

Gametic Selection, 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: mfcott@biodiversity.ubc.ca, mmosmond@zoology.ubc.ca

Contributions:

Abstract

Sex determination systems are remarkably dynamic; many studied taxa display transitions of sex-determining genes between chromosomes or the evolution of new sex-determining systems. Here, we utilize population genetic models to study the spread of novel sex-determining systems in the presence of haploid gametic selection, e.g., pollen or sperm competition. Haploid selected loci experience a form of sex-specific selection (because gametic competition occurs predominantly among haploids produced by males) and can cause sex ratios at birth to become biased (because sex ratios are determined by the fertilization success of X- versus Y-bearing pollen/sperm). Notably, we find that the spread of new genetic sex determination systems is not affected by sex ratio biases that are caused by gametic selection because sex ratios become biased after parental provisioning has occurred (even if pollen/sperm competition occurs within the mother). In addition, we find that linkage of an ancestral sex chromosome to a locus under haploid selection can favour transitions between male and female heterogamety (e.g., XY to ZW), which is not the case for any forms of diploid sex specific selection (e.g., sexually antagonistic selection). During these transitions, new sex-determining alleles spread despite breaking up favourable associations that build up between ancestral sex-determining loci and selected loci, reducing population mean fitness. Furthermore, a period of selection among haploids can favour the stable maintenance of polymorphic sex determination systems. Thus, our models offer several new insights to be explored as information about sex determination in non-model taxa accumulates.

Introduction

28 Animals and angiosperms exhibit extremely diverse sex determination sys-
tems (reviewed in Bull 1983, Charlesworth and Mank 2010, Beukeboom and
30 Perrin 2014, Bachtrog et al. 2014). Among species with genetic sex deter-
mination of diploid sexes, some taxa have heterogametic males (XY) and
32 homogametic females (XX), including mammals and most dioecious plants
(Ming et al. 2011); whereas other taxa have homogametic males (ZZ) and
34 heterogametic females (ZW), including Lepidoptera and birds. Within sev-
eral taxa, the chromosome that harbours the master sex-determining region
36 changes. For example, transitions of the master sex-determining gene be-
tween chromosomes or the evolution of new master sex-determining genes
38 have occurred in Salmonids (Li et al. 2011, Yano et al. 2012), Diptera (Vicoso
and Bachtrog 2015), and *Oryzias* (Myosho et al. 2012). In addition, many
40 gonochoric/dioecious clades with genetic sex determination exhibit transi-
tions between male (XY) and female (ZW) heterogamety, including lizards
42 (Ezaz et al. 2009), eight of 26 teleost fish families (Mank et al. 2006), true fruit
flies (Tephritids, Vicoso and Bachtrog 2015), amphibians (Hillis and Green
44 1990), the angiosperm genus *Silene* (Slancarova et al. 2013), Coleoptera and
Hemiptera (Beukeboom and Perrin 2014, plate 2). Indeed, in some cases,
46 both 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
48 *Rana rugosa* (Ogata et al. 2007). In addition, multiple transitions have oc-
curred between genetic and environmental sex determination systems, e.g.,
50 in reptiles and fishes (Conover and Heins 1987, Mank et al. 2006, Pokorná
and Kratochvíl 2009, Ezaz et al. 2009, Pen et al. 2010, Holleley et al. 2015).

52 Predominant theories in which new sex determination systems are favoured
by selection involve fitness differences between sexes (e.g., sexually antago-
54 nistic selection) or sex ratio selection. van Doorn and Kirkpatrick (2007;
2010) show that new sex determination loci can be favoured if they arise
56 in close linkage with a locus that experiences sexual antagonism. For ex-

ample, linkage allows favourable associations to build up between a male-
58 beneficial allele and a neo-Y chromosome. Such associations can favour a
new master sex-determining gene on a new chromosome (van Doorn and
60 Kirkpatrick 2007) and can also favour a transition between male and fe-
male heterogamety (e.g., a ZW to XY transition, van Doorn and Kirkpatrick
62 2010). However, any sexually-antagonistic loci that are linked to the ances-
tral sex-determination locus will develop similar, favourable associations and
64 select against the spread of a new sex-determination system.

It has been suggested that sex ratio selection could be a particularly im-
66 portant force driving transitions between sex-determining systems (Beuke-
boom and Perrin 2014, Chapter 7). For example, flexible sex determination
68 systems may be favoured in order to exploit local environmental conditions
that are optimal for males or females, which creates locally biased sex ra-
70 tios (Charnov and Bull 1977, Werren and Taylor 1984, Pen et al. 2010).
In addition, feminizing mutations may invade when female biased sex ra-
72 tios are favoured due to selection among demes (Wilson and Colwell 1981,
Vuilleumier et al. 2007). In other situations, sex ratio selection may favour
74 transitions in order to restore equal sex ratios. For example, Kozielska et al.
(2010) consider systems in which the ancestral sex chromosomes experience
76 meiotic drive (e.g., where driving X or Y chromosomes are inherited dis-
proportionately often), which causes sex ratios to become biased (Hamilton
78 1967). They find that new, unlinked sex-determining loci (masculinizing or
feminizing mutations, i.e., neo-Y or neo-W loci) can then spread, restoring
80 an even sex ratio.

