

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

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Contributions:

Abstract

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

Introduction

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

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

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

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

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

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

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

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

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

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

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

Model

We consider transitions between ancestral and novel sex-determining systems using a three-locus model, each locus having two alleles. Locus **X** is the ancestral sex-determining region, with alleles *X* and *Y* (or *Z* and *W*). Locus **A** is a locus

under selection, with alleles A and a . Locus **M** is a novel sex-determining region,
at which the null allele (M) is initially fixed in the population such that sex of
zygotes is determined by the genotype at the ancestral sex-determining region, **X**;
 XX genotypes become females and XY become males (or ZW become females
and ZZ become males). To evaluate the evolution of new sex-determination sys-
tems, we consider the invasion, fixation, maintenance, and/or loss of novel sex-
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
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
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
develop as females in a proportion (k) of the environments they experience.

In each generation, we census the genotype frequencies in male and female
gametes/gametophytes (hereafter gametes) before gametic competition. A full de-
scription of our model, including recursion equations, is given in the Appendix.
First, competition occurs among male gametes (sperm/pollen competition) and
among female gametes (egg/ovule competition) separately. Selection during ga-
metic competition depends on the **A** locus genotype, relative fitnesses are given
by w_A^φ and w_a^φ ($\varphi \in \{\text{♀}, \text{♂}\}$; see table 1). We assume that all gametes compete for
fertilization during gametic competition, which assumes a polygamous mating sys-
tem. Gametic competition in monogamous mating systems is, however, equivalent
to meiotic drive in our model (described below), as both only alter the frequency
of gametes produced by heterozygotes. After gametic competition, random mating
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
females then experience selection, with relative fitnesses w_{AA}^φ , w_{Aa}^φ , and w_{aa}^φ . The
next generation of gametes is produced by meiosis, during which recombination
and sex-specific meiotic drive can occur. Recombination (i.e., an odd number of
cross-overs) occurs between loci **X** and **A** with probability r , between loci **A** and

M with probability R , and between loci **X** and **M** with probability ρ . Any linear
 178 order of the loci can be modelled with appropriate choices of r , R , and ρ (see Ta-
 ble S.1). Individuals that are heterozygous at the **A** locus may experience meiotic
 180 drive; a gamete produced by Aa heterozygotes of sex δ bear allele A with probab-
 ity α^δ . Thus, the **A** locus can experience sex-specific gametic competition, diploid
 182 selection, and/or meiotic drive.

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	$1 - \alpha^\delta = 1/2 - \alpha_\Delta^\delta / 2$

Results

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

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

The evolution of a new sex-determination system requires that a rare mutant allele at the novel sex-determining locus, m , increases in frequency when rare. The spread of a rare mutant m at the **M** locus is determined by the leading eigenvalue, λ , of the system of eight equations describing the frequency of eggs and sperm carrying the m allele in the next generation (equations S.1). This system simplifies substantially in a number of cases of interest. Dominant neo-Y (when $k = 0$) or neo-W alleles (when $k = 1$) are only found in male diploids (neo-Y) or female diploids (neo-W) such that their growth rate ultimately depends only on the change in frequency of m -bearing gametes produced by males or by females, respectively.

Furthermore, if the m allele is fully epistatically dominant over the ancestral sex-determining system, phenotypes are not affected by the genotype at the ancestral sex-determining region (**X** locus). Thus, the invasion of rare dominant neo-Y or neo-W alleles is determined by the largest eigenvalue that solves a quadratic characteristic polynomial, $\lambda^2 + b\lambda + c = 0$ (see Appendix for a discussion of other roots).

Here, $b = -(\lambda_{mA} + \lambda_{ma}) + (\chi_{mA} + \chi_{ma})$ and $c = (\lambda_{mA} - \chi_{mA})(\lambda_{ma} - \chi_{ma}) - \chi_{mA}\chi_{ma}$, where λ_{mi} is the multiplicative growth rate of mutant haplotypes on background $i \in \{A, a\}$, without accounting for loss due to recombination, and χ_{mi} is the rate at which mutant haplotypes on background $i \in \{A, a\}$ recombine onto the other **A** locus background in heterozygotes (see 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 ancestral population, it is convenient to follow the frequency of the A allele among female gametes (eggs), p_X^\varnothing , and among X-bearing, p_X^δ , and among Y-bearing, p_Y^δ , male gametes (sperm/pollen). We also track the fraction of male gametes that are Y-bearing, q , which may deviate from 1/2 due to meiotic drive in males. We will consider only equilibrium frequencies of alleles, \hat{p}_i^\varnothing , and Y-bearing male gametes, \hat{q} , to ensure the eigenvalues of the invasion analysis are valid.

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

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

$\bar{p}^\delta = (1 - \hat{q})\hat{p}_X^\delta + q\hat{p}_Y^\delta$ 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

222 We are particularly concerned with the conditions under which a rare neo-sex-
determining allele increases in frequency, which occurs when the largest eigen-
224 value, λ , is greater than one. Given the characteristic polynomial and the Perron-
Forbenius theorem (see supplementary material for details), at least one λ is greater
226 than one when $2 + b < 0$, or $(\lambda_{mA} - \chi_{mA}) + (\lambda_{ma} - \chi_{ma}) > 2$. In words, the neo-sex-
determining allele will certainly spread when the average change in its frequency,
228 taking into account that recombination breaks up mutant *mi* haplotypes (at rate
 χ_{mi}) but ignoring the fact that recombination also creates mutant haplotypes, is
230 positive. We are also guaranteed to have at least one λ greater than one when
 $+ b + c < 0$. This is never true if neither haplotype can spread ($\lambda_{mA}, \lambda_{ma} < 1$).
232 If only one haplotype can spread then the new sex-determining allele increases in

frequency on one **A** background and declines on the other, and invasion requires

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

For example, if we assume that only the *mA* haplotype has a positive growth rate ($\lambda_{ma} < 1 < \lambda_{mA}$), the first term on the left-hand side of (1) is negative and invasion requires that the growth rate of *mA* haplotypes ($\lambda_{ma} - 1$) and the rate at which they are produced by recombination in *ma* haplotypes (χ_{ma}) are sufficiently large relative to the growth rate and recombination gain of *ma* haplotypes. i've updated this section with the correct λ and χ terms. still a bit funny that not guaranteed invasion when both lambdas greater than one, and when both are greater than one then the condition above, (1), has the inequality flipped, which makes no sense (no invasion if no recombination despite both haplotypes growing). perhaps i made a mistake somewhere? see turnoverhomhet.nb

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

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

276 Tight linkage with the ancestral sex-determining region

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

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

290 lected loci within the non-recombining region around the SDR. In essence, through
291 tight linkage with the A locus, the ancestral Y becomes strongly specialized on the
292 allele that has the highest fitness across male haploid and diploid phases. Given
293 that the ancestral Y is at this equilibrium, it is not possible for a neo-Y to create
294 males that have higher fitness than the ancestral Y.

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

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

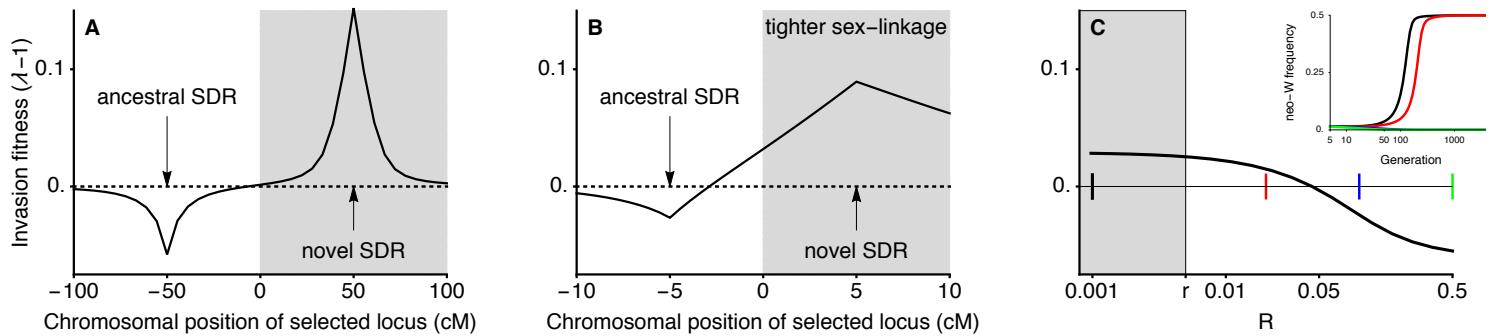


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is more loosely linked to a locus under sexually-antagonistic selection (here, without haploid selection $r^{\delta} = \alpha_{\Delta}^{\delta} = 0$). In panel A, linkage is loose enough relative to selection that the weak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

