sex chromosome evolution by considering the evolution of small amounts of recombination around the sex-determining region. Considering diploid selection alone, Otto [37] demonstrated that a small amount of recombination can be maintained by selection. Due to the asymmetrical inheritance pattern of sex chromosomes, an allele that is fixed on a Y chromosome (e.g., the A allele) can also be favoured on the X during selection in females, even if the X is fixed for the alternative allele (e.g., the a allele). With diploid selection only, this requires that the X-specific allele (a) is favoured during selection on the X in males, which only occurs when selection in males is overdominant. Assuming, for example, that

the Y is originally fixed for the A allele, recombination with the sex-determining region produces XA and Ya haplotypes in pollen or sperm. Although Ya haplotypes always have low fitness, XA haplotypes can experience a short term advantage because they next experience selection in females and selection in females can favour the A allele. For a subset of parameters, the cost of producing low fitness sons can be outweighed by this transient fitness advantage in daughters, allowing modifiers of recombination that increase recombination to invade if sufficiently loosely linked to the sex-determining region, for further discussion see [37]. Here, we perform a similar analysis in which there is also a period of selection among male haploids.

We find that mutants that increase the recombination rate are not typically favoured by selection. In particular, when the modifier locus is tightly linked to the **A** locus ($R_f \approx 0$), only mutants that suppress recombination can spread. In addition, if the allele that is fixed on the Y (e.g., A) is favoured by selection on the X in males ($w^m_{AA} > w^m_{Aa}$) but not during selection among haploids ($w_a > w_A$) increased recombination is never favoured. However, if these conditions are not met there are some parameters under which increased recombination is favoured by selection (figure S.1).

Assuming that the A allele is initially fixed on the Y, increased recombination is favoured when the cost of producing Ya pollen/sperm is be outweighed by selection in favour of XA pollen/sperm. If R_f and R_m are small, the modifier remains linked to the haplotypes it creates (XA and Ya), which always leads to a net long term disadvantage. When R_f and R_m are sufficiently large (e.g., autosomal modifiers), a modifier that increases recombination can gain a short term benefit from being found on XA pollen/sperm before recombining onto a different background. XA pollen or sperm can have a short term advantage if they have high fitness during haploid competition and/or high fitness in female diploids (all X-bearing pollen/sperm will form female diploids). Increased recombination is only consistent with XA being favoured during haploid selection and/or selection in females, see Sup. Mat. For most parameters with or without haploid selection, XA pollen/sperm either have no fitness advantage or have a transient fitness advantage that is outweighed by the cost of producing low-fitness Ya pollen/sperm. Thus, increased recombination does not usually evolve. However, with selection among haploids, it is possible for a small amount of recombination to be favoured under a less restrictive set of selective regimes in diploids, including overdominance, sexually antagonistic selection and ploidally antagonistic selection.

Discussion

Even in predominantly diploid organisms such as animals and angiosperms, there is considerable potential for selection among haploid male gametes (sperm/pollen). Here, we demonstrate that linkage between the diploid sex-determining region (XY or ZW) and a locus that experiences haploid selection is typically favoured by selection. Thus, along with selective differences between diploid sexes, selection among haploids could be a potent driver of recombination suppression on sex chromosomes.

In ZW sex determination systems, the sex ratio among diploids is unaffected by selection among male haploids. However, in XY sex determination systems, the number of males and females in each generation depends on the frequency of X and Y gametes after haploid selection. Despite this, we find that selection on recombination modifiers is not primarily driven by balancing the sex ratio of diploids. In fact, the evolution of recombination suppression should lead to Y-bearing gametes/gametophytes that have high fitness during haploid selection. Thus, we predict that sex ratios at birth can become male biased in the early stages of sex chromosome evolution.

Biased flowering sex ratios, especially male-biased sex ratios, are common among dioecious plants [40]. However, in *Rumex*, more intense pollen competition appears to result in more female biased sex-ratios among the progeny [41, 42, 43]. This phenomenon may reflect the accumulation of deleterious mutations on the Y-chromosome following recombination suppression [44, 45], as suggested by the prevalence of female sex ratio bias in species with heteromorphic rather than homomorphic sex chromosomes [40]. Thus, the net effect of experimentally manipulating the intensity of haploid selection may depend on the stage of sex chromosome degeneration, as well as the alleles associated with the Y. The increasing availability of sex-linked markers should allow sexes to be identified before reproductive maturity in plants, thus allowing changes in the sex ratio to be directly evaluated across haploid and diploid phases in species with differing degrees of Y chromosome degeneration.

