The Evolution of Sex Ratio Adjustment in the Presence of Sexually Antagonistic Selection

Gwylim S. Blackburn, Arianne Y. K. Albert, and Sarah P. Otto*

Department of Zoology and Biodiversity Research Centre, University of British Columbia, 6270 University Boulevard, Vancouver, British Columbia V6T 1Z4, Canada

Submitted January 15, 2010; Accepted May 2, 2010; Electronically published July 23, 2010

Online enhancements: appendixes.

ABSTRACT: Sex ratio adjustment (SRA) of broods has received widespread interest as a means for optimizing parental investment in offspring. Classical explanations for the evolution of SRA focus on improving offspring fitness in light of resource availability or mate attractiveness. Here, we use genetic models to demonstrate that SRA can evolve to alleviate sexual antagonism by improving the chance that the alleles of a sexually antagonistic trait are transmitted to the sex they benefit. In cases where the trait is autosomally inherited, this result is obtained regardless of whether SRA is based on the mother's or the father's genotype and irrespective of the recombination rate between the trait and SRA loci. SRA also evolves in this manner when the trait is sex-linked, provided that SRA decisions are based on the homogametic genotype (XX mothers or ZZ fathers). By contrast, when based on traits in the heterogametic sex, SRA promotes fixation of the allele that is detrimental to that sex, preventing the evolution of substantial levels of SRA. Our models indicate that the evolution of SRA in nature should be strongly influenced by the genetic architecture of the traits on which it is based and the form of selection affecting them.

Keywords: sex allocation, intralocus sexual conflict, inheritance, evolutionary theory.

Introduction

Sex ratio adjustment (SRA) of broods has been documented in numerous taxa and can result in brood sex ratio bias ranging from a few percent (e.g., Desfor et al. 2007; Servanty et al. 2007) to nearly 100% (e.g., Komdeur et al. 1997; Davison and Ward 1998; reviewed in Cockburn et al. 2002; West et al. 2002; West 2009). SRA has gained both theoretical and empirical attention as an avenue for the evolution of optimal sex allocation (investment in each sex), motivated by the idea that parents who can predict the differential fitness prospects for sons and daughters might favorably bias the sex ratio of their broods toward the fitter sex. Previous hypotheses for the adaptive value of SRA have focused on improving offspring fitness in light of a mother's condition and resource availability (Trivers and Willard 1973; West and Sheldon 2002) and on biasing brood sex ratios toward sons when mates possess attractive male-specific traits (Burley 1981, 1986; Fawcett et al. 2007). As a result, SRA has been linked to factors including social rank (e.g., Clutton-Brock et al. 1984, 1986), population density (e.g., Kruuk et al. 1999), mate quality (Long and Pischedda 2005), and local competition for mates (e.g., Herre 1985; Flanagan et al. 1998) or other resources (e.g., Gowaty 1993; Aars et al. 1995; reviewed in Hardy 2002; West 2009). However, the degree of SRA observed and the factors governing SRA decisions vary widely across species (Clutton-Brock and Iason 1986; Sheldon 1998; Cockburn et al. 2002), and several authors have suggested that we require a better understanding of the types of trait that prompt SRA and the mechanistic constraints on its evolution (Krackow 1995; Cockburn et al. 2002; West et al. 2002, 2005; West and Sheldon 2002; Uller et al. 2007; West 2009).

With this aim in mind, we evaluate the potential for SRA to alleviate sexual antagonism, which has received growing empirical support (Calsbeek and Sinervo 2004; Calsbeek and Bonneaud 2008; Connallon and Jakubowski 2009; Roulin et al. 2010) but little theoretical attention (but see Alonzo and Sinervo 2007), despite recognition of its potential importance (Fawcett et al. 2007; Patten and Haig 2009). We focus on intralocus sexual conflict, a form of sexual antagonism that arises when selection at a locus favors different alleles in males and females (Lande 1980; Rice and Chippindale 2001). Under the appropriate conditions, intralocus sexual conflict can stably maintain genetic polymorphisms (Lande 1980; Rice 1984, 1987; Albert and Otto 2005; Patten and Haig 2009), preventing either sex from reaching its optimal phenotypic state and lowering mean population fitness (Lande 1980). The possibility that SRA diminishes this effect is of broad importance, given the widespread occurrence of this source of

* Corresponding author; e-mail: otto@zoology.ubc.ca.

Am. Nat. 2010. Vol. 176, pp. 264–275. 0003-0147/2010/17603-51868$15.00.
DOI: 10.1086/655220

Table 1: Fitness of diploid sexually antagonistic trait genotypes

	Trait genotype		
	TT	*Tt*	*tt*
Female fitness	1	$1 - h_f s_f$	$1 - s_f$
Male fitness	$1 - s_m$	$1 - h_m s_m$	1

sexually antagonistic selection in nature and present efforts to identify mechanisms by which it can be resolved (Bonduriansky and Chenoweth 2009; van Doorn 2009).

We consider a trait that has opposing fitness effects in the two sexes and develop a diploid population genetic model to track the evolution of a modifier allele at a separate SRA locus. We predict that selection will favor the evolution of the modifier allele such that females bias the sex ratio of their broods according to which sexually antagonistic alleles their offspring are likely to receive, in a manner that increases the match between offspring sex and the alleles that benefit that sex. We supplement our analytical model with simulations that allow continual mutations to occur at the SRA modifier locus, in order to follow the long-term evolution of SRA and its effect on the trait. While sexually antagonistic traits are expected to arise throughout the genome, they may be more likely to remain polymorphic on sex chromosomes because of the imbalanced manner in which sex-linked genes are inherited and expressed among sexes (Rice 1984; Rice and Chippindale 2001; Gibson et al. 2002; Vicoso and Charlesworth 2006; Mank et al. 2007; Mank 2009). We therefore compare results for autosomal and sex-linked traits.

Evolution of Sex Ratio Adjustment at Autosomal Loci

Analytical Model

A single locus (**T**) governs a trait that is under sexually antagonistic selection, where one allele *T* is favored in females while the alternative allele *t* is favored in males. Such opposing selection pressures are expected for physiological, morphological, and behavioral traits that have different optima between sexes because of differences in their ecology or reproductive biology (Glucksmann 1974; Lande 1980; Rice 1984; Frayer and Wolpoff 1985; Shine 1989). Sexual antagonism can also be driven by natural and sexual selection acting in different directions; our models accommodate this scenario, provided that we assume that female preferences are fixed.

Sexually antagonistic selection is described by sex-specific fitness values (table 1), with selection coefficients against the deleterious allele in females and males given by s_f and s_m, respectively ($0 \le s_i \le 1$) and dominance coefficients in females and males given by h_f and h_m (with $0 \le h_i \le 1$). A second locus, **M**, is a modifier that allows a female to adjust the sex ratio of her brood (hereafter referred to as sex ratio adjustment [SRA]). Initially, the population is fixed for one allele (*M*), which may code for no SRA or some initial level of SRA. We then introduce a second modifier allele, *m*, and determine its fate. We assume throughout that SRA is subject only to maternal control; if paternal control were instead considered, we expect that equivalent results would be obtained (but with the sexes reversed with respect to selection and XY/ZW sex determination).

The proximate mechanisms of SRA are not well understood. Here, we assume that it can be implemented via any of the potential behavioral or physiological mechanisms of SRA, such as hormonal effects on sperm selection and gestation, postparturition maternal effects, or external environmental factors (e.g., ambient temperature; Krackow 1995; Uller et al. 2007). In addition, we impose no direct costs of SRA. Recombination between **T** and **M** occurs at rate *r*, and mutations are ignored. Standard two-locus recursions were developed and analyzed with Mathematica 6.0 (see app. A in the online edition of the *American Naturalist*).

In general, a female might adjust her sex ratio strategy in response to either her own state or that of her environment, including her social and mating environment (e.g., Flanagan et al. 1998; Alonzo and Sinervo 2007; Burton-Chellew et al. 2008). In our models, we allow a female to adjust her SRA on the basis of her own trait genotype (self-SRA) or the trait genotype of her mate (mate-SRA), assuming that trait evaluation occurs during reproduction via any feasible cognitive or physiological mechanism. Specifically, the probability that a mother produces a daughter is given by D_{ij}, where *i* represents the number of *m* alleles in her modifier genotype and *j* represents the number of *t* alleles in either her trait genotype (in the case of self-SRA) or her partner's genotype (in the case of mate-SRA; table 2). Although plausible values of D_{ij} range from 0 to 1, any errors in a female's assessment of her own or her mate's genotype would reduce the expression of SRA (bringing the D_{ij} for a female closer to her mean SRA across trait genotypes) and so would constrain the evolution of SRA. Given that the nature of these

Table 2: Sex ratio adjustment (SRA) in the case of a diploid trait and modifier

	Female trait (self-SRA) or male trait (mate-SRA)		
Female modifier	*TT*	*Tt*	*tt*
MM	D_{00}	D_{01}	D_{02}
Mm	D_{10}	D_{11}	D_{12}
mm	D_{20}	D_{21}	D_{22}

constraints is unknown, we assume that females have perfect information regarding the trait genotype, although assessment errors could easily be incorporated by appropriate choices of D_{ij} and would not alter the structure of the model.

We first identify the conditions under which a stable polymorphic equilibrium exists when the population is fixed for the *M* allele and then determine whether the *m* allele could invade if it alters SRA. Here, we present only a summary of the analyses in order to provide the reader with an intuitive understanding of the evolutionary dynamics; full analytical details are available in the Mathematica 6.0 file at http://www.zoology.ubc.ca/~otto/Research/BlackburnEtAl2010.nb. For tractability in this analysis, we assume that selection and the effect of the modifier are weak. Specifically, we assume that s_f, s_m, and the differences among the D_{ij} are all on the order of a small term, ξ. In the next section, we perform simulations that relax these assumptions.

