

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

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

* These authors contributed equally to this work

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

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

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

Contributions:

Abstract

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

Introduction

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

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

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

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

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

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

Genetic mapping experiments, which are typically designed to minimize selection in diploids, have revealed segregation distortion in various species, including mice, Drosophila, Rice, Maize, Wheat, Barley, Cotton... In some of these cases, 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 gametes only (Úbeda and Haig 2005, Lindholm et al. 2016). Gametic competition is also typically sex specific, occurring primarily among male gametes, because there are typically many more pollen/sperm than required for fertilization. Gametic competition may be particularly common in plants, in which 60-70% of all genes are expressed in the male gametophyte and these genes exhibit stronger signatures of selection than random genes (Borg et al. 2009, Arunkumar et al. 2013, Gossmann et al. 2014). In addition, artificial selection pressures applied to male gametophytes are known to cause a response to selection (e.g., Hormaza and Herrero 1996, Ravikumar et al. 2003, Hedhly et al. 2004, Clarke et al. 2004). A smaller proportion of genes are thought to be expressed and selected during competition in animal sperm, although precise estimates are uncertain (Zheng et al. 2001, Joseph and Kirkpatrick 2004, Vibranovski et al. 2010). Recent studies have demonstrated that sperm competition in animals can alter haploid allele frequencies and increase offspring fitness (Immler et al. 2014, Alavioon et al. 2017).

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

between sex-determination systems in a similar manner to sex-specific diploid selection (as explored by van Doorn and Kirkpatrick 2007; 2010). That is, 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, Fisherian sex ratio selection does not dominate and it is possible for selection on linked alleles to drive turnover between sex-determining systems despite causing increases in sex-ratio bias. In addition to considering haploid selection, another novel development in our model is that we consider loci that are in very tight linkage with the ancestral sex-determining region. We show that transitions between male and female heterogamety can then evolve despite the fact that the neo-sex-determining locus is less closely linked to a locus under selection and therefore disrupts favourable 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
₁₄₈ zygotes is determined by the genotype at the ancestral sex-determining region, **X**;
₁₅₀ XX genotypes become females and XY become males (or ZW become females and ZZ become males). To evaluate the evolution of new sex-determination systems,
₁₅₂ 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
₁₅₄ 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
₁₅₆ 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
₁₅₈ develop as females in a proportion (k) of the environments they experience.

In each generation, we census the genotype frequencies in male and female
₁₆₀ gametes/gametophytes (hereafter gametes) before gametic competition. A full description of our model, including recursion equations, is given in the Appendix.
₁₆₂ First, competition occurs among male gametes (sperm/pollen competition) and among female gametes (egg/ovule competition) separately. Selection during ga-
₁₆₄ 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
₁₆₆ fertilization during gametic competition, which assumes a polygamous mating system. Gametic competition in monogamous mating systems is, however, equivalent
₁₆₈ to meiotic drive in our model (described below), as both only alter the frequency of gametes produced by heterozygotes. After gametic competition, random mating
₁₇₀ 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
₁₇₂ 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
₁₇₄ 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
₁₇₆ **M** with probability R , and between loci **X** and **M** with probability ρ . Any linear

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

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

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

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

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 - or Sally's proof!). Here, $b = -(\lambda_{mA} + \lambda_{ma}) + (\chi_{mA} + \chi_{ma})$ and $c = (\lambda_{mA} - \chi_{mA})(\lambda_{ma} - \chi_{ma}) - \chi_{mA}\chi_{ma}$, where 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 ($k = 1$)
$\lambda_{mA} = [2(1 - \zeta)]^{-1} [\bar{p}^\delta w_A^\delta w_A^\varphi w_{AA}^\varphi + (1 - \bar{p}^\delta) w_a^\delta w_A^\varphi w_{Aa}^\varphi (1 + \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$
$\lambda_{ma} = [2(1 - \zeta)]^{-1} [(1 - \bar{p}^\delta) w_a^\delta w_a^\varphi w_{aa}^\varphi + \bar{p}^\delta w_A^\delta w_a^\varphi w_{Aa}^\varphi (1 - \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$
$\chi_{mA} = R[2(1 - \zeta)]^{-1} [(1 - \bar{p}^\delta) w_a^\delta w_A^\varphi w_{Aa}^\varphi (1 + \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$
$\chi_{ma} = R[2(1 - \zeta)]^{-1} [\bar{p}^\delta w_A^\delta w_a^\varphi w_{Aa}^\varphi (1 - \alpha_\Delta^\varphi)] / (\bar{w}_H^\varphi \bar{w}_H^\delta \bar{w}^\varphi)$

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

ζ is the zygotic sex ratio (fraction male)

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

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

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We are particularly concerned with the conditions under which a rare neo-sex-determining allele increases in frequency, which occurs when the largest eigenvalue, λ , is greater than one. Given the characteristic polynomial $f(\lambda) = \lambda^2 + b\lambda + c$ and the Perron-Forbenius theorem (guaranteeing that the leading eigenvalue is positive, unique, and real), at least one solution to $f(\lambda) = 0$ is greater than one when the polynomial has a negative slope or negative value at $\lambda = 1$ ($f'(1) = 2 + b < 0$ or $f(1) = 1 + b + c < 0$). Regardless the rate of recombination, at least one of these conditions is true if both haplotypes can spread ($\lambda_{mA}, \lambda_{ma} > 1$) and neither can be true if neither haplotype can spread ($\lambda_{mA}, \lambda_{ma} < 1$). If only one haplotype can spread then the new sex-determining allele increases in frequency on one A

background and declines on the other. Invasion then occurs if

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

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 rate of decline of *ma* haplotypes ($1 - \lambda_{ma}$) and the rate of loss of *mA* haplotypes due to recombination (χ_{mA}).

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, \hat{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^F , \hat{p}_X^D , and \hat{p}_Y^D) 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
260 can be maintained by mutation-selection balance or transiently present during the spread of beneficial alleles. However, polymorphisms maintained by selection can
262 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
264 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 $\epsilon \ll 1$).
268

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

The ancestral equilibrium allele frequencies and their stability conditions are given
272 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
274 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 ($\hat{p}_X^{\varphi} = \hat{p}_X^{\delta} = 1$) or polymorphic ($0 < \hat{p}_X^{\varphi}, \hat{p}_X^{\delta} < 1$).

278 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
280 ($\lambda_{mA} < 1$) given that the initial equilibrium is stable. Therefore, a neo-Y mutation cannot spread ($\lambda \leq 1$) in an ancestral XY system that is at equilibrium with all selected loci within the non-recombining region around the SDR. In essence, through
282 tight linkage with the **A** locus, the ancestral Y becomes strongly specialized on the allele that has the highest fitness across male haploid and diploid phases. Given
284 that the ancestral Y is at this equilibrium, it is not possible for a neo-Y to create
286

288 males that have higher fitness than the ancestral Y.

