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PLOIDALLY ANTAGONISTIC SELECTION MAINTAINS STABLE GENETIC POLYMORPHISM

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Understanding the maintenance of genetic variation in the face of selection remains a key issue in evolutionary biology. One potential mechanism for the maintenance of genetic variation is opposing selection during the diploid and haploid stages of biphasic life cycles universal among eukaryotic sexual organisms. If haploid and diploid gene expression both occur, selection can act in each phase, potentially in opposing directions. In addition, sex-specific selection during haploid phases is likely simply because male and female gametophytes/gametes tend to have contrasting life histories. We explored the potential for the maintenance of a stable polymorphism under ploidally antagonistic as well as sex-specific selection. Furthermore, we examined the role of the chromosomal location of alleles (autosomal or sex-linked). Our analyses show that the most permissible conditions for the maintenance of polymorphism occur under negative ploidy-by-sex interactions, where stronger selection for an allele in female than male diploids is coupled with weaker selection against the allele in female than male haploids. Such ploidy-by-sex interactions also promote allele frequency differences between the sexes. With constant fitness, ploidally antagonistic selection can maintain stable polymorphisms for autosomal and X-linked genes but not for Y-linked genes. We discuss the implications of our results and outline a number of biological settings where the scenarios modeled may apply.

KEY WORDS: **Alternating life-cycles, haploid selection, sexual conflict, sex chromosomes, sexually antagonistic selection.**

Evolution requires genetic variation. Without genetic variation, neither a response to selection nor genetic drift is possible. This begs the question, what maintains genetic variation? The traditional mechanisms maintaining genetic variation include a balance between mutation and drift, a balance between mutation and selection (e.g., Hill 1982), heterosis or overdominance (e.g., Gillespie 1984), and/or negative frequency-dependent selection (e.g., Trotter and Spencer 2007; for reviews see Falconer and Mackay 1996; Barton and Keightley 2002; Mackay 2010). Here, we concentrate on scenarios where diploid selection alone cannot maintain polymorphism (e.g., no overdominance). One such scenario exists where selection acts in opposite directions in the two phases (haploid and diploid) of a sexual eukaryotic life cycle.

All eukaryotic sexual organisms exhibit a life cycle alternating between a genomically doubled (typically diploid) and reduced (typically haploid) phase (Strasburger 1894; Roe 1975). Differences at the genetic, cellular, and organismal level allow selection to vary in strength and even direction between these phases. In land plants and higher animals, the diploid phase is generally the predominant phase, which can be explained by diploid advantages such as masking deleterious mutations, masking somatic mutations, or bearing more beneficial mutations (reviewed in Valero et al. 1992; Mable and Otto 1998; but see also Hughes and Otto 1999). Despite a prolonged diploid phase, extensive gene expression and selection during the haploid phase have been demonstrated in plants (e.g., Searcy and Mulcahy 1985a,b; Sari-Gorla et al. 1989; see Mascarenhas 1990; Borg et al. 2009 for review). In contrast, gene expression in the gametes of animals is thought to be largely diploid, with both homologous chromosomes contributing to gene products, although

recent research indicates that postmeiotic gene expression may be underappreciated (Zheng et al. 2001; Joseph and Kirkpatrick 2004; Krawetz 2005; Martin-DeLeon et al. 2005; Barreau et al. 2008; Vibranovski et al. 2010).

As mentioned above, selection during the different ploidy phases may contribute to the maintenance of genetic variability. The scenarios under which polymorphism can be maintained by selection have been investigated in a series of theoretical models (Scudo 1967; Harding 1975; Ewing 1977; Gregorius 1982). The general conclusion of these models is that in the absence of mutation, overdominance, or negative frequency dependence (see also Strobeck 1979), antagonistic selection is needed to maintain genetic variation. The antagonistic selection in these previous models was assumed to act either across ploidy levels (i.e., between the diploid and the haploid stage; Scudo 1967; Ewing 1977) or between the sexes exclusively at the haploid stage (Gregorius 1982). Here we generalize previous work by allowing selection to act in different ways in the two sexes and in the two ploidy phases, which allows us to develop the idea of ploidally antagonistic selection further and to investigate explicitly how ploidy-by-sex interactions influence the maintenance of genetic variation.

Before discussing the potential influence of separate sexes, we need to define "separate sexes" in some detail. For clarity, we borrow terminology from the botanical literature and refer to "sporophytes" (typically diploid) as the phase that undergoes meiosis to produce "gametophytes" (typically haploid). We use the term gametophyte to encompass either a separate life stage that can undergo mitotic cell divisions before producing gametes and/or the gametes themselves (in animals, only the latter are relevant). Gametophytes may be monoecious (producing both eggs and pollen/sperm) or dioecious (specializing on one type of gamete). Sporophytes may also be dioecious, with separate males and females, if they produce only one type of gametophyte (egg-producing or pollen/sperm-producing).

The existence of separate sexes in the gametophytic and/or sporophytic phases affects the maintenance of genetic variation because different alleles can be favored in males and females (i.e., sexual conflict; Kidwell et al. 1977; Parker 1979; reviewed in Arnqvist and Rowe 2005) and because sex linkage (e.g., XY) considerably affects the conditions for a stable polymorphism (Rice 1984). In contrast to autosomal genes that are diploid during the sporophytic stages and haploid during the gametophytic stages, X-linked genes are diploid in sporophytic females but effectively haploid in sporophytic males (assuming a degenerate Y) and occur only in a portion of male gametophytes/gametes. Finally, Y-linked genes are always effectively haploid and occur only in males (gametophytic and sporophytic).

There is increasing evidence that a large number of loci are under some sort of sex-specific selection (e.g., Delph et al. 2010; Innocenti and Morrow 2010), and in plants many loci are known to be under haploid selection (Mascarenhas 1990; Borg et al. 2009). Although haploid expression seems much less pervasive in animals, some genes show a potential for haploid selection (Joseph and Kirkpatrick 2004). Whenever diploid selection differs between the sexes and haploid selection also occurs, the full model that we develop in this article, allowing sex and ploidy differences in selection, is required to determine whether a protected polymorphism will occur. In the following, we determine the conditions for the maintenance of a genetic polymorphism under the assumption of ploidally antagonistic selection and sex-specific selection under autosomal, X-, and Y-linked inheritance.

Methods

THE MODELS

The aim of this study is to identify conditions for a stable polymorphism between alleles A and a at a locus experiencing ploidally antagonistic selection. We consider the following three scenarios. (1) Autosomal genes: selection differs between male and female sporophytes (diploid) and between male and female gametophyes (haploid). Note that selective differences between male and female sporophytes might not be restricted to dioecious sporophytes, if selection acts differently within the male and female reproductive organs of monoecious sporophytes. As special cases, our model coincides with that studied by Ewing (1977) if selection does not differ between the sexes and by Gregorius (1982) if selection differs between the sexes only in the gametophytic stage. (2) X-linked genes: selection acts on genes that are diploid in sporophytic females but hemizygous (effectively haploid) in sporophytic males. This model also describes organisms with XO or with arrhenotokous (haplodiploid) sex determination. (3) Y-linked genes: selection acts on genes that are only found in male (Y-bearing) sporophytes and gametophytes. In all three models, selection during both the gamete and the adult phase is allowed to differ between the sexes, and we allow for sex-specific dominance, as described in Table 1. The fitness regime does not require sexually antagonistic selection within either ploidy phase, although such selection can be described by letting the selection coefficients differ in sign between the sexes (i.e., $t_m t_f < 0$ for sexually antagonistic selection in the gametophytic phase or $s_m s_f < 0$ in the sporophytic phase).

