

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

Michael F Scott*¹, Matthew M Osmond*², and Sarah P Otto²

* These authors contributed equally to this work

¹ Department of Botany, University of British Columbia, #3529 - 6270 University Boulevard, Vancouver, BC, Canada V6T 1Z4

² Department of Zoology, University of British Columbia, #4200 - 6270 University Boulevard, Vancouver, BC, Canada V6T 1Z4

email: mfscott@biodiversity.ubc.ca, mmosmond@zoology.ubc.ca

Contributions:

Abstract

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

Introduction

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

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

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

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

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

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

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

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

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

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

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

Model

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

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

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

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

W_{AA} .

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

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

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

196 **Results**

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

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

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

Generic invasion by a neo-Y or neo-W

206 The evolution of a new sex-determination system requires that a rare mutant allele at the novel sex-determining locus, m , increases in frequency when rare. The
 208 spread of a rare mutant m at the **M** locus is determined by the leading eigenvalue,
 210 λ , of the system of eight equations describing the frequency of eggs and sperm
 212 carrying the m allele in the next generation (equations S.1). This system simplifies
 214 substantially in a number of cases of interest. Dominant neo-Y (when $k = 0$)
 216 or neo-W alleles (when $k = 1$) are only found in male diploids (neo-Y) or female
 218 diploids (neo-W) such that their growth rate ultimately depends only on the
 220 change in frequency of m -bearing gametes produced by males or by females, respectively.
 Furthermore, if the m allele is fully epistatically dominant over the ancestral sex-determining system, phenotypes are not affected by the genotype at the ancestral sex-determining region (**X** locus). Thus, the invasion of rare dominant neo-Y or neo-W alleles is determined by the largest eigenvalue that solves a quadratic characteristic polynomial, **Mention the possibility that the other roots yield the leading eigenvalue somewhere.** following the logic above, it seems like

we say we go from 8 eqns to 4 eqns by considering only one sex, and then from 4
 222 eqns to 2 eqns by ignoring the ancestral SD allele. why then do we have the two
 quadratic problem? $\lambda^2 + b\lambda + c = 0$. Here, $b = -(\lambda_{mA} + \lambda_{ma}) + (\chi_{mA} + \chi_{ma})$ and
 224 $c = (\lambda_{mA} - \chi_{mA})(\lambda_{ma} - \chi_{ma}) - \chi_{mA}\chi_{ma}$, where λ_{mi} is the multiplicative growth rate
 of mutant haplotypes on background $i \in \{A, a\}$, without accounting for loss due
 226 to recombination, and χ_{mi} is the rate at which mutant haplotypes on background
 $i \in \{A, a\}$ recombine onto the other A locus background in heterozygotes (see
 228 Table 2). The λ_{mi} and χ_{mi} , and thus the spread of the mutant m allele, depend on
 the frequency of alleles at the A and X loci in the ancestral population. In the an-
 230 cestral population, it is convenient to follow the frequency of the A allele among
 female gametes (eggs), p_X^{\varnothing} , and among X-bearing, p_X^{δ} , and among Y-bearing, p_Y^{δ} ,
 232 male gametes (sperm/pollen). We also track the fraction of male gametes that are
 Y-bearing, q , which may deviate from 1/2 due to meiotic drive in males. We will
 234 consider only equilibrium frequencies of alleles, \hat{p}_i^{\varnothing} , and Y-bearing male gametes,
 \hat{q} , to ensure the eigenvalues of the invasion analysis are valid.

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

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

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

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

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

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

For example, if we assume that only the mA haplotype has a positive growth rate ($\lambda_{ma} < 1 < \lambda_{mA}$), the second term on the left-hand side of (1) is negative and

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

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

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

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

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

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

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

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

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

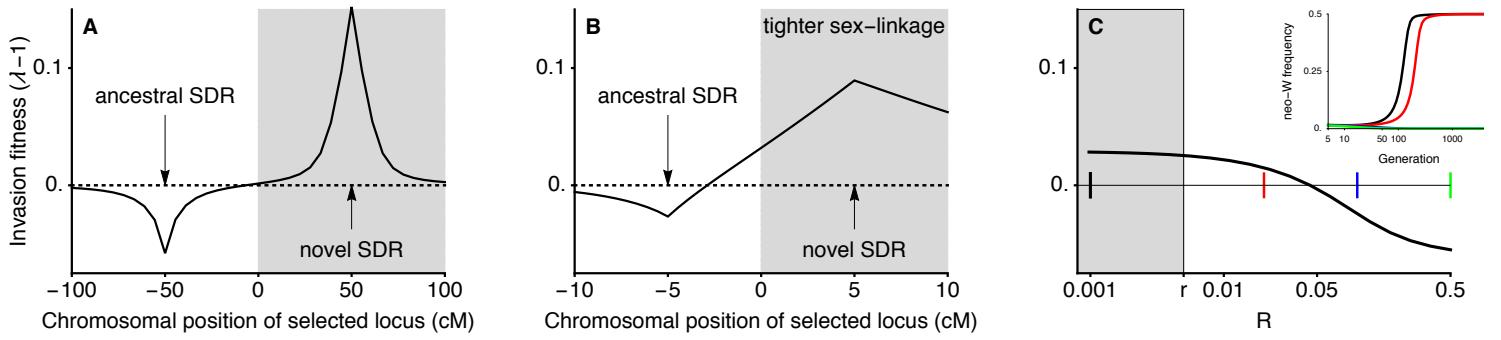


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is more loosely linked to a locus under sexually-antagonistic selection (here, without haploid selection $r^{\delta} = \alpha_{\Delta}^{\delta} = 0$). In panel A, linkage is loose enough relative to selection that the weak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

