

Haploid Selection, Sex Ratio Bias, and Transitions Between Sex-Determination Systems

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Contributions:

Abstract

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

Introduction

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

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

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

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

84 Here we use mathematical models to find the conditions under which new
sex-determination systems spread when individuals experience selection at both
86 diploid and haploid stages. Even in animal and plant species that have much
larger and more conspicuous diploid phases than haploid phases, many loci ex-
88 perience significant haploid selection through gamete competition and/or meiotic

drive (Mulcahy et al. 1996, Joseph and Kirkpatrick 2004). We use the term ‘meiotic drive’ to refer to the biased (non-Mendelian) segregation of genotypes during gamete production (from one parent) and the term ‘gametic competition’ to refer to selection upon haploid genotypes within a gamete/gametophyte pool (potentially from multiple parents); the term ‘haploid selection’ encompasses both processes.

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

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

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

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

We find that sex-ratio biases caused by haploid selection can exert Fisherian sex-ratio selection upon novel sex-determiners but that their spread is also determined by selection on genetically-associated alleles. Consequently, it is possible for selection on linked alleles to drive turnover between sex-determining systems despite causing transitory or even permanent increases in sex-ratio bias. In addition to considering haploid selection, another novel development in our model is that we consider loci that are under diploid and/or haploid selection and also in very tight linkage with the ancestral sex-determining region. Even in the absence of haploid selection, we show that transitions between male and female heterogamy can then evolve despite the fact that the neo-sex-determining locus is less closely linked to a locus under selection and therefore disrupts favourable ancestral associations between sex and the alleles selected in that sex.

Model

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

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

148 hmm, not sure. This was an idea from Sally, I think in response to terms like $2\alpha^\delta$ and $2(1 - \alpha^\delta)$. It's possible that it makes other equations less easy to understand.
150 My previous (not explained) logic was to use w and α for strong selection and s ,
 t , and α_Δ for weak selection. Just to check, I should have written $\alpha^\delta = (1 + \alpha_\Delta^\delta)/2$,
152 maybe that's where the factors of 1/2 come from? shoot, i may have changed too
much (i.e. strong selection too), but i did use the correct transformation. still, the
154 form of S.8c,d seems wrong. I'll double check it.

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

In each generation, we census the genotype frequencies in male and female
172 gametes/gametophytes (hereafter gametes) before gametic competition. A full de-
scription of our model, including recursion equations, is given in the Appendix.
174 First, competition occurs among male gametes (sperm/pollen competition) and
among female gametes (egg/ovule competition) separately. Selection during ga-
176 metic competition depends on the **A** locus genotype, relative fitnesses are given

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

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

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

¹⁹⁴ **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 (relabelling the ancestrally-heterogametic sex as female and the ancestrally-homogametic sex as male).

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, λ , 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, **Mention the possibility that the other roots yield the leading eigenvalue somewhere.** $\lambda^2 + b\lambda + c = 0$. 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 λ_{mi} is the multiplicative growth rate of mutant haplotypes on background $i \in \{A, a\}$, without accounting for loss due to recombination, and χ_{mi} is the rate at which mutant haplotypes on background

222 $i \in \{A, a\}$ recombine onto the other **A** locus background in heterozygotes (see
 223 Table 2). The λ_{mi} and χ_{mi} , and thus the spread of the mutant m allele, depend on
 224 the frequency of alleles at the **A** and **X** loci in the ancestral population. In the an-
 225 cestral population, it is convenient to follow the frequency of the A allele among
 226 female gametes (eggs), p_X^q , and among X-bearing, p_X^δ , and among Y-bearing, p_Y^δ ,
 227 male gametes (sperm/pollen). We also track the fraction of male gametes that are
 228 Y-bearing, q , which may deviate from 1/2 due to meiotic drive in males. We will
 229 consider only equilibrium frequencies of alleles, \hat{p}_i^q , and Y-bearing male gametes,
 230 \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 ($k = 0$) |
|---|
| $\lambda_{mA} = (2\zeta)^{-1} [\hat{p}_X^q w_A^q w_A^\delta w_{AA}^\delta + (1 - \hat{p}_X^q) w_a^q w_A^\delta w_{Aa}^\delta (1 + \alpha_\Delta^\delta)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^\delta)$ |
| $\lambda_{ma} = (2\zeta)^{-1} [(1 - \hat{p}_X^q) w_a^q w_a^\delta w_{aa}^\delta + \hat{p}_X^q w_A^q w_a^\delta w_{Aa}^\delta (1 - \alpha_\Delta^\delta)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^\delta)$ |
| $\chi_{mA} = R(2\zeta)^{-1} [(1 - \hat{p}_X^q) w_a^q w_A^\delta w_{Aa}^\delta (1 + \alpha_\Delta^\delta)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^\delta)$ |
| $\chi_{ma} = R(2\zeta)^{-1} [\hat{p}_X^q w_A^q w_a^\delta w_{Aa}^\delta (1 - \alpha_\Delta^\delta)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^\delta)$ |
| neo-W ($k = 1$) |
| $\lambda_{mA} = [2(1 - \zeta)]^{-1} [\bar{p}^\delta w_A^\delta w_A^q w_{AA}^q + (1 - \bar{p}^\delta) w_a^\delta w_A^q w_{Aa}^q (1 + \alpha_\Delta^q)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^q)$ |
| $\lambda_{ma} = [2(1 - \zeta)]^{-1} [(1 - \bar{p}^\delta) w_a^\delta w_a^q w_{aa}^q + \bar{p}^\delta w_A^\delta w_a^q w_{Aa}^q (1 - \alpha_\Delta^q)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^q)$ |
| $\chi_{mA} = R[2(1 - \zeta)]^{-1} [(1 - \bar{p}^\delta) w_a^\delta w_A^q w_{Aa}^q (1 + \alpha_\Delta^q)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^q)$ |
| $\chi_{ma} = R[2(1 - \zeta)]^{-1} [\bar{p}^\delta w_A^\delta w_a^q w_{Aa}^q (1 - \alpha_\Delta^q)] / (\bar{w}_H^q \bar{w}_H^\delta \bar{w}^q)$ |

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

ζ is the zygotic sex ratio (fraction male)

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

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

232 We are particularly concerned with the conditions under which a rare neo-sex-
 233 determining allele increases in frequency, which occurs when the largest eigen-

value, λ , is greater than one. If the average change in frequency of the two haplotypes that carry the m allele (Am and am) is positive, invasion will always occur, i.e., if $(\lambda_{mA} + \lambda_{ma})/2 > 1$ then $\lambda > 1$. If neither haplotype increases in frequency ($\lambda_{mA}, \lambda_{ma} < 1$), the m allele will not invade. Otherwise, the new sex-determining allele increases in frequency on one A background and declines on the other, and invasion requires

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

For example, if we assume that only the mA haplotype has a positive growth rate ($\lambda_{ma} < 1 < \lambda_{mA}$), the second term on the left-hand side of (1) is negative and invasion requires that the growth rate of mA haplotypes and the rate at which they are produced by recombination is sufficiently large relative to that of ma haplotypes. In other words, invasion requires that the average growth rate of the two haplotypes, weighted by the rates they are created by recombination, is positive.

Table 2 illustrates a number of key points about the invasion of neo-Y and 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., the first factor of the λ_{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) fitness of associated alleles (terms involving equilibrium allele frequencies, p 's). 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 is because a dominant neo-Y (neo-W) is always found in males (females), and therefore the frequency of the neo-Y (neo-W) allele, m , only changes in males (females). Finally, invasions by a neo-Y and a neo-W are qualitatively different. This is because a gamete with the ancestral- or neo-Y always pairs with a female gamete containing an X, and both develop into males. By contrast, a gamete with a neo-W can pair with an X or Y male gamete, developing into a female, while 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.

