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MAINTENANCE OR LOSS OF GENETIC VARIATION UNDER SEXUAL AND PARENTAL ANTAGONISM AT A SEX-LINKED LOCUS

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An intralocus genetic conflict occurs when a locus is selected in opposing directions in different subsets of a population. Populations with two sexes have the potential to host a pair of distinct intralocus conflicts: sexual antagonism and parental antagonism. In this article, we examine the population genetic consequences of these conflicts for X-linked genes. Both conflicts are capable of maintaining genetic variation in a population, but to different degrees. For weak sexual antagonism, the X chromosome has a higher opportunity for polymorphism than the autosomes. For parental antagonism, there is a very limited opportunity for polymorphism on the X chromosome relative to autosomes or to sexual antagonism. X-linkage introduces an asymmetry in the inheritance and expression of sexually and parentally antagonistic genes that leads to a biased fixation of alleles with certain effects. We find little support for the commonly held intuition that the X chromosome should be biased toward fixing female-beneficial alleles. Contrary to this intuition, we find that the X chromosome is biased toward fixation of male-beneficial alleles for much of the range of dominance. Additionally, we find that the X chromosome is more favorable to the fixation of alleles that are beneficial when maternally derived.

KEY WORDS: Chromosomal evolution, genetic variation, selection—sexual, sexual conflict.

What's good for the goose may not be good for the gander. This twist on an old proverb captures the notion of sexual antagonism. Traits that confer high fitness on geese (females) may confer low fitness on ganders (males), and vice versa. Previous theoretical studies have shown that sex-specific selection causes evolutionary dynamics that differ from sex-independent selection, both for autosomes (Owen 1953; Bodmer 1965; Kidwell et al. 1977) and the X chromosome (Rice 1984).

In an anisogamous diploid population, such as our geese, alleles can be maternally derived (from an egg) or paternally derived (from a sperm). Parental antagonism occurs when what is optimal in a maternally derived context is suboptimal in a paternally derived context, or vice versa. Such conflicting selection can arise when individuals interact with asymmetric kin, which are individuals for whom the relatedness of the maternally derived allele and the paternally derived allele of the focal individual differ (Haig 1997). When these two coefficients of relatedness differ, the inclusive fitness of an allele also differs in a manner that depends on its parent of origin. Previous theoretical studies have demonstrated that parent of origin-specific selection gives rise to dynamics that are different from selection schemes that do not consider parent of origin effects on fitness (Spencer 2000; Úbeda and Haig 2004).

In this article, we explore the consequences of these two selection schemes on X-linked loci. We focus on two questions. First, can sexual and parental antagonism maintain polymorphism on the X chromosome and if so, how much? Second, does X-linkage introduce a bias for resolving a conflict in favor of one sex or one parent of origin? The answer to the first question is relevant to studies that set out to measure the amount of standing

fitness variation in a population. The answer to the second question potentially informs studies that examine the evolution of sex chromosomes and genetic architecture.

Rice (1984) showed previously that polymorphism for sexually antagonistic alleles may be easier to achieve on the X chromosome than on the autosomes given the right level of dominance. Specifically, X-linkage predisposes to the invasion of female-beneficial dominant alleles and male-beneficial recessive alleles. Rice's (1984) results are specific to particular levels of dominance and are not a general statement about the extent of polymorphism of X-linked genes. Nevertheless, these results have been used as the theoretical basis for predicting that X chromosomes should be enriched for sexually antagonistic variation (Gibson et al. 2002; Connallon and Knowles 2005; Pischedda and Chippindale 2006; Foerster et al. 2007), a prediction that has been supported by empirical studies (Gibson et al. 2002; Pischedda and Chippindale 2006; Foerster et al. 2007). Other theoretical analyses, however, have come to the opposite conclusion, that the opportunity for sexually antagonistic polymorphism is smaller for X-linked loci than for autosomal loci (Curtsinger 1980; Hedrick and Parker 1997).

The X chromosome has also been proposed to favor genes that benefit females at the expense of males. This proposal originates from an oft-repeated intuition that X chromosomes should be biased toward fixation of female-beneficial alleles because X-linked alleles spend twice as much time experiencing selection in female as opposed to male bodies (Haig 2000; Parisi et al. 2003; Emerson et al. 2004; Haig 2006; Vicoso and Charlesworth 2006). Rice (1984), on the other hand, argued that the X chromosome should favor male-beneficial alleles that are recessive in their effects in female bodies but female-beneficial alleles that are dominant. Thus, X-linkage has been proposed to favor or oppose the maintenance of sexually antagonistic polymorphism and to favor or oppose female interests at the expense of male interests.

