Mating ecology explains patterns of genome elimination

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Abstract
Genome elimination – whereby an individual discards chromosomes inherited from one parent, and transmits only those inherited from the other parent – is found across thousands of animal species. It is more common in association with inbreeding, under male heterogamety, in males, and in the form of paternal genome elimination. However, the reasons for this broad pattern remain unclear. We develop a mathematical model to determine how degree of inbreeding, sex determination, genomic location, pattern of gene expression and parental origin of the eliminated genome interact to determine the fate of genome-elimination alleles. We find that: inbreeding promotes paternal genome elimination in the heterogametic sex; this may incur population extinction under female heterogamety, owing to eradication of males; and extinction is averted under male heterogamety, owing to countervailing sex-ratio selection. Thus, we explain the observed pattern of genome elimination. Our results highlight the interaction between mating system, sex-ratio selection and intragenomic conflict.

Keywords
Extinction, genomic imprinting, haplodiploidy, inbreeding, meiotic drive, paternal genome elimination, paternal genome loss, sex determination, sex ratio, sib-mating.


INTRODUCTION

Under standard mendelian inheritance, individuals receive one set of chromosomes from each of their parents, and transmit one set of chromosomes to each of their offspring, without bias according to each chromosome’s parent of origin. However, across thousands of animal species, some individuals (typically members of one sex) systematically transmit only those chromosomes that they inherited from a particular parent (Table 1 and Fig. 1; Burt & Trivers 2006). For example, in the citrus mealybug Planococcus citri, a male’s sperm carry only the chromosomes he inherited from his mother, all paternal chromosomes having been eliminated, whereas a female’s oocytes carry chromosomes from both of her parents. This ‘genome elimination’ (GE) is a whole-genome form of meiotic drive and, accordingly, its evolutionary rationale makes sense from a selfish-gene perspective: a gene that ensures it is passed on to all — as opposed to only half — of an individual’s offspring enjoys a two-fold selective advantage, and may increase in frequency unless the number of surviving offspring is more than halved as a consequence (Bull 1979). Indeed, the real evolutionary puzzle is to explain why GE occurs only in some species and not in others (Normark 2004).

There is a clear pattern in the incidence and type of GE that occurs in the animal kingdom: it is commonly found in association with inbreeding, under male heterogamety, in males, and in the form of paternal genome elimination (Table 1 and Fig. 1). This suggests that a species’ mating ecology is an important factor in predisposing it to GE. A rich literature spanning a century of work in ecology, population genetics and cytology has yielded several hypotheses as to how inbreeding impacts upon the evolution of GE, both directly and in its interaction with a species’ sex determination system (Table 2). However, these ideas lead to different — indeed, sometimes diametrically opposite — predictions, and the complexity of the problem means that a full, quantitative analysis is lacking (Bull 1983; Burt & Trivers 2006).

Here, we develop a mathematical kin selection model to determine how degree of inbreeding, mode of sex determination, genomic location, pattern of gene expression and parental origin of the eliminated genome interact to determine the fate of GE alleles. We identify those scenarios under which GE may arise in the population by performing invasion analyses, and we identify those scenarios in which GE may be maintained in the population by performing equilibrium analyses and assessing the impact of GE upon population viability. Our aim is to assess the constraints that a species’ mating ecology imposes upon its ability to evolve — and survive — GE, thereby explaining its pattern of incidence in the animal world. Although specifically focusing upon GE, our analysis yields general insights into how mating system and sex-ratio selection shape conflicts within and between individuals, with application to sex-chromosome meiotic drive, endosymbiotic parasitism and haplodiploidy.

MATERIALS AND METHODS

Mathematical model
We assume a gonochoristic, diploid population, subdivided into a large number of mating groups, each containing a large number of individuals. We denote by \( a \) the probability that a female and a male, randomly chosen from the same mating group, share the same mother. We assume that each female mates with a large number of males, and at random within her mating group, such that the probability of mating partners sharing the same father is effectively zero. Thus, \( a \) represents the incidence of mating between maternal siblings, and...
Table 1 Overview of all taxonomic groups with PGE

<table>
<thead>
<tr>
<th>Class</th>
<th>Order</th>
<th>Clade</th>
<th>Number of species</th>
<th>Type of PGE</th>
<th>Male soma</th>
<th>Sex chromosomes</th>
<th>Ancestral SD</th>
<th>Sib-mating/Inbreeding*</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collembola</td>
<td>Symphypleona</td>
<td>Symphypleona</td>
<td>1180 (6)</td>
<td>Germline PGE</td>
<td>Diploid with X elimination</td>
<td>XX/X0</td>
<td></td>
<td>Some evidence</td>
<td>(Dallai et al. 2001; Burt &amp; Trivers 2006)</td>
</tr>
<tr>
<td>Insecta</td>
<td>Coleoptera</td>
<td>Cryphalini</td>
<td>190 (1)</td>
<td>Germline PGE</td>
<td>Diploid with paternal genome silencing</td>
<td>No sex chr.</td>
<td>XX/XY</td>
<td>Strong evidence</td>
<td>(Brun et al. 1995; Borsa &amp; Kjellberg 1996; Gauthier 2010)</td>
</tr>
<tr>
<td>Insecta</td>
<td>Diptera</td>
<td>Cecidomyiidae</td>
<td>6168 (22)</td>
<td>Germline PGE</td>
<td>Diploid with X elimination</td>
<td>XX/X0</td>
<td>XX/XY</td>
<td>No evidence</td>
<td>(Painter 1966; Stuart &amp; Hatchett 1991; Burt &amp; Trivers 2006; Benatti et al. 2010)</td>
</tr>
<tr>
<td>Insecta</td>
<td>Diptera</td>
<td>Sciariidae</td>
<td>2300 (10)</td>
<td>Germline PGE</td>
<td>Diploid with X elimination</td>
<td>XX/X0</td>
<td>XX/XY</td>
<td>No evidence</td>
<td>(Metz 1938; Crouse 1960; Hag 1993b; Godoy &amp; Esteban 2001; Sánchez 2010)</td>
</tr>
<tr>
<td>Insecta</td>
<td>Hemiptera</td>
<td>Neococcoidea</td>
<td>4000 (270)</td>
<td>Germline PGE</td>
<td>Diploid with paternal genome silencing</td>
<td>No sex chr.</td>
<td>XX/X0</td>
<td>Some evidence</td>
<td>(Brown &amp; Nur 1964; Nur 1990; Gavrilov 2007; Ross et al. 2010a; Martins et al. 2012)</td>
</tr>
<tr>
<td>Insecta</td>
<td>Hemiptera</td>
<td>Diaspididae</td>
<td>2650 (130)</td>
<td>Embryonic PGE</td>
<td>Haploid (genome loss)</td>
<td>No sex chr.</td>
<td>XX/X0</td>
<td>Some evidence</td>
<td>(Brown &amp; Nur 1964; Nur 1990)</td>
</tr>
</tbody>
</table>

Rows represent independent origins of PGE, except for Diaspididae (indicated by $), which represents change in type of PGE (germline $→$ embryonic). Number of species column: estimate assumes PGE is conserved across whole clade; number of species for which there is direct evidence of PGE given in parentheses. Types of PGE: 'embryonic' when complete paternal genome is eliminated from both soma and germline in early embryogenesis; 'germline' when paternal genome only completely eliminated in the germline during spermatogenesis. Male soma and sex-chromosome information based on karyotype analysis of species with PGE. Ancestral-SD system inferred from karyotype data for diploid sister groups. Inbreeding inferred from $F_{IS}$, mating system and sex-ratio data (WM, wingless males; WF, wingless females; SB, female-biased sex ratios; PG, population-genetic evidence of low genetic diversity and excess homozygosity; PS, life history leading to strong meta-population structure; SM, frequent sib-mating).
varying this key ecological parameter allows us to explore the whole continuum from outbreeding \((a = 0)\) to chronic inbreeding \((a = 1)\). After mating, males die and mated females disperse to new patches, as in Hamilton’s (1967) classic model of local mate competition (LMC).

We consider that GE alleles may be located on autosomes or sex chromosomes, and may induce maternal genome elimination (MGE) or paternal genome elimination (PGE). We allow for the genes to be active in females or males, to induce effects either in the carrier or the carrier’s daughters or sons, and – for genes inducing GE in the carrier – to have different effects if the gene is inherited from the mother or the father (i.e. genomic imprinting; Haig 2002). We assume ‘germline’ GE, in which one parent’s genome is excluded from the focal individual’s gametes, but is present and active in the individual’s somatic tissue, as is this is likely the ancestral form of GE (see Discussion). We assume that GE in females reduces their number of surviving offspring by a fraction \(\alpha\) and that GE in males reduces their number of surviving offspring by a fraction \(\beta\), as a consequence of upsetting normal chromosomal segregation. We assume that sex is determined by the individual’s own genotype, and consider both male (XY and XO) and female (ZW and ZO) heterogamety. An important feature of the model is that GE in the heterogametic sex results in offspring sex ratio bias, towards males for MGE and towards females for PGE (Fig. 2). No sex-ratio bias obtains if GE is absent or restricted to the homogametic sex (Fig. 2).