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

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

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

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

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

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

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

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

and

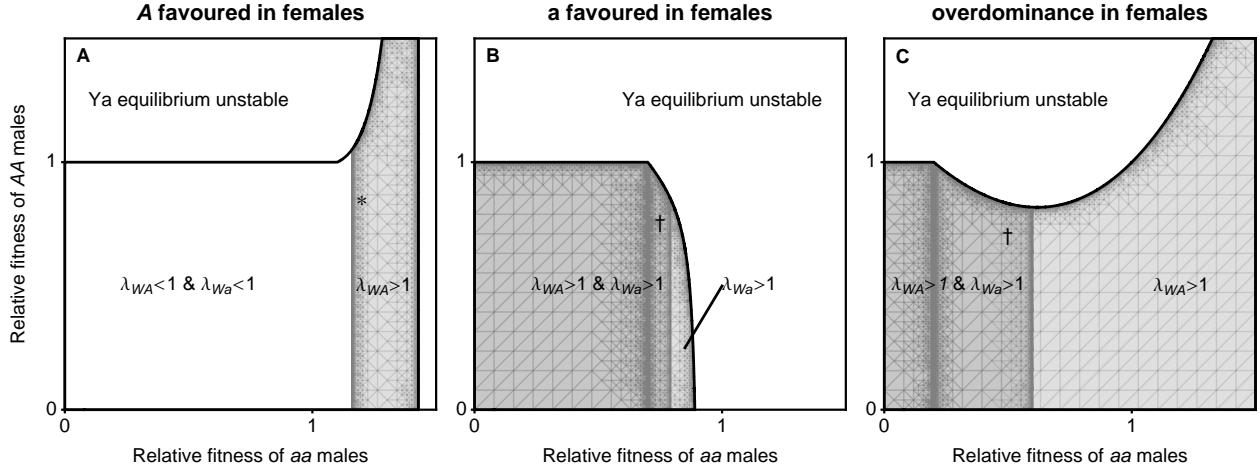


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ($r = 0$), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ($w_{Aa}^{\varphi} = 1$) and only consider stable equilibria at which both A locus alleles are maintained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

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

358 where $V_A = \bar{p}(1 - \bar{p})$ is the variance in the equilibrium frequency of A and $S_A = (D^{\delta} + \alpha_{\Delta}^{\delta} + t^{\delta}) - (D^{\varphi} + \alpha_{\Delta}^{\varphi} + t^{\varphi})$ describes sex differences in selection for the A versus
 360 a across diploid selection, meiosis, and gametic competition. The diploid selection term, $D^{\varphi} = [\bar{p}s^{\varphi} + (1 - \bar{p})h^{\varphi}s^{\varphi}] - [\bar{p}h^{\varphi}s^{\varphi} + (1 - \bar{p})]$, is the difference in fitness
 362 between A and a alleles in diploids of sex $\varphi \in \{\varphi, \delta\}$, where \bar{p} is the leading-order probability of mating with an A -bearing gamete from the opposite sex (see
 364 Appendix). The difference in A -allele-frequency among Y-bearing sperm versus X-bearing sperm is given by $\hat{p}_Y^{\delta} - \hat{p}_X^{\delta} = V_A(D^{\delta} - D^{\varphi} + \alpha_{\Delta}^{\delta} - \alpha_{\Delta}^{\varphi} + t^{\delta} - t^{\varphi})(1 - 2r)/2r$.
 366 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
 368 only if it is more closely linked to the selected locus than the ancestral sex-determining
 region (i.e., if $R < r$; note that $V_A S_A^2$ is strictly positive as long as \mathbf{A} is polymor-
 370 phic). 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),
 372 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
 374 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
 376 a neo-W is equivalent to the spread of a neo-Y ($\lambda_{W',XY} = \lambda_{Y',XY}$), such that het-
 erogametic transitions (XY to ZW or ZW to XY) can also occur only if the neo-
 378 sex-determining region is more closely linked to a locus under selection ($R < r$),
 as found by van Doorn and Kirkpatrick (2010). With haploid selection, however,
 380 the additional term in equation (3) can be positive, which can allow, for example,
 neo-W invasion ($\lambda_{W',XY} > 1$) even when the neo-sex-determining region is less
 382 closely linked to the selected locus ($R > r$).

Equation (3) shows that, with weak selection, neo-W alleles can invade an XY
 384 system for a large number of selective regimes. To clarify the parameter space
 under which $\lambda_{W',XY} > 1$, we consider several special cases. Firstly, if the \mathbf{A} locus
 386 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
 388 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
 390 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
 392 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
 394 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
 396 table 3 we give the conditions where invasion occurs when we further assume

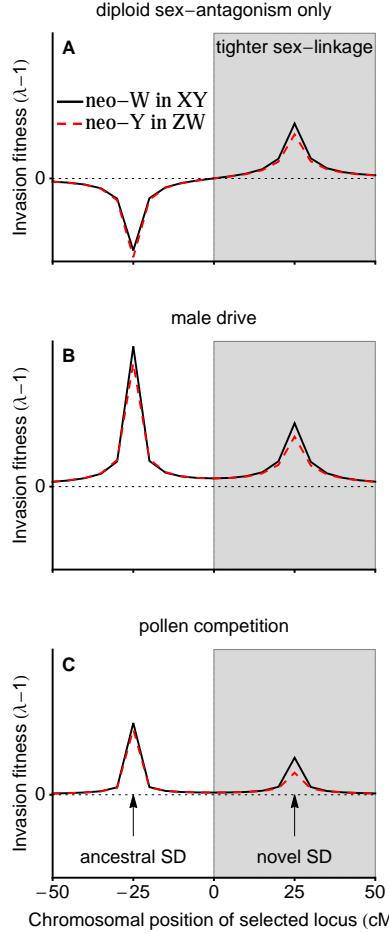


Figure 3: A neo-W can invade an XY system under a large number of selective regimes. In panel A, there is no haploid selection ($t^\delta = \alpha_\Delta^\delta = 0$) and selection in diploids is sexually antagonistic ($s^\delta = -s^q = 1/10$, $h^\delta = 1 - h^q = 3/10$), in which case the neo-sex-determining allele can only invade if it is more closely linked to the selected locus ($R < r$, gray region; but see Figure 1B for the case of very tight linkage). In panel B, male drive ($\alpha_\Delta^\delta = -1/20$, $t^\delta = \alpha_\Delta^q = 0$) opposes selection in diploids (no sex-differences: $s^\delta = 1/10$, $h^\delta = 7/10$), in which case the neo-sex-determining allele can invade regardless of linkage. In panel C, gametic competition in males ($t^\delta = -1/10$, $t^q = \alpha_\Delta^q = 0$) opposes selection in diploids (sex-differences: $s^\delta = 1/20$, $s^q = 3/20$, $h^\delta = 7/10$), in which case the neo-sex-determining allele can once again invade regardless of linkage. We use Haldane's map function (Equation 3 in Haldane 1919) to convert from map distance (centiMorgans, cM) to the probability of recombination (an odd number of cross-over events). **Check the mismatch between red and black lines here: probably because of adding or subtracting from 1. Can remove the mismatch by flipping the fitnesses between males and females (again). That is, if M_{AA} is the fitness of AA male diploids in an ancestral XY system, then M_{AA} is the fitness of AA female diploids in an ancestral ZW system. I think this makes sense in A, where we don't really want a difference between the red and black curves, but this makes less sense in B and C where we want to restrict haploid selection to males regardless of the ancestral system. We could just not flip the haploid fitnesses, but then does it make sense to flip the diploid fitnesses?**

that haploid selection only occurs in one sex (e.g., during male meiosis only) and
 398 dominance coefficients are equal in the two sexes, $h^{\varphi} = h^{\delta}$. When there is no
 gametic competition and meiotic drive is in one sex only, an unlinked neo-W can
 400 invade as long as the same allele is favoured during diploid selection in males and
 females ($s^{\varphi}s^{\delta} > 0$, see Figure 3B and Figure 4B). When there is no meiotic drive
 402 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
 404 are sex differences in selection of one type (e.g., $s^{\varphi}(s^{\delta} - s^{\varphi}) > 0$, see Figure 3C).
 These special cases indicate that neo-W invasion can occur for a relatively large
 406 fraction of the parameter space, even if the neo-W uncouples the sex-determining
 locus from a locus under selection.

