

# Haploid Selection, Sex Ratio Bias, and Transitions Between sex-determination systems

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Contributions:

## Abstract

2 Sex-determination systems are remarkably dynamic; many taxa display  
shifts in the location of sex-determining loci or the evolution of entirely  
4 new sex-determining systems. Predominant theories for why we observe  
such transitions generally conclude that novel sex-determining systems are  
6 favoured by selection if they equalise the sex ratio or increase linkage with  
a sexually-antagonistic locus. We use population genetic models to extend  
8 these theories in two ways: (1) We explicitly consider how selection on very  
tightly sex-linked loci influences the spread of novel sex-determiners. We  
10 find that tightly sex-linked genetic variation can favour the spread of new  
sex-determination systems in which the heterogametic sex changes (XY to  
12 ZW or ZW to XY) and the new sex-determining region is less closely linked  
(or unlinked) to the sex-linked locus under selection; a result that is not found  
14 with loose sex-linkage. (2) We also consider selection upon haploid geno-  
types either during gametic competition (e.g., pollen/sperm competition) or  
16 meiosis (i.e., non-Mendelian segregation); selective processes that typically  
occur in one sex or the other. With haploid selection, we again find that  
18 transitions between male and female heterogamety can occur even if the new  
sex-determining region is less closely linked to the locus under selection, and  
20 when linkage is tight haploid selection in the heterogametic sex can cause  
strong sex ratio bias, which may increase or decrease with the spread of new  
22 sex chromosomes. These results indicate that favourable associations that de-  
velop between the ancestral sex-determining locus and selected loci can be  
24 broken during the spread of a new sex-determining region. Overall, our mod-  
els provide new predictions for the types of selection and the genomic loca-  
26 tion of loci that can drive transitions between sex-determination systems.

## Introduction

28 Animals and angiosperms exhibit extremely diverse sex-determination systems  
(reviewed in Bull 1983, Charlesworth and Mank 2010, Beukeboom and Perrin  
30 2014, Bachtrog et al. 2014). Among species with genetic sex determination of  
diploid sexes, some taxa have heterogametic males (XY) and homogametic fe-  
32 males (XX), including mammals and most dioecious plants (Ming et al. 2011);  
whereas other taxa have homogametic males (ZZ) and heterogametic females (ZW),  
34 including Lepidoptera and birds. Within several taxa, the chromosome that har-  
bours the master sex-determining region changes. For example, transitions of the  
36 master sex-determining gene between chromosomes or the evolution of new mas-  
ter sex-determining genes have occurred in Salmonids (Li et al. 2011, Yano et al.  
38 2012), Diptera (Vicoso and Bachtrog 2015), and *Oryzias* (Myosho et al. 2012). In  
addition, many gonochoric clades with genetic sex determination exhibit transi-  
40 tions between male (XY) and female (ZW) heterogamety, including snakes (Gam-  
ble et al. 2017, *Current Biology*), lizards (Ezaz et al. 2009), eight of 26 teleost  
42 fish families (Mank et al. 2006), true fruit flies (Tephritids, Vicoso and Bachtrog  
2015), amphibians (Hillis and Green 1990), the angiosperm genus *Silene* (Slancar-  
44 ova et al. 2013), and Coleoptera and Hemiptera (Beukeboom and Perrin 2014, plate  
2). Indeed, in some cases, both male and female heterogametic sex-determination  
46 systems can be found in the same species, as exhibited by some cichlid species (Ser  
et al. 2010) and *Rana rugosa* (Ogata et al. 2007). In addition, multiple transitions  
48 have occurred between genetic and environmental sex-determination systems, e.g.,  
in reptiles and fishes (Conover and Heins 1987, Mank et al. 2006, Pokorná and  
50 Kratochvíl 2009, Ezaz et al. 2009, Pen et al. 2010, Holleley et al. 2015).

Predominant theories accounting for the spread of new sex-determination sys-  
52 tems by selection involve fitness differences between sexes (e.g., sexually antag-  
onistic selection) or sex-ratio selection. van Doorn and Kirkpatrick (2007; 2010)  
54 show that new sex-determining loci can be favoured if they arise in closer link-  
age with a locus that experiences sexual antagonism. Tighter linkage allows a  
56 stronger favourable association to build up between a male-beneficial allele, and

a neo-Y chromosome, for example. Such associations can favour a new master  
58 sex-determining gene on a new chromosome (van Doorn and Kirkpatrick 2007)  
and can also favour a transition between male and female heterogamety (e.g., a  
60 ZW to XY transition, van Doorn and Kirkpatrick 2010). However, any sexually-  
antagonistic loci that are more closely linked to the ancestral sex-determination  
62 locus will develop similar, favourable associations and hinder the spread of a new  
sex-determination system.

64 The sex ratio is directly affected by the sex-determination system, and it has  
therefore been suggested that sex-ratio selection is a dominant force in the evolu-  
66 tion of sex determination (e.g., Bull 1983, p 66-67; Beukeboom and Perrin 2014,  
Chapter 7). ‘Fisherian’ sex-ratio selection favours a 1:1 zygotic sex ratio when as-  
68 suming that males and females are equally costly to produce (Fisher 1930, Charnov  
1982). This follows from the fact that, for an autosomal locus, half of the ge-  
70 netic material is inherited from a male and half from a female (West 2009). Thus,  
if the population sex ratio is biased towards one sex, the average per-individual  
72 contribution of genetic material to the next generation from the opposite sex is  
greater. Therefore, a mutant that increases investment in the rarer sex will spread  
74 via the higher per-individual contributions made by that sex. In the case of sex-  
chromosome evolution, Kozielska et al. (2010) consider systems in which the an-  
76 cestral sex chromosomes experience meiotic drive (e.g., where driving X or Y  
chromosomes are inherited disproportionately often), which causes sex ratios to  
78 become biased (Hamilton 1967). They find that new, unlinked sex-determining  
loci (masculinizing or feminizing mutations, i.e., neo-Y or neo-W loci) can then  
80 spread, which restore an even sex ratio.

Here we extend current theory by using mathematical models to find the con-  
82 ditions under which new sex-determination systems spread when individuals ex-  
perience selection at both diploid and haploid stages. Even in animal and plant  
84 species that have much larger and more conspicuous diploid phases than haploid  
phases, many loci experience significant haploid selection through gamete compe-  
86 tition and/or meiotic drive (Mulcahy et al. 1996, Joseph and Kirkpatrick 2004). We

use the term ‘meiotic drive’ to refer to the biased (non-Mendelian) segregation of  
 88 genotypes during gamete production (from one parent) and the term ‘gametic com-  
 petition’ to refer to selection upon haploid genotypes within a gamete/gametophyte  
 90 pool (potentially from multiple parents); the term ‘haploid selection’ encompasses  
 both processes. Meiotic drive generally occurs either during the production of  
 92 male or female gametes only (Úbeda and Haig 2005, Lindholm et al. 2016). Be-  
 cause there are typically many more pollen/sperm than required for fertilization,  
 94 gametic competition is also typically sex specific, occurring primarily among male  
 gametes. Gametic competition may be particularly common in plants, in which 60-  
 96 70% of all genes are expressed in the male gametophyte and these genes exhibit  
 stronger signatures of selection than random genes (Borg et al. 2009, Arunkumar  
 98 et al. 2013, Gossmann et al. 2014). In addition, artificial selection pressures ap-  
 plied to male gametophytes are known to cause a response to selection (e.g., Hor-  
 100 maza and Herrero 1996, Ravikumar et al. 2003, Hedhly et al. 2004, Clarke et al.  
 2004). A smaller proportion of genes are thought to be expressed and selected dur-  
 102 ing competition in animal sperm, although precise estimates are uncertain (Zheng  
 et al. 2001, Joseph and Kirkpatrick 2004, Vibrationovski et al. 2010). Recent stud-  
 104 ies have demonstrated that sperm competition can alter haploid allele frequencies  
 and increase offspring fitness (Immler et al. 2014) (Alavioon et al. 2017). Ge-  
 106 netic mapping experiments, which are typically designed to minimize selection in  
 diploids, have revealed segregation distortion in various species, including mice,  
 108 *Drosophila*, Rice, Maize, Wheat, Barley, Cotton... In some of these cases, biased  
 segregation has been shown to be attributable to meiotic drive and/or gametic se-  
 110 lection (Leppala et al. 2013, Didion et al. 2015, 2016 Xu et al 2013 (rice), Fish-  
 man...).

112 There are various ways in which a period of haploid selection could influence  
 transitions between sex-determination systems. If we assume that haploid selec-  
 114 tion at any particular locus predominantly occurs in one sex (e.g., meiotic drive  
 during spermatogenesis), then such loci experience a form of sex-specific selec-  
 116 tion. In this respect, we might expect that haploid selection would affect transitions

between sex-determination systems in a similar manner to sex-specific diploid selection (as explored by van Doorn and Kirkpatrick 2007; 2010). That is, new masculinizing mutations (neo-Y chromosomes) could be favoured via associations with alleles that are beneficial in the male haploid stage. On the other hand, sex ratios can also become biased by linkage between the sex-determining region and a locus that harbours genetic variation in haploid fitness. For example, there are several known cases of sex-ratio bias caused by sex-linked meiotic drive alleles (Burt and Trivers 2006, Chapter 3) or selection among X- and Y-bearing pollen (Lloyd 1974, Conn and Blum 1981, Stehlik and Barrett 2005; 2006, Field et al. 2012; 2013). It is not immediately clear how the spread of new sex-determination systems would be influenced by the combination of sex-ratio biases and associations between haploid selected loci and sex-determining regions.

Our models have two important new features. Firstly, when considering loci that are under selection and also in very tight linkage with the ancestral sex-determining region we explicitly calculate equilibrium allele frequencies. This allows us to show that transitions between male and female heterogamety can evolve even when the neo-sex-determining locus is less closely linked to a locus under selection and therefore disrupts favourable ancestral associations between sex and the alleles selected in that sex. Secondly, we allow sex-specific haploid selection to occur on a locus in tight or loose linkage with the ancestral sex-determining region. We find that sex-ratio biases caused by haploid selection can exert Fisherian sex-ratio selection upon novel sex-determiners but that their spread is also determined by selection on genetically-associated alleles. Consequently, it is possible for selection on linked alleles to drive turnover between sex-determining systems despite causing transitory or even permanent increases in sex-ratio bias.

## Model

Change all  $\alpha^\phi$  to  $(1 + \alpha_\Delta^\phi)$ .

We consider transitions between ancestral and novel sex-determining systems

146 using a three-locus model, each locus having two alleles. Locus **X** is the ancestral  
 sex-determining region, with alleles  $X$  and  $Y$  (or  $Z$  and  $W$ ). Locus **A** is a locus  
 under selection, with alleles  $A$  and  $a$ . Locus **M** is a novel sex-determining region,  
 148 at which the null allele ( $M$ ) is initially fixed in the population such that sex of  
 zygotes is determined by the genotype at the ancestral sex-determining region, **X**;  
 150  $XX$  genotypes become females and  $XY$  become males (or  $ZW$  become females  
 and  $ZZ$  become males). To evaluate the evolution of new sex-determination sys-  
 152 tems, we consider the invasion, fixation, maintenance, and/or loss of novel sex-  
 determining alleles ( $m$ ) at the **M** locus. We assume that the **M** locus is epistatically  
 154 dominant over the **X** locus such that zygotes with at least one  $m$  allele develop as  
 females with probability  $k$  and as males with probability  $1 - k$ , regardless of the  
 156 **X** locus genotype. With  $k = 0$ , the  $m$  allele is a masculinizer (i.e., a neo- $Y$ ) and  
 with  $k = 1$  the  $m$  allele is a feminizer (i.e., a neo- $W$ ). With intermediate  $k$ , we can  
 158 interpret  $m$  as an environmental sex determination (ESD) allele, such that zygotes  
 develop as females in a proportion ( $k$ ) of the environments they experience. We  
 160 also analyze a model of maternally-controlled environmental sex-determination,  
 where mothers with at least one  $m$  allele produce daughters with probability  $k$ .

162 In each generation, we census the genotype frequencies in male and female  
 gametes/gametophytes (hereafter gametes) before gametic competition. A full de-  
 164 scription of our model, including recursion equations, is given in the Appendix.  
 First, competition occurs among male gametes (sperm/pollen competition) and  
 166 among female gametes (egg/ovule competition) separately. Selection during ga-  
 metic competition depends on the **A** locus genotype, relative fitnesses are given  
 168 by  $w_A^{\varnothing}$  and  $w_a^{\varnothing}$  ( $\varnothing \in \{\varnothing, \delta\}$ ; see table 1). We assume that all gametes compete for  
 fertilization during gametic competition, which assumes a polygamous mating sys-  
 170 tem. Gametic competition in monogamous mating systems is, however, equivalent  
 to meiotic drive in our model (described below), as both only alter the frequency  
 172 of gametes produced by heterozygotes. After gametic competition, random mating  
 occurs between male and female gametes. The resulting zygotes develop as males  
 174 or females, depending on their genotypes at the **X** and **M** loci. Diploid males and

females then experience selection, with relative fitnesses  $w_{AA}^{\phi}$ ,  $w_{Aa}^{\phi}$ , and  $w_{aa}^{\phi}$ . The  
 176 next generation of gametes is produced by meiosis, during which recombination  
 and sex-specific meiotic drive can occur. Recombination (i.e., an odd number of  
 178 cross-overs) occurs between loci **X** and **A** with probability  $r$ , between loci **A** and  
**M** with probability  $R$ , and between loci **X** and **M** with probability  $\chi$ . Any linear  
 180 order of the loci can be modelled with appropriate choices of  $r$ ,  $R$ , and  $\chi$  (see Ta-  
 ble S.1). Individuals that are heterozygous at the **A** locus may experience meiotic  
 182 drive; a gamete produced by  $Aa$  heterozygotes of sex  $\phi$  bear allele  $A$  with probab-  
 ility  $\alpha^{\phi}$ . Thus, the **A** locus can experience sex-specific gametic competition, diploid  
 184 selection, and/or meiotic drive.

Table 1: Relative fitness of different genotypes in sex  $\phi \in \{\varnothing, \delta\}$

Genotype	Relative fitness during gametic competition
A	$w_A^{\phi} = 1 + t^{\phi}$
a	$w_a^{\phi} = 1$
Genotype	Relative fitness during diploid selection
AA	$w_{AA}^{\phi} = 1 + s^{\phi}$
Aa	$w_{Aa}^{\phi} = 1 + h^{\phi} s^{\phi}$
aa	$w_{aa}^{\phi} = 1$
Genotype	Transmission during meiosis in $Aa$ heterozygotes
A	$\alpha^{\phi} = 1/2 + \alpha_{\Delta}^{\phi}/2$
a	$1 - \alpha^{\phi} = 1/2 - \alpha_{\Delta}^{\phi}/2$

## Results

186 The model outlined above describes both ancestrally- $XY$  and ancestrally- $ZW$   
 sex-determination systems if we relabel the two sexes as being ancestrally ‘het-  
 188 erogametic’ or ancestrally ‘homogametic’. Without loss of generality, we primar-  
 ily refer to the ancestrally heterogametic sex as male and the ancestrally homoga-



190 metic sex as female. That is, we describe an ancestral XY sex-determination system but our model is equally applicable to an ancestral ZW sex-determination system (relabelling the ancestrally-heterogametic sex as female and the ancestrally-homogametic sex as male).

