

# 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 is not predicted  
26 by previous theory. (2) We also consider selection upon haploid genotypes  
28 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 transi-  
tions between male and female heterogamety can occur even if the new  
sex-determining region is less closely linked to the locus under selection.  
Haploid selection in the heterogametic sex can also cause sex ratio biases,  
which may increase or decrease with the spread of new sex chromosomes.  
Thus, transitions between sex-determination systems cannot be simply pre-  
dicted by selection to equalise the sex-ratio. Overall, our models reveal  
that transitions between sex-determination systems, particularly transitions  
where the heterogametic sex changes, can be driven by loci in previously un-  
expected 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 (Gam-  
43 ble et al. 2017), lizards (Ezaz et al. 2009), eight of 26 teleost fish families (Mank  
44 et al. 2006), true fruit flies (Tephritids, Vicoso and Bachtrog 2015), amphibians  
45 (Hillis and Green 1990), the angiosperm genus *Silene* (Slancarova et al. 2013),  
46 the angiosperm family *Salicaceae* (Pucholt et al. 2015; 2017) and Coleoptera and  
47 Hemiptera (Beukeboom and Perrin 2014, plate 2). Indeed, in some cases, both  
48 male and female heterogametic sex-determination systems can be found in the  
49 same species, as exhibited by some cichlid species (Ser et al. 2010) and *Rana*  
50 (*rugosa* (Ogata et al. 2007, Miura 2007)). In addition, multiple transitions have  
51 occurred between genetic and environmental sex-determination systems, e.g., in  
52 reptiles and fishes (Conover and Heins 1987, Mank et al. 2006, Pokorná and Kra-  
53 tochvíl 2009, Ezaz et al. 2009, Pen et al. 2010, Holleley et al. 2015).

54 Predominant theories accounting for the spread of new sex-determination sys-  
55 tems by selection involve fitness differences between sexes (e.g., sexually antag-  
56 onistic selection) or sex-ratio selection. van Doorn and Kirkpatrick (2007; 2010)  
57 show that new sex-determining loci can be favoured if they arise in closer link-  
58 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 determined by the sex-determination system, and it  
68 has 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 Per-  
70 rin 2014, Chapter 7). ‘Fisherian’ sex-ratio selection favours a 1:1 zygotic sex ra-  
tio when assuming that males and females are equally costly to produce (Fisher  
72 1930, Charnov 1982). This follows from the fact that, for an autosomal locus,  
half of the genetic material is inherited from a male and half from a female (West  
74 2009). Thus, 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  
76 sex is 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  
78 of sex-chromosome evolution, Kozielska et al. (2010) consider systems in which  
the ancestral sex chromosomes experience meiotic drive (e.g., where driving X or  
80 Y 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.

Segregation distortion provides putative evidence of haploid selection and can sometimes be attributed to meiotic drive and/or gametic competition (Lalanne et al. 2004, Fishman and Willis 2005, Leppälä et al. 2008; 2013, Didion et al. 2015; 2016). Where it has been characterized, meiotic drive generally occurs either during the production of male or female gametes only (Úbeda and Haig 2005, Lindholm et al. 2016). Gametic competition is also typically sex specific, occurring primarily among male gametes, because there are typically many more pollen/sperm than required for fertilization. Gametic competition may be particularly common in plants, in which 60-70% of all genes are expressed in the male gametophyte and these genes exhibit stronger signatures of selection than random genes (Borg et al. 2009, Arunkumar et al. 2013, Gossman et al. 2014). In addition, artificial selection pressures applied to male gametophytes are known to cause a response to selection (e.g., Hormaza and Herrero 1996, Ravikumar et al. 2003, Hedhly et al. 2004, Clarke et al. 2004). A smaller proportion of genes are thought to be expressed and selected 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 in animals can alter haploid allele frequencies and increase offspring fitness (Immler et al. 2014, Alavioon et al. 2017).

There are various ways by which genes experiencing haploid selection could influence transitions between sex-determination systems. If we assume that haploid selection at any particular locus predominantly occurs in one sex (e.g., meiotic drive during spermatogenesis), then such loci experience a form of sex-specific selection. 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,  
120 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,  
122 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  
124 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  
126 (Lloyd 1974, Conn and Blum 1981, Stehlik and Barrett 2005; 2006, Field et al.  
128 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.

130 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  
132 by selection on genetically-associated alleles. Consequently, Fisherian sex ratio selection does not dominate and it is possible for selection on linked alleles to  
134 drive turnover between sex-determining systems despite causing increases in sex-ratio bias. In addition to considering haploid selection, another novel development  
136 in our model is that we consider loci that are in very tight linkage with the ancestral sex-determining region. We show that transitions between male and female  
138 heterogamety 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  
140 ancestral associations between sex and the alleles favoured in that sex.

## Model

142 We consider transitions between ancestral and novel sex-determining systems using a three-locus model, each locus having two alleles. Locus **X** is the ancestral  
144 sex-determining region, with alleles *X* and *Y* (or *Z* and *W*). Locus **A** is a locus under selection, with alleles *A* and *a*. Locus **M** is a novel sex-determining region,  
146 at which the null allele (*M*) is initially fixed in the population such that sex of

zygotes is determined by the genotype at the ancestral sex-determining region, **X**; 148  $XX$  genotypes become females and  $XY$  become males (or  $ZW$  become females and  $ZZ$  become males). To evaluate the evolution of new sex-determination sys- 149 tems, we consider the invasion, fixation, maintenance, and/or loss of novel sex- 150 determining alleles ( $m$ ) at the **M** locus. We assume that the **M** locus is epistatically 151 dominant over the **X** locus such that zygotes with at least one  $m$  allele develop as 152 females with probability  $k$  and as males with probability  $1 - k$ , regardless of the 153 **X** locus genotype. With  $k = 0$ , the  $m$  allele is a masculinizer (i.e., a neo-Y) and 154 with  $k = 1$  the  $m$  allele is a feminizer (i.e., a neo-W). With intermediate  $k$ , we can 155 interpret  $m$  as an environmental sex determination (ESD) allele, such that zygotes 156 develop as females in a proportion ( $k$ ) of the environments they experience.

157 In each generation, we census the genotype frequencies in male and female 158 gametes/gametophytes (hereafter gametes) before gametic competition. A full de- 159 scription of our model, including recursion equations, is given in the Appendix. 160 First, competition occurs among male gametes (sperm/pollen competition) and 161 among female gametes (egg/ovule competition) separately. Selection during ga- 162 metic competition depends on the **A** locus genotype, relative fitnesses are given 163 by  $w_A^\varphi$  and  $w_a^\varphi$  ( $\varphi \in \{\text{♀}, \text{♂}\}$ ; see table 1). We assume that all gametes compete for 164 fertilization during gametic competition, which assumes a polygamous mating sys- 165 tem. Gametic competition in monogamous mating systems is, however, equivalent 166 to meiotic drive in our model (described below), as both only alter the frequency 167 of gametes produced by heterozygotes. After gametic competition, random mating 168 occurs between male and female gametes. The resulting zygotes develop as males 169 or females, depending on their genotypes at the **X** and **M** loci. Diploid males and 170 females then experience selection, with relative fitnesses  $w_{AA}^\varphi$ ,  $w_{Aa}^\varphi$ , and  $w_{aa}^\varphi$ . The 171 next generation of gametes is produced by meiosis, during which recombination 172 and sex-specific meiotic drive can occur. Recombination (i.e., an odd number of 173 cross-overs) occurs between loci **X** and **A** with probability  $r$ , between loci **A** and 174 **M** with probability  $R$ , and between loci **X** and **M** with probability  $\rho$ . Any linear 175 order of the loci can be modelled with appropriate choices of  $r$ ,  $R$ , and  $\rho$  (see Ta-

ble S.1). Individuals that are heterozygous at the **A** locus may experience meiotic drive; a gamete produced by  $Aa$  heterozygotes of sex  $\text{♀}$  bear allele  $A$  with probability  $\alpha^{\text{♀}}$ . Thus, the **A** locus can experience sex-specific gametic competition, diploid selection, and/or meiotic drive.

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

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

## Results

The model outlined above describes both ancestrally-XY and ancestrally-ZW sex-determination systems if we relabel the two sexes as being ancestrally ‘heterogametic’ or ancestrally ‘homogametic’. Without loss of generality, we primarily refer to the ancestrally heterogametic sex as male and the ancestrally homogametic sex as female. That is, we describe an ancestral XY sex-determination system but our model is equally applicable to an ancestral ZW sex-determination system (relabeling the ancestrally-heterogametic sex as female and the ancestrally-homogametic sex as male).

190 **Generic invasion by a neo-Y or neo-W**

The evolution of a new sex-determination system requires that a rare mutant allele at the novel sex-determining locus,  $m$ , increases in frequency when rare. The spread of a rare mutant  $m$  at the **M** locus is determined by the leading eigenvalue,  $\lambda$ , of the system of eight equations describing the frequency of eggs and sperm carrying the  $m$  allele in the next generation (equations S.1). This system simplifies substantially in a number of cases of interest. Dominant neo-Y (when  $k = 0$ ) or neo-W alleles (when  $k = 1$ ) are only found in male diploids (neo-Y) or female diploids (neo-W) such that their growth rate ultimately depends only on the change in frequency of  $m$ -bearing gametes produced by males or by females, respectively. Furthermore, if the  $m$  allele is fully epistatically dominant over the ancestral sex-determining system, phenotypes are not affected by the genotype at the ancestral sex-determining region (**X** locus). Thus, the invasion of rare dominant neo-Y or neo-W alleles is determined by the largest eigenvalue that solves a quadratic characteristic polynomial,  $\lambda^2 + b\lambda + c = 0$  (see Appendix for a discussion of other roots - or Sally's proof!). Here,  $b = -(\lambda_{mA} + \lambda_{ma}) + (\chi_{mA} + \chi_{ma})$  and  $c = (\lambda_{mA} - \chi_{mA})(\lambda_{ma} - \chi_{ma}) - \chi_{mA}\chi_{ma}$ , where  $\lambda_{mi}$  is the multiplicative growth rate of mutant haplotypes on background  $i \in \{A, a\}$ , without accounting for loss due 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 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 ancestral 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,  $p_Y^{\delta}$ , 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 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. Given the characteristic polynomial  $f(\lambda) = \lambda^2 + b\lambda + c$  and the Perron-Forbenius theorem (guaranteeing that the leading eigenvalue is positive, unique, and real), at least one solution to  $f(\lambda) = 0$  is greater than one when the polynomial has a negative slope or negative value at  $\lambda = 1$  ( $f'(1) = 2 + b < 0$  or  $f(1) = 1 + b + c < 0$ ). Regardless the rate of recombination, at least one of these conditions is true if both haplotypes can spread ( $\lambda_{mA}, \lambda_{ma} > 1$ ) and neither can be true if neither haplotype can spread ( $\lambda_{mA}, \lambda_{ma} < 1$ ). If only one haplotype can spread then the new sex-determining allele increases in frequency on one A

