

# 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  
4 shifts in the location of sex-determining loci or the evolution of entirely  
6 new sex-determining systems. Predominant theories for why we observe  
8 such transitions generally conclude that novel sex-determining systems are  
10 favoured by selection if they equalise the sex ratio or increase linkage with  
12 a sexually-antagonistic locus. We use population genetic models to extend  
14 these theories in two ways: (1) We explicitly consider how selection on very  
16 tightly sex-linked loci influences the spread of novel sex-determiners. We  
18 find that tightly sex-linked genetic variation can favour the spread of new  
20 sex-determination systems in which the heterogametic sex changes (XY to  
22 ZW or ZW to XY) and the new sex-determining region is less closely linked  
24 (or unlinked) to the sex-linked locus under selection, which would not be  
26 predicted by previous theory. (2) We also consider selection upon haploid  
28 genotypes either during gametic competition (e.g., pollen/sperm competition)  
or meiosis (i.e., non-Mendelian segregation); selective processes that  
typically occur in one sex or the other. With haploid selection, we again  
find that transitions between male and female heterogamety can occur even  
if the new sex-determining region is less closely linked to the locus under se-  
lection. Haploid selection in the heterogametic sex can also cause sex ratio  
biases, which may increase or decrease with the spread of new sex chro-  
mosomes. Thus, transitions between sex-determination systems cannot be  
simply predicted by selection to equalise the sex-ratio. Overall, our models  
reveal that transitions between sex-determination systems, particularly tran-  
sitions where the heterogametic sex changes, can be driven by loci in previ-  
ously unexpected genomic locations that experience selection during diploid  
and/or haploid phases. These results might be reflected in the lability with  
which sex-determination systems evolve.

## Introduction

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

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

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

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

84 Here we use mathematical models to find the conditions under which new  
sex-determination systems spread when individuals experience selection at both  
86 diploid and haploid stages. Even in animal and plant species that have much  
larger and more conspicuous diploid phases than haploid phases, many loci ex-  
88 perience significant haploid selection through gamete competition 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 genotypes during gamete production (from one parent) and the term ‘gametic competition’ to refer to selection upon haploid genotypes within a gamete/gametophyte pool (potentially from multiple parents); the term ‘haploid selection’ encompasses both processes.

94     Genetic mapping experiments, which are typically designed to minimize selection in diploids, have revealed segregation distortion in various species, including  
96     mice, Drosophila, Rice, Maize, Wheat, Barley, Cotton... In some of these cases,  
98     biased segregation has been attributed to meiotic drive and/or gametic selection  
   (Leppala et al. 2013, Didion et al. 2015, 2016 Xu et al 2013 (rice), Fishman...).

Meiotic drive generally occurs either during the production of male or female  
100    gametes only (Úbeda and Haig 2005, Lindholm et al. 2016). Because there are  
102    typically many more pollen/sperm than required for fertilization, gametic competi-  
104    tion is also typically sex specific, occurring primarily among male gametes. Gametic  
106    competition may be particularly common in plants, in which 60-70% of all  
108    genes are expressed in the male gametophyte and these genes exhibit stronger sig-  
110    natures of selection than random genes (Borg et al. 2009, Arunkumar et al. 2013,  
112    Gossmann et al. 2014). In addition, artificial selection pressures applied to male  
114    gametophytes are known to cause a response to selection (e.g., Hormaza and Her-  
116    rero 1996, Ravikumar et al. 2003, Hedhly et al. 2004, Clarke et al. 2004). A smaller  
118    proportion of genes are thought to be expressed and selected during competition in  
   animal sperm, although precise estimates are uncertain (Zheng et al. 2001, Joseph  
   and Kirkpatrick 2004, Vibranovski et al. 2010). Recent studies have demonstrated  
   that sperm competition can alter haploid allele frequencies and increase offspring  
   fitness (Immler et al. 2014) (Alavioon et al. 2017).

114    There are various ways in which a period of haploid selection could influence  
116    transitions between sex-determination systems. If we assume that haploid selec-  
118    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-  
   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.

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. In addition to considering haploid selection, another novel development in our model is that we consider loci that are under diploid and/or haploid selection and also in very tight linkage with the ancestral sex-determining region. Even in the absence of haploid selection, we show that transitions between male and female heterogamy can then evolve despite the fact that 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.

## Model

Change all  $\alpha^{\delta}$  to  $(1 + \alpha_{\Delta}^{\delta})$ . I've attempted this everywhere except in the recursions (S.1), which seem more natural with  $\alpha$ 's. I've run into trouble in equation S.8c,d – I think we should check the Mathematica results to be sure we haven't made a

typo. This also introduced an extra 1/2 in S.6c,d that might need to be explained.

148 hmm, not sure. This was an idea from Sally, I think in response to terms like  $2\alpha^\delta$  and  $2(1 - \alpha^\delta)$ . It's possible that it makes other equations less easy to understand.

150 My previous (not explained) logic was to use  $w$  and  $\alpha$  for strong selection and  $s$ ,  
152  $t$ , and  $\alpha_\Delta$  for weak selection. Just to check, I should have written  $\alpha^\delta = (1 + \alpha_\Delta^\delta)/2$ ,  
154 maybe that's where the factors of 1/2 come from? shoot, i may have changed too  
156 much (i.e. strong selection too), but i did use the correct transformation. still, the  
form of S.8c,d seems wrong. I'll double check it. CANT FIND ANY ERRORS  
YET STILL HAVE A "2" IN S.8c. These 2s reappear in S.9 and S.10, always with

$W_{AA}$ .

We consider transitions between ancestral and novel sex-determining systems  
158 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  
160 under selection, with alleles  $A$  and  $a$ . Locus **M** is a novel sex-determining region,  
at which the null allele ( $M$ ) is initially fixed in the population such that sex of  
162 zygotes is determined by the genotype at the ancestral sex-determining region, **X**;  
 $XX$  genotypes become females and  $XY$  become males (or  $ZW$  become females  
164 and  $ZZ$  become males). To evaluate the evolution of new sex-determination sys-  
tems, we consider the invasion, fixation, maintenance, and/or loss of novel sex-  
166 determining alleles ( $m$ ) at the **M** locus. We assume that the **M** locus is epistatically  
dominant over the **X** locus such that zygotes with at least one  $m$  allele develop as  
168 females with probability  $k$  and as males with probability  $1 - k$ , regardless of the  
**X** locus genotype. With  $k = 0$ , the  $m$  allele is a masculinizer (i.e., a neo-Y) and  
170 with  $k = 1$  the  $m$  allele is a feminizer (i.e., a neo-W). With intermediate  $k$ , we can  
interpret  $m$  as an environmental sex determination (ESD) allele, such that zygotes  
172 develop as females in a proportion ( $k$ ) of the environments they experience.

In each generation, we census the genotype frequencies in male and female  
174 gametes/gametophytes (hereafter gametes) before gametic competition. A full de-  
scription of our model, including recursion equations, is given in the Appendix.  
176 First, competition occurs among male gametes (sperm/pollen competition) and

among female gametes (egg/ovule competition) separately. Selection during ga-  
178 metic competition depends on the **A** locus genotype, relative fitnesses are given  
by  $w_A^\varnothing$  and  $w_a^\varnothing$  ( $\varnothing \in \{\varnothing, \delta\}$ ; see table 1). We assume that all gametes compete for  
180 fertilization during gametic competition, which assumes a polygamous mating sys-  
tem. Gametic competition in monogamous mating systems is, however, equivalent  
182 to meiotic drive in our model (described below), as both only alter the frequency  
of gametes produced by heterozygotes. After gametic competition, random mating  
184 occurs between male and female gametes. The resulting zygotes develop as males  
or females, depending on their genotypes at the **X** and **M** loci. Diploid males and  
186 females then experience selection, with relative fitnesses  $w_{AA}^\varnothing$ ,  $w_{Aa}^\varnothing$ , and  $w_{aa}^\varnothing$ . The  
next generation of gametes is produced by meiosis, during which recombination  
188 and sex-specific meiotic drive can occur. Recombination (i.e., an odd number of  
cross-overs) occurs between loci **X** and **A** with probability  $r$ , between loci **A** and  
190 **M** with probability  $R$ , and between loci **X** and **M** with probability  $\rho$ . Any linear  
order of the loci can be modelled with appropriate choices of  $r$ ,  $R$ , and  $\rho$  (see Ta-  
192 ble S.1). Individuals that are heterozygous at the **A** locus may experience meiotic  
drive; a gamete produced by *Aa* heterozygotes of sex  $\varnothing$  bear allele *A* with probab-  
194 lity  $\alpha^\varnothing$ . Thus, the **A** locus can experience sex-specific gametic competition, diploid  
selection, and/or meiotic drive.

## 196 Results

The model outlined above describes both ancestrally-XY and ancestrally-ZW sex-  
198 determination systems if we relabel the two sexes as being ancestrally ‘heteroga-  
metic’ or ancestrally ‘homogametic’. Without loss of generality, we primarily re-  
200 fer to the ancestrally heterogametic sex as male and the ancestrally homogametic  
sex as female. That is, we describe an ancestral XY sex-determination system  
202 but our model is equally applicable to an ancestral ZW sex-determination sys-  
tem (relabelling the ancestrally-heterogametic sex as female and the ancestrally-  
204 homogametic sex as male).

Table 1: Relative fitness of different genotypes in sex  $\delta \in \{\text{♀}, \text{♂}\}$

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

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

206 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  
 208 spread of a rare mutant  $m$  at the **M** locus is determined by the leading eigenvalue,  
 210  $\lambda$ , of the system of eight equations describing the frequency of eggs and sperm  
 212 carrying the  $m$  allele in the next generation (equations S.1). This system simplifies  
 214 substantially in a number of cases of interest. Dominant neo-Y (when  $k = 0$ )  
 216 or neo-W alleles (when  $k = 1$ ) are only found in male diploids (neo-Y) or female  
 218 diploids (neo-W) such that their growth rate ultimately depends only on the  
 220 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 (**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, **Mention the possibility that the other roots yield the leading eigenvalue somewhere.** following the logic above, it seems like

we say we go from 8 eqns to 4 eqns by considering only one sex, and then from 4  
 222 eqns to 2 eqns by ignoring the ancestral SD allele. why then do we have the two  
 quadratic problem?  $\lambda^2 + b\lambda + c = 0$ . Here,  $b = -(\lambda_{mA} + \lambda_{ma}) + (\chi_{mA} + \chi_{ma})$  and  
 224  $c = (\lambda_{mA} - \chi_{mA})(\lambda_{ma} - \chi_{ma}) - \chi_{mA}\chi_{ma}$ , where  $\lambda_{mi}$  is the multiplicative growth rate  
 of mutant haplotypes on background  $i \in \{A, a\}$ , without accounting for loss due  
 226 to recombination, and  $\chi_{mi}$  is the rate at which mutant haplotypes on background  
 $i \in \{A, a\}$  recombine onto the other A locus background in heterozygotes (see  
 228 Table 2). The  $\lambda_{mi}$  and  $\chi_{mi}$ , and thus the spread of the mutant  $m$  allele, depend on  
 the frequency of alleles at the A and X loci in the ancestral population. In the an-  
 230 cestral population, it is convenient to follow the frequency of the A allele among  
 female gametes (eggs),  $p_X^{\varnothing}$ , and among X-bearing,  $p_X^{\delta}$ , and among Y-bearing,  $p_Y^{\delta}$ ,  
 232 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. We will  
 234 consider only equilibrium frequencies of alleles,  $\hat{p}_i^{\varnothing}$ , and Y-bearing male gametes,  
 $\hat{q}$ , to ensure the eigenvalues of the invasion analysis are valid.

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

neo-Y ( $k = 0$ )
$\lambda_{mA} = (2\zeta)^{-1} [\hat{p}_X^\varphi w_A^\varphi w_A^\delta w_{AA}^\delta + (1 - \hat{p}_X^\varphi) w_a^\varphi w_A^\delta w_{Aa}^\delta (1 + \alpha_\Delta^\delta)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\delta)$
$\lambda_{ma} = (2\zeta)^{-1} [(1 - \hat{p}_X^\varphi) w_a^\varphi w_a^\delta w_{aa}^\delta + \hat{p}_X^\varphi w_A^\varphi w_a^\delta w_{Aa}^\delta (1 - \alpha_\Delta^\delta)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\delta)$
$\chi_{mA} = R(2\zeta)^{-1} [(1 - \hat{p}_X^\varphi) w_a^\varphi w_A^\delta w_{Aa}^\delta (1 + \alpha_\Delta^\delta)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\delta)$
$\chi_{ma} = R(2\zeta)^{-1} [\hat{p}_X^\varphi w_A^\varphi w_a^\delta w_{Aa}^\delta (1 - \alpha_\Delta^\delta)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\delta)$
neo-W ( $k = 1$ )
$\lambda_{mA} = [2(1 - \zeta)]^{-1} [\bar{p}^\delta w_A^\delta w_A^\varphi w_{AA}^\varphi + (1 - \bar{p}^\delta) w_a^\delta w_A^\varphi w_{Aa}^\varphi (1 + \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$
$\lambda_{ma} = [2(1 - \zeta)]^{-1} [(1 - \bar{p}^\delta) w_a^\delta w_a^\varphi w_{aa}^\varphi + \bar{p}^\delta w_A^\delta w_a^\varphi w_{Aa}^\varphi (1 - \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$
$\chi_{mA} = R[2(1 - \zeta)]^{-1} [(1 - \bar{p}^\delta) w_a^\delta w_A^\varphi w_{Aa}^\varphi (1 + \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$
$\chi_{ma} = R[2(1 - \zeta)]^{-1} [\bar{p}^\delta w_A^\delta w_a^\varphi w_{Aa}^\varphi (1 - \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$

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

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

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

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

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

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

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

248 invasion requires that the growth rate of  $mA$  haplotypes and the rate at which they  
250 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  
haplotypes, weighted by the rates they are created by recombination, is positive.

