

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 where we also include 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 also cause sex ratios at birth to become biased (because sex ratios are determined by the fertilization success of X- versus Y-bearing pollen/sperm). We find that the evolution of sex determination systems where mothers determine sex at birth (e.g., environmental sex determination where sex is determined at birth) is influenced by classic Fisherian sex ratio selection. (Maybe not true???) However, 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

Animals and angiosperms exhibit extremely diverse sex determination systems, as reviewed by Bull (1983), Charlesworth and Mank (2010), Beukeboom and Perrin (2014), Bachtrog et al. (2014). Among species with genetic sex determination of diploid sexes, some taxa have heterogametic males (XY) and homogametic females (XX), including mammals and most dioecious plants (Ming et al. 2011); whereas other taxa have homogametic males (ZZ) and heterogametic females (ZW), including Lepidoptera and birds. Within several taxa, the chromosome that harbours the master sex-determining region changes. For example, transitions of the master 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), Diptera (Vicoso and Bachtrog 2015), and *Oryzias* (Myosho et al. 2012). In addition, many gonochoric/dioecious clades with genetic sex determination exhibit transitions 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 (Tephritids, Vicoso and Bachtrog 2015), amphibians (Hillis and Green 1990), the angiosperm genus *Silene* (Slancarova et al. 2013), Coleoptera and Hemiptera (Beukeboom 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 exhibited by some cichlid species (Ser et al. 2010) and *Rana rugosa* (Ogata et al. 2007). In addition, multiple transitions have occurred between genetic and environmental 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 et al. 2010, Holleley et al. 2015).

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

ample, linkage allows 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 a new chromosome (van Doorn and Kirkpatrick 2007) and can also favour a transition between male and female heterogamety (e.g., a ZW to XY transition, van Doorn and Kirkpatrick 2010). However, any sexually-antagonistic loci that are linked to the ancestral sex-determination locus will develop similar, favourable associations and select against the spread of a new sex-determination system.

It has been suggested that sex ratio selection could be a particularly important force driving transitions between sex-determining systems (Beukeboom and Perrin 2014, Chapter 7). For example, flexible sex determination systems may be favoured in order to exploit local environmental conditions that are optimal for males or females, which creates locally biased sex ratios (Charnov and Bull 1977, Werren and Taylor 1984, Pen et al. 2010). In addition, feminizing mutations may invade when female biased sex ratios are favoured due to selection among demes (Wilson and Colwell 1981, Vuilleumier et al. 2007). In other situations, sex ratio selection may favour transitions in order to restore equal sex ratios. For example, Kozielska et al. (2010) consider systems in which the ancestral sex chromosomes experience meiotic drive (e.g., where driving X or Y chromosomes are inherited disproportionately often), which causes sex ratios to become biased (Hamilton 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 an even sex ratio.

Here, we use mathematical models to find the conditions under which new sex determination systems are favoured by selection where we include a period of selection among haploid gametes/gametophytes. Selection among haploid genotypes is thought to occur primarily among pollen/sperm, which can compete whenever there are more pollen/sperm than required for fertilization (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).

NOTE RE: DRIVE. I expect drive (that occurs specifically in one sex, e.g., during spermatogenesis) to behave almost exactly like haploid selection. 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 drive systems) will only favour invasion of a more tightly linked neo-Y (worsening sex ratio biases) and could favour invasion of a neo-W. This may run counter to generic expectations from new sex chromosome systems evolving to balance the sex ratio. So, do you think it would significantly enhance the paper to model drive explicitly or just discuss it as being similar???

FOR RESULTS?

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

Model

We will consider a three locus model. Locus **X** is the ancestral sex-determining region (SDR), with alleles X and Y (or Z and W). Locus **A** is a region under 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 zygote is determined by the genotype at locus **X** (XX become females and XY males, or ZW become females and ZZ males). With at 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 is a masculinizer (e.g., a neo-Y chromosome) and with $k = 1$ the novel sex determiner is a feminizer (e.g., a neo-W chromosome). With intermediate k , locus **M** is interpreted as an environmental sex-determining region.