230 background and declines on the other. Invasion then occurs if

$$\chi_{ma}/(\lambda_{ma} - 1) + \chi_{mA}/(\lambda_{mA} - 1) < 1. \quad (1)$$

232 For example, if we assume that only the *mA* haplotype has a positive growth rate  
234 ( $\lambda_{ma} < 1 < \lambda_{mA}$ ), the first term on the left-hand side of (1) is negative and invasion  
requires that the growth rate of *mA* haplotypes ( $\lambda_{ma} - 1$ ) and the rate at which  
236 they are produced by recombination in *ma* haplotypes ( $\chi_{ma}$ ) are sufficiently large  
relative to the rate of decline of *ma* haplotypes ( $1 - \lambda_{ma}$ ) and the rate of loss of *mA*  
haplotypes due to recombination ( $\chi_{mA}$ ).

238 Table 2 illustrates a number of key points about the invasion of neo-Y and  
240 neo-W mutations. First, Fisherian sex-ratio selection will favour the spread of a  
neo-Y if the ancestral zygotic sex ratio is biased towards females,  $\zeta < 1/2$  (i.e.,  
242 the first factor of the  $\lambda_{mi}$  is greater than one for a neo-Y and less than one for a neo-  
W). However, the spread of a neo-Y (neo-W) also depends on the male (female)  
244 fitness of associated alleles (terms involving equilibrium allele frequencies,  $\hat{p}$ 's).  
246 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  
248 is because a dominant neo-Y (neo-W) is always found in males (females), and  
250 therefore the frequency of the neo-Y (neo-W) allele,  $m$ , only changes in males  
(females). Finally, invasions by a neo-Y and a neo-W are qualitatively different.  
252 This is because a gamete with the ancestral- or neo-Y always pairs with a female  
gamete containing an X, and both develop into males. By contrast, a gamete with  
254 a neo-W can pair with an X or Y male gamete, developing into a female, while  
female gametes without the neo-W can become female (when paired with X) or  
male (when paired with Y). Consequently, the types of females produced differ in  
the frequency of *A* alleles they obtain from mating.

254 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*  
256 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-  
ulation. Since only the **A** locus experiences selection directly, any deterministic

258 evolution requires that there is a polymorphism at the **A** locus. Polymorphisms  
259 can be maintained by mutation-selection balance or transiently present during the  
260 spread of beneficial alleles. However, polymorphisms maintained by selection can  
261 maintain alleles at higher allele frequencies for longer periods. Here, we focus of  
262 polymorphisms maintained by selection, where the *A* allele reaches a stable in-  
263 termediate equilibrium frequency under the ancestral sex-determination system  
264 before the neo-sex-determining allele (*m*) arises. We can analytically calculate the  
265 allele frequency of the *A* allele using two alternative simplifying assumptions: (1)  
266 the **A** locus is within (or tightly linked to) the non-recombining region around the  
267 ancestral SDR ( $r \approx 0$ ) or (2) selection is weak relative to recombination ( $s^{\delta}, t^{\delta}$ ,  
268  $\alpha_{\Delta}^{\delta}$  of order  $\epsilon \ll 1$ ).

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

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

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

males that have higher fitness than the ancestral Y.

288 Neo-W alleles, on the other hand, can invade an ancestral XY system under  
 some conditions (the full invasion conditions are given in the appendix; equations  
 290 S.6 and S.7). That is, selection on loci within the non-recombining region of the  
 SDR can favour the invasion of a less closely linked neo-W (Figure 1). In fact, with  
 292 tight linkage between the ancestral SDR and the selected locus, haploid selection  
 and/or overdominance can favour completely unlinked neo-W alleles ( $R = 1/2$ ),  
 294 allowing autosomes to become new sex chromosomes. To develop an intuition for  
 how less closely linked neo-W alleles invade, we first focus on cases where there  
 296 is no haploid selection and discuss the additional effect of haploid selection in the  
 appendix.

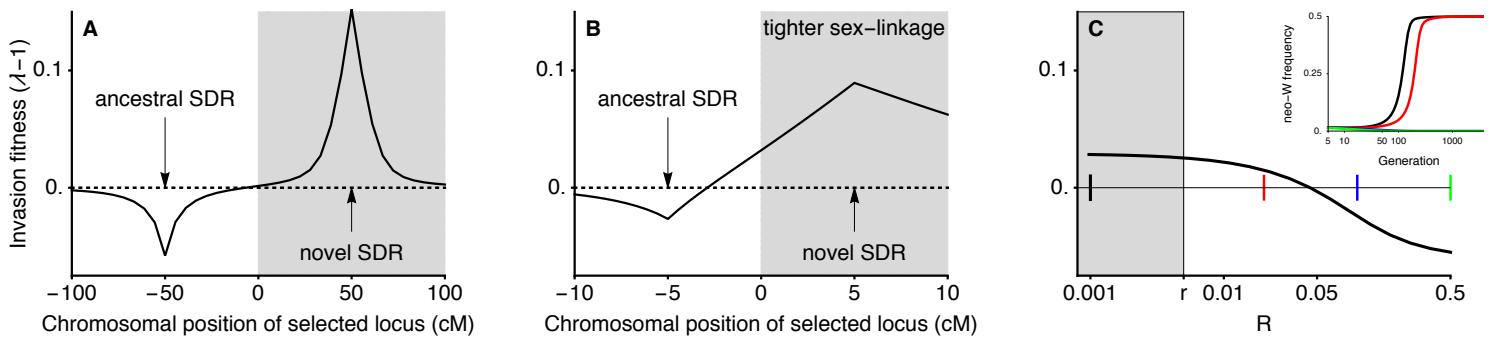


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is less tightly linked to a locus under sexually-antagonistic selection (here, without haploid selection). In panel A, linkage is loose enough relative to selection that the analytical results assuming weak selection hold, 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 analytical results assuming weak selection do not hold, and a neo-W can 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 can show that neo-W invasion fitness is always negative when  $R = 1/2$  and there is sex-antagonism but no haploid selection (see supplementary *Mathematica* file). Fitness parameters are shown by an asterisk in Figure 2A:  $w_{AA}^{\vartheta} = 1.05$ ,  $w_{aa}^{\delta} = 1.2$ ,  $w_{aa}^{\vartheta} = w_{AA}^{\delta} = 0.85$ ,  $w_{Aa}^{\delta} = 1$ ,  $t^{\vartheta} = \alpha_{\Delta}^{\vartheta} = 0$ .

298 If we categorise the  $a$  allele as being ancestrally ‘male-beneficial’ via the fact  
 that it is fixed on the Y, then  $\lambda_{mA} > 1$  indicates that the neo-W spreads when found

300 with the ancestrally ‘female-beneficial’ allele. Broadly, this is possible because  
301 the ancestral X chromosome is sometimes found in males and is therefore unable  
302 to perfectly specialise on the ‘female-beneficial’ allele. For example, when the  $a$   
303 allele is favoured in males, a polymorphism of  $A$  and  $a$  alleles can be maintained  
304 on the X despite directional selection in favour of the  $A$  allele in females ( $s^F > 0$ ,  
305  $0 < h^F < 1$ ). When the  $a$  allele is strongly favoured on X chromosomes in males  
306 ( $w_{aa}$  sufficiently large relative to  $w_{Aa}$ ), neo-W- $A$  haplotypes can spread ( $\lambda_{mA} >$   
307 1), see Figure 2A. In this case the  $a$  allele is at high frequency among ancestral  
308 XX females due to selection upon the X in males. By contrast, W- $A$  haplotypes  
309 will only create females with high fitness ( $AA$  or  $Aa$  genotypes) and can therefore  
310 spread.

When only one neo-W haplotype has a positive growth rate (see Figure 2), a  
312 neo-W can invade as long as equation (1) is satisfied, which may require that the  
313 recombination rate,  $R$ , is small enough. Nevertheless, because we assume here that  
314  $r$  is small, these results indicate that a more loosely linked sex-determining region  
315 ( $r < R$ ) can spread. Therefore, tightly sex-linked loci that experience sexually-  
316 antagonistic selection can drive heterogametic transitions in which the neo-SDR  
317 is less closely linked to the locus under selection (Figure 1).

Given that the  $a$  allele can be considered ancestrally ‘male-beneficial’ because  
318 it is fixed on the Y, it is surprising that neo-W- $a$  haplotypes can sometimes be  
319 favoured by selection in females ( $\lambda_{ma} > 1$ ). Again, this occurs because ancestral  
320 X’s also experience selection in males, in which they will always be paired with a  
321 Y- $a$ . Hence, if there is overdominance in males, X- $A$  Y- $a$  males have high fitness  
322 and the  $A$  allele is favoured by selection on the X in males. Therefore, the X can  
323 be polymorphic or even fixed for the  $A$  allele despite favouring the  $a$  allele during  
324 selection in females (e.g., see outlined region in Figure 2B and Lloyd and Webb  
325 1977, Otto 2014). In such cases, neo-W- $a$  haplotypes can spread because they  
326 create more  $Aa$  and  $aa$  females when pairing with an X from males and because  
327 they bring Y- $a$  haplotypes into females.

