Sperm Competition and the Dynamics of X Chromosome Drive: Stability and Extinction

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ABSTRACT

Several empirical studies of sperm competition in populations polymorphic for a driving X chromosome have revealed that Sex-ratio males (those carrying a driving X) are at a disadvantage relative to Standard males. Because the frequency of the driving X chromosome determines the population-level sex ratio and thus alters male and female mating rates, the evolutionary consequences of sperm competition for sex chromosome meiotic drive are subtle. As the *SR* allele increases in frequency, the ratio of females to males also increases, causing an increase in the male mating rate and a decrease in the female mating rate. While the former change may exacerbate the disadvantage of Sex-ratio males during sperm competition, the latter change decreases the incidence of sperm competition within the population. We analyze a model of the effects of sperm competition on a driving X chromosome and show that these opposing trends in male and female mating rates can result in two coexisting locally stable equilibria, one corresponding to a balanced polymorphism of the *SR* and *ST* alleles and the second to fixation of the *ST* allele. Stochastic fluctuations of either the population sex ratio or the *SR* frequency can then drive the population away from the balanced polymorphism and into the basin of attraction for the second equilibrium, resulting in fixation of the *SR* allele and extinction of the population.

ALTHOUGH a fair meiosis is required to maximize the mean fitness in a population (Charlesworth and Hartl 1978), individual alleles can gain a transmission advantage via meiotic drive. Hence, the genome has been selected to suppress such drive (Leigh 1977; Crow 1991). Nevertheless, despite the genome-wide selection against drive, individual alleles occasionally do escape such regulation, with potentially disastrous consequences for a population. When present on one of the sex chromosomes, a drive allele not only reduces mean fitness, but also results in biased sex ratios, both within individual families and at the level of entire populations (Bryant *et al.* 1982; James and Jaenike 1990). If unchecked by countervailing selection, these drive alleles may spread to fixation and result in extinction of an entire population or species (Gershenson 1928; Hamilton 1967; Lyttle 1977).

In most cases of sex chromosome meiotic drive, males that carry a particular type of X chromosome, denoted *SR* (Sex-ratio), sire primarily or exclusively female offspring, although Y drive in males and X drive in heterogametic females have also been documented. Sex chromosome meiotic drive has been documented in plants, mammals, numerous species of Diptera, and perhaps one species of butterfly (reviewed in Jaenike 2001).

Because of the capacity of driving sex-linked alleles to cause extinction, there has been considerable interest in factors holding the spread of these alleles in check. The explanations fall into four general categories. First, following the early empirical studies of Wallace (1948), a stable polymorphism may be maintained by a balance between the advantage gained through meiotic drive in males and the deleterious effects of drive alleles on female fitness (Edwards 1961; Curtsinger and Feldman 1980). One difficulty with this as a general explanation is that X drive in males is caused by genes that affect spermatogenesis (*e.g.*, Cazemajor *et al.* 2000; Wilkinson and Sanchez 2001), and there is no *a priori* reason to expect such genes to have any particular effect on females. The effects seen in females could well be due to deleterious alleles linked to the drive loci within inversions; in many cases, X drive is associated with one or more inversions.

A second possibility requires a metapopulation structure, with a global equilibrium frequency of a driving X chromosome maintained by a balance between extinction and founding of local populations (Wallace 1968). The spread of a driving X within a local population decreases its expected time to extinction, thus reducing its genetic contribution to the global population. This hypothesis has not been formally modeled for X chromosome drive. Furthermore, studies of temporal and spatial variation in frequencies of *SR* do not provide any empirical support for this scenario.

The third general mechanism by which a stable polymorphism may be achieved is via the evolution of autosomal or Y-linked suppressors of X drive (Wu 1983a; Jae-

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nike 1996; Carvalho *et al.* 1997). While such suppressors have been found in many species, it is not clear that they alone can stabilize the polymorphism. More importantly, the speed with which a driving X chromosome can spread to fixation leaves little time for the evolution of suppressors before the population is in danger of extinction.

Finally, the difficulties with the previous explanations raise the question of whether any pleiotropic effects in males could account for the polymorphism. The spread of *SR* results in an increasingly female-biased population and thus a likely increase in the rate of male mating and greater sperm depletion. Because Sex-ratio males in several species that have been studied cytologically produce about one-half as many functional sperm as do Standard (nondrive) males (Policansky and Ellison 1970; Hauschteck-Jungen and Maurer 1976; Cazemajor *et al.* 2000; Wilkinson and Sanchez 2001), the greater rate of male mating at high *SR* frequencies could take a greater toll on the fertility of Sex-ratio males. While such effects may slow the spread of *SR*, for this alone to stabilize the polymorphism requires a particular relationship between sperm numbers and offspring production (Jaenike 1996).

While empirical studies provide limited support for all of these models, these studies have also revealed another factor—sperm competition in multiply mated females—that may have profound effects on the dynamics of X chromosome drive. In experimental populations of *Drosophila pseudoobscura*, *SR* frequencies declined much more rapidly in populations where females were allowed to remate than in those where they mated only once (Beckenback 1983). Analyses of offspring production by individual multiply mated females show that sperm from Sex-ratio males is inferior to that of Standard males in sperm competition (Wu 1983b). Similar effects have subsequently been found in all other species that have been examined, including *D. simulans* (Capillon and Atlan 1999), *D. neotestacea* (James 1992), the stalk-eyed fly *Cyrtodiopsis whitei* (G. S. Wilkinson, personal communication), and the plant *Silene alba*, in which pollen competition reduces offspring production by Sex-ratio males (Taylor *et al.* 1999).

While it thus appears that reduced transmission due to sperm competition may be an important factor preventing the spread of *SR* in natural populations, it is far less clear how this might stabilize the polymorphism. As noted above, the spread of *SR* results in an increasingly female-biased population and is therefore likely to result in a decrease in the rate of female mating. Thus, sperm competition in multiply mated females will become less frequent as an agent preventing further spread of *SR*. If anything, one might expect sperm competition to be a destabilizing factor in the dynamics of the polymorphism.

Wu (1983b) made an important empirical observation that may be key to understanding *SR* dynamics when sperm competition adversely affects Sex-ratio males.

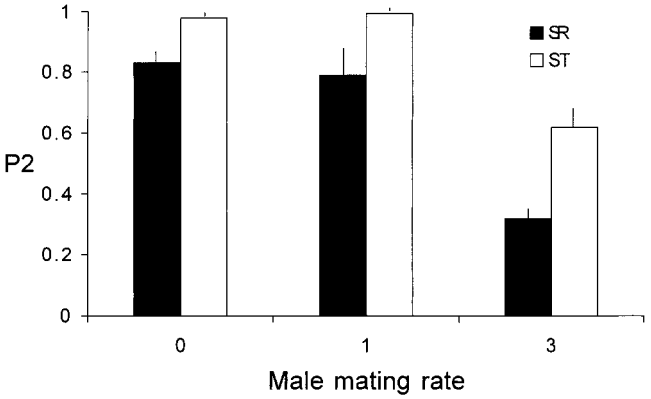

Figure 1.—Effect of male genotype (*ST* or *SR*) and male mating rate per day on *P*2, the fraction of males sired by the second male in a doubly mated female. Females were first mated to males homozygous for *vermillion*. Data are from Wu (1983b).