Results

Assuming nonoverlapping generations with all individuals in the population producing gametophytes at the same time regardless of their genotype, we derived recursion equations for each of the three models (Appendix). We then determined the conditions under which allele a could invade when rare and under which

Table 1. Fitness in gametophytes (v_i^j) and sporophytes (w_i^j) for genotype *i* in sex *j*. Fitness are given in terms of selection coefficients in gametophytes (t_j) and sporophytes (s_j), as well as dominance coefficients in sporophytes (h_j).

(A) Gametophytic phase			
	A	a	
Female:	$v_A^f=1-t_f$	$v_a^f=1$	
Male:	$v_A^m=1-t_m$	$v_a^m=1$	
(B) Sporophytic phase			
	AA	Aa	aa
Female:	$w_{AA}^f=1$	$w_{Aa}^f=1-h_f s_f$	$w_{aa}^f=1-s_f$
Male:	$w_{AA}^m=1$	$w_{Aa}^m=1-h_m s_m$	$w_{aa}^m=1-s_m$
	A	a	
Male (X or Y):	$w_A^m=1$	$w_a^m=1-s_m$	

allele A could invade when rare. If both conditions are satisfied, we conclude that a polymorphism can be maintained by selection (see Table 2 for overview).

We visualize the implications of our analytical results using graphs. Unless otherwise specified, we use the following default parameter values: $t_m = 0.3$, $t_f = 0$, $s_m = 0.1$, and $h_m = 0.1$. These specific parameters were motivated by the idea that the gametophyte most likely to disperse is the male gametophyte, which is thus more likely to be exposed to stronger selection.

MODEL 1: AUTOSOMAL GENE

Allele a invades when rare if:

$$\frac{1}{2}\frac{1-h_m s_m}{1-t_m}+\frac{1}{2}\frac{1-h_f s_f}{1-t_f} > 1, \quad (1a)$$

Table 2. Types of selection needed to maintain stable polymorphism with different forms of linkage.

Linkage scenario	Selection type
Autosomal	Strict overdominance
	Opposing selection between males and females in haploid or diploid phase (sexually antagonistic selection)
	Opposing selection between haploid and diploid phase without sex differences (ploidally antagonistic selection)
	Selective differences between sexes and phases (especially with negative sex-by-ploidy interactions)
X-linked	Similar to autosomal conditions
Y-linked	No stable polymorphism possible

which requires that the fitness of heterozygous diploids relative to resident AA diploids ($1 - h_j s_j$) is greater than the fitness of resident A haploids relative to a haploids ($1 - t_j$), in at least one of the sexes. On the other hand, allele A invades when rare if:

$$\frac{1}{2}\frac{(1-h_m s_m)(1-t_m)}{1-s_m}+\frac{1}{2}\frac{(1-h_f s_f)(1-t_f)}{1-s_f} > 1, \quad (1b)$$

which similarly requires that the fitness of heterozygous diploids relative to resident aa diploids ($(1 - h_j s_j)/(1 - s_j)$) is greater than the fitness of resident a haploids relative to A haploids ($1/(1 - t_j)$) in at least one of the sexes (Fig. 1).

Besides strict overdominance, several different scenarios can generate conditions under which both inequalities are satisfied and a polymorphism will be maintained. We first focus on several special cases that relate to previous models. Selection can be absent in the haploid phase ($t_j = 0$) but sexually antagonistic in the diploid phase ($s_m s_f < 0$), as long as the dominance coefficients are small enough that both (1a) and (1b) are satisfied, which basically ensures that the fitness of heterozygotes relative to homozygotes is greater than one when averaged across the sexes (Kidwell et al. 1977). Similarly, selection in the diploid phase can be absent or equal between sexes ($s_j = 0$ or $s_m = s_f$), but sexually antagonism in the haploid phase ($t_m t_f < 0$) can maintain polymorphism, as long as equation (A3) in the Appendix is satisfied (Gregorius 1982; Fig. 2). These two scenarios fit the classical theory of intralocus sexual conflict (Trivers 1972; Kidwell et al. 1977; Parker 1979; Arnqvist and Rowe 2005).

Alternatively, selection can favor opposite alleles in haploid and diploid phases but not be sex-specific. This is the scenario explored by Ewing (1977). Setting

$s_j = s$, $t_j = t$ and $h_j = h$, equation (1) simplifies and indicates that allele a will invade when

$$\frac{1-hs}{1-t} > 1, \quad (2a)$$

while allele A invades when

$$\frac{(1-hs)(1-t)}{1-s} > 1. \quad (2b)$$

Taken together, ploidal antagonistic selection (with s, $t > 0$) maintains a polymorphism only when dominance is sufficiently low:

$$h < \min\left[\frac{t}{s}, \frac{s-t}{s\,(1-t)}\right]. \quad (3)$$

This condition makes sense: polymorphism can be maintained only when the allele that is favored in the haploid stage is largely sheltered from selection against it in diploid heterozygotes (h small).

Equation (1) allows us to generalize beyond these special cases and consider how selective differences between the sexes and ploidy levels contribute to the maintenance of polymorphism.

