

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; a result that is not found
26 with loose sex-linkage. (2) We also consider selection upon haploid geno-
28 types 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 selection.
Haploid selection in the heterogametic sex can also cause sex ratio biases,
which may increase or decrease with the spread of new sex chromosomes.
Thus, transitions between sex-determination systems cannot be simply pre-
dicted by selection to equalise the sex-ratio. Overall, our models reveal
that transitions between sex-determination systems, particularly transitions
where the heterogametic sex changes, can be driven by loci in previously un-
expected genomic locations that experience selection during diploid and/or
haploid phases. These results might be reflected in the lability with which
sex-determination systems evolve.

Introduction

30 Animals and angiosperms exhibit extremely diverse sex-determination systems
31 (reviewed in Bull 1983, Charlesworth and Mank 2010, Beukeboom and Perrin
32 2014, Bachtrog et al. 2014). Among species with genetic sex determination of
33 diploid sexes, some taxa have heterogametic males (XY) and homogametic fe-
34 males (XX), including mammals and most dioecious plants (Ming et al. 2011);
35 whereas other taxa have homogametic males (ZZ) and heterogametic females (ZW),
36 including Lepidoptera and birds. Within several taxa, the chromosome that har-
37 bours the master sex-determining region changes. For example, transitions of the
38 master sex-determining gene between chromosomes or the evolution of new mas-
39 ter sex-determining genes have occurred in Salmonids (Li et al. 2011, Yano et al.
40 2012), Diptera (Vicoso and Bachtrog 2015), and *Oryzias* (Myosho et al. 2012). In
41 addition, many gonochoric clades with genetic sex determination exhibit transi-
42 tions between male (XY) and female (ZW) heterogamety, including snakes ([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 Switch between χ and ρ in all places because χ is used for double recombi-
149 nation events. I think we need a different variable given the ρ terms in the char-
150 acteristic polynomial. Also, where is χ used for double recombination? In other
151 papers? Here χ is the probability of an odd number of cross-overs between the
152 SDR and M loci.

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

In each generation, we census the genotype frequencies in male and female
170 gametes/gametophytes (hereafter gametes) before gametic competition. A full de-
171 scription of our model, including recursion equations, is given in the Appendix.
172 First, competition occurs among male gametes (sperm/pollen competition) and
173 among female gametes (egg/ovule competition) separately. Selection during ga-
174 metic competition depends on the **A** locus genotype, relative fitnesses are given
175 by w_A^φ and w_a^φ ($\varphi \in \{\varnothing, \delta\}$; see table 1). We assume that all gametes compete for
176 fertilization during gametic competition, which assumes a polygamous mating sys-

tem. Gametic competition in monogamous mating systems is, however, equivalent
 178 to meiotic drive in our model (described below), as both only alter the frequency
 of gametes produced by heterozygotes. After gametic competition, random mating
 180 occurs between male and female gametes. The resulting zygotes develop as males
 or females, depending on their genotypes at the **X** and **M** loci. Diploid males and
 182 females then experience selection, with relative fitnesses w_{AA}^φ , w_{Aa}^φ , and w_{aa}^φ . The
 next generation of gametes is produced by meiosis, during which recombination
 184 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
 186 **M** with probability R , and between loci **X** and **M** with probability χ . Any linear
 order of the loci can be modelled with appropriate choices of r , R , and χ (see Ta-
 188 ble S.1). Individuals that are heterozygous at the **A** locus may experience meiotic
 drive; a gamete produced by Aa heterozygotes of sex φ bear allele A with probabil-
 190 ity α^φ . Thus, the **A** locus can experience sex-specific gametic competition, diploid
 selection, and/or meiotic drive.

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

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 Aa heterozygotes
A	$\alpha^\varphi = 1/2 + \alpha_\Delta^\varphi / 2$
a	$1 - \alpha^\varphi = 1/2 - \alpha_\Delta^\varphi / 2$

¹⁹² **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 \mathbf{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 (\mathbf{X} locus). Thus, the invasion of rare dominant neo-Y or neo-W alleles is determined by the largest eigenvalue that solves a quadratic characteristic polynomial, **Mention the possibility that the other roots yield the leading eigenvalue somewhere.** $\lambda^2 + b\lambda + c = 0$. Here, $b = -(\lambda_{mA} + \lambda_{ma}) + (\rho_{mA} + \rho_{ma})$ and $c = (\lambda_{mA} - \rho_{mA})(\lambda_{ma} - \rho_{ma}) - \rho_{mA}\rho_{ma}$, where λ_{mi} is the multiplicative growth rate of mutant haplotypes on background $i \in \{A, a\}$, without accounting for loss due to recombination, and ρ_{mi} is the rate at which mutant haplotypes on background

220 $i \in \{A, a\}$ recombine onto the other **A** locus background in heterozygotes (see
 Table 2). The λ_{mi} and ρ_{mi} , and thus the spread of the mutant m allele, depend on
 222 the frequency of alleles at the **A** and **X** loci in the ancestral population. In the an-
 224 cestral population, it is convenient to follow the frequency of the A allele among
 female gametes (eggs), p_X^q , and among X-bearing, p_X^δ , and among Y-bearing, p_Y^δ ,
 male gametes (sperm/pollen). We also track the fraction of male gametes that are
 226 Y-bearing, q , which may deviate from 1/2 due to meiotic drive in males.

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

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

$\bar{p}^\delta = (1 - q)p_X^\delta + qp_Y^\delta$ 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

228

We are particularly concerned with the conditions under which a rare neo-sex-
 230 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 haplo-
 232 types 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
 234 ($\lambda_{mA}, \lambda_{ma} < 1$), 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
 236 invasion requires

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

For example, if we assume that only the mA haplotype has a positive growth rate
 238 ($\lambda_{ma} < 1 < \lambda_{mA}$), 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
 240 are produced by recombination is sufficiently large relative to that of ma haplo-
 types. In other words, invasion requires that the average growth rate of the two
 242 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
 244 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.,
 246 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)
 248 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
 250 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
 252 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.
 254 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
 256 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
 258 male (when paired with Y). Consequently, the types of females produced differ in
 the frequency of A alleles they obtain from mating.

260 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

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

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

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

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

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

tight linkage with the **A** locus, the ancestral Y becomes strongly specialized on the
292 allele that has the highest fitness across male haploid and diploid phases. Given
that the ancestral Y is at this equilibrium, it is not possible for a neo-Y to create
294 males that have higher fitness than the ancestral Y.

Neo-W alleles, on the other hand, can invade an ancestral XY system under
296 some conditions (the full invasion conditions are given in the appendix; equations
S.6 and S.7). That is, selection on loci within the non-recombining region of the
298 SDR can favour the invasion of a less closely linked neo-W, see Figure 1. This re-
sult is unexpected given the results of van Doorn and Kirkpatrick (2010), who did
300 not explicitly calculate equilibrium allele frequencies under tight linkage and gen-
erally concluded that heterogametic transitions occur when neo-sex-determining
302 alleles are in tighter linkage with loci under sex-specific diploid selection. To de-
velop an understanding (intuition) for how this happens, we focus on cases where
304 there is no haploid selection and discuss the effects of haploid selection in the
appendix.