Specifically, he mated individual females of *D. pseudoobscura* first to males homozygous for a recognizable phenotypic marker (*vermillion*) and then to either a Sex-ratio or Standard male carrying the wild-type allele at the vermillion locus. By scoring offspring eye color, he determined *P*2, the fraction of offspring sired by the second male in these multiply mated females. Wu found that *P*2 declined as a function of male mating rate substantially faster for Sex-ratio males than for Standard males (Figure 1). Thus, the outcome of sperm competition between Sex-ratio and Standard males is likely to depend on the male mating rate.

How might this affect the dynamics of the Sex-ratio polymorphism? When *SR* is rare, and thus there are equal numbers of males and females, the male mating rate is expected to be relatively low, so that Sex-ratio males will not suffer greatly in sperm competition. However, the resulting spread of *SR* brings about a more female-biased population, a greater male mating rate, and consequently reduced transmission in multiply mated females. It is thus possible that sperm competition could stabilize a *SR* polymorphism. However, beyond a certain frequency of *SR*, the ratio of females to males will become so high that multiple mating by females is unlikely, thus removing sperm competition as a factor checking the spread of *SR*. Accordingly, there may be a frequency of *SR* beyond which a driving X chromosome will spread to fixation and cause a population's extinction. In this article, we formally model this scenario and show that sperm competition can lead to coexisting stable equilibria, one corresponding to a balanced polymorphism at the drive locus and the other corresponding to fixation of the *SR* allele.

MODEL AND RESULTS

We consider the dynamics of an X-linked locus with two alleles, one of which (*SR*) causes X chromosome drive in males, while the other (*ST*) allows equal trans-

mission of the X and Y chromosomes. Sex-ratio males thus sire exclusively female progeny, whereas *ST* males sire both male and female offspring in a 1:1 ratio. In the following model, we associate a cost with the *SR* allele, which is delayed frequency dependent and realized through sperm competition within the reproductive tracts of females inseminated by both Standard and Sex-ratio males. We refer to this type of selection as delayed frequency dependent because, as shown below, the strength of selection against Sex-ratio males depends on the male mating rate, which is a function of the sex ratio of the population. In turn, the population-level sex ratio depends on the frequency of *SR* in the previous generation. However, the selective effect is delayed by a generation and is susceptible to modification by other factors such as gender-dependent mortality or migration.

The model that we explore here was developed to illustrate general dynamic properties of the Sex-ratio polymorphism, rather than to mimic the biology of any particular species. Thus, we have incorporated several simplifying assumptions about the mating biology of our hypothetical species. First, we assume that sperm competition occurs between only two males at a time and that a female does not begin oviposition until she has mated with the second male. We also assume a population with discrete generations and without adult age structure, so that male and female fertility is independent of age. Finally, we assume that priority has no effect on the outcome of sperm competition. That is, for a female that mates with a Sex-ratio and a Standard male, the fraction of offspring sired by the Sex-ratio male does not depend on whether he mated first or second.

We assume that the *SR* allele has no phenotypic manifestation in females and that Standard and Sex-ratio males are of equal viability. We first assume that in the absence of sperm competition the two types of males have equal fertility and later consider how a reduction in Sex-ratio male fertility in the absence of sperm competition affects the dynamics.

To describe the state of the population at a time t we use a three-dimensional vector (f, p_m, p_f), where f is the fraction of the population that is female, p_m is the frequency of the *SR* allele among males, and p_f is the corresponding frequency among females. We also find it useful to introduce three related variables: m, the male fraction of the population, q_m, the frequency of the *ST* allele among males, and q_f, the frequency of *ST* among females. The biological interpretation of these variables and all model parameters are summarized in Table 1.

The transmission rate of the *SR* allele relative to the *ST* allele depends both on the severity of sperm competition within a female's reproductive tract and on the frequency with which sperm competition occurs. We quantify the severity of sperm competition as $c = c(r_m)$, which is the expected proportion of the offspring produced by a single female inseminated by both male types that are sired by the Sex-ratio male, and we allow this quantity to depend on the expected number of matings, r_m, per individual male. If $c < \frac{1}{2}$, then Sex-ratio males are inferior to Standard males in sperm competition. To specify how the frequency of sperm competition depends on the population sex ratio, we set forth expressions for the probabilities of single (π_1) and multiple (π_2) matings by females (conditioned on their mating at least once) and for the probabilities that multiple mating involves two males of the same genotype or two males of different genotypes. Throughout we assume that females mate at most twice, but we take the maximal number of matings per male, denoted γ, as one of the parameters of our model.

TABLE 1

Model parameters and variables

Symbol	Interpretation
f	Proportion of females in population
m	Proportion of males in population
p_m/p_f	Frequency of *SR* allele in males/females
q_m/q_f	Frequency of *ST* allele in males/females
γ	Maximal individual male mating rate
r_m/r_f	Individual male/female mating rate
π_i, $i = 1, 2$	Probability that a mated female mates i times
$c(r_m)$	Proportion of offspring of female mated by a Sex-ratio male and a Standard male that are sired by the Sex-ratio male
$\alpha(f, p_m)$	Offspring production of Sex-ratio males relative to Standard males
β	Baseline Sex-ratio male fertility

By accounting for the frequencies of all possible crosses and the frequencies of the sexes and genotypes of the resulting offspring, we find that the values of f', p'_m, and p'_f depend on those in the previous generation as follows:

$$f' = \pi_1 p_m + \frac{1}{2}\pi_1 q_m + \pi_2 p_m^2 + \frac{1}{2}\pi_2 q_m^2 + 2\pi_2 p_m q_m\left(\frac{1}{2}(1 - c) + c\right) \quad (1)$$

$$p'_m = p_f \quad (2)$$

$$p'_f = \frac{1}{f'}\left[\pi_1 p_m p_f + \frac{1}{2}\pi_1 p_m q_f + \frac{1}{4}\pi_1 q_m p_f + \pi_2 p_m^2 p_f + \frac{1}{2}\pi_2 p_m^2 q_f + \frac{1}{4}\pi_2 q_m^2 p_f + \frac{1}{2}\pi_2 p_m q_m p_f(1 - c) + 2\pi_2 p_m q_m p_f c + \pi_2 p_m q_m q_f c\right]. \quad (3)$$

Equations 1–3 can be simplified by introducing a function $\alpha = \alpha(f, p_m)$, defined by

$$\alpha(f, p_m) = \frac{c\pi_2 + 1/2\pi_1 + p_m\pi_2(1/2 - c)}{1/2\pi_2 + 1/2\pi_1 + p_m\pi_2(1/2 - c)} = \frac{\pi_1 + \pi_2(p_m + 2cq_m)}{\pi_1 + \pi_2(q_m + 2(1 - c)p_m)}. \quad (4)$$

From the second of these two formulations, we see that

α can be interpreted as the offspring production of Sex-ratio males relative to Standard males. Equations 1–3 then take the form

$$f' = \frac{1}{2}\left(\frac{1 + (2\alpha - 1)p_m}{1 + (\alpha - 1)p_m}\right) \quad (5)$$

$$p'_m = p_f \quad (6)$$

$$p'_f = \frac{1}{2}p_f + \left(\frac{\alpha}{1 + (2\alpha - 1)p_m}\right)p_m, \quad (7)$$

which we can write succinctly as $(f', p'_m, p'_f) = F(f, p_m, p_f)$, where F is a mapping of the three-dimensional unit cube into itself. (Throughout we leave the arguments of the functions α and c implicit when these are clear from context.)