### Invasion analysis

We analyse our model using the neighbour-modulated-fitness methodology of Taylor & Frank (1996; Supporting Information). This is a recipient-centred approach to kin selection, which considers the impact of social partners on a focal individual’s fitness, and gives the same results as the actor-centred inclusive-fitness approach, which considers the impact of a focal individual on the fitness of her social

### Table: Parent-of-origin-specific genome elimination (GE)

<table>
<thead>
<tr>
<th>Parents</th>
<th>Gametes</th>
<th>Zygote</th>
<th>Soma</th>
<th>Germline</th>
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<td><img src="image3.png" alt="Image" /></td>
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</tr>
<tr>
<td>Fungus gnats</td>
<td><img src="image5.png" alt="Image" /></td>
<td><img src="image6.png" alt="Image" /></td>
<td><img src="image7.png" alt="Image" /></td>
<td><img src="image8.png" alt="Image" /></td>
</tr>
<tr>
<td>Mealybugs</td>
<td><img src="image9.png" alt="Image" /></td>
<td><img src="image10.png" alt="Image" /></td>
<td><img src="image11.png" alt="Image" /></td>
<td><img src="image12.png" alt="Image" /></td>
</tr>
<tr>
<td>Coffee borer beetle</td>
<td><img src="image13.png" alt="Image" /></td>
<td><img src="image14.png" alt="Image" /></td>
<td><img src="image15.png" alt="Image" /></td>
<td><img src="image16.png" alt="Image" /></td>
</tr>
<tr>
<td>Human body louse</td>
<td><img src="image17.png" alt="Image" /></td>
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<td><img src="image19.png" alt="Image" /></td>
<td><img src="image20.png" alt="Image" /></td>
</tr>
<tr>
<td>Armored scale insects</td>
<td><img src="image21.png" alt="Image" /></td>
<td><img src="image22.png" alt="Image" /></td>
<td><img src="image23.png" alt="Image" /></td>
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<tr>
<td>Mites</td>
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<td><img src="image28.png" alt="Image" /></td>
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**Figure 1** Parent-of-origin-specific genome elimination (GE), whereby an individual discards the chromosomes inherited from one parent, and transmits only those inherited from the other parent. We include only those cases where GE is sex-limited. Rows represent independent origins of GE. Column 1: adult generation. Column 2: gametes produced. Column 3: embryos shortly after fertilisation. Column 4: offspring soma. Column 5: offspring germline. Blue: male. Red: female. X: presence of X-chromosome (colour indicates parental origin). L in fungus-gnat entry: a germline-linked chromosome. Blue circle in mealybugs and coffee-borer-beetles entries: complete heterochromatisation of paternal genome. '?' in body-louse entry: lack of information about somatic effects. ‘Germline PGE’: eliminated genome retained throughout development but not transmitted to offspring. ‘Embryonic PGE’: eliminated genome lost early in development, resulting in haploidy.
The invasion condition for natural selection to favor an allele depends on the sex of the parent of origin and the sex of the grandparent of origin. The condition for natural selection to favor an allele increases in frequency from rarity, that is \( d \frac{g}{W} > 0 \), where \( W \) is expected relative fitness, \( g \) is genetic predisposition for GE, and \( g \) is the population frequency of GE (Taylor & Frank 1996). A zygote’s expected fitness depends on its probability of conception and its probability of surviving viability selection (both of which depend on the action of GE in the zygote’s parents) and expected mating success conditional upon surviving to adulthood (which may depend on the action of GE among those parents contributing offspring to the zygote’s future mating group).

Invasion of GE occurs when the corresponding allele increases in frequency from rarity, that is \( d \frac{W}{dg} |_{g} > 0 \). We reformulate this invasion condition to determine the potential for GE (Supporting Information). Specifically, for GE in females, the invasion condition may be expressed in the form \( \alpha < \gamma \), where \( \alpha \) is the actual cost of GE, in terms of number of surviving offspring, and \( \gamma \) is the maximum cost that is tolerated with GE still able to invade, and is a function of model parameters. A positive potential for GE (\( \gamma > 0 \)) indicates that costly GE may invade, a negative potential for GE (\( \gamma < 0 \)) indicates that GE would need to provide a benefit, in terms of number of surviving offspring, in order for it to be able to invade, and zero potential for GE (\( \gamma = 0 \)) indicates that GE cannot invade if it incurs any cost and will invade if it provides any benefit. The potential for GE may also be defined for males, by reformulating the invasion condition as \( \beta < \gamma \). In addition to allowing comparisons and contrasts across different mating ecologies, the potential for GE provides a way of gauging a particular genetic actor’s interests with respect to GE and, hence, allows quantification of genetic conflicts of interest.

### Equilibrium analysis

Focusing attention upon scenarios in which GE invades, we next investigate its subsequent evolutionary fate (Supporting Information). The condition for increase in an allele’s frequency is computed in the same way as in the invasion analysis. However, we now assume that GE incurs no viability cost (\( \alpha = \beta = 0 \)), mainly for simplicity and also because we consider that natural selection may fine-tune the mechanism to reduce such deleterious by-product effects (in reality, these are unlikely to be completely absent). Having established that GE can invade, we check for an intermediate equilibrium level by solving \( d \frac{W}{dg} |_{g} = 0 \). And we determine when GE may go to fixation according to the condition \( d \frac{W}{dg} |_{g=1} > 0 \). Straightforward fixation of GE in the heterogametic sex leads to the eradication of all individuals of one sex from the population (males for PGE, females for MGE; Fig. 2). Accordingly, we consider that fixation leads to population extinction (Hamilton 1967). Finally, for scenarios in which GE is maintained at an intermediate level, we consider its long-term fate following the establishment of a novel mechanism of sex determination, which eliminates any impact of GE upon the sex ratio (Supporting Information).

### RESULTS

#### Origin of genome elimination

We assess the impact of the degree of inbreeding, mode of sex determination, genomic location, pattern of gene expression and parental origin of the eliminated genome on the
evolutionary invasion of GE alleles. For each scenario, we calculate the potential for GE (Fig. 3). This is mediated by two key effects: first, GE is a form of genetic drive, and as such it provides a two-fold direct transmission advantage for driving genes and a corresponding direct transmission disadvantage for those genes that are driven against; second, GE may lead to sex-ratio bias among an individual’s offspring, and this may provide a selective advantage or disadvantage depending upon the direction of bias and the population’s mating system.

Drive
The immediate consequences of drive are illustrated by considering PGE in females under XY sex determination (Fig. 3 panel a). The qualitative results for this permutation of the model readily generalise to all forms of GE in the homogametic sex, which has no impact on offspring sex ratio (PGE and MGE in females under XY/XO and in males under ZW/ZO; Fig. 3 panels a, c, f and h). Here, there are four types of genic actor. First, there are genes that directly benefit from drive: the female’s maternal-origin autosomal and maternal-origin X-chromosomal genes, and her mother’s autosomal and X-chromosomal genes. These genes have positive potential for PGE: in particular, $c = 0.5$ under outbreeding ($a = 0$), reflecting how the two-fold benefit of driving can offset even a halving of offspring number. Second, there are genes who suffer from drive: the female’s paternal-origin autosomal and paternal-origin X-chromosomal genes, and her father’s autosomal and X-chromosomal genes. These genes have negative potential for PGE: in particular,
Figure 3 The origin of genome elimination (GE). Results of the invasion analyses, for: (a) Paternal genome elimination (PGE) in females under XY or XO sex determination; (b) PGE in males, XY/XO; (c) Maternal genome elimination (MGE) in females, XY/XO; (d) MGE in males, XY/XO; (e) PGE in females, ZW/ZO; (f) PGE in males, ZW/ZO; (g) MGE in females, ZW/ZO; (h) MGE in males, ZW/ZO. In each case, potential for GE is shown for each class of genic actor, for whole-range of sib-mating (0 ≤ a ≤ 1). A: autosomal gene. X: X-linked gene. Y: Y-linked gene. Z: Z-linked gene. W: W-linked gene. Unimprinted autosomal genes (black lines) have positive potential for GE only under sib-mating (a > 0), and for PGE in heterogametic males (panel b) or PGE in heterogametic females (panel e), and so these are the scenarios in which GE robustly invades.
$\gamma = -\infty$ under outbreeding ($a = 0$), reflecting how no increase in offspring number can compensate for their elimination from the individual’s gametes. Third, there are genes that are equally likely to directly benefit or suffer from drive: the female’s unimprinted autosomal and X-chromosomal genes. These genes have zero potential for PGE, $\gamma = 0$, reflecting how they are not favoured to prevent PGE if this reduces offspring number ($\alpha > 0$) and to promote PGE if this increases offspring number ($\alpha < 0$). Fourth, there are genes that are not directly affected by drive: the female’s father’s Y-chromosomal genes. Their potential for PGE is undefined, meaning that PGE is a neutral trait (West 2009).

A gene’s inclusive fitness is not solely governed by its own replicative success but also by that of its homologues, to the extent that they share identity by descent (Gardner & Welch 2011). Accordingly, inbreeding ($a > 0$) changes the above results quantitatively (but not qualitatively), such that a gene of the first type, who enjoys a direct benefit owing to drive, also suffers an indirect, kin-selected cost, owing to the disadvantage incurred by the identical-by-descent genes it drives against. Consequently, its potential for PGE falls from $\gamma = 0.5$ in the extreme of outbreeding ($a = 0$) to $\gamma = 0$ in the extreme of chronic inbreeding ($a = 1$). To the extent that PGE is controlled by such ‘maternal’ genes, sib-mating inhibits the evolution of PGE (Brown 1964; Bull 1979). Conversely, a gene of the second type, who suffers a direct cost owing to drive, also enjoys an indirect benefit, owing to the advantage accrued by its identical-by-descent homologues. Accordingly, its potential for PGE increases from $\gamma = -\infty$ in the extreme of outbreeding to $\gamma = 0$ in the extreme of chronic inbreeding. To the extent that PGE is controlled by such ‘paternal’ genes, inbreeding promotes its evolution (Burt & Trivers 2006). Inbreeding has no effect on the potential for PGE for genes of the third and fourth types, who are unaffected by drive on average or at all.

To summarise, and to generalise to other instances of GE in the homogametic sex, there is extensive scope for genetic conflicts of interest over GE, diminishing as populations are increasingly inbred, but with a robust overall advantage accruing to PGE, and a robust overall disadvantage accruing to MGE, under sib-mating, owing to the impact of GE on offspring sex ratio. Thus, MGE is relatively implausible in males under male heterogamety (Fig. 3 panel d) and in females under female heterogamety (Fig. 3 panel g), because it leads to a male-biased offspring sex ratio (Fig. 2d and g). Accordingly, we discard these model permutations from further consideration.

Sex-ratio bias

In addition to the immediate effects of drive, GE may also accrue fitness effects owing to its consequences for offspring sex ratio. Sex-ratio bias arises from GE only when this occurs in the heterogametic sex (males under XY/XO, females under ZW/ZO; Fig. 3 panels b, d, e and g). In each case, PGE yields female bias, and MGE yields male bias (Fig. 2). As a consequence of LMC, sib-mating ($a > 0$) may favour a relatively female-biased sex ratio (Hamilton 1967). This gives a selective advantage to PGE, and a selective disadvantage to MGE, in the heterogametic sex, relative to that obtained from consideration of the immediate effects of drive only (see above). This sex-ratio-selection effect need not be uniform across all genic actors, as the coefficient of inbreeding depends upon mode of inheritance, and parents may be in conflict with their offspring and each other over desired sex allocation (Hamilton 1967; Trivers 1974; Werren & Hatcher 2000). These details are captured by the model.

The resulting potential for GE is illustrated by considering it operating in heterogametic males. The potential for PGE is increased for almost all genic actors as a consequence of the LMC effect (Fig. 3 panel b). In particular, the male’s unimprinted autosomal genes exhibit positive potential for PGE, rising from $\gamma = 0$ under the extreme of outbreeding ($a = 0$) to $\gamma = 0.5$ under the extreme of chronic inbreeding ($a = 1$). The exception is the Y-chromosomal genes that maintain a potential for PGE of $\gamma = -\infty$ irrespective of the extent of sib-mating. This owes to our assumption of polyandry, which leads maternal-brothers to be unrelated through their fathers. Similarly, the potential for MGE is decreased for almost all genic actors, because the resulting male bias is selectively disadvantageous under LMC (Fig. 3 panel d). Again, the exception is for the Y-chromosomal genes, which gain the usual two-fold drive benefit and hence exhibit a potential for MGE of $\gamma = 0.5$.