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

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

Assuming that linkage is not tight, van Doorn and Kirkpatrick (2010) showed  
338 that invasion by a neo-W occurs under the same conditions as ‘fixation’ (where  
fixation indicates that the neo-W reaches its maximum frequency among eggs,  
340 which is 1/2). An equivalent analysis is not possible where we assume that linkage  
is tight. However, numerical simulations with tight linkage demonstrate that the  
342 neo-SDR does not necessarily fix, leading to the stable maintenance of a mixed  
sex-determining system, in which X, Y, Z, and W alleles all segregate (e.g., Figure  
344 S.9B-D). Within a species, both feminizing and masculinizing alleles have been  
reported in houseflies (McDonald et al. 1978), midges (Thompson 1971), frogs  
346 (Ogata et al. 2007), cichlid fish (Ser et al. 2010), tilapia (Lee et al. 2004), sea bass  
(Vandepitte et al. 2007), and lab-strains of Zebrafish (Liew et al. 2012, Wilson  
348 et al. 2014). For example, in the platyfish (*Xiphophorus maculatus*), X, Y, and  
W alleles segregate at one locus (or two closely-linked loci) near to potentially  
350 sexually-antagonistic genes for pigmentation and sexual maturity (Kallman 1965;  
1968, Wolff and Schartl 2001, Schulteis et al. 2006). Our results suggest that several  
352 forms of selection on nearby loci (i.e.,  $r$  and  $R$  small) could maintain multiple sex-  
determining alleles.

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

Assuming that selection is weak relative to all recombination rates ( $r$ ,  $R$  and  $\rho$ ),  
356 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.  
358 To leading order in selection, these are:

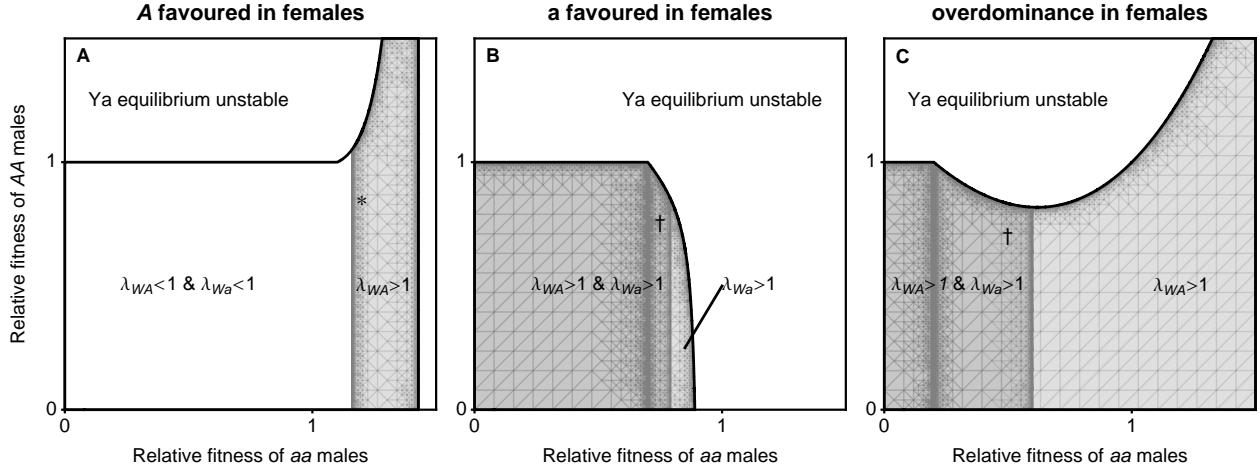


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ( $r = 0$ ), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ( $w_{Aa}^{\varphi} = 1$ ) and only consider stable equilibria at which both  $A$  locus alleles are maintained and the  $a$  allele is initially fixed on the Y, region outlined. Here, selection in females can favour the  $A$  allele (panel A,  $w_{aa}^{\varphi} = 0.85$ ,  $w_{AA}^{\varphi} = 1.05$ ), favour the  $a$  allele (panel B,  $w_{aa}^{\varphi} = 1.05$ ,  $w_{AA}^{\varphi} = 0.85$ ), or be overdominant (panel C,  $w_{aa}^{\varphi} = w_{AA}^{\varphi} = 0.6$ ). 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  $t^{\varphi} = \alpha_{\Delta}^{\varphi} = 0$ .

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

and

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

360 where  $V_A = \bar{p}(1 - \bar{p})$  is the variance in the equilibrium frequency of  $A$  and  $S_A =$   
 361  $(D^{\delta} + \alpha_{\Delta}^{\delta} + t^{\delta}) - (D^{\varphi} + \alpha_{\Delta}^{\varphi} + t^{\varphi})$  describes sex differences in selection for the  $A$   
 362 versus  $a$  across diploid selection, meiosis, and gametic competition. The diploid  
 363 selection term,  $D^{\varphi} = [\bar{p}s^{\varphi} + (1 - \bar{p})h^{\varphi}s^{\varphi}] - [\bar{p}h^{\varphi}s^{\varphi} + (1 - \bar{p})]$ , is the difference  
 364 in fitness between  $A$  and  $a$  alleles in diploids of sex  $\varphi \in \{\varphi, \delta\}$ , where  $\bar{p}$  is the

leading-order probability of mating with an  $A$ -bearing gamete from the opposite sex (equation S.4). The difference in  $A$ -allele-frequency among Y-bearing sperm versus X-bearing sperm is given by  $\hat{p}_Y^\delta - \hat{p}_X^\delta = V_A(D^\delta - D^q + \alpha_\Delta^\delta - \alpha_\Delta^q + t^\delta - t^q)(1 - 2r)/2r$ .

The neo-sex-determining allele,  $m$ , will spread if  $\lambda_{m,XY} > 1$ . Equation (2) demonstrates that, under weak selection, a neo-Y will invade an XY system if and only if it is more closely linked to the selected locus than the ancestral sex-determining region (i.e., if  $R < r$ ; note that  $V_A S_A^2$  is strictly positive as long as  $A$  is polymorphic). This echoes our tight linkage results above where a neo-Y could never invade if  $r \approx 0$ . It is also consistent with the results of van Doorn and Kirkpatrick (2007), who considered diploid selection only and also found that homogametic transitions (XY to XY or ZW to ZW) can only occur when the neo-sex-determining locus is more closely linked to a locus under sexually-antagonistic selection.

With weak selection and no haploid selection ( $t^q = \alpha_\Delta^q = 0$ ), the spread of a neo-W is equivalent to the spread of a neo-Y ( $\lambda_{W',XY} = \lambda_{Y',XY}$ ), such that heterogametic transitions (XY to ZW or ZW to XY) can also occur only if the neo-sex-determining region is more closely linked to a locus under selection ( $R < r$ ), as found by van Doorn and Kirkpatrick (2010). With haploid selection, however, the additional term in equation (3) can be positive, which can allow, for example, neo-W invasion ( $\lambda_{W',XY} > 1$ ) even when the neo-sex-determining region is less closely linked to the selected locus ( $R > r$ ).

Equation (3) shows that, with weak selection, neo-W alleles can invade an XY 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 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 no ancestral association between  $A$  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) therefore disappears and invasion depends only on the sign of  $(r - R)$ . Indeed, invasion typically occurs when the neo-W is

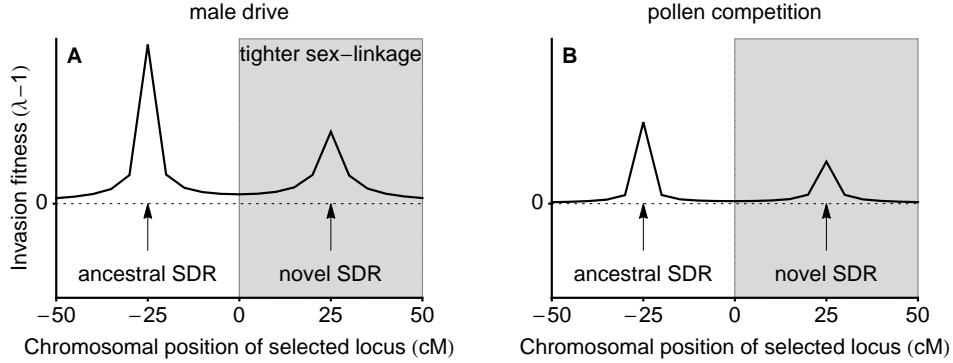


Figure 3: Ploidally-antagonistic selection allows a less tightly linked neo-W to invade. In panel A, male drive ( $\alpha_{\Delta}^{\delta} = -1/20$ ,  $t^{\delta} = \alpha_{\Delta}^{\delta} = 0$ ) opposes selection in diploids (no sex-differences:  $s^{\delta} = 1/10$ ,  $h^{\delta} = 7/10$ ), in which case the neo-sex-determining allele can invade regardless of linkage. In panel B, gametic competition in males ( $t^{\delta} = -1/10$ ,  $t^{\varphi} = \alpha_{\Delta}^{\varphi} = 0$ ) opposes selection in diploids (sex-differences:  $s^{\delta} = 1/20$ ,  $s^{\varphi} = 3/20$ ,  $h^{\delta} = 7/10$ ), 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).

more closely linked to the selected locus than the ancestral sex-determining region  
 396 (Figure 3).

Secondly, we can simplify the discussion of cases where invasion occurs despite looser sex-linkage,  $R > r$ , by focusing on the 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 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 dominance coefficients are equal in the two sexes,  $h^{\varphi} = h^{\delta}$ . When there is no gametic competition and meiotic drive is in one sex only, an unlinked neo-W can invade as long as the same allele is favoured during diploid selection in males and females ( $s^{\varphi}s^{\delta} > 0$ , see Figure 3A and Figure 4B). When there is no meiotic drive 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 are sex differences in selection of one type (e.g.,  $s^{\varphi}(s^{\delta} - s^{\varphi}) > 0$ , see Figure 3B). These special cases indicate that 410 neo-W invasion occurs for a relatively large 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	$h^\delta = h^q, t^\delta = \alpha_\Delta^q = \alpha_\Delta^\delta = 0$	$s^\delta(s^q - s^\delta) > 0$

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

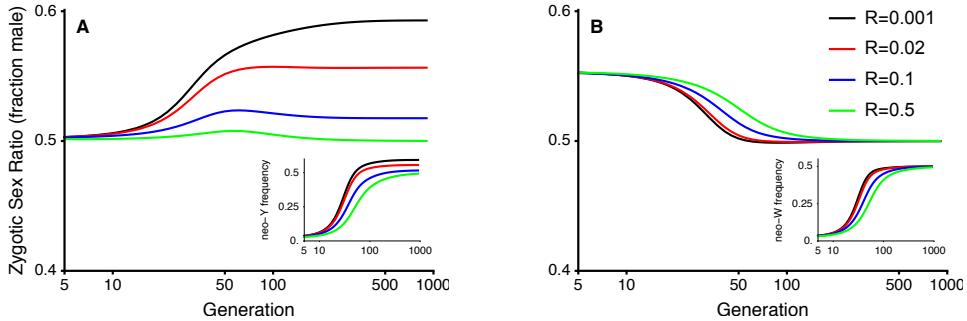


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidally antagonistic with haploid selection favouring the  $a$  allele during male meiosis. In panel A, male drive in an ancestral ZW system has no effect 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:  $s^{\varphi} = s^{\delta} = 0.2$ ,  $h^{\varphi} = h^{\delta} = 0.7$ ,  $t^{\varphi} = t^{\delta} = \alpha_{\Delta}^{\varphi} = 0$ ,  $\alpha_{\Delta}^{\delta} = -0.1$ ,  $r = 0.02$ .