We next specify how the incidence of multiple mating depends on the male-to-female ratio m/f in the population. The total potential number of matings in the population is the maximum number of matings possible for either sex, assuming unlimited availability of individuals of the opposite sex ready to mate. Taking N to be the total population size,

$$\begin{aligned}\text{total male potential matings} &= \gamma Nm\\ \text{total female potential matings} &= 2Nf,\end{aligned} \quad (8)$$

where γ is the maximum possible mating rate per individual male and the factor of 2 in the expression for female matings refers to the number of times an individual female is able to mate. The minimum of these two expressions determines the total number of matings possible in the population. To calculate the individual mating rates of the two sexes, we take this minimum and divide by the number of males or females:

$$\begin{aligned} r_m &= \min\left\{\gamma, 2\left(\frac{f}{m}\right)\right\}\\ r_f &= \min\left\{\gamma\left(\frac{m}{f}\right), 2\right\}.\end{aligned} \quad (9)$$

The importance of sperm competition depends on the prevalence of multiple mating by females, which in turn is a function of r_f. Because females mate at most twice, we note that $0 \le r_f \le 2$. We assume that if $r_f < 1$, then no females mate more than once and the probability that any given female mates exactly once is just r_f. If $r_f > 1$, then we suppose that each female mates at least once and that the probability that any given female mates exactly twice is $r_f - 1$. Recalling that π_i, $i = 1, 2$ denotes the probability that a female mates exactly i times (given that she does mate) we have

$$\begin{aligned}\pi_2 &= \max\{r_f - 1, 0\}\\ \pi_1 &= 1 - \pi_2 = \min\{2 - r_f, 1\}.\end{aligned} \quad (10)$$

The equation specifying π_2 indicates the existence of three different regimes, depending on the population-level sex ratio. In regime (i), $2/\gamma \le m/f$, and all females mate exactly twice ($\pi_1 = 0$, $\pi_2 = 1$) and are limiting for males. In regime (ii), $1/\gamma \le m/f \le 2/\gamma$, so that males mate at rate γ and some females mate fewer than two times ($\pi_1 = 2 - r_f$, $\pi_2 = r_f - 1$). Finally, in regime (iii), $m/f \le 1/\gamma$ and thus males are even more limiting for females, so that no female mates twice and some go unmated ($\pi_1 = 1$, $\pi_2 = 0$). Because α, the relative rate of *SR* transmission through males, depends on π_1 and π_2, each of these three regimes translates into a different expression for α, namely,

$$\alpha(f, p_m) = \begin{cases} \dfrac{c + p_m(1/2 - c)}{1/2 + p_m(1/2 - c)} & \text{if } \dfrac{2}{\gamma} \le \dfrac{m}{f}\\[2ex] \dfrac{(r_f - 1)\,c + (1 - 1/2r_f) + p_m(r_f - 1)(1/2 - c)}{(r_f - 1)1/2 + (1 - 1/2r_f) + p_m(r_f - 1)(1/2 - c)} & \text{if } \dfrac{1}{\gamma} \le \dfrac{m}{f} \le \dfrac{2}{\gamma}.\\[2ex] 1 & \text{if } \dfrac{m}{f} \le \dfrac{1}{\gamma}\end{cases} \quad (11)$$

Thus, as the relative proportion of males in the population declines, as would occur during spread of the *SR* allele, the population may, for a given value of γ, shift progressively from regime (i) to regime (ii) to regime (iii).

Equilibria: The equilibria of the dynamical system are obtained by solving the equation $(f, p_m, p_f) = F(f, p_m, p_f)$. Referring to Equations 5–7, we see that at any fixed point, $(\hat{f}, \hat{p}_m, \hat{p}_f)$, we have $\hat{p}_m = \hat{p}_f \equiv \hat{p}$ and that either $\alpha(\hat{f}, \hat{p}) = \frac{1}{2}$ or $\hat{p} = 0$ or 1. Generically, there are two equilibria lying on the boundary of the three-dimensional unit cube, namely

$$(\hat{f}, \hat{p}, \hat{p}) = \left(\frac{1}{2}, 0, 0\right) \text{ or } (1, 1, 1). \quad (12)$$

The first of these describes a population in which the *SR* allele is completely absent, whereas the second of these describes a population fixed for *SR* (and hence extinct, although because our model realizes this state as a fixed point, we need not implement an *ad hoc* demographic component to detect extinction). The internal equilibria correspond to fixed points $(\hat{f}, \hat{p}, \hat{p})$ at which $\alpha(\hat{f}, \hat{p}) = \frac{1}{2}$ and transmission of the *SR* allele through *SR* males and the *ST* allele through *ST* males are equivalent. Substituting $\frac{1}{2}$ for α wherever that appears in the recursion for f in Equations 4–6, we find that at such equilibria $\hat{f} = 1/(2 - \hat{p})$. Because α varies between the three regimes, we need to consider solutions of $\alpha(\hat{f}, \hat{p}) = \frac{1}{2}$ for each of these. In regime (iii), $\alpha \equiv 1$ so that there are no such solutions and hence no internal equilibria.

To find the equilibria present in regime (i), we first note that at an equilibrium $r_m = 2(\hat{f}/\hat{m}) = 2/(1 - \hat{p})$ and then set the expression given in Equation 11 equal to ½. Solving for $c(r_m)$ in terms of $\hat{p}$, we find

$$c(r_m) = c\left(\frac{2}{1-\hat{p}}\right) = \frac{1}{2}\left(\frac{1-\hat{p}}{2-\hat{p}}\right). \quad (13)$$

By setting $z = 2/(1 - \hat{p})$, this implicit equation can be recast as

$$c(z) = \frac{1}{z+2}. \quad (14)$$

All regime (i) equilibria can be found by solving for the roots of Equation 14 and then setting $\hat{p} = 1 - 2/z$ for each such root z. The requirement that these solutions belong to regime (i) imposes the constraint that $\hat{m}/\hat{f} \geq 2/\gamma$ and, using the known relationships between z, $\hat{p}$, and $\hat{f}$, this constraint is equivalent to both $z \in [2, \gamma]$ and $\hat{p} \in [0, 1 - 2/\gamma]$. We note in passing that all intersections of the two curves $c(z)$ and $1/(z + 2)$ within the specified region correspond to distinct equilibria, so that the number of regime (i) equilibria can in principle be uncountably infinite (for example, if $c(z) = 1/(z + 2)$ for all z).