To summarise, and to generalise to other instances of GE in the heterogametic sex, there is extensive scope for genetic conflicts of interest, diminishing as populations are increasingly inbred, but with a robust overall advantage accruing to PGE, and a robust overall disadvantage accruing to MGE, under sib-mating, owing to the impact of GE on offspring sex ratio. Thus, MGE is relatively implausible in males under male heterogamety (Fig. 3 panel d) and in females under female heterogamety (Fig. 3 panel g), because it leads to a male-biased offspring sex ratio (Fig. 2d and g). Accordingly, we discard these model permutations from further consideration.

Maintenance of genome elimination

We have narrowed the invasion of GE to two scenarios: PGE in males under male heterogamety (Fig. 3 panel b); and PGE in females under female heterogamety (Fig. 3 panel c). Moreover, we have identified inbreeding as an important driver of PGE in both instances. We now consider the scope for evolutionary maintenance of GE in each of these scenarios (Fig. 4).

PGE in heterogametic males

Although inbreeding facilitates the invasion of PGE in heterogametic males, this trait cannot generally increase to fixation. This is because, upon reaching a certain frequency, it will have brought the population sex ratio to its ‘optimal’ level, and further increases in the frequency of PGE are prevented by countervailing selection for reduced female bias acting...
directly on the PGE alleles (Burt & Trivers 2006). The equilibrium point depends upon the degree of inbreeding and the genomic location and mode of action of the genes underlying PGE (Fig. 4 panel a). Applying the parliament-of-genes approach, such that it is the interests of the unimprinted autosomal genes that are expected to win out, the equilibrium level of PGE increases approximately linearly, from $g/C_3 = 0$ under outbreeding ($a = 0$) to $g/C_3 = 1$ under the chronic inbreeding ($a = 1$). That is, an intermediate equilibrium obtains for all intermediate degrees of inbreeding ($0 < a < 1$).

In the longer term, it is useful to consider how this PGE may subsequently evolve should a new mode of sex determination, that eliminates the sex-ratio effects of GE, arise (Haig 1993a,b). We find that the ‘maternal’ genic actors, who derive a direct benefit from the immediate effect of drive, are favoured to increase PGE to fixation. In the event that they achieve this outcome, population viability is maintained because, under the new mode of sex determination, the fixation of PGE does not eradicate males. The ‘paternal’ genic actors are, conversely, favoured to completely suppress PGE. From the perspective of the individual’s unimprinted autosomal genes, PGE is an entirely neutral trait. Accordingly, the parliament of genes is likely to be swayed by whichever extremist faction happens to have most power, and the long-term evolutionary fate of PGE therefore depends on the details of whether control lies mostly with the ‘maternal’ genes or with the ‘paternal’ genes.

**PGE in heterogametic females**

Although countervailing selection for reduced female bias prevents fixation of PGE in heterogametic males, this need not happen for PGE in heterogametic females (Fig. 4 panel b). Indeed, if – following the parliament-of-genes logic that control of PGE lies with the female’s unimprinted autosomal genes – then our model predicts fixation of PGE in heterogametic females. This leads to the complete eradication of males, and hence population extinction. The absence of countervailing sex-ratio selection owes to PGE in heterogametic females leading to males having reduced reproductive value (Supporting Information). Indeed, in the limit of complete PGE in females, males have zero reproductive value, and all reproductive value belongs to females’ maternal-origin genes. Hamilton (1967) provides more discussion of population extinction caused by sex-ratio distorters.

**DISCUSSION**

The two-fold transmission advantage enjoyed by GE alleles has raised the problem of why such biased inheritance is observed only in some species, and in some forms, but not others. In particular, it typically occurs in association with inbreeding, under male heterogamety, in males and in the form of PGE. Our analysis has clarified that, whilst some genes do gain a transmission advantage from drive, others suffer a disadvantage from reduced transmission, and ecological factors are important in providing a robust advantage for GE. In particular, we find that: (1) inbreeding promotes the elimination of the paternal genome in the heterogametic sex (i.e. males in XY/XO systems and females in ZW/ZO systems), (2) this may lead to population extinction under female heterogamety (i.e. ZW/ZO systems), owing to the eradication of males and (c) extinction is averted under male heterogamety (i.e. XY/XO systems), owing to countervailing sex-ratio selection. That is, a species’ mating ecology imposes constraints upon its predisposition to evolve and survive GE, and this explains the widely observed pattern of PGE in heterogametic males under inbreeding.

**Intragenomic conflict over genome elimination**

Our analysis has considered genetic conflicts of interest within the nuclear family and within the individual’s own genome.
Separate consideration of the individual’s maternal-origin genes vs. the genes carried by the individual’s mother reveals that these distinct sets of genes have distinct evolutionary interests, as do the individual’s paternal-origin genes vs. the genes carried by the individual’s father. For example, genes residing on a maternal-origin autosome enjoy the full transmission benefit of PGE whereas genes residing on one of the mother’s autosomes enjoy a smaller benefit as they are not guaranteed to have been passed on to the individual who exhibits the PGE phenotype. This clarifies that intragenomic imprinting conflicts are conceptually distinct from (though may arise in similar contexts to) conflicts of interest between parents, despite these two phenomena having often been conflated (as discussed by Haig 2002).

Within male-heterogametic systems, PGE appears to be more common under XO than XY inheritance, as no PGE species with recognisable sex chromosomes has a Y chromosome (Table 1). Burt & Trivers (2006) suggested that this owes to the Y chromosome being strongly opposed to PGE in males, and inhibiting its evolution. In support of this suggestion, we find PGE is never favoured by Y-chromosomal genes, which suffer complete transmission failure under PGE, and this drive disadvantage cannot be offset by any offspring-survival or sex-ratio benefit, for any degree of inbreeding. Relaxing our assumption of polyandry could change model predictions in this respect, as this would lead to relatedness between mate competitors with respect to their Y-chromosomal genes, enhancing selection for female bias (cf Hamilton 1967), but the qualitative conclusion that the Y chromosome is less inclined to PGE than are the autosomes appears to be robust. However, in many species the Y chromosome is relatively degenerate, containing few active genes, and hence may have insufficient power to inhibit PGE. Accordingly, loss of the Y chromosome more likely occurred subsequently to the transition to PGE. Indeed, in most cases, the diploid sister groups of PGE taxa have XY sex determination (Table 1).

**Mating system and genome elimination**

Our model of sib-mating is motivated by the empirical observation that GE often coincides with a life history that leads to high levels of inbreeding (Table 1). Our model shows that sib-mating and resulting selection for female-biased sex ratios can facilitate the evolution of GE, and we suggest that this explains the apparent association. However, sib-mating need not be essential for the evolution of GE. Other forms of inbreeding, such as those arising from population viscosity, may also favour female-biased sex ratios (Gardner et al. 2009). Moreover, some dipteran species with PGE lack inbreeding altogether (Burt & Trivers 2006). It is also possible that GE drives the evolution of inbreeding, rather than the reverse, yielding an alternative explanation for their apparent association. For example, some species with PGE exhibit haploid gene expression in males (Table 1 and Fig. 1), which could promote inbreeding by purging recessive deleterious alleles and hence ameliorating the costs of homozygosity. However, inbreeding appears frequently among several PGE species that exhibit diploid-male gene expression (e.g. lice and springtails).

Our model makes a number of other assumptions about ancestral mating system. First, for analytical convenience, we have assumed extreme female promiscuity, though monandry may be more realistic for some species (Hamilton 1967). As GE in males may lead to a reduction in the number of viable sperm, lower female promiscuity could promote GE in males by reducing between-male sperm competition. Second, we have assumed a classic LMC scenario in which females mate at their natal patch prior to dispersal (Hamilton 1967), which is more realistic for some PGE species (lice, mites, coffee-borer beetles and springtails) than others (dipterans and scale insects). However, these details are not essential, and the major purpose of the model is to illustrate that any mating ecology that results in selection for sex-ratio bias may govern the evolution of GE.

**Germline vs. embryonic genome elimination**

The defining feature of PGE in males is the absence of the paternal genome among the individual’s gametes, and our model focuses upon this central feature. That is, it more closely captures the ‘germline PGE’ of those species in which elimination of the paternal genome occurs during spermatogenesis, rather than the ‘embryonic PGE’ of those species in which elimination occurs during early embryogenesis, which potentially involves additional fitness costs associated with somatically haploid males (Table 1 and Fig. 1). Such costs of haploidy mean that germline PGE is likely the ancestral form, and embryonic PGE the more derived form. Indeed, embryonic PGE has evolved from germline PGE at least in the scale insects (Nur 1990; Herrick & Seger 1999; Ross et al. 2010a).

Among those species with germline PGE, there are various somatic effects ranging from elimination of paternally derived sex chromosomes to complete transcriptional silencing of the paternal genome (Fig. 1 and Table 1). Our model provides two non-mutually exclusive explanations for this. First, once PGE has evolved, the paternally derived genes are under strong selection to evolve counteradaptations (Herrick & Seger 1999; Ross et al. 2010b). To avoid this, the maternal or maternally derived genes are selected to either: disable the paternal genome, leading to whole-genome heterochromatisation, as seen in scale insects and beetles; or else completely eliminate it, leading to embryonic PGE, as seen in mites and armoured scale insects. Second, fixation of PGE in males requires the evolution of a new sex-determining system that overrides genetic sex determination. For example, in species where sex is determined by X-dosage, the elimination of one X chromosome would convert a female (XX) into a male (X0), as occurs in springtails (Dallai et al. 2001) and Sciara flies (Haig 1993b). Indeed, sex-ratio selection might also favour the silencing or early elimination of the whole paternal genome, converting females (XX) to hemizygous males (X).

Our model explains the elimination of paternal – as opposed to maternal – chromosomes owing to its impact upon offspring sex ratio. Another reason why the paternal genome may be more vulnerable to elimination is anisogamy (Parker et al. 1972): an egg contains many more proteins and RNAs than does a sperm, so if elimination is under parental control, the mother’s interests may be expected to dominate.
This is likely to be important in the evolution of embryonic PGE, but less so in the evolution of germline PGE. Moreover, such imbalance in power fails to explain other patterns of GE, such as its confinement to males.