252 Table 2 illustrates a number of key points about the invasion of neo-Y and  
254 neo-W mutations. First, Fisherian sex-ratio selection will favour the spread of a  
256 neo-Y if the ancestral zygotic sex ratio is biased towards females,  $\zeta < 1/2$  (i.e.,  
the first factor of the  $\lambda_{mi}$  is greater than one for a neo-Y and less than one for a neo-  
258 W). However, the spread of a neo-Y (neo-W) also depends on the male (female)  
fitness of associated alleles (terms involving equilibrium allele frequencies,  $p$ 's).  
260 Second, invasion by a neo-Y (neo-W) allele does not directly depend on the fitness  
of female (male) diploids (for a given set of equilibrium allele frequencies). This  
262 is because a dominant neo-Y (neo-W) is always found in males (females), and  
therefore the frequency of the neo-Y (neo-W) allele,  $m$ , only changes in males  
264 (females). Finally, invasions by a neo-Y and a neo-W are qualitatively different.  
This is because a gamete with the ancestral- or neo-Y always pairs with a female  
266 gamete containing an X, and both develop into males. By contrast, a gamete with  
a neo-W can pair with an X or Y male gamete, developing into a female, while  
268 female gametes without the neo-W can become female (when paired with X) or  
male (when paired with Y). Consequently, the types of females produced differ in  
the frequency of  $A$  alleles they obtain from mating.

270 In order to explicitly determine the conditions under which a rare neo-sex-  
determining allele spreads, we must calculate the equilibrium frequency of the  $A$   
allele (i.e.,  $\hat{p}_X^{\varnothing}$ ,  $\hat{p}_X^{\delta}$ , and  $\hat{p}_Y^{\delta}$ ) and Y-bearing male gametes ( $\hat{q}$ ) in the ancestral pop-  
272 ulation . Since only the A locus experiences selection directly, any deterministic  
evolution requires that there is a polymorphism at the A locus. Polymorphisms  
274 can be maintained by mutation-selection balance or transiently present during the  
spread of beneficial alleles. However, polymorphisms maintained by selection can  
276 maintain alleles at higher allele frequencies for longer periods. Here, we focus of  
polymorphisms maintained by selection, where the  $A$  allele reaches a stable in-

278 intermediate equilibrium frequency under the ancestral sex-determination system  
before the neo-sex-determining allele ( $m$ ) arises. We can analytically calculate the  
280 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  
282 ancestral SDR ( $r \approx 0$ ) or (2) selection is weak relative to recombination ( $s^{\delta}, t^{\delta}$ ,  
 $\alpha_{\Delta}^{\delta}$  of order  $\epsilon \ll 1$ ).

284 **Tight linkage with the ancestral sex-determining region**

The ancestral equilibrium allele frequencies and their stability conditions are given  
286 in the appendix. When there is complete linkage between the ancestral sex-determining  
region and the **A** locus ( $r = 0$ ), either the  $A$  allele or the  $a$  allele must be fixed on  
288 the Y. Because the labelling of alleles is arbitrary, we will assume that the  $a$  locus  
is fixed on the Y ( $p_Y^{\delta} = 0$ ), without loss of generality. If there are two alleles main-  
290 tained at the **A** locus, the X can either be fixed for the  $A$  allele ( $\hat{p}_X^{\delta} = \hat{p}_X^{\delta} = 1$ ) or  
polymorphic ( $0 < \hat{p}_X^{\delta}, \hat{p}_X^{\delta} < 1$ ).

292 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*  
294 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  
296 ( $\lambda_{mA} < 1$ ) 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 se-  
298 lected 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  
300 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  
302 males that have higher fitness than the ancestral Y.

Neo-W alleles, on the other hand, can invade an ancestral XY system under  
304 some conditions (the full invasion conditions are given in the appendix; equations  
S.6 and S.7). That is, selection on loci within the non-recombining region of the  
306 SDR can favour the invasion of a less closely linked neo-W, see Figure 1. This re-

sult is unexpected given the results of van Doorn and Kirkpatrick (2010), who did  
 308 not explicitly calculate equilibrium allele frequencies under tight linkage and generally concluded that heterogametic transitions occur when neo-sex-determining  
 310 alleles are in tighter linkage with loci under sex-specific diploid selection. To develop an understanding (intuition) for how this happens, we focus on cases where  
 312 there is no haploid selection and discuss the effects of haploid selection in the appendix.

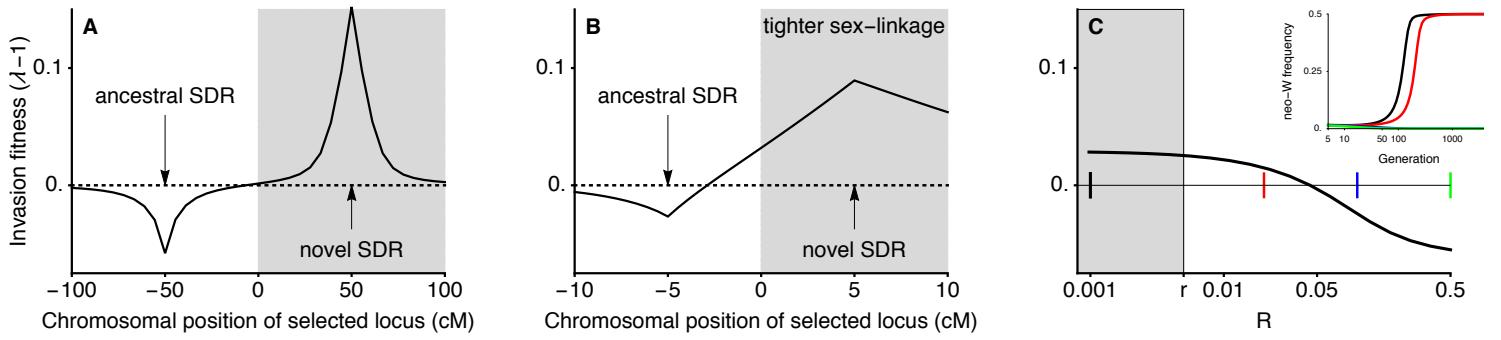


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is more loosely linked to a locus under sexually-antagonistic selection (here, without haploid selection  $r^\delta = \alpha_\Delta^\delta = 0$ ). In panel A, linkage is loose enough relative to selection that the weak selection 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). In panel C we vary the recombination rate between the neo-W and the selected locus ( $R$ ) for a fixed recombination rate between the ancestral-SDR and the selected locus ( $r = 0.005$ ). Coloured markers show recombination rates for which the temporal dynamics of invasion are plotted in the inset, demonstrating that neo-W alleles can fix (reach frequency 0.5 among female gametes) if they are more (black) or less (red) closely linked to a locus experiencing sexually-antagonistic selection. A very loosely linked neo-W does not spread in this case (blue and green lines overlap and go to 0). Indeed, we show that neo-W invasion fitness is negative when  $R = 1/2$  and  $s^{\delta} s^{\delta} < 0, 0 < h^{\delta} < 1$  in the supplementary *Mathematica* file. Fitness parameters are shown by an asterisk in Figure 2:  $w_{AA}^{\delta} = 1.05, w_{aa}^{\delta} = 1.2, w_{aa}^{\delta} = w_{AA}^{\delta} = 0.85, w_{Aa}^{\delta} = 1$ . consider removing panel A, which is repeated in Figure 3.

If we categorise the  $a$  allele as being ancestrally ‘male-beneficial’ via the fact  
 314 that it is fixed on the Y, then  $\lambda_{mA} > 1$  indicates that the neo-W spreads when found  
 316 with the ancestrally ‘female-beneficial’ allele. Broadly, this is possible because  
 318 the ancestral X chromosome is not able to perfectly specialise on the ‘female-  
 beneficial’ allele due to the fact that X’s are sometimes found in males. For ex-

ample, when the  $a$  allele is favoured in males, a polymorphism of  $A$  and  $a$  alleles  
320 can be maintained on the X despite directional selection in favour of the  $A$  allele in females ( $s^g > 0$ ,  $0 < h^g < 1$ ). Figure 2A indicates that  $\lambda_{mA}$  tends to be  
322 larger than one with sexually-antagonistic selection where the  $a$  allele is strongly  
favoured in males ( $w_{aa}$  much larger than  $w_{Aa}$ ). In this case the  $a$  allele is at high  
324 frequency among XX females is high due to selection upon the X in males. By  
contrast, W-A haplotypes will only create females with high fitness ( $AA$  or  $Aa$   
326 genotypes) and can therefore have higher fitness than ancestral females. When  
only one neo-W haplotype can has a positive growth rate (see Figure 2), a neo-W  
328 can invade as long as equation (1) is satisfied, which may require that the recom-  
bination rate,  $R$ , is small enough. Nevertheless, because we assume here that  $r$  is  
330 small, these results indicate that a more loosely linked sex-determining region can  
spread. Therefore, tightly sex-linked loci that experience sexually-antagonistic se-  
332 lection can drive heterogametic transitions in which the neo-SDR is less closely  
linked to the locus under selection (Figure 1).

334 Given that the  $a$  allele can be considered ancestrally ‘male-beneficial’ because  
it is fixed on the Y, it is surprising that neo-W- $a$  haplotypes can sometimes be  
336 favoured 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  
338 a Y- $a$ . Hence, if there is overdominance in males, X- $A$  Y- $a$  males have high fit-  
ness and the  $A$  allele is favoured by selection on the X in males. Therefore, the  
340 X can be polymorphic or even fixed for the  $A$  allele despite favouring the  $a$  allele  
during selection in females (e.g., see outlined region in Figure 2B and Lloyd and  
342 Webb 1977, Otto 2014). In such cases, neo-W- $a$  haplotypes can spread because  
they create more  $Aa$  and  $aa$  females when pairing with an X from males and be-  
344 cause they bring Y- $a$  haplotypes into females, in which case females are always  $aa$ .  
As discussed in the appendix, this scenario where neo-W’s associated with  $a$  are  
346 favoured can also occur with haploid selection, even without overdominance (e.g.,  
when  $a$  is female-beneficial and favoured by haploid selection in male gametes).

348 In some cases, both W-A and W- $a$  haplotypes can spread, e.g., when  $AA$  in-

dividuals have low fitness in females yet the *A* is polymorphic or fixed on the X  
 350 due to overdominance in males (Figure 2B and 2C). Both neo-W-*A* and neo-W-*a*  
 haplotypes then produce fewer unfit *AA* females. This is true for the neo-W-*A* hap-  
 352 lotype because it can pair with a Y-*a* haplotype and still be female. Wherever both  
 haplotypes have positive growth rates, invasion by a neo-W is expected regardless  
 354 of its linkage with the selected locus (i.e., even unlinked neo-W alleles can invade,  
 see Figures S.1 and S.2 for examples).

356 **What can we discuss about haploid selection here.** Perhaps the fact that over-  
 dominance is not required for  $\lambda_{Ma} > 1$  when there is haploid selection? We also  
 358 don't yet discuss the fact that polymorphic equilibria (mixed systems) can be sta-  
 ble.

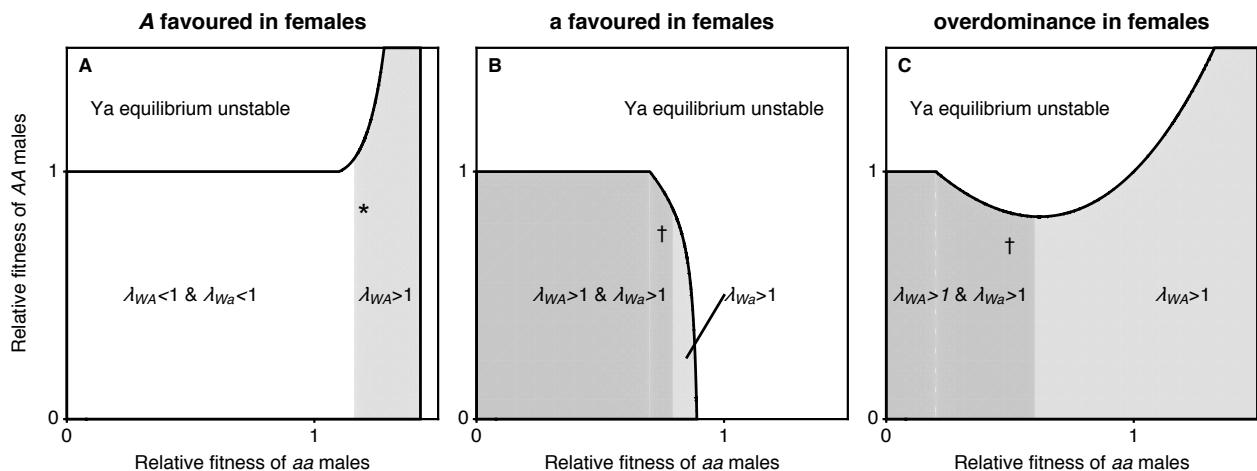


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ( $r = 0$ ), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ( $w_{Aa}^\varphi = 1$ ) and only consider stable equilibria at which both *A* locus alleles are maintained and the *a* allele is initially fixed on the Y, region outlined. Here, selection in females can favour the *A* allele (panel A,  $w_{aa}^0 = 0.85$ ,  $w_{AA}^0 = 1.05$ ), favour the *a* allele (panel B,  $w_{aa}^0 = 1.05$ ,  $w_{AA}^0 = 0.85$ ), or be overdominant (panel C,  $w_{aa}^0 = w_{AA}^0 = 0.6$ ). If  $\lambda_{wA}$  or  $\lambda_{wa}$  is greater than one, then a rare neo-W can spread for, at least, some values of  $R > r$ . For the parameter values marked with an asterisk, example invasion dynamics are shown in Figure 1C. Where both  $\lambda_{wA}$  and  $\lambda_{wa}$  are greater than one, a neo-W will spread when rare, regardless of linkage with the selected locus (for any  $R$ ). Figure S.1 shows two examples using the parameters marked with a dagger. Here, there is no haploid selection  $t^\varphi = \alpha_\Delta^\varphi = 0$ .