We next determine the regime (ii) equilibria. Noting that $r_m = \gamma$ and that $r_f = \gamma(m/f)$ and setting $\alpha = ½$ and $c = c(\gamma)$, we obtain a quadratic equation in terms of $\hat{p}$, whose roots are

$$p_\pm = \frac{1}{\gamma(1-2c)}\left[3\left(\frac{1}{2}-c\right)\gamma + \left(c-\frac{1}{2}\right) \pm \sqrt{\left(3\left(\frac{1}{2}-c\right)\gamma + \left(c-\frac{1}{2}\right)\right)^2 + 4\gamma\left(\frac{1}{2}-c\right)\left(2c\left(\gamma-1\right) + \left(\frac{3}{2}-\gamma\right)\right)}\right]. \quad (15)$$

This expression can be simplified by first expanding the products under the radical and then dividing through by $|(c - ½)|$, yielding

$$p_\pm = \mathrm{sign}\left(c-\frac{1}{2}\right)\frac{1}{2\gamma}\left[3\gamma - 1 \pm \sqrt{\gamma^2 + \left(\frac{3-2c}{1/2-c}\right)\gamma + 1}\right]. \quad (16)$$

If $c < ½$, then the expression under the radical is positive and it is clear that $p_+ > 1$ and thus is not a biologically relevant fixed point for our model. On the other hand, the expression under the radical is at least as large as γ^2 and since $\gamma \geq 1$, we see that $p_- \leq 1 - 1/2\gamma$. Regime (ii) corresponds to $\hat{p}$ values in the interval $[1 - 2/\gamma, 1]$ and so we require that $p_- \geq 1 - 2/\gamma$. Using the expression for p_- given in (16), this requirement is satisfied if and only if $c(\gamma) \leq 1/(\gamma + 2)$; *i.e.*, $c(r_m)$ must be on or below the curve $1/(z + 2)$ when $z = r_m = \gamma$.

Finally, we note that if the regime (i) equilibrium value of $\hat{p}$ is equal to $1 - 2/\gamma$, then $r_m = 2/(1 - \hat{p}) = \gamma$ and $c(r_m) = 1/(\gamma + 2)$. Solving for the regime (ii) equilibrium gives $p_- = 1 - 2/\gamma$; *i.e.*, the two equilibria coincide. This behavior is illustrated in Figure 2 for $c(z) = (½)^{bz}$ and $\gamma = 4$. The parameter b is a measure of the sensitivity of *SR* sperm competition to the population-level ratio of females to males ($z/2$), which determines the male mating rate. In this example, a unique regime (i) equilibrium exists for values of $b \in [¼(\ln(6)/\ln(2)), 1]$ and the regime (i) and regime (ii) equilibria collide at the left boundary of this interval. For values of b outside of this interval, the only equilibria are those on the boundary at (1, 1, 1) and (0.5, 0, 0). Whether a driving allele is able to invade a population and is then maintained in a stable polymorphism or else escapes to fixation depends on the stability properties of these various fixed points, which we now derive.

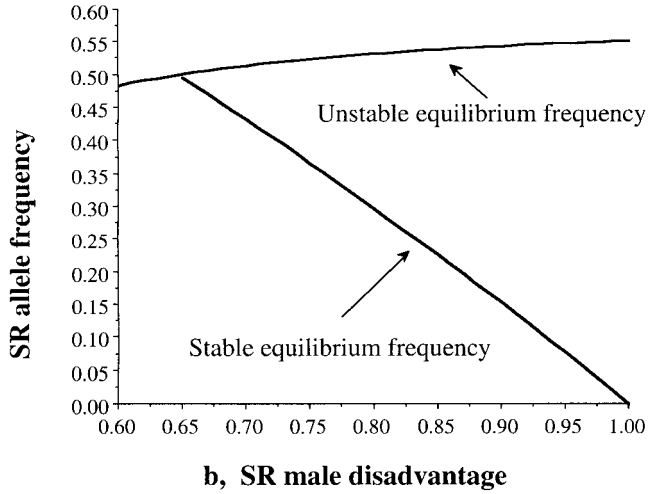

FIGURE 2.—Stable (p_s) and unstable (p_u) internal equilibria of the *SR* allele as a function of the severity of Sex-ratio male disadvantage in sperm competition with a Standard male. The boundary equilibria at $p = 0$ and $p = 1$ are not depicted. In this example the decline in Sex-ratio male fertility with male mating rate z is given by $c(z) = 0.5^{bz}$ and the maximal male mating rate is $\gamma = 4$. A stable internal equilibrium exists only for values of $b \in [¼(\ln(6)/\ln(2)), 1]$.

Local stability: Some insight into the behavior of the dynamical system given by Equations 4–6 can be gained by evaluating the local stability of the fixed points identified in the previous section. This analysis requires evaluation of the eigenvalues of the Jacobian matrix of the mapping F, which is given by

$$\begin{pmatrix} \frac{\partial f'}{\partial f} & \frac{\partial f'}{\partial p_m} & 0 \\ 0 & 0 & 1 \\ \frac{\partial p_f'}{\partial f} & \frac{\partial p_f'}{\partial p_m} & \frac{1}{2} \end{pmatrix}. \quad (17)$$

The four partial derivatives present in the Jacobian can be evaluated in terms of α and its partial derivatives (here denoted α_f and α_{p_m}) and after some simplification are

$$\frac{\partial f'}{\partial f} = \frac{1}{2}p_m(1 - p_m)\alpha_f\left(\frac{1}{1 + (\alpha - 1)p_m}\right)^2$$

$$\frac{\partial f'}{\partial p_m} = \frac{1}{2}(p_m(1 - p_m)\alpha_{p_m} + \alpha)\left(\frac{1}{1 + (\alpha - 1)p_m}\right)^2$$

$$\frac{\partial p_f'}{\partial f} = p_m(1 - p_m)\alpha_f\left(\frac{1}{1 + (2\alpha - 1)p_m}\right)^2$$

$$\frac{\partial p_f'}{\partial p_m} = (p_m(1 - p_m)\alpha_{p_m} + \alpha)\left(\frac{1}{1 + (2\alpha - 1)p_m}\right)^2. \quad (18)$$

To proceed further, we need the partial derivatives of the function α, whose value in turn depends on which regime the system is in. In regime (i), it can be shown that

$$\alpha_f = \frac{c'(r_m)}{(1 - f)^2}\left(\frac{1}{1/2 + p_m(1/2 - c_{r_m})}\right)^2$$

$$\alpha_{p_m} = \left(\frac{1/2 - c(r_m)}{1/2 + p_m(1/2 - c_{r_m})}\right)^2. \quad (19)$$