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

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

334 Given that the a allele can be considered ancestrally ‘male-beneficial’ because
it is fixed on the Y, it is surprising that neo-W- a haplotypes can sometimes be
336 favoured by selection in females ($\lambda_{ma} > 1$). Again, this occurs because ancestral
X’s also experience selection in males, in which they will always be paired with
338 a Y- a . Hence, if there is overdominance in males, X- A Y- a males have high fit-
ness and the A allele is favoured by selection on the X in males. Therefore, the
340 X can be polymorphic or even fixed for the A allele despite favouring the a allele
during selection in females (e.g., see outlined region in Figure 2B and Lloyd and
342 Webb 1977, Otto 2014). In such cases, neo-W- a haplotypes can spread because
they create more Aa and aa females when pairing with an X from males and be-
344 cause they bring Y- a haplotypes into females, in which case females are always aa .
As discussed in the appendix, this scenario where neo-W’s associated with a are
346 favoured can also occur with haploid selection, even without overdominance (e.g.,
when a is female-beneficial and favoured by haploid selection in male gametes).

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

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

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

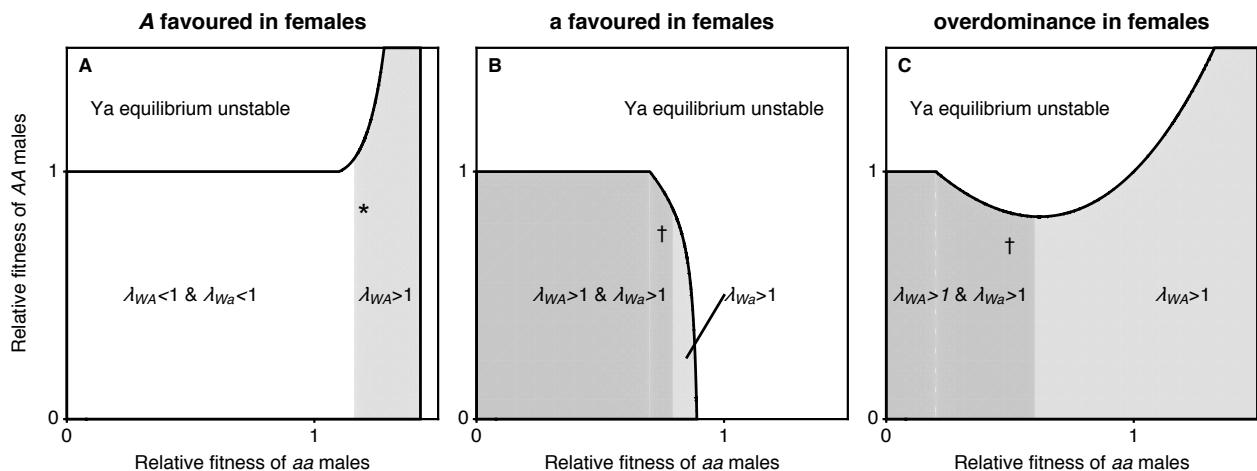


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ($r = 0$), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ($w_{Aa}^\varphi = 1$) and only consider stable equilibria at which both *A* locus alleles are maintained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

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

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

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

and

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

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

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

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

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

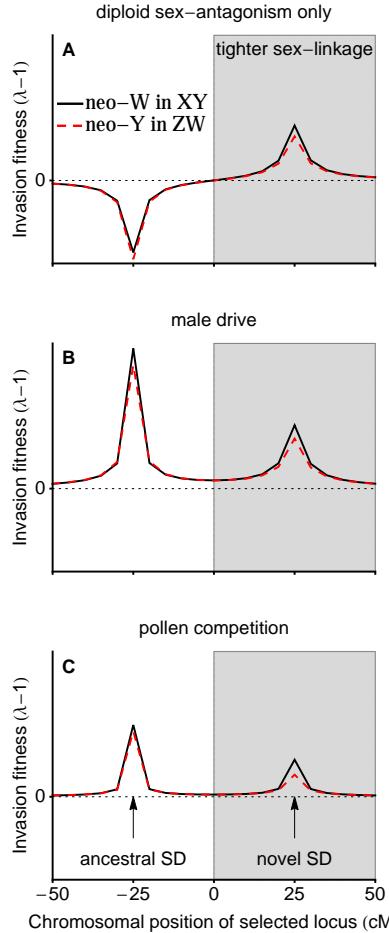


Figure 3: A neo-W can invade an XY system under a large number of selective regimes. In panel A, there is no haploid selection ($t^\delta = \alpha_\Delta^\delta = 0$) and selection in diploids is sexually antagonistic ($s^\delta = -s^q = 1/10$, $h^\delta = 1 - h^q = 3/10$), in which case the neo-sex-determining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

414 fraction of the parameter space, even if the neo-W uncouples the sex-determining
locus from a locus under selection.