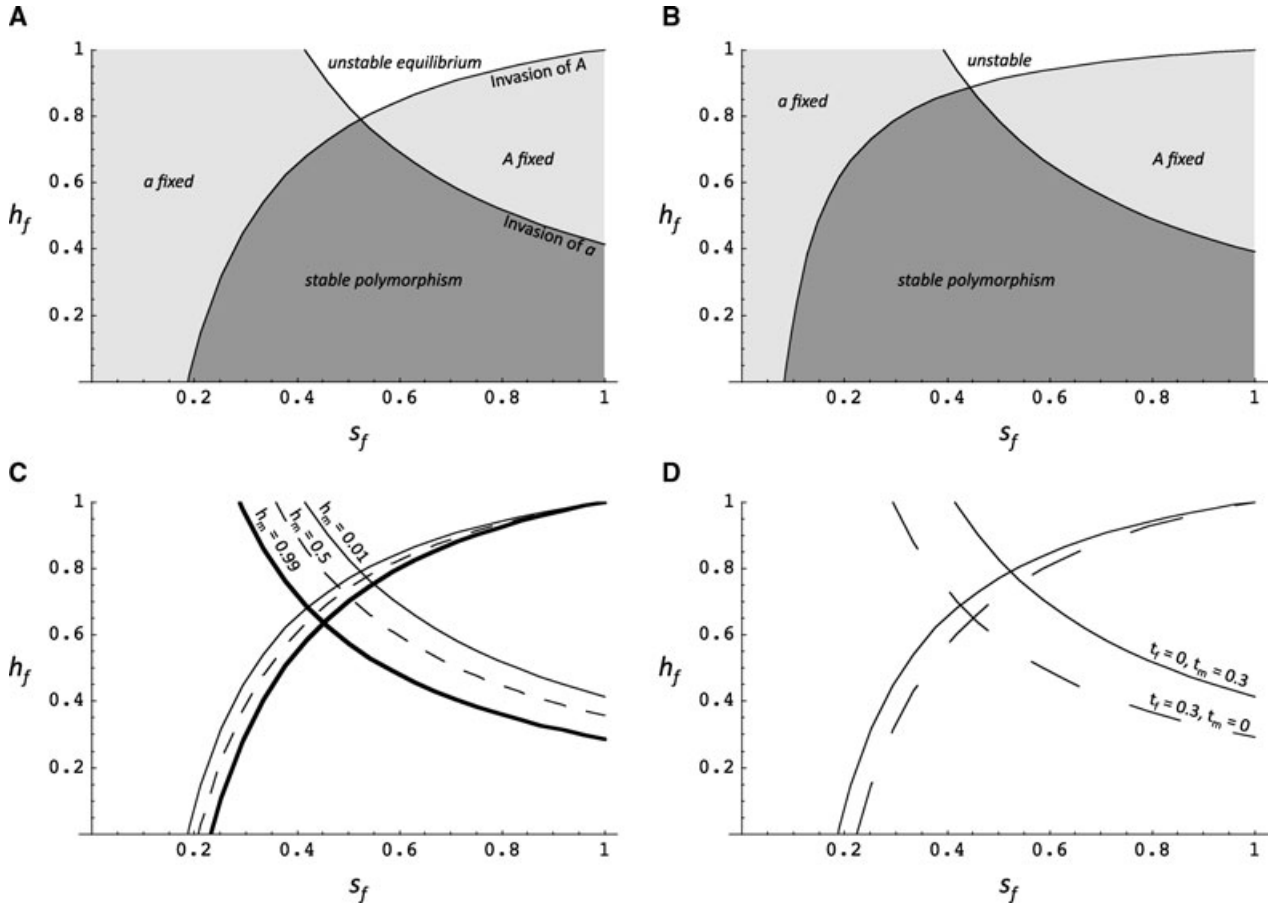

Figure 1. **Conditions favoring the maintenance of polymorphism under ploidally antagonistic selection for an autosomal gene (Model 1). (A) Results under the default parameters: $t_m = 0.3$, $t_f = 0$, $s_m = 0.1$, and $h_m = 0.1$. Curves for invasion of *a* and invasion of *A* define four regions: allele *a* goes to fixation, allele *A* goes to fixation, a stable polymorphism exists between *a* and *A* and an unstable equilibrium exists between alleles *a* and *A* (i.e., either allele *a* or *A* may fix depending on their initial frequency). (B) Allowing stronger selection in males at the sporophytic phase ($s_m = 0.25$) makes it easier to maintain a stable polymorphism in this case (with h_m small). (C) Effects of variation in parameter h_m: increasing values of h_m decrease the parameter space supporting a polymorphism, because of the reduced fitness of heterozygous males. (D) Selection acting on female gametophytes ($t_m = 0$, $t_f = 0.3$; dashed curves) narrows the condition for a stable polymorphism compared to the default parameter setting assuming selection on male gametophytes (solid curves).**

We first assume that there are slight sex differences in selection, such that $t_f = t + \delta_t$, $s_f = s + \delta_s$, and $h_f = h + \delta_h$ in females and $t_m = t - \delta_t$, $s_m = s - \delta_s$, and $h_m = h - \delta_h$ in males, where all of the δ_i terms are small. The two inequalities (1) required for polymorphism then become

$$\frac{1-hs}{1-t} + \frac{\delta_t^2(1-hs)}{(1-t)^3} - \frac{\delta_h\delta_s}{(1-t)} - \frac{\delta_t\delta_h s}{(1-t)^2} - \frac{\delta_t\delta_s h}{(1-t)^2} > 1, \tag{4a}$$

and

$$\frac{(1-hs)(1-t)}{(1-s)} + \frac{\delta_s^2(1-h)(1-t)}{(1-s)^3} - \frac{\delta_h\delta_s(1-t)}{(1-s)^2} + \frac{\delta_t\delta_h s}{(1-s)} - \frac{\delta_t\delta_s(1-h)}{(1-s)^2} > 1, \tag{4b}$$

to leading order. Adding sex-specific selection in the haploid phase or the diploid phase makes these inequalities easier to satisfy (the δ_t^2 and δ_s^2 terms, respectively), as we would expect from sexual conflict theory. Sex-specific selection in the diploid phase is especially conducive to the maintenance of polymorphism if δ_h (sex difference in dominance) and δ_s (sex difference in diploid selection) are opposite in sign, because the allele that is selected against in diploids is largely masked in the sex in which it is most deleterious and is better protected from loss. The $\delta_t\delta_h$ term makes opposite contributions to the invasion of the *a* allele ($-\delta_t\delta_h$), which is facilitated if the sporophytic sex in which *a* is better masked (lower *h*) gives rise to gametophytes of that sex in which *a* is more strongly favored (higher *t*) so that δ_h and δ_t are opposite in sign. This is to be compared with the invasion of the *A* allele ($+\delta_t\delta_h$), which is aided if the sporophytic sex in which *A* is more dominant (lower *h*) gives rise to gametophytes of that sex in which *A* is less strongly selected against (lower *t*). The most intriguing result is that polymorphism is most likely to be maintained if the sex

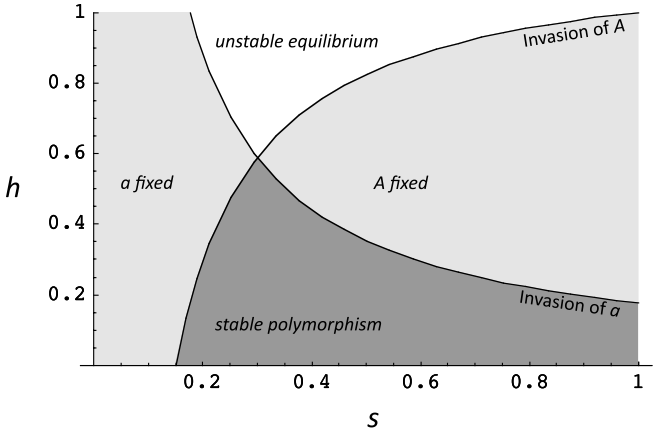

Figure 2. Sex-differences in selection in the diploid sporophytic stage are not required to maintain polymorphism (parameters: $t_m = 0.3$, $t_f = 0$, $s_j = s$, $h_j = h$).

differences in selection act in opposite directions in haploids and diploids ($-\delta_t\delta_s$ terms). That is, ploidy-by-sex interactions promote the maintenance of variation if an allele that is more strongly favored in female diploids than male diploids is more weakly selected against in female haploids than male haploids, or vice versa (Fig. 3). Essentially, even when selection acts in the same direction in both sexes, negative ploidy-by-sex interactions ensure that each allele spends less time in the sex in which it has the lowest overall fitness, allowing ploidally antagonistic selection to maintain polymorphism more readily. Inspection of equation (1) indicates that this is generally true: sex-specific selection that tugs in opposite directions in haploids and diploids is more conducive to the maintenance of polymorphism. Such negative ploidy-by-sex interactions ensure that when the denominators of the terms in equation (1) are small, the numerators are not also small.

MODEL 2: X-LINKED GENES

For an X-linked locus, allele a invades when rare if

$$\frac{1}{2}\,\frac{1-h_f s_f}{1-t_f}\left\{1+\frac{1-s_m}{1-t_m}\right\} > 1 \qquad (5a)$$

while allele A invades when rare if

$$\frac{1}{2}\,\frac{(1-h_f s_f)(1-t_f)}{1-s_f}\left\{1+\frac{1-t_m}{1-s_m}\right\} > 1 \qquad (5b)$$

Only if both conditions are met are both alleles protected from loss when rare, ensuring a polymorphism. Increasing any one of the selection coefficients (s_m, s_f, t_m, or t_f) makes one condition easier to satisfy and the other harder to satisfy, such that the net result depends on the combination of parameters under consideration. Decreasing the dominance coefficient in females (h_f), however, always makes it easier to maintain a polymorphism, because the alleles favored in haploids are better sheltered from negative selection in diploids.