#### 194 **Generic invasion by a neo-Y or neo-W**

The evolution of a new sex-determination system requires that a rare mutant allele at the novel sex-determining locus,  $m$ , increases in frequency when rare. The spread of a rare mutant  $m$  at the  $\mathbf{M}$  locus is determined by the leading eigenvalue,  $\lambda$ , of the system of eight equations describing the frequency of eggs and sperm carrying the  $m$  allele in the next generation (equations S.1). This system simplifies substantially in a number of cases of interest. Dominant neo-Y (when  $k = 0$ ) or neo-W alleles (when  $k = 1$ ) are only found in male diploids (neo-Y) or female diploids (neo-W) such that their growth rate ultimately depends only on the change in frequency of  $m$ -bearing gametes produced by males or by females, respectively. Furthermore, if the  $m$  allele is fully epistatically dominant over the ancestral sex-determining system, phenotypes are not affected by the genotype at the ancestral sex-determining region ( $\mathbf{X}$  locus). Thus, the invasion of rare dominant neo-Y or neo-W alleles is determined by the largest eigenvalue that solves a quadratic characteristic polynomial,  $\lambda^2 + b\lambda + c = 0$ . Here,  $b = -(\lambda_{mA} + \lambda_{ma}) + (\rho_{mA} + \rho_{ma})$  and  $c = (\lambda_{mA} - \rho_{mA})(\lambda_{ma} - \rho_{ma}) - \rho_{mA}\rho_{ma}$ , where  $\lambda_{mi}$  is the multiplicative growth rate of mutant haplotypes on background  $i \in \{A, a\}$ , without accounting for loss due to recombination, and  $\rho_{mi}$  is the rate at which mutant haplotypes on background  $i \in \{A, a\}$  recombine onto the other  $\mathbf{A}$  locus background in heterozygotes (see Table 2). The  $\lambda_{mi}$  and  $\rho_{mi}$ , and thus the spread of the mutant  $m$  allele, depend on the frequency of alleles at the  $\mathbf{A}$  and  $\mathbf{X}$  loci in the ancestral population. In the ancestral population, it is convenient to follow the frequency of the  $A$  allele among female gametes (eggs),  $p_X^\phi$ , and among X-bearing,  $p_X^\delta$ , and among Y-bearing,  $p_Y^\delta$ , male gametes (sperm/pollen). We also track the fraction of male gametes that are Y-bearing,  $q$ , which may deviate from  $1/2$  due to meiotic drive in males.

Table 2: Parameters determining invasion of mutant neo-Y and neo-W alleles into an ancestrally XY system

neo-Y ( $k = 0$ )
$\lambda_{mA} = \{2(1 - \zeta)\}^{-1} [p_X^\varnothing w_A^\varnothing w_A^\delta w_{AA}^\delta + 2(1 - p_X^\varnothing) w_a^\varnothing w_A^\delta w_{Aa}^\delta \alpha^\delta] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\delta)$ $\lambda_{ma} = \{2(1 - \zeta)\}^{-1} [(1 - p_X^\varnothing) w_a^\varnothing w_a^\delta w_{aa}^\delta + 2p_X^\varnothing w_A^\varnothing w_a^\delta w_{Aa}^\delta (1 - \alpha^\delta)] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\delta)$ $\rho_{mA} = R \{2(1 - \zeta)\}^{-1} [2(1 - p_X^\varnothing) w_a^\varnothing w_A^\delta w_{Aa}^\delta \alpha^\delta] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\delta)$ $\rho_{ma} = R \{2(1 - \zeta)\}^{-1} [2p_X^\varnothing w_A^\varnothing w_a^\delta w_{Aa}^\delta (1 - \alpha^\delta)] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\delta)$
neo-W ( $k = 1$ )
$\lambda_{mA} = (2\zeta)^{-1} [\bar{p}^\delta w_A^\delta w_A^\varnothing w_{AA}^\varnothing + 2(1 - \bar{p}^\delta) w_a^\delta w_A^\varnothing w_{Aa}^\varnothing \alpha^\varnothing] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\varnothing)$ $\lambda_{ma} = (2\zeta)^{-1} [(1 - \bar{p}^\delta) w_a^\delta w_a^\varnothing w_{aa}^\varnothing + 2\bar{p}^\delta w_A^\delta w_a^\varnothing w_{Aa}^\varnothing (1 - \alpha^\varnothing)] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\varnothing)$ $\rho_{mA} = R (2\zeta)^{-1} [2(1 - \bar{p}^\delta) w_a^\delta w_A^\varnothing w_{Aa}^\varnothing \alpha^\varnothing] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\varnothing)$ $\rho_{ma} = R (2\zeta)^{-1} [2\bar{p}^\delta w_A^\delta w_a^\varnothing w_{Aa}^\varnothing (1 - \alpha^\varnothing)] / (\bar{w}_H^\varnothing \bar{w}_H^\delta \bar{w}^\varnothing)$

$\bar{p}^\delta = (1 - q)p_X^\delta + qp_Y^\delta$  is the average frequency of the  $A$  allele among X- and Y-bearing male gametes.

$\zeta$  is the zygotic sex ratio (fraction female)

$\bar{w}^\varnothing$  is the mean fitness of diploids of sex  $\varnothing$ , see Table S.2

$\bar{w}_H^\varnothing$  is the mean fitness of haploids from sex  $\varnothing$ , see Table S.2

220

We are particularly concerned with the conditions under which a rare neo-sex-determining allele increases in frequency, which occurs when the largest eigenvalue,  $\lambda$ , is greater than one. If the average change in frequency of the two haplotypes that carry the  $m$  allele ( $Am$  and  $am$ ) is positive, invasion will always occur, i.e., if  $(\lambda_{mA} + \lambda_{ma})/2 > 1$  then  $\lambda > 1$ . If neither haplotype increases in frequency ( $\lambda_{mA}, \lambda_{ma} < 1$ ), the  $m$  allele will not invade. Otherwise, the new sex-determining allele increases in frequency on one **A** background and declines on the other, and invasion requires

228

$$\rho_{ma} (\lambda_{mA} - 1) + \rho_{mA} (\lambda_{ma} - 1) > 0. \quad (1)$$

For example, if we assume that only the  $mA$  haplotype has a positive growth rate

( $\lambda_{ma} < 1 < \lambda_{mA}$ ), the second term on the left-hand side of (1) is negative and

230

invasion requires that the growth rate of  $mA$  haplotypes and the rate at which they  
 232 are produced by recombination is sufficiently large relative to that of  $ma$  haplo-  
 types. In other words, invasion requires that the average growth rate of the two  
 234 haplotypes, weighted by the rates they are created by recombination, is positive.

Table 2 illustrates a number of key points about the invasion of neo-Y and  
 236 neo-W mutations. First, Fisherian sex-ratio selection will favour the spread of a  
 neo-Y if the ancestral zygotic sex ratio is biased towards females,  $\zeta > 1/2$  (i.e.,  
 238  $\zeta > 1/2$  causes the first factor of the  $\lambda_{mi}$  to be greater than one for a neo-Y and  
 vice versa for a neo-W). However, the spread of a neo-Y (neo-W) also depends on  
 240 the male (female) fitness of associated alleles (terms in square brackets). Second,  
 invasion by a neo-Y (neo-W) allele does not directly depend on the fitness of female  
 242 (male) diploids (for a given set of equilibrium allele frequencies). This is because  
 a dominant neo-Y (neo-W) is always found in males (females), and therefore the  
 244 frequency of the neo-Y (neo-W) allele,  $m$ , only changes in males (females). Finally,  
 invasions by a neo-Y and a neo-W are qualitatively different. This is because a  
 246 gamete with the ancestral- or neo-Y always pairs with a female gamete containing  
 an X, and both develop into males. By contrast, a gamete with a neo-W can pair  
 248 with an X or Y male gamete, developing into a female, while female gametes  
 without the neo-W can become female (when paired with X) or male (when paired  
 250 with Y). Consequently, the types of females produced differ in the frequency of  $A$   
 alleles they obtain from mating.

252 In order to explicitly determine the conditions under which a rare neo-sex-  
 determining allele spreads, we must calculate the equilibrium frequency of the  $A$   
 254 allele in the ancestral population (i.e.,  $\hat{p}_X^{\circ}$ ,  $\hat{p}_X^{\delta}$ , and  $\hat{p}_Y^{\delta}$ ). Since only the  $A$  locus expe-  
 riences selection directly, any deterministic evolution requires that there is a poly-  
 256 morphism at the  $A$  locus. Polymorphisms can be maintained by mutation-selection  
 balance or transiently present during the spread of beneficial alleles. However,  
 258 polymorphisms maintained by selection can maintain alleles at higher allele fre-  
 quencies for longer periods. Here, we focus of polymorphisms maintained by se-  
 260 lection, where the  $A$  allele reaches a stable intermediate equilibrium frequency

under the ancestral sex-determination system before the neo-sex-determining allele ( $m$ ) arises. We can analytically calculate the allele frequency of the  $A$  allele using two alternative simplifying assumptions: (1) the  $A$  locus is within (or tightly linked to) the non-recombining region around the ancestral SDR ( $r \approx 0$ ) or (2) selection is weak relative to recombination ( $s^\phi, t^\phi, \alpha_\Delta^\phi$  of order  $\epsilon \ll 1$ ).

Change to  $\hat{p}$  throughout as we assume that allele frequencies change slowly such that lambda is unaffected

### Tight linkage with the ancestral sex-determining region

The equilibrium allele frequencies and their stability conditions are given in the appendix, when there is complete linkage between the ancestral sex-determining region and the  $A$  locus ( $r = 0$ ). Here, either the  $A$  allele or the  $a$  allele must be fixed on the Y. Because the labelling of alleles is arbitrary, we will assume that the  $a$  locus is fixed on the Y ( $p_Y^\phi = 0$ ), without loss of generality. If there are two alleles maintained at the  $A$  locus, the X can either be fixed for the  $A$  allele ( $p_X^\phi = p_X^\phi = 1$ ) or polymorphic ( $0 < p_X^\phi, p_X^\phi < 1$ ).

A neo-Y will never invade an ancestral XY system that already has tight linkage with the locus under selection ( $r = 0$ , for details see supplementary *Mathematica* file). A neo-Y haplotype with the same allele as the ancestral Y is neutral ( $\lambda_{ma} = 1$ ) and does not change in frequency. The other neo-Y haplotype will not spread ( $\lambda_{mA} < 1$ ) given that the initial equilibrium is stable. Therefore, a neo-Y mutation cannot spread ( $\lambda \leq 1$ ) in an ancestral XY system that is at equilibrium with all selected loci within the non-recombining region around the SDR. In essence, through tight linkage with the  $A$  locus, the ancestral Y becomes strongly specialized on the allele that has the highest fitness across male haploid and diploid phases. Given that the ancestral Y is at this equilibrium, it is not possible for a neo-Y to create males that have higher fitness than the ancestral Y.

Sally edits only looked at up to this point. Next task: figures to match with this tight linkage section.

Neo-W alleles, on the other hand, can invade an ancestral XY system under

290 some conditions (the full invasion conditions are given in the appendix; equations  
 S.6 and S.7). Significantly, we note that it is possible for both neo-W haplotypes  
 292 to spread ( $\lambda_{mA} > 1$  and  $\lambda_{ma} > 1$ ), in which case neo-W invasion can occur re-  
 gardless of linkage to the selected locus. That is, selection on loci within the non-  
 294 recombining region of the SDR can favour the invasion of a less closely linked  
 neo-W (e.g., on an autosome). Although haploid selection can favour neo-W alle-  
 296 les because the ancestral sex ratio becomes male biased, this is not the only circum-  
 stance in which less tightly linked neo-W alleles invade. For example, unlinked  
 298 neo-W alleles can invade in the absence of any haploid selection. This result is  
 unexpected given the results of van Doorn and Kirkpatrick (2010), who did not ex-  
 300 plicitly calculate equilibrium allele frequencies under tight linkage and generally  
 concluded that heterogametic transitions occur when neo-sex-determining alleles  
 302 are in tighter linkage with loci under sex-specific diploid selection. Therefore, we  
 focus on cases where there is no haploid selection and discuss the effects of haploid  
 304 selection in the appendix.

If we categorise the  $a$  allele as being ancestrally ‘male-beneficial’ via the fact  
 306 that it is fixed on the Y, then  $\lambda_{mA} > 1$  indicates that the neo-W spreads when found  
 with the ancestrally ‘female-beneficial’ allele. Intuitively, this is possible because  
 308 the ancestral X chromosome is not able to specialise on the ‘female-beneficial’  
 allele due to the fact that X’s are sometimes found in males. For example, a poly-  
 310 morphism of  $A$  and  $a$  alleles can be maintained on the X despite directional se-  
 lection in favour of the  $A$  allele in females ( $s^{\varphi} > 0$ ,  $0 < h^{\varphi} < 1$ ) because the  $a$   
 312 allele is favoured in males. W- $A$  haplotypes will only create females with high  
 fitness  $AA$  or  $Aa$  genotypes and can therefore have higher fitness than ancestral  
 314 females, which sometimes also produce  $aa$  females. Thus, the neo-W can spread  
 by allowing increased specialization on female beneficial alleles.

316 Given that the  $a$  allele can be considered ancestrally ‘male-beneficial’ because  
 it is fixed on the Y, it might be surprising that neo-W- $a$  haplotypes can be favoured  
 318 by selection in females ( $\lambda_{ma} > 1$ ). Again, this occurs because ancestral X’s also  
 experience selection in males, in which they will always be paired with a Y- $a$ .

320 Hence, if there is overdominance in males, X-A Y-*a* males have high fitness and  
the *A* allele is favoured by selection on the X in males. Therefore, the X can be  
322 polymorphic or even fixed for the *A* allele despite favouring the *a* allele during  
selection in females (Lloyd and Webb 1977, Otto 2014). In such cases, neo-W-*a*  
324 haplotypes, which are never found in males, can spread because they both create  
more *Aa* and *aa* females when pairing with an X from males and they bring Y-*a*  
326 haplotypes into females, in which case females are always *aa*. Indeed, it is possible  
for both W-*A* and W-*a* haplotypes to spread, as is the case when *AA* individuals  
328 have low fitness in females yet the *A* is fixed on the X due to strong overdominance  
in males. Both neo-W-*A* and neo-W-*a* haplotypes then produce fewer unfit *AA*  
330 females. This is true for the neo-W-*A* haplotype because it can pair with a Y - *a*  
haplotype and still be female.

