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Sexual antagonism in haplodiploids

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Females and males may face different selection pressures, such that alleles conferring a benefit in one sex may be deleterious in the other. Such sexual antagonism has received a great deal of theoretical and empirical attention, almost all of which has focused on diploids. However, a sizeable minority of animals display an alternative haplodiploid mode of inheritance, encompassing both arrhenotoky, whereby males develop from unfertilized eggs, and paternal genome elimination (PGE), whereby males receive but do not transmit a paternal genome. Alongside unusual genetics, haplodiploids often exhibit social ecologies that modulate the relative value of females and males. Here, we develop a series of evolutionary-genetic models of sexual antagonism for haplodiploids, incorporating details of their molecular biology and social ecology. We find that: (1) PGE promotes female-beneficial alleles more than arrhenotoky, and to an extent determined by the timing of elimination—and degree of silencing of—the paternal genome; (2) sib-mating relatively promotes female-beneficial alleles, as do other forms of inbreeding including limited male-dispersal, oedipal-mating, and the pseudo-hermaphroditism of *Icerya purchasi*; (3) resource competition between related females inhibits the invasion of female-beneficial alleles; and (4) sexual antagonism foments conflicts between parents and offspring, endosymbionts and hosts, and maternal- and paternal-origin genes.

KEY WORDS: **Arrhenotoky, haplodiploidy, inbreeding, intralocus sexual conflict, paternal genome elimination, sexually antagonistic alleles.**

Organisms often appear remarkably well adapted to live the lives they do, as a consequence of the historical action of natural selection. Some of the best tests of our understanding of adaptation occur when organisms must make trade-offs between conflicting design objectives. Sexual antagonism is one such example, whereby genetic variants may prove beneficial to one sex but detrimental to the other. This has motivated a large body of theoretical work considering when such sexually antagonistic alleles will be able to invade (Owen 1953), how this may vary across the genome (Parsons 1961; Kidwell et al. 1977; Pamilo 1979; Rice 1984; Frank and Hurst 1996; Frank and Patten 2020; Hitchcock and Gardner 2020; Klein et al. 2021), and how we may be able to detect such alleles from population genetic data (Cheng and Kirkpatrick 2016; Kasimatis et al. 2019; Ruzicka and Connallon 2020; Ruzicka et al. 2020). This theory has been complemented more recently by molecular and quantitative genetic studies of laboratory and wild populations, both estimating the extent of sexual antagonism, and identifying specific loci at which sexually antagonistic alleles reside (Poissant et al. 2010; Mank 2017; Rowe et al. 2018; Connallon and Matthews 2019).

Almost all this research has focused on diploid, "eumendelian" (*sensu* Normark 2006) organisms. However, a sizeable minority of animals (∼15%) display an alternative, haplodiploid mode of inheritance (Normark 2003, 2006; Bachtrog et al. 2014). Haplodiploidy encompasses both arrhenotoky—whereby males develop from unfertilized eggs—and paternal genome elimination (PGE)—whereby males receive but do not transmit a paternal genome—and is employed by a diverse cast of creatures in groups as distinct as mites, nematodes, rotifers, springtails, beetles, wasps, and flies. In all of these organisms, males exclusively transmit maternal-origin genes, such that reproduction of females contributes twice as much to the ancestry of future generations as does that of males. While similarities in transmission genetics have drawn comparisons to X-linked genes (Kraaijeveld 2009; de la Filia et al. 2015), haplodiploids are not merely whole-organismal manifestations of X

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chromosomes. First, mechanisms of dosage compensation—that ensure an equal balance of X-linked versus autosomal gene products between females and males—are understood to play an important role in modulating sexual antagonism in relation to the X chromosome (Hitchcock and Gardner 2020), but it is unclear whether these mechanisms should apply in the same way in relation to arrhenotokous species in which males are haploid across their entire genome, and thus, might be able to achieve dosage compensation either passively, or through other mechanisms (e.g., additional endoreduplication) (Aron et al. 2005; Scholes et al. 2013). Second, although PGE is similar to X-linkage from a transmission perspective, this form of haplodiploidy involves males being somatically diploid through some or all of their lives (Burt and Trivers 2006; Gardner and Ross 2014; Klein et al. 2021), with concomitant gene dosage and dominance effects that may be expected to affect the balance between female- versus male-beneficial alleles.

Moreover, haplodiploids often exhibit characteristic social ecologies, including gregarious broods, chronic inbreeding, and strongly female-biased primary sex ratios (Hamilton 1967). An archetypal example is the date stone beetle (*Coccotrypes dactyliperda*), whereby a gravid female excavates a tunnel into a date seed and lays a large and heavily female-biased brood, her offspring then mate with each other, and her mated-daughters then leave to search for dates within which to raise their own families (Hamilton 1993; Spennemann 2019). While the particular niche that these species inhabit may vary substantially—from fungal-feeding to sap- or blood-sucking—they often share a similarly viscous population structure, with small, semi-isolated subpopulations, and large amounts of inbreeding (Hamilton 1967, 1978, 1993; Normark 2006). These unusual mating systems generate peculiar patterns of within-individual and between-individual relatedness, as well as differences in the scales at which the sexes compete and cooperate. Both of these factors are known to modulate the relative genetic value of males and females in the context of sex allocation (Taylor 1981; Frank 1986b; Nagelkerke and Sabelis 1996; West 2009), and thus, might also be expected to alter the outcome of sexually antagonistic selection.

Here, we investigate how the molecular biology and sexual ecology of haplodiploid organisms modulate the evolution of sexual antagonism, developing a general, theoretical overview, and presenting a series of evolutionary-genetic models to provide concrete illustration. We first consider how the genetic asymmetries found in haplodiploids are expected to alter the fate of sexually antagonistic alleles, and how this is modified by variation in the timing and expression of the paternal genome. We then explore how inbreeding alters these conditions, investigating the effects of sib-mating, lower male-dispersal, oedipal-mating, and the pseudo-hermaphroditism of *Icerya purchasi*, as well as the effect of local resource competition among females. Finally, we explore how such genetic and ecological asymmetries may foment conflicts over sexual antagonism between parents and offspring, endosymbionts and their hosts, and maternal- and paternal-origin genes.

Genetic Asymmetries

THE CONSEQUENCES OF ASYMMETRIC TRANSMISSION

In most sexual organisms, males and females pass on their maternal- and paternal-origin genes with equal frequency. In contrast, haplodiploid organisms are united by the fact that they break this fundamental symmetry, with males exclusively passing on maternal-origin genes (Normark 2006). The best-known form of this is arrhenotoky, whereby males are haploid, produced from unfertilized eggs, and thus, carry only a maternal-origin genome. Consequently, they are constrained to only ever transmit maternal-origin genes, and do so only to daughters. In another form of haplodiploidy, PGE, males are formed from fertilized eggs, and thus, initially contain both maternal- and paternal-origin genomes. However, either early during development (embryonic PGE) or during spermatogenesis (germline PGE), they eliminate their paternal genome, and thus, their sperm carries only genes of maternal origin (see Fig. 1).

These distinct transmission genetics alter the relative contributions that females and males make to the ancestry of future generations, that is, their reproductive values, which provide the weights upon selective changes occurring within these different classes of individual (Price 1970; Taylor 1990; Grafen 2006). Specifically, if we choose a random gene from the distant future and trace it back to the present generation, the probability c_f that it is currently carried by a female defines the class reproductive value of females, and the probability $c_m = 1 - c_f$ that it is carried by a male defines the class reproductive value of males. We find that—under the assumption of discrete, nonoverlapping generations—the ratio of these two reproductive values is given by $c_f/c_m = 2(1 - L)$, where L is the probability that males transmit their paternal genome. Under conventional diploidy, we have $L = 1/2$ and, hence, $c_f/c_m = 1$, that is, both sexes make an equal genetic contribution to future generations. In contrast, under haplodiploidy, we have $L = 0$ and, hence, $c_f/c_m = 2$, such that females collectively make twice the genetic contribution made by the males. In some haplodiploid species, including mealybugs and body lice, imperfect elimination of the paternal-origin genome has been documented, such that in these species males do occasionally transmit paternal-origin genes, that is, $0 < L < 1/2$ (de la Filia et al. 2018, 2019). As the extent of male paternal transmission L increases, then males obtain an increasing share of the ancestry of future generations (Gardner and Ross

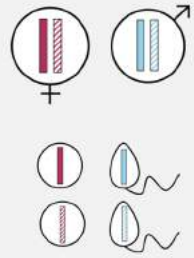
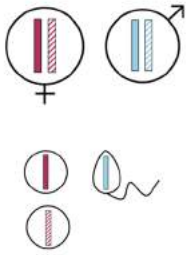
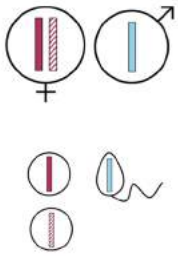
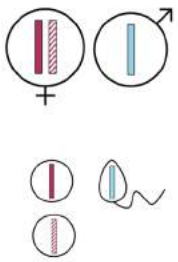
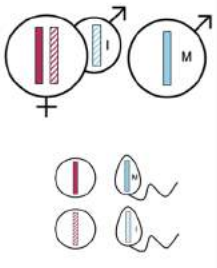
	Genetic system		Reproductive values	Examples
Eumendelian	Diploidy		$c_f = 1/2$ $c_m = 1/2$	
Haplodiploids	Germline PGE		$c_f = \frac{2-2L}{3-2L}$ $c_m = \frac{1}{3-2L}$	 **Collembola**: Symphypleona **Coleoptera**: Cryphalini, *Hypothenemus hampei* **Diptera**: Cecidomyiidae, Sciariae **Hemiptera**: Neococcoidea **Phthiraptera**: *Pediculus humanus humanus* **Psocodea**: Troctomorpha
	Embryonic PGE		$c_f = \frac{2-2L}{3-2L}$ $c_m = \frac{1}{3-2L}$	 **Acari**: Phytoseiidae, Otopheidomenidae, Ascidea **Hemiptera**: Diaspididae
	Arrhenotoky		$c_f = \frac{2}{3-\mathcal{O}}$ $c_m = \frac{1-\mathcal{O}}{3-\mathcal{O}}$	 **Coleoptera**: *Micromalthus debilis* **Hemiptera**: Aleyrodidae **Hymenoptera** **Thysanoptera** **Oxyurida** **Acari**
	Pseudo-hermaphroditism in Icerya		$c_f = \frac{2(1-\varphi)}{3-2\varphi}$ $c_i = \frac{\varphi}{3-2\varphi}$ $c_m = \frac{1-\varphi}{3-2\varphi}$	 **Hemiptera**: *Icerya purchasi*, *Gigantococcus bimaculatus*, *Crypticerya zeteki*

Figure 1. Conceptual description of the different inheritance schemes, and examples of species and groups which fall into these categories, summarized from Gardner and Ross (2014); de la Filia et al. (2015); and Hodson et al. (2017). Solid colors represent maternal-origin genes, and dashed are paternal-origin genes. In PGE systems, L is the degree of paternal genome leakage. Under arrhenotoky, $\mathcal{O}$ is the proportion of oedipal mating. In the *Iceryan* pseudo-hermaphroditism, φ is the proportion of eggs fertilised by the infectious male tissue (I), with $1-\varphi$ the proportion fertilized by true males (M). Images in order from top to bottom: *Canis familiaris* (Samantha Sturiale), Body louse (public domain), *Aulacaspis yasumatsui* (Jeffrey W. Lotz, Florida Department of Agriculture and Consumer Services, Bugwood.org), *Haplothrips subtilissimus* (Andy Murray, chaosofdelight.org), *Icerya purchasi* (public domain).

2011; Yeh and Gardner 2012), scaling between the extremes of diplodiploidy and haplodiploidy. This effect of paternal transmission in PGE species can, thus, be thought of as conceptually similar to cases of "paternal leakage" in cytoplasmic elements which also increase the reproductive value of males, and decrease that of females (Rand et al. 2001, Kuijper et al. 2015; Hitchcock and Gardner 2020).

If a sexually antagonistic allele confers a marginal fitness benefit σ to one sex, and a marginal fitness cost τ to the other, then the condition for a sexually antagonistic variant to invade from rarity will—under weak selection, outbreeding and in the absence of social interactions between relatives—be $c_f\sigma > c_m\tau$ if the allele is female-beneficial, and $c_f\tau < c_m\sigma$ if it is male-beneficial (Hitchcock and Gardner 2020). A female-beneficial allele will, therefore, invade under haplodiploidy provided $2\sigma > \tau$ while it will only invade under diploidy provided $\sigma > \tau$. Accordingly, for a given ratio of benefit and cost, the transmission genetics of haplodiploidy acts to promote the invasion of female-beneficial alleles (and inhibit the invasion of male-beneficial allele), relative to eumendelian diploidy, just as the transmission genetics of X chromosomes does (Frank and Patten 2020; Hitchcock and Gardner 2020; Klein et al. 2021). With imperfect PGE, invasion conditions will be $2(1-L)\sigma > \tau$ for a female-beneficial allele, and $\sigma > 2(1-L)\tau$ for a male-beneficial one. Thus, paternal escape relatively promotes the invasion of male-beneficial alleles, and limits the invasion of female-beneficial alleles. In natural populations, levels of paternal escape are probably relatively low (in *Planococcocus citri*, the proportion of paternal transmission was estimated to be between 0.37 and 3.39%; de la Filia et al. 2019), and thus, very similar to the conventional haplodiploid case. Nonetheless, slight differences in the degree of leakage between populations, such as those documented between ecotypes of *Pediculus humanus* (de la Filia et al. 2018), or potentially experimentally induced paternal leakage, may allow for effective comparative tests.

ASYMMETRIC PLOIDY AND GENE EFFECTS

While the different haplodiploid systems are united by their common transmission genetics, they often show distinct somatic genetics (Fig. 1). These differences in the number of gene copies carried by males and females, and the particular expression patterns of those genes, may alter the relative magnitude of allelic effects in males and females (i.e., the marginal costs τ and benefits σ described above), and thus, further shape the dynamics of sexual antagonism.

Under arrhenotoky, females carry two genes at each locus, while males carry only one. This is conceptually similar to the X chromosomes in an XO system (or an XY system insofar as there is no homologue on the Y) and, as with X chromosomes, it is not necessarily straightforward to compare relative fitness effects across ploidy levels. If an allele's effect is of similar magnitude in a homozygous and a hemizygous setting, then this will mean that alleles will typically have larger effects on average when expressed in males than in females (Charlesworth et al. 1987; Orr and Otto 1994; Hitchcock and Gardner 2020). For example, if we consider the fitness scheme outlined in Table 1, whereby an allele confers a fitness benefit S when in hemizygous or homozygous form, then assuming that the allele is vanishingly rare in the population ($p \to 0$) and that allelic effects are additive ($h_f = 1/2$), a gene expressing this variant strategy will have a marginal fitness effect of $\sigma = S/2$ if female-beneficial, but $\sigma = S$ if male-beneficial (Table 1). Alternatively, we might assume that a mutant allele's effect scales with its absolute rather than relative dosage in the genome (Frank 2003; Gardner 2012; Davies and Gardner 2014), in which case, the marginal fitness effects will not systematically differ across the sexes ($\sigma = S/2$ for both males and females). While here we follow the typical assumption of hemizygote/homozygote equivalence (Table 1), given mechanisms of apparent dosage compensation in some species—such as compensatory endoreduplication in polyploidy tissues of Hymenoptera (Rasch et al. 1977; Aron et al. 2005; Scholes et al. 2013) and differential methylation of haploid and diploid male ants (Glastad et al. 2014)—in certain tissues and biological processes it may be more accurate to assume that gene effects scale with absolute copy number, or indeed somewhere in between.

In contrast to arrhenotoky, under PGE, both males and females are initially diploid. If both gene copies within the individual are expressed then, for both males and females, the marginal fitness benefit will be $\sigma = S/2$, as is also the case for eumendelian diploidy (Table 1). However, among PGE systems there is a diversity in the extent of somatic paternal genome expression. This may occur either because the whole or a part of the paternal genome is eliminated early in development (embryonic PGE), such that somatic tissues are actually haploid, or because the paternal genome is silenced, such that certain tissues are functionally haploid (Burt and Trivers 2006; de la Filia et al. 2021). If a locus is exclusively maternally expressed, then marginal fitness effects are identical to those given for arrhenotoky. Thus, depending on species, tissue, and locus, we expect to observe a continuum between these two scenarios (Fig. 2). For simplicity, we henceforth assume that both gene copies are fully expressed under PGE, a scenario that captures autosomal expression in several PGE clades including springtails, parasitic lice, fungus gnat, and gall midge flies (de la Filia et al. 2015; Fig. 1). It also captures the evolution of a subset of genes and tissues in species, such as mealybugs, where paternal genome silencing appears to be incomplete (de la Filia et al. 2021). In contrast, species with embryonic PGE (Fig. 1) are equivalent to arrhenotokous species as males become fully haploid early in development.

Table 1. Fitness scheme for invasion analysis.

		Female benefit/Male cost	Male benefit/Female cost
Female genotypes	F_{00}	1	1
	F_{01}/F_{10}	$1 + h_f\ S$	$1 - h_f\ T$
	F_{11}	$1 + S$	$1 - T$
Male genotypes	M_{00}/M_0	1	1
	M_{01}/M_{10}	$1 - h_m\ T$	$1 + h_m\ S$
	M_{11}/M_1	$1 - T$	$1 + S$
Diploidy/	σ	$h_f S$	$h_m S$
Germline PGE	τ	$h_m T$	$h_f T$
Arrhenotoky/	σ	$h_f S$	S
Embryonic PGE	τ	T	$h_f T$

Marginal fitness effects are calculated when the allele is vanishingly rare (i.e., $\lim_{p\to 0}\sigma(p)$, $\lim_{p\to 0}\tau(p)$) and when there is no population structure.

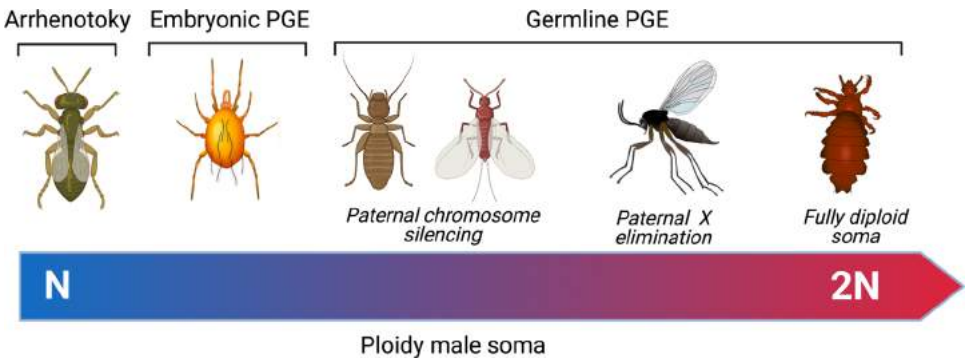


Figure 2. The continuum of male gene expression and, thus, effective ploidy level found across haplodiploid groups with representative taxa illustrated. From left to right, *Nasonia vitripennis*, predatory mite (*Phytoseiulus persimilis*), Liposcelis booklice, citrus mealybug male (*Planococcus citri*), Hessian fly (*Mayetiola destructor*), and head louse (*Pediculus humanus capitis*). Figure created with BioRender.com.

A further factor that may modulate the relative scaling of gene effects across sexes is dominance (Rice 1984; Fry 2010; Patten 2019). Dominance coefficients allow for nonadditive scalings of allelic effects between the two homozygous genotypes (Table 1). Relaxing our above assumption about additivity, a female-beneficial allele will confer a marginal fitness benefit of $\sigma = h_f\ S$ under diploidy, PGE, and arrhenotoky (Table 1), while it will confer a marginal fitness cost of $\tau = h_m\ T$ under diploidy and PGE, but $\tau = T$ under arrhenotoky (Table 1). Thus, we can see that for a given S and T the marginal cost to benefit ratio ($\tau : \sigma$) will be equal under diploidy and PGE, but will be systematically larger under arrhenotoky, with the extent determined by the values of h_f and h_m. The reverse of course will occur when the allele is male-beneficial, with the cost to benefit ratio ($\tau : \sigma$) is smaller under arrhenotoky than under PGE. Dominance effects may arise for multiple reasons including nonadditive physiology and nonlinear fitness landscapes. Depending on the assumption about the source of dominance, then different assumptions may be made about how dominance in one sex relates to dominance in the other, and similarly how the dominance of beneficial alleles relates to those of deleterious ones, with these assumptions shaping the marginal costs and benefits experienced (Fry 2010; Patten 2019). For simplicity, we restrict our attention largely to the additive case, however, the consequences of two sets of assumptions about the nature of dominance—equal dominance ($h_f = h_m$) and dominance reversals ($h_f = 1 - h_m$)—can be seen in Figure 3, with full results for arbitrary dominance to be found in the supplementary material (SM) §2.4.1.

Integrating the weightings from transmission with the marginal fitness effects, we find that, following the fitness scheme in Table 1 and assuming outbreeding, the condition for a female-beneficial allele to invade from rarity will be $2h_f S > T$ under arrhenotoky and $2h_f S > h_m T$ under male PGE. For a male-beneficial allele, the invasion conditions will be $S > 2h_f T$ and $h_m S > 2h_f T$ under arrhenotoky and male PGE, respectively (full methods can be found in SM 2.1–2.2). Note that results for arrhenotoky are identical to the invasion conditions for X-linked alleles with full dosage compensation (Rice 1984; Patten 2019), and assuming equal dominance ($h_f = h_m$) also recovers the invasion conditions for PGE reported by Klein et al. (2021).

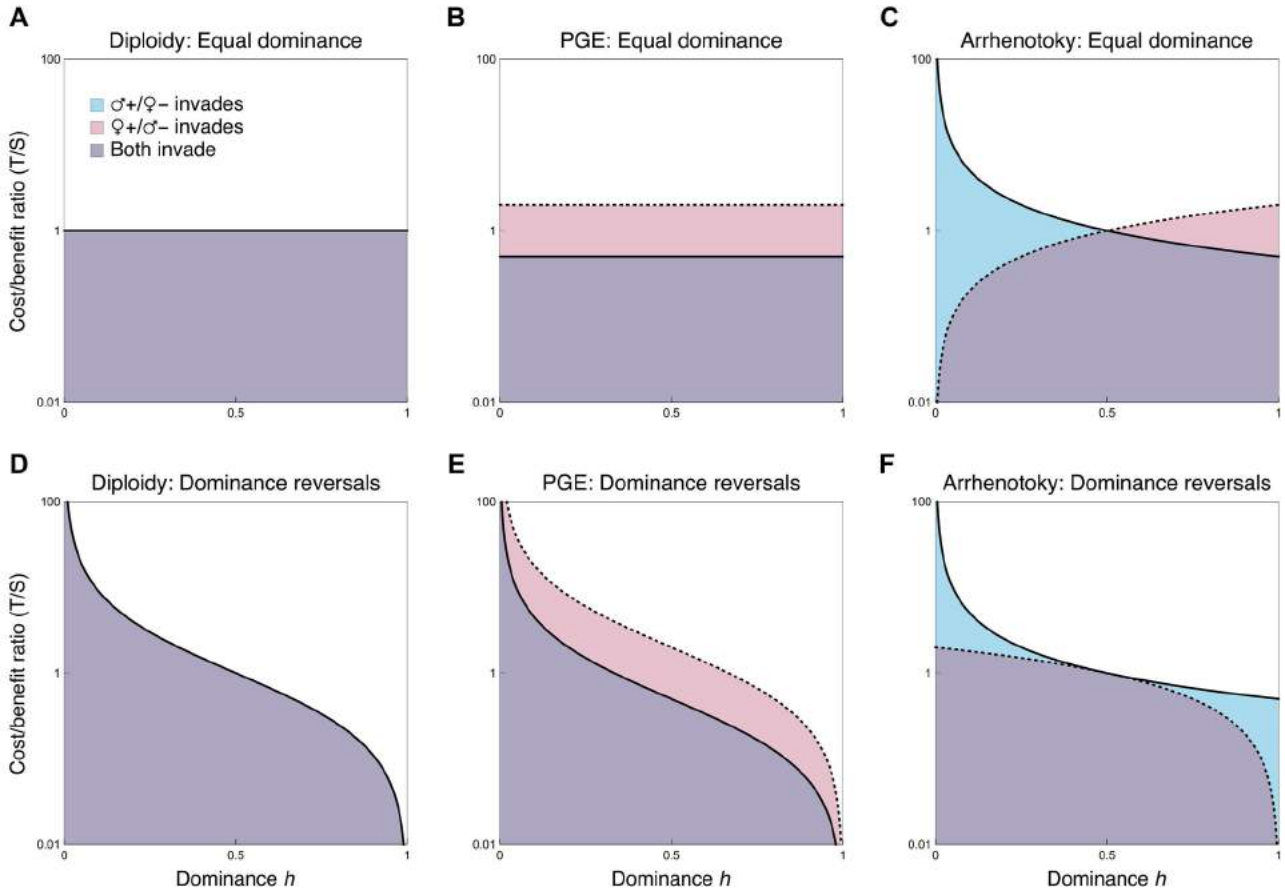


Figure 3. **Female-beneficial alleles invade more readily under germline PGE than they do under arrhenotoky. The invasion space for sexually antagonistic mutations with a given genotypic cost/benefit ratio (T/S) under different inheritance schemes and assumptions about dominance (h), with male-beneficial alleles invading beneath the solid line, and female-beneficial alleles beneath the dotted line. In the equal dominance scenarios (a-c): $h = h_f = h_m$ for both male- and female-beneficial alleles. In the reversals of dominance scenarios (d-f): $h = h_f = 1 - h_m$ for the male-beneficial scenario, and $h = 1 - h_f = h_m$ for the female-beneficial scenario.**

Under arrhenotoky (as with X-linked genes), the twofold weighting placed on females will be cancelled out by the twofold larger fitness effects in males (assuming $h_f = 1/2$). In contrast, under male PGE, where marginal fitness effects are not systematically different across sexes, this cancellation does not occur. Thus, we would generally expect relative feminization of the genome in PGE species as compared to arrhenotokous ones, as invasion conditions for female-beneficial alleles are less stringent, and those male-beneficial alleles are more stringent (Fig. 3).

Ecological Asymmetries

SIB-MATING AND ECOLOGICAL ASYMMETRIES BETWEEN THE SEXES

The above results apply to outbreeding populations with no social interactions between relatives, and therefore, it is only the direct fitness effects of alleles that required consideration. But many haplodiploid species diverge from this, with mating schemes and life cycles that result in chronic inbreeding (Hamilton 1967, 1978, 1993). These population structures may alter the relatedness within and between individuals, as well as the intensity with which males and females compete with relatives, potentially generating indirect fitness effects of sexually antagonistic alleles upon social partners. Such factors have long been recognized in sex allocation research to alter the relative value of sons and daughters (Taylor 1981; Frank 1986b; Nagelkerke and Sabelis 1996; West 2009), and thus, may be expected to play a similar role with regards to sexual antagonism.

We investigate how inbreeding may modulate sexual antagonism by modeling a population of monogamous females, in which a proportion s of females in the brood mate with their sibs, while a proportion $1 - s$ mate with males from the population at large (Fig. 4). Introducing sib-mating has multiple distinct effects upon sexual antagonism. The first is that sib-mating inflates the consanguinity of an individual to themselves, that is, their inbredness (*sensu* Frank 1986a), which has a feminization promoting effect under arrhenotoky—as a gene copy will have indirect fitness effects upon the other identical by descent gene copy

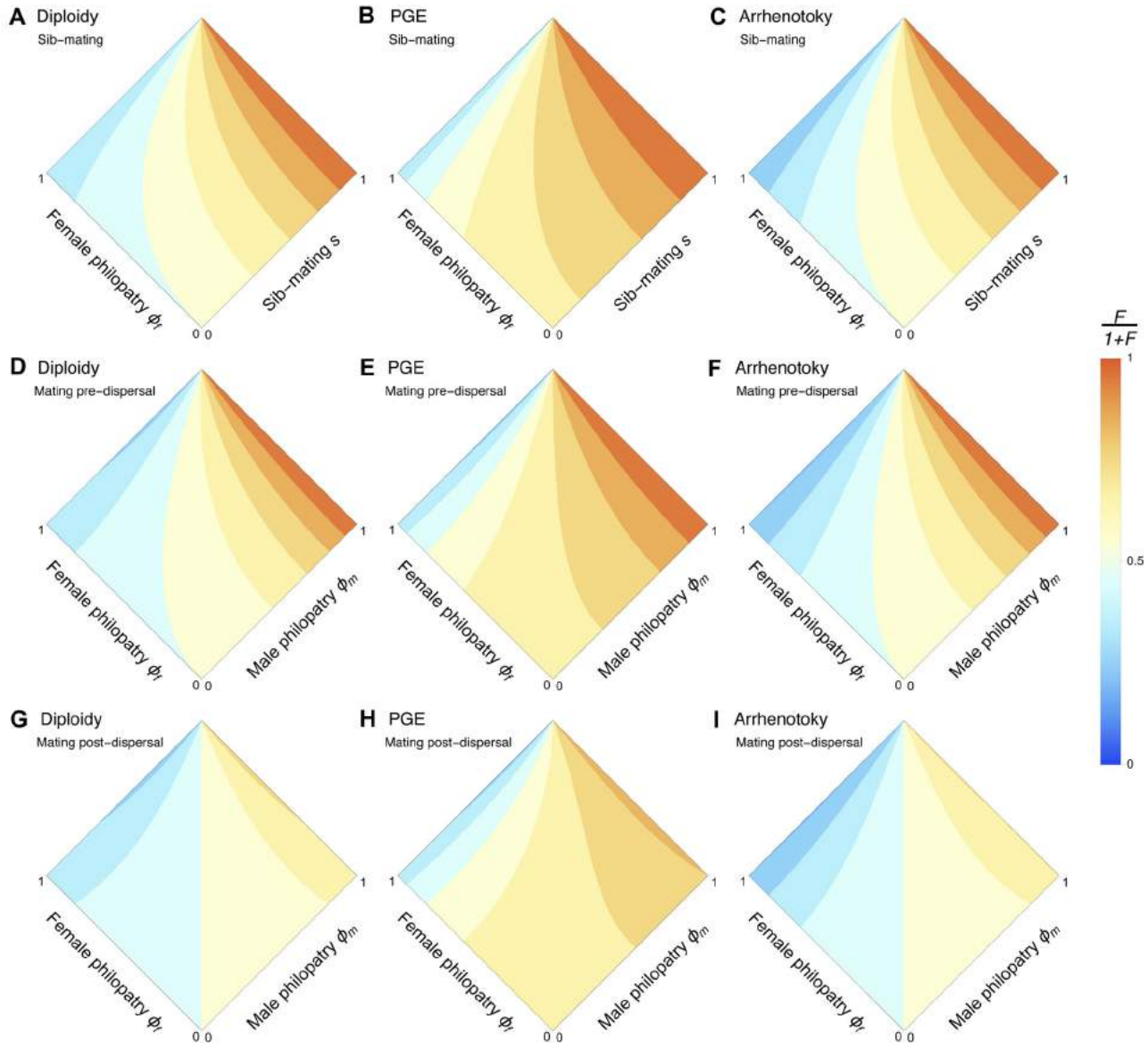


Figure 4. **Mating ecology and dispersal modulate the degree of feminization. Here, the degree of feminization, $F/(1+F)$, is plotted as a function of either the amount of male and female philopatry, or the amount of female philopatry and the proportion of sib-mating, under three inheritance systems (diploidy, germline PGE, and arrhenotoky), and for three mating ecologies (sib-mating [A-C], viscous population with mating prefemale dispersal [D-F], and viscous population with mating postfemale dispersal [G-I]).When $F/(1+F) > 0.5$, then feminization is expected, and when $F/(1+F) < 0.5$ masculinsation is expected.**

in females, but not in males, which are haploid (Tazzyman and Abbott 2015; Hitchcock and Gardner 2020)—but not for PGE or diploidy, where gene copies in both males and females experience these within individual indirect fitness effects. Second, sib-mating increases the probability that males will compete with brothers for mates, discounting the inclusive-fitness benefits of male-beneficial alleles to their male carriers, and mollifying the inclusive-fitness costs of male-deleterious alleles. Third, the direct fitness effects of alleles upon their female carriers will have indirect fitness effects upon their carriers' mates. If females sib-mate, then female-beneficial alleles will generate indirect benefits for their brothers, and female-deleterious alleles will impose indirect costs. All three of these effects have parallels in sex allocation, with increased sib-mating increasing the relatedness of a female to her daughters but not her sons under arrhenotoky, increased competition between brothers decreasing the genetic returns on males (i.e., local mate competition; Hamilton 1967), and increased sib-mating meaning that increased investment into daughters will increase the fitness of sons, either through extra mating opportunities, or through higher quality mates (Taylor 1981; Frank 1986b; West 2009;). While here we focus on the additive case, it is worth noting that sib-mating (and inbreeding

more generally) will also negate the effects of dominance by inflating the proportion of individuals who are homozygous (with concomitant effects on the maintenance of polymorphisms) (Jordan and Connallon 2014; Flintham et al. 2021; SM §2.5).

Collecting these effects, we write out the condition for a female-beneficial allele to invade. We then rearrange that condition into the form $F > T/S$, where F describes the "potential for feminization" (cf. Gardner 2010). This term bundles together the various weightings that are placed upon T and S, whether they emerge from aspects of the ecology or genetic system, providing a threshold that the cost-to-benefit ratio must not exceed in order for a female-beneficial allele to invade, and thus, F describes the stringency of those invasion conditions. For example, the result discussed above for arrhenotokous organisms, $2h_fS > T$ (Rice 1984), rearranges to give $F = 2h_f$. When $F = 1$, then the cost T simply has to be less than the benefit S for the allele to invade–as in the additive eumendelian diploid case. But as F increases, then the condition $F > T/S$ becomes easier to satisfy, and alleles which confer greater costs than benefits (i.e., $T/S > 1$) may yet be able to invade. Conversely, as F decreases, then the condition $F > T/S$ becomes harder to satisfy, and alleles which confer greater benefits than costs (i.e., $T/S < 1$) may be unable to invade. A similar approach may be taken to write out a potential for masculinzation (i.e., a male-beneficial allele invades when $M > T/S$) and, so long as the alleles under comparison do not differ with respect to dominance between the two scenarios, this is the reciprocal of the potential for feminization ($F = 1/M$). Thus, as conditions becomes less stringent for female-beneficial alleles to invade, they necessarily become more stringent for male-beneficial alleles. Therefore, when $F > 1$ feminisation is expected, and when $F < 1$ masculination is expected. Note that this potential for feminisation is distinct from other uses of feminisation in work on sex ratio distorters (e.g. Hatcher and Dunn 1995). Assuming additivity and weak selection, we find that under arrhenotoky and diploidy $F = 1/(1-s)$, and under male PGE $F = (4 - s)/(2\,(1 - s))$. Thus, we find that, across all these genetic systems, increased sib-mating promotes feminisation, with the effect being strongest under PGE (see Fig. 4). Results for nonadditive scenarios and stronger selection regimes can be found in SM §§2.3–2.5, with full methodology outlined in SM §1.1 and SM §§2.1–2.2.

So far, we have assumed that females compete globally, however, many haplodiploid species have more generally viscous populations in which females may also disperse short distances—if at all. For instance, in the date stone beetle, females may start their own families within the seed in which they were born (Spennemann 2019). Similarly, in many mealybugs, females crawl relatively small distances away from their natal patch (Varndell and Godfray 1996; Ross et al. 2010a). In these species, females may compete with sisters for breeding spots, just as their brothers competed with each other for mates, that is, local resource competition (Clark 1978). Incorporating these factors yields two further consequences for sexual antagonism. First, with limited female dispersal, direct fitness benefits to females incur indirect fitness costs to their sisters by depriving them of breeding spots, just as obtained for local mate competition in males. Second, while a fit female confers indirect fitness benefits upon brothers with whom she mates, she may also incur indirect fitness costs by competing with her brothers' mates, and thereby, indirectly depriving her brothers of reproductive success. With increasing local resource competition, the invasion condition becomes less stringent for male-beneficial alleles and more stringent for female-beneficial alleles. The dual effects of sib-mating and limited female dispersal can be seen in Figure 4, with full analytical results in SM §2.4.1.

ALTERNATIVE LIFE-CYCLES AND MODES OF INBREEDING

Above, we considered one particular inbreeding scenario, in which a fixed proportion of matings is reserved for siblings. However, the specific mechanism by which inbreeding occurs may also modulate sexual antagonism, as different mating schemes and life cycles will differ in how relatedness builds up, and how intensely males and females compete with relatives. To investigate this, we contrast the above model with an alternative involving a patch structured population in which the degree of inbreeding is modulated by the extent of dispersal (Wright 1931), whereby males remain on their natal patch with probability ϕ_m, and females with probability ϕ_f. We consider two variants, the first in which mating occurs before female dispersal (male dispersal → mating → female dispersal, DMD), and a second in which mating occurs after female dispersal (male dispersal → female dispersal → mating, DDM), the latter of which has been recently investigated by Flintham et al. (2021) for sexual antagonism in relation to diploidy and X-linkage. Comparing these results, we obtain a ranking of highest potential for feminization under sib-mating, followed by DMD, and finally DDM (Fig. 4, see SM §1.1 for life-cycle details, SM §§2.1–2.2 for Methods, and SM §2.4.1 for Results). The sib-mating and DMD scenarios are very similar, except that brothers are more likely to compete for mating opportunities in the former scenario, promoting feminization, analogous to the difference between fixed self-fertilization and mass-action selfing models of hermaphroditic plants (Jordan and Connallon 2014). Compared to DDM, both sib-mating and DMD scenarios yield a higher potential for feminization, as they involve both higher rates of consanguineous mating and also sisters conferring fitness benefits upon related mating partners, an effect that is exactly cancelled under DDM by increased competition between females and their brothers' mates. Thus, different

mating ecologies and life-cycle structures yield different patterns of feminization.

Alongside the generic demographies discussed above, haplodiploids present a striking variety of unusual lifecycles and modes of inbreeding. For illustration, we consider two scenarios in detail, both of which involve females effectively engaging in "selfing". First, oedipal mating (Fig. 1) occurs because a virgin female may produce an exclusively male brood with which she then mates, a reproductive strategy observed in groups including mites (McCulloch and Owen 2012; Tuan et al. 2016), beetles (Entwistle 1964; Jordal et al. 2001), parasitoid wasps (Browne 1922; Schneider et al. 2002), pinworms (Adamson and Ludwig 1993), and thrips (Ding et al. 2018). Second, in the scale insect *I. purchasi*, selfing is understood to occur as a consequence of a diploid female containing a transovarially transmitted haploid spermatogenic cell lineage that may fertilize her eggs (Royer 1975; Normark 2009; Ross et al. 2010b; Mongue et al. 2021). While these two systems are very different in their biological details, in both cases, we find that higher rates of "selfing" increases the potential for feminization, and do so in a fashion that is qualitatively very similar to sib-mating (see SM §§1.1, 2.1–2.2 for Methods, and SM §2.4.3 for Results).

Conflicts Over Sexual Antagonism

PARENT-OFFSPRING CONFLICT OVER SEXUALLY ANTAGONISTIC TRAITS

In the foregoing, we have assumed that the sexually antagonistic traits of interest are under the sole control of the individuals in which they are expressed. However, an individual's traits may also be influenced by social partners. In particular, parents may play an important role in shaping the traits of their offspring, whether it be through the material constitution of the zygote, the environment in which those offspring develop, or through the care that those parents provide (Mousseau and Dingle 1991; Mousseau and Fox 1998; Crean and Bonduriansky 2014; Bebbington and Groothuis 2021). For example, in the spider mite (*Tetranychus urticae*), maternal environment is known to affect offspring traits including juvenile survival (Marinosci et al. 2015), dispersal behavior (Bitume et al. 2014), and diapause induction (Oku et al. 2003). If the traits that they influence are sexually antagonistic, then parents may face a trade-off between crafting superior daughters versus superior sons. Moreover, if parents place different values upon males and females as compared to their offspring, then this may lead to parent-offspring conflict (*sensu* Trivers 1974) with respect to sexually antagonistic traits. Furthermore, if mothers and fathers also differ in their relative valuations of sons and daughters then this may lead to sexual conflict (*sensu* Trivers 1972) with respect to sexually antagonistic traits.

Focusing our attention first on genes acting through mothers, if we consider the invasion of an allele which increases the fitness of her daughters, but decreases the fitness of her sons, then for diploidy the potential for feminization may be expressed as $F = (1+s)/(1-s)$. When there is no sib-mating ($s = 0$), then this is equivalent to that for offspring, a result previously found when considering organisms with a dominant haploid phase (Patten and Haig 2009). However, under sib-mating the interests of mothers and offspring diverge, with mothers favoring a greater female bias than their offspring (Fig. 5A). This parallels a previous effect found in relation to sex allocation, whereby offspring typically favor less extreme sex ratio deviations than their parents (Trivers 1974; Werren and Hatcher 2000; Pen 2006), on account of parents being favored to maximize the success of the entire brood whereas each individual values itself more than its siblings (although see Pen (2006) for situations where this pattern may be reversed). For arrhenotoky and PGE, $F = [3-(1-s)^2]/[(1-s)(2-s)]$. Thus, for arrhenotoky, the situation is similar to diploidy, with mothers and offspring in agreement under random mating, but with mothers favouring a greater female bias when there is sib-mating (Fig. 5C). For PGE, however, when there is no sib-mating then offspring favor more female bias than their mothers, as females are twice as valuable as males from the perspective of the offspring, while sons and daughters are equally valuable from their mothers' perspective. However, this situation reverses as sib-mating increases, with mothers once again favoring more female-biased trait values than their offsprings (Fig. 5B).

Considering instead a sexually antagonistic allele that acts through fathers, we find that for diploidy the potential for feminization is the same as for mothers, $F = (1+s)/(1-s)$, and with both parents favoring a more female-biased trait value than offspring. For arrhenotoky, however, fathers favor a far more feminized trait value than either offspring or mothers, $F = [3+(1-s)^2]/[(1-s)s]$, as they only contribute genetically to their daughters in the brood. This is similar to how, under outbreeding, arrhenotokous fathers (and X chromosomes in males) favor exclusively female broods (Hamilton 1967), or investment solely into daughters (Rice et al. 2008; Friberg and Rice 2014; see also Miller et al. 2006). Nonetheless, with increased sib-mating they are increasingly related to their mates' sons, and thus place value on their fitness too, but with further sib-mating this is counteracted by the effects of increased local mate competition, once again favoring feminization (Fig. 5C). PGE yields a qualitatively similar outcome; however, as a male's paternal-origin genome is passed to neither sons nor daughters directly, then fathers are not as highly related to their daughters as compared with arrhenotoky, and they therefore, favor slightly less feminization (Fig. 5B), with the potential for feminization $F = [6-(1-s)(2-s)(1+s)]/[(1-s)(3-s)s]$.

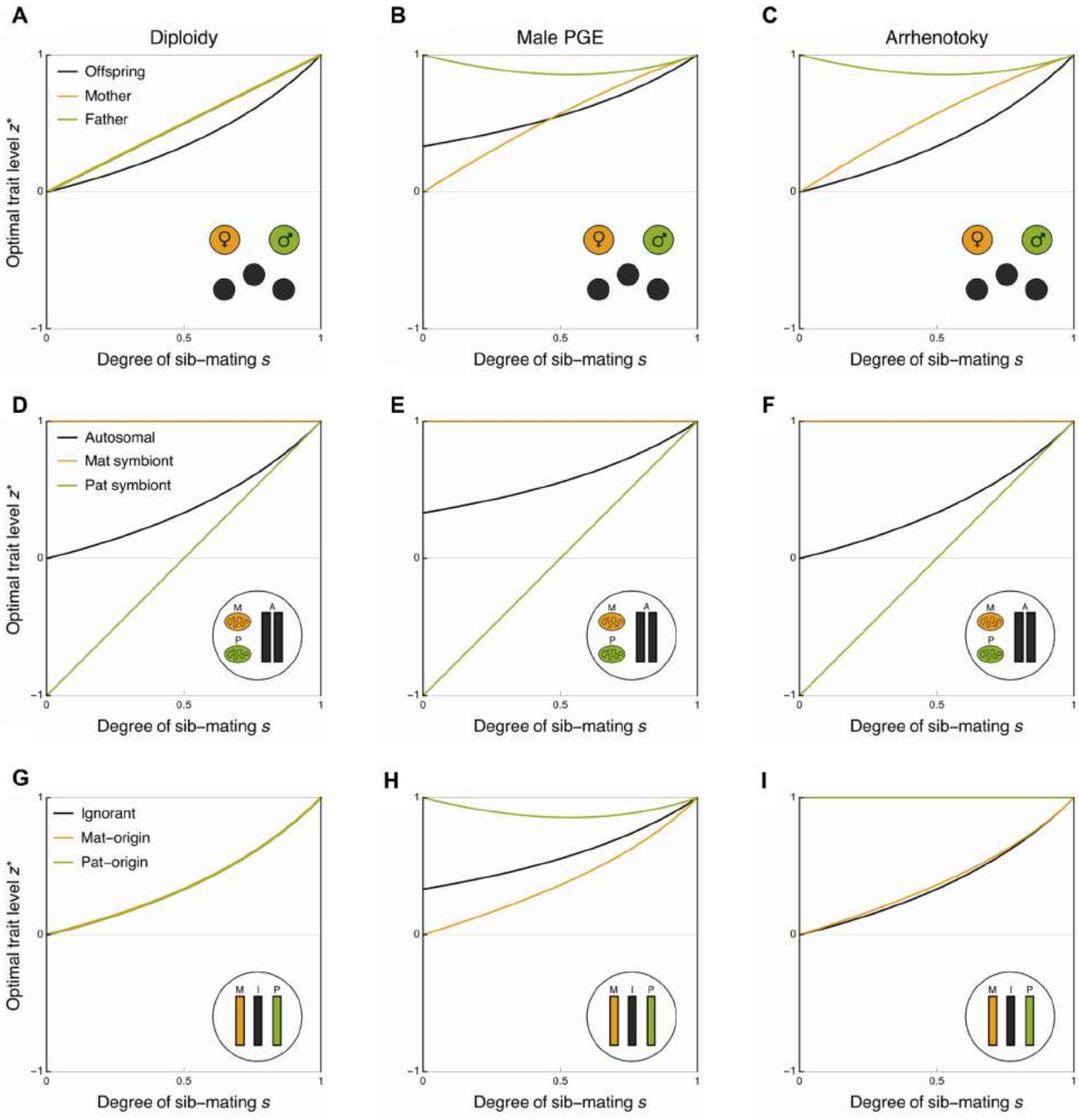


Figure 5. **Conflicts within and between individuals over sexually antagonistic traits, across different genetic systems. The optimal level of a sexually antagonistic trait *z* under diploidy, germline PGE, and arrhenotoky when control of that trait is assigned to: offspring, mothers, and fathers (A-C); autosomal genes, matrilineal cytoplasmic genes, and patrilineal cytoplasmic genes (D-F); ignorant genes, maternal-origin genes, and paternal-origin genes (G-I). In these examples, fitness is a Gaussian distributed trait with an optimum of 1 for females and −1 for males, with equal variance. Full details of methodology can be seen in §§SM 3.1–3.8.**

Full results for nonadditive scenarios can be found in SM §2.4.1.

ENDOSYMBIONTS, MITOCHONDRIA, AND GERMLINE RESTRICTED CHROMOSOMES

Thus far, we have largely treated the genome as though it is a unified entity. However, even though different genes may reside within the same body, they may nevertheless have distinct inclusive-fitness interests (Hamilton 1967; Burt and Trivers 2006; Gardner and Úbeda 2017), and thus, come into conflict over the trade-offs imposed by sexual antagonism. This is particularly relevant for haplodiploids as many contain endosymbionts which have different transmission modes to autosomal genes (Buchner 1965; Normark 2004a; Ross et al. 2012; Perlmutter and

Bordenstein 2020), and thus, may place different valuations upon males and females (Hurst 1991; Frank and Hurst 1996). Similarly, particular species also contain further unusual genomic features, such as the matrilineally inherited germline restricted E chromosomes found in gall midges (Harris et al. 2003; Normark 2004a; Hodson and Ross 2021).

For those endosymbionts and chromosomes that are strictly matrilineally inherited, they will place no direct value upon the fitness of males, bringing them into conflict with the rest of the genome (Wade 2014; Hurst and Frost 2015). These elements may also, therefore, provide a rich source of evidence for the "Mother's Curse" hypothesis, that is, that mitochondria accumulate mutations which are deleterious for males (Gemmell et al. 2004). Under full outbreeding, this conflict is at its most intense, but with increasing amounts of sib-mating the autosomes become increasingly female-biased too, aligning the interests of these two sets of genes, and thus reducing the extent of the conflict. This also applies to patrilineally inherited symbionts, which although much rarer than matrilineally inherited counterparts have been documented in a variety of species including aphids (Moran and Dunbar 2006), mosquitos (Damiani et al. 2008), leafhoppers (Watanabe et al. 2014), termites (Korb and Aanen 2003), and tsetse flies (De Vooght et al. 2015). With full outbreeding, paternally inherited genes place no value on females, but as inbreeding increases then they place an increasing value on the fitness of females, mollifying the conflict between them, autosomal, and maternal-inherited genes, as shown in Figure 5D-F.

PARENT-OF-ORIGIN SPECIFIC GENE EXPRESSION

Finally, a further intragenomic conflict that may emerge over sexual antagonism is between maternal- and paternal-origin genes (Haig 2002). The asymmetric transmission genetics that defines haplodiploidy may subsequently generate differences between maternal- and paternal-origin genes in how they value males and females, and also their relatedness to the males and females with whom they interact (Haig 1992; Queller and Strassmann 2002; Queller 2003; Wild and West 2009; Rautiala and Gardner 2016; Marshall et al. 2020).

In the simplest case, with full outbreeding, we find that if a gene is of maternal-origin it places equal value upon males and females, under diploidy, arrhenotoky, and PGE. Conversely, if it is of paternal-origin then it places equal value upon males and females under diploidy, but places no value upon males under the haplodiploid systems, as it is never transmitted by males under PGE, and is absent from males under arrhenotoky. Focusing on PGE, we can explore how, depending on which gene copy controls the trait, the potential for feminization may change. This is particularly relevant as the extent of expression in males from the maternal- and paternal-origin copies may vary across loci, tissues, and species (Burt and Trivers 2006; Gardner and Ross 2014; de la Filia et al. 2021). Allowing for a proportion y of a locus's expression in a male to come from the paternal-origin copy, and a proportion $1-y$ to come from the maternal-origin copy, we find that the potential for feminisation is $F = 1/(1-y)$. Thus, when maternal-origin genes control the trait in males ($y = 0$), then $F = 1$, equivalent to the arrhenotokous case, whie when expression is exclusively from the paternal-origin copy ($y = 1$), then $F = \infty$, that is, female-beneficial alleles will always invade, regardless of the cost they impose upon males, analogous to how paternal-origin genes may favor male suicide when there is competition between male and female siblings (Ross et al. 2011b).

As the rate of sib-mating increases, the intragenomic conflicts become more complex. We now explore the effects of parent-of-origin specific gene expression in both males and females. Allow for a proportion y of a locus's expression in males to come from their paternal-origin copy and a proportion $1-y$ from their maternal-origin copy, and allowing a proportion x of that locus's gene expression in females to come from their maternal-origin copy, and proportion $1-x$ from their paternal-origin copy. Then, we find the degree of feminisation under PGE becomes:

$$F = \frac{4 - s\,(2 - s - 2\,(1-s)\,x)}{(1-s)\,(4 - s - 2\,(2-s)\,y)}.$$

With the results for arrhenotoky generated by setting $y = 0$. We can see that assigning full control to maternal-origin copy in both sexes ($x = 1, y = 0$), conditions simplify to $F = [(2+s)(2-s)]/[(4-s)(1-s)]$, which is a monotonically increasing function of s, that is, the degree of feminization always increases as the rate of sib-mating increases. In contrast, if we assign full control to the paternal-origin genes ($x = 0, y = 1$), then $F = [3 + (1-s)^2]/[(1-s)s]$. In the absence of sib-mating ($s = 0$), then the paternal-origin copy is unrelated to the other gene copy in a male, and thus, places no value on male fitness. As the rate of sib-mating increases then the value that a paternal-origin gene places on males increases too, as that gene copy is related to the other gene copy it resides in a male with. However, with further increases in the rate of sib-mating, this is countered both by the increasing competition between related males, and also the indirect effects from related females.

Previously, intragenomic conflict between maternal- and paternal-origin genes has been suggested to drive the evolution of genomic imprinting at such loci, that is, the expression of one parental copy and the silencing of the other parental copy. This results from an escalating conflict over joint expression levels, which ultimately results in the gene copy that favors lower expression levels becoming silenced, while the one that favors higher expression levels is expressed at its optimum level, a process termed the "loudest voice prevails" principle (Haig 1996). If we apply the logic of this principle to conflict over sexually antagonistic traits then, under PGE, we may expect paternal-origin

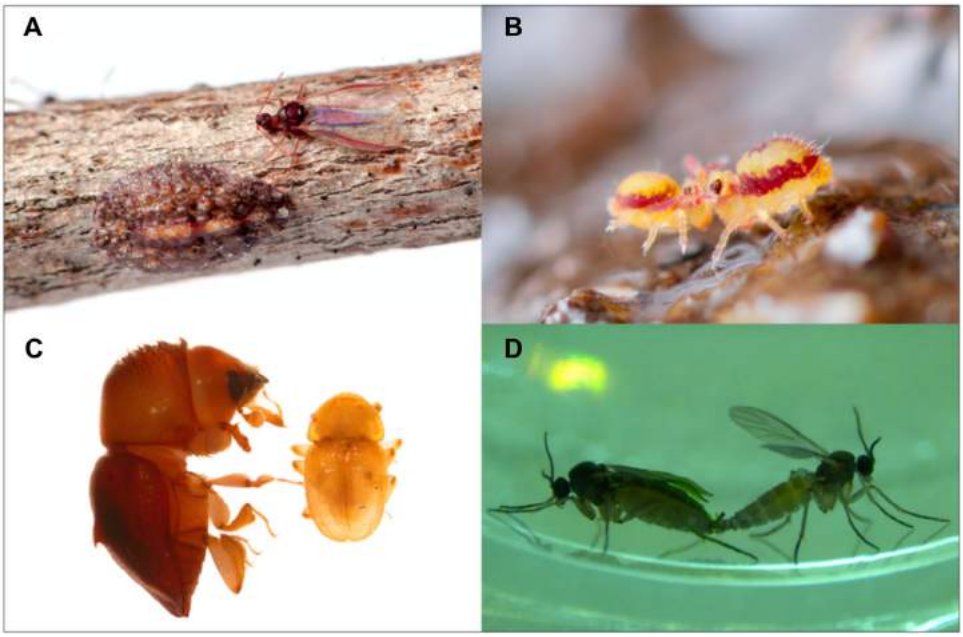


Figure 6. Examples of sexual dimorphism in haplodiploid species. (A) Soft scale insects (*Pulvinaria acericola*), female on the left, male on the right (credit: Matt Bertone). (B) Globular springtail (*Sminthurides malmgreni*), male on the left, female on the right (credit: Andy Murray, chaosofdelight.org). (C) Ambrosia beetle (*Diuncus sp.*) female on the left, male on the right (credit: Jiri Hulcr). (D) Fungus gnats (*Bradysia coprophila*), female left, male right (credit: Robert Baird).

genes to be expressed for female-beneficial trait promoters and male-beneficial trait inhibitors, while we would expect maternal-origin genes to be expressed for male-beneficial trait promoters, and female-beneficial trait inhibitors. Note that this is distinct from other theories about how sexual antagonism may give rise to genomic imprinting (Iwasa and Pomiankowski 1999, 2001; Day and Bonduriansky 2004), with predictions about the expected direction of imprint likely to differ also.

Discussion

Haplodiploid species account for a large minority of all animal species (Normark 2003, 2006; Bachtrog et al. 2014; de la Filia et al. 2015), with many striking examples of sexual dimorphism (see Fig. 1, 6). Our analyses here have shown how some of the unusual genetic and ecological asymmetries that define these groups are expected to modulate the outcome of sexual antagonism. We find that: (1) PGE promotes female-beneficial alleles more than arrhenotoky (recovering the result given recently by Klein et al. 2021); (2) the extent of this female bias is determined by the amount of paternal leakage and degree of silencing of the paternal genome; (3) the chronic sib-mating associated with many haplodiploid groups promotes feminization, with different modes of inbreeding—including limited male-dispersal, oedipal-mating, and the pseudo-hermaphroditism of *I. purchasi*—having qualitatively similar, but quantitatively different effects; (4) resource competition between related females relatively inhibits female-beneficial alleles; (5) inbreeding and asymmetric transmission may foment conflicts of interest between different parties over sexually antagonistic traits, including parents and offspring, endosymbionts and their hosts, and maternal- and paternal-origin genes; and (6) such intragenomic conflict provides a novel explanation for the evolution of genomic imprinting.

While our analysis indicates that these groups may provide a particularly rich set of comparative tests for how ecology and genetics modulate sexual antagonism, relatively little work has been carried out to investigate this. One of the reasons for this paucity of research attention is that the within-genome comparisons often used to study sexual antagonism have been considered impossible for the many haplodiploid species that lack sex chromosomes. However, this overlooks the exceptions that provide excellent opportunities for testing theory. For instance, sciarid flies not only have male PGE, but also an XO sex chromosome system (Metz 1938; Rieffel and Crouse 1966), allowing a within-organism comparison of these inheritance systems in relation to sexual antagonism. This is also true of some other groups with germline PGE such as gall midges and globular springtails (Gallun and Hatchett 1969; White 1977; Dallai 2000; Anderson et al. 2020). In these groups, we may expect female-beneficial variants to be enriched on the autosomes, while male-beneficial variants would be expected to be overrepresented on the sex chromosomes, regardless of assumptions about dominance,

making this a more straightforward prediction than between autosomes and sex chromosomes in conventional eumendelian systems (Rice 1984; Patten 2019). This is similar to how the unusual life cycle and X-chromosome transmission of pea aphids has provided an exceptional test of evolutionary theory in this area by having predictions qualitatively unaffected by dominance (Jaquiéry et al. 2013; Jaquiéry et al. 2021). In addition to X/autosome comparisons, some of these groups contain further genomic elements, such as germline-restricted chromosomes, that are maternally inherited in gall midges and show likely paternally biased inheritance in sciarid flies (Hodson and Ross 2021; Hodson et al. 2021), enabling further within-genome comparisons.

Similarly, while it has been suggested that the X chromosome should be relatively enriched for sexually antagonistic polymorphisms in eumendelian systems as compared to the autosomes (Rice 1984), again this depends on assumptions about dominance (Fry 2010; Ruzicka and Connallon 2020). We find here that the same is true of comparisons between PGE and X chromosomes or arrhenotoky, with arrhenotokous organisms ones having a higher potential for polymorphism under parallel dominance, but a smaller space for polymorphisms under dominance reversals (see SM §2.5). Additionally, such sexually antagonistic polymorphisms may be easier to detect in some haplodiploid species as compared to eumendelian ones, because the asymmetric transmission genetics means that allele frequency differences that build up between the sexes in one generation, will carry over to the next (Crow and Kimura 1970; Ruzicka and Connallon 2020).

Additionally, we find that the chronic inbreeding exhibited by many haplodiploids typically promotes feminization. This meshes with the increasing interest in the role of demography and ecology in modulating sexual antagonism (Albert and Otto 2005; Arnqvist 2011; Harts et al. 2014; Tazzyman and Abbott 2015; Connallon et al. 2019; de Vries and Caswell 2019; Hitchcock and Gardner 2020). In particular, Flintham et al. (2021) have recently shown how, in viscous populations, sex-biased dispersal may skew sexual antagonism under diploidy and X-linkage toward the sex that competes less intensely with relatives. Here, we recover that same pattern, but also find that other mating schemes that characterize haplodiploid groups can involve an additional feminizing effect, as females may confer fitness benefits upon their mates. Alongside comparisons between populations and species, one method of testing such predictions would be through the use of experimental evolution. For example, Rodrigues et al. (2021) evolved populations of the spider mite *Tetranychus urticae* under various dispersal regimes in order to investigate the evolution of sex allocation; those demographies predicted to lead to greater female bias in the sex ratio would also be expected to promote female bias in relation to sexual antagonism. Thus, under these conditions, we may expect to see either increased fixation of female-beneficial sexually antagonistic alleles and/or phenotypes moving toward the female optimum. Reinvestigation of these evolved lines or new experiments with similar design would enable testing of predictions emerging from our analysis.

Furthermore, we have shown how population structure and transmission asymmetries may foment conflicts between different genetic parties over sexually antagonistic traits. In particular, we identify potential for conflict between parents and offspring. While there has been similar work considering the differing interests between parents and offspring with regards to sex allocation (Trivers 1974; Werren and Hatcher 2000; Pen 2006), sexual antagonism provides a further arena for such conflicts of interest. While parent-offspring conflict emerges across all of our genetic systems under sib-mating, species with PGE provide a particularly interesting set of systems within which to investigate this phenomenon as, even under full outbreeding, mothers, fathers, and offspring all favor different trade-offs. Thus, depending on who controls the trait, we may expect different patterns of masculinization versus feminization. Comparisons between sperm-derived versus egg-derived products, and between those to genes expressed after the maternal-to-zygotic transition, may help reveal such conflicts over development. A further, particularly interesting case to investigate the logic of such conflicts is with the bacteriome of the armored scale insects. These are pentaploid tissues containing two complete copies of the mother's genome and a copy of the paternal-origin genome (Normark 2004b). Thus, while not identical to the parents interests, the bacteriome nonetheless might be expected to have more similar genetic interests to the mother than the offspring it resides within, and thus, the interface between them provides a within-individual arena for this parent-offspring conflict.

We have focused here on cases where there are only two classes of individual: males and females. However, many of the better known haplodiploid species—most notably the eusocial Hymentoptera—exhibit not just sex structure, but also caste structure. For instance, in the eusocial bees, wasps, and ants, in addition to reproductive females (queens) and reproductive males (drones), there is also an additional female neuter class (workers) who are morphologically, physiologically, and behaviorally distinct from the queen. While the addition of caste structure on its own is not expected to modulate sexual antagonism per se, that is, trade-offs between queens and reproductive males, if the trade-off occurs through female workers and reproductive males then results would be expected to diverge, as phenotypic effects that manifest in females would only have indirect effects through their effects on the reproductive females. Moreover, with more than two castes there is the possibility for more complex trade-offs operating across multiple classes, such as between workers and queens, workers and males, and three-way trade-offs; such

trade-offs have previously been referred to in terms of "intralocus caste antagonism" (Holman 2014; Pennell et al. 2018). A similar complexity occurs when males exhibit polyphenisms, for instance, in fig wasps between winged and nonwinged male forms (Hamilton 1979; Cook et al. 1997). Such male dimorphism can be extreme, not only concerning the presence/absence of wings, but also with respect to other aspects of morphology and behavior. If a sexually antagonistic allele affects these morphs differently, then outcomes will be more complex than those emerging from our analysis, depending on the relative fraction of male dispersers. Similarly to caste structure, this may lead to trade-offs among these male morphs, previously termed "intralocus tactical evolution" (Morris et al. 2013).

Our predictions have been derived under the assumption of nonoverlapping generations, yet age-structure may also have an important modulating effect on sexual antagonism (de Vries and Caswell 2019; Hitchcock and Gardner 2020). This may be important for two reasons. First, sex-specific age-structure may disturb the reproductive values of males and females away from the ratios given here (Grafen 2014; Hitchcock and Gardner 2020). This may be because there are sex-differences in mortality and fecundity, such as in the citrus mealybug (*P. citri*) where males live up to only 3 days post eclosion while females can live several weeks (Nelson-Rees 1960; Ross et al. 2011a), or because of other factors which can generate more cryptic age structure such as partial bivoltinism (Seger 1983; Grafen 1986), sperm storage, or worker reproduction (Benford 1978; Charnov 1978; Alpedrinha et al. 2013). Second, population viscosity may generate competition between parents and offspring (Irwin and Taylor 2001; Ronce and Promislow 2010). Coupled with other aspects of sex-biased demography, such as sex-biased dispersal (Johnstone and Cant 2008, 2010), then this may reduce the magnitude of costs or benefits to one sex more than the other, and thus, bias the outcome of sexual antagonism toward one sex. An example relevant to this is the aforementioned date-stone beetle where a single female may spawn up to five generations within a single drupe over the spring and summer (Spennemann 2019), thus generating potentially strong inter- and intragenerational kin competition.

Finally, we have considered mating to be the only social interaction between males and females. Yet invasion conditions for sexually antagonistic alleles are liable to be modulated by more extensive and complex intersexual interactions. For instance, intrabrood competition may result in male-beneficial alleles decreasing the fitness of females both through the direct effect of those alleles being expressed by females, but also through those females being outcompeted by their brothers (and vice versa, for female-beneficial alleles). The extent of such competition will vary with ecological context. For instance, bark beetles are understood to experience intense sib-competition, while phloem feeders are less likely to do so (Normark 2004a, 2006). Intense intrabrood competition is also an ecology well-suited to the evolution of cytoplasmic male killing (Hurst 1991; Hamilton 1993; Normark 2004a). Moreover, we have assumed that there is an asymmetry in which female-beneficial variants improve the likelihood of a mating pair winning a breeding opportunity (as it is competitiveness of females that determines this), while male-beneficial variants have no such effect. While this does adequately capture the ecology of many haplodiploid species, there are scenarios in which this assumption need not hold. For instance, males may have beneficial fitness effects upon their mates if there is paternal care, as in the case of the mud daubers (Brockmann 1980; Bragato Bergamaschi et al. 2015) and the solitary apid bee, *Ceratina nigrolabiata* (Mikát et al. 2019), or if sperm is a limiting factor on the rate of reproduction. Alternatively, males may also have deleterious fitness effects if they exhibit harming traits such as the traumatic insemination observed in some groups of pinworms (Adamson 1989).

In conclusion, we have explored how genetic and ecological asymmetries that characterize haplodiploid groups are expected to modulate sexual antagonism, and how these may, in turn, foment conflicts both between and within individuals over such traits. Exploring the consequences of these unusual genetic systems and life cycles has previously offered rich insights into sex allocation (Charnov 1982; West 2009), and thus, leveraging the natural diversity within these groups may also deepen our understanding of sexual antagonism and the evolution of sexual dimorphism. Gene expression studies increasingly look for sex-biased gene expression in such nonmodel and noneumendelian species, and our predictions will facilitate interpretation of these data, as well as identifying where future research effort may be most fruitfully focused. Finally, many of the species that reproduce through arrhenotoky or PGE are pests and parasites of humans, livestock, and crops, for example, the coffee borer beetle, hessian fly, head lice, and the citrus mealybug. Improved understanding of the evolutionary consequences of these unusual lifecycles and genetics, therefore, also has practical relevance in guiding our use of chemical, biological, and genetic controls.

AUTHOR CONTRIBUTIONS

T.J.H., A.G., and L.R. jointly designed the study, T.J.H. performed the analysis, and T.J.H., A.G., and L.R. wrote the manuscript.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Supplementary material

Supplementary Material for:

Sexual antagonism in haplodiploids

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Contents

Outline **2**

1 General Background **3**

1.1 Life cycles . . . 3

1.2 Genetic systems . . . 4

2 Invasion analysis **6**

2.1 Notation . . . 6

2.2 Methodology . . . 6

2.3 Fitness schemes . . . 10

2.4 Invasion conditions . . . 12

2.5 Potential for polymorphism . . . 19

3 Kin selection model of a sexually antagonistic trait **26**

3.1 General conditions . . . 26

3.2 Fitness functions . . . 27

3.3 Genotype to phenotype mapping . . . 28

3.4 Marginal fitness effects . . . 29

3.5 Consanguinities . . . 31

3.6 Reproductive value . . . 35

3.7 Condition for increase . . . 36

3.8 Trait Optima . . . 37

4 References **38**

Outline

This Supplementary Material contains background on the models, and additional results, for those conditions presented in the main text of *Sexual antagonism in haplodiploids*. The document is organised as follows. **Section 1** summarises the general background and associated notation for the life-cycles and genetic systems analysed. **Section 2** provides details of the invasion analysis of a sexually antagonistic allele across these different life-cycles and genetic systems. **Section 3** is a kin-selection analysis of a sexually antagonistic trait using the methodology of Taylor and Frank (1996). Together these approaches provide complementary insights. The invasion analysis allows us to consider arbitrary strength of selection, and the effects of specific genetic parameters, such as dominance. The latter, by honing in on additivity and weak selection, both aids in interpretation of the invasion conditions and allows us to relax some of the assumptions about the ecology.

1 General Background

1.1 Life cycles

We consider three core life-cycle variants that apply to most of our genetic systems: a fixed sib-mating scenario, a viscous population with mating occuring prior to female dispersal (dispersal→mating→dispersal, DMD) and a viscous population where mating occurs after female dispersal (dispersal→dispersal→mating, DDM). These three scenarios are described below and illustrated in Figure S1. In all three cases we consider an infinite population, split into a large number of patches (i.e. an infinite island model (Wright, 1931)). We assume that on each patch there is one, singly mated, female breeder, who lays a large brood of κ offspring, of which of which a proportion $1-z$ are female, and z are male. We also consider two further life-cycles which are specific to particular genetic systems: oedipal mating, and pseudo-hermaphroditism in *Icerya purchasi*.

Sib-mating scenario. In the fixed sib-mating scenario, with probability s females mate with brothers, and with probability $1-s$ mate with males from the population at large. This captures scenarios where there may be a temporal separation between sib-mating and outbreeding, analogous to both "prior selfing" and "delayed selfing" models in hermaphroditic plants, where there is no direct competition between pollen from self and from other plants (Lloyd and Schoen, 1992; Lloyd, 1992; Jordan and Connallon, 2014). After mating, females then either disperse with probability d_f, or remain on their natal patch with probability $1-d_f$. They then compete for the breeding spot on the patch, unsuccessful females then die, and the life-cycle begins again.

Viscous population, mating before female dispersal. In the mating pre-dispersal scenario (DMD), males first either disperse with probability d_m or remain on their natal patch with probability $1-d_m$, females then mate with the males on their patch. Thus this is analogous to "mass-action" models of selfing in hermaphroditic plants where an individual's own pollen does directly compete with pollen from the wider population (Holsinger, 1991; Jordan and Connallon, 2014). After mating, females either disperse with probability d_f, or remain on their natal patch with probability $1-d_f$. Once again the females compete for the breeding spot on the patch, unsuccessful females then die, and the life-cycle begins anew.

Viscous population, mating post female dispersal. In the mating post-dispersal scenario (DDM), both males and females initially disperse from the patch, with probabilities d_m and d_f respectively, or remain on the natal patch with probabilities $1-d_m$ and $1-d_f$ respectively. Females then mate with males on their patch, before competing for breeding spots. Unsuccessful females then die, and the life-cycle begins once more. This life-cycle is very similar to that previously analysed by Flintham and colleagues (Flintham, Savolainen, and Mullon, 2021).

Oedipal mating. For arrhenotokous organisms, females need not mate in order to produce offspring, and this opens up further variants of the life-cycles above. One particular case is oedipal mating, which has been documented in a range of arrhenotokous organisms (referenced in main text). In these cases a virgin female may initially lay a brood of exclusively male offspring, mating with one of her sons, before laying a bisexual brood. To incorporate this, we allow a proportion $\mathcal{O}$ of females to oedipally mate, and a proportion $1-\mathcal{O}$ to

mate with a random male in the population. Two variants of this life-cycle can be envisaged depending on whether it assumed that brothers compete to mate with their mother (we denote this type I oedipal mating), or alternatively the mother simply mates with the first male who ecloses (type II oedipal mating).

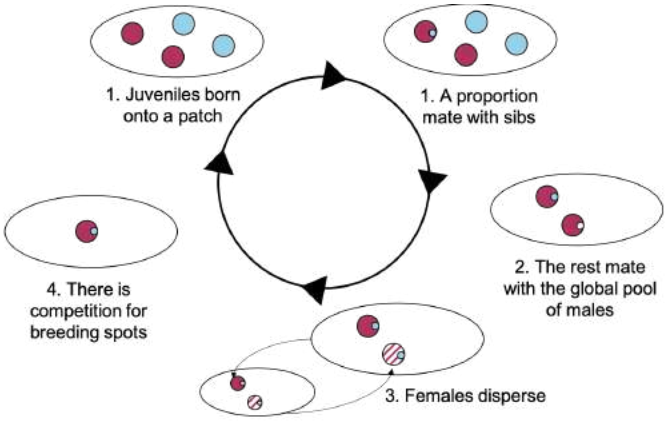
Pseudo-hermaphroditism in Icerya. *Icerya purchasi* is among the few "hermaphroditic" species of insect that are known, with diploid females and haploid males. However, in addition to these two genomes, in females there is a third genome belonging to an invasive lineage of spermatogenic tissue(Mongue et al., 2021). This tissue can both fertilise the eggs of the female, but is also directly transmitted to female offspring. Females therefore can either "self", mating with this invasive male tissue, or "outbreed", mating with a true male. We allow for this by setting a proportion φ of females to self, and proportion $1-\varphi$ mate with males as normal.

1.2 Genetic systems

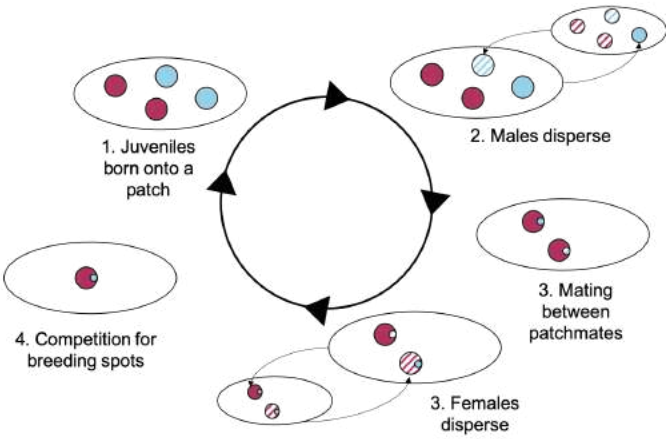
In the main text we described four main types of genetic system: endosymbionts/cytoplasmic elements, diploidy, male paternal genome elimination (PGE), arrhenotoky, and pseudo-hermaphroditism in Icerya. Here we consider three further genetic systems: sexual haploidy, whereby both males and females are haploid undergo sexual reproduction to produce new haploid offspring, female maternal genome elimination (MGE), where females are initially diploid but eliminate their maternal-origin genes when producing eggs, and paterothylotoky, where females are haploid producing either empty eggs which become fertilised to become females or haploid eggs which become fertilised to become males.

Of these wider sets of systems, only haploidy is well described and observed in nature, found in groups of green algae, brown algae, and bryophytes (Bachtrog et al., 2014; Coelho et al., 2018). Whilst examples of paterothylotoky have not been observed, these results are equivalent to those for the non-pseudoautosomal region of Z chromosomes in ZW chromosome systems. Finally, again whilst there are no known species that exhibit female MGE, there are various documented examples of androgenesis, whereby genes contributed by females are eliminated shortly after fertilisation (Burt and Trivers, 2006; Schwander and Oldroyd, 2016), thus a sex-specific version of this is not inconceivable. Moreover, these systems provide a comparison to investigate and distinguish between the effects of sex-specific aspects of ecology and genetics on the invasion conditions.

Fixed sib-mating



Viscous population: Mating pre-female dispersal



Viscous population: Mating post-female dispersal

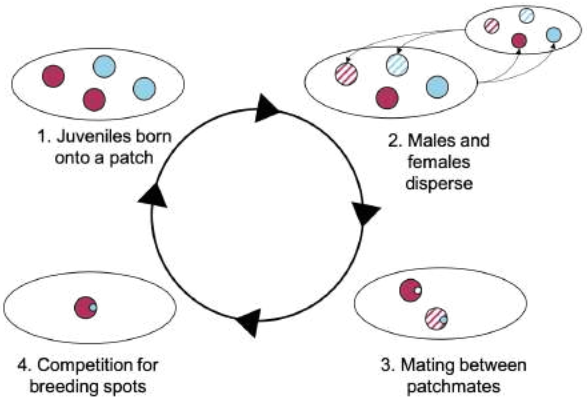


Figure S1: The structure of the three main life-cycles that we consider

2 Invasion analysis

For the invasion analysis we first write out recursion equations describing the frequency of different mating pairs. We then make weak selection approximations of these recursion equations, and use these to analytically find the invasion conditions for a sexually antagonistic allele. Finally, we use these recursion equations to find the invasion boundaries for stronger selection regimes, and thus the parameter space for stable polymorphisms.

2.1 Notation

We consider the conditions when a mutant allele will be able to invade a population which is monomorphic for a resident allele. For a particular genetic system, we write out the genotype of a female as a_i where $a_i \in A$, with A being the set of all possible female genotypes in that system, and the genotype of a male as b_i, where $b_i \in B$, with B being the set of all possible male genotypes, and the genotype of a mating pair c_i, with $c_i \in C$, where C is the set of all possible mating pairs.

We write out the frequency of a particular mating pair genotype at time t as f_{c_i}, and the frequency at time $t+1$ to be f'_{c_i}. The number of females of genotype a_j produced by mating pair c_i is given by x_{a_j,c_i}, and the number of males of genotype k produced by genotype c_i is y_{b_k,c_i}. Let w_{a_j,c_i} be the fitness of an individual female who has genotype a_j and who comes from mating pair of genotype c_i, and let v_{b_k,c_i} be the fitness of an individual male with genotype b_k and who comes from a mating pair with genotype c_i. We write out the mean fitness of the females produced by a mating pair of genotype c_i to be $\overline{w}_{c_i} = (1/x_{c_i})\sum_j x_{a_j,c_i} w_{a_j,c_i}$, and the mean fitness of the males produced by a mating pair of genotype c_i to be $\overline{v}_{c_i} = (1/y_{c_i})\sum_k y_{b_k,c_i} v_{b_k,c_i}$, where the number of females produced by a genotype c_i is $x_{c_i} = \sum_j x_{a_j,c_i}$ and the number of males $y_{c_i} = \sum_k y_{b_k,c_i}$.

2.2 Methodology

2.2.1 Recursion equations

Fixed sib-mating scenario

$$\begin{aligned} f'_{\{a_j,b_k\}} = \sum_i f_{c_i}[(1-d_f)\left(\frac{x_{a_j,c_i} w_{a_j,c_i}}{\alpha_i}(s\frac{y_{b_k,c_i} v_{b_k,c_i}}{y_{c_i}\overline{v}_{c_i}} + (1-s)\sum_l f_{c_l}\frac{y_{b_k,c_l} v_{b_k,c_l}}{\overline{yv}}\right) \\ + d_f \sum_{\mathbb{i}} f_{c_{\mathbb{i}}}\frac{x_{a_j,c_i} w_{a_j,c_i}}{\alpha_{\mathbb{i}}}(s\frac{y_{b_k,c_i} v_{b_k,c_i}}{y_{c_i}\overline{v}_{c_i}} + (1-s)\sum_l f_{c_l}\frac{y_{b_k,c_l} v_{b_k,c_l}}{\overline{yv}})] \end{aligned} \tag{S2.2.1a}$$

Where α_i is the competitiveness of patch type c_i for females:

$$\alpha_i = (1-d_f)x_{c_i}\overline{w}_{c_i} + d_f\overline{xw} \tag{S2.2.1b}$$

With the mean female fitness given by:

$$\overline{w} = \sum_i f_{c_i}\overline{w}_{c_i} = \sum_i f_{c_i}\sum_j \frac{x_{a_j,c_i}}{x_{c_i}} w_{a_j,c_i} \tag{S2.2.1c}$$

And the mean male fitness given by:

$$\overline{v} = \sum_i f_{c_i}\overline{v}_{c_i} = \sum_i f_{c_i}\sum_k \frac{y_{b_k,c_i}}{y_{c_i}} v_{b_k,c_i} \tag{S2.2.1d}$$

Viscous population, mating pre-female dispersal

$$f'_{\{a_j,b_k\}} = \sum_i f_{c_i}[(1-d_f)\left(\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_i}((1-d_m)\frac{y_{b_k,c_i}v_{b_k,c_i}}{\beta_i} + d_m\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\beta_i})\right) + d_f\sum_{\mathbb{i}} f_{c_{\mathbb{i}}}\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_{\mathbb{i}}}((1-d_m)\frac{y_{b_k,c_i}v_{b_k,c_i}}{\beta_i} + d_m\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\beta_i})] \tag{S2.2.2a}$$

Where α_i is the competitiveness of patch type c_i for females:

$$\alpha_i = (1-d_f)x_{c_i}\overline{w}_{c_i} + d_f\overline{xw} \tag{S2.2.2b}$$

And the relative competitiveness of patch type c_i for males β_i is:

$$\beta_i = (1-d_m)y_{c_i}\overline{v}_{c_i} + d_m\overline{yv} \tag{S2.2.2c}$$

With the mean female fitness given by:

$$\overline{w} = \sum_i f_{c_i}\overline{w}_{c_i} = \sum_i f_{c_i}\sum_j \frac{x_{a_j,c_i}}{x_{c_i}}w_{a_j,c_i} \tag{S2.2.2d}$$

And the mean female fitness given by:

$$\overline{v} = \sum_i f_{c_i}\overline{v}_{c_i} = \sum_i f_{c_i}\sum_k \frac{y_{b_k,c_i}}{y_{c_i}}v_{b_k,c_i} \tag{S2.2.2e}$$

Viscous population, mating post-female dispersal

$$f'_{\{a_j,b_k\}} = \sum_i f_{c_i}[(1-d_f)\left(\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_i}((1-d_m)\frac{y_{b_k,c_i}v_{b_k,c_i}}{\beta_i} + d_m\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\beta_i})\right) + d_f\sum_{\mathbb{i}} f_{c_{\mathbb{i}}}\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_{\mathbb{i}}}((1-d_m)\frac{y_{b_k,c_{\mathbb{i}}}v_{b_k,c_{\mathbb{i}}}}{\beta_{\mathbb{i}}} + d_m\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\beta_{\mathbb{i}}})] \tag{S2.2.3a}$$

Where α_i is the competitiveness of patch type c_i for females:

$$\alpha_i = (1-d_f)x_{c_i}\overline{w}_{c_i} + d_f\overline{xw} \tag{S2.2.3b}$$

And the relative competitiveness of patch type c_i for males β_i is:

$$\beta_i = (1-d_m)y_{c_i}\overline{v}_{c_i} + d_m\overline{yv} \tag{S2.2.3c}$$

With the mean female fitness given by:

$$\overline{w} = \sum_i f_{c_i}\overline{w}_{c_i} = \sum_i f_{c_i}\sum_j \frac{x_{a_j,c_i}}{x_{c_i}}w_{a_j,c_i} \tag{S2.2.3d}$$

And the mean male fitness given by:

$$\overline{v} = \sum_i f_{c_i}\overline{v}_{c_i} = \sum_i f_{c_i}\sum_k \frac{y_{b_k,c_i}}{y_{c_i}}v_{b_k,c_i} \tag{S2.2.3e}$$

Oedipal mating - Type I We notate the fraction of oedipal mating to be $\mathcal{O}$. Also, as when a female mates she produces a brood of offspring herself, we write out the number of these males of genotype b_k to be y_{b_k,a_j}, and her total number of males to be y_{a_j}, with the mean fitness of those males being $\overline{v}_{a_j} = \sum_k y_{b_k,a_j}v_{b_k,a_j}$.

$$f'_{\{a_j,b_k\}} = \sum_i f_{c_i}[(1-d_f)\left(\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_i}(\mathcal{O}\frac{y_{b_k,a_j}v_{b_k,a_j}}{y_{a_j}\overline{v}_{a_j}} + (1-\mathcal{O})\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\overline{yv}}\right) + d_f\sum_{\mathbb{i}} f_{c_{\mathbb{i}}}\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_{\mathbb{i}}}(\mathcal{O}\frac{y_{b_k,a_j}v_{b_k,a_j}}{y_{a_j}\overline{v}_{a_j}} + (1-\mathcal{O})\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\overline{yv}})] \tag{S2.2.4a}$$

Where α_i is the competitiveness of patch type c_i for females:

$$\alpha_i = (1-d_f)x_{c_i}\overline{w}_{c_i} + d_f\overline{xw} \tag{S2.2.4b}$$

With the mean female fitness given by:

$$\overline{w} = \sum_i f_{c_i}\overline{w}_{c_i} = \sum_i f_{c_i}\sum_j \frac{x_{a_j,c_i}}{x_{c_i}}w_{a_j,c_i} \tag{S2.2.4c}$$

With the mean male fitness by:

$$\overline{v} = \sum_i f_{c_i}\overline{v}_{c_i} = \sum_i f_{c_i}\sum_k \frac{y_{b_k,c_i}}{y_{c_i}}v_{b_k,c_i} \tag{S2.2.4d}$$

Oedipal mating - Type II We notate the fraction of oedipal mating to be $\mathcal{O}$. Also, as when a female mates she produces a brood of offspring herself, we write out the number of these males of genotype b_k to be y_{b_k,a_j}, and her total number of males to be y_{a_j}.

$$f'_{\{a_j,b_k\}} = \sum_i f_{c_i}[(1-d_f)\left(\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_i}(\mathcal{O}\frac{y_{b_k,a_j}}{y_{a_j}} + (1-\mathcal{O})\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\overline{yv}}\right) + d_f\sum_{\mathbb{i}} f_{c_{\mathbb{i}}}\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_{\mathbb{i}}}(\mathcal{O}\frac{y_{b_k,a_j}}{y_{a_j}} + (1-\mathcal{O})\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\overline{yv}})] \tag{S2.2.5a}$$

Where α_i is the competitiveness of patch type c_i for females:

$$\alpha_i = (1-d_f)x_{c_i}\overline{w}_{c_i} + d_f\overline{xw} \tag{S2.2.5b}$$

With the mean female fitness given by:

$$\overline{w} = \sum_i f_{c_i}\overline{w}_{c_i} = \sum_i f_{c_i}\sum_j \frac{x_{a_j,c_i}}{x_{c_i}}w_{a_j,c_i} \tag{S2.2.5c}$$

With the mean male fitness given by:

$$\overline{v} = \sum_i f_{c_i}\overline{v}_{c_i} = \sum_i f_{c_i}\sum_k \frac{y_{b_k,c_i}}{y_{c_i}}v_{b_k,c_i} \tag{S2.2.5d}$$

Pseudo-hermaphroditism in Icerya We notate the fraction of females who are mated by the infectious haploid spermatogenic tissue to to be φ, and the proportion mated by 'true' males to be $1-\varphi$. Similar to in the oedipal mating scenario, when a female 'selfs' we imagine that she produces a brood of males of which she then mates with one, in this case we notate it $\mathbb{y}_{b_k,a_j}$, as is distinct from the genotypes of 'true' males she would produce.

$$f'_{\{a_j,b_k\}} = \sum_i f_{c_i}[(1-d_f)\left(\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_i}(\varphi\frac{\mathbb{y}_{b_k,a_j}}{\mathbb{y}_{a_j}} + (1-\varphi)\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\overline{yv}}\right) + d_f\sum_{\mathbb{i}} f_{c_{\mathbb{i}}}\frac{x_{a_j,c_i}w_{a_j,c_i}}{\alpha_{\mathbb{i}}}(\varphi\frac{\mathbb{y}_{b_k,a_j}}{\mathbb{y}_{a_j}} + (1-\varphi)\sum_l f_{c_l}\frac{y_{b_k,c_l}v_{b_k,c_l}}{\overline{yv}})] \tag{S2.2.6a}$$

Where α_i is the competitiveness of patch type c_i for females:

$$\alpha_i = (1-d_f)x_{c_i}\overline{w}_{c_i} + d_f\overline{xw} \tag{S2.2.6b}$$

With the mean female fitness given by:

$$\overline{w} = \sum_i f_{c_i}\overline{w}_{c_i} = \sum_i f_{c_i}\sum_j \frac{x_{a_j,c_i}}{x_{c_i}} w_{a_j,c_i} \tag{S2.2.6c}$$

And mean male fitness given by:

$$\overline{v} = \sum_i f_{c_i}\overline{v}_{c_i} = \sum_i f_{c_i}\sum_k \frac{y_{b_k,c_i}}{y_{c_i}} v_{b_k,c_i} \tag{S2.2.6d}$$

2.2.2 Jacobians

Using these recursion equations we can ask when the mutant allele will be able to invade from rarity. If we denote the mating pair purely made up the resident allele c_*, then we want to consider the equilibrium point $f_{c_*} = 1$. If this is unstable then the mutant allele mating pair genotypes will be able to invade. To determine the stability, we first calculate the Jacobian matrix $\mathbf{J}$, analysed when the mating pair genotypes containing the mutant allele are vanishingly rare in the population (Otto and Day, 2011). Each entry of the matrix is given by:

$$\mathbf{J}_{a,b} = \frac{\partial f'_{c_a}}{\partial f'_{c_b}}\Big|_{c_*=1} \tag{S2.2.7}$$

If the leading eigenvalue of this matrix is greater than one, $\lambda_{max} > 1$ then the mutant mating pairs will increased in frequency, and thus the mutant allele will be able to invade.

2.2.3 Weak selection approximations

For many of these scenarios it is not tractable to find full analytical solutions. Instead we approximate the conditions for invasion when the mutant allele has a vanishingly small fitness effect as compared to the resident allele. If this is the case our fitness effects are of order δ, and we can write the largest eigenvalue as:

$$\lambda_{max} \approx 1 + \delta\lambda \tag{S2.2.8}$$

We then substitute this back into our characteristic equation and perform a first order Taylor expansion, ignoring terms of order δ^2 and higher. We then solve for λ, and compute our condition for invasion. The invasion conditions for the sib-mating, DMD, and DDM life-cycles under different fitness scemes can be found in Tables S3-S8. The invasion conditions for the oedipdal mating and the pseudo-hermaphroditism of Icerya can be found in Table S9.

2.2.4 Numerical solutions

We also find numerical approximations for the invasion boundary under stronger selection regimes by first finding the largest eigenvalue for specific values, and then interpolating the boundary where $\lambda_{max} = 1$ by using Mathematica's ListContour function (Inc., n.d.). These results can be seen in Figures S6-S8.

2.3 Fitness schemes

Here, we analyse three separate fitness schemes 1) that used by Rice 1984, and 2) that used by Kidwell et al. (1977), which can be seen in Table S1. We also use a further fitness scheme 3) to investigate parent-of-origin expression patterns, this can be seen in Table S2. For the case of Iceryan hermaphroditism, we treat females as though they are genetically triploid, but that the phenotype is purely determined by the two genotypes in the diploid female, and as such, the third genome of the spermatogenic lineage has no impact upon the phenotype, i.e. $w_{000} = w_{001} = w_{00}$, $w_{010} = w_{011} = w_{01}$,$w_{100} = w_{101} = w_{10}$, and $w_{110} = w_{111} = w_{11}$.

	Rice: F+/M-	Rice: M+/F-	Kidwell
w_0	1	1	$1-u_f$
w_1	$1+S$	$1-T$	1
w_{00}	1	1	$1-u_f$
w_{01}	$1+h_fS$	$1-h_fT$	$1-h_fu_f$
w_{10}	$1+h_fS$	$1-h_fT$	$1-h_fu_f$
w_{11}	$1+S$	$1-T$	1
v_0	1	1	1
v_1	$1-T$	$1+S$	$1-u_m$
v_{00}	1	1	1
v_{01}	$1-h_mT$	$1+h_mS$	$1-h_mu_m$
v_{10}	$1-h_mT$	$1+h_mS$	$1-h_mu_m$
v_{11}	$1-T$	$1+S$	$1-u_m$

Table S1: Fitness scheme used for the invasion analysis of a sexually antagonistic allele. w_i is the fitness of a female when an individual of genotype i has control over the sexually antagonistic trait. Thus, when the trait is under offspring control i represents her genotype, when the trait is under maternal control then i represents her mothers genotype, and when under paternal control i represents her fathers genotype. Similarly v_i represents the fitness of a male where the controlling genotype is i. In the two Rice scenarios, S represents the benefit of the allele when homozygous, T the cost, and h_f and h_m are the dominance coefficients for the allelic effect in males and females respectively (Rice, 1984; Patten, 2019). In the Kidwell scenario (Kidwell et al., 1977; Flintham, Savolainen, and Mullon, 2021), the fittest genotype has fitness 1, with the cost u_f to females and u_m to males. Again h_f and h_m are the dominance coefficients for the effect in males and females. For the case of Iceryan hermaphroditism, we assume that the notionally triploid females (diploid females with the additional haploid spermatogenic tissue) are equivalent to 'true' diploid females.

	Offspring control		Maternal control		Paternal control	
	F+/M-	M+/F-	F+/M-	M+/F-	F+/M-	M+/F-
w_0	1	1	1	1	1	1
w_1	$1+S$	$1-T$	$1+S$	$1-T$	$1+S$	$1-T$
w_{00}	1	1	1	1	1	1
w_{01}	$1+(1-x)S$	$1-(1-x)T$	$1+(1-x)S$	$1-(1-x)T$	$1+yS$	$1-yT$
w_{10}	$1+xS$	$1-Tx$	$1+xS$	$1-xT$	$1+(1-y)S$	$1-(1-y)T$
w_{11}	$1+S$	$1-T$	$1+S$	$1-T$	$1+S$	$1-T$
v_0	1	1	1	1	1	1
v_1	$1-T$	$1+S$	$1-T$	$1+S$	$1-T$	$1+S$
v_{00}	1	1	1	1	1	1
v_{01}	$1-Ty$	$1+yS$	$1-(1-x)T$	$1+(1-x)S$	$1-yT$	$1+yS$
v_{10}	$1-(1-y)T$	$1+(1-y)S$	$1-xT$	$1+xS$	$1-(1-y)T$	$1+(1-y)S$
v_{11}	$1-T$	$1+S$	$1-T$	$1+S$	$1-T$	$1+S$

Table S2: Fitness scheme used for the invasion analysis of an imprinted sexually antagonistic allele. w_i is the fitness of a female when an individual of genotype i has control over the sexually antagonistic trait, similarly v_i represents the fitness of a male where the controlling genotype is i. S represents the benefit of the allele when homozygous, T the cost, and x is the fraction of expression that comes from a maternal-origin gene copy in a female, and and y is the fraction of expression that comes from the paternal-origin copy in a male. For the case of Iceryan hermaphroditism, we assume that the notionally triploid females (diploid females with the additional haploid spermatogenic tissue) are equivalent to 'true' diploid females.

2.4 Invasion conditions

2.4.1 Rice fitness scheme

Following the Rice fitness scheme outlined in Table S1, and using the methodology outlined in the sections above, we generate the invasion conditions for a female beneficial allele under weak selection (Table S3-S5). These conditions have been rearranged into the form $T/S < F$, where the right hand side of these equations, F, can be defined as the 'potential for feminisation', as is also described in the main text. This provides a measure for how stringent invasion conditions are for female beneficial alleles. Conditions for male beneficial alleles can easily be recovered from these equations as the condition for a male beneficial allele is simply $T/S < M$, where M is the potential for masculinisation and $M = 1/F$. Plots of these invasion conditions can also be seen in Figures S2-S4.

We can also see in Table S3 some of the key results in the main text. When there is no population structure, i.e. $s = 0$, $\phi_m = 1 - d_m = 0$, $\phi_f = 1 - d_f = 0$, then we recover the following results.
For arrhenotoky:

$$F = 2h_f \tag{S2.4.1}$$

For male PGE:

$$F = \frac{2h_f}{h_m} \tag{S2.4.2}$$

For diploidy:

$$F = \frac{h_f}{h_m} \tag{S2.4.3}$$

In the sib-mating scenario, when $h_f = 1/2$ and $\phi_f = 1 - d_f = 0$ then we recover the results for diploidy and arrhenotoky of:

$$F = \frac{1}{1-s} \tag{S2.4.4}$$

And for male PGE:

$$F = \frac{4-s}{2(1-s)} \tag{S2.4.5}$$

Equations used for the plots found in Figure 3 of the main text can be regenerated from Table S3, by setting $h_f = h_m = 1/2$.

The results for maternal and paternal control can be seen in Tables S4 and S5 respectively, and the results in the main text can be generated by setting $h_f = 1/2$ and $\phi_f = 1 - d_f = 0$. With these simplifications, the results for a maternally controlled trait for arrhenotoky and male PGE are:

$$F = \frac{2 + (2-s)s}{(2-s)(1-s)} \tag{S2.4.6}$$

And for diploidy:

$$F = \frac{1+s}{1-s} \tag{S2.4.7}$$

Under paternal control, the results for arrhenotoky become:

$$F = \frac{4 + (2-s)s}{s(1-s)} \tag{S2.4.8}$$

For male PGE:

$$F = \frac{4 - s(1 - (2-s)s))}{(3-s)(1-s)s} \tag{S2.4.9}$$

And for diploidy:

$$F = \frac{1+s}{1-s} \tag{S2.4.10}$$

	Sib-mating	Viscous population: DMD	Viscous population: DMD
Haploidy	$\frac{T}{S} < \frac{(s+1)\phi_f^2-2}{2(s-1)}$	$\frac{T}{S} < \frac{\phi_f^2(\phi_m+1)-2}{\phi_m^2+\phi_m-2}$	$\frac{T}{S} < \frac{\phi_f(\phi_f+\phi_m)-2}{\phi_m(\phi_f+\phi_m)-2}$
Diploidy	$\frac{T}{S} < \frac{\left(4(s-1)h_f-s\right)\left((s+1)\phi_f^2-2\right)}{8(s-1)^2h_m-2(s-1)s}$	$\frac{T}{S} < \frac{\left(\phi_f^2(\phi_m+1)-2\right)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-1)(\phi_m+2)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < \frac{(\phi_f(\phi_f+\phi_m)-2)((4h_f-1)\phi_f\phi_m-4h_f)}{(\phi_m(\phi_f+\phi_m)-2)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Arrhenotoky	$\frac{T}{S} < -\frac{\left(4(s-1)h_f-s\right)\left(3\phi_f^2+s-4\right)}{(s-4)(s-2)(s-1)}$	$\frac{T}{S} < -\frac{\left(3\phi_f^2+\phi_m-4\right)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-2)(\phi_m-1)((\phi_m-1)\phi_m-4)}$	$\frac{T}{S} < \frac{\left(\left(\phi_f^2-2\right)\phi_f\phi_m-3\phi_f^2+4\right)((4h_f-1)\phi_f\phi_m-4h_f)}{(\phi_f\phi_m-2)(\phi_f(\phi_m^2-3)\phi_m-2\phi_m^2+4)}$
Paterothylotoky	$\frac{T}{S} < -\frac{(s-2)\left((s+2)\phi_f^2+s-4\right)}{(s-4)(s-1)(4(s-1)h_m-s)}$	$\frac{T}{S} < -\frac{(\phi_m-2)\left(\phi_f^2(\phi_m+2)+\phi_m-4\right)}{(\phi_m-1)((\phi_m-2)\phi_m-4)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < \frac{(\phi_f\phi_m-2)\left(\left(\phi_f^2-3\right)\phi_f\phi_m-2\phi_f^2+4\right)}{(\phi_f(\phi_m^2-2)\phi_m-3\phi_m^2+4)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Male PGE	$\frac{T}{S} < \frac{\left(4(s-1)h_f-s\right)\left(3\phi_f^2+s-4\right)}{8(s-1)^2h_m-2(s-1)s}$	$\frac{T}{S} < \frac{\left(3\phi_f^2+\phi_m-4\right)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-1)(\phi_m+2)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < -\frac{\left(\left(\phi_f^2-2\right)\phi_f\phi_m-3\phi_f^2+4\right)((4h_f-1)\phi_f\phi_m-4h_f)}{(\phi_m(\phi_f+\phi_m)-2)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Female PGE	$\frac{T}{S} < \frac{\left(4(s-1)h_f-s\right)\left(((s-3)s-1)\phi_f^2-(s-2)s+2\right)}{(s-4)(s-1)(4(s-1)h_m-s)}$	$\frac{T}{S} < \frac{\left(\phi_f^2((\phi_m-3)\phi_m-1)-(\phi_m-2)\phi_m+2\right)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-1)((\phi_m-2)\phi_m-4)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < -\frac{(\phi_f(\phi_f+\phi_m)-2)((4h_f-1)\phi_f\phi_m-4h_f)}{(\phi_f(\phi_m^2-2)\phi_m-3\phi_m^2+4)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Cytoplasmic	$\frac{T}{S} < \frac{(2\mathbb{L}-1)\mathbb{L}+1)\phi_f^2(\mathbb{L}(s-1)+1)+\mathbb{L}(-2\mathbb{L}s+s+1)-1}{\mathbb{L}(s-1)}$	$\frac{T}{S} < \frac{(2(\mathbb{L}-1)\mathbb{L}+1)\phi_f^2(-\mathbb{L}+\mathbb{L}\phi_m+1)+\mathbb{L}+(1-2\mathbb{L})\mathbb{L}\phi_m-1}{\mathbb{L}(\phi_m-1)((2(\mathbb{L}-1)\mathbb{L}+1)\phi_m+1)}$	$\frac{T}{S} < \frac{(\mathbb{L}-1)\left((2(\mathbb{L}-1)\mathbb{L}+1)\phi_f^2-2(\mathbb{L}-1)\mathbb{L}\phi_f\phi_m-1\right)}{\mathbb{L}\phi_m(2(\mathbb{L}-1)\mathbb{L}(\phi_f-\phi_m)-\phi_m)+\mathbb{L}}$

Table S3: Invasion conditions for female beneficial alleles under various genetic systems, and in various life-cycle structures, when the trait is under offspring control, and selection is weak. $\mathbb{L}$ is the extent of paternal leakage, s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Sib-mating	Viscous population: DMD	Viscous population: DDM
Haploidy	$\frac{T}{S} < \frac{(s+1)\left(\phi_f^2-1\right)}{s-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m^2-1}$
Diploidy	$\frac{T}{S} < \frac{(s+1)\left(\phi_f^2-1\right)\left(4(s-1)h_f-s\right)}{(s-1)(4(s-1)h_m-s)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-1)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\left((4h_f-1)\phi_f\phi_m-4h_f\right)}{(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Arrhenotoky	$\frac{T}{S} < \frac{((s-2)s-2)\left(\phi_f^2-1\right)\left(4(s-1)h_f-s\right)}{(s-2)(s-1)(4(s-1)h_m-s)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\left((\phi_m-2)\phi_m-2\right)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-2)(\phi_m-1)(\phi_m+1)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < -\frac{2\left(\phi_f^2-1\right)\left((4h_f-1)\phi_f\phi_m-4h_f\right)}{(\phi_m^2-1)\left(\phi_f\phi_m-2\right)\left(\phi_f(4h_m-1)\phi_m-4h_m\right)}$
Paterothylotoky	$\frac{T}{S} < \frac{s(2s-5)\left(\phi_f^2-1\right)}{2(s-2)(s-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\phi_m(2\phi_m-5)}{2(\phi_m-2)(\phi_m-1)(\phi_m+1)}$	$\frac{T}{S} < -\frac{\phi_f\left(\phi_f^2-1\right)\phi_m}{2(\phi_m^2-1)(\phi_f\phi_m-2)}$
Male PGE	$\frac{T}{S} < \frac{((s-2)s-2)\left(\phi_f^2-1\right)\left(4(s-1)h_f-s\right)}{(s-2)(s-1)(4(s-1)h_m-s)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\left((\phi_m-2)\phi_m-2\right)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-2)(\phi_m-1)(\phi_m+1)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < -\frac{2\left(\phi_f^2-1\right)\left((4h_f-1)\phi_f\phi_m-4h_f\right)}{(\phi_m^2-1)\left(\phi_f\phi_m-2\right)\left(\phi_f(4h_m-1)\phi_m-4h_m\right)}$
Female PGE	$\frac{T}{S} < \frac{s(s^2-7)\left(\phi_f^2-1\right)\left(4(s-1)h_f-s\right)}{(s-1)((s-1)s-4)(4(s-1)h_m-s)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\phi_m(\phi_m^2-7)\left(4h_f(\phi_m-1)-\phi_m\right)}{(\phi_m-1)(\phi_m+1)((\phi_m-1)\phi_m-4)\left(4h_m(\phi_m-1)-\phi_m\right)}$	$\frac{T}{S} < -\frac{\phi_f\left(\phi_f^2-1\right)\phi_m\left(\phi_f\phi_m-3\right)\left((4h_f-1)\phi_f\phi_m-4h_f\right)}{(\phi_m^2-1)\left(\phi_f\phi_m(\phi_f\phi_m-1)-4\right)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Cytoplasmic	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\mathbb{L}(s-1)+1)}{\mathbb{L}(s-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\left(-\mathbb{L}+\mathbb{L}\phi_m+1\right)}{\mathbb{L}(\phi_m^2-1)}$	$\frac{T}{S} < -\frac{(\mathbb{L}-1)\left(\phi_f^2-1\right)}{\mathbb{L}(\phi_m^2-1)}$

Table S4: Invasion conditions for female beneficial alleles under various genetic systems, and in various life-cycle structures, when the trait is under maternal control, and selection is weak. $\mathbb{L}$ is the extent of paternal leakage, s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Sib-mating	Viscous population: DMD	Viscous population: DDM
Haploidy	$\frac{T}{S} < \frac{(s+1)\left(\phi_f^2-1\right)}{s-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m^2-1}$
Diploidy	$\frac{T}{S} < \frac{(s+1)\left(\phi_f^2-1\right)(4(s-1)h_f-s)}{(s-1)(4(s-1)h_m-s)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(4h_f(\phi_m-1)-\phi_m)}{(\phi_m-1)(4h_m(\phi_m-1)-\phi_m)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)((4h_f-1)\phi_f\phi_m-4h_f)}{(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Arrhenotoky	$\frac{T}{S} < \frac{((s-2)s+4)\left(\phi_f^2-1\right)}{(s-1)s}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)((\phi_m-2)\phi_m+4)}{\phi_m(\phi_m^2-1)}$	$\frac{T}{S} < -\frac{2\left(\phi_f^2-1\right)(\phi_f\phi_m-2)}{\phi_f\phi_m(\phi_m^2-1)}$
Paterothylotoky	$\frac{T}{S} < \frac{(s+2)\left(\phi_f^2-1\right)(4(s-1)h_f-s)}{8(s-1)^2h_m-2(s-1)s}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m+2)(4h_f(\phi_m-1)-\phi_m)}{2(\phi_m^2-1)(4h_m(\phi_m-1)-\phi_m)}$	$\frac{T}{S} < -\frac{\left(\phi_f^2-1\right)(\phi_f\phi_m-2)((4h_f-1)\phi_f\phi_m-4h_f)}{2(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Male PGE	$\frac{T}{S} < \frac{(s((s-2)s-1)-4)\left(\phi_f^2-1\right)(4(s-1)h_f-s)}{(s-3)(s-1)s(4(s-1)h_m-s)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m((\phi_m-2)\phi_m-1)-4)(4h_f(\phi_m-1)-\phi_m)}{(\phi_m-3)(\phi_m-1)\phi_m(\phi_m+1)(4h_m(\phi_m-1)-\phi_m)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_f\phi_m(\phi_f\phi_m-1)-4)((4h_f-1)\phi_f\phi_m-4h_f)}{\phi_f\phi_m(\phi_m^2-1)(\phi_f\phi_m-3)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Female PGE	$\frac{T}{S} < \frac{(s+2)\left(\phi_f^2-1\right)(4(s-1)h_f-s)}{8(s-1)^2h_m-2(s-1)s}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m+2)(4h_f(\phi_m-1)-\phi_m)}{2(\phi_m^2-1)(4h_m(\phi_m-1)-\phi_m)}$	$\frac{T}{S} < -\frac{\left(\phi_f^2-1\right)(\phi_f\phi_m-2)((4h_f-1)\phi_f\phi_m-4h_f)}{2(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}$
Cytoplasmic	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\mathbb{L}(s-1)+1)}{\mathbb{L}(s-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(-\mathbb{L}+\mathbb{L}\phi_m+1)}{\mathbb{L}(\phi_m^2-1)}$	$\frac{T}{S} < -\frac{(\mathbb{L}-1)\left(\phi_f^2-1\right)}{\mathbb{L}(\phi_m^2-1)}$

Table S5: Invasion conditions for female beneficial alleles under various genetic systems, and in various life-cycle structures, when the trait is under paternal control, and selection is weak. $\mathbb{L}$ is the extent of paternal leakage, s is the degree of sib-mating, $\phi_f = 1 - d_f$ is the degree of female philopatry and $\phi_m = 1 - d_m$ is the degree of male philopatry.

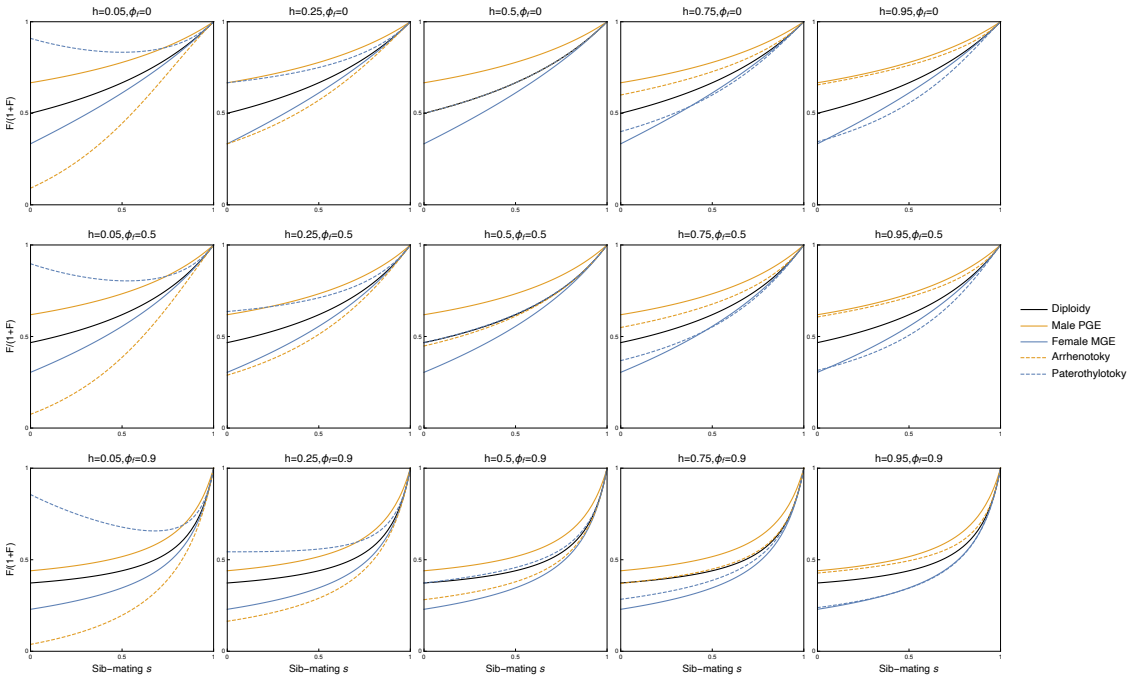


Figure S2: Potential for feminisation across some of the different genetic systems we consider as a function of dominance h, sib-mating s, and female philopatry $\phi_f = 1 - d_f$

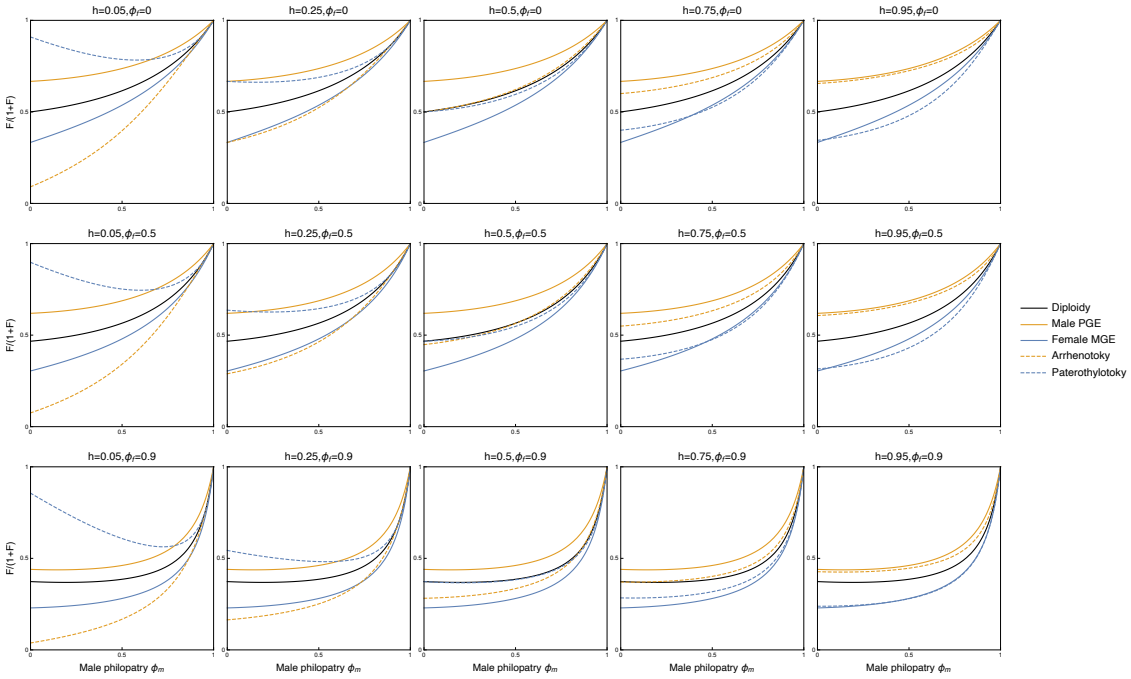


Figure S3: Potential for feminisation across some of the different genetic systems we consider when mating occurs pre-female dispersal, as a function of dominance h, male philopatry $\phi_m = 1-d_m$, and female philopatry $\phi_f = 1-d_f$

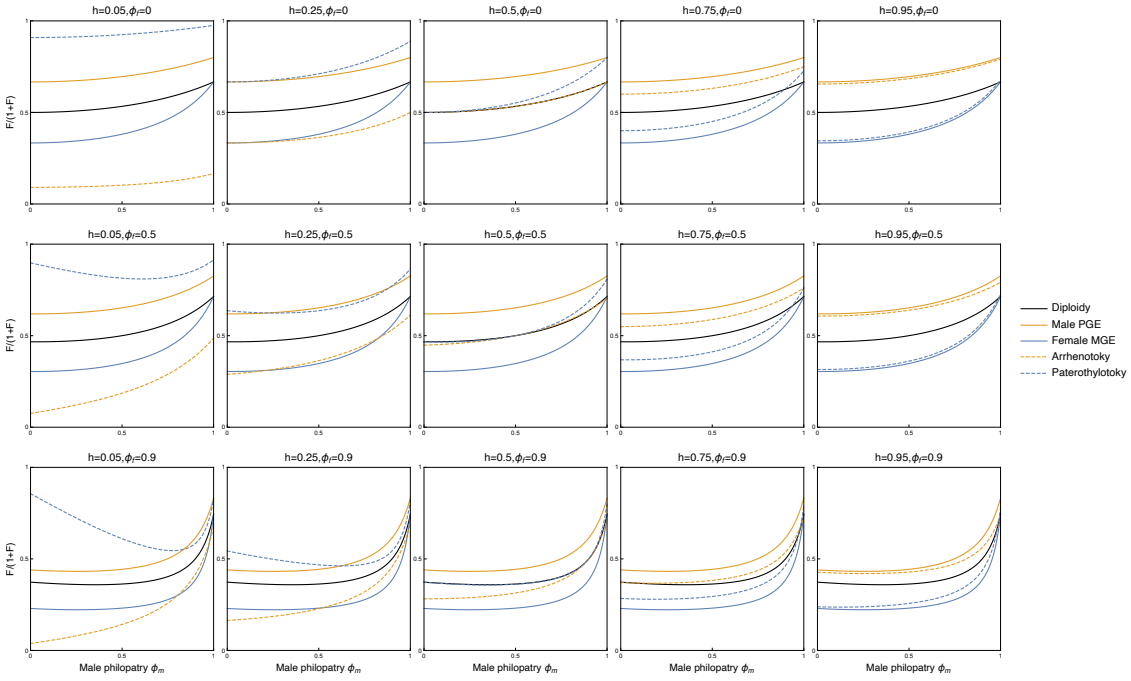


Figure S4: Potential for feminisation across some of the different genetic systems we consider when mating occurs post-female dispersal, as a function of dominance h, male philopatry $\phi_m = 1-d_m$, and female philopatry $\phi_f = 1-d_f$

2.4.2 Imprinting fitness scheme

We can alternatively use the fitness scheme seen in Table S2, where we allow control of the trait to be assigned disproportionately either to the maternal-origin or paternal-origin copy within an individual. with the results for offspring control, maternal control, and paternal control found in Tables S6-S8. The fraction of expression that derives from the maternal-origin copy in a female is denoted x and the fraction of expression coming from the paternal-origin copy in a male is denoted y. Here the results for imprinting discussed in the main text can be found, in particular the result for offspring control under male PGE can be generated by assuming $\phi_f = 1 - d_f = 0$ giving:

$$F = \frac{4 - s(2 - s - 2(1-s)x}{(1-s)(4 - s - 2(2-s)y)} \tag{S2.4.11}$$

	Sib-mating	Viscous population: DMD	Viscous population: DDM
Diploidy	$\frac{T}{S} < \frac{(s+1)\phi_f^2 - 2}{2(s-1)}$	$\frac{T}{S} < \frac{\phi_f^2(\phi_m+1)-2}{\phi_m^2+\phi_m-2}$	$\frac{T}{S} < \frac{\phi_f(\phi_f+\phi_m)-2}{\phi_m(\phi_f+\phi_m)-2}$
Arrhenotoky	$\frac{T}{S} < \frac{\phi_f^2((s-2)s(2x-1)+2(x-2))+s(-2sx+s+2x-2)+4}{(s-4)(s-1)}$	$\frac{T}{S} < \frac{\phi_f^2((2x-1)(\phi_m-2)\phi_m+2(x-2))+\phi_m(-2x\phi_m+\phi_m+2x-2)+4}{(\phi_m-1)((\phi_m-1)\phi_m-4)}$	$\frac{T}{S} < \frac{2\phi_f\left(\phi_m\left(-(x-1)\phi_f^2-1\right)+(x-2)\phi_f\right)+4}{\phi_f(\phi_m^2-3)\phi_m-2\phi_m^2+4}$
Paterothylotoky	$\frac{T}{S} < \frac{(s+2)\phi_f^2+s-4}{2(s-1)(s(y-1)+2)}$	$\frac{T}{S} < \frac{\phi_f^2(\phi_m+2)+\phi_m-4}{2(\phi_m-1)((y-1)\phi_m^2+\phi_m+2)}$	$\frac{T}{S} < \frac{\phi_f\left(2\phi_f-\left(\phi_f^2-3\right)\phi_m\right)-4}{2\phi_f\phi_m((y-1)\phi_m^2+1)-2(y-2)\phi_m^2-4}$
Male PGE	$\frac{T}{S} < \frac{\phi_f^2(s^2-2(s-1)^2x-2s+4)+s(2(s-1)x-s+2)-4}{(s-1)(2(s-2)y-s+4)}$	$\frac{T}{S} < \frac{\phi_f^2(-(2x-1)(\phi_m-2)\phi_m-2x+4)+\phi_m((2x-1)\phi_m-2x+2)-4}{(\phi_m-1)((2y-1)\phi_m^2+\phi_m-4y+4)}$	$\frac{T}{S} < \frac{2\phi_f(\phi_f((x-1)\phi_f\phi_m-x+2)+\phi_m)-4}{\phi_f\phi_m((2y-1)\phi_m^2-4y+3)-2(y-1)(\phi_m^2-2)}$
Female PGE	$\frac{T}{S} < \frac{\phi_f^2(-2(s-1)^2x+s+2)+2(s-2)(s-1)x+s-4}{2(s-1)(s(y-1)+2)}$	$\frac{T}{S} < \frac{\phi_f^2(\phi_m(-2x\phi_m+4x+1)-2x+2)+2x(\phi_m-3)\phi_m+\phi_m+4x-4}{2(\phi_m-1)((y-1)\phi_m^2+\phi_m+2)}$	$\frac{T}{S} < \frac{\phi_f\phi_m\left((2x-1)\phi_f^2-4x+3\right)-2(x-1)\left(\phi_f^2-2\right)}{2\phi_f\phi_m((y-1)\phi_m^2+1)-2(y-2)\phi_m^2-4}$

Table S6: Invasion conditions for female beneficial alleles under various genetic systems, and in various life-cycle structures, when the trait is under offspring control, and selection is weak. x is the proportion of expression that comes from the maternal-origin gene in females, y is the proportion of expression that comes from the paternal-origin gene in males, s is the degree of sib-mating, $\phi_f = 1 - d_f$ is the degree of female philopatry and $\phi_m = 1 - d_m$ is the degree of male philopatry.

	Sib-mating	Viscous population: DMD	Viscous population: DDM
Diploidy	$\frac{T}{S} < \frac{(s+1)\left(\phi_f^2-1\right)}{s-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m^2-1}$
Arrhenotoky	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(2(s-1)sx-s-2)}{(s-2)(s-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m(2x\phi_m-2x-1)-2)}{(\phi_m-2)(\phi_m-1)(\phi_m+1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)((2x-1)\phi_f\phi_m(\phi_f\phi_m-1)-2)}{(\phi_m^2-1)(\phi_f\phi_m-2)}$
Paterothylotoky	$\frac{T}{S} < \frac{s(2s-5)\left(\phi_f^2-1\right)}{2(s-2)(s-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\phi_m(2\phi_m-5)}{2(\phi_m-2)(\phi_m-1)(\phi_m+1)}$	$\frac{T}{S} < -\frac{\phi_f\left(\phi_f^2-1\right)\phi_m}{2(\phi_m^2-1)(\phi_f\phi_m-2)}$
Male PGE	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(2(s-1)sx-s-2)}{(s-2)(s-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m(2x\phi_m-2x-1)-2)}{(\phi_m-2)(\phi_m-1)(\phi_m+1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)((2x-1)\phi_f\phi_m(\phi_f\phi_m-1)-2)}{(\phi_m^2-1)(\phi_f\phi_m-2)}$
Female PGE	$\frac{T}{S} < \frac{s\left(\phi_f^2-1\right)((s-3)(s-1)x+2s-5)}{(s-1)((s-4)(s-1)x+2(s-2))}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)\phi_m(\phi_m(x\phi_m-4x+2)+3x-5)}{(\phi_m^2-1)(\phi_m(x\phi_m-5x+2)+4(x-1))}$	$\frac{T}{S} < \frac{\phi_f\left(\phi_f^2-1\right)\phi_m(x\phi_f\phi_m-x-1)}{(\phi_m^2-1)(\phi_f\phi_m(x\phi_f\phi_m-5x+2)+4(x-1))}$

Table S7: Invasion conditions for female beneficial alleles under various genetic systems, and in various life-cycle structures, when the trait is under maternal control, and selection is weak. x is the proportion of expression that comes from the maternal-origin gene in females, y is the proportion of expression that comes from the paternal-origin gene in males, s is the degree of sib-mating, $\phi_f = 1 - d_f$ is the degree of female philopatry and $\phi_m = 1 - d_m$ is the degree of male philopatry.

	Sib-mating	Viscous population: DMD	Viscous population: DDM
Diploidy	$\frac{T}{S} < \frac{(s+1)\left(\phi_f^2-1\right)}{s-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m-1}$	$\frac{T}{S} < \frac{\phi_f^2-1}{\phi_m^2-1}$
Arrhenotoky	$\frac{T}{S} < \frac{((s-2)s+4)\left(\phi_f^2-1\right)}{(s-1)s}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)((\phi_m-2)\phi_m+4)}{\phi_m(\phi_m^2-1)}$	$\frac{T}{S} < -\frac{2\left(\phi_f^2-1\right)(\phi_f\phi_m-2)}{\phi_f\phi_m(\phi_m^2-1)}$
Paterothylotoky	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(s((s-1)s(2y-1)-1)-2)}{(s-1)((s-1)s(2y-1)-2)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m((2y-1)(\phi_m-1)\phi_m-1)-2)}{(\phi_m^2-1)((2y-1)(\phi_m-1)\phi_m-2)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_f\phi_m-2)}{(\phi_m^2-1)((2y-1)\phi_f\phi_m(\phi_f\phi_m-1)-2)}$
Male PGE	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)((s^3-5s+4)y-s^2+2s-4)}{(s-1)s((s-1)y-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m(\phi_m(y\phi_m-1)-5y+2)+4(y-1))}{\phi_m(\phi_m^2-1)(y\phi_m-y-1)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_f\phi_m(y\phi_f\phi_m-5y+2)+4(y-1))}{\phi_f\phi_m(\phi_m^2-1)(y\phi_f\phi_m-y-1)}$
Female PGE	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(s((s-1)s(2y-1)-1)-2)}{(s-1)((s-1)s(2y-1)-2)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_m((2y-1)(\phi_m-1)\phi_m-1)-2)}{(\phi_m^2-1)((2y-1)(\phi_m-1)\phi_m-2)}$	$\frac{T}{S} < \frac{\left(\phi_f^2-1\right)(\phi_f\phi_m-2)}{(\phi_m^2-1)((2y-1)\phi_f\phi_m(\phi_f\phi_m-1)-2)}$

Table S8: Invasion conditions for female beneficial alleles under various genetic systems, and in various life-cycle structures, when the trait is under paternal control, and selection is weak. x is the proportion of expression that comes from the maternal-origin gene in females, y is the proportion of expression that comes from the paternal-origin gene in males, s is the degree of sib-mating, $\phi_f = 1 - d_f$ is the degree of female philopatry and $\phi_m = 1 - d_m$ is the degree of male philopatry.

2.4.3 Pseudo-hermaphroditism in Icerya and oedipal mating

In addition to the core life-cycles and genetics analysed, we also consider two further ways that inbreeding may occur: oedipal mating (which is only possible for arrhenotokous species) and pseudo-hermaphroditism in Icerya. As defined earlier type I oedipal mating is where male offspring compete to mate with their mother, whilst in type II oedipal mating the mother simply mates with a random son and so there is no additional selection in this step. The results for these can be seen in Table S9 and plots comparing them to the arrhenotokous sib-mating scenario can be seen in S5.

	Rice fitness scheme	Imprinting fitness scheme
Oedipal mating - type I	$\frac{T}{S} < -\frac{(2(\mathcal{O}-1)h_f-\mathcal{O})\left((\mathcal{O}+3)\phi_f^2-4\right)}{4(\mathcal{O}-1)}$	$\frac{T}{S} < \frac{\phi_f^2((\mathcal{O}-1)x+2)-2}{2(\mathcal{O}-1)}$
Oedipal mating - type II	$\frac{T}{S} < \frac{(2(\mathcal{O}-1)h_f-\mathcal{O})\left((\mathcal{O}+3)\phi_f^2-4\right)}{2(\mathcal{O}-2)(\mathcal{O}-1)}$	$\frac{T}{S} < \frac{\phi_f^2(-\mathcal{O}x+x-2)+2}{(\mathcal{O}-2)(\mathcal{O}-1)}$
Pseudo-hermaphroditism in Icerya	$\frac{T}{S} < -\frac{(2(\varphi-1)h_f-\varphi)\left((\varphi^2-3)\phi_f^2-2\varphi+4\right)}{2(\varphi-2)(\varphi-1)}$	$\frac{T}{S} < \frac{\phi_f^2(\varphi^2+\varphi^2(-x)+x-2)-\varphi^2+2(\varphi-1)\varphi x+2}{(\varphi-2)(\varphi-1)}$

Table S9: Invasion conditions for female beneficial alleles under various genetic systems, and in various life-cycle structures, when the trait is under offspring control, and selection is weak. $\mathcal{O}$ is the proportion of females who oedipally mate, φ is the proportion of females who 'self' in the Iceryan system.

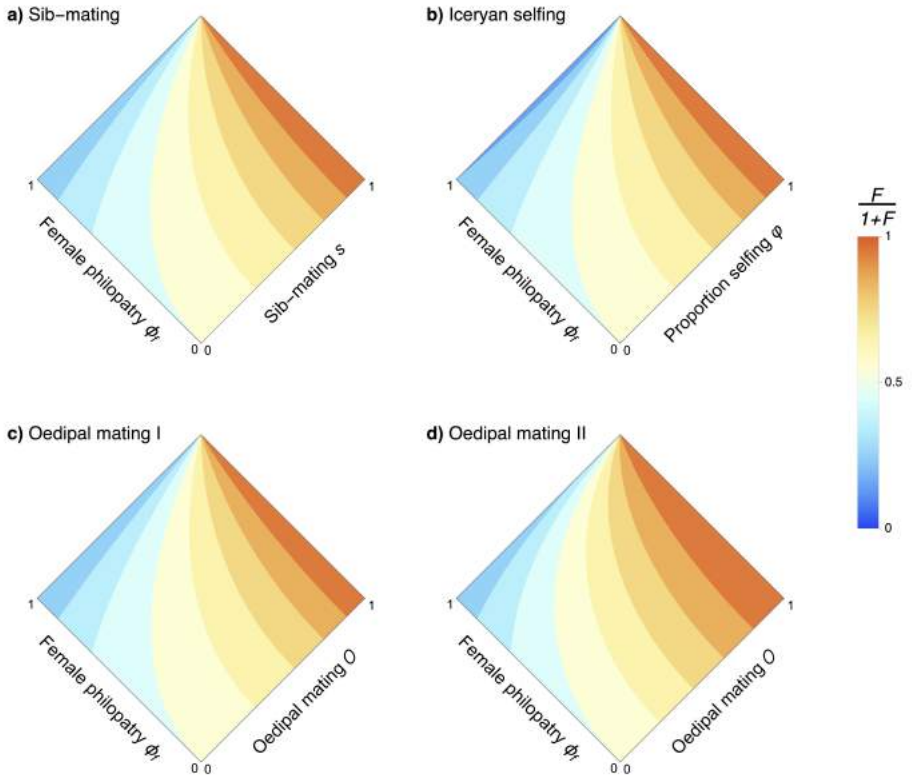


Figure S5: Potential for feminisation across different mechanisms of inbreeding: a) sib-mating, b) pseudo-hermaphroditism in Icerya, c) oedipal mating type I, and d) oedipal mating type II.

2.5 Potential for polymorphism

To investigate the potential for polymorphism we use two approaches, first we use to fitness scheme from Kidwell (1977) (Table S1) and write out explicit conditions for protected polymorphisms under weak selection. Results for the potential for polymorphism under offspring control can be seen in Tables S10-S12, conditions when under maternal control can be seen in Tables . From these we can see that the potential for polymorphism is reduced by inbreeding, and this effect is stronger under sib-mating and DMD than under DDM.

We also use the numerical results from the Rice fitness scheme (Table S1) and plot these out. This allows us to see the potential for polymorphism under stronger selection regimes and more clearly illustrates how the potential for polymorphism may differ for male and female beneficial alleles.

	Conditions for stable polymorphism
Diploidy	$-\frac{2(s-1)(4(s-1)h_m-3s+4)}{\left(4(s-1)h_f-s\right)\left((s+1)\phi_f^2-2\right)} < \frac{u_f}{u_m} < \frac{2(s-1)(s-4(s-1)h_m)}{\left(4(s-1)h_f-3s+4\right)\left((s+1)\phi_f^2-2\right)}$
Arrhenotoky	$-\frac{(s-4)(s-2)(s-1)}{\left(4(s-1)h_f-s\right)\left(3\phi_f^2+s-4\right)} < \frac{u_f}{u_m} < \frac{(s-4)(s-2)(s-1)}{\left(4(s-1)h_f-3s+4\right)\left(3\phi_f^2+s-4\right)}$
Paterothylotoky	$\frac{(s-4)(s-1)(4(s-1)h_m-3s+4)}{(s-2)\left((s+2)\phi_f^2+s-4\right)} < \frac{u_f}{u_m} < \frac{(s-4)(s-1)(s-4(s-1)h_m)}{(s^2-4)\phi_f^2+s^2-6s+8}$
Male PGE	$-\frac{2(s-1)(4(s-1)h_m-3s+4)}{\left(4(s-1)h_f-s\right)\left(3\phi_f^2+s-4\right)} < \frac{u_f}{u_m} < \frac{2(s-1)(s-4(s-1)h_m)}{\left(4(s-1)h_f-3s+4\right)\left(3\phi_f^2+s-4\right)}$
Female MGE	$-\frac{(s-4)(s-1)(4(s-1)h_m-3s+4)}{\left(4(s-1)h_f-s\right)\left(((s-3)s-1)\phi_f^2-(s-2)s+2\right)} < \frac{u_f}{u_m} < \frac{(s-4)(s-1)(s-4(s-1)h_m)}{\left(4(s-1)h_f-3s+4\right)\left(((s-3)s-1)\phi_f^2-(s-2)s+2\right)}$

Table S10: Weak selection approximations for conditions for the maintenance of a stable polymorphism with sib-mating under various genetic systems, when the trait is under offspring control. s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$-\frac{(\phi_m-1)(\phi_m+2)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2(\phi_m+1)-2\right)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-1)(\phi_m+2)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2(\phi_m+1)-2\right)(4h_f(\phi_m-1)-3\phi_m+4)}$
Arrhenotoky	$-\frac{(\phi_m-2)(\phi_m-1)((\phi_m-1)\phi_m-4)}{\left(3\phi_f^2+\phi_m-4\right)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < \frac{(\phi_m-2)(\phi_m-1)((\phi_m-1)\phi_m-4)}{\left(3\phi_f^2+\phi_m-4\right)(4h_f(\phi_m-1)-3\phi_m+4)}$
Paterothylotoky	$\frac{(\phi_m-1)((\phi_m-2)\phi_m-4)(4h_m(\phi_m-1)-3\phi_m+4)}{(\phi_m-2)\left(\phi_f^2(\phi_m+2)+\phi_m-4\right)} < \frac{u_f}{u_m} < -\frac{(\phi_m-1)((\phi_m-2)\phi_m-4)(4h_m(\phi_m-1)-\phi_m)}{(\phi_m-2)\left(\phi_f^2(\phi_m+2)+\phi_m-4\right)}$
Male PGE	$-\frac{(\phi_m-1)(\phi_m+2)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(3\phi_f^2+\phi_m-4\right)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-1)(\phi_m+2)(4h_m(\phi_m-1)-\phi_m)}{\left(3\phi_f^2+\phi_m-4\right)(4h_f(\phi_m-1)-3\phi_m+4)}$
Female MGE	$-\frac{(\phi_m-1)((\phi_m-2)\phi_m-4)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2((\phi_m-3)\phi_m-1)-(\phi_m-2)\phi_m+2\right)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-1)((\phi_m-2)\phi_m-4)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2((\phi_m-3)\phi_m-1)-(\phi_m-2)\phi_m+2\right)(4h_f(\phi_m-1)-3\phi_m+4)}$

Table S11: Weak selection approximations for the conditions for the maintenance of a stable polymorphism with mating pre-female dispersal (DMD) under various genetic systems, when the trait is under offspring control. s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$-\frac{(\phi_m(\phi_f+\phi_m)-2)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{(\phi_f(\phi_f+\phi_m)-2)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < -\frac{(\phi_m(\phi_f+\phi_m)-2)(\phi_f(4h_m-1)\phi_m-4h_m)}{(\phi_f(\phi_f+\phi_m)-2)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Arrhenotoky	$\frac{(\phi_f\phi_m-2)(\phi_f(\phi_m^2-3)\phi_m-2\phi_m^2+4)}{\left(\left(\phi_f^2-2\right)\phi_f\phi_m-3\phi_f^2+4\right)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < -\frac{(\phi_f\phi_m-2)(\phi_f(\phi_m^2-3)\phi_m-2\phi_m^2+4)}{\left(\left(\phi_f^2-2\right)\phi_f\phi_m-3\phi_f^2+4\right)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Paterothylotoky	$-\frac{(\phi_f(\phi_m^2-2)\phi_m-3\phi_m^2+4)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{(\phi_f\phi_m-2)\left(\left(\phi_f^2-3\right)\phi_f\phi_m-2\phi_f^2+4\right)} < \frac{u_f}{u_m} < \frac{(\phi_f(\phi_m^2-2)\phi_m-3\phi_m^2+4)(\phi_f(4h_m-1)\phi_m-4h_m)}{(\phi_f\phi_m-2)\left(\left(\phi_f^2-3\right)\phi_f\phi_m-2\phi_f^2+4\right)}$
Male PGE	$\frac{(\phi_m(\phi_f+\phi_m)-2)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{\left(\left(\phi_f^2-2\right)\phi_f\phi_m-3\phi_f^2+4\right)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < \frac{(\phi_m(\phi_f+\phi_m)-2)(\phi_f(4h_m-1)\phi_m-4h_m)}{\left(\left(\phi_f^2-2\right)\phi_f\phi_m-3\phi_f^2+4\right)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Female MGE	$\frac{(\phi_f(\phi_m^2-2)\phi_m-3\phi_m^2+4)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{(\phi_f(\phi_f+\phi_m)-2)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < \frac{(\phi_f(\phi_m^2-2)\phi_m-3\phi_m^2+4)(\phi_f(4h_m-1)\phi_m-4h_m)}{(\phi_f(\phi_f+\phi_m)-2)((4h_f-3)\phi_f\phi_m-4h_f+4)}$

Table S12: Weak selection approximations for conditions for the maintenance of a stable polymorphism with mating post-female dispersal (DDM) under various genetic systems, when the trait is under offspring control. s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$\frac{-4(s-1)^2h_m+3s^2-7s+4}{(s+1)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < \frac{(s-1)(s-4(s-1)h_m)}{(s+1)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$
Arrhenotoky	$-\frac{(s-2)(s-1)(4(s-1)h_m-3s+4)}{((s-2)s-2)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < \frac{(s-2)(s-1)(s-4(s-1)h_m)}{((s-2)s-2)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$
Paterothylotoky	n/a
Male PGE	$-\frac{(s-2)(s-1)(4(s-1)h_m-3s+4)}{((s-2)s-2)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < \frac{(s-2)(s-1)(s-4(s-1)h_m)}{((s-2)s-2)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$
Female MGE	$-\frac{(s-1)((s-1)s-4)(4(s-1)h_m-3s+4)}{s\left(s^2-7\right)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < -\frac{(s-1)((s-1)s-4)(4(s-1)h_m-s)}{s\left(s^2-7\right)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$

Table S13: Weak selection approximations for conditions for the maintenance of a stable polymorphism with sib-mating under various genetic systems, when the trait is under maternal control. s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$-\frac{(\phi_m-1)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-1)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)(4h_f(\phi_m-1)-3\phi_m+4)}$
Arrhenotoky	$-\frac{(\phi_m-2)(\phi_m-1)(\phi_m+1)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)((\phi_m-2)\phi_m-2)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-2)(\phi_m-1)(\phi_m+1)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)((\phi_m-2)\phi_m-2)(4h_f(\phi_m-1)-3\phi_m+4)}$
Paterothylotoky	n/a
Male PGE	$-\frac{(\phi_m-2)(\phi_m-1)(\phi_m+1)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)((\phi_m-2)\phi_m-2)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-2)(\phi_m-1)(\phi_m+1)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)((\phi_m-2)\phi_m-2)(4h_f(\phi_m-1)-3\phi_m+4)}$
Female MGE	$-\frac{(\phi_m-1)(\phi_m+1)((\phi_m-1)\phi_m-4)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)\phi_m(\phi_m^2-7)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-1)(\phi_m+1)((\phi_m-1)\phi_m-4)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)\phi_m(\phi_m^2-7)(4h_f(\phi_m-1)-3\phi_m+4)}$

Table S14: Weak selection approximations for the conditions for the maintenance of a stable polymorphism with mating pre-female dispersal (DMD) under various genetic systems, when the trait is under maternal control.s is the degree of sib-mating, $\phi_f = 1 - d_f$ is the degree of female philopatry and $\phi_m = 1 - d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$-\frac{(\phi_m^2-1)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{\left(\phi_f^2-1\right)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < -\frac{(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}{\left(\phi_f^2-1\right)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Arrhenotoky	$\frac{(\phi_m^2-1)(\phi_f\phi_m-2)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{2\left(\phi_f^2-1\right)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < \frac{(\phi_m^2-1)(\phi_f\phi_m-2)(\phi_f(4h_m-1)\phi_m-4h_m)}{2\left(\phi_f^2-1\right)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Paterothylotoky	n/a
Male PGE	$\frac{(\phi_m^2-1)(\phi_f\phi_m-2)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{2\left(\phi_f^2-1\right)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < \frac{(\phi_m^2-1)(\phi_f\phi_m-2)(\phi_f(4h_m-1)\phi_m-4h_m)}{2\left(\phi_f^2-1\right)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Female MGE	$-\frac{(\phi_m^2-1)(\phi_f\phi_m(\phi_f\phi_m-1)-4)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{\phi_f\left(\phi_f^2-1\right)\phi_m(\phi_f\phi_m-3)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < -\frac{(\phi_m^2-1)(\phi_f\phi_m(\phi_f\phi_m-1)-4)(\phi_f(4h_m-1)\phi_m-4h_m)}{\phi_f\left(\phi_f^2-1\right)\phi_m(\phi_f\phi_m-3)((4h_f-3)\phi_f\phi_m-4h_f+4)}$

Table S15: Weak selection approximations for conditions for the maintenance of a stable polymorphism with mating post-female dispersal (DDM) under various genetic systems, when the trait is under maternal control. s is the degree of sib-mating, $\phi_f = 1 - d_f$ is the degree of female philopatry and $\phi_m = 1 - d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$\frac{-4(s-1)^2h_m+3s^2-7s+4}{(s+1)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < \frac{(s-1)(s-4(s-1)h_m)}{(s+1)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$
Arrhenotoky	n/a
Paterothylotoky	$-\frac{2(s-1)(4(s-1)h_m-3s+4)}{(s+2)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < \frac{2(s-1)(s-4(s-1)h_m)}{(s+2)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$
Male PGE	$-\frac{(s-3)(s-1)s(4(s-1)h_m-3s+4)}{(s((s-2)s-1)-4)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < -\frac{(s-3)(s-1)s(4(s-1)h_m-s)}{(s((s-2)s-1)-4)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$
Female MGE	$-\frac{2(s-1)(4(s-1)h_m-3s+4)}{(s+2)\left(\phi_f^2-1\right)(4(s-1)h_f-s)} < \frac{u_f}{u_m} < \frac{2(s-1)(s-4(s-1)h_m)}{(s+2)\left(\phi_f^2-1\right)(4(s-1)h_f-3s+4)}$

Table S16: Weak selection approximations for conditions for the maintenance of a stable polymorphism with sib-mating under various genetic systems, when the trait is under paternal control.s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$-\frac{(\phi_m-1)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-1)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)(4h_f(\phi_m-1)-3\phi_m+4)}$
Arrhenotoky	n/a
Paterothylotoky	$-\frac{2(\phi_m^2-1)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)(\phi_m+2)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{2(\phi_m^2-1)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)(\phi_m+2)(4h_f(\phi_m-1)-3\phi_m+4)}$
Male PGE	$-\frac{(\phi_m-3)(\phi_m-1)\phi_m(\phi_m+1)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)(\phi_m((\phi_m-2)\phi_m-1)-4)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{(\phi_m-3)(\phi_m-1)\phi_m(\phi_m+1)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)(\phi_m((\phi_m-2)\phi_m-1)-4)(4h_f(\phi_m-1)-3\phi_m+4)}$
Female MGE	$-\frac{2(\phi_m^2-1)(4h_m(\phi_m-1)-3\phi_m+4)}{\left(\phi_f^2-1\right)(\phi_m+2)(4h_f(\phi_m-1)-\phi_m)} < \frac{u_f}{u_m} < -\frac{2(\phi_m^2-1)(4h_m(\phi_m-1)-\phi_m)}{\left(\phi_f^2-1\right)(\phi_m+2)(4h_f(\phi_m-1)-3\phi_m+4)}$

Table S17: Weak selection approximations for the conditions for the maintenance of a stable polymorphism with mating pre-female dispersal (DMD) under various genetic systems, when the trait is under paternal control.s is the degree of sib-mating, $\phi_f = 1-d_f$ is the degree of female philopatry and $\phi_m = 1-d_m$ is the degree of male philopatry.

	Conditions for stable polymorphism
Diploidy	$-\frac{(\phi_m^2-1)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{(\phi_f^2-1)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < -\frac{(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}{(\phi_f^2-1)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Arrhenotoky	n/a
Paterothylotoky	$\frac{2(\phi_m^2-1)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{(\phi_f^2-1)(\phi_f\phi_m-2)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < \frac{2(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}{(\phi_f^2-1)(\phi_f\phi_m-2)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Male PGE	$-\frac{\phi_f\phi_m(\phi_m^2-1)(\phi_f\phi_m-3)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{(\phi_f^2-1)(\phi_f\phi_m(\phi_f\phi_m-1)-4)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < -\frac{\phi_f\phi_m(\phi_m^2-1)(\phi_f\phi_m-3)(\phi_f(4h_m-1)\phi_m-4h_m)}{(\phi_f^2-1)(\phi_f\phi_m(\phi_f\phi_m-1)-4)((4h_f-3)\phi_f\phi_m-4h_f+4)}$
Female MGE	$\frac{2(\phi_m^2-1)(\phi_f(4h_m-3)\phi_m-4h_m+4)}{(\phi_f^2-1)(\phi_f\phi_m-2)((4h_f-1)\phi_f\phi_m-4h_f)} < \frac{u_f}{u_m} < \frac{2(\phi_m^2-1)(\phi_f(4h_m-1)\phi_m-4h_m)}{(\phi_f^2-1)(\phi_f\phi_m-2)((4h_f-3)\phi_f\phi_m-4h_f+4)}$

Table S18: Weak selection approximations for conditions for the maintenance of a stable polymorphism with mating post-female dispersal (DDM) under various genetic systems, when the trait is under paternal control. s is the degree of sib-mating, $\phi_f = 1 - d_f$ is the degree of female philopatry and $\phi_m = 1 - d_m$ is the degree of male philopatry.

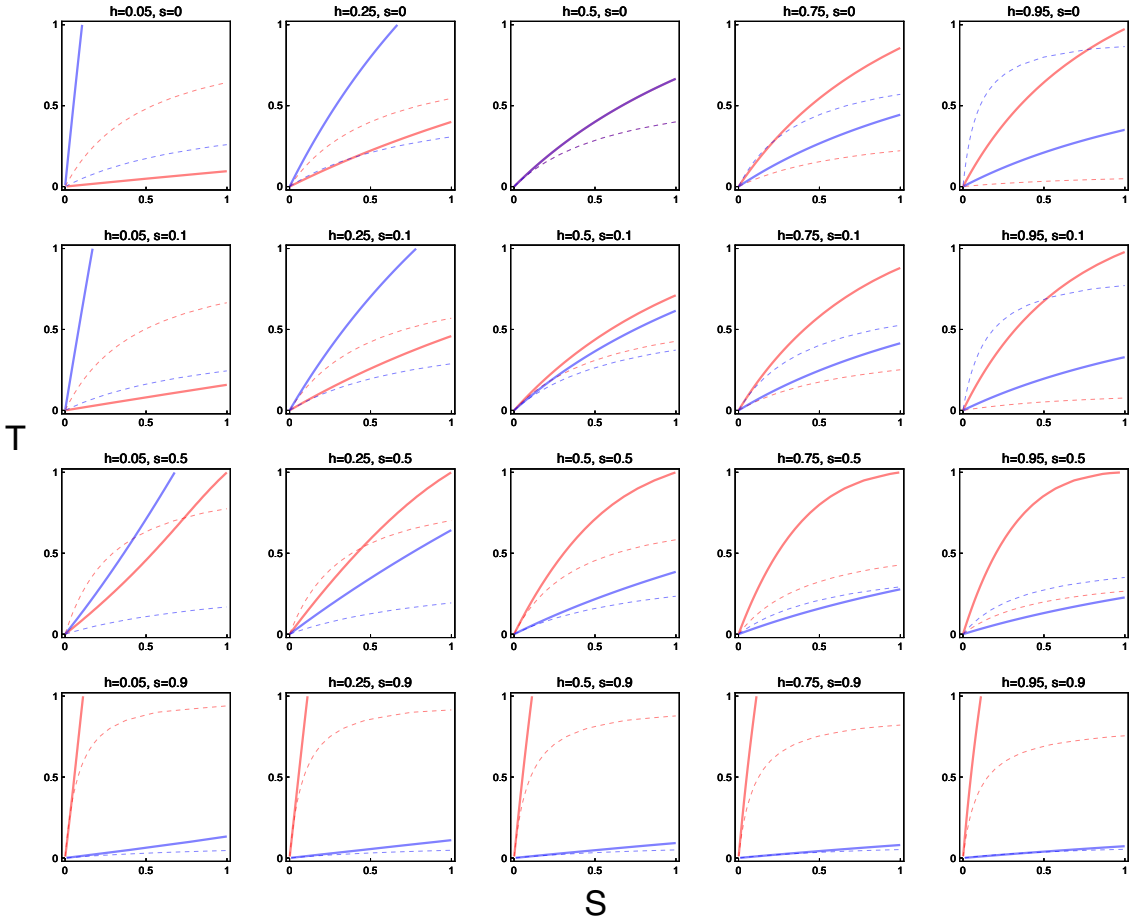


Figure S6: Potential for polymorphism for male and female beneficial alleles under arrhenotoky with sib-mating, across different levels of sib-mating s, and dominance coefficients h, where dominance is parallel across sexes $h = h_f = h_m$

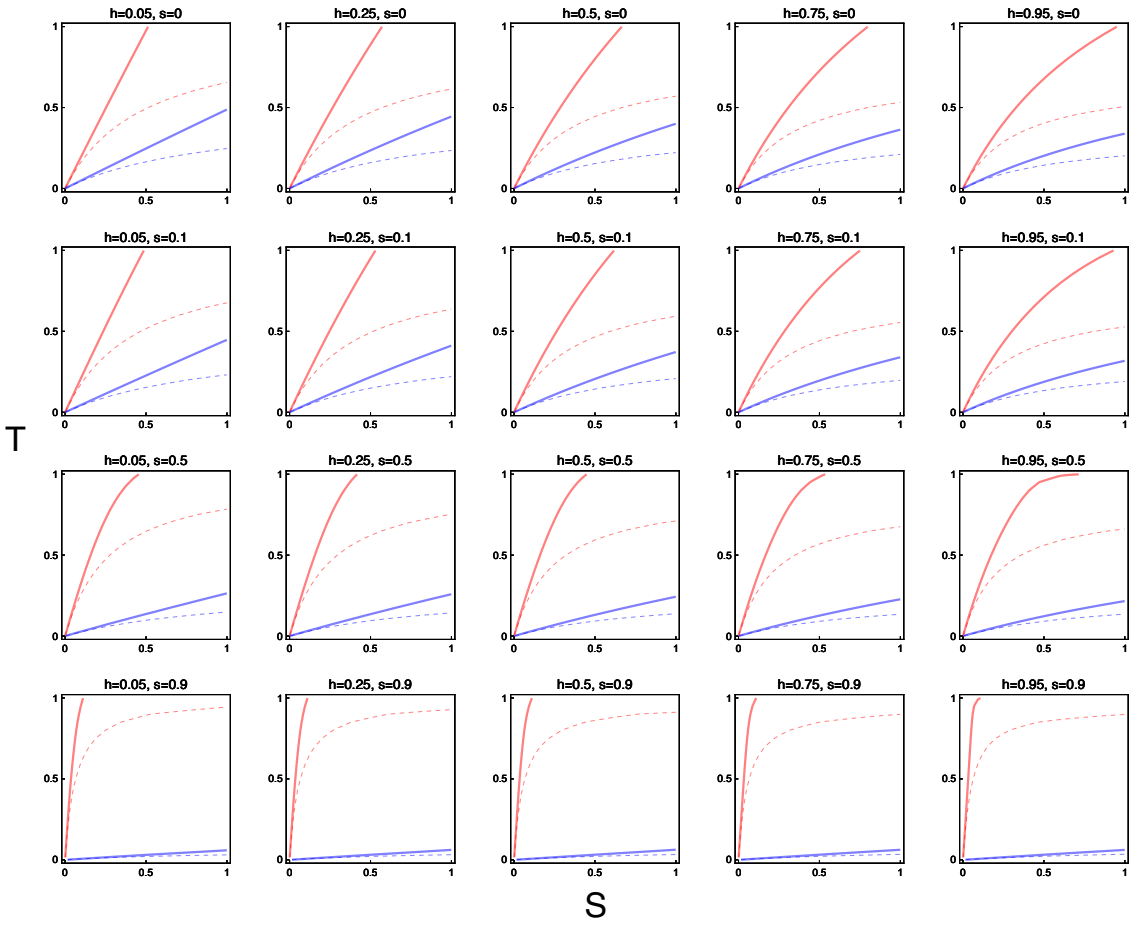


Figure S7: Potential for polymorphism for male and female beneficial alleles under male PGE with sib-mating, across different levels of sib-mating s, and dominance coefficients h, where dominance is parallel across sexes $h = h_f = h_m$

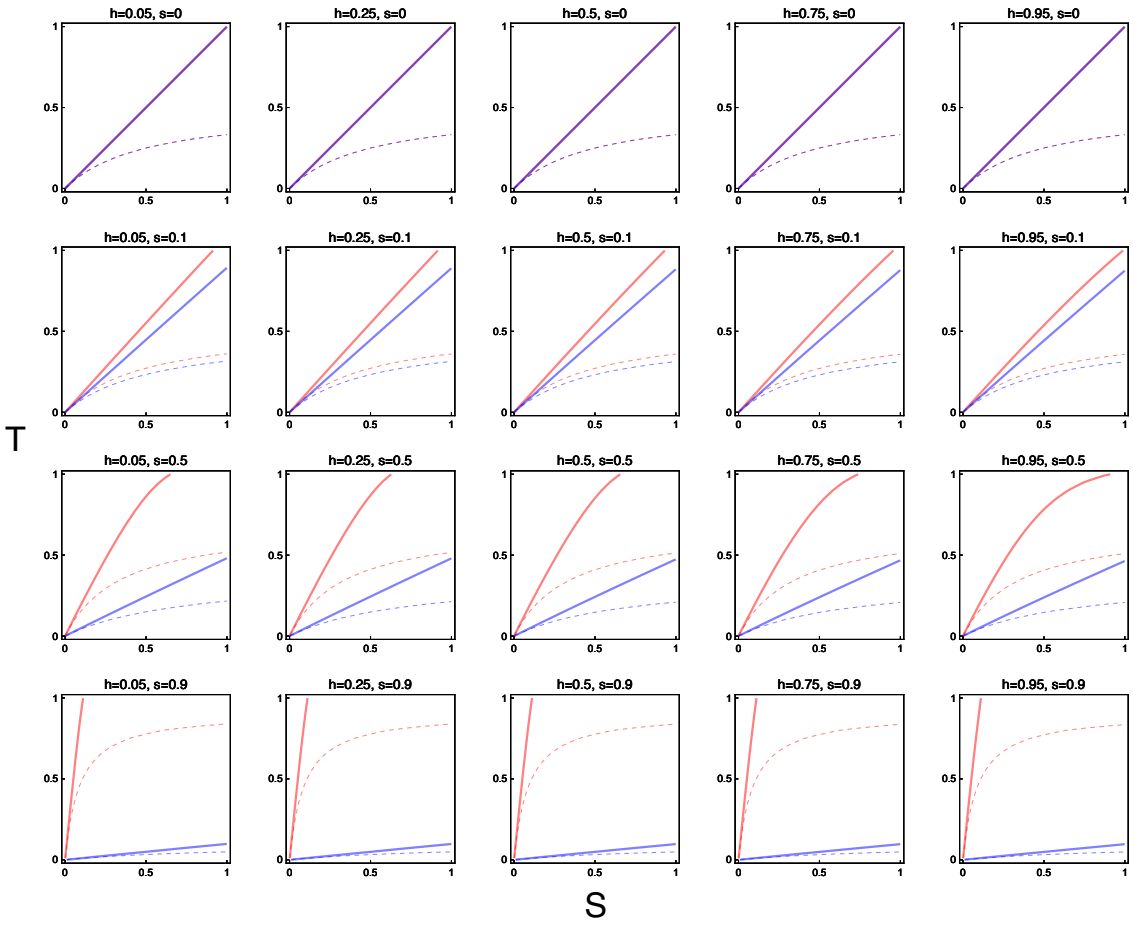


Figure S8: Potential for polymorphism for male and female beneficial alleles under diploidy with sib-mating, across different levels of sib-mating s, and dominance coefficients h, where dominance is parallel across sexes $h = h_f = h_m$

3 Kin selection model of a sexually antagonistic trait

Alongside the above invasion analysis, we also perform a kin-selection analysis of a sexually antagonistic trait ϕ, using the direct fitness methodology of Taylor and Frank (1996). By assuming additivity and weak selection, we can more straightforwardly partition the different effects of the above life-cycles on sexual antagonism into their component parts. Doing this enables a more general analysis of how different life-cycle structures and mating schemes may impact sexual antagonism. We also then use this to understand how, assuming specific fitness functions, this will alter the optimum of such traits, and how, depending on who controls such traits, the optima reached may differ.

3.1 General conditions

We represent the relative fitness of an individual in our population by W, where W is function of $\boldsymbol{\phi}$, a vector of the different values of our sexually antagonistic trait ϕ in our population, including our focal individual. We consider a locus which affects this trait, and denote the genic value of a gene drawn at random from the population at this locus g, and the population average of this trait value to be $\overline{g}$. The condition for natural selection to favour an increase in the level of this sexually antagonistic trait is:

$$\frac{dW}{dg}\Big|_{g=\overline{g}} > 0 \tag{S3.1.1a}$$

And similarly for a decrease in this trait value:

$$\frac{dW}{dg}\Big|_{g=\overline{g}} < 0 \tag{S3.1.1b}$$

If our population is composed of different types of individual, as it is the cases we investigate, then these individuals may be differently affected by changes in the trait value. We write the relative fitness of a focal individual of class j as W_j. Furthermore, these different types of individual may, on average, make differential contributions to the ancestry of the population. To account for this, we must weight the effects in these different classes of individual by that class's reproductive value c_j, which is the product of the relative abundance of that class u_j, and the expected contribution of individuals of that class to the future ancestry of the population v_j, hence $c_\mathrm{j} = u_\mathrm{j} v_\mathrm{j}$. These class reproductive values are normalised such that $\sum_\mathrm{j} c_\mathrm{j} = 1$. Doing this, our condition for increase becomes:

$$\frac{dW}{dg} = \sum_\mathrm{j} c_\mathrm{j} \frac{dW_\mathrm{j}}{dg_\mathrm{j}} > 0 \tag{S3.1.2}$$

Where g_j denotes the genic value of a gene picked randomly at the locus in a focal individual of class j, and once again the condition is evaluated when the population is monomorphic for the trait value $g_\mathrm{j} = g = \overline{g}$. Using the chain rule, we can expand the $dW_\mathrm{j}/dg_\mathrm{j}$ terms, separating the direct effects of the an individual's trait value upon their own fitness, and the effects of the trait values of other social partners upon their fitness. If we index individuals in our population by i then:

$$\frac{dW_\mathrm{j}}{dg_\mathrm{j}} = \sum_\mathrm{i} \frac{\partial W_\mathrm{j}}{\partial \phi_\mathrm{i}} \frac{d\phi_\mathrm{i}}{dG_\mathrm{i}} \frac{dG_\mathrm{i}}{dg_\mathrm{i}} \frac{dg_\mathrm{i}}{dg_\mathrm{j}} \tag{S3.1.3}$$

Where $d\phi_i/dG_i = \gamma$ is the mapping of breeding value to phenotype, which we assume to be a constant across our individuals, dG_i/dg_i is the change in an individual's genetic value with a change in the genetic value at a single locus, which is 1 under adding genetics and $1/n_i$ under averaging genetics with n_i the ploidy of the ith individual (Frank, 2003; Gardner, 2012), and $dg_i/dg_j = \rho_{i,j}$ is the consanguinity between our focal individual of class j, and the individual i. Our total condition for increase can then be written for averaging genetics as:

$$\frac{dW}{dg} = \sum_j \sum_i c_j \frac{\partial W_j}{\partial \phi_i} \frac{\rho_{i,j}}{n_i} > 0 \tag{S3.1.4a}$$

And for adding genetics:

$$\frac{dW}{dg} = \sum_j \sum_i c_j \frac{\partial W_j}{\partial \phi_i} \rho_{i,j} > 0 \tag{S3.1.4b}$$

Further class structure The above analysis assumes that the different genes within an individual contribute equally to the genic value of the individual that they reside within, and also are transmitted equally from that individual. However, this may not be always be the case, for example if there is genomic imprinting, then one of the two parent-of-origin copies may solely determine the phenotype, or alternatively if there is paternal-genome elimination, then only one of the maternal-origin gene copy will be transmitted. To account for this, we now further decompose our population into different classes of genes, where j now indexes a focal gene in class j, for example "female, maternal-origin", and i indexes all of the individual gene copies in our population, $\mathbb{i}$ indexes the individuals in the population, and W is now the relative fitness of an individual gene copy, rather than individual. Our condition for increase now becomes:

$$\frac{dW_j}{dg_j} = \sum_i \frac{\partial W_j}{\partial \phi_{\mathbb{i}}} \frac{d\phi_{\mathbb{i}}}{dG_{\mathbb{i}}} \frac{dG_{\mathbb{i}}}{dg_i} \frac{dg_i}{dg_j} \tag{S3.1.5}$$

Much remains the same as before, however now the term $dG_{\mathbb{i}}/dg_i$ captures the relative effect of a specific gene copy i on it's individual's genetic value $G_{\mathbb{i}}$, rather than being an average across the gene copies in that individual. For instance, if there is genomic imprinting, with the paternal-origin copy silenced, then if that gene copy is of maternal-origin then $dG_{\mathbb{f}}/dg_{f_M} = 1$, whilst if that gene is of paternal-origin $dG_{\mathbb{f}}/dg_{f_P} = 0$. Similarly, a male paternal-origin gene's fitness will be altered differently to that of a male maternal-origin gene under paternal-genome elimination, i.e. $\partial W_{m_P}/\partial \phi_{\mathbb{i}} = 0$.

3.2 Fitness functions

General model We can describe three life-cycles described previously with the following model, where W_f is the relative fitness of an individual female, and W_m is the relative fitness of an individual male. The competitiveness of a female w, and a male v, are functions of a sexually antagonistic trait ϕ. We denote the phenotype of our focal female as ϕ_x^f, our focal male as ϕ_x^m, female and male patchmates as ϕ_y^f and ϕ_y^m, and of females and males in the wider population as ϕ_z^f and ϕ_z^m.

Individuals may compete with both the global pool of individuals, or with natal patch mates. We can capture the variation between these extremes with the term a, the spatial scale of competition (Frank, 1998). a_{ff} is the scale of competition for females upon related females, a_{mm} is the scale of competition for males upon

related males, and a_{fm} is the scale of competition for females upon related males. Individuals may also mate with individuals from the global pool or with natal patch mates, the probability that an individual mates with a patch mate is given by ψ, and the probability that they mate with an individual from the rest of the population is $(1-\psi)$. The values for the scales of competition that emerge from the different life-cycles described can be seen in Table S19.

$$W_{\mathrm{f_M}}, W_{\mathrm{f_P}} = \frac{w[\phi^{\mathrm{f}}_{\mathrm{x}}]}{a_{\mathrm{ff}}\, w[\phi^{\mathrm{f}}_{\mathrm{y}}] + (1-a_{\mathrm{ff}})\, w[\phi^{\mathrm{f}}_{\mathrm{z}}]} \tag{S3.2.1a}$$

$$W_{\mathrm{m_M}}, W_{\mathrm{m_P}} = \left(\frac{v[\phi^{\mathrm{m}}_{\mathrm{x}}]}{a_{\mathrm{mm}}\, v[\phi^{\mathrm{m}}_{\mathrm{y}}] + (1-a_{\mathrm{mm}})\, v[\phi^{\mathrm{m}}_{\mathrm{z}}]}\right)\left(\frac{\psi\, w[\phi^{\mathrm{f}}_{\mathrm{y}}] + (1-\psi)\, w[\phi^{\mathrm{f}}_{\mathrm{z}}]}{a_{\mathrm{fm}}\, w[\phi^{\mathrm{f}}_{\mathrm{y}}] + (1-a_{\mathrm{fm}})\, w[\phi^{\mathrm{f}}_{\mathrm{z}}]}\right) \tag{S3.2.1b}$$

Specific Gaussian model The above model leaves open the specific relationship between the sexually antagonistic trait and competitiveness. We construct a toy model where the competitiveness of females w and males v are two Gaussian functions of the trait ϕ, normally distributed around the female and male optima, ϕ^{f^*} and ϕ^{m^*} respectively, with standard deviations SD_{f} and SD_{m} respectively:

$$w[\phi] = N[\phi^{\mathrm{f}^*}, SD_{\mathrm{f}}] \tag{S3.2.2a}$$

$$v[\phi] = N[\phi^{\mathrm{m}^*}, SD_{\mathrm{m}}] \tag{S3.2.2b}$$

	a_{ff}	a_{fm}	a_{mm}
Fixed sib-mating	$(1-d_{\mathrm{f}})^2$	$(1-d_{\mathrm{f}})^2$	s
Mating pre-dispersal	$(1-d_{\mathrm{f}})^2$	$(1-d_{\mathrm{f}})^2$	$(1-d_{\mathrm{m}})^2$
Mating post-dispersal	$(1-d_{\mathrm{f}})^2$	$(1-d_{\mathrm{m}})\,(1-d_{\mathrm{f}})$	$(1-d_{\mathrm{m}})^2$

Table S19: The sex-specific scales of competition in our three different life-cycles

3.3 Genotype to phenotype mapping

We now look at how the genetic values at different gene positions of an individual g map into the breeding value of the individual G, and therefore the trait value ϕ of that individual. For haploid systems there is only a single gene determining the phenotype, and therefore it is straightforwardly determined by this gene copy. For diploid systems (including arrhenotoky and paterothylotoky), multiple gene positions may contribute to the phenotype, and these different gene positions may have different amounts of influence on the breeding value. We assign a fraction $\mathscr{X}$ of the breeding value in females to the maternal-origin gene, and fraction $(1-\mathscr{X})$ to the paternal-origin gene. In males, we assign a fraction $(1-\mathscr{Y})$ of the breeding value to the maternal-origin gene and a fraction $\mathscr{Y}$ to the paternal-origin gene. Earlier we also discussed how the choice of adding vs averaging genetics is important when comparing across ploidy levels. To allow for this choice we weight the breeding value in females and males by α and β respectively. In all cases there is an uncorrelated error denoted by ϵ.

Additionally, it may be that the trait value ϕ of our focal individual is not necessarily under the control of that individual themselves, but instead is determined by the breeding value of another individual. In particular here, we consider the influence of parents. We denote the value of the parents of our focal individual by $\mathbb{x}$. Thus

the maternal-origin gene of our focal individual's mother would be denoted $\mathbb{x}_{\mathrm{f_M}}$, and the paternal-origin gene of this focal individual's father by $\mathbb{x}_{\mathrm{m_P}}$.

3.3.1 Haploidy

Offspring control

$$\phi_{\mathrm{x}}^{\mathrm{f}} = G_{\mathrm{x}}^{\mathrm{f}} + \epsilon = g_{\mathrm{x,f}} + \epsilon \tag{S3.3.1a}$$

$$\phi_{\mathrm{x}}^{\mathrm{m}} = G_{\mathrm{x}}^{\mathrm{m}} + \epsilon = g_{\mathrm{x,m}} + \epsilon \tag{S3.3.1b}$$

Maternal control

$$\phi_{\mathrm{x}}^{\mathrm{f}} = G_{\mathbb{x}}^{\mathrm{f}} + \epsilon = g_{\mathbb{x},\mathrm{f}} + \epsilon \tag{S3.3.2a}$$

$$\phi_{\mathrm{x}}^{\mathrm{m}} = G_{\mathbb{x}}^{\mathrm{f}} + \epsilon = g_{\mathbb{x},\mathrm{f}} + \epsilon \tag{S3.3.2b}$$

Paternal control

$$\phi_{\mathrm{x}}^{\mathrm{f}} = G_{\mathbb{x}}^{\mathrm{m}} + \epsilon = g_{\mathbb{x},\mathrm{m}} + \epsilon \tag{S3.3.3a}$$

$$\phi_{\mathrm{x}}^{\mathrm{m}} = G_{\mathbb{x}}^{\mathrm{m}} + \epsilon = g_{\mathbb{x},\mathrm{m}} + \epsilon \tag{S3.3.3b}$$

3.3.2 Diploidy

Offspring control

$$\phi_{\mathrm{x}}^{\mathrm{f}} = G_{\mathrm{x}}^{\mathrm{f}} + \epsilon = \alpha\left(\mathscr{X} g_{\mathrm{x,f_M}} + (1-\mathscr{X}) g_{\mathrm{x,f_P}}\right) + \epsilon \tag{S3.3.4a}$$

$$\phi_{\mathrm{x}}^{\mathrm{m}} = G_{\mathrm{x}}^{\mathrm{m}} + \epsilon = \beta\left((1-\mathscr{Y}) g_{\mathrm{x,m_M}} + \mathscr{Y} g_{\mathrm{x,m_P}}\right) + \epsilon \tag{S3.3.4b}$$

Maternal control

$$\phi_{\mathrm{x}}^{\mathrm{f}} = G_{\mathbb{x}}^{\mathrm{f}} + \epsilon = \alpha\left(\mathscr{X} g_{\mathbb{x},\mathrm{f_M}} + (1-\mathscr{X}) g_{\mathbb{x},\mathrm{f_P}}\right) + \epsilon \tag{S3.3.5a}$$

$$\phi_{\mathrm{x}}^{\mathrm{m}} = G_{\mathbb{x}}^{\mathrm{f}} + \epsilon = \alpha\left(\mathscr{X} g_{\mathbb{x},\mathrm{f_M}} + (1-\mathscr{X}) g_{\mathbb{x},\mathrm{f_P}}\right) + \epsilon \tag{S3.3.5b}$$

Paternal control

$$\phi_{\mathrm{x}}^{\mathrm{m}} = G_{\mathbb{x}}^{\mathrm{m}} + \epsilon = \beta\left((1-\mathscr{Y}) g_{\mathbb{x},\mathrm{m_M}} + \mathscr{Y} g_{\mathbb{x},\mathrm{m_P}}\right) + \epsilon \tag{S3.3.6a}$$

$$\phi_{\mathrm{x}}^{\mathrm{m}} = G_{\mathbb{x}}^{\mathrm{m}} + \epsilon = \beta\left((1-\mathscr{Y}) g_{\mathbb{x},\mathrm{m_M}} + \mathscr{Y} g_{\mathbb{x},\mathrm{m_P}}\right) + \epsilon \tag{S3.3.6b}$$

3.4 Marginal fitness effects

With these fitness functions, and the mapping of genotype to phenotype, we can now look at how the fitness of our focal individual changes with the changing genetic values of self and social partners. To get the marginal fitness effect of different actors, we can differentiate our fitness functions by the values of the various gene positions, evaluated when there is vanishingly small genetic variation, $g_{\mathrm{x,i}} = g_{\mathrm{y,i}} = g_{\mathrm{z,i}} = \overline{g}$.

For the general model, we notate the marginal change in the competitiveness in females with respect to the trait value by:

$$\frac{d\,w[\phi]/d\phi}{w[\phi]} = \sigma \tag{S3.4.1a}$$

And in males:

$$\frac{d\,v[\phi]/d\phi}{v[\phi]} = \tau \tag{S3.4.1b}$$

When a trait is sexually antagonistic, τ and σ will have opposite signs. The values of σ and τ will depend on the specific mapping of our trait into competitiveness. In our toy model:

$$\sigma = \left(\phi^{\mathrm{f}^*} - \alpha\phi^{\mathrm{f}}\right)\frac{1}{SD_{\mathrm{f}}^2} \tag{S3.4.2a}$$

$$\tau = \left(\phi^{\mathrm{m}^*} - \beta\phi^{\mathrm{m}}\right)\frac{1}{SD_{\mathrm{m}}^2} \tag{S3.4.2b}$$

The marginal fitness effects under haploidy, diploidy, and under offspring, maternal, and paternal control, can be seen in Table S20.

	$\partial W_{\mathrm{f_M}}$	$\partial W_{\mathrm{f_P}}$	$\partial W_{\mathrm{m_M}}$	$\partial W_{\mathrm{m_P}}$
$\partial x_{\mathrm{f_M}}$	$\alpha\sigma\mathscr{X}$	$\alpha\sigma\mathscr{X}$	0	0
$\partial y_{\mathrm{f_M}}$	$-\alpha\sigma\mathscr{X}\,a_{\mathrm{ff}}$	$-\alpha\sigma\mathscr{X}\,a_{\mathrm{ff}}$	$\alpha\sigma\mathscr{X}\left(\psi - a_{\mathrm{fm}}\right)$	$\alpha\sigma\mathscr{X}\left(\psi - a_{\mathrm{fm}}\right)$
$\partial x_{\mathrm{f_P}}$	$\alpha\sigma(1-\mathscr{X})$	$\alpha\sigma(1-\mathscr{X})$	0	0
$\partial y_{\mathrm{f_P}}$	$-\alpha\sigma(1-\mathscr{X})a_{\mathrm{ff}}$	$-\alpha\sigma(1-\mathscr{X})a_{\mathrm{ff}}$	$\alpha\sigma(1-\mathscr{X})\left(\psi - a_{\mathrm{fm}}\right)$	$\alpha\sigma(1-\mathscr{X})\left(\psi - a_{\mathrm{fm}}\right)$
$\partial x_{\mathrm{m_M}}$	0	0	$\beta\tau(1-\mathscr{Y})$	$\beta\tau(1-\mathscr{Y})$
$\partial y_{\mathrm{m_M}}$	0	0	$-\beta\tau(1-\mathscr{Y})a_{\mathrm{mm}}$	$-\beta\tau(1-\mathscr{Y})a_{\mathrm{mm}}$
$\partial x_{\mathrm{m_P}}$	0	0	$\beta\tau\mathscr{Y}$	$\beta\tau\mathscr{Y}$
$\partial y_{\mathrm{m_P}}$	0	0	$-\beta\tau\mathscr{Y}\,a_{\mathrm{mm}}$	$-\beta\tau\mathscr{Y}\,a_{\mathrm{mm}}$

Table S20: Marginal fitness effects for different genetic actors on self and social partners under diploidy.

3.5 Consanguinities

To calculate the consanguinities between different gene positions, we first write out recursions describing the probability of identity by descent (Bulmer, 1994). We then assume that the consanguinity coefficients have reached their quasi-equilibrium values which is a reasonable assumption if selection is weak (Gardner, West, and Wild, 2011).

We denote the probability of being IBD between two gene positions within an individual as $\rho^{i}_{g_1,g_2}$, and the probability of being IBD between two gene positions within a patch as $\rho^{p}_{g_1,g_2}$. We denote the probability of being IBD between a mother and an offspring $\rho^{Mot}_{g_1,g_2}$, where g_1 is the gene position in the mother, and g_2 the gene position in the offspring. We denote the probability of being IBD between a father and offspring $\rho^{Fat}_{g_1,g_2}$, where g_1 is the gene position in the father, and g_2 the gene position in the offspring.

The probability that a maternal-origin gene came from a maternal-origin gene is $\mathscr{A}$, and the probability that a paternal-origin gene came from a paternal-origin gene is $\mathscr{B}$. Such that for diploidy $\mathscr{A} = 1/2, \mathscr{B} = 1/2$, for arrhenotoky/male PGE $\mathscr{A} = 1/2, \mathscr{B} = 0$, and for paterothylotoky/female PGE $\mathscr{A} = 0, \mathscr{B} = 1/2$. The probability that an individual descended from a mating between patchmates is ψ. The values for ψ for our different life-cycles/mating systems are: $\psi = s$ for sib-mating, $\psi = (1 - d_{\mathrm{m}})$ for DMD, and $\psi = (1 - d_{\mathrm{m}})(1 - d_{\mathrm{f}})$ for DDM.

The consanguinities between these gene positions under these different genetic systems can be found in Table S21-S24.

3.5.1 Haploidy

Within individuals

$$\rho^{\mathrm{i}}_{\mathrm{f}} = \rho^{\mathrm{i}}_{\mathrm{m}} = 1 \tag{S3.5.1}$$

Between patchmates

$$\rho^{\mathrm{p}}_{\mathrm{f,f}} = (1-\mathscr{A})(\mathscr{A}\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi + (1-\mathscr{A})) + \mathscr{A}((1-\mathscr{A})\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi + \mathscr{A}) \tag{S3.5.2a}$$

$$\rho^{\mathrm{p}}_{\mathrm{f,m}} = (1-\mathscr{A})(\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi(1-\mathscr{B}) + \mathscr{B}) + \mathscr{A}(\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi\mathscr{B} + (1-\mathscr{B})) \tag{S3.5.2b}$$

$$\rho^{\mathrm{p}}_{\mathrm{m,m}} = \mathscr{B}(\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi(1-\mathscr{B}) + \mathscr{B}) + (1-\mathscr{B})(\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi\mathscr{B} + (1-\mathscr{B})) \tag{S3.5.2c}$$

Mothers and offspring

$$\rho^{\mathrm{Mot}}_{\mathrm{f,f}} = (1-\mathscr{A})\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi + \mathscr{A} \tag{S3.5.3a}$$

$$\rho^{\mathrm{Mot}}_{\mathrm{f,m}} = \rho^{\mathrm{p}}_{\mathrm{f,m}}\psi\mathscr{B} + (1-\mathscr{B}) \tag{S3.5.3b}$$

Fathers and offspring

$$\rho^{\mathrm{Fat}}_{\mathrm{m,f}} = \mathscr{A}\rho^{\mathrm{p}}_{\mathrm{f,m}}\psi + (1-\mathscr{A}) \tag{S3.5.4a}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m,m}} = \rho^{\mathrm{p}}_{\mathrm{f,m}}\psi(1-\mathscr{B}) + \mathscr{B} \tag{S3.5.4b}$$

3.5.2 Diploidy

Within individuals

$$\rho^{\mathrm{i}}_{\mathrm{f_M,f_M}} = \rho^{\mathrm{i}}_{\mathrm{f_P,f_P}} = \rho^{\mathrm{i}}_{\mathrm{m_M,m_M}} = \rho^{\mathrm{i}}_{\mathrm{m_P,m_P}} = 1 \tag{S3.5.5a}$$

$$\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}} = \psi\left(\mathscr{A}\left((1-\mathscr{B})\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}} + \mathscr{B}\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\right) + (1-\mathscr{A})\left((1-\mathscr{B})\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}} + \mathscr{B}\rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}\right)\right) \tag{S3.5.5b}$$

$$\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}} = \psi\left(\mathscr{A}\left((1-\mathscr{B})\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}} + \mathscr{B}\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\right) + (1-\mathscr{A})\left((1-\mathscr{B})\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}} + \mathscr{B}\rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}\right)\right) \tag{S3.5.5c}$$

Between patchmates

$$\rho^{p}_{\mathrm{f_M,f_M}} = \mathscr{A}\left(\mathscr{A}\rho^{\mathrm{i}}_{\mathrm{f_M,f_M}} + (1-\mathscr{A})\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}\right) + (1-\mathscr{A})\left(\mathscr{A}\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}} + (1-\mathscr{A})\rho^{\mathrm{i}}_{\mathrm{f_M,f_M}}\right) \tag{S3.5.6a}$$

$$\rho^{\mathrm{p}}_{\mathrm{f_M,f_P}} = \psi\left(\mathscr{A}\left((1-\mathscr{B})\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}} + \mathscr{B}\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\right) + (1-\mathscr{A})\left((1-\mathscr{B})\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}} + \mathscr{B}\rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}\right)\right) \tag{S3.5.6b}$$

$$\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}} = (1-\mathscr{A})\left(\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}\mathscr{A} + 1(1-\mathscr{A})\right) + \mathscr{A}\left(\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}(1-\mathscr{A}) + 1\mathscr{A}\right) \tag{S3.5.6c}$$

$$\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}} = \psi\left(\mathscr{A}\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}(1-\mathscr{B}) + \rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\mathscr{B}\right) + (1-\mathscr{A})\left(\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}(1-\mathscr{B}) + \rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}\mathscr{B}\right)\right) \tag{S3.5.6d}$$

$$\rho^{\mathrm{p}}_{\mathrm{f_P,f_P}} = (1-\mathscr{B})\left(\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}\mathscr{B} + 1(1-\mathscr{B})\right) + \mathscr{B}\left(\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}(1-\mathscr{B}) + 1\mathscr{B}\right) \tag{S3.5.6e}$$

$$\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}} = \psi\left((1-\mathscr{B})\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}\mathscr{A} + \rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}(1-\mathscr{A})\right) + \mathscr{B}\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\mathscr{A} + \rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}(1-\mathscr{A})\right)\right) \tag{S3.5.6f}$$

$$\rho^{\mathrm{p}}_{\mathrm{f_P,m_P}} = (1-\mathscr{B})\left(\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}\mathscr{B} + 1(1-\mathscr{B})\right) + \mathscr{B}\left(\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}(1-\mathscr{B}) + 1\mathscr{B}\right) \tag{S3.5.6g}$$

$$\rho^{\mathrm{p}}_{\mathrm{m_M,m_M}} = (1-\mathscr{A})\left(\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}\mathscr{A} + 1(1-\mathscr{A})\right) + \mathscr{A}\left(\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}(1-\mathscr{A}) + 1\mathscr{A}\right) \tag{S3.5.6h}$$

$$\rho^{\mathrm{p}}_{\mathrm{m_M,m_P}} = \psi\left(\mathscr{A}\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}(1-\mathscr{B}) + \rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\mathscr{B}\right) + (1-\mathscr{A})\left(\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}(1-\mathscr{B}) + \rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}\mathscr{B}\right)\right) \tag{S3.5.6i}$$

$$\rho^{\mathrm{p}}_{\mathrm{m_P,m_P}} = (1-\mathscr{B})\left(\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}\mathscr{B} + 1(1-\mathscr{B})\right) + \mathscr{B}\left(\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}(1-\mathscr{B}) + 1\mathscr{B}\right) \tag{S3.5.6j}$$

Mothers and offspring

$$\rho^{\mathrm{Mat}}_{\mathrm{f_M,f_M}} = \rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}(1-\mathscr{A}) + 1\mathscr{A} \tag{S3.5.7a}$$

$$\rho^{\mathrm{Mat}}_{\mathrm{f_M,f_P}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}(1-\mathscr{B}) + \rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\mathscr{B}\right) \tag{S3.5.7b}$$

$$\rho^{\mathrm{Mot}}_{\mathrm{f_M,m_M}} = \rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}(1-\mathscr{A}) + 1\mathscr{A} \tag{S3.5.7c}$$

$$\rho^{\mathrm{Mot}}_{\mathrm{f_M,m_P}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\mathscr{B} + \rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}(1-\mathscr{B})\right) \tag{S3.5.7d}$$

$$\rho^{\mathrm{Mot}}_{\mathrm{f_P,f_M}} = \rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}\mathscr{A} + 1(1-\mathscr{A}) \tag{S3.5.7e}$$

$$\rho^{\mathrm{Mot}}_{\mathrm{f_P,f_P}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}(1-\mathscr{B}) + \rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}\mathscr{B}\right) \tag{S3.5.7f}$$

$$\rho^{\mathrm{Mot}}_{\mathrm{f_P,m_M}} = \rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}\mathscr{A} + 1(1-\mathscr{A}) \tag{S3.5.7g}$$

$$\rho^{\mathrm{Mot}}_{\mathrm{f_P,m_P}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}(1-\mathscr{B}) + \rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}\mathscr{B}\right) \tag{S3.5.7h}$$

	Haploidy
$\rho^{\mathrm{p}}_{\mathrm{f,f}}$	$\frac{2\mathbb{L}^2-2\mathbb{L}+1}{2\mathbb{L}^2\psi-2\mathbb{L}\psi+1}$
$\rho^{\mathrm{p}}_{\mathrm{f,m}}$	$\frac{2\mathbb{L}^2-2\mathbb{L}+1}{2\mathbb{L}^2\psi-2\mathbb{L}\psi+1}$
$\rho^{\mathrm{p}}_{\mathrm{m,m}}$	$\frac{2\mathbb{L}^2-2\mathbb{L}+1}{2\mathbb{L}^2\psi-2\mathbb{L}\psi+1}$
$\rho^{\mathrm{Mot}}_{\mathrm{f,f}}$	$\frac{2\mathbb{L}^2\psi-\mathbb{L}\psi-\mathbb{L}+1}{2\mathbb{L}^2\psi-2\mathbb{L}\psi+1}$
$\rho^{\mathrm{Mot}}_{\mathrm{f,m}}$	$\frac{2\mathbb{L}^2\psi-\mathbb{L}\psi-\mathbb{L}+1}{2\mathbb{L}^2\psi-2\mathbb{L}\psi+1}$
$\rho^{\mathrm{Fat}}_{\mathrm{m,f}}$	$\frac{2\mathbb{L}^2\psi-3\mathbb{L}\psi+\mathbb{L}+\psi}{2\mathbb{L}^2\psi-2\mathbb{L}\psi+1}$
$\rho^{\mathrm{Fat}}_{\mathrm{m,m}}$	$\frac{2\mathbb{L}^2\psi-3\mathbb{L}\psi+\mathbb{L}+\psi}{2\mathbb{L}^2\psi-2\mathbb{L}\psi+1}$

Table S21: Consanguinities between different gene positions under haploidy. Under our fixed sib-mating scenario $\psi = s$, under our DMD scenario $\psi = 1 - d_{\mathrm{m}}$, and under the DDM scenario $\psi = (1 - d_{\mathrm{f}})(1 - d_{\mathrm{m}})$, $\mathbb{L}$ represents the proportion of paternal transmission.

Fathers and offspring

$$\rho^{\mathrm{Fat}}_{\mathrm{m_M,f_M}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}\mathscr{A} + \rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}(1-\mathscr{A})\right) \tag{S3.5.8a}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m_M,f_P}} = \rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}\mathscr{B} + 1\,(1-\mathscr{B}) \tag{S3.5.8b}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m_M,m_M}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}\mathscr{A} + \rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}(1-\mathscr{A})\right) \tag{S3.5.8c}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m_M,m_P}} = \rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}\mathscr{B} + 1\,(1-\mathscr{B}) \tag{S3.5.8d}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m_P,f_M}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\mathscr{A} + \rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}(1-\mathscr{A})\right) \tag{S3.5.8e}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m_P,f_P}} = \rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}(1-\mathscr{B}) + 1\mathscr{B} \tag{S3.5.8f}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m_P,m_M}} = \psi\left(\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}\mathscr{A} + \rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}(1-\mathscr{A})\right) \tag{S3.5.8g}$$

$$\rho^{\mathrm{Fat}}_{\mathrm{m_P,m_P}} = \rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}(1-\mathscr{B}) + 1\mathscr{B} \tag{S3.5.8h}$$

	Diploidy	Male PGE	Female MGE
$\rho^{\mathrm{i}}_{\mathrm{f_M,f_P}}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$
$\rho^{\mathrm{i}}_{\mathrm{m_M,m_P}}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$
$\rho^{\mathrm{p}}_{\mathrm{f_M,f_M}}$	$\frac{\psi-2}{3\psi-4}$	$\frac{2-\psi}{4-3\psi}$	1
$\rho^{\mathrm{p}}_{\mathrm{f_M,f_P}}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$
$\rho^{\mathrm{p}}_{\mathrm{f_M,m_M}}$	$\frac{\psi-2}{3\psi-4}$	$\frac{2-\psi}{4-3\psi}$	1
$\rho^{\mathrm{p}}_{\mathrm{f_M,m_P}}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$
$\rho^{\mathrm{p}}_{\mathrm{f_P,f_P}}$	$\frac{\psi-2}{3\psi-4}$	1	$\frac{2-\psi}{4-3\psi}$
$\rho^{\mathrm{p}}_{\mathrm{f_P,m_M}}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$
$\rho^{\mathrm{p}}_{\mathrm{f_P,m_P}}$	$\frac{\psi-2}{3\psi-4}$	1	$\frac{2-\psi}{4-3\psi}$
$\rho^{\mathrm{p}}_{\mathrm{m_M,m_M}}$	$\frac{\psi-2}{3\psi-4}$	$\frac{2-\psi}{4-3\psi}$	1
$\rho^{\mathrm{p}}_{\mathrm{m_M,m_P}}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$	$\frac{\psi}{4-3\psi}$
$\rho^{\mathrm{p}}_{\mathrm{m_P,m_P}}$	$\frac{\psi-2}{3\psi-4}$	1	$\frac{2-\psi}{4-3\psi}$

Table S22: Consanguinities between different gene positions in offspring under our different genetical systems. Under our fixed sib-mating scenario $\psi = s$, under our labile sib-mating scenario $\psi = 1-d_m$, and under our viscous population $\psi = (1-d_f)(1-d_m)$. Here we have assumed that in both the male PGE and female MGE scenarios $\mathbb{L} = 0$.

	Diploidy	Male PGE	Female PGE
$\rho^{\mathrm{Mot}}_{\mathrm{f_M,f_M}}$	$\frac{\psi-2}{3\psi-4}$	$\frac{\psi-2}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$
$\rho^{\mathrm{Mot}}_{\mathrm{f_M,f_P}}$	$-\frac{\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$
$\rho^{\mathrm{Mot}}_{\mathrm{f_M,m_M}}$	$\frac{\psi-2}{3\psi-4}$	$\frac{\psi-2}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$
$\rho^{\mathrm{Mot}}_{\mathrm{f_M,m_P}}$	$-\frac{\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$
$\rho^{\mathrm{Mot}}_{\mathrm{f_P,f_M}}$	$\frac{\psi-2}{3\psi-4}$	$\frac{\psi-2}{3\psi-4}$	1
$\rho^{\mathrm{Mot}}_{\mathrm{f_P,f_P}}$	$-\frac{\psi}{3\psi-4}$	$-\frac{\psi^2}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$
$\rho^{\mathrm{Mot}}_{\mathrm{f_P,m_M}}$	$\frac{\psi-2}{3\psi-4}$	$\frac{\psi-2}{3\psi-4}$	1
$\rho^{\mathrm{Mot}}_{\mathrm{f_P,m_P}}$	$-\frac{\psi}{3\psi-4}$	$-\frac{\psi^2}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$

Table S23: Consanguinities between different gene positions in mothers and offspring under our different genetical systems. Under our fixed sib-mating scenario $\psi = s$, under our labile sib-mating scenario $\psi = 1-d_{\mathrm{m}}$, and under our viscous population $\psi = (1-d_{\mathrm{f}})(1-d_{\mathrm{m}})$. Here we have assumed that in both the male PGE and female MGE scenarios $\mathbb{L} = 0$.

	Diploidy	Male PGE	Female PGE
$\rho^{\text{Fat}}_{\text{m}_\text{M},\text{f}_\text{M}}$	$-\frac{\psi}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$	$-\frac{\psi^2}{3\psi-4}$
$\rho^{\text{Fat}}_{\text{m}_\text{M},\text{f}_\text{P}}$	$\frac{\psi-2}{3\psi-4}$	1	$\frac{\psi-2}{3\psi-4}$
$\rho^{\text{Fat}}_{\text{m}_\text{M},\text{m}_\text{M}}$	$-\frac{\psi}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$	$-\frac{\psi^2}{3\psi-4}$
$\rho^{\text{Fat}}_{\text{m}_\text{M},\text{m}_\text{P}}$	$\frac{\psi-2}{3\psi-4}$	1	$\frac{\psi-2}{3\psi-4}$
$\rho^{\text{Fat}}_{\text{m}_\text{P},\text{f}_\text{M}}$	$-\frac{\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$
$\rho^{\text{Fat}}_{\text{m}_\text{P},\text{f}_\text{P}}$	$\frac{\psi-2}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$	$\frac{\psi-2}{3\psi-4}$
$\rho^{\text{Fat}}_{\text{m}_\text{P},\text{m}_\text{M}}$	$-\frac{\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$	$\frac{(\psi-2)\psi}{3\psi-4}$
$\rho^{\text{Fat}}_{\text{m}_\text{P},\text{m}_\text{P}}$	$\frac{\psi-2}{3\psi-4}$	$-\frac{\psi}{3\psi-4}$	$\frac{\psi-2}{3\psi-4}$

Table S24: Consanguinities between different gene positions in fathers and offspring under our different genetical systems. Under our fixed sib-mating scenario $\psi = s$, under our labile sib-mating scenario $\psi = 1 - d_\text{m}$, and under our viscous population $\psi = (1 - d_\text{f})(1 - d_\text{m})$. Here we have assumed that in both the male PGE and female MGE scenarios $\mathbb{L} = 0$.

3.6 Reproductive value

Reproductive value provides a measure of an individual gene's, or a class of genes', expected asymptotic contribution to future generations. We denote the reproductive value of class i by c_i. This can be calculated by writing a gene flow matrix for a monomorphic population. This gene flow matrix is essentially describing a Markov process of a gene's state, going back in time (Grafen, 2006). We notate the probability that a gene in class i came from class j in the previous timestep by $\pi_{\text{i,j}}$. If we write this gene flow matrix out, then the dominant left eigenvector of this matrix gives us the class reproductive value weightings for our different gene positions (Taylor, 1990; Taylor, 1996). Solving for our different genetic systems we get the class reproductive values seen in Table S25.

$$\begin{pmatrix} c_{\text{f}_\text{M}} & c_{\text{f}_\text{P}} & c_{\text{m}_\text{M}} & c_{\text{m}_\text{P}} \end{pmatrix} = \begin{pmatrix} c_{\text{f}_\text{M}} & c_{\text{f}_\text{P}} & c_{\text{m}_\text{M}} & c_{\text{m}_\text{P}} \end{pmatrix} \begin{pmatrix} \pi_{\text{f}_\text{M},\text{f}_\text{M}} & \pi_{\text{f}_\text{M},\text{f}_\text{P}} & \pi_{\text{f}_\text{M},\text{m}_\text{M}} & \pi_{\text{f}_\text{M},\text{m}_\text{P}} \\ \pi_{\text{f}_\text{P},\text{f}_\text{M}} & \pi_{\text{f}_\text{P},\text{f}_\text{P}} & \pi_{\text{f}_\text{P},\text{m}_\text{M}} & \pi_{\text{f}_\text{P},\text{m}_\text{P}} \\ \pi_{\text{m}_\text{M},\text{f}_\text{M}} & \pi_{\text{m}_\text{M},\text{f}_\text{P}} & \pi_{\text{m}_\text{M},\text{m}_\text{M}} & \pi_{\text{m}_\text{M},\text{m}_\text{P}} \\ \pi_{\text{m}_\text{P},\text{f}_\text{M}} & \pi_{\text{m}_\text{P},\text{f}_\text{P}} & \pi_{\text{m}_\text{P},\text{m}_\text{M}} & \pi_{\text{m}_\text{P},\text{m}_\text{P}} \end{pmatrix} \tag{S3.6.1}$$

	$c_{\mathrm{f_M}}$	$c_{\mathrm{f_P}}$	$c_{\mathrm{m_M}}$	$c_{\mathrm{m_P}}$	c_{i}
Haploidy	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	N/A
Diploidy	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{3}$	N/A	N/A
Arrhenotoky	N/A	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{3}$	N/A
Paterothylotoky	$\frac{\mathbb{L}-1}{2\mathbb{L}-3}$	$\frac{\mathbb{L}-1}{2\mathbb{L}-3}$	$\frac{\mathbb{L}-1}{2\mathbb{L}-3}$	$\frac{\mathbb{L}}{3-2\mathbb{L}}$	N/A
Male PGE	$\frac{\mathbb{L}}{3-2\mathbb{L}}$	$\frac{\mathbb{L}-1}{2\mathbb{L}-3}$	$\frac{\mathbb{L}-1}{2\mathbb{L}-3}$	$\frac{\mathbb{L}-1}{2\mathbb{L}-3}$	N/A
Female PGE	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	N/A
Icerya system	$\frac{\varphi-1}{2\varphi-3}$	$\frac{\varphi-1}{2\varphi-3}$	$\frac{\varphi-1}{2\varphi-3}$	0	$\frac{\varphi}{3-2\varphi}$

Table S25: Class reproductive values for the gene positions in various genetic systems. Where $c_{\mathrm{f_M}}$ are female maternal-origin genes, $c_{\mathrm{f_P}}$ are female paternal-origin genes, $c_{\mathrm{m_M}}$ are male maternal-origin genes, $c_{\mathrm{m_P}}$ are male paternal-origin genes, and c_{i} is the infectious male lineage

3.7 Condition for increase

Putting the reproductive values, consanguinities, and marginal fitness effects together, we can write the condition for increase in our diploid systems with offspring control as so:

$$\frac{\tau}{\sigma} < \frac{\alpha}{\beta}\left(\frac{\mathscr{X}\, w_{\mathrm{f_M}} + (1-\mathscr{X})\, w_{\mathrm{f_P}}}{(1-\mathscr{Y})\, w_{\mathrm{m_M}} + \mathscr{Y}\, w_{\mathrm{m_P}})}\right) \tag{S3.7.1}$$

Where $w_{\mathrm{f_M}}$ is the inclusive fitness (IF) effect to a female maternal-origin copy of an increase in the trait value, $w_{\mathrm{f_P}}$ is the IF effect to a female paternal-origin gene copy of a change in the trait value, $w_{\mathrm{m_M}}$ is the IF effect to a male maternal-origin gene copy of a change in the trait value, and $w_{\mathrm{m_P}}$ is the IF effect to a male paternal-origin gene copy of a change in the trait value. The α,β,$\mathscr{X}$, and $\mathscr{Y}$, scale the relative phenotypic effects of these gene copies on males and females. Where the inclusive fitness effects experienced by our different genetic actors is:

$$\begin{aligned} w_{\mathrm{f_M}} =& c_{\mathrm{f_P}}\left(\rho^{\mathrm{i}}_{\mathrm{f_M},\mathrm{f_P}} - a_{\mathrm{ff}}\rho^{\mathrm{p}}_{\mathrm{f_M},\mathrm{f_P}}\right) + c_{\mathrm{f_M}}\left(1 - a_{\mathrm{ff}}\rho^{\mathrm{p}}_{\mathrm{f_M},\mathrm{f_M}}\right) \\ &+ c_{\mathrm{m_P}}\left(\psi - a_{\mathrm{mf}}\right)\rho^{\mathrm{p}}_{\mathrm{f_M},\mathrm{m_P}} + c_{\mathrm{m_M}}\left(\psi - a_{\mathrm{mf}}\right)\rho^{\mathrm{p}}_{\mathrm{f_M},\mathrm{m_M}} \end{aligned} \tag{S3.7.2a}$$

$$\begin{aligned} w_{\mathrm{f_P}} =& c_{\mathrm{f_M}}\left(\rho^{\mathrm{i}}_{\mathrm{f_M},\mathrm{f_P}} - a_{\mathrm{ff}}\rho^{\mathrm{p}}_{\mathrm{f_M},\mathrm{f_P}}\right) + c_{\mathrm{f_P}}\left(1 - a_{\mathrm{ff}}\rho^{\mathrm{p}}_{\mathrm{f_P},\mathrm{f_P}}\right) \\ &+ c_{\mathrm{m_P}}\left(\psi - a_{\mathrm{mf}}\right)\rho^{\mathrm{p}}_{\mathrm{f_P},\mathrm{m_P}} + c_{\mathrm{m_M}}\left(\psi - a_{\mathrm{mf}}\right)\rho^{\mathrm{p}}_{\mathrm{f_P},\mathrm{m_M}} \end{aligned} \tag{S3.7.2b}$$

$$w_{\mathrm{m_M}} = c_{\mathrm{m_P}}\left(\rho^{\mathrm{i}}_{\mathrm{m_M},\mathrm{m_P}} - a_{\mathrm{mm}}\rho^{\mathrm{p}}_{\mathrm{m_M},\mathrm{m_P}}\right) + c_{\mathrm{m_M}}\left(1 - a_{\mathrm{mm}}\rho^{\mathrm{p}}_{\mathrm{m_M},\mathrm{m_M}}\right) \tag{S3.7.2c}$$

$$w_{\mathrm{m_P}} = c_{\mathrm{m_M}}\left(\rho^{\mathrm{i}}_{\mathrm{m_M},\mathrm{m_P}} - a_{\mathrm{mm}}\rho^{\mathrm{p}}_{\mathrm{m_M},\mathrm{m_P}}\right) + c_{\mathrm{m_P}}\left(1 - a_{\mathrm{mm}}\rho^{\mathrm{p}}_{\mathrm{m_P},\mathrm{m_P}}\right) \tag{S3.7.2d}$$

3.8 Trait Optima

To find the optimal trait level in our toy model, we set our condition for increase to zero, and then solve for ϕ^*. In the main text this is referred to as z^*.

	Trait optima ϕ^*
Haploidy	$\frac{\hat{z}_f \mathrm{SD}_m^2\left((2(\mathbb{L}-1)\mathbb{L}+1)(d_f-2)d_f(\mathbb{L}(s-1)+1)+2\mathbb{L}(\mathbb{L}-1)^2(s-1)\right)+\mathbb{L}(s-1)\mathrm{SD}_f^2\hat{z}_m}{(2(\mathbb{L}-1)\mathbb{L}+1)(d_f-2)d_f\mathrm{SD}_m^2(\mathbb{L}(s-1)+1)+\mathbb{L}(s-1)\left(\mathrm{SD}_f^2+2(\mathbb{L}-1)^2\mathrm{SD}_m^2\right)}$
Arrhenotoky/MPGE	$\frac{\alpha\hat{z}_f\mathrm{SD}_m^2\left((d_f-2)d_f((s-2)s(2\mathscr{X}-1)+2(\mathscr{X}-2))-2(s-1)\mathscr{X}\right)-\beta(s-1)\mathrm{SD}_f^2\hat{z}_m(2(s-2)\mathscr{Y}-s+4)}{\alpha^2\mathrm{SD}_m^2\left((d_f-2)d_f((s-2)s(2\mathscr{X}-1)+2(\mathscr{X}-2))-2(s-1)\mathscr{X}\right)-\beta^2(s-1)\mathrm{SD}_f^2(2(s-2)\mathscr{Y}-s+4)}$
Paterothylotoky/FMGE	$\frac{\alpha\hat{z}_f\mathrm{SD}_m^2\left((d_f-2)d_f\left(2(s-1)^2\mathscr{X}-s-2\right)+2(s-1)(\mathscr{X}-1)\right)-2\beta(s-1)\mathrm{SD}_f^2\hat{z}_m(s(\mathscr{Y}-1)+2)}{\alpha^2\mathrm{SD}_m^2\left((d_f-2)d_f\left(2(s-1)^2\mathscr{X}-s-2\right)+2(s-1)(\mathscr{X}-1)\right)-2\beta^2(s-1)\mathrm{SD}_f^2(s(\mathscr{Y}-1)+2)}$
Diploidy	$\frac{\alpha\hat{z}_f\mathrm{SD}_m^2\left((s+1)(d_f-2)d_f+s-1\right)+2\beta(s-1)\mathrm{SD}_f^2\hat{z}_m}{\alpha^2\mathrm{SD}_m^2\left((s+1)(d_f-2)d_f+s-1\right)+2\beta^2(s-1)\mathrm{SD}_f^2}$

Table S26: Optima for a normally distributed trait when under offspring control, and there is sib-mating

	Trait optima ϕ^*
Haploidy	$\frac{(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2(\mathbb{L}(s-1)+1)+\mathbb{L}(s-1)\mathrm{SD}_f^2\hat{z}_m}{(d_f-2)d_f\mathrm{SD}_m^2(\mathbb{L}(s-1)+1)+\mathbb{L}(s-1)\mathrm{SD}_f^2}$
Arrhenotoky/MPGE	$\frac{(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2(2(s-1)s\mathscr{X}-s-2)+(s-2)(s-1)\mathrm{SD}_f^2\hat{z}_m}{\alpha(d_f-2)d_f\mathrm{SD}_m^2(2(s-1)s\mathscr{X}-s-2)+\alpha(s-2)(s-1)\mathrm{SD}_f^2}$
Paterothylotoky/FMGE	$\frac{s(2s-5)(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2+2(s-2)(s-1)\mathrm{SD}_f^2\hat{z}_m}{\alpha s(2s-5)(d_f-2)d_f\mathrm{SD}_m^2+2\alpha(s-2)(s-1)\mathrm{SD}_f^2}$
Diploidy	$\frac{(s+1)(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2+(s-1)\mathrm{SD}_f^2\hat{z}_m}{\alpha(s+1)(d_f-2)d_f\mathrm{SD}_m^2+\alpha(s-1)\mathrm{SD}_f^2}$

Table S27: Optima for a normally distributed trait when under maternal control, and there is sib-mating

	Trait optima ϕ^*
Haploidy	$\frac{(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2(\mathbb{L}(s-1)+1)+\mathbb{L}(s-1)\mathrm{SD}_f^2\hat{z}_m}{(d_f-2)d_f\mathrm{SD}_m^2(\mathbb{L}(s-1)+1)+\mathbb{L}(s-1)\mathrm{SD}_f^2}$
Arrhenotoky/MPGE	$\frac{((s-2)s+4)(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2+(s-1)s\mathrm{SD}_f^2\hat{z}_m}{\beta((s-2)s+4)(d_f-2)d_f\mathrm{SD}_m^2+\beta(s-1)s\mathrm{SD}_f^2}$
Paterothylotoky/FMGE	$\frac{(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2(s((s-1)s(2\mathscr{Y}-1)-1)-2)+(s-1)\mathrm{SD}_f^2\hat{z}_m((s-1)s(2\mathscr{Y}-1)-2)}{\beta\left((d_f-2)d_f\mathrm{SD}_m^2(s((s-1)s(2\mathscr{Y}-1)-1)-2)+(s-1)\mathrm{SD}_f^2((s-1)s(2\mathscr{Y}-1)-2)\right)}$
Diploidy	$\frac{(s+1)(d_f-2)d_f\hat{z}_f\mathrm{SD}_m^2+(s-1)\mathrm{SD}_f^2\hat{z}_m}{\beta(s+1)(d_f-2)d_f\mathrm{SD}_m^2+\beta(s-1)\mathrm{SD}_f^2}$

Table S28: Optima for a normally distributed trait when under paternal control, and there is sib-mating

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