The analytical results for autosomal loci are identical under self-SRA and mate-SRA, so they are not distinguished in this section. With the *M* allele fixed, opposing selection pressures in males and females can, under appropriate conditions, maintain variation in a trait, with the equilibrium frequency of allele *T* equal to

$$\hat{p}_T = \frac{(1-h_f)s_f - h_m s_m - H\theta}{(1-2h_f)s_f + (1-2h_m)s_m + (1-2H)\theta} + O(\xi), \quad (1)$$

where $O(\xi)$ represents terms that are of order ξ and hence smaller than the leading term. The term *H* measures the dominance of the SRA strategy in *MM* females: $H = (D_{01} - D_{02})/(D_{00} - D_{02})$. The term θ represents the effect of Fisherian sex ratio selection, which acts to maintain a balanced population-wide sex ratio (Fisher 1930; Bodmer and Edwards 1960; Kolman 1960) and takes the form

$$\theta = \left(\overline{D} - \frac{1}{2}\right)\frac{\Delta D}{\overline{D}(1-\overline{D})}, \quad (2)$$

where $\overline{D}$ is the mean sex ratio at birth in the population (proportion females) and ΔD is the difference in SRA in response to *TT* versus *tt* genotypes ($\Delta D = D_{00} - D_{02}$). The term θ is nonzero whenever the population-wide sex ratio departs from 1/2 and the *T* and *t* alleles are associated with different offspring sex ratios. For example, if the population is female biased ($\overline{D} > 1/2$) and *T*-bearing females are more likely to produce daughters ($\Delta D > 0$), then θ has a positive value and drives down the equilibrium frequency of the *T* allele.

A stability analysis indicated that the polymorphic equilibrium is stable only when the denominator of equation (1) is positive. Polymorphism is likely when the dominance coefficients are sufficiently low that *Tt* heterozygotes are more fit than the homozygotes when averaged across the sexes. We expect to see the persistence of high levels of sexually antagonistic fitness variation only at autosomal loci that allow a stable polymorphism, and we therefore focus exclusively on such loci.

Next, a new SRA modifier allele, *m*, was introduced to the polymorphic equilibrium represented by equation (1). Our goal was to assess which conditions would permit the modifier to spread in the population while preserving this equilibrium. The leading eigenvalue (λ) within the characteristic polynomial of the local stability matrix is useful in this regard, because this value describes when the equilibrium remains stable in the presence of *m*. Specifically, the modifier can invade whenever λ has a value greater than 1, and selection will thus favor any *m* allele that places λ within this range of values. We determined how the parameters in our model affect λ by representing it as a power series, $\lambda = \lambda_0 + \lambda_1\xi + \lambda_2\xi^2 + \ldots$, where ξ represents the order of terms assumed to be small (the selection coefficients and the differences in D_{ij}). Solving for the successive terms in this series, we obtain

$$\lambda = 1 - \frac{1}{2}\frac{[\overline{D} - (1/2)]}{\overline{D}(1-\overline{D})}F_1 + \hat{p}_T\hat{p}_t s_m[(1-h_m)\hat{p}_T + h_m\hat{p}_t]F_2 + O(\xi^3), \quad (3)$$

where

$$F_1 = \hat{p}_T^2(D_{10} - D_{00}) + 2\hat{p}_T\hat{p}_t(D_{11} - D_{01}) + \hat{p}_t^2(D_{12} - D_{02}),$$

$$F_2 = \hat{p}_T(D_{10} - D_{00}) + (\hat{p}_t - \hat{p}_T)(D_{11} - D_{01}) - \hat{p}_t(D_{12} - D_{02}).$$

While equation (3) appears not to depend on selection in females, this is because we have used equation (1) to rewrite s_f in terms of s_m and $\hat{p}_T$ to simplify the presentation (see the Mathematica file).

We can see that the first line of equation (3) dominates the value of λ whenever the population-wide sex ratio departs substantially from 1/2, exerting Fisherian sex ratio selection in favor of strategies that equalize the sex ratio. For example, if the population is female biased ($\overline{D} > 1/2$), selection favors any *m* allele that increases the production of sons, causing the term F_1 to be negative (i.e., causing λ to be greater than 1). If the population-wide sex ratio

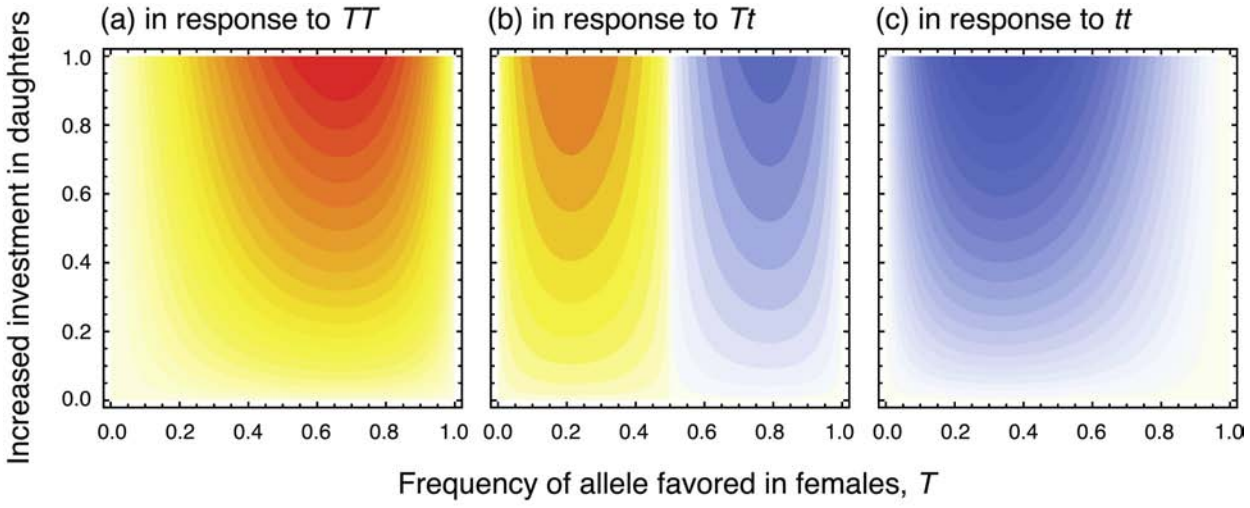


Figure 1: Strength of selection for a sex ratio adjustment (SRA) modifier that increases the production of daughters when the trait genotype of the mother (self-SRA) or father (mate-SRA) is *TT* (*a*), *Tt* (*b*), or *tt* (*c*). The strength of selection acting on the modifier is defined as $\lambda - 1$, which describes the rate of change of allele *m* while rare in a manner equivalent to direct selection on the **M** locus. Equation (3) was used for λ, setting $h_m = 1/2$ and assuming that the population sex ratio remains balanced ($\overline{D} = 1/2$). Positive selection increases in strength from white (0) to red ($2s_m/27$), while negative selection increases in strength from white (0) to blue ($-2s_m/27$). The amount of change in D_{ij} in response to a particular genotype is given by the *Y*-axis (e.g., the *Y*-axis of *a* is $D_{10} - D_{00}$).

is nearly 1/2, then the term $\overline{D} - 1/2$ is small (of order ξ) and the second line of equation (3) also contributes to the fate of the new modifier allele. The sign of the second line depends on the sign of F_2, which contains the terms that drive the evolution of SRA (fig. 1). Specifically, the term $\hat{p}_T(D_{10} - D_{00})$ indicates that the system will evolve to produce more daughters when in *TT* females (self-SRA) or in matings with *TT* males (mate-SRA). The term $-\hat{p}_t(D_{12} - D_{02})$ indicates that the system will evolve to produce fewer daughters when in *tt* females (self-SRA) or in matings with *tt* males (mate-SRA). Finally, the evolution of the SRA strategy in heterozygotes, represented by the term $(\hat{p}_t - \hat{p}_T)(D_{11} - D_{01})$, favors production of daughters when *T* is rare and production of sons when *t* is rare. To understand this result, consider the case when *T* is rare. Mothers that are *Tt* (self-SRA) or have mated with *Tt* males (mate-SRA) will produce offspring that are more likely to carry the *T* allele than the rest of the population (who are predominantly *tt*). Because the *T* allele benefits females, female offspring will be more fit relative to the rest of the population, while male offspring will be less fit, thus explaining why SRA among *Tt* individuals evolves to favor daughters when *T* is rare. Reversing this logic explains why SRA among *Tt* individuals evolves to favor sons when *t* is rare.

This local-stability analysis demonstrates that SRA evolves as expected: favoring daughters when parents carry the allele advantageous in females (*T*) and favoring sons when parents carry the allele advantageous in males (*t*). In fact, as long as the population sex ratio remains nearly equal and assessment errors are low, stronger and stronger SRA is expected to evolve, and equilibrium (1) will remain valid and stable throughout this process. The evolution of SRA can be rapid, with selection on the modifier on the order of the strength of selection at the **T** locus times the extent to which SRA is altered by the new modifier allele (eq. [3]). The fact that *r* does not enter into the leading-order terms of λ implies that SRA evolution should be insensitive to the recombination rate between the trait and the modifier.

Simulating the Long-Term Evolution of SRA in Response to Autosomal Loci

We next built an individual-based simulation that allowed multiple alleles to arise at the modifier locus, enabling us to track the long-term evolution of SRA in the presence of sexual antagonism. Only self-SRA was simulated, because the results for mate-SRA were expected to be similar. To reduce the number of parameters, we assumed complete additivity at the modifier locus—$D_{i1} = (D_{i0} + D_{i2})/2$; $D_{1j} = (D_{0j} + D_{2j})/2$—so that each modifier allele could be fully characterized by its action in homozygotes. Specifically, we represented the SRA strategy of a homozygous modifier genotype, say *MM*, as $\{D_{00}, D_{02}\}$, where

the two elements of this vector give the offspring sex ratio when the mother is *TT* and *tt*, respectively.

