Self-fertilization and inbreeding limit the scope for sexually antagonistic polymorphism

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Abstract

Sexual antagonism occurs when there is a positive intersexual genetic correlation in trait expression but opposite fitness effects of the trait(s) in males and females. As such, it constrains the evolution of sexual dimorphism and may therefore have implications for adaptive evolution. There is currently considerable evidence for the existence of sexually antagonistic genetic variation in laboratory and natural populations, but how sexual antagonism interacts with other evolutionary phenomena is still poorly understood in many cases. Here we explore how self-fertilization and inbreeding affect the maintenance of polymorphism for sexually antagonistic loci. We expected *a priori* that selfing should reduce the region of polymorphism, since inbreeding reduces the frequency of heterozygotes and speeds fixation. This expectation was supported, but although previous results suggest that the more an allele that is deleterious to one sex is dominant in that sex, the smaller the region of parameter space that will admit polymorphism, we found that this effect is weakened by self-fertilisation. However, the effect of inbreeding is not strong enough to completely cancel out the effect of dominance: for a given frequency of inbreeding, it will still be the case that the more dominant the alleles are in their deleterious context, the smaller the region of parameter space in which they can exist at polymorphism.

Introduction

Sexual antagonism occurs when there is a positive intersexual genetic correlation in trait expression but opposite fitness effects of the trait(s) in males and females (Bonduriansky & Chenoweth, 2009; it is also known as intralocus sexual conflict when the intersexual genetic correlation is for the same trait in both sexes). As such, it constrains the evolution of sexual dimorphism and may therefore have implications for adaptive evolution. It is also taxonomically ubiquitous and therefore of considerable potential importance in natural populations (Bonduriansky & Chenoweth, 2009; Cox & Calsbeek, 2009). Although in much of the literature sexual antagonism is discussed in terms of phenotypic traits, this phenomenon is most easily modeled as a genotype-by-sex effect on fitness, where a single allele has opposing effects on male and female fitness.

The role of partial selfing in influencing the dynamics of a sexually antagonistic locus has, however, rarely previously been considered (but see Jordan & Connallon, 2014). This is an important gap in our knowledge, given that a large number of plant and animal taxa are hermaphroditic and partially or completely selfing (Goodwillie *et al.*, 2005; Jarne & Auld, 2006). It has previously been assumed that, all else being equal, sexually antagonistic alleles in hermaphrodites should exhibit the same dynamics as autosomal sexually antagonistic loci in separate-sexed organisms (Arnqvist and Rowe, 2005; Morgan, 1994), since there is nothing in classic models of sexually antagonistic alleles that requires that the sexes be separate in the species being modelled (Kidwell *et al.*, 1977). This implies that selection must be strong and approximately equal in magnitude across the sexes in order for polymorphism to be maintained (Kidwell *et al.*, 1977). Selfing should reduce the region of polymorphism, since inbreeding reduces the frequency of heterozygotes and

speeds fixation, even when there is overdominance (or heterozygote advantage; Hayman, 1953; Hayman & Mather, 1953). One might therefore expect *a priori* that partially selfing hermaphrodites should exhibit lower levels of sexually antagonistic genetic variation compared to separate-sexed species or obligate outcrossers. However recent work by Fry (2009) demonstrates that dominance effects can have considerable influence on the region of parameter space permitting polymorphism.

Here, we extend the framework developed by Kidwell *et al.* (1977) and Fry (2009) to investigate how rate of selfing and dominance interact to affect the maintenance of polymorphism at a sexually antagonistic locus. Although Rice (1984) and Fry's (2009) results suggest that sex-specific dominance of fitness is an important factor in the maintenance of sexually antagonistic polymorphism, it is not immediately obvious how these effects will be modulated by inbreeding. We found that although inbreeding reduces the region of parameter space permitting polymorphism overall, it can offset some of the effects of sex-specific dominance demonstrated by Fry (2009).