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

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

408 Previous research suggests that when the ancestral sex-determining locus is
 linked to a locus that experiences haploid selection (e.g., meiotic drive), a new,
 410 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
 412 ancestral-SDR ($r < 1/2$) and experiences meiotic drive in males only (e.g., during
 spermatogenesis but not during oogenesis, $\alpha_{\Delta}^{\delta} \neq 0, \alpha_{\Delta}^{\varphi} = 0$), without gametic
 414 competition ($t^{\varphi} = t^{\delta} = 0$). In this case, the zygotic sex ratio can be initially biased
 only if the ancestral sex-determining system is XY (Figure 4B). We might there-
 416 fore expect a difference in the potential for XY to ZW and ZW to XY transitions.
 However, to leading order with selection weak relative to recombination, we find
 418 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
 420 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
 422 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
 424 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
 426 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
 428 sex ratio evolves to become biased towards males.

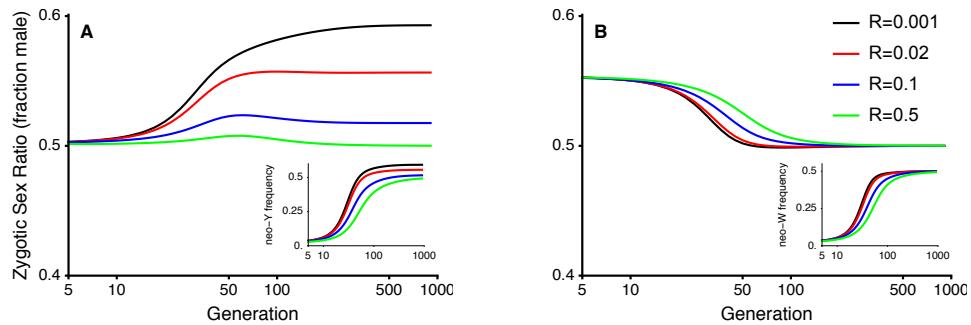


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidally antagonistic with haploid selection favouring the a allele during male meiosis. In panel A, male drive in an ancestral ZW system has no effect on the zygotic sex ratio, yet a neo-Y can invade and replace the ancestral sex-determination system (inset shows neo-Y frequency among male gametes, the ancestral W also goes to fixation during this transition). When $R < 1/2$, the neo-Y becomes associated with the allele favoured by drive, causing the zygotic sex ratio to become biased, hence the frequency of neo-Y among male gametes can be higher than 0.5 (inset). In panel B, male drive in an ancestral XY system causes a male bias, allowing a neo-W to invade and replace the ancestral sex-determination system (inset shows neo-W frequency among female gametes, the ancestral Y also goes to fixation), which balances the zygotic sex ratio. Parameters: $s^{\varphi} = s^{\delta} = 0.2$, $h^{\varphi} = h^{\delta} = 0.7$, $t^{\varphi} = t^{\delta} = \alpha_{\Delta}^{\varphi} = 0$, $\alpha_{\Delta}^{\delta} = -0.1$, $r = 0.02$.

The green curves in Figure 4 demonstrate a case where transitions between
 430 male and female heterogamety occur even though the new sex-determining re-
 gion is unlinked to a locus that experiences haploid and diploid selection. We
 432 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
 434 because the zygotic sex ratio is ancestrally male biased. However, in Figure 4A, an

unlinked neo-Y spreads despite the fact that the ancestral zygotic sex ratio is even.

436 In this case, the male meiotic drive allele, a , is initially more common among
ancestral-Z-bearing eggs than ancestral-W-bearing eggs because the Z is found in
438 males more often than the W and $r < 1/2$ (equation S.5). Polymorphism at the A
locus is maintained by counter-selection against the a allele in diploids and there-
440 fore ancestral-ZZ males have generally low diploid fitness. A freely recombining
neo-Y ($R = 1/2$) is not directly favoured or disfavoured by male meiotic drive
442 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
444 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
446 transitions can occur when $R > r$ is that the neo-SDR determines sex in the
diploid phase but recombination occurs before any subsequent haploid selection.

448 **Environmental sex determination**

We next consider the case where the new sex-determining mutation, m , causes sex
450 to be determined probabilistically or by heterogeneous environmental conditions
(environmental sex determination, ESD), with individuals carrying allele m devel-
452 oping as females with probability k . Here, we do not assume that the environmen-
tal conditions that determine sex also differentially affect the fitness of males versus
454 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-
456 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
458 reviews by Charnov 1982, Bull 1983, West 2009).

The characteristic polynomial determining the eigenvalues (equations S.1) does
460 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-
462 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 $k = 1$.

464 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
 466 spread of which is given by

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

468 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
 470 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
 472 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
 474 (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
 476 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
 478 system because they generate females that are either rare or have high fitness, in
 the same manner as a neo- W .

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

484 plain the invasion of ESD under weak selection. Specifically, with male haploid
485 selection, the neo-ESD is equally likely to invade when it equalizes the zygotic sex
486 ratio (through $\lambda_{W',XY}$) and when it doesn't (through $\lambda_{Y',ZW}$). In addition, we note
487 that ESD may not invade, even if the sex ratio is initially biased (e.g., with drive in
488 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

490 Two predominant theories explaining the remarkably high frequency of transitions
491 between sex-determination systems are sexually-antagonistic selection and sex-
492 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
493 arise in closer linkage with a sexually-antagonistic locus (van Doorn and Kirk-
494 patrick 2007; 2010). The latter predicts that new sex-determining systems are
495 generally favoured if they result in more equal sex-ratios than the ancestral sys-
496 tem. Firstly, we show that selection (including sexually-antagonistic selection) on
497 loci within or near the non-recombining region of the ancestral sex-determining
498 region can favour heterogametic transitions (XY to ZW or ZW to XY) to new sex-
499 determining systems that are less closely linked to the selected loci (e.g., see Figure
500 1). Secondly, assuming that selection is weak relative to recombination ('weak se-
501 lection'), we show that new sex-determining alleles are typically favoured if they
502 are more closely linked to a locus under haploid selection, which is the only con-
503 dition favouring homogametic transitions (XY to XY or ZW to ZW). In addition,
504 with haploid selection and weak selection, heterogametic transitions (XY to ZW
505 or ZW to XY) can occur even when the new sex-determining region is less closely
506 linked to the locus under selection (e.g., see Figure 4). need to mention sex ratio
507 here

508 Sex-ratio biases caused by haploid selection can facilitate heterogametic transi-
509 tions between sex-determining systems. For instance, alleles favoured by haploid
510 selection in males often become associated with the Y, which leads to a male-

512 biased zygotic sex-ratio. This male bias increases the potential for a neo-W to
513 invade (Table 2), which can equalize the sex-ratio (e.g., see Figure 4B, for related
514 examples see Kozielska et al. 2010, Úbeda et al. 2015). However, sex-ratio se-
515 lection can be overwhelmed by additional selective effects (e.g., when a linked
516 allele is beneficial for male diploids but detrimental for female diploids; Table 3),
517 preventing the neo-W from invading. Indeed, transitions between sex-determining
518 systems can even lead to stronger sex-ratio biases. For example, where a neo-Y
519 invades and is linked with a locus that experiences haploid selection in male ga-
520 metes, the sex ratio evolves to become biased (e.g., see Figure 4A and step 1 in
521 Úbeda et al. 2015). Furthermore, with weak selection, we find that there is no
522 difference in conditions allowing XY to ZW and ZW to XY transitions, indicating
523 that sex chromosome transitions are not predominantly predicted by their effect on
524 the sex-ratio (i.e., the sex-ratio bias created by male haploid selection facilitates
525 the spread of a neo-W into an XY system the same way that male haploid selection
526 drives the spread of a neo-Y into a ZW system with a 1:1 sex ratio). Thus, haploid
527 selection can favour heterogametic transitions both via sex-ratio selection and via
528 fitness effects of alleles that are associated with the neo-sex-determining allele,
529 and these selection pressures are predicted to often be of equal magnitude when
530 selection is weak.