332 In Figure ??A we show the region of parameter space within which both neo-W  
haplotypes invade ( $\lambda_{mA} > 1$  and  $\lambda_{ma} > 1$ ) when there is overdominance in females  
334 and no haploid selection (corresponding to Figure 2a in Otto 2014). Wherever  
both haplotypes have positive growth rates (gray region of Figure ??), invasion  
336 by a neo-W is expected regardless of its linkage with the selected locus (i.e., even  
unlinked neo-W alleles can invade). In regions where only one haplotype can  
338 spread (white region of Figure ??), a neo-W can invade as long as equation (1) is  
satisfied, which can require that the recombination rate,  $R$ , is small enough and  
340 yet still indicates that more loosely linked sex-determining regions can spread. It  
is also possible for haploid selection to drive the invasion of a loosely linked neo-  
342 W. Take, for instance, selection directionally favouring *A* in both diploid sexes and  
meiotic drive in males. Figure ??B then shows that ploidy-antagonistic selection  
344 can allow both neo-W haplotypes to invade.

### Loose linkage with the ancestral sex-determining region

346 Assuming that selection is weak relative to all recombination rates ( $r$ ,  $R$  and  $\chi$ ),  
we denote the leading eigenvalues describing the invasion of a neo-Y ( $k = 0$ ) and  
348 a neo-W ( $k = 1$ ) into an ancestrally XY system by  $\lambda_{Y',XY}$  and  $\lambda_{W',XY}$ , respectively,

which are

$$\lambda_{Y',XY} = 1 + V_A S_A^2 \frac{(r-R)}{rR} + O(\epsilon^3) \quad (2)$$

350 and

$$\lambda_{W',XY} = \lambda_{Y',XY} + (2\alpha_\Delta^\delta - 2\alpha_\Delta^\varphi + t^\delta - t^\varphi) (\hat{p}_Y^\delta - \hat{p}_X^\delta) / 2 + O(\epsilon^3) \quad (3)$$

where  $V_A = \bar{p}(1 - \bar{p})$  is the variance in the frequency of  $A$  and  $S_A = (D^\delta + \alpha_\Delta^\delta + t^\delta) - (D^\varphi + \alpha_\Delta^\varphi + t^\varphi)$  describes sex differences in selection for the  $A$  versus  $a$  across diploid selection, meiosis, and gametic competition. The diploid selection term,  $D^\varphi = (\bar{p}s^\varphi + (1 - \bar{p})h^\varphi s^\varphi) - (\bar{p}h^\varphi s^\varphi + (1 - \bar{p}))$ , is the difference in fitness between  $A$  and  $a$  alleles in diploids of sex  $\varphi \in \{\varphi, \delta\}$ , where  $\bar{p}$  is the leading-order probability of mating with an  $A$ -bearing gamete from the opposite sex (see Appendix).

The neo-sex-determining allele,  $m$ , will spread if  $\lambda_{m,XY} > 1$ . Equation (2) demonstrates that under weak selection a neo-Y will invade an XY system if and only if it is more closely linked to the selected locus than the ancestral sex-determining region (i.e., if  $R < r$ ; note that  $V_A$  and  $S_A^2$  are strictly positive as long as  $\mathbf{A}$  is polymorphic). This echoes our tight linkage results above and the results of van Doorn and Kirkpatrick (2007), who considered diploid selection only and also found that homogametic transitions (XY to XY or ZW to ZW) can only occur when the neo-sex-determining locus is more closely linked to a locus under sexually-antagonistic selection.

Equation (3) shows that, in contrast to the tight linkage results of the previous section, with weak selection and no haploid selection ( $t^\varphi = \alpha_\Delta^\varphi = 0$ ), as considered by van Doorn and Kirkpatrick (2010), the spread of a neo-W is equivalent to the spread of a neo-Y ( $\lambda_{W',XY} = \lambda_{Y',XY}$ ), such that heterogametic transitions (XY to ZW or ZW to XY) can also occur only if the neo-sex-determining region is more closely linked to a locus under selection ( $R < r$ ). However, if there is any haploid selection, the additional term in equation (3) can be positive, which can allow,

for example, neo-W invasion ( $\lambda_{W',XY} > 1$ ) even when the neo-sex-determining  
 374 region is less closely linked to the selected locus ( $R > r$ ). These transitions are  
 unusual because, when  $R > r$ , associations that selection has built up between  
 376 alleles more favourable in one sex and alleles that determine sex will be weak-  
 ened. Mean diploid fitness therefore decreases during heterogametic transitions  
 378 that create looser sex-linkage (Figure ??B,D).

Equation (3) shows that, with weak selection, neo-W alleles can invade an XY  
 380 system for a large number of selective regimes. To clarify the parameter space  
 under which  $\lambda_{W',XY} > 1$ , we consider several special cases. Firstly, if the A locus  
 382 is unlinked to the ancestral sex-determining region ( $r = 1/2$ ), a more closely linked  
 neo-W ( $R < 1/2$ ) can always invade because there is then no association between  
 384 A alleles and sex chromosomes,  $(\hat{p}_Y^\delta - \hat{p}_X^\delta) = 0$ . The second term in equation (3)  
 then disappears and invasion depends only on the sign of  $(r - R)$ . Indeed, invasion  
 386 typically occurs when the neo-W is more closely linked to the selected locus than  
 the ancestral sex-determining region, under a variety of selective regimes (Figure  
 388 4). Secondly, we can simplify cases where invasion occurs despite looser sex-  
 linkage,  $R > r$ , using the special case where  $R = 1/2$  and  $r < 1/2$  (e.g., the  
 390 selected locus is on the ancestral sex chromosome and the novel sex-determining  
 locus arises on an autosome). In table 3 we give the conditions where invasion  
 392 occurs when we further assume that haploid selection only occurs in one sex (e.g.,  
 during male meiosis only) and dominance coefficients are equal in the two sexes,  
 394  $h^\varnothing = h^\delta$ . When there is no gametic competition and meiotic drive is in one sex  
 only, an unlinked neo-W can invade as long as the same allele is favoured during  
 396 diploid selection in males and females ( $s^\varnothing s^\delta > 0$ , see Figure 4B). When there  
 is no meiotic drive and gametic competition occurs in one sex only, an unlinked  
 398 neo-W can invade as long as the same allele is favoured in male and female diploid  
 selection and there are sex differences in selection of one type (e.g.,  $s^\varnothing(s^\delta - s^\varnothing) > 0$ ,  
 400 see Figure 4C,D). These special cases indicate that neo-W invasion can occur for  
 a relatively large fraction of parameter space, even if the neo-W uncouples the  
 402 sex-determining locus from a locus under selection.



Table 3: Invasion conditions for unlinked neo-W ( $R = 1/2$ ,  $r < 1/2$ ) into ancestral XY with one form of haploid selection

Scenario	Assumptions	neo-W spreads ( $\lambda_{W',XY} > 1$ ) if
male drive only	$h^\sigma = h^\varphi, t^\varphi = t^\sigma = \alpha_\Delta^\varphi = 0$	$s^\varphi s^\sigma > 0$
female drive only	$h^\sigma = h^\varphi, t^\varphi = t^\sigma = \alpha_\Delta^\sigma = 0$	$s^\varphi s^\sigma > 0$
sperm competition only	$h^\sigma = h^\varphi, t^\varphi = \alpha_\Delta^\varphi = \alpha_\Delta^\sigma = 0$	$s^\varphi(s^\sigma - s^\varphi) > 0$
egg competition only	$h^\sigma = h^\varphi, t^\sigma = \alpha_\Delta^\varphi = \alpha_\Delta^\sigma = 0$	$s^\sigma(s^\varphi - s^\sigma) > 0$

Previous research suggests that when the ancestral sex-determining locus is  
404 linked to a locus that experiences haploid selection (e.g., meiotic drive), a new,  
unlinked sex-determining locus invades in order to restore equal sex ratios (Koziel-  
406 ska et al. 2010). Consider, for example, the case where the **A** locus is linked to the  
ancestral-SDR ( $r < 1/2$ ) and experiences meiotic drive in males only (e.g., during  
408 spermatogenesis but not during oogenesis,  $\alpha^\sigma \neq 1/2$ ,  $\alpha^\varphi = 1/2$ ). Disregarding  
gametic competition ( $t^\varphi = t^\sigma = 0$ ) such that zygotic sex ratios are only biased  
410 by meiotic drive in males. In this case, the zygotic sex ratio can be initially bi-  
ased only if the ancestral sex-determining system is XY (Figure ??B). We might  
412 therefore expect a difference in the potential for XY to ZW and ZW to XY tran-  
sitions. However, to leading order with selection weak relative to recombination,  
414 we find that sex ratio selection (first terms in table 2) is equal in magnitude to  
the fitness effects of alleles associated with new sex-determining alleles (second  
416 terms in table 2). Thus, invasion by a neo-W into an XY system and invasion by  
a neo-Y into a ZW system occur under the same conditions ( $\lambda_{Y',XY} = \lambda_{W',ZW}$   
418 and  $\lambda_{Y',ZW} = \lambda_{W',XY}$ , at least up to order  $\epsilon^2$ ). As selection becomes stronger (or  
linkage becomes tighter), this symmetry between sex-ratio selection and haploid  
420 selection is lost, causing differences in the strength of selection favouring the two  
heterogametic transitions (compare red to black near -25cM and 25 cM in Figure  
422 4).

## Environmental sex determination

424 We next consider the case where the new sex-determining mutation,  $m$ , causes sex  
to be determined probabilistically or by heterogeneous environmental conditions  
426 (environmental sex determiner, ESD). We assume that individuals carrying the  $m$   
allele develop as females with probability  $k$  (e.g., in a fraction  $k$  of the environ-  
428 ments they randomly experience). The characteristic polynomial determining the  
eigenvalues of the 8 equation system (equations S.1) does not reduce for ESD mu-  
430 tants as it does for  $k = 0$  or  $k = 1$ . We therefore focus on weak selection here.  
Assuming weak selection, the spread of these mutations is given by

$$\begin{aligned} \lambda_{ESD',XY} = & 1 + (1 - 2k)^2 V_A S_A^2 \frac{r - R}{rR} \\ & + \frac{k(\hat{p}_Y^\delta - \hat{p}_X^\delta)}{2} \left( k(2\alpha_\Delta^\delta - 2\alpha_\Delta^\varnothing + t^\delta - t^\varnothing) - 4(1 - k)S_A \right) + O(\epsilon^3), \end{aligned} \quad (4)$$

432 which reduces to  $\lambda_{Y',XY}$  when  $k = 0$  and  $\lambda_{W',XY}$  when  $k = 1$ .

Of particular interest are ESD mutations that cause half of their carriers to  
434 develop as females and half as males ( $k = 1/2$ , creating equal sex ratios), the  
spread of which is given by

$$\lambda_{ESD',XY} = 1 + \frac{1}{2} \frac{(\lambda_{Y',XY} - 1) + (\lambda_{W',XY} - 1)}{2} \Big|_{R=1/2} + O(\epsilon^3), \quad (5)$$

436 where we have indicated that  $\lambda_{Y',XY}$  and  $\lambda_{W',XY}$  are evaluated at  $R = 1/2$ . That is,  
recombination between the selected locus and the novel sex-determining locus,  $R$ ,  
438 doesn't enter into the  $k = 1/2$  results. This is because sex is essentially randomized  
each generation, preventing associations from building up between allele  $A$  and  
sex. An important result from equation (5) is that ESD can invade if there is haploid  
440 selection. When evaluated at  $R = 1/2$ ,  $\lambda_{Y',XY} \leq 1$  but  $\lambda_{W',XY}$  can be greater than  
one if there is haploid selection, as discussed above. Previous studies where ESD  
442 is favoured have typically assumed that environmental conditions (e.g., maternal

condition, mate quality, age, or host size) can differentially affect the fitness of males versus females such that ESD invades because it allows sex determination to depend on the environment (reviewed in Charnov 1982, Bull 1983, West 2009). Here, ESD mutations can spread because they generate females that are either rare or have high fitness, in the same manner as a neo-W.

Equation (5) also shows that invasion by a novel ‘perfect’ ESD (equal sex ratio,  $k = 1/2$ ) mutation is the same for an ancestrally XY or ZW system (since  $\lambda_{Y',XY} = \lambda_{W',ZW}$ ,  $\lambda_{W',XY} = \lambda_{Y',ZW}$ ). Thus, by the same argument as above (if drive only occurs in males then the sex ratio is only biased when the ancestral sex-determination system is XY), Fisherian sex-ratio selection alone does not explain the invasion of an offspring-controlled neo-ESD allele under weak selection. Rather, the neo-ESD gets half of the fitness of a feminizing mutation (neo-W) and half of the fitness of a masculinizing mutation (neo-Y), but only has an effect one half of the time (the other half of the time it produces the same sex as the ancestral system would have, to leading order). The net result can be that perfect ESD will not invade, even if current sex ratios are biased. For example, if there is haploid selection in males (either drive or pollen/sperm competition) but the conditions in table 3 are not met, perfect ESD will not invade, even though it would equalize the zygotic sex ratio from an initially biased case (assuming  $r < 1/2$ ).

## Discussion

Two predominant theories explaining the remarkably high frequency of transitions between sex-determination systems are sexually-antagonistic selection and sex-ratio selection (reviewed in Blaser et al. 2012) (van Doorn, 2014, sexual development). The former predicts that neo-sex-determining alleles can invade when they arise in closer linkage with a sexually-antagonistic locus (van Doorn and Kirkpatrick 2007; 2010). The latter predicts that neo-W alleles will invade an XY system when there is a male bias caused by haploid selection in males, and vice-versa, a neo-Y will invade a ZW system when there is a female bias caused by

472 haploid selection in females (Kozielska et al. 2010, Úbeda et al. 2015). Here we  
have shown that both predictions must be amended when recombination is weak  
474 relative to selection or selection happens in both diploid and haploid phases.

When the rate of recombination between the ancestral sex-determining locus  
476 and a locus under selection is small relative to the strength of selection (i.e., sex-  
linkage is tight, or selection is strong), heterogametic transitions (XY to ZW or  
478 ZW to XY) that reduce sex-linkage are possible, with or without haploid selection  
or sexually-antagonistic selection (Figure ??). The likelihoods of these transitions  
480 are driven by sex-ratio selection, direct selection on alleles linked to the neo-sex-  
determining allele, the ability of the neo-sex-determining allele to avoid selection  
482 in one sex, and the ability of the neo-sex-determining allele to bring alleles on the  
sex-specific chromosome in the ancestor into the other sex (given that the neo-sex  
484 determining allele is epistatically dominant to its predecessor). This possibility  
that looser sex-linkage could evolve, even in the absence of haploid selection (Fig-  
486 ure ??A), was overlooked in van Doorn and Kirkpatrick (2010), likely because  
they did not explicitly calculate the resident equilibria (equation S.2; Lloyd and  
488 Webb 1977, Otto 2014). Interestingly, there is substantial overlap between the pa-  
rameter space that allows both neo-W-A and neo-W-a haplotypes to spread in an  
490 XY system and that which selects for increased recombination between X and Y  
chromosomes (e.g., compare gray region of Figure ??A with coloured regions of  
492 Figure 2(a) in Otto 2014). This makes sense, as when both neo-W haplotypes can  
spread the neo-W can invade despite reducing sex-linkage, i.e., the rate of recom-  
494 bination between the sex-determining allele and the selected locus increases.