Here, we use mathematical models to find the conditions under which new
82 sex determination systems are favoured by selection when there is a period
of selection among haploid gametes/gametophytes. Selection among hap-
84 loid genotypes is thought to occur primarily among pollen/sperm, which can
compete whenever there are more pollen/sperm than required for fertiliza-
86 tion (Mulcahy et al. 1996, Joseph and Kirkpatrick 2004). Haploid selection

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 cause the frequency of resistant alleles to increase (e.g., Hormaza and Herrero 1996, Ravikumar et al. 2003, Hedhly et al. 2004, Clarke et al. 2004). A smaller (but non-negligible) proportion of genes are thought to be expressed and selected in animal sperm, although precise estimates are uncertain (Zheng et al. 2001, Joseph and Kirkpatrick 2004, Vibranovski et al. 2010). **add something about meiotic drive here?**

There are various ways in which a period of haploid selection could influence transitions between sex determination systems. Firstly, if we assume that haploid selection at any particular locus predominantly occurs in one sex (e.g., pollen/sperm competition), then such loci experience a form of sex-specific selection. In this respect, we might expect that haploid selection might affect transitions between sex determination systems in a similar manner to sex-specific diploid selection (as explored by van Doorn and Kirkpatrick 2007; 2010). That is, new masculinizing mutations (neo-Y chromosomes) could be favoured via linkage associations with alleles that are beneficial in pollen/sperm. However, sex ratios can also become biased if there is linkage between the sex-determining region and a locus that harbours genetic variation in haploid fitness. For example, differences in fitness between X- and Y-bearing pollen tubes can cause the sex ratio among seeds to become biased when there is pollen competition (Lloyd 1974, Conn and Blum 1981, Stehlik and Barrett 2005; 2006, Field et al. 2012; 2013). It is not immediately clear how the spread of new sex determination systems would be influenced by the combination of sex ratio biases and favourable associations between haploid selected loci and sex-determining regions.

Surprisingly, our models show that haploid selection influences the evolution of new sex determination systems in a way that is distinct from both

diploid sex-specific selection and sex ratio selection. We find that new genetic
118 sex determination systems are not affected by any sex ratio biases caused by
associations between sex-determining regions and haploid selected loci. In
120 addition, we find that associations that build up between an ancestral sex-
determining locus and a haploid-selected locus can favour transitions between
122 male and female heterogamety (e.g., a neo-W allele arising at a previously
autosomal locus spreads in an ancestrally XY system), despite the fact that
124 these ancestral associations were built up by selection. This does not occur
in models that do not include haploid selection.

126 NOTE RE: DRIVE. I expect drive (that occurs specifically in one sex,
e.g., during spermatogenesis) to behave almost exactly like haploid selection.
128 That is, I think that a XY-linked driver that is maintained by selection (e.g.,
because it causes sterility when homozygous, which is common in known
130 drive systems) will only favour invasion of a more tightly linked neo-Y (wors-
ening sex ratio biases) and could favour invasion of a neo-W. This may run
132 counter to generic expectations from new sex chromosome systems evolving
to balance the sex ratio. So, do you think it would significantly enhance the
134 paper to model drive explicitly or just discuss it as being similar???

FOR RESULTS?

136 FROM PREVIOUS PAPER: The maintenance of polymorphism at loci
that experience sex specific selection in both haploid and diploid phases was
138 considered by Immler et al. Immler et al. (2012), demonstrating that poly-
morphisms can be maintained by sexually antagonistic selection or overdom-
140 inance as well as by conflicting selection pressures in haploids and diploids
(haploid-diploid conflict or ploidy antagonistic selection) or a combination
142 of these selective regimes.

Model

144 We consider a three locus model. Locus **X** is the ancestral sex-determining
 region, with alleles X and Y (or Z and W). Locus **A** is a region under
 146 selection, with alleles A and a . And locus **M** is a novel sex-determining
 region, with alleles M and m . With genotype MM at locus **M**, the sex of a
 148 zygote is determined by the genotype at locus **X** (XX become females and
 XY become males, or ZW become females and ZZ become males). With at
 150 least one m allele at locus **M**, a zygote develops as a female with probability
 k and as a male with probability $1 - k$. With $k = 0$, the novel sex determiner
 152 is a masculinizer (i.e., a neo-Y) and with $k = 1$ the novel sex determiner is a
 feminizer (i.e., a neo-W). With intermediate k , locus **M** is interpreted as an
 154 offspring controlled environmental sex-determining region. Finally, we also
 analyze a model of maternally-controlled environmental sex-determination
 156 (ESD), where mothers with at least one m allele produce daughters with
 probability k .

158 The life-cycle begins with competition between haploid gametes/gametophytes
 (hereafter gametes) from each sex, where selection depends on the sex of the
 160 diploid they came from and their allele at the **A** locus. Gametes from males
 then pair randomly with gametes from females. The resulting zygotes de-
 162 velop as males or females, depending on their genotypes at the **X** and **M**
 loci (and the **M** genotype of their mother in the case of maternal control).
 164 Diploids compete with others of the same sex, where selection depends on
 the sex of the individual and its genotype at the **A** locus. This is followed by
 166 meiosis with recombination and sex-specific meiotic drive. Recombination
 occurs between loci **X** and **A** with probability r , between loci **A** and **M** with
 168 probability R , and between loci **X** and **M** with probability χ . Any order of
 the loci can be modelled with appropriate choices of r , R , and χ . We model
 170 meiotic drive at the viability locus only; individuals of sex i who are het-
 erozygous at the **A** locus produce haploids bearing allele A with probability
 172 α^i . We track the frequency of haploid genotypes produced by each sex from

one generation to the next (recursion equations in Sup. Mat.).

174 With allele M fixed at the **M** locus, sex is determined by locus **X** and an
equilibrium is reached at locus **A**. We then examine the ability of a rare, novel
176 sex-determiner, m , to increase in frequency from this equilibrium. Numerical
simulations are used to examine when an invading mutation goes to fixation.

