

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

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Contributions:

Abstract

Sex-determination systems are remarkably dynamic; many taxa display shifts in the location of sex-determining loci or the evolution of entirely new sex-determining systems. Predominant theories for why we observe such transitions generally conclude that novel sex-determining systems are favoured by selection if they equalise the sex ratio or increase linkage with a sexually-antagonistic locus. We use population genetic models to extend these theories in two ways: (1) We explicitly consider how selection on very tightly sex-linked loci influences the spread of novel sex-determiners. We find that tightly sex-linked genetic variation can favour the spread of new sex-determination systems in which the heterogametic sex changes (XY to ZW or ZW to XY) and the new sex-determining region is less closely linked (or unlinked) to the sex-linked locus under selection, which is not predicted by previous theory. (2) We also consider selection upon haploid genotypes either during gametic competition (e.g., pollen/sperm competition) or meiosis (i.e., non-Mendelian segregation); selective processes that typically occur in one sex or the other. With haploid selection, we again find that transitions between male and female heterogamety can occur even if the new sex-determining region is less closely linked to the locus under 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 predicted 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 unexpected genomic locations that experience selection during diploid and/or haploid phases. These results might be reflected in the lability with which sex-determination systems evolve.

Introduction

30 Animals and angiosperms exhibit extremely diverse sex-determination systems
(reviewed in Bull 1983, Charlesworth and Mank 2010, Beukeboom and Perrin
32 2014, Bachtrog et al. 2014). Among species with genetic sex determination of
diploid sexes, some taxa have heterogametic males (XY) and homogametic fe-
34 males (XX), including mammals and most dioecious plants (Ming et al. 2011);
whereas other taxa have homogametic males (ZZ) and heterogametic females (ZW),
36 including Lepidoptera and birds. Within several taxa, the chromosome that har-
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-
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
addition, many gonochoric clades with genetic sex determination exhibit transi-
42 tions between male (XY) and female (ZW) heterogamety, including snakes (Gam-
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
(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
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
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
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-
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-
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)
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.

Segregation distortion provides putative evidence of haploid selection and can sometimes be attributed to meiotic drive and/or gametic competition (Lalanne et al. 2004, Fishman and Willis 2005, Leppälä et al. 2008; 2013, Didion et al. 2015; 2016). Where it has been characterized, 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, Vibrationovski 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 by which genes experiencing 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,
120 new masculinizing mutations (neo-Y chromosomes) could be favoured via associ-
ations with alleles that are beneficial in the male haploid stage. On the other hand,
122 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
124 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
126 (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
128 systems would be influenced by the combination of sex-ratio biases and associa-
tions between haploid selected loci and sex-determining regions.

130 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 deter-
132 mined by selection on genetically-associated alleles. Consequently, Fisherian sex
ratio selection does not dominate and it is possible for selection on linked alleles to
134 drive turnover between sex-determining systems despite causing increases in sex-
ratio bias. In addition to considering haploid selection, another novel development
136 in our model is that we consider loci that are in very tight linkage with the ances-
tral sex-determining region. We show that transitions between male and female
138 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
140 ancestral associations between sex and the alleles favoured in that sex.

Model

142 We consider transitions between ancestral and novel sex-determining systems us-
ing a three-locus model, each locus having two alleles. Locus **X** is the ancestral
144 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,
146 at which the null allele (*M*) is initially fixed in the population such that sex of

zygotes is determined by the genotype at the ancestral sex-determining region, **X**;
 148 XX genotypes become females and XY become males (or ZW become females
 and ZZ become males). To evaluate the evolution of new sex-determination sys-
 150 tems, we consider the invasion, fixation, maintenance, and/or loss of novel sex-
 determining alleles (m) at the **M** locus. We assume that the **M** locus is epistatically
 152 dominant over the **X** locus such that zygotes with at least one m allele develop as
 females with probability k and as males with probability $1 - k$, regardless of the
 154 **X** locus genotype. With $k = 0$, the m allele is a masculinizer (i.e., a neo-Y) and
 with $k = 1$ the m allele is a feminizer (i.e., a neo-W). With intermediate k , we can
 156 interpret m as an environmental sex determination (ESD) allele, such that zygotes
 develop as females in a proportion (k) of the environments they experience.

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

ble S.1). Individuals that are heterozygous at the **A** locus may experience meiotic
 178 drive; a gamete produced by *Aa* heterozygotes of sex ϕ bear allele *A* with probability α^ϕ . Thus, the **A** locus can experience sex-specific gametic competition, diploid
 180 selection, and/or meiotic drive.

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

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

Results

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

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

$\bar{p}^{\delta} = (1 - \hat{q})\hat{p}_X^{\delta} + \hat{q}\hat{p}_Y^{\delta}$ 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}^{\varnothing} is the mean fitness of diploids of sex \varnothing , see Table S.2

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

220 We are particularly concerned with the conditions under which a rare neo-sex-
determining allele increases in frequency, which occurs when the largest eigen-
222 value, 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 pos-
224 itive, 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 $\lambda = 1$ ($f'(1) = 2 + b < 0$
226 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
228 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**

230 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
 232 ($\lambda_{ma} < 1 < \lambda_{mA}$), 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
 234 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
 236 haplotypes due to recombination (χ_{mA}).

Table 2 illustrates a number of key points about the invasion of neo-Y and
 238 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.,
 240 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)
 242 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
 244 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
 246 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.
 248 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
 250 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
 252 male (when paired with Y). Consequently, the types of females produced differ in
 the frequency of A alleles they obtain from mating.

254 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
 256 allele (i.e., \hat{p}_X^o , \hat{p}_X^s , and \hat{p}_Y^s) and Y-bearing male gametes (\hat{q}) in the ancestral pop-
 ulation. Since only the A locus experiences selection directly, any deterministic

258 evolution requires that there is a polymorphism at the **A** locus. Polymorphisms
 can be maintained by mutation-selection balance or transiently present during the
 260 spread of beneficial alleles. However, polymorphisms maintained by selection can
 maintain alleles at higher allele frequencies for longer periods. Here, we focus of
 262 polymorphisms maintained by selection, where the *A* allele reaches a stable in-
 termediate 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)
 266 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^ϕ, t^ϕ ,
 268 α_Δ^ϕ of order $\epsilon \ll 1$).

Tight linkage with the ancestral sex-determining region

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

A neo-Y will never invade an ancestral XY system that already has tight linkage
 278 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 ($\lambda_{ma} = 1$)
 280 and does not change in frequency. The other neo-Y haplotype will not spread
 ($\lambda_{mA} < 1$) given that the initial equilibrium is stable. Therefore, a neo-Y mutation
 282 cannot spread ($\lambda \leq 1$) in an ancestral XY system that is at equilibrium with all se-
 lected loci within the non-recombining region around the SDR. In essence, through
 284 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
 286 that the ancestral Y is at this equilibrium, it is not possible for a neo-Y to create

males that have higher fitness than the ancestral Y.

288 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
290 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
292 tight linkage between the ancestral SDR and the selected locus, haploid selection
and/or overdominance can favour completely unlinked neo-W alleles ($R = 1/2$),
294 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
296 is no haploid selection and discuss the additional effect of haploid selection in the
appendix.

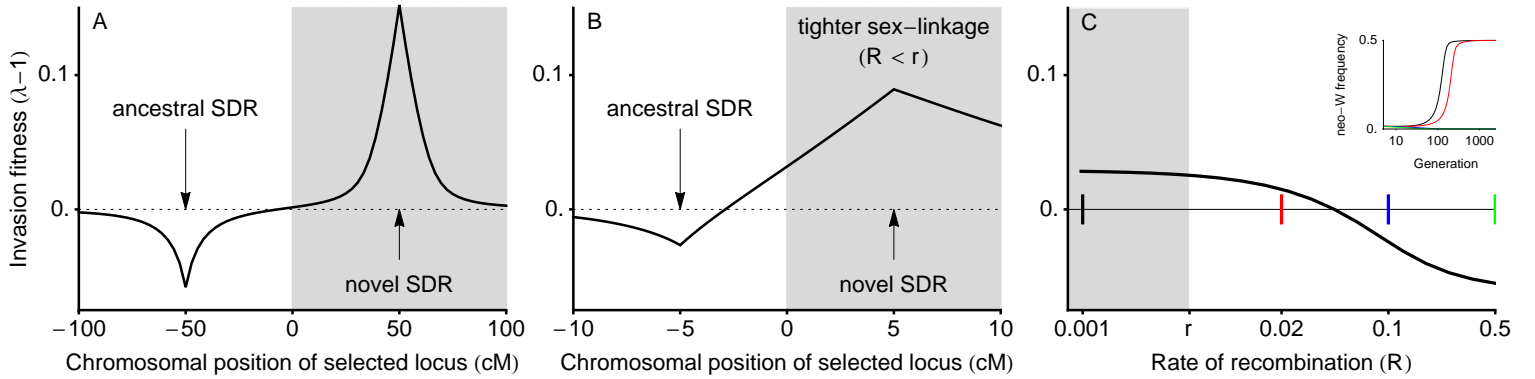


Figure 1: Transitions between XY and ZW systems can occur even when the neo-SDR is less tightly linked to a locus under sexually-antagonistic selection (here, without haploid selection). In panel A, linkage is loose enough relative to selection that the analytical results assuming weak selection hold, 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: $w_{AA}^{\circ} = 1.05$, $w_{aa}^{\circ} = 1.2$, $w_{aa}^{\delta} = w_{AA}^{\delta} = 0.85$, $w_{Aa}^{\delta} = 1$, $t^{\delta} = \alpha_{\Delta}^{\delta} = 0$.