360 **Loose linkage with the ancestral sex-determining region**

Assuming that selection is weak relative to all recombination rates ( $r$ ,  $R$  and  $\rho$ ),  
 362 we denote the leading eigenvalues describing the invasion of a neo-Y ( $k = 0$ ) and  
 a neo-W ( $k = 1$ ) into an ancestrally XY system by  $\lambda_{Y',XY}$  and  $\lambda_{W',XY}$ , respectively.  
 364 To leading order in selection, these are:

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

and

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

366 where  $V_A = \bar{p}(1 - \bar{p})$  is the variance in the equilibrium frequency of  $A$  and  $S_A = (D^\delta + \alpha_\Delta^\delta + t^\delta) - (D^q + \alpha_\Delta^q + t^q)$  describes sex differences in selection for the  $A$  versus  
 368  $a$  across diploid selection, meiosis, and gametic competition. The diploid selection term,  $D^\delta = [\bar{p}s^\delta + (1 - \bar{p})h^\delta s^\delta] - [\bar{p}h^\delta s^\delta + (1 - \bar{p})]$ , is the difference in fitness  
 370 between  $A$  and  $a$  alleles in diploids of sex  $\delta \in \{\text{♀}, \text{♂}\}$ , where  $\bar{p}$  is the leading-order probability of mating with an  $A$ -bearing gamete from the opposite sex (see  
 372 Appendix). The difference in  $A$ -allele-frequency among Y-bearing sperm versus X-bearing sperm is given by  $\hat{p}_Y^\delta - \hat{p}_X^\delta = V_A (D^\delta - D^q + \alpha_\Delta^\delta - \alpha_\Delta^q + t^\delta - t^q) (1 - 2r) / 2r$ .  
 374 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  
 376 only if it is more closely linked to the selected locus than the ancestral sex-determining region (i.e., if  $R < r$ ; note that  $V_A S_A^2$  is strictly positive as long as  $A$  is polymorphic). This echoes our tight linkage results above where a neo-Y could never invade if  $r \approx 0$  and is consistent with the results of van Doorn and Kirkpatrick (2007),  
 380 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  
 382 more closely linked to a locus under sexually-antagonistic selection.

With weak selection and no haploid selection ( $t^\delta = \alpha_\Delta^\delta = 0$ ), the spread of

- 384 a neo-W is equivalent to the spread of a neo-Y ( $\lambda_{W',XY} = \lambda_{Y',XY}$ ), such that het-  
 385 erogametic transitions (XY to ZW or ZW to XY) can also occur only if the neo-  
 386 sex-determining region is more closely linked to a locus under selection ( $R < r$ ),  
 387 as found by van Doorn and Kirkpatrick (2010). With haploid selection, however,  
 388 the additional term in equation (3) can be positive, which can allow, for example,  
 389 neo-W invasion ( $\lambda_{W',XY} > 1$ ) even when the neo-sex-determining region is less  
 390 closely linked to the selected locus ( $R > r$ ).

Equation (3) shows that, with weak selection, neo-W alleles can invade an XY  
 392 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  
 394 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  
 396 *A* alleles and sex chromosomes in males,  $(\hat{p}_Y^\delta - \hat{p}_X^\delta) = 0$ , see equation (S.5). The  
 second term in equation (3) then disappears and invasion depends only on the sign  
 398 of  $(r - R)$ . Indeed, invasion typically occurs when the neo-W is more closely linked  
 to the selected locus than the ancestral sex-determining region, under a variety of  
 400 selective regimes (Figure 3). Secondly, we can simplify the discussion of cases  
 where invasion occurs despite looser sex-linkage,  $R > r$ , by focussing on the  
 402 special case where  $R = 1/2$  and  $r < 1/2$  (e.g., the selected locus is on the ancestral  
 sex chromosome and the novel sex-determining locus arises on an autosome). In  
 404 table 3 we give the conditions where invasion occurs when we further assume  
 that haploid selection only occurs in one sex (e.g., during male meiosis only) and  
 406 dominance coefficients are equal in the two sexes,  $h^q = h^\delta$ . When there is no  
 gametic competition and meiotic drive is in one sex only, an unlinked neo-W can  
 408 invade as long as the same allele is favoured during diploid selection in males and  
 females ( $s^q s^\delta > 0$ , see Figure 3B and Figure 4B). When there is no meiotic drive  
 410 and gametic competition occurs in one sex only, an unlinked neo-W can invade as  
 long as the same allele is favoured in male and female diploid selection and there  
 412 are sex differences in selection of one type (e.g.,  $s^q(s^\delta - s^q) > 0$ , see Figure 3C).  
 These special cases indicate that neo-W invasion can occur for a relatively large

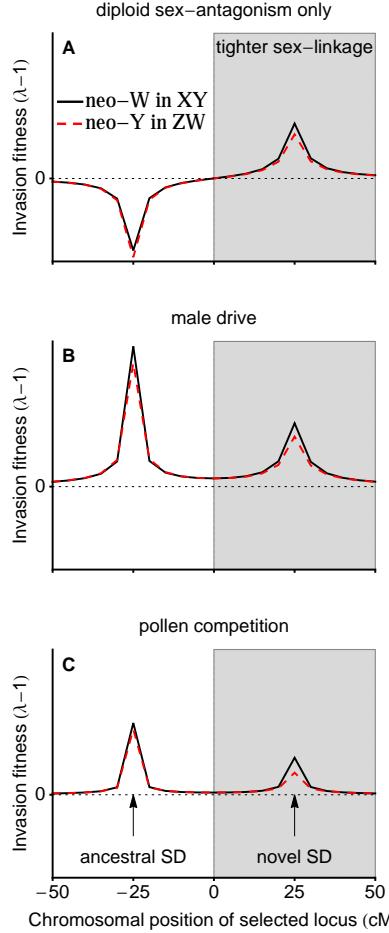


Figure 3: A neo-W can invade an XY system under a large number of selective regimes. In panel A, there is no haploid selection ( $t^\delta = \alpha_\Delta^\delta = 0$ ) and selection in diploids is sexually antagonistic ( $s^\delta = -s^q = 1/10$ ,  $h^\delta = 1 - h^q = 3/10$ ), in which case the neo-sex-determining 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 ( $\alpha_\Delta^\delta = -1/20$ ,  $t^\delta = \alpha_\Delta^\delta = 0$ ) 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, gametic competition in males ( $t^\delta = -1/10$ ,  $t^q = \alpha_\Delta^q = 0$ ) opposes selection in diploids (sex-differences:  $s^\delta = 1/20$ ,  $s^q = 3/20$ ,  $h^\delta = 7/10$ ), 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). **Check the mismatch between red and black lines here: probably because of adding or subtracting from 1. Can remove the mismatch by flipping the fitnesses between males and females (again). That is, if  $M_{AA}$  is the fitness of AA male diploids in an ancestral XY system, then  $M_{AA}$  is the fitness of AA female diploids in an ancestral ZW system. I think this makes sense in A, where we don't really want a difference between the red and black curves, but this makes less sense in B and C where we want to restrict haploid selection to males regardless of the ancestral system. We could just not flip the haploid fitnesses, but then does it make sense to flip the diploid fitnesses?**

<sup>414</sup> fraction of the parameter space, even if the neo-W uncouples the 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	$h^\delta = h^q, t^q = t^\delta = \alpha_\Delta^q = 0$	$s^q s^\delta > 0$
female drive only	$h^\delta = h^q, t^q = t^\delta = \alpha_\Delta^\delta = 0$	$s^q s^\delta > 0$
sperm competition only	$h^\delta = h^q, t^q = \alpha_\Delta^q = \alpha_\Delta^\delta = 0$	$s^q(s^\delta - s^q) > 0$
egg competition only	$h^\delta = h^q, t^\delta = \alpha_\Delta^q = \alpha_\Delta^\delta = 0$	$s^\delta(s^q - s^\delta) > 0$

<sup>416</sup> Previous research suggests that when the ancestral sex-determining locus is linked to a locus that experiences haploid selection (e.g., meiotic drive), a new, <sup>418</sup> unlinked sex-determining locus invades in order to restore equal sex ratios (Kozielska et al. 2010). Consider, for example, the case where the A locus is linked to the <sup>420</sup> ancestral-SDR ( $r < 1/2$ ) and experiences meiotic drive in males only (e.g., during spermatogenesis but not during oogenesis,  $\alpha_\Delta^\delta \neq 0, \alpha_\Delta^q = 0$ ), without gametic <sup>422</sup> competition ( $t^q = t^\delta = 0$ ). In this case, the zygotic sex ratio can be initially biased only if the ancestral sex-determining system is XY (Figure 4B). We might therefore <sup>424</sup> expect a difference in the potential for XY to ZW and ZW to XY transitions. However, to leading order with selection weak relative to recombination, we find <sup>426</sup> that sex ratio selection favours the spread of a neo-W (through the first terms in table 2) by an amount that is equal in magnitude to the fitness effects of alleles <sup>428</sup> associated with new sex-determining alleles (second terms in table 2). Thus, invasion by a neo-W into an XY system and invasion by a neo-Y into a ZW system <sup>430</sup> occur under the same conditions ( $\lambda_{Y',XY} = \lambda_{W',ZW}$  and  $\lambda_{Y',ZW} = \lambda_{W',XY}$ , at least to order  $\epsilon^2$ ). For example, in Figure 4B neo-W alleles invade an ancestrally-XY <sup>432</sup> system where females are initially rare because the ancestral-Y is associated with a male meiotic drive allele. However, Figure 4A shows that a neo-Y can invade <sup>434</sup> an ancestrally-ZW system under the same conditions. In fact, where  $R < 1/2$  the neo-Y becomes associated with the male meiotic drive allele such that the zygotic

436 sex ratio evolves to become biased towards males.

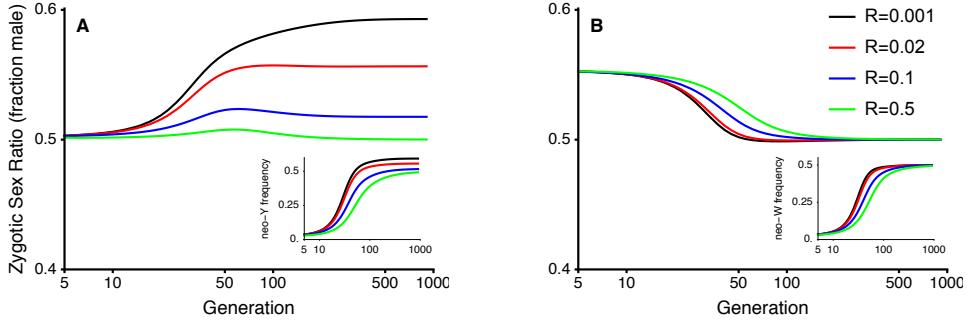


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidally antagonistic with haploid selection favouring the  $a$  allele during male meiosis ( $s^q = s^\delta = 0.2$ ,  $h^q = h^\delta = 0.7$ ,  $t^q = t^\delta = \alpha_\Delta^\delta = 0$ ). In panel A, male drive in an ancestral ZW system has no affect on the zygotic sex ratio, yet a neo-Y can invade and replace the ancestral sex-determination system (inset shows neo-Y frequency among male gametes, the ancestral W also goes to fixation during this transition). When  $R < 1/2$ , the neo-Y becomes associated with the allele favoured by drive, causing the zygotic sex ratio to become biased, hence the frequency of neo-Y among male gametes can be higher than 0.5 (inset). In panel B, male drive in an ancestral XY system causes a male bias, allowing a neo-W to invade and replace the ancestral sex-determination system (inset shows neo-W frequency among female gametes, the ancestral Y also goes to fixation), which balances the zygotic sex ratio. Parameters:  $s^q = s^\delta = 0.2$ ,  $h^q = h^\delta = 0.7$ ,  $t^q = t^\delta = \alpha_\Delta^\delta = 0$ ,  $\alpha_\Delta^\delta = -0.2$ ,  $r = 0.02$ .