We have shown that the spread of new sex determination systems can be driven
531 by loci experiencing haploid selection. Because haploid selection can cause tran-
532 sitions that increase or decrease sex-linkage, haploid selection may lead to less
533 stability, and greater potential for cycling, in sex-determination systems (e.g., the
534 final state of the red line in Figure 4A is the starting state in Figure 4B). In par-
535 ticular, if haploid selection is strong but selective differences between male and
536 female diploids are weak, we find that heterogametic transitions (XY to ZW or
537 vice versa) are favoured more strongly than homogametic transitions (e.g., with
538 $|D^\delta - D^\Omega| << |\alpha_\Delta^\delta - \alpha_\Delta^\Omega + t^\delta - t^\Omega|$ we have $\lambda_{W',XY} > \lambda_{Y',XY}$; equations 3 and S.5).
539 Turnovers driven by haploid selection may help to explain the relative rarity of
540 heteromorphic sex chromosomes in plants, which are thought to experience more

542 selection during their multicellular haploid stage. For example, among relatively
543 few dioecious clades in which multiple species have well characterized sex chro-
544 mosomes (Ming et al. 2011), heterogametic transitions have been inferred in *Silene*
545 subsection *Otites* (Slancarova et al. 2013) and in *Salicaceae* (Pucholt et al. 2015,
546 2017). Furthermore, assuming that transitions from dioecy to hermaphroditism
547 (equal parental investment in male and female gametes) are favoured in a simi-
548 lar manner to the ESD examined here (equal probability of zygotes developing as
549 males or females), our results suggest that competition during the haploid stage
550 could drive transitions between dioecy and hermaphroditism, which are frequent
551 in plants (Käfer et al., 2017, Sabath et al., 2017).

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

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

592 Another simplification that we made is that meiotic drive involves only a single
593 locus with two alleles. However, many meiotic drive systems involve an interac-
594 tion with another locus at which alleles may ‘suppress’ the action of meiotic drive
595 (Burt and Trivers 2006, Lindholm et al. 2016) Taylor,1999. Thus, the dynamics
596 of meiotic drive alleles can be heavily dependent on the interaction between two
597 loci and the recombination rate between them, which in turn can be affected by
598 sex-linkage if there is reduced recombination between sex chromosomes (Hurst
599 and Pomiankowski 1991). Furthermore, in some cases, a driving allele may act
600 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

602 drive allele (Holman et al. 2015). In polygamous mating systems, the intensity of
603 pollen/sperm competition can depend on the density of males available to donate
604 pollen/sperm, which can itself depend on the sex ratio (Taylor and Jaenike 2002).
605 In terms of our model, this implies that the strength of gametic competition (t^δ)
606 may both determine and be determined by the sex ratio. How the evolution of
607 new sex-determining mechanisms could be influenced by two-locus meiotic drive
608 and/or by ecological feedbacks under different mating systems remains to be stud-
ied.

610 We have shown that tight sex-linkage and haploid selection can drive previ-
611 ously unexpected transitions between sex-determination systems. In particular,
612 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
614 purely sexually-antagonistic selection, eliminating the need for differences in se-
lection between male and female diploids. Perhaps counterintuitively, transitions
616 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
618 pivotal factor driving transitions between sex-determination systems. Overall, our
results suggest several new scenarios under which new sex-determination systems
620 are favoured, which could help to explain why the evolution of sex-determination
systems is so dynamic.

622 Discuss polymorphic mating systems somewhere? Say that haploid selection
623 makes this particularly likely (I think there are examples with gametic competition
and weak selection, whereas the vD&K, 2010 results suggest that it's not possible
with weak selection and diploid selection alone)? This might be best as a section
626 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
628 pasted from from Vuilleumier et al. 2007 and vD&K, 2010, might be added to this
section.

630 “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.

632 2012), crustaceans (e.g. Battaglia 1958; Battaglia & Malesani 1959; Voordouw
633 & Anholt 2002), bivalves (Haley 1977; Saavedra et al. 1997), gastropods (Yusa
634 2007a,b), and polychaetes (Bacci 1965, 1978; Premoli et al. 1996).” From Vuilleumier
635 et al. 2007: “Polymorphism for sex-determining genes within or among pop-
636 ulations has been reported in many species including houseflies, midges, woodlice,
637 platyfish, cichlid fish, and frogs (Gordon, 1944; Kallman, 1970; Thompson, 1971;
638 Macdonald, 1978; Bull, 1983; Rigaud et al., 1997; Caubet et al., 2000; Lande et
639 al., 2001; Ogata et al., 2003; Lee et al., 2004; Mank et al., 2006).” Also check
640 Kallman (1984) -from vD&K, 2010.

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Appendix

828 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

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

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

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

Table S.1: Values of ρ for different loci orders assuming no interference and $r, R \in (0, 1/2)$.
 write all in form of first line?
 so that 1/2 cases are okay (can't determine chi if R is 1/2 in second line, or if r is 1/2 in third line)

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

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

competition) in the next generation are given by

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

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

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

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

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

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

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

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

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

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

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

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

880

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, Immler et al. 2012), or a combination of these selective regimes.

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

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

Sex & Life Cycle Stage	Mean Fitness
female gametes (\bar{w}_H^{\varnothing})	$p_X^{\varnothing} w_A^{\varnothing} + (1 - p_X^{\varnothing}) w_a^{\varnothing}$
male gametes (\bar{w}_H^{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
902 strengths of selection and recombination. However, here, we focus on two regimes
903 (tight linkage and weak selection) in order to make fewer assumptions about fit-
904 nesses.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

924 properties are unaffected by small amounts of recombination between the SDR
 925 and A locus, although equilibrium frequencies may be slightly altered. For the a
 926 allele to be stably fixed on the Y we need $\bar{w}_{Y_a}^\delta > \bar{w}_{YA}^\delta$ where $\bar{w}_{Y_a}^\delta = w_a^\delta [1 -$
 $\alpha_\Delta^\delta] w_A^q w_{Aa}^\delta + (1 - \hat{p}_X^q) w_a^q w_{aa}^\delta]$ and $\bar{w}_{YA}^\delta = w_A^\delta [\hat{p}_X^q w_A^q w_{AA}^\delta + (1 - \hat{p}_X^q) (1 + \alpha_\Delta^\delta) w_a^q w_{Aa}^\delta]$.
 927 That is, Y-a haplotypes must have higher fitness than Y-A haplotypes. Substitut-
 928 ing in $\hat{p}_X^q = \hat{p}_X^q$ from above, fixation of the a allele on the Y requires that $\gamma_i > 0$
 929 where $\gamma_{(A)} = w_a^\delta [(1 - \alpha_\Delta^\delta) w_{Aa}^\delta \phi + w_{aa}^\delta \psi] - w_A^\delta [w_{AA}^\delta \phi + (1 + \alpha_\Delta^\delta) w_{Aa}^\delta \psi]$ for equilib-
 930 rium (A) and $\gamma_{(B)} = (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta - w_A^\delta w_{AA}^\delta$ for equilibrium (B). Stability of a
 931 polymorphism on the X chromosome (equilibrium A) further requires that $\phi > 0$
 932 and $\psi > 0$. Fixation of the a allele on the X (equilibrium B) can be stable only if
 933 equilibrium (A) is not, as it requires $\psi < 0$ and $2w_A^q w_{AA}^q > (1 - \alpha_\Delta^q) w_a^q w_{Aa}^q$ or just
 934 $4w_A^q w_{AA}^q < (1 - \alpha_\Delta^q) w_a^q w_{Aa}^q$ (which prevents $\psi > 0$).

935 **check last condition and the stability condition below are correct** The last con-
 936 dition looks good to me, although in your Turnover-norec-MFS.nb you look at YA
 937 fixed, so you have to flip everything (so I made Turnover-norec-MFS-MMO.nb to
 938 do this). The one issue I can find here is that you can also prevent $\lambda > 1$ when
 939 the slope and intercept of the quadratic at $\lambda = 1$ are negative (you only looked at
 940 both being positive). In this case we need $4w_A^q w_{AA}^q < (1 - \alpha_\Delta^q) w_a^q w_{Aa}^q$, which also
 941 prevents $\psi > 0$. I've added this in. It could also be the case that the slope and inter-
 942 cept are the same sign but the roots are imaginary - but this is never the case here.
 943 Stability condition below looks good to me (from matt version of turnoverSOM-
 944 MIKE.nb).