The emergence of both haploid expression profiles [18, 23] and a larger number of sex chromosome systems [46, 5, 47, 48, 49] provides an excellent opportunity to evaluate whether sex chromosomes are enriched for genes selected during the haploid phase, as predicted by our models. If possible, a stronger signal of association with sex-determining regions should occur among loci explicitly shown to exhibit variation in haploid competitive ability [50] or loci where mutants affect fitness in both haploid and diploid phases [51]. Finally, we predict that the strength of haploid competition partly determines whether and how fast recombination suppression evolves. Evaluating a related hypothesis, Lenormand and Duthiel [52] correlate heterochiasmy (differences in autosomal recombination between sexes) with the degree of sex specific haploid selection, using outcrossing rate as a proxy for male haploid selection. We would predict a similar pattern for recombination suppression around sex-determining regions. Estimates of pollen limitation could also be used as proxy for the intensity of haploid competition [53, 54].

As in a previous analysis by Otto [37], we find that a small amount of recombination can be selectively maintained around the sex-determining region. Otto [37] considered only diploid selection and found that overdominance in males was required for recombination to be selectively maintained. Here, we include a period of selection among haploids and find that increased recombination can be favoured with various forms of selection among diploids, including sexually antagonistic selection and ploidally antagonistic selection (figure S.1), as long as the allele fixed on the Y is favoured in haploids and/or females. However, increased recombination is never favoured when modifiers of recombination act locally, such that they are also closely linked to the sex-determining region. Therefore, while these dynamics may influence the maintenance of small amounts of recombination around sex-determining regions when polymorphisms with the right form of selection arise (e.g., within the coloured regions in figure S.1), suppressed recombination will be favoured in most circumstances.

Meiotic drive is not exactly equivalent to a period of haploid selection. In particular, meiotic drive can only occur in heterozygotes and often involves an interaction with a separate susceptible/resistant locus [11]. However, meiotic drive is also usually sex specific, either acting during spermatogenesis in males or polar body formation in females. In this respect, we expect loci experiencing meiotic drive to behave similarly to those experiencing haploid selection. In particular, we predict that selection should favour linkage between

alleles favoured by drive and the sex chromosome for the sex in which drive occurs (e.g., with the Y or Z when drive occurs during spermatogenesis). Despite theoretical interest in related topics [55, 56, 57, 58, 59], such as the evolution of recombination between an X chromosome that experiences drive and another selected locus [55, 59], this process has yet to be explicitly modelled and is worthy of future exploration.

Overall, as well as providing several predictions, our model offers a new perspective on drivers of sex chromosome evolution. Traditionally, sex differences in selection are thought to provide the raw material driving recombination suppression on sex chromosomes. However, even where diploid sexes exhibit very few morphological or ecological differences, the selective environment of their haploid gametes may be very divergent. We have shown that this condition - differences in fitness among pollen or sperm - should also favour suppressed recombination. Consequently, our view of sex chromosome evolution is expanded to incorporate the degree of sex specific selection in haploids along with that in diploids.

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Recursion Equations

In each generation we census the genotype frequencies in male and female haploids before haploid selection, e.g., sperm/pollen and eggs/ovules. Before haploid selection, the frequency of X-bearing male and female haploids are given by X_i^m and X_i^f and the frequency of Y-bearing haploids is given by Y_i^m where the index i specifies genotypes MA, Ma, mA, and ma. Selection then occurs among male haploids according to the **A** locus allele, k, carried by individuals with genotype i. Assuming that the fraction of X-bearing haploids produced by males is f, the genotype frequencies after haploid selection are $X_i^{m,s} = fw_k X_i^m/\bar{w}_H$ and $Y_i^{m,s} = (1-f)w_k Y_i^m/\bar{w}_H$, where $\bar{w}_H = \sum_{i=1}^4 fw_k X_i^m + (1-f)w_k Y_i^m$ is the mean fitness of male haploids. Random mating then occurs between gametes to produce diploid females with genotype ij at frequency $x_{ij} = X_i^f X_j^{m,s}$ and diploid males at frequency $y_{ij} = X_i^f Y_j^{m,s}$. In females, individuals with genotype ij are equivalent to those with genotype ji. For simplicity we denote the frequency of genotype ij in females to the average of these frequencies, $x_{ij} = (X_i^f X_j^{m,s} + X_j^f X_i^{m,s})/2$. Note that the sex ratio before diploid selection depends both on the production of X-bearing haploids by fathers (f) and on haploid selection (w_k). However, f does not enter into any results, indicating that the main force driving recombination evolution is not to balance the current sex ratio.

Table S.1. Fitness of different genotypes.

