

Gametic Selection, Meiotic Drive, Sex Ratio Bias, and Transitions Between Sex Determination Systems

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

* These authors contributed equally to this work

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

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

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

Contributions:

Abstract

2 Sex determination systems are remarkably dynamic; many studied taxa
display transitions of sex-determining genes between chromosomes or the
4 evolution of entirely new sex-determining systems. Predominant theories in
which new sex-determining systems are favoured by selection generally con-
6 clude that that novel sex determination systems are favoured if they equalise
the sex ratio or increase linkage between the sex-determining region and a
8 sexually-antagonistic locus. We use population genetic models to extend
these theories in two ways: (1) We explicitly consider how selection on very
10 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
12 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
14 (or unlinked) to the sex linked locus under selection; a result that is not found
with loose sex-linkage. (2) We also consider selection upon haploid geno-
16 types either during gametic competition (e.g., pollen/sperm competition) or
meiosis (i.e., non-Mendelian segregation); selective processes that typically
18 occur in one sex or the other. As well as having sex-specific fitness conse-
quences, haploid selection can cause the zygotic sex ratio to become biased
20 because sex ratios are determined by the production and fertilization success
of X- versus Y-bearing pollen/sperm (or Z- versus W-bearing ovules/eggs).
22 Consequently, selection for XY to ZW transitions and ZW to XY transitions
can be assymetrical when linkage between the ancestral sex-determining lo-
24 cus and a locus under haploid selection is tight, in which case ancestral sex
ratio biases can be strong. With looser linkage and haploid selection, we
26 again find that transitions between male and female heterogamety (XY to
ZW or ZW to XY) can occur even if the new sex-determining region is less
28 closely linked to the locus under selection. That is, favourable associations
that develop between the ancestral sex-determining locus and selected loci
30 can be broken during the spread of a new sex-determining region. Overall,
our models provide new predictions for the types of selection and the ge-
32 nomic location of loci that can drive transitions between sex-determination

systems.

34 abstract word count: ≈ 350

Introduction

36 Animals and angiosperms exhibit extremely diverse sex determination systems (re-
viewed in Bull 1983, Charlesworth and Mank 2010, Beukeboom and Perrin 2014,
38 Bachtrog et al. 2014). Among species with genetic sex determination of diploid
sexes, some taxa have heterogametic males (XY) and homogametic females (XX),
40 including mammals and most dioecious plants (Ming et al. 2011); whereas other
taxa have homogametic males (ZZ) and heterogametic females (ZW), including
42 Lepidoptera and birds. Within several taxa, the chromosome that harbours the
master sex-determining region changes. For example, transitions of the master
44 sex-determining gene between chromosomes or the evolution of new master sex-
determining genes have occurred in Salmonids (Li et al. 2011, Yano et al. 2012),
46 Diptera (Vicoso and Bachtrog 2015), and *Oryzias* (Myosho et al. 2012). In ad-
dition, many gonochoric clades with genetic sex determination exhibit transitions
48 between male (XY) and female (ZW) heterogamety, including lizards (Ezaz et al.
2009), eight of 26 teleost fish families (Mank et al. 2006), true fruit flies (Tephri-
50 tids, Vicoso and Bachtrog 2015), amphibians (Hillis and Green 1990), the an-
giosperm genus *Silene* (Slancarova et al. 2013), Coleoptera and Hemiptera (Beuke-
52 boom and Perrin 2014, plate 2). Indeed, in some cases, both male and female
heterogametic sex determination systems can be found in the same species, as ex-
54 hibited by some cichlid species (Ser et al. 2010) and *Rana rugosa* (Ogata et al.
2007). In addition, multiple transitions have occurred between genetic and en-
56 vironmental sex determination systems, e.g., in reptiles and fishes (Conover and
Heins 1987, Mank et al. 2006, Pokorná and Kratochvíl 2009, Ezaz et al. 2009, Pen
58 et al. 2010, Holleley et al. 2015).

Predominant theories in accounting for the spread of new sex determination
60 systems by selection involve fitness differences between sexes (e.g., sexually antag-
onistic selection) or sex ratio selection. van Doorn and Kirkpatrick (2007; 2010)
62 show that new sex-determining loci can be favoured if they arise in closer link-
age with a locus that experiences sexual antagonism. For example, linkage allows
64 favourable associations to build up between a male-beneficial allele and a neo-Y

chromosome. Such associations can favour a new master sex-determining gene on
66 a new chromosome (van Doorn and Kirkpatrick 2007) and can also favour a tran-
sition between male and female heterogamety (e.g., a ZW to XY transition, van
68 Doorn and Kirkpatrick 2010). However, any sexually-antagonistic loci that are
more closely linked to the ancestral sex-determination locus will develop similar,
70 favourable associations and select against the spread of a new sex-determination
system. Here, we extend these studies by explicitly calculating the the equilib-
72 rium allele frequencies of loci that are very tightly linked to the ancestral sex-
determining region.

74 The sex ratio is directly affected by the sex determination system, it has there-
fore been suggested that sex ratio selection is a dominant force in the evolution of
sex determination (e.g., Bull 1983, p66-67; Beukeboom and Perrin 2014, Chapter
7). ‘Fisherian’ sex ratio selection favours a 1:1 zygotic sex ratio when assuming
78 that males and females are equally costly to produce (Fisher 1930, Charnov 1982).
This follows from the fact that, for an autosomal locus, half of the genetic material
80 is inherited from a male, and half from a female (West 2009). Thus, if the popula-
tion sex ratio is biased towards females, the average per-individual contribution of
82 genetic material to the next generation from males is greater than the contribution
from females (and vice versa for male-biased sex ratios). Therefore, a mutant that
84 increases investment in males (e.g., increases the proportion of males produced)
will spread via the higher per-individual contributions made by males. In the case
86 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
88 Y chromosomes are inherited disproportionately often), which causes sex ratios
to become biased (Hamilton 1967). They find that new, unlinked sex-determining
90 loci (masculinizing or feminizing mutations, i.e., neo-Y or neo-W loci) can then
spread, which restore an even sex ratio.

92 Here, we use mathematical models to find the conditions under which new
sex determination systems are favoured when loci experience haploid selection.
94 Haploid genotypes at many loci experience selection during gamete competition

and/or meiotic drive (Mulcahy et al. 1996, Joseph and Kirkpatrick 2004). We
96 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 com-
98 petition’ to refer to selection upon haploid genotypes within a gamete/gametophyte
pool (potentially from by multiple parents); the term ‘haploid selection’ encom-
100 passes both processes. Meiotic drive generally occurs either during the production
of male or female gametes only (Úbeda and Haig 2005, Lindholm et al. 2016). Be-
102 cause there are typically many more pollen/sperm than required for fertilization,
gametic competition is also typically sex specific, occurring primarily among male
104 gametes. 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
106 stronger signatures of selection than random genes (Borg et al. 2009, Arunkumar
et al. 2013, Gossmann et al. 2014). In addition, artificial selection pressures ap-
108 plied to male gametophytes are known to cause a response to selection (e.g., Hor-
maza and Herrero 1996, Ravikumar et al. 2003, Hedhly et al. 2004, Clarke et al.
110 2004) and gametic selection appears to occur during the creation of F2 crosses
(Kumar, 2007). A much smaller proportion of genes are thought to be expressed
112 and selected during competition in animal sperm, although precise estimates are
uncertain (Zheng et al. 2001, Joseph and Kirkpatrick 2004, Vibranovski et al. 2010,
114 Immler et al. 2014).

There are various ways in which a period of haploid selection could influence
116 transitions between sex determination systems. Firstly, if we assume that haploid
selection at any particular locus predominantly occurs in one sex (e.g., meiotic
118 drive during spermatogenesis), then such loci experience a form of sex-specific
selection. In this respect, we might expect that haploid selection would affect
120 transitions between sex determination systems in a similar manner to sex-specific
diploid selection (as explored by van Doorn and Kirkpatrick 2007; 2010). That
122 is, new masculinizing mutations (neo-Y chromosomes) could be favoured via asso-
ciations with alleles that are beneficial in the male haploid stage. However, sex
124 ratios can also become biased by linkage between the sex-determining region and

a locus that harbours genetic variation in haploid fitness. For example, there are
126 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
128 (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
130 systems would be influenced by the combination of sex ratio biases and associa-
tions between haploid selected loci and sex-determining regions.