945 Selection weak relative to recombination (weak selection)

946 Here, we assume that selection and meiotic drive are weak relative to recombi-
 947 nation ($s^\delta, t^\delta, \alpha_\Delta^\delta$ of order ϵ). The maintenance of a polymorphism at the A locus
 948 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 ($\hat{p}_X^\varphi = \hat{p}_X^\delta = \hat{p}_Y^\delta = \bar{p}$) 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 $O(\epsilon)$:

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

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

964 Invasion conditions

Cover the other parts of the characteristic polynomial here.

966 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
968 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)
970 and is epistatically dominant to the ancestral sex-determining locus, we need only
two recursion equations (e.g., tracking the change in the frequency of neo-Y-A and
972 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
974 (Table 2).

For tight linkage between the ancestral sex-determining locus and the selected
976 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
978 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)

980 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
982 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*
984 notebook for proof).

Starting with the simpler equilibrium (B), the terms of the characteristic poly-
986 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,
 988 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-
 990 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
 992 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
 994 develop as the rarer, female, sex.

Secondly, haploid selection in females selects on neo-W haplotypes directly. At
 996 equilibrium (B), the fitness of female gametes under the ancestral sex-determining
 system is w_A^φ such that the relative fitnesses of neo-W- A and neo-W- a haplotypes
 998 during female gametic competition are w_A^φ/w_A^φ and w_a^φ/w_A^φ (see terms in equation
 S.6). Meiotic drive in females will also change the proportion of gametes that carry
 1000 the A versus a alleles, which will be produced by heterozygous females in propor-
 tions $(1 + \alpha_\Delta^\varphi)/2$ and $(1 - \alpha_\Delta^\varphi)/2$, respectively. These terms are only associated with
 1002 heterozygous females, i.e., they are found alongside w_{Aa}^φ .

Thirdly, haploid selection in males affects the diploid genotypes of females
 1004 by altering the allele frequencies in the male gametes that female gametes pair
 with. At equilibrium (B), neo-W female gametes will mate with X- A male ga-
 metes with probability $w_A^\delta(1 + \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ and Y- a male gametes with probability
 1006

$w_a^\delta(1 - \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$, where the $2\bar{w}_H^\delta$ terms have been canceled in equation (S.6)
 1008 (as mentioned above). Thus, for example, neo-W-A haplotypes are found in AA
 female diploids with probability $w_A^\delta(1 + \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ (first term in square brackets
 1010 in the numerator of equation S.6a) and in Aa female diploids with probability
 $w_a^\delta(1 - \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ (see equation S.6c and the second term in square brackets in
 1012 the numerator of equation S.6a).

The other terms in equations (S.6) are more easily interpreted if we assume that
 1014 there is no haploid selection in either sex, in which case $\lambda_{mA} > 1$ when $w_{Aa}^q > w_{AA}^q$
 and $\lambda_{ma} > 1$ when $(w_{Aa}^q + w_{aa}^q)/2 > w_{AA}^q$. These conditions cannot be met under
 1016 purely sexually-antagonistic selection, where A is directionally favoured in females
 $(w_{AA}^q > w_{Aa}^q > w_{aa}^q)$ and a is directionally favoured in males ($w_{AA}^\delta > w_{Aa}^\delta > w_{aa}^\delta$).
 1018 Essentially, the X is then already as specialized as possible for the female beneficial
 allele (A is fixed on the X), and the neo-W often makes daughters with the Y- a
 1020 haplotype, increasing the flow of a alleles into females, which reduces the fitness
 of those females.

1022 If selection doesn't uniformly favour A in females, however, neo-W- A haplo-
 types and/or neo-W- a haplotypes can spread ($\lambda_{mA} > 1$ and/or $\lambda_{ma} > 1$) at this
 1024 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 $w_{Aa}^q > w_{AA}^q$, as stated
 1026 above. In this case the a allele is favoured by selection in females despite A being
 fixed on the X. For this equilibrium to be stable (i.e., to keep A fixed on the X),
 1028 X- a cannot be overly favoured in females and X- A must be sufficiently favoured
 in males (for example, by overdominance in males, remembering that a is fixed
 1030 on the Y). Specifically, from the stability conditions for equilibrium (B), we must
 have $w_{Aa}^q < 2w_{AA}^q$ and $w_{Aa}^\delta / [(w_{aa}^\delta + w_{Aa}^\delta)/2] > w_{Aa}^q / w_{AA}^q$.

1032 Still considering $w_{Aa}^q > w_{AA}^q$, the neo-W can also spread alongside the a allele
 $(\lambda_{ma} > 1)$ if w_{aa}^q is large enough such that $(w_{Aa}^q + w_{aa}^q)/2 > w_{AA}^q$. This can occur
 1034 with overdominance or directional selection for a in females (Figure 2B,C). [mike,](#)
[you might want to check these last two statements i've edited, and the following.](#)
 1036 In this case, a is not favoured enough in females near the equilibrium (comparing

Aa to *AA* genotypes) to prevent *A* from stably fixing on the X, and yet the neo-W can spread with *a* because it produces female *aa* individuals by capturing Y-*a* haplotypes.

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

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

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

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

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

where

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

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

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

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

As with equilibrium (B), haploid selection again modifies invasion fitnesses 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 longer always homozygote *AA* and males are no longer always heterozygote *Aa*. Thus the effect of haploid selection in males is reduced, as is the difference in fitness between neo-W haplotypes and resident X haplotypes, as both can be on any diploid or haploid background.

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
 1052 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
 1054 (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 $w_{AA}^{\delta} < w_{Aa}^{\delta} < w_{aa}^{\delta}$).
 1056 The neo-W-*A* can spread as long as it becomes associated with females that bear
 more *A* alleles than observed at equilibrium (A).

Without haploid selection, the neo-W-*a* haplotype spreads ($\lambda_{ma} > 1$) if and
 1058 only if

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

1060 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
 1062 circumstances. For example, with overdominance in males there is selection for
 increased *A* frequencies on X chromosomes in males, which are always paired
 1064 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
 1066 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
 1068 more females bearing more *a* alleles than the ancestral X chromosome does.

Supplementary Figures

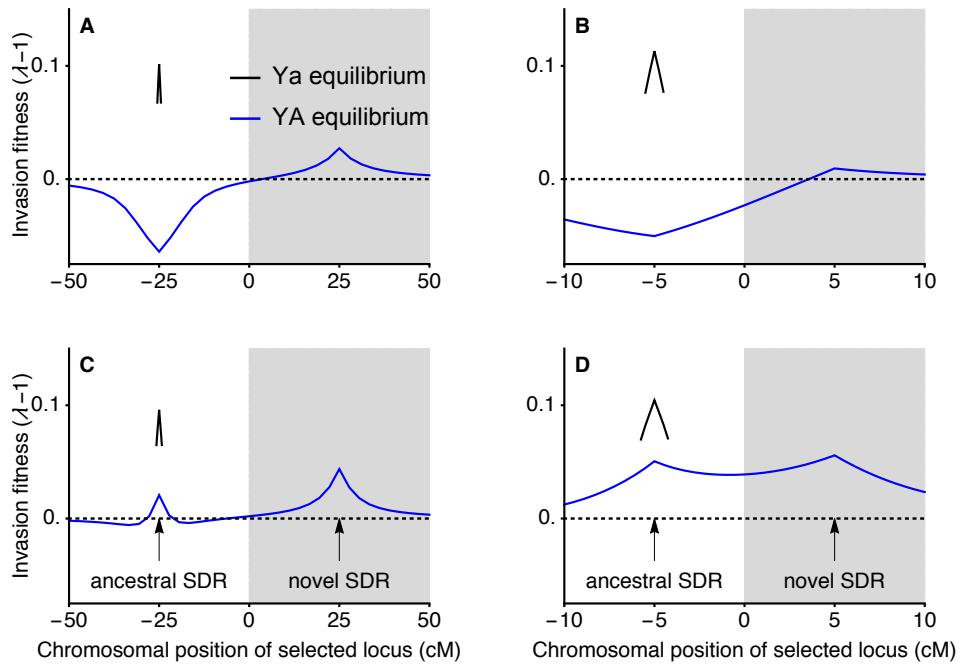


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ($r \approx 0$). In panels A and B, the a allele is favoured in females ($w_{aa}^{\text{♀}} = 1.05$, $w_{Aa}^{\text{♀}} = 1$, $w_{AA}^{\text{♀}} = 0.85$) and selection in males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