416 Previous research suggests that when the ancestral sex-determining locus is

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

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

linked to a locus that experiences haploid selection (e.g., meiotic drive), a new,
418 unlinked sex-determining locus invades in order to restore equal sex ratios (Kozielska et al. 2010). Consider, for example, the case where the A locus is linked to the
420 ancestral-SDR ($r < 1/2$) and experiences meiotic drive in males only (e.g., during spermatogenesis but not during oogenesis, $\alpha_\Delta^\delta \neq 0, \alpha_\Delta^q = 0$), without gametic
422 competition ($t^q = t^\delta = 0$). In this case, the zygotic sex ratio can be initially biased only if the ancestral sex-determining system is XY (Figure 4B). We might therefore
424 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
426 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
428 associated with new sex-determining alleles (second terms in table 2). Thus, invasion by a neo-W into an XY system and invasion by a neo-Y into a ZW system
430 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
432 system where females are initially rare because the ancestral-Y is associated with a male meiotic drive allele. However, Figure 4A shows that a neo-Y can invade
434 an ancestrally-ZW system under the same conditions. In fact, where $R < 1/2$ the neo-Y becomes associated with the male meiotic drive allele such that the zygotic
436 sex ratio evolves to become biased towards males.

The green curves in Figure 4 demonstrate a case where transitions between
438 male and female heterogametey occur even though the new sex-determining re-

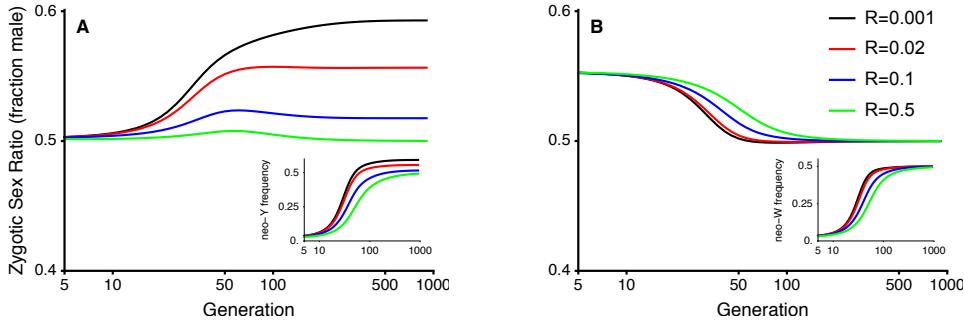


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidally antagonistic with haploid selection favouring the a allele during male meiosis ($s^q = s^\delta = 0.2$, $h^q = h^\delta = 0.7$, $t^q = t^\delta = \alpha_\Delta^\delta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

gion is unlinked to a locus that experiences haploid and diploid selection. We
 440 use these green curves to discuss why heterogametic transitions can occur when
 $R = 1/2$ and $r < 1/2$, as in Table 3. In Figure 4B, an unlinked neo-W can spread
 442 because the zygotic sex ratio is ancestrally male biased. However, in Figure 4A, an
 444 unlinked neo-Y spreads despite the fact that the ancestral zygotic sex ratio is even.
 446 In this case, the the male meiotic drive allele, a , is initially more common among
 448 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
 450 locus is maintained by counter-selection against the a allele in diploids and there-
 452 fore ancestral-ZZ males have generally low diploid fitness. A freely recombining
 454 neo-Y ($R = 1/2$) is not directly favoured or disfavoured by male meiotic drive
 because it is equally likely to be segregate with the A or a allele when found in
 a heterozygote. The neo-Y spreads because it produces males with high diploid
 fitness through matings with ancestral-W-bearing female gametes, which are more
 likely to carry the A allele. Thus, a key factor in explaining why heterogametic
 transitions can occur when $R > r$ is that that the neo-SDR determines sex in the

diploid phase but recombination occurs before any subsequent haploid selection.

456 Environmental sex determination

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

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

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

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

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

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

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 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 gets half of the fitness of a feminizing mutation (neo- W) and half of the fitness of a masculinizing mutation (neo- Y), but only has an effect one half of the time (the other half of the time it produces the same sex as the ancestral system would have, to leading order). As discussed above, $\lambda_{Y',XY|R=1/2}$ is necessarily less than one, but $\lambda_{W',XY|R=1/2}$ can be greater than one if there is haploid selection. That is, when there is haploid selection, ESD mutations can invade an ancestrally-XY system because they generate females that are either rare or have high fitness, in the same manner as a neo- W .