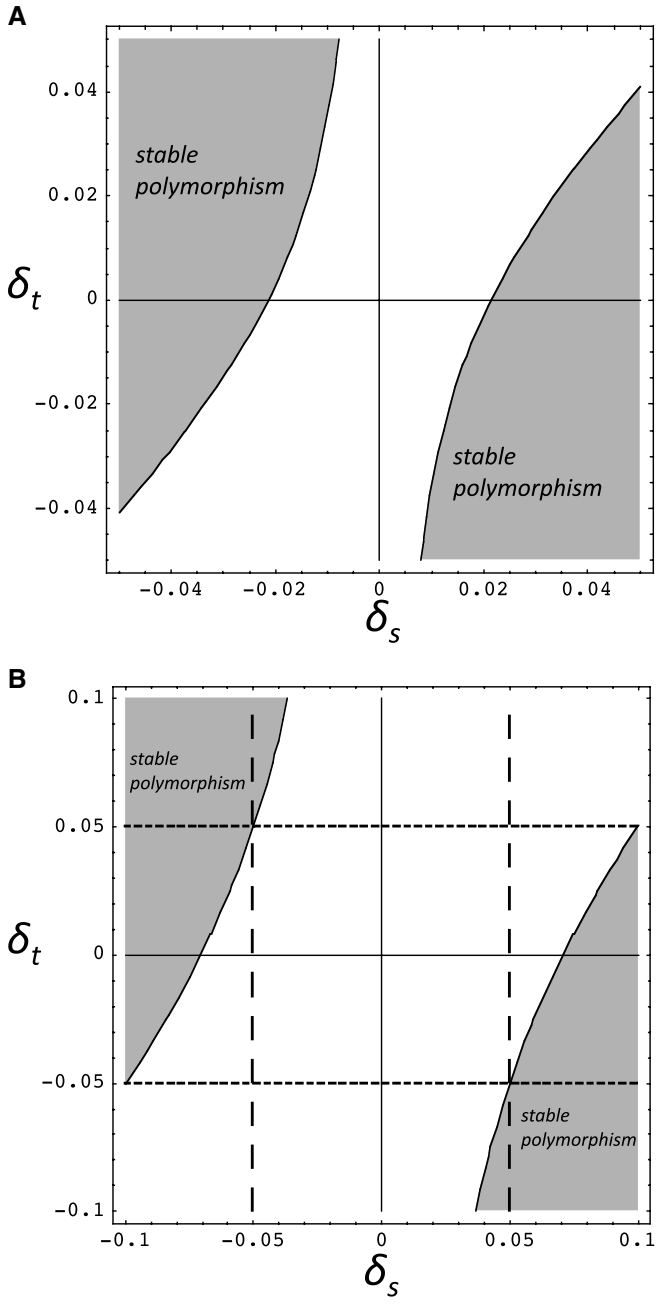

Figure 3. Negative ploidy-by-sex interactions facilitate the maintenance of polymorphism. Parameters with ploidally antagonistic selection were chosen ($t = 0.05$, $s = 0.05$) such that polymorphism would not be maintained in the absence of sex differences in selection (origin on graphs) but could be with sex differences in selection, δ_t and δ_s (see eq. 4). (A) With strong masking ($h = 0.01$), sex-differences in selection allow the maintenance of polymorphism in the shaded areas even though selection is not sexually antagonistic in either ploidy phase (i.e., selection acts in the same direction in haploid males and females and in the opposite direction in diploid males and females because δ_t and δ_s remain smaller in magnitude than t and s throughout this panel). (B) With weaker masking ($h = 0.1$), polymorphism can be maintained only when selection is sexually antagonistic, acting in opposite directions on

Note that the form of equation (5) with sex-linkage is slightly more complicated than the equivalent expression (1) for autosomal loci. In the autosomal case, the selection coefficients in males are separate from those in females (see eq. 1). This occurs because genotype frequencies are initially identical in male and female sporophytes before selection. Diploid selection then acts on male sporophytes followed by haploid selection in their gametophytes (which are all male), with the same occurring in females. Consequently, what matters is that rare alleles are favored, on average, across the two sexes. In contrast, genotypes differ even in newly formed sporophytes in the X-linked case because of the hemizygosity of male sporophytes, which always inherit their X from female gametophytes.

Comparing the conditions for maintenance of a polymorphism for an autosomal gene (1) with those for an X-linked gene (5) shows that it is easier for allele a to invade when rare in the autosomal case if

$$\frac{(1-h_m s_m)}{(1-h_f s_f)} > \frac{(1-s_m)}{(1-t_f)} \tag{6a}$$

and for the A allele to invade when rare if

$$\frac{(1-h_m s_m)}{(1-h_f s_f)} > \frac{(1-t_f)}{(1-s_f)}. \tag{6b}$$

The more fit the heterozygous males are in the autosomal case (lower h_m), the more readily autosomal genes will maintain polymorphism compared to X-linked genes. Increasing the strength of ploidally antagonistic selection (increasing t_f) makes one of the conditions easier to satisfy in the autosomal model and the other one harder. Thus, in general, one cannot say a priori whether autosomal or X-linkage will be more conducive to the maintenance of polymorphism with ploidally antagonistic selection (Fig. 4). We note that this is true also for sexually antagonistic selection (Fry 2010).

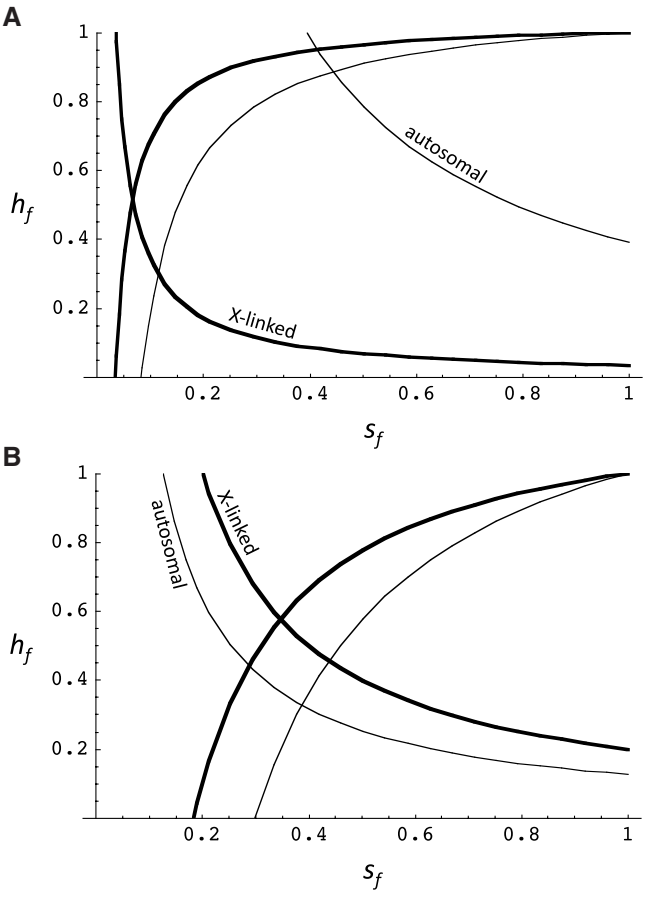

Figure 4. Comparing the maintenance of polymorphism for autosomal (thin) and X-linked (thick) genes. (A) Using the default parameters ($t_m = 0.3$, $t_f = 0$, $s_m = 0.1$, and $h_m = 0.1$), the conditions for a stable polymorphism are more restrictive for X-linked genes than for autosomal genes. (B) The conditions for a polymorphism are more restrictive for autosomal genes, however, if selection is stronger on female gametophytes than on male gametophytes (i.e., $t_m = 0$, $t_f = 0.3$) and on male sporophytes ($s_m = 0.25$ and $h_m = 0.99$).