Model

Our model is based on a classic framework for the investigation of sexually antagonistic alleles (Kidwell *et al.*, 1977). Our population is made up of a large number of diploid hermaphroditic individuals. We focus on a single locus, at which there are two alleles, denoted *A* and *a*. This means that every individual is one of three genotypes: *AA*, *Aa*, or *aa*, each of which confers a different fitness. Two key simplifying assumptions of the model are that:

1) Genotype does not influence rate of selfing
2) Genotype does not influence offspring production in self-fertilisation events

Although these assumptions are unlikely to hold in all cases, we believe that they are reasonably biologically realistic. Selfing is usually considered to be a form of reproductive assurance (Goodwillie *et al.*, 2005), and as such might often be determined by ecological factors (such as probability of encountering a potential mate) rather than pure genetic propensity (assumption 1). In addition, reproductive assurance will be ineffective if selfed gametes are subject to the same selection pressures as outcrossed gametes (assumption 2). We do however consider alternative scenarios briefly in the Discussion (see below).

We model generations as being discrete and non-overlapping. Within each generation, the life cycle goes as follows. We first census the genotypes in the population, and denote the frequency of genotype *AA* by p, and the frequency of genotype *aa* by q. Then the frequency of heterozygote *Aa* types is $1 - p - q$.

After censusing, random mating occurs. A proportion F of matings are self-fertilisation, while the remaining $(1 - F)$ matings are outbreeding events. For the self-fertilisation events there is no effect of genotype on offspring production. The genotypes of the offspring from self-fertilisation will depend on the parental genotype. Homozygous *AA* or *aa* individuals will produce their own genotypes for offspring, while heterozygous *Aa* individuals will have ¼ of their offspring of genotype *AA*, ½ genotype *Aa*, and ¼ genotype *aa*. Thus the frequency of each genotype in the next generation due to offspring from inbreeding events is as follows.

AA: $F\,(p + \tfrac{1}{4}(1 - p - q))$; *Aa*: $\tfrac{1}{2}\,F(1 - p - q)$; *aa*: $F\,(q + \tfrac{1}{4}(1 - p - q))$.

Because we are assuming random mating we can model each outbreeding event as being the result of the combination of sperm and eggs from randomly drawn individuals, with the probability of drawing a given genotype in a given sex role being proportional to the frequency of that genotype, and to its fitness in that sex role.

Fitness differs across genotypes and across each sex role, as summarised in Table 1. The *A* allele is female-beneficial, male-deleterious, so that bearing an *A* allele makes a hermaphroditic individual better at the female role but worse at the male role. Conversely, the *a* allele is female-deleterious, male-beneficial, so that bearing an *a* allele makes an individual worse at the female role but better at the male role. These deleterious and beneficial effects are summarised by the parameters s_f and s_m, which represent the selection coefficients, and h_f and h_m, which represent the dominance coefficients (Table 1).

Assuming a large population, the frequency of each genotype in the next generation due to offspring from outbreeding events is as follows. The frequency of *AA* individuals is

$$(1-F)\frac{\left(p+\frac{1}{2}(1-p-q)\left(1-h_f s_f\right)\right)\left(p(1-s_m)+\frac{1}{2}(1-p-q)(1-h_m s_m)\right)}{\bar{w}_f \bar{w}_m}$$

and the frequency of *aa* individuals is

$$(1-F)\frac{\left(\frac{1}{2}(1-p-q)\left(1-h_f s_f\right)+q\left(1-s_f\right)\right)\left(\frac{1}{2}(1-p-q)(1-h_m s_m)+q\right)}{\bar{w}_f \bar{w}_m}$$

where $\bar{w}_f$ and $\bar{w}_m$ are respectively the mean fitness in the female and male roles,

$$\bar{w}_f = p + (1-p-q)\left(1-h_f s_f\right) + q\left(1-s_f\right)$$

$$\bar{w}_m = p(1-s_m) + (1-p-q)\left(1-h_f s_f\right) + q$$

The frequency of *Aa* individuals is the balancing expression so that these three frequencies add to (1 – *F*).

Putting together both self-fertilisation and outbreeding events, we can derive expressions for the change in frequency of genotypes *AA* and *aa* from one generation to the next, denoted Δp and Δq respectively, as

$$\Delta p = F\left(p + \frac{1}{4}(1-p-q)\right)$$

$$+(1-F)\frac{\left(p + \frac{1}{2}(1-p-q)\left(1-h_f s_f\right)\right)\left(p(1-s_m) + \frac{1}{2}(1-p-q)(1-h_m s_m)\right)}{\overline{w}_f \overline{w}_m} - p$$

$$\Delta q = F\left(\frac{1}{4}(1-p-q) + q\right)$$

$$+(1-F)\frac{\left(\frac{1}{2}(1-p-q)\left(1-h_f s_f\right) + q(1-s_f)\right)\left(\frac{1}{2}(1-p-q)(1-h_m s_m) + q\right)}{\overline{w}_f \overline{w}_m} - q \qquad (1)$$

Using equations (1) we can establish whether the *A* and *a* alleles are protected from extinction when rare, and consequently whether polymorphism is protected or not (Appendix), depending on the values of parameters F, s_f, s_m, h_f, and h_m. When $F = 0$, we recover the classic results for this model (Fry, 2009; Kidwell *et al.*, 1977). Therefore in our analysis here we focus on the effect of F on the region admitting polymorphism.