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

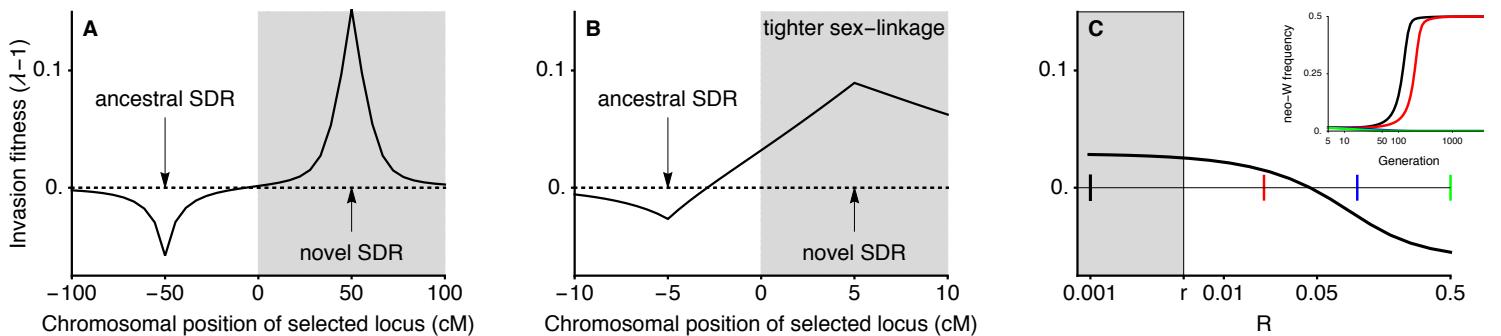


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is less tightly linked to a locus under sexually-antagonistic selection (here, without haploid selection). In panel A, linkage is loose enough relative to selection that the analytical results assuming weak selection hold, and a neo-W can only invade when it is more tightly linked with the selected locus ($R < r$; shaded region). In panel B, linkage is tight enough relative to selection that the analytical results assuming weak selection do not hold, and a neo-W can invade even when it is less tightly linked with the selected locus ($r < R$; unshaded region). In panel C we vary the recombination rate between the neo-W and the selected locus (R) for a fixed recombination rate between the ancestral-SDR and the selected locus ($r = 0.005$). Coloured markers show recombination rates for which the temporal dynamics of invasion are plotted in the inset, demonstrating that neo-W alleles can fix (reach frequency 0.5 among female gametes) if they are more (black) or less (red) closely linked to a locus experiencing sexually-antagonistic selection. A very loosely linked neo-W does not spread in this case (blue and green lines overlap and go to 0). Indeed, we can show that neo-W invasion fitness is always negative when $R = 1/2$ and there is sex-antagonism but no haploid selection (see supplementary *Mathematica* file). Fitness parameters are shown by an asterisk in Figure 2A: $w_{AA}^{\delta} = 1.05$, $w_{aa}^{\delta} = 1.2$, $w_{aa}^{\delta} = w_{AA}^{\delta} = 0.85$, $w_{Aa}^{\delta} = 1$, $t^{\delta} = \alpha_{\Delta}^{\delta} = 0$.

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

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

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

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

330 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
332 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-
334 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
336 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).

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

haploid selection?

356 Loose linkage with the ancestral sex-determining region

Assuming that selection is weak relative to all recombination rates (r , R and ρ),
358 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.

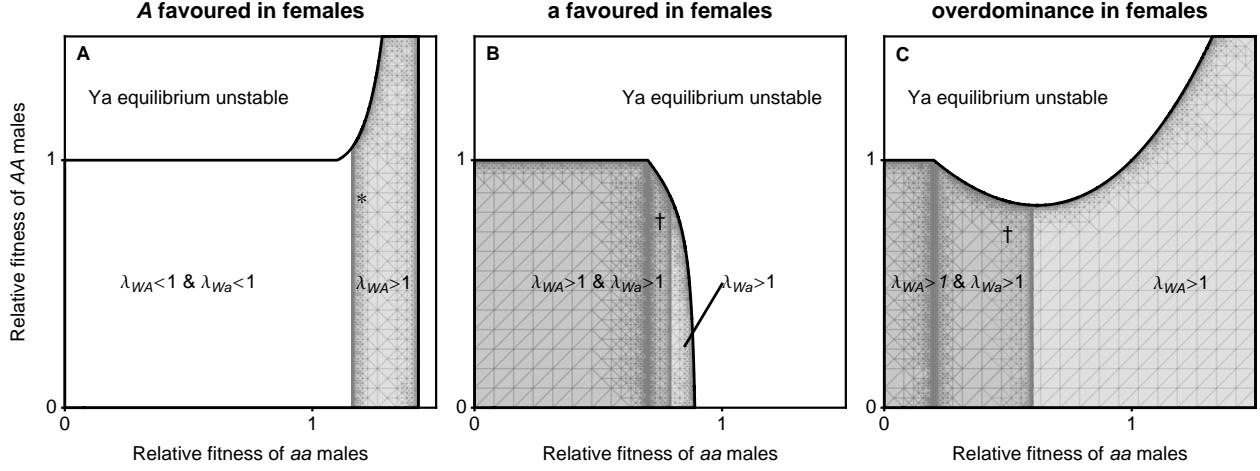


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ($r = 0$), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ($w_{Aa}^{\delta} = 1$) and only consider stable equilibria at which both A locus alleles are maintained and the a allele is initially fixed on the Y, region outlined. Here, selection in females can favour the A allele (panel A, $w_{aa}^{\delta} = 0.85$, $w_{AA}^{\delta} = 1.05$), favour the a allele (panel B, $w_{aa}^{\delta} = 1.05$, $w_{AA}^{\delta} = 0.85$), or be overdominant (panel C, $w_{aa}^{\delta} = w_{AA}^{\delta} = 0.6$). If λ_{wA} or λ_{wa} is greater than one, then a rare neo-W can spread for, at least, some values of $R > r$. For the parameter values marked with an asterisk, example invasion dynamics are shown in Figure 1C. Where both λ_{wA} and λ_{wa} are greater than one, a neo-W will spread when rare, regardless of linkage with the selected locus (for any R). Figure S.1 shows two examples using the parameters marked with a dagger. Here, there is no haploid selection $t^{\delta} = \alpha_{\Delta}^{\delta} = 0$.

³⁶⁰ To leading order in selection, these are:

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

and

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

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

366 in fitness between A and a alleles in diploids of sex $\delta \in \{\text{♀}, \text{♂}\}$, where \bar{p} is the
 367 leading-order probability of mating with an A -bearing gamete from the opposite
 368 sex (equation S.4). The difference in A -allele-frequency among Y-bearing sperm
 369 versus X-bearing sperm is given by $\hat{p}_Y^\delta - \hat{p}_X^\delta = V_A(D^\delta - D^\text{♀} + \alpha_\Delta^\delta - \alpha_\Delta^\text{♀} + t^\delta -$
 370 $t^\text{♀})(1 - 2r)/2r$.

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

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

Equation (3) shows that, with weak selection, neo-W alleles can invade an XY
 390 system for a large number of selective regimes. To clarify the parameter space
 391 under which $\lambda_{W',XY} > 1$, we consider several special cases. Firstly, if the A locus is
 392 unlinked to the ancestral sex-determining region ($r = 1/2$), a more closely linked
 393 neo-W ($R < 1/2$) can always invade because there is no ancestral association
 394 between A alleles and sex chromosomes in males, $(\hat{p}_Y^\delta - \hat{p}_X^\delta) = 0$, see equation
 (S.5). The second term in equation (3) therefore disappears and invasion depends