Genotype	A	a	AA	Aa	aa
Fitness in males	w_A	w_a	w_{AA}^m	w_{Aa}^m	w_{aa}^m
Fitness in females	1	1	w_{AA}^f	w_{Aa}^f	w_{aa}^f

Table S.2. Marginal fitnesses of YA and Xa haplotypes

$\bar{w}_{YA}^m$	$= (w_A(p_{Xf}w_{AA}^f + (1-p_{Xf})w_{Aa}^f))$
$\bar{w}_{Xa}^{mat,m}$	$= p_{Ym}w_A w_{Aa}^m + (1-p_{Ym})w_a w_{aa}^m$
$\bar{w}_{Xa}^{pat,f}$	$= p_{Xf}w_a w_{Aa}^f + (1-p_{Xf})w_a w_{aa}^f$
$\bar{w}_{Xa}^{mat,f}$	$= p_{Xm}w_A w_{Aa}^f + (1-p_{Xm})w_a w_{aa}^f$

Selection among diploids then occurs according to the diploid genotype at the **A** locus, k, for an individual of type ij (see Table S.1). The diploid frequencies after selection are given by $x_{ij}^s = w_k^f x_{ij}/\bar{w}^f$ in females and $y_{ij}^s = w_k^m y_{ij}/\bar{w}^m$ in males, where $\bar{w}^f = \sum_{i=1}^4\sum_{j=1}^4 w_k^f x_{ij}$ and $\bar{w}^m = \sum_{i=1}^4\sum_{j=1}^4 w_k^m y_{ij}$ are the mean fitnesses of females and males, respectively. Finally, these diploids undergo meiosis to produce the next generation. The haplotype frequencies in the next generation of eggs/ovules is given by:

$$X_{MA}^{f'} = \left(\sum_{j=1}^4 x_{1j}^s\right) - R_f(x_{14}^s - x_{23}^s) \tag{S.1a}$$

$$X_{Ma}^{f'} = \left(\sum_{j=1}^4 x_{2j}^s\right) + R_f(x_{14}^s - x_{23}^s) \tag{S.1b}$$

$$X_{mA}^{f'} = \left(\sum_{j=1}^4 x_{3j}^s\right) + R_f(x_{14}^s - x_{23}^s) \tag{S.1c}$$

$$X_{ma}^{f'} = \left(\sum_{j=1}^4 x_{4j}^s\right) - R_f(x_{14}^s - x_{23}^s) \tag{S.1d}$$

which only involve the recombination rate between the **A** locus and the **M** locus in females (R_f). In males, recombination between the SDR and the **A** locus or the **M** also affects the frequencies of haplotypes produced. The frequency of haplotypes among X-bearing sperm/pollen (before haploid selection) in the next generation are given by

$$\begin{aligned} X_{MA}^{m'} = &\left(\sum_{j=1}^4 y_{1j}^s\right) - r_{MM}(y_{12}^s - y_{21}^s) \\ &- (R_m + r_{Mm} - 2\chi)(y_{13}^s - y_{31}^s) - (R_m + r_{Mm} - \chi)y_{14}^s \\ &+ (r_{Mm} - \chi)y_{41}^s + \chi\ y_{23}^s + (r_{Mm} - \chi)y_{32}^s \end{aligned} \tag{S.2a}$$

$$\begin{aligned} X_{Ma}^{m'} = &\left(\sum_{j=1}^{4} y_{2j}^{s}\right) - r_{MM}(y_{21}^{s} - y_{12}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{24}^{s} - y_{42}^{s}) - (R_m + r_{Mm} - \chi)y_{23}^{s} \\ &+ (r_{Mm} - \chi)y_{32}^{s} + \chi\ y_{14}^{s} + (r_{Mm} - \chi)y_{41}^{s} \end{aligned} \tag{S.2b}$$

$$\begin{aligned} X_{mA}^{m'} = &\left(\sum_{j=1}^{4} y_{3j}^{s}\right) - r_{mm}(y_{34}^{s} - y_{43}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{31}^{s} - y_{13}^{s}) - (R_m + r_{Mm} - \chi)y_{32}^{s} \\ &+ (r_{Mm} - \chi)y_{23}^{s} + \chi\ y_{41}^{s} + (r_{Mm} - \chi)y_{14}^{s} \end{aligned} \tag{S.2c}$$

$$\begin{aligned} X_{ma}^{m'} = &\left(\sum_{j=1}^{4} y_{4j}^{s}\right) - r_{mm}(y_{43}^{s} - y_{34}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{42}^{s} - y_{24}^{s}) - (R_m + r_{Mm} - \chi)y_{41}^{s} \\ &+ (r_{Mm} - \chi)y_{14}^{s} + \chi\ y_{32}^{s} + (r_{Mm} - \chi)y_{23}^{s} \end{aligned} \tag{S.2d}$$

and the frequencies of Y-bearing sperm/pollen haplotypes (before haploid selection) are given by

$$\begin{aligned} Y_{MA}^{m'} = &\left(\sum_{j=1}^{4} y_{1j}^{s}\right) - r_{MM}(y_{21}^{s} - y_{12}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{31}^{s} - y_{13}^{s}) - (R_m + r_{Mm} - \chi)y_{41}^{s} \\ &+ (r_{Mm} - \chi)y_{14}^{s} + \chi\ y_{32}^{s} + (r_{Mm} - \chi)y_{23}^{s} \end{aligned} \tag{S.3a}$$