Significantly, equation (5) is the same whether ESD is invading an ancestrally XY or ZW system (because $\lambda_{Y',XY} = \lambda_{W',ZW}$ and $\lambda_{W',XY} = \lambda_{Y',ZW}$). Thus, because the sex ratio is only biased by male haploid selection when the ancestral sex-determination system is XY, Fisherian sex-ratio selection alone does not explain 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 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 males only, $r < 1/2$, $h^{\varphi} = h^{\delta}$, and $s^{\varphi}s^{\delta} < 0$, such that $\lambda_{W',XY} < 1$, see Table 3).

Discussion

Two predominant theories explaining the remarkably high frequency of transitions between sex-determination systems are sexually-antagonistic selection and sex-ratio selection (reviewed in Blaser et al. 2012) (van Doorn, 2014, sexual development). The former predicts that neo-sex-determining alleles can invade when they arise in closer linkage with a sexually-antagonistic locus (van Doorn and Kirk-

patrick 2007; 2010). The latter predicts that new sex-determining systems are
504 generally favoured if they result in more equal sex-ratios than the ancestral sys-
tem. Firstly, we show that selection (including sexually-antagonistic selection) on
506 loci within or near the non-recombining region of the ancestral sex-determining
region can favour heterogametic transitions (XY to ZW or ZW to XY) to new sex-
508 determining systems that are less closely linked to the selected loci (e.g., see Figure
1). Secondly, assuming that selection is weak relative to recombination ('weak se-
510 lection'), we show that new sex-determining alleles are typically favoured if they
are more closely linked to a locus under haploid selection, which is the only con-
512 dition favouring homogametic transitions (XY to XY or ZW to ZW). In addition,
with haploid selection and weak selection, heterogametic transitions (XY to ZW
514 or ZW to XY) can occur even when the new sex-determining region is less closely
linked to the locus under selection (e.g., see Figure 4). **need to mention sex ratio**
516 **here**

Sex-ratio biases caused by haploid selection can facilitate heterogametic transi-
518 tions between sex-determining systems. For instance, alleles favoured by haploid
selection in males often become associated with the Y, which leads to a male-
520 biased zygotic sex-ratio. This male bias increases the potential for a neo-W to
invade (Table 2), which can equalize the sex-ratio (e.g., see Figure 4B, for related
522 examples see Kozielska et al. 2010, Úbeda et al. 2015). However, sex-ratio se-
lection can be overwhelmed by additional selective effects (e.g., when a linked
524 allele is beneficial for male diploids but detrimental for female diploids; Table 3),
preventing the neo-W from invading. Indeed, transitions between sex-determining
526 systems can even lead to stronger sex-ratio biases. For example, where a neo-Y
invades and is linked with a locus that experiences haploid selection in male ga-
528 metes, the sex ratio evolves to become biased (e.g., see Figure 4A and step 1 in
Úbeda et al. 2015). Furthermore, with weak selection, we find that there is no
530 difference in conditions allowing XY to ZW and ZW to XY transitions, indicating
532 that sex chromosome transitions are not predominantly predicted by their effect on
the sex-ratio (i.e., the sex-ratio bias created by male haploid selection facilitates

the spread of a neo-W into an XY system the same way that male haploid selection
534 drives the spread of a neo-Y into a ZW system with a 1:1 sex ratio). Thus, haploid
selection can favour heterogametic transitions both via sex-ratio selection and via
536 fitness effects of alleles that are associated with the neo-sex-determining allele,
and these selection pressures are predicted to often be of equal magnitude when
538 selection is weak.

We have shown that the spread of new sex determination systems can be driven
540 by loci experiencing haploid selection. Because haploid selection can cause transi-
tions that increase or decrease sex-linkage, haploid selection may lead to less
542 stability, and greater potential for cycling, in sex-determination systems (e.g., the
final state of the red line in Figure 4A is the starting state in Figure 4B). In par-
544 ticular, if haploid selection is strong but selective differences between male and
female diploids are weak, we find that heterogametic transitions (XY to ZW or
546 vice versa) are favoured more strongly than homogametic transitions (e.g., with
 $|D^\delta - D^Q| << |\alpha_\Delta^\delta - \alpha_\Delta^Q + t^\delta - t^Q|$ we have $\lambda_{W',XY} > \lambda_{Y',XY}$; equations 3 and S.5).
548 Turnovers driven by haploid selection may help to explain the relative rarity of
heteromorphic sex chromosomes in plants, which are thought to experience more
550 selection during their multicellular haploid stage. For example, among relatively
few dioecious clades in which multiple species have well characterized sex chro-
552 mosomes (Ming et al. 2011), heterogametic transitions have been inferred in *Silene*
subsection *Otites* (Slancarova et al. 2013) and in *Salicaceae* (Pucholt et al. 2015,
554 2017). Furthermore, assuming that transitions from dioecy to hermaphroditism
(equal parental investment in male and female gametes) are favoured in a simi-
556 lar manner to the ESD examined here (equal probability of zygotes developing as
males or females), our results suggest that competition during the haploid stage
558 could drive transitions between dioecy and hermaphroditism, which are frequent
in plants (Käfer et al., 2017, Sabath et al., 2017).