298 If we categorise the a allele as being ancestrally ‘male-beneficial’ via the fact

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

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

Given that the a allele can be considered ancestrally ‘male-beneficial’ because
 318 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
 320 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
 322 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
 324 selection in females (e.g., see outlined region in Figure 2B and Lloyd and Webb
 326 1977, Otto 2014). In such cases, neo-W- a haplotypes can spread because they
 create more Aa and aa females when pairing with an X from males and because
 328 they bring Y- a haplotypes into females.

In some cases, both W-A and W-a haplotypes can spread, e.g., when AA individuals have low fitness in females yet the A is polymorphic or fixed on the X due to overdominance in males (Figure 2B and 2C). Both neo-W-A and neo-W-a haplotypes then produce fewer unfit AA females. This is true for the neo-W-A haplotype 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 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).

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 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 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 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 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 (Vandeputte 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 W alleles segregate at one locus (or two closely-linked loci) near to potentially sexually-antagonistic genes for pigmentation and sexual maturity (Kallman 1965; 1968, Volff 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-determining alleles.

Loose linkage with the ancestral sex-determining region

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

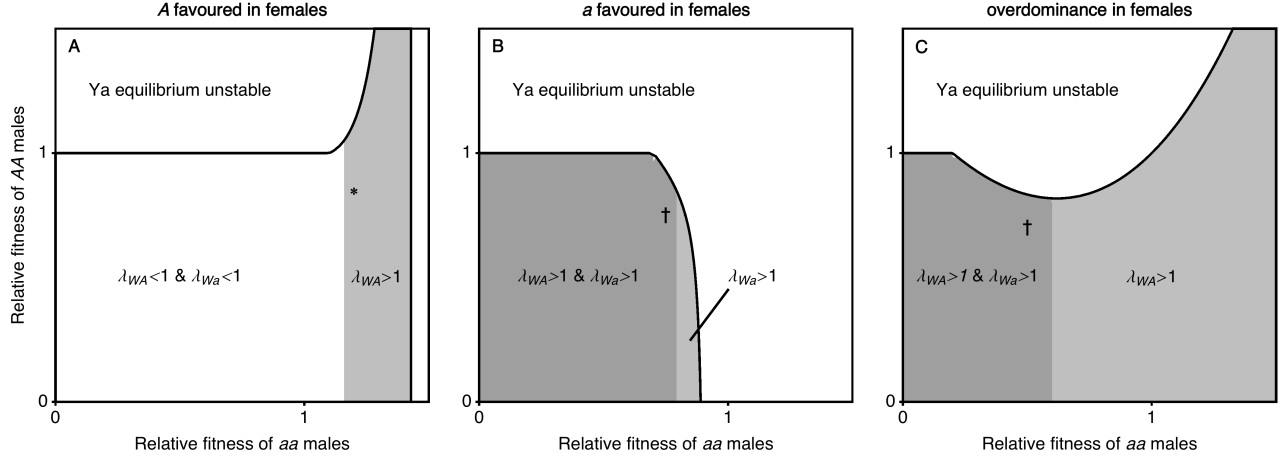


Figure 2: When the ancestral-XY locus is tightly linked to a locus under selection ($r = 0$), one or both neo-W haplotypes can spread. We vary the fitness of male homozygotes relative to heterozygotes ($w_{Aa}^{\varnothing} = 1$) and only consider stable equilibria at which both A locus alleles are maintained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

358 To leading order in selection, these are:

$$\lambda_{Y',XY} = 1 + \frac{1}{4} V_A S_A^2 \frac{(r - R)}{rR} + O(\epsilon^3) \quad (2)$$

and

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

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

leading-order probability of mating with an A -bearing gamete from the opposite sex (equation S.4). The difference in A -allele-frequency among Y-bearing sperm versus X-bearing sperm is given by $\hat{p}_Y^\delta - \hat{p}_X^\delta = V_A(D^\delta - D^\varnothing + \alpha_\Delta^\delta - \alpha_\Delta^\varnothing + t^\delta - t^\varnothing)(1 - 2r)/(2r)$.

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

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

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

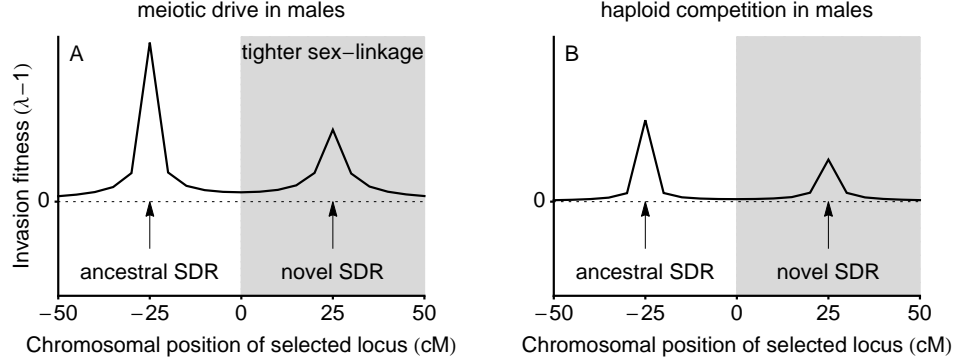


Figure 3: Ploidally-antagonistic selection allows a less tightly linked neo-W to invade. In panel A, male drive ($\alpha_{\Delta}^{\delta} = -1/20$, $r^{\delta} = \alpha_{\Delta}^{\varnothing} = 0$) opposes selection in diploids (no sex-differences: $s^{\delta} = 1/10$, $h^{\delta} = 7/10$), in which case the neo-sex-determining allele can invade regardless of linkage. In panel B, gametic competition in males ($r^{\delta} = -1/10$, $r^{\varnothing} = \alpha_{\Delta}^{\delta} = 0$) opposes selection in diploids (sex-differences: $s^{\delta} = 3/20$, $s^{\varnothing} = 1/20$, $h^{\delta} = 7/10$), 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).

curs 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, $h^{\varnothing} = h^{\delta}$. 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 ($s^{\varnothing}s^{\delta} > 0$, 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., $s^{\varnothing}(s^{\delta} - s^{\varnothing}) > 0$, see Figure 3B). These special cases indicate that 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	$h^\delta = h^\varphi, t^\varphi = t^\delta = \alpha_\Delta^\varphi = 0$	$s^\varphi s^\delta > 0$
female drive only	$h^\delta = h^\varphi, t^\varphi = t^\delta = \alpha_\Delta^\delta = 0$	$s^\varphi s^\delta > 0$
sperm competition only	$h^\delta = h^\varphi, t^\varphi = \alpha_\Delta^\varphi = \alpha_\Delta^\delta = 0$	$s^\varphi(s^\delta - s^\varphi) > 0$
egg competition only	$h^\delta = h^\varphi, t^\delta = \alpha_\Delta^\varphi = \alpha_\Delta^\delta = 0$	$s^\delta(s^\varphi - s^\delta) > 0$

412 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,
414 unlinked sex-determining locus invades in order to restore equal sex ratios (Koziel-
ska et al. 2010). Consider, for example, the case where the **A** locus is linked to the
416 ancestral-SDR ($r < 1/2$) and experiences meiotic drive in males only (e.g., dur-
ing spermatogenesis but not during oogenesis, $\alpha_\Delta^\delta \neq 0$, $\alpha_\Delta^\varphi = 0$), without gametic
418 competition ($t^\varphi = t^\delta = 0$). In this case, the zygotic sex ratio can be initially biased
only if the ancestral sex-determining system is XY (Figure 4B). We might there-
420 fore expect a difference in the potential for XY to ZW and ZW to XY transitions.
However, to leading order with selection weak relative to recombination, we find
422 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
424 associated with new sex-determining alleles (second terms in table 2). Thus, in-
vasion by a neo-W into an XY system and invasion by a neo-Y into a ZW system
426 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
428 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
430 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
432 sex ratio evolves to become biased towards males.