Haig (2000, 2006) has suggested that X chromosomes should show a matrilineal bias in the fixation of parentally antagonistic genes. Haig argues from the view that matrilineal interests have a leg up on patrilineal interests because X-linked alleles are twice as often maternally derived versus paternally derived, although these ideas have yet to be tested in a population genetic framework. There has been no previous theoretical exploration of the opportunity for parentally antagonistic polymorphism on sex chromosomes. In what follows, we fill this gap for X-linked parental antagonism and attempt to resolve some of the apparent disagreements in the theoretical development of X-linked sexual antagonism.

Population Dynamics

We assume an infinite population size, no mutation, and random union of gametes in the model below. Let p_e and p_s be the frequencies of allele A_1 in eggs and sperm, q_e and q_s be the corresponding frequencies for A_2. Let v_i be the fitness of a male with the A_iY genotype and w_{ij} be the fitness of a female with an A_iA_j genotype such that $v_1, v_2, w_{11}, w_{12}, w_{21}, w_{22}$ are the relative fitnesses of the genotypes $A_1Y, A_2Y, A_1A_1, A_1A_2, A_2A_1$, and A_2A_2, respectively.

If $w_{11} > w_{12} = w_{21} > w_{22}$ and $v_2 > v_1$, then such a locus is sexually antagonistic in its effects, where A_1 is arbitrarily selected to be the female-beneficial allele. This special case has been considered before by Haldane (1926), Bennett (1957, 1958), Sprott (1957), Mandel (1959), Parsons (1961), Haldane and Jayakar (1964), Rice (1984), Gavrilets and Rice (2006), and Engelstädter and Haig (2008).

When $w_{21} > w_{11}$, $w_{22} > w_{12}$ and $v_2 > v_1$, then such a locus is parentally antagonistic, where A_2 is arbitrarily selected to be the allele that is beneficial when maternally derived. This definition of parental antagonism sees the A_2 allele experiencing higher marginal fitness (i.e., the fitness of an allele averaged over all the genotypes in which it occurs; Rice 2004 p. 9) than the A_1 allele when maternally derived and the A_1 allele experiencing higher marginal fitness than the A_2 allele when paternally derived at any allele frequency.

The recursion equations give the next generation's allele frequencies as a function of the current generation's:

$$p_e' = \frac{1}{2} \cdot \frac{2p_e p_s w_{11} + p_e q_s w_{12} + q_e p_s w_{21}}{p_e p_s w_{11} + p_e q_s w_{12} + q_e p_s w_{21} + q_e q_s w_{22}} \qquad (1)$$
$$p_s' = \frac{p_e v_1}{p_e v_1 + q_e v_2},$$

(Haldane and Jayakar 1964).

This system has two trivial equilibria, $p_e = p_s = 0$ and $p_e = p_s = 1$. It also has the following polymorphic equilibrium, which can be obtained from equation (2) by setting $p_e' = p_e = \hat{p}_e$ and $p_s' = p_s = \hat{p}_s$:

$$\hat{p}_e = \frac{1}{2} \cdot \frac{v_1 w_{21} + v_2(w_{12} - 2w_{22})}{v_1(w_{21} - w_{11}) + v_2(w_{12} - w_{22})} \qquad (2)$$
$$\hat{p}_s = \frac{\hat{p}_e v_1}{\hat{p}_e v_1 + \hat{q}_e v_2}.$$

The stability of this equilibrium was examined by Haldane and Jayakar (1964) for $w_{12} = w_{21}$. A formal analysis of the stability of this equilibrium that allows parent of origin-specific fitness is beyond the scope of the current investigation.