$$\begin{aligned} Y_{Ma}^{m'} = &\left(\sum_{j=1}^{4} y_{2j}^{s}\right) - r_{MM}(y_{12}^{s} - y_{21}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{42}^{s} - y_{24}^{s}) - (R_m + r_{Mm} - \chi)y_{32}^{s} \\ &+ (r_{Mm} - \chi)y_{23}^{s} + \chi\ y_{41}^{s} + (r_{Mm} - \chi)y_{14}^{s} \end{aligned} \tag{S.3b}$$

$$\begin{aligned} Y_{mA}^{m'} = &\left(\sum_{j=1}^{4} y_{3j}^{s}\right) - r_{mm}(y_{43}^{s} - y_{34}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{13}^{s} - y_{31}^{s}) - (R_m + r_{Mm} - \chi)y_{23}^{s} \\ &+ (r_{Mm} - \chi)y_{32}^{s} + \chi\ y_{14}^{s} + (r_{Mm} - \chi)y_{41}^{s} \end{aligned} \tag{S.3c}$$

$$\begin{aligned} Y_{ma}^{m'} = &\left(\sum_{j=1}^{4} y_{4j}^{s}\right) - r_{mm}(y_{34}^{s} - y_{43}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{24}^{s} - y_{42}^{s}) - (R_m + r_{Mm} - \chi)y_{14}^{s} \\ &+ (r_{Mm} - \chi)y_{41}^{s} + \chi\ y_{23}^{s} + (r_{Mm} - \chi)y_{32}^{s} \end{aligned} \tag{S.3d}$$

Invasion of recombination modifiers

Invasion of modifiers that create a strong linkage between the X and a allele is determined by the largest solution to the characteristic polynomial

$$\lambda_{Xa}{}^2 - \lambda_{Xa}\bar{w}_{Xa}^{mat,f}/\bar{w}^f - (\bar{w}_{Xa}^{pat,f}/\bar{w}^f)(\bar{w}_{Xa}^{mat,m}/\bar{w}^m) = 0. \tag{S.4}$$

This can be solved for λ_{Xa} if we assume that the selected locus is initially loosely linked to the SDR (r_{MM}) and that there are no sex differences in selection ($w_{ij}^m = w_{ij}^f = w_{ij}$). The equilibrium frequency of the A allele when maintained at a polymorphic equilibrium by selection is then

$$\hat{p}_{Xm} = \hat{p}_{Ym} = \hat{p}_{Xf} = \frac{2w_a w_{aa} - w_{Aa}(w_A + w_a)}{2\left(w_A(w_{AA} - w_{Aa}) + w_a(w_{aa} - w_{Aa})\right)}. \tag{S.5}$$

This equilibrium is valid and stable when

$$\begin{aligned} &w_{Aa}(w_A + w_a) > 2w_A w_{AA} \text{ and} \\ &w_{Aa}(w_A + w_a) > 2w_a w_{aa}. \end{aligned} \tag{S.6}$$

Therefore, a polymorphism can be maintained either if there is heterozygote advantage in diploids ($w_{Aa} > w_{aa}$ and $w_{Aa} > w_{AA}$) or if there is antagonistic selection between haploids and diploids (e.g., $w_A > w_a$ and $w_{aa} > w_{Aa} > w_{AA}$) or a combination of both (Immler et al. 2012).

After this equilibrium is reached, the invasion of a modifier that brings the A allele into linkage with the Y is given by

$$\lambda_{YA} = 1 + \frac{(w_A - w_a)w_{Aa}(w_A + w_a)(w_{Aa}(w_A + w_a) - 2w_{AA}w_A)}{(w_A + w_a)({w_{Aa}}^2(w_A + w_a)^2 - 4w_A w_{AA} w_a w_{aa})}, \tag{S.7}$$

where $\lambda_{YA} > 1$ indicates that the modifier increases in frequency. Given that a polymorphism at the **A** locus is initially stable (conditions S.6 are met) the sign of $\lambda_{YA} - 1$ depends on the sign of $w_A - w_a$. That is, modifiers that bring the allele favoured in haploids (e.g., A when $w_A > w_a$) into tight linkage with the Y will spread.

Similarly, condition **2** for the invasion of modifiers that bring the a allele into tight linkage with the X chromosome is satisfied if

$$\frac{(w_A - w_a)w_{Aa}(w_A + w_a)(w_{Aa}(w_A + w_a) - 2w_{AA}w_A)}{2(w_A + w_a)(w_{Aa}(w_A + w_a) - w_A w_{AA} - w_a w_{aa})} > 0, \tag{S.8}$$

which requires $w_A > w_a$, given that conditions **S.6** are met. These results indicate that recombination modifiers invade if they bring the X into tight linkage with the allele that is less fit during haploid selection, even without the weak selection assumptions in equation **4** and without sex differences in selection in the diploid phase.