Results

We can use (1) to calculate expressions a^* and A^* corresponding to protection when rare of *a* and *A*, respectively. The female-deleterious, male-beneficial allele *a* is protected from extinction where rare if

$$s_m > a^* = \frac{\left(F + 2h_f(1-F)\right)s_f}{2 - F\left(1-s_f\right) + 2(1-F)(h_f s_f - h_m)}$$

while the female-beneficial, male-deleterious allele *A* is protected from extinction when rare if

$$s_m < A^* = \frac{\left(F + 2(1-h_f)(1-F)\right)s_f}{\left(2h_m + F(1-2h_m)\right)(1-s_f)}$$

Then, for a given value of s_f, we know that the region of values of s_m admitting polymorphism will be where $a^* < s_m < A^*$. Thus the size of this region, which we denote by R, is equal to $R = A^* - a^*$. We want to know how changing F changes the value of R: if $\partial R/\partial F > 0$ then increasing F increases the region of parameter space that leads to polymorphism (and if $\partial R/\partial F < 0$ then increasing F decreases the region of parameter space that leads to polymorphism). We want to know the effect of F on these thresholds. Thus, we calculate

$$\frac{\partial a^*}{\partial F} = \frac{2(1 - h_f - h_m)s_f}{\left(2 - F(1 - s_f) + 2(1 - F)(h_f s_f - h_m)\right)^2}$$

$$\frac{\partial A^*}{\partial F} = \frac{2(h_f + h_m - 1)s_f}{\left(2h_m + F(1 - 2h_m)\right)^2(1 - s_f)}$$

and since $\partial R/\partial F = \partial A^*/\partial F - \partial a^*/\partial F$,

$$\frac{\partial R}{\partial F} = (h_f + h_m - 1)\left(\frac{2s_f}{\left(2h_m + F(1 - 2h_m)\right)^2(1 - s_f)} + \frac{2s_f}{\left(2 - F(1 - s_f) + 2(1 - F)(h_f s_f - h_m)\right)^2}\right)$$

Since the quotients in the right-hand brackets must both be positive (as in both cases the numerator and the denominator are positive), it follows that the sign of $\partial R/\partial F$ is the same as the sign of $h_f + h_m - 1$: increasing F increases the region of parameter space for polymorphism if $h_f + h_m > 1$, and reduces it (as well as reducing the region of unstable equilibrium) if $h_f + h_m < 1$ (Figure 1).

Interestingly, this result relates to two previous findings. Kidwell *et al.* (1977) showed that $h_f + h_m = 1$ represents a dividing point between models with a single stable polymorphic equilibrium ($h_f + h_m < 1$) and those with multiple equilibria ($h_f + h_m > 1$). Fry (2009) showed that in the absence of inbreeding, the higher the value of $h_f + h_m$, the smaller the region of parameter space admitting polymorphism. For any

fixed value of F this remains the case in our model. However, this dominance effect is weakened by inbreeding, because inbreeding results in fewer heterozygotes, and consequently the effect of h_f and h_m is weakened (Figure 1). The results when $F = 1$ are identical to the case when $h_f = h_m = 0.5$.

For weak selection (e.g. parameter values $0 < s_f, s_m < 0.1$) there is very little scope for polymorphism when $h_f + h_m > 1$ (Figure 1, see also Fry (2009)) regardless of the value of F. However, when $h_f + h_m < 1$, the range of parameter values for which there can be a sexually antagonistic polymorphism due to weakly selected alleles is severely curtailed by self-fertilisation (Figure 2).