When A_1 is rare and p_e and p_s are close to zero, (1) approximates to:

$$p_e' = p_e \frac{w_{12}}{2w_{22}} + p_s \frac{w_{21}}{2w_{22}} \qquad (3)$$
$$p_s' = p_e \frac{v_1}{v_2}.$$

The Jacobian of this system of equations linearizes allele frequency change near the equilibrium $p_e = p_s = 0$ and allows us to test its stability. The equilibrium is unstable, i.e., the A_1 allele

can invade, if the larger eigenvalue of the Jacobian is greater than one (Hartl and Clark 1989). Thus, the invasion condition for A_1 is:

$$\frac{v_1}{v_2} > \frac{2w_{22} - w_{12}}{w_{21}}. \quad (4)$$

The invasion condition for A_2 can be found in a similar manner:

$$\frac{v_2}{v_1} > \frac{2w_{11} - w_{21}}{w_{12}}. \quad (5)$$

A protected polymorphism exists if inequalities (4) and (5) are simultaneously satisfied, which means that neither allele can go to fixation and eliminate variation for those values of the parameters

$$\frac{w_{12}}{2w_{11} - w_{21}} > \frac{v_1}{v_2} > \frac{2w_{22} - w_{12}}{w_{21}}. \quad (6)$$

It is useful to reparameterize the above model in terms of selection coefficients (s, t) and a dominance parameter (h). For sexual antagonism, $v_1 = 1 - t$; $v_2 = 1$; $w_1 = 1$; $w_{12} = w_{21} = 1 - hs$; $w_2 = 1 - s$ (where $0 < s, t \leq 1$; $0 \leq h \leq 1$). Using these terms, inequality (6) becomes

$$\frac{2hs}{1 + hs} < t < \frac{2s(1 - h)}{1 - hs}. \quad (7)$$

For comparison, invasion conditions on the autosomes are independent of h when dominance is the same in both sexes. [Varying dominance between the two sexes permits a variety of equilibrium states (Owen 1953; Kidwell et al. 1977), but we restrict our consideration to equivalent dominance in the two sexes for the sake of tractability.] The opportunity for polymorphism under sexual antagonism on the autosomes is given by

$$\frac{s}{1 + s} < t < \frac{s}{1 - s} \quad (8)$$

(Kidwell et al. 1977). Thus, sexual antagonism creates the possibility for polymorphism without there being overdominance for fitness in either sex.

For parental antagonism, we have arbitrarily chosen A_2 as the allele with a fitness advantage when maternally derived (and fitness disadvantage when paternally derived). In males, $v_1 = 1 - t$ and $v_2 = 1$. In females, $w_{21} = 1$, $w_{11} = 1 - a\psi$, $w_{22} = 1 - b\psi$, and $w_{12} = 1 - \psi$. Inequality (6) becomes

$$\frac{1 - 2a}{1 - 2a\psi} < \frac{t}{\psi} < 2b - 1, \quad (9)$$

in which t and ψ act as selection coefficients and a and b serve as scalars that relate homozygous fitness to the heterozygous extremes. For comparison, on the autosomes, the fitness parameterizations are the same in both sexes and equivalent to the parameterization of the X in females given above. Polymorphism under parental antagonism is protected on an autosome provided $a, b \geq 0.5$ (Úbeda and Haig 2004), which ensures that the average of the two heterozygote fitnesses is greater than both of the homozygous fitnesses.

Opportunity for Polymorphism on the X

Inequality (6) describes the conditions for protected polymorphism and defines the region of parameter space that provides this opportunity. If this area is narrow or biologically unrealistic then intralocus conflict is an unlikely explanation of genetic and fitness variation in nature. If this area is large and does not require any special pleading about the nature of mutations, then intralocus conflict may well contribute to natural variation. We obtain the relative proportions of regions of parameter space by simulation.

A stable sexually antagonistic polymorphism is found in roughly 28% of the volume of the unit cube defined by $0 \leq s, t, h \leq 1$. As suggested by Rice (1984), the opportunity for polymorphism increases with increasing dominance of the female-beneficial allele, A_1 (Fig. 1). If the dominance parameter is selected from a uniform distribution in the range $0 \leq h \leq 0.5$ (female-beneficial allele partially dominant), the opportunity for polymorphism is roughly 49%; by contrast, if the dominance parameter is selected from $0.5 \leq h \leq 1$ (female-beneficial allele partially recessive), the opportunity for polymorphism is roughly 8% (Table 1). Our analysis did not consider the possibility of under-dominance ($h > 1$) or overdominance ($h < 0$). For comparison, on autosomes, the space contained within the bounds of inequality (8) represents roughly 38% of parameter space and is unaffected by dominance (Prout 2000). Thus, an X-linked locus has a smaller total opportunity for polymorphism than an autosomal locus, as shown before (Curtsinger 1980).