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

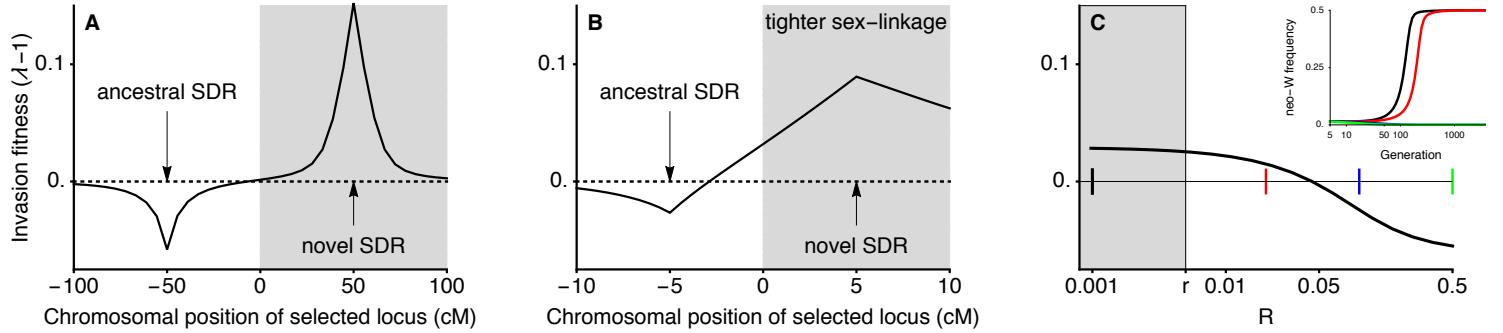


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is more loosely linked to a locus under sexually-antagonistic selection (here, without haploid selection $r^{\delta} = \alpha_{\Delta}^{\delta} = 0$). In panel A, linkage is loose enough relative to selection that the weak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

bination rate, R , is small enough. Nevertheless, because we assume here that r is
 322 small, these results indicate that a more loosely linked sex-determining region can
 spread. Therefore, tightly sex-linked loci that experience sexually-antagonistic se-
 324 lection 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
 326 it is fixed on the Y, it is surprising that neo-W- a haplotypes can sometimes be
 328 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
 330 a Y- a . Hence, if there is overdominance in males, X- A Y- a males have high fit-
 ness and the A allele is favoured by selection on the X in males. Therefore, the
 332 X can be polymorphic or even fixed for the A allele despite favouring the a allele

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

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

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

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

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

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

and

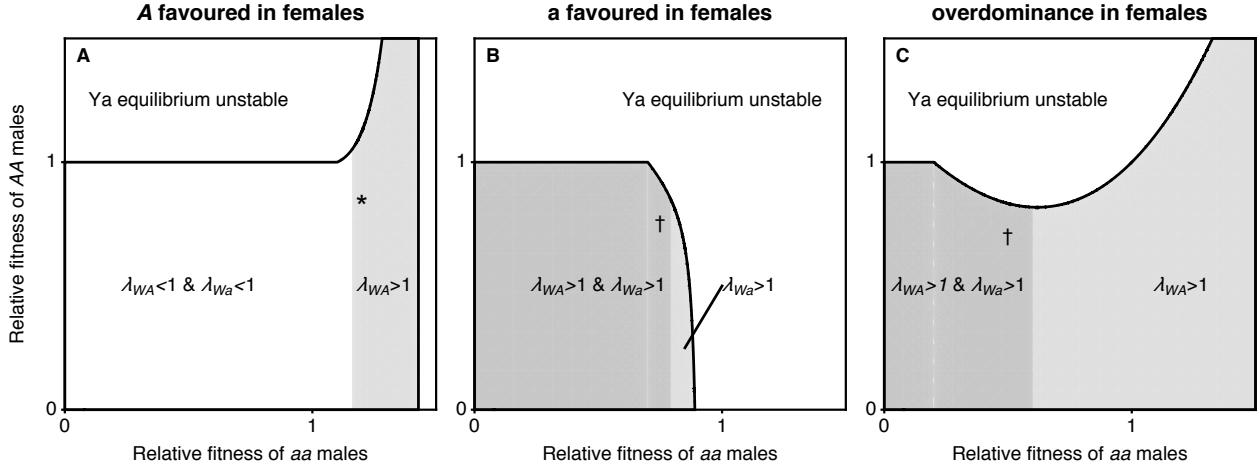


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ($r = 0$), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ($w_{Aa}^\delta = 1$) and only consider stable equilibria at which both A locus alleles are maintained 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 $t^\delta = \alpha_\Delta^\delta = 0$.

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

358 where $V_A = \bar{p}(1 - \bar{p})$ is the variance in the frequency of A and $S_A = (D^\delta + \alpha_\Delta^\delta + t^\delta) - (D^\varphi + \alpha_\Delta^\varphi + t^\varphi)$ describes sex differences in selection for the A versus a across
 360 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
 362 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).

364 The neo-sex-determining allele, m , will spread if $\lambda_{m,XY} > 1$. Equation (2)
 demonstrates that under weak selection a neo-Y will invade an XY system if and
 366 only if it is more closely linked to the selected locus than the ancestral sex-determining

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

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

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

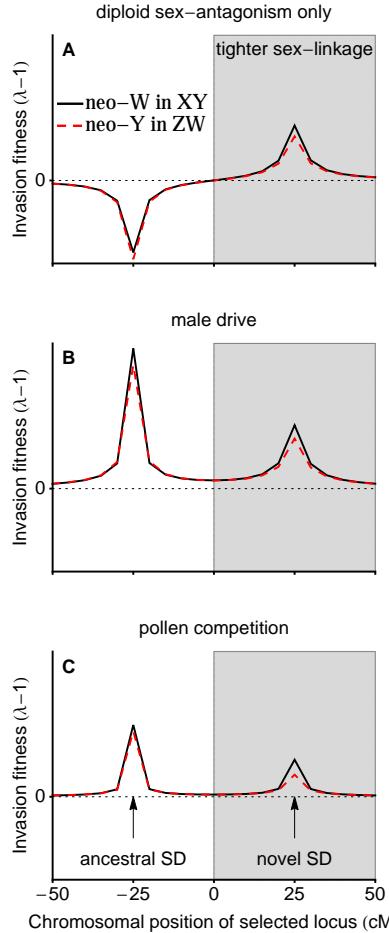


Figure 3: A neo-W can invade an XY system under a large number of selective regimes. In panel A, there is no haploid selection ($t^\delta = \alpha_\Delta^\delta = 0$) and selection in diploids is sexually antagonistic ($s^\delta = -s^q = 1/10$, $h^\delta = 1 - h^q = 3/10$), in which case the neo-sex-determining 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 ($t^\delta = -1/10$, $t^q = \alpha_\Delta^q = 0$) opposes selection in diploids (sex-differences: $s^\delta = 1/20$, $s^q = 3/20$, $h^\delta = 7/10$), in which case the neo-sex-determining allele can once again invade regardless of linkage. We use Haldane's map function (Equation 3 in Haldane 1919) to convert from map distance (centiMorgans, cM) to the probability of recombination (an odd number of cross-over events). **Check the mismatch between red and black lines here: probably because of adding or subtracting from 1.**

gametic competition and meiotic drive is in one sex only, an unlinked neo-W can
 398 invade as long as the same allele is favoured during diploid selection in males and females ($s^q s^\delta > 0$, see Figure 3B and Figure 4B). When there is no meiotic drive

