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Evolutionary Consequences of Cytoplasmically Inherited Feminizing Factors

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Evolutionary consequences of cytoplasmically inherited feminizing factors

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SUMMARY

We develop a model to analyse the population and evolutionary consequences of parasitic sex ratio distortion to a particular class of systems, where the sex ratio organism (SRO) acts on host sex ratio by converting genotypic males into phenotypic females. Our model differs from previous approaches in that we explicitly distinguish between the processes of SRO transmission (infection) and SRO expression (SRO-induced feminization). We conclude that the evolutionarily stable host sex ratio will be biased towards the non-transmitting sex, provided that the SRO transmission and feminization efficiencies are not both 100%. Feedback between SRO prevalence and host sex ratio may drive to monogeny (the situation in which uninfected hosts produce only the non-transmitting sex). However, for many combinations of transmission and feminization efficiency, this feedback interaction does not lead to the exclusive production of males by uninfected females.

1. INTRODUCTION

Cytoplasmically inherited factors that distort sex ratio (sex ratio organism: SRO) are of evolutionary interest because they may drive hosts towards new mechanisms of sex determination, and can potentially drive host populations to extinction. Novel mechanisms of sex determination may arise in the host owing to SRO driven selection for non-fisherian sex ratios. SROs with high transmission rates have the potential to drive host populations extinct owing to the absence of one host sex.

Lewis (1941) and Howard (1942) were the first to suggest that cytoplasmically inherited organisms may increase in abundance by distorting host sex ratio in favour of the transmitting sex. We can envisage several possible mechanisms of sex ratio distortion by cytoplasmically inherited factors. Werren (1987) distinguishes between sex ratio genes, acting on sex ratio propensities in the mother, and sex determination genes, acting on the sex of individual offspring. Sex ratio may also be distorted by killing males (Skinner 1985; Hurst & Majerus 1993). In this paper, we consider the evolution of systems in which the SRO acts by manipulation of sex determination; this form of SRO has been considered by Bull (1983) and Taylor (1990).

Werren (1987) modelled sex ratio evolution and population dynamics for a sex ratio manipulator. This model is appropriate to some SROs of hymenopterans, where the sex of hosts is under haplodiploid control (Skinner 1982). However, SROs of crustaceans employ the alternative strategy of manipulating sex determination in infected offspring (Bull 1983; Rousset *et al.* 1992; Dunn *et al.* 1993). In these cases, the SRO infects a proportion of offspring, and then feminizes some fraction of these. Hence these SROs convert genetic males into phenotypic females. In these systems, transmission of the parasite and feminization are distinct processes. Transmission and expression have been considered separately in other SRO systems (Uyenoyama & Feldman 1978; Beukeboom & Werren 1993).

Various studies of SROs in Crustacea provide evidence for the distinction between transmission and feminization. Populations of *Gammarus duebeni* are infected by a microsporidian sex ratio organism (Bulnheim & Vavra 1968). The parasite is transovarially transmitted (in the cytoplasm of the eggs) from mother to offspring (Bulnheim 1967; Smith & Dunn 1991; Dunn *et al.* 1993). Infected offspring are feminized by the SRO, probably by indirect suppression of androgen gland development during embryogenesis (Bulnheim 1978). Feminization increases the transmission base to future host generations by converting males, which do not transmit the SRO, into females. Artificial infection experiments leading to partial feminization (Bulnheim 1977) and the occurrence of SRO infected males (Bulnheim 1978) may indicate that transovarial transmission is not a prerequisite for feminization, and that feminization after infection may occasionally fail. Artificial infection with SROs leads to feminization in *Armadillidium vulgare* (Juchault & Mocquard 1989) and *Orchestia gammarellus* (Ginsburger-Vogel & Desportes 1979). Incompletely feminized but infected intersex individuals have also been observed in *O. gammarellus* (Ginsburger-Vogel *et al.* 1980). Juchault *et al.* (1992) and Rigaud & Juchault (1992) identify separate resistance genes in *A. vulgare* that act against SRO transmission and feminization.

As SROs lead to a bias in the host population sex ratio in favour of the transmitting sex, selection will favour host (autosomal) genes that bias sex ratio towards the (rarer) non-transmitting sex (Werren 1987), in accord

with Fisher (1930). Such compensating sex ratio genes will be favoured provided that within-lineage transmission of the SRO is not certain. The ability of SROs to increase in host populations is dependent on the sex ratio produced by uninfected hosts (Bull 1983). The interdependence of SRO prevalence and host sex ratio may have the potential for positive feedback: compensatory shifts in host sex ratio allow increased parasite prevalence, which may drive sex ratio selection still further (Werren 1987). This may result in monogeny, in which uninfected females produce only male offspring; hence the sex of hosts is determined entirely on the basis of presence or absence of the SRO (Bull 1983). Monogeny is predicted provided that the host species possesses sufficient heritable variation in sex ratio, and that the within-lineage transmission efficiency of the SRO is not 100% (Werren 1987). Evidence for monogeny exists for *A. vulgare* (Juchault *et al.* 1992). However, many other populations and associations clearly have not evolved to monogeny, and we may be left with the uneasy conclusion that there is insufficient heritable variation in host sex ratio (Bull & Charnov 1988).

SROs with high within-lineage rates of transmission are predicted to spread rapidly throughout a host population, reaching an equilibrium prevalence (Bull 1983; Skinner 1987; Werren 1987). If transmission is complete, SROs without deleterious effects on host fecundity or survival are expected to reach fixation (100% prevalence). However, SROs with high transmission rates and no detrimental effects on host fecundity or survival are supported at rather low prevalences in field populations (Bull 1983; Werren 1987; Rigaud *et al.* 1992; Juchault *et al.* 1992; Beukeboom & Werren 1992). Subdivided host population structure may account for the low prevalence of one SRO in *Nasonia vitripennis* (Werren & Beukeboom 1993). However, there is no general explanation for the discrepancy between observed prevalence and prediction.

In this paper, we analyse the population dynamics and evolution of the host sex ratio towards monogeny for associations in which the SRO distorts sex determination by feminization of host offspring. We develop from previous analysis (Bull 1983) in that we distinguish explicitly between the processes of SRO transmission and feminization. Our models were developed with reference to the SRO–*G. duebeni* system described by Dunn *et al.* (1993), following the framework provided by Werren (1987).

2. THE MODEL

We develop a model of SRO action in which the sex ratio of host offspring is distorted by conversion of genetic males into phenotypic females, and in which the processes of SRO transmission and expression are distinct. We identify the ESS (Maynard Smith & Price 1973) sex ratio for uninfected hosts in an SRO-infected population, to compare the evolutionary consequences with those observed by Werren (1987), who considered SROs that distort sex ratio directly. Evolution to a biased ESS sex ratio requires sex ratio plasticity (heritable variation in sex ratio). This assumption may be met in *G. duebeni*, some populations of which exhibit adaptive sex ratio variation with respect to seasonal effects (Naylor *et al.* 1988; Watt & Adams 1994). Selection for modifiers of SRO action are not considered here so as to maintain consistency with Werren (1987). We investigate the evolutionary consequences of this association in three parts as follows.