In the main text and above, we consider the invasion of recombination suppressors that bring the a allele into tight linkage with the X when the **A** locus is initially loosely linked to the SDR ($r_{MM} = 1/2$) such that $\hat{p}_{Xm} = \hat{p}_{Ym}$. Here, we consider cases where $r_{MM} < 1/2$ and define the difference in the frequency of the A allele between X- and Y-bearing pollen/sperm as $\delta_{XY} = \hat{p}_{Ym} - \hat{p}_{Xm}$. We assume that selection is weak relative to recombination such that δ, δ_{XY}, and δ_H are all small (of order ϵ^2). Invasion is then given by

$$\lambda'_{Xa} = \lambda_{Xa}\left(1 - (1 - 2\ r_{MM})(3 + 2w^f_{Aa}/\bar{w}^f)\right) + \frac{w^f_{Aa}\delta_{XY}}{3\bar{w}^f} \tag{S.9}$$

Under the conditions where $\lambda_{Xa} > 1$, we would expect that the a allele is associated with the X such that $\delta_{XY} < 0$. Thus, **S.9** indicates that selection in favour of modifiers that suppress recombination is less strong when $r_{MM} < 1/2$ ($\lambda'_{Xa} < \lambda_{Xa}$), in which case intralocus conflicts are initially partially resolved by reduced recombination.

Invasion of Modifiers That Increase Recombination from an Initially Low Level

We consider a population in which linkage is tight between the **A** locus and the SDR (r_{MM} is of order ϵ, where the M allele is initially fixed). Recombination has no effect if the **A** locus is fixed for one allele, we therefore focus on the five equilibria that maintain both A and a alleles, of which four are given to leading order by:

$$\begin{aligned}
&(A) \quad \hat{p}_{Ym} = 0,\ \hat{p}_{Xf} = \frac{\alpha}{\alpha + \beta},\ \hat{p}_{Xm} = \frac{w^m_{Aa}\alpha}{w^m_{Aa}\alpha + w^m_{aa}\beta}\\
&(A') \quad \hat{p}_{Ym} = 1,\ \hat{p}_{Xf} = 1 - \frac{\alpha'}{\alpha' + \beta'},\ \hat{p}_{Xm} = 1 - \frac{w^m_{Aa}\alpha'}{w^m_{Aa}\alpha' + w^m_{aa}\beta'}\\
&(B) \quad \hat{p}_{Ym} = 0,\ \hat{p}_{Xf} = 1,\ \hat{p}_{Xm} = 1\\
&(B') \quad \hat{p}_{Ym} = 1,\ \hat{p}_{Xf} = 0,\ \hat{p}_{Xm} = 0
\end{aligned}$$

$$\begin{aligned}
\alpha &= w^f_{Aa}(w^m_{aa}w_a + w^m_{Aa}w_A) - 2w^f_{aa}w^m_{aa}w_a\\
\alpha' &= w^f_{Aa}(w^m_{AA}w_A + w^m_{Aa}w_a) - 2w^f_{AA}w^m_{AA}w_A\\
\beta &= w^f_{Aa}(w^m_{aa}w_a + w^m_{Aa}w_A) - 2w^f_{AA}w^m_{Aa}w_A\\
\beta' &= w^f_{Aa}(w^m_{AA}w_A + w^m_{Aa}w_a) - 2w^f_{aa}w^m_{Aa}w_a
\end{aligned}$$

A fifth equilibrium (C) also exists where A is present at an intermediate frequency on the Y chromosome ($0 < \hat{p}_Y < 1$). However, equilibrium (C) is never locally stable when $r_{MM} \approx 0$ and is therefore not considered further. Thus, the Y can either be fixed for the a allele (equilibria A and B) or the A allele (equilibria A' and B'). The X chromosome can then either be polymorphic (equilibria A and A') or fixed for the alternative allele (equilibria B and B'). Since equilibria (A) and (B) are equivalent to equilibria (A') and (B') with the labelling of A and a alleles interchanged, we discuss only equilibria (A') and (B'), in which the YA haplotype is favoured (as in the previous section), without loss of generality.

We next calculate when (A') and (B') are locally stable for $r_{MM} = 0$. According to the 'small parameter theory' (Karlin and McGregor 1972*a*;*b*), these stability properties are unaffected by small amounts of recombination between the SDR and **A** locus, although equilibrium frequencies may be slightly altered. For the A allele to be stably fixed on the Y requires that $\bar{w}^m_{YA} > \bar{w}^m_{Ya}$, where the marginal fitnesses of YA and Ya haplotypes are $\bar{w}^m_{YA}$ (as above) and $\bar{w}^m_{Ya} = w^m_{Aa}p_{Xf} + w^m_{aa}(1 - p_{Xf})$, respectively. Substituting $\hat{p}_{Xf}$ from above, fixation of the A allele on the Y requires that $\gamma_i > 0$ where $\gamma_{(A')} = w_A(w^m_{Aa}\alpha' + w^m_{AA}\beta') - w_a(w^m_{aa}\alpha' + w^m_{Aa}\beta')$ for equilibrium (A') and $\gamma_{(B')} = w^m_{Aa}w_A - w^m_{aa}w_a$ for equilibrium (B'). Stability of a polymorphism on the X chromosome (equilibrium A') further requires that $\alpha' > 0$ and $\beta' > 0$. Fixation of the a allele on the X (equilibrium B') is

mutually exclusive with (A') and requires that $\beta' < 0$. We will assume that these conditions are met such that population has reached a stable equilibrium at the **A** locus when considering evolution at the modifier locus.