The green curves in Figure 4 show transitions between male and female heterogamety even though the new sex-determining region is unlinked to a locus that experiences haploid and diploid selection. We 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 because the zygotic sex ratio is ancestrally male biased. In Figure 4A, an unlinked neo-Y spreads despite the fact that the ancestral zygotic sex ratio is even. In this case, 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 males more often than the W ( $\hat{p}_W^{\varphi} - \hat{p}_Z^{\varphi} > 0$ , equation S.5). Polymorphism at the  $A$  locus is maintained by counter-selection against the  $a$  allele in diploids and therefore ancestral-ZZ males have generally low diploid fitness. The neo-Y spreads because it produces males with high diploid fitness through matings with ancestral-W-bearing female gametes, which are more likely to carry the  $A$  allele. A freely recombining neo-Y ( $R = 1/2$ ) is equally likely to be segregate with the  $A$  or  $a$  allele and is therefore unaffected by male meiotic drive. Thus, a key factor in explaining why heterogametic transitions can

occur when  $R > r$  is that the neo-SDR determines sex in the diploid phase but  
 450 recombination occurs before any subsequent haploid selection.

### Environmental sex determination

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

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

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

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

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

spread of which is given by

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

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

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

<sup>492</sup> **Discussion**

Two predominant theories explaining the remarkably high frequency of transitions between sex-determination systems are sexually-antagonistic selection and sex-ratio selection (reviewed in Blaser et al. 2012, van Doorn 2014). The former predicts that neo-sex-determining alleles can invade when they arise in closer linkage with a sexually-antagonistic locus (van Doorn and Kirkpatrick 2007; 2010). The latter predicts that new sex-determining systems are generally favoured if they result in more equal sex-ratios than the ancestral system. Firstly, we show that selection (including sexually-antagonistic selection) on loci within or near the non-recombinating region of the ancestral sex-determining region can favour heterogametic transitions (XY to ZW or ZW to XY) to new sex-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 selection'), 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 condition favouring homogametic transitions (XY to XY or ZW to ZW). In addition, with haploid selection and weak selection, heterogametic transitions (XY to ZW 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).

Sex-ratio biases caused by haploid selection can facilitate heterogametic transitions between sex-determining systems. For instance, alleles favoured by haploid selection in males often become associated with the Y, which leads to a male-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 examples see Kozielska et al. 2010, Úbeda et al. 2015). However, sex-ratio selection can be overwhelmed by additional selective effects (e.g., when a linked allele is beneficial for male diploids but detrimental for female diploids; Table 3), preventing the neo-W from invading. Indeed, transitions between sex-determining 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 ga-

metes, 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  $|D^\delta - D^0| << |\alpha_\Delta^\delta - \alpha_\Delta^0 + t^\delta - t^0|$  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

552 could drive transitions between dioecy and hermaphroditism, which are frequent  
553 in plants (Käfer et al. 2017, Goldberg et al. 2017).

554 In support of their role in sex chromosome turnover, genes expected to be under  
555 sexually-antagonistic selection (e.g., those causing bright male colouration) have  
556 been found on recently derived sex chromosomes (Lindholm and Breden 2002,  
557 Tripathi et al. 2009, Ser et al. 2010). Our results show that, if loci experiencing  
558 overdominance and/or sexually-antagonistic selection can be identified in close  
559 linkage with the ancestral sex-determining locus (rather than only the novel sex-  
560 determining locus), then they could also be implicated in driving heterogametic  
561 transitions between sex-determination systems. In addition, we show haploid se-  
562 lection on loci around either the ancestral- or the novel-sex-determining regions  
563 could have had a role in driving sex chromosome turnover. A recent transcrip-  
564 tome analysis in *Rumex*, suggests a role for gametic competition in the evolution  
565 of sex-determination systems, showing that Y-linked genes are have higher expres-  
566 sion in haploid pollen than autosomal genes ([check this is accurate](#)). Interestingly,  
567 haploid-expression is also more common on the autosome that is orthologous to the  
568 sex chromosomes in closely related species suggesting that new sex chromosomes  
569 may have been favoured through their association with haploid selected alleles on  
570 these chromosomes ([Sandler et al., 2017, Personal Communication](#)).

571 We assume that sex-determining alleles do not experience direct selection ex-  
572 cept via their associations with sex and selected alleles. However, in some cases,  
573 there may be significant degeneration around the sex-limited allele (Y or W) in the  
574 ancestral sex-determining region because recessive deleterious mutations and/or  
575 deletions accumulate around the Y or W sex-determining regions (Rice 1996,  
576 Charlesworth and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). During  
577 heterogametic transitions (XY to ZW or ZW to XY), but not homogametic transi-  
578 tions (XY to XY or ZW to ZW), any recessive deleterious alleles linked to the Y or  
579 W are revealed to selection in YY or WW individuals (Bachtrog et al. 2014). This  
580 phenomenon was studied by van Doorn and Kirkpatrick (2010), who found that  
581 degeneration can prevent fixation of a neo-W or a neo-Y allele, leading to a mixed

582 sex-determination system where the ancestral and new sex-determining loci are  
583 both segregating. However, they noted that very rare recombination events around  
584 the ancestral sex-determining region can allow these heterogametic transitions to  
585 complete. Degeneration around the Y or W could explain why heterogametic transi-  
586 tions are not observed to be much more common than homogametic transitions  
587 despite the fact that our models demonstrate that they are favoured under a wider  
588 range of conditions. For example, Vicoso and Bachtrog (2015) found a dozen  
589 sex chromosome configurations among Dipteran species but only one transition  
590 between male and female heterogametey.

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

We have shown that tight sex-linkage and haploid selection can drive previ-  
608 ously unexpected transitions between sex-determination systems. In particular,  
609 both can select for neo-sex-determining loci that are more loosely linked. In ad-  
610 dition, haploid selection alone can cause transitions analogous to those caused by

612 purely sexually-antagonistic selection, eliminating the need for differences in se-  
613 lection between male and female diploids. Perhaps counterintuitively, transitions  
614 involving haploid selection can be driven by sex-ratio selection or cause sex-ratio  
615 biases to evolve. We conclude that haploid selection should be considered as a  
616 pivotal factor driving transitions between sex-determination systems. Overall, our  
617 results suggest several new scenarios under which new sex-determination systems  
618 are favoured, which could help to explain why the evolution of sex-determination  
systems is so dynamic.

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# Appendix

## 876 Recursion Equations

In each generation we census the genotype frequencies in male and female ga-  
878 metes/gametophytes (hereafter, gametes) between meiosis (and any meiotic drive)  
and gametic competition. At this stage we denote the frequencies of X- and Y-  
880 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  
882 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  
884 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  
886 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  
888 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.

890 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  
892 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  
894 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  
896 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  $yy_{ij} = (y_i^{\varphi,s} y_j^{\delta,s} + y_j^{\varphi,s} y_i^{\delta,s})/2$ .

898 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  
900  $k_{bc}$ . 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  $yy_{ij}^\Omega = k_{bc} yy_{ij}$ .  
902 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

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

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

916 Finally, these diploids undergo meiosis to produce the next generation of ga-  
 917 metes. Recombination and sex-specific meiotic drive occur during meiosis. Here,  
 918 we allow any relative locations for the SDR, **A**, and **M** loci by using three param-  
 919 eters to describe the recombination rates between them.  $R$  is the recombination  
 920 rate between the **A** locus and the **M** locus,  $\rho$  is the recombination rate between  
 921 the **M** locus and the **X** locus, and  $r$  is the recombination rate between the **A** locus  
 922 and the **X** locus. Table S.1 shows replacements that can be made for each possi-  
 923 ble ordering of the loci assuming that there is no cross-over interference. During  
 924 meiosis in sex  $\delta$ , meiotic drive occurs such that, in  $Aa$  heterozygotes, a fraction  
 925  $\alpha^\delta$  of gametes produced carry the  $A$  allele and  $(1 - \alpha^\delta)$  carry the  $a$  allele.

Table S.1: Substitutions for different loci orders assuming no interference.

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

926 Among gametes from sex  $\delta$ , the frequencies of haplotypes (before gametic

competition) in the next generation are given by

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

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

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

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

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

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

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

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

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

928

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, Immel 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  
950 strengths of selection and recombination. However, here, we focus on two regimes  
(tight linkage and weak selection) in order to make fewer assumptions about fit-  
952 nesses.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

972 erties are unaffected by small amounts of recombination between the SDR and **A**  
 973 locus, although equilibrium frequencies may be slightly altered. For the *a* allele to  
 974 be stably fixed on the Y we need  $\bar{w}_{Y_a}^\delta > \bar{w}_{Y_A}^\delta$  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  $\bar{w}_{Y_A}^\delta = w_A^\delta [\hat{p}_X^\varphi w_A^\varphi w_{AA}^\delta + (1 - \hat{p}_X^\varphi)(1 + \alpha_\Delta^\delta) w_a^\varphi w_{Aa}^\delta]$ . That is,  
 975 Y-*a* haplotypes must have higher fitness than Y-*A* haplotypes. Substituting in  $\hat{p}_X^\varphi$   
 976 from equation (S.2), fixation of the *a* allele on the Y requires that  $\gamma_i > 0$  where  
 977  $\gamma_{(A)} = w_a^\delta [(1 - \alpha_\Delta^\delta) w_{Aa}^\delta \phi + w_{aa}^\delta \psi] - w_A^\delta [w_{AA}^\delta \phi + (1 + \alpha_\Delta^\delta) w_{Aa}^\delta \psi]$  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 poly-  
 978 980 morphism 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  
 982 equilibrium  $(A)$  is not, as it requires  $\psi < 0$  and  $2w_A^\varphi w_{AA}^\varphi > (1 - \alpha_\Delta^\varphi) w_a^\varphi w_{Aa}^\varphi$  or just  
 $4w_A^\varphi w_{AA}^\varphi < (1 - \alpha_\Delta^\varphi) w_a^\varphi w_{Aa}^\varphi$  (which also prevents  $\psi > 0$ ).