400 and gametic competition occurs in one sex only, an unlinked neo-W can invade as
 long as the same allele is favoured in male and female diploid selection and there
 402 are sex differences in selection of one type (e.g., $s^{\varphi}(s^{\delta} - s^{\varphi}) > 0$, see Figure 3C).
 These special cases indicate that neo-W invasion can occur for a relatively large
 404 fraction of the parameter space, even if the neo-W uncouples the sex-determining
 locus from a locus under selection.

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

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

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

422 system where females are initially rare because the ancestral-Y is associated with
 423 a male meiotic drive allele. However, Figure 4A shows that a neo-Y can invade
 424 an ancestrally-ZW system under the same conditions. In fact, where $R < 1/2$ the
 425 neo-Y becomes associated with the male meiotic drive allele such that the zygotic
 426 sex ratio evolves to become biased towards males.

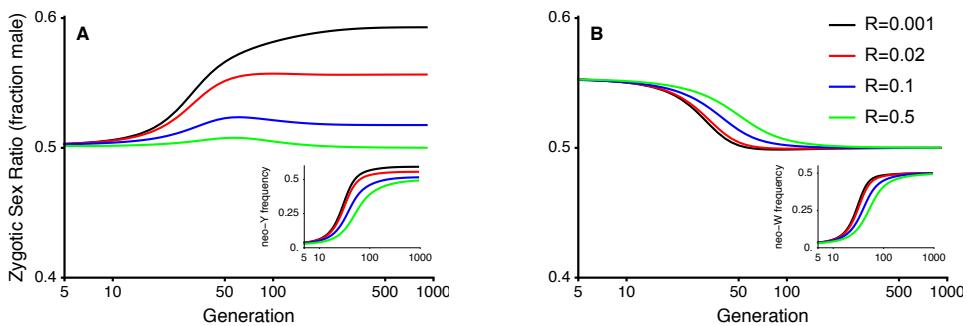


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidally antagonistic with haploid selection favouring the a allele during male meiosis ($s^{\varphi} = s^{\delta} = 0.2$, $h^{\varphi} = h^{\delta} = 0.7$, $t^{\varphi} = t^{\delta} = \alpha_{\Delta}^{\delta} = 0$). 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: $s^{\varphi} = s^{\delta} = 0.2$, $h^{\varphi} = h^{\delta} = 0.7$, $t^{\varphi} = t^{\delta} = \alpha_{\Delta}^{\delta} = 0$, $\alpha_{\Delta}^{\delta} = -0.2$, $r = 0.02$.

The green curves in Figure 4 demonstrate a case where transitions between
 428 male and female heterogamety occur even though the new sex-determining re-
 gion is unlinked to a locus that experiences haploid and diploid selection. We
 430 use these green curves to discuss why heterogametic transitions can occur when
 432 $R = 1/2$ and $r < 1/2$, as in Table 3. In Figure 4B, an unlinked neo-W can spread
 434 because the zygotic sex ratio is ancestrally male biased. However, in Figure 4A, an
 436 unlinked neo-Y spreads despite the fact that the ancestral zygotic sex ratio is even.
 In this case, the the male meiotic drive allele, a , is initially more common among
 ancestral-Z-bearing eggs than ancestral-W-bearing eggs because the Z is found in
 males more often than the W and $r < 1/2$ (equation S.5). Polymorphism at the **A**
 locus is maintained by counter-selection against the a allele in diploids and there-

438 fore ancestral-ZZ males have generally low diploid fitness. A freely recombining
 439 neo-Y ($R = 1/2$) is not directly favoured or disfavoured by male meiotic drive
 440 because it is equally likely to be segregate with the A or a allele when found in
 441 a heterozygote. The neo-Y spreads because it produces males with high diploid
 442 fitness through matings with ancestral-W-bearing female gametes, which are more
 443 likely to carry the A allele. Thus, a key factor in explaining why heterogametic
 444 transitions can occur when $R > r$ is that the neo-SDR determines sex in the
 445 diploid phase but recombination occurs before any subsequent haploid selection.

446 **Environmental sex determination**

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

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

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

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

462 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
 464 spread of which is given by

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

466 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
 and the novel sex-determining locus, R , doesn't enter into the $k = 1/2$ results. This
 468 is because sex is essentially randomized each generation, preventing associations
 from building up between allele A and sex. Equation (5) shows that the neo-ESD
 470 gets half of the fitness of a feminizing mutation (neo- W) and half of the fitness
 of a masculinizing mutation (neo- Y), but only has an effect one half of the time
 472 (the other half of the time it produces the same sex as the ancestral system would
 have, to leading order). As discussed above, $\lambda_{Y',XY|R=1/2}$ is necessarily less than
 474 one, but $\lambda_{W',XY|R=1/2}$ can be greater than one if there is haploid selection. That
 is, when there is haploid selection, ESD mutations can invade an ancestrally-XY
 476 system because they generate females that are either rare or have high fitness, in
 the same manner as a neo- W .