(a) We consider the dynamics of SRO invasion and equilibrium prevalence in an infinite host population, using standard methods in population genetics.

(b) We derive the ESS host sex ratio (i.e. that sex ratio which when produced by hosts cannot be invaded by host genes coding for any other sex ratio), in the presence of parasitism, as presented in Werren (1987), after Uyenoyama & Feldman (1978).

(c) We investigate whether the interaction between parasite prevalence, as modelled in (*a*), and host sex ratio, as modelled in (*b*), will have regions of costability.

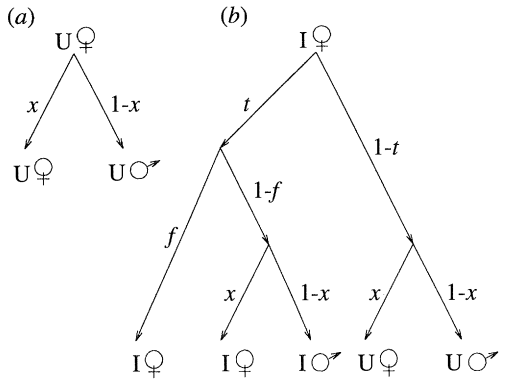

Figure 1. Mechanism of sex determination in hosts associated with feminizing SROs. (*a*) Sex ratio of offspring of uninfected (U) females, expressed as the proportion of females. (*b*) Sex determination of offspring of infected (I) females is overridden by transmission (t) and feminization (f) by the SRO.

(*a*) *Population dynamics of* SRO

Consider a population of hosts, of which a proportion P of females are infected. The remaining $(1-P)$ females produce brood with a sex ratio x in that each offspring develops as a female with probability x. Infected females are of fitness W relative to uninfected females (W expresses relative survival) and infection is transmitted to a proportion t of their brood. The remaining $(1-t)$ of brood have their sex determined as for the offspring of uninfected mothers (i.e. become female with probability x). The SRO acts on a proportion f of brood that it has infected, converting these to females. The remaining $(1-f)$ brood, although infected, are not feminized by the parasite. Their sex is determined in accordance to the host's underlying primary sex ratio x. This mechanism can be represented as a decision tree (figure 1) with parameters defined as follows.

P, frequency of sex ratio organism (SRO) in adult females;

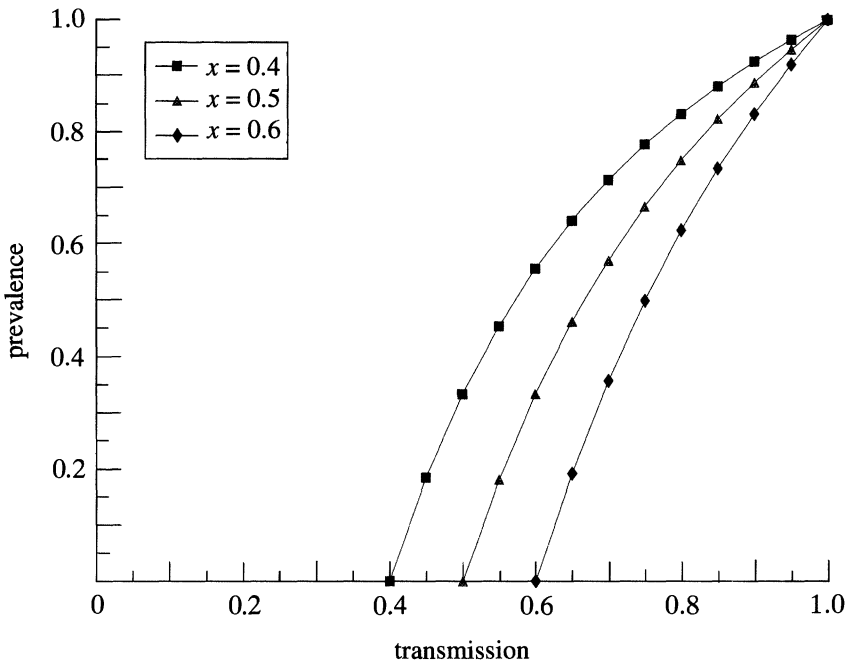

Figure 2. Equilibrium prevalence of SRO in an infinite host population. The effect of the sex ratio produced by uninfected hosts (x) on prevalence at equilibrium (P^*).

W, survival of SRO females relative to uninfected females;

x, probability of becoming female if uninfected or unfeminized;

t, proportion of SRO brood that inherit SRO (transmission efficiency);

f, proportion of all infected brood that become female as a result of SRO action.

This model assumes that infected individuals that are not actively feminized are identical to uninfected individuals in terms of rules for sex determination. This assumption will not hold under all circumstances (for instance, when the transmitting sex is also the heterogametic sex). We have also assumed that infected unfeminized females transmit the SRO in an identical manner to infected, actively feminized females. (Hence we assume: $W_c = W_i$ where W_i is the fitness of infected, feminized hosts (relative to uninfected hosts) and W_c is the relative fitness of genetically female carriers; $t_c = t_i$ where t_i is the SRO transmission efficiency from feminized hosts to their offspring and t_c the transmission efficiency from carriers; $f_c = f_i$ where f_i is the feminization efficiency of SROs inherited from feminized hosts and f_c the feminization efficiency of SROs inherited from carriers.) These assumptions were deemed appropriate to the biological system that motivated this work (*G. duebeni* and microsporidian SRO: Dunn *et al.* 1993). We consider the effects of relaxing these simplifying assumptions in the discussion (see also the appendix).

(i) *Invasion conditions*

The proportion of females infected in successive generations is given by

$$P' = \frac{PWtf + PWt(1-f)x}{PWtf + PWt(1-f)x + P(1-t)x + (1-P)x}. \quad (1)$$

The SRO can increase initially providing

$$Wt(f + x - fx) > x. \quad (2)$$

Hence, successful invasion requires that infected females produce more infected daughters than uninfected females produce daughters. This argument has been presented verbally by Howard (1942), and by Bull (1983). SROs may achieve the invasion criterion by imparting a fitness benefit and/or feminizing host offspring. If infected females do not differ in fitness to uninfected females, we obtain Bull's (1983) result that transmission efficiency must exceed host sex ratio. When transmission and feminization are complete ($t = f = 1$), $W > x$ is the condition for invasion: an SRO with deleterious effects can invade if it is sufficiently efficient at transmission and feminization. Without feminization, we obtain $Wt > 1$; the parasite must enhance host fitness in order to invade, whatever its transmission rate. This result is in agreement with Fine (1975, 1984) and Anderson & May (1981), considering vertically transmitted parasites that do not affect sex determination.