178 Results

Resident equilibrium and stability

180 With allele M fixed, we follow the dynamics of the frequency of A in gametes
from the homogametic sex (e.g., in eggs from an XX female, p_X^f), the fre-
182 quencies of A in the two types of gametes from the heterogametic sex (e.g., in
sperm from an XY male that are X-bearing, p_X^m , and Y-bearing, p_Y^m), and the
184 frequencies of the sex-determining factors in gametes from the heterogametic
sex (e.g., the frequency of Y in sperm, q). Assuming selection and meiotic
186 drive are weak relative to recombination, the differences in the frequencies
of A in each type of gamete are small, as is the bias in the sex-determining
188 factor from the heterogametic sex, and we can solve for the mean frequency
of A across all types (p_A), the difference in the frequencies of A between two
190 of the three types, and the bias in the frequency of the sex-determining fac-
tor, to first order in selection. Linear stability analysis can then be used to
192 determine the stability of this equilibrium. Without haploid selection or mei-
otic drive our results reduce to those of van Doorn and Kirkpatrick (2007).
194 However, with haploid selection or meiotic drive a stable polymorphism at
locus **A** no longer requires sexually antagonistic selection.

196 Sex chromosome turnover

The spread of a rare mutant m at the **M** locus in such a population is deter-
198 mined by the leading eigenvalue, λ , of the system described by the equations

for the next generation frequency of haploid genotypes with the mutation.
 200 Below we present the analytic results for invasion into an XY system assum-
 ing no competition among gametes from females, no meiotic drive in females,
 202 and linear arrangement **XAM**. Equivalent results for invasions into a ZW
 system are derived by consistently switching the roles of males and females.

204 **Neo- Y and neo- W**

A rare, dominant neo- Y ($k = 0$) or neo- W ($k = 1$) is always expected to
 206 invade an ancestral XY system when the average growth rate of the mutant
 haplotypes (Am and am) is positive, $(g_A + g_a)/2 > 0$. When the growth
 208 rates of the mutant haplotypes without recombination ($R = 0$) are negative,
 $g_i^* < 0 \forall i \in \{A, a\}$, where $g_i < g_i^*$, the new sex-determining allele does not
 210 invade.

Otherwise, the new sex-determining allele increases in frequency on one
 212 **A** background and declines on the other, and invasion requires

$$R \left[\frac{p_X^f w_a^m (1 - \alpha^m)}{g_A^*} + \frac{(1 - p_X^f) w_A^m \alpha^m}{g_a^*} \right] w_{Aa}^m < \nu^m, \quad (1)$$

for the neo- Y , and

$$R \left[\frac{(1 - \bar{p}^m) w_a^m}{g_A^*} + \frac{\bar{p}^m w_A^m}{g_a^*} \right] w_{Aa}^f < \nu^f \quad (2)$$

214 for the neo- W . Here $\bar{p}^m = (1 - q)p_X^m + qp_Y^m$ is the average frequency of
 A in gametes produced by males, w_j^i is the relative viability fitness of sex
 216 $i \in \{m, f\}$ depending on their haploid or diploid genotype at the **A** locus
 (with $j \in \{A, a, AA, Aa, aa\}$) and $\nu^i = p_X^f p_{\mathbf{X}^i}^m w_a^m w_{AA}^i + p_X^f (1 - p_{\mathbf{X}^i}^m) w_a^m w_{Aa}^i +$
 218 $(1 - p_X^f) p_{\mathbf{X}^i}^m w_a^m w_{Aa}^i + (1 - p_X^f) (1 - p_{\mathbf{X}^i}^m) w_a^m w_{aa}^i$, with $\mathbf{X}^m = Y$ and $\mathbf{X}^f = X$,
 is the mean relative fitness of resident individuals of sex i . Although male
 220 meiotic drive does not explicitly appear in Equation 2, it does affect the
 average frequency of A in gametes from males, \bar{p}^m , and thus can play a role

222 in neo-W invasion.

Equations (1) and (2) show that the new sex-determining allele is ex-
 224 pected to invade for any recombination rate, R , when the net flow of double
 recombinants is from the less fit to the more fit **A** background (making the
 226 terms inside the square brackets in Equations 1 and 2 negative). When the
 net flow of double recombinants is from the more fit to the less fit haplotype,
 228 the new sex-determining allele can still invade when the rate of recombination
 between it and the selected locus, R , is small enough.

Assuming weak selection and meiotic drive, we can explicitly solve for the
 invasion fitness of the new sex-determining allele, m , into the ancestral XY
 232 system, giving

$$\lambda_{m,XY} \approx 1 + V_A S_A C_m, \quad (3)$$

where $V_A = p_A(1 - p_A)$ is the variance at the **A** locus. We will consider
 234 haploid selection and meiotic drive separately. With haploid selection and
 no drive we have $S_A = s^f(t^m)^2 / [rR(s^f + s^m)^2]$, where s^f and s^m are the
 236 respective selection coefficients for A in diploid females and males, t^m is the
 selection coefficient for A in gametes from males, and we've assumed equal
 238 dominance coefficients in the two sexes ($h^f = h^m$). With drive and no haploid
 selection one replaces t^m with $\alpha^m - 1/2$.