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

Discussion

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

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

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

528 We have shown that the spread of new sex determination systems can be driven
by loci experiencing haploid selection. Because haploid selection can cause tran-
530 sitions that increase or decrease sex-linkage, haploid selection may lead to less
stability, and greater potential for cycling, in sex-determination systems (e.g., the
532 final state of the red line in Figure 4A is the starting state in Figure 4B). In par-
ticular, if haploid selection is strong but selective differences between male and
534 female diploids are weak, we find that heterogametic transitions (XY to ZW or
vice versa) are favoured more strongly than homogametic transitions (e.g., with
536 $|D^\delta - D^\varnothing| << |\alpha_\Delta^\delta - \alpha_\Delta^\varnothing + t^\delta - t^\varnothing|$ we have $\lambda_{W',XY} > \lambda_{Y',XY}$; equations 3 and S.5).
Turnovers driven by haploid selection may help to explain the relative rarity of
538 heteromorphic sex chromosomes in plants, which are thought to experience more
selection during their multicellular haploid stage. For example, among relatively
540 few dioecious clades in which multiple species have well characterized sex chro-
mosomes (Ming et al. 2011), heterogametic transitions have been inferred in *Silene*
542 subsection *Otites* (Slancarova et al. 2013) and in *Salicaceae* (Pucholt et al. 2015,
2017). Furthermore, assuming that transitions from dioecy to hermaphroditism
544 (equal parental investment in male and female gametes) are favoured in a simi-
lar manner to the ESD examined here (equal probability of zygotes developing as
546 males or females), our results suggest that competition during the haploid stage

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

In support of their role in sex chromosome turnover, genes expected to be un-
550 der sexually-antagonistic selection (e.g., those causing bright male colouration)
have been found on recently derived sex chromosomes (Lindholm and Breden
552 2002, Tripathi et al. 2009, Ser et al. 2010). Our results show that, if loci ex-
periencing overdominance and/or sexually-antagonistic selection can be identi-
554 fied 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
556 heterogametic transitions between sex-determination systems. As noted by van
Doorn and Kirkpatrick (2010), it would be prudent to compare closely related
558 clades in order to determine whether observed polymorphisms predate a transi-
tion in sex-determination or arose afterwards. In addition, we show haploid se-
560 lection on loci around either the ancestral- or the novel-sex-determining regions
could have had a role in driving sex chromosome turnover. A recent transcrip-
562 tome analysis in *Rumex*, suggests a role for gametic competition in the evolution
of sex-determination systems, showing that Y-linked genes are have higher expres-
564 sion 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
566 sex chromosomes in closely related species suggesting that new sex chromosomes
may have been favoured through their association with haploid selected alleles on
568 these chromosomes (Sandler et al., 2017, Personal Communication).

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

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

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

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

Discuss polymorphic mating systems somewhere? Say that haploid selection makes this particularly likely (I think there are examples with gametic competition 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 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 pasted from from Vuilleumier et al. 2007 and vD&K, 2010, might be added to this section.

“Polygenic sex determination has been reported in many plants (e.g. Shannon & Holsinger 2007), fishes (Vandeputte et al. 2007; Ser et al. 2010; Liew et al. 2012), crustaceans (e.g. Battaglia 1958; Battaglia & Malesani 1959; Voordouw & Anholt 2002), bivalves (Haley 1977; Saavedra et al. 1997), gastropods (Yusa 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, 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 al., 2001; Ogata et al., 2003; Lee et al., 2004; Mank et al., 2006).” Also check

Kallman (1984) -from vD&K, 2010.

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824 **Appendix**

Recursion Equations

826 In each generation we census the genotype frequencies in male and female ga-
metes/gametophytes (hereafter, gametes) between meiosis (and any meiotic drive)
828 and gametic competition. At this stage we denote the frequencies of X- and Y-
bearing gametes from males and females x_i^\varnothing and y_i^\varnothing , where $\varnothing \in \{\delta, \Omega\}$ specifies
830 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
832 $1 = MA$, $2 = Ma$, $3 = mA$, and $4 = ma$. The gamete frequencies from each sex
sum to one, $\sum_i x_i^\varnothing + y_i^\varnothing = 1$.

834 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 ($w_1^\varnothing =$
836 $w_3^\varnothing = w_A^\varnothing$, $w_2^\varnothing = w_4^\varnothing = w_a^\varnothing$, see Table 1). The genotype frequencies after gametic
competition are $x_i^{\varnothing,s} = w_i x_i^\varnothing / \bar{w}_H^\varnothing$ and $y_i^{\varnothing,s} = w_i y_i^\varnothing / \bar{w}_H^\varnothing$, where $\bar{w}_H^\varnothing = \sum_i w_i x_i^\varnothing +$
838 $w_i y_i^\varnothing$ is the mean fitness of male ($\varnothing = \delta$) or female ($\varnothing = \Omega$) gametes.

840 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
842 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
844 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
846 these frequencies, $xx_{ij} = (x_i^{\varnothing,s} x_j^{\delta,s} + x_j^{\varnothing,s} x_i^{\delta,s})/2$ and $yy_{ij} = (y_i^{\varnothing,s} y_j^{\delta,s} + y_j^{\varnothing,s} y_i^{\delta,s})/2$.

848 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
850 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
852 $xy_{ij}^\delta = (1 - k_{bc}) xy_{ij}$, and YY males frequencies are $yy_{ij}^\delta = (1 - k_{bc}) yy_{ij}$. This

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

860 Selection among diploids then occurs according to the diploid genotype at the
A locus, $l \in \{AA, Aa, aa\}$, for an individual of type ij (see Table 1). The diploid
 862 frequencies after selection in sex $\hat{\varphi}$ are given by $xx_{ij}^{\hat{\varphi},s} = w_l^{\hat{\varphi}} xx_{ij}/\bar{w}^{\hat{\varphi}}$, $xy_{ij}^{\hat{\varphi},s} =$
 864 $w_l^{\hat{\varphi}} xy_{ij}/\bar{w}^{\hat{\varphi}}$, and $yy_{ij}^{\hat{\varphi},s} = w_l^{\hat{\varphi}} yy_{ij}/\bar{w}^{\hat{\varphi}}$, where $\bar{w}^{\hat{\varphi}} = \sum_{i=1}^4 \sum_{j=1}^4 w_l^{\hat{\varphi}} xx_{ij} + w_l^{\hat{\varphi}} xy_{ij} +$
 $w_l^{\hat{\varphi}} yy_{ij}$ is the mean fitness of individuals of sex $\hat{\varphi}$.

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

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

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

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

⁸⁷⁶ competition) in the next generation are given by

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

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

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

$$\begin{aligned}
x_4^{\phi'} = & xx_{44}^{\phi,s} + xx_{34}^{\phi,s}/2 + (xx_{14}^{\phi,s} + xx_{24}^{\phi,s})\alpha^\phi \\
& - R(xx_{14}^{\phi,s} - xx_{23}^{\phi,s})\alpha^\phi \\
& (xy_{44}^{\phi,s} + xy_{42}^{\phi,s})/2 + (xy_{41}^{\phi,s} + xy_{43}^{\phi,s})(1 - \alpha^\phi) \\
& - r(xy_{43}^{\phi,s} - xy_{34}^{\phi,s})(1 - \alpha^\phi) - \chi(xy_{42}^{\phi,s} - xy_{24}^{\phi,s})/2 \\
& + \left\{ -(R + r + \chi)xy_{41}^{\phi,s} + (r + \chi - R)xy_{14}^{\phi,s} \right. \\
& \left. + (R + r - \chi)xy_{32}^{\phi,s} + (R + \chi - r)xy_{23}^{\phi,s} \right\} (1 - \alpha^\phi)/2
\end{aligned} \tag{S.1d}$$