Other forms of genome elimination

Our model has considered the most frequently occurring form of GE, whereby one haploid genome is eliminated according to its parent of origin. However, there are a number of reproductive systems that exhibit alternative forms of GE, typically involving hybridisation events where one species temporarily ‘borrows’ a genome from a related species, to express in its somatic tissues, but eliminates it from the germline (Beukeboom & Vrijenhoek 1998; Burt & Trivers 2006). In many, but not all, of these cases the hybridogenetic species is all-female. Our model shows that, in principle, this mating system could have evolved via PGE in heterogametic females, with extinction avoided despite the eradication of males because females were able to mate with males from a closely related species, but this is unlikely to be of empirical importance. First, if this were how hybridogenesis typically evolved, we would expect an overrepresentation of ancestral female heterogamy among these taxa, and although ZW is present in the ancestors of two hybridrogenetic teleost fish, the ancestors of hybridrogenetic frogs and stick insects were clearly XY (The Tree of Sex Consortium 2014). Second, in all cases, hybridogenesis apparently arose from an ancient hybridisation event, of which GE was a consequence rather than a cause (Burt & Trivers 2006).

However, there is one other case where genome elimination is dependent upon parent of origin. Androgenesis – found in two species of ant, four species of clam and one species of cypress tree – involves only sperm-contributing genes to zygotes, and maternal genomes being entirely eliminated (Beukeboom & Vrijenhoek 1998; Burt & Trivers 2006). In ants, it evolved from an already-asymmetric inheritance system (haplodiploidy) and resulted from selection pressures particular to eusociality. In clams and cypress, it evolved from hermaphroditism and occurs in every offspring (unlike PGE, which is restricted to males; Burt & Trivers 2006), so it resembles a modified form of parthenogenesis (with all reproductive value belonging to males rather than females) and may be better captured by classic models for the evolution of parthenogenesis.

Finally, PGE shares several key features with haplodiploidy (Hartl & Brown 1970; Bull 1979; Burt & Trivers 2006). In particular, all genes transmitted by males derive from their mothers, and are passed on only by their daughters. PGE and haplodiploidy often co-occur in closely related taxonomic groups, including scale insects, mites and beetles, which suggest that similar selection pressures underlie the evolution of both genetic systems. In addition to the drive benefits that are enjoyed by maternal-origin genes, the offspring sex ratio bias effect captured in our model may also apply to the evolution of haplodiploidy, as mating with a haploid male results in female bias that may be favoured under sib-mating (Hamilton 1967; Borgia 1980; Bull 1983). Sib-mating may also promote the evolution of haplodiploidy in other ways, unrelated to the effects considered in the present model, for example by purging recessive deleterious alleles and hence raising the viability of haploid males (Brown 1964). However, an alternative explanation for this empirical association is that haplodiploidy may evolve via PGE (Schrader & Hughes-Schrader 1931). This possibility remains to be formally explored, and presents an exciting avenue for future theoretical and empirical study.

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AUTHORSHIP

AG and LR designed the study and wrote the manuscript, AG performed the mathematical analysis.

REFERENCES


1. PGE in females and/or males under XY/XO inheritance: invasion analysis

1.1. Autosomal genes

Classes – There are 8 classes of individual, classified according to their sex $s \in \{f,m\}$, the grandparent of origin of their maternal-origin gene $T \in \{M,P\}$ and the grandparent of origin of their paternal-origin gene $U \in \{M,P\}$. Each class is notated in the form $sTU$, i.e. $fMM$, $fMP$, $fPM$, $fPP$, $mMM$, $mMP$, $mPM$ and $mPP$. 

Survival – Picking a zygote at random, it has some probability of belonging to each of the 8 classes, depending on whether its parents exhibited genome elimination. We model this as if there were 8 potential zygotes, of which one is chosen to come into existence. Moreover, the realised zygote may not survive to maturity, owing to viability costs associated with the genome elimination behaviour of its parents. Denoting the probability that the zygote’s mother exhibited PGE by $\phi$, the probability that the zygote’s father exhibited PGE by $\mu$, the viability cost associated with female PGE by $\alpha$ and the viability cost associated with male PGE by $\beta$, the probability that each potential zygote survives to maturity is: $S_{MM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/4)\phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$; $S_{MP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/4)\phi(1-\mu)(1-\alpha)$; $S_{PM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)(1-\phi)\mu(1-\beta)$; $S_{PP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu)$; $S_{MM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/4)\phi(1-\mu)(1-\alpha)$; $S_{MP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu)$; $S_{PM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu)$; and $S_{PP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu)$.

Reproductive success – Upon survival to maturity, every female achieves a unit of expected reproductive success, and every male achieves $(1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta)$ units of expected reproductive success, where: $z(\phi',\mu',\alpha,\beta) = (\sum_{s(T,M,P)\in(M,P)} S_{sTU}(\phi',\mu',\alpha,\beta))/(\sum_{s(T,M,P)\in(M,P)} S_{sTU}(\phi',\mu',\alpha,\beta))$ is the sex ratio (proportion male) in his mating group; $\phi'$ is the level of PGE among the females contributing offspring to the mating group; and $\mu'$ is the level of PGE among the fathers contributing offspring to the mating group. Thus, contingent upon survival, expected reproductive success is $R_{TU}(\phi',\mu',\alpha,\beta) = 1$ for females and $R_{TU}(\phi',\mu',\alpha,\beta) = (1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta)$ for males.

Fitness – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. $w_{sTU}(\phi,\mu,\mu',\alpha,\beta) = S_{sTU}(\phi,\mu,\alpha,\beta) \times R_{sTU}(\phi',\mu',\alpha,\beta)$. Because PGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by $\bar{w}_{sTU}(\alpha,\beta) = w_{sTU}(0,0,0,0,\alpha,\beta)$, and each individual’s fitness can be expressed relative to the average of their class, by $W_{sTU}(\phi,\mu,\mu',\alpha,\beta) = w_{sTU}(\phi,\mu,\mu',\alpha,\beta)/\bar{w}_{sTU}(\alpha,\beta)$. 

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Gene fitness – There are 16 classes of gene, because every class of individual carries one maternal-origin gene and one paternal-origin gene. Thus, gene classes are denoted in the form sTUv, where v ∈ {1,2} according to whether the gene is of maternal (v = 1) or paternal (v = 2) origin. The fitness of a gene, defined in terms of its probability of being transmitted to a potential (rather than a realised) zygote, is proportional to the fitness of the individual who carries it. Consequently, the relative fitness of a gene is equal to the relative fitness of its carrier: W_{STU1}(φ,μ,μ′,α,β) = W_{STU2}(φ,μ,μ′,α,β) = W_{STU}(φ,μ,μ′,α,β).

Reproductive value – The flow of genes between classes determines each class’s reproductive value. Each gene’s class in any generation may provide some information and some uncertainty about that gene’s origin in the previous generation. For example, genes of class fMM1 in the present generation are derived from genes of class fTU1 in the previous generation, where T = M or P with equal probability and U = M or P with equal probability. Consequently, the fMM1 gene derived from a fMM1 gene with probability φ_{fMM1->fMM1} = ¼, from a fMP1 gene with probability φ_{fMM1->fMP1} = ¼, from a fM1 gene with probability φ_{fMM1->fM1} = ¼ and from a fP1 gene with probability φ_{fMM1->fP1} = ¼. The reproductive value of a given class is shared among all the classes who contribute genes to that class, in proportion to the contributions that they make: C_{STUV} = \sum_{s,\{f,m\},T,\{M,P\},U,\{1,2\}} s*T*U*v* C_{sTUv}. This defines a system of 16 linear equations, which can be written as c = c • G, where c = \{c_{fMM1},c_{fMM2},c_{fMP1},...,c_{fMP2}\} is the vector of class reproductive values and G is the gene-flow matrix. Solving (i.e. finding the left eigenvector of G corresponding to the eigenvalue 1) obtains C_{STUV} = 1/16 for all gene classes.

PGE in females – Genes can be assigned genic values g according to their heritable tendency for any trait of interest. The condition for natural selection to favour an increase in this trait is dW/dg > 0, where W = \sum_{s,\{f,m\},T,\{M,P\},U,\{1,2\}} C_{STUV} W_{STUV}. Here, g is the genic value associated with an autosomal gene in a potential zygote. Thus, the condition for increase in female PGE is \sum_{s,\{f,m\},T,\{M,P\},U,\{1,2\}} C_{STUV} (dW_{STUV}/dg) \times (dφ/dG) \times (dG/dg_{STUV}) > 0, where G is the average genic value among those genes at the same locus whose expression controls PGE in the focal individual’s mother. Consequently: dφ/dG is an arbitrary mapping between genic value and phenotypic value, and can be set to 1; and dG/dg_{STUV} = p_{STUV} is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier. The consanguinities will depend upon which class or classes of genes control PGE in females -- see the Consanguinity section below, and Table A1.1.1, for details.

PGE in males – Similarly, the condition for increase in male PGE is \sum_{s,\{f,m\},T,\{M,P\},U,\{1,2\}} C_{STUV} (((dW_{STUV}/dg) \times (dμ/dG) \times (dG/dg_{STUV}) + (dW_{STUV}/dμ′) \times (dμ′/dG) \times (dG/dg_{STUV})) > 0, where G is the average genic value among those genes at the same locus whose expression controls PGE in the focal individual’s father, and
$G'$ is the average genic value among those genes at the same locus whose expression controls PGE in the males contributing offspring to the focal individual's mating group. Here: $d\mu/dG = d\mu'/dG' = 1$; $dG/dq_{STUV} = q_{STUV}$ is the consanguinity between the focal class-sTUV gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier; and $dG'/dq_{STUV} = q_{STUV}'$ is the consanguinity between the focal class-sTUV gene and the genes, residing at the same locus, who control PGE in the males who contribute offspring to the focal individual’s mating group. Note that, because $dW_{STUV}/d\mu' = 0$ for all classes of genes in females, we need only calculate consanguinities $q_{mtUV}$ for classes of genes in males. Again, the consanguinities will depend upon which class or classes of genes control PGE in males -- see the Consanguinity section below, and Table A1.1.2, for details.

Consanguinity – The coefficient of inbreeding is the consanguinity of mating partners. This may be expressed as $\rho = a((1/4) \times (1+\rho)/2 + (1/2) \times \rho + (1/4) \times \rho)$. That is: with probability $a$ the male and female share the same mother; so with probability $\frac{1}{4}$ we pick the maternal-origin genes from both individuals and hence their consanguinity is simply the consanguinity of their mother to herself, or $(1+\rho)/2$; and with probability $\frac{1}{2}$ we pick the maternal-origin gene from one of the mating partners and the paternal-origin gene from the other, in which case their consanguinity is that of mating partners, or $\rho$; and with probability $\frac{1}{4}$ we pick the paternal-origin genes from both individuals, in which case their consanguinity is that of two males in the same mating group, or $\rho$. Solving this equation obtains $\rho = a/(8-7a)$, so that there is full outbreeding in the absence of sib mating ($\rho = 0$ when $a = 0$) and there is full inbreeding if all mating is between sibs ($\rho = 1$ when $a = 1$).