For the neo- Y we have $C_Y = (r - R)s^f$ with haploid selection and $C_Y =$
 $4(r - R)s^f$ with drive. The neo- Y can therefore invade whenever it is in
 242 tighter linkage with the selected locus than the ancestral sex-determining
 locus, $r > R$, provided locus **A** is polymorphic ($V_A > 0$), there is diploid
 244 selection in females ($s^f \neq 0$), and there is either haploid selection between
 gametes from males ($t^m \neq 0$) or meiotic drive in males ($\alpha^m \neq 1/2$). This is
 246 similar to the conclusion reached concerning sexual-antagonistic selection in
 van Doorn and Kirkpatrick (2007) and reduces to their weak-linkage results
 248 exactly when we do not assume equal dominance coefficients in the two sexes,
 there is no haploid selection or meiotic drive, and there is free recombination
 250 between locus **A** and one of the sex-determining regions.

For the neo- W we have $C_W = [(2r(1 - R) - R)s^f + (1 - 2r)Rs^m]/2$ with
haploid selection and $C_W = 4[r(1 - 2R)s^f + (1 - 2r)Rs^m]/2$ with drive.
In this case, even when the novel sex-determining locus is in looser linkage
with the selected locus than the ancestral sex-determining locus is, $r < R$,
a neo- W can invade (Figures 1 – 3). This does not occur in models with
only sexually-antagonistic selection (van Doorn and Kirkpatrick 2010). For
example, with $R = 1/2$ the neo- W invades if there is any linkage between
the ancestral sex-determining and selected loci ($r < 1/2$), there is selection
among gametes in males ($t^m \neq 0$), and there is selection for or against A in
both males and females ($s^m s^f > 0$) that is stronger in males than in females
($|s^m| > |s^f|$). With meiotic drive and $R = 1/2$, all that is required for neo- W
invasion is $r < 1/2$ and $s^m s^f > 0$. Our results reduce to the weak-linkage
results of (van Doorn and Kirkpatrick 2010, Equation 3) when we do not
assume equal dominance coefficients in the two sexes and there is no haploid
selection or meiotic drive.

Offspring-controlled neo-ESD

The growth rate of a rare, dominant offspring-controlled neo-ESD region that
produces males or females with equal probability ($k = 1/2$) is

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

Thus with $k = 1/2$ the neo-ESD gets half of the advantages of a neo- W and
half that of a 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). Re-
combination between the selected locus and the novel sex-determining locus,
 R , doesn't enter into the $k = 1/2$ results because sex is essentially random-
ized each generation, preventing associations from building up between allele
 A and sex.

276 Maternally-controlled neo-ESD

One might think that when the sex of zygotes is under the control of mothers,
278 there would be strong selection to balance the sex ratio among zygotes. However, we find that, as with offspring control, under weak selection the invasion
280 fitness of a sex-determiner that is maternally controlled can be written

$$\lambda_{k,XY} \approx 1 + V_A S_A C_k, \quad (5)$$

where C_k is a term that depends on k . Of particular interest is $k = 1/2$ (i.e.,
282 when the mother perfectly balances the sex ratio of her offspring). When both recombination rates are small we have $C_{1/2} \approx R(s^m - s^f)/8 = \lim_{r \rightarrow 0} C_1/4$.
284 This implies that, at least under tight linkage, the invasion of maternally-controlled ESD is independent of R (because $S_A \propto R^{-1}$) and can invade
286 whenever a neo- W can (which can invade even when it biases the sex ratio further; Figures 1 – 2).

288 Discussion

Brief results summary.

290 DRAFT (improve): In Úbeda et al. (2015), the new sex determining locus spreads because it arises in linkage with a locus that experiences drive. They
292 assume that drive occurs predominantly in one sex, e.g., during spermatogenesis or a 'killer' sperm. A driving allele is maintained at an intermediate
294 frequency by selection, e.g., because it causes male sterility when homozygous (because all male sperm are killed). Y chromosomes that arise in linkage with
296 the driving allele spread because they allow drive to occur more often, thus genetic sex determination with a sex ratio bias evolves. Thus Úbeda et al.
298 (2015) also find that genetic sex determiners can invade, despite causing sex ratios to become biased. Finally, they show that autosomal 'restorers' that
300 negate the effects of meiotic drive can invade and restore an equal sex ratio.

OTHER RESTORERS. Indeed, alleles that negate the effect of sex-linked
302 meiotic drivers and restore equal sex ratios have been identified (Stalker
1961, Smith 1975). A similar process occurs with cytoplasmic male sterility
304 alleles (that cause biased sex ratios) and nuclear ‘restorer’ genotypes (Frank
1989). When sex ratio bias occurs due to haploid selection, a natural class
306 of sex ratio ‘restorers’ exist because haploid selection often occurs in a con-
text that is determined by the diploid parents. For example, the intensity of
308 pollen competition can be manipulated by altering style length (Travers and
Shea 2001, Lankinen and Skogsmyr 2001, Ruane 2009), delaying stigma re-
310 ceptivity (Galen et al. 1986, Lankinen and Madjidian 2011) and/or delaying
pollen tube growth in the pistil (Herrero 2003). Where the X and Y have
312 fitness differences, Hough et al. (2013) and Otto et al. (2015) demonstrated
that mothers should generally evolve to balance sex ratios by reducing the
314 intensity of haploid competition.

Despite the fact that sex ratio restorers can evolve, we find that sex
316 chromosome turnover occurs regardless of sex ratio bias (Figure 3).

FROM THESIS: However, reducing competition among haploids also re-
318 duces the potential for harmful deleterious mutations to be purged. When
deleterious mutations are included, the optimal intensity of haploid selection
320 can reflect a balance between maximizing offspring fitness and equalizing sex
ratios.