132 Our models tracking the spread of new sex determination systems therefore
have two important new features. Firstly, we consider loci that are under selec-
134 tion and also in very tight linkage with the ancestral sex-determining region. Sec-
ondly, we allow sex-specific haploid selection to occur on a locus in tight or loose
136 linkage with the ancestral sex-determining region. We find that sex ratio biases
caused by haploid selection can exert Fisherian sex ratio selection upon novel sex-
138 determiners but that their spread is also determined by the fitness of the alleles that
are associated with them. Indeed, it is only when haploid selected loci are tightly
140 linked to the ancestral sex-determining region (and so sex ratio biases are initially
large) that we see an asymmetry between selection for XY to ZW transitions and
142 ZW to XY transitions, e.g., because haploid selection in males only causes bi-
ased zygotic sex ratios in an ancestrally XY system. In addition, we show that
144 transitions between male and female heterogamety can evolve even when the neo-
sex-determining locus is less closely linked to a locus under selection and therefore
146 disrupts favourable ancestral associations between sex and the alleles selected in
that sex. Such transitions are not favoured in models lacking tight linkage and/or
148 haploid selection.

Model

150 We consider the transition between an ancestral and novel sex determination sys-
tems using a three locus model. Locus **X** is the ancestral sex-determining region,
152 with alleles *X* and *Y* (or *Z* and *W*). Locus **A** is a locus under selection, with al-

leles A and a . Locus \mathbf{M} is a novel sex-determining region, at which the null allele
 154 (M) is initially fixed in the population such that sex of zygotes is determined by
 the genotype at the ancestral sex-determining region, \mathbf{X} ; XX genotypes become
 156 females and XY become males (or ZW become females and ZZ become males).
 To evaluate the evolution of new sex-determination systems, we consider the in-
 158 vasion, fixation, maintenance, and/or loss of novel sex-determining alleles (m) at
 the \mathbf{M} locus. We assume that the \mathbf{M} locus is epistatically dominant over the \mathbf{X}
 160 locus such that zygotes with at least one m allele develop as females with proba-
 bility k and as males with probability $1 - k$, regardless of the \mathbf{X} locus genotype.
 162 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 , the m allele confers
 164 environmental sex determination (ESD) such that zygotes develop as females in
 a proportion (k) of the environments they experience. Finally, we also analyze a
 166 model of maternally-controlled environmental sex-determination, where mothers
 with at least one m allele produce daughters with probability k .

168 In each generation, we census the genotype frequencies in male and female
 gametes/gametophytes (hereafter gametes) before gametic competition. A full de-
 170 scription of our model, including recursion equations, is given in the Appendix.
 First, competition occurs among male gametes (sperm/pollen competition) and
 172 among female gametes (egg/ovule competition) separately. Selection during ga-
 metic competition depends on the \mathbf{A} locus genotype, relative fitnesses are given
 174 by $w_A^{\mathfrak{f}}$ and $w_a^{\mathfrak{f}}$ ($\mathfrak{f} \in \{\mathfrak{f}, \mathfrak{m}\}$; see table 1). We assume that all gametes compete for
 fertilization during gametic competition, which is not the case for monogamous
 176 mating systems where gametes from only one mating partner are present. Ga-
 metic competition in monogamous mating systems is equivalent to meiotic drive
 178 in our model, which only alters the frequency of gametes produced by heterozy-
 gotes. After gametic competition, random mating occurs between male and female
 180 gametes. The resulting zygotes develop as males or females, depending on their
 genotypes at the \mathbf{X} and \mathbf{M} loci (and the \mathbf{M} genotype of their mother in the case
 182 of maternal control) as described above. Diploid males and females then expe-

184 rience selection, relative fitnesses are given by w_g^δ in males and w_g^φ in females,
 where g is the diploid genotype at the **A** locus ($g \in \{AA, Aa, aa\}$). The next gen-
 186 eration of gametes is then produced by meiosis, during which recombination and
 sex-specific meiotic drive can occur. Recombination (i.e., an odd number of cross-
 188 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 χ . Therefore, any order
 of the loci can be modelled with appropriate choices of r , R , and χ (see Table
 190 S.1). Males/females that are heterozygous at the **A** locus may experience meiotic
 drive; Aa heterozgotes of sex φ produce gametes bearing allele A with probability
 192 α^φ . Thus, the **A** locus can experience sex-specific gametic competition, diploid
 selection and/or meiotic drive.

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

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

194 Results

The only asymmetry between males and females in our model is that, under the
 196 ancestral sex determination system, males develop with genotype XY (or ZZ)
 and females with genotype XX (or ZW). Therefore, the model outlined above

describes both ancestrally- XY and ancestrally- ZW sex determination systems if we relabel the two sexes as being ancestrally ‘heterogametic’ or ancestrally ‘homogametic’. Without loss of generality, we primarily refer to the ancestrally heterogametic sex as male and the ancestrally homogametic sex as female. That is, we describe an ancestral XY sex determination system but our model can easily be applied to an ancestral ZW sex determination system.

Turnover between sex-determination systems

The evolution of a new sex determination system requires that a rare mutant allele, m , at the novel sex-determining locus increases in frequency when rare. The spread of a rare mutant m at the \mathbf{M} locus is determined by the leading eigenvalue, λ , of the system of eight equations describing the next generation frequency of eggs and sperm carrying the mutation, (S.1c), (S.1d), (S.1g), (S.1h). 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 (for a neo-Y) or by females (for a neo-W). Furthermore, if the m allele is fully dominant over the ancestral sex-determining system, phenotypes are not affected by the genotype at the ancestral sex-determining region (\mathbf{X} locus). Thus, the invasion of rare dominant neo-Y or neo-W alleles is determined by the largest eigenvalue that solves the quadratic characteristic polynomial $\lambda^2 + b\lambda + c = 0$. In this case $b = -(\lambda_{mA} + \lambda_{ma})$ and $c = \lambda_{mA}\lambda_{ma} - \rho_{mA}\rho_{ma}$, where λ_{mi} is the (discrete time) growth rate of mutant haplotypes on background $i \in \{A, a\}$, accounting for loss due to recombination, and ρ_{mi} is the rate of addition of mutant haplotypes onto background $i \in \{A, a\}$ due to recombination, see table 2. The spread of the mutant m allele depends on the frequency of alleles at the other loci in the ancestral population. In the ancestral population, it is convenient to follow the frequency of the A allele in female gametes (eggs) from an XX female, p_X^ϕ , and in X -bearing, p_X^δ , and Y -bearing, p_Y^δ , male gametes (sperm). 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.

228 I now remove the zygotic sex ratio ζ from the mean fitnesses. The mean fitnesses will have to be adjusted in a corresponding way.

230 CHECK: DO THE HAPLOID MEAN FITNESSES HAVE TO BE IN THE DENOMINATOR?

232

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

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

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

R is the probability of recombination between loci \mathbf{A} and \mathbf{M} .

ζ is the zygotic sex ratio and \bar{w}^{φ} is the mean fitness of sex φ , see Table S.2.

234 We are particularly concerned with the conditions under which a rare neo-sex-determining allele increases in frequency, which occurs when the largest eigen-
236 value, λ , is greater than one. If the average change in frequency of the two haplotypes that carry the m allele (Am and am) is positive, invasion will always occur
238 (i.e., if $\{(\lambda_{mA} - 1) + (\lambda_{ma} - 1)\} / 2 > 0$ then $\lambda > 1$). If neither haplotype increases in frequency ($\lambda_{mA}, \lambda_{ma} < 1$), the m allele will not invade. Otherwise, the new sex-determining allele increases in frequency on one \mathbf{A} background and declines on
240

the other and invasion depends on the recombination rate between the **M** and **A**
 242 loci (R) see equations (S.5) and (S.6).

Table 2 illustrates a number of key points about the invasion of neo-Y and
 244 neo-W mutations. Firstly, Fisherian sex ratio selection will favour the spread of
 a neo-Y if the ancestral zygotic sex ratio is biased towards males, $\zeta > 1/2$, and
 246 vice versa for a neo-W, see terms in square brackets. However, the spread of a
 neo-Y (neo-W) also depends on the male (female) fitness of alleles that they are
 248 associated with, see terms in curly brackets. Secondly, since a dominant neo-Y is
 always found in males, the allele frequencies at the neo-Y (**M**) locus only change in
 250 males. Therefore, invasion by a neo-Y allele does not involve any female diploid
 selection terms (w_g°). Similarly, invasion by a neo-W is driven by the fitness of
 252 female gametes and diploids and does not involve any direct selection in male
 diploids.

254 Finally, the diploid fitness terms in Table 2 are weighted by the probability
 of producing those genotypes through matings with gametes of the opposite sex.
 256 For example, matings between a neo-Y-bearing male gamete and an A -bearing
 female gamete occur with probability $p_X^\circ w_A^\circ / \bar{w}_H^\circ$. The probability that a neo-W
 258 bearing female gamete mates with an A -bearing male gamete is $\bar{p}^\delta w_A^\delta / \bar{w}_H^\delta$, where
 $\bar{p}^\delta = p_Y^\delta q + p_X^\delta (1 - q)$ is the frequency of the A allele among both X- and Y-
 260 bearing male gametes. That is, in the case of a neo-W, female diploids can result
 from matings with either an X-bearing or a Y-bearing sperm, resulting in zygotes
 262 that will develop as females. However, females that do not carry the neo-W only
 result from matings with X-bearing sperm. Therefore, eggs with and without a
 264 neo-W can differ in the frequency of A alleles they obtain from matings with male
 gametes. Invasions by a neo-Y and a neo-W differ in this respect because sperm
 266 with or without a neo-Y allele both mate with X-bearing female gametes only.