(ii) *Equilibrium SRO prevalence*

For fixed W, t, f and x, the SRO will reach an equilibrium frequency

$$P^* = \frac{Wt(f + x - fx) - x}{Wt(f + x - fx) - tx}. \quad (3)$$

Higher values of W, t, and f allow the SRO to maintain infection at a greater equilibrium frequency. P^* is also dependent on host sex ratio (figure 2); more male-biased ratios result in increased P^*.

Equation (3) indicates that SROs with complete transmission reach fixation ($P^* = 1$), even if feminization is incomplete. This follows by definition from the mechanism: the only daughters produced by infected females are themselves infected, even if they have not been directly feminized. When feminization

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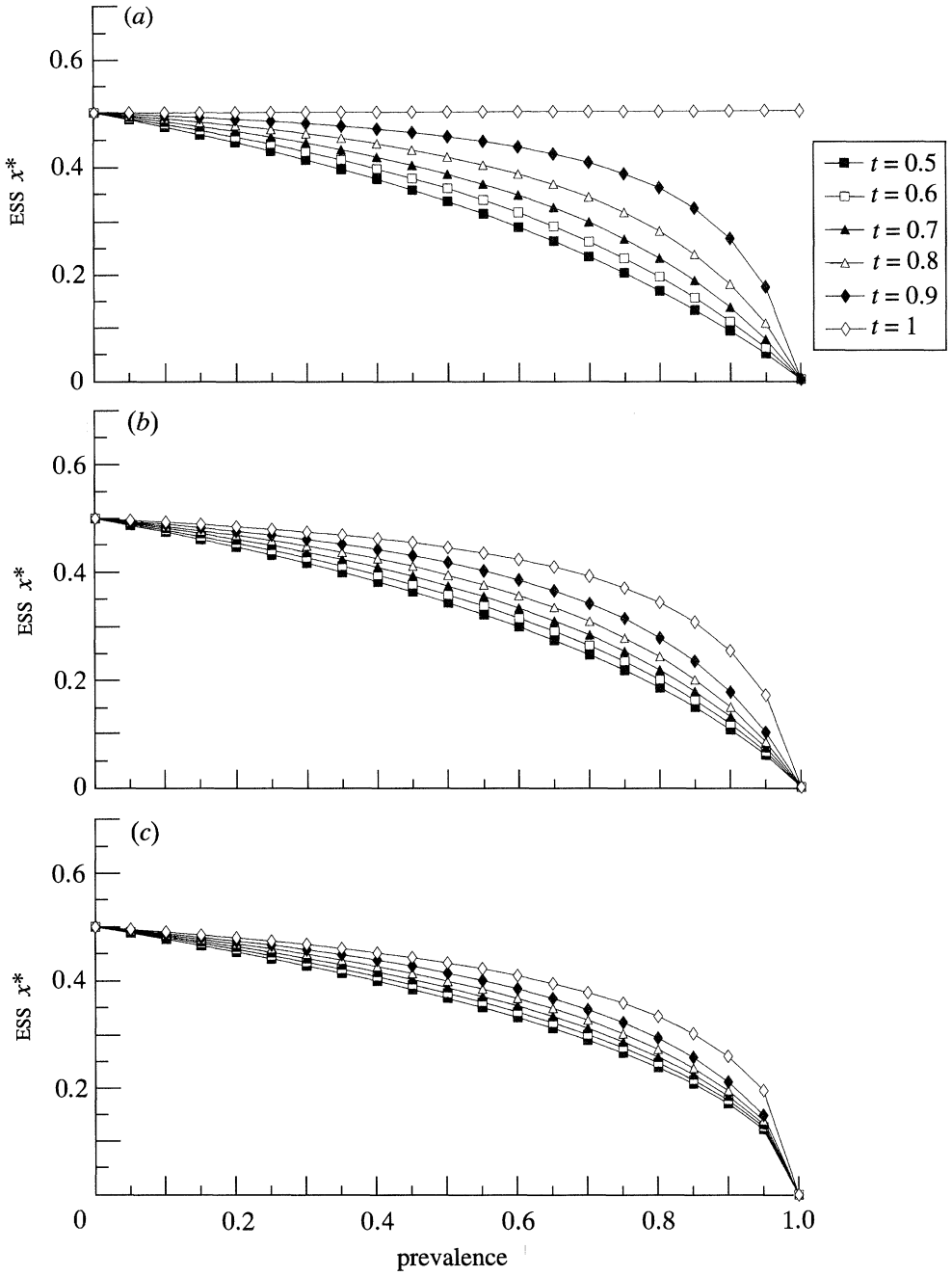

Figure 3. The evolutionarily stable sex ratio (ESS x^*) for uninfected hosts in a population containing SROs. The relation between x^* and SRO prevalence for given transmission efficiencies is plotted (*a*) when feminization (f) is complete, (*b*) for $f = 0.7$ and (*c*) for $f = 0.5$.

efficiency is high, few or no sons will be produced by infected mothers. Hence when $t = 1$ and $f = 1$, we obtain the paradoxical situation that the SRO rises rapidly to fixation, but drives its host population (and hence itself) extinct owing to lack of males (see also Werren 1987). However, incomplete feminization ($f < 1$) will result in the production of some (putatively infected) males. Under such conditions the SRO would go to fixation in females, but incomplete expression would continue to yield some males that may enable maintenance of the host population.

(*b*) *ESS host sex ratio*

We derive the unbeatable host sex ratio in a population infected with an SRO following Werren (1987). We assume the host population is of infinite size, with mating at random with respect to infection status. The SRO is assumed to be at equilibrium prevalence in the population. We consider an autosomal sex ratio locus with two alleles (A or a) that operates in diploid uninfected hosts to determine sex. The A allele is dominant to a, but a is near fixation. Hence most hosts are of genotype aa and become female with probability x. Rarely, hosts are genotype Aa, and become female at an arbitrarily different probability x_a. Infected offspring develop according to figure 1, and express their genotype if uninfected or unfeminized. We obtain the evolutionarily stable (ESS; Maynard Smith