In sum, although previous results suggest that the more an allele that is deleterious to one sex is dominant in that sex, the smaller the region of parameter space that will admit polymorphism (Fry, 2009), this effect is weakened by self-fertilisation (Figure 1). In partially selfing hermaphrodites we would therefore expect a greater number of dominant sexually antagonistic alleles remaining at polymorphism (and fewer recessive alleles) than if there were no selfing, at least for strongly selected loci. However, the effect of inbreeding is not strong enough to completely cancel out the effect of dominance; for a given frequency of inbreeding, it will still be the case that the more dominant the alleles are in their deleterious context, the smaller the region of parameter space in which they can exist at polymorphism (Figure 2).

Discussion

In hermaphrodites, the ability to self-fertilise will affect the maintenance or otherwise of sexually antagonistic polymorphisms. We expected *a priori* that selfing should reduce the region of polymorphism, since inbreeding reduces the frequency of

heterozygotes and speeds fixation (Hayman, 1953; Hayman & Mather, 1953). Although this expectation was supported, our results show that there is an interactive effect between the degree of selfing and dominance. This is perhaps unsurprising, because both the degree of overdominance and the rate of inbreeding are known to affect the region of polymorphism (with overdominance increasing and inbreeding decreasing the size of the region; Hayman, 1953; Hayman & Mather, 1953; Kimura and Ohta, 1971). By allowing sex-specific dominance effects, this results in net overdominance for fitness (Fry, 2009), causing the observed interactive effect between selfing and dominance. In particular, for weakly selected sexually antagonistic alleles which are on average partially recessive in their deleterious state, the range of parameter space allowing for a polymorphic equilibrium is strongly restricted in the case where there is inbreeding (Figure 2); if the alleles are on average partially dominant in their deleterious state, the region of unstable equilibrium is decreased for weakly selected loci, and the parameter space allowing for polymorphism for strongly selected loci is increased by inbreeding, but remains small. Although the outcome will depend on the distribution of dominance coefficients and fitness effects, we might expect that the more self-fertilisation occurs in a hermaphroditic species, the fewer sexually antagonistic polymorphisms will exist overall (assuming that most sexually antagonistic selection is weak).

It is of course well-established that selfing can lead to inbreeding depression, but that in habitually selfing organisms the benefits of selfing should outweigh the costs of inbreeding depression (Goodwillie *et al.*, 2005), leading us to make the two key assumptions listed in the Model section above. We assumed that genotype does not influence the rate of selfing, but this might not be the case if, for example, allocation to sperm versus eggs influences the decision to self or outcross.

For example, an individual investing heavily in sperm will have limited fecundity during selfing, but may have high production of offspring during outcrossing, leading to a higher rate of outcrossing in individuals with male-benefit/female-detriment alleles. We also assumed that selfed gametes will not experience selection. This assumption is realistic if most of the selection during outcrossing is due to extrinsic factors, such as sexual conflicts with the mating partner (Anthes & Michiels, 2007; Koene, 2006; Koene *et al.*, 2005) or energetic or predation costs of finding a mate (Jennions & Petrie, 1997). It becomes less realistic if the sexually antagonistic alleles cause intrinsic fitness differences (e.g. poor survival of gametes). Sperm (or pollen) limitation is unlikely to be a major limiting factor in fecundity when selfing (but see Hodgkin & Barnes, 1991), but it is not unlikely that mutations affecting egg quality/survival would have an effect even on the production of selfed offspring. If sperm are accompanied by toxic seminal fluid used in sperm competition when outcrossing, then this could also contribute to lower egg survival, even when selfing (Koene *et al.*, 2010; Schärer *et al.*, 2014). Indeed, a recent model by Jordan & Connallon (Jordan & Connallon, 2014) which takes asymmetric selection effects across sex functions into account and assumes that selfed gametes experience selection via inbreeding depression, found that selfing expands the region of parameter space which is favourable to female-beneficial alleles but restricts it for male-beneficial alleles. Collectively, their results and those presented here suggest that the ultimate effect of inbreeding on the maintenance of polymorphism is to some extent dependent on the assumptions about selection and genetic transmission to selfed versus outcrossed offspring.

It is also worth noting that our model, although originally constructed with hermaphrodites in mind, is equally applicable to separate-sexed organisms with

respect to inbreeding instead of selfing (Appendix). This generates some interesting avenues for future exploration, especially for populations with high levels of inbreeding, such as island populations (e.g. Grant *et al.*, 2003), or populations with low dispersal levels due to habitat fragmentation (e.g. Andersen *et al.*, 2004).