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

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

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

988 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  
 989 frequencies of *A* in each type of gamete are the same ( $\hat{p}_X^\varphi = \hat{p}_X^\delta = \hat{p}_Y^\delta = \bar{p}$ ) and  
 990 given, to leading order, by

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

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

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

where  $V_A = \bar{p}(1 - \bar{p})$  is the variance in the frequency of  $A$  and  $D^\varnothing = [\bar{p}s^\varnothing + (1 - \bar{p})h^\varnothing s^\varnothing] - [\bar{p}h^\varnothing s^\varnothing + (1 - \bar{p})]$  corresponds to the difference in fitness between  $A$  and  $a$  alleles in diploids of sex  $\varnothing \in \{\varnothing, \delta\}$  ( $\bar{p}$  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 ( $\alpha_\Delta^{\varnothing} = t^{\varnothing} = 0$ ) our results reduce to those of van Doorn and Kirkpatrick (2007).

## 1002 Invasion conditions

Cover the other parts of the characteristic polynomial here. Waiting for Sally's proof!

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

For tight linkage between the ancestral sex-determining locus and the selected locus we can calculate each of these terms exactly, while for weak selection we

1016 take a Taylor series of the leading eigenvalue. With weak selection, the leading eigenvalue,  $\lambda$ , for any  $k$ , is given up to order  $\epsilon^2$  by equation (4).

1018 **Tight linkage between A and X (recombination weak relative to selection)**

1020 Here, we explore the conditions under which a neo-W invades an XY system assuming that the A locus is initially in tight linkage with the ancestral sex-determining region ( $r \approx 0$ ). We disregard neo-Y mutations, which never spread given that the 1022 ancestral population is at a stable equilibrium (see supplementary *Mathematica* notebook for proof).

1024 Starting with the simpler equilibrium ( $B$ ), the terms of the characteristic polynomial are

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

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

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

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

1026 Haploid selection impacts the spread of neo-W haplotypes in three ways. Firstly, the zygotic sex ratio becomes male biased,  $\zeta > 1/2$ , when the  $a$  allele (which is 1028 fixed on the Y) is favoured during competition among male gametes or by meiotic 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 1030 equations (S.6) to leave the term  $[w_a^\delta(1 - \alpha_\Delta^\delta)]^{-1}$ . Male biased sex ratios facilitate 1032 the spread of a neo-W because neo-W alleles cause the zygotes that carry them to develop as the rarer, female, sex.

1034 Secondly, haploid selection in females selects on neo-W haplotypes directly. At  
 1035 equilibrium ( $B$ ), the fitness of female gametes under the ancestral sex-determining  
 1036 system is  $w_A^{\varphi}$  such that the relative fitnesses of neo-W- $A$  and neo-W- $a$  haplotypes  
 1037 during female gametic competition are  $w_A^{\varphi}/w_A^{\varphi}$  and  $w_a^{\varphi}/w_A^{\varphi}$  (see terms in equation  
 1038 S.6). Meiotic drive in females will also change the proportion of gametes that carry  
 1039 the  $A$  versus  $a$  alleles, which will be produced by heterozygous females in propor-  
 1040 tions  $(1 + \alpha_{\Delta}^{\varphi})/2$  and  $(1 - \alpha_{\Delta}^{\varphi})/2$ , respectively. These terms are only associated with  
 1041 heterozygous females, i.e., they are found alongside  $w_{Aa}^{\varphi}$ .

1042 Thirdly, haploid selection in males affects the diploid genotypes of females  
 1043 by altering the allele frequencies in the male gametes that female gametes pair  
 1044 with. At equilibrium ( $B$ ), neo-W female gametes will mate with X- $A$  male ga-  
 1045 metes with probability  $w_A^{\delta}(1 + \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$  and Y- $a$  male gametes with probability  
 1046  $w_a^{\delta}(1 - \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$ , where the  $2\bar{w}_H^{\delta}$  terms have been canceled in equation (S.6)  
 1047 (as mentioned above). Thus, for example, neo-W- $A$  haplotypes are found in  $AA$   
 1048 female diploids with probability  $w_A^{\delta}(1 + \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$  (first term in square brack-  
 1049 ets in the numerator of equation S.6a) and in  $Aa$  female diploids with probability  
 1050  $w_a^{\delta}(1 - \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$  (see equation S.6c and the second term in square brackets in  
 1051 the numerator of equation S.6a).

1052 The other terms in equations (S.6) are more easily interpreted if we assume that  
 1053 there is no haploid selection in either sex, in which case  $\lambda_{mA} > 1$  when  $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$   
 1054 and  $\lambda_{ma} > 1$  when  $(w_{Aa}^{\varphi} + w_{aa}^{\varphi})/2 > w_{AA}^{\varphi}$ . These conditions cannot be met under  
 1055 purely sexually-antagonistic selection, where  $A$  is directionally favoured in females  
 1056 ( $w_{AA}^{\varphi} > w_{Aa}^{\varphi} > w_{aa}^{\varphi}$ ) and  $a$  is directionally favoured in males ( $w_{AA}^{\delta} > w_{Aa}^{\delta} > w_{aa}^{\delta}$ ).  
 1057 Essentially, the X is then already as specialized as possible for the female beneficial  
 1058 allele ( $A$  is fixed on the X), and the neo-W often makes daughters with the Y- $a$   
 1059 haplotype, increasing the flow of  $a$  alleles into females, which reduces the fitness  
 1060 of those females.

1061 If selection doesn't uniformly favour  $A$  in females, however, neo-W- $A$  haplo-  
 1062 types and/or neo-W- $a$  haplotypes can spread ( $\lambda_{mA} > 1$  and/or  $\lambda_{ma} > 1$ ) at this  
 1063 equilibrium. A neo-W can spread alongside the  $A$  allele ( $\lambda_{mA} > 1$ ), despite the

1064 fact that a neo-W brings Y-*a* haplotypes into females, when  $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$ , as stated  
 above. In this case the *a* allele is favoured by selection in females despite *A* being  
 1066 fixed on the X. For this equilibrium to be stable (i.e., to keep *A* fixed on the X),  
 X-*a* cannot be overly favoured in females and X-*A* must be sufficiently favoured  
 1068 in males (for example, by overdominance in males, remembering that *a* is fixed  
 on the Y). Specifically, from the stability conditions for equilibrium (B), we must  
 1070 have  $w_{Aa}^{\varphi} < 2w_{AA}^{\varphi}$  and  $w_{Aa}^{\delta}/[(w_{aa}^{\delta} + w_{Aa}^{\delta})/2] > w_{Aa}^{\varphi}/w_{AA}^{\varphi}$ .

Still considering  $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$ , the neo-W can also spread alongside the *a* allele  
 1072 ( $\lambda_{ma} > 1$ ) if  $w_{aa}^{\varphi}$  is large enough such that  $(w_{Aa}^{\varphi} + w_{aa}^{\varphi})/2 > w_{AA}^{\varphi}$ . This can occur  
 with overdominance or directional selection for *a* in females (Figure 2B,C). In this  
 1074 case, *a* is favoured in females (comparing *Aa* to *AA* genotypes in females) but *A*  
 is fixed on the X due to selection in males. The neo-W-*a* haplotype can spread  
 1076 because it produces females with higher fitness *Aa* and *aa* genotypes.

Similar equations can be derived for equilibrium (A) by substituting the equi-  
 1078 librium allele frequencies into Table 2

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

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

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

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

where

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

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

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

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

1080 As with equilibrium (B), haploid selection again modifies invasion fitnesses  
 by altering the sex-ratio and the diploid genotypes of females and directly select-  
 1082 ing upon female gametes. The only difference is that resident XX females are no  
 longer always homozygote *AA* and males are no longer always heterozygote *Aa*.  
 1084 Thus the effect of haploid selection in males is reduced, as is the difference in fit-  
 ness between neo-W haplotypes and resident X haplotypes, as both can be on any  
 1086 diploid or haploid background.

1088 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  
 and only if

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

1090 where  $\phi - \psi = w_{AA}^\varphi w_{Aa}^\delta - w_{aa}^\varphi w_{aa}^\delta$  and both  $\phi$  and  $\psi$  are positive when equilibrium  
 (A) is stable. In contrast to equilibrium (B), a neo-W haplotype can spread under  
 1092 purely sexually-antagonistic selection ( $w_{aa}^\varphi < w_{Aa}^\varphi < w_{AA}^\varphi$  and  $w_{AA}^\delta < w_{Aa}^\delta < w_{aa}^\delta$ ).  
 The neo-W-*A* can spread as long as it becomes associated with females that bear  
 1094 more *A* alleles than observed at equilibrium (A).

Without haploid selection, the neo-W-*a* haplotype spreads ( $\lambda_{ma} > 1$ ) if and  
 1096 only if

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

This condition cannot be met with purely sexually antagonistic selection (as both  
1098 terms on the left-hand side would then be negative), but it can be met under other  
circumstances. For example, with overdominance in males there is selection for  
1100 increased *A* frequencies on X chromosomes in males, which are always paired  
with Y-*a* haplotypes. Directional selection for *a* in females can then maintain a  
1102 polymorphism at the **A** locus on the X. This scenario selects for a modifier that  
increases recombination between the sex chromosomes (e.g., blue region of Figure  
1104 2d in Otto 2014) and facilitates the spread of neo-W-*a* haplotypes, which create  
more females bearing more *a* alleles than the ancestral X chromosome does.