264 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
 266 allele (i.e., \hat{p}_X^{\varnothing} , \hat{p}_X^{δ} , and \hat{p}_Y^{δ}) and Y-bearing male gametes (\hat{q}) in the ancestral pop-
 268 ulation . Since only the A locus experiences selection directly, any deterministic
 270 evolution requires that there is a polymorphism at the A locus. Polymorphisms
 can be maintained by mutation-selection balance or transiently present during the
 272 spread of beneficial alleles. However, polymorphisms maintained by selection can
 maintain alleles at higher allele frequencies for longer periods. Here, we focus of
 274 polymorphisms maintained by selection, where the A allele reaches a stable in-
 termediate equilibrium frequency under the ancestral sex-determination system
 276 before the neo-sex-determining allele (m) arises. We can analytically calculate the
 278 allele frequency of the A allele using two alternative simplifying assumptions: (1)
 the A locus is within (or tightly linked to) the non-recombining region around the
 ancestral SDR ($r \approx 0$) or (2) selection is weak relative to recombination (s^{δ} , t^{δ} ,
 α_{Δ}^{δ} of order $\epsilon \ll 1$).

Tight linkage with the ancestral sex-determining region

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

A neo-Y will never invade an ancestral XY system that already has tight linkage
 288 with the locus under selection ($r = 0$, for details see supplementary *Mathematica*
 file). A neo-Y haplotype with the same allele as the ancestral Y is neutral ($\lambda_{ma} = 1$)
 290 and does not change in frequency. The other neo-Y haplotype will not spread
 ($\lambda_{mA} < 1$) given that the initial equilibrium is stable. Therefore, a neo-Y mutation
 292 cannot spread ($\lambda \leq 1$) in an ancestral XY system that is at equilibrium with all se-

lected loci within the non-recombining region around the SDR. In essence, through
294 tight linkage with the **A** locus, the ancestral Y becomes strongly specialized on the
allele that has the highest fitness across male haploid and diploid phases. Given
296 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.

298 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
300 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, see Figure 1. This re-
302 sult is unexpected given the results of van Doorn and Kirkpatrick (2010), who did
not explicitly calculate equilibrium allele frequencies under tight linkage and gen-
304 erally concluded that heterogametic transitions occur when neo-sex-determining
alleles are in tighter linkage with loci under sex-specific diploid selection. To de-
306 velop an understanding (intuition) for how this happens, we focus on cases where
there is no haploid selection and discuss the effects of haploid selection in the
308 appendix.

If we categorise the *a* allele as being ancestrally ‘male-beneficial’ via the fact
310 that it is fixed on the Y, then $\lambda_{mA} > 1$ indicates that the neo-W spreads when found
with the ancestrally ‘female-beneficial’ allele. Broadly, this is possible because
312 the ancestral X chromosome is not able to perfectly specialise on the ‘female-
beneficial’ allele due to the fact that X’s are sometimes found in males. For ex-
314 ample, when the *a* allele is favoured in males, a polymorphism of *A* and *a* alleles
can be maintained on the X despite directional selection in favour of the *A* al-
316 lele in females ($s^q > 0, 0 < h^q < 1$). Figure 2A indicates that λ_{mA} tends to be
larger than one with sexually-antagonistic selection where the *a* allele is strongly
318 favoured in males (w_{aa} much larger than w_{Aa}). In this case the *a* allele is at high
frequency among XX females is high due to selection upon the X in males. By
320 contrast, W-A haplotypes will only create females with high fitness (*AA* or *Aa*
genotypes) and can therefore have higher fitness than ancestral females. When
322 only one neo-W haplotype can has a positive growth rate (see Figure 2), a neo-W

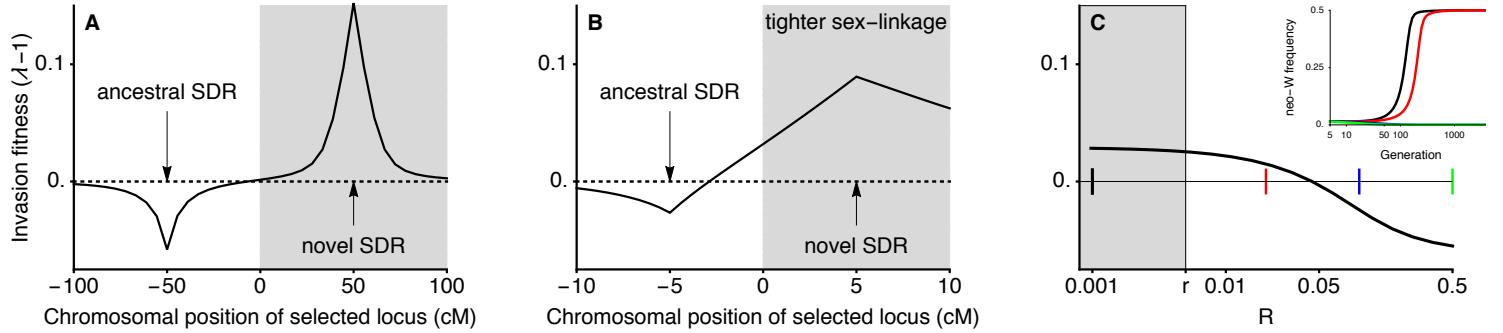


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is more loosely linked to a locus under sexually-antagonistic selection (here, without haploid selection $r^\delta = \alpha_A^\delta = 0$). In panel A, linkage is loose enough relative to selection that the weak selection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

can invade as long as equation (1) is satisfied, which may require that the recombination rate, R , is small enough. Nevertheless, because we assume here that r is small, these results indicate that a more loosely linked sex-determining region can spread. Therefore, tightly sex-linked loci that experience sexually-antagonistic selection can drive heterogametic transitions in which the neo-SDR is less closely linked to the locus under selection (Figure 1).

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

X can be polymorphic or even fixed for the *A* allele despite favouring the *a* allele
 336 during selection in females (e.g., see outlined region in Figure 2B and Lloyd and
 Webb 1977, Otto 2014). In such cases, neo-W-*a* haplotypes can spread because
 338 they create more *Aa* and *aa* females when pairing with an X from males and be-
 cause they bring Y-*a* haplotypes into females, in which case females are always *aa*.
 340 As discussed in the appendix, this scenario where neo-W's associated with *a* are
 favoured can also occur with haploid selection, even without overdominance (e.g.,
 342 when *a* is female-beneficial and favoured by haploid selection in male gametes).

In some cases, both W-*A* and W-*a* haplotypes can spread, e.g., when *AA* in-
 344 dividuals 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*
 346 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
 348 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,
 350 see Figures S.1 and S.2 for examples).