The comparison between the opportunity for polymorphism at X-linked and autosomal loci is based on a consideration of all possible combinations of selection coefficients and dominance values under the assumption of a uniform distribution of these parameters in the interval [0,1]. The uniform distribution assumes that allele effects are equally likely to be dominant or recessive and that strong sexual antagonism is as likely as weak sexual antagonism. If the comparison is restricted to weaker selection ($0 \leq s, t \leq 0.1$, $0 \leq h \leq 1$), the conclusion from before is reversed: a stable polymorphism is found in ~21% of that parameter space for X-linked loci but only in ~6% of the parameter space for autosomal loci (Fig. 2). Curtsinger (1980) did not find this reversal for weaker selection, perhaps because male and female heterozygotes were constrained to have equal fitness in his treatment. In our model, the opportunity for polymorphism is more sensitive to the strength of selection on autosomes than on the X.

For parental antagonism, the ranges $0 \leq a, b, t, \psi \leq 1$ define a hypercube in which the inequalities from (9) sit. Polymorphism is most likely for small values of t (which minimizes directional

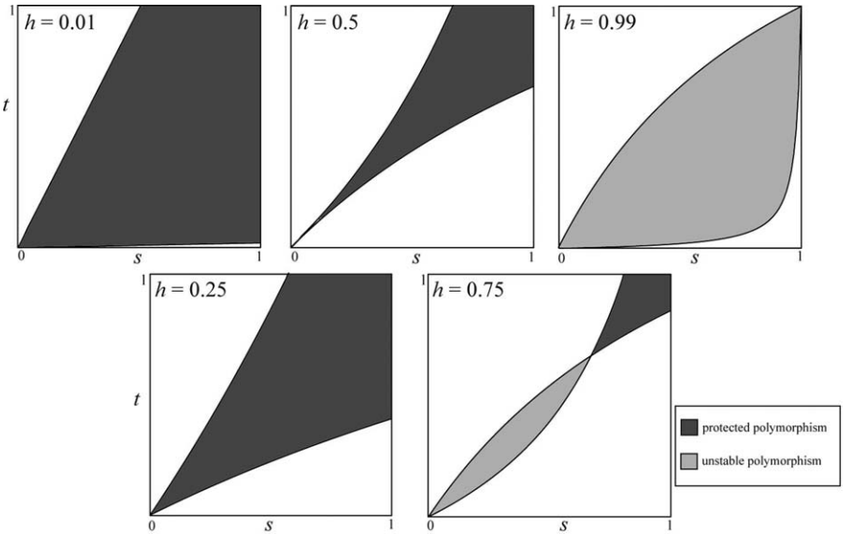

Figure 1. Opportunity for polymorphism on the X for varying dominance. The darkly shaded regions contain the values of the selection coefficients, *s* and *t*, that give rise to a protected polymorphism in the population. The upper left unshaded region of each plot represents all pairs of selection coefficients that lead to a stable fixation of the male-beneficial allele (A_2). The lower right unshaded region represents all pairs of selection coefficients that lead to stable fixation of the female-beneficial allele (A_1). The lightly shaded regions contain those pairs of selection coefficients that permit an unstable polymorphic equilibrium. As the dominance of the A_2 allele increases (i.e., with increasing *h*) the opportunity for polymorphism shrinks and the possibility for an unstable polymorphic equilibrium increases. Curves are taken from inequality (7).

selection in males), intermediate values of a, and larger values of ψ and b (which together have the ability to offset any directional selection that acts in males). In other words, one needs a particular—and perhaps unrealistic—type of gene action for parental antagonism to maintain X-linked polymorphism under parental antagonism.

If all four parameter values are independently drawn at random from a uniform distribution between zero and one, we find that the opportunity for polymorphism is a mere ~7%. For comparison, the opportunity for polymorphism under parental antagonism on the autosomes is 25%. However, the autosomal figure is reached without the same special pleading about gene action. This particular ~7% of parameter space for X chromosomes, with its special requirements for a, b, t, and ψ, may be a region of the space of all possible mutations that real genes rarely visit.