From this coefficient can be defined other consanguinities between mating partners: $\rho_{m1} = a((1/2) \times (1+\rho)/2 + (1/2) \times \rho)$, from the perspective of the female’s maternal-origin gene; $\rho_{p1} = a \rho$, from the perspective of the female’s paternal-origin gene; $\rho_{M1} = a((1/2) \times (1+\rho)/2 + (1/2) \times \rho)$, from the perspective of the male’s maternal-origin gene; $\rho_{P1} = a \rho$, from the perspective of the male’s paternal-origin gene; $\rho_{MM} = a(1+\rho)/2$, from the perspective of both mating partners’ maternal genes; $\rho_{MP} = a \rho$, from the perspective of the female’s maternal gene and the male’s paternal gene; $\rho_{PM} = a \rho$, from the perspective of the female’s paternal gene and the male’s maternal gene; and $\rho_{PP} = a \rho$, from the perspective of both mating partners’ paternal genes. And these coefficients define all the consanguinities needed to solve the model (listed in Tables A1.1.1 & A1.1.2).

Potential for PGE – The condition for increase in PGE is $dW/dg > 0$. The left-hand side of this inequality is the marginal fitness and, above, we have calculated these for female PGE and for male PGE, for different scenarios regarding which genes control PGE. Evaluating the marginal fitnesses at $\phi = \phi' = \phi = \mu = \mu' = \bar{\mu} = 0$, the inequalities give the condition for invasion of female or male PGE in a non-PGE population. Setting the marginal fitness for female PGE equal to zero, and solving for $\alpha$, obtains the threshold cost of female PGE $\gamma$ such that invasion will occur if $\alpha < \gamma$ and invasion will not occur if $\alpha > \gamma$. This defines the potential for female PGE. If $\gamma > 0$, then it is possible for costly PGE to invade (provided the cost is sufficiently small), [3]
and if $\gamma < 0$, then it is possible that beneficial PGE (i.e. providing a viability benefit rather than a viability cost) may not invade (provided the benefit is sufficiently small). Thus, the potential $\gamma$ provides a quantitative measure of the extent to which a particular class of genes desires PGE in females. Similarly, setting the marginal fitness for male PGE to zero, and solving for $\beta$, obtains the potential for male PGE $\gamma$. These potentials are illustrated in Figure 3 of the main text.

### 1.2. X-linked genes

**Classes** – There are 4 classes of individual, classified according to their sex $s \in \{f,m\}$ and the grandparent of origin of their maternal-origin gene $T \in \{M,P\}$. If the individual is female, then all of her paternal-origin X-linked genes are derived from her paternal grandmother. And if the individual is male, he has no paternal-origin X-linked genes. Thus, each class is notated in the form sTUv, where $U \in \{M,\}$, i.e. fMM, fPM, mM- and mP-.

**Survival** – The probabilities of survival for each class are: $S_{fMM}(\phi, \mu, \alpha, \beta) = (1/4)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$; $S_{fPM}(\phi, \mu, \alpha, \beta) = (1/4)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$; $S_{mMM}(\phi, \mu, \alpha, \beta) = (1/4)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$; $S_{mPM}(\phi, \mu, \alpha, \beta) = (1/4)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$.
Expected reproductive success contingent upon surviving to maturity, i.e. Fitness $S$ is given by the product of survival to maturity and expected reproductive success is $R_{SU} = (1/4)(1 - \phi)(1 - \mu) + (1/2)(1 - \phi)(1 - \mu); S_{m}\cdot(\phi,\mu,\alpha,\beta) = (1/4)(1 - \phi)(1 - \mu) + (1/2)\phi(1 - \mu)(1 - \alpha)$; and $S_{mp}\cdot(\phi,\mu,\alpha,\beta) = (1/4)(1 - \phi)(1 - \mu)$.

**Reproductive success** – Contingent upon survival, expected reproductive success is $R_{SU} = (1/4)(1 - \phi)(1 - \mu) + (1/2)(1 - \phi)(1 - \mu); S_{m}\cdot(\phi,\mu,\alpha,\beta) = (1/4)(1 - \phi)(1 - \mu) + (1/2)\phi(1 - \mu)(1 - \alpha)$; and $S_{mp}\cdot(\phi,\mu,\alpha,\beta) = (1/4)(1 - \phi)(1 - \mu)$.

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e.
\[ w_{\text{STU}}(\phi, \mu, \mu', \alpha, \beta) = S_{\text{STU}}(\phi, \mu, \alpha, \beta) \times R_{\text{STU}}(\mu', \mu, \alpha, \beta). \] 

As before, because PGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by \( \bar{w}_{\text{STU}}(\alpha, \beta) = w_{\text{STU}}(0, 0, 0, \alpha, \beta) \), and each individual’s fitness can be expressed relative to the average of their class, by \( W_{\text{STU}}(\phi, \mu, \mu', \alpha, \beta) = w_{\text{STU}}(\phi, \mu, \mu', \alpha, \beta) / \bar{w}_{\text{STU}}(\alpha, \beta) \).

**Gene fitness** – There are 6 classes of gene, because individuals of each of the two female classes carry separate maternal-origin and paternal-origin genes, whereas individuals of each of the two male classes carry only maternal-origin genes. As before, gene classes are denoted in the form \( sTUv \), where \( v \in \{1, 2\} \) according to whether the gene is of maternal (\( v = 1 \)) or paternal (\( v = 2 \)) origin. The relative fitness of a gene in a female is given by \( W_{\text{TP}1}(\phi, \mu, \mu', \alpha, \beta) = W_{\text{TP}2}(\phi, \mu, \mu', \alpha, \beta) = W_{\text{TP}0}(\phi, \mu, \mu', \alpha, \beta) \) and the relative fitness of a gene in a male is given by \( W_{\text{MT}-1}(\phi, \mu, \mu', \alpha, \beta) = W_{\text{MT}0}(\phi, \mu, \mu', \alpha, \beta) \).

**Reproductive value** – As before, each gene’s class in any generation may provide some information and some uncertainty about that gene’s origin in the previous generation. For example, genes of class fMM1 in the present generation are derived from genes of class fTM1 in the previous generation, where \( T = M \) or \( P \) with equal probability. Consequently, the fMM1 gene derived from a fMM1 gene with probability \( \phi_{\text{IMM}1-\text{IMM}1} = \frac{1}{2} \) and from a fPM1 gene with probability \( \phi_{\text{IMM}1-\text{PM}1} = \frac{1}{2} \). The corresponding system of 6 linear equations may be written in linear algebraic form as \( c = c \cdot G \), where \( c = \{c_{\text{MM1}}, c_{\text{MM2}}, c_{\text{PM1}}, c_{\text{PM2}}, c_{\text{MM1-PM1}}, c_{\text{MM1-PM1}}\} \) is the vector of class reproductive values and \( G \) is the gene-flow matrix. Solving obtains \( c_{\text{STU}} = 1/6 \) for all gene classes.

**PGE in females** – Following the same procedure as before, the condition for increase in female PGE is \( c_{\text{MM1}} ((dW_{\text{IMM}1}/d\phi) \times (d\phi/dG) \times (dG/dg_{\text{MM}1}) + c_{\text{MM2}} ((dW_{\text{IMM}2}/d\phi) \times (d\phi/dG) \times (dG/dg_{\text{MM}2})) + c_{\text{PM1}} ((dW_{\text{IPM}1}/d\phi) \times (d\phi/dG) \times (dG/dg_{\text{PM}1}) + c_{\text{PM2}} ((dW_{\text{IPM}2}/d\phi) \times (d\phi/dG) \times (dG/dg_{\text{PM}2}) + c_{\text{MM1-PM1}} ((dW_{\text{MM1-PM1}}/d\phi) \times (d\phi/dG) \times (dG/dg_{\text{MM1-PM1}})) + c_{\text{MM1-PM1}} ((dW_{\text{MM1-PM1}}/d\phi) \times (d\phi/dG) \times (dG/dg_{\text{MM1-PM1}})) > 0 \). Once again: \( d\phi/dG \) is an arbitrary mapping between genic value and phenotypic value, and can be set to 1; and \( dG/dg_{\text{STU}} = p_{\text{STU}} \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier.

**PGE in males** – Similarly, the condition for increase in male PGE is \( c_{\text{MM1}} ((dW_{\text{IMM}1}/d\mu) \times (d\mu/dG) \times (dG/dg_{\text{MM}1}) + c_{\text{MM2}} ((dW_{\text{IMM}2}/d\mu) \times (d\mu/dG) \times (dG/dg_{\text{MM}2})) + c_{\text{PM1}} ((dW_{\text{IPM}1}/d\mu) \times (d\mu/dG) \times (dG/dg_{\text{PM}1}) + c_{\text{PM2}} ((dW_{\text{IPM}2}/d\mu) \times (d\mu/dG) \times (dG/dg_{\text{PM}2}) + c_{\text{MM1-PM1}} ((dW_{\text{MM1-PM1}}/d\mu) \times (d\mu/dG) \times (dG/dg_{\text{MM1-PM1}}) + c_{\text{MM1-PM1}} ((dW_{\text{MM1-PM1}}/d\mu) \times (d\mu/dG) \times (dG/dg_{\text{MM1-PM1}})) > 0 \), where: \( d\mu/dG = d\mu'/dG' = 1 \); \( dG/dg_{\text{STU}} = q_{\text{STU}} \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier; and \( dG'/dg_{\text{MTU}} = q'_{\text{MTU}} \) is the consanguinity between the focal class-sTUv gene and
the genes, residing at the same locus, who control PGE in the males who contribute offspring to the focal individual’s mating group.

**Consanguinity** – The coefficient of inbreeding for X-linked genes is \( \rho^* = a(1/2) \times (1+\rho^*)/2 + (1/2) \times \rho^* \). That is: with probability \( a \) the male and female share the same mother; so with probability \( 1/2 \) we pick the maternal-origin genes from the female and hence their consanguinity is simply the consanguinity of their mother to herself, or \((1+\rho^*)/2\); and with probability \( 1/2 \) we pick the paternal-origin gene from the female, in which case their consanguinity is that of mating partners, or \( \rho^* \).