A recent model suggests that sexual antagonism and demography can interact to cause extinction of populations located in patches that are beneficial to male fitness and detrimental to female fitness (Harts *et al.*, 2014). This is because populations collapse if there are too few reproducing females. However populations which are declining in numbers should also become more inbred, as a result of the decreasing effective population size. If inbreeding facilitates purging of sexually antagonistic alleles, it would be interesting to see how this may influence outcomes in the model above.

The effect of inbreeding on the genomic location of polymorphic sexually antagonistic alleles is also an interesting issue. In a seminal paper, Rice (1984) argued that the X-chromosome should harbour increased levels of sexually antagonistic genetic variation because male-benefit loci that are recessive in females will be expressed in hemizygous males, but largely escape counter-selection in females at low to intermediate frequencies. Conversely, dominant female-benefit loci will also be more common on the X than on the autosomes, despite their deleterious effect in males, because the X spends more time in females than the autosomes (2/3 versus ½) and therefore experiences stronger total female-specific selection. However Fry's (2009) results suggest that when there is overdominance for fitness, then polymorphism of autosomal sexually antagonistic loci becomes possible over a wider region of parameter space. It is therefore currently unclear whether sex chromosomes are inevitably hotspots for sexual antagonism. Inbreeding could also play a role,

because any X- or Z-linked locus that is not completely recessive in the homogametic sex will be partially dominant overall (i.e. $h_f + h_m > 1$ will always hold true when $h_{homogametic} > 0$ because $h_{heterogametic} = 1$), and therefore subject to an increased range of polymorphism with increasing inbreeding level, according to the results in our model.

In sum, we show that although inbreeding reduces the region of parameter space permitting polymorphism overall, it can offset some of the effects of dominance demonstrated by Fry (2009). This means that although hermaphrodites with high levels of inbreeding are perhaps unlikely to harbour significant sexually antagonistic genetic variation, those segregating sexually antagonistic loci that do exist may be more likely to be partially dominant.

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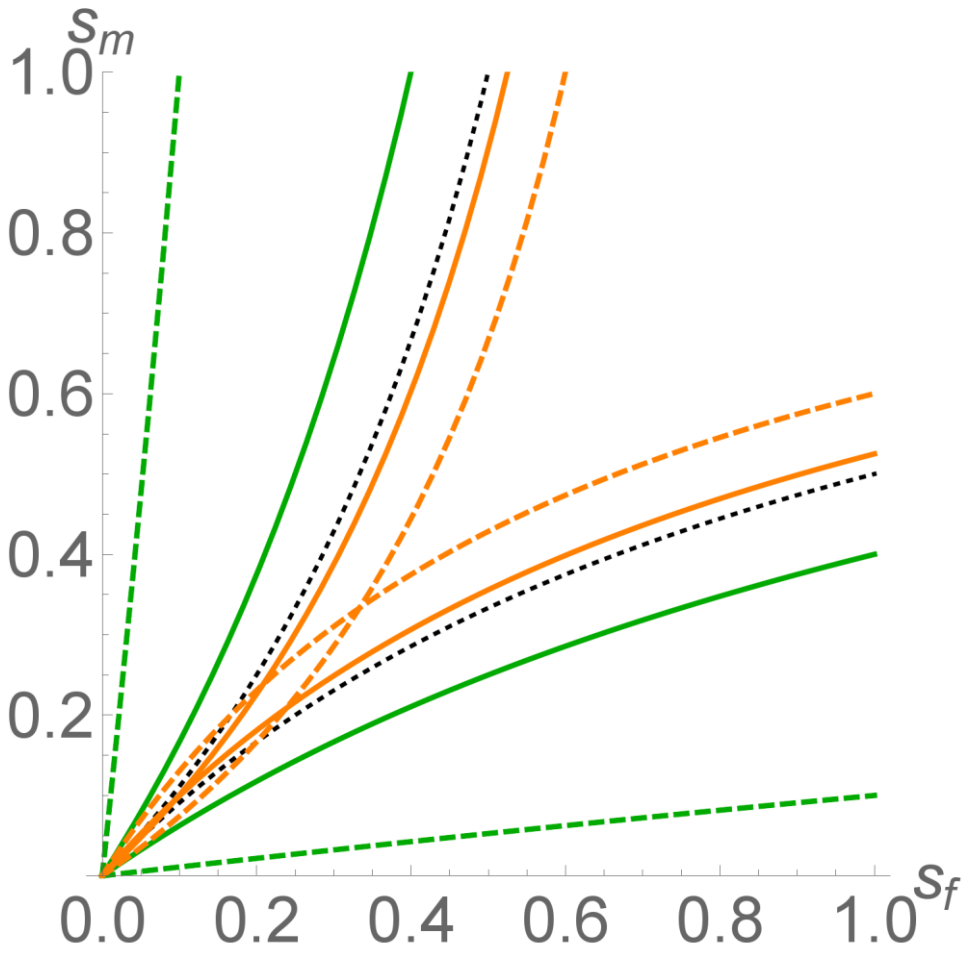