MODEL 3: Y-LINKED GENES

For a Y-linked locus, allele a invades when rare if

$$\frac{1-s_m}{1-t_m} > 1 \tag{7a}$$

whereas allele A invades when rare if

$$\frac{1-t_m}{1-s_m} > 1. \tag{7b}$$

One, and only one, of these conditions can be satisfied, demonstrating that ploidally antagonistic selection cannot maintain polymorphism for Y-linked genes. One allele always has a higher fitness, when averaged across the gametophytic and sporophytic phases, and this allele goes to fixation.

Figure 3. male and female gametophytes (large δ_t) or on male and female sporophytes (large δ_s). The dashed horizontal lines and dashed vertical lines indicate the regions outside of which selection becomes sexually antagonistic in the sporophytic phase and gametophytic phase, respectively. In both panels, negative ploidy-by-sex interactions (δ_t opposite in sign to δ_s; top left and bottom right corners) are more conducive to the maintenance of polymorphism than positive interactions.

Discussion

Ploidally antagonistic selection can maintain polymorphism, but whether it does so depends on the nature of ploidy-by-sex interactions and the sex linkage of the genes under selection.

Polymorphism can be protected from the fixation of either allele with ploidally antagonistic selection at autosomal or X-linked genes, with or without sex differences in selection, but not at Y-linked genes. The fact that allelic variation cannot be maintained for Y-linked genes is unsurprising because the alleles are effectively under haploid selection that varies over time, as the alleles pass through gametophytic and sporophytic stages, and whichever allele has the highest geometric mean fitness is expected to fix (Dempster 1955). In contrast, haploid selection only in the gametophytic phase can maintain a polymorphism if there is sexually antagonistic selection (Gregorius 1982); in this case, selection in the two sexes mimics spatially varying selection in two patches, which can maintain a polymorphism even in a haploid model (Levene 1953; Strobeck 1979). Our models show that, in addition to the previously described scenarios of ploidally antagonistic selection (Ewing 1977) and sexually antagonistic selection at the haploid stage (Gregorius 1982), genetic variation can be maintained if selection acts differentially during the two phases (haploid and diploid) and the two sexes (males and females). Interestingly, we have shown that, with ploidally antagonistic selection, introducing selective differences between the sexes in either the haploid or diploid phase can facilitate the maintenance of polymorphism (see eq. 4), even when only the strength—and not the direction—of selection differs between the sexes. Facilitation occurs whenever selection exhibits negative ploidy-by-sex interactions, where the sporophyte sex in which allele *A* is more strongly favored gives rise to gametophytes (of the same sex) in which allele *a* is less strongly favored compared to the other sex. Negative ploidy-by-sex interactions also promote divergence in allele frequencies between the two sexes, such that each allele is better protected from loss in one of the sexes (see eq. 4 and Fig. 3). Finally, even in standard cases of sexually antagonistic selection among diploids, our full model is necessary to determine whether there is a protected polymorphism when selection also acts in the haploid phase. Below, we discuss the implications of our models in more detail and put them into a biological context.

BIOLOGICAL ASPECTS OF THE MODELS AND PREDICTIONS

Model 1—autosomal genes

Any genes active during pollen development (according to some studies up to 60%; see Mascarenhas 1990; Borg et al. 2009 for review) and expressed also in tissues of the sporophytes could be under ploidally antagonistic selection. Empirical evidence for the positive effect of pollen selection for the fitness of subsequent sporophyte generations is abundant (see Hormaza and Herrero 1992 for review). Haploid selection has been successfully used in agriculture to obtain plants that are more suitable for rearing under specific conditions, because selection on pollen is more effective and time and space efficient than selection on diploid plants. Such evidence includes selection for increased resistance, as in the tomato *Solanum lycopersicum*, where pollen were selected for increased temperature resistance (Zamir et al. 1982; Zamir and Vallejos 1983) and in maize *Zea mays*, where pollen selection was very effective in obtaining herbicide resistance in subsequent sporophyte generations (Frascaroli and Songstad 2001). Similarly, herbicide tolerance selected in maize at the haploid pollen level resulted in herbicide tolerance of the diploid sporophytes (Frascaroli et al. 1994). Studies quantifying the strength and direction of selection in haploid and diploid phases are much less common. A study of selection on body size in haploid and diploid kelp (*Lessonia nigrescens*) found statistical support only for selection in diploids (Martínez and Santelices 1998).

Additional evidence for ploidally antagonistic selection is available from studies investigating sex differences in selection across the life cycle. For example, in a study of selection on the phosphoglucoisomerase locus in the annual plant *Clarkia unguiculata* (Travers and Mazer 2001), an allele favored in male pollen tube growth was shown to have antagonistic effects on female fertility in diploids. This example exhibits negative ploidy-by-sex interactions, where selection acts primarily on male gametophytes and on female sporophytes to favor different alleles. Our work shows that this form of selection is more conducive to the maintenance of polymorphism than if selection was antagonistic in only one sex or in only one ploidy phase. Similarly, in the monkeyflower *Mimulus gutatus*, a meiotically driven allele *D* favored in female meiosis (akin to haploid selection) is associated with reduced male fertility in homozygous carriers of the allele (Fishman and Saunders 2008), again generating a negative ploidy-by-sex interaction. Other studies suggest that sex-specific effects on haploid and diploid selection may be fairly commonplace. In the red alga *Asparagopsis aramata*, for example, male gametophytes had the lowest secondary metabolites and a high nutrient content, making them most attractive to grazing by the sea hare *Aplysia parvula*, whilst the female gametophyte had the highest content of secondary metabolites (Vergés et al. 2008). In contrast, the diploid carposporophytes were least consumed. Similarly, a recent study in the self-compatible plant *Collinsia heterophylla* showed experimentally that pollen that induced earlier stigma receptivity caused a reduction in seed numbers (Lankinen and Kiboi 2007), and hence a potential conflict between male pollen and female fitness may exist in this species.

As mentioned in the Introduction, haploid selection in animals is thought to be minimal due to limited expression of the haploid genome (Joseph and Kirkpatrick 2004). Although many genes are expressed during spermatogenesis and are sperm specific, expression patterns within sperm remain effectively diploid due to the existence of cytoplasmic bridges among spermatids. Cytoplasmic bridges allow postmeiotic exchange of transcription

products between haploid spermatids. Nevertheless, any gene that is expressed in spermatids but not shared through cytoplasmic bridges is a potential candidate for haploid selection, one example being the *Spam1* gene expressed in mouse spermatids (Zheng et al. 2001; Martin-DeLeon et al. 2005). *Spam1,* an autosomal gene found in house mice *Mus musculus,* determines the egg-binding capacity of sperm, and disruption of its translation results in reduced fertility (Zheng et al. 2001), which exposes it to haploid selection. To date, gene expression at the gamete stage has been studied in only very few species, mostly *Drosophila* (Barreau et al. 2008; Vibranovski et al. 2010) and mice (Zheng et al. 2001; Martin-DeLeon et al. 2005) but next generation sequencing methods promise to rapidly expand our knowledge of postmeiotic gene expression also in nonmodel organisms. In the species investigated so far, a potential for haploid selection has been revealed by evidence for at least some postmeiotic gene expression. Future studies are needed to increase our understanding of how widespread haploid selection in animals might be.