Under weak selection (or loose sex-linkage), transitions to new sex-determining  
496 systems can occur when they arise more closely linked to a sexually-antagonistic  
locus (van Doorn and Kirkpatrick 2007; 2010). Our results show that genetic vari-  
498 ation at loci that experience haploid selection can generate selection in favour of  
new sex-determining systems in a similar way. New sex-determining alleles are  
500 again favoured if they are more closely linked to a locus under haploid selection.  
However, with haploid selection, heterogametic transitions (XY to ZW or ZW to

502 XY) can also occur when the new sex-determining region is less closely linked  
 to the locus under selection. Neo-W (neo-Y) alleles invade when their fitness in  
 504 females (males) is greater than the mean fitness of females (males) under the an-  
 cestral sex-determination system and/or females (males) are the rarer sex. With  
 506 sexually-antagonistic selection (between diploid sexes) only, linkage between a se-  
 lected locus and the sex-determining region strengthens associations between male  
 508 beneficial alleles and the male-determining allele (Y or Z) and between female ben-  
 efitial alleles and the female-determining allele (X or W). Thus, the mean fitness  
 510 of both males and females increases with closer linkage to the sex-determining re-  
 gion. Therefore, new sex-determining alleles only invade if they are more closely  
 512 linked than the ancestral sex-determining region. However, if there is haploid se-  
 lection on loci linked to an XY (ZW) sex-determining region, selection can main-  
 514 tain polymorphisms at which the product of the frequency of females (males) and  
 the mean fitness of females (males) is lower than it would be without sex-linkage.  
 516 In these cases, unlinked neo-W (neo-Y) alleles can increase the frequency and/or  
 fitness of the only sex they are found in, at a cost to the other sex, and invade despite  
 518 lowering population mean fitness (Figure ??).

Sex ratio biases caused by gametic competition or meiotic drive have been  
 520 shown to exert Fisherian sex-ratio selection on various autosomal (Stalker 1961,  
 Smith 1975, Frank 1989, Hough et al. 2013, Úbeda et al. 2015, Otto et al. 2015) and  
 522 sex-linked (Kozielska et al. 2010) modifiers. We find that sex-ratio biases caused  
 by haploid selection can also affect transitions between sex-determining systems  
 524 (e.g., see  $\zeta$  terms in Table 2). For instance, when an allele that drives in males  
 is linked to an XY locus it will often become associated with the Y and therefore  
 526 produce a male bias ( $\zeta < 1/2$ ). This male bias increases the potential for a neo-W  
 to invade (as we then have  $(2\zeta)^{-1} > 1$  in Table 2), which can equalize the sex-  
 528 ratio (for related examples see Kozielska et al. 2010, Úbeda et al. 2015). However,  
 this sex-ratio selection can be overwhelmed by drive itself or additional selective  
 530 effects (e.g., when a linked allele is beneficial for male diploids but detrimental  
 for female diploids; Table 3), preventing the neo-W from invading. Indeed, these

532 additional selective effects can even favour transitions between sex-determining  
systems that create new sex-ratio biases (Úbeda et al. 2015). For example, in an  
534 ancestral ZW system, an allele that drives only in males can allow a linked neo-  
Y to invade, despite the fact it creates a male bias (Figure ??C). Furthermore,  
536 with weak selection, there is no asymmetry between XY to ZW and ZW to XY  
transitions, indicating that sex-ratio selection does not dominate (i.e., the sex-ratio  
538 bias created by haploid selection impacts the spread of a neo-W into an XY system  
the same way it impacts the spread of a neo-Y into a ZW system with a 1:1 sex  
540 ratio). An asymmetry can develop when sex-linkage is tight (e.g., Figure 4 near  
-25cM and 25cM) but under most circumstances we do not predict asymmetry  
542 between XY to ZW and ZW to XY transitions despite the presence/absence of sex  
ratio selection. Thus, haploid selection can favour heterogametic transitions both  
544 via sex-ratio selection (as predicted by Kozielska et al. 2010) and via fitness effects  
of alleles that are associated with the neo-sex-determining allele (as predicted in  
546 step 1 by Úbeda et al. 2015), and these selection pressures are predicted to often  
be of roughly equal magnitude.

548 We assume that sex-determining alleles do not experience direct selection ex-  
cept via their associations with sex and alleles at a selected locus. However, in  
550 some cases, there may be significant degeneration around the sex-limited allele (Y  
or W) in the ancestral sex-determining region because recessive deleterious muta-  
552 tions and/or deletions may fix around the Y or W allele (Rice 1996, Charlesworth  
and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). During heterogametic  
554 transitions (XY to ZW or ZW to XY), the formally sex-limited allele fixes such  
that all individuals have YY or WW genotypes (Figure ??). Any recessive delete-  
556 rious alleles linked to the Y or W will therefore be revealed to selection during a  
heterogametic transition. This phenomenon was studied by van Doorn and Kirk-  
558 patrick (2010), who found that degeneration can prevent fixation of a neo-W or  
a neo-Y allele, leading to a mixed sex-determination system where the ancestral-  
560 and neo- sex-determining loci are both polymorphic. However, they noted that  
very rare recombination events around the ancestral sex-determining region can

562 allow these heterogametic transitions to complete. While not explicitly studied,  
we also predict that Y or W degeneration would prevent fixation of the new sex-  
564 determiners considered here.

In addition, our model of meiotic drive is simple, involving a single locus with  
566 two alleles. However, many meiotic drive systems involve an interaction with an-  
other locus at which alleles may ‘suppress’ the action of meiotic drive (Burt and  
568 Trivers 2006, Lindholm et al. 2016). Thus, the dynamics of meiotic drive alleles  
can be heavily dependent on the interaction between two loci and the recombina-  
570 tion rate between them, which in turn can be affected by sex-linkage if there is re-  
duced recombination between sex chromosomes (Hurst and Pomiankowski 1991).  
572 Furthermore, in some cases, a driving allele may act by killing any gametes that  
carry a ‘target’ allele at another locus, in which case there is a two-locus drive sys-  
574 tem and the total number of gametes produced can be reduced by meiotic drive.  
Where gamete number is reduced by meiotic drive, the number of mates competing  
576 for fertilization (mating system) can affect the equilibrium frequency of a meiotic  
drive allele (Holman et al. 2015). In polygamous mating systems, the intensity  
578 of pollen/sperm competition can depend on the density of males available to do-  
nate pollen/sperm, which can itself depend on the sex ratio (Taylor and Jaenike  
580 2002). Since the sex ratio is partly determined by the sex-determination system,  
the evolution of new sex-determination system could be influenced by these dy-  
582 namics. How the evolution of new sex-determining mechanisms could be influ-  
enced by two-locus meiotic drive and/or by ecological feedbacks under different  
584 mating systems remains to be studied.

The hypotheses presented here can be empirically investigated in a similar  
586 manner to the idea that transitions between sex-determining systems are favoured  
by linkage to sexually-antagonistic variation. In the case of sexually-antagonistic  
588 variation, one supporting observation is that genes expected to be under sexually-  
antagonistic selection (e.g., those causing bright male colouration) have been found  
590 on recently derived sex chromosomes (Lindholm and Breden 2002, Tripathi et al.  
2009, Ser et al. 2010). Our results suggest that polymorphic loci that are ancestrally

sex-linked and under sex-specific selection could also drive heterogametic transitions between sex-determination systems. As noted by van Doorn and Kirkpatrick (2010), it would be prudent to compare closely related clades in order to determine whether observed polymorphisms pre-dates a transition in sex-determination or arose afterwards, particularly because sex-linkage allows sexually-antagonistic selection to maintain polymorphisms under a different and larger parameter space (Rice 1987, Jordan and Charlesworth 2011). As with sexually-antagonistic selection, the presence of haploid selected loci around ancestral- or novel-sex-determining regions could support their role in sex chromosome turnover. A recent transcriptome analysis in *Rumex*, suggests a role for haploid competition in the evolution of sex-determination systems by showing that Y-linked genes are overexpressed in pollen but not in male diploids, indicating variation currently or previously maintained by haploid selection; over-expression also occurs on the autosome that is orthologous to the sex chromosomes in closely related species (Sandler et al., 2017, Personal Communication).

Taken at face value, our results indicate that transitions in heterogamete (XY to ZW or vice versa) are more likely than transitions in homogamete when genetic conflict is predominately between the haploids of each sex (e.g., with  $|D^\delta - D^\varphi| \ll |\alpha_\Delta^\delta - \alpha_\Delta^\varphi + t^\delta - t^\varphi|$  we have  $\lambda_{W',XY} > \lambda_{Y',XY}$ ; equations 3 and S.5). In addition, because haploid selection can cause transitions that increase or decrease sex-linkage, haploid selection may lead to less stability, and greater potential for cycling, in sex-determination systems (e.g., the final state in Figure ??C is the starting state in Figure ??B). Potentially, successive heterogametic transitions between master regulators of sex-determination could be inferred from careful examination of the molecular pathways by which sex is determined. Our predictions could also be examined using a suitable proxy for haploid selection, for example, Lenormand and Dutheil (2005) use the outcrossing rate in plants as a proxy for the strength of pollen competition. Furthermore, assuming that transitions from dioecy to hermaphroditism (equal parental investment in male and female gametes) are favoured in a similar manner to the ESD examined here (equal probability of



zygotes developing as males or females), our results suggest that haploid competition during the multicellular haploid stage could drive transitions between dioecy and hermaphroditism in plants (Käfer et al., 2017, Sabath et al., 2017). In animals, one might expect gametic competition to be stronger in species where sperm is required to live for a long time after spermatogenesis because transcripts shared during spermatogenesis may become depleted, revealing the haploid phenotype of the sperm (Immler et al. 2014). Given the caveats mentioned above about the form of meiotic drive modelled, we would also expect that heterogametic transitions in sex determination would be more common in clades where there is meiotic drive.

We have shown that tight sex-linkage and haploid selection can drive previously unexpected transitions between sex-determination systems. In particular, both can select for neo-sex-determining loci that are more loosely linked. In addition, haploid selection alone can cause transitions analogous to those caused by purely sexually-antagonistic selection, eliminating the need for differences in selection between male and female diploids. Perhaps counterintuitively, transitions involving haploid selection can be driven by sex-ratio selection, or cause sex-ratio biases to evolve. We therefore argue that haploid selection should be considered, alongside sexually-antagonistic and sex-ratio selection, as a potentially pivotal factor in the evolution of many sex-determination systems. Overall, our results suggest several new scenarios under which new sex-determination systems are favoured, including sex-specific selection on ancestrally sex-linked loci, which could help to explain why the evolution of sex-determination systems is so dynamic.

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## Figures

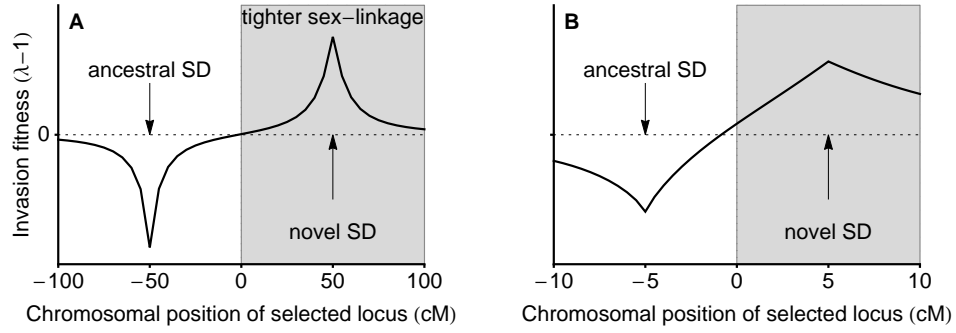


Figure 1: In the absence of haploid selection, tight linkage allows looser sex-linkage to evolve in transitions between XY and ZW systems. In panel A, linkage is loose enough relative to selection that the weak selection analytical results hold, and a neo-W can only invade when it is more tightly linked with the selected locus ( $R < r$ ; shaded region). In panel B, linkage is tight enough relative to selection that the weak selection analytical results do not hold, and a neo-W can only invade even when it is less tightly linked with the selected locus ( $r < R$ ; unshaded region). Parameters:  $w_{AA}^{\varnothing} = w_{Aa}^{\varnothing} = 0.85$ ,  $w_{Aa}^{\sigma} = 1$ ,  $w_{Aa}^{\delta} = 0.925$ ,  $t^{\varnothing} = \alpha_{\Delta}^{\varnothing} = 0$ .

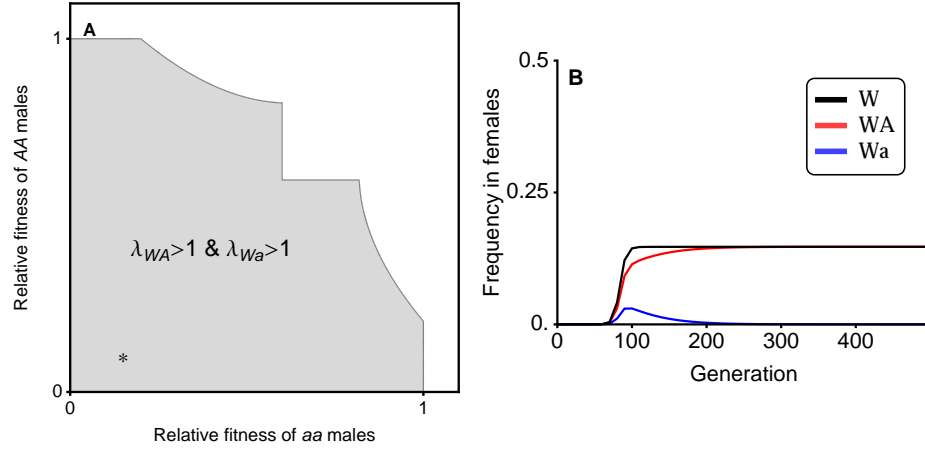


Figure 2: In the absence of haploid selection, both neo-W haplotypes can spread in an XY system when there is overdominance. Panel A shows the range of male homozygote fitnesses that allow both neo-W haplotypes to invade. Panel B shows the temporal dynamics of the invading neo-W haplotypes; in this case the neo-W does not fix, leading to a polymorphic sex-determining system. Asterisk in panel A marks parameter values ( $w_{aa}^{\delta} = 0.15$ ,  $w_{AA}^{\delta} = 0.1$ ) used in panel B. Parameters:  $w_{AA}^{\varnothing} = w_{aa}^{\varnothing} = 0.6$ ,  $w_{Aa}^{\varnothing} = 1$ ,  $t^{\varnothing} = \alpha_{\Delta}^{\varnothing} = 0$ ,  $r = R = 0$ .