The life-cycle begins with competition between haploid gametes/gametophytes within each sex, where selection depends on the sex of the diploid they came from and their allele at the **A** locus. Gametes/gametophytes from males then mate randomly with gametes/gametophytes from females. The resulting zygotes develop as males or females, depending on their genotypes at the **X** and **M** loci. Diploids then 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 meiosis with recombination. Recombination 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 . Any order of the loci can be modelled with appropriate choices of r , R , and χ . We track the frequency of haploid genotypes produced by each sex from one generation to the next (recursion equations in Sup. Mat.).

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 sex-determiner m to increase in frequency at this equilibrium. Numerical simulations are used to examine when an invading mutation goes to fixation.

Results

Resident equilibrium and stability

With allele M fixed, we follow the dynamics of the frequency of A in gametes/gametophytes from the homogametic sex (e.g., in eggs from an XX female, p_{Xf}) and the frequencies of A in the two types of gametes/gametophytes from the heterogametic sex (e.g., in sperm from an XY male that are X-bearing, p_{Xm} , and Y-bearing, p_{Ym}). Assuming selection is weak relative to recombination, the differences in the frequencies of A in each type of gamete/gametophyte are small, and we can solve for the mean frequency of A across all types (p_A) as well as the difference in the frequencies of A between two of the three types, to first order in selection. Linear stability analysis can then be used to determine the stability of this equilibrium. With no haploid selection and $k = 0$ (neo- Y invading a ZW system) or $k = 1$ (neo- W invading an XY system), our results reduce to those of van Doorn and Kirkpatrick (2010).

Sex chromosome turnover

The spread of a rare mutant m at the \mathbf{M} locus in such a population is determined by the leading eigenvalue, λ , of the system described by the equations for the next generation frequency of haploid genotypes with the mutation. Below we present the results for invasion into an XY system assuming equal dominance coefficients in the two sexes, no competition among gametes/gametophytes from females, and linear arrangement **XAM**. Equivalent results for invasions into a ZW system are given in the Sup. Mat.

Neo-Y

The growth rate of a rare, dominant neo-Y region ($k = 0$) is

$$\lambda_{Y,XY} = 1 + V_A \frac{(r - R)}{rR} \frac{s_f^2}{(s_f + s_m)^2} t_m^2, \quad (1)$$

where $V_A = p_A(1 - p_A)$ is the variance at the **A** locus, s_f and s_m are the respective selection coefficients for A in diploid females and males, and t_m is the selection coefficient for A in gametes/gametophytes from males. The neo-Y can therefore invade whenever it is in tighter linkage with the selected locus than the ancestral sex-determining locus, $r > R$, provided locus **A** is polymorphic ($V_A > 0$) and there is selection among females ($s_f \neq 0$) and gametes/gametophytes from males ($t_m \neq 0$).

Neo-W

The growth rate of a rare, dominant neo-W region ($k = 1$) is

$$\lambda_{W,XY} = 1 + V_A \frac{s_f ((2r(1 - R) + R)s_f + (1 - 2r)Rs_m)}{2rR(s_f + s_m)^2} t_m^2. \quad (2)$$

In this case, even when the novel sex-determining locus is in looser linkage with the selected locus than is the ancestral sex-determining locus, $r < R$, a novel sex-determiner can invade. For example, with $R = 1/2$ the neo-W invades if there is some linkage between the ancestral sex-determining and selected loci ($r < 1/2$), there is selection among gametes/gametophytes 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 females than in males ($|s_f| > |s_m|$).

Neo-ESD

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

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

Thus the $k = 1/2$ case gets half of the advantages of a neo- W and half that of a neo- Y . Recombination between the selected locus and the novel sex-determining locus, R , 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.

Discussion

2 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
4 assume that drive occurs predominantly in one sex, e.g., during spermatogenesis or a 'killer' sperm. A driving allele is maintained at an intermediate
6 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
8 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.
10 (2015) also find that genetic sex determiners can invade, despite causing sex ratios to become biased. Finally, they show that autosomal 'restorers' that
12 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
14 meiotic drivers and restore equal sex ratios have been identified (Stalker 1961, Smith 1975). A similar process occurs with cytoplasmic male sterility
16 alleles (that cause biased sex ratios) and nuclear 'restorer' genotypes (Frank 1989). When sex ratio bias occurs due to haploid selection, a natural class

18 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
20 pollen competition can be manipulated by altering style length (Travers and
Shea 2001, Lankinen and Skogsmyr 2001, Ruane 2009), delaying stigma re-
22 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
24 fitness differences, Hough et al. (2013) and Otto et al. (2015) demonstrated
that mothers should generally evolve to balance sex ratios by reducing the
26 intensity of haploid competition.