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

Loose linkage with the ancestral sex-determining region

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

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

360 and

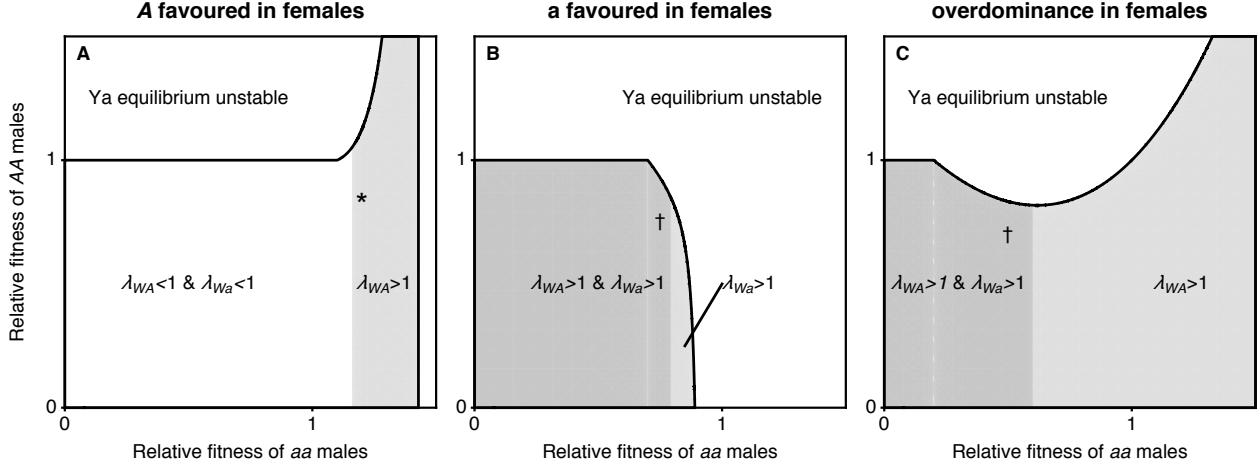


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ($r = 0$), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ($w_{Aa}^{\delta} = 1$) and only consider stable equilibria at which both A locus alleles are maintained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

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

where $V_A = \bar{p}(1 - \bar{p})$ is the variance in the equilibrium frequency of A and $S_A = (D^{\delta} + \alpha_{\Delta}^{\delta} + t^{\delta}) - (D^{\varphi} + \alpha_{\Delta}^{\varphi} + t^{\varphi})$ describes sex differences in selection for the A versus a across diploid selection, meiosis, and gametic competition. The diploid selection term, $D^{\delta} = [\bar{p}s^{\delta} + (1 - \bar{p})h^{\delta}s^{\delta}] - [\bar{p}h^{\delta}s^{\delta} + (1 - \bar{p})]$, is the difference in fitness between A and a alleles in diploids of sex $\delta \in \{\varphi, \delta\}$, where \bar{p} is the leading-order probability of mating with an A -bearing gamete from the opposite sex (see Appendix). The difference in A -allele-frequency among Y-bearing sperm versus X-bearing sperm is given by $\hat{p}_Y^{\delta} - \hat{p}_X^{\delta} = V_A(D^{\delta} - D^{\varphi} + \alpha_{\Delta}^{\delta} - \alpha_{\Delta}^{\varphi} + t^{\delta} - t^{\varphi})(1 - 2r)/2r$.

The neo-sex-determining allele, m , will spread if $\lambda_{m,XY} > 1$. Equation (2)

370 demonstrates that under weak selection a neo-Y will invade an XY system if and
371 only if it is more closely linked to the selected locus than the ancestral sex-determining
372 region (i.e., if $R < r$; note that $V_A S_A^2$ is strictly positive as long as A is polymor-
373 phic). This echoes our tight linkage results above where a neo-Y could never invade
374 if $r \approx 0$ and is consistent with the results of van Doorn and Kirkpatrick (2007),
375 who considered diploid selection only and also found that homogametic transitions
376 (XY to XY or ZW to ZW) can only occur when the neo-sex-determining locus is
377 more closely linked to a locus under sexually-antagonistic selection.

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

386 Equation (3) shows that, with weak selection, neo-W alleles can invade an XY
387 system for a large number of selective regimes. To clarify the parameter space
388 under which $\lambda_{W',XY} > 1$, we consider several special cases. Firstly, if the A locus
389 is unlinked to the ancestral sex-determining region ($r = 1/2$), a more closely linked
390 neo-W ($R < 1/2$) can always invade because there is then no association between
391 A alleles and sex chromosomes in males, $(\hat{p}_Y^\delta - \hat{p}_X^\delta) = 0$, see equation (S.5). The
392 second term in equation (3) then disappears and invasion depends only on the sign
393 of $(r - R)$. Indeed, invasion typically occurs when the neo-W is more closely linked
394 to the selected locus than the ancestral sex-determining region, under a variety of
395 selective regimes (Figure 3). Secondly, we can simplify the discussion of cases
396 where invasion occurs despite looser sex-linkage, $R > r$, by focussing on the
397 special case where $R = 1/2$ and $r < 1/2$ (e.g., the selected locus is on the ancestral
398 sex chromosome and the novel sex-determining locus arises on an autosome). In
399 table 3 we give the conditions where invasion occurs when we further assume

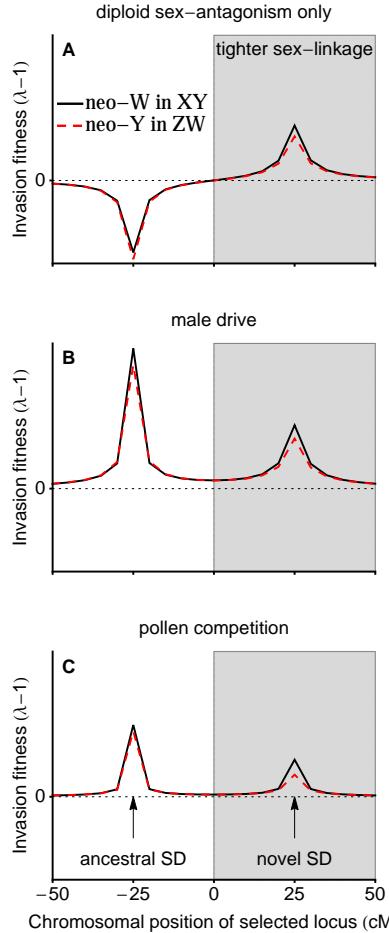


Figure 3: A neo-W can invade an XY system under a large number of selective regimes. In panel A, there is no haploid selection ($t^\delta = \alpha_\Delta^\delta = 0$) and selection in diploids is sexually antagonistic ($s^\delta = -s^q = 1/10$, $h^\delta = 1 - h^q = 3/10$), in which case the neo-sex-determining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

400 that haploid selection only occurs in one sex (e.g., during male meiosis only) and
 dominance coefficients are equal in the two sexes, $h^q = h^\delta$. When there is no
 402 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
404 females ($s^{\varphi} s^{\delta} > 0$, see Figure 3B and Figure 4B). When there is no meiotic drive
and gametic competition occurs in one sex only, an unlinked neo-W can invade as
406 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 3C).
408 These special cases indicate that neo-W invasion can occur for a relatively large
fraction of the parameter space, even if the neo-W uncouples the sex-determining
410 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 | $h^{\delta} = h^{\varphi}, t^{\varphi} = t^{\delta} = \alpha_{\Delta}^{\varphi} = 0$ | $s^{\varphi} s^{\delta} > 0$ |
| female drive only | $h^{\delta} = h^{\varphi}, t^{\varphi} = t^{\delta} = \alpha_{\Delta}^{\delta} = 0$ | $s^{\varphi} s^{\delta} > 0$ |
| sperm competition only | $h^{\delta} = h^{\varphi}, t^{\varphi} = \alpha_{\Delta}^{\varphi} = \alpha_{\Delta}^{\delta} = 0$ | $s^{\varphi}(s^{\delta} - s^{\varphi}) > 0$ |
| egg competition only | $h^{\delta} = h^{\varphi}, t^{\delta} = \alpha_{\Delta}^{\varphi} = \alpha_{\Delta}^{\delta} = 0$ | $s^{\delta}(s^{\varphi} - s^{\delta}) > 0$ |