322 As part of a collaborative project (Otto et al. 2015), I considered the
evolution of the haploid ‘selective arena’ in cases where the X chromosome
324 harbours a polymorphism that affects haploid fitness. Mothers again primar-
ily evolve to restore equal sex ratios. However, modifying haploid selection
326 also affects the X-linked genotypes that are inherited by offspring. Specif-
ically, increasing the intensity of haploid selection increases the proportion
328 of daughters (all progeny of X-bearing sperm/pollen are female) that inherit
the allele with high haploid fitness. If this allele has high fitness in daugh-
330 ters, mothers can be selected to increase the intensity of haploid selection;

otherwise, decreased selection among haploids is favoured. Thus, because
332 altering haploid selection intensity affects the alleles that are inherited by
daughters, mothers can favour slightly biased sex ratios. In addition, I found
334 that stronger sex ratio biases can be favoured by paternal manipulations of
the haploid ‘selective arena’ because fathers are strongly selected to maximize
336 their own siring success (above selection to equalize the sex ratio).

Discuss patterns that might be looked for:

338 In broadcast spawning animal species (e.g., corals, many fish) and species
where sperm typically requires greater longevity, haploid selection may be
340 stronger because transcripts shared during spermatogenesis may become de-
pleted (Immler et al. 2014). also, mating systems (e.g., fewer alleles are
342 available during haploid competition in monogamous species), selfing rates,
and estimates of pollen limitation could be used as indicators of the intensity
344 of haploid selection

We have results where polygenic sex determination is sometimes stable, may
346 be worth mentioning:

“Polygenic sex determination has been reported in many plants (e.g.
348 Shannon & Holsinger 2007), fishes (Vandeputte et al. 2007; Ser et al. 2010;
Liew et al. 2012), crustaceans (e.g. Battaglia 1958; Battaglia & Malesani
1959; Voordouw & Anholt 2002), bivalves (Haley 1977; Saavedra et al. 1997),
350 gastropods (Yusa 2007a,b), and polychaetes (Bacci 1965, 1978; Premoli et al.
1996).” From Vuilleumier et al. 2007: “Polymorphism for sex-determining
352 genes within or among populations has been reported in many species in-
cluding houseflies, midges, woodlice, platyfish, cichlid fish, and frogs (Gor-
don, 1944; Kallman, 1970; Thompson, 1971; Macdonald, 1978; Bull, 1983;
356 Rigaud et al., 1997; Caubet et al., 2000; Lande et al., 2001; Ogata et al.,
2003; Lee et al., 2004; Mank et al., 2006).”

358 (Check with Jim Bull that it’s ok before including this speculation:) Fi-
nally, Hamilton (1967) pointed out that biased sex ratios can affect popu-
360 lation size because the number of offspring in each generation is typically

determined by the number of females. Population density can, in turn, affect the intensity of pollen/sperm competition in future generations because fewer males are available to donate pollen/sperm in a particular area. Thus, a feedback could occur between population densities and haploid selection, which has not yet been investigated.

References

- Arunkumar, R., E. B. Josephs, R. J. Williamson, and S. I. Wright. 2013. Pollen-specific, but not sperm-specific, genes show stronger purifying selection and higher rates of positive selection than sporophytic genes in *Capsella grandiflora*. *Molecular biology and evolution* 30:2475–2486.
- Bachtrog, D., J. E. Mank, C. L. Peichel, M. Kirkpatrick, S. P. Otto, T.-L. Ashman, M. W. Hahn, J. Kitano, I. Mayrose, R. Ming, N. Perrin, L. Ross, N. Valenzuela, J. C. Vamasi, and Tree of Sex Consortium. 2014. Sex determination: why so many ways of doing it? *PLoS Biol* 12:e1001899.
- Beukeboom, L. W., and N. Perrin. 2014. The evolution of sex determination. Oxford University Press, Oxford, UK.
- Borg, M., L. Brownfield, and D. Twell. 2009. Male gametophyte development: a molecular perspective. *Journal of Experimental Botany* 60:1465–1478.
- Bull, J. J. 1983. Evolution of sex determining mechanisms. The Benjamin Cummings Publishing Company.
- Charlesworth, D., and J. E. Mank. 2010. The birds and the bees and the flowers and the trees: lessons from genetic mapping of sex determination in plants and animals. *Genetics* 186:9–31.
- Charnov, E. L., and J. Bull. 1977. When is sex environmentally determined? *Nature* 266:828–830.

- 386 Clarke, H. J., T. N. Khan, and K. H. M. Siddique. 2004. Pollen selection
for chilling tolerance at hybridisation leads to improved chickpea cultivars.
388 Euphytica 139:65–74.
- Conn, J. S., and U. Blum. 1981. Sex ratio of *Rumex hastatulus*: the effect of
390 environmental factors and certation. Evolution 35:1108–1116.
- Conover, D. O., and S. W. Heins. 1987. Adaptive variation in environmental
392 and genetic sex determination in a fish. Nature 326:496–498.
- Ezaz, T., S. D. Sarre, and D. O’Meally. 2009. Sex chromosome evolution
394 in lizards: independent origins and rapid transitions. Cytogenetic and
Genome Research 127:249–260.
- 396 Field, D. L., M. Pickup, and S. C. H. Barrett. 2012. The influence of polli-
nation intensity on fertilization success, progeny sex ratio, and fitness in a
398 wind-pollinated, dioecious plant. International Journal of Plant Sciences
173:184–191.
- 400 ———. 2013. Comparative analyses of sex-ratio variation in dioecious flow-
ering plants. Evolution 67:661–672.
- 402 Frank, S. A. 1989. The Evolutionary Dynamics of Cytoplasmic Male Sterility.
American Naturalist 133:345–376.
- 404 Galen, C., J. A. Shykoff, and R. C. Plowright. 1986. Consequences of stigma
receptivity schedules for sexual selection in flowering plants. American
406 Naturalist pages 462–476.
- Gossmann, T. I., M. W. Schmid, U. Grossniklaus, and K. J. Schmid. 2014.
408 Selection-driven evolution of sex-biased genes Is consistent with sexual
selection in *Arabidopsis thaliana*. Molecular biology and evolution 31:574–
410 583.
- Hamilton, W. D. 1967. Extraordinary sex ratios. Science 156:477–488.