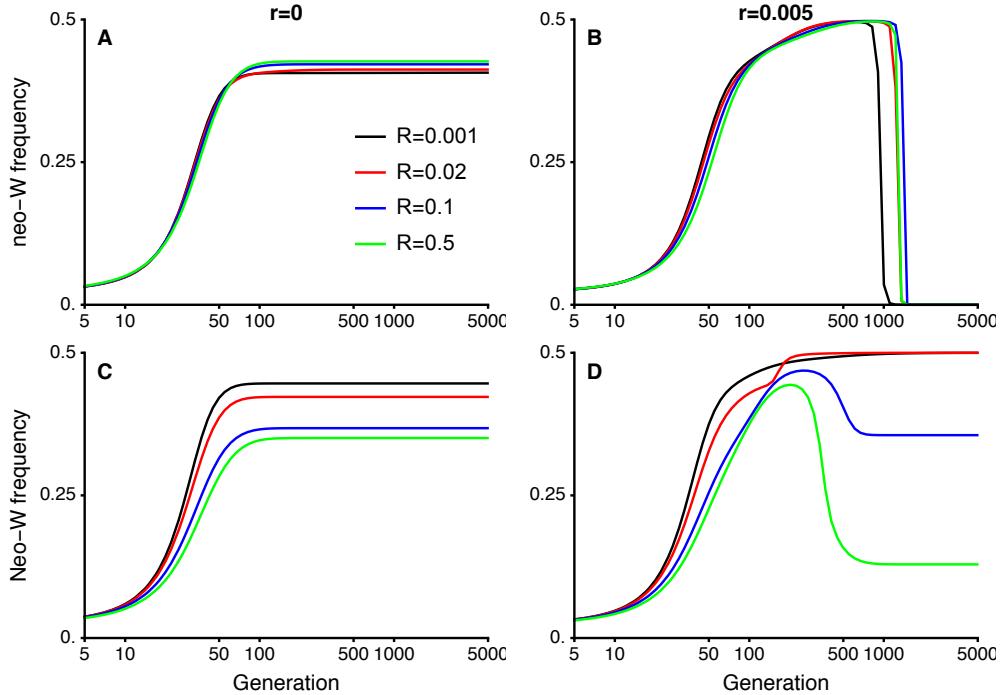


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among 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 ($w_{aa}^o = 1.05$, $w_{Aa}^o = 1$, $w_{AA}^o = 0.85$) and there is overdominant selection in males ($w_{aa}^\delta = w_{AA}^\delta = 0.75$) in panels A and B. In panels C and D, selection in males and females is overdominant ($w_{aa}^o = w_{AA}^o = 0.6$, $w_{aa}^\delta = 0.5$, $w_{AA}^\delta = 0.7$, $w_{Aa}^\delta = 1$). These parameters are marked by a dagger in Figure 2. Here, there is no haploid selection $t^\delta = \alpha_\Delta^\delta = 0$.

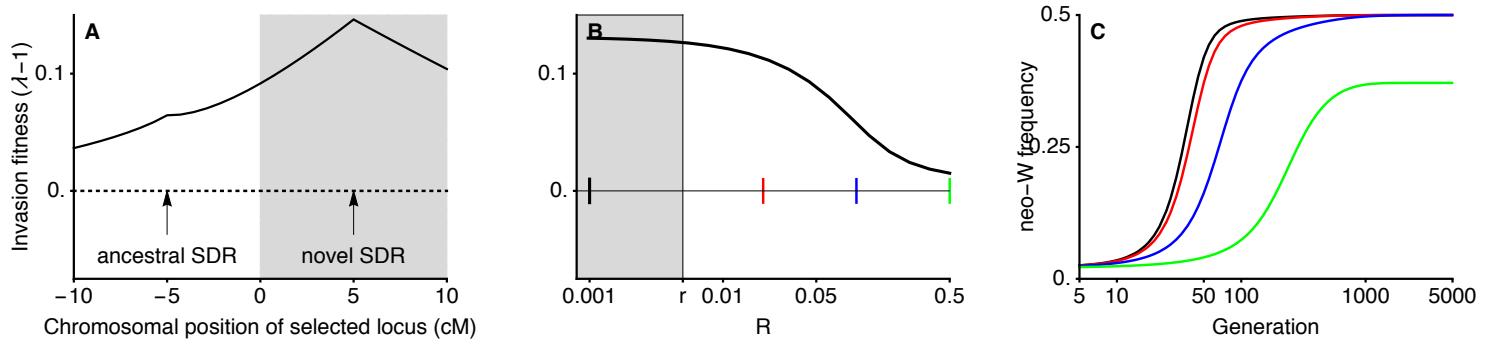


Figure S.3: When there is haploid selection and sexually-antagonistic selection, a neo-W may invade for any R . Check that we mention Sally's result that invasion cannot occur with sexually-antagonistic selection and $R = 1/2$ (currently only in legend for figure 1) Add asterisk to Figure S.4A to show parameters used in this plot. $w_{AA}^{\delta} = 1.05$, $w_{Aa}^{\delta} = 1$, $w_{aa}^{\delta} = 0.85$, $w_{AA}^{\sigma} = 0.85$, $w_{aa}^{\sigma} = 1.05$, $\alpha_{\Delta}^{\delta} = -0.08$.

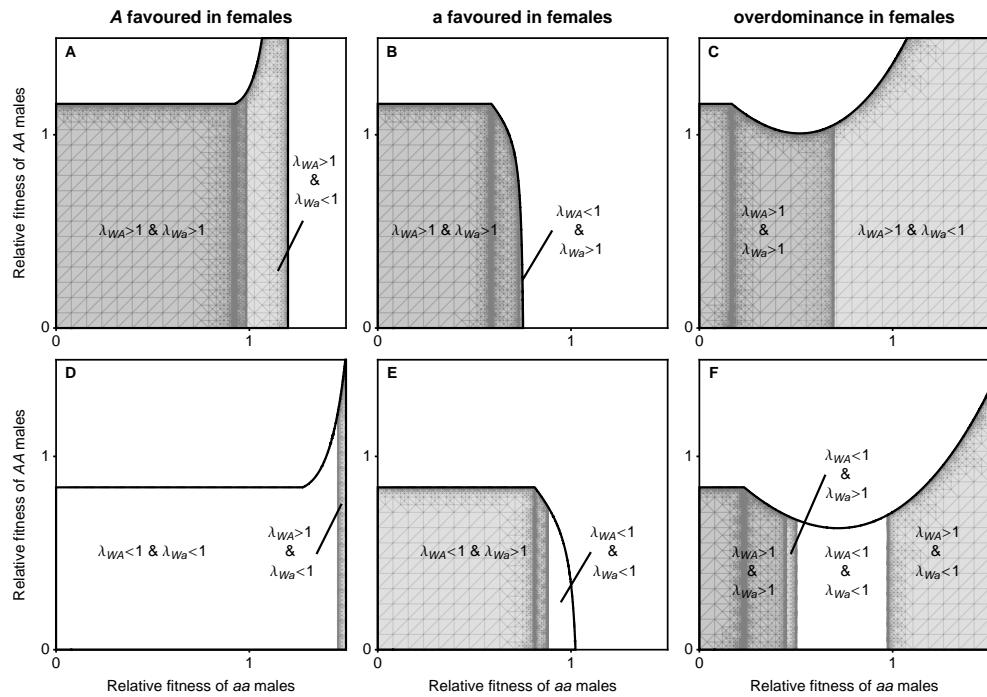


Figure S.4: ABC, $\alpha_{\Delta}^{\delta} = -0.08$ DEF, $\alpha_{\Delta}^{\delta} = 0.08$.