Previous research suggests that when the ancestral sex-determining locus is
412 linked to a locus that experiences haploid selection (e.g., meiotic drive), a new,
unlinked sex-determining locus invades in order to restore equal sex ratios (Koziel-
414 ska et al. 2010). Consider, for example, the case where the A locus is linked to the
ancestral-SDR ($r < 1/2$) and experiences meiotic drive in males only (e.g., dur-
416 ing spermatogenesis but not during oogenesis, $\alpha_{\Delta}^{\delta} \neq 0, \alpha_{\Delta}^{\varphi} = 0$), without gametic
competition ($t^{\varphi} = t^{\delta} = 0$). In this case, the zygotic sex ratio can be initially biased
418 only if the ancestral sex-determining system is XY (Figure 4B). We might there-
fore expect a difference in the potential for XY to ZW and ZW to XY transitions.
420 However, to leading order with selection weak relative to recombination, we find
that sex ratio selection favours the spread of a neo-W (through the first terms in
422 table 2) by an amount that is equal in magnitude to the fitness effects of alleles
associated with new sex-determining alleles (second terms in table 2). Thus, in-
424 vasion by a neo-W into an XY system and invasion by a neo-Y into a ZW system

occur under the same conditions ($\lambda_{Y',XY} = \lambda_{W',ZW}$ and $\lambda_{Y',ZW} = \lambda_{W',XY}$, at least to order ϵ^2). For example, in Figure 4B neo-W alleles invade an ancestrally-XY system where females are initially rare because the ancestral-Y is associated with a male meiotic drive allele. However, Figure 4A shows that a neo-Y can invade an ancestrally-ZW system under the same conditions. In fact, where $R < 1/2$ the neo-Y becomes associated with the male meiotic drive allele such that the zygotic sex ratio evolves to become biased towards males.

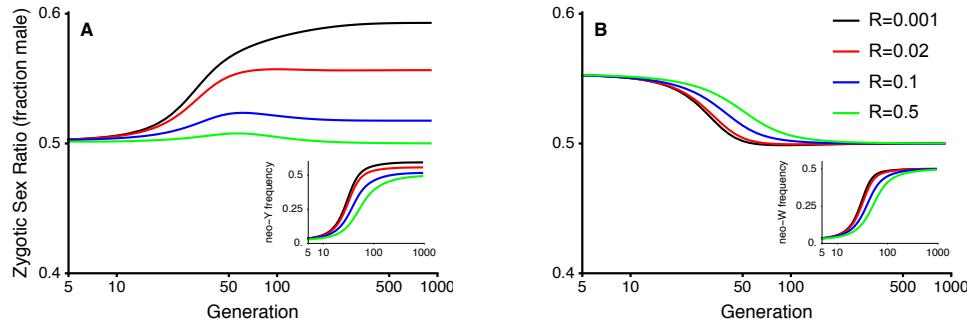


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidally antagonistic with haploid selection favouring the a allele during male meiosis ($s^q = s^\delta = 0.2$, $h^q = h^\delta = 0.7$, $t^q = t^\delta = \alpha_\Delta^\delta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

The green curves in Figure 4 demonstrate a case where transitions between male and female heterogamety occur 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. However, 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 and $r < 1/2$ (equation S.5). Polymorphism at the A
442 locus is maintained by counter-selection against the a allele in diploids and therefore ancestral-ZZ males have generally low diploid fitness. A freely recombining
444 neo-Y ($R = 1/2$) is not directly favoured or disfavoured by male meiotic drive because it is equally likely to be segregate with the A or a allele when found in
446 a heterozygote. The neo-Y spreads because it produces males with high diploid fitness through matings with ancestral-W-bearing female gametes, which are more
448 likely to carry the A allele. Thus, a key factor in explaining why heterogametic transitions can occur when $R > r$ is that the neo-SDR determines sex in the
450 diploid phase but 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 developing as females with probability k . Here, we do not assume that the environmental
456 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 fitness; 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

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

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

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

⁴⁸⁴ Significantly, equation (5) is the same whether ESD is invading an ancestrally XY or ZW system (because $\lambda_{Y',XY} = \lambda_{W',ZW}$ and $\lambda_{W',XY} = \lambda_{Y',ZW}$). Thus, because the sex ratio is only biased by male haploid selection when the ancestral ⁴⁸⁶ sex-determination system is XY, Fisherian sex-ratio selection alone does not ex-

plain the invasion of ESD under weak selection. Specifically, with male haploid
488 selection, 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
490 that 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).

492 Discussion

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

Sex-ratio biases caused by haploid selection can facilitate heterogametic transi-
512 tions between sex-determining systems. For instance, alleles favoured by haploid
selection in males often become associated with the Y, which leads to a male-
514 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
516 examples see Kozielska et al. 2010, Úbeda et al. 2015). However, sex-ratio se-
lection can be overwhelmed by additional selective effects (e.g., when a linked
518 allele is beneficial for male diploids but detrimental for female diploids; Table 3),
preventing the neo-W from invading. Indeed, transitions between sex-determining
520 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-
522 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
524 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
526 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
528 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
530 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
532 selection is weak.

We have shown that the spread of new sex determination systems can be driven
534 by loci experiencing haploid selection. Because haploid selection can cause tran-
sitions that increase or decrease sex-linkage, haploid selection may lead to less
536 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 par-
538 ticular, if haploid selection is strong but selective differences between male and
female diploids are weak, we find that heterogametic transitions (XY to ZW or
540 vice versa) are favoured more strongly than homogametic transitions (e.g., with
 $|D^\delta - D^\varphi| \ll |\alpha_\Delta^\delta - \alpha_\Delta^\varphi + t^\delta - t^\varphi|$ we have $\lambda_{W',XY} > \lambda_{Y',XY}$; equations 3 and S.5).
542 Turnovers driven by haploid selection may help to explain the relative rarity of
heteromorphic sex chromosomes in plants, which are thought to experience more
544 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 *Orites* (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 could drive transitions between dioecy and hermaphroditism, which are frequent in plants (Käfer et al., 2017, Sabath et al., 2017).

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

We assume that sex-determining alleles do not experience direct selection ex-

cept via their associations with sex and selected alleles. However, in some cases,
576 there may be significant degeneration around the sex-limited allele (Y or W) in the
ancestral sex-determining region because recessive deleterious mutations and/or
578 deletions accumulate around the Y or W sex-determining regions (Rice 1996,
Charlesworth and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). During
580 heterogametic transitions (XY to ZW or ZW to XY), but not homogametic transi-
tions (XY to XY or ZW to ZW), any recessive deleterious alleles linked to the Y or
582 W are revealed to selection in YY or WW individuals (Bachtrog et al. 2014). This
phenomenon was studied by van Doorn and Kirkpatrick (2010), who found that
584 degeneration can prevent fixation of a neo-W or a neo-Y allele, leading to a mixed
sex-determination system where the ancestral and new sex-determining loci are
586 both segregating. However, they noted that very rare recombination events around
the ancestral sex-determining region can allow these heterogametic transitions to
588 complete. Degeneration around the Y or W could explain why heterogametic transi-
tions are not observed to be much more common than homogametic transitions
590 despite the fact that our models demonstrate that they are favoured under a wider
range of conditions. For example, Vicoso and Bachtrog (2015) found a dozen
592 sex chromosome configurations among Dipteran species but only one transition
between male and female heterogamety.

594 Another simplification that we made is that meiotic drive involves only a single
locus with two alleles. However, many meiotic drive systems involve an interac-
596 tion with another locus at which alleles may ‘suppress’ the action of meiotic drive
(Burt and Trivers 2006, Lindholm et al. 2016) Taylor,1999. Thus, the dynamics
598 of meiotic drive alleles can be heavily dependent on the interaction between two
loci and the recombination rate between them, which in turn can be affected by
600 sex-linkage if there is reduced recombination between sex chromosomes (Hurst
and Pomiankowski 1991). Furthermore, in some cases, a driving allele may act
602 by killing any gametes that carry a ‘target’ allele at another locus, in which case
there can be fertility effects which can affect the equilibrium frequency of a meiotic
604 drive allele (Holman et al. 2015). In polygamous mating systems, the intensity of

pollen/sperm competition can depend on the density of males available to donate
606 pollen/sperm, which can itself depend on the sex ratio (Taylor and Jaenike 2002). In terms of our model, this implies that the strength of gametic competition (t^δ)
608 may both determine and be determined by the sex ratio. How the evolution of new sex-determining mechanisms could be influenced by two-locus meiotic drive
610 and/or by ecological feedbacks under different mating systems remains to be studied.