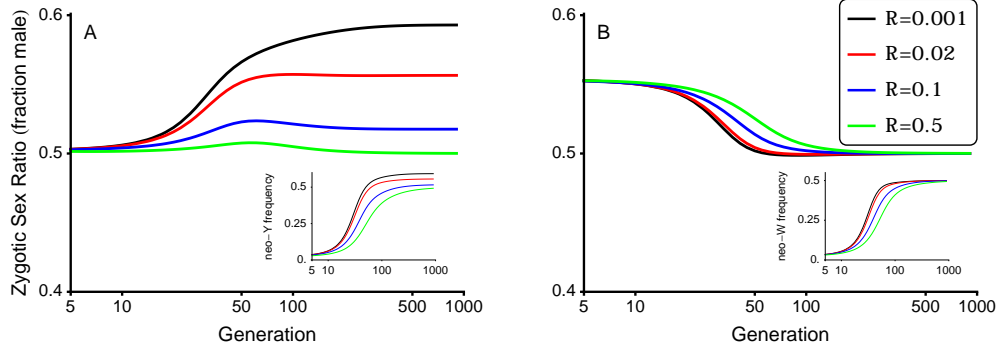


Figure 4: Fisherian sex-ratio selection alone is not a good predictor of turnover between sex-determining systems. In this figure, selection is ploidal antagonistic with haploid selection favouring the a allele during male meiosis. In panel A, male drive in an ancestral ZW system has no affect on the 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: $s^{\text{Z}} = s^{\text{X}} = 0.2$, $h^{\text{Z}} = h^{\text{X}} = 0.7$, $t^{\text{Z}} = t^{\text{X}} = \alpha_{\Delta}^{\text{Z}} = 0$, $\alpha_{\Delta}^{\text{X}} = -0.1$, $r = 0.02$.

The green curves in Figure 4 show transitions between male and female het-
 434 erogametety 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
 436 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 ances-
 438 trally 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 male meiotic drive allele, a , is
 440 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 ($\hat{p}_W^{\text{Z}} - \hat{p}_Z^{\text{Z}} > 0$, equation
 442 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
 444 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
 446 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 meiotic drive.
 448 Thus, a key factor in explaining why heterogametic transitions can occur when

$R > r$ is that the neo-SDR determines sex in the diploid phase but recombination
 450 occurs before any subsequent haploid selection.

Environmental sex determination

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

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

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

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

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

spread of which is given by

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

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

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

Discussion

Two predominant theories explaining the remarkably high frequency of transitions between sex-determination systems are sexually-antagonistic selection and sex-ratio selection (reviewed in Blaser et al. 2012, van Doorn 2014). 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
 522 Úbeda et al. 2015). Furthermore, with weak selection, we find that there is no
 524 difference in conditions allowing XY to ZW and ZW to XY transitions, indicating
 that sex chromosome transitions are not predominantly predicted by their effect on
 526 the sex-ratio (i.e., the sex-ratio bias created by male haploid selection facilitates
 the spread of a neo-W into an XY system the same way that male haploid selection
 528 drives the spread of a neo-Y into a ZW system with a 1:1 sex ratio). Thus, haploid
 selection can favour heterogametic transitions both via sex-ratio selection and via
 530 fitness effects of alleles that are associated with the neo-sex-determining allele,
 and these selection pressures are predicted to often be of equal magnitude when
 532 selection is weak.

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

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

554 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
556 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
558 overdominance and/or sexually-antagonistic selection can be identified in close
linkage with the ancestral sex-determining locus (rather than only the novel sex-
560 determining locus), then they could also be implicated in driving heterogametic
transitions between sex-determination systems. In addition, we show haploid se-
562 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-
564 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-
566 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
568 sex chromosomes in closely related species suggesting that new sex chromosomes
may have been favoured through their association with haploid selected alleles on
570 these chromosomes (Sandler et al., 2017, Personal Communication).

We assume that sex-determining alleles do not experience direct selection ex-
572 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
574 ancestral sex-determining region because recessive deleterious mutations and/or
deletions accumulate around the Y or W sex-determining regions (Rice 1996,
576 Charlesworth and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). During
heterogametic transitions (XY to ZW or ZW to XY), but not homogametic transi-
578 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
580 phenomenon was studied by van Doorn and Kirkpatrick (2010), who found that
degeneration can prevent fixation of a neo-W or a neo-Y allele, leading to a mixed

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

Another simplification that we made is that meiotic drive involves only a single locus with two alleles. However, many meiotic drive systems involve an interaction with another locus at which alleles may ‘suppress’ the action of meiotic drive (Burt and Trivers 2006, Lindholm et al. 2016). Thus, the dynamics of meiotic drive alleles can be heavily dependent on the interaction between two loci and the recombination rate between them, which in turn can be affected by sex-linkage if there is reduced recombination between sex chromosomes (Hurst and Pomiankowski 1991). Furthermore, in some cases, a driving allele may act by killing any gametes that carry a ‘target’ allele at another locus, in which case there can be fertility effects which can affect the equilibrium frequency of a meiotic drive allele (Holman et al. 2015). In polygamous mating systems, the intensity of pollen/sperm competition can depend on the density of males available to donate pollen/sperm, which can itself depend on the sex ratio (Taylor and Jaenike 2002). In terms of our model, this implies that the strength of gametic competition (r^{δ}) may both determine and be determined by the sex ratio. How the evolution of new sex-determining mechanisms could be influenced by two-locus meiotic drive and/or by ecological feedbacks under different mating systems remains to be studied.

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

612 purely sexually-antagonistic selection, eliminating the need for differences in se-
lection between male and female diploids. Perhaps counterintuitively, transitions
614 involving haploid selection can be driven by sex-ratio selection or cause sex-ratio
biases to evolve. We conclude that haploid selection should be considered as a
616 pivotal factor driving transitions between sex-determination systems. Overall, our
results suggest several new scenarios under which new sex-determination systems
618 are favoured, which could help to explain why the evolution of sex-determination
systems is so dynamic.

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Appendix

876 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} . The superscript $\phi \in \{\sigma, \varphi\}$ specifies the sex of the diploid that the gamete came from. The subscript $i \in \{1, 2, 3, 4\}$ specifies the genotype at the selected locus **A** and at the novel sex-determining locus **M**, where $1 = AM$, $2 = aM$, $3 = Am$, and $4 = am$. The gamete frequencies from each sex sum to one, $\sum_i x_i^{\phi} + y_i^{\phi} = 1$.

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 ($w_1^{\phi} = w_3^{\phi} = w_A^{\phi}$, $w_2^{\phi} = w_4^{\phi} = w_a^{\phi}$, see Table 1). The genotype frequencies after gametic competition are $x_i^{\phi,s} = w_i x_i^{\phi} / \bar{w}_H^{\phi}$ and $y_i^{\phi,s} = w_i y_i^{\phi} / \bar{w}_H^{\phi}$, where $\bar{w}_H^{\phi} = \sum_i w_i x_i^{\phi} + w_i y_i^{\phi}$ is the mean fitness of male ($\phi = \sigma$) or female ($\phi = \varphi$) 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, $xx_{ij} = (x_i^{\varphi,s} x_j^{\sigma,s} + x_j^{\varphi,s} x_i^{\sigma,s})/2$ and $yy_{ij} = (y_i^{\varphi,s} y_j^{\sigma,s} + y_j^{\varphi,s} y_i^{\sigma,s})/2$.

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 $xx_{ij}^{\varphi} = k_{bc} xx_{ij}$, XY females are given by $xy_{ij}^{\varphi} = k_{bc} xy_{ij}$, and YY females are given by $yy_{ij}^{\varphi} = k_{bc} yy_{ij}$. Similarly, XX male frequencies are $xx_{ij}^{\sigma} = (1 - k_{bc}) xx_{ij}$, XY male frequencies are $xy_{ij}^{\sigma} = (1 - k_{bc}) xy_{ij}$, and YY males frequencies are $yy_{ij}^{\sigma} = (1 - k_{bc}) yy_{ij}$. This

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

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

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

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

competition) in the next generation are given by

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

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

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

$$\begin{aligned}
x_4^{\tilde{\phi}'} = & x x_{44}^{\tilde{\phi},s} + x x_{34}^{\tilde{\phi},s}/2 + (x x_{14}^{\tilde{\phi},s} + x x_{24}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(x x_{14}^{\tilde{\phi},s} - x x_{23}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{44}^{\tilde{\phi},s} + x y_{42}^{\tilde{\phi},s})/2 + (x y_{41}^{\tilde{\phi},s} + x y_{43}^{\tilde{\phi},s})(1 - \alpha^{\tilde{\phi}}) \\
& - r(x y_{43}^{\tilde{\phi},s} - x y_{34}^{\tilde{\phi},s})(1 - \alpha^{\tilde{\phi}}) - \rho(x y_{42}^{\tilde{\phi},s} - x y_{24}^{\tilde{\phi},s})/2 \\
& + [-(R + r + \rho) x y_{41}^{\tilde{\phi},s} + (R + \rho - r) x y_{14}^{\tilde{\phi},s} \\
& + (R + r - \rho) x y_{32}^{\tilde{\phi},s} + (R + \rho - r) x y_{23}^{\tilde{\phi},s}] (1 - \alpha^{\tilde{\phi}})/2
\end{aligned} \tag{S.1d}$$

$$\begin{aligned}
y_1^{\tilde{\phi}'} = & y y_{11}^{\tilde{\phi},s} + y y_{13}^{\tilde{\phi},s}/2 + (y y_{12}^{\tilde{\phi},s} + y y_{14}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(y y_{14}^{\tilde{\phi},s} - y y_{23}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{11}^{\tilde{\phi},s} + x y_{31}^{\tilde{\phi},s})/2 + (x y_{21}^{\tilde{\phi},s} + x y_{41}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - r(x y_{21}^{\tilde{\phi},s} - x y_{12}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} - \rho(x y_{31}^{\tilde{\phi},s} - x y_{13}^{\tilde{\phi},s})/2 \\
& + [-(R + r + \rho) x y_{41}^{\tilde{\phi},s} + (R + \rho - r) x y_{14}^{\tilde{\phi},s} \\
& + (R + r - \rho) x y_{32}^{\tilde{\phi},s} + (R + \rho - r) x y_{23}^{\tilde{\phi},s}] \alpha^{\tilde{\phi}}/2
\end{aligned} \tag{S.1e}$$