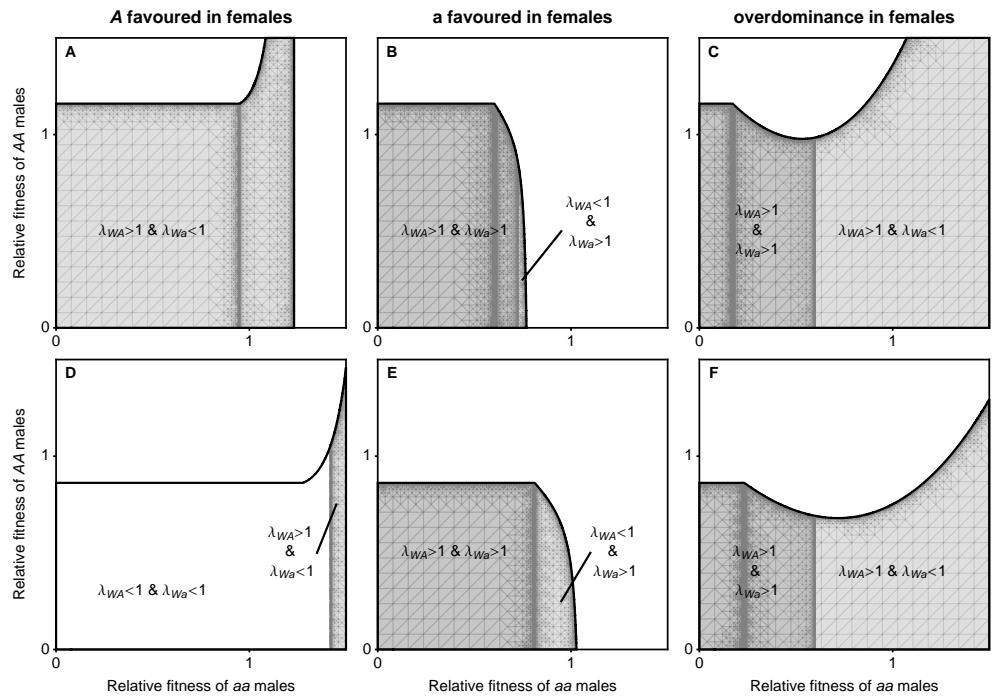


Figure S.5: ABC, $w_a^\delta = 1.16$, $w_A^\delta = 1$. DEF, $w_a^\delta = 1$, $w_A^\delta = 1.16$. I thought that re-running this with $w_a^\delta = 1.16$, $w_A^\delta = 0.84$ and $w_a^\delta = 0.84$, $w_A^\delta = 1.16$ will mean that it matches exactly with the meiotic drive example. Checking for 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. trouble is selection only in heterozygotes with drive?

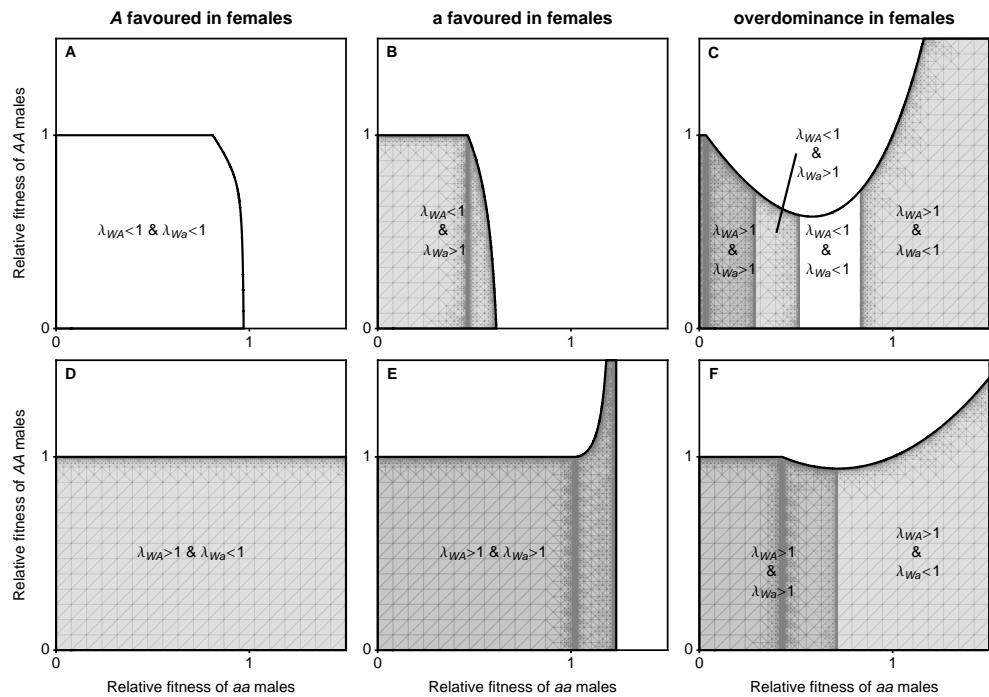


Figure S.6: ABC, $\alpha_{\Delta}^{\varnothing} = -0.08$ DEF, $\alpha_{\Delta}^{\varnothing} = 0.08$. λ s are increased for the haplotype that is favoured by female haploid selection, the stability conditions are also affected.

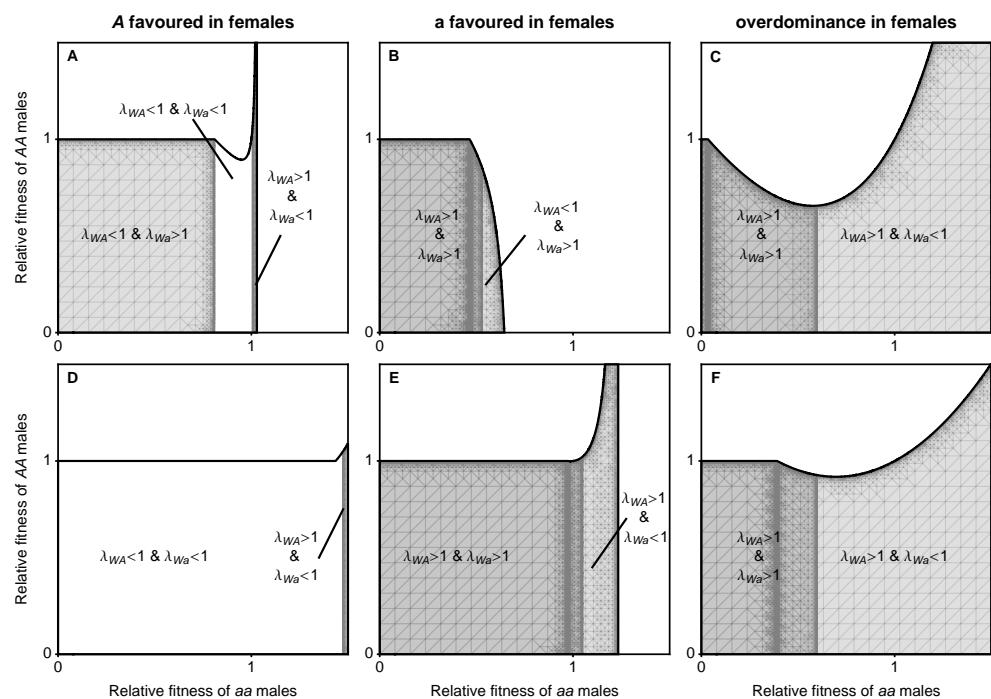


Figure S.7: ABC, $w_a^0 = 1.16$, $w_A^0 = 1$. DEF, $w_a^0 = 1$, $w_A^0 = 1.16$.