The green curves in Figure 4 demonstrate a case where transitions between  
 438 male and female heterogamety occur even though the new sex-determining re-  
 gion is unlinked to a locus that experiences haploid and diploid selection. We  
 440 use these green curves to discuss why heterogametic transitions can occur when  
 $R = 1/2$  and  $r < 1/2$ , as in Table 3. In Figure 4B, an unlinked neo-W can spread  
 442 because the zygotic sex ratio is ancestrally male biased. However, in Figure 4A, an  
 unlinked neo-Y spreads despite the fact that the ancestral zygotic sex ratio is even.  
 444 In this case, the the male meiotic drive allele,  $a$ , is initially more common among  
 ancestral-Z-bearing eggs than ancestral-W-bearing eggs because the Z is found in  
 446 males more often than the W and  $r < 1/2$  (equation S.5). Polymorphism at the A  
 locus is maintained by counter-selection against the  $a$  allele in diploids and there-  
 448 fore ancestral-ZZ males have generally low diploid fitness. A freely recombining  
 neo-Y ( $R = 1/2$ ) is not directly favoured or disfavoured by male meiotic drive  
 450 because it is equally likely to be segregate with the  $A$  or  $a$  allele when found in  
 a heterozygote. The neo-Y spreads because it produces males with high diploid

452 fitness through matings with ancestral-W-bearing female gametes, which are more  
 453 likely to carry the  $A$  allele. Thus, a key factor in explaining why heterogametic  
 454 transitions can occur when  $R > r$  is that the neo-SDR determines sex in the  
 diploid phase but recombination occurs before any subsequent haploid selection.

456 **Environmental sex determination**

We next consider the case where the new sex-determining mutation,  $m$ , causes sex  
 458 to be determined probabilistically or by heterogeneous environmental conditions  
 (environmental sex determination, ESD), with individuals carrying allele  $m$  devel-  
 460 oping as females with probability  $k$ . Here, we do not assume that the environmen-  
 461 tal conditions that determine sex also differentially affect the fitness of males versus  
 462 females. Such correlations can favour environmental sex-determination systems  
 463 that allow each sex to be produced in the environment in which it has highest fit-  
 464 ness; in the absence of these correlations, previous theory would predict that ESD  
 465 is favoured when it produces more equal sex ratios than the ancestral system (see  
 466 reviews by Charnov 1982, Bull 1983, West 2009).

The characteristic polynomial determining the eigenvalues (equations S.1) does  
 468 not factor for ESD mutants as it does for  $k = 0$  or  $k = 1$ . We therefore focus  
 on weak selection here. Assuming weak selection, the spread of the new sex-  
 470 determining region is given by

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

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

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

<sup>474</sup> spread of which is given by

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

<sup>476</sup> where  $\lambda_{Y',XY|R=1/2}$  and  $\lambda_{W',XY|R=1/2}$  represent  $\lambda_{Y',XY}$  and  $\lambda_{W',XY}$  when evaluated  
<sup>476</sup> at  $R = 1/2$  (equations 2 and 3). That is, recombination between the selected locus  
and the novel sex-determining locus,  $R$ , doesn't enter into the  $k = 1/2$  results. This  
<sup>478</sup> is because sex is essentially randomized each generation, preventing associations  
from building up between allele  $A$  and sex. Equation (5) shows that the neo-ESD  
<sup>480</sup> 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  
<sup>482</sup> (the other half of the time it produces the same sex as the ancestral system would  
have, to leading order). As discussed above,  $\lambda_{Y',XY|R=1/2}$  is necessarily less than  
<sup>484</sup> one, but  $\lambda_{W',XY|R=1/2}$  can be greater than one if there is haploid selection. That  
is, when there is haploid selection, ESD mutations can invade an ancestrally-XY  
<sup>486</sup> system because they generate females that are either rare or have high fitness, in  
the same manner as a neo- $W$ .

<sup>488</sup> Significantly, equation (5) is the same whether ESD is invading an ancestrally  
XY or ZW system (because  $\lambda_{Y',XY} = \lambda_{W',ZW}$  and  $\lambda_{W',XY} = \lambda_{Y',ZW}$ ). Thus, be-  
<sup>490</sup> cause the sex ratio is only biased by male haploid selection when the ancestral  
sex-determination system is XY, Fisherian sex-ratio selection alone does not ex-  
<sup>492</sup> plain the invasion of ESD under weak selection. Specifically, with male haploid  
selection, the neo-ESD is equally likely to invade when it equalizes the zygotic sex  
<sup>494</sup> ratio (through  $\lambda_{W',XY}$ ) and when it doesn't (through  $\lambda_{Y',ZW}$ ). In addition, we note  
that ESD may not invade, even if the sex ratio is initially biased (e.g., with drive in  
<sup>496</sup> males only,  $r < 1/2$ ,  $h^{\varphi} = h^{\delta}$ , and  $s^{\varphi}s^{\delta} < 0$ , such that  $\lambda_{W',XY} < 1$ , see Table 3).

## Discussion

498 Two predominant theories explaining the remarkably high frequency of transitions  
between sex-determination systems are sexually-antagonistic selection and sex-  
500 ratio selection (reviewed in Blaser et al. 2012) (van Doorn, 2014, sexual develop-  
ment). The former predicts that neo-sex-determining alleles can invade when they  
502 arise in closer linkage with a sexually-antagonistic locus (van Doorn and Kirk-  
patrick 2007; 2010). The latter predicts that new sex-determining systems are  
504 generally favoured if they result in more equal sex-ratios than the ancestral sys-  
tem. Firstly, we show that selection (including sexually-antagonistic selection) on  
506 loci within or near the non-recombining region of the ancestral sex-determining  
region can favour heterogametic transitions (XY to ZW or ZW to XY) to new sex-  
508 determining systems that are less closely linked to the selected loci (e.g., see Figure  
1). Secondly, assuming that selection is weak relative to recombination ('weak se-  
510 lection'), we show that new sex-determining alleles are typically favoured if they  
are more closely linked to a locus under haploid selection, which is the only con-  
512 dition favouring homogametic transitions (XY to XY or ZW to ZW). In addition,  
with haploid selection and weak selection, heterogametic transitions (XY to ZW  
514 or ZW to XY) can occur even when the new sex-determining region is less closely  
linked to the locus under selection (e.g., see Figure 4). need to mention sex ratio  
516 here

518 Sex-ratio biases caused by haploid selection can facilitate heterogametic transi-  
tions between sex-determining systems. For instance, alleles favoured by haploid  
selection in males often become associated with the Y, which leads to a male-  
520 biased zygotic sex-ratio. This male bias increases the potential for a neo-W to  
invade (Table 2), which can equalize the sex-ratio (e.g., see Figure 4B, for related  
522 examples see Kozielska et al. 2010, Úbeda et al. 2015). However, sex-ratio se-  
lection can be overwhelmed by additional selective effects (e.g., when a linked  
524 allele is beneficial for male diploids but detrimental for female diploids; Table 3),  
preventing the neo-W from invading. Indeed, transitions between sex-determining  
526 systems can even lead to stronger sex-ratio biases. For example, where a neo-Y

invades and is linked with a locus that experiences haploid selection in male gametes, the sex ratio evolves to become biased (e.g., see Figure 4A and step 1 in Úbeda et al. 2015). Furthermore, with weak selection, we find that there is no difference in conditions allowing XY to ZW and ZW to XY transitions, indicating that sex chromosome transitions are not predominantly predicted by their effect on the sex-ratio (i.e., the sex-ratio bias created by male haploid selection facilitates the spread of a neo-W into an XY system the same way that male haploid selection drives the spread of a neo-Y into a ZW system with a 1:1 sex ratio). Thus, haploid selection can favour heterogametic transitions both via sex-ratio selection and via fitness effects of alleles that are associated with the neo-sex-determining allele, and these selection pressures are predicted to often be of equal magnitude when selection is weak.

We have shown that the spread of new sex determination systems can be driven by loci experiencing haploid selection. 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 of the red line in Figure 4A is the starting state in Figure 4B). In particular, if haploid selection is strong but selective differences between male and female diploids are weak, we find that heterogametic transitions (XY to ZW or vice versa) are favoured more strongly than homogametic transitions (e.g., with  $|D^\delta - D^Q| << |\alpha_\Delta^\delta - \alpha_\Delta^Q + t^\delta - t^Q|$  we have  $\lambda_{W',XY} > \lambda_{Y',XY}$ ; equations 3 and S.5). Turnovers driven by haploid selection may help to explain the relative rarity of heteromorphic sex chromosomes in plants, which are thought to experience more selection during their multicellular haploid stage. For example, among relatively few dioecious clades in which multiple species have well characterized sex chromosomes (Ming et al. 2011), heterogametic transitions have been inferred in *Silene* subsection *Otites* (Slancarova et al. 2013) and in *Salicaceae* (Pucholt et al. 2015, 2017). 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 competition during the haploid stage  
558 could drive transitions between dioecy and hermaphroditism, which are frequent  
in plants (Käfer et al., 2017, Sabath et al., 2017).

560 In support of their role in sex chromosome turnover, genes expected to be un-  
der sexually-antagonistic selection (e.g., those causing bright male colouration)  
562 have been found on recently derived sex chromosomes (Lindholm and Breden  
2002, Tripathi et al. 2009, Ser et al. 2010). Our results show that, if loci ex-  
564 periencing overdominance and/or sexually-antagonistic selection can be identi-  
fied in close linkage with the ancestral sex-determining locus (rather than only  
566 the novel sex-determining locus), then they could also be implicated in driving  
heterogametic transitions between sex-determination systems. As noted by van  
568 Doorn and Kirkpatrick (2010), it would be prudent to compare closely related  
clades in order to determine whether observed polymorphisms predate a transi-  
570 tion in sex-determination or arose afterwards. In addition, we show haploid se-  
lection on loci around either the ancestral- or the novel-sex-determining regions  
572 could have had a role in driving sex chromosome turnover. A recent transcript-  
ome analysis in *Rumex*, suggests a role for gametic competition in the evolution  
574 of sex-determination systems, showing that Y-linked genes are have higher expres-  
sion in haploid pollen than autosomal genes (check this is accurate). Interestingly,  
576 haploid-expression is also more common on the autosome that is orthologous to the  
sex chromosomes in closely related species suggesting that new sex chromosomes  
578 may have been favoured through their association with haploid selected alleles on  
these chromosomes (Sandler et al., 2017, Personal Communication).

580 We assume that sex-determining alleles do not experience direct selection ex-  
cept via their associations with sex and selected alleles. However, in some cases,  
582 there may be significant degeneration around the sex-limited allele (Y or W) in the  
ancestral sex-determining region because recessive deleterious mutations and/or  
584 deletions accumulate around the Y or W sex-determining regions (Rice 1996,  
Charlesworth and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). During  
586 heterogametic transitions (XY to ZW or ZW to XY), but not homogametic transi-

tions (XY to XY or ZW to ZW), any recessive deleterious alleles linked to the Y or  
588 W are revealed to selection in YY or WW individuals (Bachtrog et al. 2014). This  
phenomenon was studied by van Doorn and Kirkpatrick (2010), who found that  
589 degeneration can prevent fixation of a neo-W or a neo-Y allele, leading to a mixed  
sex-determination system where the ancestral and new sex-determining loci are  
590 both segregating. However, they noted that very rare recombination events around  
the ancestral sex-determining region can allow these heterogametic transitions to  
591 complete. Degeneration around the Y or W could explain why heterogametic transi-  
tions are not observed to be much more common than homogametic transitions  
592 despite the fact that our models demonstrate that they are favoured under a wider  
range of conditions. For example, Vicoso and Bachtrog (2015) found a dozen  
593 sex chromosome configurations among Dipteran species but only one transition  
594 between male and female heterogametey.

600 Another simplification that we made is that meiotic drive involves only a single  
locus with two alleles. However, many meiotic drive systems involve an interac-  
602 tion with another locus at which alleles may ‘suppress’ the action of meiotic drive  
(Burt and Trivers 2006, Lindholm et al. 2016) Taylor,1999. Thus, the dynamics  
604 of meiotic drive alleles can be heavily dependent on the interaction between two  
loci and the recombination rate between them, which in turn can be affected by  
606 sex-linkage if there is reduced recombination between sex chromosomes (Hurst  
and Pomiankowski 1991). Furthermore, in some cases, a driving allele may act  
608 by killing any gametes that carry a ‘target’ allele at another locus, in which case  
there can be fertility effects which can affect the equilibrium frequency of a meiotic  
610 drive allele (Holman et al. 2015). In polygamous mating systems, the intensity of  
pollen/sperm competition can depend on the density of males available to donate  
612 pollen/sperm, which can itself depend on the sex ratio (Taylor and Jaenike 2002).  
In terms of our model, this implies that the strength of gametic competition ( $t^d$ )  
614 may both determine and be determined by the sex ratio. How the evolution of  
new sex-determining mechanisms could be influenced by two-locus meiotic drive  
616 and/or by ecological feedbacks under different mating systems remains to be stud-

ied.

618 We have shown that tight sex-linkage and haploid selection can drive previously  
619 unexpected transitions between sex-determination systems. In particular,  
620 both can select for neo-sex-determining loci that are more loosely linked. In addition,  
621 haploid selection alone can cause transitions analogous to those caused by  
622 purely sexually-antagonistic selection, eliminating the need for differences in se-  
623 lection between male and female diploids. Perhaps counterintuitively, transitions  
624 involving haploid selection can be driven by sex-ratio selection or cause sex-ratio  
625 biases to evolve. We conclude that haploid selection should be considered as a  
626 pivotal factor driving transitions between sex-determination systems. Overall, our  
627 results suggest several new scenarios under which new sex-determination systems  
628 are favoured, which could help to explain why the evolution of sex-determination  
systems is so dynamic.

630 Discuss polymorphic mating systems somewhere? Say that haploid selection  
631 makes this particularly likely (I think there are examples with gametic competition  
632 and weak selection, whereas the vD&K, 2010 results suggest that it's not possible  
633 with weak selection and diploid selection alone)? This might be best as a section  
634 added to the appendix. When giving an example of polymorphic, make sure it's  
635 not just that variation was lost at the A locus. The following examples copied and  
636 pasted from from Vuilleumier et al. 2007 and vD&K, 2010, might be added to this  
section.