In order to explicitly determine the conditions under which a rare neo-sex-
 268 determining allele spreads, we must calculate the frequency of the A allele in the
 ancestral population (i.e., p_X° , p_X^δ , and p_Y^δ). We assume that the A allele reaches a
 270 stable equilibrium frequency under the ancestral sex-determination system before

the neo-sex-determining allele (m) arises. We can analytically calculate the allele
 272 frequency of the A allele using two alternative simplifying assumptions: (1) as-
 suming that the A locus is within the non-recombining region around the ancestral
 274 SDR (or within tight linkage, $r \approx 0$) or (2) assuming that selection is weak relative
 to recombination ($s^\phi, t^\phi, \alpha_\Delta^\phi$ of order ϵ).

276 When there is tight linkage between the ancestral sex-determining region and
 the A locus ($r = 0$), either the A allele or the a allele must be fixed on the Y.
 278 Because the labelling of alleles is arbitrary, we will assume that the a locus is fixed
 on the Y without loss of generality ($p_Y^\phi = 0$). If there are two alleles maintained at
 280 the A locus, the X can either be fixed for the A allele ($p_X^\phi = p_X^\phi = 1$) or polymorphic
 ($0 < p_X^\phi, p_X^\phi < 1$). These equilibrium allele frequencies and their stability conditions
 282 are given in the appendix.

A neo-Y will never invade an ancestral XY system that already has tight linkage
 284 with the locus under selection ($r = 0$). When then neo-Y is also tightly linked
 ($R = 0$) a neo-Y will either remain linked to the A allele or to the a allele and so
 286 invasion is given directly by the larger of λ_{mA} or λ_{ma} (evaluated with $R = 0$). A
 neo-Y can either be linked to the same allele as the ancestral Y, in which case it
 288 is a neutral mutation with no effect ($\lambda_{ma} = 1$), or be linked to the alternative A
 allele, in which case it will not spread given that the initial equilibrium is stable
 290 ($\lambda_{mA} < 1$). Given that λ_{mA} and λ_{ma} both decrease with increasing R , more loosely
 linked neo-Y alleles also do not spread ($\lambda < 1$ when $R > 0$).

292 However, under some conditions, a neo-W can invade an ancestral XY system.
 When the neo-W is also tightly linked ($R = 0$), it can spread in linkage with either
 294 the allele that is fixed on the Y or the allele that is more common on the X, i.e.,
 under some conditions $\lambda_{ma} > 1$ and/or $\lambda_{mA} > 1$. These conditions are given in
 296 more detail in the appendix.

Under weak selection, we denote the leading eigenvalues describing the inva-
 298 sion 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, which are given by

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

300 and

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

where $V_A = \bar{p}(1-\bar{p})$ is the variance in the frequency of A and $S_A = (D^\delta + \alpha_\Delta^\delta + t^\delta) -$
 302 $(D^\varnothing + \alpha_\Delta^\varnothing + t^\varnothing)$ is the difference in fitness in males versus females for the A allele
 against the a allele across diploid selection, gametic competition, and meiosis.
 304 $D^\varnothing = (\bar{p}s^\varnothing + (1-\bar{p})h^\varnothing s^\varnothing) - (\bar{p}h^\varnothing s^\varnothing + (1-\bar{p}))$ is the difference in fitness between
 A and a alleles in diploids of sex $\varnothing \in \{\varnothing, \delta\}$; \bar{p} is the leading-order probability of
 306 mating with an A -bearing gamete from the opposite sex, see appendix.

The neo-sex-determining allele m will spread if $\lambda_{m,XY} > 1$. Equation (1)
 308 demonstrates that a neo-Y will invade 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
 310 that V_A and S_A^2 are strictly positive as long as \mathbf{A} is polymorphic). This result echoes
 that of van Doorn and Kirkpatrick (2007), who considered diploid selection only
 312 and also found that homogametic transitions (XY to XY or ZW to ZW) can oc-
 cur when the neo-sex-determining locus is more closely linked to a locus under
 314 sexually-antagonistic selection.

Equation (2) shows that if there is no selection upon haploid genotypes ($t^\varnothing =$
 316 $\alpha_\Delta^\varnothing = 0$), as considered by van Doorn and Kirkpatrick (2010), the spread of a
 neo-W is equivalent to the spread of a neo-Y ($\lambda_{W',XY} = \lambda_{Y',XY}$) such that het-
 318 erogametic 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 ($R < r$).
 320 However, if there is any haploid selection, the additional term in equation (2) can
 be positive, which can allow, for example, neo-W invasion ($\lambda_{W',XY} > 1$) even when
 322 the neo-sex-determining region is less closely linked to the selected locus ($R > r$).
 These transitions are unusual because, when $R > r$, associations that have built up

324 between alleles more favourable in one sex and that sex will be weakened. There-
fore, mean fitness can decrease (Figure 2B,D).

326 We find that 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
328 consider several special cases. Firstly, if the **A** locus is unlinked to the ancestral
sex-determining region ($r = 1/2$), a more closely linked neo-W ($R < 1/2$) can
330 always invade because $(\hat{p}_Y^\delta - \hat{p}_X^\delta) = 0$ such that the second term in equation (2)
disappears and invasion depends only on the sign of $(r - R)$. Indeed, invasion
332 typically occurs when the neo-W is more closely linked to the selected locus than
the ancestral sex-determining region (Figure 3). Secondly, we can simplify cases
334 where invasion occurs despite $R > r$ using the special case where $R = 1/2$ and
 $r < 1/2$. In table 3 we give the conditions where invasion occurs where we further
336 assume that haploid selection only occurs in one sex (e.g., during male meiosis
only) and dominance coefficients are equal in the two sexes, $h^\varphi = h^\delta$. Where
338 there is no gametic competition and meiotic drive in one sex only, an unlinked
neo-W can invade as long as the same allele is favoured during diploid selection in
340 males and females ($s^\varphi s^\delta > 0$, see Figure 3B). Where there is no meiotic drive and
gametic competition occurs in one sex only, an unlinked neo-W can invade as long
342 as the same allele is favoured in male and female diploid selection and there are sex
differences in selection of one type (e.g., $s^\varphi(s^\delta - s^\varphi) > 0$, see Figure 3C,D). These
344 special cases indicate that neo-W invasion can occur for a relatively large fraction
of parameter space, even if the neo-W uncouples the sex-determining locus from
346 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	$h^\delta = h^\varphi, t^\delta = \alpha_\Delta^\varphi = \alpha_\Delta^\delta = 0$	$s^\delta(s^\varphi - s^\delta) > 0$

Previous research suggests, when the ancestral sex-determining locus is linked
 348 to a locus that experiences haploid selection (e.g., meiotic drive), a new, unlinked
 sex-determining locus invades in order to restore equal sex ratios (Kozielska et al.
 350 2010). Our model provides a good opportunity to determine whether Fisherian
 sex ratio selection provides a useful explanation for the evolution of new sex-
 352 determining loci in other contexts. Consider, for example, the case where the **A**
 locus is linked to the ancestral-SDR ($r < 1/2$) and experiences meiotic drive in
 354 males only (e.g., during spermatogenesis but not during oogenesis, $\alpha^\delta \neq 1/2$,
 $\alpha^\varnothing = 1/2$). We will also disregard gametic competition ($t^\varnothing = t^\delta = 0$) such that
 356 zygotic sex ratios are only biased by meiotic drive in males. In this case, the zy-
 gotic sex ratio can be initially biased only if the ancestral sex-determining system
 358 is XY (Figure 1B). If the ancestral sex-determining system is ZW, the zygotic sex
 ratio will be 1:1 because diploid sex is determined by the proportion of Z-bearing
 360 versus W-bearing eggs (and meiosis in females is fair, Figure 1D). Thus, if the
 zygotic sex ratio is crucial to the evolution of new genetic sex-determining sys-
 362 tems, invasion into ZW and XY systems will be distinct. However, we find that
 invasion by a homogametic neo-sex-determining allele (XY to XY, or ZW to ZW)
 364 or by a heterogametic neo-sex-determining allele (XY to ZW or ZW to XY) oc-
 cur under the same conditions. That is, we can show that $\lambda_{Y',XY} = \lambda_{W',ZW}$ and
 366 $\lambda_{Y',ZW} = \lambda_{W',XY}$ (at least up to order ϵ^3 ; for a numerical example, compare Figure
 1A,B to Figure 1C,D).