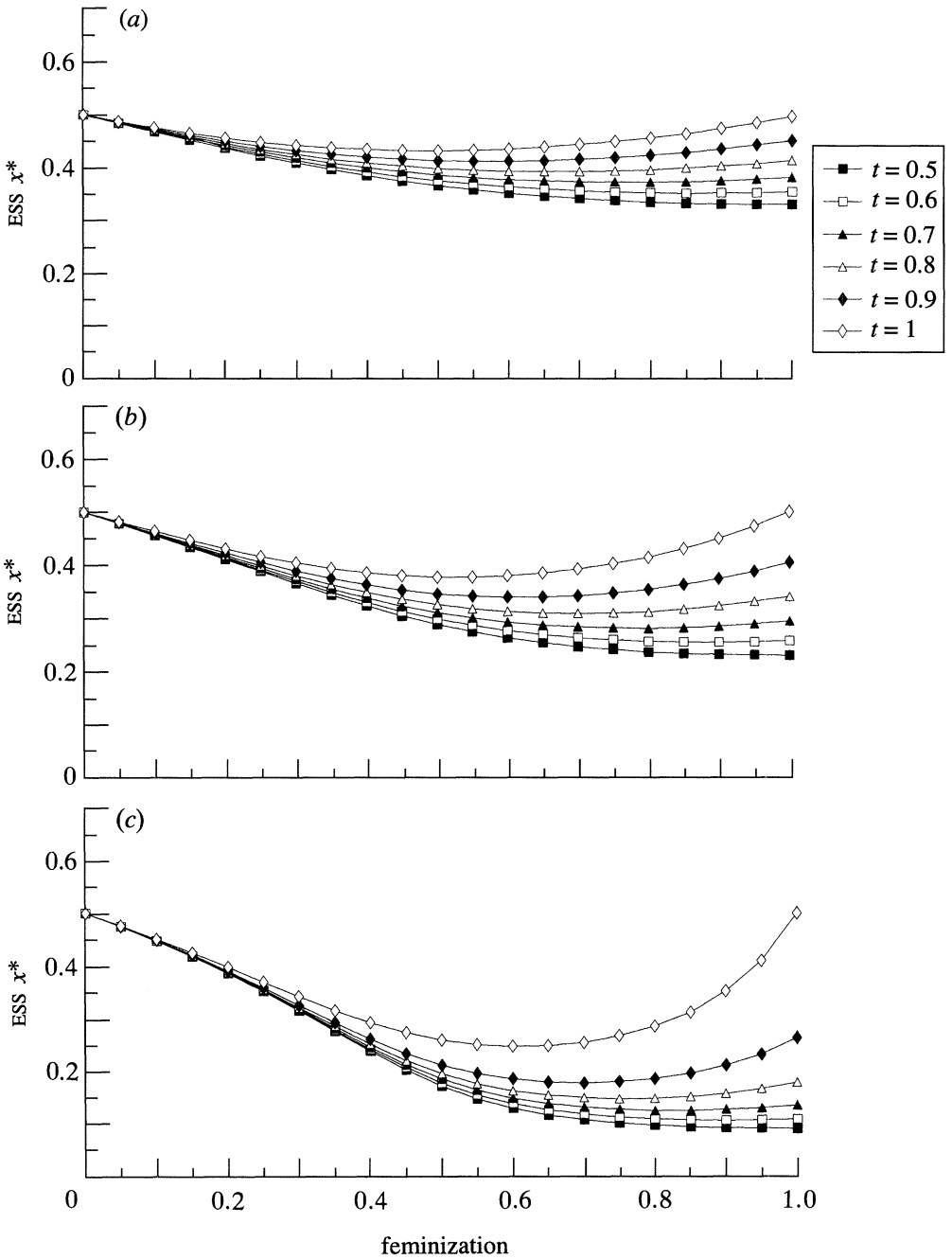

Figure 4. The effect of feminization on ESS sex ratio. ESS x^* is plotted for given transmission efficiencies when (*a*) SRO prevalence $P = 0.5$, (*b*) $P = 0.7$ and (*c*) $P = 0.9$.

1976*a*, *b*) host sex ratio by finding the sex ratio x^* that, when produced by allele *a*, allows *a* to resist invasion by the *A* allele producing any other sex ratio.

The transmission dynamics of the *A* allele in a population containing the SRO at prevalence *P* are described by the following matrix, where homozygous *AA* individuals and heterozygous $Aa \times Aa$ matings have been discounted from the analysis, as *A* is assumed to be rare:

$$\begin{pmatrix} \epsilon'_1 \\ \epsilon'_2 \\ \epsilon'_3 \end{pmatrix} = \begin{pmatrix} \frac{(1-x_a)(1-Ptf)}{2M} & \frac{(1-P)(1-x_a)}{2M} & \frac{P(1-x_a)(1-tf)}{2M} \\ \frac{x_a(1-Pt)}{2F} & \frac{(1-P)x_a}{2F} & \frac{P(1-t)x_a}{2F} \\ \frac{WPt(f+x_a-fx_a)}{2J} & 0 & \frac{WPt(f+x_a-fx_a)}{2J} \end{pmatrix} \begin{pmatrix} \epsilon_1 \\ \epsilon_2 \\ \epsilon_3 \end{pmatrix}, \quad (4)$$

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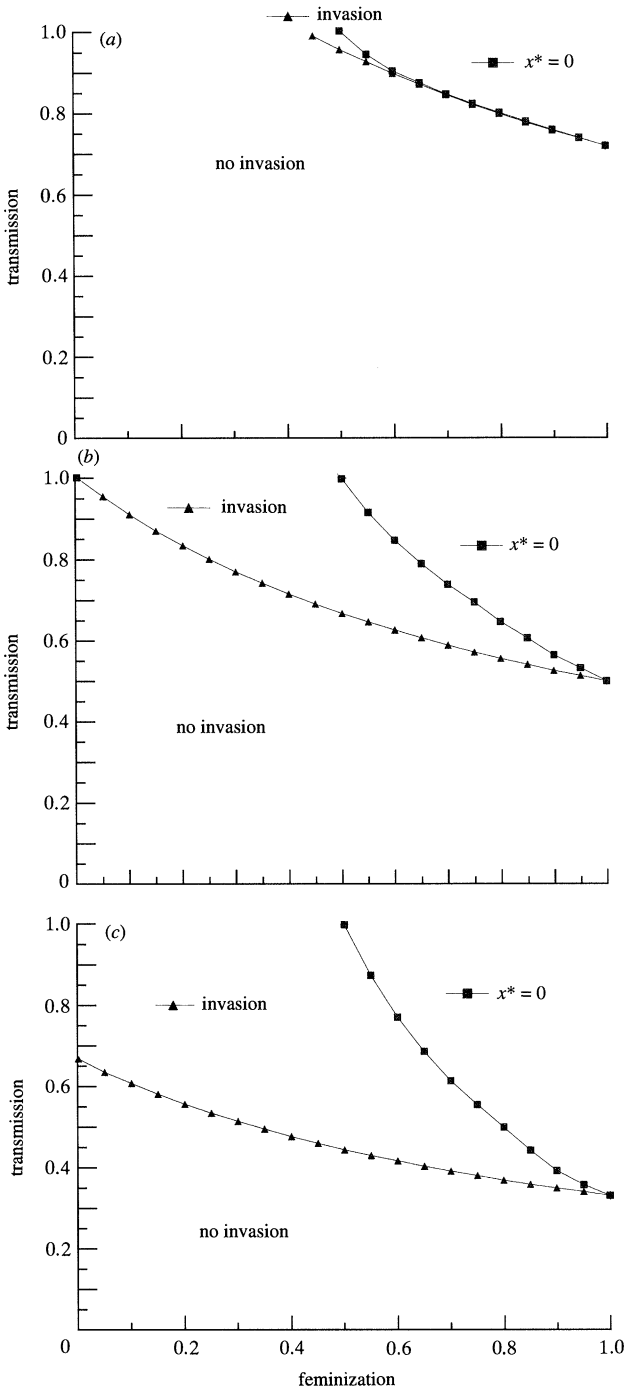