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

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

632 “Polygenic sex determination has been reported in many plants (e.g. Shannon & Holsinger 2007), fishes (Vandepitte et al. 2007; Ser et al. 2010; Liew et al.
634 2012), crustaceans (e.g. Battaglia 1958; Battaglia & Malesani 1959; Voordouw

& Anholt 2002), bivalves (Haley 1977; Saavedra et al. 1997), gastropods (Yusa 636 2007a,b), and polychaetes (Bacci 1965, 1978; Premoli et al. 1996).” From Vuilleumier et al. 2007: “Polymorphism for sex-determining genes within or among populations has been reported in many species including houseflies, midges, woodlice, 638 platyfish, cichlid fish, and frogs (Gordon, 1944; Kallman, 1970; Thompson, 1971; Macdonald, 1978; Bull, 1983; Rigaud et al., 1997; Caubet et al., 2000; Lande et 640 al., 2001; Ogata et al., 2003; Lee et al., 2004; Mank et al., 2006).” Also check 642 Kallman (1984) -from vD&K, 2010.

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Appendix

830 Recursion Equations

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

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

Random mating then occurs between gametes to produce diploid zygotes. The frequencies of XX zygotes are then denoted as xx_{ij} , XY zygotes as xy_{ij} , and YY zygotes as yy_{ij} , where **A** and **M** locus genotypes are given by $i, j \in \{1, 2, 3, 4\}$, as above. In XY zygotes, the haplotype inherited from an X-bearing gamete is given by i and the haplotype from a Y-bearing gamete is given by j . In XX and YY zygotes, individuals with diploid genotype ij are equivalent to those with diploid genotype ji ; for simplicity, we use xx_{ij} and yy_{ij} with $i \neq j$ to denote the average of these frequencies, $xx_{ij} = (x_i^{\varphi,s} x_j^{\delta,s} + x_j^{\varphi,s} x_i^{\delta,s})/2$ and $yy_{ij} = (y_i^{\varphi,s} y_j^{\delta,s} + y_j^{\varphi,s} y_i^{\delta,s})/2$.

Denoting the **M** locus genotype by $b \in \{MM, Mm, mm\}$ and the **X** locus genotype by $c \in \{XX, XY, YY\}$, zygotes develop as females with probability k_{bc} . Therefore, the frequencies of XX females are given by $xx_{ij}^\Omega = k_{bc} xx_{ij}$, XY females are given by $xy_{ij}^\Omega = k_{bc} xy_{ij}$, and YY females are given by $yy_{ij}^\Omega = k_{bc} yy_{ij}$. Similarly, XX male frequencies are $xx_{ij}^\delta = (1 - k_{bc}) xx_{ij}$, XY male frequencies are $xy_{ij}^\delta = (1 - k_{bc}) xy_{ij}$, and YY males frequencies are $yy_{ij}^\delta = (1 - k_{bc}) yy_{ij}$. This

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

Selection among diploids then occurs according to the diploid genotype at the
 865 **A** locus, $l \in \{AA, Aa, aa\}$, for an individual of type ij (see Table 1). The diploid
 866 frequencies after selection in sex $\hat{\varphi}$ are given by $xx_{ij}^{\hat{\varphi},s} = w_l^{\hat{\varphi}} xx_{ij}/\bar{w}^{\hat{\varphi}}$, $xy_{ij}^{\hat{\varphi},s} =$
 867 $w_l^{\hat{\varphi}} xy_{ij}/\bar{w}^{\hat{\varphi}}$, and $yy_{ij}^{\hat{\varphi},s} = w_l^{\hat{\varphi}} yy_{ij}/\bar{w}^{\hat{\varphi}}$, where $\bar{w}^{\hat{\varphi}} = \sum_{i=1}^4 \sum_{j=1}^4 w_l^{\hat{\varphi}} xx_{ij} + w_l^{\hat{\varphi}} xy_{ij} +$
 868 $w_l^{\hat{\varphi}} yy_{ij}$ is the mean fitness of individuals of sex $\hat{\varphi}$.

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

Table S.1: ρ substitutions for different loci orders (assuming no interference)

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

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

competition) in the next generation are given by

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

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

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

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

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

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

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

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

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

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

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

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

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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.

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Resident equilibrium and stability

In the resident population (allele M fixed), we follow the frequency of A in X-bearing female gametes, p_X^{φ} , and X-bearing male gametes, p_X^{δ} , and Y-bearing male gametes, p_Y^{δ} . 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.

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

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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^{δ}) | $\bar{p}^{\delta} w_A^{\delta} + (1 - \bar{p}^{\delta}) w_a^{\delta}$ |
| 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}^{δ}) | $\zeta^{-1} [p_X^{\varnothing} w_A^{\varnothing} p_Y^{\delta} w_A^{\delta} w_{AA}^{\delta} + (1 - p_X^{\varnothing}) w_a^{\varnothing} p_Y^{\delta} w_A^{\delta} w_{Aa}^{\delta} + p_X^{\varnothing} w_A^{\varnothing} (1 - p_Y^{\delta}) w_a^{\delta} w_{Aa}^{\delta} + (1 - p_X^{\varnothing}) w_a^{\varnothing} (1 - p_Y^{\delta}) w_a^{\delta} w_{aa}^{\delta}] / (\bar{w}_H^{\varnothing} \bar{w}_H^{\delta})$ |
| fraction zygotes male (ζ) | $q [p_Y^{\delta} w_A^{\delta} + (1 - p_Y^{\delta}) w_a^{\delta}] / \bar{w}_H^{\delta}$ |

bility can be calculated analytically without assuming anything about the relative
904 strengths of selection and recombination. However, here, we focus on two regimes
905 (tight linkage and weak selection) in order to make fewer assumptions about fit-
906 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-
908 tral population when the recombination rate between the X and A loci is small
910 (r of order ϵ). Selection at the A locus will not affect evolution at the novel sex-
912 determining locus, M, if one allele is fixed on all backgrounds. We therefore focus
on the five equilibria that maintain both A and a alleles, four of which are given to
leading order by:

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

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

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

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

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

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

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

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

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

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

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

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

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

check last condition and the stability condition below are correct

938 Selection weak relative to recombination (weak selection)

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

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

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

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

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

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

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

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

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

956 Invasion conditions

Cover the other parts of the characteristic polynomial here.

958 A rare neo-Y or neo-W will spread from a given ancestral equilibrium when
the leading eigenvalue, λ , of the Jacobian matrix derived from the eight mutant
960 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)
962 and is epistemically dominant to the ancestral sex-determining locus, we need only
two recursion equations (e.g., tracking the change in the frequency of neo-Y- A and
964 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
966 (Table 2).

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

⁹⁷⁰ eigenvalue, λ , for any k , is given up to order ϵ^2 by equation (4).

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

⁹⁷² Here, we explore the conditions under which a neo-W invades an XY system as-
 suming that the A locus is initially in tight linkage with the ancestral sex-determining
⁹⁷⁴ region ($r \approx 0$). We disregard neo-Y mutations, which never spread given that the
 ancestral population is at a stable equilibrium (see supplementary *Mathematica*
⁹⁷⁶ notebook for proof).