368 We next consider the case where the new sex-determining mutation, m , causes
 sex to be determined stochastically or by environmental conditions (environmental
 370 sex determiner, ESD). We assume that individuals carrying the m allele develop as
 females in a fraction, k , of the environments they experience. The spread of these
 372 mutations is given by

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

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

374 Under Fisherian sex ratio selection, autosomal modifiers favour equal invest-
 376 ment in male and female offspring, i.e., a 1:1 sex ratio (Fisher 1930, Charnov
 378 1982, West 2009). Therefore, a novel environmental sex-determiner that causes
 half of its carriers to become female and half to become male ($k = 1/2$) will be in
 males half of the time and in females half of the time (like an autosome). In addi-
 380 tion, these novel sex-determination alleles equalize the sex ratio and so one might
 expect them to be favoured by Fisherian sex ratio selection when the resident sex
 ratio is biased. However, we find that the growth rate of a rare, dominant offspring-
 382 controlled neo-ESD allele that produces males or females with equal probability
 ($k = 1/2$) is

$$\lambda_{ESD',XY} = 1 + \frac{1}{2} \frac{(\lambda_{Y',XY} - 1) + (\lambda_{W',XY} - 1)}{2} \Big|_{R=1/2} + O(\epsilon^3), \quad (4)$$

384 where we have indicated that $\lambda_{Y',XY}$ and $\lambda_{W',XY}$ are evaluated at $R = 1/2$. Re-
 combination between the selected locus and the novel sex-determining locus, R ,
 386 doesn't enter into the $k = 1/2$ results because sex is essentially randomized each
 generation, preventing associations from building up between allele A and sex.

388 Equation (4) shows that invasion by a novel 'perfect' ESD (equal sex ratio,
 $k = 1/2$) mutation is the same for an ancestrally XY or ZW system (since $\lambda_{Y',XY} =$
 390 $\lambda_{W',ZW}$, $\lambda_{W',XY} = \lambda_{Y',ZW}$). Thus, by the same argument as above (if drive occurs
 in males only then the sex ratio is only biased when the ancestral sex-determination
 392 system is XY), Fisherian sex ratio selection does not explain invasion by an offspring-
 controlled neo-ESD locus. Rather, the neo-ESD gets half of the fitness of a femi-

394 nizing mutation (neo- W) and half of the fitness of a masculinizing mutation (neo- Y),
 396 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). The net result
 can be that perfect ESD will not invade, even if current sex ratios are biased. For
 398 example, if there is haploid selection in males (either drive or pollen/sperm com-
 petition) but the conditions in table 3 are not met, perfect ESD will not invade,
 400 even though it would equalize the zygotic sex ratio from an initially biased case
 (assuming $r < 1/2$).

402 Fisherian sex ratio selection is sometimes considered in terms of balancing
 parental investment in male versus female offspring (Charnov 1982). In addition,
 404 under environmental sex-determination, the proportion of males/females is some-
 times controlled by the mother, e.g., the proportion of eggs laid in warm versus
 406 cold environments could determine the sex ratio of offspring. We therefore also
 considered the invasion of a neo-sex-determining allele (m) in a model in which
 408 mothers that have at least one m allele produce daughters with probability k . As
 with offspring-controlled ESD, for all $k \in \{0, 1/2, 1\}$, we find that invasion into
 410 an ancestrally XY system is the same as invasion into an ancestrally ZW system (at
 least up to order ϵ^3), implying transitions between genetic sex determination and
 412 maternally controlled environmental sex determination are not driven by Fisherian
 sex ratio selection on biased zygotic sex ratios.

414 Discussion

Because linkage between haploid selected loci and sex-determining regions causes
 416 biased zygotic sex ratios (Hamilton 1967, Burt and Trivers 2006, Field et al. 2012;
 2013), one might expect Fisherian sex ratio selection to drive the spread of new
 418 sex-determining systems that bring the sex ratio closer to 50:50. Fisherian sex
 ratio selection follows from the fact that, for an autosomal locus, half of the ge-
 420 netic material is inherited from a male, and half from a female (Fisher 1930, West
 2009). Thus, if the population sex ratio is biased towards females, the average

422 per-individual contribution of genetic material to the next generation from males
is greater than the contribution from females (and vice versa for male-biased sex
424 ratios). Therefore, a mutant that increases investment in males will spread via
the higher per-individual contributions made by males. An implicit assumption
426 of Fisherian sex ratio selection is that the mutant allele is autosomal and has the
same inheritance pattern as the non-mutant allele. The mutations we consider
428 here, neo-sex-determining alleles, break this assumption. For example, the suc-
cess of neo-Y/neo-W mutations depends only on the number of alleles contributed
430 by males/females (Table 2). In this respect, a neo-W is similar to a cytoplasmic el-
ement, which also does not experience selection to balance sex ratios (Frank 1989,
432 Werren and Beukeboom 1998, Chase 2007). Even mutants that are equally likely
to be found in males or females, such as an environmental sex determination mu-
434 tation (equation 4), are not strictly autosomal if they determine sex. Thus, despite
the fact that sex ratio biases caused by gametic competition or meiotic drive have
436 been shown to exert Fisherian sex ratio selection on various autosomal modifiers
(Stalker 1961, Smith 1975, Frank 1989, Hough et al. 2013, Úbeda et al. 2015, Otto
438 et al. 2015), we do not find evidence of Fisherian sex ratio selection acting dur-
ing invasion by neo-sex-determination systems (e.g., see Figure 1 and Úbeda et al.
440 2015, in which a neo-Y invades despite biasing sex ratios).

We note two other ways in which sex determination has been shown to relate
442 to zygotic sex ratios. Firstly, female-biased sex ratios can be favoured when there
is local mate competition, where all matings are between siblings and assuming
444 one male can inseminate many females (Hamilton 1967). Therefore, with local
mate competition, feminizing mutations can spread because they bias the sex ratio
446 towards females (Wilson and Colwell 1981, Vuilleumier et al. 2007). Secondly,
environmental conditions (e.g., maternal condition, mate quality, age, or host size)
448 can differentially affect the fitness of males versus females such that the optimal al-
location to males/females depends on the environment (Trivers and Willard 1973,
450 Charnov and Bull 1977, Charnov 1982). In such cases, flexible sex determination
systems may evolve in order to allow the zygotic sex ratio to be determined in a

452 way that depends on the environment (Charnov and Bull 1977, Werren and Taylor
1984, Pen et al. 2010). In this study, we do not consider environmental condi-
454 tion dependence or local mate competition (reviewed in Charnov 1982, Bull 1983,
West 2009).

456 It has previously been demonstrated that new sex-determining systems can
evolve if there is genetic variation maintained by sexually-antagonistic selection
458 (van Doorn and Kirkpatrick 2007; 2010). In particular, transitions to new sex-
determining systems can occur when new sex-determining regions are more closely
460 linked to a sexually-antagonistic locus. Our results show that genetic variation at
loci that experience haploid selection can also generate selection in favour of new
462 sex-determining systems. New sex-determining alleles are again favoured if they
are linked with a locus under haploid selection and the ancestral sex-determination
464 locus is not. However, with haploid selection, heterogametic transitions (XY to
ZW or ZW to XY) can also occur when the new sex-determining region is less
466 closely linked to the locus under selection.

Neo-W (neo-Y) alleles invade when their fitness in females (males) is greater
468 than the mean fitness of females (males) under the ancestral sex determination
system. With sexually antagonistic selection (between diploid sexes) only, linkage
470 between a selected locus and the sex-determining region strengthens associations
between male beneficial alleles and the male-determining allele (Y or Z) and be-
472 tween female beneficial alleles and the female-determining allele (X or W). Thus,
the mean fitness of both males and females increases with closer linkage to the sex-
474 determining region. Therefore, new sex-determining alleles only invade if they are
more closely linked than the ancestral sex-determining region. However, if there
476 is haploid selection on loci linked to an XY (ZW) sex-determining region, selec-
tion can maintain polymorphisms at which the mean fitness of females (males)
478 or males is lower than it would be without sex-linkage. In these cases, unlinked
neo-W (neo-Y) alleles can invade, see figure 2.

480 We assume that sex-determining alleles do not experience direct selection ex-
cept via their associations with sex and alleles at a selected locus. However, in

482 some cases, there may be significant degeneration around the sex-limited allele (Y
or W) in the ancestral sex determining region because recessive deleterious muta-
484 tions and/or deletions may fix around the Y or W allele (Rice 1996, Charlesworth
and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). During heterogametic
486 transitions (XY to ZW or ZW to XY), the formally sex-limited allele fixes such
that all individuals have YY or WW genotypes (Figure 1). Any recessive delete-
488 rious alleles linked to the Y or W will therefore be revealed to selection during a
heterogametic transition. This phenomenon was studied by van Doorn and Kirk-
490 patrick (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-
492 and neo- sex-determining loci are both polymorphic. However, they noted that
very rare recombination events around the ancestral sex-determining region can
494 allow these heterogametic transitions to complete. While not explicitly studied,
we also predict that Y or W degeneration would prevent fixation of the new sex-
496 determiners considered here.