Model 2—X-linked genes

With X-linkage or arrhenotoky, alleles under selection spend two-thirds of their time in females and one-third in males. Sex chromosomes have independently evolved several times in flowering plants and animals (Charlesworth 2002; Mank et al. 2006). In many cases, the Y (or W) chromosome shows signs of deterioration (Charlesworth 2002; Jamilena et al. 2008; Marais et al. 2008). However, sex chromosomes in plants (and particularly Y chromosomes) are considerably larger than autosomes (Parker 1990) and contain a suite of essential genes. There is some evidence that a male-specific gene responsible for pollen development (*MROS3*) is located on the X chromosome in *S. latifolia* without a fully functional homologue on the Y chromosome (Kejnovsky et al. 2001). Our results provide a framework for determining whether selection acting on such genes will maintain polymorphism, in those cases where selection acts at both the gametophytic and sporophytic stages.

In animals, despite the fact that many X-linked genes are known to be involved in spermatogenesis, postmeiotic transcription on the X chromosome may be limited as a result of meiotic sex chromosome inactivation (Lifschytz and Lindsley 1972; Graves 1995; Vallender and Lahn 2004; Ellis and Affara 2006; Vicoso and Charlesworth 2006), although this may vary across taxa (Parsch 2009).

Model 3—Y-linked genes

Y-linked genes spend their entire time in males and are effectively continuously in a haploid state. In dioecious flowering plants with chromosomal sex determination, the Y chromosome may be larger (e.g., 1.4 times larger) than the X chromosome and both sex chromosomes are substantially larger than any of the autosomes (Kejnovsky et al. 2001). In these plant species, the Y chromosome often contains many essential genes (e.g., Matsunaga et al. 1996; Matsunaga et al. 1997; Delichere et al. 1999). Our model suggests, however, that we would not expect selection to maintain polymorphism at these genes, even with ploidally antagonistic selection, because whichever allele has the higher geometric mean fitness will go to fixation.

PLOIDALLY ANTAGONISTIC SELECTION AND SEX DIFFERENCES IN SELECTION

Under the assumption of differential selection between the sexes during both diploid and haploid life stages, our analysis shows that polymorphism is most likely to be maintained with negative ploidy-by-sex interactions (eq. 4; Fig. 3). Specifically, an allele that favors success of gametophytes/gametes more in males than in females is more likely to remain polymorphic if it has a weaker deleterious effect on the resulting male offspring than on the resulting female offspring, which also promotes differences in allele frequency between males and females. Even without sexually antagonistic selection in either ploidy phase, intralocus conflict across ploidy levels is more likely to maintain variation when the sexes diverge in allele frequency. A suggested resolution of intralocus sexual conflict within a ploidy level is the occurrence of sex-limited gene expression (Rice 1984). Whether there has been selection to limit expression of genes to one or the other ploidy phase remains to be examined, although we note that ploidy-limited gene expression is pronounced in those flowering plants where the transcriptomes of pollen and vegetative tissues have been compared (e.g., Becker et al. 2003; Wei et al. 2010) and is virtually complete in animals (Joseph and Kirkpatrick 2004). That these phenomena might have evolved to resolve ploidally antagonistic selection is an intriguing possibility.

CONCLUSIONS

Our models show that polymorphism can be maintained under ploidally antagonistic selection and that sex differences in selection promote polymorphism, even if these sex differences are not so strong as to cause sexually antagonistic selection. Our results also show that negative ploidy-by-sex interactions, where stronger selection for an allele in diploids of one sex is coupled with weaker selection against that allele in haploids of the same sex, are especially conducive to the maintenance of genetic variation. Future empirical work is now needed to determine whether such sex differences in selection and interactions across ploidy stages facilitate the maintenance of genetic variation in natural systems.

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LITERATURE CITED

Arnqvist, G., and L. Rowe. 2005. Sexual conflict. Univ. Press, Princeton, NJ.

Barreau, C., E. Benson, E. Gudmannsdottir, F. Newton, and H. White-Cooper. 2008. Post-meiotic transcription in *Drosophila* testes. Development 135:1897–1902.

Barton, N., and P. Keightley. 2002. Understanding quantitative genetic variation. Nat. Rev. Genet. 3:11–21.

Becker, J. D., L. C. Boavida, J. Carneiro, M. Haury, and J. A. Feijó. 2003. Transcriptional profiling of Arabidopsis tissues reveals the unique characteristics of the pollen transcriptome. Plant Physiol. 133:713–725.

Borg, M., L. Brownfield, and D. Twell. 2009. Male gametophyte development: a molecular perspective. J. Exp. Bot. 60:1465–1478.

Charlesworth, D. 2002. Plant sex determination and sex chromosomes. Heredity 88:94–101.

Delichere, C., J. Veuskens, M. Hernould, N. Barbacar, A. Mouras, I. Negrutiu, and F. Moneger. 1999. SlY1, the first active gene cloned from a plant Ychromosome, encodes a WD-protein. EMBO J. 18:4169–4179.

Delph, L. F., A. M. Arntz, C. Scotti-Saintagne, and I. Scotti. 2010. The genomic architecture of sexual dimorphism in the dioecious plant *Silene latifolia*. Evolution 64:2873–2886.

Dempster, E. 1955. Maintenance of genetic heterogeneity. Cold Spring Harb. Symp. Quant. Biol. 20:25–32.

Ellis, P., and N. Affara. 2006. Spermatogenesis and sex chromosome gene content: an evolutionary perspective. Human Fertil. 9:1–7.

Ewing, E. 1977. Selection at the haploid and diploid phases: cyclical variation. Genetics 87:195–208.

Falconer, D., and T. Mackay. 1996. Introduction to quantitative genetics. Longman, Harlow.

Fishman, L., and A. Saunders. 2008. Centromere-associated female meiotic drive entails male fitness costs in monkeyflowers. Science 322:1559–1562.

Frascaroli, E., and D. Songstad. 2001. Pollen genotype selection for a simply inherited qualitative factor determining resistance to chlorsulfuron in maize. Theor. Appl. Genet. 102:342–346.

Frascaroli, E., S. Galletti, and P. Landi. 1994. Haplo-diploid gene expression and pollen selection for tolerance to acetochlor in maize. Theor. Appl. Genet. 88:780–784.

Fry, J. 2010. The genomic location of sexually antagonistic variation: some cautionary comments. Evolution 64:1510–1516.

Gillespie, J. 1984. Pleiotropic overdominance and the maintenance of genetic variation in polygenic characters. Genetics 107:321–330.

Graves, J. 1995. The origin and function of the mammalian Y chromosome and Y-borne genes—an evolving understanding. BioEssays 17:311–321.

Gregorius, H.-R. 1982. Selection in diplo-haplonts. Theor. Pop. Biol. 21: 289–300.

Harding, J. 1975. Models for gamete competition and self fertilization as components of natural selection in populations of higher plants. Pp. 243–255 *in* D. Mulcahy, ed. Gamete competition in plants and animals. Elsevier, New York.

Hill, W. 1982. Rates of change in quantitative traits from fixation of new mutations. Proc. Natl. Acad. Sci. USA 79:142–145.

Hormaza, J., and M. Herrero. 1992. Pollen selection. Theor. Appl. Genet. 83:663–672.

Hughes, J. S., and S. P. Otto. 1999. Ecology and the evolution of biphasic life cycles. Am. Nat. 154:306–320.

Innocenti, P., and E. H. Morrow. 2010. The sexually antagonistic genes of *Drosophila melanogaster*. Public Libr. Sci. Biol. 8:e1000335.

Jamilena, M., B. Mariotti, and S. Manzano. 2008. Plant sex chromosomes: molecular structure and function. Cytogenet. Genome Res. 120: 255–264.