Likewise, in regime (ii), we have

$$\alpha_f = \frac{\gamma(1/2 - c(r_m))}{2f^2}\left(\frac{1}{(r_f - 1)1/2 + (1 - 1/2r_f) + p_m(r_f - 1)(1/2 - c(r_m))}\right)$$

$$\alpha_{p_m} = \left(\frac{(r_f - 1)(1/2 - c(r_m))}{(r_f - 1)1/2 + (1 - 1/2r_f) + p_m(r_f - 1)(1/2 - c(r_m))}\right). \quad (20)$$

The eigenvalues of the Jacobian of F at each fixed point can then be found by solving for the roots of the characteristic polynomial, $\chi_J(\lambda) = \det(J - \lambda I)$, where I is the three-by-three identity matrix. We first consider the two boundary equilibria, (½, 0, 0) and (1, 1, 1). For the first of these we define $\alpha_0 = 2c(2)$ and then note that the characteristic polynomial of the Jacobian is $-\lambda(\lambda^2 - \frac{1}{2}\lambda - \alpha_0)$, with roots $\lambda = 0$ and $\lambda_\pm = \frac{1}{2}(\frac{1}{2} \pm \sqrt{\frac{1}{4} + 4\alpha_0})$. For (½, 0, 0) to be stable, it is necessary that $|\lambda_\pm| < 1$, which holds if and only if $c(2) < \frac{1}{4}$. If this inequality is satisfied, then the cost of sperm competition for a Sex-ratio male competing with a population of Standard males is sufficiently high that it exceeds the twofold advantage gained from the meiotic drive and prevents invasion of the population by the driving *SR* allele. For the equilibrium corresponding to fixation of the *SR* allele (and hence extinction) the characteristic polynomial is $-\lambda(\lambda^2 - \frac{1}{2}\lambda - \frac{1}{4})$ and the eigenvalues are $\lambda = 0$ and $\lambda_\pm = \frac{1}{4} \pm \frac{1}{4}\sqrt{5}$. In this case, all three of the eigenvalues are <1 in modulus and so this equilibrium is always stable.

We now consider the stability of the two types of internal equilibria. There are two simplifications of the characteristic polynomial that facilitate this analysis. Recall that at internal equilibria, $\alpha = \frac{1}{2}$. We show that at these same equilibria $\alpha_{p_m} = \frac{1}{4}$. In regime (i), we have

$$\alpha_{p_m} = \left(\frac{1/2 - c(r_m)}{1/2 + p_m(1/2 - c(r_m))}\right)^2$$

$$= \left(\frac{1/2 + p_m(1/2 - c(r_m)) - (c(r_m) + p_m(1/2 - c(r_m)))}{1/2 + p_m(1/2 - c(r_m))}\right)^2$$

$$= (1 - \alpha)^2$$

$$= \frac{1}{4}.$$

A similar calculation establishes the same result for regime (ii). We next observe that the characteristic polynomial factors into the product of a quadratic polynomial and a monomial. From the general form of the Jacobian given in Equation 17, we find that the characteristic polynomial is

$$\det(J - \lambda I) = -\lambda^3 + \left(\frac{1}{2} + \frac{\partial f'}{\partial f}\right)\lambda^2 + \left(\frac{\partial p_f'}{\partial p_m} - \frac{1\partial f'}{2\,\partial f}\right)\lambda - \left(\frac{\partial f'}{\partial f}\frac{\partial p_f'}{\partial p_m} - \frac{\partial f'}{\partial p_m}\frac{\partial p_f'}{\partial f}\right). \quad (21)$$

Substitution of the expressions for the partial derivatives of F into the constant term in the characteristic polynomial shows that this term in fact vanishes, allowing us to write

$$\det(J - \lambda I) = -\lambda\left(\lambda^2 - \left(\frac{1}{2} + \frac{\partial f'}{\partial f}\right)\lambda - \left(\frac{\partial p_f'}{\partial p_m} - \frac{1\partial f'}{2\,\partial f}\right)\right). \quad (22)$$

The roots of this simplified equation are $\lambda = 0$ and

$$\lambda_\pm = \frac{1}{2}\left[\frac{1}{2} + \frac{\partial f'}{\partial f} \pm \sqrt{\left(\frac{1}{2} - \frac{\partial f'}{\partial f}\right)^2 + 4\frac{\partial p_f'}{\partial p_m}}\right]. \quad (23)$$

For regime (i) equilibria, we calculate

$$\frac{\partial f'}{\partial f} = 2\left(\frac{\hat{p}}{1 - \hat{p}}\right)(2 - \hat{p})^2 c'(r_m)$$

$$\frac{\partial p_f'}{\partial p_m} = \frac{1}{2} + \frac{1}{4}\hat{p}(1 - \hat{p}).$$

Substituting these into the expression for $\lambda_\pm$ gives

$$\lambda_\pm = \frac{1}{4} + \left(\frac{\hat{p}}{1 - \hat{p}}\right)(2 - \hat{p})^2 c'(r_m) \pm \sqrt{9 + 4\hat{p}(1 - \hat{p}) - 8\left(\frac{\hat{p}}{1 - \hat{p}}\right)(2 - \hat{p})^2 c'(r_m) + 16\left(\frac{\hat{p}}{1 - \hat{p}}\right)^2 (2 - \hat{p})^4 c'(r_m)^2}. \quad (24)$$

We now derive conditions on $c = c(r_m)$, which are necessary and sufficient for stability of regime (i) equilibria. Because the expression under the radical in Equation 24 is nonnegative, the eigenvalues $\lambda_\pm$ are real and so stability is equivalent to the condition $-1 < \lambda_- \leq \lambda_+ < 1$. Because the middle inequality is satisfied automatically, it suffices to determine the conditions under which the outer two hold. A little algebra shows that

$$\lambda_+ < 1 \quad \text{iff } c'(r_m) < -\frac{1}{4}\left(\frac{1-\hat{p}}{2-\hat{p}}\right)^2 \tag{25}$$

and that

$$\lambda_- > -1 \quad \text{iff } c'(r_m) > \frac{1}{3}\left[\frac{4}{\hat{p}(1-\hat{p})} - 1\right]\left(-\frac{1}{4}\left(\frac{1-\hat{p}}{2-\hat{p}}\right)^2\right). \tag{26}$$

These conditions can be interpreted geometrically, as follows. Define $\phi(z) = 1/(z+2)$ and recall that regime (i) equilibria are in one-to-one correspondence with the intersections of the curves $c(z)$ and $\phi(z)$ over the interval $[0, 1 - 2/\gamma]$. Differentiating ϕ at the point $r_m = 2/(1-\hat{p})$ gives

$$\phi'(r_m) = -\left(\frac{1}{r_m+2}\right)^2 = -\frac{1}{4}\left(\frac{1-\hat{p}}{2-\hat{p}}\right)^2 \equiv \xi. \tag{27}$$

Then the conditions for stability of the regime (i) equilibrium $(\hat{f}, \hat{p}, \hat{p})$ can be rewritten as

$$\frac{1}{3}\left[\frac{4}{\hat{p}(1-\hat{p})} - 1\right]\xi < c'(r_m) < \xi. \tag{28}$$

Thus, for stability of the equilibrium corresponding to an intersection of the curves c and ϕ, the slope of c at that point must be more negative than that of ϕ, but not too negative. One consequence of this condition is that, provided c is a continuous function, equilibria corresponding to adjacent intersections of the two curves cannot both be stable. Likewise, in the degenerate case where c is coincident with ϕ so that there is an entire interval of regime (i) equilibria, we will have $\lambda_+ = 1$ and all such equilibria will be neutrally stable. For the more realistic cases, however, these results show that there may be a stable equilibrium frequency of the *SR* allele at a relatively low frequency.