Figure 1

The effect of inbreeding and dominance on the maintenance of sexually antagonistic polymorphisms in hermaphrodites. The area between two matching curves is where polymorphism is admitted. The green curves correspond to the case where $h_f = h_m = 0.1$, so that the allele that is deleterious in each sex is partially recessive in that sex. The orange curves correspond to the case where $h_f = h_m = 0.6$, so that the allele that is deleterious in each sex is partially dominant in each sex. The dashed curves represent where $F = 0$, the situation where there is no inbreeding. The solid curves represent the case where $F = 0.75$, so that three quarters of matings are self-fertilisation. For the green curves, this results in a smaller area of polymorphism, while for the orange curves, it results in a larger area of polymorphism. The black dotted line is the

asymptotic limit $F = 1$. It exactly corresponds to the case in which $F = 0$ and $h_f = h_m = 0.5$.

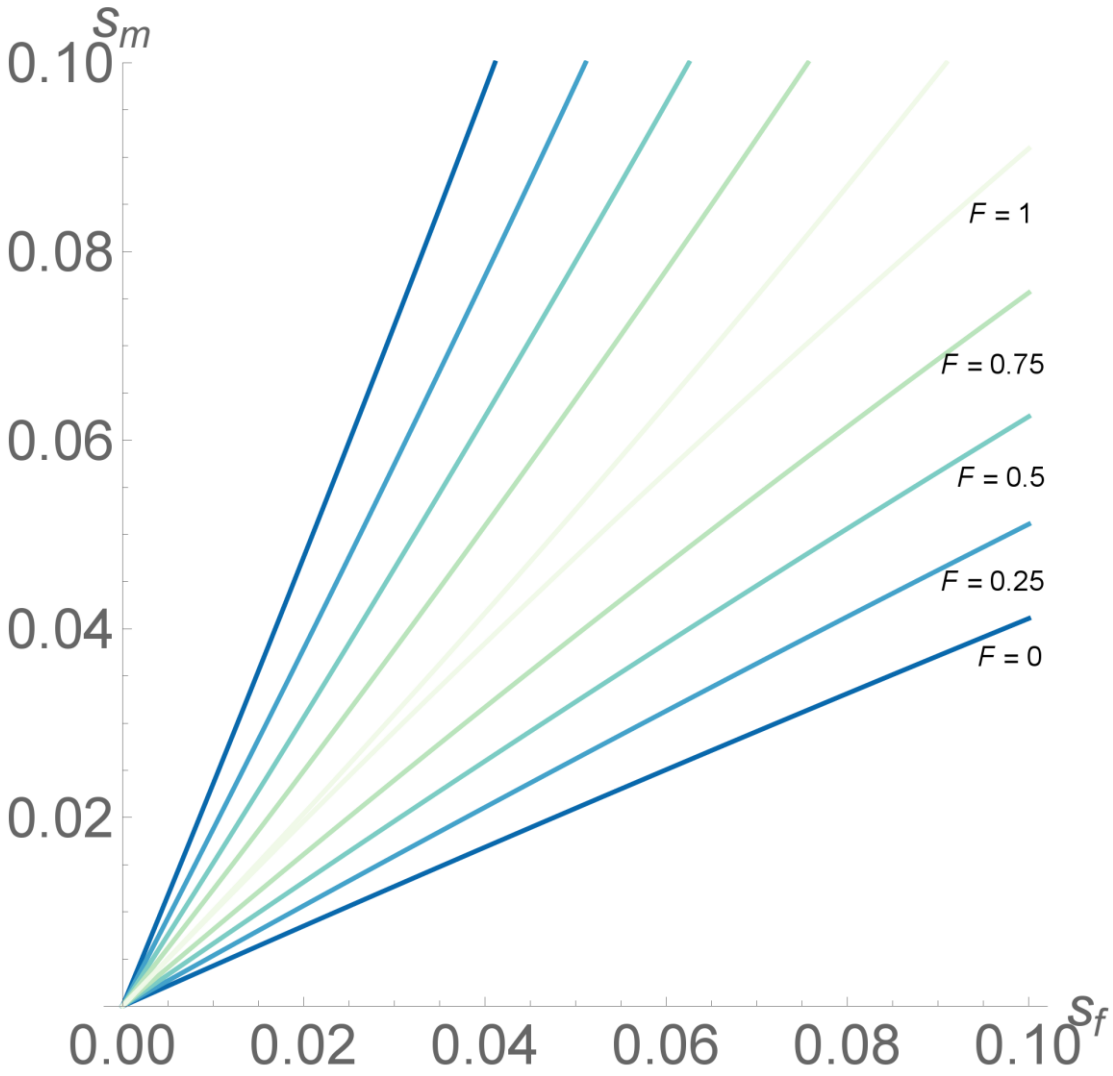

Figure 2

The effect of inbreeding on the maintenance of weakly sexually antagonistic polymorphisms. The region between any two matching-coloured lines admits a stable sexually antagonistic polymorphism. The pairs of matching lines correspond to the cases where F = 0, 0.25, 0.5, 0.75, and 1, respectively, as marked. Here $h_f = h_m = 0.3$; as inbreeding increases, the region admitting polymorphism decreases in size, to the limiting case where $F = 1$. For values of $h_f + h_m > 1$, the region admitting polymorphism is contained within the region for $F = 1$, and consequently for these dominance parameters there is very little scope for polymorphism under weak selection.

Tables

Genotype	Fitness in female role	Fitness in male role
AA	1	$1 - s_m$
Aa	$1 - h_f s_f$	$1 - h_m s_m$
aa	$1 - s_f$	1

Table 1: Fitness in different sex roles when outbreeding

Appendix

Stability of equilibria

Using equations (1) we can define the function $g[p, q] = (\Delta p, \Delta q)$, defined for all possible values of p and q (i.e. on the standard 2-simplex). We know that $g[1, 0] = (0, 0)$ (corresponding to fixation of the *A* allele), and $g[0, 1] = (0, 0)$ (corresponding to fixation of the *a* allele). To determine whether either of these two equilibria are stable we consider the Jacobian matrix **J** of the function g,