### 1106 Tight Linkage and Haploid Selection

Generally, haploid selection expands the scenarios under which neo-W alleles can  
1108 spread. For example, when selection is sexually-antagonistic in diploids ( $s^{\varphi} s^{\delta} < 0$   
and  $0 < h^{\varphi} < 1$ ) an unlinked neo-W ( $R = 1/2$ ) cannot invade unless there is also  
1110 haploid selection, c.f., Figure 1 and Figure S.3. Secondly, with haploid selection,  
overdominance ( $w_{aa}^{\varphi} < w_{Aa}^{\varphi}$  &  $w_{AA}^{\varphi} < w_{Ad}^{\varphi}$ ) is not required for neo-W-*a* haplo-  
1112 types to spread ( $\lambda_{ma} > 1$ ), Figures S.4-S.7. Finally, haploid selection can maintain  
a polymorphism in the face of directional selection in male and female diploids  
1114 (ploidally-antagonistic selection). When selection is ploidally-antagonistic, neo-  
W alleles often spread, for at least some values of  $R$ , Figure S.8.

1116 As discussed above, male haploid selection alters the sex ratio and the alleles  
carried by male gametes that female gametes pair with. Male haploid selection  
1118 in favour of the *a* allele ( $\alpha_{\Delta}^{\delta} < 0$ ,  $w_A^{\delta} < w_a^{\delta}$ ) generates male-biased sex ratios  
at equilibria (A) and (B), where Y-*a* is fixed ( $\hat{p}_Y^{\delta} = 0$ ). Male-biased sex-ratios  
1120 facilitate the spread of neo-W-*A* and neo-W-*a* haplotypes (increasing  $\lambda_{WA}$  and  
 $\lambda_{Wa}$ ). Panels A-C in Figures S.4 and S.5 show that neo-W haplotypes tend to  
1122 spread for a wider range of parameters when sex ratios are male biased, compared  
to Figure 2 without haploid selection. By contrast, male haploid selection in favour  
1124 of the *A* allele generates female-biased sex ratios and reduces  $\lambda_{WA}$  and  $\lambda_{Wa}$ , as  
demonstrated by panels D-F in Figures S.4 and S.5.

<sup>1126</sup> Female haploid selection generates direct selection on the neo-W-*A* and neo-  
<sup>1128</sup> W-*a* haplotypes as they spread in females. Thus, female haploid selection in favour  
of the *a* allele tends to increase  $\lambda_{W_a}$  and decrease  $\lambda_{W_A}$ , as shown by panels A-C in  
Figures S.6 and S.7. Conversely, female haploid selection in favour of the *A* allele  
<sup>1130</sup> increases  $\lambda_{W_A}$  and decreases  $\lambda_{W_a}$ , see panels D-F in Figures S.6 and S.7.

## Supplementary Figures

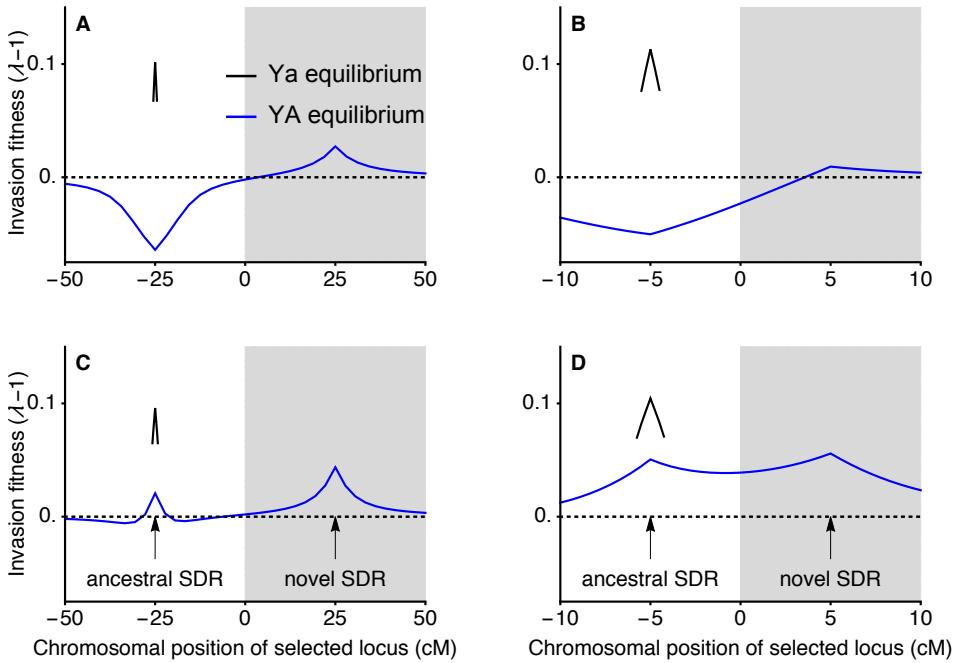


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ( $r \approx 0$ ). In panels A and B, the  $a$  allele is favoured in females ( $w_{aa}^{\varnothing} = 1.05$ ,  $w_{Aa}^{\varnothing} = 1$ ,  $w_{AA}^{\varnothing} = 0.85$ ) 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 ( $w_{aa}^{\varnothing} = w_{AA}^{\varnothing} = 0.6$ ,  $w_{aa}^{\delta} = 0.5$ ,  $w_{Aa}^{\delta} = 0.7$ ,  $w_{Aa}^{\varnothing} = 1$ ). There is no haploid selection  $t^{\varnothing} = \alpha_{\Delta}^{\varnothing} = 0$ . These parameters are marked by daggers in Figure 2B and C, which show that neo-W invasion is expected for any  $R$  ( $\lambda_{WA}, \lambda_{Wa} > 1$ ) when the  $a$  allele is nearly fixed on the Y (black lines in this figure). Equilibria where the  $A$  allele is more common among Y-bearing male gametes can also be stable and allow neo-W invasion 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, in the absence of haploid selection, neo-W alleles should spread only if they are more tightly linked to the selected locus (positive invasion fitness if and only if the selected locus is in the grey region). However, when linkage is tight (panels C and D and when the selected locus is near the SDR in all panels), this prediction breaks down.

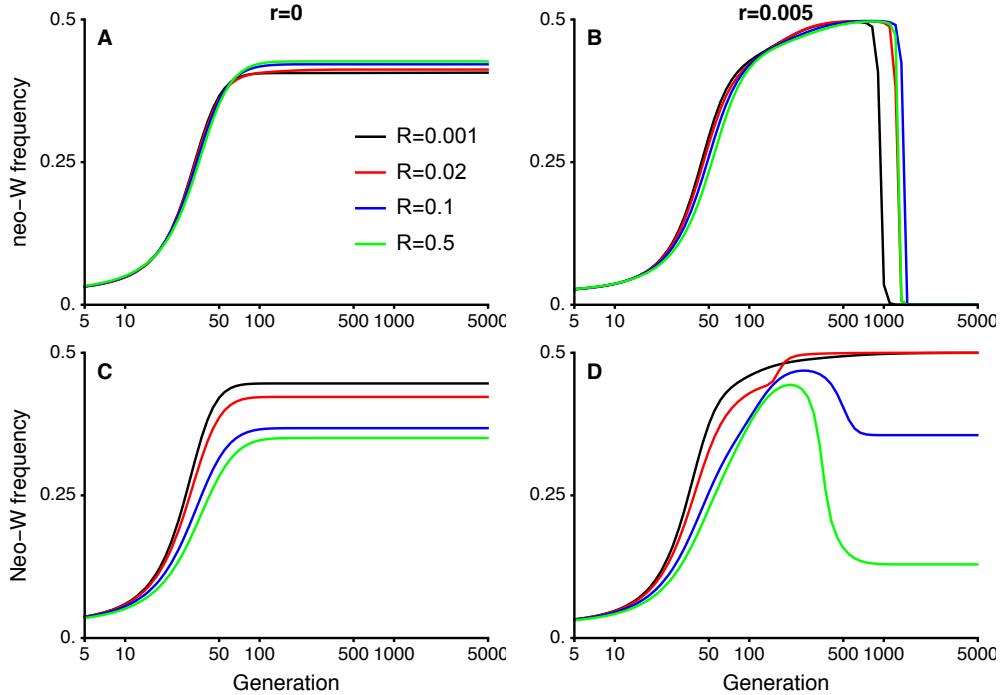


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among 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 ( $w_{aa}^o = 1.05$ ,  $w_{Aa}^o = 1$ ,  $w_{AA}^o = 0.85$ ) 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  $t^\delta = \alpha_\Delta^\delta = 0$ .

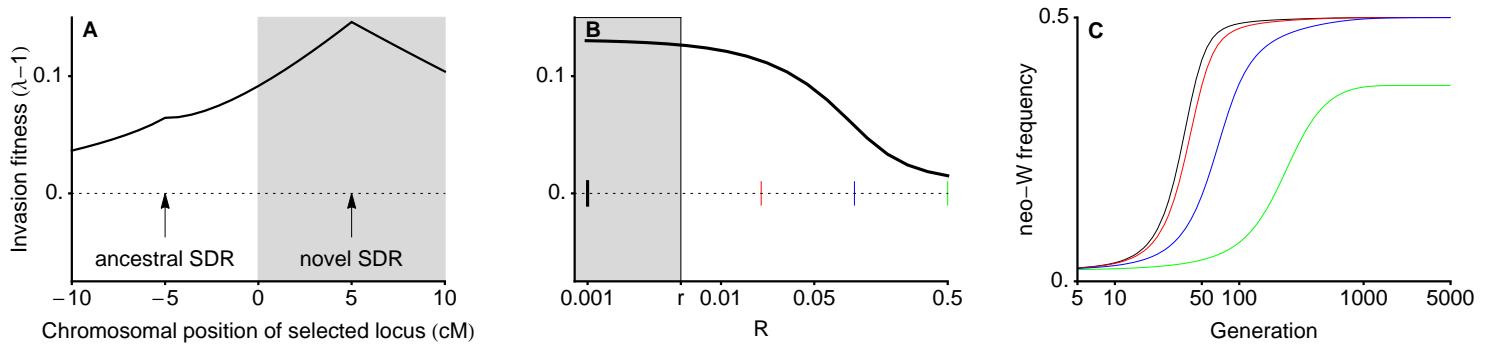


Figure S.3: When there is sexually-antagonistic selection and haploid selection, a neo-W may invade for any  $R$ . Panel A shows that the invasion fitness of a neo-W is positive where linkage is tight, even when  $r < R$  (unshaded region). (remove A?) In panel B, we vary the recombination rate between 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 neo-W invasion are plotted in panel C (black  $R = 0.001$ , red  $R = 0.02$ , blue  $R = 0.1$ , green  $R = 0.5$ ). The diploid selection parameters used in this plot are the same as in Figure 1, marked by an asterisk in Figure S.4A:  $w_{AA}^{\delta} = 1.05$ ,  $w_{Aa}^{\delta} = 1$ ,  $w_{aa}^{\delta} = 0.85$ ,  $w_{AA}^{\vartheta} = 0.85$ ,  $w_{Aa}^{\vartheta} = 1.05$ ,  $w_{aa}^{\vartheta} = -0.08$ , except that there is also male meiotic drive in favour of the  $a$  allele,  $\alpha_{\Delta}^{\delta} = -0.08$ . When  $R = 0.5$  (green curve), the neo-W does not reach fixation and X,Y,Z, and W alleles are all maintained in the population, see Figure S.9C.