The regime (ii) equilibria are always unstable. To see this, observe that

$$\frac{\partial f'}{\partial f} = \frac{1}{2}\hat{p}(1-\hat{p})\alpha_f\left(\frac{1}{1-1/2\hat{p}}\right)^2$$

$$\frac{\partial p_2'}{\partial p_1} = \frac{1}{2} + \frac{1}{4}\hat{p}(1-\hat{p}).$$

Algebraic manipulation of Equation 23 then shows that

$$\lambda_+ > 1 \quad \text{iff } 4\hat{p}(1-\hat{p}) > -8\frac{\partial f'}{\partial f}. \tag{29}$$

However, in regime (ii), $\alpha_f > 0$ and hence $\partial f'/\partial f > 0$. It follows that $\lambda_+ > 1$ and thus regime (ii) equilibria, whenever they exist, are unstable. Thus, a spread of *SR* can bring about a change in the male/female ratio in the population, resulting in a shift from regime (i) to regime (ii), which in turn will allow the *SR* allele to spread unchecked to fixation.

Baseline reductions in *SR* fertility: Even in the absence of sperm competition and frequent male mating, some empirical studies have shown that Sex-ratio males are less fertile than Standard males (*e.g.*, Policansky 1979; Wu 1983a; Jaenike 1996). The above model can be generalized to take into account a baseline reduction in Sex-ratio male fertility by multiplying the function α by a constant $\beta \in [0, 1]$. Since the derivations and calculations do not differ substantially from those encountered in the preceding sections, we present only the results of our analysis here.

The boundary equilibria are unchanged, but their stability now depends on β. The point $(\frac{1}{2}, 0, 0)$ is stable if and only if $c(2) < 1/4\beta$, while the equilibrium at $(1, 1, 1)$ is stable provided $\beta > \frac{1}{2}$. In contrast, both the positions and stability properties of the two types of internal equilibria vary with β. Regime (i) equilibria are still defined implicitly by the intersections of two curves $c(z)$ and $\phi(z)$. Taking $z = 2/(1-\hat{p})$, $(1/(2-\hat{p}), \hat{p}, \hat{p})$ is a regime (i) equilibrium if and only if

$$c(z) = \frac{(1-\beta)z - (1-2\beta)}{z + 2(2\beta - 1)} \tag{30}$$

and $\hat{p} = 1 - 2/z \in [0, 1 - 2/\lambda]$. As before, the stability of regime (i) equilibria can be expressed in terms of the derivatives of $c(z)$ and $\phi(z)$ at their points of intersection. Writing $\phi(z) = (1-\beta)z - (1-2\beta)/(z + 2(2\beta - 1))$, let ξ denote the derivative of ϕ at a value of z corresponding to an equilibrium frequency $\hat{p}$ and note that $\xi = -(2\beta-1)^2/4\,((1-\hat{p})/(2\beta - (2\beta-1)\hat{p}))^2$. Then the equilibrium is stable provided that $c'(r_m)$ satisfies the two inequalities,

$$\frac{1}{3}\left[\frac{\beta}{(2\beta-1)^2}\frac{4}{\hat{p}(1-\hat{p})} - 1\right]\xi < c'(r_m) < \xi. \tag{31}$$

Finally, regime (ii) equilibria are still obtained from one of the roots of a quadratic equation and have the form

$$\hat{p} = \frac{1}{2\gamma}\left[\left(\frac{4\beta-1}{2\beta-1}\right)\gamma - 1 - \sqrt{\left(\frac{\gamma}{2\beta-1}\right)^2 + \left(\frac{2c - 4\beta + 1}{c - 1/2}\right)\left(\frac{\gamma}{2\beta-1}\right) + 1}\right]. \tag{32}$$

These equilibria remain unstable for all values of the parameters.

Global dynamics: The analyses presented above determine all equilibria of the dynamical system presented in Equations 1–3 and determine the local stability properties of these. When an equilibrium is locally stable, we know that there exists a neighborhood of that equilibrium with the property that any trajectory of the system started within that neighborhood converges to the equilibrium. Thus, for those values of the parameters for which a stable internal equilibrium exists, we know that sperm competition is sufficient to maintain a polymorphism at the driving locus and that if the system is

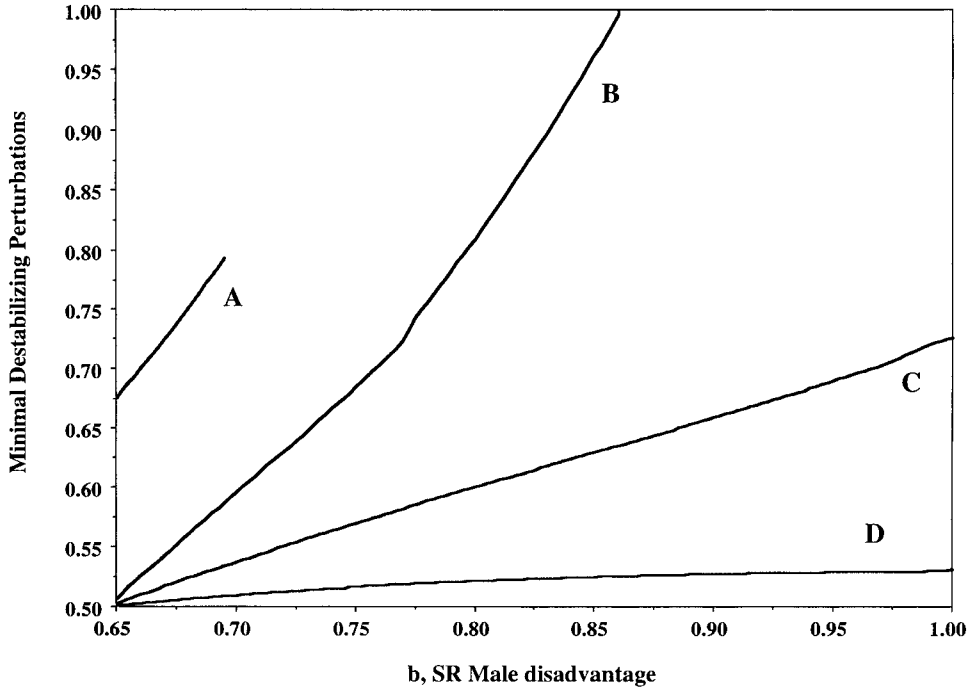

FIGURE 3.—Minimal perturbations of the state variables away from the stable internal equilibrium that result in fixation of the *SR* allele. A curve is shown for each state variable when individually perturbed (A–C) and a fourth curve (D) is shown for concomitant perturbation of the male and female frequencies of *SR*. A, B, and C correspond to the state variables f, p_m, and p_f, respectively. All unperturbed state variables were started at the corresponding equilibrium values. The minimal perturbation was determined by starting the perturbed state variable(s) at nonnegative multiples of 0.001 and finding the smallest of these (if any) such that the resulting numerical trajectory converges to (1, 1, 1). In this example the decline in Sex-ratio male fertility with male mating rate z is given by $c(z) = 0.5^{bz}$ and the maximal male mating rate is $\gamma = 4$. Observe that for two of the state variables, f and p_m, there exist values of the parameter b such that no destabilizing perturbations were found. The corresponding curves, A and B, are therefore not defined on the entire interval [0.65, 1].