638 “Polygenic sex determination has been reported in many plants (e.g. Shannon  
639 & Holsinger 2007), fishes (Vandeputte et al. 2007; Ser et al. 2010; Liew et al.  
640 2012), crustaceans (e.g. Battaglia 1958; Battaglia & Malesani 1959; Voordouw  
641 & Anholt 2002), bivalves (Haley 1977; Saavedra et al. 1997), gastropods (Yusa  
642 2007a,b), and polychaetes (Bacci 1965, 1978; Premoli et al. 1996).” From Vuilleu-  
643 mier et al. 2007: “Polymorphism for sex-determining genes within or among pop-  
644 ulations has been reported in many species including houseflies, midges, woodlice,  
645 platyfish, cichlid fish, and frogs (Gordon, 1944; Kallman, 1970; Thompson, 1971;  
646 Macdonald, 1978; Bull, 1983; Rigaud et al., 1997; Caubet et al., 2000; Lande et

al., 2001; Ogata et al., 2003; Lee et al., 2004; Mank et al., 2006).” Also check  
648 Kallman (1984) -from vD&K, 2010.

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# Appendix

## 836 Recursion Equations

In each generation we census the genotype frequencies in male and female gametes/gametophytes (hereafter, gametes) between meiosis (and any meiotic drive) and gametic competition. At this stage we denote the frequencies of X- and Y-bearing gametes from males and females  $x_i^\varphi$  and  $y_i^\varphi$ , where  $\varphi \in \{\delta, \Omega\}$  specifies the sex of the diploid that the gamete came from.  $i \in \{1, 2, 3, 4\}$  specifies the genotype at the selected locus **A** and at the novel sex-determining locus **M** where  $1 = MA$ ,  $2 = Ma$ ,  $3 = mA$ , and  $4 = ma$ . The gamete frequencies from each sex sum to one,  $\sum_i x_i^\varphi + y_i^\varphi = 1$ .

Competition then occurs among gametes of the same sex (e.g., among eggs and among sperm separately) according to the genotype at the **A** locus ( $w_1^\varphi = w_3^\varphi = w_A^\varphi$ ,  $w_2^\varphi = w_4^\varphi = w_a^\varphi$ , see Table 1). The genotype frequencies after gametic competition are  $x_i^{\varphi,s} = w_i x_i^\varphi / \bar{w}_H^\varphi$  and  $y_i^{\varphi,s} = w_i y_i^\varphi / \bar{w}_H^\varphi$ , where  $\bar{w}_H^\varphi = \sum_i w_i x_i^\varphi + w_i y_i^\varphi$  is the mean fitness of male ( $\varphi = \delta$ ) or female ( $\varphi = \Omega$ ) gametes.

Random mating then occurs between gametes to produce diploid zygotes. The frequencies of XX zygotes are then denoted as  $xx_{ij}$ , XY zygotes as  $xy_{ij}$ , and YY zygotes as  $yy_{ij}$ , where **A** and **M** locus genotypes are given by  $i, j \in \{1, 2, 3, 4\}$ , as above. In XY zygotes, the haplotype inherited from an X-bearing gamete is given by  $i$  and the haplotype from a Y-bearing gamete is given by  $j$ . In XX and YY zygotes, individuals with 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,  $xx_{ij} = (x_i^{\varphi,s} x_j^{\delta,s} + x_j^{\varphi,s} x_i^{\delta,s})/2$  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 genotype by  $c \in \{XX, XY, YY\}$ , zygotes develop as females with probability  $k_{bc}$ . Therefore, the frequencies of XX females are given by  $xx_{ij}^\Omega = k_{bc} xx_{ij}$ , XY females are given by  $xy_{ij}^\Omega = k_{bc} xy_{ij}$ , and YY females are given by  $yy_{ij}^\Omega = k_{bc} yy_{ij}$ . Similarly, XX male frequencies are  $xx_{ij}^\delta = (1 - k_{bc}) xx_{ij}$ , XY male frequencies are  $xy_{ij}^\delta = (1 - k_{bc}) xy_{ij}$ , and YY males frequencies are  $yy_{ij}^\delta = (1 - k_{bc}) yy_{ij}$ . This

864 notation allows both the ancestral and novel sex-determining regions to determine  
 865 zygotic sex according to an XY system, a ZW system, or an environmental sex-  
 866 determining system. In addition, we can consider any epistatic dominance rela-  
 867 tionship between the two sex-determining loci. Here, we assume that the ancestral  
 868 sex-determining system (**X** locus) is XY ( $k_{MMXX} = 1$  and  $k_{MMXY} = k_{MMYY} = 0$ )  
 869 or ZW ( $k_{MMZZ} = 0$  and  $k_{MMZW} = k_{MMWW} = 1$ ) and epistematically recessive to a  
 870 dominant novel sex-determining locus, **M** ( $k_{Mmc} = k_{mmc} = k$ ).

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

875 Finally, these diploids undergo meiosis to produce the next generation of ga-  
 876 metes. Recombination and sex-specific meiotic drive occur during meiosis. Here,  
 877 we allow any relative locations for the SDR, **A**, and **M** loci by using three param-  
 878 eters to describe the recombination rates between them.  $R$  is the recombination  
 879 rate between the **A** locus and the **M** locus,  $\rho$  is the recombination rate between the  
 880 **M** locus and the **X** locus, and  $r$  is the recombination rate between the **A** locus and  
 881 the **X** locus. Table S.1 shows the value of  $\rho$  in the absence of cross-over inter-  
 882 ference for each possible ordering of the loci. During meiosis in sex  $\hat{\varphi}$ , meiotic drive  
 883 occurs such that, in  $Aa$  heterozygotes, a fraction  $\alpha^{\hat{\varphi}}$  of gametes produced carry the  
 884  $A$  allele and  $(1 - \alpha^{\hat{\varphi}})$  carry the  $a$  allele.

Table S.1: Values of  $\rho$  for different loci orders assuming no interference and  $r, R \in (0, 1/2)$ .

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

885 Among gametes from sex  $\hat{\varphi}$ , the frequencies of haplotypes (before gametic

competition) in the next generation are given by

$$\begin{aligned}
x_1^{\phi'} = & xx_{11}^{\phi,s} + xx_{13}^{\phi,s}/2 + (xx_{12}^{\phi,s} + xx_{14}^{\phi,s})\alpha^{\phi} \\
& - R(xx_{14}^{\phi,s} - xx_{23}^{\phi,s})\alpha^{\phi} \\
& + (xy_{11}^{\phi,s} + xy_{13}^{\phi,s})/2 + (xy_{12}^{\phi,s} + xy_{14}^{\phi,s})\alpha^{\phi} \\
& - r(xy_{12}^{\phi,s} - xy_{21}^{\phi,s})\alpha^{\phi} - \rho(xy_{13}^{\phi,s} - xy_{31}^{\phi,s})/2 \\
& + [-(R+r+\rho)xy_{14}^{\phi,s} + (R+\rho-r)xy_{41}^{\phi,s} \\
& + (R+r-\rho)xy_{23}^{\phi,s} + (R+\rho-r)xy_{32}^{\phi,s}] \alpha^{\phi}/2
\end{aligned} \tag{S.1a}$$

$$\begin{aligned}
x_2^{\phi'} = & xx_{22}^{\phi,s} + xx_{24}^{\phi,s}/2 + (xx_{12}^{\phi,s} + xx_{23}^{\phi,s})\alpha^{\phi} \\
& - R(xx_{23}^{\phi,s} - xx_{14}^{\phi,s})\alpha^{\phi} \\
& (xy_{22}^{\phi,s} + xy_{24}^{\phi,s})/2 + (xy_{21}^{\phi,s} + xy_{23}^{\phi,s})(1 - \alpha^{\phi}) \\
& - r(xy_{21}^{\phi,s} - xy_{12}^{\phi,s})(1 - \alpha^{\phi}) - \rho(xy_{24}^{\phi,s} - xy_{42}^{\phi,s})/2 \\
& + [-(R+r+\rho)xy_{23}^{\phi,s} + (R+\rho-r)xy_{32}^{\phi,s} \\
& + (R+r-\rho)xy_{14}^{\phi,s} + (R+\rho-r)xy_{41}^{\phi,s}] (1 - \alpha^{\phi})/2
\end{aligned} \tag{S.1b}$$

$$\begin{aligned}
x_3^{\phi'} = & xx_{33}^{\phi,s} + xx_{13}^{\phi,s}/2 + (xx_{23}^{\phi,s} + xx_{34}^{\phi,s})\alpha^{\phi} \\
& - R(xx_{23}^{\phi,s} - xx_{14}^{\phi,s})\alpha^{\phi} \\
& (xy_{33}^{\phi,s} + xy_{31}^{\phi,s})/2 + (xy_{32}^{\phi,s} + xy_{34}^{\phi,s})\alpha^{\phi} \\
& - r(xy_{34}^{\phi,s} - xy_{43}^{\phi,s})\alpha^{\phi} - \rho(xy_{31}^{\phi,s} - xy_{13}^{\phi,s})/2 \\
& + [-(R+r+\rho)xy_{32}^{\phi,s} + (R+\rho-r)xy_{23}^{\phi,s} \\
& + (R+r-\rho)xy_{41}^{\phi,s} + (R+\rho-r)xy_{14}^{\phi,s}] \alpha^{\phi}/2
\end{aligned} \tag{S.1c}$$

$$\begin{aligned}
x_4^{\phi'} = & xx_{44}^{\phi,s} + xx_{34}^{\phi,s}/2 + (xx_{14}^{\phi,s} + xx_{24}^{\phi,s})\alpha^\phi \\
& - R(xx_{14}^{\phi,s} - xx_{23}^{\phi,s})\alpha^\phi \\
& (xy_{44}^{\phi,s} + xy_{42}^{\phi,s})/2 + (xy_{41}^{\phi,s} + xy_{43}^{\phi,s})(1 - \alpha^\phi) \\
& - r(xy_{43}^{\phi,s} - xy_{34}^{\phi,s})(1 - \alpha^\phi) - \rho(xy_{42}^{\phi,s} - xy_{24}^{\phi,s})/2
\end{aligned} \tag{S.1d}$$

$$\begin{aligned}
y_1^{\phi'} = & yy_{11}^{\phi,s} + yy_{13}^{\phi,s}/2 + (yy_{12}^{\phi,s} + yy_{14}^{\phi,s})\alpha^\phi \\
& - R(yy_{14}^{\phi,s} - yy_{23}^{\phi,s})\alpha^\phi \\
& (xy_{11}^{\phi,s} + xy_{31}^{\phi,s})/2 + (xy_{21}^{\phi,s} + xy_{41}^{\phi,s})\alpha^\phi \\
& - r(xy_{21}^{\phi,s} - xy_{12}^{\phi,s})\alpha^\phi - \rho(xy_{31}^{\phi,s} - xy_{13}^{\phi,s})/2
\end{aligned} \tag{S.1e}$$

$$\begin{aligned}
y_2^{\phi'} = & yy_{22}^{\phi,s} + yy_{24}^{\phi,s}/2 + (yy_{12}^{\phi,s} + yy_{23}^{\phi,s})\alpha^\phi \\
& - R(yy_{23}^{\phi,s} - yy_{14}^{\phi,s})\alpha^\phi \\
& (xy_{22}^{\phi,s} + xy_{42}^{\phi,s})/2 + (xy_{12}^{\phi,s} + xy_{32}^{\phi,s})(1 - \alpha^\phi) \\
& - r(xy_{12}^{\phi,s} - xy_{21}^{\phi,s})(1 - \alpha^\phi) - \rho(xy_{42}^{\phi,s} - xy_{24}^{\phi,s})/2
\end{aligned} \tag{S.1f}$$

$$\begin{aligned}
y_3^{\phi'} = & yy_{33}^{\phi,s} + yy_{13}^{\phi,s}/2 + (yy_{23}^{\phi,s} + yy_{34}^{\phi,s})\alpha^\phi \\
& - R(yy_{23}^{\phi,s} - yy_{14}^{\phi,s})\alpha^\phi \\
& (xy_{33}^{\phi,s} + xy_{13}^{\phi,s})/2 + (xy_{23}^{\phi,s} + xy_{43}^{\phi,s})\alpha^\phi \\
& - r(xy_{43}^{\phi,s} - xy_{34}^{\phi,s})\alpha^\phi - \rho(xy_{13}^{\phi,s} - xy_{31}^{\phi,s})/2
\end{aligned} \tag{S.1g}$$

$$\begin{aligned}
& + [-(R + r + \rho)xy_{23}^{\phi,s} + (R + \rho - r)xy_{32}^{\phi,s}] \\
& + (R + r - \rho)xy_{14}^{\phi,s} + (R + \rho - r)xy_{41}^{\phi,s}] \alpha^\phi / 2
\end{aligned} \tag{S.1g}$$

$$\begin{aligned}
y_4^{\delta'} = & yy_{44}^{\delta,s} + yy_{34}^{\delta,s}/2 + (yy_{14}^{\delta,s} + yy_{24}^{\delta,s})\alpha^{\delta} \\
& - R(yy_{14}^{\delta,s} - yy_{23}^{\delta,s})\alpha^{\delta} \\
& (xy_{44}^{\delta,s} + xy_{24}^{\delta,s})/2 + (xy_{14}^{\delta,s} + xy_{34}^{\delta,s})(1 - \alpha^{\delta}) \\
& - r(xy_{34}^{\delta,s} - xy_{43}^{\delta,s})(1 - \alpha^{\delta}) - \rho(xy_{24}^{\delta,s} - xy_{42}^{\delta,s})/2 \\
& + [-(R + r + \rho)xy_{14}^{\delta,s} + (R + \rho - r)xy_{41}^{\delta,s} \\
& + (R + r - \rho)xy_{23}^{\delta,s} + (R + \rho - r)xy_{32}^{\delta,s}](1 - \alpha^{\delta})/2
\end{aligned} \tag{S.1h}$$

888

The full system is therefore described by 16 recurrence equations (three diallelic loci in two sexes,  $2^3 \times 2 = 16$ ). However, not all diploid types are produced under certain sex-determination systems. For example, with the  $M$  allele fixed and an ancestral  $XY$  sex determination, there are  $XX$  males,  $XY$  females, or  $YY$  females ( $x_3^{\delta} = x_4^{\delta} = y_4^{\delta} = y_3^{\delta} = y_i^{\delta} = 0$ ). In this case, the system only involves six recursion equations, which we assume below to calculate the equilibria.