$$\begin{aligned}
y_2^{\tilde{\phi}'} = & y y_{22}^{\tilde{\phi},s} + y y_{24}^{\tilde{\phi},s}/2 + (y y_{12}^{\tilde{\phi},s} + y y_{23}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(y y_{23}^{\tilde{\phi},s} - y y_{14}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{22}^{\tilde{\phi},s} + x y_{42}^{\tilde{\phi},s})/2 + (x y_{12}^{\tilde{\phi},s} + x y_{32}^{\tilde{\phi},s})(1 - \alpha^{\tilde{\phi}}) \\
& - r(x y_{12}^{\tilde{\phi},s} - x y_{21}^{\tilde{\phi},s})(1 - \alpha^{\tilde{\phi}}) - \rho(x y_{42}^{\tilde{\phi},s} - x y_{24}^{\tilde{\phi},s})/2 \\
& + [-(R + r + \rho) x y_{32}^{\tilde{\phi},s} + (R + \rho - r) x y_{23}^{\tilde{\phi},s} \\
& + (R + r - \rho) x y_{41}^{\tilde{\phi},s} + (R + \rho - r) x y_{14}^{\tilde{\phi},s}] (1 - \alpha^{\tilde{\phi}})/2
\end{aligned} \tag{S.1f}$$

$$\begin{aligned}
y_3^{\tilde{\phi}'} = & y y_{33}^{\tilde{\phi},s} + y y_{13}^{\tilde{\phi},s}/2 + (y y_{23}^{\tilde{\phi},s} + y y_{34}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - R(y y_{23}^{\tilde{\phi},s} - y y_{14}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& (x y_{33}^{\tilde{\phi},s} + x y_{13}^{\tilde{\phi},s})/2 + (x y_{23}^{\tilde{\phi},s} + x y_{43}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} \\
& - r(x y_{43}^{\tilde{\phi},s} - x y_{34}^{\tilde{\phi},s}) \alpha^{\tilde{\phi}} - \rho(x y_{13}^{\tilde{\phi},s} - x y_{31}^{\tilde{\phi},s})/2 \\
& + [-(R + r + \rho) x y_{23}^{\tilde{\phi},s} + (R + \rho - r) x y_{32}^{\tilde{\phi},s} \\
& + (R + r - \rho) x y_{14}^{\tilde{\phi},s} + (R + \rho - r) x y_{41}^{\tilde{\phi},s}] \alpha^{\tilde{\phi}}/2
\end{aligned} \tag{S.1g}$$

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

928

The full system is therefore described by 16 recurrence equations (three diallelic
930 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
932 ancestral XY sex-determining system, there are XX males, XY females, or YY
females ($x_3^{\phi} = x_4^{\phi} = y_4^{\phi} = y_3^{\phi} = y_i^{\phi} = 0$). In this case, the system only involves six
934 recursion equations, which we assume below to calculate the equilibria.

Resident equilibria and stability

In the resident population (allele M fixed), we follow the frequency of A in X-
936 bearing female gametes, p_X^{ϕ} , and X-bearing male gametes, p_X^{δ} , and Y-bearing male
938 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 deter-
mine the frequencies of the six resident gamete types: $x_1^{\phi} = \hat{p}_X^{\phi}$, $x_2^{\phi} = 1 - \hat{p}_X^{\phi}$,
940 $x_1^{\delta} = (1 - q)\hat{p}_X^{\delta}$, $x_2^{\delta} = (1 - q)(1 - \hat{p}_X^{\delta})$, $y_1^{\delta} = q\hat{p}_Y^{\delta}$, and $y_2^{\delta} = q(1 - \hat{p}_Y^{\delta})$. Mean
942 fitnesses in the resident population are given in table S.2.

Various forms of selection can maintain a polymorphism at the A locus, in-
944 cluding sexually antagonistic selection, overdominance, conflicts between diploid
selection and selection upon haploid genotypes (ploidy antagonistic selection,
946 Immler et al. 2012), or a combination of these selective regimes. [add reference or
say "see below"](#)

948 In particular special cases, e.g., no sex-differences in selection or meiotic drive

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

($s^\delta = s^\varnothing$, $h^\delta = h^\varnothing$, and $\alpha^\delta = \alpha^\varnothing = 1/2$), the equilibrium allele frequency and sta-
950 bility can be calculated analytically without assuming anything about the relative
strengths of selection and recombination. However, here, we focus on two regimes
952 (tight linkage and weak selection) in order to make fewer assumptions about fit-
nesses.

954 **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-
956 tral population when the recombination rate between the X and A loci is small
(r of order ϵ). Selection at the A locus will not affect evolution at the novel sex-
958 determining locus, M, if one allele is fixed on all backgrounds. We therefore focus
on the five equilibria that maintain both A and a alleles, four of which are given to
960 leading order by:

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

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

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

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

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

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

$$\begin{aligned} \phi &= (1 + \alpha_\Delta^\varnothing) w_A^\varnothing w_{Aa}^\varnothing \left[w_a^\delta w_{aa}^\delta + (1 + \alpha_\Delta^\delta) w_A^\delta w_{Aa}^\delta \right] / 2 - w_a^\delta w_A^\varnothing w_{aa}^\delta w_{aa}^\varnothing \\ \psi &= (1 - \alpha_\Delta^\varnothing) w_a^\varnothing w_{Aa}^\varnothing \left[w_a^\delta w_{aa}^\delta + (1 + \alpha_\Delta^\delta) w_A^\delta w_{Aa}^\delta \right] / 2 - (1 + \alpha_\Delta^\delta) w_A^\varnothing w_A^\delta w_{Aa}^\delta w_{AA}^\varnothing \\ \phi' &= (1 - \alpha_\Delta^\varnothing) w_a^\varnothing w_{Aa}^\varnothing \left[w_A^\delta w_{AA}^\delta + (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta \right] / 2 - w_A^\varnothing w_A^\delta w_{AA}^\delta w_{AA}^\varnothing \\ \psi' &= (1 + \alpha_\Delta^\varnothing) w_A^\varnothing w_{Aa}^\varnothing \left[w_A^\delta w_{AA}^\delta + (1 - \alpha_\Delta^\delta) w_a^\delta w_{Aa}^\delta \right] / 2 - (1 - \alpha_\Delta^\delta) w_a^\varnothing w_a^\delta w_{Aa}^\delta w_{Aa}^\varnothing \end{aligned}$$

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

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

erties 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 $\bar{w}_{Ya}^\delta > \bar{w}_{YA}^\delta$ where $\bar{w}_{Ya}^\delta = w_a^\delta [\hat{p}_X^\varphi (1 - \alpha_\Delta^\delta) w_A^\varphi w_{Aa}^\delta + (1 - \hat{p}_X^\varphi) w_a^\varphi w_{aa}^\delta]$ and $\bar{w}_{YA}^\delta = w_A^\delta [\hat{p}_X^\varphi w_A^\varphi w_{AA}^\delta + (1 - \hat{p}_X^\varphi)(1 + \alpha_\Delta^\delta) w_a^\varphi w_{Aa}^\delta]$. That is, Y- a haplotypes must have higher fitness than Y- A haplotypes. Substituting in \hat{p}_X^φ from equation (S.2), 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 $\psi < 0$.

Selection weak relative to recombination (weak selection)

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

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

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

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

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

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

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

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

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

1002 Invasion conditions

1004 **Cover the other parts of the characteristic polynomial here. Waiting for Sally's proof!**

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

1014 The general conditions for the invasion of a neo-sex-determining allele are given in the main text, in terms of the growth rates of the mutant haplotypes in

1016 the absence of recombination (λ_{mi}) and the rate that recombination destroys them
 (χ_{mi}). For tight linkage between the ancestral sex-determining locus and the se-
 1018 lected locus we can calculate these terms explicitly (see below). For weak selection
 we can take a Taylor series of the leading eigenvalue. The leading eigenvalue, λ ,
 1020 for any k , is given up to order ϵ^2 by equation (4).

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

1022 Here, we explore the conditions under which a neo-W invades an XY system as-
 suming that the A locus is initially in tight linkage with the ancestral sex-determining
 1024 region ($r \approx 0$). We disregard neo-Y mutations, which never spread given that the
 ancestral population is at a stable equilibrium (see supplementary *Mathematica*
 1026 notebook for proof).