To facilitate comparison with previous work, we followed the form of the model 1 simulations in Fawcett et al. (2007), used to explore the evolution of SRA in the presence of sexual selection (in that study, the trait was expressed only in males). In particular, mutations occurred at the modifier at a rate of $\mu = 0.05$ per allele per generation, with each mutation causing a $+1/250$ or a $-1/250$ change in one of the pair of strategies characterizing an allele (e.g., a modifier allele with strategies {124/250, 121/250} might mutate to {123/250, 121/250}). In the standard set of simulations, selection coefficients were set to $s_f = s_m = 1/6$ and the dominance coefficients to $h_f = h_m = 1/10$ (dominance was not present in the haploid model considered by Fawcett et al.). The population was held fixed at 4,000 diploid individuals (Fawcett et al. modeled the same number of alleles but in 8,000 haploid individuals, and they also allowed slight variation over time in the population size, depending on the sex ratio). The trait and modifier loci were assumed to be unlinked ($r = 1/2$). We placed no intrinsic limit on the degree of SRA control females can possess, in order to let SRA evolve freely within the context of sexually antagonistic selection.

Over 50,000 generations, strong SRA occurred in the predicted direction (fig. 2), increasing the mean fitness of each sex in the population (fig. 3). Mean male fitness (*blue curve*) reached a slightly higher value than mean female fitness (*red curve*), in accordance with the final frequency of the female-benefiting *T* allele being slightly below 0.5 in figure 2. Both the pattern and the speed of SRA evolution were comparable to those observed by Fawcett et al. (2007) in simulations of Fisherian sexual selection with recurrent deleterious mutations or sexual selection on traits indicating male condition. Note, however, that the simulations of Fawcett et al. included two loci contributing to SRA (doubling the SRA mutation rate) and assumed that individuals were haploid (effectively increasing the strength of selection relative to the diploid model used here), which suggests that sexual antagonism is at least as effective as sexual selection in generating SRA.

In contrast to the predictions of the analytical model, the speed of SRA evolution was relatively slow in the simulations, taking tens of thousands of generations, as emphasized also by Fawcett et al. (2007). We explored two potential explanations for this result. First, it is possible that most mutations were prevented from spreading to high frequency because they perturbed the sex ratio from 1/2. To test this explanation, we ran the simulations again while allowing each mutation to have equal and opposite effect in *TT* and *tt* mothers while keeping the magnitude

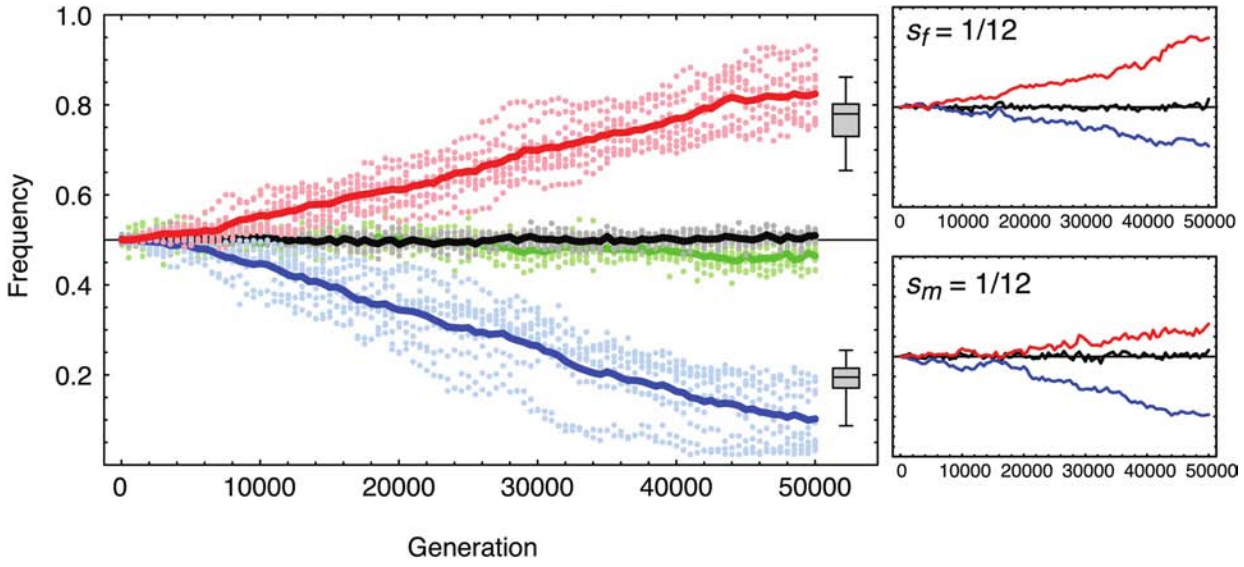


Figure 2: Evolution of sex ratio adjustment in response to sexual antagonism under autosomal inheritance. Curves show the average frequency of daughters born in response to *TT* (*red*) or *tt* (*blue*) genotypes, the overall sex ratio (*black*), and the frequency of the *T* allele (*green*). Dotted curves represent replicate simulations ($n = 10$), and solid curves plot their means. Box plots show the 25% and 75% quantiles (with whiskers showing the range) for the entire population of strategies present in the first replicate at 50,000 generations. In the standard set of simulations (*left*), $s_f = s_m = 1/6$, $h_f = h_m = 1/10$, and the mutation rate was set to $\mu = 0.05$. The panels to the right show the effect of weaker selection on the trait in females (*top*) and males (*bottom*).

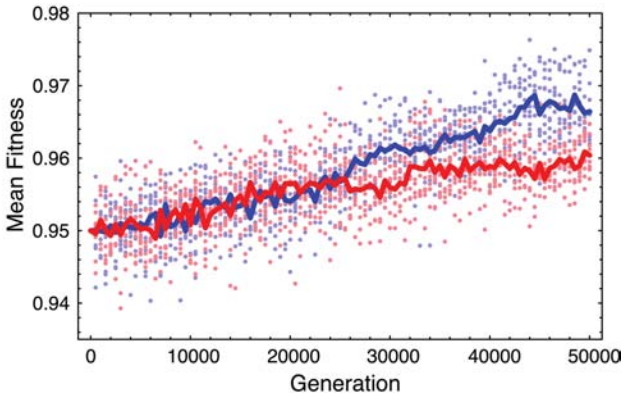


Figure 3: Change in mean fitness among females (*red*) and males (*blue*) during the course of sex ratio adjustment evolution. The simulations are the same as those reported in the left-hand panel of figure 2.

of the mutation at 1/250 (that is, the mutational effect was either $\{+1/500, -1/500\}$ or $\{-1/500, +1/500\}$). Under the default parameter settings, it took on average 31,116 generations (SE = 2,643) for an SRA difference of 0.5 to evolve, which was not significantly faster than when mutations affected the response in *TT* or *tt* mothers separately. Second, the speed of SRA evolution might have been limited by the amount of genetic variance in SRA, which is expected to be proportional to the mutation rate at the modifier locus times the square of the effect size of modifier mutations (Dieckmann and Law 1996). Simulation results for a range of effect sizes are shown in figure 4 for both the default mutation rate, $\mu = 0.05$, and $\mu = 0.005$. The time required to produce an SRA strategy differing by more than 0.5 between *TT* and *tt* mothers declined dramatically as the mutation rate and effect size of the modifier increased. We thus conclude that the most important factor limiting the speed of SRA evolution in these simulations was the amount of genetic variation for SRA.

The simulations considered above involved symmetrical selection in males and females. Because the trait allele then equilibrated at a frequency of 0.5, extremely biased SRA could evolve where *TT* and *tt* genotypes nearly always resulted in the production of daughters and sons, respectively. When the frequency of *T* departs from 1/2 because of sex differences in the strength of selection, the evolution of extremely biased SRA is hampered because it would cause the population sex ratio to depart from 1/2. For example, if the frequency of *t* were 0.9 and if *tt* genotypes resulted in the exclusive production of sons, then the sex ratio would become very biased (>80% male). Consistent with this interpretation, we found that when selection was weaker in females ($s_f = 1/12$ and $s_m = 1/6$, leading to $\hat{p}_T = 0.3$), SRA evolved to a lower level in response to the more common *tt* genotypes than in response to the rarer *TT* genotypes, ensuring that the sex ratio remained near 1/2 (*black curve* in fig. 2, *top-right panel*). The exact opposite was seen when selection was weaker in males ($s_f = 1/6$ and $s_m = 1/12$, leading to $\hat{p}_T = 0.7$; fig. 2, *bottom-right panel*). We expect this constraint to be weaker if SRA in *Tt* mothers were free to evolve rather than being fixed at the average of *TT* and *tt* mothers. Nevertheless, these examples illustrate the main restriction on the evolution of SRA: Fisherian sex ratio selection prevents the evolution of substantial SRA in any case that leads to a large departure from a balanced sex ratio within the population.

Evolution of Sex Ratio Adjustment at Sex-Linked Loci

Analytical Model

Here, we summarize the outcomes for X and Z linkage; detailed quantitative results are presented in appendix C in the online edition of the *American Naturalist*, and the entire analysis is available in the Mathematica file. If a sex ratio of 1/2 ($\theta = 0$) is assumed, stability requires only that the female-benefiting allele *T* be partially recessive in females (with X linkage) or that the male-benefiting allele *t* be partially recessive in males (with Z linkage; table B1 in the online edition of the *American Naturalist*). In the case of X linkage, we consider both autosomal and sex-linked modifiers when assessing whether a modifier allele altering offspring sex ratios could spread in a population

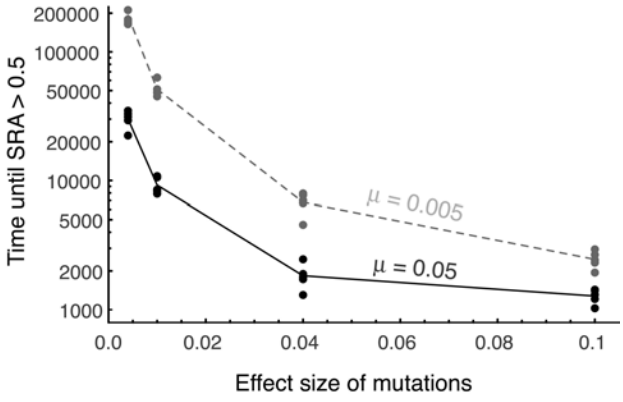


Figure 4: Speed of sex ratio adjustment (SRA) evolution in relation to mutation rate and effect size. A mutation at the modifier locus caused one of the two elements of its SRA strategy $\{D_{ij}, D_{kl}\}$ to change by an amount determined by the effect size (set to 1/250, 1/100, 1/25, or 1/10), with positive and negative effects being equally likely. Curves connect the mean values of five replicates (*dots*) at different effect sizes. Remaining parameter values are as in figure 2, where the effect size was 1/250.

that is polymorphic for a sex-linked trait. With a Z-linked modifier and maternal sex ratio control, however, modifier alleles that increase the proportion of sons are always favored, regardless of the trait carried, because the Z chromosome possessed by mothers passes only from mothers to sons. This induces strong meiotic drive that can lead to "extraordinary" sex ratio imbalances and potentially even population extinction (Hamilton 1967). Thus, we consider only autosomal modifiers of SRA in the presence of a Z-linked trait.