$$\mathbf{J} = \begin{pmatrix} \frac{\partial \Delta p}{\partial p} & \frac{\partial \Delta p}{\partial q} \\ \frac{\partial \Delta q}{\partial p} & \frac{\partial \Delta q}{\partial q} \end{pmatrix}$$

For each fixed point, we evaluate **J** and calculate its eigenvalues. If they are all negative for a given equilibrium point, that point is stable (thus if any of the eigenvalues are positive, the equilibrium point is unstable). If the equilibrium point at (1, 0) is unstable, then *a* is protected from extinction when rare (corresponding to the condition $s_m > a^*$ given in the main text). If the equilibrium point at (0, 1) is unstable, then *A* is protected from extinction from rare (corresponding to the condition $s_m < A^*$ given in the main text). If both alleles are protected from extinction when they are rare, then we have a protected polymorphism.

Applicability of model to separate-sexed species

Although the model was constructed to consider hermaphrodites, it can also apply to separate-sexed species. Because separate-sexed species cannot self-fertilise, the definition of F as the proportion of self-fertilising events cannot be maintained. Instead, F is taken to be a measure of the additional probability with which an individual will mate with a partner sharing the same genotype at the *A*/*a* locus of

interest (Appendix Table 1). Thus F can be seen as a measure of the level of inbreeding that is occurring in the population.

Focal genotype	Mating partner's genotype	Probability of that partner genotype under random mating	Probability of that partner genotype with "self-fertilisation frequency" F
AA	*AA*	p	$p + F(1 - p)$
	Aa	$1 - p - q$	$(1 - F)(1 - p - q)$
	aa	q	$(1 - F)\,q$
Aa	*AA*	p	$(1 - F)\,p$
	Aa	$1 - p - q$	$1 - p - q + F(p + q)$
	aa	q	$(1 - F)\,q$
aa	*AA*	p	$(1 - F)\,p$
	Aa	$1 - p - q$	$(1 - F)(1 - p - q)$
	Aa	q	$q + F(1 - q)$

Appendix Table 1: Application of F to separate-sexed species