Starting with the simpler equilibrium (B), the terms of the characteristic poly-
 1028 nomial are

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

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

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

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

Haploid selection impacts the spread of neo-W haplotypes in three ways. Firstly,
 1030 the zygotic sex ratio becomes male biased, $\zeta > 1/2$, when the a allele (which is
 fixed on the Y) is favoured during competition among male gametes or by meiotic
 1032 drive in males. Specifically, at equilibrium (B), the fraction female is $1 - \zeta =$
 $w_A^\delta(1 + \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ where $2\bar{w}_H^\delta = [w_a^\delta(1 - \alpha_\Delta^\delta) + w_A^\delta(1 + \alpha_\Delta^\delta)]$ has been canceled

1034 out in equations (S.6) to leave the term $[w_A^\delta(1 + \alpha_\Delta^\delta)]^{-1}$. Male biased sex ratios
 1036 facilitate the spread of a neo-W because neo-W alleles cause the zygotes that carry
 them to develop as the rarer, female, sex.

Secondly, haploid selection in females selects on neo-W haplotypes directly. At
 1038 equilibrium (B), the fitness of female gametes under the ancestral sex-determining
 system is w_A^\varnothing such that the relative fitnesses of neo-W- A and neo-W- a haplotypes
 1040 during female gametic competition are $w_A^\varnothing/w_A^\varnothing$ and $w_a^\varnothing/w_A^\varnothing$ (see terms in equation
 S.6). Meiotic drive in females will also change the proportion of gametes that carry
 1042 the A versus a alleles, which will be produced by heterozygous females in propor-
 tions $(1 + \alpha_\Delta^\varnothing)/2$ and $(1 - \alpha_\Delta^\varnothing)/2$, respectively. These terms are only associated with
 1044 heterozygous females, i.e., they are found alongside w_{Aa}^\varnothing .

Thirdly, haploid selection in males affects the diploid genotypes of females
 1046 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-
 1048 metes with probability $w_A^\delta(1 + \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ and Y- a male gametes with probability
 $w_a^\delta(1 - \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$, where the $2\bar{w}_H^\delta$ terms have been canceled in equation (S.6)
 1050 (as mentioned above). Thus, for example, neo-W- A haplotypes are found in AA
 female diploids with probability $w_A^\delta(1 + \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ (first term in square brack-
 1052 ets in the numerator of equation S.6a) and in Aa female diploids with probability
 $w_a^\delta(1 - \alpha_\Delta^\delta)/(2\bar{w}_H^\delta)$ (see equation S.6c and the second term in square brackets in
 1054 the numerator of equation S.6a).

The other terms in equations (S.6) are more easily interpreted if we assume that
 1056 there is no haploid selection in either sex, in which case $\lambda_{mA} > 1$ when $w_{Aa}^\varnothing > w_{AA}^\varnothing$
 and $\lambda_{ma} > 1$ when $(w_{Aa}^\varnothing + w_{aa}^\varnothing)/2 > w_{AA}^\varnothing$. These conditions cannot be met under
 1058 purely sexually-antagonistic selection, where A is directionally favoured in females
 ($w_{AA}^\varnothing > w_{Aa}^\varnothing > w_{aa}^\varnothing$) and a is directionally favoured in males ($w_{AA}^\delta > w_{Aa}^\delta > w_{aa}^\delta$).
 1060 Essentially, the X is then already as specialized as possible for the female beneficial
 allele (A is fixed on the X), and the neo-W often makes daughters with the Y- a
 1062 haplotype, increasing the flow of a alleles into females, which reduces the fitness
 of those females.

1064 If selection doesn't uniformly favour A in females, however, neo-W- A haplo-
 1066 types 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
 1068 fact that a neo-W brings Y- a haplotypes into females, when $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$, as stated
 above. In this case the a allele is favoured by selection in females despite A being
 1070 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
 1072 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 $w_{Aa}^{\varphi} < 2w_{AA}^{\varphi}$ and $w_{Aa}^{\sigma} / [(w_{aa}^{\sigma} + w_{Aa}^{\sigma})/2] > w_{Aa}^{\varphi} / w_{AA}^{\varphi}$.
 1074 Still considering $w_{Aa}^{\varphi} > w_{AA}^{\varphi}$, the neo-W can also spread alongside the a allele
 ($\lambda_{ma} > 1$) if w_{aa}^{φ} is large enough such that $(w_{Aa}^{\varphi} + w_{aa}^{\varphi})/2 > w_{AA}^{\varphi}$. This can occur
 1076 with overdominance or directional selection for a in females (Figure 2B,C). In this
 case, a is favoured in females (comparing Aa to AA genotypes in females) but A
 1078 is fixed on the X due to selection in males. The neo-W- a haplotype can spread
 because it produces females with higher fitness Aa and aa genotypes.
 1080 Similar equations can be derived for equilibrium (A) by substituting the equi-
 librium allele frequencies into Table 2

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

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

$$\chi_{mA} = \frac{a}{b} \frac{R}{2} [w_{Aa}^{\varphi} (1 + \alpha_{\Delta}^{\varphi}) w_a^{\sigma} c] / w_a^{\varphi} \quad (\text{S.7c})$$

$$\chi_{ma} = \frac{a}{b} \frac{R}{2} [w_{Aa}^{\varphi} (1 - \alpha_{\Delta}^{\varphi}) w_{Aa}^{\sigma} w_A^{\sigma} (1 + \alpha_{\Delta}^{\sigma}) \phi] / w_A^{\varphi} \quad (\text{S.7d})$$

1082 where

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

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

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

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

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

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

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

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

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

1100 This condition cannot be met with purely sexually antagonistic selection (as both
 1102 terms on the left-hand side would then be negative), but it can be met under other
 1104 circumstances. For example, with overdominance in males there is selection for
 1106 increased A frequencies on X chromosomes in males, which are always paired
 1108 with $Y-a$ haplotypes. Directional selection for a in females can then maintain a
 polymorphism at the A locus on the X. This scenario selects for a modifier that
 increases recombination between the sex chromosomes (e.g., blue region of Figure
 2d in Otto 2014) and facilitates the spread of neo-W- a haplotypes, which create
 more females bearing more a alleles than the ancestral X chromosome does.

Tight Linkage and Haploid Selection

1110 is this specific in any way to tight linkage, $r \approx 0$? i see most of the plots referred
 1112 to are tight linkage plots...

1112 Generally, haploid selection expands the scenarios under which neo-W alle-
 1114 les can spread. For example, when selection is sexually-antagonistic in diploids
 1116 ($s^{\varphi}s^{\delta} < 0$ and $0 < h^{\delta} < 1$) an unlinked neo-W ($R = 1/2$) cannot invade un-
 less there is also haploid selection (Figures 1 and S.3). Secondly, with haploid
 1118 selection, overdominance ($w_{aa}^{\delta} < w_{Aa}^{\delta}$ & $w_{AA}^{\delta} < w_{Aa}^{\delta}$) is not required for neo-
 W- a haplotypes to spread ($\lambda_{ma} > 1$) (Figures S.4-S.7). Haploid selection and
 overdominance allow unlinked neo-W alleles to invade even when there is tight
 linkage between the ancestral SDR and the selected locus (Figure S.10). Finally,
 1120 haploid selection can maintain a polymorphism in the face of directional selection
 in male and female diploids (ploiddally-antagonistic selection). When selection is
 1122 ploiddally-antagonistic, neo-W alleles often spread, for at least some values of R
 (Figure S.8).

1124 As discussed above, male haploid selection alters the sex ratio and the alleles
 1126 carried by male gametes that female gametes pair with. Male haploid selection
 in favour of the a allele ($\alpha_{\Delta}^{\delta} < 0$, $w_A^{\delta} < w_a^{\delta}$) generates male-biased sex ratios
 at equilibria (A) and (B), where $Y-a$ is fixed ($\hat{p}_Y^{\delta} = 0$). Male-biased sex-ratios
 1128 facilitate the spread of neo-W- A and neo-W- a haplotypes (increasing λ_{WA} and

1130 λ_{W_a}). Panels A-C in Figures S.4 and S.5 show that neo-W haplotypes tend to
spread for a wider range of parameters when sex ratios are male biased, compared
to Figure 2 without haploid selection. By contrast, male haploid selection in favour
1132 of the A allele generates female-biased sex ratios and reduces λ_{WA} and λ_{Wa} , as
demonstrated by panels D-F in Figures S.4 and S.5.

1134 Female haploid selection generates direct selection on the neo-W- A and neo-
W- a haplotypes as they spread in females. Thus, female haploid selection in favour
1136 of the a allele tends to increase λ_{Wa} and decrease λ_{WA} , as shown by panels A-C in
Figures S.6 and S.7. Conversely, female haploid selection in favour of the A allele
1138 increases λ_{WA} and decreases λ_{Wa} , see panels D-F in Figures S.6 and S.7.