⁹⁷⁸ Starting with the simpler equilibrium (B), the terms of the characteristic poly-
 nomial are

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

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

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

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

Haploid selection impacts the spread of neo-W haplotypes in three ways. Firstly,
⁹⁸⁰ the zygotic sex ratio becomes male biased, $\zeta > 1/2$, when the a allele (which is
 fixed on the Y) is favoured during competition among male gametes or by mei-
⁹⁸² otic drive in males. Specifically, at equilibrium (B), the sex ratio is $\zeta = w_a^\delta(1 -$
 $\alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ where $2\bar{w}_H^\delta = [w_a^\delta(1 - \alpha_\Delta^\delta) + w_A^\delta(1 + \alpha_\Delta^\delta)]$ has been canceled out in
⁹⁸⁴ equations (S.6) to leave the term $[w_a^\delta(1 - \alpha_\Delta^\delta)]^{-1}$. Male biased sex ratios facilitate
 the spread of a neo-W because neo-W alleles cause the zygotes that carry them to
⁹⁸⁶ develop as the rarer, female, sex.

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

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

Thirdly, haploid selection in males affects the diploid genotypes of females
 995 by altering the allele frequencies in the male gametes that female gametes pair
 996 with. At equilibrium (*B*), neo-W female gametes will mate with X-*A* male ga-
 997 metes with probability $w_A^{\delta}(1 + \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$ and Y-*a* male gametes with probability
 998 $w_a^{\delta}(1 - \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$, where the $2\bar{w}_H^{\delta}$ terms have been canceled in equation (S.6)
 1000 (as mentioned above). Thus, for example, neo-W-*A* haplotypes are found in *AA*
 1001 female diploids with probability $w_A^{\delta}(1 + \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$ (first term in square brack-
 1002 ets in the numerator of equation S.6a) and in *Aa* female diploids with probability
 1003 $w_a^{\delta}(1 - \alpha_{\Delta}^{\delta})/(2\bar{w}_H^{\delta})$ (see equation S.6c and second term in square brackets in the
 1004 numerator of equation S.6a).

The other terms in equations (S.6) are more easily interpreted if we assume that
 1005 there is no haploid selection in either sex, in which case $\lambda_{mA} > 1$ when $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$
 1006 and $\lambda_{ma} > 1$ when $(w_{Aa}^{\varphi} + w_{aa}^{\varphi})/2 > w_{AA}^{\varphi}$. These conditions cannot be met under
 1007 purely sexually-antagonistic selection, where *A* is directionally favoured in females
 1008 ($w_{AA}^{\varphi} > w_{Aa}^{\varphi} > w_{aa}^{\varphi}$) and *a* is directionally favoured in males ($w_{AA}^{\delta} > w_{Aa}^{\delta} > w_{aa}^{\delta}$).
 1009 Essentially, the X is then already as specialized as possible for the female beneficial
 1010 allele (*A* is fixed on the X), and the neo-W often makes daughters with the Y-*a*
 1011 haplotype, increasing the flow of *a* alleles into females, which reduces the fitness
 1012 of those females.

1014 If selection doesn't uniformly favour *A* in females, however, neo-W-*A* hap-
 1015 lotypes and/or neo-W-*a* haplotypes can spread ($\lambda_{mA} > 1$ and/or $\lambda_{ma} > 1$) at this
 1016 equilibrium. A neo-W can spread alongside the *A* allele ($\lambda_{mA} > 1$), despite the
 fact that a neo-W brings Y-*a* haplotypes into females, when $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$. In this

1018 case the a allele is favoured by selection in females despite A being fixed on the
 X. For this equilibrium to be stable, $X-A$ must be sufficiently favoured in males
 1020 (for example, by overdominance in males, remembering that a is fixed on the Y)
 to keep A fixed on the X. Specifically, from the stability conditions for equilibrium
 1022 (B), we must have $w_{Aa}^\delta / [(w_{aa}^\delta + w_{Aa}^\delta)/2] > w_{Aa}^\varphi / w_{AA}^\varphi$.

Still considering $w_{Aa}^\varphi > w_{AA}^\varphi$, the neo-W can also spread alongside the a allele
 1024 ($\lambda_{ma} > 1$) if there is sufficiently strong underdominance in females ($w_{aa}^\varphi > w_{Aa}^\varphi$)
 [this is describing directional selection, not overdominance - check the conditions
 1026 for the case we want to talk about], such that $(w_{Aa}^\varphi + w_{aa}^\varphi)/2 > w_{AA}^\varphi$. In this case,
 a is not favored in females near the equilibrium where females are AA (comparing
 1028 Aa to AA genotypes) and yet the neo-W can spread with a because it produces
 female aa individuals by capturing Y-a haplotypes.

1030 Similar equations can be derived for equilibrium (A) by substituting the equi-
 librium allele frequencies into Table 2

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

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

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

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

1032 where

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

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

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

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

As with equilibrium (B), haploid selection again modifies invasion fitnesses
 1034 by altering the sex-ratio and the diploid genotypes of females and directly selecting upon female gametes. The only difference is that resident XX females are no
 1036 longer always homozygote AA and males are no longer always heterozygote Aa . Thus the effect of haploid selection in males is reduced, as is the difference in fitness
 1038 between neo-W haplotypes and resident X haplotypes, as both can be on any diploid or haploid background.

1040 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
 1042

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

where $\phi - \psi = w_{AA}^\varphi w_{Aa}^\delta - w_{aa}^\varphi w_{aa}^\delta$ and both ϕ and ψ are positive when equilibrium
 1044 (A) is stable. In contrast to equilibrium (B), a neo-W haplotype can spread under purely sexually-antagonistic selection ($w_{aa}^\varphi < w_{Aa}^\varphi < w_{AA}^\varphi$ and $w_{AA}^\delta < w_{Aa}^\delta < w_{aa}^\delta$).
 1046 The neo-W- A can spread as long as it becomes associated with females that bear more A alleles than observed at equilibrium (A).

1048 Without haploid selection, the neo-W- a haplotype spreads ($\lambda_{ma} > 1$) if and only if

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

1050 This condition cannot be met with purely sexually antagonistic selection (as both
 terms on the left-hand side would then be negative), but it can be met under other
 1052 circumstances. For example, with overdominance in males there is selection for
 increased *A* frequencies on X chromosomes in males, which are always paired
 1054 with Y-*a* haplotypes. Directional selection for *a* in females can then maintain a
 polymorphism at the A locus on the X. This scenario selects for a modifier that
 1056 increases recombination between the sex chromosomes (e.g., blue region of Figure
 2d in Otto 2014) and facilitates the spread of neo-W-*a* haplotypes, which create
 1058 more females bearing more *a* alleles than the ancestral X chromosome does.