Solving this equation obtains \( \rho^* = a/(4-3a) \). From this coefficient can be defined other consanguinities between mating partners: \( \rho_{M}^* = a(1+\rho^*)/2 \), from the perspective of the female’s maternal-origin gene; and \( \rho_{P}^* = a \rho^* \), from the perspective of the female’s paternal-origin gene. Moreover, the consanguinity between two males in the same mating group is \( \psi^* = a(1+\rho^*)/2 \). These coefficients define all the consanguinities needed to solve the model (listed in Tables A1.2.1 & A1.2.2).

**Potential for PGE** – The potentials for female and male PGE are calculated as described above. These are illustrated in Figure 3 of the main text.

### 1.3. Y-linked genes

**Classes** – There are 2 classes of individual, classified according to their sex \( s \in \{f,m\} \). If the individual is female, then she has no Y-linked genes. And if the individual is male, then all of his Y-linked genes are paternal in origin (and came from his paternal grandfather). Thus, class is notated in the form \( s \in \{f,m\} \).

**Survival** – The probabilities of survival for each class are \( S_s(\phi,\mu,\alpha,\beta) = (1/2)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta) \) and \( S_m(\phi,\mu,\alpha,\beta) = (1/2)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) \).

**Reproductive success** – Contingent upon survival, expected reproductive success is \( R_s(\phi,\mu,\alpha,\beta) = 1 \) for females and \( R_m(\phi,\mu,\alpha,\beta) = (1-\phi)(1-\mu)(1-\alpha) + \phi \mu (1-\alpha)(1-\beta) \) for males, where \( \zeta(\phi,\mu,\alpha,\beta) = S_m(\phi,\mu,\alpha,\beta)/(S_l(\phi,\mu,\alpha,\beta) + S_m(\phi,\mu,\alpha,\beta)) \).

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. \( w_s(\phi,\mu,\alpha,\beta) = S_s(\phi,\mu,\alpha,\beta) \times R_s(\phi,\mu,\alpha,\beta) \). As before, because PGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by \( \bar{w}_s(\alpha,\beta) = w_s(0,0,0,0,\alpha,\beta) \), and each individual’s fitness can be expressed relative to the average of their class, by \( W_s(\phi,\mu,\alpha,\beta) = w_s(\phi,\mu,\mu,\alpha,\beta)/\bar{w}_s(\alpha,\beta) \).

**Gene fitness** – There is only one class of Y-linked gene, because females carry no Y-linked genes and males carry one class of Y-linked genes. Accordingly, the relative fitness of a Y-linked gene in a male is simply \( W_m(\phi,\mu,\mu,\alpha,\beta) \).
Reproductive value – Since there is only one class of Y-linked gene, all reproductive value belongs to this class: \( c_m = 1 \).

**PGE in females** – Following the same procedure as before, the condition for increase in female PGE is \( c_m \left( \left( \frac{dW_m}{d\phi} \right) \times \left( \frac{d\phi}{dG} \right) \times \left( \frac{dG}{d\rho_m} \right) \right) > 0 \). Once again: \( \frac{d\phi}{dG} \) is an arbitrary mapping between genic value and phenotypic value, and can be set to 1; and \( \frac{dG}{d\rho_m} = p_m \) is the consanguinity between the focal gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier.

**PGE in males** – Similarly, the condition for increase in male PGE is \( c_m \left( \left( \frac{dW_m}{d\mu} \right) \times \left( \frac{d\mu}{dG} \right) \times \left( \frac{dG}{d\rho_m} \right) \right) \left( \left( \frac{dW_m}{d\mu'} \right) \times \left( \frac{d\mu'}{dG'} \right) \times \left( \frac{dG'}{d\rho_{m'}} \right) \right) > 0 \), where: \( \frac{d\mu}{dG} = \frac{d\mu'}{dG'} = 1 \)
\( \text{d}u'/\text{d}G = 1; \text{d}G/\text{d}g_m = q_m \) is the consanguinity between the focal class-\( sTUv \) gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier; and \( \text{d}G'/\text{d}g_m = q'_m \) is the consanguinity between the focal class-\( sTUv \) gene and the genes, residing at the same locus, who control PGE in the males who contribute offspring to the focal individual’s mating group.

**Consanguinity** – Coefficients of inbreeding are undefined for Y-linked genes, because females do not carry any such genes. The consanguinity between two males in the same mating group is \( \psi^{**} = a\psi^{**} \); that is, with probability \( a \) they share the same mother, and hence their consanguinity is that of their fathers, i.e. two males in the same mating group. Solving yields \( \psi^{**} = 0 \) for all \( 0 < a < 1 \). This coefficient defines all the consanguinities needed to solve the model (listed in Tables A1.3.1 & A1.3.2).

**Potential for PGE** – The potentials for female and male PGE are calculated as described above. These are illustrated in Figure 3 of the main text.

2. MGE in females and/or males under XY/XO inheritance: invasion analysis

2.1. Autosomal genes

**Classes** – There are 8 classes of individual, classified according to their sex \( s \in \{f,m\} \), the grandparent of origin of their maternal-origin gene \( T \in \{M,P\} \) and the grandparent of origin of their paternal-origin gene \( U \in \{M,P\} \). Each class is notated in the form \( sTU \), i.e. \( fMM, fMP, fPM, fPP, mMM, mMP, mPM \) and \( mPP \).

**Survival** – Denoting the probability that the zygote’s mother exhibited MGE by \( \phi \), the probability that the zygote’s father exhibited MGE by \( \mu \), the viability cost associated with female MGE by \( \alpha \) and the viability cost associated with male MGE by \( \beta \), the probability that each potential zygote survives to maturity is:

\[
S_{MM}(\phi,\mu,\alpha,\beta) = \frac{(1/8)(1-\phi)(1-\mu)}{1-(1/8)(1-\phi)(1-\mu)}; \\
S_{MP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu); \\
S_{PM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu); \\
S_{PP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu); \\
S_{MM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu); \\
m_{MP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu); \\
m_{PM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu); \\
m_{PP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu).
\]

**Reproductive success** – Upon survival to maturity, every female achieves a unit of expected reproductive success, and every male achieves \( (1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta) \) units of expected reproductive success, where:

\[
z(\phi',\mu',\alpha,\beta) = \frac{\sum_{s(T\in\{M,P\},U\in\{M,P\}} S_{sTU}(\phi',\mu',\alpha,\beta)}{\sum_{s(T\in\{M,P\},U\in\{M,P\}} S_{sTU}(\phi',\mu',\alpha,\beta)}}
\]

in his mating group; \( \phi' \) is the level of MGE among the females contributing offspring to the mating group; and \( \mu' \) is the level of MGE among the fathers contributing offspring to the mating group. Thus, contingent upon survival, expected reproductive success is \( R_{TU}(\phi',\mu',\alpha,\beta) = 1 \) for females and \( R_{sTU}(\phi',\mu',\alpha,\beta) = (1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta) \) for males.
Average genic value among those genes at the same locus whose expression controls reproductive value of a population in which genome elimination is vanishingly rare. Consequently, the probability of being transmitted to a potential (rather than a realized) zygote, is proportional to the fitness of the individual who carries it. Consequently, the condition for natural selection to favour an increase in a gene is equal to the relative fitness of its carrier: $W_{STU}(\phi, \phi', \mu, \mu', \alpha, \beta) = W_{STU2}(\phi, \phi', \mu, \mu', \alpha, \beta) = W_{STU}(\phi, \phi', \mu, \mu', \alpha, \beta)$.

Reproductive value – Because class reproductive values are calculated in a population in which genome elimination is vanishingly rare, these are exactly the same as calculated in section 1.1, i.e. $c_{STUV} = 1/16$ for all gene classes.

**MGE in females** – The condition for natural selection to favour an increase in MGE is $\sum_{s=\{m\},T=\{M,M',P\},U=\{M,P\},v=\{1,2\}} c_{STUV} \left( dW_{STUV}/d\phi \right) \times (d\phi/dG) \times (dG/dg_{STUV}) > 0$, where $G$ is the average genic value among those genes at the same locus whose expression controls MGE in the focal individual’s mother. Consequently: $d\phi/dG$ is an arbitrary mapping.

### Table A1.3.1. Consanguinities for the female PGE invasion analysis: Y-linked genes.

The only Y-linked actor class is the female’s father’s Y-linked genes ($Y_{Fat}$). Shown here are their consanguinity $p_m$ to the single recipient class $m$ among the focal female’s sons.

<table>
<thead>
<tr>
<th>Recipient Class</th>
<th>Actor Class</th>
<th>$Y_{Fat}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$m$</td>
<td>$\alpha\psi^{**}$</td>
<td>$\alpha\psi^{**}$</td>
</tr>
</tbody>
</table>

### Table A1.3.2. Consanguinities for the male PGE invasion analysis: Y-linked genes.

The two Y-linked actor classes are the male’s own Y-linked genes ($Y$) and the Y-linked genes of his father ($Y_{Fat}$). Shown here are their consanguinity $q_m$ to the recipient class $m$ among the focal male’s sons, and the consanguinity $q_{m'}$ to the recipient class $m'$ in the males who compete for mates with the focal male’s sons.

<table>
<thead>
<tr>
<th>Recipient Class</th>
<th>Actor Class</th>
<th>$Y$</th>
<th>$Y_{Fat}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$m$</td>
<td>$1$</td>
<td>$1$</td>
<td>$\alpha\psi^{**}$</td>
</tr>
<tr>
<td>$m'$</td>
<td>$\alpha\psi^{**}$</td>
<td>$\alpha\psi^{**}$</td>
<td></td>
</tr>
</tbody>
</table>
between genic value and phenotypic value, and can be set to 1; and \( \frac{dG}{dg_{sTU}} = p_{sTU} \) is the consanguinity between the focal class-\( sTU \) gene and the genes, residing at the same locus, who control MGE in the mother of the focal gene’s carrier. The consanguinities will depend upon which class or classes of genes control MGE in females – and these are exactly the same as those listed in Table A1.1.1.

**MGE in males** – Similarly, the condition for increase in male MGE is

\[
\sum_{s \in \{f,m\}, T \in \{M,P\}, U \in \{M,P\}, v \in \{1,2\}} C_{sTUv} \left( (\frac{dW_{sTU}}{d\mu}) \times (\frac{d\mu}{dG}) \times (\frac{dG}{dg_{sTU}}) + (\frac{dW_{sTU}}{d\mu'}) \times (\frac{d\mu'}{dg'}) \times (\frac{dG}{dg_{sTU}}) \right) > 0,
\]

where \( G \) is the average genic value among those genes that control MGE in the focal individual’s father, and \( G' \) is the average genic value among those genes at the same locus whose expression controls MGE in the focal individual’s mother.

Consanguinity – As noted above, because consanguinities are calculated in a population from which genomic elimination is absent, these are exactly the same as those listed in Tables A1.1.1 & A1.1.2.