To consider recombination rate evolution, we evaluate whether a mutant allele, m, can invade if it modifies the recombination rate between **A** and the SDR by a small amount ($|r_{mm} - r_{MM}|$ and $|r_{Mm} - r_{MM}|$ are of order ϵ). As above, we use the leading eigenvalue, λ, from a local stability analysis to evaluate the spread of a rare mutant modifier, where now λ_i determines invasion into a population at equilibrium i. Firstly, because stability of equilibrium (A') requires that $\alpha' > 0$ and $\beta' > 0$ and all fitnesses must be non-negative, we can define the following series of κ terms, which must be positive when (A') is locally stable.

$$
\begin{aligned}
\kappa_1 &= w^f_{aa}\alpha' + w^f_{Aa}\beta' \\
\kappa_2 &= w^f_{Aa}\alpha' + w^f_{AA}\beta' \\
\kappa_3 &= w^m_{Aa}\alpha' + w^m_{AA}\beta' \\
\kappa_4 &= w^f_{aa}\alpha' + w^f_{AA}\beta' \\
\kappa_5 &= w^m_{Aa}w_a + w^m_{AA}w_A \\
\kappa_6 &= w^m_{Aa}w_a w^m_{AA}w_A \\
\kappa_7 &= w^f_{aa}w^m_{Aa}w_a\alpha' + w^f_{AA}w^m_{AA}w_A\beta' \\
\kappa_8 &= w^m_{aa}\alpha'\alpha' + 2w^m_{Aa}\alpha'\beta' + w^m_{AA}\beta'\beta' \\
\kappa_9 &= w^m_{Aa}w_a\alpha' + w^m_{AA}w_A\beta' \\
\kappa_{10} &= w^f_{Aa}\kappa_9 + 2\kappa_6\kappa_4/\kappa_5
\end{aligned}
$$

These are useful in determining the magnitude of $\lambda_{(A')}$, which determines invasion of modifiers and is given by

$$
\lambda_{(A')} = 1 + (r_{Mm} - r_{MM})\frac{w^m_{Aa}\alpha' K_1}{w_a R_m(w^m_{aa}\alpha' + w^m_{Aa}\beta')K_2} \tag{S.10}
$$

where we neglect terms of order ϵ^2 and higher and K_2 is strictly positive,

$$
\begin{aligned}
K_2 =& R_f 2w^f_{Aa}\kappa_3\kappa_5(\alpha' + \beta')\kappa_{10} + R_f R_m w^m_{Aa}w^m_{AA}2w_a w_A K_3\kappa_3\kappa_4/\kappa_5 \\
&+ R_m w^m_{Aa}w^m_{AA}(1 - 2R_f)\big(w_a\beta'\kappa_1(2w^m_{AA}w_A\kappa_2 + \kappa_{10}) + w_A\alpha'\kappa_2(2w^m_{Aa}w_a\kappa_1 + \kappa_{10})\big)
\end{aligned}
$$

such that $\lambda_{(A')} > 1$ if and only if $(r_{Mm} - r_{MM})K_1 > 0$, where

$$
\begin{aligned}
K_1 =& -(1 - 2R_f)R_m\gamma_{(A')}\kappa_1\kappa_2\kappa_6 - R_m R_f\gamma_{(A')}\kappa_4\kappa_6\big(\kappa_7/\kappa_5 + w^f_{Aa}(\alpha' + \beta')/2\big) \\
&- R_f\gamma_{(A')}w^f_{Aa}w_a\kappa_1\kappa_3\kappa_5 \\
&+ R_f w^f_{Aa}w^m_{Aa}(\gamma_{(A')}\alpha' + R_m w_a\kappa_8)\big((w^m_{Aa} - w^m_{AA})w_a w_A\kappa_4 + (w_A - w_a)w^f_{Aa}\kappa_5(\alpha' + \beta')/2\big)
\end{aligned}
$$

Modifiers that increase recombination ($r_{Mm} - r_{MM} > 0$) therefore only spread if $K_1 > 0$. Only the last term of K_1 can be positive, and this term can only be positive if either $w^m_{Aa} > w^m_{AA}$ or $w_A > w_a$. Thus, for increased recombination to be favoured by selection ($K_1 > 0$), heterozygous males must be more fit that males homozygous for the allele fixed on the Y and/or the allele fixed on the Y must be favoured during haploid selection. Since the A allele is fixed on the Y, $w^m_{Aa} > w^m_{AA}$ implies that X chromosomes bearing the a allele are favoured during selection in males. If a polymorphism is maintained on the X (equilibrium A'), counter-selection must favour the A allele during haploid selection and/or selection in females when $w^m_{Aa} > w^m_{AA}$. In addition, when linkage between the modifier locus and the selected locus is tight (at least in females, $R_f = 0$), K_1 is always negative and increased recombination is never favoured.