The results differ between self-SRA and mate-SRA when there is sex linkage, so we must now treat these scenarios separately. In the case of a sex-linked trait and SRA based on the homogametic sex (self-SRA based on XX females or mate-SRA based on ZZ males), the evolution of SRA is qualitatively the same as in the autosomal model (app. C). However, dramatically different results are obtained when SRA occurs in response to the hemizygous genotype (mate-SRA based on XY males or self-SRA based on ZW females): SRA then directly affects the allele frequency dynamics at the trait locus. The reason is that SRA induces transmission distortion when traits in the heterogametic sex are used to adjust offspring sex ratios. Consider mate-based SRA involving *T*-bearing and *t*-bearing X chromosomes in XY males. Because *T* is favored in females, SRA is expected to evolve such that females mated to *T*-bearing males produce more daughters, which causes the X chromosome carrying the *T* allele (X^T) to be inherited by more offspring than the Y chromosome. In contrast, females mated to *t*-bearing males evolve to produce more sons, which causes the Y chromosome to be inherited and reduces the transmission of the *t* allele to offspring. The opposite tendency develops in the case of self-SRA with ZW sex determination; *T*-bearing females evolve a daughter-biased sex ratio, which results in an increased inheritance of W instead of Z^T, reducing transmission of the *T* allele. Thus, when traits in the heterogametic sex are used to adjust offspring sex ratios, SRA evolves in a manner that induces a transmission disadvantage against the allele that is fitter in the heterogametic sex. As a result, if SRA becomes too extreme, the trait polymorphism can be lost, with allele *T* fixed in XY systems and *t* fixed in ZW systems. In addition, if SRA is based on the hemizygous sex and the modifier is autosomal, it is also possible for SRA evolution to halt at a low level ($\Delta D_{\text{hemi}} = s_{\text{m}}/4$ for mate-SRA in XY systems and $\Delta D_{\text{hemi}} = s_{\text{f}}/4$ for self-SRA in ZW systems, where $\Delta D_{\text{hemi}} = D_{00} - D_{01}$, as defined in table B2). This occurs because of genetic associations that develop between the modifier and trait loci, inhibiting further evolution of SRA even though the trait locus remains polymorphic (see app. C).

In summary, while sex linkage can make it easier for sexually antagonistic selection to maintain polymorphism for a trait in the first place, it can also constrain the evolution of offspring SRA in response to this trait. Specifically, whenever the genotype of the heterogametic sex is used to adjust the offspring sex ratio (mate-SRA with X-linked traits and self-SRA with Z-linked traits), SRA evolves only up to a point at which either the trait polymorphism is lost or selection on the modifier changes sign, preventing the further evolution of SRA.

Simulating the Long-Term Evolution of SRA in Response to Sex-Linked Loci

We explored the evolution of SRA over longer periods of time, using simulations with an X-linked trait. We were particularly interested in testing the predictions made by our analyses concerning the role of transmission distortion. The simulations were built as described for the case of an autosomal trait and run for equivalent parameter values. We first simulated self-SRA with an X-linked trait and an X-linked modifier. The analysis predicted that SRA should evolve in a manner similar to the autosomal case but be faster by a factor of approximately 4/3 (see app. C). The simulation results in figure 5*a* confirm this prediction: SRA in response to *TT* genotypes and in *tt* genotypes evolved at nearly the rate predicted from a regression based on the autosomal case (fig. 2), with slopes multiplied by 4/3.

Under mate-based SRA, a different picture emerged. Mothers evolved to alter their brood sex ratio such that X^TY males produced more daughters and X^tY males produced more sons, resulting in transmission distortion that caused *T* alleles to increase rapidly from the frequency of 1/2 predicted by natural selection alone (*green curves* in fig. 5*b*, 5*c*). As expected with an X-linked trait and an X-linked modifier (see eq. [C1] in the online edition of the *American Naturalist*), SRA evolved only to the point where the trait polymorphism was lost. As the *T* allele rose in frequency, the sex ratio in matings with *T*-bearing males became more and more constrained to remain near 1/2 (*red curve*; see inset in fig. 5*b*), to ensure that the population-wide sex ratio was even. Meanwhile, the sex ratio in matings with *t*-bearing males declined (*blue curves*), until the frequency of *T* reached 1. At this point, the sex ratio strategy in matings with *t*-bearing males was free to drift around neutrally.

Finally, we simulated the case of mate-SRA with an X-linked trait locus and an autosomal modifier locus. For the parameters used in figure 5, SRA was predicted to evolve only up to an intermediate level, with $\Delta D_{\text{hemi}} = s_{\text{m}}/4$ (eq. [C2] in the online edition of the *American Naturalist*). Assuming a population at equilibrium (table B1) with an even population-wide sex ratio, the analysis predicts that SRA should have stopped evolving once mothers

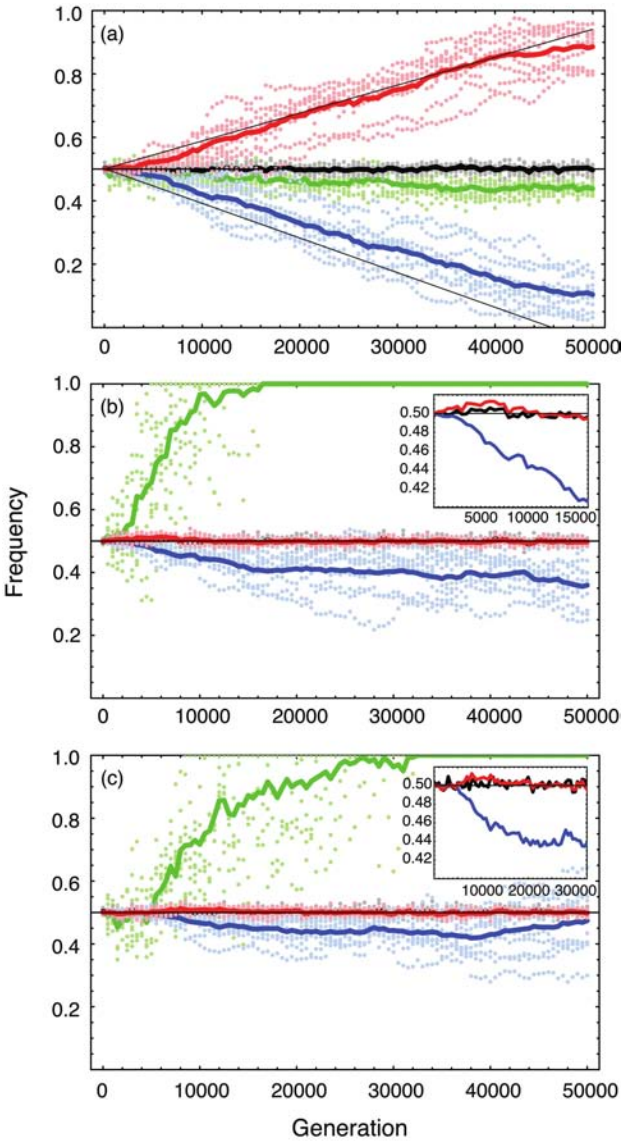


Figure 5: Evolution of sex ratio adjustment (SRA) with sex linkage. Colors and parameters are as in figure 2. The curves in *a* represent self-SRA in the case of an X-linked trait and an X-linked modifier. Straight lines show the predicted slope of SRA, calculated as the autosomal case in figure 2 but with the slope multiplied by 4/3 (see app. C in the online edition of the *American Naturalist*). The bottom panels illustrate the evolution of mate-SRA with an X-linked trait and an X-linked modifier (*b*) and with an X-linked trait and an autosomal modifier (*c*). Insets show greater resolution near $p_T = 0.5$.

produce 51% daughters when mated to *T*-bearing males and 47% daughters when mated to *t*-bearing males, with an equilibrium frequency of *T* of $\hat{p}_T = 0.8$. While the simulation results (fig. 5*c*) suggest that the system briefly hovered near this point, the system exhibited substantial fluctuations, and ultimately the *T* allele fixed in all 10 replicates. The same result was obtained in simulations initiated using the exact modifier strategy and *T*-allele frequency at the point where SRA evolution is expected to halt (results not shown), indicating that the *T* allele fixed because of fluctuations about the equilibrium value. We suspect that these fluctuations were caused either by random genetic drift (given the finite population size of 4,000 diploids) or by the stochastic sequence of modifier mutations that happened to arise during the simulations, causing the sex ratio to depart too far from 1/2 or causing too much transmission bias on the *T* allele.

Overall, the simulations with sex linkage confirm one of our main results: basing offspring sex ratios on the trait of a hemizygous individual (mate-based SRA in the case of male heterogamety) generates a transmission bias that is generally too strong to permit the evolution of high levels of SRA. Instead, only a low sex ratio bias evolves, followed by fixation of the allele that benefits the homogametic sex. While we had predicted an intermediate evolutionarily stable strategy in the case of an autosomal modifier, the simulations indicated that this strategy is not robust to departures from the assumptions of the analytical model (infinite population size, weak selection, weak SRA), and again we observed fixation of the allele that benefits the homogametic sex.