Supplementary Figures

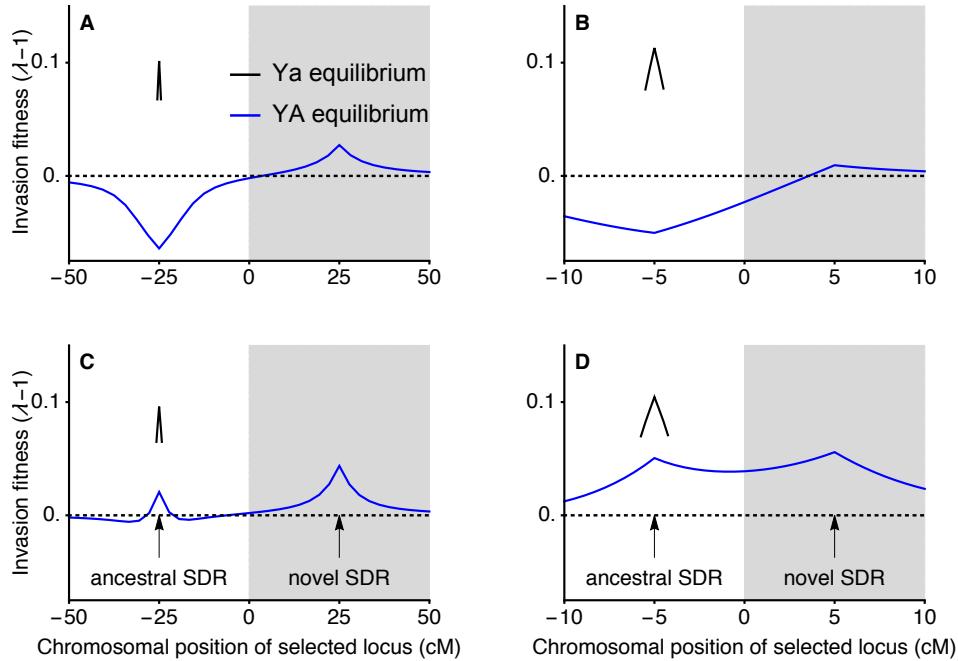


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ($r \approx 0$). In panels A and B, the a allele is favoured in females ($w_{aa}^{\varphi} = 1.05$, $w_{Aa}^{\varphi} = 1$, $w_{AA}^{\varphi} = 0.85$) and selection in males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

1060 Add Sally's figure showing lambda for small r near equil A versus near equil
 1061 B. Add references to this figure to appendix where we discuss whether lambdas
 1062 can be greater than 1 with sexually antagonistic selection.

1063 Perhaps it would also be useful to add an 8 panel figure that features ploidally
 1064 antagonistic selection. For each type of haploid selection (gametic competition/
 1065 meiotic drive in males/females), give a regionplot where $h^{\delta} = h^{\varphi}$, e.g., $h^{\delta} =$
 1066 $h^{\varphi} = 0.75$ (or perhaps the value of h we use in the regionplots we have, in which
 1067 $w_{aa} = 0.85$, $w_{Aa} = 1$, $w_{AA} = 1.05$). Matt made a figure like this before but

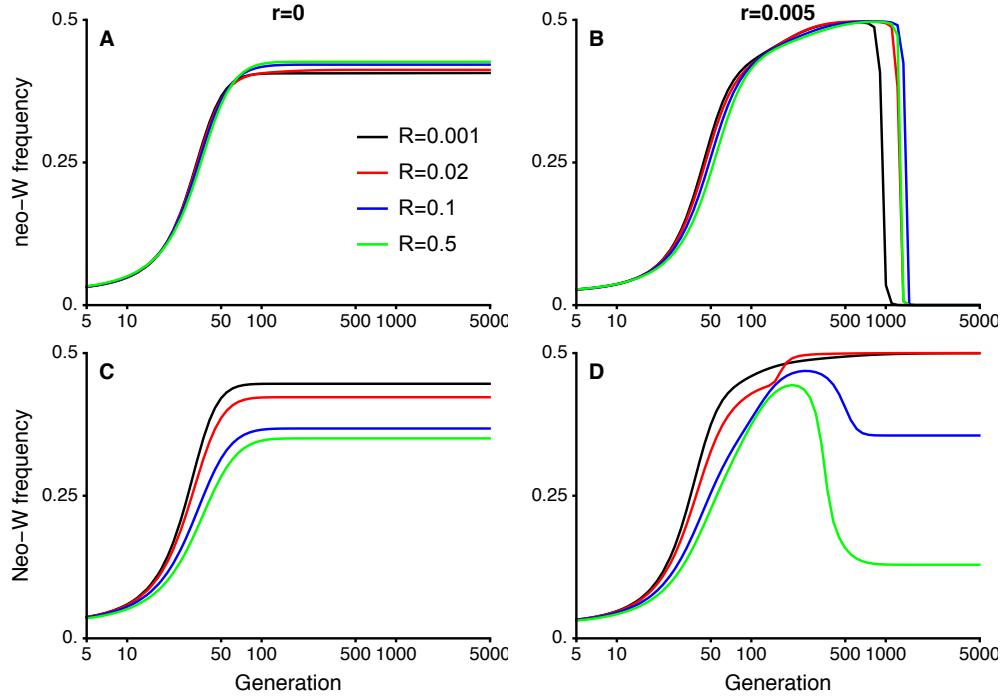


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among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

1068 both *Ya* and *YA* equilibria were plotted and there was no outline showing where
 1069 the *Ya* equilibrium is stable (as in Figure 2). In Matts plot the axes were s^δ and
 1070 α_Δ^δ . Add an asterisk to each region plot and show invasion in another panel, using
 1071 those parameters and various R (e.g., in the style of S.2). In an email, Sally has an
 1072 example of ploidally-antagonistic selection where the neo-W fixes and $R = 1/2$.

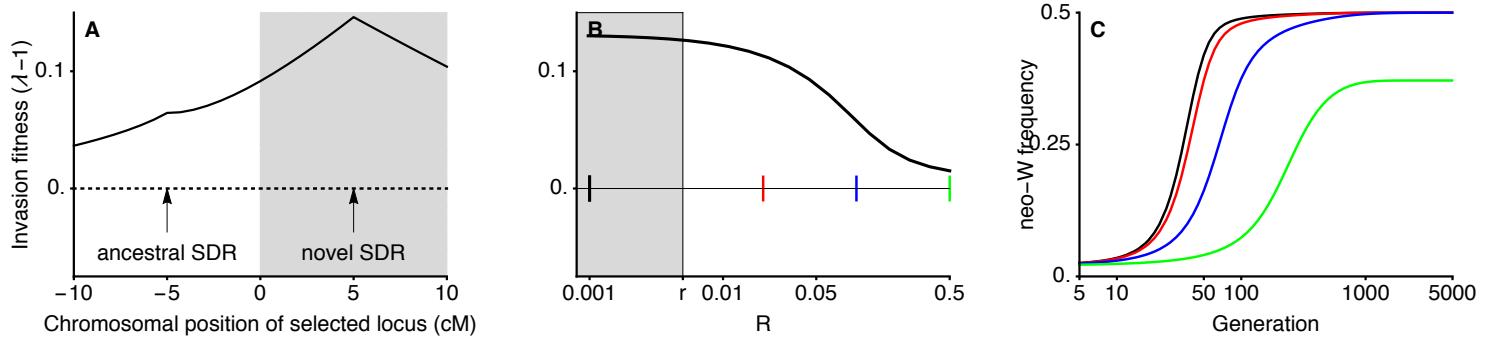


Figure S.3: When there is haploid selection and sexually-antagonistic selection, a neo-W may invade for any R . Check that we mention Sally's result that invasion cannot occur with sexually-antagonistic selection and $R = 1/2$ (currently only in legend for figure 1) Add asterisk to Figure S.5B to show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

This would cover that case and more.

1074 We could also give versions of Figure 2 where there is also haploid selection
 1075 of various types. Haploid selection can favour A or a , so this would involve 4x
 1076 6-panel figures. Started looking at this in Figure S.4 and Figure S.5, add female
 1077 haploid selection. Try to integrate into the discussion of haploid selection? e.g.,
 1078 male haploid selection ones generally show effect of sex ratio, increasing both
 lambdas when female biased (top rows).

1080 Perhaps, for one set of parameters, we should plot the dynamics of all the dif-
 1081 ferent alleles. E.g., we could use the same parameters used in 4. The main purpose
 1082 would be to show what happens to the ancestral SDR during turnover. We could
 1083 also show an example where XY and ZW sex determining systems are both poly-
 1084 morphic and stable (e.g., using one of the curves in Figure S.2 and the green curve
 1085 in Figure S.3). I think there are also examples with looser sex linkage and pollen
 1086 competition that lead to a mixed sex-determination system. We should probably
 have a short section in the appendix discussing this.