In support of their role in sex chromosome turnover, genes expected to be un-
560 der sexually-antagonistic selection (e.g., those causing bright male colouration)
562 have been found on recently derived sex chromosomes (Lindholm and Breden

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

We assume that sex-determining alleles do not experience direct selection except 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 ancestral sex-determining region because recessive deleterious mutations and/or deletions accumulate around the Y or W sex-determining regions (Rice 1996, Charlesworth and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). During heterogametic transitions (XY to ZW or ZW to XY), but not homogametic transitions (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 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 sex-determination system where the ancestral and new sex-determining loci are both segregating. However, they noted that very rare recombination events around

the ancestral sex-determining region can allow these heterogametic transitions to
594 complete. Degeneration around the Y or W could explain why heterogametic transitions
595 are not observed to be much more common than homogametic transitions
596 despite the fact that our models demonstrate that they are favoured under a wider
range of conditions. For example, Vicoso and Bachtrog (2015) found a dozen
598 sex chromosome configurations among Dipteran species but only one transition
between male and female heterogamety.

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

618 We have shown that tight sex-linkage and haploid selection can drive previ-
ously unexpected transitions between sex-determination systems. In particular,
620 both can select for neo-sex-determining loci that are more loosely linked. In ad-
dition, haploid selection alone can cause transitions analogous to those caused by
622 purely sexually-antagonistic selection, eliminating the need for differences in se-

lection between male and female diploids. Perhaps counterintuitively, transitions
624 involving haploid selection can be driven by sex-ratio selection or cause sex-ratio
biases to evolve. We conclude that haploid selection should be considered as a
626 pivotal factor driving transitions between sex-determination systems. Overall, our
results suggest several new scenarios under which new sex-determination systems
628 are favoured, which could help to explain why the evolution of sex-determination
systems is so dynamic.

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

638 “Polygenic sex determination has been reported in many plants (e.g. Shannon
& Holsinger 2007), fishes (Vandeputte et al. 2007; Ser et al. 2010; Liew et al.
640 2012), crustaceans (e.g. Battaglia 1958; Battaglia & Malesani 1959; Voordouw
& Anholt 2002), bivalves (Haley 1977; Saavedra et al. 1997), gastropods (Yusa
642 2007a,b), and polychaetes (Bacci 1965, 1978; Premoli et al. 1996).” From Vuilleu-
mier et al. 2007: “Polymorphism for sex-determining genes within or among pop-
644 ulations has been reported in many species including houseflies, midges, woodlice,
platyfish, cichlid fish, and frogs (Gordon, 1944; Kallman, 1970; Thompson, 1971;
646 Macdonald, 1978; Bull, 1983; Rigaud et al., 1997; Caubet et al., 2000; Lande et
al., 2001; Ogata et al., 2003; Lee et al., 2004; Mank et al., 2006).” Also check
648 Kallman (1984) -from vD&K, 2010.

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Appendix

836 Recursion Equations

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

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

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

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

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

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

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

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

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

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

competition) in the next generation are given by

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

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

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

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

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

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

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

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

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

888

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

Resident equilibrium and stability

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

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

In particular special cases, e.g., no sex-differences in selection or meiotic drive ($s^{\delta} = s^{\varphi}$, $h^{\delta} = h^{\varphi}$, and $\alpha^{\delta} = \alpha^{\varphi} = 1/2$), the equilibrium allele frequency and sta-

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

check last condition and the stability condition below are correct

944 Selection weak relative to recombination (weak selection)

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

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

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

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

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

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

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

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

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

962 Invasion conditions

Cover the other parts of the characteristic polynomial here.

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

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

⁹⁷⁶ eigenvalue, λ , for any k , is given up to order ϵ^2 by equation (4).

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

⁹⁷⁸ Here, we explore the conditions under which a neo-W invades an XY system as-
 suming that the A locus is initially in tight linkage with the ancestral sex-determining
⁹⁸⁰ 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*
⁹⁸² notebook for proof).

⁹⁸⁴ Starting with the simpler equilibrium (B), the terms of the characteristic poly-
 nomial are

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

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

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

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

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

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

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

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

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

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

1024 case the a allele is favoured by selection in females despite A being fixed on the
 X. For this equilibrium to be stable, $X-A$ must be sufficiently favoured in males
 1026 (for example, by overdominance in males, remembering that a is fixed on the Y)
 to keep A fixed on the X. Specifically, from the stability conditions for equilibrium
 1028 (B), we must have $w_{Aa}^\delta / [(w_{aa}^\delta + w_{Aa}^\delta)/2] > w_{Aa}^\varphi / w_{AA}^\varphi$.