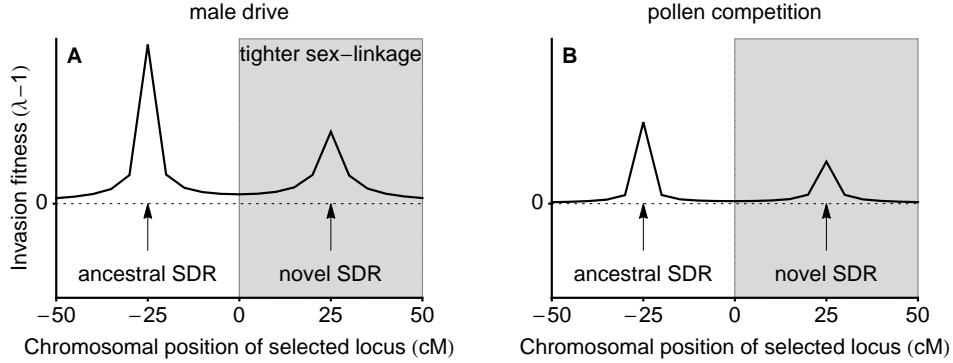


Figure 3: Ploidally-antagonistic selection allows a less tightly linked neo-W to invade. In panel A, male drive ($\alpha_{\Delta}^{\delta} = -1/20$, $t^{\delta} = \alpha_{\Delta}^{\delta} = 0$) opposes selection in diploids (no sex-differences: $s^{\delta} = 1/10$, $h^{\delta} = 7/10$), in which case the neo-sex-determining allele can invade regardless of linkage. In panel B, gametic competition in males ($t^{\delta} = -1/10$, $t^{\varphi} = \alpha_{\Delta}^{\varphi} = 0$) opposes selection in diploids (sex-differences: $s^{\delta} = 1/20$, $s^{\varphi} = 3/20$, $h^{\delta} = 7/10$), in which case the neo-sex-determining allele can once again invade regardless of linkage. We use Haldane's map function (Equation 3 in Haldane 1919) to convert from map distance (centiMorgans, cM) to the probability of recombination (an odd number of cross-over events).

only on the sign of $(r - R)$. Indeed, invasion typically occurs when the neo-W is more closely linked to the selected locus than the ancestral sex-determining region (Figure 3).

Secondly, we can simplify the discussion of cases where invasion occurs despite looser sex-linkage, $R > r$, by focusing on the special case where $R = 1/2$ and $r < 1/2$ (e.g., the selected locus is on the ancestral sex chromosome and the novel sex-determining locus arises on an autosome). In Table 3 we give the conditions where invasion occurs when we further assume that haploid selection only occurs in one sex (e.g., during male meiosis only) and dominance coefficients are equal in the two sexes, $h^{\varphi} = h^{\delta}$. When there is no gametic competition and meiotic drive is in one sex only, an unlinked neo-W can invade as long as the same allele is favoured during diploid selection in males and females ($s^{\varphi}s^{\delta} > 0$, see Figure 3A and Figure 4B). When there is no meiotic drive and gametic competition occurs in one sex only, an unlinked neo-W can invade as long as the same allele is favoured in male and female diploid selection and there are sex differences in selection of one type (e.g., $s^{\varphi}(s^{\delta} - s^{\varphi}) > 0$, see Figure 3B). These special cases indicate that

- ⁴¹² neo-W invasion occurs for a relatively large fraction of the parameter space, even if the neo-W uncouples the sex-determining locus from a locus under selection.

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

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

⁴¹⁴ 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, ⁴¹⁶ 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 ⁴¹⁸ ancestral-SDR ($r < 1/2$) and experiences meiotic drive in males only (e.g., during spermatogenesis but not during oogenesis, $\alpha_\Delta^\delta \neq 0, \alpha_\Delta^q = 0$), without gametic ⁴²⁰ competition ($t^q = t^\delta = 0$). In this case, the zygotic sex ratio can be initially biased only if the ancestral sex-determining system is XY (Figure 4B). We might therefore ⁴²² 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 ⁴²⁴ 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 ⁴²⁶ 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 ⁴²⁸ 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 ⁴³⁰ 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 ⁴³² 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

434 sex ratio evolves to become biased towards males.

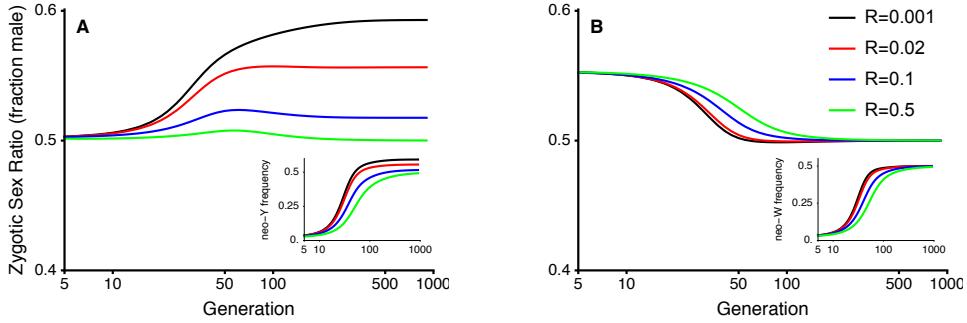


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidally antagonistic with haploid selection favouring the a allele during male meiosis. In panel A, male drive in an ancestral ZW system has no affect on the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

The green curves in Figure 4 shows transitions between male and female heterogametey even though the new sex-determining region is unlinked to a locus that experiences haploid and diploid selection. We use these green curves to discuss why heterogametic transitions can occur when $R = 1/2$ and $r < 1/2$, as in Table 3. In Figure 4B, an unlinked neo-W can spread because the zygotic sex ratio is ancestrally male biased. In Figure 4A, an unlinked neo-Y spreads despite the fact that the ancestral zygotic sex ratio is even. In this case, the the male meiotic drive allele, a , is initially more common among ancestral-Z-bearing eggs than ancestral-W-bearing eggs because the Z is found in males more often than the W ($\hat{p}_W^{\varphi} - \hat{p}_Z^{\varphi} > 0$, equation S.5). Polymorphism at the A locus is maintained by counter-selection against the a allele in diploids and therefore ancestral-ZZ males have generally low diploid fitness. The neo-Y spreads because it produces males with high diploid fitness through matings with ancestral-W-bearing female gametes, which are more likely to carry the A allele. A freely recombining neo-Y ($R = 1/2$) is equally likely to be segregate with the A or a allele and is therefore unaffected by male

450 meiotic drive. Thus, a key factor in explaining why heterogametic transitions can
 451 occur when $R > r$ is that the neo-SDR determines sex in the diploid phase but
 452 recombination occurs before any subsequent haploid selection.

Environmental sex determination

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

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

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

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

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

spread of which is given by

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

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

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

⁴⁹⁴ **Discussion**

Two predominant theories explaining the remarkably high frequency of transitions between sex-determination systems are sexually-antagonistic selection and sex-ratio selection (reviewed in Blaser et al. 2012, van Doorn 2014). The former predicts that neo-sex-determining alleles can invade when they arise in closer linkage with a sexually-antagonistic locus (van Doorn and Kirkpatrick 2007; 2010). The latter predicts that new sex-determining systems are generally favoured if they result in more equal sex-ratios than the ancestral system. Firstly, we show that selection (including sexually-antagonistic selection) on loci within or near the non-recombining region of the ancestral sex-determining region can favour heterogametic transitions (XY to ZW or ZW to XY) to new sex-determining systems that are less closely linked to the selected loci (e.g., see Figure 1). Secondly, assuming that selection is weak relative to recombination ('weak selection'), we show that new sex-determining alleles are typically favoured if they are more closely linked to a locus under haploid selection, which is the only condition favouring homogametic transitions (XY to XY or ZW to ZW). In addition, with haploid selection and weak selection, heterogametic transitions (XY to ZW or ZW to XY) can occur even when the new sex-determining region is less closely linked to the locus under selection (e.g., see Figure 4).