Discussion

Alleviation of Sexual Antagonism via SRA

Our results indicate that sex ratio adjustment (SRA) can evolve in the presence of intralocus sexual conflict, diminishing sexually antagonistic fitness effects by increasing the match between offspring sex and the alleles benefiting that sex (e.g., fig. 2). Thus, SRA can be viewed as a mechanism by which sexual antagonism can be resolved (Alonzo and Sinervo 2007; Fawcett et al. 2007; Patten and Haig 2009), improving the fitness of both sexes (fig. 3). In this respect, our models corroborate a previous game theory analysis on side-blotched lizards *Uta stansburiana* by Alonzo and Sinervo (2007). We extend these findings by establishing an explicit genetic framework for the evolutionary dynamics and exploring the evolution of SRA under different modes of inheritance. The analytical models revealed that sexually antagonistic selection on SRA can be strong, proportional in strength to the product of the effect size of the modifier and the strength of selection on the trait. Our simulations corroborated these results: provided that sufficient genetic variation exists at the SRA locus (fig. 4), SRA for autosomal traits can evolve rapidly, increasing the mean fitness in each sex (fig. 3). However, we also find that differences in genetic architecture strongly

influence SRA evolution. In particular, SRA relieving sex-linked sexual antagonism is likely to persist only when based on the homogametic sex (female characteristics in XY systems and male characteristics in ZW systems; fig. 5*a*). The reciprocal pattern is likely to be transient (fig. 5*b*, 5*c*); systems lacking SRA may include cases where sex-linked polymorphisms have been lost because of the evolution of SRA based on traits in the heterogametic sex.

SRA alters gene expression at the level of the individual, manipulating whether an allele is expressed in a daughter or a son. While we have considered only a single gene, it is likely that multiple loci throughout the genome experience intralocus sexual antagonism, and future work would do well to examine how this affects the evolution of SRA. If a mother adjusts her brood sex ratio in response to only one of several sexually antagonistic traits, we expect that genetic variation for the other traits would act in a way similar to errors in genotype assessment: both, at least occasionally, would cause mothers to produce more of the less fit sex (because of a mistaken assessment of the genomewide advantage of producing daughters vs. sons or to an incorrect assessment of the **T**-locus genotype). On the other hand, if multiple polymorphic genes contribute to the same trait, then SRA could potentially evolve to alleviate sexual antagonism at more than one gene at the same time, strengthening selection for SRA. In any case, we expect that selection will continue to favor the evolution of SRA in the presence of multiple genes, as long as there remains a net sexually antagonistic fitness effect. Numerous empirical examples confirm the presence of net sexually antagonistic fitness effects with respect to individual traits (e.g., Rice 1996; Merila et al. 1997; Long and Rice 2007) or genomewide fitness differences (e.g., Chippindale et al. 2001; Gibson et al. 2002; Prasad et al. 2007; reviewed in Bonduriansky and Chenoweth 2009). Provided that females can detect and respond to this variation, we expect SRA to evolve as in our models.

Identifying Causes of SRA

Variation in patterns of SRA in nature might stem in part from the action of several different mechanisms, and distinguishing among alternatives remains a major empirical and theoretical challenge (Cockburn et al. 2002; West 2009). Toward this goal, our results indicate that the effects of sexual antagonism must be considered in addition to classic conditional sex allocation hypotheses. Few studies have measured the sex-specific fitness effects of traits subject to SRA, so it is unclear whether SRA commonly helps to alleviate sexually antagonistic selection in nature. However, several recent cases support the prediction that broods with skewed sex ratios will exhibit diminished sexually antagonistic fitness effects. Connallon and Jakubowski (2009) reported that in the fruit fly *Drosophila melanogaster*, females produced female-biased broods when mated to males who carried alleles with positive fitness effects in daughters. Similarly, in barn owls *Tyco alba*, parents possessing relatively small spots on their plumage are more likely to produce sons, mitigating the sexually antagonistic fitness effects associated with this trait (Roulin et al. 2010). A related pattern occurs in side-blotched lizards *Uta stansburiana* (Calsbeek and Sinervo 2004) and brown anoles *Anolis sagrei* (Calsbeek and Bonneaud 2008): although sex ratios within broods are typically balanced in both species, females have been shown to reduce sexual conflict with respect to body size by mating multiple times and then cryptically assigning sperm from relatively large sires to sons and that from small sires to daughters (Calsbeek and Sinervo 2002, 2004; Calsbeek and Bonneaud 2008).

While we have focused on SRA only in response to traits exhibiting sexually antagonistic selection, different forms of selection may act simultaneously, driving SRA in similar or opposite directions. For example, SRA that facilitates the transmission of an allele conferring attractiveness to sons (Burley 1981) should be strengthened if that trait also has sexually antagonistic effects, as depicted in our mate-SRA models. By contrast, mothers in good condition are expected to evolve increased investment in sons when additional resources disproportionately increase the fitness of sons relative to daughters (Trivers and Willard 1973), whereas our self-SRA models suggest this effect would be counteracted if female condition is based on sexually antagonistic selection. On the other hand, the selection pressures in this latter example would act in the same direction if additional resource investment disproportionately benefits daughters (see also Silk 1983; West 2009).

Disentangling the effects of multiple selection pressures on SRA evolution in an experimental context could entail varying the strength of individual selective forces and then tracking the evolution of SRA. In systems where this is not feasible, the issue could instead be approached empirically by measuring the relevant parameters (e.g., the strength of sexual selection on males, the impact of parental condition on offspring fitness, and selection coefficients in each sex) and determining the relative strength of selection on an SRA modifier arising from the different forces. Theoretical analyses like ours assist these approaches by supplying estimates of the direction and strength of selection on SRA modifiers resulting from a given form of selection (e.g., as given by eq. [3]). Similar estimates from models investigating other forms of selection are needed for a comprehensive theoretical framework in this respect. The sum total of the SRA changes expected under each force considered in isolation would provide an estimate of the rate and direction of SRA evolution,

supplemented by simulations to assess departures from this additive expectation.

Alternative Mechanisms to Alleviate Sexual Antagonism

Sexual antagonism is widespread (Chippindale et al. 2001; Foerster et al. 2007) and may affect diverse evolutionary processes (Parker and Partridge 1998; Bonduriansky and Chenoweth 2009; van Doorn 2009), highlighting the need to identify mechanisms that affect its strength and persistence (Bonduriansky and Chenoweth 2009). Sexually dimorphic trait expression is considered the principal means to resolve intralocus sexual conflict (Lande 1980; Rice 1984; Cox and Calsbeek 2009; van Doorn 2009), where dimorphism arises via the evolution of genomic imprinting or modifiers that result in sex-limited expression (reviewed in Ellegren and Parsch 2007; Bonduriansky and Chenoweth 2009). SRA represents an alternative potential strategy toward this end, biasing the inheritance of sexually antagonistic traits such that daughters are more likely to inherit alleles that are favored in females and vice versa for sons. SRA might be particularly important in this regard if the evolution of sexual dimorphism is constrained: for example, if the genes underlying the trait are highly correlated between sexes (Lande 1980) or have pleiotropic functions (Badyaev 2002; Ellegren and Parsch 2007; van Doorn 2009). Disparities in nature where the observed degree of sexual dimorphism does not completely remove sexually antagonistic selection suggest that such constraints might be common (Cox and Calsbeek 2009). For example, male yellow-pine chipmunks *Tamias amoenus* are close to their optimal body size, but females are displaced from their optimum (Schulte-Hostedde et al. 2002), whereas, in house finches *Carpodacus mexicanus*, neither sex expresses optimal body size (Badyaev and Martin 2000). Numerous other examples exist (Forsman 1995; Björklund and Senar 2001; reviewed in Cox and Calsbeek 2009), and they include cases in which sexual dimorphism is absent despite the presence of sexual antagonism (Merila et al. 1997). Indeed, Cox and Calsbeek (2009) provide evidence that residual sexual antagonism is widespread even among species exhibiting strong sexual dimorphism, suggesting that traits may frequently be displaced from their optimal value in each sex (see also Roulin et al. 2010). Hence, SRA might often have the opportunity to evolve in conjunction with sexual dimorphism, mitigating sexually antagonistic selection to a greater degree than either mechanism alone.

Conclusions

Our study indicates that sexually antagonistic selection poses a potent source of selection for differential sex ratio allocation and that, in turn, SRA evolution can help to diminish sexual conflict. The impact of SRA on sexual antagonism likely varies among populations according to the genetic variation available at potential modifier loci, the mode of inheritance of sexually antagonistic traits, the action of other putative selective pressures on SRA, and the potential for alternative strategies to mediate the conflicting fitness pressures on daughters and sons (e.g., sex-limited gene expression, genomic imprinting). We expect that consideration of these factors will help to better explain variation in patterns of SRA in nature.

Acknowledgments

We would like to thank T. W. Fawcett for sharing simulation details and D. E. Irwin, W. P. Maddison, L. K. M'Gonigle, and three anonymous reviewers for helpful comments on the manuscript. We are also grateful to R. G. FitzJohn, W. P. Maddison, and L. K. M'Gonigle for computer support. Funding was provided by the Natural Sciences and Engineering Research Council of Canada through a Discovery Grant to W. P. Maddison (G.S.B.), a postdoctoral fellowship (A.Y.K.A.), and a Discovery Grant (S.P.O.).

Literature Cited

Aars, J., H. P. Andreassen, and R. A. Ims. 1995. Root voles: litter sex ratio variation in fragmented habitat. Journal of Animal Ecology 64:459–472.

Albert, A. Y. K., and S. P. Otto. 2005. Sexual selection can resolve sex-linked sexual antagonism. Science 310:119–121.

Alonzo, S. H., and B. Sinervo. 2007. The effect of sexually antagonistic selection on adaptive sex ratio allocation. Evolutionary Ecology Research 9:1097–1117.

Badyaev, A. V. 2002. Growing apart: an ontogenetic perspective on the evolution of sexual size dimorphism. Trends in Ecology & Evolution 17:369–378.

Badyaev, A. V., and T. E. Martin. 2000. Sexual dimorphism in relation to current selection in the house finch. Evolution 54:987–997.

Björklund, M., and J. C. Senar. 2001. Sex differences in survival selection in the serin, *Serinus serinus*. Journal of Evolutionary Biology 14:841–849.