$$\begin{aligned}
y_1^{\phi'} = & yy_{11}^{\phi,s} + yy_{13}^{\phi,s}/2 + (yy_{12}^{\phi,s} + yy_{14}^{\phi,s})\alpha^\phi \\
& - R(yy_{14}^{\phi,s} - yy_{23}^{\phi,s})\alpha^\phi \\
& (xy_{11}^{\phi,s} + xy_{31}^{\phi,s})/2 + (xy_{21}^{\phi,s} + xy_{41}^{\phi,s})\alpha^\phi \\
& - r(xy_{21}^{\phi,s} - xy_{12}^{\phi,s})\alpha^\phi - \chi(xy_{31}^{\phi,s} - xy_{13}^{\phi,s})/2 \\
& + \left\{ -(R + r + \chi)xy_{41}^{\phi,s} + (r + \chi - R)xy_{14}^{\phi,s} \right. \\
& \left. + (R + r - \chi)xy_{32}^{\phi,s} + (R + \chi - r)xy_{23}^{\phi,s} \right\} \alpha^\phi/2
\end{aligned} \tag{S.1e}$$

$$\begin{aligned}
y_2^{\phi'} = & yy_{22}^{\phi,s} + yy_{24}^{\phi,s}/2 + (yy_{12}^{\phi,s} + yy_{23}^{\phi,s})\alpha^\phi \\
& - R(yy_{23}^{\phi,s} - yy_{14}^{\phi,s})\alpha^\phi \\
& (xy_{22}^{\phi,s} + xy_{42}^{\phi,s})/2 + (xy_{12}^{\phi,s} + xy_{32}^{\phi,s})(1 - \alpha^\phi) \\
& - r(xy_{12}^{\phi,s} - xy_{21}^{\phi,s})(1 - \alpha^\phi) - \chi(xy_{42}^{\phi,s} - xy_{24}^{\phi,s})/2 \\
& + \left\{ -(R + r + \chi)xy_{32}^{\phi,s} + (r + \chi - R)xy_{23}^{\phi,s} \right. \\
& \left. + (R + r - \chi)xy_{41}^{\phi,s} + (R + \chi - r)xy_{14}^{\phi,s} \right\} (1 - \alpha^\phi)/2
\end{aligned} \tag{S.1f}$$

$$\begin{aligned}
y_3^{\phi'} = & yy_{33}^{\phi,s} + yy_{13}^{\phi,s}/2 + (yy_{23}^{\phi,s} + yy_{34}^{\phi,s})\alpha^\phi \\
& - R(yy_{23}^{\phi,s} - yy_{14}^{\phi,s})\alpha^\phi \\
& (xy_{33}^{\phi,s} + xy_{13}^{\phi,s})/2 + (xy_{23}^{\phi,s} + xy_{43}^{\phi,s})\alpha^\phi \\
& - r(xy_{43}^{\phi,s} - xy_{34}^{\phi,s})\alpha^\phi - \chi(xy_{13}^{\phi,s} - xy_{31}^{\phi,s})/2 \\
& + \left\{ -(R + r + \chi)xy_{23}^{\phi,s} + (r + \chi - R)xy_{32}^{\phi,s} \right. \\
& \left. + (R + r - \chi)xy_{14}^{\phi,s} + (R + \chi - r)xy_{41}^{\phi,s} \right\} \alpha^\phi/2
\end{aligned} \tag{S.1g}$$

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

878 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
880 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
882 ($x_3^{\delta} = x_4^{\delta} = y_4^{\delta} = y_3^{\delta} = y_i^{\delta} = 0$). In this case, the system only involves six recursion
equations, which we assume below to calculate the equilibria.

884 **Resident equilibrium and stability**

In the resident population (allele *M* fixed), we follow the frequency of *A* in X-
886 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
888 may deviate from 1/2 due to meiotic drive in males. These four variables deter-
mine the frequencies of the six resident gamete types: $x_1^{\varphi} = p_X^{\varphi}$, $x_2^{\varphi} = 1 - p_X^{\varphi}$,
890 $x_1^{\delta} = (1 - q)p_X^{\delta}$, $x_2^{\delta} = (1 - q)(1 - p_X^{\delta})$, $y_1^{\delta} = qp_Y^{\delta}$, and $y_2^{\delta} = q(1 - p_Y^{\delta})$. Mean
fitnesses in the resident population are given in table S.2.

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

896 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)	$q [p_Y^{\delta} w_A^{\delta} + (1 - p_Y^{\delta}) w_a^{\delta}] / \bar{w}_H^{\delta}$

ability can be calculated analytically without assuming anything about the relative strengths of selection and recombination. However, here, we focus on two regimes (tight linkage and weak selection) in order to make fewer assumptions about fitnesses.

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

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

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

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

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

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

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

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

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

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

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

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

A fifth equilibrium (*C*) also exists where *A* is present at an intermediate frequency

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

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

properties are unaffected by small amounts of recombination between the SDR
922 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 $\bar{w}_{Y_a}^\delta = w_a^\delta [p_X^\delta (1 -$
924 $\alpha_\Delta^\delta) w_A^\delta w_{Aa}^\delta + (1 - p_X^\delta) w_a^\delta w_{aa}^\delta]$ and $\bar{w}_{YA}^\delta = w_A^\delta [p_X^\delta w_A^\delta w_{AA}^\delta + (1 - p_X^\delta) (1 + \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta]$.
That is, Y-*a* haplotypes must have higher fitness than Y-*A* haplotypes. Substituting
926 in $p_X^\delta = \hat{p}_X^\delta$ 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 equilib-
928 rium (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 $\phi > 0$
930 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 $w_A^\delta w_{AA}^\delta > (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta$.

932 **check last condition and the stability condition below are correct**

Selection weak relative to recombination (weak selection)

934 Here, we assume that selection and meiotic drive are weak relative to recombina-
tion ($s^\varphi, t^\varphi, \alpha_\Delta^\varphi$ of order ϵ). The maintenance of a polymorphism at the A locus
936 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.

938 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 ($\hat{p}_X^\varphi = \hat{p}_X^\delta = \hat{p}_Y^\delta = \bar{p}$) and
940 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 $O(\epsilon)$:

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

942 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
944 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
946 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
948 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
950 (2007).

Invasion conditions

952 **Cover the other parts of the characteristic polynomial here.**

A rare neo-Y or neo-W will spread from a given ancestral equilibrium when
954 the leading eigenvalue, λ , of the Jacobian matrix derived from the eight mutant
recursion equations (given by S.1c,d,g,h), evaluated at the ancestral equilibrium, is
956 greater than one. However, because a neo-Y (neo-W) is always in males (females)
and is epistatically dominant to the ancestral sex-determining locus, we need only
958 two recursion equations (e.g., tracking the change in the frequency of neo-Y- A and
neo-Y- a gametes from males) and thus the leading eigenvalue is the largest solution
960 to a quadratic characteristic polynomial $\lambda^2 + b\lambda + c = 0$ as described in the text
(Table 2).