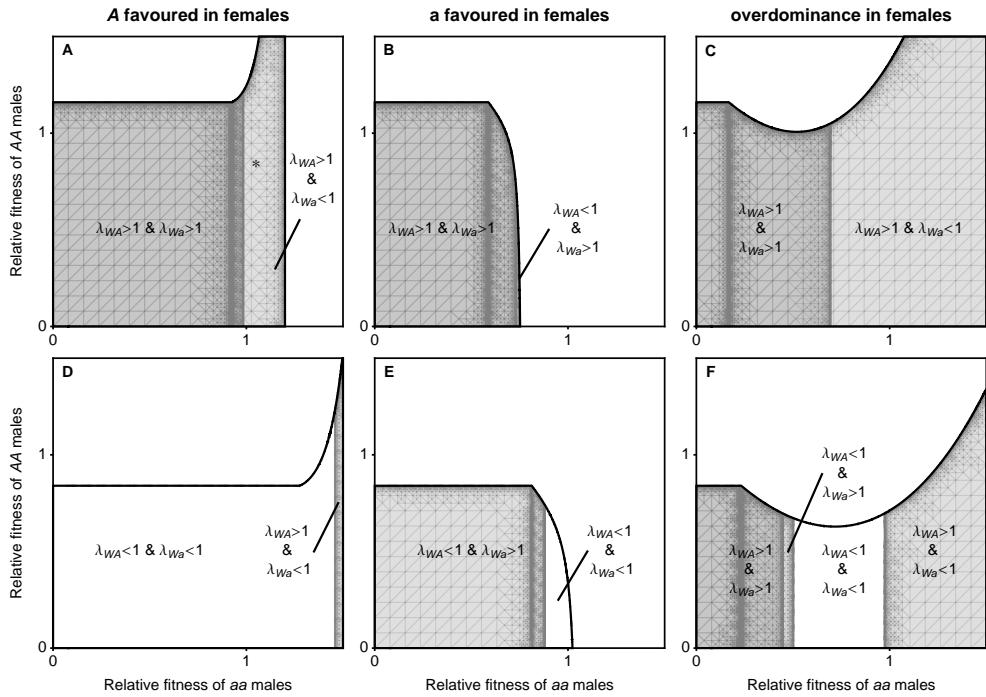


Figure S.4: Meiotic drive in males affects whether neo-W-*A* and neo-W-*a* haplotypes spread when the ancestral-XY locus is tightly linked to a locus under selection ( $r = 0$ ). We vary the fitness of male homozygotes relative to heterozygotes ( $w_{Aa}^{\varnothing} = 1$ ) and only consider stable equilibria at which both *A* locus allele are maintained and the *a* allele is initially fixed on the Y, region outlined. In panels A-C, meiotic drive in males favours the *a* allele ( $\alpha_{\Delta}^{\delta} = -0.16$ ), creating male-biased sex ratios and generally increasing  $\lambda_{WA}$  and  $\lambda_{Wa}$ . By contrast,  $\lambda_{WA}$  and  $\lambda_{Wa}$  tend to be reduced when meiotic drive in males favours the *A* allele ( $\alpha_{\Delta}^{\delta} = 0.16$ ), panels D-F. We consider three forms of selection in females: directional selection in favour of the *A* allele (panels A and D,  $w_{aa}^{\varnothing} = 0.85$ ,  $w_{AA}^{\varnothing} = 1.05$ ), direction selection in favour of the *a* allele (panels B and E,  $w_{aa}^{\varnothing} = 1.05$ ,  $w_{AA}^{\varnothing} = 0.85$ ), and overdominance (panels C and F,  $w_{aa}^{\varnothing} = 0.6$ ,  $w_{AA}^{\varnothing} = 0.6$ ).

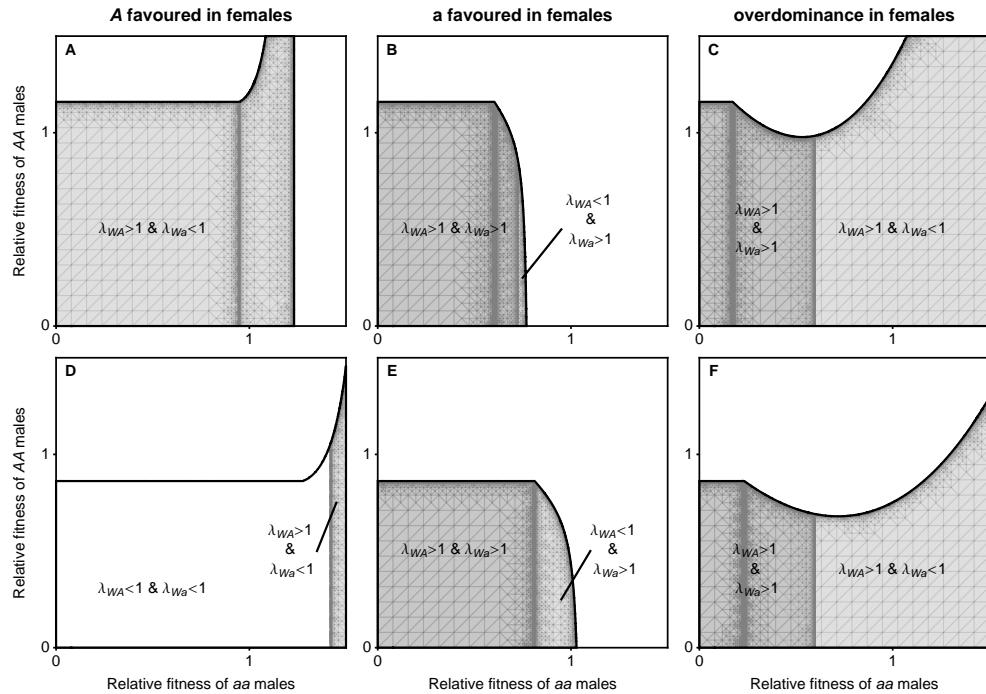


Figure S.5: Parameters for which neo-W-*A* and neo-W-*a* haplotypes spread when there is male gametic competition at a locus that is tightly linked to the ancestral-XY locus. Diploid selection parameters ( $w_{ij}^\delta$ ) are the same as those in Figure S.4. The *a* allele is favoured during male gametic competition in Panels A-C ( $w_a^\delta = 1.16$ ,  $w_A^\delta = 1$ ), which creates male biased sex-ratios and increases  $\lambda_{WA}$  and  $\lambda_{Wa}$ . On the other hand, the *A* allele is favoured during male gametic competition in Panels D-F ( $w_a^\delta = 1$ ,  $w_A^\delta = 1.16$ ) and  $\lambda_{WA}$  and  $\lambda_{Wa}$  tend to be reduced. Compared to the meiotic drive parameters in Figure S.4, the effect of these male gametic competition parameters on the sex ratio is smaller. For example, in Figure S.4A-C, the ancestral sex ratio is  $\alpha^\delta = 0.58$  at equilibrium (B) and in panels A-C of this plot, the ancestral sex ratio is  $w_a^\delta/(w_A^\delta + w_a^\delta) = 0.537$  at equilibrium (B).

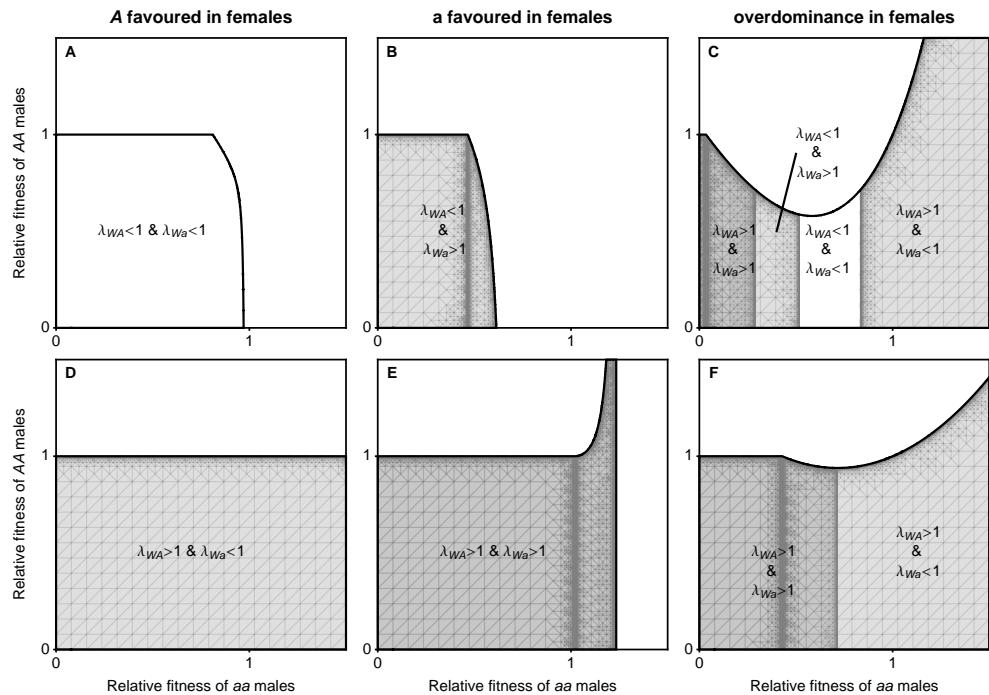


Figure S.6: Parameters for which neo-W-*A* and neo-W-*a* haplotypes spread when there is female meiotic drive at a locus that is tightly linked to the ancestral-XY locus. Diploid selection parameters ( $w_{ij}^\phi$ ) are the same as those in Figure S.4 and S.5. The *a* allele is favoured by meiotic drive in females in Panels A-C ( $\alpha_\Delta^\phi = -0.16$ ), which increases  $\lambda_{Wa}$  and decreases  $\lambda_{WA}$ . Female meiotic drive in favour of the *A* allele (panels D-F,  $\alpha_\Delta^\phi = -0.16$ ) has the opposite effect.