**Potential for MGE** – The potential for female MGE and the potential for male MGE are calculated in the same way as before, and these are illustrated in Figure 3 of the main text.

2.2. X-linked genes

**Classes** – There are 4 classes of individual, classified according to their sex \( s \in \{f,m\} \) and the grandparent of origin of their maternal-origin gene \( T \in \{M,P\} \). If the individual is female, then all of her paternal-origin X-linked genes are derived from her paternal grandmother. And if the individual is male, he has no paternal-origin X-linked genes. Thus, each class is notated in the form \( sTU \), where \( U \in \{M,-\} \), i.e. \( fMM, fPM, mM- \) and \( mP- \).

**Survival** – The probabilities of survival for each class are: \( S_{fMM}(\phi_1, \alpha, \beta) = \frac{1}{4}(1-\phi)(1-\mu) \); \( S_{fPM}(\phi, \mu, \alpha, \beta) = \frac{1}{4}(1-\phi)(1-\mu) + \frac{1}{2}(1-\phi)(1-\mu)(1-\alpha) \); \( S_{mM-}(\phi, \mu, \alpha, \beta) = \frac{1}{4}(1-\phi)(1-\mu) + \frac{1}{2}(1-\phi)(1-\mu)(1-\alpha) + \frac{1}{2}(1-\phi)(1-\mu)(1-\beta) \); and \( S_{mP-}(\phi, \mu, \alpha, \beta) = \frac{1}{4}(1-\phi)(1-\mu) + \frac{1}{2}(1-\phi)(1-\mu)(1-\alpha) + \frac{1}{2}(1-\phi)(1-\mu)(1-\beta) \).
Reproductive success – Contingent upon survival, expected reproductive success is 
\[ R_{TU}(\phi',\mu',\alpha,\beta) = 1 \] for females and 
\[ R_{mTU}(\phi',\mu',\alpha,\beta) = (1 - z(\phi',\mu',\alpha,\beta)) / z(\phi',\mu',\alpha,\beta) \] for males, where 
\[ z(\phi',\mu',\alpha,\beta) = (S_{MM}(\phi',\mu',\alpha,\beta) + S_{mP}(\phi',\mu',\alpha,\beta)) / (S_{MM}(\phi',\mu',\alpha,\beta) + S_{mP}(\phi',\mu',\alpha,\beta)) \].

Fitness – Expected fitness is given by the product of survival to maturity and 
expected reproductive success contingent upon surviving to maturity, i.e. 
\[ w_{STU}(\phi,\mu',\alpha,\beta) = S_{STU}(\phi,\mu,\alpha,\beta) \times R_{STU}(\phi',\mu',\alpha,\beta). \] As before, because MGE is 
vanishingly rare in the population, the average fitness among all individuals of a 
particular class is given by 
\[ w_{STU}(\alpha,\beta) = w_{STU}(0,0,0,\alpha,\beta), \] and each individual’s 
fitness can be expressed relative to the average of their class, by 
\[ W_{STU}(\phi,\mu',\alpha,\beta) = w_{STU}(\phi,\mu',\alpha,\beta) / w_{STU}(\alpha,\beta). \]

Gene fitness – There are 6 classes of gene, because individuals of each of the two 
female classes carries separate maternal-origin and paternal-origin genes, whereas 
individuals of each of the two male classes carry only maternal-origin genes. As 
before, gene classes are denoted in the form sTUv, where \( v \in \{1,2\} \) according to 
whether the gene is of maternal (\( v = 1 \)) or paternal (\( v = 2 \)) origin. The relative fitness 
of a gene in a female is given by 
\[ W_{TP1}(\phi,\mu',\alpha,\beta) = W_{TP2}(\phi,\mu',\alpha,\beta) = \]
\[ W_{mT-1}(\phi,\mu',\alpha,\beta) = \]
\[ W_{mT}(\phi,\mu',\alpha,\beta). \]

Reproductive value – Because class reproductive values are calculated in a 
population in which genome elimination is vanishingly rare, these are exactly the 
same as calculated in section 1.2, i.e. \( c_{sTUv} = 1/6 \) for all gene classes.

MGE in females – Following the same procedure as before, the condition for increase 
in female MGE is 
\[ c_{MM1} \left( (dW_{IMM1}/d\phi) \times (d\phi/dG) \times (dG/dg_{MM1}) \right) + c_{MM2} \left( (dW_{IMM2}/d\phi) \times (d\phi/dG) \times (dG/dg_{MM2}) \right) + c_{PM1} \left( (dW_{IPM1}/d\mu) \times (d\mu/dG) \times (dG/dg_{PM1}) \right) + c_{PM2} \left( (dW_{IPM2}/d\mu) \times (d\mu/dG) \times (dG/dg_{PM2}) \right) + c_{mM-1} \left( (dW_{mM-1}/d\phi) \times (d\phi/dG) \times (dG/dg_{mM-1}) \right) + c_{mM-1} \left( (dW_{mM-1}/d\mu) \times (d\mu/dG) \times (dG/dg_{mM-1}) \right) > 0. \] Once again: \( d\phi/dG \) is an 
arbitrary mapping between genic value and phenotypic value, and can be set to 1; 
and \( dG/dg_{STU} = p_{sTUv} \) is the consanguinity between the focal class-sTUv gene and the 
genes, residing at the same locus, who control MGE in the mother of the focal gene’s 
carrier.

MGE in males – Similarly, the condition for increase in male MGE is 
\[ c_{MM1} \left( (dW_{IMM1}/d\mu) \times (d\mu/dG) \times (dG/dg_{MM1}) \right) + c_{MM2} \left( (dW_{IMM2}/d\mu) \times (d\mu/dG) \times (dG/dg_{MM2}) \right) + c_{PM1} \left( (dW_{IPM1}/d\mu) \times (d\mu/dG) \times (dG/dg_{PM1}) \right) + c_{PM2} \left( (dW_{IPM2}/d\mu) \times (d\mu/dG) \times (dG/dg_{PM2}) \right) + c_{mM-1} \left( (dW_{mM-1}/d\mu) \times (d\mu/dG) \times (dG/dg_{mM-1}) \right) + c_{mM-1} \left( (dW_{mM-1}/d\mu) \times (d\mu/dG) \times (dG/dg_{mM-1}) \right) + \right) > 0, \] where: \( d\mu/dG \neq d\mu'/dG' \neq 1; dG/dg_{sTUv} = q_{sTUv} \) is the consanguinity between the focal class-sTUv gene and the 
genes, residing at the same locus, who control MGE in the father of the focal gene’s carrier; 
and \( dG'/dG_{mTUv} = q_{mTUv} \) is the consanguinity between the focal class-sTUv gene and
the genes, residing at the same locus, who control MGE in the males who contribute offspring to the focal individual’s mating group.

**Consanguinity** – As noted above, because consanguinities are calculated in a population from which genomic elimination is absent, these are exactly the same as those listed in Tables A1.2.1 & A1.2.2.

**Potential for MGE** – The potential for female MGE and the potential for male MGE are calculated in the same way as before, and these are illustrated in Figure 3 of the main text.

### 2.3. Y-linked genes

**Classes** – There are 2 classes of individual, classified according to their sex $s \in \{f, m\}$. If the individual is female, then she has no Y-linked genes. And if the individual is male, then all of his Y-linked genes are paternal in origin (and came from his paternal grandfather). Thus, class is notated in the form $s \in \{f, m\}$.

**Survival** – The probabilities of survival for each class are $S_f(\phi, \mu, \alpha, \beta) = (1/2)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha)$ and $S_m(\phi, \mu, \alpha, \beta) = (1/2)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$.

**Reproductive success** – Contingent upon survival, expected reproductive success is $R_f(\phi', \mu', \alpha, \beta) = 1$ for females and $R_m(\phi', \mu', \alpha, \beta) = (1-z(\phi', \mu', \alpha, \beta))/z(\phi', \mu', \alpha, \beta)$ for males, where $z(\phi', \mu', \alpha, \beta) = S_m(\phi', \mu', \alpha, \beta)/(S_f(\phi', \mu', \alpha, \beta) + S_m(\phi', \mu', \alpha, \beta))$.

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. $w_s(\phi, \phi', \mu, \mu', \alpha, \beta) = S_s(\phi, \mu, \alpha, \beta) \times R_s(\phi', \mu', \alpha, \beta)$. As before, because MGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by $\overline{w}_s(\alpha, \beta) = w_s(0, 0, 0, 0, \alpha, \beta)$, and each individual’s fitness can be expressed relative to the average of their class, by $W_s(\phi, \phi', \mu, \mu', \alpha, \beta) = w_s(\phi, \phi', \mu, \mu', \alpha, \beta)/\overline{w}_s(\alpha, \beta)$.

**Gene fitness** – There is only one class of Y-linked gene, because females carry no Y-linked genes and males carry one class of Y-linked genes. Accordingly, the relative fitness of a Y-linked gene in a male is simply $W_m(\phi, \phi', \mu, \mu', \alpha, \beta)$.

**Reproductive value** – Since there is only one class of Y-linked gene, all reproductive value belongs to this class: $c_m = 1$.

**MGE in females** – Following the same procedure as before, the condition for increase in female MGE is $c_m ((dW_m/d\phi) \times (d\phi/dG) \times (dG/d\phi_m)) > 0$. Once again: $d\phi/dG$ is an arbitrary mapping between genic value and phenotypic value, and can be set to 1; and $dG/d\phi_m = p_m$ is the consanguinity between the focal gene and the genes, residing at the same locus, who control MGE in the mother of the focal gene’s carrier.
\( MGE \textit{ in males} \) – Similarly, the condition for increase in male MGE is \( c_m ((d W_m /d\mu) \times (d\mu/dG) \times (dG/dg_m)) > 0 \), where: \( d\mu/dG = \mu' /dG' = 1; dG/dg_m = q_m \) is the consanguinity between the focal class-\( sTUv \) gene and the genes residing at the same locus, who control MGE in the father of the focal gene’s carrier; and \( dG'/dg_m = q_m \) is the consanguinity between the focal class-\( sTUv \) gene and the genes, residing at the same locus, who control MGE in the males who contribute offspring to the focal individual’s mating group.

\( \textit{Consanguinity} \) – As noted above, because consanguinities are calculated in a population from which genomic elimination is absent, these are exactly the same as those listed in Tables A1.3.1 & A1.3.2.

\( \textit{Potential for MGE} \) – The potential for female MGE and the potential for male MGE are calculated in the same way as before, and these are illustrated in Figure 3 of the main text.