Sex Bias and Parent of Origin Bias on the X Chromosome

Under sexual antagonism, roughly 36% of the volume of the unit cube of parameter space corresponds to values that lead to stable fixation of the male-beneficial allele and roughly 29% of the volume corresponds to values that lead to stable fixation of the female-beneficial allele (Fig. 1, Table 1). The region of the total parameter space that permits stable fixation of the male-beneficial allele is ~27% larger than that for fixation of the female-beneficial allele, suggesting that the X chromosome is biased in favor of fixing male-beneficial alleles (Table 1). The bias holds for recessive male-beneficial alleles ($0 \leq h \leq 0.5$) as Rice (1984) predicts but also holds for dominant male-beneficial alleles ($0.5 \leq h \leq 1$) (Table 1). The bias is present but smaller for alleles that are closer to perfect additivity ($0.25 \leq h \leq 0.75$) (Table 1).

This result runs counter to the oft-repeated intuition that the X chromosome should be biased to evolve toward the female optimum because it spends twice as much time in females as in males (Haig 2000, 2006; Parisi et al. 2003; Emerson et al. 2004; Vicoso and Charlesworth 2006). The invasion condition for A_1 (4) from above can be written in a form that superficially resembles this intuition

$$\frac{v_1 - v_2}{v_2} + 2\left(\frac{w_h - w_2}{w_h}\right) > 0, \tag{10}$$

in which $w_h = w_{12} = w_{21}$. Loosely interpreted, this equation states that a rare sexually antagonistic allele can invade if its initial relative benefit in males exceeds twice its relative cost in females or if its relative benefit in females is more than half its relative cost in males. However, if the fitness effects of a rare allele are not completely dominant, the allele is expected to have a smaller effect on the fitness of heterozygous females than of hemizygous males [i.e., $(v_1 - v_2)$ will be greater in absolute value than $(w_h - w_2)$]. Thus, the numerators in (10) will be of unequal magnitude. In the case of perfect additivity ($h = 0.5$),

Table 1. Division of parameter space under sexually antagonistic selection on the X chromosome for various parameterizations of dominance (h). Numbers are given as proportions of parameter space that give rise to protected polymorphism, permit an unstable polymorphism, or lead to fixation of one or the other allele. Numbers in parentheses are these proportions of the parameter space for the weakest 1% of selection coefficients ($0 \le s, t \le 0.1$). For the four columns at right, the dominance parameter is chosen at random from a uniform distribution within the specified range. Within nearly every parameterization, the likelihood of fixation of the male-beneficial allele (A_2) is higher than that for the female-beneficial allele (A_1).

	h=0.01	h=0.25	h=0.5	h=0.75	h=0.99	$0<h<0.5$	$0.25<h<0.75$	$0.5<h<1$	$0<h<1$
Polymorphism	0.74 (0.74)	0.48 (0.49)	0.24 (0.04)	0.08 (0)	~0 (0)	0.49 (0.42)	0.25 (0.12)	0.08 (~0)	0.28 (0.21)
Unstable polymorphism	0 (0)	0 (0)	0 (0)	0.08 (0.40)	0.55 (0.73)	0 (0)	0.01 (0.09)	0.14 (0.40)	0.07 (0.18)
Male fix	0.25 (0.25)	0.30 (0.26)	0.38 (0.48)	0.41 (0.34)	0.39 (0.26)	0.31 (0.34)	0.38 (0.41)	0.42 (0.36)	0.36 (0.35)
Female fix	0.01 (0.01)	0.21 (0.25)	0.38 (0.48)	0.43 (0.26)	0.06 (0.01)	0.21 (0.24)	0.36 (0.38)	0.36 (0.26)	0.29 (0.25)

the average effect of a rare allele in females is expected to be half the average effect in males, offsetting the twofold greater occurrence of the allele in females and removing any bias (Fig. 1). We find that the X chromosome is biased in favor of males if mutations have a uniform distribution of h and similar magnitudes of fitness effects in hemizygous males and homozygous females.