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

32 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
34 harbours a polymorphism that affects haploid fitness. Mothers again primar-
ily evolve to restore equal sex ratios. However, modifying haploid selection
36 also affects the X-linked genotypes that are inherited by offspring. Specif-
ically, increasing the intensity of haploid selection increases the proportion
38 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-
40 ters, mothers can be selected to increase the intensity of haploid selection;
otherwise, decreased selection among haploids is favoured. Thus, because
42 altering haploid selection intensity affects the alleles that are inherited by
daughters, mothers can favour slightly biased sex ratios. In addition, I found
44 that stronger sex ratio biases can be favoured by paternal manipulations of
the haploid ‘selective arena’ because fathers are strongly selected to maximize
46 their own siring success (above selection to equalize the sex ratio).

Discuss patterns that might be looked for:

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

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

“Polygenic sex determination has been reported in many plants (e.g.
58 Shannon & Holsinger 2007), fishes (Vandeputte et al. 2007; Ser et al. 2010;
Liew et al. 2012), crustaceans (e.g. Battaglia 1958; Battaglia & Male-
60 sani 1959; Voordouw & Anholt 2002), bivalves (Haley 1977; Saavedra et
al. 1997), gastropods (Yusa 2007a,b), and polychaetes (Bacci 1965, 1978;
62 Premoli et al. 1996).” From Vuilleumier et al. 2007: “Polymorphism for sex-
determining genes within oramong populations has been reported in many
64 speciesincluding houseflies, midges, woodlice, platyfish, cichlidfish, and frogs
(Gordon, 1944; Kallman, 1970; Thomp-son, 1971; Macdonald, 1978; Bull,
66 1983; Rigaud et al., 1997; Caubet et al., 2000; Lande et al., 2001; Ogataet
al., 2003; Lee et al., 2004; Mank et al., 2006).

68 Several aspects of the relationship between haploid selection (e.g., pollen
or sperm competition) and sex ratios remain to be explored. For example,
70 new sex-determining systems (particularly transitions between male and fe-
male heterogamety) can be favoured in order to restore equal sex ratios in
72 populations that have a sex ratio bias (Bull 1983, Kozielska et al. 2010, Úbeda
et al. 2015). Based on the results of Chapter ??, we would expect that sex
74 ratio biases would occur via associations between sex-determining loci and
loci that experience haploid selection. However, these associations should
76 also select against transitions between sex-determining systems, as has been
found with sexually antagonistic selection (van Doorn and Kirkpatrick 2007;

2010). It is not 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. Finally, Hamilton (1967) pointed out that biased sex ratios can affect population 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. Vamossi, 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
 104 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?
 106 266:828–830.
- Clarke, H. J., T. N. Khan, and K. H. M. Siddique. 2004. Pollen selection
 108 for chilling tolerance at hybridisation leads to improved chickpea cultivars.
 110 *Euphytica* 139:65–74.
- Conover, D. O., and S. W. Heins. 1987. Adaptive variation in environmental
 112 and genetic sex determination in a fish. *Nature* 326:496–498.
- Ezaz, T., S. D. Sarre, and D. O’Meally. 2009. Sex chromosome evolution
 114 in lizards: independent origins and rapid transitions. *Cytogenetic and
 Genome Research* 127:249–260.
- Frank, S. A. 1989. The Evolutionary Dynamics of Cytoplasmic Male Sterility.
 116 *American Naturalist* 133:345–376.
- Galen, C., J. A. Shykoff, and R. C. Plowright. 1986. Consequences of stigma
 118 receptivity schedules for sexual selection in flowering plants. *American
 120 Naturalist* pages 462–476.
- Gossmann, T. I., M. W. Schmid, U. Grossniklaus, and K. J. Schmid. 2014.
 122 Selection-driven evolution of sex-biased genes Is consistent with sexual
 selection in *Arabidopsis thaliana*. *Molecular biology and evolution* 31:574–
 124 583.
- Hamilton, W. D. 1967. Extraordinary sex ratios. *Science* 156:477–488.
- Hedhly, A., J. I. Hormaza, and M. Herrero. 2004. Effect of temperature
 126 on pollen tube kinetics and dynamics in sweet cherry, *Prunus avium*
 128 (Rosaceae). *American journal of botany* 91:558–564.