In addition, our model of meiotic drive is simple, involving a single locus with
498 two alleles. However, many meiotic drive systems involve an interaction with an-
other locus at which alleles may ‘suppress’ the action of meiotic drive (Burt and
500 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 recombina-
502 tion rate between them, which in turn can be affected by sex-linkage if there is re-
duced recombination between sex chromosomes (Hurst and Pomiankowski 1991).
504 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 is a two-locus drive sys-
506 tem and the total number of gametes produced can be reduced by meiotic drive.
Where gamete number is reduced by meiotic drive, the number of mates com-
508 peting for fertilization (mating system) can affect the equilibrium frequency of a
meiotic drive allele (Holman et al. 2015). In polygamous mating systems, the in-
510 tensity 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

512 2002). Since the sex ratio is partly determined by the sex determination system, the
evolution of new sex determination system could be influenced by these dynam-
514 ics. How the evolution of new sex-determining mechanisms could be influenced
by two-locus meiotic drive and/or by ecological feedbacks under different mating
516 systems remains to be studied.

The hypotheses presented here can be empirically investigated in a similar
518 manner to the idea that transitions between sex-determining systems are favoured
by linkage to sexually antagonistic variation. In the case of sexually antagonis-
520 tic variation, one supporting observation is that genes that appear to experience
sexually-antagonistic selection have been found on recently derived sex chromo-
522 somes (Lindholm and Breden 2002, Tripathi et al. 2009, Ser et al. 2010). However,
it is possible that sexually antagonistic variation accumulated after sex chromo-
524 some transitions because linkage with the sex-determining regions allows sexu-
ally antagonistic selection to maintain polymorphisms under a larger parameter
526 space (Rice 1987, Jordan and Charlesworth 2011). We note that linkage with sex
chromosomes is not, a priori, more permissive to the maintenance of ploidy an-
528 tagonistic variation (Immler et al. 2012). However, as with sexually-antagonistic
variation, a comparison between closely related clades could indicate whether
530 a polymorphism pre-dates a transition in sex-determination or arose afterwards.
Secondly, we have shown that new sex-determination systems can be favoured if
532 either the ancestral sex-determining region or the new sex-determining region are
linked to loci under haploid selection. Therefore, the presence of haploid selected
534 loci around ancestral- or new- sex-determining regions could support their role in
sex chromosome turnover.

536 Taken at face value, our results indicate that transitions in heterogamety (XY
to ZW or vice versa) are more likely to be favoured by selection if there is selection
538 upon both haploid and diploid genotypes rather than diploid selection alone. This
prediction could be examined using a suitable proxy for haploid selection, for ex-
540 ample, Lenormand and Dutheil (2005) use the outcrossing rate in plants as a proxy
for the strength of pollen competition. In animals, one might expect gametic com-

542 petition to be stronger in species where sperm is required to live for a long time
after spermatogenesis because transcripts shared during spermatogenesis may be-
544 come depleted, revealing the haploid phenotype of the sperm (Immler et al. 2014).
Given the caveats mentioned above about the form of meiotic drive modelled, we
546 would also expect that heterogametic transitions in sex determination would be
more common in clades where there is meiotic drive.

548 We have shown that haploid selection can drive transitions between sex de-
termination systems, such that haploid selection should be incorporated into the
550 factors that influence the evolution of sex determination. However, the particular
way in which transitions are affected by haploid selection is not intuitively obvious.
552 Firstly, sex-specific haploid selection affects turnovers between sex determination
systems in a manner that is qualitatively different from diploid sex-specific selec-
554 tion. In particular, closer linkage between a sex-determining locus and a selected
locus is not always favoured during heterogametic transitions when there is hap-
556 loid selection. Secondly, even though haploid selection is a source of zygotic sex
ratio biases, Fisherian sex ratio selection does not have good explanatory power
558 in our models in determining whether various sex-determination systems evolve;
this result is surprising given that sex ratios are ultimately determined via the sex-
560 determination system.

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756 **Figures**

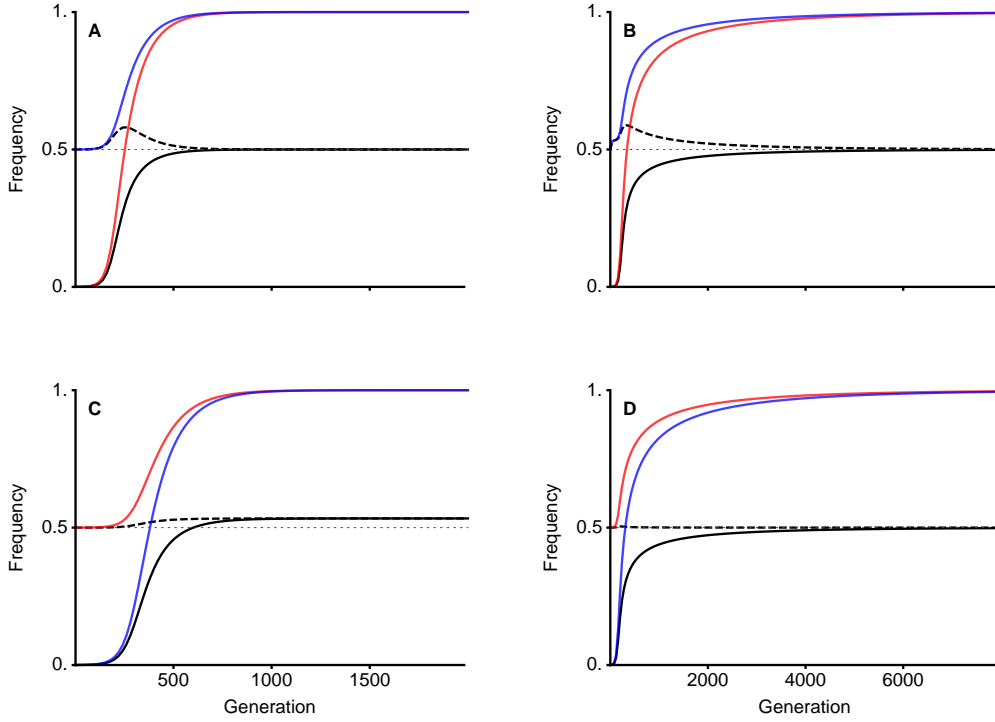


Figure 1: Heterogametic transitions from XY to ZW sex determination (neo-W frequency shown by black lines, panels A and B) or from ZW to XY (neo-Y frequency shown by black lines, panels C and D) occur similarly regardless of sex ratio biases present before (B versus D) or after (C versus A, dashed lines show male frequency). During invasion by a neo-ZW sex determination system (A and B), the ancestral Y fixes in both males and females (blue and red lines). Similarly, the ancestral W allele fixes in males and females (blue and red lines) during a ZW to XY transition. In this plot, there is no gametic competition ($r^{\text{♀}} = r^{\text{♂}} = 0$) and meiotic drive occurs during male meiosis only ($\alpha_{\Delta}^{\text{♀}} = 0$, $\alpha_{\Delta}^{\text{♂}} = -1/5$). Therefore, sex ratio biases can only arise when the **A** locus is linked to an XY sex-determining locus. In panels A and C, the neo-sex-determining locus is more closely linked to the **A** locus than the ancestral sex-determining region ($r = 1/2$, $R = 1/20$) such that a neo-Y can cause biased sex ratios (panel C). In panels B and D, the ancestral sex-determining locus is more closely linked to the **A** locus than the neo-sex-determining locus ($r = 1/20$, $R = 1/2$). Therefore, an ancestral XY sex determination can have a biased zygotic sex ratio that becomes unbiased after an unlinked neo-W invades (B). However, in panel D, a unlinked neo-Y invades an ancestral ZW sex determination system in a similar manner but no biases to the zygotic sex ratio occur. With diploid selection alone, neo-sex-determining loci do not spread if they are less closely linked to the **A** locus than the ancestral sex-determining locus (see equation (2) and Figure 3A). In this plot there are no sex differences in selection and an equilibrium is maintained because selection in diploids opposes meiotic drive, $s^{\text{♀}} = s^{\text{♂}} = 1/5$, $h^{\text{♀}} = h^{\text{♂}} = 7/10$.

Aesthetic adjustments: Could add titles to the columns/rows: neo-W for row 1, neo-Y for row 3, $r = 0.5$, $R = 0.05$ for column 1 and $r = 0.05$, $R = 0.5$ for column 2. Could adjust padding (too much whitespace where there is no axis label). It also seems could increase ratio of font size relative to plot size to make figure more compact. Matt - could you uncomment the line legends in the Mathematica file (function not included in my Mathematica version).