Figure 5. Solution regions for ESS sex ratio in the presence of coevolution. Combinations of transmission (t) and feminization (f) that satisfy the conditions for invasion and lie below the bound for monogeny ($x^* = 0$) result in a male biased sex ratio at equilibrium. (a) Relative fitness of infected individuals $W = 0.7$, (b) $W = 1$ and (c) $W = 1.5$.

where:

ϵ_1 is the frequency of *Aa* among adult males,

ϵ_2 is the frequency of *Aa* among adult uninfected females,

ϵ_3 is the frequency of *Aa* among adult infected females,

ϵ_1' is the appropriate frequency in the following generation,

x is the proportion of *aa* offspring of uninfected mothers that become female,

x_a is the proportion of *Aa* offspring of uninfected mothers that become female and other parameters are as above, with

$$\left.\begin{aligned} M &= (1-x)(1-Ptf), \\ F &= x(1-Pt), \\ J &= WPt(f+x-fx). \end{aligned}\right\} \quad (5)$$

The ESS sex ratio (denoted x^*) is determined by forming the characteristic equation of the transmission matrix, differentiating with respect to x, setting the differential = 0, then setting eigenvalue = 1 and $x_a = x$, and solving for x (see Werren 1987). These manipulations were done with use of algebraic software packages. Differentiation and substitution for

eigenvalue and x_a yield the quadratic numerator in x (ax^2+bx+c), where

$$\left.\begin{aligned} a &= 2(1-f)(1-Ptf)(2Pt-P-1),\\ b &= 1-3f+P-fP-2Pt+4fPt+2f^2Pt\\ &\quad -2fP^2t+2f^2P^2t+2fP^2t^2-4f^2P^2t^2,\\ c &= f(1-P)(1-Pt). \end{aligned}\right\} \quad (6)$$

The ESS is found by solving for x in the above quadratic; general roots exist provided $a \neq 0$. Since the term in x^2 has the factor $(1-f)$, x^* for $f = 1$ (complete feminization) must be considered separately. When $f = 1$, the numerator simplifies to a linear equation in x, yielding

$$x^* = \tfrac{1}{2}(1-P)/(1-Pt). \quad (7)$$

Hence the ESS sex ratio under complete feminization is dependent on SRO prevalence P and SRO transmission rate t. When the SRO is absent ($P = 0$), we retrieve $x^* = \frac{1}{2}$ (Fisher 1930). When transmission is complete ($t = 1$), we also obtain $x^* = \frac{1}{2}$, as predicted by Werren (1987). Hence, under complete transmission and feminization by the SRO, compensatory sex ratio evolution is not favoured in the host.

When feminization is incomplete ($f < 1$), we return to the general solution for x^*. The solutions for x^* are dependent on f, t and P, and do not appear to be amenable to simplification. The lower root yields biologically feasible solutions ($0 < x^* < 1$).

The relation between x^* and P, t and f is plotted in figures 3 and 4. The results are in general agreement with those for the sex ratio distorter system (Werren 1987). For any given transmission rate less than unity, increase in parasite prevalence results in a more male-biased ESS sex ratio (figure 3). As prevalence reaches 100%, x^* reaches zero; hence when the SRO is at fixation, the host ESS is to produce exclusively male offspring. This result holds for all values of f. When feminization is incomplete, the ESS x^* is biased towards production of males ($x^* < \frac{1}{2}$) for all non-zero values of P and t. In contrast to Werren (1987), compensatory sex ratio evolution is favoured even when transmission is complete, provided feminization is incomplete (figure 4). When some individuals escape feminization, gene flow between infected and uninfected subpopulations continues, and selection on sex ratio is permitted.

Increasing efficiencies of transmission and feminization lead to increasing separation between infected and uninfected host populations. When t and f are both complete, there is no gene flow between infected and uninfected subpopulations, and hence no selection for compensatory sex ratios. Low values for feminization do not induce much compensatory sex ratio shift, as the SRO no longer distorts host sex ratio to such an extreme (figure 4). At the limit, $f = 0$ represents a cytoplasmically inherited factor that does not distort sex ratio. Under these circumstances, we expect (and obtain) $x^* = \frac{1}{2}$.

In summary, the ESS sex ratio becomes more strongly biased towards the non-transmitting sex as SRO prevalence increases. Shifts in host sex ratio are favoured except when:

(*a*) $f = 0$, the SRO does not distort primary sex ratio; or

(*b*) $f = t = 1$, infected and uninfected hosts form separate subpopulations.

(*c*) *Coevolution to costability*

ESS sex ratio is dependent on SRO prevalence (figure 3), which is itself dependent on sex ratio (figure 2). This relation has feedback potential, in that decreasing x^* will result in increasing P^* which in turn selects for a further decrease in x^*. We now analyse whether interaction between parasite prevalence and host ESS sex ratio will lead to costable values for P and x (denoted P^{**} and x^{**}), or whether the interaction will drive x to zero and hence host sex determination to monogeny.

When feminization is complete, substitution for x^* (equation (7)) into P^* (equation (3)) yields

$$(P-1)(1-Pt)(2Wt-1) = 0. \quad (8)$$

The equality is satisfied when $P = 1$, $P = t = 1$ and $Wt = \frac{1}{2}$. The third condition cannot be satisfied by any SRO that originally invaded a host population at fisherian equilibrium (invasion condition for $x = \frac{1}{2}$; equation (2)); hence there are no stable values for P other than $P^{**} = 1$. Substituting $P = 1$ into equation (7), we identify the costable ESS sex ratio $x^{**} = 0$. For SROs with complete feminization, feedback in the relation between prevalence and ESS sex ratio will drive the SRO to fixation and the host to monogeny (in agreement with Bull (1983) and Werren (1987)). This conclusion holds provided there is sufficient heritable variation for host sex ratio to allow x to evolve to zero. Feedback to monogeny occurs provided transmission is incomplete; when $t = 1$, ESS x^* remains at $\frac{1}{2}$, and there is no feedback between prevalence and sex ratio (see also Werren 1987).

For SROs with incomplete feminization, we substitute for P^* (equation (3)) into the quadratic in x^* (equation (6)), yielding a cubic in x (see appendix). This equation does not appear to be amenable to simplification when the parameter space is left free. However, by setting $W = 1$ (infected and uninfected individuals of equal fitness), we obtain a three factor expression (presented in the appendix), the only possible solution for x being

$$x^{**} = (2tf-1)/[2t(f-1)],$$
$$x^{**} \geqslant 0 \text{ when } tf \leqslant \tfrac{1}{2}. \quad (9)$$

Costable ESS sex ratio is dependent on SRO feminization and transmission efficiency. For combinations of f and t that satisfy $ft < \frac{1}{2}$, there are non-zero solutions for x^{**}. Hence feedback between SRO prevalence and host ESS sex ratio does not always result in evolution to monogeny, when SRO expression (feminization) is incomplete. Previous analyses have concluded that interaction between SROs with incomplete transmission will result in monogeny (Bull 1983; Werren 1987). The solution regions for x^{**} in terms of f and t are plotted in figure 5.