## Resident equilibrium and stability

In the resident population (allele  $M$  fixed), we follow the frequency of  $A$  in X-bearing female gametes,  $p_X^{\varphi}$ , and X-bearing male gametes,  $p_X^{\delta}$ , and Y-bearing male gametes,  $p_Y^{\delta}$ . We also track the total frequency of Y among male gametes,  $q$ , which may deviate from 1/2 due to meiotic drive in males. These four variables determine the frequencies of the six resident gamete types:  $x_1^{\varphi} = \hat{p}_X^{\varphi}$ ,  $x_2^{\varphi} = 1 - \hat{p}_X^{\varphi}$ ,  $x_1^{\delta} = (1 - q)\hat{p}_X^{\delta}$ ,  $x_2^{\delta} = (1 - q)(1 - \hat{p}_X^{\delta})$ ,  $y_1^{\delta} = q\hat{p}_Y^{\delta}$ , and  $y_2^{\delta} = q(1 - \hat{p}_Y^{\delta})$ . Mean fitnesses in the resident population are given in table S.2.

Various forms of selection can maintain a polymorphism at the **A** locus, including sexually antagonistic selection, overdominance, conflicts between diploid selection and selection upon haploid genotypes (ploidally antagonistic selection, Immler et al. 2012), or a combination of these selective regimes.

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-

Table S.2: Mean fitnesses and zygotic sex ratio in the resident population ( $M$  fixed, XY sex determination).

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

bility can be calculated analytically without assuming anything about the relative  
910 strengths of selection and recombination. However, here, we focus on two regimes  
911 (tight linkage and weak selection) in order to make fewer assumptions about fit-  
912 nesses.

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

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

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

$$\hat{p}_X^\delta = \frac{w_a^\delta \phi}{w_a^\delta \phi + w_A^\delta \psi}, \quad \hat{p}_X^\delta = \frac{(1 + \alpha_\Delta^\delta) w_{Aa}^\delta \phi}{(1 + \alpha_\Delta^\delta) w_{Aa}^\delta \phi + w_{AA}^\delta \psi}$$

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

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

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

$$(B') \quad \hat{p}_Y^\delta = 1, \quad \hat{p}_X^\delta = 0, \quad \hat{p}_X^\delta = 0, \quad \hat{q} = (1 + \alpha_\Delta^\delta)/2 \quad (\text{S.2d})$$

$$\phi = (1 + \alpha_\Delta^\delta) w_A^\delta w_{Aa}^\delta [w_a^\delta w_{aa}^\delta + (1 + \alpha_\Delta^\delta) w_A^\delta w_{Aa}^\delta] / 2 - w_a^\delta w_a^\delta w_{aa}^\delta w_{aa}^\delta$$

$$\psi = (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta [w_a^\delta w_{aa}^\delta + (1 + \alpha_\Delta^\delta) w_A^\delta w_{Aa}^\delta] / 2 - (1 + \alpha_\Delta^\delta) w_A^\delta w_A^\delta w_{Aa}^\delta w_{AA}^\delta$$

$$\phi' = (1 - \alpha_\Delta^\delta) w_A^\delta w_{AA}^\delta [w_A^\delta w_{AA}^\delta + (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta] / 2 - w_A^\delta w_A^\delta w_{AA}^\delta w_{AA}^\delta$$

$$\psi' = (1 + \alpha_\Delta^\delta) w_A^\delta w_{Aa}^\delta [w_A^\delta w_{AA}^\delta + (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta] / 2 - (1 - \alpha_\Delta^\delta) w_a^\delta w_a^\delta w_{Aa}^\delta w_{aa}^\delta$$

920 A fifth equilibrium (*C*) also exists where *A* is present at an intermediate frequency  
 921 on the Y chromosome ( $0 < \hat{p}_Y^\delta < 1$ ). However, equilibrium (*C*) is never locally  
 922 stable when  $r \approx 0$  and is therefore not considered further. Thus, the Y can either  
 923 be fixed for the *a* allele (equilibria *A* and *B*) or the *A* allele (equilibria *A'* and  
 924 *B'*). The X chromosome can then either be polymorphic (equilibria *A* and *A'*) or fixed for the  
 925 alternative allele (equilibria *B* and *B'*). Since equilibria (*A*) and  
 926 (*B*) are equivalent to equilibria (*A'*) and (*B'*) with the labelling of *A* and *a* alleles  
 927 interchanged, we discuss only equilibria (*A*) and (*B*), in which the Y is fixed for the  
 928 *a* allele. If there is no haploid selection ( $\alpha_\Delta^\delta = 0, w_A^\delta = w_a^\delta = 1$ ), these equilibria  
 929 are equivalent to those found by Lloyd and Webb (1977) and Otto (2014).

930 We next calculate when (*A*) and (*B*) are locally stable for  $r = 0$ . According  
 931 to the ‘small parameter theory’ (Karlin and McGregor 1972*a;b*), these stability

properties are unaffected by small amounts of recombination between the SDR and A locus, although equilibrium frequencies may be slightly altered. For the  $a$  allele to be stably fixed on the Y we need  $\bar{w}_{Y_a}^\delta > \bar{w}_{YA}^\delta$  where  $\bar{w}_{Y_a}^\delta = w_a^\delta [\hat{p}_X^\varphi (1 - \alpha_\Delta^\delta) w_A^\varphi w_{Aa}^\delta + (1 - \hat{p}_X^\varphi) w_a^\varphi w_{aa}^\delta]$  and  $\bar{w}_{YA}^\delta = w_A^\delta [\hat{p}_X^\varphi w_A^\varphi w_{AA}^\delta + (1 - \hat{p}_X^\varphi)(1 + \alpha_\Delta^\delta) w_a^\varphi w_{Aa}^\delta]$ . That is, Y-a haplotypes must have higher fitness than Y-A haplotypes. Substituting in  $\hat{p}_X^\varphi = \hat{p}_X^\delta$  from above, fixation of the  $a$  allele on the Y requires that  $\gamma_i > 0$  where  $\gamma_{(A)} = w_a^\delta [(1 - \alpha_\Delta^\delta) w_{Aa}^\delta \phi + w_{aa}^\delta \psi] - w_A^\delta [w_{AA}^\delta \phi + (1 + \alpha_\Delta^\delta) w_{Aa}^\delta \psi]$  for equilibrium (A) and  $\gamma_{(B)} = (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta - w_A^\delta w_{AA}^\delta$  for equilibrium (B). Stability of a polymorphism on the X chromosome (equilibrium A) further requires that  $\phi > 0$  and  $\psi > 0$ . Fixation of the  $a$  allele on the X (equilibrium B) can be stable only if equilibrium (A) is not and requires  $\psi < 0$  and  $w_A^\varphi w_{AA}^\varphi > (1 - \alpha^\varphi) w_a^\varphi w_{Aa}^\varphi$ .

check last condition and the stability condition below are correct

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

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

$$0 < -[(1 - h^\varphi)s^\varphi + (1 - h^\delta)s^\delta + t^\varphi + t^\delta + \alpha_\Delta^\varphi + \alpha_\Delta^\delta] \quad (\text{S.3})$$

and  $0 < h^\varphi s^\varphi + h^\delta s^\delta + t^\varphi + t^\delta + \alpha_\Delta^\varphi + \alpha_\Delta^\delta.$

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

Given that a polymorphism is maintained at the A locus by weak selection, the frequencies of A in each type of gamete are the same ( $\hat{p}_X^\varphi = \hat{p}_X^\delta = \hat{p}_Y^\varphi = \bar{p}$ ) and given, to leading order, by

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

Differences in frequency between gamete types are of  $O(\epsilon)$ :

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

where  $V_A = \bar{p}(1-\bar{p})$  is the variance in the frequency of  $A$  and  $D^\delta = [\bar{p}s^\delta + (1-\bar{p})h^\delta s^\delta] - [\bar{p}h^\delta s^\delta + (1-\bar{p})]$  corresponds to the difference in fitness between  $A$  and  $a$  alleles in diploids of sex  $\delta \in \{\text{♀}, \text{♂}\}$  ( $\bar{p}$  is the leading-order probability of mating with an  $A$ -bearing gamete from the opposite sex). The frequency of  $Y$  among male 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  $A$  allele in males,  $q = 1/2 + \alpha_\Delta^\delta (\hat{p}_Y^\delta - \hat{p}_X^\delta)/2 + O(\epsilon^3)$ . Without gametic competition or drive ( $\alpha_\Delta^\delta = t^\delta = 0$ ) our results reduce to those of van Doorn and Kirkpatrick (2007).

## 962 Invasion conditions

Cover the other parts of the characteristic polynomial here.

964 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  
966 recursion 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 (females)  
968 and is epistemically dominant to the ancestral sex-determining locus, we need only  
two recursion equations (e.g., tracking the change in the frequency of neo-Y- $A$  and  
970 neo-Y- $a$  gametes from males) and thus the leading eigenvalue is the largest solution  
to a quadratic characteristic polynomial  $\lambda^2 + b\lambda + c = 0$  as described in the text  
972 (Table 2).

For tight linkage between the ancestral sex-determining locus and the selected  
974 locus we can calculate each of these terms exactly, while for weak selection we  
take a Taylor series of the leading eigenvalue. With weak selection, the leading

<sup>976</sup> eigenvalue,  $\lambda$ , for any  $k$ , is given up to order  $\epsilon^2$  by equation (4).

### Tight linkage between A and X (recombination weak relative to selection)

<sup>978</sup> Here, we explore the conditions under which a neo-W invades an XY system as-  
 suming that the A locus is initially in tight linkage with the ancestral sex-determining  
<sup>980</sup> region ( $r \approx 0$ ). We disregard neo-Y mutations, which never spread given that the  
 ancestral population is at a stable equilibrium (see supplementary *Mathematica*  
<sup>982</sup> notebook for proof).

<sup>984</sup> Starting with the simpler equilibrium ( $B$ ), the terms of the characteristic poly-  
 nomial are

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

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

$$\chi_{mA} = \frac{1}{2} [w_a^\delta(1 - \alpha_\Delta^\delta)]^{-1} \frac{w_A^\varphi}{w_A^\varphi} \frac{[w_a^\delta(1 - \alpha_\Delta^\delta)w_{Aa}^\varphi(1 + \alpha_\Delta^\varphi)]}{w_{AA}^\varphi} \frac{R}{2} \quad (\text{S.6c})$$

$$\chi_{ma} = \frac{1}{2} [w_a^\delta(1 - \alpha_\Delta^\delta)]^{-1} \frac{w_A^\varphi}{w_A^\varphi} \frac{[w_A^\delta(1 + \alpha_\Delta^\delta)w_{Aa}^\varphi(1 - \alpha_\Delta^\varphi)]}{w_{AA}^\varphi} \frac{R}{2} \quad (\text{S.6d})$$

Haploid selection impacts the spread of neo-W haplotypes in three ways. Firstly,  
<sup>986</sup> the zygotic sex ratio becomes male biased,  $\zeta > 1/2$ , when the  $a$  allele (which is  
 fixed on the Y) is favoured during competition among male gametes or by mei-  
<sup>988</sup> otic drive in males. Specifically, at equilibrium ( $B$ ), the sex ratio is  $\zeta = w_a^\delta(1 -$   
 $\alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$  where  $2\bar{w}_H^\delta = [w_a^\delta(1 - \alpha_\Delta^\delta) + w_A^\delta(1 + \alpha_\Delta^\delta)]$  has been canceled out in  
<sup>990</sup> equations (S.6) to leave the term  $[w_a^\delta(1 - \alpha_\Delta^\delta)]^{-1}$ . Male biased sex ratios facilitate  
 the spread of a neo-W because neo-W alleles cause the zygotes that carry them to  
<sup>992</sup> develop as the rarer, female, sex.

Secondly, haploid selection in females selects on neo-W haplotypes directly. At

994 equilibrium (*B*), the fitness of female gametes under the ancestral sex-determining  
 995 system is  $w_A^{\varphi}$  such that the relative fitnesses of neo-W-*A* and neo-W-*a* haplotypes  
 996 during female gametic competition are  $w_A^{\varphi}/w_A^{\varphi}$  and  $w_a^{\varphi}/w_A^{\varphi}$  (see terms in equation  
 S.6). Meiotic drive in females will also change the proportion of gametes that carry  
 998 the *A* versus *a* alleles, which will be produced by heterozygous females in propor-  
 999 tions  $(1 + \alpha_{\Delta}^{\varphi})/2$  and  $(1 - \alpha_{\Delta}^{\varphi})/2$ , respectively. These terms are only associated with  
 1000 heterozygous females, i.e., they are found alongside  $w_{Aa}^{\varphi}$ .