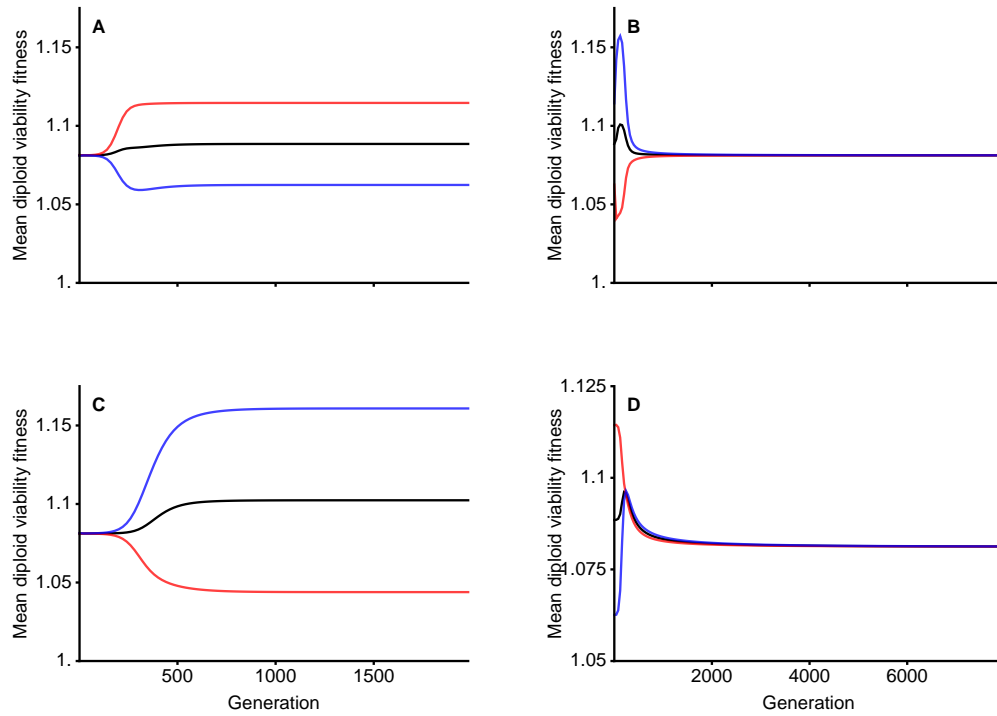


Figure 2: Here, we plot how male mean fitness (blue lines), female mean fitness (red lines), and population mean fitness (male mean fitness plus female mean fitness, black lines) changes during the transitions between sex-determination systems shown in Figure 1. Here we multiply male mean fitness and female mean fitness by two so that we can show it on the same scale as population mean fitness. The mean fitness of females increases during the spread of neo-W alleles (A and B) and the mean fitness of males increases during the spread of neo-Y alleles (C and D). However, when a neo-sex determining system evolves that is less closely linked to a locus under selection (B and D), population mean fitness decreases. **Could add titles to the columns/rows: neo-W for row 1, neo-Y for row 3, $r = 0.5$, $R = 0.05$ for column 1 and $r = 0.05$, $R = 0.5$ for column 2. & possibly adjust padding (too much whitespace?). Matt - could you uncomment the line legends in the Mathematica file (function not included in my Mathematica version).**

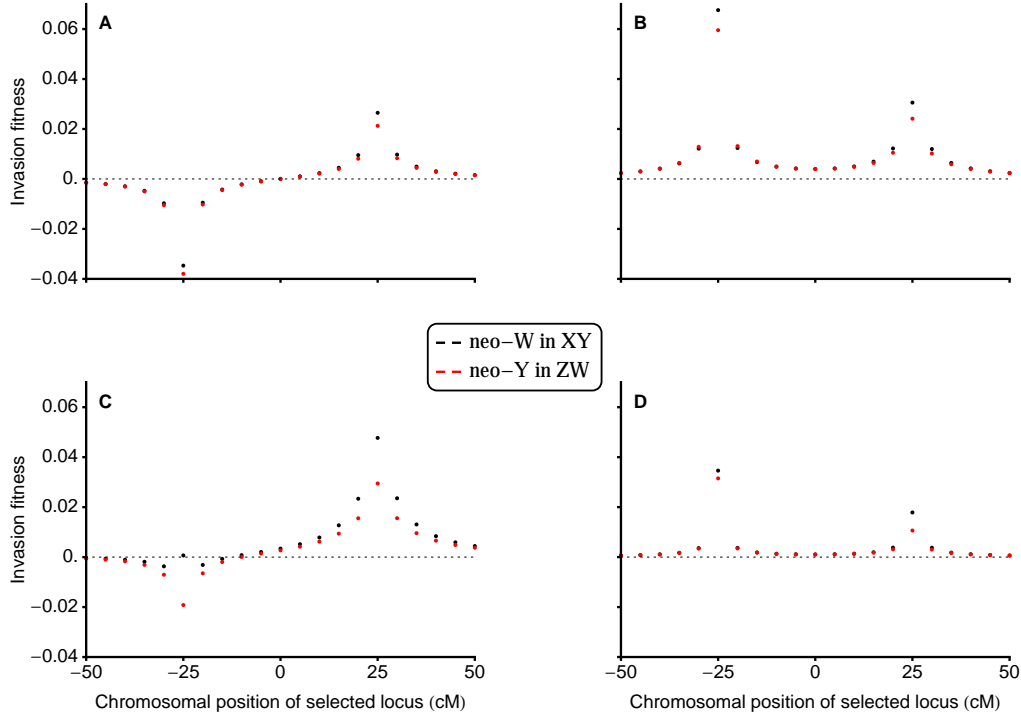


Figure 3: The invasion fitness of a neo-W allele plotted against the relative location of a locus under direct selection, **A**, for various selective regimes. We assume that the ancestral sex-determining locus is located at -0.25, the novel sex-determining locus is located at 0.25 and that there is a polymorphism at the **A** locus maintained by selection. We used Haldane's map function (Equation 3 in Haldane 1919) to convert from map distance (centiMorgans) to the probability of a cross-over event. In **A**, there is no haploid selection ($r^\delta = \alpha_\Delta^\delta = 0$) and selection in diploids is sexually antagonistic (following van Doorn and Kirkpatrick 2010), in which case a neo-W can only invade if it is more closely linked to the selected locus ($s^\varnothing = 1/10$, $h^\varnothing = 7/10$, $s^\delta = -1/10$, $h^\delta = 3/10$). In **B-D** we include haploid selection and assume that selection in diploids is not sexually-antagonistic ($s^\varnothing s^\delta > 0$). A polymorphism can then be maintained by opposing selection between the haploid and diploid phases. In **B**, there is drive in favour of the *a* allele in males ($\alpha_\Delta^\delta = -1/10$), no female meiotic drive or gametic competition, $r^\delta = \alpha_\Delta^\delta = 0$, and equal selection in diploid sexes ($s^\varnothing = s^\delta = 1/10$, $h^\varnothing = h^\delta = 7/10$). In this case, a neo-W can invade even when the selected locus is more closely linked to the ancestral sex determining locus (see Table 3 and Figure 1). In **C** and **D**, there is gametic competition among male gametes only (favouring *a*, $r^\delta = -1/10$) and no meiotic drive or gametic competition in females ($r^\varnothing = \alpha_\Delta^\delta = 0$). In this case, the neo-W does not invade if $s^\varnothing > s^\delta$ (panel **C**: $s^\varnothing = 3/20$, $s^\delta = 1/20$) but does if $s^\varnothing < s^\delta$ (panel **D**: $s^\varnothing = 1/20$, $s^\delta = 3/20$), see Table 3. **I suspect that panel C has a region where no equilibrium is maintained (CHECK! Maybe include different parameters here or remove the part when no equilibrium).** Currently use different parameters for **B** than using in figure 1 (selection/drive twice as strong in turnover figure). This plot would also benefit from titles giving, e.g., “sexually-antagonistic selection, $s^\varnothing s^\delta < 0$ ” for **A**, “male meiotic drive, $s^\varnothing s^\delta > 0$ ” for **B**

Appendix

758 Recursion Equations

In each generation we census the genotype frequencies in male and female gametes/gametophytes (hereafter, gametes) after meiosis (and any meiotic drive) and immediately before gametic competition. At this stage, the frequencies of X-bearing male and female gametes are given by X_i^δ and X_i^φ and the frequencies of Y-bearing gametes are given by Y_i^δ and Y_i^φ where the index i specifies genotypes $MA = 1$, $Ma = 2$, $mA = 3$, and $ma = 4$ ($\sum_{i=1}^4 Y_i^\delta + X_i^\delta = 1$ and $\sum_{i=1}^4 Y_i^\varphi + X_i^\varphi = 1$). Competition then occurs among gametes of the same sex (e.g., among eggs and among sperm separately) according to the **A** locus allele, g ($g \in A, a$, see Table 1), carried by individuals with genotype i . The genotype frequencies after gametic competition are $X_i^{\delta,s} = w_g X_i^\delta / \bar{w}_H^\delta$ and $Y_i^{\delta,s} = w_g Y_i^\delta / \bar{w}_H^\delta$, where $\bar{w}_H^\delta = \sum_{i=1}^4 w_g X_i^\delta + w_g Y_i^\delta$ is the mean fitness of male ($\delta = \delta$) or female ($\delta = \varphi$) gametes. Random mating then occurs between gametes to produce diploid zygotes with genotype ij at the **A** and **M** loci, such that XX zygotes are denoted xx_{ij} , XY zygotes are xy_{ij} , and YY zygotes are yy_{ij} . In XX and YY zygotes, individuals with genotype ij are equivalent to those with genotype ji ; for simplicity, we denote the frequency of genotype ij to the average of these frequencies, $xx_{ij} = (X_i^{\varphi,s} X_j^{\delta,s} + X_j^{\varphi,s} X_i^{\delta,s})/2$ and $yy_{ij} = (Y_i^{\varphi,s} Y_j^{\delta,s} + Y_j^{\varphi,s} Y_i^{\delta,s})/2$.