- 412 Hedhly, A., J. I. Hormaza, and M. Herrero. 2004. Effect of temperature
on pollen tube kinetics and dynamics in sweet cherry, *Prunus avium*
414 (Rosaceae). American journal of botany 91:558–564.
- Herrero, M. 2003. Male and female synchrony and the regulation of mating
416 in flowering plants. Philosophical Transactions of the Royal Society B:
Biological Sciences 358:1019–1024.
- 418 Hillis, D. M., and D. M. Green. 1990. Evolutionary changes of heterogametic
sex in the phylogenetic history of amphibians. Journal of Evolutionary
420 Biology 3:49–64.
- Holleley, C. E., D. O’Meally, S. D. Sarre, J. A. Marshall Graves, T. Ezaz,
422 K. Matsubara, B. Azad, X. Zhang, and A. Georges. 2015. Sex reversal
triggers the rapid transition from genetic to temperature-dependent sex.
424 Nature 523:79–82.
- Hormaza, J. I., and M. Herrero. 1996. Male gametophytic selection as a plant
426 breeding tool. Scientia horticulturae 65:321–333.
- Hough, J., S. Immler, S. Barrett, and S. P. Otto. 2013. Evolutionarily stable
428 sex ratios and mutation load. Evolution 7:1915–1925.
- Immler, S., G. Arnqvist, and S. P. Otto. 2012. Ploidally antagonistic selection
430 maintains stable genetic polymorphism. Evolution 66:55–65.
- Immler, S., C. Hotzy, G. Alavioon, E. Petersson, and G. Arnqvist. 2014.
432 Sperm variation within a single ejaculate affects offspring development in
Atlantic salmon. Biology letters 10:20131040.
- 434 Joseph, S., and M. Kirkpatrick. 2004. Haploid selection in animals. Trends
in Ecology & Evolution 19:592–597.

- 436 Kozielska, M., F. J. Weissing, L. W. Beukeboom, and I. Pen. 2010. Segrega-
tion distortion and the evolution of sex-determining mechanisms. *Heredity*
438 104:100–112.
- Lankinen, A., and J. A. Madjidian. 2011. Enhancing pollen competition
440 by delaying stigma receptivity: Pollen deposition schedules affect siring
ability, paternal diversity, and seed production in *Collinsia heterophylla*
442 (Plantaginaceae). *American journal of botany* 98:1191–1200.
- Lankinen, A., and I. Skogsmyr. 2001. Evolution of pistil length as a choice
444 mechanism for pollen quality. *Oikos* 92:81–90.
- Li, J., R. B. Phillips, A. S. Harwood, B. F. Koop, and W. S. Davidson. 2011.
446 Identification of the Sex Chromosomes of Brown Trout (*Salmo trutta*)
and Their Comparison with the Corresponding Chromosomes in Atlantic
448 Salmon (*Salmo salar*) and Rainbow Trout (*Oncorhynchus mykiss*). *Cyto-*
genetic and Genome Research 133:25–33.
- 450 Lloyd, D. G. 1974. Female-predominant sex ratios in angiosperms, vol. 32.
Heredity.
- 452 Mank, J. E., D. E. L. Promislow, and J. C. Avise. 2006. Evolution of alter-
native sexdetermining mechanisms in teleost fishes. *Biological Journal of*
454 *the Linnean Society* 87:83–93.
- Ming, R., A. Bendahmane, and S. S. Renner. 2011. Sex chromosomes in land
456 plants. [dx.doi.org](https://doi.org/10.1093/aob/mbr244) 62:485–514.
- Mulcahy, D. L., M. Sari-Gorla, and G. B. Mulcahy. 1996. Pollen selection -
458 past, present and future. *Sexual Plant Reproduction* 9:353–356.
- Myosho, T., H. Otake, H. Masuyama, M. Matsuda, Y. Kuroki, A. Fujiyama,
460 K. Naruse, S. Hamaguchi, and M. Sakaizumi. 2012. Tracing the Emergence
of a Novel Sex-Determining Gene in Medaka, *Oryzias luzonensis*. *Genetics*
462 191:163–170.