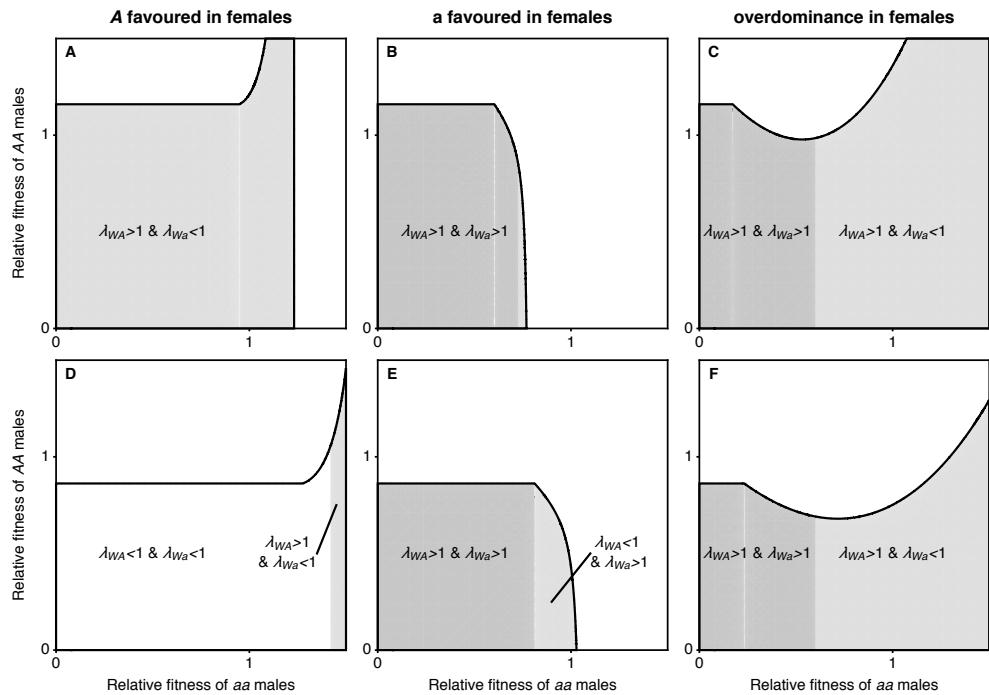


Figure S.4: ABC, $w_a^\delta = 1.16$, $w_A^\delta = 1$. DEF, $w_a^\delta = 1$, $w_A^\delta = 1.16$. I thought that re-running this with $w_a^\delta = 1.16$, $w_A^\delta = 0.84$ and $w_a^\delta = 0.84$, $w_A^\delta = 1.16$ will mean that it matches exactly with the meiotic drive example. Checking for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

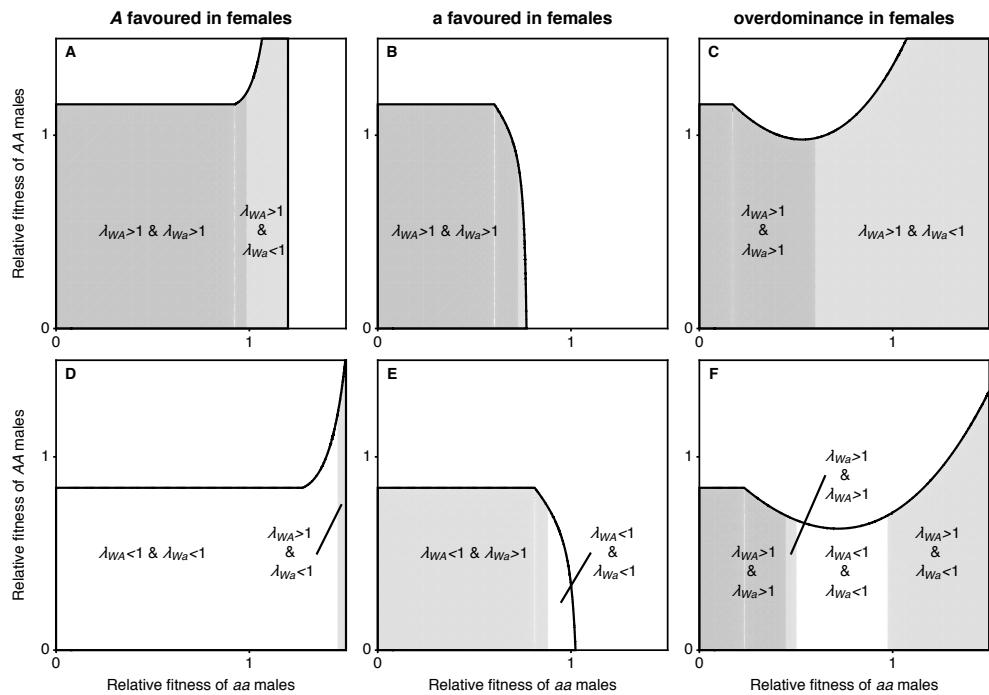


Figure S.5: ABC, $\alpha_{\Delta}^{\delta} = -0.08$ DEF, $\alpha_{\Delta}^{\delta} = 0.08$. Panel F mislabelled, should have $\lambda_{Wa} > 1$ & $\lambda_{WA} < 1$ as the upper label that has the line.

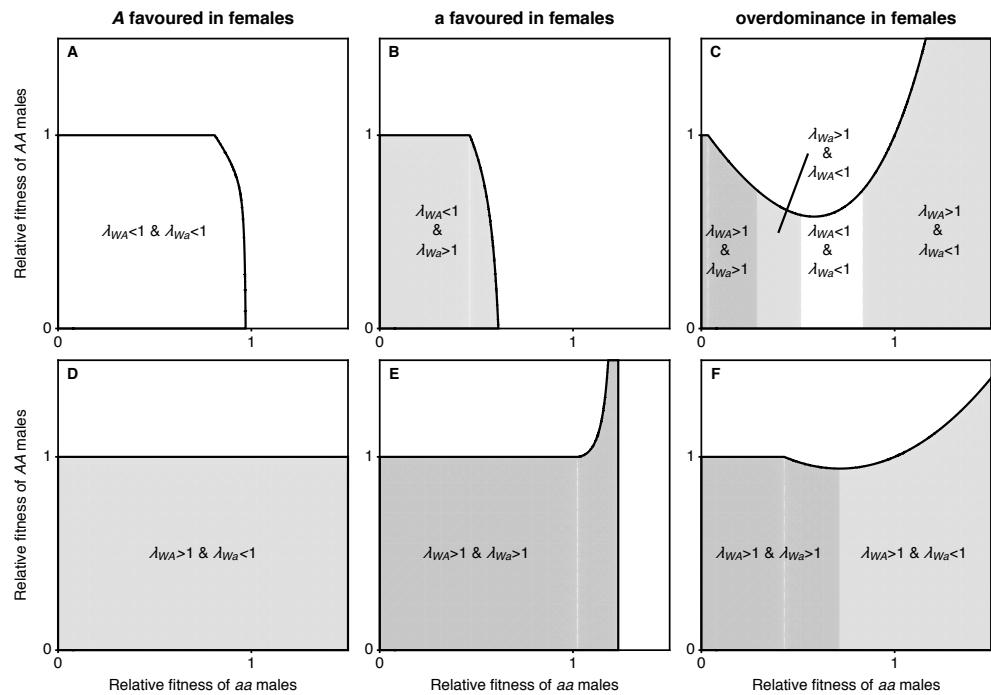


Figure S.6: ABC, $\alpha_{\Delta}^{\varnothing} = -0.08$ DEF, $\alpha_{\Delta}^{\varnothing} = 0.08$. λ s are increased for the haplotype that is favoured by female haploid selection, the stability conditions are also affected.