Under parental antagonism, we find 14 times as many parameter combinations that result in fixation of A_2, the maternally derived favored allele, as result in fixation of A_1, the paternally derived favored allele. We draw our parameter values from a uniform distribution, although we expect to obtain a similar bias for any other mathematically or biologically reasonable assumption about what distribution the parameters take. Although this bias runs in the same direction as an intuitive prediction (Haig 2006) that relies on there being twice as many maternally derived alleles as paternally derived alleles, the concordance is obtained more by luck than good judgment. The invasion condition for A_1 (4) can be rearranged to give

$$\frac{v_1 - v_2}{v_2} + \frac{w_{12} - w_{22}}{w_{21}} + \frac{w_{21} - w_{22}}{w_{21}} > 0. \qquad (11)$$

The left-hand side of this inequality has three terms that correspond roughly to the fitness effect of a maternally derived allele in males, a maternally derived allele in females, and a paternally derived allele in females. The first of these, the effect of the allele in males, is on average the largest in absolute value and therefore has more to do with directing any bias than the other two terms, which, on average, exactly offset each other. Selection in females is unbiased with respect to parental origin but selection in males acts solely on maternally derived alleles in a directional fashion, introducing a bias in favor of fixing alleles that are favored when maternally derived.

Discussion

We have shown that sexual antagonism and parental antagonism are both capable of maintaining genetic and fitness variation at X-linked loci. The conditions for polymorphism due to parental antagonism on the X are restrictive, however. We have also shown that the X chromosome is more likely to fix male-beneficial alleles than female-beneficial alleles and that a commonly held intuition that predicts otherwise is incorrect due to an erroneous assumption. Additionally, X-linkage introduces a strong bias in favor of fixing alleles that are beneficial when maternally derived.

Parameter space is carved up according to inequality (6) regardless of any assumptions we may make about the distributions of parameter values. To arrive at the numerical figures, we presented for the opportunity for polymorphism and the direction of evolutionary bias, though, we assumed that parameters took

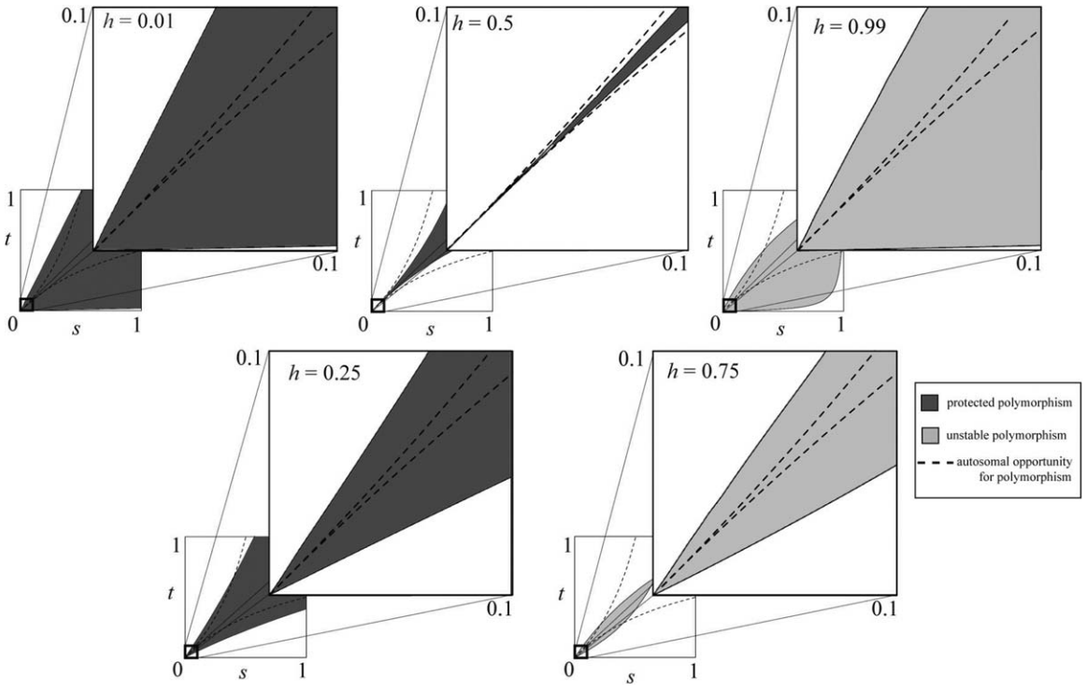

Figure 2. A comparison of the opportunity for polymorphism on the X chromosome and the autosomes for weaker selection. If selection coefficients in females and males (*s* and *t*, respectively) are confined to $0 \leq s, t \leq 0.1$, the opportunity for polymorphism on the X chromosome (darkly shaded region) exceeds that on the autosomes (area between the dashed lines) by ∼21% to ∼6%, respectively. This is in contrast to the case when selection coefficients take values from the entire permissible range, $0 < s, t \leq 1$ (Fig. 1), which sees X chromosomes having a smaller opportunity for polymorphism than autosomes (∼28% to ∼38%, respectively). Curves for both the X chromosome (solid lines) and autosomes (dashed lines) are taken from inequalities (7) and (8).