The behaviour of x^{**} when $W \neq 1$ was investigated by numerical calculation, by repeated substitution into the expressions for ESS x^* (equation (6)) and P^* (equation (3)), starting with an initial host sex ratio of $x = \frac{1}{2}$. Equilibrium values for P^{**} and x^{**} were found

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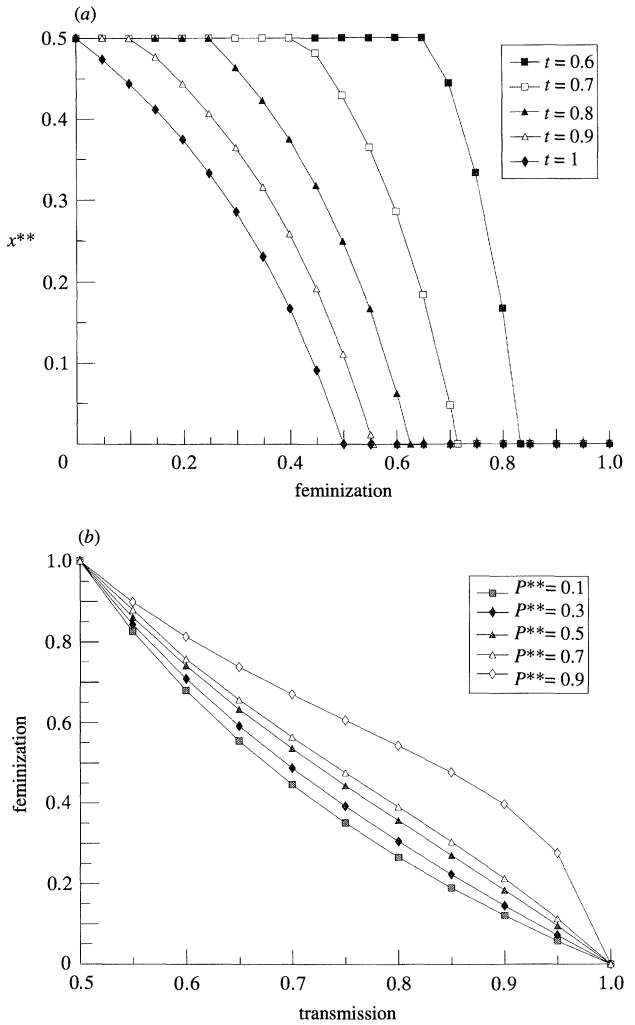

Figure 6. Costable ESS host sex ratio and equilibrium prevalence of SRO. (*a*) ESS sex ratio (x^{**}) is plotted for given efficiencies of transmission (t), for equal fitness of infected and uninfected hosts ($W = 1$). (*b*) Combinations of feminization and transmission that lead to a polymorphic equilibrium in female hosts when coevolution is permitted. Values of f and t that result in given SRO prevalences at equilibrium are plotted.

by iterating the process over many generations, until P and x appeared stable. For most values of f and t, x^{**} either reached zero or obtained an apparent non-zero equilibrium with 200 iterations. Non-zero equilibrium values for x^{**} remained unchanged when iterations were allowed to continue for 10000 iterations.

Regions of stable non-zero x^{**} and monogeny were investigated by numerical calculation. Any triplet of f, t and W occupies one of four regions (figure 5):

I, SRO cannot become established (invasion condition not met);
II, x^{**} evolves to a stable, non-zero value;
III, x^{**} evolves to zero (monogeny);
IV, x^{**} remains unbiased ($= \frac{1}{2}$; when $t = f = 1$).

The position of the lower bound (between regions I and II) was calculated directly from the invasion condition (equation (2)). The position of the upper bound (between regions II and III) was calculated directly for the case when $W = 1$, and was estimated by using binary search techniques otherwise.

It can be shown that no type III solutions exist when feminization efficiency is less than $\frac{1}{2}$ (see appendix). An intuitive explanation for this can be observed for the case of complete transmission. With $t = 1$, $P^* = 1$; only infected females exist in the population. From Fisher (1930), host genes that favour the production of the rarer sex will be selected provided they are expressed (which still occurs when feminization fails). For $f < \frac{1}{2}$, monogeny ($x = 0$) would result in all mothers producing male-biased progenies, and hence an $x^{**} > 0$ (at which number of males equals number of females) will be favoured. We have also observed that no type II solutions exist when feminization is complete (equation (7)). These two observations allow us to fix both endpoints of the upper bound, and the lower bound can be calculated directly with reference to the invasion criterion. Hence the complete solution space can be reasonably well characterized for any value of W. Figure 5 shows that as W is increased the region of stable non-zero x^{**} increases. For relative fitness much less than $W = 0.7$, monogeny is effectively the only solution.

Host sex ratio may evolve to a stable, non-zero value under interaction with SRO prevalence, for intermediate values of feminization efficiency and transmission (figure 6*a*). The costable value of x^{**} lies above 0 and below $\frac{1}{2}$; i.e. the sex ratio produced by uninfected hosts

after coevolution will be skewed toward the non-transmitting sex. The precise value of x^{**} is dependent on f, t and W. Higher values of f and t lead to stronger feedback between sex ratio and prevalence, resulting in monogeny ($x^{**} = 0$) and SRO fixation ($P^{**} = 1$).

When x^{**} is stable above zero, the SRO does not reach fixation. The costable value for P^{**} when $W = 1$ is given by

$$P^{**} = (tf+t-1)/[tf(2t-1)]. \quad (10)$$

Hence, combinations of t and f lead to one of four evolutionary outcomes for SRO prevalence and ESS sex ratio. Biologically feasible values of t and f exist that result in any value of x^{**} between 0 and $\frac{1}{2}$ (figure 6*a*) and any value of P^{**} between 0 and 1 (figure 6*b*). Monogeny and SRO fixation will not necessarily be the consequence, even when sufficient heritable variation in host sex ratio exists. The evolutionary outcome depends on the precise efficiencies of transmission and feminization. In particular, if (and only if) feminization is incomplete ($f < 1$), host sex ratio may evolve to a stable value that is skewed toward the non-transmitting sex, but does not code for the exclusive production of this sex.

3. DISCUSSION

Werren (1987) and Bull (1983) discuss a number of reasons why coevolution between SRO and host may not necessarily produce feedback over host sex ratio and lead to monogeny. In this paper, we introduce a further reason, that of incomplete SRO expression (feminization efficiency less than unity). Interaction between host ESS sex ratio and SRO prevalence leads to one of four outcomes:

I, SRO is unable to invade host population (t, f, W do not meet invasion condition);
II, SRO reaches equilibrium prevalence ($P^{**} < 1$), host sex ratio evolves to a male-biased equilibrium value ($0 < x^{**} < \frac{1}{2}$);
III, SRO reaches fixation ($P^{**} = 1$), uninfected offspring always develop as males ($x^{**} = 0$);
IV, SRO reaches fixation; ESS sex ratio remains unbiased (when $t = f = 1$).