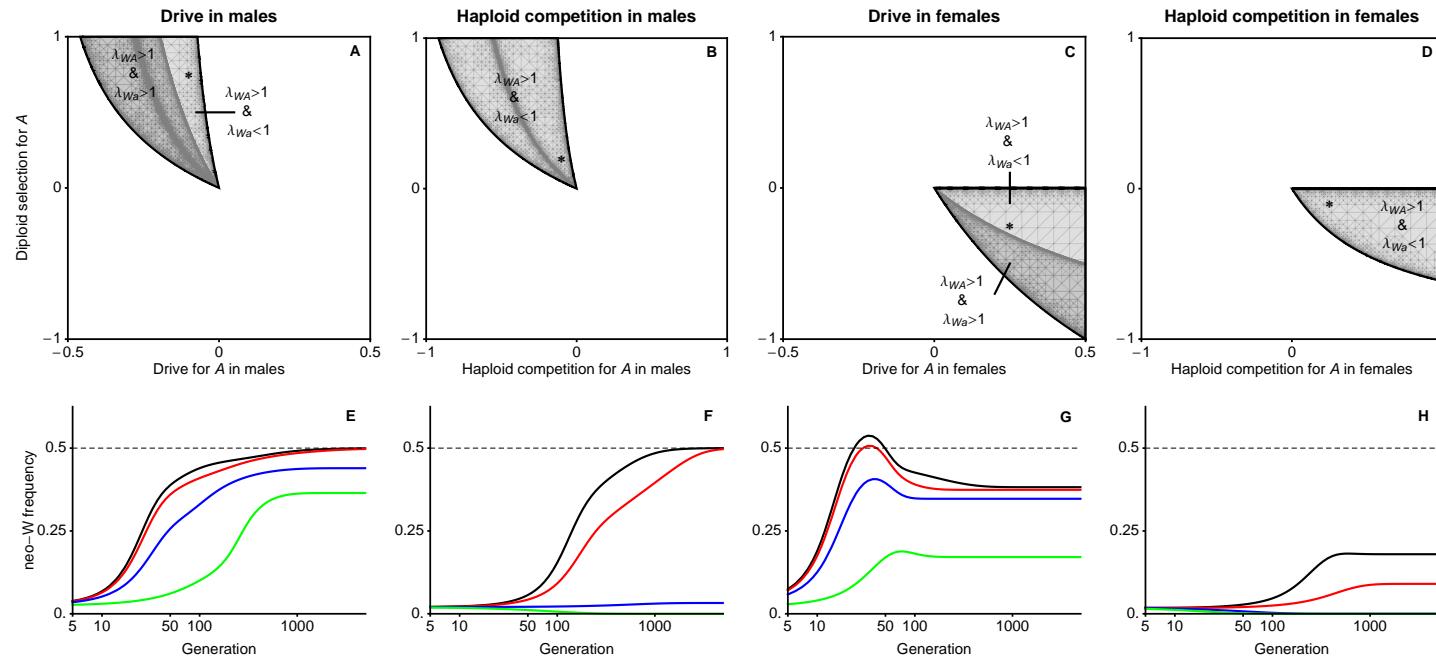


Figure S.8: A-D show when each of the neo-W haplotypes invade an internally stable equilibrium with a fixed on the Y (found by setting $r = 0$). The y-axis shows directional selection in diploids of both sexes, $s^{\vartheta} = s^{\delta}$, and the x-axes show sex-specific drive, $\alpha_{\Delta}^{\vartheta}$,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

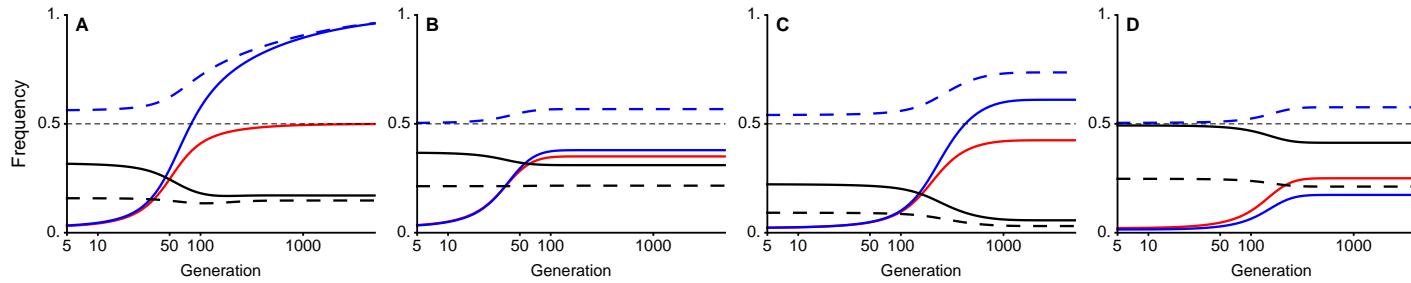


Figure S.9: Dynamics of all sex-determining alleles in each sex (males dashed). Red is neo-W, Blue is Y, Black is A. Panel A has the same parameters as the green curve in Figure 4B (ploidy-antagonism with male drive). Panel B has the same parameters as the green curve in Figure S.2C (overdominance in both sexes). Panel C has the same parameters as the green curve in Figure S.4C (sexual-antagonism with male drive). Panel D has the same parameters as the red curve in Figure S.8F, except $r = 0$ (ploidy-antagonism with pollen competition). Panel A shows complete sex-determination turnover (XY \rightarrow ZW) whereas panels B-D show the evolution of polymorphic sex-determining systems (X and Y still segregating and the neo-W has a frequency in females that is less than 1/2). All begin from equilibrium that would have a fixed on the Y if $r = 0$. All maintain a polymorphism at A locus.

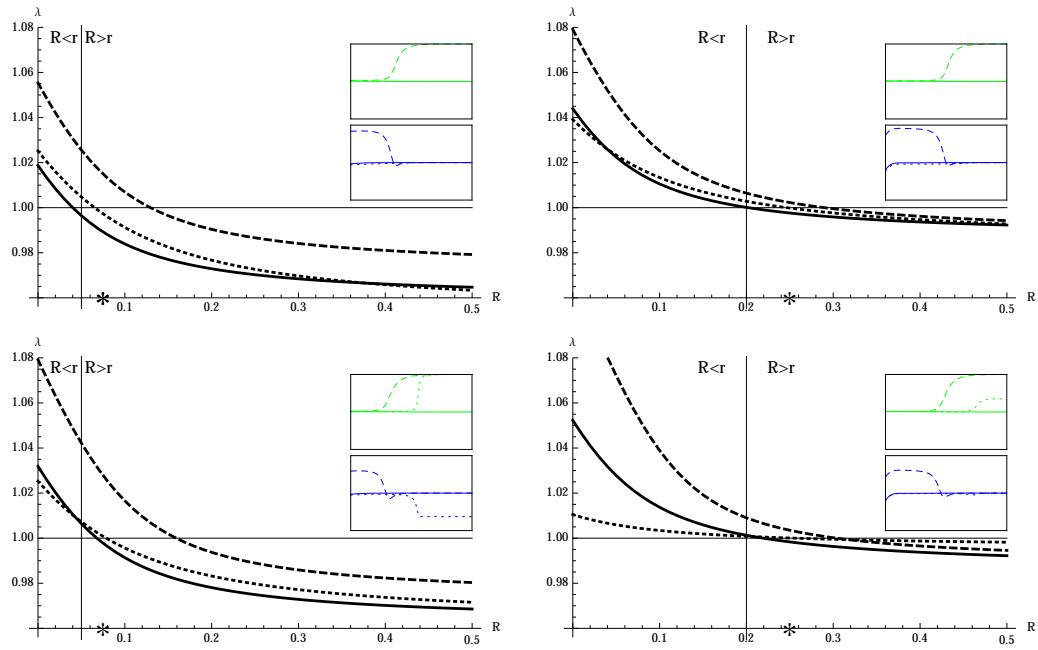


Figure S.10: [is this the one?](#)

1070 Add Sally's figure showing lambda for small r near equil A versus near equil
1072 B. Add references to this figure to appendix where we discuss whether lambdas
can be greater than 1 with sexually antagonistic selection. not sure which one you
are talking about, but see Figure S.10

1074 Perhaps it would also be useful to add an 8 panel figure that features ploidally
antagonistic selection. For each type of haploid selection (gametic competition/
1076 meiotic drive in males/females), give a regionplot where $h^{\delta} = h^{\varphi}$, e.g., $h^{\delta} =$
 $h^{\varphi} = 0.75$ (or perhaps the value of h we use in the regionplots we have, in which
1078 $w_{aa} = 0.85$, $w_{Aa} = 1$, $w_{AA} = 1.05$). Matt made a figure like this before but
both Ya and YA equilibria were plotted and there was no outline showing where
1080 the Ya equilibrium is stable (as in Figure 2). In Matts plot the axes were s^{φ} and
 $\alpha_{\Delta}^{\varphi}$. Add an asterisk to each region plot and show invasion in another panel, using
1082 those parameters and various R (e.g., in the stye of S.2). In an email, Sally has an
example of ploidally-antagonistic selection where the neo-W fixes and $R = 1/2$.
1084 This would cover that case and more. made an attempt (Figure S.8)

1086 We could also give versions of Figure 2 where there is also haploid selection
of various types. Haploid selection can favour A or a , so this would involve 4x
1088 6-panel figures. Started looking at this in Figure S.5 and Figure S.4, add female
haploid selection. Try to integrate into the discussion of haploid selection? e.g.,
1090 male haploid selection ones generally show effect of sex ratio, increasing both
lambdas when female biased (top rows). these figures are now done (S.4-S.7)
(ensuring frequencies between 0 and 1), but yet to discuss in text.

1092 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
1094 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-
1096 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
1098 competition that lead to a mixed sex-determination system. We should probably
have a short section in the appendix discussing this. made an attempt with Figure

₁₁₀₀ S.9, but yet to discuss in text