initiated sufficiently close to that equilibrium, then sperm competition will cause it to subsequently converge to that equilibrium. Likewise, for those parameter values for which (1, 1, 1) is a stable equilibrium, perturbations that cause all three state variables (f, p_m, p_f) to exceed some threshold <1 will result in fixation of the *SR* allele.

However, these results do not guarantee that a population in which a *SR* allele initially appears at a low frequency will arrive at the balanced polymorphism, provided this exists, nor do they tell us the size of perturbations away from the balanced polymorphism required to fix the *SR* allele. To investigate these issues, we resorted to numerical iteration of the dynamical system specified by Equations 1–3, with the function $c(z)$ taken to be $\frac{1}{2}^{bz}$ and the maximal male mating rate γ set equal to 4. Recall that for this specification of $c(z)$, the local stability analysis shows that a stable internal equilibrium exists for any value of b in the interval $(1/4 \ln(6)/\ln(2), 1)$ (see Figure 2). For values of b less than the left endpoint of this interval, (0.5, 0, 0) is an unstable equilibrium and (1, 1, 1) is the unique stable equilibrium, while for values of b greater than the right endpoint, (1, 1, 1) is unstable and (0.5, 0, 0) is the sole stable equilibrium with nonnegative coordinates.

Numerical iteration of the dynamical system for values of b within the specified interval suggests that whenever the system is started at an initial point with all three state variables less than or equal to their corresponding values at the predicted stable internal equilibrium, the system subsequently converges to that equilibrium. These iterations were carried out with both the values of b and the coordinates of the initial points taken to be any nonnegative multiple of 0.01 consistent with the specified constraints. For example, if the value of f at the equilibrium was 0.031, then the initial values of f ranged over the set 0, 0.01, 0.02, 0.03. Likewise, for positive values of b outside of the interval for which stable internal equilibria exist, all iterations converged to the corresponding stable boundary equilibrium whenever the initial point was taken to lie in the interior of the state space. In no case did we observe trajectories suggestive of either periodic or chaotic dynamics, although we note that for certain choices of the function $c(z)$ [*e.g.*, such that $c(z)$ has multiple intersections with the curve $\phi(z) = 1/(z + 2)$] these may be possible.

In Figure 3, we plot the minimal perturbations of single state variables away from the stable internal equilibrium required to trigger fixation of the *SR* allele. These were found numerically for each of the three state variables by tracking the evolution of the dynamical system from an initial state with two of these variables started at their equilibrium values and with the remaining variable started at a value that was stepped up from 0 in increments of 0.001 until the smallest such value was found from which the resulting trajectory converges to (1, 1, 1). We observed numerically that for all nonnegative values of this third state variable less than that critical value the trajectory converges to the stable internal equilibrium, whereas for all values at or above the critical value the system converges to (1, 1, 1). Figure 3 also includes a similar plot of the minimal perturba-

tion of the population frequency of *SR* required to trigger convergence to (1, 1, 1) when the initial values of the male and female frequencies are equal and that of the population sex ratio is set at the equilibrium value.

In addition to providing some indication that the local stability analysis adequately describes the global dynamics of the SR system, at least for this choice of $c(z)$, Figure 3 shows that the susceptibility of a population to escape from a balanced polymorphism at the drive locus depends on which state variables are subject to the largest stochastic fluctuations. Increases in the population-level sex ratio can result in escape, but only for a limited range of parameter values in which the stable and unstable equilibria are situated near to one another in the phase space. This observation suggests that differences in mortality or migration between the sexes are unlikely to result in fixation of the *SR* allele. In contrast, increases in the frequency of the driving allele can lead to fixation over a larger set of parameter values, with concomitant male and female increases being most effective and increases among males alone being least effective. Increases among females only lead to fixation much more readily than do increases among males only, in part because each female carries two X chromosomes whereas each male carries only one X chromosome, the female contribution to the population frequency of X-linked alleles being that much greater. However, this same difference in copy number means that the effects of drift on the sex-specific frequencies of the *SR* allele will be greatest among males. Whether fluctuations in the frequency of *SR* among males or among females are more likely to trigger escape from the balanced polymorphism may therefore depend on the nonlinear structure of the dynamical system.

DISCUSSION

The model developed in this article was motivated by the observation that poor sperm (or pollen) competition seems to be a generic feature of X drive systems, having been found in plants and several species of Diptera (*e.g.*, Wu 1983b; James 1992; Capillon and Atlan 1999; Taylor *et al.* 1999; Wilkinson and Fry 2001). However, one might intuitively expect that *SR* dynamics governed by sperm competition would not be internally stable for two reasons. First, as the *SR* allele increases in frequency, the increasing female to male ratio in the population brings about a decrease in the incidence of multiple mating by females, leading to diminished levels of sperm competition. Second, the increasing frequency of Sex-ratio males in the population means that sperm competition, when it occurs, occurs less and less often between Sex-ratio and Standard males.

To address these seemingly contradictory results, we considered how variation in success in sperm competition as a function of the male mating rate would affect the dynamics. Our results show that if the success of sperm from Sex-ratio males declines with male mating rate, then sperm competition alone can be sufficient to stabilize the polymorphism and thus forestall the extinction event that is likely with unchecked meiotic drive on a sex chromosome. However, our model also demonstrates that this protection is not absolute and that suitably large perturbations of either the population sex ratio or of the frequency of the *SR* allele lead to extinction. Should the fraction of females in the population become too large, the frequency of multiple mating by individual females will decline to the point at which sperm competition becomes unimportant, thus releasing the *SR* allele from stabilizing selection and allowing it to spread to fixation.

Two biologically independent factors determine how sperm competition affects the dynamics of a driving X chromosome. The first depends on the effect of male mating rate on sperm competition. For there to be a stable *SR-ST* polymorphism, the sperm competitive ability of Sex-ratio males relative to Standard males must decline neither too slowly nor too rapidly as the male mating rate increases. In particular, a fixed difference in fertility between *SR* and *ST* males [$c(z) \equiv c \in (0, 1)$] that is realized only when there is sperm competition between the two types will not result in a stable polymorphism. Changes in the shape of the function c, possibly due to genetic changes in the population, may create or eliminate equilibria or stabilize or destabilize those already present. Given the central role played by c in these dynamics, empirical studies should focus on how the outcome of *SR-ST* sperm competition is affected by the male mating rate.