776 Denoting the **M** locus genotype by b ($b \in MM, Mm, mm$) and the **X** locus genotype by c ($c \in XX, XY, YY$), 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 $xx_{ij}^\delta = (1 - k_{bc})xx_{ij}$, XY male frequencies are $xy_{ij}^\delta = (1 - k_{bc})xy_{ij}$, and YY males frequencies are $yy_{ij}^\delta = (1 - k_{bc})yy_{ij}$. This 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 relationship between the two sex-determining loci. Typically, we assume

786 that the ancestral sex-determining system (**X** locus) is XY ($k_{MMXX} = 1$ and
 $k_{MMXY} = k_{MYY} = 0$) and epistatically recessive to a dominant novel sex-
788 determining locus, **M** ($k_{Mmc} = k_{mmc} = k$).

Selection among diploids then occurs according to the diploid genotype at the
790 **A** locus, h , for an individual of type ij ($h \in AA, Aa, aa$, see Table 1). The
diploid frequencies after selection in sex ϕ are given by $xx_{ij}^{\phi,s} = w_h^{\phi}xx_{ij}/\bar{w}^{\phi}$,
792 $xy_{ij}^{\phi,s} = w_h^{\phi}xy_{ij}/\bar{w}^{\phi}$, and $yy_{ij}^{\phi,s} = w_h^{\phi}yy_{ij}/\bar{w}^{\phi}$, where $\bar{w}^{\phi} = \sum_{i=1}^4 \sum_{j=1}^4 w_h^{\phi}xx_{ij} +$
 $w_h^{\phi}xy_{ij} + w_h^{\phi}yy_{ij}$ is the mean fitness of individuals of sex ϕ .

794 Finally, these diploids undergo meiosis to produce the next generation of ga-
metes. Recombination and sex-specific meiotic drive occur during meiosis. Here,
796 we allow the relative locations of the SDR, **A**, and **M** loci to be generic by using
three parameters to describe the recombination rates between them. R is the re-
798 combination rate between the **A** locus and the **M** locus, χ is the recombination rate
between the **M** locus and the **X** locus, and r is the recombination rate between the
800 **A** locus and the **X** locus. Table S.1 gives substitutions for χ for defined relative
locations of these loci. During meiosis in sex ϕ , meiotic drive occurs such that, in
802 Aa heterozygotes, a fraction α^{ϕ} of gametes produced carry the A allele and $(1 - \alpha^{\phi})$
carry the a allele.

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

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

804 Among gametes from sex ϕ (sperm/pollen when $\phi = \sigma$, eggs/ovules when
 $\phi = \varphi$), the frequencies of haplotypes (before gametic competition) in the next
806 generation are given by

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

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

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

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

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

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

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

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

808 The full system is therefore described by 16 recurrence equations (three loci, each
with two alleles, and two gamete sexes yields 16 combinations). However, some
810 diploid types are not produced under a given sex determination system. For exam-
ple, with the M allele fixed and ancestral XY sex determination, there are no XX
812 males, XY females, or YY females ($xx_{11}^{\phi}, xx_{12}^{\phi}, xx_{22}^{\phi}, xy_{11}^{\phi}, xy_{12}^{\phi}, xy_{22}^{\phi}, yy_{11}^{\phi}, yy_{12}^{\phi}$,
and yy_{22}^{ϕ} are all 0). In this case, the system only involves six recursion equations
814 because there is only one M locus allele and no Y-bearing female gametes. This
six-equation system yields equilibrium (S.3).

816 Resident equilibrium and stability

In the resident population (allele M fixed), we follow the frequency of A in female
818 gametes (eggs) from an XX female, p_X^{ϕ} , and in X-bearing, p_X^{ϕ} , and Y-bearing,
 p_Y^{ϕ} , male gametes (sperm). We also track the total frequency of Y among male
820 gametes, q , which may deviate from 1/2 due to meiotic drive in males. Within this
resident population (when m is absent) we can then describe frequencies among
822 different gamete types, which are given by $X_{MA}^{\phi} = p_X^{\phi}$, $X_{Ma}^{\phi} = (1 - p_X^{\phi})$, $X_{MA}^{\delta} =$
 $(1 - q)p_X^{\phi}$, $X_{Ma}^{\delta} = (1 - q)(1 - p_X^{\phi})$, $Y_{MA}^{\phi} = qp_Y^{\phi}$, and $Y_{Ma}^{\phi} = q(1 - p_Y^{\phi})$. Mean
824 fitnesses in this resident population are given in table S.2.

Various forms of selection can maintain a polymorphism at the A locus, includ-
826 ing sexually antagonistic selection, overdominance and conflicts between diploid
selection and selection upon haploid genotypes (ploiddally antagonistic selection,

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

Table S.2: mean fitnesses in resident (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^σ)	$\bar{p}^\sigma w_A^\sigma + (1 - \bar{p}^\sigma) w_a^\sigma$
females (\bar{w}^\varnothing)	$\frac{\{p_X^\varnothing w_A^\varnothing p_X^\sigma w_A^\sigma w_{AA}^\varnothing + (1 - p_X^\varnothing) w_a^\varnothing p_X^\sigma w_A^\sigma w_{Aa}^\varnothing + p_X^\varnothing w_A^\varnothing (1 - p_X^\sigma) w_a^\sigma w_{Aa}^\varnothing + (1 - p_X^\varnothing) w_a^\varnothing (1 - p_X^\sigma) w_a^\sigma w_{aa}^\varnothing\}}{\{\bar{w}_H^\varnothing \bar{w}_H^\sigma\}}$
males (\bar{w}^σ)	$\frac{\{p_X^\varnothing w_A^\varnothing p_Y^\sigma w_A^\sigma w_{AA}^\sigma + (1 - p_X^\varnothing) w_a^\varnothing p_Y^\sigma w_A^\sigma w_{Aa}^\sigma + p_X^\varnothing w_A^\varnothing (1 - p_Y^\sigma) w_a^\sigma w_{Aa}^\sigma + (1 - p_X^\varnothing) w_a^\varnothing (1 - p_Y^\sigma) w_a^\sigma w_{aa}^\sigma\}}{\{\bar{w}_H^\varnothing \bar{w}_H^\sigma\}}$

Recombination weak relative to selection

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

$$\begin{aligned}
(A) \quad \hat{p}_Y^\delta = 0, \quad \hat{q} &= \frac{1}{2} - \frac{(\alpha^\delta - 1/2)w_{Aa}^\delta \Phi}{w_{Aa}^\delta \Phi + w_{aa}^\delta \Psi}, \\
\hat{p}_X^\varnothing &= \frac{w_a^\varnothing \Phi}{w_a^\varnothing \Phi + w_A^\varnothing \Psi}, \quad \hat{p}_X^\delta = \frac{2\alpha^\delta w_{Aa}^\delta \Phi}{2\alpha^\delta w_{Aa}^\delta \Phi + w_{AA}^\delta \Psi} \\
(A') \quad \hat{p}_Y^\delta = 0, \quad \hat{q} &= \frac{1}{2} + \frac{(\alpha^\delta - 1/2)w_{Aa}^\delta \Phi'}{w_{Aa}^\delta \Phi' + w_{AA}^\delta \Psi'}, \\
\hat{p}_X^\varnothing &= 1 - \frac{w_A^\varnothing \Phi'}{w_A^\varnothing \Phi' + w_a^\varnothing \Psi'}, \quad \hat{p}_X^\delta = 1 - \frac{2(1 - \alpha^\delta)w_{Aa}^\delta \Phi'}{2(1 - \alpha^\delta)w_{Aa}^\delta \Phi' + w_{aa}^\delta \Psi'} \\
(B) \quad \hat{p}_Y^\delta &= 0, \quad \hat{p}_X^\varnothing = 1, \quad \hat{p}_X^\delta = 1, \quad \hat{q} = (1 - \alpha^\delta) \\
(B') \quad \hat{p}_Y^\delta &= 1, \quad \hat{p}_X^\varnothing = 0, \quad \hat{p}_X^\delta = 0, \quad \hat{q} = \alpha^\delta
\end{aligned}$$