- Ogata, M., Y. Hasegawa, H. Ohtani, M. Mineyama, and I. Miura. 2007. The
 464 ZZ/ZW sex-determining mechanism originated twice and independently
 during evolution of the frog, *Rana rugosa*. *Heredity* 100:92–99.
- Otto, S. P., M. F. Scott, and S. Immler. 2015. Evolution of haploid selection
 466 in predominantly diploid organisms. *Proceedings of the National ...*
- Pen, I., T. Uller, B. Feldmeyer, A. Harts, G. M. While, and E. Wapstra.
 468 2010. Climate-driven population divergence in sex-determining systems.
 470 *Nature* 468:436–438.
- Pokorná, M., and L. Kratochvíl. 2009. Phylogeny of sexdetermining mech-
 472 anisms in squamate reptiles: are sex chromosomes an evolutionary trap?
Zoological Journal of the ... 156:168–183.
- Ravikumar, R. L., B. S. Patil, and P. M. Salimath. 2003. Drought tolerance in
 474 sorghum by pollen selection using osmotic stress. *Euphytica* 133:371–376.
- Ruane, L. G. 2009. Post-pollination processes and non-random mating among
 476 compatible mates. *Evolutionary Ecology Research* 11:1031–1051.
- Ser, J. R., R. B. Roberts, and T. D. Kocher. 2010. Multiple interacting loci
 478 control sex determination in lake Malawi cichlid fish. *Evolution* 64:486–
 480 501.
- Slancarova, V., J. Zdanska, B. Janousek, M. Talianova, C. Zschach, J. Zlu-
 482 vova, J. Siroky, V. Kovacova, H. Blavet, J. Danihelka, B. Oxelman, A. Wid-
 mer, and B. Vyskot. 2013. Evolution of sex determination systems with
 484 heterogametic males and females in *Silene*. *Evolution* 67:3669–3677.
- Smith, D. A. S. 1975. All-female broods in the polymorphic butterfly *Danaus*
 486 *chrysippus* L. and their ecological significance. *Heredity* 34:363–371.
- Stalker, H. D. 1961. The Genetic Systems Modifying Meiotic Drive in
 488 *Drosophila Paramelanica*. *Genetics* .

- 490 Stehlik, I., and S. Barrett. 2005. Mechanisms governing sex-ratio variation
in dioecious *Rumex nivalis*. *Evolution* 59:814–825.
- 492 Stehlik, I., and S. C. H. Barrett. 2006. Pollination intensity influences sex
ratios in dioecious *Rumex nivalis*, a wind-pollinated plant. *Evolution*
60:1207–1214.
- 494 Travers, S. E., and K. Shea. 2001. Selection on pollen competitive ability in
relation to stochastic factors influencing pollen deposition. *Evolutionary*
496 *Ecology Research* 3:729–745.
- Úbeda, F., M. M. Patten, and G. Wild. 2015. On the origin of sex chromo-
498 somes from meiotic drive. *Proceedings of the Royal Society B: Biological*
Sciences 282:20141932.
- 500 van Doorn, G. S., and M. Kirkpatrick. 2007. Turnover of sex chromosomes
induced by sexual conflict. *Nature* 449:909–912.
- 502 ———. 2010. Transitions Between Male and Female Heterogamety Caused
by Sex-Antagonistic Selection. *Genetics* 186:629–645.
- 504 Vibranovski, M. D., D. S. Chalopin, H. F. Lopes, M. Long, and T. L. Karr.
2010. Direct evidence for postmeiotic transcription during *Drosophila*
506 *melanogaster* spermatogenesis. *Genetics* 186:431–433.
- Vicoso, B., and D. Bachtrog. 2015. Numerous transitions of sex chromosomes
508 in Diptera. *PLoS Biol* 13:e1002078.
- Vuilleumier, S., R. Lande, J. J. M. van Alphen, and O. Seehausen. 2007.
510 Invasion and fixation of sex-reversal genes. *Journal of Evolutionary Biology*
20:913–920.
- 512 Werren, J. H., and P. D. Taylor. 1984. The effects of population recruitment
on sex ratio selection. *The American Naturalist* 124:143–148.

- 514 Wilson, D. S., and R. K. Colwell. 1981. Evolution of sex ratio in structured
demes. *Evolution* 35:882–897.
- 516 Yano, A., B. Nicol, E. Jouanno, E. Quillet, A. Fostier, R. Guyomard,
and Y. Guiguen. 2012. The sexually dimorphic on the Y-chromosome
518 gene (sdY) is a conserved male-specific Y-chromosome sequence in many
salmonids. *Evolutionary Applications* 6:486–496.
- 520 Zheng, Y., X. Deng, and P. A. Martin-DeLeon. 2001. Lack of sharing of
Spam1 (Ph-20) among mouse spermatids and transmission ratio distortion.
522 *Biology of Reproduction* 64:1730–1738.