- Herrero, M. 2003. Male and female synchrony and the regulation of mating
 130 in flowering plants. *Philosophical Transactions of the Royal Society B: Biological Sciences* 358:1019–1024.
- Hillis, D. M., and D. M. Green. 1990. Evolutionary changes of heterogametic
 132 sex in the phylogenetic history of amphibians. *Journal of Evolutionary*
 134 *Biology* 3:49–64.
- Holleley, C. E., D. O’Meally, S. D. Sarre, J. A. Marshall Graves, T. Ezaz,
 136 K. Matsubara, B. Azad, X. Zhang, and A. Georges. 2015. Sex reversal
 triggers the rapid transition from genetic to temperature-dependent sex.
 138 *Nature* 523:79–82.
- Hormaza, J. I., and M. Herrero. 1996. Male gametophytic selection as a plant
 140 breeding tool. *Scientia horticultrae* 65:321–333.
- Hough, J., S. Immler, S. Barrett, and S. P. Otto. 2013. Evolutionarily stable
 142 sex ratios and mutation load. *Evolution* 7:1915–1925.
- Immler, S., G. Arnqvist, and S. P. Otto. 2012. Ploidally antagonistic selection
 144 maintains stable genetic polymorphism. *Evolution* 66:55–65.
- Immler, S., C. Hotzy, G. Alavioon, E. Petersson, and G. Arnqvist. 2014.
 146 Sperm variation within a single ejaculate affects offspring development in
 Atlantic salmon. *Biology letters* 10:20131040.
- Joseph, S., and M. Kirkpatrick. 2004. Haploid selection in animals. *Trends*
 148 *in Ecology & Evolution* 19:592–597.
- Kozielska, M., F. J. Weissing, L. W. Beukeboom, and I. Pen. 2010. Segrega-
 150 tion distortion and the evolution of sex-determining mechanisms. *Heredity*
 152 104:100–112.
- Lankinen, A., and J. A. Madjidian. 2011. Enhancing pollen competition
 154 by delaying stigma receptivity: Pollen deposition schedules affect siring

- ability, paternal diversity, and seed production in *Collinsia heterophylla*
 156 (Plantaginaceae). American journal of botany 98:1191–1200.
- Lankinen, A., and I. Skogsmyr. 2001. Evolution of pistil length as a choice
 158 mechanism for pollen quality. Oikos 92:81–90.
- Li, J., R. B. Phillips, A. S. Harwood, B. F. Koop, and W. S. Davidson. 2011.
 160 Identification of the Sex Chromosomes of Brown Trout (*Salmo trutta*)
 and Their Comparison with the Corresponding Chromosomes in Atlantic
 162 Salmon (*Salmo salar*) and Rainbow Trout (*Oncorhynchus mykiss*). Cyto-
 genetic and Genome Research 133:25–33.
- 164 Mank, J. E., D. E. L. Promislow, and J. C. Avise. 2006. Evolution of alter-
 native sexdetermining mechanisms in teleost fishes. Biological Journal of
 166 the Linnean Society 87:83–93.
- Ming, R., A. Bendahmane, and S. S. Renner. 2011. Sex chromosomes in land
 168 plants. dx.doi.org 62:485–514.
- Mulcahy, D. L., M. Sari-Gorla, and G. B. Mulcahy. 1996. Pollen selection -
 170 past, present and future. Sexual Plant Reproduction 9:353–356.
- Myosho, T., H. Otake, H. Masuyama, M. Matsuda, Y. Kuroki, A. Fujiyama,
 172 K. Naruse, S. Hamaguchi, and M. Sakaizumi. 2012. Tracing the Emergence
 of a Novel Sex-Determining Gene in Medaka, *Oryzias luzonensis*. Genetics
 174 191:163–170.
- Ogata, M., Y. Hasegawa, H. Ohtani, M. Mineyama, and I. Miura. 2007. The
 176 ZZ/ZW sex-determining mechanism originated twice and independently
 during evolution of the frog, *Rana rugosa*. Heredity 100:92–99.
- 178 Otto, S. P., M. F. Scott, and S. Immler. 2015. Evolution of haploid selection
 in predominantly diploid organisms. Proceedings of the National