$$\begin{aligned}
\Phi &= \alpha^\varnothing w_A^\varnothing w_{Aa}^\varnothing (w_a^\delta w_{aa}^\delta + 2\alpha^\delta w_A^\delta w_{Aa}^\delta) - w_a^\delta w_a^\varnothing w_{aa}^\delta w_{aa}^\varnothing \\
\Psi &= (1 - \alpha^\varnothing) w_a^\varnothing w_{Aa}^\varnothing (w_a^\delta w_{aa}^\delta + 2\alpha^\delta w_A^\delta w_{Aa}^\delta) - 2\alpha^\delta w_A^\delta w_A^\varnothing w_{Aa}^\delta w_{AA}^\varnothing \\
\Phi' &= (1 - \alpha^\varnothing) w_a^\varnothing w_{Aa}^\varnothing (w_A^\delta w_{AA}^\delta + 2(1 - \alpha^\delta) w_a^\delta w_{Aa}^\delta) - w_A^\delta w_A^\varnothing w_{AA}^\delta w_{AA}^\varnothing \\
\Psi' &= \alpha^\varnothing w_A^\varnothing w_{Aa}^\varnothing (w_A^\delta w_{AA}^\delta + 2(1 - \alpha^\delta) w_a^\delta w_{Aa}^\delta) - 2(1 - \alpha^\delta) w_a^\delta w_a^\varnothing w_{Aa}^\delta w_{aa}^\varnothing
\end{aligned}$$

836 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
838 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
840 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
842 (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
844 a allele. If there is no haploid selection ($\alpha^\delta = 1/2$, $w_g^\delta = 1$), these equilibria are
equivalent to those found by [Otto \(2014\)](#) and [Lloyd \(197?, see Otto for reference\)](#).

846 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

848 properties are unaffected by small amounts of recombination between the SDR and
 A locus, although equilibrium frequencies may be slightly altered. For the a allele
 850 to be stably fixed on the Y requires that $\bar{w}_{Ya}^\delta > \bar{w}_{YA}^\delta$ where $\bar{w}_{Ya}^\delta = w_a^\delta(2p_X^\varnothing(1 -$
 $\alpha^\delta)w_A^\varnothing w_{Aa}^\delta + (1 - p_X^\varnothing)w_a^\varnothing w_{aa}^\delta)$ and $\bar{w}_{YA}^\delta = w_A^\delta(p_X^\varnothing w_A^\varnothing w_{AA}^\delta + 2(1 - p_X^\varnothing)\alpha^\delta w_a^\varnothing w_{aa}^\delta)$.
 852 That is, Ya haplotypes must have higher fitness than YA haplotypes. Substituting
 \hat{p}_{Xf} from above, fixation of the A allele on the Y requires that $\gamma_i > 0$ where $\gamma_{(A)} =$
 854 $w_a^\delta(2(1 - \alpha^\delta)w_{Aa}^\delta \Phi + w_{aa}^\delta \Psi) - w_A^\delta(2\alpha^\delta w_{Aa}^\delta \Phi + w_{aa}^\delta \Psi)$ for equilibrium (A) and
 $\gamma_{(B)} = 2(1 - \alpha^\delta)w_a^\delta w_{Aa}^\delta - w_A^\delta w_{AA}^\delta$ for equilibrium (B). Stability of a polymorphism
 856 on the X chromosome (equilibrium A) further requires that $\Phi > 0$ and $\Psi > 0$.
 Fixation of the a allele on the X (equilibrium B) is mutually exclusive with (A)
 858 and requires that $\Psi < 0$ and $w_A^\varnothing w_{AA}^\varnothing > (1 - \alpha^\varnothing)w_a^\varnothing w_{Aa}^\varnothing$.

Selection weak relative to recombination

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

$$0 < -((1 - h^\varnothing)s^\varnothing + (1 - h^\delta)s^\delta + t^\varnothing + t^\delta + \alpha_\Delta^\varnothing + \alpha_\Delta^\delta) \quad (\text{S.2})$$

and $0 < (h^\varnothing s^\varnothing + h^\delta s^\delta + t^\varnothing + t^\delta + \alpha_\Delta^\varnothing + \alpha_\Delta^\delta).$

which indicates that a polymorphism is maintained under various selective regimes.
 864 In particular special cases, e.g., no sex-differences in selection or meiotic drive
 $(s^\delta = s^\varnothing, h^\delta = h^\varnothing, \text{ and } \alpha^\delta = \alpha^\varnothing = 1/2)$, the equilibrium allele frequency and
 866 stability can be calculated analytically without assuming weak selection. How-
 ever, here, we focus on weak selection in order to make fewer assumptions about
 868 fitnesses.

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

$$\bar{p} = \frac{h^{\varphi}s^{\varphi} + h^{\delta}s^{\delta} + t^{\varphi} + t^{\delta} + \alpha_{\Delta}^{\varphi} + \alpha_{\Delta}^{\delta}}{(2h^{\varphi} - 1)s^{\varphi} + (2h^{\delta} - 1)s^{\delta}} + O(\epsilon). \quad (\text{S.3})$$

872 Differences in frequency between gamete types are of order ϵ to leading order and
given by

$$\begin{aligned} \hat{p}_X^{\delta} - \hat{p}_X^{\varphi} &= V_A (D^{\delta} - D^{\varphi} + \alpha_{\Delta}^{\delta} - \alpha_{\Delta}^{\varphi}) + O(\epsilon^2) \\ \hat{p}_Y^{\delta} - \hat{p}_X^{\varphi} &= V_A (D^{\delta} - D^{\varphi} + \alpha_{\Delta}^{\delta} - \alpha_{\Delta}^{\varphi} + (1 - 2r)(t^{\delta} - t^{\varphi})) / 2r + O(\epsilon^2) \\ \hat{p}_Y^{\delta} - \hat{p}_X^{\delta} &= V_A (D^{\delta} - D^{\varphi} + \alpha_{\Delta}^{\delta} - \alpha_{\Delta}^{\varphi} + t^{\delta} - t^{\varphi})(1 - 2r) / 2r + O(\epsilon^2) \end{aligned} \quad (\text{S.4})$$

874 where $V_A = \bar{p}(1 - \bar{p})$ is the variance in the frequency of A and $D^{\varphi} = (\bar{p}s^{\varphi} + (1 - \bar{p})h^{\varphi}s^{\varphi}) - (\bar{p}h^{\delta}s^{\delta} + (1 - \bar{p}))$ corresponds to the difference in fitness between A and
876 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
878 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
880 or drive ($\alpha_{\Delta}^{\varphi} = t^{\varphi} = 0$), these results reduce to those of van Doorn and Kirkpatrick
882 (2007).

Invasion conditions

884 Here, we determine whether a rare neo- Y or neo- W allele spreads when rare, which occurs when $\lambda > 1$. If the average change in frequency of the two haplotypes that
886 carry the m allele (Am and am) is positive, invasion will always occur (i.e., if $\{(\lambda_{mA} - 1) + (\lambda_{ma} - 1)\} / 2 > 0$ then $\lambda > 1$, see table 2 for λ_{mi}). If neither haplo-
888 type increases in frequency ($\lambda_{mA}, \lambda_{ma} < 1$), the m allele will not invade. Otherwise, the new sex-determining allele increases in frequency on one A background and

890 declines on the other, and invasion requires

$$R \left[\frac{p_X^\varphi w_A^\varphi w_a^\delta (1 - \alpha^\delta)}{\bar{w}_H^\varphi \bar{w}_H^\delta (\lambda_{mA} - 1)} + \frac{(1 - p_X^\varphi) w_a^\varphi w_A^\delta \alpha^\delta}{\bar{w}_H^\varphi \bar{w}_H^\delta (\lambda_{ma} - 1)} \right] \frac{w_{Aa}^\delta}{q \bar{w}^\delta} < 1, \quad (\text{S.5})$$

892 for the neo- Y , and

$$R \left[\frac{\bar{p}^\delta w_A^\delta w_a^\varphi (1 - \alpha^\varphi)}{\bar{w}_H^\delta \bar{w}_H^\varphi (\lambda_{mA} - 1)} + \frac{(1 - \bar{p}^\delta) w_a^\delta w_A^\varphi \alpha^\varphi}{\bar{w}_H^\delta \bar{w}_H^\varphi (\lambda_{ma} - 1)} \right] \frac{w_{Aa}^\varphi}{(1 - q) \bar{w}^\varphi} < 1, \quad (\text{S.6})$$

for the neo- W .