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

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

We have shown that the spread of new sex determination systems can be driven by loci experiencing haploid selection. Because haploid selection can cause transitions that increase or decrease sex-linkage, haploid selection may lead to less stability, and greater potential for cycling, in sex-determination systems (e.g., the final state of the red line in Figure 4A is the starting state in Figure 4B). In particular, if haploid selection is strong but selective differences between male and female diploids are weak, we find that heterogametic transitions (XY to ZW or vice versa) are favoured more strongly than homogametic transitions (e.g., with $|D^\delta - D^0| << |\alpha_\Delta^\delta - \alpha_\Delta^0 + t^\delta - t^0|$ we have $\lambda_{W',XY} > \lambda_{Y',XY}$; Equations 3 and S.5). Turnovers driven by haploid selection may help to explain the relative rarity of heteromorphic sex chromosomes in plants, which are thought to experience more selection during their multicellular haploid stage. For example, among relatively few dioecious clades in which multiple species have well characterized sex chromosomes (Ming et al. 2011), heterogametic transitions have been inferred in *Silene* subsection *Otites* (Slancarova et al. 2013) and in *Salicaceae* (Pucholt et al. 2015; 2017). Furthermore, assuming that transitions from dioecy to hermaphroditism (equal parental investment in male and female gametes) are favoured in a similar manner to the ESD examined here (equal probability of zygotes developing as males or females), our results suggest that competition during the haploid stage

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

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

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

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

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

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

614 purely sexually-antagonistic selection, eliminating the need for differences in se-
615 lection between male and female diploids. Perhaps counterintuitively, transitions
616 involving haploid selection can be driven by sex-ratio selection or cause sex-ratio
617 biases to evolve. We conclude that haploid selection should be considered as a
618 pivotal factor driving transitions between sex-determination systems. Overall, our
619 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.

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Appendix

844 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

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

Selection among diploids then occurs according to the diploid genotype at the
 880 **A** locus, $l \in \{AA, Aa, aa\}$, for an individual of type ij (see Table 1). The diploid
 881 frequencies after selection in sex φ are given by $xx_{ij}^{\varphi,s} = w_l^\varphi xx_{ij}/\bar{w}^\varphi$, $xy_{ij}^{\varphi,s} =$
 882 $w_l^\varphi xy_{ij}/\bar{w}^\varphi$, and $yy_{ij}^{\varphi,s} = w_l^\varphi yy_{ij}/\bar{w}^\varphi$, where $\bar{w}^\varphi = \sum_{i=1}^4 \sum_{j=1}^4 w_l^\varphi xx_{ij} + w_l^\varphi xy_{ij} +$
 883 $w_l^\varphi yy_{ij}$ is the mean fitness of individuals of sex φ .

884 Finally, these diploids undergo meiosis to produce the next generation of ga-
 885 metes. Recombination and sex-specific meiotic drive occur during meiosis. Here,
 886 we allow any relative locations for the SDR, **A**, and **M** loci by using three param-
 887 eters to describe the recombination rates between them. R is the recombination
 888 rate between the **A** locus and the **M** locus, ρ is the recombination rate between
 889 the **M** locus and the **X** locus, and r is the recombination rate between the **A** locus
 890 and the **X** locus. Table S.1 shows replacements that can be made for each possi-
 891 ble ordering of the loci assuming that there is no cross-over interference. During
 892 meiosis in sex φ , meiotic drive occurs such that, in Aa heterozygotes, a fraction
 893 α^φ of gametes produced carry the A allele and $(1 - \alpha^\varphi)$ carry the a allele.

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

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

894 Among gametes from sex φ , the frequencies of haplotypes (before gametic

competition) in the next generation are given by

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

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

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

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

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

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

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

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

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

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

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

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

896

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

Resident equilibrium and stability

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

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

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

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

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

bility can be calculated analytically without assuming anything about the relative
918 strengths of selection and recombination. However, here, we focus on two regimes
(tight linkage and weak selection) in order to make fewer assumptions about fit-
920 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-
922 tral population when the recombination rate between the X and A loci is small
924 (r of order ϵ). Selection at the A locus will not affect evolution at the novel sex-
determining locus, M, if one allele is fixed on all backgrounds. We therefore focus
926 on the five equilibria that maintain both A and a alleles, four of which are given to
leading order by:

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Selection weak relative to recombination (weak selection)

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

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

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

⁹⁶⁶ 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
⁹⁶⁸ 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

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

⁹⁷⁰ 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
⁹⁷² or drive ($\alpha_\Delta^\varphi = t^\varphi = 0$) our results reduce to those of van Doorn and Kirkpatrick
⁹⁷⁴ (2007).

980 **Invasion conditions**

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

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

992 For tight linkage between the ancestral sex-determining locus and the selected
locus we can calculate each of these terms exactly, while for weak selection we
994 take a Taylor series of the leading eigenvalue. With weak selection, the leading
eigenvalue, λ , for any k , is given up to order ϵ^2 by equation (4).

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

Here, we explore the conditions under which a neo-W invades an XY system as-
998 suming that the A locus is initially in tight linkage with the ancestral sex-determining
region ($r \approx 0$). We disregard neo-Y mutations, which never spread given that the
1000 ancestral population is at a stable equilibrium (see supplementary *Mathematica*
notebook for proof).

1002 Starting with the simpler equilibrium (B), the terms of the characteristic poly-
nomial are

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

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

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

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

1004 Haploid selection impacts the spread of neo-W haplotypes in three ways. Firstly,
 1006 the zygotic sex ratio becomes male biased, $\zeta > 1/2$, when the a allele (which is
 1008 fixed on the Y) is favoured during competition among male gametes or by meiotic
 drive in males. Specifically, at equilibrium (B), the sex ratio is $\zeta = w_a^\delta(1 -$
 1010 $\alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ where $2\bar{w}_H^\delta = [w_a^\delta(1 - \alpha_\Delta^\delta) + w_A^\delta(1 + \alpha_\Delta^\delta)]$ has been canceled out in
 equations (S.6) to leave the term $[w_a^\delta(1 - \alpha_\Delta^\delta)]^{-1}$. Male biased sex ratios facilitate
 1012 the spread of a neo-W because neo-W alleles cause the zygotes that carry them to
 1014 develop as the rarer, female, sex.

1012 Secondly, haploid selection in females selects on neo-W haplotypes directly. At
 1014 equilibrium (B), the fitness of female gametes under the ancestral sex-determining
 1016 system is w_A^φ such that the relative fitnesses of neo-W- A and neo-W- a haplotypes
 1018 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
 the A versus a alleles, which will be produced by heterozygous females in propor-
 1020 tions $(1 + \alpha_\Delta^\varphi)/2$ and $(1 - \alpha_\Delta^\varphi)/2$, respectively. These terms are only associated with
 1022 heterozygous females, i.e., they are found alongside w_{Aa}^φ .

1020 Thirdly, haploid selection in males affects the diploid genotypes of females
 1022 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

1024 $w_a^\delta(1 - \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$, where the $2\bar{w}_H^\delta$ terms have been canceled in equation (S.6)
 (as mentioned above). Thus, for example, neo-W-A haplotypes are found in *AA*
 1026 female diploids with probability $w_A^\delta(1 + \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ (first term in square brackets
 in the numerator of equation S.6a) and in *Aa* female diploids with probability
 1028 $w_a^\delta(1 - \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ (see equation S.6c and the second term in square brackets in
 the numerator of equation S.6a).

1030 The other terms in equations (S.6) are more easily interpreted if we assume that
 there is no haploid selection in either sex, in which case $\lambda_{mA} > 1$ when $w_{Aa}^? > w_{AA}^?$
 1032 and $\lambda_{ma} > 1$ when $(w_{Aa}^? + w_{aa}^?)/2 > w_{AA}^?$. These conditions cannot be met under
 purely sexually-antagonistic selection, where *A* is directionally favoured in females
 1034 ($w_{AA}^? > w_{Aa}^? > w_{aa}^?$) and *a* is directionally favoured in males ($w_{AA}^\delta > w_{Aa}^\delta > w_{aa}^\delta$).
 Essentially, the X is then already as specialized as possible for the female beneficial
 1036 allele (*A* is fixed on the X), and the neo-W often makes daughters with the Y-*a*
 haplotype, increasing the flow of *a* alleles into females, which reduces the fitness
 1038 of those females.