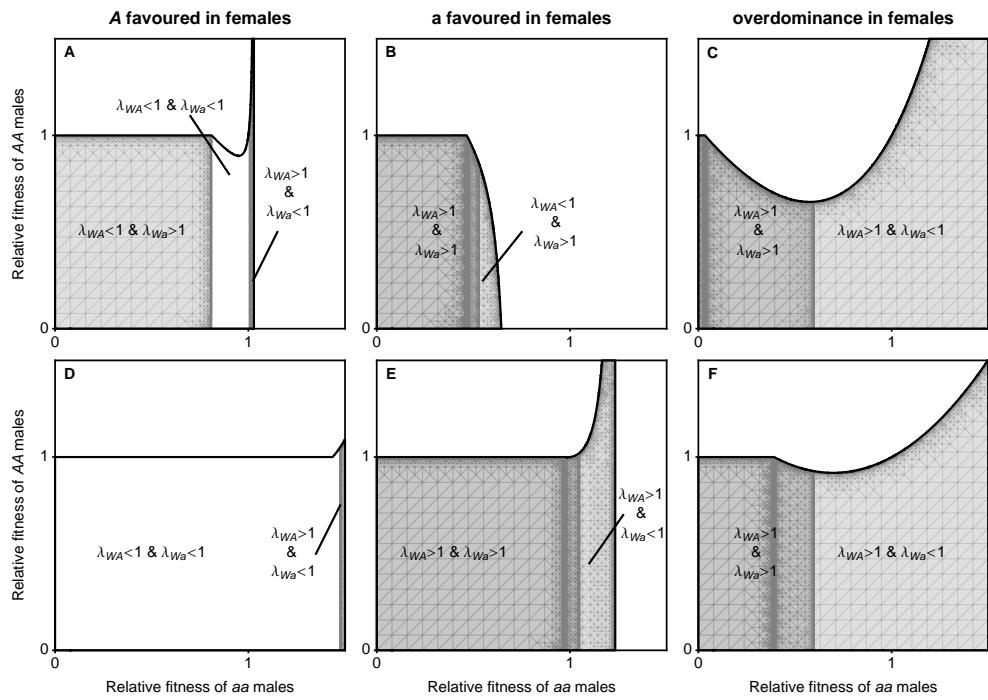


Figure S.7: Parameters for which neo-W-A and neo-W-a haplotypes spread when there is female meiotic drive at a locus that is tightly linked to the ancestral-XY locus. Diploid selection parameters ( $w_{ij}^g$ ) are the same as those in Figure S.4, S.5, and S.6. The *a* allele is favoured during female gametic competition in females in Panels A-C ( $\alpha_{\Delta}^g = -0.16$ ), which increases  $\lambda_{Wa}$  and decreases  $\lambda_{WA}$ . The *A* allele is favoured during gametic competition in panels D-F ( $\alpha_{\Delta}^g = -0.16$ ), giving the opposite effect on  $\lambda_{Wa}$  and  $\lambda_{WA}$ .

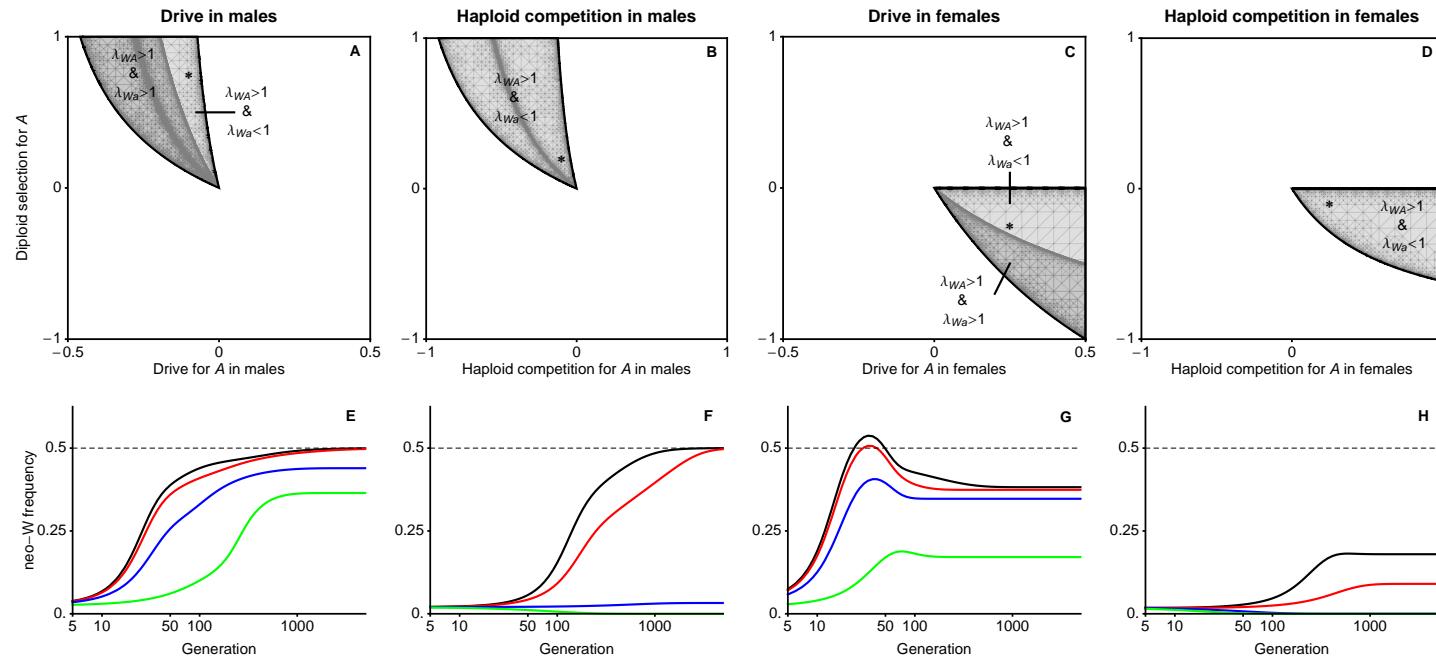


Figure S.8: A-D show when each of the neo-W haplotypes invade an internally stable equilibrium with  $a$  fixed on the Y (found by setting  $r = 0$ ). The y-axis shows directional selection in diploids of both sexes,  $s^{\varphi} = s^{\delta}$ , and the x-axes show sex-specific drive,  $\alpha_{\Delta}^{\varphi}$ , or haploid competition,  $t^{\varphi}$ . The top left and bottom right quadrants therefore imply ploidally-antagonistic selection (and these are the only places where neo-W haplotypes can invade). Dominance is equal in both sexes,  $h^{\varphi} = h^{\delta} = 3/4$ . E-F show the temporal dynamics of neo-W frequency in females with parameters given by the asterisks in the corresponding A-D plot, with  $r = 1/200$ , for four different  $R$ . Black  $R = 1/1000$ , Red  $R = 2/100$ , Blue  $R = 1/10$ , Green  $R = 1/2$ . Dashed line in E-H gives “fixation” of neo-W (all females heterozygous ZW).

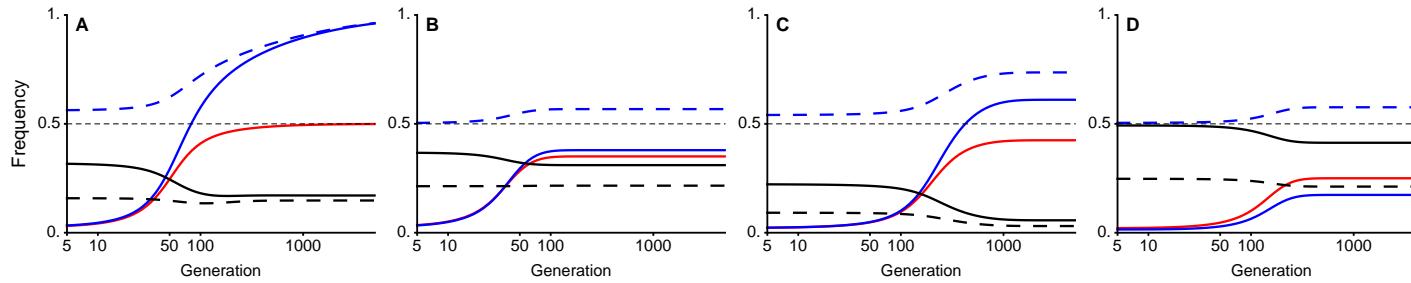


Figure S.9: Dynamics of sex-determining alleles during invasion by a neo-W allele. The curves show the frequencies of the neo-W (red), ancestral-Y (Blue), and A allele among female gametes (solid curves) and among male gametes (dashed curves). In panel A, there is a complete transition from XY sex determination (XX-ZZ females and XY-ZZ males) to ZW sex determination (YY-ZW females and YY-ZZ males). In panels B-D polymorphism is maintained at both the ancestral XY locus and the neo-ZW locus, such that there are males with genotypes XY-ZZ or YY-ZZ and females with genotypes XX-ZZ, XX-ZW, XY-ZW, or YY-ZW. In panel A, selection is ploidally antagonistic with drive in males (parameters as in the green curve in Figure 4B). In panel B, there is overdominance in both sexes (parameters as the green curve in Figure S.2C). In panel C, there is male meiotic drive and sexually-antagonistic selection in diploids (parameters as the green curve in Figure S.4C). (remove D?) Panel D has the same parameters as the red curve in Figure S.8F, except  $r = 0$  (ploidy-antagonism with pollen competition). In all cases, the initial equilibrium frequency has  $a$  near fixed on the Y.