Joseph, S. B., and M. Kirkpatrick. 2004. Haploid selection in animals. Trends Ecol. Evol. 19:592–597.

Kejnovsky, E., J. Vrána, S. Matsunaga, P. Soucek, J. Siroky, J. Dolezel, and B. Vyskot. 2001. Localization of male-specifically expressed *MROS* genes of *Silene latifolia* by PCR on flow-sorted sex chromosomes and autosomes. Genetics 158:1269–1277.

Kidwell, J. F., M. Clego, F. Stewart, and T. Prout. 1977. Regions of stable equilibria for models of differential selection in the two sexes under random mating. Genetics 85:171–183.

Krawetz, S. 2005. Paternal contribution: new insights and future challenges. Nat. Rev. Genet. 6:633–642.

Lankinen, A., and S. Kiboi. 2007. Pollen donor identity affects timing of stigma receptivity in *Collinsia heterophylla* (Plantaginaceae): a sexual conflict during pollen competition? Am. Nat. 170:854–863.

Levene, H. 1953. Genetic equilibrium when more than one ecological niche is available. Am. Nat. 87:331–333.

Lifschytz, E., and D. Lindsley. 1972. The role of X chromosome inactivation during spermatogenesis. Proc. Natl. Acad. Sci. USA 69:182–186.

Mable, B., and S. Otto. 1998. The evolution of life cycles with haploid and diploid phases. BioEssays 20:453–462.

Mackay, T. 2010. Mutations and quantitative genetic variation: lessons from *Drosophila*. Phil. Trans. R. Soc. Lond. B. 365:1229–1239.

Mank, J. E., D. E. L. Promislow, and J. C. Avise. 2006. Evolution of alternative sex-determining mechanisms in teleost fishes. Biol. J. Linn. Soc. 87: 83–93.

Marais, G., M. Nicolas, R. Bergero, P. Chambrier, E. Kejnovsky, F. Monéger, R. Hobza, A. Widmer, and D. Charlesworth. 2008. Evidence for degeneration of the Y chromosome in the dioecious plant *Silene latifolia*. Curr. Biol. 18:545–549.

Martin-DeLeon, P., H. Zhang, C. Morales, Y. Zhao, M. Rulon, B. Barnoski, H. Chen, and D. Galileo. 2005. *Spam1*-associated transmission ratio distortion in mice: elucidating the mechanism. Reprod. Biol. Endocrinol. 3:32.

Martínez, E., and B. Santelices. 1998. Selective mortaility on haploid and diploid microscopic stages of *Lessonia nigrescens* Bory (Phaeophyta, Laminariales). J. Exp. Mar. Biol. Ecol. 229:219–239.

Mascarenhas, J. 1990. Gene activity during pollen development. Ann. Rev. Plant Physiol. Plant Mol. Biol. 41:317–338.

Matsunaga, S., S. Kawano, H. Takano, H. Uchida, A. Sakai, and T. Kuroiwa. 1996. Isolation and developmental expression of male reproductive organ-specific genes in a dioecious campion, *Melandrium album* (*Silene latifolia*). Plant J. 10:679–689.

Matsunaga, S., S. Kawano, and T. Kuroiwa. 1997. MROS1, a male stamen-specific gene in the dioecious campion *Sliene latifolia* is expressed in mature pollen. Plant Cell Physiol. 38:499–502.

Parker, G. A. 1979. Sexual selection and sexual conflict. Pp. 123–166 *in* M. Blum, and N. Blum, eds. Sexual selection and reproductive competition in insects. Blackwell Scientific, Oxford.

Parker, J. 1990. Sex-chromosome and sex differentiation in flowering plants. Chromosomes Today 10:187–198.

Parsch, J. 2009. X Chromosome: expression and escape. PLoS Genet. 5:e1000724.

Rice, W. R. 1984. Sex chromosomes and the evolution of sexual dimorphism. Evolution 38:735–742.

Roe, K. 1975. Origin of the alternation of generations in plants: reconsideration of the traditional theories. Biologist 57:1–13.

Sari-Gorla, M., E. Ottaviano, E. Frascaroli, and P. Landi. 1989. Herbicide-tolerant corn by pollen selection. Sex Plant Reprod. 2:65–69.

Scudo, F. 1967. Selection on both haplo and diplophase. Genetics 56:693–704.

Searcy, K., and D. Mulcahy. 1985a. Pollen selection and the gametophytic expression of metal tolerance in *Silene dioica* (Caryophyllaceae) and *Mimulus guttatus* (Scrophulariaceae). Am. J. Bot. 72:1700–1702.

Searcy, K., and D. Mulcahy. 1985b. Pollen tube competition and selection for metal tolerance in *Silene dioica* (Caryophyllaceae) and *Mimulus guttatus* (Scrophulariaceae). Am. J. Bot. 72:1695–1699.

Strasburger, E. 1894. The periodic reduction of the number of the chromosomes in the life-history of living organisms. Ann. Bot. 8:281–316.

Strobeck, C. 1979. Haploid selection with *n* alleles in *m* niches. Am. Nat. 113:439–444.

Travers, S., and S. Mazer. 2001. Trade-offs between male and female reproduction associated with allozyme variation in phosphoglucoisomerase in an annual plant (*Clarkia unguiculata* : Onagraceae). Evolution 55:2421–2428.

Trivers, R. L. 1972. Parental investment and sexual selection. Pp. 136–179 *in* B. Campbell, ed. Sexual selection and the descent of man 1871–1971. Aldine-Atherton, Chicago.

Trotter, M., and H. Spencer. 2007. Frequency-dependent selection and the maintenance of genetic variation: exploring the parameter space of the mutliallelic pairwise interaction model. Genetics 176:1729–1740.

Valero, M., S. Richerd, V. Perrot, and C. Desombe. 1992. Evolution of alternation of haploid and diploid phases in life cycles. Trends Ecol. Evol. 7:25–29.

Vallender, E., and B. Lahn. 2004. How mammalian sex chromosomes acquired their peculiar gene content. BioEssays 26:159–169.

Vergés, A., N. Paul, and P. Steinberg. 2008. Sex and life-history stage alter herbivore response to a chemically defended red alga. Ecology 89:1334–1343.

Vibranovski, M., D. Chalopin, H. Lopes, M. Long, and T. Karr. 2010. Direct evidence for postmeiotic transcription during *Drosophila melanogaster* spermatogenesis. Genetics 186:431–433.

Vicoso, B., and B. Charlesworth. 2006. Evolution on the X chromosome: unusual patterns and processes. Nat. Rev. Genet. 7:645–653.

Wei, L. Q., W. Y. Xu, Z. Y. Deng, Z. Su, Y. B. Xue, and T. Wang. 2010. Genome-scale analysis and comparison of gene expression profiles in developing and germinated pollen in *Oryza sativa*. BMC Genomics 11:338.

Yanchukov, A. 2009. One- and two-locus population models with differential viability between sexes: parallels between haploid parental selection and genomic imprinting. Genetics 182:1117–1127.

Zamir, D., S. Tanksley, and R. Jones. 1982. Haploid selection for low temperature tolerance of tomato pollen. Genetics 101:129–137.

Zamir, D., and E. Vallejos. 1983. Temperature effects on haploid selection of tomato microscopres and pollen grains. Pp. 335–342 *in* D. Mulcahy, and E. Ottaviano, eds. Pollen: biology and implications for plant breeding. Elsevier, New York.

Zheng, Y., X. Deng, and P. Martin-DeLeon. 2001. Lack of sharing of *Spam1* (Ph-20) among mouse spermatids and transmission ratio distortion. Biol. Reprod. 64:1730–1738.