Supplementary Figures

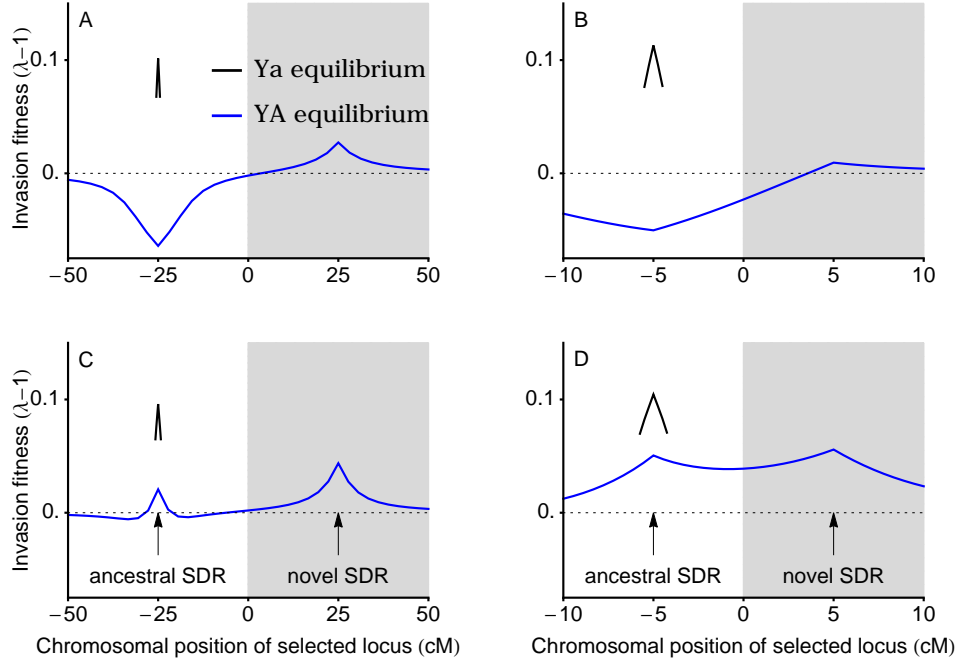


Figure S.1: Neo-W alleles can spread when loci under diploid selection are tightly linked to the ancestral sex determining locus ($r \approx 0$). In panels A and B, the a allele is favoured in females ($w_{aa}^{\varnothing} = 1.05$, $w_{Aa}^{\varnothing} = 1$, $w_{AA}^{\varnothing} = 0.85$) and selection in males is overdominant ($w_{aa}^{\sigma} = w_{AA}^{\sigma} = 0.75$). In panels C and D, selection in males and females is overdominant ($w_{aa}^{\varnothing} = w_{AA}^{\varnothing} = 0.6$, $w_{aa}^{\sigma} = 0.5$, $w_{AA}^{\sigma} = 0.7$, $w_{Aa}^{\sigma} = 1$). There is no haploid selection $r^{\varnothing} = \alpha_{\Delta}^{\varnothing} = 0$. These parameters are marked by daggers in Figure 2B and C, which show that neo-W invasion is expected for any \bar{R} ($\lambda_{WA}, \lambda_{Wa} > 1$) when the a allele is nearly fixed on the Y (black lines in this figure; not stable for $r \gg 0$). 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 if and 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 B and D and when the selected locus is near the SDRs in all panels), this weak selection prediction can break down.

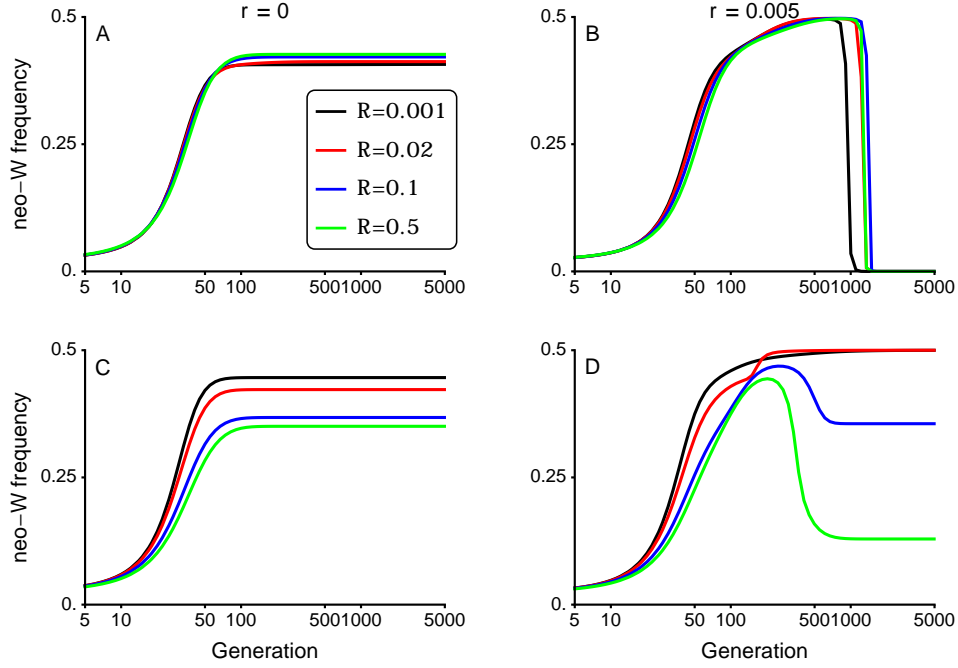


Figure S.2: Following invasion by a neo-W allele, there can be a complete transition to a new sex-determination system, maintenance of polymorphism at both ancestral-XY and neo-ZW sex determining regions, or loss of the new sex-determining allele. Here we plot the frequency of the neo-W allele among 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 (equilibrium A'), such that all females have the high fitness genotype aa and all males are 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 lines in Figure S.1A and S.1B near the ancestral SDR). In contrast, the neo-W is not lost in panel D as it is favoured near $r \approx 0$ (see blue lines in Figure S.1C and S.1D near the ancestral SDR). Fitness parameters are the same as in Figure S.1; in panels A and B the a allele is favoured in females ($w_{aa}^{\varnothing} = 1.05$, $w_{Aa}^{\varnothing} = 1$, $w_{AA}^{\varnothing} = 0.85$) while there is overdominance in males ($w_{aa}^{\delta} = w_{AA}^{\delta} = 0.75$) and in panels C and D, there is overdominance in both sexes ($w_{aa}^{\varnothing} = w_{AA}^{\varnothing} = 0.6$, $w_{aa}^{\delta} = 0.5$, $w_{AA}^{\delta} = 0.7$, $w_{Aa}^{\delta} = 1$). These parameters are marked by a dagger in Figure 2. Here, there is no haploid selection $I^{\delta} = \alpha_{\Delta}^{\delta} = 0$.

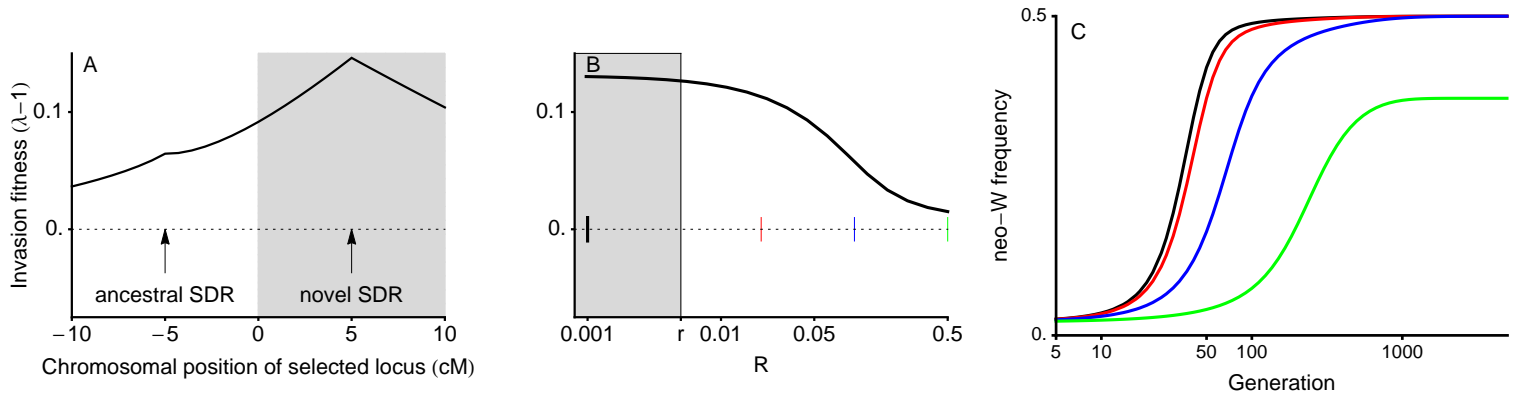


Figure S.3: When there is sexually-antagonistic selection and haploid selection, a neo-W may invade for any R . Panel A shows that the invasion fitness of a neo-W is positive where linkage is tight, even when $r < R$ (unshaded region). In panel B, we vary the recombination rate between the neo-W and 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 neo-W invasion are plotted in panel C (black $R = 0.001$, red $R = 0.02$, blue $R = 0.1$, green $R = 0.5$). The diploid selection parameters used in this plot are the same as in Figure 1. There is also meiotic drive in males favouring a ($\alpha_{\Delta}^{\delta} = -0.08$), this full set of parameters is marked by an asterisk in Figure S.4A. When $R = 0.5$ (green curve), the neo-W does not reach fixation and X,Y,Z, and W alleles are all maintained in the population, see Figure S.9C.