values from a uniform distribution between 0 and 1. The utility of our numerical results is found not in their quantitative predictions but in the qualitative comparisons between X chromosomes and autosomes. For instance, our numerical result that shows polymorphism is more likely on the X chromosome (∼21%) than on an autosome (∼6%) for weaker sexual antagonism ($0 \leq s, t \leq 0.1$) is more valuable for demonstrating the heightened sensitivity of autosomal polymorphism to the strength of selection than for making any prediction about how much sexually antagonistic variation is harbored on X chromosomes in nature. With this comparison in hand, we can reconcile the empirical results on the degree of polymorphism on the X chromosome (Gibson et al. 2002; Pischedda and Chippindale 2006) with the theoretical prediction of the opportunity for polymorphism by suggesting that most sexually antagonistic alleles segregating in populations are weakly selected. Further, we think these qualitative results on the opportunity for polymorphism and the direction of bias under sexual and parental antagonism hold up if any of our assumptions are relaxed and can only be reversed with special pleading. To eliminate the male bias we found for X-linked sexual antagonism, for example, mutations would have to have weaker effects in hemizygous males than in homozygous females or mutations would have to more often be female beneficial. This requires special assumptions about male developmental systems or about mutational effects, respectively.

Our results relate to previous theoretical work surrounding sex-linked polymorphism and sexual antagonism. Miller et al. (2006) and Rice et al. (2008) have examined sex-linked parental effects with sexually antagonistic consequences and found that maintaining genetic variation for an effect deriving from the heterogametic parent is precluded. This is in contrast to our results above for X-linked direct effects, in which variation is more likely to be found on the X under sexual antagonism, at least for weak selection. This outcome arises in the parental effect models because of the asymmetry of inheritance of the sex chromosomes from the heterogametic parent, which ensures that the expression of a gene in the parent is uncorrelated with its fitness effects in one sex of offspring. For example, X-linked paternal effect genes will only be passed to daughters and not to sons, and will therefore only be selected for their effects on daughters. Sexual antagonism at such a locus is therefore resolved in favor of daughter-beneficial interests. Sexual antagonism for parental effects does not generally preclude the maintenance of variation, however. For effects deriving from the X chromosome of the homogametic parent,

the conditions are equivalent to those for autosomal parental effects, which are themselves equivalent to autosomal direct effects (Gavrilets and Rice 2006; Patten and Haig 2009).

The theoretical development above may be relevant to empirical studies of sexually dimorphic gene expression, as this is one the potential resolution to intralocus sexual conflict (Rice 1984). A number of empirical studies have shown the X chromosome to be unique with respect to sexually dimorphic gene expression (Vicoso and Charlesworth 2006; Mank 2009), as might be expected from the uniqueness of our theoretical predictions for the X chromosome. However, these empirical studies taken together do not offer any consistent pattern for the evolution of sexual dimorphism on the X. For instance, one finds that male-specific genes are under-represented on the X in *Drosophila* (Parisi et al. 2003) but are overrepresented in human (Lercher et al. 2003). Perhaps the different modes of dosage compensation limit the parameter space that mutations can visit in such a way that gives rise to these incompatible results; perhaps the evolution of sexual dimorphism is sensitive to the population size differences between these taxa; or, perhaps sexually antagonistic selection is not the most important force underlying the evolution of sexually dimorphic gene expression. Regardless of the actual cause of sexually dimorphic gene expression in nature, the theoretical development we offer above is general enough to serve as a theoretical basis for any claim about sexual antagonism's role. Care should be taken, though, in extending our model to any genomics study, because our use of the term "gene" covers more than simply the transcribed and translated regions of the genome.

Finally, any results here also apply to Z chromosomes, provided sex- and parent-specific labels are reversed accordingly. Further, these results apply to organisms subject to any mode of dosage compensation, except for the imprinted X-inactivation that is found in marsupials and in the extra-embryonic membranes of some eutherians (Payer and Lee 2008).

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