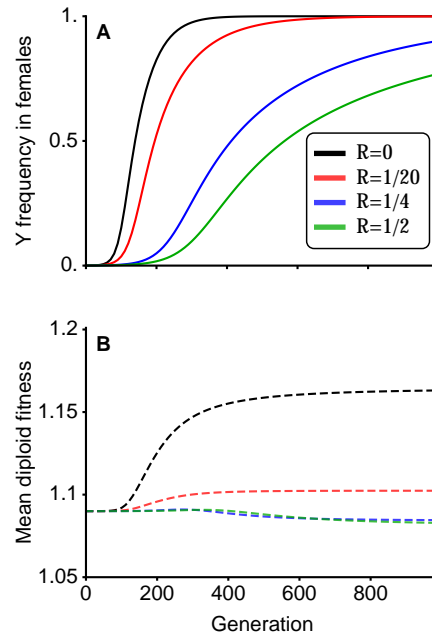


Figure 3: With weak selection, a less tightly linked neo-W can invade (and fix in) an XY system (causing the Y to fix in females) when there is some haploid selection (blue and green curves in panel A), and this invasion decreases mean diploid fitness (green and blue curves in panel B). Parameters:  $s^{\delta} = 0.2$ ,  $h^{\delta} = 0.7$ ,  $t^{\delta} = \alpha_{\Delta}^{\delta} = 0$ ,  $\alpha_{\Delta}^{\circ} = -0.1$ ,  $r = 1/10$ .

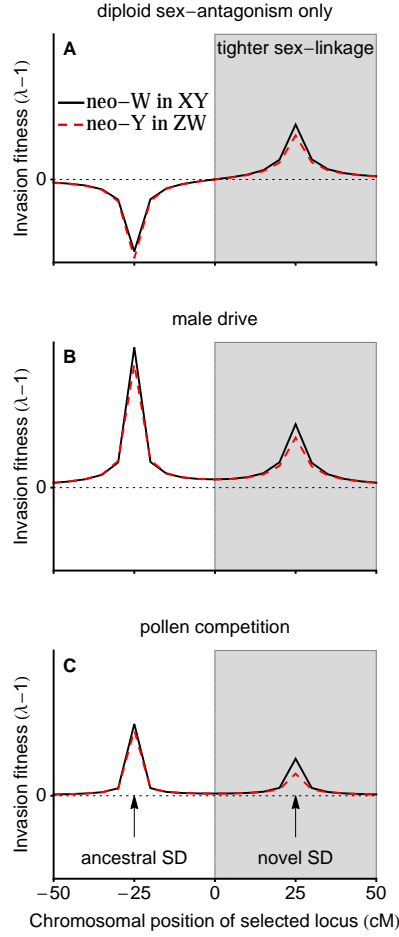


Figure 4: A neo-W can invade an XY system under a large number of selective regimes. In panel A, there is no haploid selection ( $t^{\delta} = \alpha_{\Delta}^{\delta} = 0$ ) and selection in diploids is sexually antagonistic ( $s^{\delta} = -s^{\varnothing} = 1/10$ ,  $h^{\delta} = 1 - h^{\varnothing} = 3/10$ ), in which case the neo-sex-determining allele can only invade if it is more closely linked to the selected locus ( $R < r$ , gray region; but see Figure 1B for the case of very tight linkage). In panel B, male drive ( $\alpha_{\Delta}^{\delta} = -1/20$ ,  $t^{\delta} = \alpha_{\Delta}^{\varnothing} = 0$ ) opposes selection in diploids (no sex-differences:  $s^{\delta} = 1/10$ ,  $h^{\delta} = 7/10$ ), in which case the neo-sex-determining allele can invade regardless of linkage. In panel C, haploid competition in males ( $t^{\delta} = -1/10$ ,  $t^{\varnothing} = \alpha_{\Delta}^{\delta} = 0$ ) opposes selection in diploids (sex-differences:  $s^{\delta} = 3s^{\varnothing} = 3/20$ ,  $h^{\delta} = 7/10$ ), in which case the neo-sex-determining allele can once again invade regardless of linkage. We use Haldane's map function (Equation 3 in Haldane 1919) to convert from map distance (centiMorgans, cM) to the probability of recombination (an odd number of cross-over events).

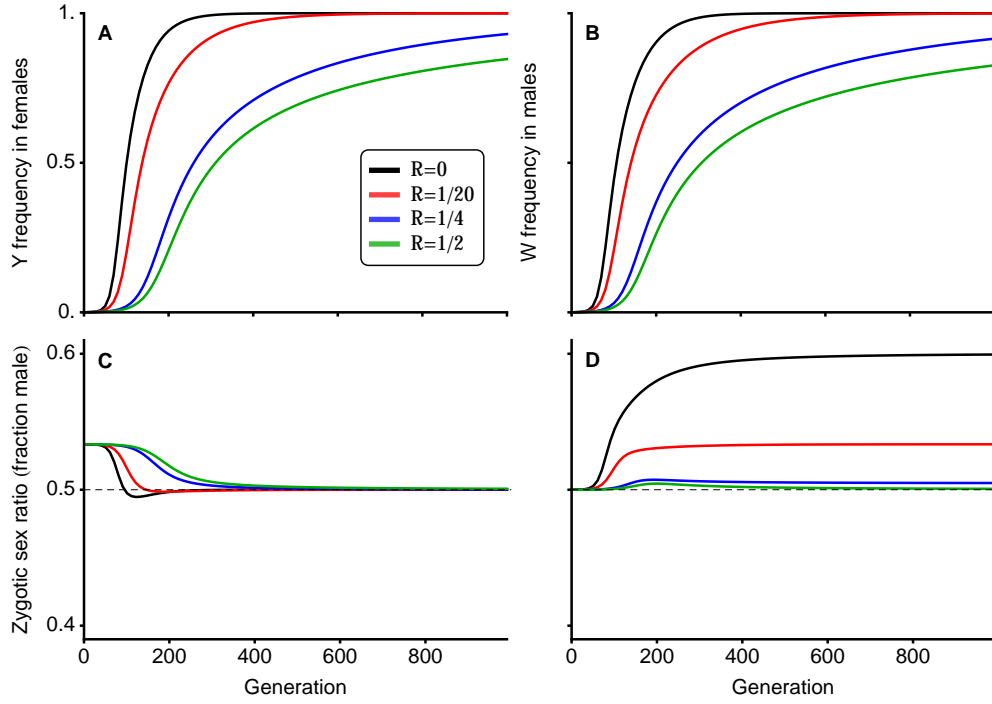


Figure 5: Sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In panels A and C, male drive in an ancestral XY system causes a male bias, allowing a neo-W to invade and fix (causing the Y to fix in females), which balances the sex ratio (it also avoids selection in males, and thus makes better females in this case of sex-specific ploidy-antagonistic selection). In panels B and D, male drive in an ancestral ZW system has no effect on the sex ratio, yet a neo-Y can invade and fix (causing the W to fix in males) because it becomes linked with the driving allele, and this imparts a sex ratio bias. Parameters:  $s^{\varnothing} = s^{\delta} = 0.2$ ,  $h^{\varnothing} = h^{\delta} = 0.7$ ,  $t^{\varnothing} = t^{\delta} = \alpha_{\Delta}^{\delta} = 0$ ,  $\alpha_{\Delta}^{\varnothing} = -1/10$ ,  $r = 1/20$ .

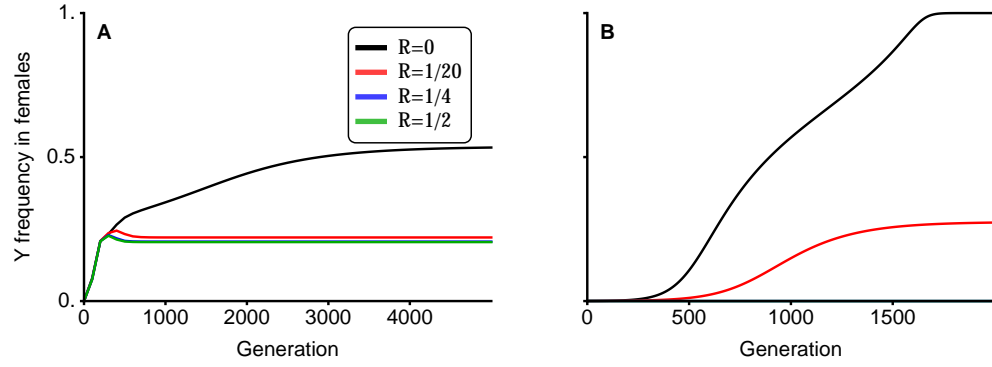


Figure 6: Tight linkage (relative to selection) and/or haploid selection can cause stable polymorphic sex-determining systems. In panel A, there is strong overdominance in both sexes and no haploid selection (parameters as in Figure 2B with  $r = 1/10$ ), in which case a neo-W can invade regardless of sex-linkage, but cannot not fix (i.e., the Y does not fix in females, and X, Y, W, and Z all segregate at equilibrium). In panel B, there is sex-antagonistic selection at the diploid stage and drive in females (parameters as in Figure 1 with  $\alpha_{\Delta}^{\circ} = 1/20$  and  $r = 1/10$ ), in which case a sufficiently tightly linked neo-W can invade ( $R < \sim r$ ), but only fixes if  $R = 0$ .

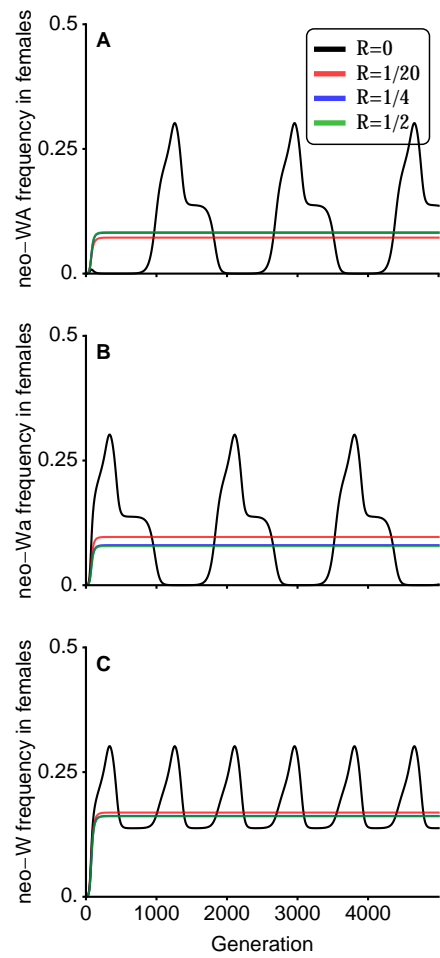


Figure 7: Thought I'd show this just for fun (not for publication).



## 832 Appendix

### Recursion Equations

834 Should we adjust the subscripts throughout this subsection? Right now we end up  
 re-defining  $i$  and  $j$  (when switching from haploid to diploid; this might have been  
 836 my doing!) and then introduce three new subscripts  $b$ ,  $c$ , and  $l$ , all of which can  
 be derived from  $i$  and  $j$ . Might be more straightforward to just use  $p_{x_1, x_2, a_1, a_2, m_1, m_2}^{\phi}$   
 838 where 1 is maternal and 2 is paternal? We then no longer have to switch indices  
 from haploid to diploid and the connection to other variables is clear:  $b = m_1 m_2$ ,  
 840  $c = x_1 x_2$ , and  $l = a_1 a_2$ . I guess the downside will be re-writing the recursion  
 equations... which is why I haven't gone ahead and tried this.

842 In each generation we census the genotype frequencies in male and female gametes/gametophytes (hereafter, gametes) between meiosis (and any meiotic drive)  
 844 and gametic competition. At this stage we denote the frequencies of X- and Y-bearing gametes from males and females  $x_{ij}^{\phi}$  and  $y_{ij}^{\phi}$ , where  $\phi \in \{\delta, \varphi\}$  specifies the sex of the diploid that the gamete came from,  $i \in \{A, a\}$  specifies the allele at the selected locus **A**, and  $j \in \{M, m\}$  specifies the allele at the novel sex-determining locus **M**. The gamete frequencies from each sex sum to one,  
 848  $\sum_{i,j} x_{ij}^{\phi} + y_{ij}^{\phi} = 1$ .

850 Competition then occurs among gametes of the same sex (e.g., among eggs and among sperm separately) according to the **A** locus allele,  $i$  (see Table 1). The  
 852 genotype frequencies after gametic competition are  $x_{ij}^{\phi, s} = w_i x_{ij}^{\phi} / \bar{w}_H^{\phi}$  and  $y_{ij}^{\phi, s} = w_i y_{ij}^{\phi} / \bar{w}_H^{\phi}$ , where  $\bar{w}_H^{\phi} = \sum_{i,j} w_i x_{ij}^{\phi} + w_i y_{ij}^{\phi}$  is the mean fitness of male ( $\phi = \delta$ ) or  
 854 female ( $\phi = \varphi$ ) gametes.

Random mating then occurs between gametes to produce diploid zygotes. To  
 856 shorten notation we now use index  $i$  (and  $j$ ) to denote the alleles at both the **A** and **M** loci and label  $MA = 1$ ,  $Ma = 2$ ,  $mA = 3$ , and  $ma = 4$ , such that  
 858  $i, j \in \{1, 2, 3, 4\}$ . The frequencies of  $XX$  zygotes are then denoted as  $xx_{ij}$ ,  $XY$  zygotes as  $xy_{ij}$ , and  $YY$  zygotes as  $yy_{ij}$ . In  $XX$  and  $YY$  zygotes, individuals with  
 860 diploid genotype  $ij$  are equivalent to those with diploid genotype  $ji$ ; for simplicity,

we use  $xx_{ij}$  and  $yy_{ij}$  with  $i \neq j$  to denote the average of these frequencies,  $xx_{ij} =$   
 862  $(x_i^{\varphi,s} x_j^{\delta,s} + x_j^{\varphi,s} x_i^{\delta,s})/2$  and  $yy_{ij} = (y_i^{\varphi,s} y_j^{\delta,s} + y_j^{\varphi,s} y_i^{\delta,s})/2$ .