- 180 Pen, I., T. Uller, B. Feldmeyer, A. Harts, G. M. While, and E. Wapstra.
2010. Climate-driven population divergence in sex-determining systems.
182 Nature 468:436–438.
- Pokorná, M., and L. Kratochvíl. 2009. Phylogeny of sexdetermining mech-
184 anisms in squamate reptiles: are sex chromosomes an evolutionary trap?
Zoological Journal of the ... 156:168–183.
- 186 Ravikumar, R. L., B. S. Patil, and P. M. Salimath. 2003. Drought tolerance in
sorghum by pollen selection using osmotic stress. Euphytica 133:371–376.
- 188 Ruane, L. G. 2009. Post-pollination processes and non-random mating among
compatible mates. Evolutionary Ecology Research 11:1031–1051.
- 190 Ser, J. R., R. B. Roberts, and T. D. Kocher. 2010. Multiple interacting loci
control sex determination in lake Malawi cichlid fish. Evolution 64:486–
192 501.
- Slancarova, V., J. Zdanska, B. Janousek, M. Talianova, C. Zschach, J. Zlu-
194 vova, J. Siroky, V. Kovacova, H. Blavet, J. Danihelka, B. Oxelman, A. Wid-
mer, and B. Vyskot. 2013. Evolution of sex determination systems with
196 heterogametic males and females in *Silene*. Evolution 67:3669–3677.
- Smith, D. A. S. 1975. All-female broods in the polymorphic butterfly *Danaus*
198 *chrysippus* L. and their ecological significance. Heredity 34:363–371.
- Stalker, H. D. 1961. The Genetic Systems Modifying Meiotic Drive in
200 *Drosophila Paramelanica*. Genetics .
- Travers, S. E., and K. Shea. 2001. Selection on pollen competitive ability in
202 relation to stochastic factors influencing pollen deposition. Evolutionary
Ecology Research 3:729–745.

- 204 Úbeda, F., M. M. Patten, and G. Wild. 2015. On the origin of sex chromo-
somes from meiotic drive. *Proceedings of the Royal Society B: Biological*
206 *Sciences* 282:20141932.
- van Doorn, G. S., and M. Kirkpatrick. 2007. Turnover of sex chromosomes
208 induced by sexual conflict. *Nature* 449:909–912.
- . 2010. Transitions Between Male and Female Heterogamety Caused
210 by Sex-Antagonistic Selection. *Genetics* 186:629–645.
- Vibranovski, M. D., D. S. Chalopin, H. F. Lopes, M. Long, and T. L. Karr.
212 2010. Direct evidence for postmeiotic transcription during *Drosophila*
melanogaster spermatogenesis. *Genetics* 186:431–433.
- 214 Vicoso, B., and D. Bachtrog. 2015. Numerous transitions of sex chromosomes
in Diptera. *PLoS Biol* 13:e1002078.
- 216 Vuilleumier, S., R. Lande, J. J. M. van Alphen, and O. Seehausen. 2007.
Invasion and fixation of sex-reversal genes. *Journal of Evolutionary Biology*
218 20:913–920.
- Werren, J. H., and P. D. Taylor. 1984. The effects of population recruitment
220 on sex ratio selection. *The American Naturalist* 124:143–148.
- Wilson, D. S., and R. K. Colwell. 1981. Evolution of sex ratio in structured
222 demes. *Evolution* 35:882–897.
- Yano, A., B. Nicol, E. Jouanno, E. Quillet, A. Fostier, R. Guyomard,
224 and Y. Guiguen. 2012. The sexually dimorphic on the Y-chromosome
gene (sdY) is a conserved male-specific Y-chromosome sequence in many
226 salmonids. *Evolutionary Applications* 6:486–496.
- Zheng, Y., X. Deng, and P. A. Martin-DeLeon. 2001. Lack of sharing of
228 Spam1 (Ph-20) among mouse spermatids and transmission ratio distortion.
Biology of Reproduction 64:1730–1738.