The precise values of stable ESS sex ratio and SRO prevalence are dependent on transmission (t) and feminization (f) efficiency, and the relative fitness of infected females (W). When infected and uninfected females are of equal fitness, combinations of t and f that allow invasion but satisfy $tf < \frac{1}{2}$ will lead to a stable non-zero ESS sex ratio.

Werren (1987) concludes that interaction between SROs and host populations may result in states I, IV, or monogeny ($x^{**} = 0$) but not fixation ($P^{**} < 1$). When the SRO acts by distorting sex ratio directly (see, for example: Skinner 1982; Werren 1987), no parallel to incomplete expression as defined in this paper exists. Bull (1983), considering sex determination distorters, concluded that states I and III could occur, but he did not distinguish between transmission and expression.

The novel conclusion of state II in this paper would appear to result from the distinction made between transmission and feminization. The f system modelled here differs from the f-free system considered by Bull (1983) in that the absolute number of infected females in consecutive generations is now dependent on host sex ratio. This is due to the production of a subclass of infected individuals that become female as a result of host gene action rather than parasite-induced feminization ('carriers'). The relative importance of the dependent subclass in contributing infected females is highest when feminization efficiency is low. Since carriers contribute less to P' as x decreases, the relation between P and x may now potentially lead to stability.

In this paper we assume that the transmission and feminization characteristics and fitness effects of SROs in carriers are identical to those of SROs in actively feminized hosts. Numerical calculations when this assumption is relaxed (see appendix) reveal no qualitative difference: non-zero costable sex ratios are still observed. Hence the assumption of equality between actively feminized and carrier females does not appear to be responsible for the existence of state II. When the SROs from carriers do not transmit or feminize at all, the model collapses onto that of Bull (1983). The absolute production of infected females is no longer dependent on sex ratio, and ESS sex ratio evolves to monogeny (state II is no longer observed). This suggests that state II is only possible in systems with an x-dependent subclass of infected females. Further, this subclass must also transmit SROs capable of feminization to at least some of their offspring.

The distinction between transmission and feminization also allows infected females to produce some males, even if transmission is complete. This source of males may be sufficient to stabilize sex ratio coevolution before monogeny is reached. If feminization were particularly inefficient (e.g. $f < \frac{1}{2}$ for $t = 1$), monogeny would result in infected mothers producing more males than females. When feminization is assumed complete (as in Bull 1983), this situation can never arise, as it requires a transmission efficiency too low to permit initial invasion by the SRO.

By introducing an explicit term for the process of feminization we develop a more general model for this class of SROs. Both W and f are important in determining whether a particular factor can invade a host population: factors that do not feminize must impart a fitness benefit to infected individuals. Factors that distort sex determination to a sufficient degree may invade even if they have a negative effect on host fitness. The introduction of f enables the unification of several previous models of vertical transmission. The models of Bull (1983), and some aspects of Fine (1975) and Anderson & May (1981), represent limiting cases at the ends of a continuum of possible feminization efficiencies; the first when $f = 1$ and the other two when $f = 0$.

The model presented here encompasses cytoplasmically inherited factors that do not distort sex ratio, partial feminizers and complete feminizers within a single framework. However, it may not be appropriate in all cases. The assumption of equality between actively feminized and carrier females does

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not take into account the possibility that these classes of female are correlated with resistant host genotypes or dosage effects on SRO expression. If there is no cost to resistance, resistant host genotypes are expected to spread until the population sex ratio returns to $\frac{1}{2}$ (Taylor 1990). If resistance is costly, we expect the spread of resistance to depend on cost in addition to population sex ratio (see Hurst & Pomiankowski (1991) for analysis of a related system). Hence the evolution of resistant hosts offers an alternative route by which monogeny is avoided. Dependence of feminization and/or transmission success on dosage of infection is an interesting (and extremely complex) possibility that we shall consider elsewhere. We still obtain states I to IV if two subclasses of infected females are distinguished (see appendix). It may, however, be more appropriate to model such phenomena with continuous variables rather than fixed proportions operating in very limited classes of individuals.

Any factor that is uniparentally inherited will, in principle, be selected to bias host sex ratio towards the transmitting sex (Hamilton 1967). Sex ratio distortion may be achieved by a variety of means (reviewed in Bull 1983; Hurst 1993). Apart from mitochondrial involvement in cytoplasmic male sterility in plants (reviewed in Vedel *et al.* 1994), organelles and cytoplasmically inherited symbionts of insects have not generally been implicated in sex ratio distortion (see, for example: Margulis 1970; Grun 1976; Eberhard 1980; Houk & Griffith 1980; Cosmides & Tooby 1981; Awahmukalah & Brooks 1983; Douglas 1989). This may indicate constraints on the ability of these factors to manipulate host sex ratio, or host genetic strategies for resistance to sex distortion (Bull & Charnov 1988). With reference to feminizing strategies, f and W may be negatively correlated: distortion of sex determination may lead to a reduction in host fitness, which is not outweighed by the benefits of feminization.

Several examples of sex determination distorters occur in the Crustacea (reviewed in Bull 1983; Smith & Dunn 1991; Rousset *et al.* 1992; Hurst 1993). It is possible that the mechanism of sex determination in crustaceans (Legrand *et al.* 1987) leaves them vulnerable to parasitic sex manipulation (unpublished data) or that the costs to host fitness of partial failure of sex manipulation are relatively low. The occurrence of intersexes (individuals exhibiting both male and female morphologies) has been linked to infection with SROS (Bulnheim 1978; Ginsburger-Vogel *et al.* 1980). If feminization evolves through intermediate stages, it may only be observed in associations where the products of partial feminization have some degree of female function (as evidenced by Bulnheim 1965; Dunn *et al.* 1990), and hence contribute towards SRO transmission.

The model highlights the importance of accurate parameter estimates if population and evolutionary outcomes are to be understood. In practice, it may be difficult to obtain good estimates of feminization efficiency; for instance, the SRO may degenerate in male-determined tissue. Conflation of feminization and transmission may lead to incorrect predictions concerning the evolutionary outcome of the association, as $f < 1$ enables the maintenance of host populations through male production, despite high SRO transmission and apparent near-fixation prevalence. Further, $f < 1$ introduces the possibility of stable, non-zero male-biased ESS sex ratio and intermediate SRO prevalence at equilibrium.