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

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

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

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

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

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

1038 where

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

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

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

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

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

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

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

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

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

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

1056 This condition cannot be met with purely sexually antagonistic selection (as both terms on the left-hand side would then be negative), but it can be met under other
 1058 circumstances. For example, with overdominance in males there is selection for increased A frequencies on X chromosomes in males, which are always paired
 1060 with Y- a haplotypes. Directional selection for a in females can then maintain a polymorphism at the A locus on the X. This scenario selects for a modifier that
 1062 increases recombination between the sex chromosomes (e.g., blue region of Figure

2d in Otto 2014) and facilitates the spread of neo-W-*a* haplotypes, which create
 1064 more females bearing more *a* alleles than the ancestral X chromosome does.

Supplementary Figures

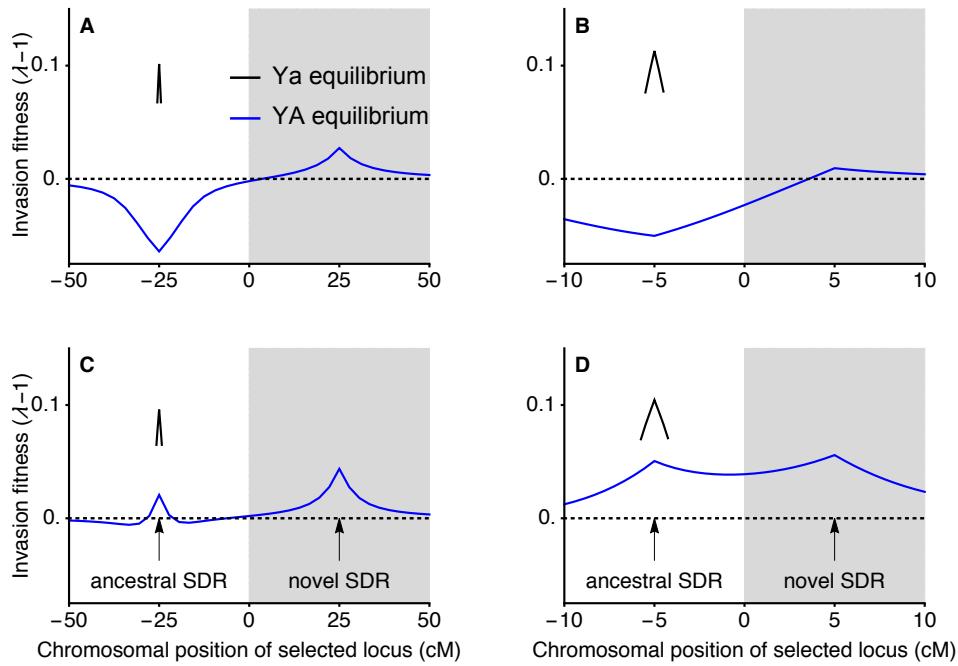


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ($r \approx 0$). In panels A and B, the *a* allele is favoured in females ($w_{aa}^{\varphi} = 1.05$, $w_{Aa}^{\varphi} = 1$, $w_{AA}^{\varphi} = 0.85$) and selection in males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

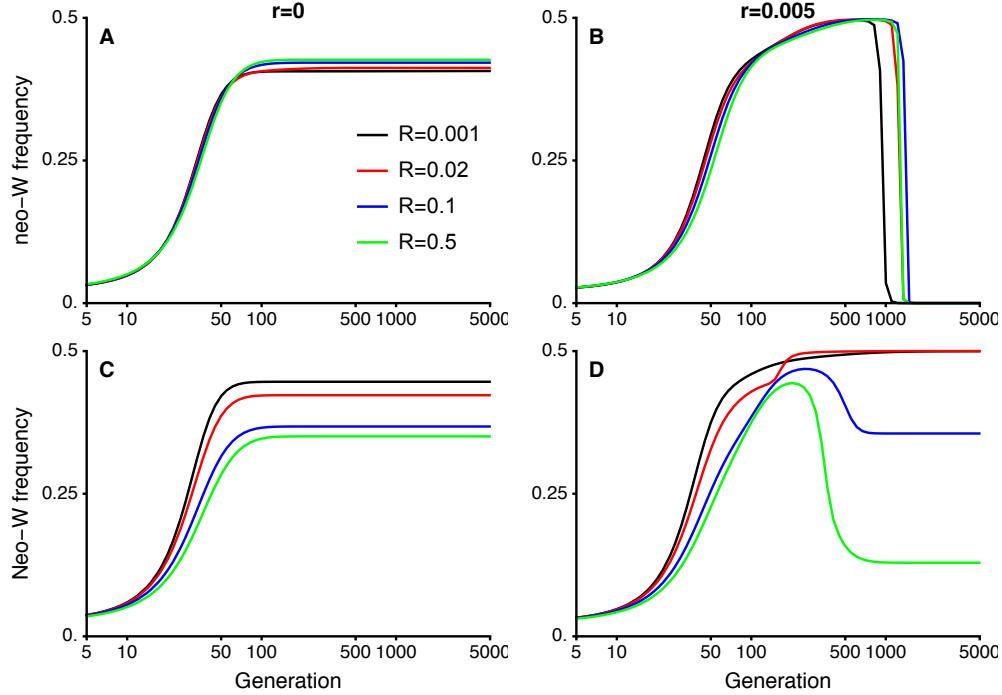


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among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