If selection doesn't uniformly favour *A* in females, however, neo-W-*A* haplotypes and/or neo-W-*a* haplotypes can spread ($\lambda_{mA} > 1$ and/or $\lambda_{ma} > 1$) at this equilibrium. A neo-W can spread alongside the *A* allele ($\lambda_{mA} > 1$), despite the fact that a neo-W brings Y-*a* haplotypes into females, when $w_{Aa}^? > w_{AA}^?$, as stated 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), 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 on the Y). Specifically, from the stability conditions for equilibrium (B), we must have $w_{Aa}^? < 2w_{AA}^?$ and $w_{Aa}^\delta / [(w_{aa}^\delta + w_{Aa}^\delta)/2] > w_{Aa}^? / w_{AA}^?$.

Still considering $w_{Aa}^? > w_{AA}^?$, the neo-W can also spread alongside the *a* allele ($\lambda_{ma} > 1$) if $w_{aa}^?$ is large enough such that $(w_{Aa}^? + w_{aa}^?)/2 > w_{AA}^?$. This can occur with overdominance or directional selection for *a* in females (Figure 2B,C). 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-

1054 W can spread with a because it produces female aa individuals by capturing Y- a
 1055 haplotypes. mike, could you check this paragraph? i fixed an old problem, and
 1056 want to make sure i haven't introduced any new errors

Similar equations can be derived for equilibrium (A) by substituting the equi-
 1058 librium allele frequencies into Table 2

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

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

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

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

where

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

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

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

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

1060 As with equilibrium (B), haploid selection again modifies invasion fitnesses
 1061 by altering the sex-ratio and the diploid genotypes of females and directly select-
 1062 ing upon female gametes. The only difference is that resident XX females are no
 1063 longer always homozygote AA and males are no longer always heterozygote Aa .
 1064 Thus the effect of haploid selection in males is reduced, as is the difference in fit-
 1065 ness between neo-W haplotypes and resident X haplotypes, as both can be on any
 1066 diploid or haploid background.

The other terms are easier to interpret in the absence of haploid selection. For

¹⁰⁶⁸ instance, without haploid selection, the neo-W-*A* haplotype spreads ($\lambda_{mA} > 1$) if and only if

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

¹⁰⁷⁰ where $\phi - \psi = w_{AA}^{\varphi}w_{Aa}^{\delta} - w_{aa}^{\varphi}w_{aa}^{\delta}$ and both ϕ and ψ are positive when equilibrium (A) is stable. In contrast to equilibrium (B), a neo-W haplotype can spread under ¹⁰⁷² purely sexually-antagonistic selection ($w_{aa}^{\varphi} < w_{Aa}^{\varphi} < w_{AA}^{\varphi}$ and $w_{AA}^{\delta} < w_{Aa}^{\delta} < w_{aa}^{\delta}$). The neo-W-*A* can spread as long as it becomes associated with females that bear ¹⁰⁷⁴ more *A* alleles than observed at equilibrium (A).

¹⁰⁷⁶ Without haploid selection, the neo-W-*a* haplotype spreads ($\lambda_{ma} > 1$) if and only if

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

¹⁰⁷⁸ 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 circumstances. For example, with overdominance in males there is selection for ¹⁰⁸⁰ increased *A* frequencies on X chromosomes in males, which are always paired with Y-*a* haplotypes. Directional selection for *a* in females can then maintain a ¹⁰⁸² polymorphism at the A locus on the X. This scenario selects for a modifier that increases recombination between the sex chromosomes (e.g., blue region of Figure ¹⁰⁸⁴ 2d in Otto 2014) and facilitates the spread of neo-W-*a* haplotypes, which create more females bearing more *a* alleles than the ancestral X chromosome does.

¹⁰⁸⁶ Supplementary Figures

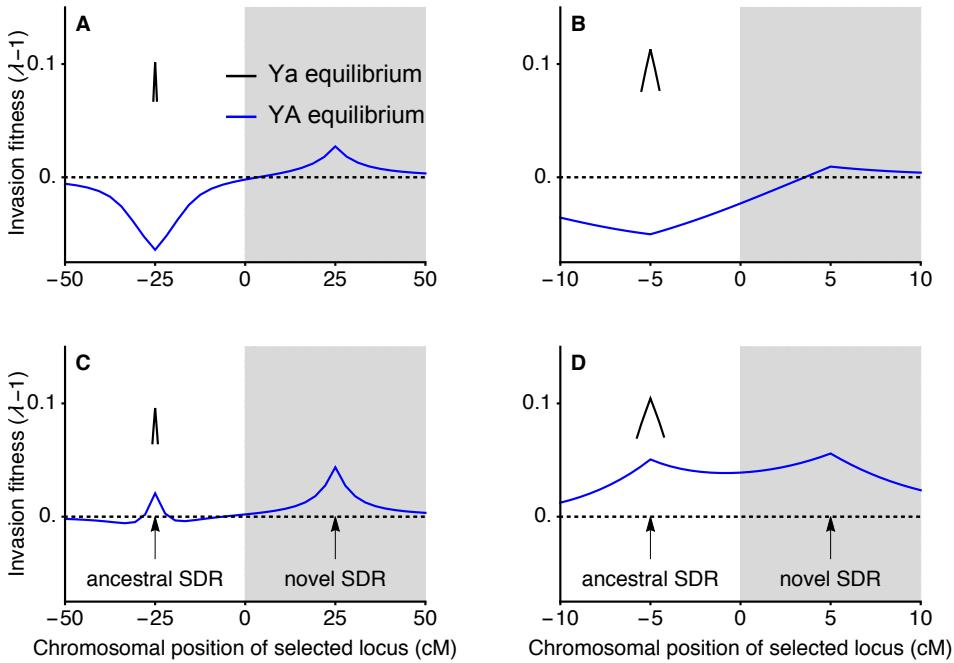


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ($r \approx 0$). In panels A and B, the a allele is favoured in females ($w_{aa}^{\varnothing} = 1.05$, $w_{Aa}^{\varnothing} = 1$, $w_{AA}^{\varnothing} = 0.85$) and selection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

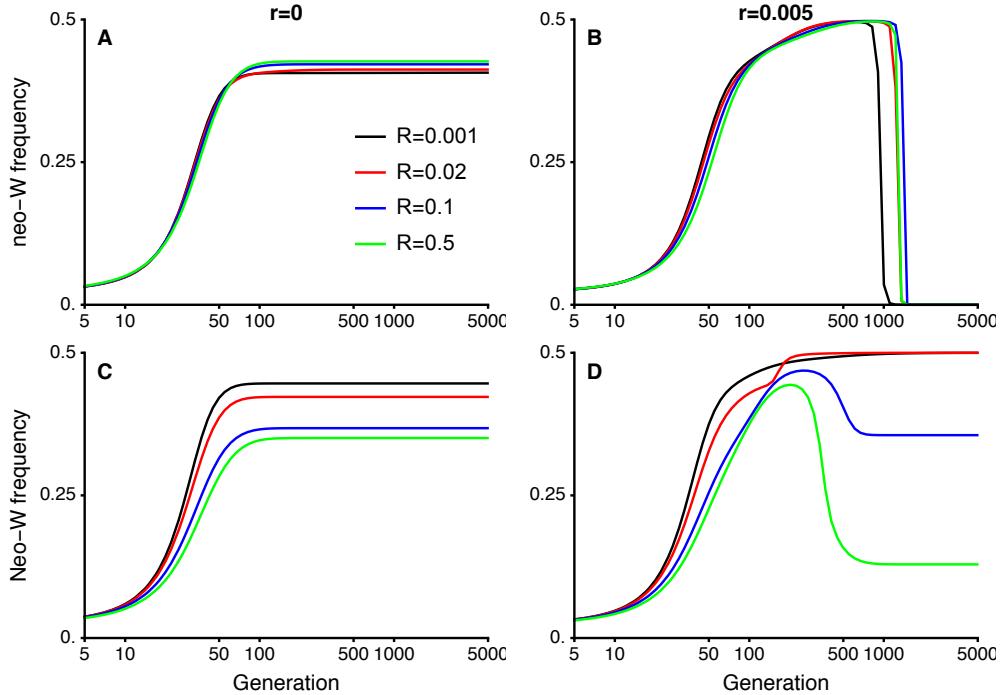


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among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