Denoting the **M** locus genotype by  $b \in \{MM, Mm, mm\}$  and the **X** locus  
 864 genotype by  $c \in \{XX, XY, YY\}$ , zygotes develop as females with probability  
 $k_{bc}$ . Therefore, the frequencies of  $XX$  females are given by  $xx_{ij}^{\varphi} = k_{bc}xx_{ij}$ ,  $XY$   
 866 females are given by  $xy_{ij}^{\varphi} = k_{bc}xy_{ij}$ , and  $YY$  females are given by  $yy_{ij}^{\varphi} = k_{bc}yy_{ij}$ .  
 Similarly,  $XX$  male frequencies are  $xx_{ij}^{\delta} = (1 - k_{bc})xx_{ij}$ ,  $XY$  male frequencies  
 868 are  $xy_{ij}^{\delta} = (1 - k_{bc})xy_{ij}$ , and  $YY$  males frequencies are  $yy_{ij}^{\delta} = (1 - k_{bc})yy_{ij}$ . This  
 notation allows both the ancestral and novel sex-determining regions to determine  
 870 zygotic sex according to an  $XY$  system, a  $ZW$  system, or an environmental sex-  
 determining system. In addition, we can consider any epistatic dominance rela-  
 872 tionship between the two sex-determining loci. For example, here we assume that  
 the ancestral sex-determining system (**X** locus) is  $XY$  ( $k_{MMXX} = 1$  and  $k_{MMXY} =$   
 874  $k_{MMYY} = 0$ ) or  $ZW$  ( $k_{MMZZ} = 0$  and  $k_{MMZW} = k_{MMWW} = 1$ ) and epistatically  
 recessive to a dominant novel sex-determining locus, **M** ( $k_{Mmc} = k_{mmc} = k$ ).

Selection among diploids then occurs according to the diploid genotype at the  
 876 **A** locus,  $l \in \{AA, Aa, aa\}$ , for an individual of type  $ij$  (see Table 1). The diploid  
 878 frequencies after selection in sex  $\varphi$  are given by  $xx_{ij}^{\varphi,s} = w_l^{\varphi}xx_{ij}/\bar{w}^{\varphi}$ ,  $xy_{ij}^{\varphi,s} =$   
 $w_l^{\varphi}xy_{ij}/\bar{w}^{\varphi}$ , and  $yy_{ij}^{\varphi,s} = w_l^{\varphi}yy_{ij}/\bar{w}^{\varphi}$ , where  $\bar{w}^{\varphi} = \sum_{i=1}^4 \sum_{j=1}^4 w_l^{\varphi}xx_{ij} + w_l^{\varphi}xy_{ij} +$   
 880  $w_l^{\varphi}yy_{ij}$  is the mean fitness of individuals of sex  $\varphi$ .

Finally, these diploids undergo meiosis to produce the next generation of gam-  
 882 etes. Recombination and sex-specific meiotic drive occur during meiosis. Here,  
 we allow any relative locations for the SDR, **A**, and **M** loci by using three param-  
 884 eters to describe the recombination rates between them.  $R$  is the recombination  
 rate between the **A** locus and the **M** locus,  $\chi$  is the recombination rate between the  
 886 **M** locus and the **X** locus, and  $r$  is the recombination rate between the **A** locus and  
 the **X** locus. Table S.1 shows how  $\chi$  can be substituted to give any linear order of  
 888 loci. During meiosis in sex  $\varphi$ , meiotic drive occurs such that, in  $Aa$  heterozygotes,  
 a fraction  $\alpha^{\varphi}$  of gametes produced carry the  $A$  allele and  $(1 - \alpha^{\varphi})$  carry the  $a$  allele.

890 Among gametes from sex  $\varphi$  (sperm/pollen when  $\varphi = \delta$ , eggs/ovules when

Table S.1:  $\chi$  substitutions for different loci orders (assuming no interference)

Order of loci	
SDR-A-M	$\chi = R(1 - r) + r(1 - R)$
SDR-M-A	$\chi = (r - R)/(1 - 2R)$
A-SDR-M	$\chi = (R - r)/(1 - 2r)$

$\phi = \varphi$ ), the frequencies of haplotypes (before gametic competition) in the next  
892 generation are given by

$$\begin{aligned}
x_{MA}^{\phi'} = & x x_{11}^{\phi,s} + x x_{13}^{\phi,s}/2 + (x x_{12}^{\phi,s} + x x_{14}^{\phi,s})\alpha^{\phi} \\
& - R(x x_{14}^{\phi,s} - x x_{23}^{\phi,s})\alpha^{\phi} \\
& + (x y_{11}^{\phi,s} + x y_{13}^{\phi,s})/2 + (x y_{12}^{\phi,s} + x y_{14}^{\phi,s})\alpha^{\phi} \\
& - r(x y_{12}^{\phi,s} - x y_{21}^{\phi,s})\alpha^{\phi} - \chi(x y_{13}^{\phi,s} - x y_{31}^{\phi,s})/2 \\
& + \{ -(R + r + \chi)x y_{14}^{\phi,s} + (r + \chi - R)x y_{41}^{\phi,s} \\
& + (R + r - \chi)x y_{23}^{\phi,s} + (R + \chi - r)x y_{32}^{\phi,s} \}\alpha^{\phi}/2
\end{aligned} \tag{S.1a}$$

$$\begin{aligned}
x_{Ma}^{\phi'} = & x x_{22}^{\phi,s} + x x_{24}^{\phi,s}/2 + (x x_{12}^{\phi,s} + x x_{23}^{\phi,s})\alpha^{\phi} \\
& - R(x x_{23}^{\phi,s} - x x_{14}^{\phi,s})\alpha^{\phi} \\
& (x y_{22}^{\phi,s} + x y_{24}^{\phi,s})/2 + (x y_{21}^{\phi,s} + x y_{23}^{\phi,s})(1 - \alpha^{\phi}) \\
& - r(x y_{21}^{\phi,s} - x y_{12}^{\phi,s})(1 - \alpha^{\phi}) - \chi(x y_{24}^{\phi,s} - x y_{42}^{\phi,s})/2 \\
& + \{ -(R + r + \chi)x y_{23}^{\phi,s} + (r + \chi - R)x y_{32}^{\phi,s} \\
& + (R + r - \chi)x y_{14}^{\phi,s} + (R + \chi - r)x y_{41}^{\phi,s} \}(1 - \alpha^{\phi})/2
\end{aligned} \tag{S.1b}$$

$$\begin{aligned}
x_{mA}^{\tilde{\phi}'} = & x x_{33}^{\tilde{\phi},s} + x x_{13}^{\tilde{\phi},s} / 2 + (x x_{23}^{\tilde{\phi},s} + x x_{34}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(x x_{23}^{\tilde{\phi},s} - x x_{14}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{33}^{\tilde{\phi},s} + x y_{31}^{\tilde{\phi},s}) / 2 + (x y_{32}^{\tilde{\phi},s} + x y_{34}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - r(x y_{34}^{\tilde{\phi},s} - x y_{43}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} - \chi(x y_{31}^{\tilde{\phi},s} - x y_{13}^{\tilde{\phi},s}) / 2 \\
& + \{ -(R + r + \chi) x y_{32}^{\tilde{\phi},s} + (r + \chi - R) x y_{23}^{\tilde{\phi},s} \\
& + (R + r - \chi) x y_{41}^{\tilde{\phi},s} + (R + \chi - r) x y_{14}^{\tilde{\phi},s} \} \alpha^{\tilde{\phi}} / 2
\end{aligned} \tag{S.1c}$$

$$\begin{aligned}
x_{ma}^{\tilde{\phi}'} = & x x_{44}^{\tilde{\phi},s} + x x_{34}^{\tilde{\phi},s} / 2 + (x x_{14}^{\tilde{\phi},s} + x x_{24}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(x x_{14}^{\tilde{\phi},s} - x x_{23}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{44}^{\tilde{\phi},s} + x y_{42}^{\tilde{\phi},s}) / 2 + (x y_{41}^{\tilde{\phi},s} + x y_{43}^{\tilde{\phi},s}) (1 - \alpha^{\tilde{\phi}}) \\
& - r(x y_{43}^{\tilde{\phi},s} - x y_{34}^{\tilde{\phi},s}) (1 - \alpha^{\tilde{\phi}}) - \chi(x y_{42}^{\tilde{\phi},s} - x y_{24}^{\tilde{\phi},s}) / 2 \\
& + \{ -(R + r + \chi) x y_{41}^{\tilde{\phi},s} + (r + \chi - R) x y_{14}^{\tilde{\phi},s} \\
& + (R + r - \chi) x y_{32}^{\tilde{\phi},s} + (R + \chi - r) x y_{23}^{\tilde{\phi},s} \} (1 - \alpha^{\tilde{\phi}}) / 2
\end{aligned} \tag{S.1d}$$

$$\begin{aligned}
y_{MA}^{\tilde{\phi}'} = & y y_{11}^{\tilde{\phi},s} + y y_{13}^{\tilde{\phi},s} / 2 + (y y_{12}^{\tilde{\phi},s} + y y_{14}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(y y_{14}^{\tilde{\phi},s} - y y_{23}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{11}^{\tilde{\phi},s} + x y_{31}^{\tilde{\phi},s}) / 2 + (x y_{21}^{\tilde{\phi},s} + x y_{41}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - r(x y_{21}^{\tilde{\phi},s} - x y_{12}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} - \chi(x y_{31}^{\tilde{\phi},s} - x y_{13}^{\tilde{\phi},s}) / 2 \\
& + \{ -(R + r + \chi) x y_{41}^{\tilde{\phi},s} + (r + \chi - R) x y_{14}^{\tilde{\phi},s} \\
& + (R + r - \chi) x y_{32}^{\tilde{\phi},s} + (R + \chi - r) x y_{23}^{\tilde{\phi},s} \} \alpha^{\tilde{\phi}} / 2
\end{aligned} \tag{S.1e}$$

$$\begin{aligned}
y_{Ma}^{\tilde{\phi}'} = & y y_{22}^{\tilde{\phi},s} + y y_{24}^{\tilde{\phi},s} / 2 + (y y_{12}^{\tilde{\phi},s} + y y_{23}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(y y_{23}^{\tilde{\phi},s} - y y_{14}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{22}^{\tilde{\phi},s} + x y_{42}^{\tilde{\phi},s}) / 2 + (x y_{12}^{\tilde{\phi},s} + x y_{32}^{\tilde{\phi},s}) (1 - \alpha^{\tilde{\phi}}) \\
& - r(x y_{12}^{\tilde{\phi},s} - x y_{21}^{\tilde{\phi},s}) (1 - \alpha^{\tilde{\phi}}) - \chi(x y_{42}^{\tilde{\phi},s} - x y_{24}^{\tilde{\phi},s}) / 2 \\
& + \{ -(R + r + \chi) x y_{32}^{\tilde{\phi},s} + (r + \chi - R) x y_{23}^{\tilde{\phi},s} \\
& + (R + r - \chi) x y_{41}^{\tilde{\phi},s} + (R + \chi - r) x y_{14}^{\tilde{\phi},s} \} (1 - \alpha^{\tilde{\phi}}) / 2
\end{aligned} \tag{S.1f}$$

$$\begin{aligned}
y_{mA}^{\phi'} = & yy_{33}^{\phi,s} + yy_{13}^{\phi,s}/2 + (yy_{23}^{\phi,s} + yy_{34}^{\phi,s})\alpha^{\phi} \\
& - R(yy_{23}^{\phi,s} - yy_{14}^{\phi,s})\alpha^{\phi} \\
& (xy_{33}^{\phi,s} + xy_{13}^{\phi,s})/2 + (xy_{23}^{\phi,s} + xy_{43}^{\phi,s})\alpha^{\phi} \\
& - r(xy_{43}^{\phi,s} - xy_{34}^{\phi,s})\alpha^{\phi} - \chi(xy_{13}^{\phi,s} - xy_{31}^{\phi,s})/2 \\
& + \{ -(R + r + \chi)xy_{23}^{\phi,s} + (r + \chi - R)xy_{32}^{\phi,s} \\
& + (R + r - \chi)xy_{14}^{\phi,s} + (R + \chi - r)xy_{41}^{\phi,s} \}\alpha^{\phi}/2
\end{aligned} \tag{S.1g}$$

$$\begin{aligned}
y_{ma}^{\phi'} = & yy_{44}^{\phi,s} + yy_{34}^{\phi,s}/2 + (yy_{14}^{\phi,s} + yy_{24}^{\phi,s})\alpha^{\phi} \\
& - R(yy_{14}^{\phi,s} - yy_{23}^{\phi,s})\alpha^{\phi} \\
& (xy_{44}^{\phi,s} + xy_{24}^{\phi,s})/2 + (xy_{14}^{\phi,s} + xy_{34}^{\phi,s})(1 - \alpha^{\phi}) \\
& - r(xy_{34}^{\phi,s} - xy_{43}^{\phi,s})(1 - \alpha^{\phi}) - \chi(xy_{24}^{\phi,s} - xy_{42}^{\phi,s})/2 \\
& + \{ -(R + r + \chi)xy_{14}^{\phi,s} + (r + \chi - R)xy_{41}^{\phi,s} \\
& + (R + r - \chi)xy_{23}^{\phi,s} + (R + \chi - r)xy_{32}^{\phi,s} \}(1 - \alpha^{\phi})/2
\end{aligned} \tag{S.1h}$$

894 The full system is therefore described by 16 recurrence equations (three diallelic  
 loci in two sexes,  $2^3 \times 2 = 16$ ). However, some diploid types are not produced  
 896 under a given sex-determination system. For example, with the  $M$  allele fixed and  
 ancestral  $XY$  sex determination, there are no  $m$  alleles,  $XX$  males,  $XY$  females,  
 898 or  $YY$  females ( $xx_{11}^{\phi} = xx_{12}^{\phi} = xx_{22}^{\phi} = xy_{11}^{\phi} = xy_{12}^{\phi} = xy_{21}^{\phi} = xy_{22}^{\phi} = yy_{11}^{\phi} =$   
 $yy_{12}^{\phi} = yy_{22}^{\phi} = 0$ ). In this case, the system only involves six recursion equations,  
 900 which yields equilibrium (S.4).

## Resident equilibrium and stability

902 In the resident population (allele  $M$  fixed), we choose to follow the frequency  
 of  $A$  in female gametes (eggs) from an  $XX$  female,  $p_X^{\phi}$ , and in  $X$ -bearing,  $p_X^{\delta}$ ,  
 904 and  $Y$ -bearing,  $p_Y^{\delta}$ , male gametes (sperm). We also track the total frequency of  
 $Y$  among male gametes,  $q$ , which may deviate from  $1/2$  due to meiotic drive in  
 906 males. These four variables determine the frequencies of the six resident gamete

types:  $x_{MA}^{\varphi} = p_X^{\varphi}$ ,  $x_{Ma}^{\varphi} = 1 - p_X^{\varphi}$ ,  $x_{MA}^{\delta} = (1 - q)p_X^{\delta}$ ,  $x_{Ma}^{\delta} = (1 - q)(1 - p_X^{\delta})$ ,  
 908  $y_{MA}^{\delta} = qp_Y^{\delta}$ , and  $y_{Ma}^{\delta} = q(1 - p_Y^{\delta})$ . Mean fitnesses in the resident population are  
 given in table S.2.