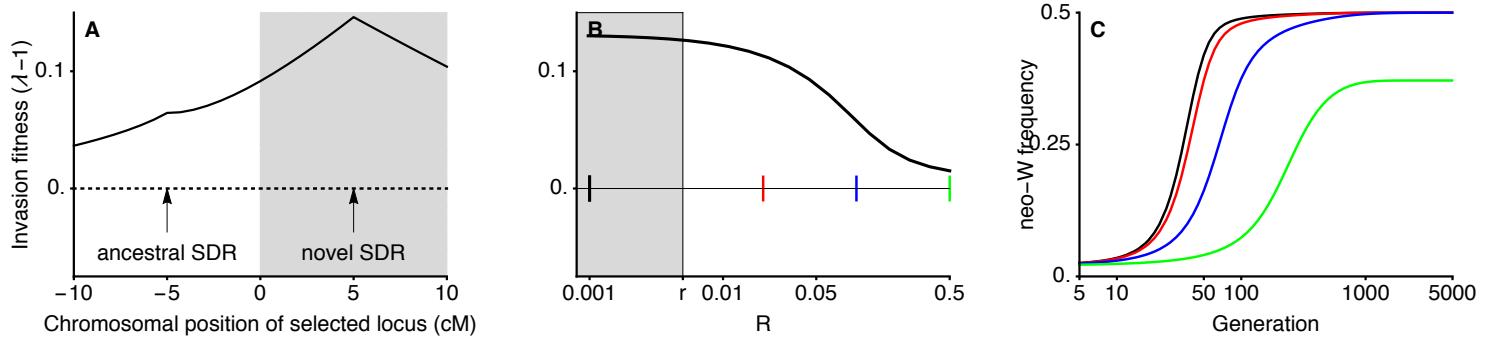


Figure S.3: When there is haploid selection and sexually-antagonistic selection, a neo-W may invade for any R . Check that we mention Sally's result that invasion cannot occur with sexually-antagonistic selection and $R = 1/2$ (currently only in legend for figure 1) Add asterisk to Figure S.5B to show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

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

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

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

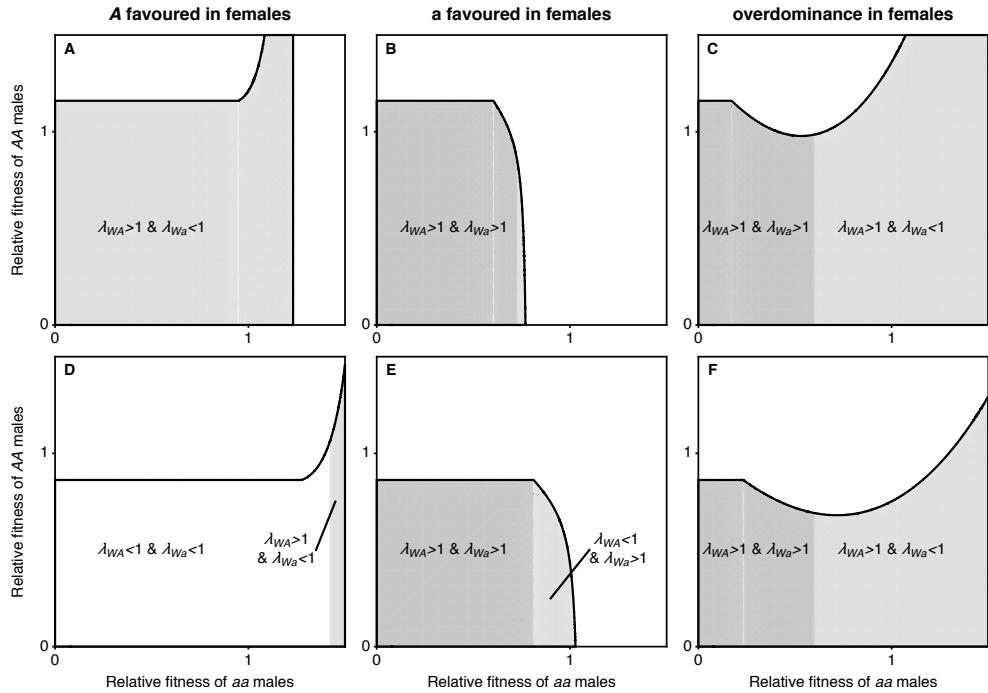


Figure S.4: ABC, $w_a^\delta = 1.16$, $w_A^\delta = 1$. DEF, $w_a^\delta = 1$, $w_A^\delta = 1.16$. I thought that re-running this with $w_a^\delta = 1.16$, $w_A^\delta = 0.84$ and $w_a^\delta = 0.84$, $w_A^\delta = 1.16$ will mean that it matches exactly with the meiotic drive example. Checking for panel A, the result was qualitatively similar (e.g., region where both 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.