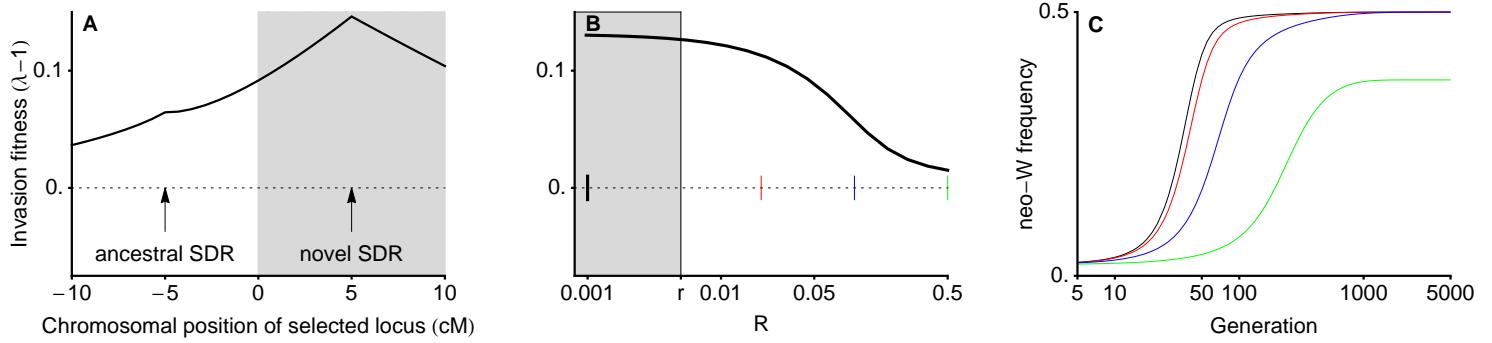


Figure S.3: When there is sexually-antagonistic selection and haploid selection, a neo-W may invade for any R . Check that we mention Sally's result that invasion cannot occur with sexually-antagonistic selection and $R = 1/2$ (currently only in legend for figure 1). This is now discussed in a round-about way at the beginning of the tight linkage invasion section in the main text. Parameters given by asterisk in Figure S.4: $w_{AA}^{\varphi} = 1.05$, $w_{Aa}^{\varphi} = 1$, $w_{aa}^{\varphi} = 0.85$, $w_{AA}^{\delta} = 0.85$, $w_{Aa}^{\delta} = 1.05$, $w_{aa}^{\delta} = 0.85$, $\alpha_{\Delta}^{\varphi} = -0.08$.

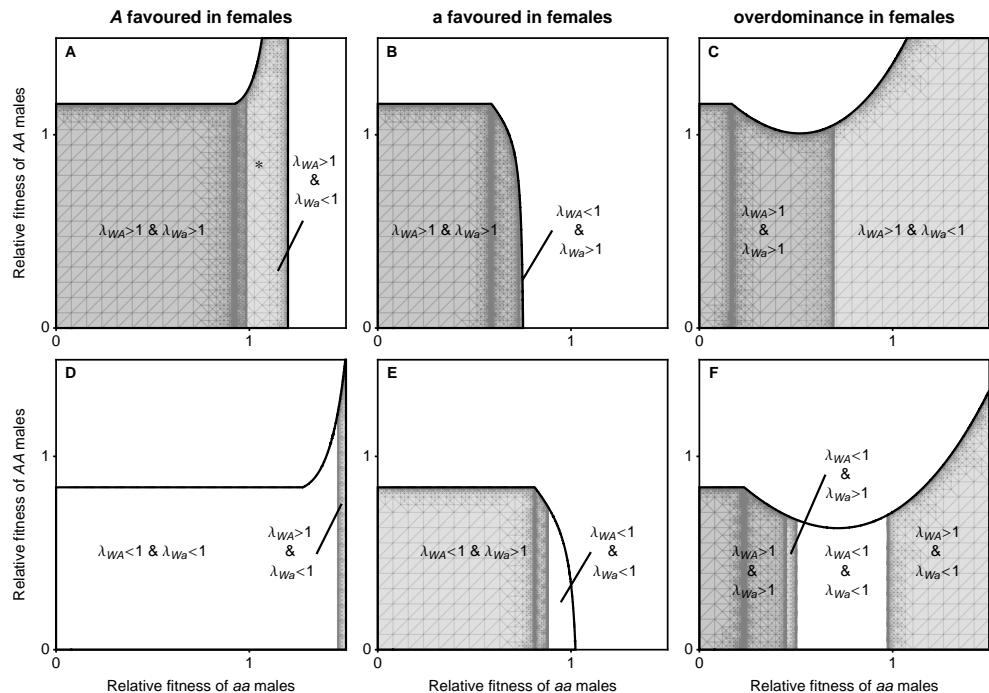


Figure S.4: ABC, $\alpha_{\Delta}^{\varphi} = -0.08$ DEF, $\alpha_{\Delta}^{\varphi} = 0.08$.

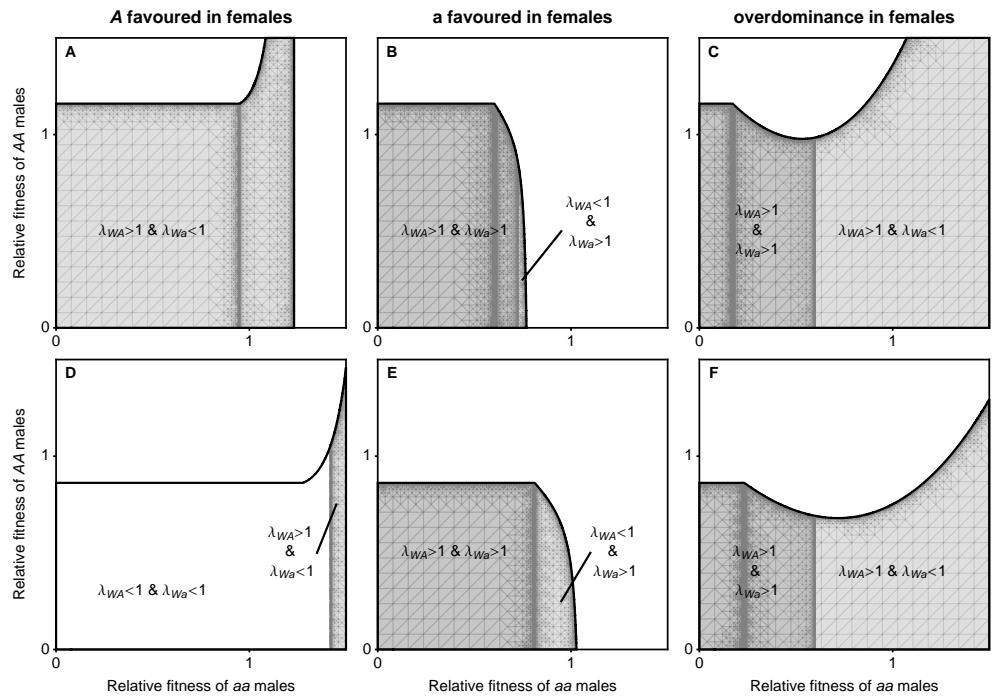


Figure S.5: ABC, $w_a^\delta = 1.16$, $w_A^\delta = 1$. DEF, $w_a^\delta = 1$, $w_A^\delta = 1.16$. I thought that re-running this with $w_a^\delta = 1.16$, $w_A^\delta = 0.84$ and $w_a^\delta = 0.84$, $w_A^\delta = 1.16$ will mean that it matches exactly with the meiotic drive example. Checking for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