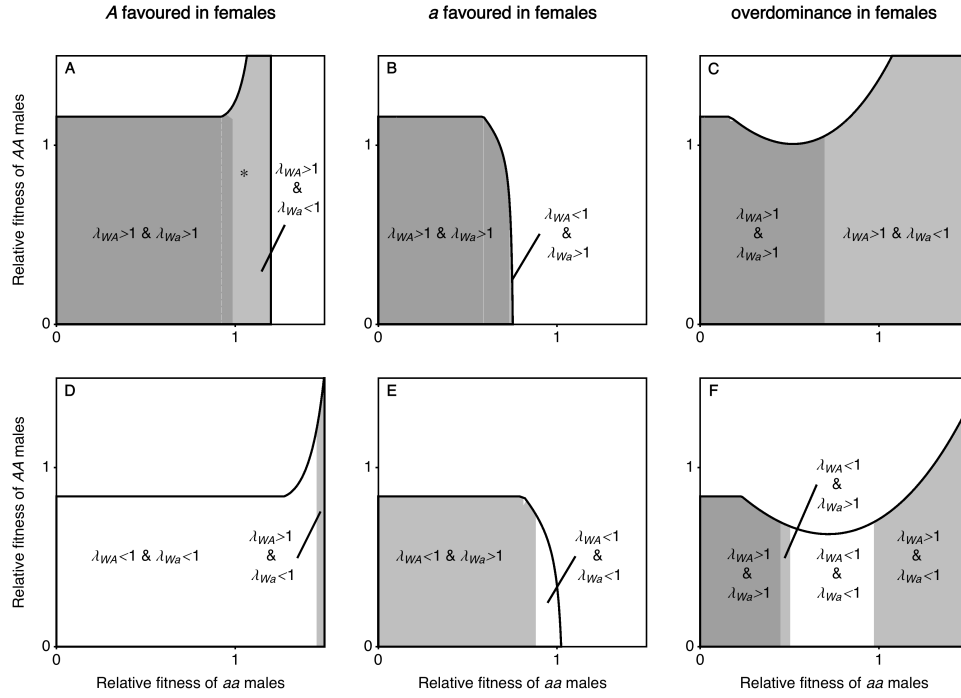


Figure S.4: Meiotic drive in males affects whether neo-W-A and neo-W-a haplotypes spread when the ancestral-XY locus is tightly linked to a locus under selection ($r = 0$). We vary the fitness of male homozygotes relative to heterozygotes ($w_{Aa}^{\varnothing} = 1$) and only consider stable equilibria at which both A locus allele are maintained and the *a* allele is initially fixed on the Y, region outlined. In panels A-C, meiotic drive in males favours the *a* allele ($\alpha_{\Delta}^{\delta} = -0.16$), creating male-biased sex ratios and generally increasing λ_{WA} and λ_{Wa} . By contrast, λ_{WA} and λ_{Wa} tend to be reduced when meiotic drive in males favours the *A* allele ($\alpha_{\Delta}^{\delta} = 0.16$), panels D-F. We consider three forms of selection in females: directional selection in favour of the *A* allele (panels A and D, $w_{aa}^{\varnothing} = 0.85$, $w_{AA}^{\varnothing} = 1.05$), direction selection in favour of the *a* allele (panels B and E, $w_{aa}^{\varnothing} = 1.05$, $w_{AA}^{\varnothing} = 0.85$), and overdominance (panels C and F, $w_{aa}^{\varnothing} = w_{AA}^{\varnothing} = 0.6$).

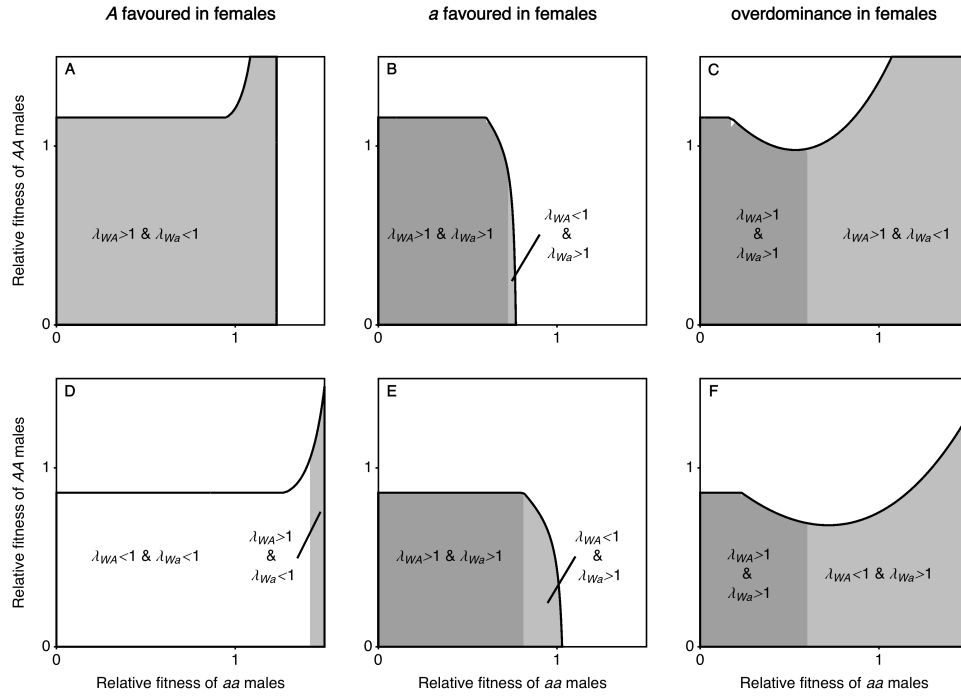


Figure S.5: Parameters for which neo-W-A and neo-W-a haplotypes spread when there is male gametic competition at a locus that is tightly linked to the ancestral-XY locus. Diploid selection parameters (w_{ij}^δ) are the same as those in Figure S.4. The *a* allele is favoured during male gametic competition in Panels A-C ($w_a^\delta = 1.16$, $w_A^\delta = 1$), which creates male biased sex-ratios and increases λ_{WA} and λ_{Wa} . On the other hand, the *A* allele is favoured during male gametic competition in Panels D-F ($w_a^\delta = 1$, $w_A^\delta = 1.16$) and λ_{WA} and λ_{Wa} tend to be reduced. Compared to the meiotic drive parameters in Figure S.4, the effect of these male gametic competition parameters on the sex ratio is smaller. For example, in Figure S.4A-C, the ancestral sex ratio is $\alpha^\delta = 0.58$ at equilibrium (B) and in panels A-C of this plot, the ancestral sex ratio is $w_a^\delta / (w_A^\delta + w_a^\delta) = 0.537$ at equilibrium (B).

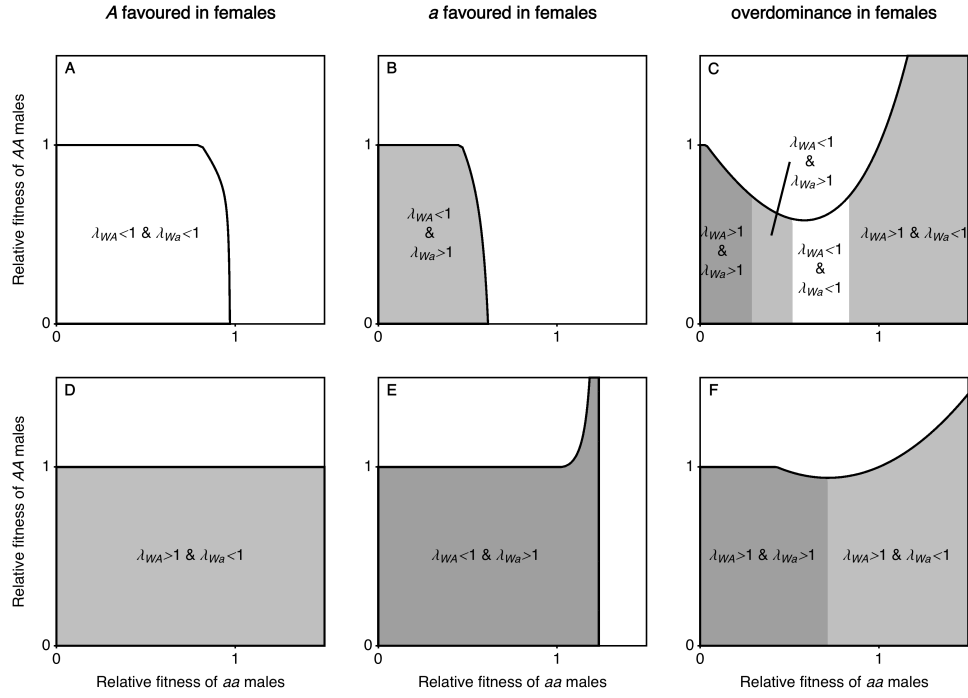


Figure S.6: Parameters for which neo-W-*A* and neo-W-*a* haplotypes spread when there is female meiotic drive at a locus that is tightly linked to the ancestral-XY locus. Diploid selection parameters (w_{ij}^{δ}) are the same as those in Figure S.4 and S.5. The *a* allele is favoured by meiotic drive in females in Panels A-C ($\alpha_{\Delta}^{\delta} = -0.16$), which increases λ_{Wa} and decreases λ_{WA} . Female meiotic drive in favour of the *A* allele (panels D-F, $\alpha_{\Delta}^{\delta} = -0.16$) has the opposite effect.