We next consider the invasion of a recombination modifier into a population at equilibrium (B'). Local stability of this equilibrium requires that $(-\beta') > 0$ and $\gamma_{(B')} > 0$. Ignoring terms of order ϵ^2 and higher,

$$
\lambda_{(B')} = 1 + \frac{(r_{Mm} - r_{MM})K_4}{4(\gamma_{(B')} + R_m w^m_{aa}w_a)((-\beta') + w^f_{Aa}(R_f w^m_{Aa}w_a + R_m w^m_{AA}w_A(1 - R_f))}
$$

where

$$
\begin{aligned}
K_4 =& -2\gamma_{(B')}(-\beta') - (2R_f + R_m(1 - R_f))w^f_{Aa}w^m_{AA}w_A\gamma_{(B')} \\
&- R_m(-\beta')w^m_{aa}w_a \\
&+ R_f(w_A - w_a)w^f_{Aa}w^m_{Aa}(2\gamma_{(B')} + R_m w^m_{aa}w_a) \\
&+ R_f R_m(w^m_{Aa} - w^m_{AA})w^f_{Aa}w^m_{Aa}w_a w_A
\end{aligned}
$$

Therefore $\lambda_{(B')} > 1$ if and only if $(r_{Mm} - r_{MM})K_4 > 0$. The only terms in K_4 that can be positive again involve the factors $(w_A - w_a)$ and $(w^m_{Aa} - w^m_{AA})$, such that either $w^m_{Aa} > w^m_{AA}$ or $w_a > w_A$ are again necessary (but not sufficient) conditions for the invasion of modifiers that increase recombination.

Finally, we re-write the condition $K_4 > 0$ to obtain

$$w^f_{aa} < w^f_{Aa}\left(1 - \gamma_{(B')}R_f(2 - R_m)R_m) - \gamma_{(B')}(w^m_{Aa} - w^m_{AA})K_5 + (w_A - w_a)K_6\right)/K_7 \tag{S.11}$$

where the following terms are positive

$$\begin{aligned}
K_5 =& (1 - R_f)(2\gamma_{(B')}(1 - R_m) + R_m w^m_{Aa} w_a)/w^m_{Aa} \\
K_6 =& (R_f R_m w_A {w^m_{Aa}}^2 + (w^m_{AA}(1 - R_f) + R_f w^m_{Aa})(2\gamma_{(B')}(1 - R_m) + w^m_{Aa} w_A R_m)) \\
K_7 =& 4\gamma_{(B')} + 2w^m_{aa} w_a R_m
\end{aligned}$$

Thus, if haploid selection favours the A allele, then condition **S.11** can be met whether selection among diploid females favours allele A or a ($w^f_{aa} < w^f_{Aa}$ or $w^f_{aa} > w^f_{Aa}$). However, if haploid selection favours the a allele ($w_a > w_A$), the evolution of increased recombination requires that $w^m_{Aa} > w^m_{AA}$ (see above), and equation **S.11** shows that selection must favour the A allele during selection in females ($w^f_{aa} < w^f_{Aa}$). Thus, increased recombination is only favoured if the A allele is favoured during selection in females ($w^f_{aa} < w^f_{Aa}$) and/or the A allele is favoured during haploid selection ($w_A > w_a$). Only under these conditions is it possible for recombination between the XA and Ya to produce XA gametes that are favoured over the short term (in daughters and/or gametes/gametophytes, respectively).

One might not expect selection to favour XA haplotypes because an A allele on an average X background should either have the same fitness as an a allele (when a polymorphism is maintained, equilibrium A') or lower fitness (when A is fixed, equilibrium B'). However, an XA haplotype created by recombination in males is found in a male haploid (pollen or sperm), not on an average X background (which is weighted across X-bearing male sperm/pollen and female eggs/ovules). Increased recombination does not evolve if R_f and R_m are small because the modifier remains linked to the haplotypes it creates, which will eventually be found on all backgrounds. However, when R_f and R_m are sufficiently large, modifiers that increase recombination can gain a transient fitness advantage. XA pollen/sperm haplotypes can gain a transient fitness advantage during haploid selection and/or selection in females. The evolution of increased recombination is only consistent with this form of selection.