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

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

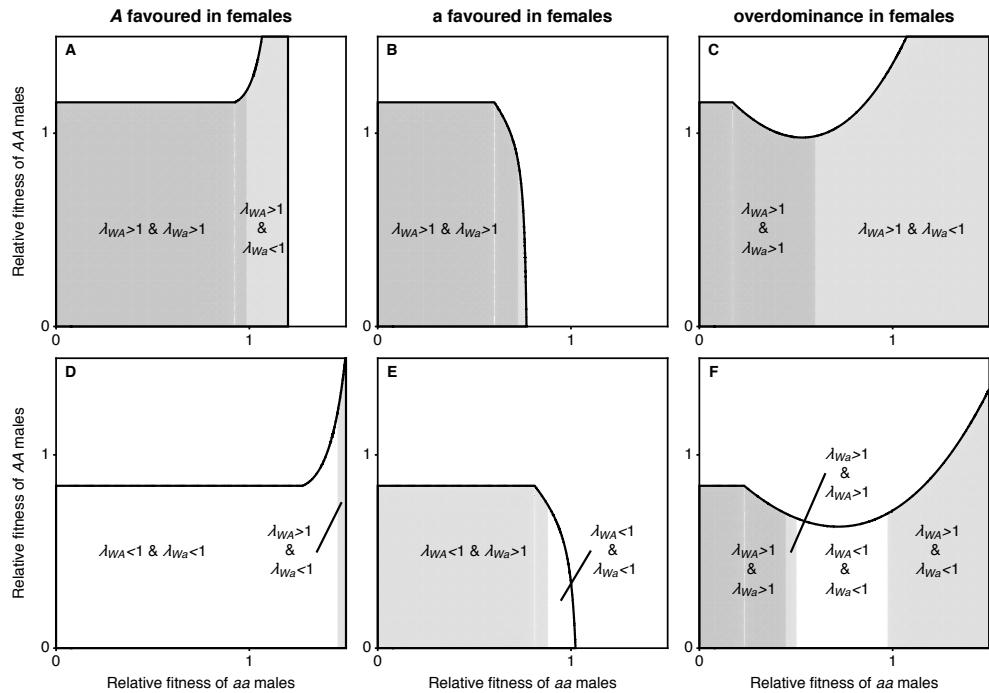


Figure S.5: ABC, $\alpha_{\Delta}^{\delta} = -0.08$ DEF, $\alpha_{\Delta}^{\delta} = 0.08$. Panel F mislabelled, should have $\lambda_{Wa} > 1$ & $\lambda_{WA} < 1$ as the upper label that has the line.

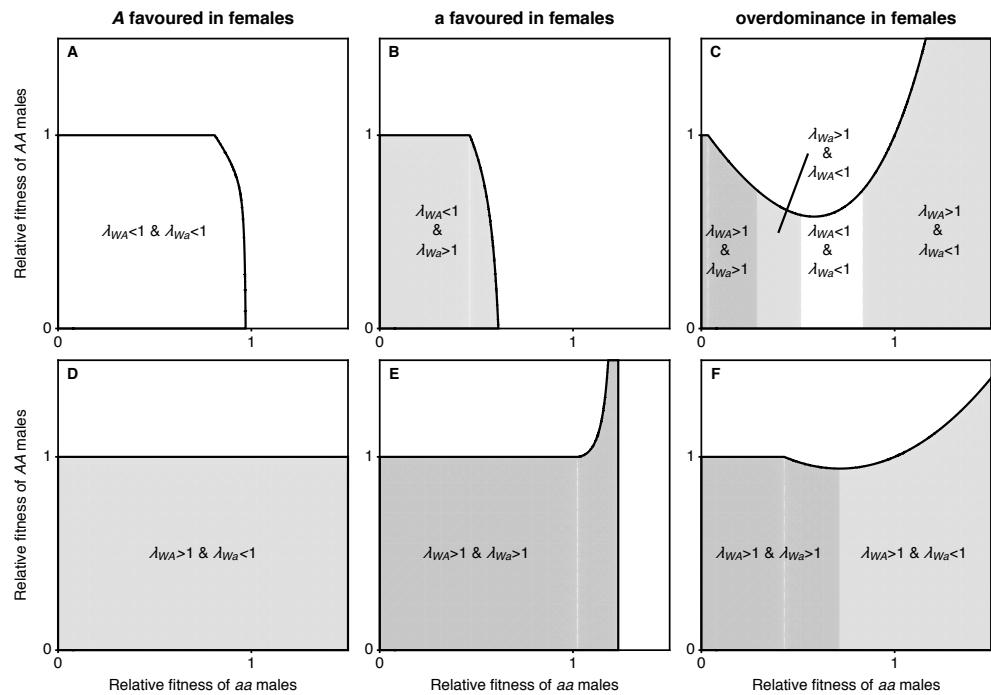


Figure S.6: ABC, $\alpha_{\Delta}^{\varnothing} = -0.08$ DEF, $\alpha_{\Delta}^{\varnothing} = 0.08$. λ s are increased for the haplotype that is favoured by female haploid selection, the stability conditions are also affected.