Thirdly, haploid selection in males affects the diploid genotypes of females  
 1002 by altering the allele frequencies in the male gametes that female gametes pair  
 1003 with. At equilibrium (*B*), neo-W female gametes will mate with X-*A* male ga-  
 1004 metes with probability  $w_A^{\delta}(1 + \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$  and Y-*a* male gametes with probability  
 1005  $w_a^{\delta}(1 - \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$ , where the  $2\bar{w}_H^{\delta}$  terms have been canceled in equation (S.6)  
 1006 (as mentioned above). Thus, for example, neo-W-*A* haplotypes are found in *AA*  
 1007 female diploids with probability  $w_A^{\delta}(1 + \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$  (first term in square brack-  
 1008 ets in the numerator of equation S.6a) and in *Aa* female diploids with probability  
 1009  $w_a^{\delta}(1 - \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$  (see equation S.6c and second term in square brackets in the  
 1010 numerator of equation S.6a).

The other terms in equations (S.6) are more easily interpreted if we assume that  
 1012 there is no haploid selection in either sex, in which case  $\lambda_{mA} > 1$  when  $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$   
 1013 and  $\lambda_{ma} > 1$  when  $(w_{Aa}^{\varphi} + w_{aa}^{\varphi})/2 > w_{AA}^{\varphi}$ . These conditions cannot be met under  
 1014 purely sexually-antagonistic selection, where *A* is directionally favoured in females  
 1015 ( $w_{AA}^{\varphi} > w_{Aa}^{\varphi} > w_{aa}^{\varphi}$ ) and *a* is directionally favoured in males ( $w_{AA}^{\delta} > w_{Aa}^{\delta} > w_{aa}^{\delta}$ ).  
 1016 Essentially, the X is then already as specialized as possible for the female beneficial  
 1017 allele (*A* is fixed on the X), and the neo-W often makes daughters with the Y-*a*  
 1018 haplotype, increasing the flow of *a* alleles into females, which reduces the fitness  
 of those females.

1020 If selection doesn't uniformly favour *A* in females, however, neo-W-*A* haplo-  
 1021 types and/or neo-W-*a* haplotypes can spread ( $\lambda_{mA} > 1$  and/or  $\lambda_{ma} > 1$ ) at this  
 1022 equilibrium. A neo-W can spread alongside the *A* allele ( $\lambda_{mA} > 1$ ), despite the  
 fact that a neo-W brings Y-*a* haplotypes into females, when  $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$ . In this

1024 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  
 1026 (for example, by overdominance in males, remembering that  $a$  is fixed on the Y)  
 to keep  $A$  fixed on the X. Specifically, from the stability conditions for equilibrium  
 1028 (B), we must have  $w_{Aa}^\delta / [(w_{aa}^\delta + w_{Aa}^\delta)/2] > w_{Aa}^\varphi / w_{AA}^\varphi$ .

Still considering  $w_{Aa}^\varphi > w_{AA}^\varphi$ , the neo-W can also spread alongside the  $a$  allele  
 1030 ( $\lambda_{ma} > 1$ ) if there is sufficiently strong underdominance in females ( $w_{aa}^\varphi > w_{Aa}^\varphi$ )  
 [this is describing directional selection, not overdominance - check the conditions  
 1032 for the case we want to talk about], such that  $(w_{Aa}^\varphi + w_{aa}^\varphi)/2 > w_{AA}^\varphi$ . In this case,  
 a is not favored in females near the equilibrium where females are AA (comparing  
 1034 Aa to AA genotypes) and yet the neo-W can spread with  $a$  because it produces  
 female aa individuals by capturing Y-a haplotypes.

1036 Similar equations can be derived for equilibrium (A) by substituting the equi-  
 librium allele frequencies into Table 2

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

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

$$\chi_{mA} = \frac{aR}{b2} \left[ w_{Aa}^\varphi (1 + \alpha_\Delta^\varphi) w_a^\delta \frac{c}{d} \right] / w_a^\varphi \quad (\text{S.7c})$$

$$\chi_{ma} = \frac{aR}{b2} \left[ w_{Aa}^\varphi (1 - \alpha_\Delta^\varphi) w_{Aa}^\delta w_A^\delta (1 + \alpha_\Delta^\delta) \phi \right] / w_A^\varphi \quad (\text{S.7d})$$

1038 where

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

$$b = w_{AA}^\varphi \phi [w_{Aa}^\delta w_A^\delta (1 + \alpha_\Delta^\delta) \phi] + w_{Aa}^\varphi \psi [w_{Aa}^\delta w_A^\delta (1 + \alpha_\Delta^\delta) \phi + w_{AA}^\delta w_a^\delta \psi] + w_{aa}^\varphi \psi (w_{AA}^\delta w_a^\delta \psi) \quad (\text{S.8b})$$

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

$$d = w_{Aa}^\delta (1 + \alpha_\Delta^\delta) \phi + w_{aa}^\delta \psi \quad (\text{S.8d})$$

As with equilibrium (B), haploid selection again modifies invasion fitnesses  
 1040 by altering the sex-ratio and the diploid genotypes of females and directly selecting upon female gametes. The only difference is that resident XX females are no  
 1042 longer always homozygote  $AA$  and males are no longer always heterozygote  $Aa$ . Thus the effect of haploid selection in males is reduced, as is the difference in fit-  
 1044 ness between neo-W haplotypes and resident X haplotypes, as both can be on any diploid or haploid background.

1046 The other terms are easier to interpret in the absence of haploid selection. For instance, without haploid selection, the neo-W- $A$  haplotype spreads ( $\lambda_{mA} > 1$ ) if  
 1048 and only if

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

where  $\phi - \psi = w_{AA}^{\varphi}w_{Aa}^{\delta} - w_{aa}^{\varphi}w_{aa}^{\delta}$  and both  $\phi$  and  $\psi$  are positive when equilibrium  
 1050 (A) is stable. In contrast to equilibrium (B), a neo-W haplotype can spread under purely sexually-antagonistic selection ( $w_{aa}^{\varphi} < w_{Aa}^{\varphi} < w_{AA}^{\varphi}$  and  $w_{AA}^{\delta} < w_{Aa}^{\delta} < w_{aa}^{\delta}$ ).  
 1052 The neo-W- $A$  can spread as long as it becomes associated with females that bear more  $A$  alleles than observed at equilibrium (A).

1054 Without haploid selection, the neo-W- $a$  haplotype spreads ( $\lambda_{ma} > 1$ ) if and only if

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

1056 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  
 1058 circumstances. For example, with overdominance in males there is selection for increased  $A$  frequencies on X chromosomes in males, which are always paired  
 1060 with Y- $a$  haplotypes. Directional selection for  $a$  in females can then maintain a polymorphism at the A locus on the X. This scenario selects for a modifier that  
 1062 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  
 1064 more females bearing more *a* alleles than the ancestral X chromosome does.

## Supplementary Figures

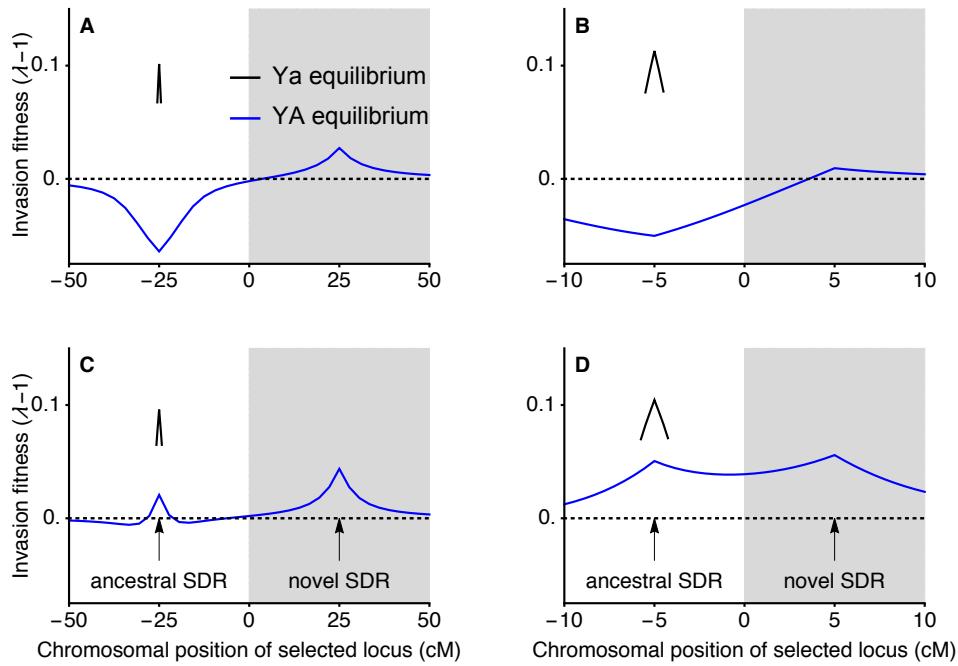


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ( $r \approx 0$ ). In panels A and B, the *a* allele is favoured in females ( $w_{aa}^{\varphi} = 1.05$ ,  $w_{Aa}^{\varphi} = 1$ ,  $w_{AA}^{\varphi} = 0.85$ ) and selection in males is overdominant ( $w_{aa}^{\delta} = w_{AA}^{\delta} = 0.75$ ). In panels C and D, selection in males and females is overdominant ( $w_{aa}^{\varphi} = w_{AA}^{\varphi} = 0.6$ ,  $w_{aa}^{\delta} = 0.5$ ,  $w_{AA}^{\delta} = 0.7$ ,  $w_{Aa}^{\delta} = 1$ ). These parameters are marked by a dagger in Figure 2, which shows that neo-W invasion is expected for any  $R$  when the *a* allele is nearly fixed on the Y (black lines). Equilibria where the *A* allele is more common among Y-bearing male gametes can also be stable for these parameters (blue lines). The weak selection approximation holds when all recombination rates are large relative to selection (around 0 in panels A and C), in which case neo-W alleles should spread if they are more tightly linked to the selected locus (positive invasion fitness in the grey region). However, when linkage is tight (panels C and D and when the selected locus is near the SDR), this prediction breaks down. Here, there is no haploid selection  $t^{\varphi} = \alpha_{\Delta}^{\varphi} = 0$ .

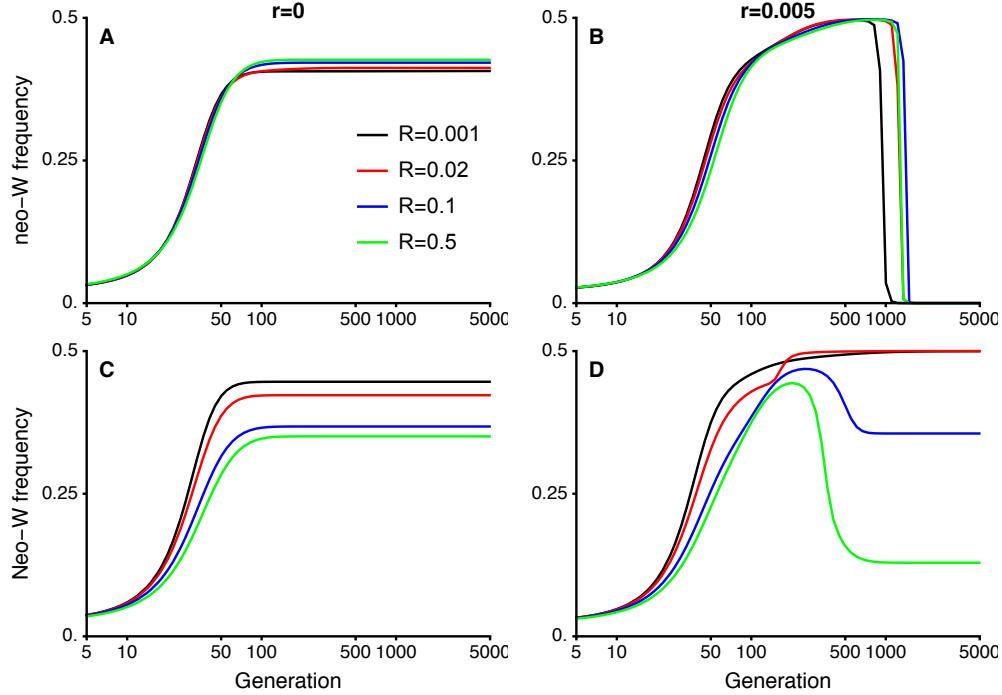


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among female gametes; as the neo-W reaches frequency 0.5, polymorphism at the ancestral XY locus is lost with Y becoming fixed such that sex is determined only by the ZW allele carried by a female gamete. Panels A, C and D show cases where a steady state is reached with the neo-W at a frequency below 0.5, in which case ancestral-X and Y alleles also both segregate. In all cases, we assume that the  $a$  allele is initially more common than the  $A$  allele on the Y (Y- $a$  is fixed when  $r = 0$ ). When  $r > 0$  (panels B and D), Y- $A$  haplotypes created by recombination can become more common than Y- $a$  haplotypes as the neo-W spreads. In B, this leads to loss of the neo-W and the system goes to an equilibrium with X- $a$  and Y- $A$  haplotypes fixed (A'), such that all females have the high fitness genotype  $aa$  and all males  $Aa$ . For the parameters in B, neo-W alleles have negative invasion fitness when the Y- $A$  haplotype is ancestrally more common than Y- $a$  (see blue line in Figure S.2A and S.2B). In contrast, the neo-W is not lost in panel D (see blue line in Figure S.2C and S.2D). Fitness parameters are the same as in Figure S.2, the  $a$  allele is favoured in females ( $w_{aa}^o = 1.05$ ,  $w_{Aa}^\delta = 1$ ,  $w_{AA}^\delta = 0.85$ ) and there is overdominant selection in males ( $w_{aa}^\delta = w_{AA}^\delta = 0.75$ ) in panels A and B. In panels C and D, selection in males and females is overdominant ( $w_{aa}^o = w_{AA}^o = 0.6$ ,  $w_{aa}^\delta = 0.5$ ,  $w_{AA}^\delta = 0.7$ ,  $w_{Aa}^\delta = 1$ ). These parameters are marked by a dagger in Figure 2. Here, there is no haploid selection  $t^\delta = \alpha_\Delta^\delta = 0$ .