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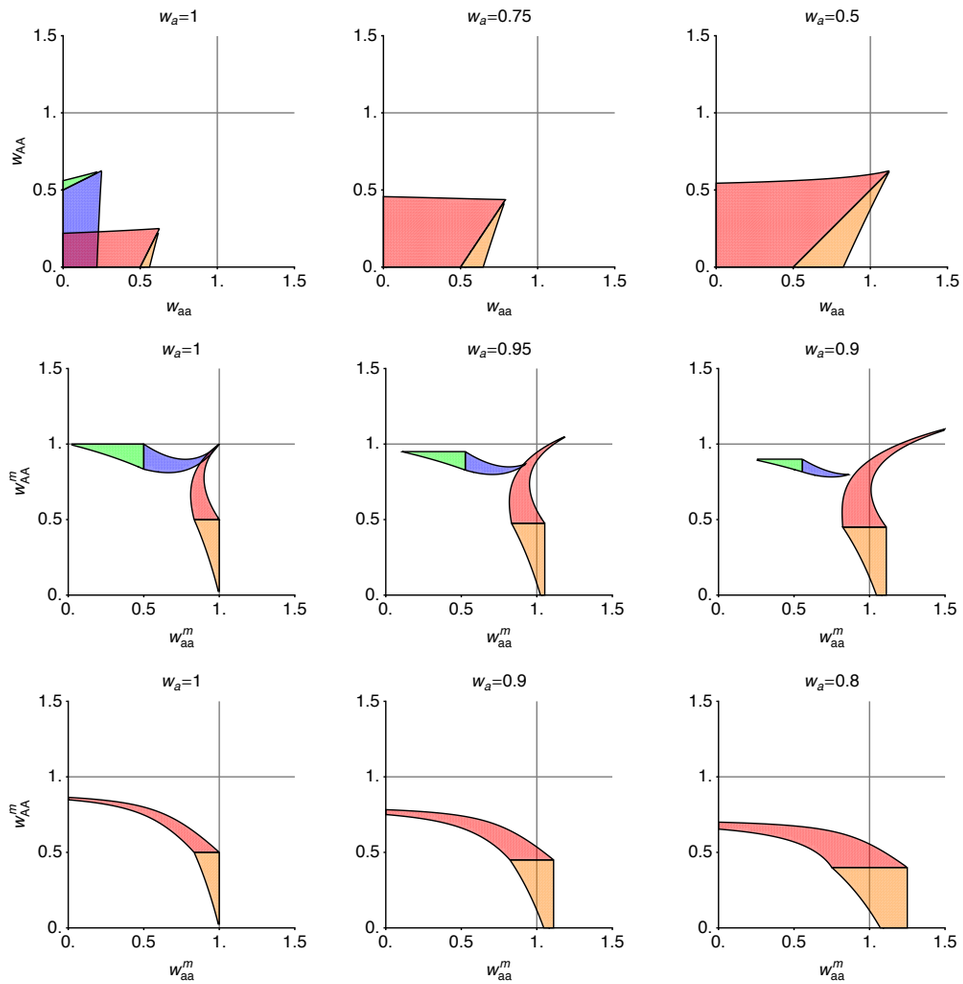

Fig. S.1. Selection can favour increased recombination between the sex-determining region (SDR) and a selected locus that is closely linked ($r_{ij} \approx 0$), even when selection in males is not overdominant. Coloured regions show where increased recombination is favoured in a population at equilibrium (A) in blue, (B) in green, (A') in red, and (B') in orange. Since this model is symmetrical, red/orange regions can be exchanged with blue/green regions if the labelling of A and a alleles is switched. Across columns we vary the fitness of a-bearing haploids relative to the A-bearing haploids ($w_A = 1$). Grey lines show the fitness of heterozygous diploids $w^k_{ij} = 1$. In the first row, there are no differences in selection between male and female diploids ($w^f_{ij} = w^m_{ij} = w_{ij}$), where w_{aa} and w_{AA} are varied along the x and y axes, respectively. As haploid selection becomes stronger, increased recombination can evolve with weaker overdominance in diploids and also with ploidally antagonistic selection ($w_{aa} > 1 > w_{AA}$). In the second and third rows, we consider sex differences in selection, where w^m_{aa} and w^m_{AA} are varied along the x and y axes ($w^m_{Aa} = 1$). In the second row, where selection in females is overdominant ($w^f_{AA} = 0.75$, $w^f_{Aa} = 1$, $w^f_{aa} = 0.75$), increased recombination can be favoured when selection is directional (or underdominant) in males and haploid selection is moderately strong. In the third row, selection favours the A allele in females ($w^f_{AA} = 1.05$, $w^f_{Aa} = 1$, $w^f_{aa} = 0.75$) and increased recombination can also be favoured with sexually antagonistic selection ($w^m_{AA} < 1 < w^m_{aa}$). For this plot, we assume that the modifier of recombination is unlinked ($R_f = R_m = 1/2$).