The second factor influencing *SR* dynamics is the dependence of male and female mating rates on the sex ratio of the population. In essence, as the ratio of females to males increases, the per capita rate of female mating is expected to decrease (as males become more limiting), while that for males increases. We have assumed that females can mate at most twice and that the variances in mating success for each sex are minimal given the expected mating rates specified by Equation 10. A model in which females were allowed to mate more than twice would be substantially more complicated than that analyzed here, but would retain stable and unstable internal equilibria as well as the stable equilibrium at (1, 1, 1). Thus, its dynamics would be qualitatively similar to those found in the simpler version that we chose to investigate. The same is true if we generalize the model by allowing greater variance in mating success. Although the formulation of α would change and could be made to vary smoothly with changes in the sex ratio, as long as the asymptotic behavior of the model for sex ratios close to 0 or 1 was similar to that investigated here, the dynamical behaviors would be similar as well. The conclusions drawn from our model will break down, of course, if mating rates are uncoupled from the sex ratio, as might happen, for

example, if male-female encounter rates, rather than gamete production, limit offspring production.

Once the system shifts into the basin of attraction of the fixed point at (1, 1, 1), numerical iterations of the model show that the *SR* allele is rapidly fixed, typically exceeding a frequency of 0.999 in <50 generations. Consequently, populations headed for immediate extinction due to X drive are unlikely to be observed in nature. There are, however, anecdotal reports of cage populations of Drosophila going all female and subsequently suffering extinction. Examples include *D. neotestacea* (D. Grimaldi, personal communication) and *D. quinaria* (L. Tompkins, personal communication). Although X chromosome drive was not known in either species when these observations were made, it was discovered subsequently in both of them (James and Jaenike 1990; Jaenike 1996). The dramatic change in sex ratio in these cage populations is exactly what would be expected for an *SR*-polymorphic system shifted into the (1, 1, 1) basin of attraction. Cage populations, because of their small size, are likely to experience greater stochastic fluctuations in *SR* frequency and mating rate and thus may be much more vulnerable than natural populations to shifts away from the stable internal equilibrium. It would be interesting to start large cage populations at several initial frequencies of *SR* to determine whether in fact there is a region in which fixation of the *SR* allele is driven deterministically.

What conditions in nature are likely to promote the fixation of a sex-ratio distorter previously kept in check by sperm competition? In our model, escape from the internal equilibrium requires a decrease in the male mating rate that is sufficient to limit the frequency with which individual females mate with both a Sex-ratio and a Standard male. This could be brought about by a change in the population-level sex ratio, with a higher female/male ratio reducing the rate of multiple mating by females, or by a change in population density or environmental conditions that limits the encounter rates of males and females. In small populations, various types of demographic stochasticity could bring about shifts in the population-level sex ratio. If the stable regime (i) equilibrium is sufficiently close to the basin of attraction for fixation of *SR*, genetic drift at the *SR* locus or sampling variance for the X *vs.* Y chromosomes contributed by males may cause a shift from a stable polymorphism to fixation of *SR*.

More importantly, we suspect, changes in population density may also play a role in triggering the fixation of sex-ratio distorters. A decline in population density may relax selection against *SR* in two ways, both of which depend on the rate at which individuals mate. First, as population density decreases, the incidence of multiple mating by females is expected to decline. Thus, there is likely to be less sperm competition at low population density. Second, at low population density, the rate of encounter between males and females may be sufficiently low that the rate of male mating is also depressed, regardless of the population-level sex ratio. Recall that it is the increasing male mating rate in our model that plays a key role in checking the spread of *SR*. For both of these reasons, therefore, we expect that populations may be especially vulnerable to increases in *SR* frequency when environmental conditions cause reductions in population density. In support of this idea, James (1992) found that the success of wild-caught *D. neotestacea* Sex-ratio males in laboratory assays of sperm competition increased over a 1-month period of declining population density in the wild.

If poor performance in sperm competition is a pleiotropic correlate of *SR*, then the effect of sperm competition on *SR* dynamics is likely to apply throughout the history of a *SR* polymorphism within a species, from its initial appearance to final fixation, loss, or stabilization via additional mechanisms. These additional stabilizing mechanisms include the evolution of Y-linked and autosomal suppressors of X drive (Jaenike 1996; Carvalho *et al.* 1997) and the association of *SR* with inversions that tie up large blocks of the genome. Such inversions can be stably maintained, for instance, through heterozygote superiority, independently of the presence of the *SR* polymorphism. Elsewhere, we have argued that suppressors and inversions are a derived condition in *SR*-polymorphic species (Jaenike 1996). Even if sperm competition does result in a balanced polymorphism, selection for Fisherian sex ratios may subsequently recruit additional mechanisms that suppress X chromosome drive. Indeed, because frequency-dependent stabilization of the *SR* polymorphism leaves the population vulnerable to random demographic and environmental events that can lead to deterministic fixation of the *SR* allele and extinction, maintenance of the *SR* polymorphism in the long term is contingent on the appearance of alternative stabilizing mechanisms. This reasoning suggests that the role of sperm competition in stabilizing X chromosome drive may be transient, yet critical to the survival of a population or species invaded by driving sex chromosomes. Given sufficient time, suppressors or inversions linking deleterious mutations to the distorter may arise, but in a newly exposed population these controls are unlikely to be present. Pleiotropic effects of drive, in particular those revealed in sperm competition, provide a plausible explanation as to how an otherwise rapidly fixing *SR* allele can be stabilized in the absence of a preexisting mechanism that is biologically independent of the drive locus.

In this initial exploration of the effect of sperm competition on *SR* dynamics, we have deliberately assumed a simple model of sperm competition. In essence, our model assumes sperm mixing within the female reproductive tract, with no advantage to either the first or second male. Empirical studies on Drosophila reveal that the mechanisms of sperm competition can be more complex, including both bulk displacement and inca-

pacitation of one male's sperm by that of another (Price *et al.* 1999). Under these and other mechanisms, the order of mating can play a central role in determining paternity, with advantage most commonly accruing to the last male to mate (Harshman and Prout 1994; Civetta 1999). Provided that the mean advantage of *ST* males over *SR* males exhibits the same qualitative dependence on mating rate as supposed by the model, the predicted dynamics of the *SR* allele will apply to more complicated mating schemes allowing for priority and more than two matings per female. Likewise, the outcome of sperm competition can depend not only on the genotypes of the males (*SR vs. ST* in our model), but also on that of the females in which the sperm competition occurs (Clark and Begun 1998; Clark *et al.* 1999). Despite these complexities, we suspect that our basic conclusion—that sperm competition can simultaneously stabilize a sex-ratio distorter polymorphism and leave a population susceptible to fixation of the distorter allele—is independent of the specific mechanism of this competition.

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