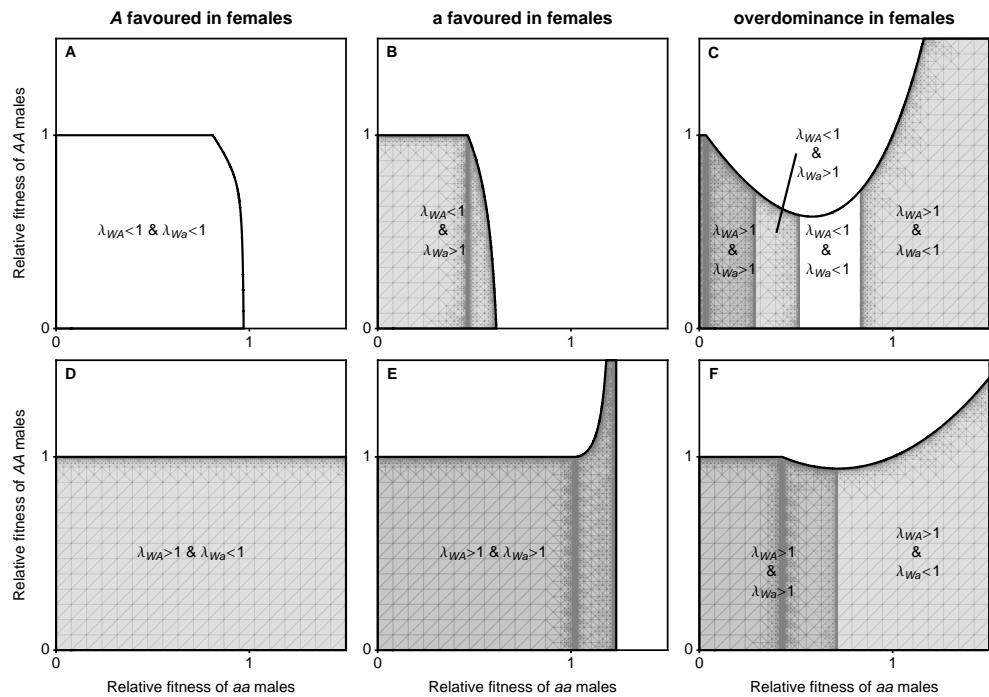


Figure S.6: ABC, $\alpha_{\Delta}^{\varnothing} = -0.08$ DEF, $\alpha_{\Delta}^{\varnothing} = 0.08$. λ s are increased for the haplotype that is favoured by female haploid selection, the stability conditions are also affected.

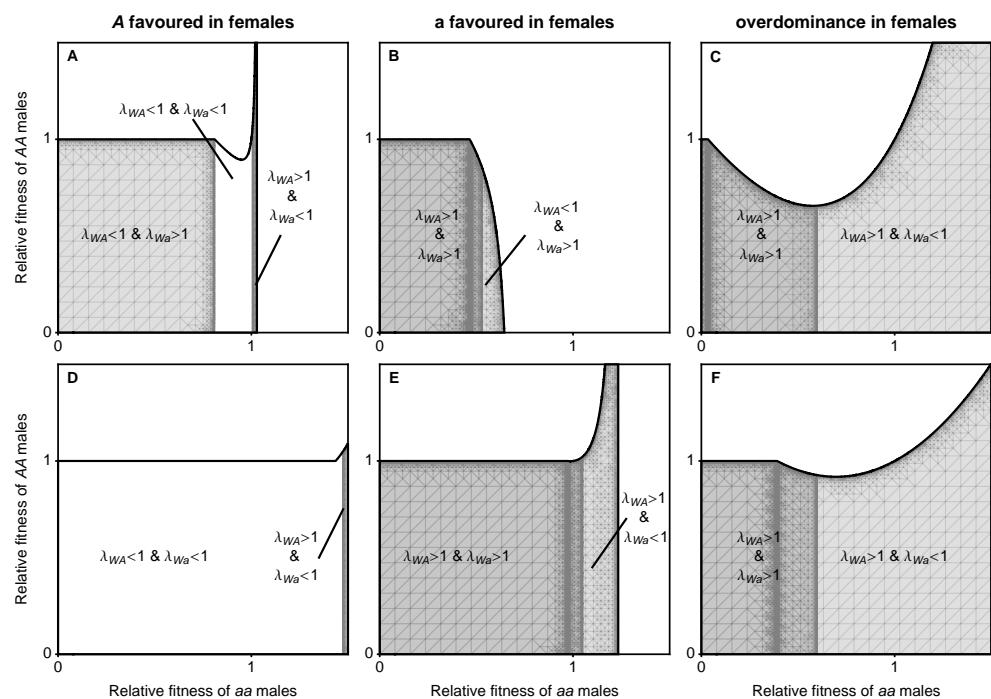


Figure S.7: ABC, $w_a^0 = 1.16$, $w_A^0 = 1$. DEF, $w_a^0 = 1$, $w_A^0 = 1.16$.