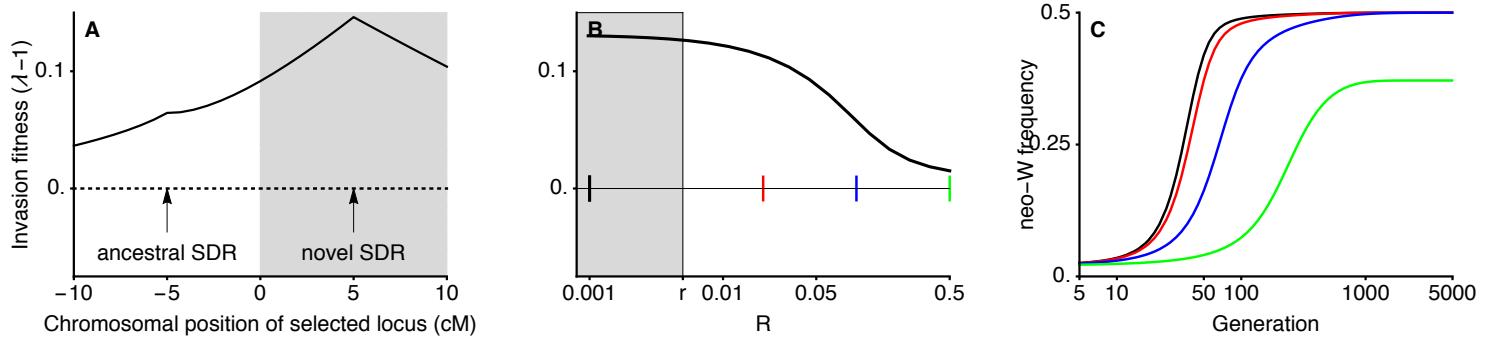


Figure S.3: When there is haploid selection and sexually-antagonistic selection, a neo-W may invade for any  $R$ . Check that we mention Sally's result that invasion cannot occur with sexually-antagonistic selection and  $R = 1/2$  (currently only in legend for figure 1) Add asterisk to Figure S.5B to show parameters used in this plot.  $w_{aa}^{\varphi} = 1.05$ ,  $w_{Aa}^{\delta} = 1$ ,  $w_{AA}^{\varphi} = 0.85$ ,  $w_{aa}^{\delta} = 0.85$ ,  $w_{AA}^{\varphi} = 1.05$ ,  $\alpha_{\Delta}^{\delta} = -0.08$ .

1066        Add Sally's figure showing lambda for small  $r$  near equil A versus near equil  
 1067        B. Add references to this figure to appendix where we discuss whether lambdas  
 1068        can be greater than 1 with sexually antagonistic selection.

1070        Perhaps it would also be useful to add an 8 panel figure that features ploidally  
 1071        antagonistic selection. For each type of haploid selection (gametic competition/  
 1072        meiotic drive in males/females), give a regionplot where  $h^{\delta} = h^{\varphi}$ , e.g.,  $h^{\delta} =$   
 1073         $h^{\varphi} = 0.75$  (or perhaps the value of  $h$  we use in the regionplots we have, in which  
 1074         $w_{aa} = 0.85$ ,  $w_{Aa} = 1$ ,  $w_{AA} = 1.05$ ). Matt made a figure like this before but  
 1075        both Ya and YA equilibria were plotted and there was no outline showing where  
 1076        the Ya equilibrium is stable (as in Figure 2). In Matt's plot the axes were  $s^{\delta}$  and  
 1077         $\alpha_{\Delta}^{\delta}$ . Add an asterisk to each region plot and show invasion in another panel, using  
 1078        those parameters and various  $R$  (e.g., in the style of S.2). In an email, Sally has an  
 1079        example of ploidally-antagonistic selection where the neo-W fixes and  $R = 1/2$ .  
 1080        This would cover that case and more.

1081        We could also give versions of Figure 2 where there is also haploid selection  
 1082        of various types. Haploid selection can favour  $A$  or  $a$ , so this would involve 4x  
 1083        6-panel figures. Started looking at this in Figure S.4 and Figure S.5, add female

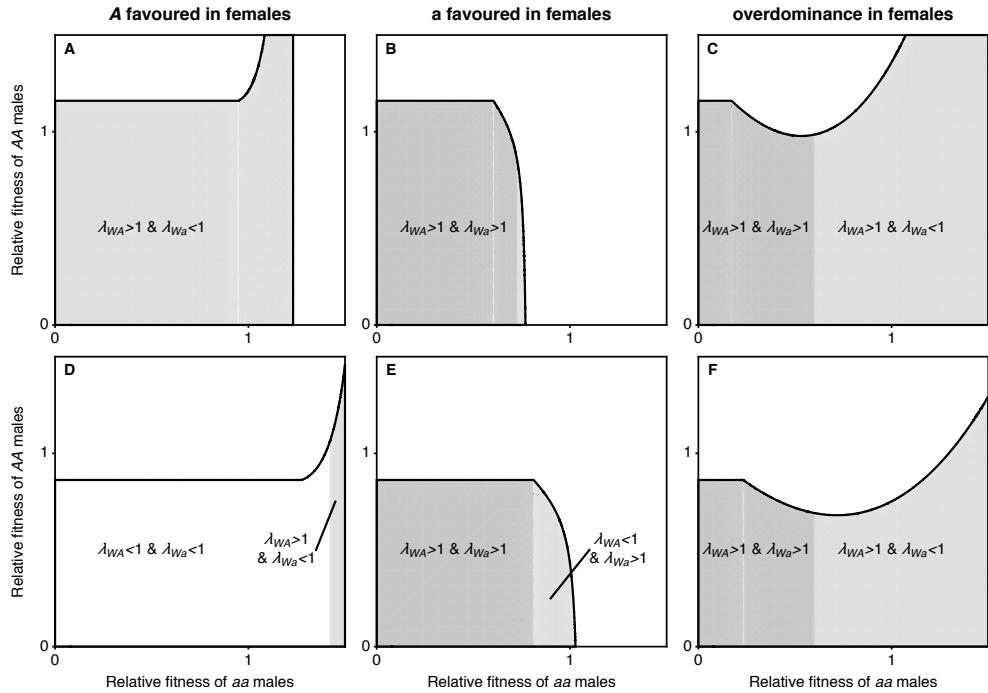


Figure S.4: ABC,  $w_a^\delta = 1.16$ ,  $w_A^\delta = 1$ . DEF,  $w_a^\delta = 1$ ,  $w_A^\delta = 1.16$ . I thought that re-running this with  $w_a^\delta = 1.16$ ,  $w_A^\delta = 0.84$  and  $w_a^\delta = 0.84$ ,  $w_A^\delta = 1.16$  will mean that it matches exactly with the meiotic drive example. Checking for panel A, the result was qualitatively similar (e.g., region where both  $\lambda$ 's are greater than one in panel A, as you might expect from equation (S.6), however the region where a polymorphism is stable is also altered so they don't exactly match.

haploid selection. Try to integrate into the discussion of haploid selection? e.g.,  
 1084 male haploid selection ones generally show effect of sex ratio, increasing both  
 lambdas when female biased (top rows).

1086 Perhaps, for one set of parameters, we should plot the dynamics of all the dif-  
 ferent alleles. E.g., we could use the same parameters used in 4. The main purpose  
 1088 would be to show what happens to the ancestral SDR during turnover. We could  
 also show an example where XY and ZW sex determining systems are both poly-  
 1090 morphic and stable (e.g., using one of the curves in Figure S.2 and the green curve  
 in Figure S.3). I think there are also examples with looser sex linkage and pollen  
 1092 competition that lead to a mixed sex-determination system. We should probably  
 have a short section in the appendix discussing this.

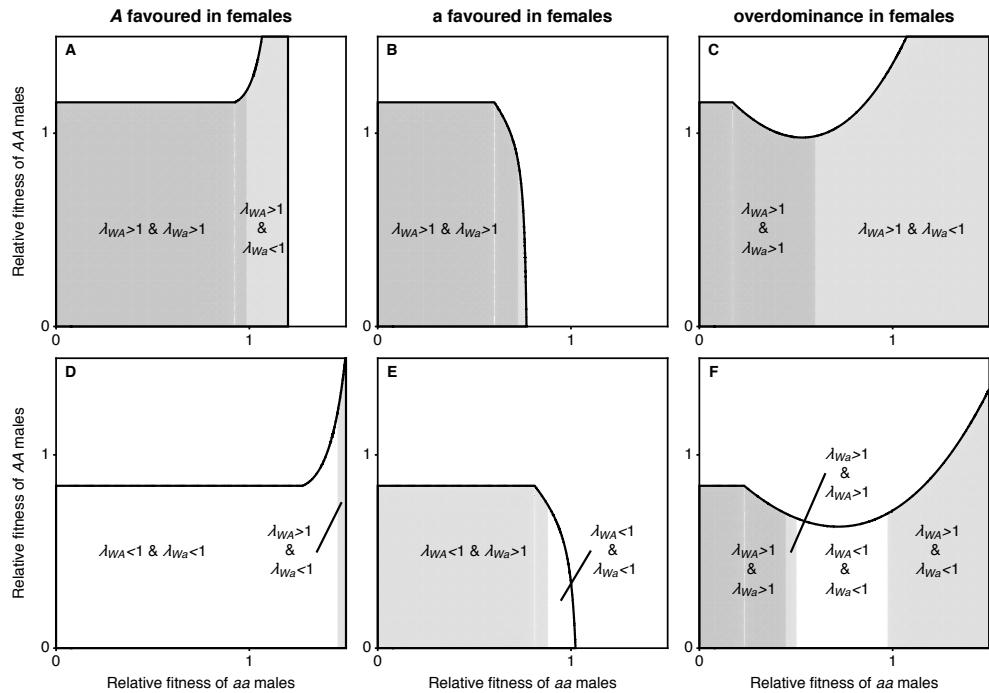


Figure S.5: ABC,  $\alpha_{\Delta}^{\delta} = -0.08$  DEF,  $\alpha_{\Delta}^{\delta} = 0.08$ . Panel F mislabelled, should have  $\lambda_{Wa} > 1$  &  $\lambda_{WA} < 1$  as the upper label that has the line.

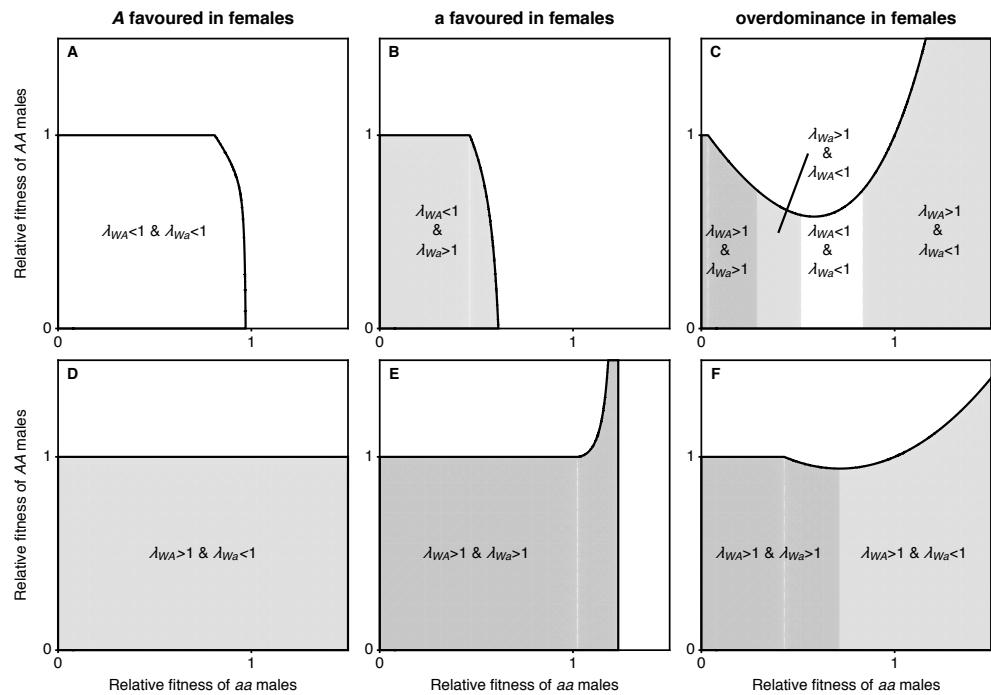


Figure S.6: ABC,  $\alpha_{\Delta}^{\varnothing} = -0.08$  DEF,  $\alpha_{\Delta}^{\varnothing} = 0.08$ .  $\lambda$ s are increased for the haplotype that is favoured by female haploid selection, the stability conditions are also affected.