Bodmer, W. F., and A. W. F. Edwards. 1960. Natural selection and the sex ratio. Annals of Human Genetics 24:239–244.

Bonduriansky, R., and S. F. Chenoweth. 2009. Intralocus sexual conflict. Trends in Ecology & Evolution 24:280–288.

Burley, N. 1981. Sex ratio manipulation and selection for attractiveness. Science 211:721–722.

———. 1986. Sex-ratio manipulation in color-banded populations of zebra finches. Evolution 40:1191–1206.

Burton-Chellew, M. N., T. Koevoets, B. K. Grillenberger, E. M. Sykes, S. L. Underwood, R. Bijlsma, J. Gadau, et al. 2008. Facultative sex ratio adjustment in natural populations of wasps: cues of local mate competition and the precision of adaptation. American Naturalist 172:393–404.

Calsbeek, R., and C. Bonneaud. 2008. Postcopulatory fertilization bias as a form of cryptic sexual selection. Evolution 62:1137–1148.

Calsbeek, R., and B. Sinervo. 2002. Uncoupling direct and indirect

components of female choice in the wild. Proceedings of the National Academy of Sciences of the USA 99:14897–14902.

———. 2004. Within-clutch variation in offspring sex determined by differences in sire body size: cryptic mate choice in the wild. Journal of Evolutionary Biology 17:464–470.

Chippindale, A. K., J. R. Gibson, and W. R. Rice. 2001. Negative genetic correlation for adult fitness between sexes reveals ontogenetic conflict in *Drosophila*. Proceedings of the National Academy of Sciences of the USA 98:1671–1675.

Clutton-Brock, T. H., and G. R. Iason. 1986. Sex ratio variation in mammals. Quarterly Review of Biology 61:339–374.

Clutton-Brock, T. H., S. D. Albon, and F. E. Guinness. 1984. Maternal dominance, breeding success and birth sex ratios in red deer. Nature 308:358–360.

———. 1986. Great expectations: dominance, breeding success and offspring sex ratios in red deer. Animal Behaviour 34:460–471.

Cockburn, A., S. Legge, and M. C. Double. 2002. Sex ratios in birds and mammals: can the hypotheses be disentangled? Pages 266–286 *in* I. C. W. Hardy, ed. Sex ratios: concepts and research methods. Cambridge University Press, Cambridge.

Connallon, T., and E. Jakubowski. 2009. Association between sex ratio distortion and sexually antagonistic fitness consequences of female choice. Evolution 63:2179–2183.

Cox, R. M., and R. Calsbeek. 2009. Sexually antagonistic selection, sexual dimorphism, and the resolution of intralocus sexual conflict. American Naturalist 173:176–187.

Davison, M. J., and S. J. Ward. 1998. Prenatal bias in sex ratios in a marsupial, *Antechinus agilis*. Proceedings of the Royal Society B: Biological Sciences 265:2095–2099.

Desfor, K. B., J. J. Boomsma, and P. Sunde. 2007. Tawny owls *Strix aluco* with reliable food supply produce male-biased broods. Ibis 149:98–105.

Dieckmann, U., and R. Law. 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. Journal of Mathematical Biology 34:579–612.

Ellegren, H., and J. Parsch. 2007. The evolution of sex-biased genes and sex-biased gene expression. Nature Reviews Genetics 8:689–698.

Fawcett, T. W., B. Kuijper, I. Pen, and F. J. Weissing. 2007. Should attractive males have more sons? Behavioral Ecology 18:71–80.

Fisher, R. A. 1930. The genetical theory of natural selection. Clarendon, Oxford.

Flanagan, K. E., S. A. West, and H. C. J. Godfray. 1998. Local mate competition, variable fecundity and information use in a parasitoid. Animal Behaviour 56:191–198.

Foerster, K., T. Coulson, B. C. Sheldon, J. M. Pemberton, T. H. Clutton-Brock, and L. E. B. Kruuk. 2007. Sexually antagonistic genetic variation for fitness in red deer. Nature 447:1107–1109.

Forsman, A. 1995. Opposing fitness consequences of colour pattern in male and female snakes. Journal of Evolutionary Biology 8:53–70.

Frayer, D. W., and M. H. Wolpoff. 1985. Sexual dimorphism. Annual Review of Anthropology 14:429–473.

Gibson, J. R., A. K. Chippindale, and W. R. Rice. 2002. The X chromosome is a hot spot for sexually antagonistic fitness variation. Proceedings of the Royal Society B: Biological Sciences 269:499–505.

Glucksmann, A. 1974. Sexual dimorphism in mammals. Biological Reviews of the Cambridge Philosophical Society 49:423–475.

Gowaty, P. A. 1993. Differential dispersal, local resource competition, and sex ratio variation in birds. American Naturalist 141:263–280.

Hamilton, W. D. 1967. Extraordinary sex ratios. Science 156:477–488.

Hardy, I. C. W. 2002. Sex ratios: concepts and research methods. Cambridge University Press, Cambridge.

Herre, E. A. 1985. Sex ratio adjustment in fig wasps. Science 228: 896–898.

Kolman, W. A. 1960. The mechanism of natural selection for the sex ratio. American Naturalist 94:373–377.

Komdeur, J., S. Daan, J. Tinbergen, and C. Mateman. 1997. Extreme adaptive modification in sex ratio of the Seychelles warbler's eggs. Nature 385:522–525.

Krackow, S. 1995. Potential mechanisms for sex ratio adjustment in mammals and birds. Biological Reviews of the Cambridge Philosophical Society 70:225–241.

Kruuk, L. E. B., T. H. Clutton-Brock, S. D. Albon, J. M. Pemberton, and F. E. Guinness. 1999. Population density affects sex ratio variation in red deer. Nature 399:459–461.

Lande, R. 1980. Sexual dimorphism, sexual selection, and adaptation in polygenic characters. Evolution 34:292–305.

Long, T. A. F., and A. Pischedda. 2005. Do female *Drosophila melanogaster* adaptively bias offspring sex ratios in relation to the age of their mate? Proceedings of the Royal Society B: Biological Sciences 272:1781–1787.

Long, T. A. F., and W. R. Rice. 2007. Adult locomotory activity mediates intralocus sexual conflict in a laboratory-adapted population of *Drosophila melanogaster*. Proceedings of the Royal Society B: Biological Sciences 274:3105–3112.

Mank, J. E. 2009. Sex chromosomes and the evolution of sexual dimorphism: lessons from the genome. American Naturalist 173: 141–150.

Mank, J. E., E. Axelsson, and H. Ellegren. 2007. Fast-X on the Z: rapid evolution of sex-linked genes in birds. Genome Research 17: 618–624.

Merila, J., B. C. Sheldon, and H. Ellegren. 1997. Antagonistic natural selection revealed by molecular sex identification of nestling collared flycatchers. Molecular Ecology 6:1167–1175.

Parker, G. A., and L. Partridge. 1998. Sexual conflict and speciation. Philosophical Transactions of the Royal Society B: Biological Sciences 353:261–274.

Patten, M. M., and D. Haig. 2009. Parental sex discrimination and intralocus sexual conflict. Biology Letters 5:667–670.

Prasad, N. G., S. Bedhomme, T. Day, and A. K. Chippindale. 2007. An evolutionary cost of separate genders revealed by male-limited evolution. American Naturalist 169:29–37.

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

———. 1987. The accumulation of sexually antagonistic genes as a selective agent promoting the evolution of reduced recombination between primitive sex chromosomes. Evolution 41:911–914.

———. 1996. Sexually antagonistic male adaptation triggered by experimental arrest of female evolution. Nature 381:232–234.

Rice, W. R., and A. K. Chippindale. 2001. Intersexual ontogenetic conflict. Journal of Evolutionary Biology 14:685–693.

Roulin, A., R. Altwegg, H. Jensen, I. Steinsland, and M. Schaub. 2010. Sex-dependent selection on an autosomal melanic female ornament promotes the evolution of sex ratio bias. Ecology Letters 13: 616–626.

Schulte-Hostedde, A. I., J. S. Millar, and H. L. Gibbs. 2002. Female-

biased sexual size dimorphism in the yellow-pine chipmunk (*Tamias amoenus*): sex-specific patterns of annual reproductive success and survival. Evolution 56:2519–2529.

Servanty, S., J. M. Gaillard, D. Allaine, S. Brandt, and E. Baubet. 2007. Litter size and fetal sex ratio adjustment in a highly polytocous species: the wild boar. Behavioral Ecology 18:427–432.

Sheldon, B. C. 1998. Recent studies of avian sex ratios. Heredity 80: 397–402.

Shine, R. 1989. Ecological causes for the evolution of sexual dimorphism: a review of the evidence. Quarterly Review of Biology 64: 419–461.

Silk, J. B. 1983. Local resource competition and facultative adjustment of sex ratios in relation to competitive abilities. American Naturalist 121:56–66.

Trivers, R. L., and D. E. Willard. 1973. Natural selection of parental ability to vary the sex ratio of offspring. Science 179:90–92.

Uller, T., I. Pen, E. Wapstra, L. W. Beukeboom, and J. Komdeur. 2007. The evolution of sex ratios and sex-determining systems. Trends in Ecology & Evolution 22:292–297.

van Doorn, G. S. 2009. Intralocus sexual conflict. Pages 52–71 *in* C. D. Schlichting and T. A. Mousseau, eds. The year in evolutionary biology 2009. Annals of the New York Academy of Sciences 1168. Wiley-Blackwell, Oxford.

Vicoso, B., and B. Charlesworth. 2006. Evolution on the X chromosome: unusual patterns and processes. Nature Reviews Genetics 7:645–653.