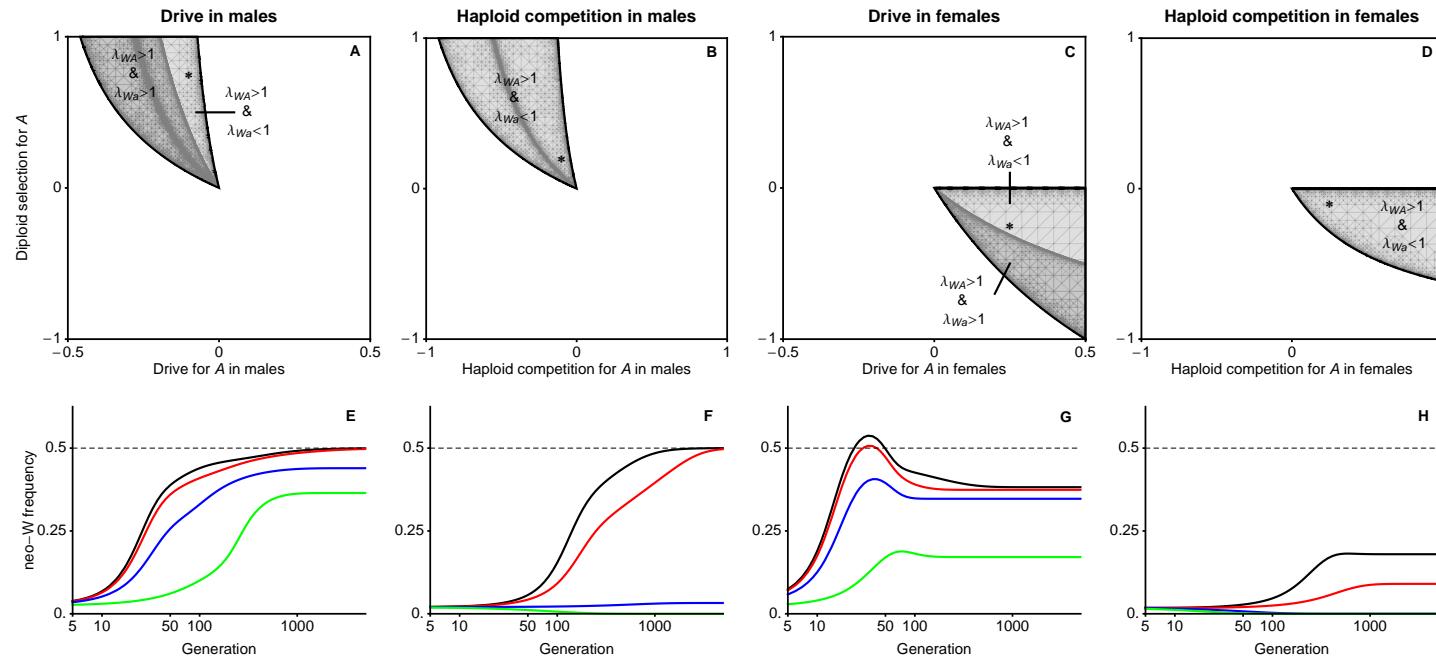


Figure S.8: A-D show when each of the neo-W haplotypes invade an internally stable equilibrium with a fixed on the Y (found by setting $r = 0$). The y-axis shows directional selection in diploids of both sexes, $s^{\varphi} = s^{\delta}$, and the x-axes show sex-specific drive, $\alpha_{\Delta}^{\varphi}$,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

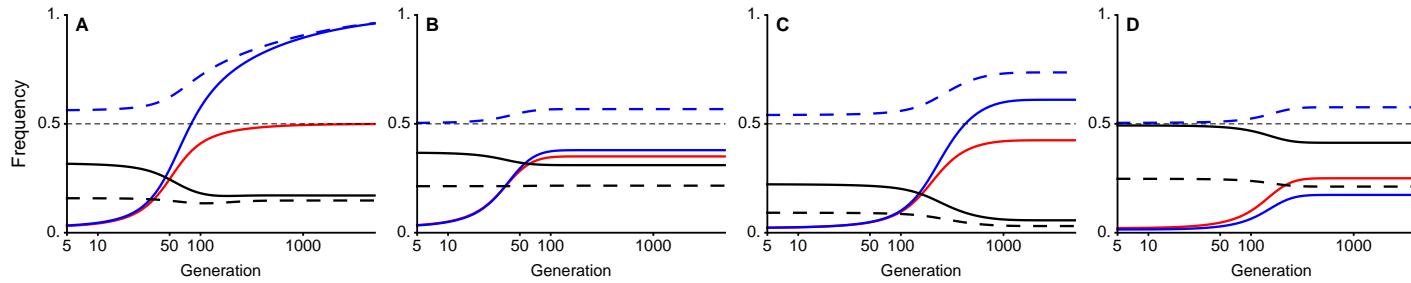


Figure S.9: Dynamics of all sex-determining alleles in each sex (males dashed). Red is neo-W, Blue is Y, Black is A. Panel A has the same parameters as the green curve in Figure 4B (ploidy-antagonism with male drive). Panel B has the same parameters as the green curve in Figure S.2C (overdominance in 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.

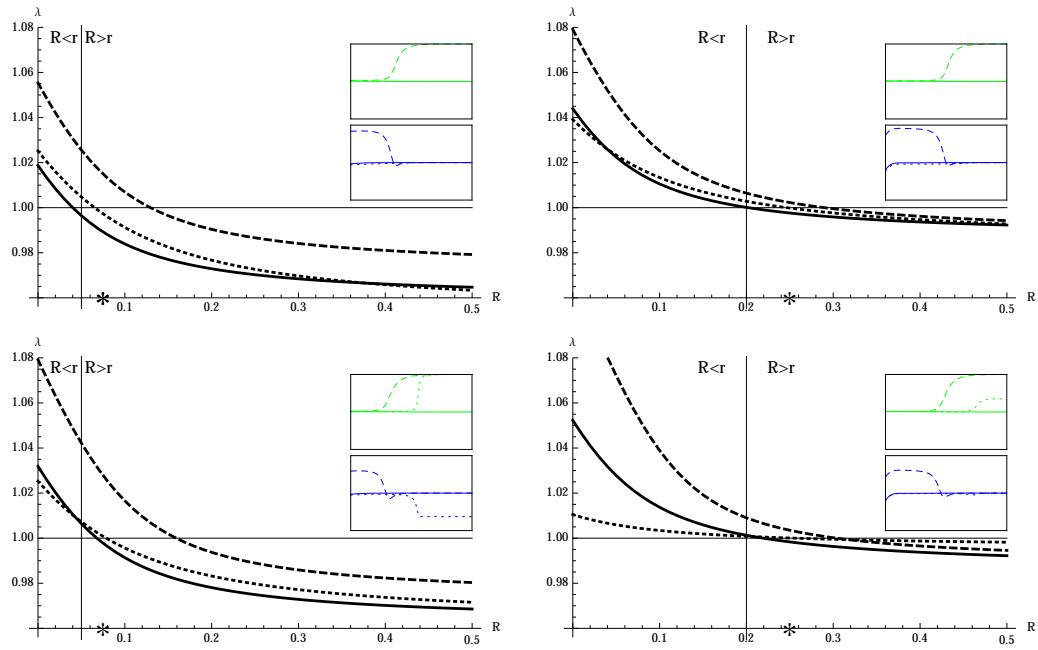


Figure S.10: [is this the one?](#)

1088 Add Sally's figure showing lambda for small r near equil A versus near equil
B. Add references to this figure to appendix where we discuss whether lambdas
can be greater than 1 with sexually antagonistic selection. not sure which one you
1090 are talking about, but see Figure S.10

1092 Perhaps it would also be useful to add an 8 panel figure that features ploidally
antagonistic selection. For each type of haploid selection (gametic competition/
meiotic drive in males/females), give a regionplot where $h^\delta = h^q$, e.g., $h^\delta =$
1094 $h^q = 0.75$ (or perhaps the value of h we use in the regionplots we have, in which
 $w_{aa} = 0.85$, $w_{Aa} = 1$, $w_{AA} = 1.05$). Matt made a figure like this before but
1096 both Ya and YA equilibria were plotted and there was no outline showing where
the Ya equilibrium is stable (as in Figure 2). In Matts plot the axes were s^q and
1098 α_Δ^q . Add an asterisk to each region plot and show invasion in another panel, using
those parameters and various R (e.g., in the stye of S.2). In an email, Sally has an
1100 example of ploidally-antagonistic selection where the neo-W fixes and $R = 1/2$.
This would cover that case and more. made an attempt (Figure S.8)

1102 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
1104 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.,
1106 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)
1108 (ensuring frequencies between 0 and 1), but yet to discuss in text.

1110 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
would be to show what happens to the ancestral SDR during turnover. We could
1112 also show an example where XY and ZW sex determining systems are both poly-
morphous and stable (e.g., using one of the curves in Figure S.2 and the green curve
1114 in Figure S.3). I think there are also examples with looser sex linkage and pollen
competition that lead to a mixed sex-determination system. We should probably
1116 have a short section in the appendix discussing this. made an attempt with Figure

S.9, but yet to discuss in text