910 Various forms of selection can maintain a polymorphism at the **A** locus, in-  
 cluding sexually antagonistic selection, overdominance, conflicts between diploid  
 912 selection and selection upon haploid genotypes (ploiddally antagonistic selection,  
 Immler et al. 2012), and a combination of these selective regimes.

Table S.2: mean fitnesses in the resident population ( $M$  fixed, XY sex determination)

Sex & Life Cycle Stage	Mean Fitness
female gametes ( $\bar{w}_H^{\varphi}$ )	$p_X^{\varphi} w_A^{\varphi} + (1 - p_X^{\varphi}) w_a^{\varphi}$
male gametes ( $\bar{w}_H^{\delta}$ )	$\bar{p}^{\delta} w_A^{\delta} + (1 - \bar{p}^{\delta}) w_a^{\delta}$
females ( $\bar{w}^{\varphi}$ )	$\{p_X^{\varphi} w_A^{\varphi} p_X^{\delta} w_A^{\delta} w_{AA}^{\varphi} + (1 - p_X^{\varphi}) w_a^{\varphi} p_X^{\delta} w_A^{\delta} w_{Aa}^{\varphi} + p_X^{\varphi} w_A^{\varphi} (1 - p_X^{\delta}) w_a^{\delta} w_{Aa}^{\varphi} + (1 - p_X^{\varphi}) w_a^{\varphi} (1 - p_X^{\delta}) w_a^{\delta} w_{aa}^{\varphi}\} / \{\bar{w}_H^{\varphi} \bar{w}_H^{\delta} \zeta\}$
males ( $\bar{w}^{\delta}$ )	$\{p_X^{\varphi} w_A^{\varphi} p_Y^{\delta} w_A^{\delta} w_{AA}^{\delta} + (1 - p_X^{\varphi}) w_a^{\varphi} p_Y^{\delta} w_A^{\delta} w_{Aa}^{\delta} + p_X^{\varphi} w_A^{\varphi} (1 - p_Y^{\delta}) w_a^{\delta} w_{Aa}^{\delta} + (1 - p_X^{\varphi}) w_a^{\varphi} (1 - p_Y^{\delta}) w_a^{\delta} w_{aa}^{\delta}\} / \{\bar{w}_H^{\varphi} \bar{w}_H^{\delta} (1 - \zeta)\}$
zygotic sex ratio $\zeta$	$\{(1 - q)(p_X^{\delta} w_A^{\delta} + (1 - p_X^{\delta}) w_a^{\delta})\} / \bar{w}_H^{\delta}$

914 In particular special cases, e.g., no sex-differences in selection or meiotic drive  
 ( $s^{\delta} = s^{\varphi}$ ,  $h^{\delta} = h^{\varphi}$ , and  $\alpha^{\delta} = \alpha^{\varphi} = 1/2$ ), the equilibrium allele frequency and sta-  
 916 bility can be calculated analytically without assuming anything about the relative  
 strengths of selection and recombination. However, here, we focus on two regimes  
 918 (tight linkage and weak selection) in order to make fewer assumptions about fit-  
 nesses.

920 **Recombination weak relative to selection (tight linkage between A and X)**

We first calculate the equilibrium frequency of the Y and A alleles in the ancestral  
 922 population when the recombination rate between the X and A loci is small ( $r$  of  
 order  $\epsilon$ ). The A locus will not affect evolution at the novel sex-determining locus,  
 924 M, if one A allele is fixed on all backgrounds. We therefore focus on the five  
 equilibria that maintain both A and a alleles, four of which are given to leading  
 926 order by:

$$(A) \quad \hat{p}_Y^\delta = 0, \quad \hat{q} = \frac{1}{2} - \frac{(\alpha^\delta - 1/2)w_{Aa}^\delta \phi}{w_{Aa}^\delta \phi + w_{aa}^\delta \psi}, \quad (S.2a)$$

$$\hat{p}_X^\varnothing = \frac{w_a^\varnothing \phi}{w_a^\varnothing \phi + w_A^\varnothing \psi}, \quad \hat{p}_X^\delta = \frac{2\alpha^\delta w_{Aa}^\delta \phi}{2\alpha^\delta w_{Aa}^\delta \phi + w_{AA}^\delta \psi}$$

$$(A') \quad \hat{p}_Y^\delta = 1, \quad \hat{q} = \frac{1}{2} + \frac{(\alpha^\delta - 1/2)w_{Aa}^\delta \phi'}{w_{Aa}^\delta \phi' + w_{AA}^\delta \psi'}, \quad (S.2b)$$

$$\hat{p}_X^\varnothing = 1 - \frac{w_A^\varnothing \phi'}{w_A^\varnothing \phi' + w_a^\varnothing \psi'}, \quad \hat{p}_X^\delta = 1 - \frac{2(1 - \alpha^\delta)w_{Aa}^\delta \phi'}{2(1 - \alpha^\delta)w_{Aa}^\delta \phi' + w_{aa}^\delta \psi'}$$

$$(B) \quad \hat{p}_Y^\delta = 0, \quad \hat{p}_X^\varnothing = 1, \quad \hat{p}_X^\delta = 1, \quad \hat{q} = 1 - \alpha^\delta \quad (S.2c)$$

$$(B') \quad \hat{p}_Y^\delta = 1, \quad \hat{p}_X^\varnothing = 0, \quad \hat{p}_X^\delta = 0, \quad \hat{q} = \alpha^\delta \quad (S.2d)$$

$$\begin{aligned} \phi &= \alpha^\varnothing w_A^\varnothing w_{Aa}^\varnothing (w_a^\delta w_{aa}^\delta + 2\alpha^\delta w_A^\delta w_{Aa}^\delta) - w_a^\delta w_a^\varnothing w_{aa}^\delta w_{aa}^\varnothing \\ \psi &= (1 - \alpha^\varnothing) w_a^\varnothing w_{Aa}^\varnothing (w_a^\delta w_{aa}^\delta + 2\alpha^\delta w_A^\delta w_{Aa}^\delta) - 2\alpha^\delta w_A^\delta w_A^\varnothing w_{Aa}^\delta w_{AA}^\varnothing \\ \phi' &= (1 - \alpha^\varnothing) w_a^\varnothing w_{Aa}^\varnothing (w_A^\delta w_{AA}^\delta + 2(1 - \alpha^\delta) w_a^\delta w_{Aa}^\delta) - w_A^\delta w_A^\varnothing w_{AA}^\delta w_{AA}^\varnothing \\ \psi' &= \alpha^\varnothing w_A^\varnothing w_{Aa}^\varnothing (w_A^\delta w_{AA}^\delta + 2(1 - \alpha^\delta) w_a^\delta w_{Aa}^\delta) - 2(1 - \alpha^\delta) w_a^\delta w_a^\varnothing w_{Aa}^\delta w_{aa}^\varnothing \end{aligned}$$

A fifth equilibrium (C) also exists where A is present at an intermediate frequency  
 928 on the Y chromosome ( $0 < \hat{p}_Y^\delta < 1$ ). However, equilibrium (C) is never locally  
 stable when  $r \approx 0$  and is therefore not considered further. Thus, the Y can either  
 930 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')

932 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  
 934 interchanged, we discuss only equilibria  $(A)$  and  $(B)$ , in which the Y is fixed for the  
 $a$  allele. If there is no haploid selection ( $\alpha^\phi = 1/2$ ,  $w_A^\phi = w_a^\phi = 1$ ), these equilibria  
 936 are equivalent to those found by Lloyd and Webb (1977) and Otto (2014).

We next calculate when  $(A)$  and  $(B)$  are locally stable for  $r = 0$ . According  
 938 to the ‘small parameter theory’ (Karlin and McGregor 1972a;b), these stability  
 properties are unaffected by small amounts of recombination between the SDR and  
 940 **A** locus, although equilibrium frequencies may be slightly altered. For the  $a$  allele  
 to be stably fixed on the Y requires that  $\bar{w}_{Ya}^\phi > \bar{w}_{YA}^\phi$  where  $\bar{w}_{Ya}^\phi = w_a^\phi(2p_X^\phi(1 -$   
 942  $\alpha^\phi)w_A^\phi w_{Aa}^\phi + (1 - p_X^\phi)w_a^\phi w_{aa}^\phi)$  and  $\bar{w}_{YA}^\phi = w_A^\phi(p_X^\phi w_A^\phi w_{AA}^\phi + 2(1 - p_X^\phi)\alpha^\phi w_a^\phi w_{Aa}^\phi)$ .  
 That is,  $Ya$  haplotypes must have higher fitness than  $YA$  haplotypes. Substituting  
 944 in  $p_X^\phi = \hat{p}_X^\phi$  from above, fixation of the  $a$  allele on the Y requires that  $\gamma_i > 0$  where  
 $\gamma_{(A)} = w_a^\phi(2(1 - \alpha^\phi)w_{Aa}^\phi \phi + w_{aa}^\phi \psi) - w_A^\phi(w_{AA}^\phi \phi + 2\alpha^\phi w_{Aa}^\phi \psi)$  for equilibrium  
 946  $(A)$  and  $\gamma_{(B)} = 2(1 - \alpha^\phi)w_a^\phi w_{Aa}^\phi - w_A^\phi w_{AA}^\phi$  for equilibrium  $(B)$ . Stability of a  
 polymorphism on the X chromosome (equilibrium  $A$ ) further requires that  $\phi > 0$   
 948 and  $\psi > 0$ . Fixation of the  $a$  allele on the X (equilibrium  $B$ ) is mutually exclusive  
 with equilibrium  $(A)$  and requires  $\psi < 0$  and  $w_A^\phi w_{AA}^\phi > (1 - \alpha^\phi)w_a^\phi w_{Aa}^\phi$ .

#### 950 **Selection weak relative to recombination (weak selection)**

Here, we assume that selection and meiotic drive are weak relative to recombina-  
 952 tion ( $s^\phi$ ,  $t^\phi$ ,  $\alpha_\Delta^\phi$  of order  $\epsilon$ ). The maintenance of a polymorphism at the **A** locus  
 then requires that

$$\begin{aligned} 0 &< -((1 - h^\phi)s^\phi + (1 - h^\phi)s^\phi + t^\phi + t^\phi + \alpha_\Delta^\phi + \alpha_\Delta^\phi) \\ \text{and } 0 &< (h^\phi s^\phi + h^\phi s^\phi + t^\phi + t^\phi + \alpha_\Delta^\phi + \alpha_\Delta^\phi). \end{aligned} \quad (\text{S.3})$$

954 which indicates that a polymorphism can be maintained by various selective regimes.

Given that a polymorphism is maintained at the **A** locus by selection, with  
 956 weak selection and drive the frequencies of  $A$  in each type of gamete are the same



( $\hat{p}_X^\varnothing = \hat{p}_X^\sigma = \hat{p}_Y^\sigma = \bar{p}$ ) and given, to leading order, by

$$\bar{p} = \frac{h^\varnothing s^\varnothing + h^\sigma s^\sigma + t^\varnothing + t^\sigma + \alpha_\Delta^\varnothing + \alpha_\Delta^\sigma}{(2h^\varnothing - 1)s^\varnothing + (2h^\sigma - 1)s^\sigma} + O(\epsilon). \quad (\text{S.4})$$

958 Differences in frequency between gamete types are of order  $\epsilon$  and given, to leading order, by

$$\begin{aligned} \hat{p}_X^\sigma - \hat{p}_X^\varnothing &= V_A (D^\sigma - D^\varnothing + \alpha_\Delta^\sigma - \alpha_\Delta^\varnothing) + O(\epsilon^2) \\ \hat{p}_Y^\sigma - \hat{p}_X^\varnothing &= V_A (D^\sigma - D^\varnothing + \alpha_\Delta^\sigma - \alpha_\Delta^\varnothing + (1 - 2r)(t^\sigma - t^\varnothing)) / 2r + O(\epsilon^2) \\ \hat{p}_Y^\sigma - \hat{p}_X^\sigma &= V_A (D^\sigma - D^\varnothing + \alpha_\Delta^\sigma - \alpha_\Delta^\varnothing + t^\sigma - t^\varnothing)(1 - 2r) / 2r + O(\epsilon^2) \end{aligned} \quad (\text{S.5})$$

960 where  $V_A = \bar{p}(1 - \bar{p})$  is the variance in the frequency of  $A$  and  $D^\varnothing = (\bar{p}s^\varnothing + (1 - \bar{p})h^\varnothing s^\varnothing) - (\bar{p}h^\sigma s^\sigma + (1 - \bar{p}))$  corresponds to the difference in fitness between  $A$  and  
962  $a$  alleles in diploids of sex  $\varnothing \in \{\varnothing, \sigma\}$  ( $\bar{p}$  is the leading-order probability of mating with an  $A$ -bearing gamete from the opposite sex). The frequency of  $Y$  among male  
964 gametes depends upon the difference in the frequency of the  $A$  allele between  $X$ - and  $Y$ -bearing male gametes and the strength of meiotic drive in favour of the  $A$   
966 allele in males,  $q = 1/2 + \alpha_\Delta^\sigma(\hat{p}_Y^\sigma - \hat{p}_X^\sigma)/2 + O(\epsilon^3)$ . Without gametic competition or drive ( $\alpha_\Delta^\varnothing = t^\varnothing = 0$ ) our results reduce to those of van Doorn and Kirkpatrick  
968 (2007).

## Invasion conditions

970 A rare neo- $Y$  or neo- $W$  will spread from a given ancestral equilibrium when the leading eigenvalue,  $\lambda$ , of the Jacobian matrix derived from the eight mutant re-  
972 cursion equations (given by S.1c,d,g,h), evaluated at the ancestral equilibrium, is greater than one. However, because a neo- $Y$  (neo- $W$ ) is always in males (fe-  
974 males) and is epistatically dominant to the ancestral sex-determining locus, we need only two recursion equations (e.g., tracking the change in the frequency of  
976 neo- $Y$ - $A$  and neo- $Y$ - $a$  gametes from males) and thus the leading eigenvalue is

the largest solution to a quadratic characteristic polynomial  $\lambda^2 + b\lambda + c = 0$ .  
 978 It can be shown (see supplementary Mathematica file) that the coefficients are  
 $b = -(\lambda_{mA} + \lambda_{ma}) + (\rho_{mA} + \rho_{ma})$  and  $c = (\lambda_{mA} - \rho_{mA})(\lambda_{ma} - \rho_{ma}) - \rho_{mA}\rho_{ma}$ , where  
 980  $\lambda_{mi}$  is the multiplicative growth rate of the frequency of mutants on background  
 $i \in \{A, a\}$ , without accounting for loss due to recombination, and  $\rho_{mi}$  is the rate at  
 982 which mutants on background  $i \in \{A, a\}$  recombine onto the other **A** locus back-  
 ground in heterozygotes. The leading eigenvalue is then greater than one whenever  
 984  $\lambda_{mA} > 1$  and  $\lambda_{ma} > 1$ , less than one whenever  $\lambda_{mA} < 1$  and  $\lambda_{ma} < 1$ , and greater  
 than one whenever  $\lambda_{mA} > 1$  or  $\lambda_{ma} > 1$  and  $\rho_{ma}(\lambda_{mA} - 1) + \rho_{mA}(\lambda_{ma} - 1) > 0$ .  
 986 For tight linkage between the ancestral sex-determining locus and the selected  
 locus we can calculate each of these terms exactly, while for weak selection we  
 988 take a Taylor series of the leading eigenvalue.