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

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

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

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

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

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

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

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

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

Haploid selection impacts the spread of neo-W haplotypes in three ways. Firstly, 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 meiotic drive in males. Specifically, at equilibrium (B), the sex ratio is $\zeta = w_a^\delta(1 - \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ where $2\bar{w}_H^\delta = [w_a^\delta(1 - \alpha_\Delta^\delta) + w_A^\delta(1 + \alpha_\Delta^\delta)]$ has been canceled out in 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 develop as the rarer, female, sex.

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

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

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

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

If selection doesn't uniformly favour A in females, however, neo-W- A haplotypes and/or neo-W- a haplotypes can spread ($\lambda_{mA} > 1$ and/or $\lambda_{ma} > 1$) at this 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

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 (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 (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 ($\lambda_{ma} > 1$) if there is sufficiently strong underdominance in females ($w_{aa}^\varphi > w_{Aa}^\varphi$) [this is describing directional selection, not overdominance - check the conditions 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 Aa to AA genotypes) and yet the neo-W can spread with a because it produces female aa individuals by capturing Y- a haplotypes.

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

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

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

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

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

where

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

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

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

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

1028 As with equilibrium (B), haploid selection again modifies invasion fitnesses
 1030 by altering the sex-ratio and the diploid genotypes of females and directly select-
 1031 ing upon female gametes. The only difference is that resident XX females are no
 longer always homozygote *AA* and males are no longer always heterozygote *Aa*.
 1032 Thus the effect of haploid selection in males is reduced, as is the difference in fit-
 1033 ness between neo-W haplotypes and resident X haplotypes, as both can be on any
 1034 diploid or haploid background.

The other terms are easier to interpret in the absence of haploid selection. For
 1035 instance, without haploid selection, the neo-W-*A* haplotype spreads ($\lambda_{mA} > 1$) if
 1036 and only if

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

1037 where $\phi - \psi = w_{AA}^\varphi w_{Aa}^\delta - w_{aa}^\varphi w_{aa}^\delta$ and both ϕ and ψ are positive when equilibrium
 1038 (A) is stable. In contrast to equilibrium (B), a neo-W haplotype can spread under
 1039 purely sexually-antagonistic selection ($w_{aa}^\varphi < w_{Aa}^\varphi < w_{AA}^\varphi$ and $w_{AA}^\delta < w_{Aa}^\delta < w_{aa}^\delta$).
 1040 The neo-W-*A* can spread as long as it becomes associated with females that bear
 1041 more *A* alleles than observed at equilibrium (A).

Without haploid selection, the neo-W-*a* haplotype spreads ($\lambda_{ma} > 1$) if and
 1042 only if

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

This condition cannot be met with purely sexually antagonistic selection (as both
 1046 terms on the left-hand side would then be negative), but it can be met under other
 circumstances. For example, with overdominance in males there is selection for
 1048 increased *A* frequencies on X chromosomes in males, which are always paired
 with Y-*a* haplotypes. Directional selection for *a* in females can then maintain a
 1050 polymorphism at the A locus on the X. This scenario selects for a modifier that
 increases recombination between the sex chromosomes (e.g., blue region of Figure
 1052 2d in Otto 2014) and facilitates the spread of neo-W-*a* haplotypes, which create
 more females bearing more *a* alleles than the ancestral X chromosome does.

1054 **Supplementary Figures**

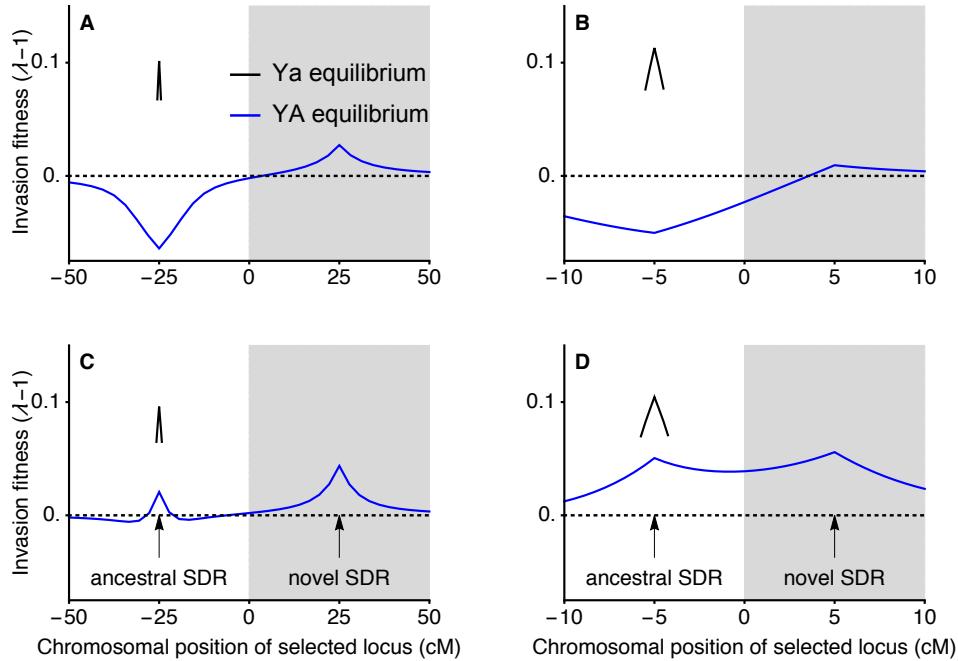


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ($r \approx 0$). In panels A and B, the a allele is favoured in females ($w_{aa}^{\text{♀}} = 1.05$, $w_{Aa}^{\text{♀}} = 1$, $w_{AA}^{\text{♀}} = 0.85$) and selection in males is overdominant ($w_{aa}^{\text{♂}} = w_{AA}^{\text{♂}} = 0.75$). In panels C and D, selection in males and females is overdominant ($w_{aa}^{\text{♀}} = w_{AA}^{\text{♀}} = 0.6$, $w_{Aa}^{\text{♀}} = 0.5$, $w_{AA}^{\text{♂}} = 0.7$, $w_{Aa}^{\text{♂}} = 1$). 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 $r^{\text{♂}} = \alpha_{\Delta}^{\text{♂}} = 0$.