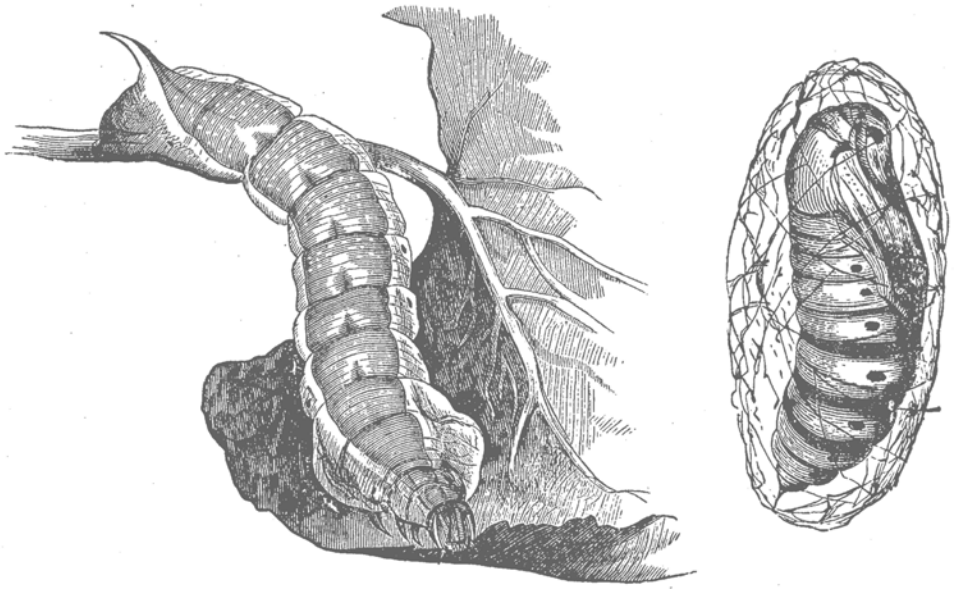
West, S. A. 2009. Sex allocation. Princeton University Press, Princeton, NJ.

West, S. A., and B. C. Sheldon. 2002. Constraints in the evolution of sex ratio adjustment. Science 295:1685–1688.

West, S. A., S. E. Reece, and B. C. Sheldon. 2002. Sex ratios. Heredity 88:117–124.

West, S. A., D. M. Shuker, and B. C. Sheldon. 2005. Sex-ratio adjustment when relatives interact: a test of constraints on adaptation. Evolution 59:1211–1228.

Associate Editor: Stuart West
Editor: Donald L. DeAngelis



Vine dresser and pupa (*Choerocampa pampinatrix*), "a single caterpillar of which will sometimes strip a small vine of its leaves in a few nights and sometimes nips off bunches of half-grown grapes." From "Review of Second Annual Report on the Noxious, Beneficial and Other Insects, of the State of Missouri" (*American Naturalist*, 1870, 4:610–615).

 DOI: 10.1086/655220

Appendix A from G. S. Blackburn et al., "The Evolution of Sex Ratio Adjustment in the Presence of Sexually Antagonistic Selection" (Am. Nat., vol. 176, no. 3, p. 264)

Developing Recursion Equations

To illustrate how the recursions were developed for an SRA modifier in the face of sexually antagonistic selection, we focus on the case of X linkage, which is simpler to display than the autosomal case. Table A1 lists the available genotypes in the population, in terms of the SRA modifier (**M**) and sexually antagonistic trait (**T**), when loci are X-linked.

We assume natural selection or sexual selection acts only at **T**. The frequency of each female genotype after selection and before mating is determined by its relative fitness, $x_n^{\mathrm{s}} = x_n W_n / \overline{W}_{\mathrm{f}}$, where W_n is the fitness of genotype x_n and

$$\overline{W}_{\mathrm{f}} = x_1 + x_2(1 - h_{\mathrm{f}}s_{\mathrm{f}}) + x_3(1 - s_{\mathrm{f}}) + x_4 + x_5(1 - h_{\mathrm{f}}s_{\mathrm{f}}) + x_6(1 - h_{\mathrm{f}}s_{\mathrm{f}}) + x_7(1 - s_{\mathrm{f}}) + x_8 + x_9(1 - h_{\mathrm{f}}s_{\mathrm{f}}) + x_{10}(1 - s_{\mathrm{f}})$$

is the mean fitness across all female genotypes. For example, the frequency of *TM*/*tM* after selection becomes $x_2^{\mathrm{s}} = x_2(1 - h_{\mathrm{f}}s_{\mathrm{f}})/\overline{W}_{\mathrm{f}}$. The frequency after selection of each male genotype is similarly given by $y_n^{\mathrm{s}} = y_n W_n / \overline{W}_{\mathrm{m}}$, with a mean male fitness of

$$\overline{W}_{\mathrm{m}} = y_1(1 - s_{\mathrm{m}}) + y_2 + y_3(1 - s_{\mathrm{m}}) + y_4.$$

We assume that, once selection has acted on the genotypes, parents mate randomly, produce offspring, and die. Thus, recursion equations representing the frequency of each genotype in females after one generation of reproduction (x_1') consist of the sum of all pairwise parental gametic combinations that can produce that genotype, subject to the rate of recombination (r) between **M** and **T** in mothers during gametogenesis and the probability D_{ij} that mothers with genotype i and referring to genotype j (either her own [self-SRA; table 2] or her mate's [mate-SRA; table B2] genotype) produce a daughter. For example, in the case of mate-SRA, *TM*/*TM* daughters are produced by the adult mating combinations *TM*/*TM* × *TM*, *TM*/*tM* × *TM*, *TM*/*Tm* × *TM*, *TM*/*tm* × *TM*, and *Tm*/*tM* × *TM* at a total frequency of

$$x_1' = x_1^{\mathrm{s}}y_1^{\mathrm{s}}D_{00} + \frac{1}{2}x_2^{\mathrm{s}}y_1^{\mathrm{s}}D_{00} + \frac{1}{2}x_4^{\mathrm{s}}y_1^{\mathrm{s}}D_{10} + \frac{1}{2}x_5^{\mathrm{s}}(1 - r)y_1^{\mathrm{s}}D_{10} + \frac{1}{2}(x_6^{\mathrm{s}}r)y_1^{\mathrm{s}}D_{10}.$$

Similarly, *TM*/*tM* daughters occur at a frequency of

$$x_2' = x_3^{\mathrm{s}}y_1^{\mathrm{s}}D_{00} + x_1^{\mathrm{s}}y_2^{\mathrm{s}}D_{01} + \frac{1}{2}x_2^{\mathrm{s}}y_1^{\mathrm{s}}D_{00} + \frac{1}{2}x_7^{\mathrm{s}}y_1^{\mathrm{s}}D_{10} + \frac{1}{2}x_2^{\mathrm{s}}y_2^{\mathrm{s}}D_{01} + \frac{1}{2}x_4^{\mathrm{s}}y_2^{\mathrm{s}}D_{11} + \frac{1}{2}x_6^{\mathrm{s}}(1 - r)y_1^{\mathrm{s}}D_{10} + \frac{1}{2}(x_5^{\mathrm{s}}r)y_1^{\mathrm{s}}D_{10} + \frac{1}{2}x_5^{\mathrm{s}}(1 - r)y_2^{\mathrm{s}}D_{11} + \frac{1}{2}(x_6^{\mathrm{s}}r)y_2^{\mathrm{s}}D_{11}.$$

The same logic applies to males. For example, *TM* sons occur at a frequency of

$$
\begin{aligned}
y_1' = {} & x_1^s[y_1^s(1 - D_{00}) + y_2^s(1 - D_{01}) + y_3^s(1 - D_{00}) + y_4^s(1 - D_{01})] \\
& + \frac{1}{2}x_2^s[y_1^s(1 - D_{00}) + y_2^s(1 - D_{01}) + y_3^s(1 - D_{00}) + y_4^s(1 - D_{01})] \\
& + \frac{1}{2}x_4^s[y_1^s(1 - D_{10}) + y_2^s(1 - D_{11}) + y_3^s(1 - D_{10}) + y_4^s(1 - D_{11})] \\
& + \frac{1}{2}x_5^s(1 - r)[y_1^s(1 - D_{10}) + y_2^s(1 - D_{11}) + y_3^s(1 - D_{10}) + y_4^s(1 - D_{11})] \\
& + \frac{1}{2}x_6^s r[y_1^s(1 - D_{10}) + y_2^s(1 - D_{11}) + y_3^s(1 - D_{10}) + y_4^s(1 - D_{10})].
\end{aligned}
$$

The same method is used to generate recursion equations for the remaining eight female and three male genotypes listed in table A1. Analogous methods are used to construct recursion equations for Z-linked **M** and **T** loci, but in this case the genotypes listed in table A1 are switched between sexes. Finally, in the case of autosomal loci, both male and female genotypes resemble those listed for females in table A1, and recombination occurs in both parents. All recursion equations are presented and analyzed in the Mathematica file at http://www.zoology.ubc.ca/~otto/Research/BlackburnEtAl2010.nb.

Table A1
Female and male genotypes with X-linked trait and modifier loci

Female genotype	Male genotype
$x_1 = TM/TM$	$y_1 = TM$
$x_2 = TM/tM$	$y_2 = tM$
$x_3 = tM/tM$	$y_3 = Tm$
$x_4 = TM/Tm$	$y_4 = tm$
$x_5 = TM/tm$	
$x_6 = Tm/tM$	
$x_7 = tM/tm$	
$x_8 = Tm/Tm$	
$x_9 = Tm/tm$	
$x_{10} = tm/tm$	

 DOI: 10.1086/655220

Appendix B from G. S. Blackburn et al., "The Evolution of Sex Ratio Adjustment in the Presence of Sexually Antagonistic Selection" (Am. Nat., vol. 176, no. 3, p. 264)

Polymorphic Equilibria for Sex-Linked Traits

The equilibrium frequency of T when the **T** locus is sex-linked, $\hat{p}_T$, is given in table B1 for different forms of SRA, assuming that both SRA and selection are weak (derivations are presented in the Mathematica file at http://www.zoology.ubc.ca/~otto/Research/BlackburnEtAl2010.nb).