Theoretical models of SRO dynamics appear to be at odds with empirical data, in that observed prevalences of SROS are lower than predicted equilibrium prevalences (Bull 1983; Werren 1987; Rigaud *et al.* 1992; Werren & Beukeboom 1993). A number of explanations for this discrepancy have been suggested, including demic population structure and local mate competition (Werren & Beukeboom 1993) and resistance strategies in the host (Uyenoyama & Feldman 1978; Bull 1993; Taylor 1990). Prevalence estimates may also be limited by random loss of SROS from host subpopulations (Rigaud *et al.* 1992) and by population level selection, whereby host subpopulations go extinct as the SRO reaches fixation (Werren 1987). The effect of these processes on mean prevalences is unclear, and will be highly dependent on relative rates of SRO loss, subpopulation extinction and recolonization.

Differential extinction of populations may influence the patterns of SRO associations we currently observe, in that only those SROS with combinations of transmission and feminization that enable the SRO to invade and allow maintenance of the host population may persist over time. The results presented in this paper indicate that, even if evolutionary interaction between host sex ratio and SRO prevalence is permitted, SROS will not necessarily go to fixation or result in monogeny in the host population. The evolutionary outcome is dependent on efficiencies of SRO transmission and expression and on the precise relation between prevalence and sex ratio. Host ESS sex ratio may stabilize at a non-zero value only if the SRO does not always feminize, and all infected females can transmit feminizing SROS to at least some of their offspring.

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APPENDIX

(*a*) *ESS host sex ratio solutions in the presence of coevolution*

When P^* (equation (2)) is substituted into ESS x^* (equation (6)), we obtain the following numerator, set to 0:

$$px^3 + qx^2 + rx + s = 0, \qquad \text{(A 1)}$$

where

$$\begin{aligned} p = {} & 2 - 4Wt - 12t^3f^3W^2 + 4Wf - 2Wf^2 + 14Wtf \\ & + 12W^2tf^2 - 4f - 4W^2t^2f - 12W^2tf + 4W^2t \\ & - 2W - 16f^2tW + 4f^4W^3t^2 + 6f^3Wt - 4tf^3W^2 \\ & + 2f^2 + 12W^2t^2f^2, \end{aligned}$$

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$$
\begin{aligned}
q &= W-1-2W^2t-14Wtf-16W^2t^2f^2+2W^2t^2f \\
&\quad +26t^2f^3W^2-12f^4W^2t^2+24f^2tW \\
&\quad -26W^2tf^2+12tf^3W^2+16W^2tf+3Wf^2 \\
&\quad -12f^3Wt+3f-4Wf-2f^2+2Wt, \\
r &= 12f^4W^2t^2-12tf^3W^2+4W^2t^2f^2-9f^2tW \\
&\quad +16W^2tf^2+3Wtf+6f^3Wt-4W^2tf-16t^2f^3W, \\
s &= 4tf^3W^2+f^2tW+2t^2f^3W^2 \\
&\quad -Wf^2-4f^4W^2t^2-2W^2tf^2.
\end{aligned} \tag{A 2}
$$

When $W = 1$, this simplifies to

$$
\begin{aligned}
0 &= (1-t)f(1-2xt-2tf+2xtf) \\
&\quad \times (2x^2t-xt+5xtf-4x^2tf-tf+2f^2t \\
&\quad -4xf^2t+2x^2f^2t-x^2-f+x^2f).
\end{aligned} \tag{A 3}
$$

Numerical substitutions over the biological range $(0 < t, f < 1)$ indicate that the factor in x^2 does not yield valid solutions for x: x is found to be either complex, < 0 or > 1. The linear factor in x yields solutions over the biological range $(0 \leqslant x \leqslant 1)$:

$$
x^{**} = (2tf-1)/[2t(f-1)]. \tag{A 4}
$$

(*b*) *Conditions for* $x^{**} = 0$

We can prove that $f = \frac{1}{2}$ yields the solution $x^{**} = 0$, for all W when t is close to 1, by the following steps.

(i) Substitute $f = \frac{1}{2}$ in equation (6).

(ii) Substitute $t = 1-\epsilon$ and approximate for ϵ small $(\epsilon^2 \to 0)$. This yields

$$
-\tfrac{1}{4}\epsilon x(2W^2x+W^2+W^2x^2-3Wx-3Wx^2+2x^2) = 0. \tag{A 5}
$$

(iii) The quadratic factor in x has solutions

$$
x^{**} = \left(\frac{W}{1-W}, \frac{W}{2-W}\right). \tag{A 6}
$$

For $x^* \leqslant \frac{1}{2}$, W in the above solutions must satisfy

$$
W \leqslant \tfrac{1}{3}, \quad W \leqslant \tfrac{2}{3}. \tag{A 7}
$$

(iv) For the SRO to invade when $f = \frac{1}{2}$, W must satisfy the following (by substituting for f in equation (1)):

$$
\tfrac{1}{2}Wt(1+x) > x \Rightarrow W > \tfrac{2}{3}. \tag{A 8}
$$

Hence, no solutions for x^{**} are valid from the quadratic factor, since W is strictly greater than $\frac{2}{3}$ from the invasion condition. Hence, the only valid solution for x in equation (A 5) is $x^{**} = 0$.

(*c*) *Distinction between subclasses of infected females*

The iterations (A 9) describe the model in which the transmission and feminization efficiencies (t_i, f_i respectively) of SROs in actively feminized hosts are distinguished from the transmission and feminization efficiencies of SROs in carrier females (t_c,f_c). Carriers are those hosts that inherit the SRO but become female as a result of host gene action rather than parasite induced feminization. Carrier and feminized females were assumed to be of equal fitness to uninfected females. P, P' represent prevalence of actively feminized females from one generation to next; Q, Q' represent prevalence of carriers.

$$
\begin{aligned}
P' &= (Pt_if_i+Qt_cf_c)/F_a, \\
Q' &= [Pt_i(1-f_i)\,x+Qt_c(1-f_c)\,x]/F_a, \\
F_a &= Pt_if_i+Qt_cf_c+Pt_i(1-f_i)\,x+Qt_c(1-f_c)\,x \\
&\quad +P(1-t_i)\,x+Q(1-t_c)\,x+(1-P-Q)\,x.
\end{aligned} \tag{A 9}
$$

For given values of f_i, t_i, f, t_c, numerical methods were used to calculate (1) equilibrium prevalences P^* and Q^* and (2) ESS host sex ratio x^*. Numerical methods were used to calculate costable prevalences P^{**} and Q^{**} and sex ratio x^{**}, by repeated substitution and iteration of processes (1) and (2) until either monogeny or stability was observed. The solution was deemed stable when x^{**} remained unchanged over 1000 iterations. The model yielded similar qualitative results to those for the case when $t_i = t_c$ and $f_i = f_c$ (all states I to IV were observed). The SRO was unable to invade ($P^* = 0$) for small parameter values. Monogeny ($x^{**} = 0$) was observed for high t_i, f_i, t_c, f_c, for $f_i = 1$, for $t_c = 0$ and for $f_c = 0$. Some intermediate values of the parameters ($f_c < f_i$ and/or $t_c < t_i$) yielded costable non-zero sex ratios $(0 < x^{**} < \frac{1}{2})$ and intermediate prevalences $(0 < P^{**} < 1)$.

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