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

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

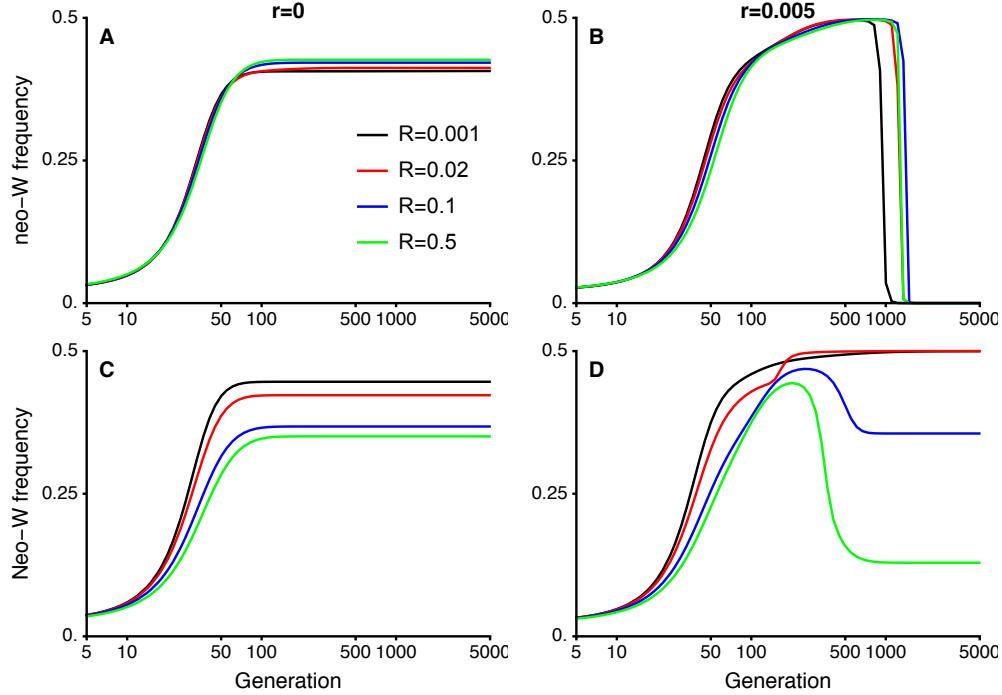


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among 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 (*Ya* is fixed when $r = 0$). When $r > 0$ (panels B and D), *YA* haplotypes created by recombination can become more common than *Ya* haplotypes as the neo-W spreads. In B, this leads to loss of the neo-W and the system goes to an equilibrium with *Xa* and *YA* 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 *YA* haplotype is ancestrally more common than *Ya* (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 ($w_{aa}^{\varphi} = 1.05$, $w_{Aa}^{\varphi} = 1$, $w_{AA}^{\varphi} = 0.85$) 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 ($w_{aa}^{\varphi} = w_{AA}^{\varphi} = 0.6$, $w_{aa}^{\delta} = 0.5$, $w_{AA}^{\delta} = 0.7$, $w_{Aa}^{\delta} = 1$). These parameters are marked by a dagger in Figure 2. Here, there is no haploid selection $t^{\varphi} = \alpha_{\Delta}^{\varphi} = 0$.

both *Ya* and *YA* equilibria were plotted and there was no outline showing where
 1064 the *Ya* equilibrium is stable (as in Figure 2). In Matts plot the axes were s^{φ} and
 $\alpha_{\Delta}^{\varphi}$. Add an asterisk to each region plot and show invasion in another panel, using
 1066 those parameters and various R (e.g., in the style of S.2). In an email, Sally has an
 example of ploidally-antagonistic selection where the neo-W fixes and $R = 1/2$.

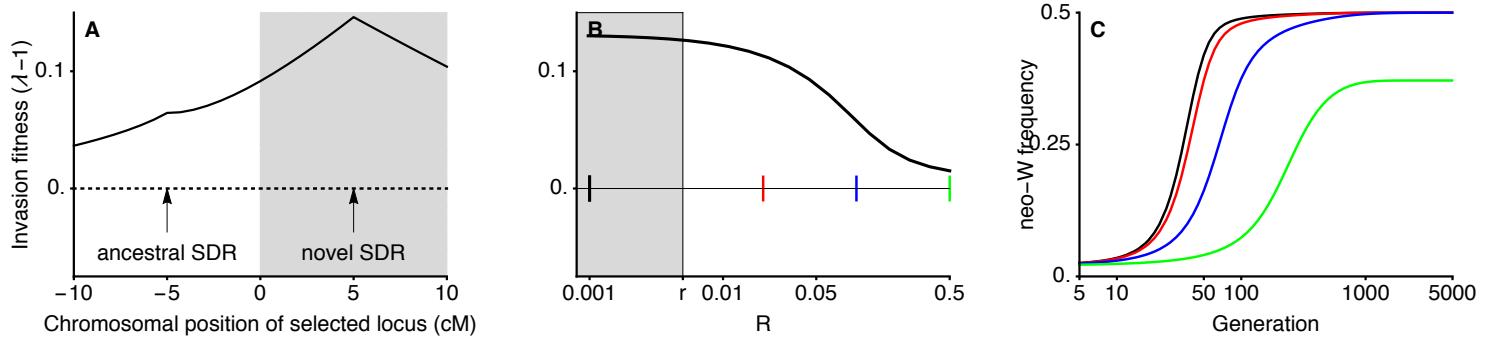


Figure S.3: When there is haploid selection and sexually-antagonistic selection, a neo-W may invade for any R . Check that we mention Sally's result that invasion cannot occur with sexually-antagonistic selection and $R = 1/2$ (currently only in legend for figure 1) Add asterisk to Figure S.5B to show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

1068 This would cover that case and more.

We could also give versions of Figure 2 where there is also haploid selection
 1070 of various types. Haploid selection can favour A or a , so this would involve 4x
 1072 6-panel figures. Started looking at this in Figure S.4 and Figure S.5, add female
 1074 haploid selection. Try to integrate into the discussion of haploid selection? e.g.,
 male haploid selection ones generally show effect of sex ratio, increasing both
 lambdas when female biased (top rows).

Perhaps, for one set of parameters, we should plot the dynamics of all the dif-
 1076 ferent alleles. E.g., we could use the same parameters used in 4. The main purpose
 would be to show what happens to the ancestral SDR during turnover. We could
 1078 also show an example where XY and ZW sex determining systems are both poly-
 morphic and stable (e.g., using one of the curves in Figure S.2 and the green curve
 1080 in Figure S.3). I think there are also examples with looser sex linkage and pollen
 competition that lead to a mixed sex-determination system. We should probably
 1082 have a short section in the appendix discussing this.