### Recombination weak relative to selection (tight linkage between **A** and **X**)

990 Here, we explore the conditions under which a neo-W invades an XY system as-  
 suming that the **A** locus is initially in close linkage with the ancestral sex-determining  
 992 region ( $r \approx 0$ ). We disregard neo-Y mutations, which never spread given that the  
 ancestral population is at a stable equilibrium.  
 994 Starting with the simpler equilibrium (**B**), the terms of the characteristic poly-  
 nomial are

$$\lambda_{mA} = (2\alpha^\delta)^{-1} \frac{w_A^\varnothing [w_A^\delta w_{AA}^\varnothing \alpha^\delta + 2w_a^\delta w_{Aa}^\varnothing \alpha^\varnothing (1 - \alpha^\delta)]}{w_A^\delta w_A^\varnothing w_{AA}^\varnothing} \quad (\text{S.6a})$$

$$\lambda_{ma} = (2\alpha^\delta)^{-1} \frac{w_a^\varnothing [w_a^\delta w_{aa}^\varnothing (1 - \alpha^\delta) + 2w_A^\delta w_{Aa}^\varnothing (1 - \alpha^\varnothing) \alpha^\delta]}{w_A^\delta w_A^\varnothing w_{AA}^\varnothing} \quad (\text{S.6b})$$

$$\rho_{mA} = (2\alpha^\delta)^{-1} \frac{Rw_a^\delta w_A^\varnothing w_{Aa}^\varnothing \alpha^\varnothing (1 - \alpha^\delta)}{w_A^\delta w_A^\varnothing w_{AA}^\varnothing} \quad (\text{S.6c})$$

$$\rho_{ma} = (2\alpha^\delta)^{-1} \frac{Rw_A^\delta w_a^\varnothing w_{Aa}^\varnothing (1 - \alpha^\varnothing) \alpha^\delta}{w_A^\delta w_A^\varnothing w_{AA}^\varnothing} \quad (\text{S.6d})$$

996 Haploid selection impacts the spread of neo-W haplotypes in three ways. Firstly,  
 the zygotic sex ratio becomes male biased ( $\zeta < 1/2$ ; at equilibrium (B) the sex  
 998 ratio is  $\zeta = \alpha^\delta w_A^\delta / [(1 - \alpha^\delta)w_a^\delta + \alpha^\delta w_A^\delta]$  when the  $a$  allele (which is fixed on  
 the Y) is favoured during competition among male gametes or by meiotic drive  
 1000 in males. This facilitates the spread of a neo-W because neo-W alleles cause the  
 zygotes that carry them to develop as the rarer, female, sex. Secondly, haploid  
 1002 selection in males affects the diploid genotypes of females by altering the allele  
 frequencies in the male gametes that female gametes pair with. For instance, be-  
 1004 cause an epistatically dominant neo-W always causes its carrier to become female,  
 it creates females who carry either Y- $a$  or X genotypes from their father. Thus, be-  
 1006 cause when there is a polymorphism the X carries some non-zero frequency of  $A$ ,  
 haploid selection in males impacts the diploid genotypes of females (e.g., creating  
 1008 more  $Aa$  females when drive in males favours Y- $a$ ). How this affects the spread  
 of the neo-W then depends on diploid and haploid selection in females. Thirdly,  
 1010 female drive and gamete competition directly select on neo-W haplotypes. Drive  
 for  $A$  in females favours neo-W- $A$  haplotypes, at a cost to neo-W- $a$  haplotypes, and  
 1012 vice-versa when there is drive for  $a$ . The impact of this drive depends on how of-  
 ten XX and neo-W females are heterozygous. Competition among female gametes  
 1014 acts similarly, and depends on the frequency of  $A$  on resident X chromosomes  
 (e.g., competition among eggs has no affect on the initial spread of the neo-W- $A$   
 1016 haplotype when  $A$  is fixed on the X). Because haploid selection in females favours  
 one neo-W haplotype at the expense of the other, recombination off the favoured  
 1018 background becomes more detrimental as it becomes more favoured. Thus higher  
 rates of recombination between the neo-W and the selected locus,  $R$ , can lead to  
 1020 smaller leading eigenvalues when there is haploid selection in females.

The other terms in equations (S.6) are more easily interpreted if we assume  
 1022 that there is no haploid selection in either sex, in which case  $\lambda_{mA} > 1$  when  $w_{Aa}^\varnothing >$   
 $w_{AA}^\varnothing$  and  $\lambda_{ma} > 1$  when  $(w_{Aa}^\varnothing + w_{aa}^\varnothing)/2 > w_{AA}^\varnothing$ . These conditions cannot be met  
 1024 under purely sexually-antagonistic selection, where  $a$  is directionally favoured in  
 males ( $w_{AA}^\delta > w_{Aa}^\delta > w_{aa}^\delta$ ) and  $A$  is directionally favoured in females ( $w_{AA}^\varnothing >$

1026  $w_{Aa}^{\varnothing} > w_{aa}^{\varnothing}$ ). Essentially, the X is already as specialized as possible for the female  
beneficial allele (XA is fixed), and the neo-W often makes daughters with the Y-a  
1028 haplotype, increasing the flow of  $a$  alleles into females, which reduces the fitness  
of those females.

1030 If selection doesn't uniformly favour  $A$  in females, however, neo-W- $A$  haplo-  
types and/or neo-W- $a$  haplotypes can spread ( $\lambda_{mA} > 1$  and/or  $\lambda_{ma} > 1$ ) at this  
1032 equilibrium. A neo-W can spread alongside the  $A$  allele ( $\lambda_{mA} > 1$ ), despite the  
fact that a neo-W brings Ya haplotypes into females, when  $w_{Aa}^{\varnothing} > w_{AA}^{\varnothing}$ . In this  
1034 case the  $a$  allele is favoured by selection in females despite  $A$  being fixed on the  
X. For this equilibrium to be stable, X- $A$  must be sufficiently favoured in males  
1036 to keep the frequency of XA at one (specifically, from the stability conditions, we  
must have  $w_{Aa}^{\delta} / ((w_{aa}^{\delta} + w_{Aa}^{\delta}) / 2) > w_{Aa}^{\varnothing} / w_{AA}^{\varnothing}$ ).

1038 Under this same condition,  $w_{Aa}^{\varnothing} > w_{AA}^{\varnothing}$ , the neo-W can also spread alongside  
the  $a$  allele ( $\lambda_{ma} > 1$ ) if there is sufficiently strong underdominance in females  
1040 ( $w_{aa}^{\varnothing} > w_{Aa}^{\varnothing}$ ), such that  $(w_{Aa}^{\varnothing} + w_{aa}^{\varnothing}) / 2 > w_{AA}^{\varnothing}$ . In this case,  $a$  is not favored in  
females near the equilibrium where females are  $AA$  (comparing  $Aa$  to  $AA$  geno-  
1042 types) and yet the neo-W can spread with  $a$  because it produces female  $aa$  individ-  
uals by capturing Y- $a$  haplotypes.

1044 When both haplotypes can spread on their own ( $\lambda_{mA} > 1$  and  $\lambda_{ma} > 1$ ), the  
neo-W invades regardless the recombination rate between it and the selected locus,  
1046  $R$ . When neither haplotype can spread ( $\lambda_{mA} < 1$  and  $\lambda_{ma} < 1$ ) the neo-W can never  
invade. And when only one haplotype can spread on its own the neo-W invades  
1048 only when the rate of recombination onto the favourable background is sufficiently  
larger than the rate of recombination off this background (i.e., equation 1 is satis-  
1050 fied).

Similar equations can be derived for equilibrium (A) by subbing the equilib-  
1052 rium allele frequencies into Table 2.

$$\lambda_{mA} = \frac{a}{b} \left[ w_{AA}^{\varphi} w_{Aa}^{\delta} w_A^{\delta} \alpha^{\delta} \phi + 2w_{Aa}^{\varphi} \alpha^{\varphi} w_a^{\delta} \frac{c}{d} \right] / w_a^{\varphi} \quad (\text{S.7a})$$

$$\lambda_{ma} = \frac{a}{b} \left[ 2w_{Aa}^{\varphi} (1 - \alpha^{\varphi}) w_{Aa}^{\delta} w_A^{\delta} \alpha^{\delta} \phi + w_{aa}^{\varphi} w_a^{\delta} \frac{c}{d} \right] / w_A^{\varphi} \quad (\text{S.7b})$$

$$\rho_{mA} = \frac{a}{b} R \left[ 2w_{Aa}^{\varphi} \alpha^{\varphi} w_a^{\delta} \frac{c}{d} \right] / w_a^{\varphi} \quad (\text{S.7c})$$

$$\rho_{ma} = \frac{a}{b} R \left[ 2w_{Aa}^{\varphi} (1 - \alpha^{\varphi}) w_{Aa}^{\delta} w_A^{\delta} \alpha^{\delta} \phi \right] / w_A^{\varphi} \quad (\text{S.7d})$$

where

$$a = w_a^{\varphi} \phi + w_A^{\varphi} \psi \quad (\text{S.8a})$$

$$b = w_{AA}^{\varphi} \phi (2w_{Aa}^{\delta} w_A^{\delta} \alpha^{\delta} \phi) + w_{Aa}^{\varphi} \psi (2w_{Aa}^{\delta} w_A^{\delta} \alpha^{\delta} \phi + w_{AA}^{\delta} w_a^{\delta} \psi) + w_{aa}^{\varphi} \psi (w_{AA}^{\delta} w_a^{\delta} \psi) \quad (\text{S.8b})$$

$$c = 2(w_{Aa}^{\delta} \phi)^2 (1 - \alpha^{\delta}) \alpha^{\delta} + w_{Aa}^{\delta} \phi (w_{AA}^{\delta} \psi + w_{aa}^{\delta} \psi \alpha^{\delta}) + w_{aa}^{\delta} \psi w_{AA}^{\delta} \psi \quad (\text{S.8c})$$

$$d = 2w_{Aa}^{\delta} \alpha^{\delta} \phi + w_{aa}^{\delta} \psi \quad (\text{S.8d})$$

1054 As with equilibrium (B), haploid selection again modifies invasion fitnesses  
by altering the sex-ratio and the diploid genotypes of females and directly select-  
1056 ing upon female gametes. The only difference is that resident XX females are no  
longer always homozygote *AA* and males are no longer always heterozygote *Aa*.  
1058 Thus the effect of haploid selection in males is reduced, as is the difference in fit-  
ness between neo-W haplotypes and resident X haplotypes, as both can be on any  
1060 diploid or haploid background.

The other terms are easier to interpret in the absence of haploid selection. For  
1062 instance, without haploid selection, the neo-W-*A* haplotype spreads ( $\lambda_{mA} > 1$ ) if  
and only if

$$2(w_{Aa}^{\varphi} - w_{aa}^{\varphi}) w_{AA}^{\delta} \psi^2 > (w_{AA}^{\varphi} - w_{Aa}^{\varphi}) w_{Aa}^{\delta} \phi (\phi - \psi) \quad (\text{S.9})$$

1064 where  $\phi - \psi = w_{AA}^{\varphi} w_{Aa}^{\delta} - w_{aa}^{\varphi} w_{aa}^{\delta}$  and both  $\phi$  and  $\psi$  are positive when equilibrium

(A) is stable. In contrast to equilibrium (B), a neo-W haplotype can spread under purely sexually-antagonistic selection ( $w_{AA}^\delta > w_{Aa}^\delta > w_{aa}^\delta$  and  $w_{AA}^\varphi > w_{Aa}^\varphi > w_{aa}^\varphi$ ). In this case, the neo-W-A haplotype can spread, despite producing a lot of *Aa* daughters by obtaining the *a* from Y-gametes, when *aa* females, which the neo-W-A never makes, are strongly selected against. This can be intuited from the fact that (S.9) will be more easily met when  $w_{Aa}^\varphi - w_{aa}^\varphi \approx w_{Aa}^\varphi$  and  $w_{AA}^\varphi - w_{Aa}^\varphi \approx 0$ , implying  $w_{aa}^\varphi \approx 0$  and  $w_{Aa}^\varphi \approx w_{AA}^\varphi$  (although this is complicated by the fact that  $w_{aa}^\varphi$  and  $w_{Aa}^\varphi$  affect  $\phi$  and  $\psi$  too, the intuition holds).

Without haploid selection, the neo-W-*a* haplotype spreads ( $\lambda_{ma} > 1$ ) if and only if

$$(w_{aa}^\varphi + w_{Aa}^\varphi - 2w_{AA}^\varphi)w_{Aa}^\delta \phi^2 + (w_{aa}^\varphi - w_{Aa}^\varphi)(w_{Aa}^\delta + 2w_{AA}^\delta)\phi\psi > 0 \quad (\text{S.10})$$

This condition cannot be met with purely sexually antagonistic selection (as both terms on the left-hand side would then be negative), but it can be met under other circumstances. For example, with overdominance in males there is selection for increased *A* frequencies on X chromosomes in males, which are always paired with Y-*a* haplotypes. Then, directional selection for *a* in females maintains a polymorphism at the **A** locus on the X and by creating selection for decreased *A* frequencies on X chromosomes in females. This scenario selects for a modifier that increases recombination between the sex chromosomes (e.g., blue region of Figure 2d in Otto 2014) and facilitates the spread of neo-W-*a* haplotypes, which create more heterozygote and *aa* females than ancestral X chromosomes do.

As with equilibrium (B), if both haplotypes can spread ( $\lambda_{mA} > 1$  and  $\lambda_{ma} > 1$ ) then the neo-W invades under any rate of recombination with the selected locus,  $R \geq 0$ . In addition, even when only one haplotype can spread (e.g., under purely sexually-antagonistic selection  $\lambda_{mA} > 1$  and  $\lambda_{ma} < 1$ ), neo-W invasion can still occur under modest rates of recombination between the novel sex-determining and selected loci.

### **Selection weak relative to recombination (weak selection)**

<sup>1092</sup> With weak selection the leading eigenvalue,  $\lambda$ , for any  $k$ , is given up to order  $\epsilon^2$  by equation 4. Scenarios leading to  $\lambda > 1$  are discussed in the main text.