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

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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, reviewed in 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 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 the same species, including cichlid species (Ser et al. 2010) and *Rana rugosa* (Ogata et al. 2007).

Depending on the prominence of transitions to ESD in the manuscript include something like (currently quoted):

"Transitions have repeatedly occurred between environmental sex determination and genotypic sex determination, as exemplified by the distribution of temperature sex determination among reptiles: either temperature or genes provide the initial trigger in closely related species (Ewert and Nelson 1991; Pokorna and Kratochvil 2009; Ezaz et al. 2009) or even conspecific populations (Pen et al. 2010). Similar situations are found in fishes (e.g.,

Conover and Heins 1978a)."

We have results where polygenic sex determination is sometimes stable:

"Polygenic sex determination has been reported in many plants (e.g. 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), gastropods (Yusa 2007a,b), and polychaetes (Bacci 1965, 1978; Premoli et al. 1996)."

Description of sex ratio adjustment and sexual antagonism theories:

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 example, 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.

Sex ratio selection might be a particularly important force driving transitions between sex-determining systems (Beukeboom and Perrin 2014, Chapter 7). For example, feminizing mutations may invade when female biased sex ratios are favoured due to interdemic selection (Wilson and Colwell 1981, Vuilleumier et al. 2007 CITE) or local mate competition (Hamilton 1967, Reinhold 1996, Werren and Taylor 1984, Pen et al. 2010 CITE). 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., driving X or Y chromosomes are inherited disproportionately often), which leads to biased sex ratios. They find that new, unlinked sex-determining loci (masculinizing or feminizing mutations) can then spread, restoring an even sex ratio.

For example, new sex-determining systems (particularly transitions between male and female heterogamety) can be favoured in order to restore equal sex ratios in populations that have a sex ratio bias (Bull 1983, Kozielska et al. 2010, Úbeda et al. 2015).

Sex ratios can also become biased due to meiotic drive; in a classic paper, Hamilton (1967) showed that X- or Y-linked alleles that experience meiotic drive will bias sex ratios. He assumed that driving alleles are under directional selection and spread to fixation but such alleles can also be maintained at intermediate frequencies by selection (Feldman and Otto 1989, Holman et al. 2015). When sex ratios are biased, other loci are expected to evolve to restore equal sex ratios. Indeed, alleles that negate the effect of sex-linked meiotic drivers and restore equal sex ratios have been identified (Stalker 1961, Smith 1975). A similar process occurs with cytoplasmic male sterility alleles (that cause biased sex ratios) and nuclear 'restorer' genotypes (Frank 1989).

When sex ratio bias occurs due to haploid selection, a natural class of sex ratio 'restorers' exist because haploid selection often occurs in a context that is determined by the diploid parents. For example, the intensity of pollen competition can be manipulated by altering style length (Travers and Shea 2001, Lankinen and Skogsmyr 2001, Ruane 2009), delaying stigma receptivity (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 fixed fitness differences, Hough et al. (2013) demonstrated that mothers should generally evolve to balance sex ratios by reducing the intensity of haploid competition. However, reducing competition among haploids also reduces the potential for harmful deleterious mutations to be purged. When deleterious mutations are included, the optimal intensity of haploid selection can reflect

a balance between maximizing offspring fitness and equalizing sex ratios.

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

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.

We add haploid selection (and justification, see below)
Mention Hough study for sex ratio adjustment here or in discussion? Also discuss the fact that, in terms of recombination suppression, haploid selection among male gametes generates selection pressure similar to that of male specific selection.
What will be the result: where there is sex biases and sex-specific selection

FROM PREVIOUS PAPER:

To the extent that pollen and sperm success reflects differences in their haploid genotypes, selection among these gametes/gametophytes is qualitatively distinct from selection among diploid males. That is, diploids cannot be assigned fitness values that also account for the fitness of their haploid

gametes Immler et al. (2012). In plants, selection among haploid male gametophytes is thought to be pervasive Skogsmyr and Lankinen (2002), Moore and Pannell (2011), Marshall and Evans (2016); in *Arabidopsis*, 60-70% of all genes are expressed during the haploid phase Borg et al. (2009), and pollen expressed genes exhibit stronger signatures of purifying selection and positive selection Arunkumar et al. (2013), Gossmann et al. (2014). For agricultural breeding, pollen has been exposed to a variety of selection pressures *in vivo* and *in vitro*, including temperature Hedhly et al. (2004), Clarke et al. (2004), herbicides Frascaroli and Songstad (2001), metals Searcy and Mulcahy (1985), water stress Ravikumar et al. (2003), and pathogens Ravikumar et al. (2012), resulting in an increased frequency of resistant genotypes among the diploid sporophytic offspring. In animals, expression during the haploid sperm stage is traditionally thought to be suppressed Hecht (1998), although recent evidence suggests that the extent and selective importance of post-meiotic gene expression may be underestimated Zheng et al. (2001), Joseph and Kirkpatrick (2004), Vibranovski et al. (2010), Immler et al. (2014).

Here, suggest that the canonical view (no haploid expression in animals, genome highly chromatinized in sperm and not expressed) might be based on model organisms, such as mice, where sperm is sufficiently short-lived that transcripts provisioned during spermatogenesis may be sufficient without further haploid transcription (although note that the Vibranovski lab results are in mice showing some transcription does occur). In broadcast spawning animal species (e.g., corals, many fish) and species where sperm typically requires greater longevity, expression of the haploid genotype may be required (Immler paper indicates this, but not that strongly - as I remember). We can use this suggestion in discussion to speculate in what species the processes we study might be looked for (i.e.., animals with multiple matings, broadcast spawning and/or long-lived sperm and outcrossing/non-pollen-limited plants).

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 ploidally antagonistic selection) or a combination of these selective regimes.

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???

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 haploid gametes/gametophytes experiencing se-

lection that depends on 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. There is then a period of diploid selection that again depends on genotypes at the **A** locus, 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

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. Without haploid selection this reduces to the results of (van Doorn and Kirkpatrick 2010).

Discussion

FROM THESIS: Generally, any sex-linked gene that harbours genetic variation in haploid fitness should cause sex ratios to become biased. Sex ratio bias caused by pollen competition has previously been discussed in the context of Y-linked deleterious mutations, which are thought to build up after recombination suppression evolves (Lloyd 1974, Stehlik and Barrett 2005). Sex ratios can also become biased due to meiotic drive; in a classic paper, Hamilton (1967) showed that X- or Y-linked alleles that experience meiotic drive will bias sex ratios. He assumed that driving alleles are under directional selection and spread to fixation but such alleles can also be maintained at intermediate frequencies by selection (Feldman and Otto 1989, Holman et al. 2015). When sex ratios are biased, other loci are expected to evolve to restore equal sex ratios. Indeed, alleles that negate the effect of sex-linked meiotic drivers and restore equal sex ratios have been identified (Stalker 1961, Smith 1975). A similar process occurs with cytoplasmic male sterility alleles (that cause biased sex ratios) and nuclear 'restorer' genotypes (Frank 1989).

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Several aspects of the relationship between haploid selection (e.g., pollen or sperm competition) and sex ratios remain to be explored. For example, new sex-determining systems (particularly transitions between male and female heterogamety) can be favoured in order to restore equal sex ratios in 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 ratio biases would occur via associations between sex-determining loci and loci that experience haploid selection. However, these associations should 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.

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