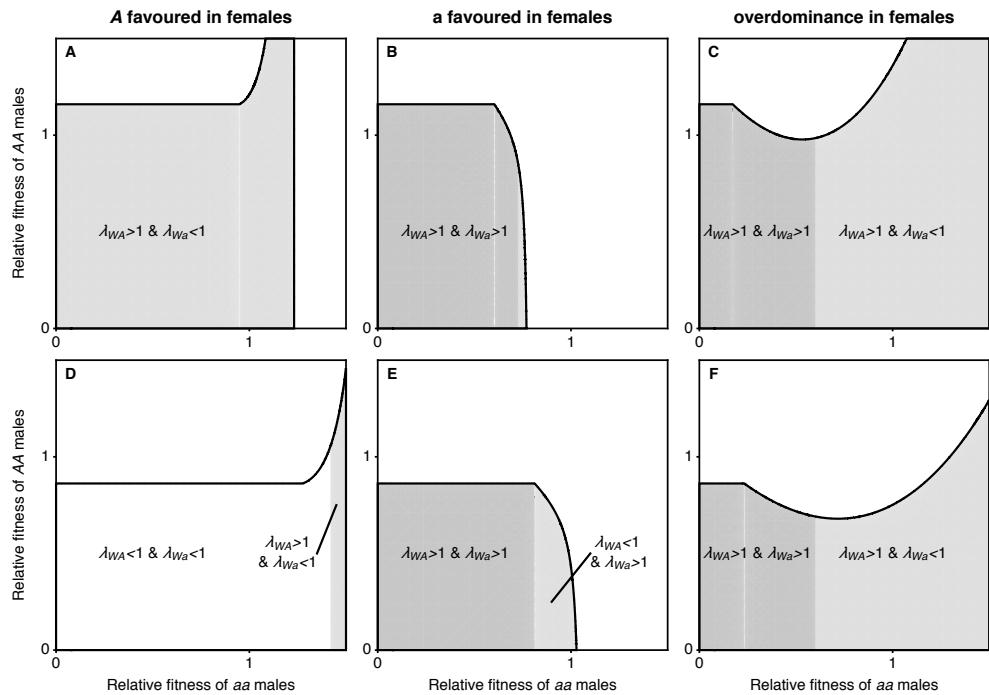


Figure S.4: ABC, $w_a^\delta = 1.16$, $w_A^\delta = 1$. DEF, $w_a^\delta = 1$, $w_A^\delta = 1.16$. I thought that re-running this with $w_a^\delta = 1.16$, $w_A^\delta = 0.84$ and $w_a^\delta = 0.84$, $w_A^\delta = 1.16$ will mean that it matches exactly with the meiotic drive example. Checking for 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.

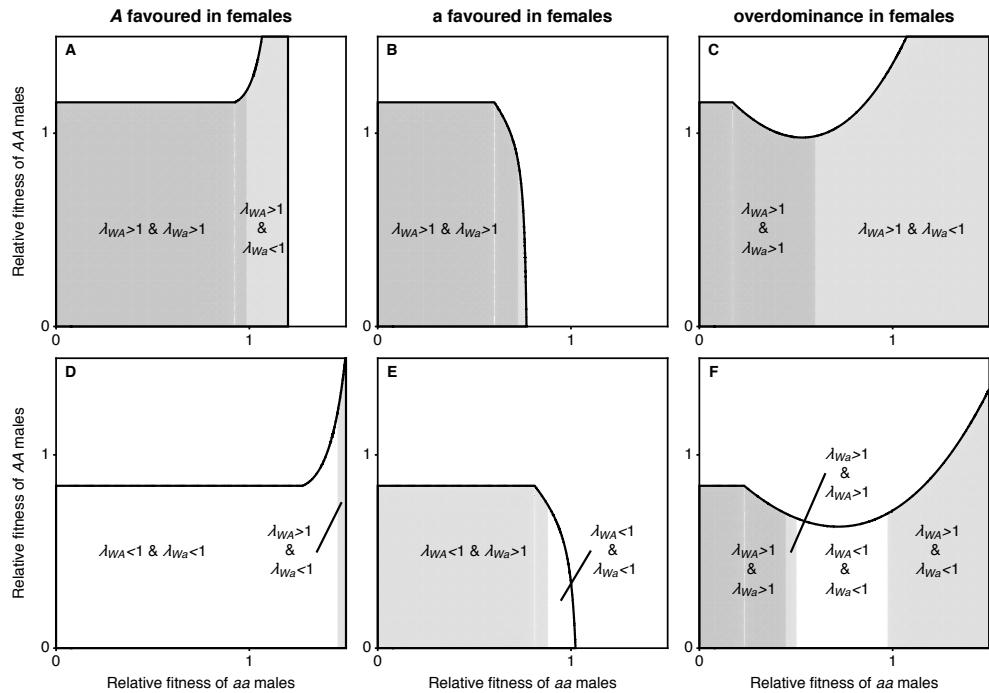


Figure S.5: ABC, $\alpha_{\Delta}^{\delta} = -0.08$ DEF, $\alpha_{\Delta}^{\delta} = 0.08$. Panel F mislabelled, should have $\lambda_{Wa} > 1$ & $\lambda_{WA} < 1$ as the upper label that has the line.

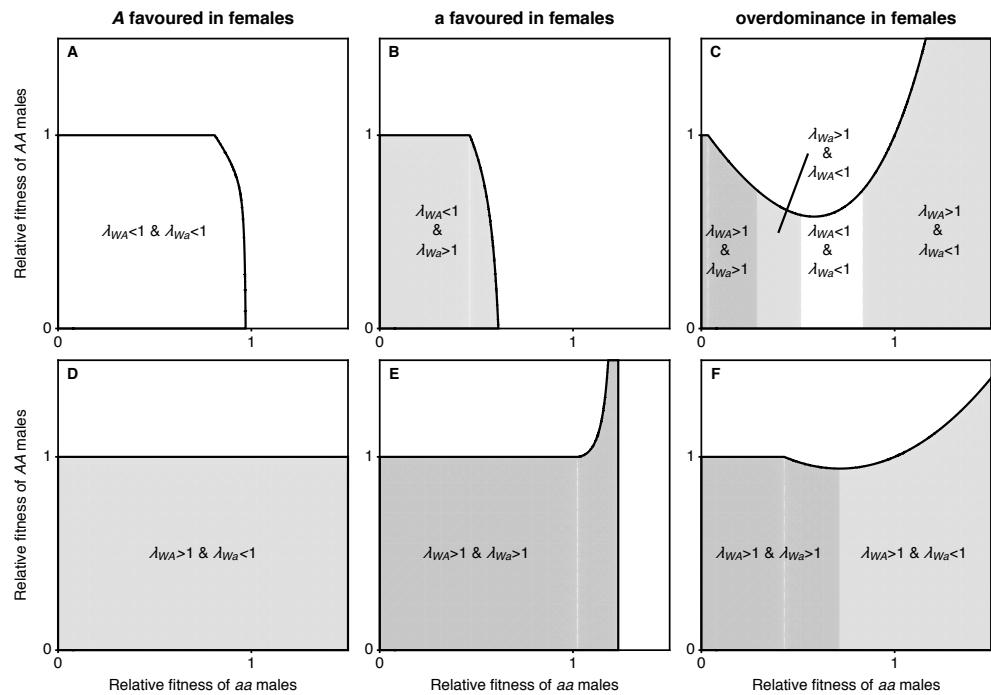


Figure S.6: ABC, $\alpha_{\Delta}^{\varnothing} = -0.08$ DEF, $\alpha_{\Delta}^{\varnothing} = 0.08$. λ s are increased for the haplotype that is favoured by female haploid selection, the stability conditions are also affected.