The appropriate values of D_{ij} are given by table 2 for cases in which SRA depends on the homogametic individual's genotype (self-SRA with X linkage, mate-SRA with Z linkage). The formulas for these cases depend on H, which represents the dominance of the SRA strategy in response to a Tt genotype ($H = (D_{01} - D_{02})/(D_{00} - D_{02})$), and θ (see eq. [2]), which describes the relationship between $\overline{D}$, the mean sex ratio in the population, and ΔD, the difference in SRA between responses to a TT and a tt genotype (i.e., $D_{00} - D_{02}$). Table B2 should be consulted for cases in which SRA is based on the heterogametic sex (mate-SRA with X linkage and self-SRA with Z linkage). The formulas in these cases depend on the difference in SRA between responses to a T and a t genotype in a hemizygous individual ($\Delta D_{\text{hemi}} = D_{00} - D_{01}$). Note that when sex ratios are altered in response to the heterogametic sex, the equilibrium frequency of T depends strongly on the SRA strategy via the term ΔD_{hemi}, even if the population sex ratio remains balanced ($\theta = 0$). The effect of SRA on the equilibrium trait frequency reflects transmission distortion, which is biased against the allele favored in the heterogametic sex.

A stability analysis was performed for each polymorphic equilibrium, with the assumption that the M allele is fixed in the population. In each case the equilibrium is stable only if its denominator is positive.

Table B1
Equilibrium frequency, $\hat{p}_T$, of a sex-linked trait in the presence of SRA

	Self-SRA	Mate-SRA
X-linked	$\dfrac{(1-h_f)s_f - (1/2)s_m - H\theta}{(12h_f)s_f + (12H)\theta}$	$\dfrac{(1-h_f)s_f - (1/2)s_m + \Delta D_{\text{hemi}}/(2\overline{D})}{(12h_f)s_f}$
Z-linked	$\dfrac{(1/2)s_f - h_m s_m - \Delta D_{\text{hemi}}/[2(1-\overline{D})]}{(12h_m)s_m}$	$\dfrac{(1/2)s_f - h_m s_m - H\theta}{(12h_m)s_m + (12H)\theta}$

Table B2
SRA in the presence of a hemizygous trait and diploid modifier

	Female trait (self-SRA) or male trait (mate-SRA)	
Female modifier	T	t
MM	D_{00}	D_{01}
Mm	D_{10}	D_{11}
mm	D_{20}	D_{21}

 DOI: 10.1086/655220

Appendix C from G. S. Blackburn et al., "The Evolution of Sex Ratio Adjustment in the Presence of Sexually Antagonistic Selection" (Am. Nat., vol. 176, no. 3, p. 264)

Invasion of an SRA Modifier at Sex-Linked Loci

Here, we summarize results for sex-linked traits in the same format as presented for autosomally linked traits in the main text. Complete analyses can be found in the Mathematica file at http://www.zoology.ubc.ca/~otto/Research/BlackburnEtAl2010.nb.

X-Linked Loci

In the case of an X-linked trait and SRA based on the homogametic sex (self-SRA), sex ratio allocation evolves in a qualitatively similar manner to the autosomal model, with the following slight changes. (1) For self-SRA with an X-linked trait and an X-linked modifier, the leading eigenvalue, λ, is given by equation (3), but with h_m set to 1/2 and the F_1 and F_2 terms multiplied by 4/3. (2) For self-SRA with an X-linked trait and an autosomal modifier, the leading eigenvalue, λ, is given by equation (3) but with h_m set to 1/2 and the F_2 term multiplied by 6/5.

Thus, we again expect offspring sex ratios to be adjusted in favor of daughters among *TT* mothers and in favor of sons among *tt* mothers. As long as the population sex ratio remains near 1/2, stronger and stronger SRA is expected to evolve, and the equilibrium (table B1) will remain valid and stable throughout this process.

In contrast, with mate-SRA and an X-linked trait, selection on the modifier changes form because of the transmission distortion caused by adjusting offspring sex ratios based on the trait of the heterogametic sex. If the modifier is also X-linked, then the leading eigenvalue becomes

$$\lambda = 1 - \frac{2}{3}\frac{[\overline{D} - (1/2)]}{\overline{D}(1-\overline{D})}\{\hat{p}_T(D_{10} - D_{00}) + \hat{p}_t(D_{11} - D_{01})\}$$
$$+ \frac{1}{3}\hat{p}_T\hat{p}_t\left(s_m - \frac{\Delta D_{hemi}}{\overline{D}}\right)\{(D_{10} - D_{00}) - (D_{11} - D_{01})\} + O(\xi^3). \quad \text{(C1)}$$

Initially, if the population sex ratio is balanced ($\overline{D} = 1/2$) and there is no SRA ($\Delta D_{hemi} = 0$), the second line dominates equation (C1), and SRA should evolve in the expected direction. Specifically, modifiers can invade if they cause mothers to produce more daughters when mated with *T*-bearing males ($D_{10} - D_{00} > 0$) and more sons when mated with *t*-bearing males ($D_{11} - D_{01} < 0$, as defined in table B2). As stronger SRA evolves, it can be shown that the term $s_m - \Delta D_{hemi}/\overline{D}$ remains positive as long as the trait polymorphism is maintained. Thus, equation (C1) indicates that more and more extreme SRA should evolve (assuming that the population-wide sex ratio remains near 1/2). At some point, however, the evolution of SRA causes ΔD_{hemi} to become so large that the trait polymorphism is lost because of the transmission distortion favoring *T*-bearing X chromosomes. This occurs when the equation for $\hat{p}_T$ given in table B1 for mate-SRA with X linkage reaches 1.

If, instead, the modifier is autosomal with mate-SRA and an X-linked trait, then the leading eigenvalue becomes

$$\lambda = 1 - \frac{1}{2}\frac{[\overline{D} - (1/2)]}{\overline{D}(1-\overline{D})}\{\hat{p}_T(D_{10} - D_{00}) + \hat{p}_t(D_{11} - D_{01})\}$$
$$+ \frac{1}{5}\hat{p}_T\hat{p}_t\left(s_m - 2\frac{\Delta D_{hemi}}{\overline{D}}\right)\{(D_{10} - D_{00}) - (D_{11} - D_{01})\} + O(\xi^3). \quad \text{(C2)}$$

If the population sex ratio is balanced ($\overline{D} = 1/2$) and there is no SRA ($\Delta D_{\text{hemi}} = 0$), SRA evolves as in equation (C1). Again, as stronger SRA evolves, the transmission distortion can become so strong that the trait polymorphism is lost. It is also possible, however, that as stronger SRA evolves, the term $s_{\text{m}} - 2\Delta D_{\text{hemi}}/\overline{D}$ can switch signs, even under conditions that maintain the trait polymorphism. Thus, more extreme SRA should evolve only up to an intermediate level, given by $\Delta D_{\text{hemi}} = s_{\text{m}}/4$ (assuming $\overline{D} = 1/2$), at which point further increases in SRA are selected against, even though the trait remains polymorphic. A numerical analysis suggests that when $\Delta D_{\text{hemi}} \approx s_m/4$, a new modifier allele that further increases SRA (producing more daughters in matings with *T*-bearing males and/or more sons in matings with *t*-bearing males) becomes genetically associated with *TT* females, which experience only a modest selective advantage over *Tt* females, and with *T* males, which are strongly selected against. The association with *T*-bearing males occurs because the new modifier experiences a stronger transmission bias against X′ in matings with X′Y males. Thus, further increases in SRA become increasingly opposed by natural selection, halting the evolution of further SRA despite the fact that a sexually antagonistic polymorphism remains.

Z-Linked Loci

In the case of a Z-linked trait, an autosomal modifier, and SRA based on the homogametic sex (self-SRA), the leading eigenvalue governing the spread of a new modifier allele is

$$\lambda = 1 - \frac{1}{2}\frac{[\overline{D} - (1/2)]}{\overline{D}(1 - \overline{D})}\{\hat{p}_T(D_{10} - D_{00}) + \hat{p}_t(D_{11} - D_{01})\}$$
$$+ \frac{1}{5}\hat{p}_T\hat{p}_t\left[s_{\text{f}} - 2\frac{\Delta D_{\text{hemi}}}{(1 - \overline{D})}\right]\{(D_{10} - D_{00}) - (D_{11} - D_{01})\} + O(\xi^3), \tag{C3}$$

where all D_{ij} are given by table B2, with self-SRA and $\Delta D_{\text{hemi}} = D_{00} - D_{01}$. As stronger SRA evolves, the term $s_{\text{m}} - 2\Delta D_{\text{hemi}}/(1 - \overline{D})$ can become negative, even under conditions that maintain the trait polymorphism. Thus, equation (C3) indicates that more extreme SRA should evolve up to an intermediate level, given by $\Delta D_{\text{hemi}} = s_{\text{f}}/4$ (assuming $\overline{D} = 1/2$), at which point further increases in SRA are selected against, even though the trait remains polymorphic. As SRA evolves and ΔD_{hemi} becomes more positive, the system equilibrates at a point $\hat{p}_T$ where the *t* allele declines, on average, across the sexes because of natural selection but increases in frequency because of transmission distortion in its favor. A numerical analysis suggests that when $\Delta D_{\text{hemi}} = s_{\text{f}}/4$, a new modifier allele that further increases SRA (producing more daughters among *T*-bearing mothers and/or more sons among *t*-bearing mothers) becomes genetically associated with *tt* males, which experience only a modest selective advantage over *Tt* males, and with *t*-bearing females, which are strongly selected against. Thus, further increases in SRA become increasingly opposed by natural selection, halting the evolution of further SRA despite the fact that a sexually antagonistic polymorphism remains.

In contrast, if there is mate-SRA with a Z-linked trait and an autosomal modifier, this transmission bias does not arise, because the mate is ZZ and passes each Z chromosome equitably to both sons and daughters. In this case, the evolution of offspring sex ratios is qualitatively similar to the autosomal case, with the following slight changes. The leading eigenvalue, λ, is given by equation (3) but with h_{m} set to 1/2 and the F_2 term multiplied by $6s_{\text{f}}/5s_{\text{m}}$.

Thus, we again expect offspring sex ratios to be adjusted in favor of daughters among *T*-bearing mothers and in favor of sons among *t*-bearing mothers. As long as the population sex ratio remains near 1/2, stronger and stronger SRA is expected to evolve, and the equilibrium (table B1) will remain valid and stable throughout this process.