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Appendix: Recursion equations

MODEL 1: AUTOSOMAL GENES

We census the population at the beginning of the haploid gametophytic phase, at which point alleles A and a are present at frequencies p_{m0} and q_{m0} in males and at frequencies p_{f0} and q_{f0} in females (where $p_{j0} + q_{j0} = 1$). After selection among gametophytes, the frequencies of allele A in male gametes ($p_{m1} = 1 - q_{m1}$) and female gametes ($p_{f1} = 1 - q_{f1}$) become:

$$p_{m1} = \frac{p_{m0} v_A^m}{p_{m0} v_A^m + q_{m0} v_a^m} \tag{A1a}$$

$$p_{f1} = \frac{p_{f0} v_A^f}{p_{f0} v_A^f + q_{f0} v_a^m}. \tag{A1b}$$

Random mating between male and female gametes then occurs, followed by selection in the diploid phase. Finally, the diploids undergo meiosis to produce haploid gametophytes, giving us the allele frequencies in the next generation

$$p'_{m0} = \frac{p_{m1} p_{f1} w_{AA}^m + \frac{1}{2}(p_{m1} q_{f1} + p_{f1} q_{m1}) w_{Aa}^m}{W_{mS}} \tag{A2a}$$

$$p'_{f0} = \frac{p_{m1} p_{f1} w_{AA}^f + \frac{1}{2}(p_{m1} q_{f1} + p_{f1} q_{m1}) w_{Aa}^f}{W_{fS}}, \tag{A2b}$$

where the denominators describe the mean diploid fitness in males

$$W_{mS} = p_{m1} q_{f1} w_{AA}^m + (p_{m1} q_{f1} + p_{f1} q_{m1}) w_{Aa}^m + q_{m1} q_{f1} w_{aa}^m \tag{A3a}$$

and in females:

$$W_{fS} = p_{m1} q_{f1} w_{AA}^f + (p_{m1} q_{f1} + p_{f1} q_{m1}) w_{Aa}^f + q_{m1} q_{f1} w_{aa}^f. \tag{A3b}$$

Gregorius (1982) considered a special case of this model where selection in the diploid phase does not differ among sexes ($w_i^f = w_i^m$). In this case, equations (A2a) and (A2b) are equivalent, and there is no difference between the frequency of A in male and female gametophytes ($p_{m0} = p_{f0}$). As shown by Gregorius (1982), the dynamics are then equivalent to those observed in a "standard" diploid model (i.e., where selection operates only in the diploid phase, there are no selective differences between males and females, and mating is random), as long as we use the following compound fitness for the diploid genotypes:

$$\begin{aligned} Fitness(AA) &= v_A^m \; v_A^f \; w_{AA} \\ Fitness(Aa) &= \frac{1}{2}\left(v_A^m \; v_a^f + v_a^m \; v_A^f\right) \; w_{Aa} \\ Fitness(aa) &= v_a^m \; v_a^f \; w_{aa}. \end{aligned}$$

The above compound fitness can be thought of as the geometric mean fitness of a genotype across the alternating generations, where male and female gametophytes are paired at random into "virtual diploids." Ploidally antagonistic selection, where an allele associated with a high homozygous diploid fitness lowers fitness in haploids (e.g., w_{AA} high and v_A^j low), thus acts in essentially the same manner as reducing the fitness of homozygotes, *Fitness*(AA) and *Fitness*(aa), in the standard diploid model, with the result that

the system is more likely to display heterozygous advantage. That is, *AA* and *aa* genotypes experience greater variation in fitness over the life cycle than *Aa* genotypes under ploidally antagonistic selection, resulting in a lower geometric mean fitness. Similarly, sexually antagonistic selection, where different alleles are favored in male and female gametophytes (e.g., v_A^m and v_a^f high), acts essentially in the same manner as increasing the fitness of heterozygotes, *Fitness*(*Aa*), again promoting heterozygous advantage. Gregorius (1982) observed that even with selection acting only in haploids ($w_i^j = 1$), polymorphism is maintained as long as there is sexually antagonistic selection with $v_A^m v_A^f$ and $v_a^m v_a^f$ both less than $(v_A^m v_a^f + v_a^m v_A^f)/2$. In terms of selection coefficients, this requires that the haploid selection coefficients satisfy:

$$t_f t_m < (t_f + t_m)/2 < 0. \tag{A3}$$

In this case, sexually antagonistic selection in the haploid phase is the mechanism maintaining genetic variation, and ploidally antagonistic selection is absent. A similar result is found with imprinting in the diploid phase (Yanchukov 2009), which mimics haploid selection.

The main result of Gregorius (1982) showing equivalence to the standard diploid model no longer holds, however, when there are selective differences among the sexes in the diploid phase. With sex-specific diploid selection, the frequencies of allele *A* in male and female gametophytes are no longer equal (see eq. A2), and the model requires that we keep track of two variables (here, p_{m0} and p_{f0}). Thus, regardless of how we choose diploid fitness (assuming these to be constant and mating to be random), the general model of sex-specific selection in both haploid and diploid phases is not equivalent to the standard diploid model.

MODEL 2: X-LINKED GENES

Allele frequencies after selection at the haploid gametophytic stage continue to be described by equation (A1). The only difference is that half of the male gametophytes carry the Y chromosome and do not experience selection with respect to the *A*/*a* alleles. Restricting our attention to the X-bearing male gametophytes, however, selection continues to act according to (A1a). After mating and selection at the diploid sporophytic stage, meiosis generates a new generation of haploid gametophytes, with the frequency of *A* among X-bearing male gametophytes in the next generation given by:

$$p'_{m0} = \frac{p_{f1} w_A^m}{p_{f1} w_A^m + q_{f1} w_a^m}. \tag{A5}$$

The frequency of *A* among female gametophytes is again given by (A2b). Notice that male gametophytes are produced by male sporophytes (so A5 depends only on the fitness of male sporophytes), but the X chromosome passed on from this male sporophyte to its X-bearing male gametophytes must have been inherited from a female gamete (so A5 depends only on the allele frequencies in female gametophytes). This model also describes other genetic systems where the male sporophyte effectively inherits a haploid genome only from the female gametophyte, including XO genetic systems and arrhenotokous (haplodiploid) sexual systems.

MODEL 3: Y-LINKED GENES

As only male gametophytes carry the Y chromosome, selection in the gametophytic stage is given by equation (A1a). Selection subsequently acts in males at the diploid sporophytic stage, leading to a frequency of Y in the next generation of male gametophytes given by

$$p'_{m0} = \frac{p_{m1} w_A^m}{p_{m1} w_A^m + q_{m1} w_a^m}. \tag{A6}$$

ANALYSES

The above models are fully described by the two recursion equations relating the allele frequencies in male and female gametophytes in the next generation, p'_{m0} and p'_{f0}, to those in the previous generation, p_{m0} and p_{f0}. We determined the conditions under which a protected polymorphism must exist by finding when allele *a* could invade when rare and when *A* could invade when rare. Specifically, we determined when the leading eigenvalue of the 2×2stability matrix evaluated at $q_{m0} = q_{f0} = 0$ was greater than one (*a* can invade), and similarly, when the leading eigenvalue of the 2×2 stability matrix evaluated at $p_{m0} = p_{f0} = 0$ was greater than one (*A* can invade). It should be noted that throughout this article, our focus is only on cases where there is a protected polymorphism, so that both *A* and *a* alleles can invade when rare. It is possible that there are additional locally stable polymorphic equilibria alongside a locally stable fixation state (see, e.g., Kidwell et al. 1977), but we have not analyzed the conditions under which this occurs.