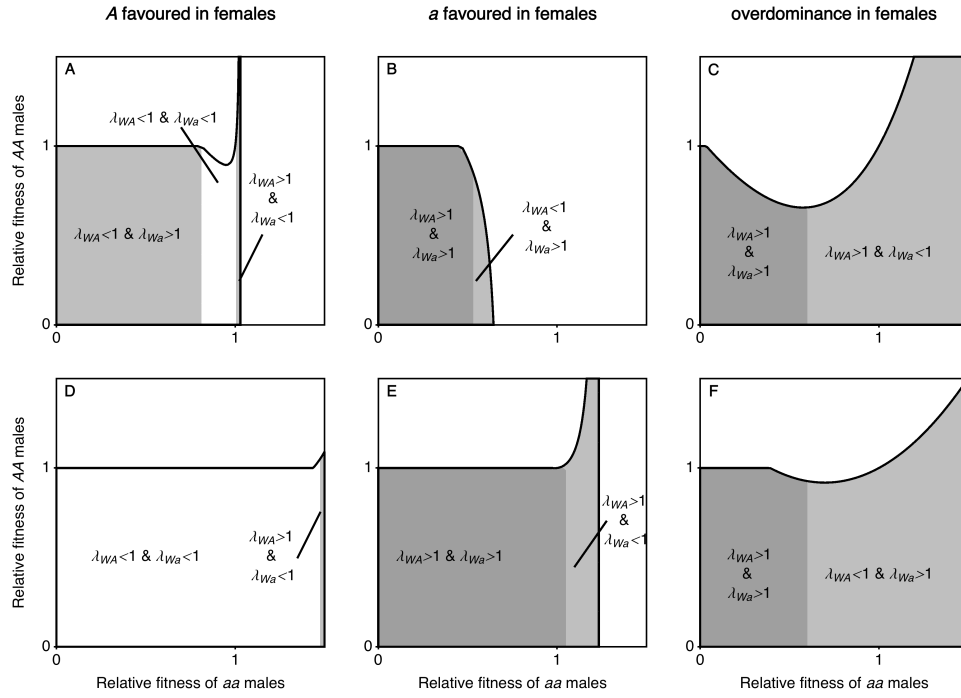


Figure S.7: Parameters for which neo-W-*A* and neo-W-*a* haplotypes spread when there is female gametic competition at a locus that is tightly linked to the ancestral-XY locus. Diploid selection parameters (w_{ij}^{ϕ}) are the same as those in Figure S.4, S.5, and S.6. The *a* allele is favoured during female gametic competition in females in Panels A-C ($w_a^{\phi} = 1.16$, $w_A^{\phi} = 1$), which increases λ_{Wa} and decreases λ_{WA} . The *A* allele is favoured during gametic competition in panels D-F ($w_a^{\phi} = 1$, $w_A^{\phi} = 1.16$), giving the opposite effect on λ_{Wa} and λ_{WA} .

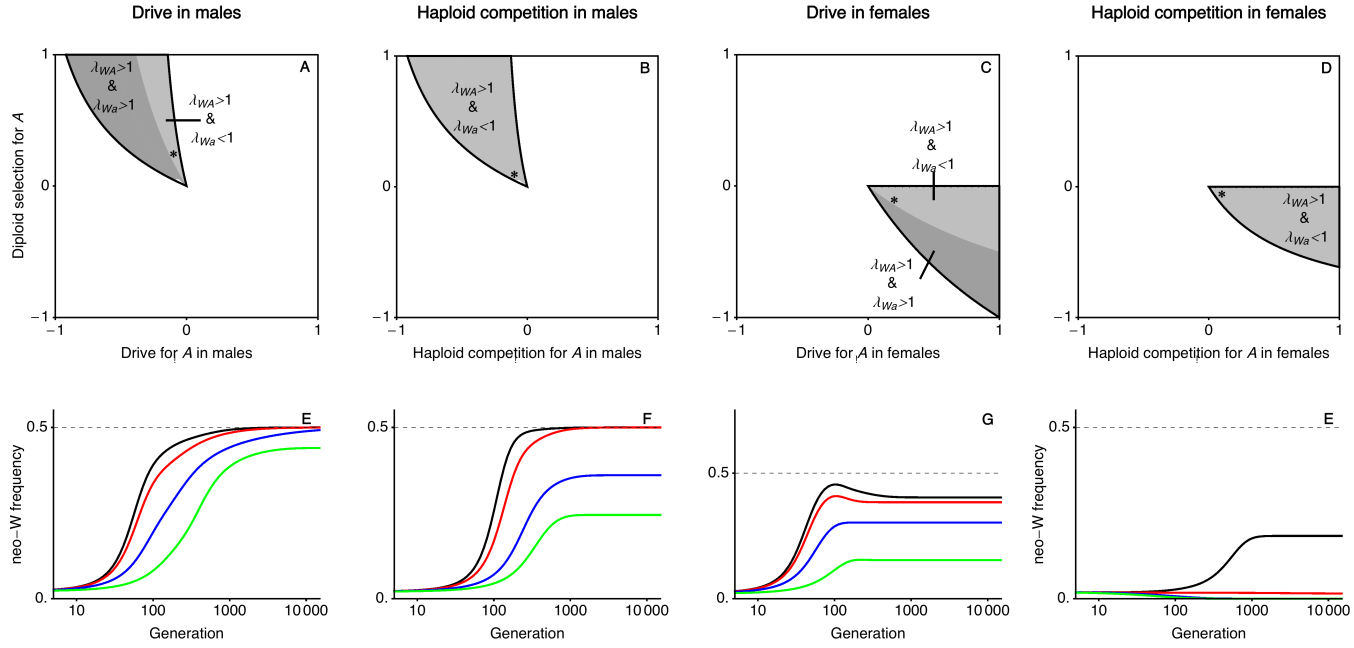


Figure S.8: A-D show when each of the neo-W haplotypes invade an internally stable equilibrium with a fixed on the Y (found by setting $r = 0$). The y-axis shows directional selection in diploids of both sexes, $s^{\varnothing} = s^{\delta}$, and the x-axes show sex-specific drive, α_{Δ}^{δ} , or haploid competition, t^{δ} . 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, $h^{\varnothing} = h^{\delta} = 3/4$. 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).

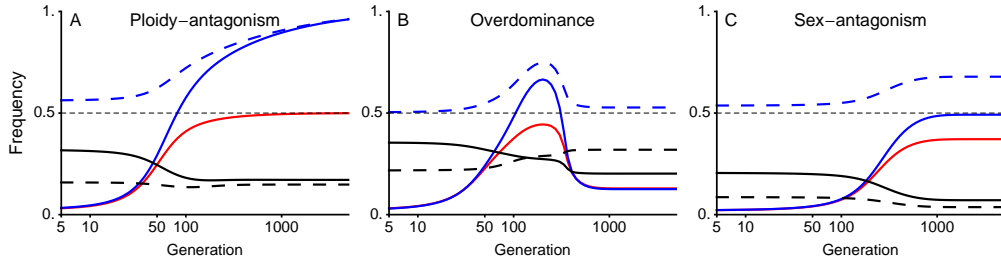


Figure S.9: Dynamics of sex-determining alleles during invasion by a neo-W allele. The curves show the frequencies of the neo-W (red), ancestral-Y (Blue), and *A* allele among female gametes (solid curves) and among male gametes (dashed curves). In panel A, there is a complete transition from XY sex determination (XX-ZZ females and XY-ZZ males) to ZW sex determination (YY-ZW females and YY-ZZ males). In panels B and C a polymorphism is maintained at both the ancestral XY locus and the neo-ZW locus, such that there are males with genotypes XY-ZZ or YY-ZZ and females with genotypes XX-ZZ, XX-ZW, XY-ZW, or YY-ZW. In panel A, selection is ploidy-antagonistic with drive in males (parameters as in the green curve in Figure 4B). In panel B, there is overdominance in both sexes and no haploid selection (parameters as in the green curve in Figure S.2C). In panel C, there is sexually-antagonistic selection in diploids with drive in males (parameters as in the green curve in Figure S.4C). In all cases, the initial equilibrium frequency has *a* near fixation on the Y.

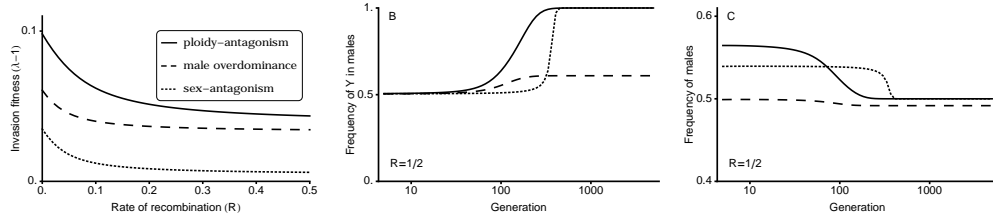


Figure S.10: An unlinked neo-W can invade a perfectly linked system with overdominance or haploid selection. Here overdominance leads to a polymorphic sex-determining system. Before invasion the population is at equilibrium B. Parameters: