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A gene's-eye view of sexual antagonism

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Females and males may face different selection pressures. Accordingly, alleles that confer a benefit for one sex often incur a cost for the other. Classic evolutionary theory holds that the X chromosome, whose sex-biased transmission sees it spending more time in females, should value females more than males, whereas autosomes, whose transmission is unbiased, should value both sexes equally. However, recent mathematical and empirical studies indicate that male-beneficial alleles may be more favoured by the X chromosome than by autosomes. Here we develop a gene's-eye-view approach that reconciles the classic view with these recent discordant results, by separating a gene's valuation of female versus male fitness from its ability to induce fitness effects in either sex. We use this framework to generate new comparative predictions for sexually antagonistic evolution in relation to dosage compensation, sex-specific mortality and assortative mating, revealing how molecular mechanisms, ecology and demography drive variation in masculinization versus feminization across the genome.

1. Introduction

New genomic approaches paint an increasingly vivid picture of the extent of sexual antagonism across the genome, identifying specific loci at which fixed or segregating alleles increase the fitness of their female carriers while decreasing the fitness of their male carriers, or *vice versa* [1,2]. The overall action of natural selection on such alleles depends on how the benefits enjoyed by one sex are balanced by costs incurred by the other, and since different parts of the genome are expected to place different values on the fitness of females and males this is predicted to lead to an intragenomic conflict of interest with respect to sexually antagonistic traits [3–8]. Conventionally, the X chromosome has been viewed as placing twice as much value on the fitness of females as it does the fitness of males, on account of it spending twice as much evolutionary time in the bodies of females than in the bodies of males, whereas the autosomes have been viewed as placing equal value on each sex on account of them spending an equal portion of evolutionary time being carried by males and females [9–16]. Accordingly, the X chromosome and the autosomes have been regarded as being locked in an intragenomic conflict, in which the former favours phenotypes that are relatively closer to the female optimum and the latter favour phenotypes that are relatively closer to the male optimum [7,8].

However, this view has been challenged by recent mathematical analysis which has indicated that male-beneficial alleles may be more—not less—readily favoured at X-linked loci than at autosomal loci [17,18]. Specifically, this work suggests that while the condition for an autosomal sexually antagonistic allele to invade from rarity is the same irrespective of which sex obtains the benefit, the condition for an X-linked sexually antagonistic allele to invade from rarity is almost always less stringent when males obtain the benefit and females suffer the cost than when females obtain the benefit and males suffer the cost, where benefits and costs are defined according to how the allele's homozygous and hemizygous genotype fitnesses differ from those of the resident allele. Empirical support for masculinized X chromosomes has been found in humans [19], aphids [20] and stalk-eyed flies [21]. These surprising results have been

interpreted as directly contradicting evolutionary biologists' classic understanding of intragenomic conflict [17].

Here, we show that these results are, in fact, fully consistent with the classic view, by taking an explicit gene's-eye-view approach that considers the inclusive-fitness interests of a single gene rather than a whole genotype [8,22]. By partitioning a gene's 'agenda' (valuation of female versus male fitness) from its 'power' (ability to exert fitness effects upon females versus males), we show that the classic view concerns a gene's agenda and the discordant results emerge from sex differences in power. We use this framework to generate new comparative predictions for sexually antagonistic evolution in relation to dosage compensation, sex-specific mortality and assortative mating, revealing how molecular mechanisms, ecology and demography drive variation in masculinization and feminization across the genome.

2. Results

We begin by recapping the puzzling mathematical results that have motivated our analysis. Traditionally, X-linked genes, for which there is a double-dose in females in comparison with males, have been viewed as placing twice as great a value upon the fitness of females as that of males, on account of their spending twice as much evolutionary time in the bodies of females as opposed to males [8,23–26]. However, specific population-genetic models of sexual antagonism have cast doubt on this principle. If a mutant allele confers a fitness benefit S to one sex and confers a fitness cost T to the other sex when in its homozygous/hemizygous form, then in the absence of dominance effects the condition for natural selection to favour invasion of the allele from rarity turns out to be $S > T$ for both X-linked and autosomal genes, irrespective of which sex obtains the benefit [15,17]. That is, the X chromosome does not appear to be particularly biased towards female-beneficial alleles versus their male-beneficial counterparts.

The situation is more complex in the presence of dominance effects. Rice [15] showed that whereas the condition for a sexually antagonistic allele to invade from rarity on an autosome remains $S > T$, the corresponding condition for the X chromosome is $S > 2hT$ if it is male-beneficial and $S > T/(2h)$ if it is female-beneficial, where h is the dominance coefficient. Accordingly, if the degree of dominance is the same for both male-beneficial and female-beneficial alleles, then the X chromosome is expected to become masculinized if mutations are typically recessive, and feminized if they are typically dominant [15]. However, consideration of the curvature of the fitness landscape in the interval between the male and female optima has suggested that dominance coefficients will typically be reversed in comparisons of beneficial versus deleterious alleles, such that the heterozygote fitnesses are given by $(1-h)S$ and hT, respectively [17,27]. This yields the conditions $(1-h)S > hT$ for autosomal alleles, $S > 2hT$ for male-beneficial X-linked alleles, and $S > T/(2(1-h))$ for female-beneficial X-linked alleles (note that these results are exact in the limit of weak selection; expressions for stronger selection are provided in the electronic supplementary material). Accordingly, over almost all dominance coefficients, the X chromosome promotes male-beneficial alleles over their female-beneficial counterparts [17].

How can these results be reconciled with the view that X-linked genes place greater value upon the fitness of females than that of males? The key is to take an explicitly genic, rather than genotypic, approach. In the absence of dominance, the marginal fitness effect that a single gene has in the context of the sex in which it is advantageous is $\sigma = S/2$ if this sex is diploid at the focal locus (which is the case for both females and males if the gene is autosomal, and is the case for females if the gene is X-linked) and is $\sigma = S$ if this sex is haploid at the focal locus (which is the case for males if the gene is X-linked). Likewise, the fitness effect that the gene has in the context of the sex in which it is disadvantageous is $\tau = T/2$ if this sex is diploid at the focal locus and is $\tau = T$ if this sex is haploid at the focal locus. Accordingly, if autosomal genes place equal value on the fitness of females and males, then the condition for invasion of a mutant allele is $\sigma > \tau$, which is equivalent to $S > T$, in agreement with the above analysis. And if X-linked genes place twice the value on the fitness of females that they do males, then the condition for invasion of a mutant allele is $2\sigma > \tau$ when the allele benefits females and $\sigma > 2\tau$ when the allele benefits males, which in both cases is equivalent to $S > T$, again in agreement with the above analysis. The same logic can be used to recover the results for the dominance and reversal-of-dominance scenarios (see electronic supplementary material for details).

In other words, the X-masculinization results are entirely in line with the classic view of how X chromosomes and autosomes value female and male fitness. This equivalence has, until now, been obscured by a focus on whole genotypes and genotypic fitnesses, rather than on single genes and the fitness effects for which they—and they alone—are responsible. Specifically, X-linked genes do place an extra twofold weighting on their fitness effects in females, as a consequence of such genes spending a greater fraction of their evolutionary time in females. In this sense, X-linked genes have a female-biased agenda. However, since a gene's impact upon the phenotype may become diluted as it moves from a haploid to a diploid setting [28,29], the relative power of an X-linked gene to induce fitness effects may be lower in a female carrier than in a male. This power asymmetry creates a bias towards male-beneficial strategies that may counteract, and even overturn, the X-linked gene's more fundamental female-biased agenda.

More generally, the inclusive-fitness consequences of a gene's actions may be partitioned into three basic components: fitness effects, reproductive value and relatedness [8,30]. The fitness effects are the quantities that vary as a consequence of the gene adopting alternative strategies and represent the gene's power to shape the world. Reproductive value and relatedness together provide a currency conversion that translates these fitness effects into the gene's own inclusive-fitness valuation of any given strategy [31,32], and these dictate its agenda. The particular biological circumstances in which a gene finds itself will modulate all three components of inclusive fitness, and by investigating the modulating effects of molecular mechanisms, ecology and demography we are better able to predict and understand the relative feminization versus masculinization of sex chromosomes across different loci, populations and species (figure 1).

(a) Fitness effects

First, we consider those factors that shape the magnitude of costs and benefits in the two sexes (figure 2). One such factor is dosage compensation. It is often assumed that the

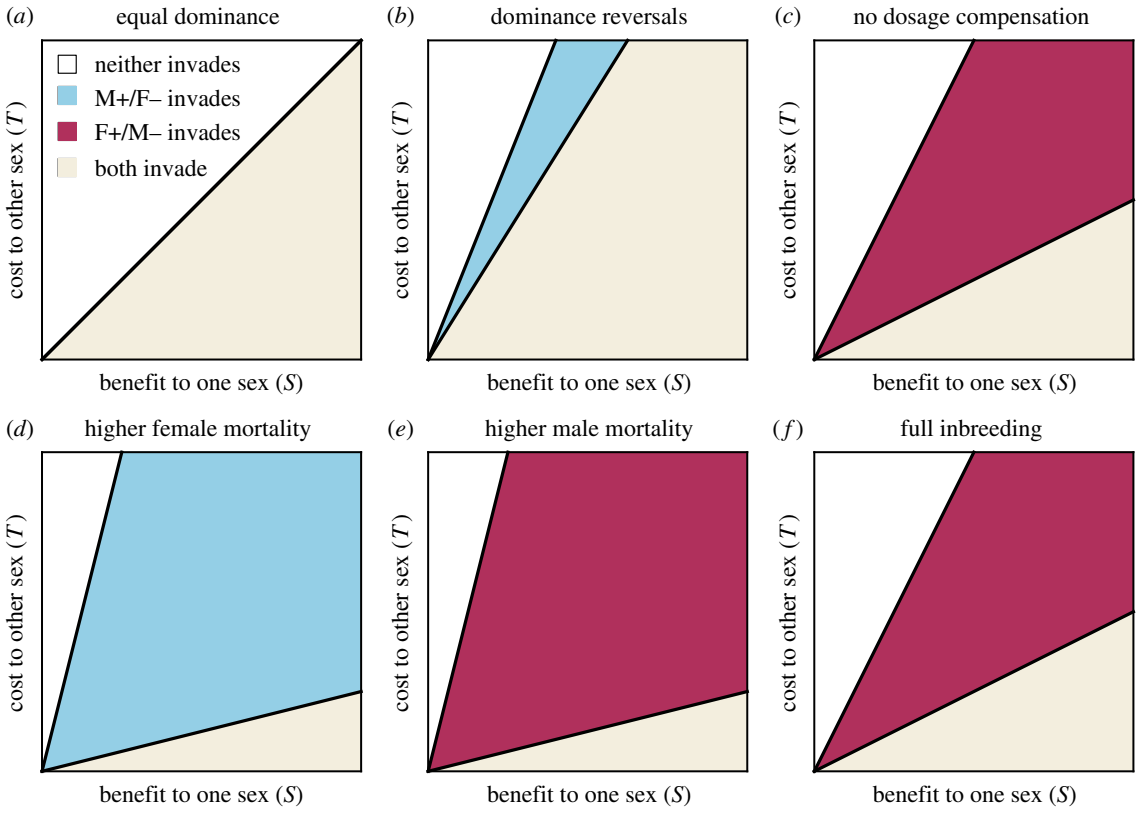

Figure 1. Comparison of invasion conditions for sexually antagonistic alleles on the X chromosome under weak selection. Shaded areas beneath the line indicate where an allele can invade from rarity given a whole-genotype fitness cost (T) to one sex and benefit to the other (S). (*a*) With equal dominance in the two sexes ($h = 1/2$), (*b*) with reversals of dominance between the two sexes ($h = 1/5$), (*c*) with equal dominance and no dosage compensation ($h = 1/2$, $\gamma = 0$), (*d*) with higher female mortality ($x = 3/2, y = 3$), (*e*) with higher male mortality ($x = 3, y = 3/2$), (*f*) with full inbreeding ($\phi = 1$). (Online version in colour.)

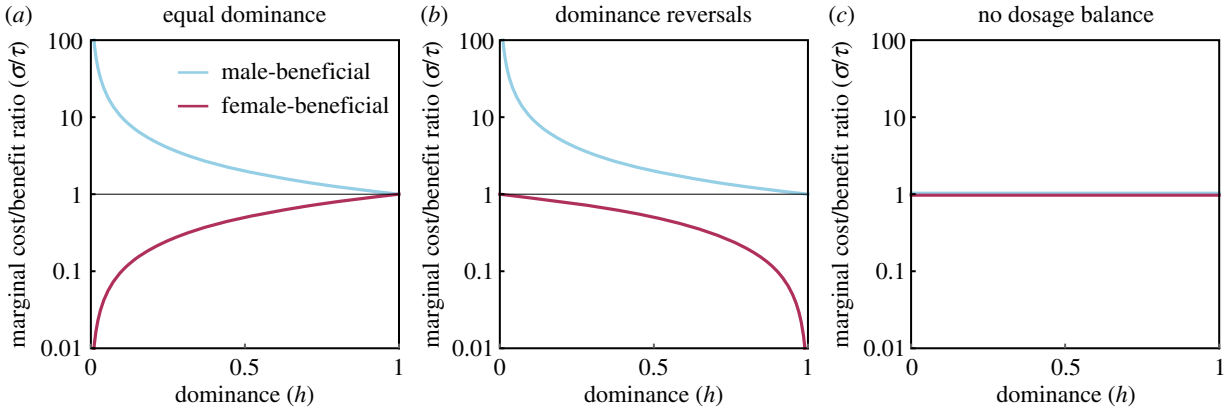

Figure 2. Ratios of marginal 'gene-level' fitness costs and benefits (σ/τ) for sexually antagonistic alleles on the X chromosome under differing assumptions about dominance and dosage balance. Note the ratios are plotted on a logarithmic scale. (*a*) Equal dominance in the two sexes. (*b*) Reversals of dominance between the two sexes. (*c*) Equal dominance in the two sexes, but no dosage balance. (Online version in colour.)

phenotypes of a mutant homozygote and hemizygote are comparable [33], and consequently that the fitness effect of a single mutant X-linked allele is greater in males. This assumption is often justified by pointing to the existence of mechanisms that scale gene expression to maintain a constant X : autosome ratio of gene products across the two sexes, despite variation in the number of X chromosomes [33]. However, it is now clear that dosage-balancing mechanisms are not universal and vary across species, genes and developmental stages [34–36]. We explore the effects of this variation by introducing a parameter γ that scales the mutant fitness effect in the heterogametic sex between the extremes of no dosage compensation ($\gamma = 0$)—and thus comparable to the heterozygote—and full dosage compensation ($\gamma = 1$)—and thus comparable to the homozygote. Under additivity, the ratio of the marginal costs and benefits $\sigma : \tau$ in the two sexes is $(1 + \gamma)S : T$ when male-beneficial and $S : (1 + \gamma)T$ when female-beneficial. Accordingly, in the limit of full dosage compensation ($\gamma = 1$), the marginal fitness effect in males is double that in females. But, as dosage balance decreases ($\gamma < 1$), then the marginal fitness effect in males is reduced, making conditions for invasion of female-beneficial alleles less stringent and thus driving greater feminization. Other factors may also modulate the marginal fitness effects in a

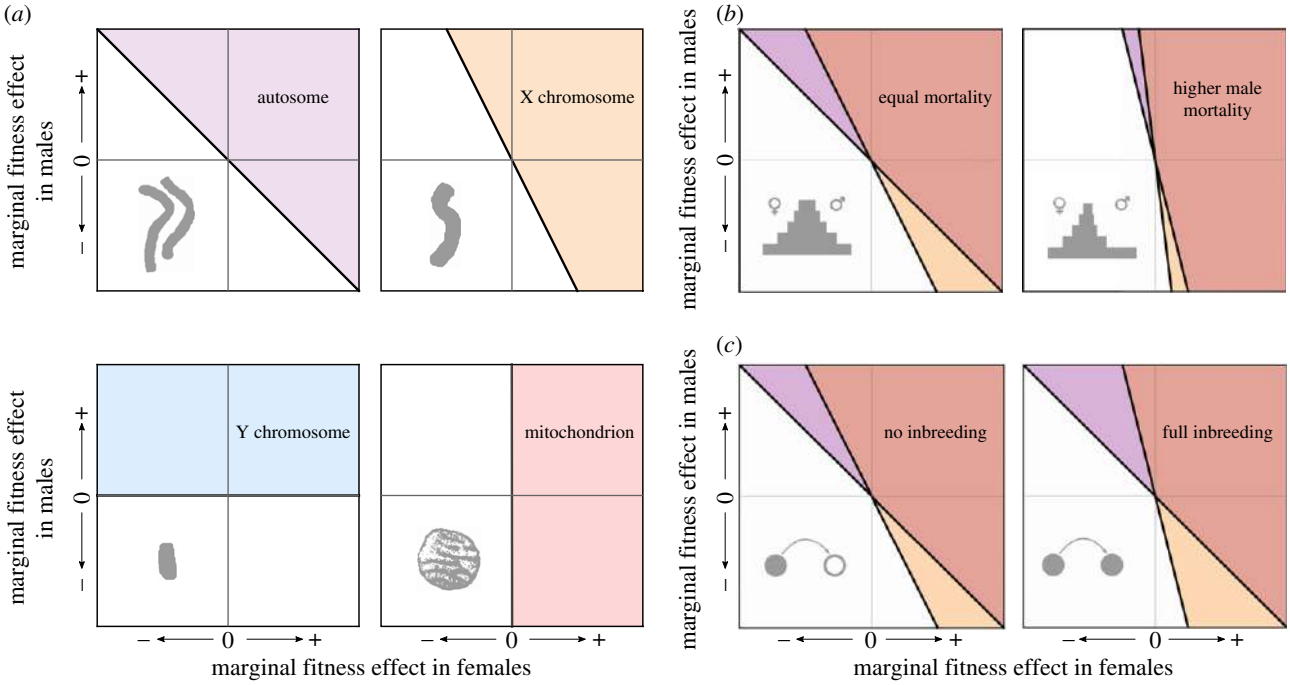

Figure 3. The invasion space for new mutations, illustrating the weightings put on marginal gene-level fitness effects in females and in males. (*a*) Across different portions of the genome (for simplicity, we assume here that mitochondria have exclusively maternal transmission), (*b*) for survival effects on autosomes and X chromosomes with equal mortality ($x = 2, y = 2$) and higher male mortality ($x = 3, y = 3/2$), (*c*) for autosomes and X chromosomes with full outbreeding ($\phi = 0$) and full inbreeding ($\phi = 1$). (Online version in colour.)

similar fashion, for instance if selection occurs predominantly in the haploid rather than diploid state of the life cycle [37], if loci are expressed in a parent-of-origin-specific manner, or if there remain functional homologues on the Y chromosome (see electronic supplementary material).

(b) Reproductive value

Second, we consider reproductive value (figure 3). The traditional view is that a twofold weighting of female fitness effects arises because twice as many of the X-linked genes of future generations will descend from females, as compared with males, in the present generation [23,24] and, accordingly, selective effects in females are expected to shape future generations twice as strongly as are selective effects in males. However, this need not be the case in populations with overlapping generations, in which sex-biases in the stable age distribution may have a modulating effect on the reproductive values of females and males with respect to autosomal and X-chromosomal genes ([38,39]; figure 3*b*). Specifically, the ratio of female to male class reproductive values in an age-structured population is $x : y$ for autosomal genes and $2x : y$ for X-linked genes, where x is the average age of a newborn's mother and y is the average age of a newborn's father. The existence of overlapping generations means that individuals may contribute genes to the future in two ways—through survival and through reproduction—and our analysis reveals that these alternative routes are differently affected by sexual antagonism. Survival effects are weighted by the ratio of the reproductive values of female versus male survivors, which is $(x-1) : (y-1)$ for autosomes and $2(x-1) : (y-1)$ for X chromosomes. Hence, under the assumption of age-independent mortality and fecundity rates, if a sexually antagonistic X-linked allele affects survival, then it will invade when male-beneficial if $2(x-1)\tau < (y-1)\sigma$ and when female-beneficial if $(x-1)\sigma > (y-1)\tau$. By contrast, if the allele affects fertility, then its fitness effects in males and females are valued according to their respective genetic shares of their newborn offspring. For the X chromosome, this means fertility effects are weighted in the typical 2 : 1 ratio (see electronic supplementary material).

(c) Relatedness

Third, we consider relatedness (figure 3). Factors such as population structure and mating system may generate genetic correlations between homologous genes residing within the same individual, i.e. inbredness [40], and the traditional coefficient of inbreeding provides a measure of the relatedness between these homologues. For X-chromosomal genes, this affects males and females differently, as while females are diploid at their X-linked loci and hence can be inbred, this is not possible for males on account of their haploidy at X-linked loci [41]. As inbredness increases, we find that an X-linked gene in a female values not only its direct fitness impact on itself, but also its indirect fitness impact on the other, related, gene copy. This increases the relative importance of fitness effects in females (figure 3*c*). To illustrate, under a regime of assortative mating of degree ϕ, the condition for a male-beneficial allele to invade on the X chromosome is $\sigma > 2(\tau + \phi\tau')$, and for a female-beneficial allele is $2(\sigma + \phi\sigma') > \tau$, where σ' and τ' are the indirect fitness effects. Thus, a higher degree of assortative mating can push the invasion conditions in favour of female-beneficial alleles, even if fitness effects are of a greater magnitude in males (see electronic supplementary material).

3. Discussion

Taking a gene-centred approach to the problem of sexual antagonism has two major advantages. First, it provides conceptual clarity, resolving apparent contradictions between the female-biased agendas of X-chromosomal genes and the male-biased

outcomes of certain population-genetic analyses. Second, it provides a simple and practical way to separate and properly understand the factors that affect the outcome of sexually antagonistic selection. By considering in turn how different biological contexts may modulate fitness effects, reproductive values and relatedness, we can more easily generate new testable hypotheses about sexually antagonistic selection and intragenomic conflicts (some examples are given in table 1). While sexual antagonism and sex chromosome evolution have been historically well-studied topics [9,13–16], there remain significant gaps in theoretical understanding [49–52]. Here, we have shown how a gene's-eye–view approach may facilitate incorporation of salient aspects of real-world biology into future models, making them more empirically informative.

One possible avenue for future empirical investigation concerns the relationship between dosage compensation and sex-biased gene expression. While previous work has focused on how and why such dosage compensation systems may have evolved [53–56], less emphasis has been placed on how sexual antagonism may manifest differently in different dosage compensation systems (but see [42,57,58]). Given biologists' increasing knowledge of a variety of sex chromosome systems and their dosage compensation mechanisms [34–36], this presents an exciting opportunity for comparative work, both within and between species. As dosage compensation is reduced in the gonads of many species [35], we would expect greater relative feminization in gonad-expressed genes as compared with those expressed in somatic tissues. Additionally, the degree of dosage compensation may vary across sex chromosomes themselves; for example, in *Drosophila melanogaster* it is thought that the completeness of dosage compensation varies with distance from the high-affinity sites where the dosage compensation complex binds [59]. Therefore, we would expect male-beneficial alleles to invade more readily at loci close to these sites, yielding a negative relationship between male-biased gene expression and distance from these binding sites. Current evidence is mixed as to whether these new predictions are met [42–46], which may in part be due to other effects of dosage compensation upon the distribution of sex-biased genes [58]. Similar—but reversed—predictions would also apply to the Z chromosome, with increased masculinization expected for loci, tissues and species that have lower dosage compensation.

Moreover, species vary greatly in the pace and span of life [60], and within many species differences also occur between the sexes [61]. As we have shown, sex differences in life-history parameters can play an important role in shaping sexually antagonistic traits, with genes ultimately placing more value on the sex in which they spend more time. In our illustrative model, an asymmetry in mean parental age arises as a consequence of sex-specific mortality (figures 1 and 2). Thus, a novel—albeit crude—prediction would be that those organisms that typically have higher male mortality, such as mammals [48,62], will have relatively feminized genomes, while those with female-biased mortality, such as birds [63,64], will be relatively masculinzed. However, factors other than mortality may also affect mean parental age. For example, the two sexes may enter reproductive maturity at different times, and fecundity/mating success may vary with age. Consequently, one sex could have a higher mortality rate—and thus a shorter expected lifespan—yet have a higher mean parental age. An example of this is in humans, where although men typically have a higher mortality rate, the average father is older than the average mother [65,66]. This may explain why, despite women having longer lifespans in almost all societies [67], they nonetheless senesce at a faster rate [68,69], a phenomenon that is referred to in the medical literature as the 'male–female health-survival paradox' [70,71]. While previous suggestions have been made in relation to menopause, and women's lack of direct reproduction in old age [72], the present analysis identifies the more general asymmetry in mean age of parentage in humans—whereby fathers are typically older than mothers—as a potential driver of these differences between the sexes. Additionally, for those sexually antagonistic variants affecting senescence, the later-reproducing sex would be favoured, thus further exacerbating sexual dimorphism in senescence. With demographic and genetic data on sex-specific vital rates and patterns of senescence becoming increasingly available [73–75], similar hypotheses relating intralocus sexual conflict and differences in mean parental age to sex differences in senescence and sex-biased gene expression will become testable not only in humans, but across the tree of life.

Furthermore, we have found that the asymmetry on the X chromosome between an intragenomic 'social' setting (females) and an 'asocial' one (males) means that relatedness between homologous genes may also play an important role in modulating sexual antagonism. While positive relatedness (i.e. due to inbreeding) pushes invasion conditions in favour of female-beneficial alleles, scenarios with negative relatedness (i.e. due to inbreeding avoidance) would do the opposite: with beneficial effects in females being offset by benefits to negatively related gene copies, and deleterious effects being countered by costs to negatively related gene copies (a gene-level form of spite; [22,76]). Despite the potential importance of this effect of assortative mating, few studies have explicitly considered mating scheme or population structure with regard to sexual antagonism, and those that have done so have focused instead on how these may modulate the potential for polymorphism [77–79], rather than their impact on feminization/masculinization. Specific mating systems may introduce further complications involving the relatedness of different individuals to one another—such as local mate and resource competition [80]. Although not considered here, such intrasexual and intersexual cooperative and competitive interactions can modulate the relative value of males and females [30,80], and thus potentially modulate feminization versus masculinzation of the genome. This may occur even for those genes inherited exclusively from one sex [81,82]. Combining both intra-organismal and inter-organismal social interactions provides opportunities for investigating not only how social interaction may modulate sexual antagonism but also how sexual antagonism may modulate social interaction [83,84].

Our analysis has focused mainly upon those X-linked genes for which there is no homologue on the Y chromosome, but similar principles also apply to pseudoautosomal regions. Although the dynamics of these regions are typically more complicated [85–87]—as allele frequencies may differ between males and females even if selection is weak—boundary cases are readily interpretable. We find that when recombination in males is free ($r \approx 1/2$), then these regions will evolve similarly to 'true' autosomal genes, whereas when there is no recombination—yet there remain functional copies on the Y chromosome—then X-linked genes are expected to become feminized, as while there remains the typical 2 : 1 weighting on females, the marginal fitness effects of new mutations may be expected to be of similar magnitude in males and females (see electronic supplementary material).

Table 1. Some empirical predictions emerging from this analysis and possible avenues for testing. n.a., not applicable.

		predictions				
factor	measure of value	autosomes	X chromosomes	Z chromosomes	cytoplasmic genes	potential empirical tests
dosage compensation	marginal fitness effects	n.a.	increased dosage compensation → increased masculinization	increased dosage compensation → increased feminization	n.a.	• comparisons across loci with different extents of dosage compensation (e.g. distance to high-affinity sites in *Drosophila melanogaster* [42–46]) • comparisons across different tissue types, (e.g. soma versus gonads of *Tribolium castanea* [47]) • comparisons across different species with different degrees of dosage compensation (e.g. global versus local dosage compensation [35])
mean parental age	reproductive value	• survival effects: higher mean paternal age → greater masculinization; higher mean maternal age → greater feminization • fertility effects: no change				• comparisons across different species with different, sex-specific mean parental ages (e.g. variation across mammals with different sex-specific mortality rates [48]) • experimental evolution with direct manipulation of parental age
assortative mating	relatedness	no change	increased inbredness →greater feminization	increased inbredness →greater masculinization	no change	• comparisons across species with different mating structures/variation in inbredness • experimental evolution manipulating the degree of inbredness

However, if the Y chromosome degenerates, and if dosage compensation arises, then the marginal fitness effect in males will likely be larger, and thus male-beneficial alleles may more readily invade. From this, we would anticipate that X-linked alleles fixed prior to Y degeneration are more female-biased than those fixed subsequently.

Finally, while our main focus has been upon XY and XO sex determination systems, our general analysis also applies to other systems. Our results for X chromosomes can be directly applied to Z chromosomes simply by switching the roles of female and male. Similarly, the results we have obtained for autosomal regions—including those relating to age structure—will also apply to other systems with similar transmission genetics, including species that employ environmental sex determination. Along the continuum of sex-bias, the Y and W chromosomes occupy the extreme ends, as these are exclusively restricted to males and females, respectively (figure 3), and although cytoplasmically inherited genes, such as those carried by mitochondria and chloroplasts, are most often maternally transmitted, and thus expected to show extreme female bias [3], they may fall anywhere along this spectrum, depending on a combination of their mode of inheritance [88–90] and the nature of the population's age structure (see electronic supplementary material). Identifying the factors that shape the valuations these different genomic factions place on males and females—and the power they have in these different contexts—yields a richer understanding not only of the evolution of sexual dimorphism, but also of the array of intragenomic conflicts that these sex differences foment.

Data accessibility. There are no associated data. Details of the mathematical models are provided in the electronic supplementary material.

Authors' contributions. T.J.H. and A.G. jointly designed the study, performed the analysis and wrote the manuscript.

Competing interests. The authors declare that they have no conflicts of interest.

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A gene's-eye view of sexual antagonism:

Supplementary Material

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Contents

1 Summary

This document contains the methodology behind, and further results pertaining to, *"A gene's-eye view of sexual antagonism"*. The mathematical methods used to generate the results and the reasoning behind certain modelling choices can be found in the section **Methodology**. The results found in the main text can be seen in the section **Results**. In addition this section also contains the invasion conditions for stronger selection regimes, as well as scenarios not discussed fully in the main text including: cytoplasmic genes, genomic imprinting, and haploid selection.

2 Methodology

We consider a series of population genetic models of sexual antagonism. We consider a single locus, and ask whether a rare mutant allele X_1 will be able to invade a population of a given resident allele X_0, and how these conditions will depend on its genomic location, and assumptions about dosage, mating scheme, and age-structure.

2.1 General methodology

We take a "gene" to mean a particular copy of a nonrecombining sequence at some locus in some individual. This gene may exist in various states of the world. It may be in a female or male, it may be of maternal-origin or paternal-origin, it may be in a juvenile or adult. We refer to the particular state of the world that a gene finds itself in as its *class*, which is analogous to *context* (*sensu* Kirkpatrick, Johnson, and Barton, 2002). A gene may adopt a particular strategy or "allele", either the mutant X_1 or the resident X_0. The mutant allele frequency in class i is notated p_i.

Each generation, genes may flow between classes. The probability that a randomly sampled gene in class i at time t came from class j at time $t-1$ is notated $\pi_{i,j}$. Whereby both i and j belong to the same set of total possible classes $i, j \in I$. As the probabilities must sum to 1, then $\sum_j \pi_{i,j} = 1$. These probabilities are the usual backwards transition probabilities found in a gene flow matrix, and thus the dominant left-eigenvector of this matrix gives us the associated class reproductive values (Taylor, 1990; Taylor, 1996).

To calculate whether the mutant allele will invade from rarity, we can construct recursion equations describing the allele frequency in class i at time t, as a function of the allele frequency in the other classes at time $t-1$, we can write out recursion equations in the following form, with the allele frequency at the next time-step given by:

$$p_i'' = \sum_j \pi_{i,j} p_j' = \sum_j \pi_{i,j} p_j w_{i,j} \tag{1}$$

where $\pi_{i,j}$ is the probability that a randomly sampled gene in class i came from class j in the previous time-step, and $w_{i,j}$ is the relative fitness in the path going from class j to class i. $w_{i,j}$ will typically be a function of the allele frequency in the different classes, and $\pi_{i,j}$ will be a function of both the allele frequency in different classes and the fitness. For the scenarios we consider here, when the mutant is vanishingly rare we can reasonably approximate the actual values of $\pi_{i,j}$ with those calculated for a

population which is monomorphic for the resident strategy $\tilde{\pi}_{i,j}$.

$$\begin{aligned} p_i'' &= \sum_j \pi_{i,j} p_j' \\ &= \sum_j (\tilde{\pi}_{i,j} + \delta\tilde{\pi}_{i,j})(p_j + \delta p_j) \\ &= \sum_j (\tilde{\pi}_{i,j} p_j + \tilde{\pi}_{i,j}\delta p_j + \delta\tilde{\pi}_{i,j} p_j + \delta\tilde{\pi}_{i,j}\delta p_j) \\ &= \sum_j \tilde{\pi}_{i,j} p_j' + O(\delta^2) \end{aligned} \tag{2}$$

Using these recursion equations we can ask when the mutant allele will be able to invade from rarity. If the $p = 0$ equilibrium point is unstable then the mutant will be able to invade. To determine the stability, we first calculate the Jacobian matrix $\mathbf{J}$, analysed when the allele is vanishingly rare in the population (Otto and Day, 2011). Each entry of the matrix is given by:

$$\mathbf{J}_{i,j} = \left.\frac{\partial p_i''}{\partial p_j}\right|_{p_i, p_j = 0} \tag{3}$$

If the leading eigenvalue of this matrix is greater than one, $\lambda_{max} > 1$ then the mutant allele will be able to invade. We can write out our eigenvalue, and thus our condition for increase, in the following form:

$$\lambda_{max} = 1 + \sum_i c_i a_i V_i > 1 \tag{4}$$

Where c_i is the class reproductive value (Fisher, 1930; Price and Smith, 1972; Grafen, 2006), a_i is the marginal fitness effect (i.e. Fisher's average effect (Fisher, 1930; Falconer, 1985)), and V_i is the genetic variance in class i (Fisher, 1918). When either selection is weak, or the mutant has low penetrance (e.g. Taylor, 1990; Seger and Stubblefield, 2002), then the variance is approximately equal in all classes. In which case we can write the condition for invasion as:

$$\sum_i c_i a_i > 0 \tag{5}$$

2.2 Transmission

The backwards transmission probabilities for the autosomal, pseudo-autosomal and X-linked cases can be seen in Figure S1.

For the mitochondria, in order to describe some of the diversity of transmission scenarios seen in nature (e.g. Birky Jr, 2001; Greiner, Sobanski, and Bock, 2015), we adopt the following scheme. We assume that individuals contain - and thus transmit- only a single mitochondrial type, but that they may inherit either parental type with a given probability. We allow this to be specific to each sex, such that males may disproportionately inherit a mitochondrial type from fathers, and females from mothers for example. This again can be seen in Figure S1.

2.3 Mating

To incorporate both haploid selection, and also avoid gene frequency change induced by assortative mating itself, we adopt the following scheme. Haploid gametes are produced, undergo selection, then a fraction ϕ of these haploid gametes are selected and preferentially pair with a gamete with the same

allele. This produces diploid genotypes with the following frequencies:

$$
\begin{aligned}
F_{11}, M_{11} = G_{11}(p_f, p_m) &= (1-\phi)p_f p_m + \phi \begin{cases} p_m & \text{if } p_f \geq p_m \\ p_f & \text{if } p_f \leq p_m \end{cases} \\
F_{01}, M_{01} = G_{01}(p_f, p_m) &= (1-\phi)(1-p_f)p_m + \phi \begin{cases} 0 & \text{if } p_f \geq p_m \\ p_m - p_f & \text{if } p_f \leq p_m \end{cases} \\
F_{10}, M_{10} = G_{10}(p_f, p_m) &= (1-\phi)p_f(1-p_m) + \phi \begin{cases} p_f - p_m & \text{if } p_f \geq p_m \\ 0 & \text{if } p_f \leq p_m \end{cases} \\
F_{00}, M_{00} = G_{00}(p_f, p_m) &= (1-\phi)(1-p_f)(1-p_m) + \phi \begin{cases} 1-p_f & \text{if } p_f \geq p_m \\ 1-p_m & \text{if } p_f \leq p_m \end{cases}
\end{aligned}
\tag{6}
$$

When selection is weak, $p_f \approx p_m \approx p$, and thus genotype frequencies can be approximated to:

$$
\begin{aligned}
F_{11} &\approx M_{11} \approx (1-\phi)p^2 + \phi p \\
F_{01} &\approx M_{01} \approx (1-\phi)(1-p)p \\
F_{10} &\approx M_{10} \approx (1-\phi)p(1-p) \\
F_{00} &\approx M_{00} \approx (1-\phi)(1-p)^2 + \phi(1-p)
\end{aligned}
\tag{7}
$$

This also has the useful property that when selection is weak, our assortative parameter ϕ, is equivalent to Wright's inbreeding coefficient (Wright, 1922), the kin-selection coefficient of genetic relatedness between the maternal-origin and paternal-origin genes (Hamilton, 1964).

2.4 Overlapping generations

> "To what extent will persons of this age, on the average, contribute to the ancestry of future generations? The question is one of some interest, since the direct action of Natural Selection must be proportional to this contribution." – (Fisher, 1930, p27)

We may also allow for overlapping generations in our analysis (e.g. Charlesworth, 1994; Caswell, 2001). Commonly, the flow of individuals through different phases of the life-cycle is described in the language of birth rates and survival probabilities. We can take these parameters and translate them into backwards transition probabilities instead, $\pi_{i,j}$, allowing us to describe our population in terms of class reproductive values, i.e. the share of a future population's ancestry that different age classes will have. These provide the correct weightings for allele-frequency changes such that asymptotic change is recovered (Fisher, 1930; Price and Smith, 1972; Taylor, 1990). Thus class reproductive value also describes the relative importance of selection on different age cohorts.

We first analyse the asexual case, extend it to sexually reproducing individuals, and then apply it to our model.

2.4.1 Asexual reproduction

Let us take a population of size n, which is growing at rate λ, and has attained a stable age distribution. There are n_a individuals of age a, and they have an effective birth rate of b_a, i.e. they produce b_a new individuals in the next census point. This may be thought of as a compound of the actual offspring

produced, and the survival of those offspring to the first census. Individuals then survive from age 1 to age a with probability l_a. This can also be written in terms of mortality rates, where μ_a is the probability of death in the interval $a-1$ to a.

$$l_a = \prod_{i=2}^{a}(1-\mu_i) \tag{8}$$

If b is the per capita birth rate of the population, then the relative size of each age class is:

$$u_a = \frac{n_a}{n} = b\frac{l_a}{\lambda^a} \tag{9}$$

Thus the probability that a gene sampled in an age 1 individual came from an age a individual in the previous census is given by:

$$\pi_{1,a} = \frac{b_a}{b}u_a = \frac{b_a l_a}{\lambda^a} \tag{10}$$

For $a > 1$, $\pi_{a,a-1} = 1$. Using these backward transition probabilities we can calculate the reproductive value of the different age-classes.

$$c_a = \sum_{i=1}^{\infty}\pi_{a,i}c_i = \pi_{a,a+1}c_{a+1} + \pi_{1,a}c_1 = c_{a+1} + \frac{b_a l_a}{\lambda^a}c_1 = c_1\sum_{i=a}^{\infty}\frac{b_i l_i}{\lambda^i} \tag{11}$$

This provides the appropriate weighting for the allele-frequency across different age classes, and thus provides a measure of the force of selection on that age class (Medawar, 1946; Medawar, 1952; Hamilton, 1966). Focusing on the newborn cohort ($a = 1$), we can also see that their reproductive value is equivalent to $1/T$, where T is the mean parental age, a classic measure of generation time (Hamilton, 1966; Charlesworth, 1994). We can see this as:

$$T = \frac{\sum_{a=1}^{\infty} a n_a b_a}{\sum_{a=1}^{\infty} n_a b_a} = \sum_{a=1}^{\infty} a\frac{b_a l_a}{\lambda^a} \tag{12}$$

$$1 = \sum_{a=1}^{\infty} c_a = \sum_{a=1}^{\infty}\left(c_1\sum_{i=a}^{\infty}\frac{b_i l_i}{\lambda^i}\right) = c_1\sum_{a=1}^{\infty}\left(\sum_{i=a}^{\infty}\frac{b_i l_i}{\lambda^i}\right) = c_1\sum_{a=1}^{\infty} a\frac{b_a l_a}{\lambda^a} = c_1 T \tag{13}$$

Equation 11 provides an expression for the reproductive value of the age a class, and thus how a change in allele frequency in that class should be weighted. This weighting is composed of two processes - survival and reproduction - which selection may act on differently.

$$c_a = c_{a,r} + c_{a,s} \tag{14}$$

The force of selection on reproduction is given by the value of the newborn individuals that class a produces. If all newborn individuals are the same, then this is simply their share in the newborn class:

$$c_{a,r} = \pi_{1,a}c_1 = c_1\frac{b_a l_a}{\lambda^a} = \frac{1}{T}\frac{b_a l_a}{\lambda^a} \tag{15}$$

The force of selection on survival is given by the value of survivors that class a produces, i.e. the age $a+1$ cohort.

$$c_{a,s} = c_{a+1} = c_1\sum_{i=a+1}^{\infty}\frac{b_i l_i}{\lambda^i} = \frac{1}{T}\sum_{i=a+1}^{\infty}\frac{b_i l_i}{\lambda^i} \tag{16}$$

We can see how these equations also match with Hamilton's expressions for the force of selection on survival and reproduction. Our equation 15 is the same as his equation 8, and our equation 16 is the same as his equation 25, except ours is relative birth rate, and his absolute (Hamilton, 1966).

As well as classes, reproductive value is often framed in terms of individuals $v_a = c_a/u_a$. Commonly, individual reproductive value is scaled such that the reproductive value of a newborn female is 1, which in the discrete time case is $v_1 = 1$ (Fisher, 1930). With the above notation:

$$\frac{v_a}{v_1} = \frac{c_a}{u_a}\frac{u_1}{c_1} = \frac{u_1}{u_a}\sum_{i=a}^{\infty}\frac{b_i l_i}{\lambda^i} = \frac{\lambda^{a-1}}{l_a}\sum_{i=a}^{\infty}\frac{b_i l_i}{\lambda^i} \tag{17}$$

This is a discrete time analogue of Hamilton's equation 22.

2.4.2 Sexual reproduction

> "If we consider the aggregate of an entire generation of such offspring it is clear that the total reproductive value of the males in this group is exactly equal to the total value of all the females, because each sex must supply half the ancestry of all future generations of the species" – (Fisher, 1930, p142)

We can introduce sexual reproduction for arbitrary ploidy in the following way. Let us now split our population into two portions, females n_f, and males n_m. Let us retain the above notation for the female specific parameters, and let us introduce κ_a for the male specific survival probabilities, and ν_a for the mortalities.

$$\kappa_a = \prod_{i=2}^{a}(1-\nu_i) \tag{18}$$

And rather than b_a for birth rate, we have m_a for the fertilisation rate at age a. We also allow individuals to vary their sex-ratio strategy as a function of age, where z_a is the fraction of males produced by females of age a, and ζ_a is the fraction of males from male fertilisations of age a. We place the following constraints on our system:

$$\begin{aligned} n_f\overline{b} &= \sum_{a=1}^{\infty} n_{f,a}b_a = \sum_{a=1}^{\infty} n_{m,a}m_a = n_m\overline{m} \\ n_f\overline{b}(1-\overline{z}) &= \sum_{a=1}^{\infty} n_{f,a}b_a(1-z_a) = \sum_{a=1}^{\infty} n_{m,a}m_a(1-z_a) = n_m\overline{m}(1-\overline{\zeta}) \\ n_f\overline{b}\overline{z} &= \sum_{a=1}^{\infty} n_{f,a}b_a z_a = \sum_{a=1}^{\infty} n_{m,a}m_a z_a = n_m\overline{m}\overline{\zeta} \end{aligned} \tag{19}$$

From this we can see that the mean maternal age for a female and male are respectively:

$$\begin{aligned} T_{f,f} &= \frac{\sum_{a=1}^{\infty} a n_{f,a}b_a(1-z_a)}{\sum_{a=1}^{\infty} n_{f,a}b_a(1-z_a)} = \sum_{a=1}^{\infty} a\frac{b_a l_a(1-z_a)}{\lambda^a} \\ T_{f,m} &= \frac{\sum_{a=1}^{\infty} a n_{f,a}b_a z_a}{\sum_{a=1}^{\infty} n_{f,a}b_a z_a} = \sum_{a=1}^{\infty} a\frac{b_a l_a z_a}{\lambda^a} \end{aligned} \tag{20}$$

And the mean paternal age for a female and male respectively are:

$$\begin{aligned} T_{m,f} &= \frac{\sum_{a=1}^{\infty} a n_{m,a}m_a(1-\zeta_a)}{\sum_{a=1}^{\infty} n_{m,a}m_a(1-\zeta_a)} = \sum_{a=1}^{\infty} a\frac{m_a\kappa_a(1-\zeta_a)}{\lambda^a} \\ T_{m,m} &= \frac{\sum_{a=1}^{\infty} a n_{m,a}m_a\zeta_a}{\sum_{a=1}^{\infty} n_{m,a}m_a\zeta_a} = \sum_{a=1}^{\infty} a\frac{m_a\kappa_a\zeta_a}{\lambda^a} \end{aligned} \tag{21}$$

To allow for arbitrary ploidy, we notate the genetic share of an individual female that a mother gets α, and the genetic share of a son that a father gets β. We now calculate the backward transition probabilities for our four cases:

$$\begin{aligned} \pi_{\{f,a\},\{f,1\}} &= \alpha\frac{b_a l_a(1-z_a)}{\lambda^a} \\ \pi_{\{f,a\},\{m,1\}} &= (1-\beta)\frac{b_a l_a z_a}{\lambda^a} \\ \pi_{\{m,a\},\{f,1\}} &= (1-\alpha)\frac{m_a\kappa_a(1-\zeta_a)}{\lambda^a} \\ \pi_{\{m,a\},\{m,1\}} &= \beta\frac{m_a\kappa_a\zeta_a}{\lambda^a} \end{aligned} \tag{22}$$

With these backward transition probabilities, we can now calculate the class reproductive values for females of age a:

$$\begin{aligned} c_{f,a} &= c_{f,a+1} + \pi_{\{f,a\},\{f,1\}}c_{f,1} + \pi_{\{f,a\},\{m,1\}}c_{m,1} \\ &= c_{f,1}\alpha\sum_{i=a}^{\infty}\frac{b_i l_i(1-z_i)}{\lambda^i} + c_{m,1}(1-\beta)\sum_{i=a}^{\infty}\frac{b_i l_i z_i}{\lambda^i} \end{aligned} \tag{23}$$

And males of age a:

$$\begin{aligned} c_{m,a} =& c_{m,a+1} + \pi_{\{m,a\},\{f,1\}} c_{f,1} + \pi_{\{m,a\},\{m,1\}} c_{m,1} \\ =& c_{f,1}(1-\alpha) \sum_{i=a}^{\infty} \frac{m_i \kappa_i (1-\zeta_i)}{\lambda^i} + c_{m,1} \beta \sum_{i=a}^{\infty} \frac{m_i \kappa_i \zeta_i}{\lambda^i} \end{aligned} \tag{24}$$

We can then write the total reproductive value of males and females in terms of the newborns, mean parental ages, and sex-specific shares in offspring.

$$\begin{aligned} c_f = \sum_{a=1}^{\infty} c_{f,a} =& c_{f,1} \alpha T_{f,f} + c_{m,1}(1-\beta) T_{f,m} \\ c_m = \sum_{a=1}^{\infty} c_{m,a} =& c_{f,1}(1-\alpha) T_{m,f} + c_{m,1} \beta T_{m,m} \end{aligned} \tag{25}$$

We can also write the total class reproductive values of males and females as:

$$\begin{aligned} c_f =& \alpha c'_{f,1} + (1-\beta) c'_{m,1} + \sum_{a=2}^{\infty} c'_{f,a} = \alpha c'_{f,1} + (1-\beta) c'_{m,1} + c'_f - c'_{f,1} \\ c_m =& (1-\alpha) c'_{f,1} + \beta c'_{m,1} + \sum_{a=2}^{\infty} c'_{m,a} = (1-\alpha) c'_{f,1} + \beta c'_{m,1} + c'_m - c'_{m,1} \end{aligned} \tag{26}$$

As the class reproductive values are constant over time. We can drop the primes and rearrange to:

$$\frac{c_{f,1}}{c_{m,1}} = \frac{1-\beta}{1-\alpha} \tag{27}$$

Under diploidy ($\alpha = 1/2, \beta = 1/2$), this means that the class reproductive value of newborn males and females is equal:

$$\frac{c_{f,1}}{c_{m,1}} = \frac{1/2}{1/2} = 1 \tag{28}$$

It is this constraint - that the reproductive value of the newborn females is equal to that of the newborn males - which underpins the classical sex-ratio argument that a parent should invest in the rarer sex (Fisher, 1930; Edwards, 1998). Thus we can see how this argument holds even with overlapping generations (Goodman, 1982; Grafen, 2014).

Similarly, we can recover results for haplodiploidy/X-chromosomes ($\alpha = 1/2, \beta = 0$) as a special case of equation 27:

$$\frac{c_{f,1}}{c_{m,1}} = \frac{1}{1/2} = 2 \tag{29}$$

Once again, it is this constraint that underpins sex-ratio arguments relating to haplodiploids. We can again see how this is remains under age-structure (Gardner, 2014).

Putting together equations 25 and 27, along with the constraint that $c_f + c_m = 1$, we can write the class reproductive values of newborns as so:

$$\begin{aligned} c_{f,1} &= (1-\beta)/\overline{T} \\ c_{m,1} &= (1-\alpha)/\overline{T} \end{aligned} \tag{30}$$

Where:

$$\overline{T} = \alpha(1-\beta) T_{f,f} + (1-\alpha)(1-\beta) T_{f,m} + (1-\alpha)(1-\beta) T_{m,f} + (1-\alpha)\beta T_{m,m} \tag{31}$$

And so we can express the class reproductive values for males and females as a whole cohort:

$$\frac{c_f}{c_m} = \left(\frac{1-\beta}{1-\alpha} \right) \left(\frac{\alpha T_{f,f} + (1-\alpha) T_{f,m}}{(1-\beta) T_{m,f} + \beta T_{m,m}} \right) \tag{32}$$

Once again, we can recover the results for autosomes as a special case ($\alpha = 1/2, \beta = 1/2$) (Grafen, 2014):

$$\frac{c_f}{c_m} = \frac{T_{f,f} + T_{f,m}}{T_{m,f} + T_{m,m}} = \frac{T_f}{T_m} \tag{33}$$

And extend it to other inheritance systems, e.g. haplodiploidy ($\alpha = 1/2, \beta = 0$):

$$\frac{c_f}{c_m} = 2\left(\frac{T_{f,f} + T_{f,m}}{2T_{m,f}}\right) = 2\frac{T_f}{T_m} \tag{34}$$

2.4.3 Weights on survival and reproduction

> "From an individual's point of view, survival must be weighted by the individual's reproductive value in the next time period. Current fecundity must be weighted by the reproductive value of offspring in the next time period"– (Frank, 1998, p171)

As with the asexual case, in the sexual case, for each age class, the force of selection upon fecundity relative to survival effects will be given by the relative value of the survivors produced, compared to the newborns created. For our model, we assume that individuals do not undergo senescence, and thus fecundity, sex-ratio strategy, and mortality rates remain constant with respect to age. As a consequence, we need not track each age-class separately, but can combine them instead into a single class, of which a fraction of the next generation will come from survival, and a fraction from reproduction.

We allow for sex-specific mortality, with the probability of survival each generation being $1-\mu$ for females, and $1-\nu$ for males. The fraction of individuals that came through reproduction is simply n_1/n, and the fraction through survival is $\sum_{a=2}^{\infty} n_a/n = 1 - n_1/n$.

$$\begin{aligned}
\frac{n_{f,1}}{n_f} &= \frac{n_f\lambda^{-1}b(1-z)}{n_f\lambda^{-1}b(1-z) + n_f\lambda^{-1}(1-\mu)} = \frac{b(1-z)}{b(1-z)+(1-\mu)} = \frac{\lambda-(1-\mu)}{\lambda} \\
\frac{n_{m,1}}{n_m} &= \frac{n_m\lambda^{-1}bz}{n_m\lambda^{-1}bz + n_m\lambda^{-1}(1-\nu)} = \frac{bz}{bz+(1-\nu)} = \frac{\lambda-(1-\nu)}{\lambda}
\end{aligned} \tag{35}$$

And thus the fraction of individuals who came from survival in the previous generation is given by:

$$\begin{aligned}
\sum_{a=2}^{\infty}\frac{n_{f,a}}{n_f} &= 1 - \frac{\lambda-(1-\mu)}{\lambda} = \frac{1-\mu}{\lambda} \\
\sum_{a=2}^{\infty}\frac{n_{m,a}}{n_m} &= 1 - \frac{\lambda-(1-\nu)}{\lambda} = \frac{1-\nu}{\lambda}
\end{aligned} \tag{36}$$

When the population is constant in size, then the fraction of newborns and survivors is simply μ and $1-\mu$ for females, and ν and $1-\nu$ for males.

As with above, the weights on survival and reproduction can be written in terms of class reproductive values. Weights on reproduction are given by the class reproductive values of the newborn individuals, and the relative shares that males and females have in them:

$$\begin{aligned}
\frac{c_{f,r}}{c_{m,r}} &= \frac{\alpha c_{f,1} + (1-\beta)c_{m,1}}{(1-\alpha)c_{f,1} + \beta c_{m,1}} \\
&= \frac{\alpha(1-\beta) + (1-\beta)(1-\alpha)}{(1-\alpha)(1-\beta) + \beta(1-\alpha)}
\end{aligned} \tag{37}$$

For diploidy ($\alpha = 1/2, \beta = 1/2$) this simplifies to 1, and for haplodiploidy/X chromosomes ($\alpha = 1/2, \beta = 0$) this simplifies to 2. Weights on survival are given by the reproductive value of the surviving males and females.

$$\begin{aligned}
\frac{c_{f,s}}{c_{m,s}} &= \frac{\sum_{a=1}^{\infty} c_{f,a+1}}{\sum_{a=1}^{\infty} c_{m,a+1}} = \frac{c_f - c_{f,1}}{c_m - c_{m,1}} \\
&= \left(\frac{1-\beta}{1-\alpha}\right)\left(\frac{\alpha T_{ff} + (1-\alpha)T_{fm} - 1}{(1-\beta)T_{mf} + \beta T_{mm} - 1}\right) \\
&= \left(\frac{1-\beta}{1-\alpha}\right)\left(\frac{T_f - 1}{T_m - 1}\right)
\end{aligned} \tag{38}$$

In our specific model, outlined above, the mean parental ages are:

$$T_f = \sum_{a=1}^{\infty} a \frac{l_a}{\lambda^a} b = a \frac{(1-\mu)^{a-1}}{\lambda^a} = \frac{\lambda}{\lambda - (1-\mu)} \tag{39}$$

$$T_m = \sum_{a=1}^{\infty} a \frac{\kappa_a}{\lambda^a} m = a \frac{(1-\nu)^{a-1}}{\lambda^a} = \frac{\lambda}{\lambda - (1-\nu)} \tag{40}$$

Which in our specific model means the relative weighting on survival simplifies under diploidy ($\alpha = 1/2, \beta = 1/2$) to:

$$\frac{c_{f,s}}{c_{m,s}} = \left(\frac{1-\mu}{1-\nu}\right)\left(\frac{\lambda - (1-\nu)}{\lambda - (1-\mu)}\right) \tag{41}$$

And for haplodiploidy/X-chromosomes ($\alpha = 1/2, \beta = 0$):

$$\frac{c_{f,s}}{c_{m,s}} = 2\left(\frac{1-\mu}{1-\nu}\right)\left(\frac{\lambda - (1-\nu)}{\lambda - (1-\mu)}\right) \tag{42}$$

2.5 Fitness

In choosing the fitness scheme a number of assumptions must be made. Here we explain the reasoning behind the different assumptions. The fitness scheme itself can be seen in Table S2.

2.5.1 Dominance in the two sexes

The dominance coefficient h, typically scales a fitness effect between the two homozygote states. If gene effects are purely additive then $h = 1/2$. In previous models (e.g. Rice, 1984), it has been assumed that dominance is equivalent, or at least comparable, in the two sexes. In comparison, a series of more recent models have instead assumed reversals of dominance between the two sexes (Fry, 2010; Jordan and Charlesworth, 2012; Patten, 2019). Much of this debate appears centred round phenotypic vs fitness conceptions of the work the dominance coefficient is doing. If phenotypic, then the dominance coefficient would be expected due to the non-linearities in the allelic effect on the phenotype. If the gene is acting on a similar phenotype, and through a similar pathway in the two sexes, then it would make sense that dominance would be similar in the two sexes. In contrast, if we take a fitness view, where by the homozygote genotype moves amounts S and T in fitness space. Then, due to the expected non-linearities in the fitness landscape, the dominance coefficients may be different. In particular, it has been argued that if they are moving in opposite directions with respect to their respective optima then reversals of dominance may be expected. Discussion of these points can be found in (Fry, 2010, Patten, 2019, Connallon and Chenoweth, 2019). To incorporate both of these we consider both 'equal dominance' $h_f = h_m = h$, and 'reversals of dominance' $h_f = 1 - h_m = h$ scenarios.

2.5.2 Magnitude of fitness effects in the two sexes

A second point revolves around how to compare the fitness effect in a heterozygote/homozygote to that in a hemizygote. Typically, it is assumed that the fitness effects in the hemizygote/homozygote are comparable. However, this implicitly assumes an averaging rather than an adding view of gene effects (Frank, 2003, Gardner, 2012). In general, there is not a clear theoretical argument as to which approach should be taken. Empirically, dosage balancing mechanisms, such as X-inactivation or increased gene expression in one sex, may justify an averaging rather than adding approach, as the

amount of gene product in comparison to the autosomes will be the same in the two sexes (Mank, 2013; Gu and Walters, 2017; Muyle, Shearn, and Marais, 2017).

However, this may not hold if a variant's effects are due to absolute physical copies of the gene, or if dosage balancing mechanisms do not ensure that the amount of mutant gene product is equivalent between the homozygote and the hemizygote. In these cases, the hemizygote may be regarded as more comparable to the heterozygote - an adding approach. In reality, it is likely that the extent of dosage balance - which we notate γ - will scale between these two extremes.

2.5.3 Parent-of-origin effects

Finally, genes may also have parent-of-origin effects (Ferguson-Smith, 2011). The best understood form of this is genomic imprinting, whereby a gene's level of expression depends on its parent of origin, and usually involves the silencing of a gene originating from one parent and the expression of its homologue originating from the other. This is an important aspect to consider as various mechanisms of dosage balance on sex chromosomes rely on parent-of-origin specific mechanisms. The best understood of which is in the marsupials, where X-inactivation is always paternal (Graves, 2016). Recently, there has also been evidence for genomic imprinting as a form of dosage compensation in *Silene latifolia* (Muyle, Zemp, et al., 2018), although see Krasovec et al. (2019) for an alternative interpretation.

It has also been proposed that genomic imprinting may evolve as a consequence of sexually antagonistic selection (Day and Bonduriansky, 2004). If so, then sex-specific imprinting would be expected, i.e. daughters express their maternal-origin gene copy and sons their paternal-origin gene copy. To explore these diverse pieces of biology we consider four scenarios: A) no parent-of-origin effects, B) maternal-origin silencing, C) paternal-origin silencing, D) sex-specific imprinting. For the X chromosome, we assume that the imprinting has no impact in males, and as such the imprinting only affects females.

2.5.4 Selection in haploids vs diploids

Selection may occur during haploid as well as diploid phases (reviewed by Immler (2019)). As with the homozygote/hemizygote comparison above it is not clear whether gene effects should be added or averaged. In practice, most modellers assume averaging effects (e.g. Immler, Arnqvist, and Otto, 2012). We follow that precedent here when comparing selection in haploids and diploids.

2.5.5 Table of genotypic fitnesses

Autosomal/Pseudoautosomal		F_{00}	F_{10}	F_{01}	F_{11}	M_{00}	M_{10}	M_{01}	M_{11}	f_0	f_1	m_0	m_1
No PoO effects	M+/F-	1	$1-h_fT$	$1-h_fT$	$1-T$	1	$1+h_mS$	$1+h_mS$	$1+S$	1	1	1	1
	F+/M-	1	$1+h_fS$	$1+h_fS$	$1+S$	1	$1-h_mT$	$1-h_mT$	$1-T$	1	1	1	1
Paternal-origin silencing	M+/F-	1	$1-T$	1	$1-T$	1	$1+S$	1	$1+S$	1	1	1	1
	F+/M-	1	$1+S$	1	$1+S$	1	$1-T$	1	$1-T$	1	1	1	1
Maternal-origin silencing	M+/F-	1	1	$1-T$	$1-T$	1	1	$1+S$	$1+S$	1	1	1	1
	F+/M-	1	1	$1+S$	$1+S$	1	1	$1-T$	$1-T$	1	1	1	1
Sex-specific imprinting	M+/F-	1	$1-T$	1	$1-T$	1	1	$1+S$	$1+S$	1	1	1	1
	F+/M-	1	$1+S$	1	$1+S$	1	1	$1-T$	$1-T$	1	1	1	1
Haploid selection	M+/F-	1	1	1	1	1	1	1	1	1	$1-T$	1	$1+S$
	F+/M-	1	1	1	1	1	1	1	1	1	$1+S$	1	$1-T$

Table S1: Fitness scheme for autosomal and pseudoautosomal genes

X-linked		F_{00}	F_{10}	F_{01}	F_{11}	M_0	M_1	f_0	f_1	m_0	m_1
No PoO effects	M+	1	$1-h_fT$	$1-h_fT$	$1-T$	1	$1+(h_m+\gamma(1-h_m))S$	1	1	1	1
	F+	1	$1+h_fS$	$1+h_fS$	$1+S$	1	$1-(h_m+\gamma(1-h_m))T$	1	1	1	1
Paternal-origin silencing	M+	1	$1-T$	1	$1-T$	1	$1+S$	1	1	1	1
	F+	1	$1+S$	1	$1+S$	1	$1-T$	1	1	1	1
Maternal-origin silencing	M+	1	1	$1-T$	$1-T$	1	$1+S$	1	1	1	1
	F+	1	1	$1+S$	$1+S$	1	$1-T$	1	1	1	1
Sex-specific imprinting	M+	1	$1-T$	1	$1-T$	1	$1+S$	1	1	1	1
	F+	1	$1+S$	1	$1+S$	1	$1-T$	1	1	1	1
Haploid selection	M+	1	1	1	1	1	1	1	$1-T$	1	$1+S$
	F+	1	1	1	1	1	1	1	$1+S$	1	$1-T$

Table S2: Fitness scheme for X-linked genes

Mitochondria		F_0	F_1	M_0	M_1	f_0	f_1	m_0	m_1
No PoO effects	M+	1	$1-T$	1	$1+S$	1	1	1	1
	F+	1	$1+S$	1	$1-T$	1	1	1	1
Paternal-origin silencing	M+	1	$1-T$	1	$1+S$	1	1	1	1
	F+	1	$1+S$	1	$1-T$	1	1	1	1
Maternal-origin silencing	M+	1	$1-T$	1	$1+S$	1	1	1	1
	F+	1	$1+S$	1	$1-T$	1	1	1	1
Sex-specific imprinting	M+	1	$1-T$	1	$1+S$	1	1	1	1
	F+	1	$1+S$	1	$1-T$	1	1	1	1
Haploid selection	M+	1	1	1	1	1	$1-T$	1	$1+S$
	F+	1	1	1	1	1	$1+S$	1	$1-T$

Table S3: Fitness scheme for mitochondrial genes

2.6 Recurrence equations

2.6.1 Genotype recursions

We initially write out our recurrence equations in terms of genotype frequencies. For the autosomal/pseudoautosomal case, there are 4 possible genotypes per sex. As the frequency of the genotypes within one sex must sum to 1, then we can fully describe out population with 6 variables. Similarly we need 4 variables for the X-linked case, and 2 for the mitochondrial case.

Autosomal

$$\begin{aligned}
F'_{10} =& \rho_F \left(F_{10} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_F) G_{10}(f'_1, m'_1) \\
F'_{01} =& \rho_F \left(F_{01} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_F) G_{01}(f'_1, m'_1) \\
F'_{11} =& \rho_F \left(F_{11} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_F) G_{11}(f'_1, m'_1) \\
M'_{10} =& \rho_M \left(M_{10} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_M) G_{10}(f'_1, m'_1) \\
M'_{01} =& \rho_M \left(M_{01} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_M) G_{01}(f'_1, m'_1) \\
M'_{11} =& \rho_M \left(M_{11} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_M) G_{11}(f'_1, m'_1)
\end{aligned} \tag{43}$$

Where:

$$\rho_F = \frac{1-\mu}{\lambda}; \rho_M = \frac{1-\nu}{\lambda} \tag{44}$$

$$\begin{aligned}
f'_1 &= \left(F_{01} \frac{\omega_{F01}}{2\overline{\omega}_F} + F_{10} \frac{\omega_{F10}}{2\overline{\omega}_F} + F_{11} \frac{\omega_{F11}}{\overline{\omega}_F} \right) \frac{\omega_{f1}}{\overline{\omega}_f} \\
m'_1 &= \left(M_{01} \frac{\omega_{M01}}{2\overline{\omega}_M} + M_{10} \frac{\omega_{M10}}{2\overline{\omega}_M} + M_{11} \frac{\omega_{M11}}{\overline{\omega}_M} \right) \frac{\omega_{m1}}{\overline{\omega}_m}
\end{aligned} \tag{45}$$

$$\begin{aligned}
\overline{\psi}_F =& \psi_{F00}(1 - F_{01} - F_{10} - F_{11}) + \psi_{F01} F_{01} + \psi_{F10} F_{10} + \psi_{F11} F_{11} \\
\overline{\psi}_M =& \psi_{M00}(1 - M_{01} - M_{10} - M_{11}) + \psi_{M01} M_{01} + \psi_{M10} M_{10} + \psi_{M11} M_{11}
\end{aligned} \tag{46}$$

$$\begin{aligned}
\overline{\omega}_F =& \omega_{F00}(1 - F_{01} - F_{10} - F_{11}) + \omega_{F01} F_{01} + \omega_{F10} F_{10} + \omega_{F01} F_{11} \\
\overline{\omega}_M =& \omega_{M00}(1 - M_{01} - M_{10} - M_{11}) + \omega_{M01} M_{01} + \omega_{M10} M_{10} + \omega_{M01} M_{11}
\end{aligned} \tag{47}$$

$$\begin{aligned}
\overline{\omega}_f =& \left(F_{01} \frac{\omega_{F01}}{2\overline{\omega}_F} + F_{10} \frac{\omega_{F10}}{2\overline{\omega}_F} + F_{11} \frac{\omega_{F11}}{\overline{\omega}_F} \right) \omega_{f1} \\
&+ \left(1 - F_{01} \frac{\omega_{F01}}{2\overline{\omega}_F} - F_{10} \frac{\omega_{F10}}{2\overline{\omega}_F} - F_{11} \frac{\omega_{F11}}{\overline{\omega}_F} \right) \omega_{f0} \\
\overline{\omega}_m =& \left(M_{01} \frac{\omega_{M01}}{2\overline{\omega}_M} + M_{10} \frac{\omega_{M10}}{2\overline{\omega}_M} + M_{11} \frac{\omega_{M11}}{\overline{\omega}_M} \right) \omega_{m1} \\
&+ \left(1 - M_{01} \frac{\omega_{M01}}{2\overline{\omega}_M} - M_{10} \frac{\omega_{M10}}{2\overline{\omega}_M} - M_{11} \frac{\omega_{M11}}{\overline{\omega}_M} \right) \omega_{m0}
\end{aligned} \tag{48}$$

X-linked

$$\begin{aligned}
F'_{10} =& \rho_F \left(F_{10} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_F) G_{10}(f'_1, m'_1) \\
F'_{01} =& \rho_F \left(F_{01} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_F) G_{01}(f'_1, m'_1) \\
F'_{11} =& \rho_F \left(F_{11} \frac{\psi_{F10}}{\overline{\psi}_F} \right) + (1-\rho_F) G_{11}(f'_1, m'_1) \\
M'_1 =& \rho_M \left(M_1 \frac{\psi_{M1}}{\overline{\psi}_M} \right) + (1-\rho_M) f'_1
\end{aligned} \tag{49}$$

$$
\begin{aligned}
f_1' &= \left(F_{01} \frac{\omega_{F01}}{2\overline{\omega}_F} + F_{10} \frac{\omega_{F10}}{2\overline{\omega}_F} + F_{11} \frac{\omega_{F11}}{\overline{\omega}_F} \right) \frac{\omega_{f1}}{\overline{\omega}_f} \\
m_1' &= \left(M_1 \frac{\omega_{M1}}{\overline{\omega}_M} \right) \frac{\omega_{m1}}{\overline{\omega}_m}
\end{aligned}
\tag{50}
$$

$$
\rho_F = \frac{1-\mu}{\lambda}; \rho_M = \frac{1-\nu}{\lambda} \tag{51}
$$

Where:

$$
\begin{aligned}
\overline{\psi}_F =& \psi_{F00}(1 - F_{01} - F_{10} - F_{11}) + \psi_{F01}F_{01} + \psi_{F10}F_{10} + \psi_{F11}F_{11} \\
\overline{\psi}_M =& \psi_{M1}M_1 + \psi_{M0}(1 - M_1)
\end{aligned}
\tag{52}
$$

$$
\begin{aligned}
\overline{\omega}_F =& \omega_{F00}(1 - F_{01} - F_{10} - F_{11}) + \omega_{F01}F_{01} + \omega_{F10}F_{10} + \omega_{F01}F_{11} \\
\overline{\omega}_M =& \omega_{M1}M_1 + \omega_{M0}(1 - M_1)
\end{aligned}
\tag{53}
$$

$$
\begin{aligned}
\overline{\omega}_f =& \left(F_{01} \frac{\omega_{F01}}{2\overline{\omega}_F} + F_{10} \frac{\omega_{F10}}{2\overline{\omega}_F} + F_{11} \frac{\omega_{F11}}{\overline{\omega}_F} \right) \omega_{f1} \\
&+ \left(1 - F_{01} \frac{\omega_{F01}}{2\overline{\omega}_F} - F_{10} \frac{\omega_{F10}}{2\overline{\omega}_F} - F_{11} \frac{\omega_{F11}}{\overline{\omega}_F} \right) \omega_{f0} \\
\overline{\omega}_m =& \left(\frac{\omega_{M1}}{\overline{\omega}_M} M_1 \right) \omega_{m1} + \left(1 - \frac{\omega_{M1}}{\overline{\omega}_M} M_1 \right) \omega_{m0}
\end{aligned}
\tag{54}
$$

Mitochondria

$$
\begin{aligned}
F_1' =& \rho_F \left(F_1 \frac{\psi_{F1}}{\overline{\psi}_F} \right) \\
&+ (1 - \rho_F) \left(\mathbb{P}_f G_{10}(f_1', m_1') + (1 - \mathbb{P}_f) G_{01}(f_1', m_1') + G_{11}(f_1', m_1') \right) \\
M_1' =& \rho_M \left(M_1 \frac{\psi_{M1}}{\overline{\psi}_M} \right) \\
&+ (1 - \rho_M) \left((1 - \mathbb{P}_m) G_{10}(f_1', m_1') + \mathbb{P}_m G_{01}(f_1', m_1') + G_{11}(f_1', m_1') \right)
\end{aligned}
\tag{55}
$$

$$
\begin{aligned}
f_1' &= \left(F_1 \frac{\omega_{F1}}{\overline{\omega}_F} \right) \frac{\omega_{f1}}{\overline{\omega}_f} \\
m_1' &= \left(M_1 \frac{\omega_{M1}}{\overline{\omega}_M} \right) \frac{\omega_{m1}}{\overline{\omega}_m}
\end{aligned}
\tag{56}
$$

2.6.2 Allele frequency recursions

We can convert the above genotype recursions into allele frequency recursions using the following recipes.

Autosomal/Pseudoautosomal

$$
\begin{aligned}
p_{ff}' =& F_{10}' + F_{11}' \\
p_{fm}' =& F_{01}' + F_{11}' \\
p_{mf}' =& M_{10}' + M_{11}' \\
p_{mm}' =& M_{01}' + M_{11}' \\
D_f' =& F_{11}' - (F_{10}' + F_{11}')(F_{01}' + F_{11}') \\
D_m' =& M_{11}' - (M_{10}' + M_{11}')(M_{01}' + M_{11}')
\end{aligned}
\tag{57}
$$

Where:

$$\begin{aligned} F_{10} =& p_{ff}(1-p_{fm}) - \mathcal{D}_f \\ F_{01} =& (1-p_{ff})p_{fm} - \mathcal{D}_f \\ F_{11} =& p_{ff}p_{fm} + \mathcal{D}_f \\ M_{10} =& p_{mf}(1-p_{mm}) - \mathcal{D}_m \\ M_{01} =& (1-p_{mf})p_{mm} - \mathcal{D}_m \\ M_{11} =& p_{mf}(1-p_{mm}) - \mathcal{D}_m \end{aligned} \tag{58}$$

X-linked

$$\begin{aligned} p'_{ff} =& F'_{10} + F'_{11} \\ p'_{fm} =& F'_{01} + F'_{11} \\ p'_{m} =& M'_{1} \\ D'_{f} =& F'_{11} - (F'_{10} + F'_{11})(F'_{01} + F'_{11}) \end{aligned} \tag{59}$$

Where:

$$\begin{aligned} F_{10} =& p_{ff}(1-p_{fm}) - \mathcal{D}_f \\ F_{01} =& (1-p_{ff})p_{fm} - \mathcal{D}_f \\ F_{11} =& p_{ff}p_{fm} + \mathcal{D}_f \\ M_{1} =& p_{m} \end{aligned} \tag{60}$$

Mitochondrial

$$\begin{aligned} p'_{f} =& F'_{1} \\ p'_{m} =& M'_{1} \end{aligned} \tag{61}$$

Where:

$$\begin{aligned} F_{1} =& p_{f} \\ M_{1} =& p_{m} \end{aligned} \tag{62}$$

3 Results

This section contains the invasion conditions for both the mutant, and the resident allele, for both selection on fertility effects, selection on survival effects, under both weak and strong selection.

3.1 Selection on fertility effects

In this section, selection acts on fertility such that the ω's of our recursion equations are as outlined in the fitness tables. There is no selection on survival and thus the ψ's in the recursion equations are all simply 1. There is no assortative mating, so $\phi = 0$.

Full invasion conditions for these different scenarios can be seen in Tables S5,S7,S9. Weak selection approximations can be see in Tables S4,S6,S8. Plots of these invasion conditions can be found in Figures S2,S3,S4,S5.

3.2 Selection on fertility effects with inbreeding

As in the previous section, selection acts on fertility such that the ω's of our recursion equations are as outlined in the fitness tables. There is no selection on survival and thus the ψ's in the recursion equations are all equal to 1. We now allow for arbitrary amounts of assortative mating. In the assortative mating scheme it needs to be known whether the allele frequency is higher in male or female gametes post-selection. For the scenarios we consider here, we can *a priori* make assumptions about when the allele frequency will be higher in males or females. We make the assumption that those scenarios where the allele is female beneficial, will generate a higher frequency in female gametes, and when it is male beneficial, there will be a higher frequency in male gametes. This was consistent with numerical iterations of our recursion equations which do not make these assumptions.

Full invasion conditions for these scenarios can be seen in Tables S9,S11,S13. Weak selection approximations can be seen in Tables S8,S10,S12. Plots of these invasion conditions can be found in Figures S6,S7,S8,S9.

3.3 Selection on survival effects

In this section, selection acts on survival such that the ψ's of our recursion equations are as outlined in the fitness tables. There is no selection on survival and thus the ω's in the recursion equations are all simply 1. There is no inbreeding, and thus $\phi = 0$.

Full invasion conditions for these scenarios can be seen in Table S15,S17,S19. Weak selection approximations can be seen in Tables S14,S16,S18. Plots of these invasion conditions can be found in Figures S10,S11,S12,S13.

4 Figures

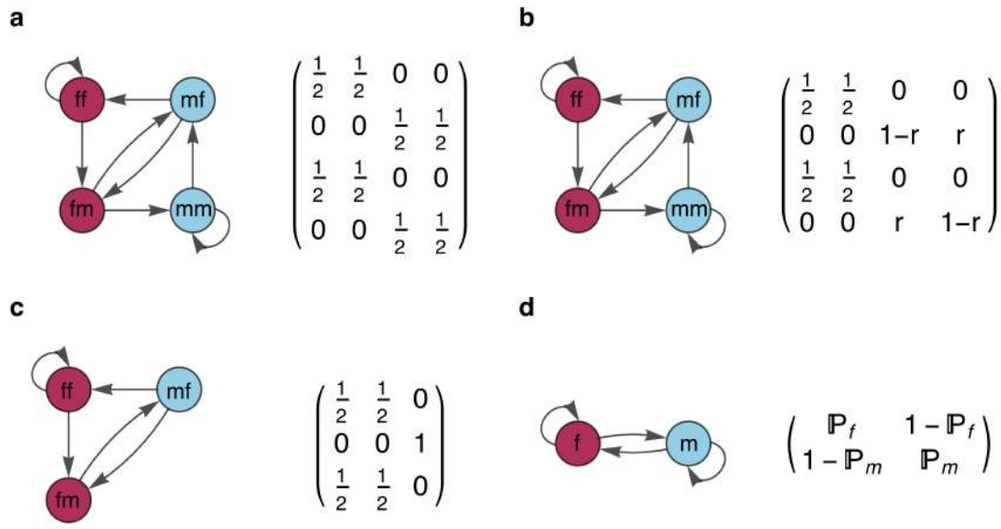

Figure S1: Gene flow matrices for the different portions of the genome we consider here. a) Autosomal genes, **b)** pseudoautosomal genes, **c)** x-linked genes, **d)** mitochondrial genes.

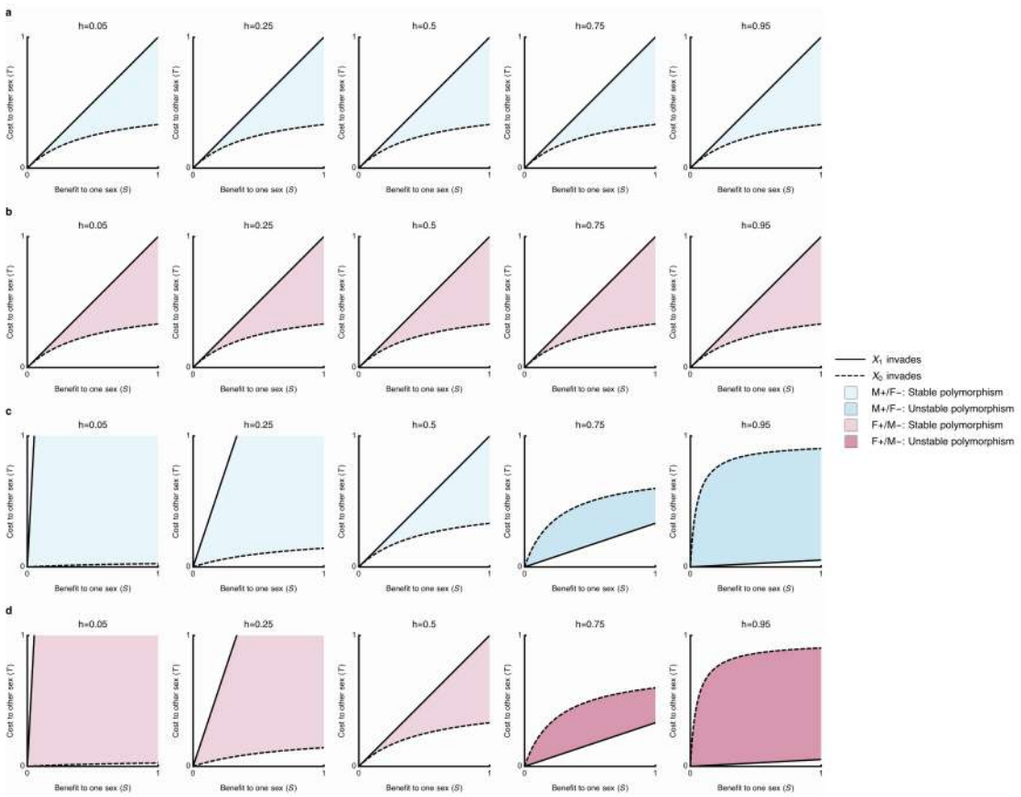

Figure S2: Invasion conditions on an autosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete outbreeding ($\phi = 0$), and fitness effects affect fertility. **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

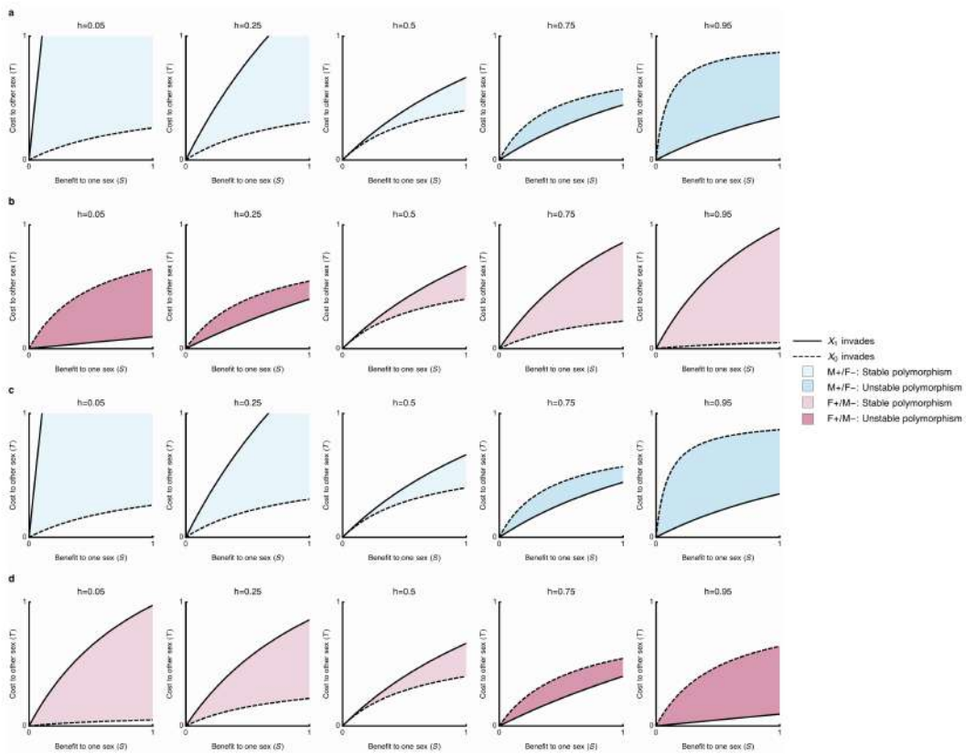

Figure S3: Invasion conditions on the X chromosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete outbreeding ($\phi = 0$), and fitness effects affect fertility. **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

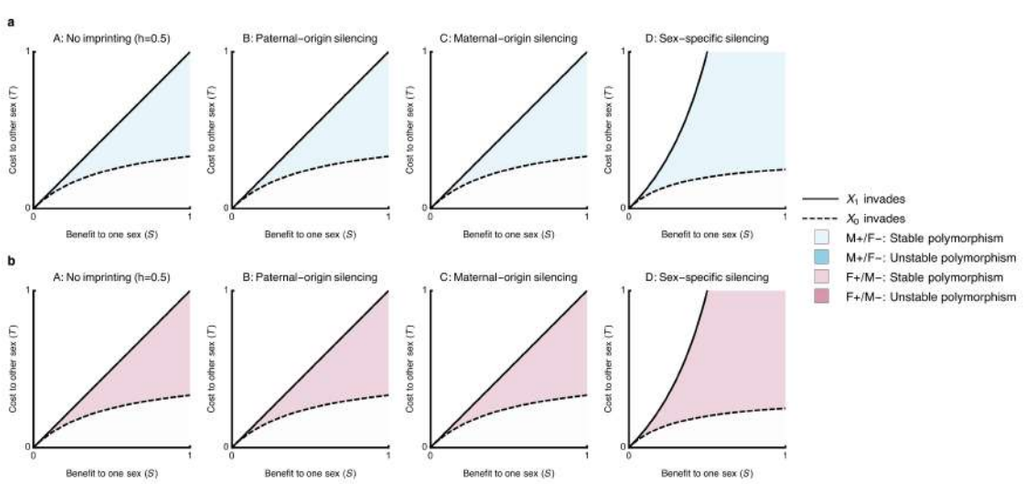

Figure S4: Invasion conditions on an autosome for the mutant allele (solid lines), and the resident allele (dashed lines),when there is complete outbreeding ($\phi = 0$), and fitness effects affect fertility. **a)** Where the allele is beneficial in males, costly in females. **b)** Where the allele is beneficial in females, costly in males.

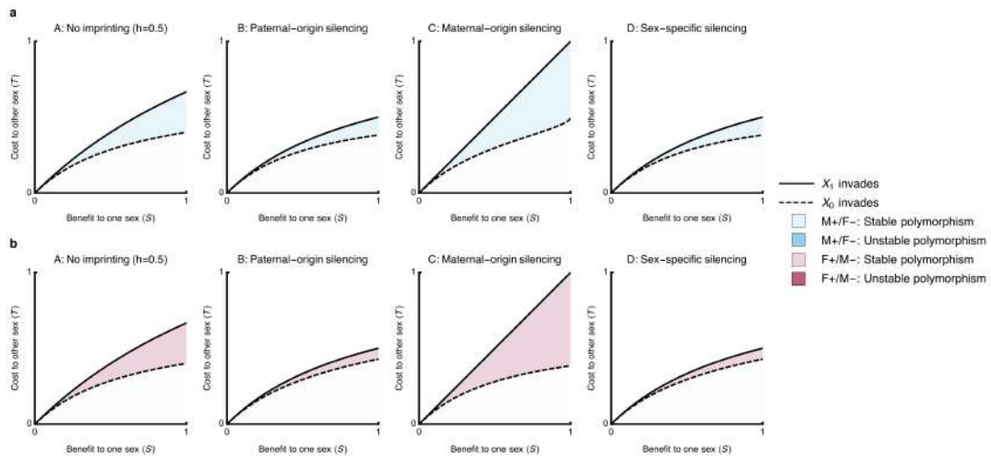

Figure S5: Invasion conditions on an X chromosome for the mutant allele (solid lines), and the resident allele (dashed lines),when there is complete outbreeding ($\phi = 0$), and fitness effects affect fertility. **a)** Where the allele is beneficial in males, costly in females. **b)** Where the allele is beneficial in females, costly in males.

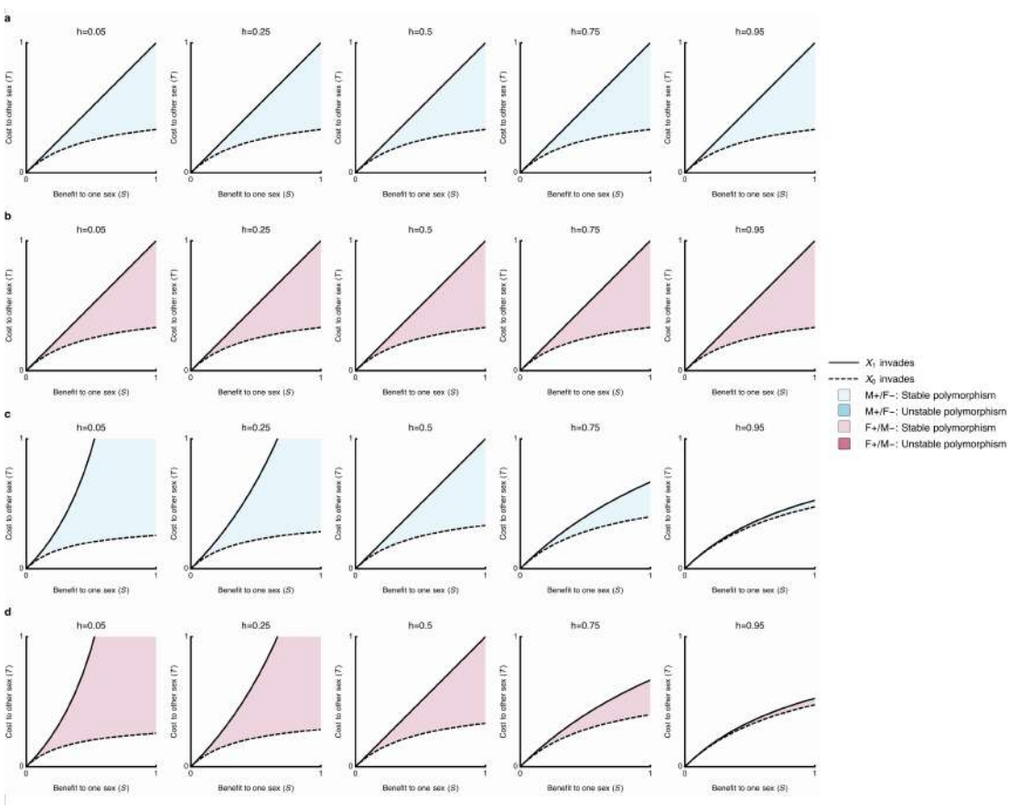

Figure S6: Invasion conditions on an autosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete inbreeding ($\phi = 1$), and fitness effects affect fertility. **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

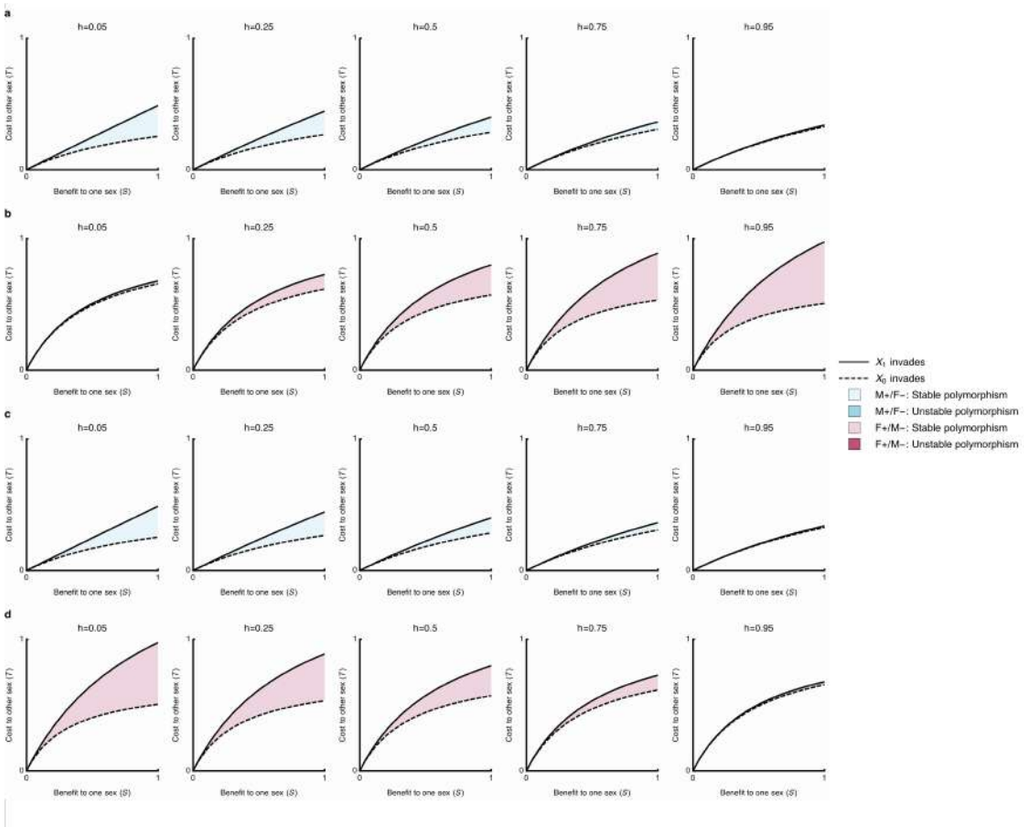

Figure S7: Invasion conditions on the X chromosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete inbreeding ($\phi = 1$), and fitness effects affect fertility. **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

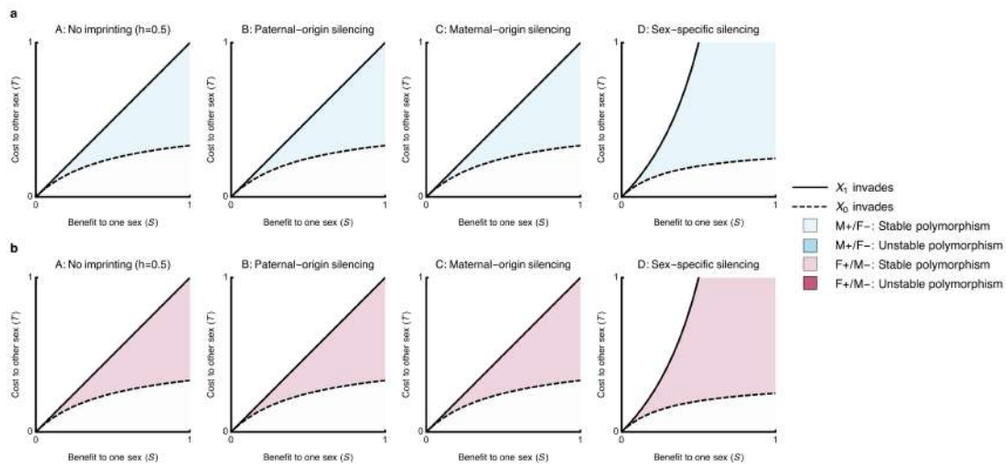

Figure S8: Invasion conditions on an autosome for the mutant allele (solid lines), and the resident allele (dashed lines),when there is complete inbreeding ($\phi = 1$), and fitness effects affect fertility. **a)** Where the allele is beneficial in males, costly in females. **b)** Where the allele is beneficial in females, costly in males.

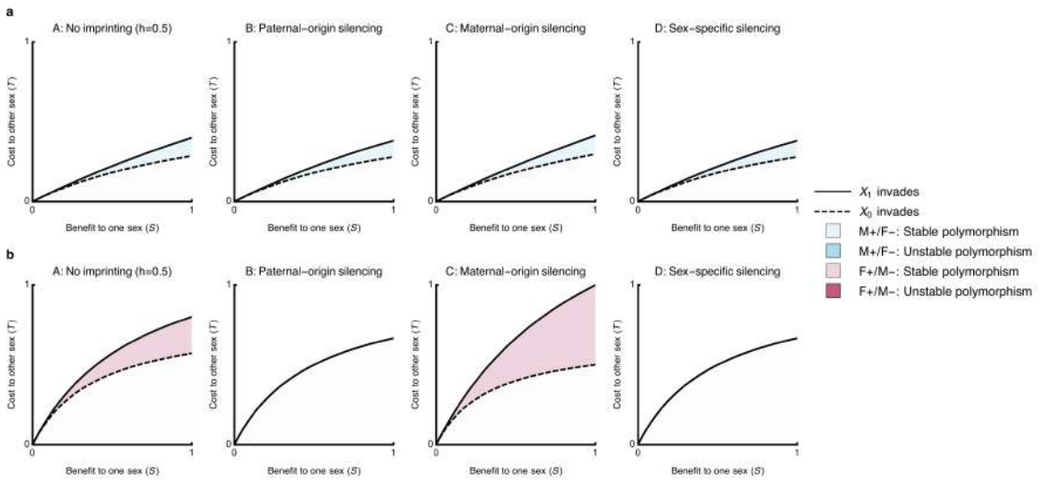

Figure S9: Invasion conditions on an X chromosome for the mutant allele (solid lines), and the resident allele (dashed lines),when there is complete inbreeding ($\phi = 1$), and fitness effects affect fertility. **a)** Where the allele is beneficial in males, costly in females. **b)** Where the allele is beneficial in females, costly in males.

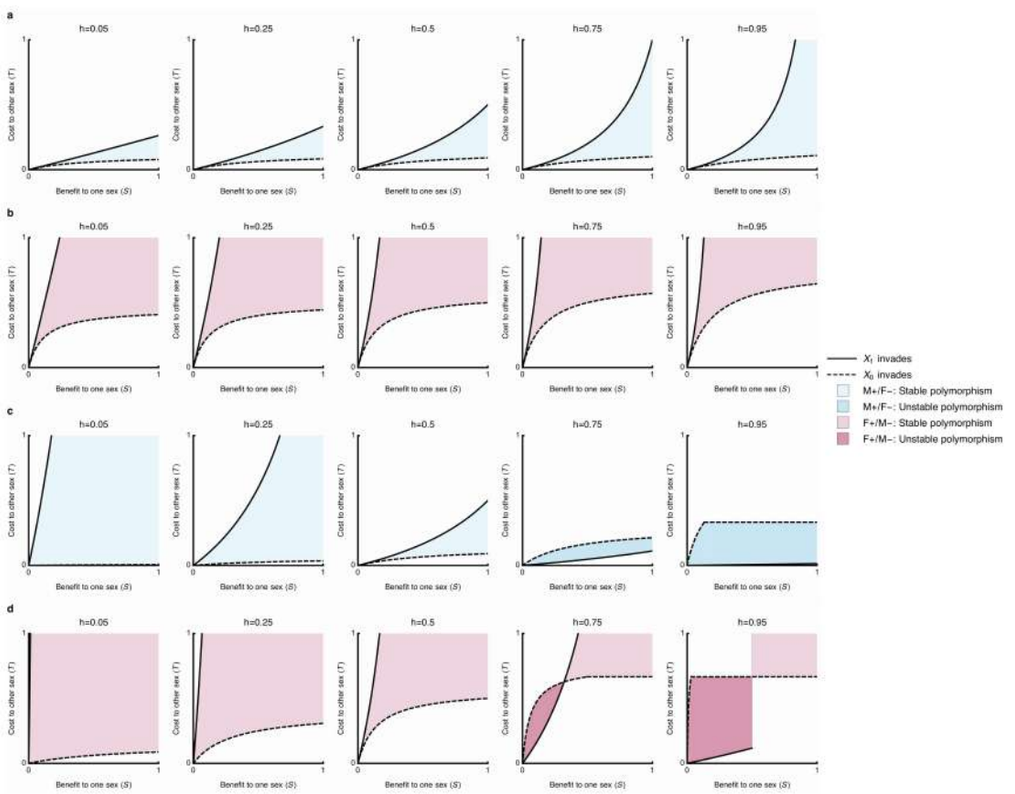

Figure S10: Invasion conditions on an autosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete outbreeding ($\phi = 0$), fitness effects affects survival, and there is higher male mortality ($\mu = 1/3, \nu = 2/3$). **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

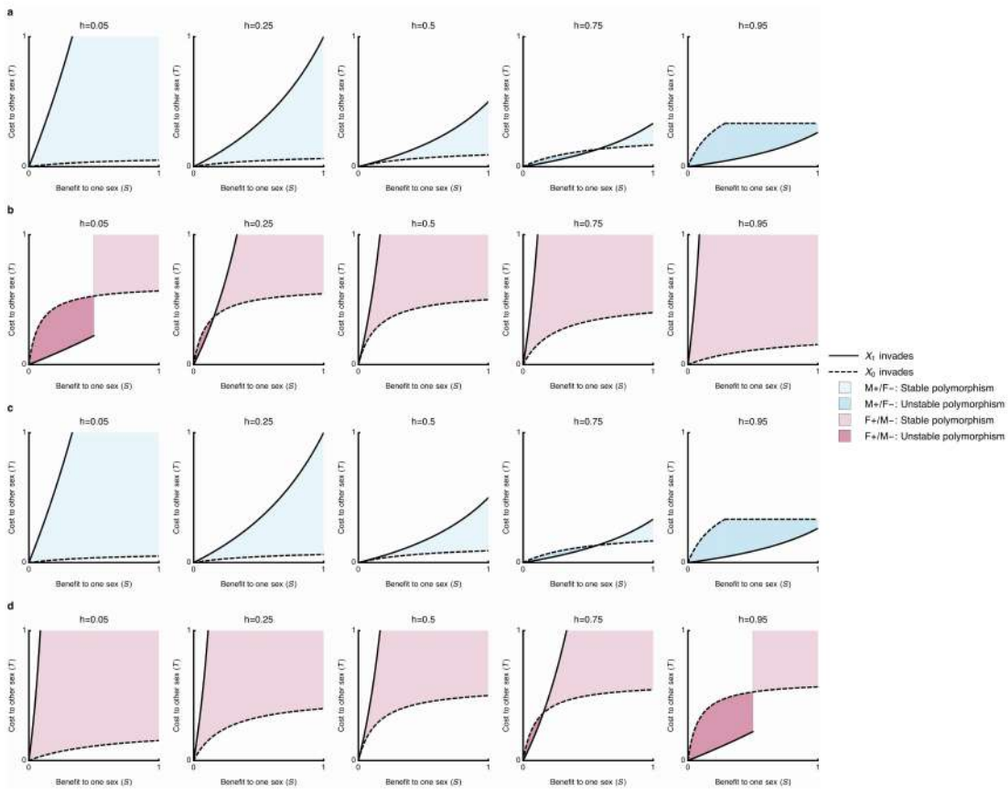

Figure S11: Invasion conditions on the X chromosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete outbreeding ($\phi = 0$), fitness effects affects survival, and there is higher male mortality ($\mu = 1/3, \nu = 2/3$). **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

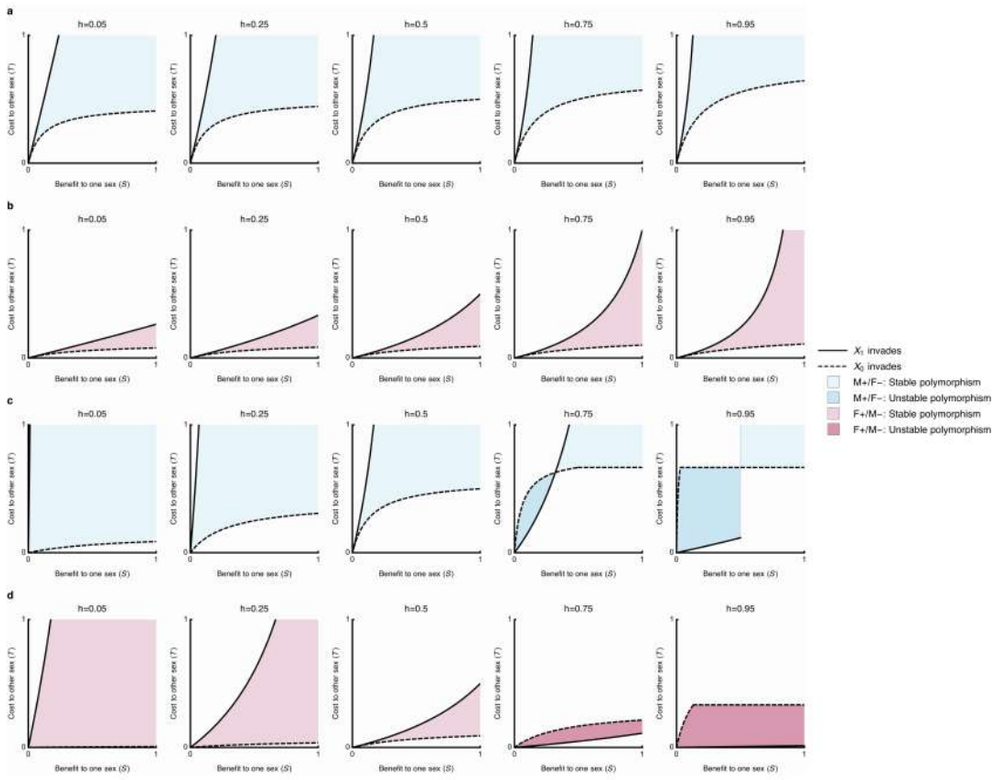

Figure S12: Invasion conditions on an autosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete outbreeding ($\phi = 0$), fitness effects affects survival, and there is higher female mortality ($\mu = 2/3, \nu = 1/3$). **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

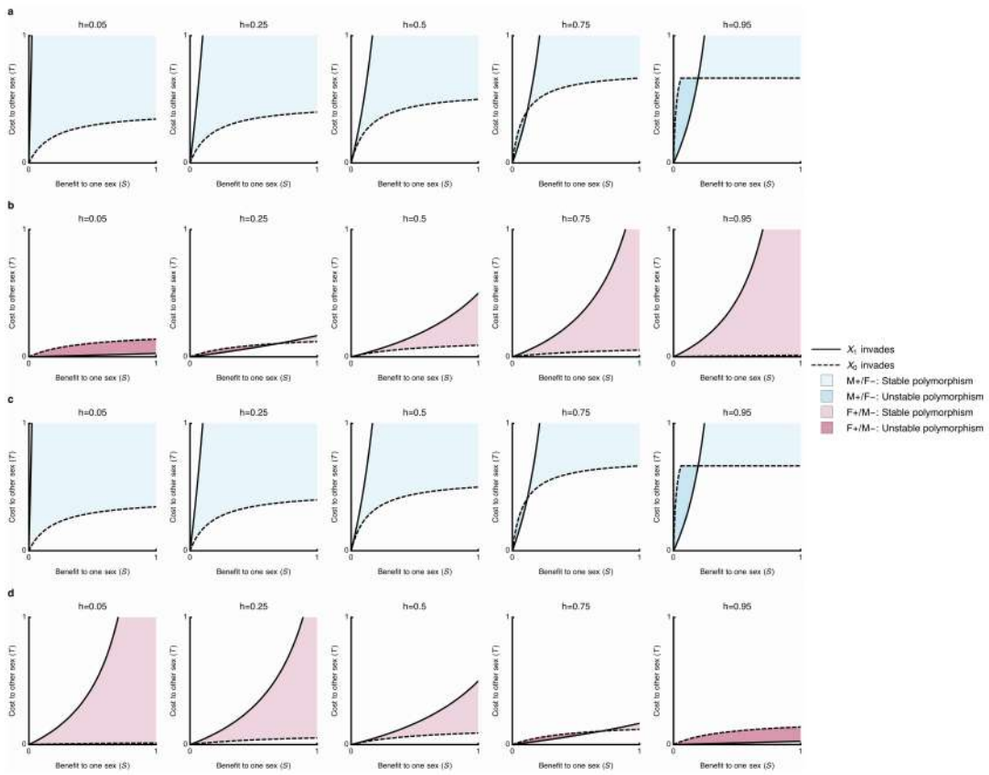

Figure S13: Invasion conditions on the X chromosome for the mutant allele (solid lines), and the resident allele (dashed lines), when there is complete outbreeding ($\phi = 0$), fitness effects affects survival, and there is higher female mortality ($\mu = 2/3, \nu = 1/3$). **a)** Dominance equal in the two sexes where allele is beneficial in males, costly in females. **b)** Dominance equal in the two sexes where allele is beneficial in females, costly in males. **c)** Reversals of dominance, where the allele is beneficial in males, costly in females. **d)** Reversals of dominance, where the allele is beneficial in females, costly in males.

5 Tables

		X_1	X_0
No PoO Effects	M+/F-	$T < \frac{h_m S}{h_f}$	$T > \frac{(h_m-1)S}{h_f-1}$
	F+/M-	$T < \frac{h_f S}{h_m}$	$T > \frac{(1-h_f)S}{1-h_m}$
All other scenarios	M+/F-	$T < S$	$T > S$
	F+/M-	$T < S$	$T > S$

Table S4: Weak selection approximations for invasion conditions for autosomal genes, with selection acting on fertility effects and full outbreeding ($\phi = 0$).

		X_1	X_0
No PoO Effects	M+/F-	$\frac{h_f T}{h_m} < S$	$\frac{T(1-h_f)}{T(2-h_f-h_m)+(1-h_m)} > S$
	F+/M-	$T < \frac{h_f S}{h_m}$	$T > \frac{(1-h_f)S}{S(2-h_f-h_m)+(1-h_m)}$
Paternal-origin silencing	M+/F-	$T < S$	$\frac{T}{1-2T} > S$
	F+/M-	$T < S$	$T > \frac{S}{2S+1}$
Maternal-origin silencing	M+/F-	$T < S$	$\frac{T}{1-2T} > S$
	F+/M-	$T < S$	$T > \frac{S}{2S+1}$
Sex-specific imprinting	M+/F-	$\frac{T}{T+1} < S$	$\frac{T}{1-3T} > S$
	F+/M-	$T < \frac{S}{1-S}$	$T > \frac{S}{3S+1}$
Haploid selection	M+/F-	$T < S$	$\frac{T}{1-2T} > S$
	F+/M-	$T < S$	$T > \frac{S}{2S+1}$

Table S5: Invasion conditions for autosomal genes, with selection acting on fertility effects and full outbreeding ($\phi = 0$).

		X_1	X_0
No PoO Effects	M+/F-	$\frac{2h_f T}{\gamma+(1-\gamma)h_m} < S$	$\frac{2(1-h_f)T}{\gamma+h_m(1-\gamma)} > S$
	F+/M-	$T < \frac{2h_f S}{\gamma+h_m(1-\gamma)}$	$T > \frac{2(1-h_f)S}{\gamma+h_m(1-\gamma)}$
All imprinting	M+/F-	$T < S$	$T > S$
	F+/M-	$T < S$	$T > S$
Haploid selection	M+/F-	$T < \frac{S}{2}$	$T > \frac{S}{2}$
	F+/M-	$T < 2S$	$T > 2S$

Table S6: Weak selection approximations of invasion conditions for X-linked genes, with selection acting on fertility effects and full outbreeding ($\phi = 0$).

		X_1	X_0
No PoO Effects	M+/F-	$\frac{2h_fT}{(1-h_fT)(\gamma+(1-\gamma)h_m)} < S$	$\frac{2(1-h_f)T}{(1+(h_f-2)T)(\gamma+h_m(1-\gamma))} > S$
	F+/M-	$T < \frac{2h_fS}{(h_fS+1)(\gamma+(1-\gamma)h_m)}$	$T > \frac{2(1-h_f)S}{(1+(2-h_f)S)(\gamma+(1-\gamma)h_m)}$
Paternal-origin silencing	M+/F-	$\frac{(1+\rho_f)T}{1+\rho_f-T} < S$	$T > \frac{(\rho_f+1)S}{\rho_f+2\rho_fS+S+1}$
	F+/M-	$\frac{(\rho_f+1)T}{1+\rho_f-T} < S$	$T > \frac{(1+\rho_f)S}{1+\rho_f+2\rho_fS+S}$
Maternal-origin silencing	M+/F-	$\frac{(1+\rho_f)T}{1+\rho_f-\rho_fT} < S$	$T > \frac{(1+\rho_f)S}{1+2S+\rho_f+S\rho_f}$
	F+/M-	$\frac{(1+\rho_f)T}{1+\rho_f-\rho_fT} < S$	$T > \frac{(1+\rho_f)S}{1+2S+\rho_f+\rho_fS}$
Sex-specific imprinting	M+/F-	$\frac{(1+\rho_f)T}{1+\rho_f-T} < S$	$T > \frac{(1+\rho_f)S}{1+S+\rho_f+2S\rho_f}$
	F+/M-	$\frac{(1+\rho_f)T}{1+\rho_f-T} < S$	$T > \frac{(1+\rho_f)S}{1+S+\rho_f+2S\rho_f}$
Haploid selection	M+/F-	$-\frac{2T}{T-1} < S$	$\frac{2T}{1-2T} > S$
	F+/M-	$T < \frac{2S}{S+1}$	$T > \frac{2S}{2S+1}$

Table S7: Invasion conditions for X-linked genes, with selection acting on fertility effects and full outbreeding ($\phi = 0$), where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

		X_1	X_0
All scenarios	M+/F-	$\frac{(1-\mathbb{P}_m)T}{1-\mathbb{P}_f} < S$	$\frac{(1-\mathbb{P}_m)T}{1-\mathbb{P}_f} > S$
	F+/M-	$T < \frac{(1-\mathbb{P}_m)S}{1-\mathbb{P}_f}$	$T > \frac{(1-\mathbb{P}_m)S}{1-\mathbb{P}_f}$

Table S8: Weak selection approximations for invasion conditions for mitochondrial genes, with selection acting on fertility effects.

		X_1	X_0
All scenarios	M+/F-	$\frac{(1-\mathbb{P}_m)T}{T(1-\mathbb{P}_f-\mathbb{P}_m)+(1-\mathbb{P}_f)} < S$	$\frac{(1-\mathbb{P}_m)T}{1-\mathbb{P}_f-T} > S$
	F+/M-	$T < \frac{(1-\mathbb{P}_m)S}{S(1-\mathbb{P}_f-\mathbb{P}_m)+(1-\mathbb{P}_f)}$	$T > \frac{(1-\mathbb{P}_m)S}{1-\mathbb{P}_f}$

Table S9: Invasion conditions for mitochondrial genes, with selection acting on fertility effects.

		X_1	X_0
No PoO Effects	M+/F-	$\frac{T(h_f(-\phi)+h_f+\phi)}{h_m(-\phi)+h_m+\phi} < S$	$\frac{T(h_f(\phi-1)+1)}{h_m(\phi-1)+1} > S$
	F+/M-	$T < \frac{S(h_f(-\phi)+h_f+\phi)}{h_m(-\phi)+h_m+\phi}$	$T > \frac{S(h_f(\phi-1)+1)}{h_m(\phi-1)+1}$
All other scenarios	M+/F-	$T < S$	$T > S$
	F+/M-	$T < S$	$T > S$

Table S10: Weak selection approximations for invasion conditions for autosomal genes, with selection acting on fertility effects with inbreeding.

		X_1	X_0
No PoO Effects	M+/F-	$\frac{T(h_f(-\phi)+h_f+\phi)}{-h_fT\phi+h_m(T-1)\phi+h_m+\phi} < S$	$\frac{h_fT(\phi-1)+T}{(h_f-2)T+h_m(-2T\phi+T+\phi-1)+1} > S$
	F+/M-	$T < \frac{S(h_f(-\phi)+h_f+\phi)}{-h_fS\phi+h_m(S-1)\phi+h_m+\phi}$	$T > \frac{h_fS(\phi-1)+S}{S(2h_f\phi-h_f+2)+h_m(-S+\phi-1)+1}$
Paternal-origin silencing	M+/F-	$T < S$	$\frac{T}{1-2T} > S$
	F+/M-	$T < S$	$T > \frac{S}{2S+1}$
Maternal-origin silencing	M+/F-	$T < S$	$\frac{T}{1-2T} > S$
	F+/M-	$T < S$	$T > \frac{S}{2S+1}$
Sex-specific imprinting	M+/F-	$\frac{T}{T+1} < S$	$\frac{T}{1-3T} > S$
	F+/M-	$T < \frac{S}{1-S}$	$T > \frac{S}{3S+1}$
Haploid selection	M+/F-	$T < S$	$\frac{T}{1-2T} > S$
	F+/M-	$T < S$	$T > \frac{S}{2S+1}$

Table S11: Invasion conditions for autosomal genes, with selection acting on fertility effects with inbreeding.

		X_1	X_0
No PoO Effects	M+/F-	$\frac{2T(h_f(-\phi)+h_f+\phi)}{\gamma-_m+h_m} < S$	$\frac{2T(h_f(\phi-1)+1)}{\gamma-\gamma h_m+h_m} > S$
	F+/M-	$T < -\frac{2S(h_f(-\phi)+h_f+\phi)}{(\gamma-1)h_m-\gamma}$	$T > \frac{2S(h_f(\phi-1)+1)}{\gamma-\gamma h_m+h_m}$
All imprinting	M+/F-	$T(\phi+1) < S$	$T(\phi+1) > S$
	F+/M-	$T < S(\phi+1)$	$T > S(\phi+1)$
Haploid selection	M+/F-	$2T < S$	$2T > S$
	F+/M-	$T < 2S$	$T > 1S$

Table S12: Weak selection approximations of invasion conditions for X-linked genes, with selection acting on fertility effects and inbreeding.

		X_1	X_0
No PoO Effects	M+/F-	$-\frac{2T(h_f(-\phi)+h_f+\phi)}{(h_fT-1)(\gamma-\gamma h_m+h_m)} < S$	$\frac{2T(h_f(\phi-1)+1)}{((h_f-2)T+1)(\gamma-\gamma h_m+h_m)} > S$
	F+/M-	$T < \frac{2S(h_f(-\phi)+h_f+\phi)}{((\gamma-1)h_m-\gamma)(2(h_f-1)S\phi-h_fS-1)}$	$T > \frac{2S(h_f(\phi-1)+1)}{(\gamma-\gamma h_m+h_m)(S(h_f(2\phi-1)+2)+1)}$
Paternal-origin silencing	M+/F-	$\frac{T(\phi+1)(\rho_f(T\phi+2)-T\phi+2)}{2(\rho_f-T+1)} < S$	$-\frac{T(\phi+1)((3T-2)\rho_f+T-2)}{(T(5T-6)+2)\rho_f+(T-4)T+2} > S$
	F+/M-	$T < \frac{S(\phi+1)(\rho_f+1)}{\rho_f+S\phi(\rho_f+1)+S+1}$	$T > \frac{S(\phi+1)(\rho_f(S(\phi-3)-2)-S(\phi+1)-2)}{\rho_f(S^2((\phi-2)\phi-5)-2S(\phi+3)-2)-S(S(\phi+1)^2+2(\phi+2))-2}$
Maternal-origin silencing	M+/F-	$\frac{T(\phi+1)(\rho_f(T\phi-2)-T\phi-2)}{2(T-1)\rho_f-2} < S$	$-\frac{T(\phi+1)((T-2)\rho_f+3T-2)}{((T-4)T+2)\rho_f+T(5T-6)+2} > S$
	F+/M-	$T < \frac{S(\phi+1)(\rho_f+1)}{\rho_f(S\phi+S+1)+S\phi+1}$	$T > \frac{S(\phi+1)(\rho_f(S\phi+S+2)-S(\phi-3)+2)}{\rho_f(S^2(\phi+1)^2+2S(\phi+2)+2)+S^2(5-(\phi-2)\phi)+2S(\phi+3)+2}$
Sex-specific imprinting	M+/F-	$\frac{T(\phi+1)(-\rho_f(T\phi+2)+T\phi-2)}{2(-\rho_f+T-1)} < S$	$-\frac{T(\phi+1)((3T-2)\rho_f+T-2)}{(T(5T-6)+2)\rho_f+(T-4)T+2} > S$
	F+/M-	$T < \frac{S(\phi+1)(\rho_f+1)}{\rho_f+S\phi(\rho_f+1)+S+1}$	$T > \frac{S(\phi+1)(\rho_f(S(\phi-3)-2)-S(\phi+1)-2)}{\rho_f(S^2((\phi-2)\phi-5)-2S(\phi+3)-2)-S(S(\phi+1)^2+2(\phi+2))-2}$
Haploid selection	M+/F-	$-\frac{2T}{T-1} < S$	$\frac{2T}{1-2T} > S$
	F+/M-	$T < \frac{2S}{S+1}$	$T > \frac{2S}{2S+1}$

Table S13: Invasion conditions for X-linked genes, with selection acting on fertility effects and full outbreeding ($\phi = 0$), where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

		X_1	X_0
No PoO Effects	M+/F-	$T < \frac{(1-\rho_f)\rho_m S(h_m(1-\phi)+\phi)}{\rho_f(1-\rho_m)(h_f(1-\phi)+\phi)}$	$T > \frac{(1-\rho_f)\rho_m S(1-h_m(1-\phi))}{\rho_f(1-\rho_m)(1-h_f(1-\phi))}$
	F+/M-	$T < \frac{\rho_f(1-\rho_m)S(h_f(1-\phi)+\phi)}{(1-\rho_f)\rho_m(h_m(1-\phi)+\phi)}$	$T > \frac{\rho_f(1-\rho_m)S(1-h_f(1-\phi))}{(1-\rho_f)\rho_m(1-h_m(1-\phi))}$
All imprinting	M+/F-	$T < \frac{(1-\rho_f)\rho_m S}{\rho_f(1-\rho_m)}$	$T > \frac{(1-\rho_f)\rho_m S}{\rho_f(1-\rho_m)}$
	F+/M-	$T < \frac{\rho_f(1-\rho_m)S}{(1-\rho_f)\rho_m}$	$T > \frac{\rho_f(1-\rho_m)S}{(1-\rho_f)\rho_m}$

Table S14: Weak selection approximations for invasion conditions for autosomal genes, with selection acting on survival effects, where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

		X_1	X_0
No PoO Effects	M+/F-	$\min[\frac{1-\rho_m}{\rho_m}, \frac{h_f\rho_f T - h_f\rho_f\rho_m T}{h_m\rho_m(2h_f\rho_f T-\rho_f+1)}] < S$	$T > \min[1-\rho_f, \frac{(1-h_m)(1-\rho_f)\rho_m S}{\rho_m(\rho_f(h_f(2h_m-1)S+h_f-h_mS-1)-h_mS+S)+(1-h_f)\rho_f(S+1)}]$
	F+/M-	$\min[\frac{1-\rho_f}{\rho_f}, \frac{h_m\rho_m T - h_m\rho_f\rho_m T}{h_f\rho_f(2h_m\rho_m T-\rho_m+1)}] < S$	$T > \min[1-\rho_m, \frac{(h_f-1)\rho_f(\rho_m-1)S}{2h_fh_m\rho_f\rho_mS-h_f\rho_f\rho_mS-h_f\rho_fS+h_m\rho_f\rho_m-h_m\rho_m-h_m\rho_f\rho_mS-h_m\rho_mS-\rho_f\rho_m+\rho_m+\rho_fS+\rho_mS}]$
Paternal-origin silencing	M+/F-	$\frac{\rho_f(1-\rho_m)T}{\rho_m(-\rho_f+2\rho_fT+1)} < S$	$T > \frac{(1-\rho_f)\rho_mS}{-\rho_f\rho_m+\rho_f+\rho_fS+\rho_mS}$
	F+/M-	$\frac{(1-\rho_f)\rho_mT}{\rho_f(-\rho_m+2\rho_mT+1)} < S$	$T > \frac{\rho_f(1-\rho_m)S}{-\rho_f\rho_m+\rho_m+\rho_fS+\rho_mS}$
Maternal-origin silencing	M+/F-	$\frac{\rho_f(1-\rho_m)T}{\rho_m(-\rho_f+2\rho_fT+1)} < S$	$T > \frac{(1-\rho_f)\rho_mS}{-\rho_f\rho_m+\rho_f+\rho_fS+\rho_mS}$
	F+/M-	$\frac{(1-\rho_f)\rho_mT}{\rho_f(-\rho_m+2\rho_mT+1)} < S$	$T > \frac{\rho_f(1-\rho_m)S}{-\rho_f\rho_m+\rho_m+\rho_fS+\rho_mS}$
Sex-specific imprinting	M+/F-	$\frac{\rho_f(1-\rho_m)T}{\rho_m(-\rho_f+3\rho_fT+1)} < S$	$T > \frac{(1-\rho_f)\rho_mS}{-\rho_f\rho_m+\rho_f+\rho_f\rho_mS+\rho_fS+\rho_mS}$
	F+/M-	$\frac{(1-\rho_f)\rho_mT}{\rho_f(-\rho_m+3\rho_mT+1)} < S$	$T > \frac{\rho_f(1-\rho_m)S}{-\rho_f\rho_m+\rho_m+\rho_f\rho_mS+\rho_fS+\rho_mS}$

Table S15: Invasion conditions for autosomal genes, with selection acting on survival effects and full outbreeding ($\phi = 0$), where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

		X_1	X_0
No PoO Effects	M+/F-	$T < \frac{(1-\rho_f)\rho_m S(\gamma+(1-\gamma)h_m)}{2\rho_f(1-\rho_m)(h_f(-\phi)+h_f+\phi)}$	$T > \frac{(1-\rho_f)\rho_m S(\gamma+(1-\gamma)h_m)}{2\rho_f(1-\rho_m)(1-h_f(1-\phi))}$
	F+/M-	$T < \frac{2\rho_f(1-\rho_m)S(h_f(1-\phi)+\phi)}{(1-\rho_f)\rho_m(\gamma(1-h_m)+h_m)}$	$T > \frac{2\rho_f(1-\rho_m)S(h_f(1-\phi)+\phi)}{(1-\rho_f)\rho_m(\gamma(1-h_m)+h_m)}$
All imprinting	M+/F-	$T < \frac{(1-\rho_f)\rho_m S}{\rho_f(1-\rho_m)(\phi+1)}$	$T > \frac{(1-\rho_f)\rho_m S}{\rho_f(1-\rho_m)(\phi+1)}$
	F+/M-	$T < \frac{\rho_f(1-\rho_m)S(\phi+1)}{(1-\rho_f)\rho_m}$	$T > \frac{\rho_f(1-\rho_m)S(\phi+1)}{(1-\rho_f)\rho_m}$

Table S16: Weak selection approximations for invasion conditions for X-linked genes, with selection acting on survival effects, where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

		X_1	X_0
No PoO Effects	M+/F-	$\frac{2h_f\rho_f(1-\rho_m)T}{\rho_m(\gamma+(1-\gamma)h_m)(1-\rho_f(1-2h_fT))} < S$	$T > \min[1-\rho_f, \frac{(1-\rho_f)\rho_m S(\gamma+(1-\gamma)h_m)}{\rho_f(2h_f((\gamma-1)h_mS+\rho_m-\gamma S-1)+(\gamma-1)h_m(\rho_m-2)S-2\rho_m-\gamma\rho_mS+2\gamma S+2)+\rho_mS(\gamma-\gamma h_m+h_m)}]$
	F+/M-	$\min[\frac{1-\rho_f}{\rho_f}, \frac{(1-\rho_f)\rho_mT(\gamma+(1-\gamma)h_m)}{2h_f\rho_f((1-\gamma)h_m\rho_mT-\rho_m+\gamma\rho_mT+1)}] < S$	$T > \frac{2(1-h_f)\rho_f(1-\rho_m)S}{(\gamma+(1-\gamma)h_m)(\rho_fS(-2h_f-\rho_m+2)+(1-\rho_f)\rho_m+\rho_mS)}$
Paternal-origin silencing	M+/F-	$\frac{\rho_f(\rho_f+1)(1-\rho_m)T}{\rho_m+\rho_f^2\rho_m(2T-1)} < S$	$T > \min[1-\rho_f, \frac{\left(1-\rho_f^2\right)\rho_mS}{\rho_f^2(-\rho_m+S+1)-\rho_f(\rho_m-1)(S+1)+\rho_mS}]$
	F+/M-	$\min[\frac{1-\rho_f}{\rho_f}, \frac{\left(1-\rho_f^2\right)\rho_mT}{\rho_f(\rho_f-\rho_m+\rho_f\rho_m(2T-1)+1)}] < S$	$T > \frac{\rho_f(\rho_f+1)(1-\rho_m)S}{\rho_f^2(-\rho_m)+\rho_m+S\left(\rho_f^2-\rho_f\rho_m+\rho_f+\rho_m\right)}$
Maternal-origin silencing	M+/F-	$\frac{\rho_f(1-\rho_m)T}{\rho_m-\rho_f\rho_m(1-T)} < S$	$T > \min[1-\rho_f, \frac{(1-\rho_f)\rho_mS}{\rho_f(1-\rho_m)(S+1)+\rho_mS}]$
	F+/M-	$\min[\frac{1-\rho_f}{\rho_f}, \frac{(1-\rho_f)\rho_mT}{\rho_f-\rho_f\rho_m(1-T)}] < S$	$T > \frac{\rho_f(1-\rho_m)S}{(1-\rho_f)\rho_m+\rho_f(1-\rho_m)S+\rho_mS}$
Sex-specific imprinting	M+/F-	$\frac{\rho_f(\rho_f+1)(1-\rho_m)T}{\rho_f^2\rho_m\rho_m(1-2T)} < S$	$T > 1-\rho_f, \frac{\left(1-\rho_f^2\right)\rho_mS}{\rho_f^2(-\rho_m+S+1)+\rho_f(1-\rho_m)(S+1)+\rho_mS}$
	F+/M-	$\frac{1-\rho_f}{\rho_f}, \frac{\left(1-\rho_f^2\right)\rho_mT}{\rho_f(\rho_f-\rho_m+\rho_f\rho_m(-(1-2T))+1)} < S$	$T > \frac{\rho_f(\rho_f+1)(1-\rho_m)S}{\rho_f^2(-\rho_m)+\rho_m+S\left(\rho_f^2-\rho_f\rho_m+\rho_f+\rho_m\right)}$

Table S17: Invasion conditions for X-linked genes, with selection acting on survival effects under full outbreeding ($\phi = 0$),where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

		X_1	X_0
No PoO Effects	M+/F-	$T < \frac{(1-\mathbb{P}_f)(1-\rho_f)\rho_m S}{(1-\mathbb{P}_m)\rho_f(1-\rho_m)}$	$T > \frac{(1-\mathbb{P}_f)(1-\rho_f)\rho_m S}{(1-\mathbb{P}_m)\rho_f(1-\rho_m)}$
	F+/M-	$T < \frac{(1-\mathbb{P}_m)\rho_f(1-\rho_m)S}{(1-\mathbb{P}_f)(1-\rho_f)\rho_m}$	$T > \frac{(1-\mathbb{P}_m)\rho_f(1-\rho_m)S}{(1-\mathbb{P}_f)(1-\rho_f)\rho_m}$

Table S18: Weak selection approximations for invasion conditions for mitochondrial genes, with selection acting on survival effects, where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

		X_1	X_0
All scenarios	M+/F-	$\frac{(1-\mathbb{P}_m)\rho_f(1-\rho_m)T}{\rho_m(-\mathbb{P}_f(1-\rho_f)-\rho_f(1-T)+1)} < S$	$T > \frac{(1-\mathbb{P}_f)(1-\rho_f)\rho_m S}{(1-\mathbb{P}_m)\rho_f(1-\rho_m)+\rho_f S(-(1-\mathbb{P}_f)\rho m-\mathbb{P}_m(1-\rho_m)+1)+(1-\mathbb{P}_f)\rho_m S}$
	F+/M-	$\frac{(1-\mathbb{P}_f)(1-\rho_f)\rho_m T}{\rho_f(-\mathbb{P}_m(1-\rho_m)-\rho_m(1-T)+1)} < S$	$T > \frac{(1-\mathbb{P}_m)\rho_f(1-\rho_m)S}{(1-\mathbb{P}_f)(1-\rho_f)\rho_m+S(-(1-\mathbb{P}_f)\rho_f\rho_m-\mathbb{P}_f\rho_m-\mathbb{P}_m\rho_f(1-\rho_m)+\rho_f+\rho_m)}$

Table S19: Invasion conditions for mitochondrial genes, with selection acting on survival effects, where $\rho_f = (1-\mu)/\lambda$, and $\rho_m = (1-\nu)/\lambda$.

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