Figures

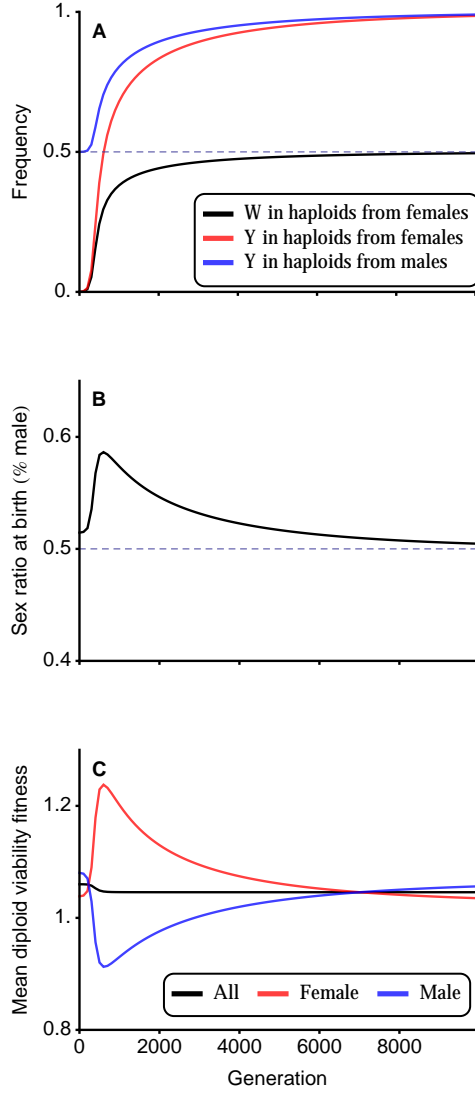


Figure 1: Haploid selection allows a neo- W to invade an ancestral XY system and fix (**A**) despite temporarily biasing the sex ratio further (**B**) and decreasing mean diploid viability fitness (**C**). Complete turnover between genetic sex-determination systems occurs despite the neo- W being less tightly linked to the selected locus than the ancestral sex-determining locus is, $R > r$. Parameters: $k = 1$, $s^f = 0.05$, $s^m = 0.15$, $h^f = h^m = 0.7$, $t^f = 0$, $t^m = -0.1$, $\alpha^m = \alpha^f = 1/2$, $r = 0.01$, $R = 0.05$.

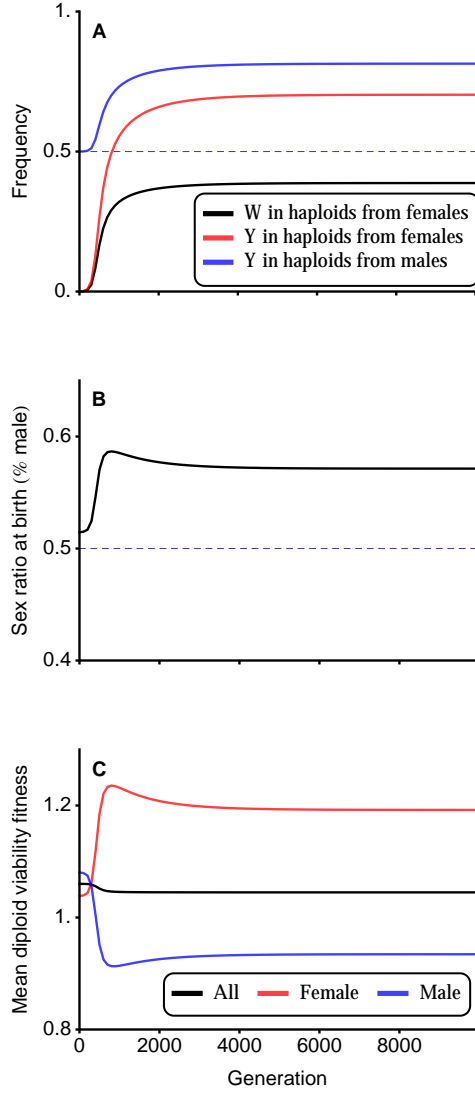


Figure 2: Haploid selection allows a completely unlinked neo- W to invade an ancestral XY system (**A**) despite further biasing the sex ratio (**B**) and decreasing mean diploid viability fitness (**C**). The neo- W does not fix (although variation at the **A** locus is maintained, $V_A > 0$), resulting in a polymorphic sex-determination system. Parameters as in Figure 1 but with $R = 0.5$.

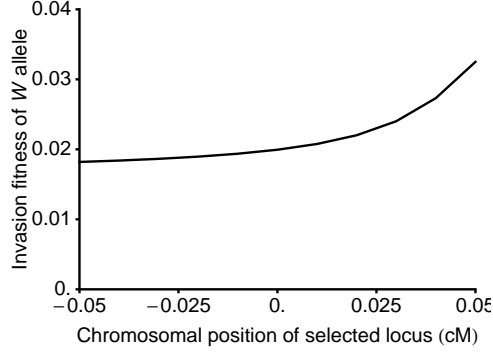


Figure 3: Haploid selection allows a neo- W to invade an ancestral XY system regardless of how tightly it and the ancestral sex-determining locus are linked to the selected locus. The ancestral sex-determining locus is located at -0.05 and the novel sex-determining locus is located at 0.05, such that the probability of a cross-over between them is ≈ 0.1 . The x-axis gives the position of the locus under haploid selection. We used Haldane's map function (Equation 3 in ?) to convert from map distance (centiMorgans) to the probability of a cross-over event. Parameters as in Figure 1.

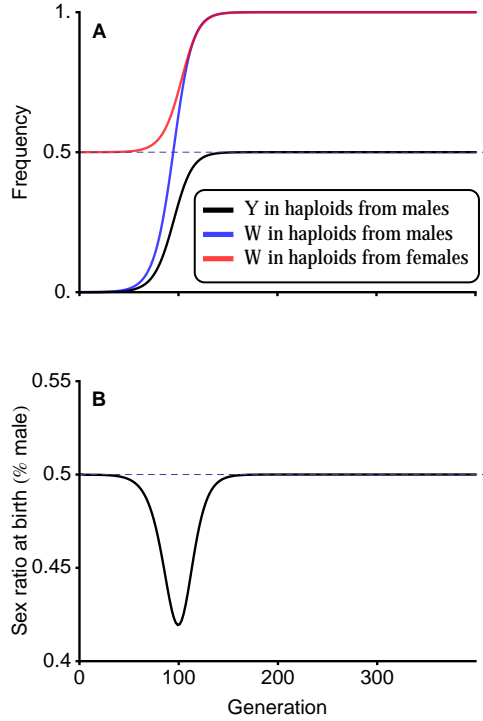


Figure 4: Meiotic drive allows a neo- Y to invade an ancestral ZW system and fix (**A**) despite temporarily biasing the sex ratio (**B**). Parameters: $k = 0$, $s^f = s^m = t^f = t^m = 0$, $\alpha^m = 0.4$, $\alpha^f = 1/2$, $r = 0$, $R = 0$.