3. PGE in females and/or males under ZW inheritance: invasion analysis

3.1. \textit{Autosomal genes}

\( \textit{Classes} \) – There are 8 classes of individual, classified according to their sex \( s \in \{f,m\} \), the grandparent of origin of their maternal-origin gene \( T \in \{M,P\} \) and the grandparent of origin of their paternal-origin gene \( U \in \{M,P\} \). Each class is notated in the form \( sTU \), i.e. \( fMM, fMP, fPM, fPP, mMM, mMP, mPM \) and \( mPP \).

\( \textit{Survival} \) – Picking a zygote at random, it has some probability of belonging to each of the 8 classes, depending on whether its parents exhibited genome elimination. We model this as if there were 8 potential zygotes, of which one is chosen to come into existence. Moreover, the realised zygote may not survive to maturity, owing to viability costs associated with the genome elimination behaviour of its parents. Denoting the probability that the zygote’s mother exhibited PGE by \( \phi \), the probability that the zygote’s father exhibited PGE by \( \mu \), the viability cost associated with female PGE by \( \alpha \) and the viability cost associated with male PGE by \( \beta \), the probability that each potential zygote survives to maturity is: \( S_{MM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta) \); \( S_{MP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) \); \( S_{PM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) \); \( S_{PP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) \); \( S_{MMP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) \); \( S_{MPM}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) \); \( S_{MMMP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) \); and \( S_{MPMP}(\phi,\mu,\alpha,\beta) = (1/8)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/4)(1-\phi)\mu(1-\beta) \).

\( \textit{Reproductive success} \) – Upon survival to maturity, every female achieves a unit of expected reproductive success, and every male achieves \((1-z(\phi,\mu,\alpha,\beta)) / z(\phi,\mu,\alpha,\beta)\) units of expected reproductive success, where: \( z(\phi,\mu,\alpha,\beta) = (\Sigma_{s\in\{f,m\},T\in\{M,P\},U\in\{M,P\}} S_{sTU}(\phi,\mu,\alpha,\beta)) \) is the sex ratio (proportion male)
in his mating group; \( \phi' \) is the level of PGE among the females contributing offspring to the mating group; and \( \mu' \) is the level of PGE among the fathers contributing offspring to the mating group. Thus, contingent upon survival, expected reproductive success is \( R_{TU}(\phi', \mu', \alpha, \beta) = 1 \) for females and \( R_{mTU}(\phi', \mu', \alpha, \beta) = (1 - z(\phi', \mu', \alpha, \beta))/z(\phi', \mu', \alpha, \beta) \) for males.

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e.

\[ w_{STU}(\phi, \phi', \mu, \mu', \alpha, \beta) = S_{STU}(\phi, \mu, \alpha, \beta) \times R_{STU}(\phi', \mu', \alpha, \beta). \]

Because PGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by 
\[ \overline{w}_{TU}(\alpha, \beta) = w_{STU}(0, 0, 0, 0, \alpha, \beta), \]
and each individual’s fitness can be expressed relative to the average of their class, by

\[ W_{STU}(\phi, \phi', \mu, \mu', \alpha, \beta) = \frac{w_{STU}(\phi, \phi', \mu, \mu', \alpha, \beta)}{\overline{w}_{TU}(\alpha, \beta)}. \]

**Gene fitness** – There are 16 classes of gene, because every class of individual carries one maternal-origin gene and one paternal-origin gene. Thus, gene classes are denoted in the form \( sTUv \), where \( v \in \{1, 2\} \) according to whether the gene is of maternal \( (v = 1) \) or paternal \( (v = 2) \) origin. The fitness of a gene, defined in terms of its probability of being transmitted to a potential (rather than a realised) zygote, is proportional to the fitness of the individual who carries it. Consequently, the relative fitness of a gene is equal to the relative fitness of its carrier:

\[ W_{STU}(\phi, \phi', \mu, \mu', \alpha, \beta) = W_{STU2}(\phi, \phi', \mu, \mu', \alpha, \beta) = W_{STU}(\phi, \phi', \mu, \mu', \alpha, \beta). \]

**Reproductive value** – The flow of autosomal genes between classes is exactly the same under XY/XO and ZZ inheritance, so the class reproductive values are identical to those derived in section 1.1.1, i.e. \( c_{STUv} = 1/16 \) for all gene classes.

**PGE in females** – Genes can be assigned genic values \( g \) according to their heritable tendency for any trait of interest. The condition for natural selection to favour an increase in this trait is \( dW/dg > 0 \), where

\[ W = \sum_{s \in \{f,m\}, T \in \{M,P\}, U \in \{M,P\}, v \in \{1, 2\}} c_{STUv} W_{STUv}. \]

Here, \( g \) is the genic value associated with an autosomal gene in a potential zygote. Thus, the condition for increase in female PGE is

\[ \sum_{s \in \{f,m\}, T \in \{M,P\}, U \in \{M,P\}, v \in \{1, 2\}} c_{STUv} ((dW_{STUv}/dg) \times (d\phi/dG) \times (dG/dg_{SUv}) + (dW_{STUv}/d\phi') \times (d\phi'/dG') \times (dG'/dG_{STUv})) > 0, \]

where \( G \) is the average genic value among those genes at the same locus whose expression controls PGE in the focal individual’s mother, and \( G' \) is the average genic value among those genes at the same locus whose expression controls PGE in the females contributing offspring to the focal individual’s mating group. Here: \( d\phi/dG = \phi'/G' = 1; \) \( dG/dg_{STUv} = p_{STUv} \) is the consanguinity between the focal class-\( sTUv \) gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier; and \( dG'/dg_{STUv} = p_{STUv} \) is the consanguinity between the focal class-\( sTUv \) gene and the genes, residing at the same locus, who control PGE in the females who contribute offspring to the focal individual’s mating group. Note that, because \( dW_{STUv}/d\phi' = 0 \) for all classes of genes in females, we need only calculate consanguinities \( p_{mSTUv} \) for classes of genes in males. Again, the consanguinities will
depend upon which class or classes of genes control PGE in females -- see the Consanguinity section below.

**PGE in males** – Similarly, the condition for increase in male PGE is \( \sum_{s \in \{f,m\}, T \in \{M,P\}, U \in \{M,P\}, v \in \{1,2\}} G_{STU} \left( (dW_{STU}/d\mu) \times (d\mu/dG) \times (dG/dg_{STU}) \right) > 0 \), where \( G \) is the average genic value among those genes at the same locus whose expression controls PGE in the focal individual’s father. Here, \( d\mu/dG = 1 \), and \( dG/dg_{STU} = q_{STU} \) is the consanguinity between the focal class-sTUV gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier. Again, the consanguinities will depend upon which class or classes of genes control PGE in males -- see the Consanguinity section below.

**Consanguinity** – The inheritance of autosomal genes is exactly the same under XY/XO and ZW inheritance so, just as in section 1.1: the consanguinity of mating partners is \( \rho = a/(8-7a) \). Moreover, as before, we have \( \rho_M = a((1/2) \times (1+p)/2 + (1/2) \times p) \), \( \rho_F = a \rho \), \( \rho_M = a((1/2) \times (1+p)/2 + (1/2) \times p) \), \( \rho_P = a \rho \), \( \rho_{MP} = a \rho \) and \( \rho_{PP} = a \rho \). These coefficients define all the consanguinities needed to solve the model (listed in Tables A3.1.1 & A3.1.2).

**Potential for PGE** – The potential for female PGE and the potential for male PGE are calculated in the same way as before, and these are illustrated in Figure 3 of the main text.

**3.2. Z-linked genes**

**Classes** – There are 4 classes of individual, classified according to their sex \( s \in \{f,m\} \) and the grandparent of origin of their paternal-origin gene \( U \in \{M,P\} \). If the individual is female, she has no maternal-origin Z-linked genes. And if the individual is male, then all of his maternal-origin Z-linked genes are derived from his maternal grandfather. Thus, each class is notated in the form \( sTU \), where \( T \in \{M,r\} \), i.e. \( f-M, f-P, mPM \) and \( mPP \).

**Survival** – The probabilities of survival for each class are: \( S_{f-M}(\phi,\mu,\alpha,\beta) = (1/4)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta) \); \( S_{f-P}(\phi,\mu,\alpha,\beta) = (1/4)(1-\phi)(1-\mu) + (1/2)\phi(1-\mu)(1-\alpha) \); \( S_{mPM}(\phi,\mu,\alpha,\beta) = (1/4)(1-\phi)(1-\mu) + (1/2)(1-\phi)\mu(1-\beta) \); and \( S_{mPP}(\phi,\mu,\alpha,\beta) = (1/4)(1-\phi)(1-\mu) \).

**Reproductive success** – Contingent upon survival, expected reproductive success is \( R_{STU}(\phi',\mu',\alpha,\beta) = 1 \) for females and \( R_{mSTU}(\phi',\mu',\alpha,\beta) = (1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta) \) for males, where \( z(\phi',\mu',\alpha,\beta) = (S_{mPM}(\phi',\mu',\alpha,\beta) + S_{mPP}(\phi',\mu',\alpha,\beta))/(S_{f-M}(\phi',\mu',\alpha,\beta) + S_{f-P}(\phi',\mu',\alpha,\beta) + S_{mPM}(\phi',\mu',\alpha,\beta) + S_{mPP}(\phi',\mu',\alpha,\beta)) \).

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. \( w_{STU}(\phi,\phi',\mu,\mu',\alpha,\beta) = S_{STU}(\phi,\mu,\alpha,\beta) \times R_{STU}(\phi',\mu',\alpha,\beta) \). As before, because PGE is
<table>
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<tr>
<th>Recipient Class</th>
<th>Actor Class</th>
<th>A&lt;sub&gt;Mat&lt;/sub&gt;</th>
<th>A&lt;sub&gt;Pat&lt;/sub&gt;</th>
<th>A&lt;sub&gt;Mo&lt;/sub&gt;</th>
<th>A&lt;sub&gt;Fa&lt;/sub&gt;</th>
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<td>ρ&lt;sub&gt;PM&lt;/sub&gt;</td>
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<tr>
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<td>ρ</td>
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<td>ρ&lt;sub&gt;MP&lt;/sub&gt;</td>
<td>ρ&lt;sub&gt;PP&lt;/sub&gt;</td>
<td>a ρ</td>
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<tr>
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<td>(1+ρ)/2</td>
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<td>ρ&lt;sub&gt;MP&lt;/sub&gt;</td>
<td>ρ&lt;sub&gt;PP&lt;/sub&gt;</td>
<td>a ρ</td>
<td>a ρ</td>
</tr>
</tbody>
</table>

| mMM1’           | a(1+ρ)/2    | a               | a ρ             | a(1+ρ)/2    | a ρ         |
| mMM2’           | a ρ<sub>M</sub> | a ρ<sub>MM</sub>| a ρ<sub>PM</sub>| a<sup>2</sup>(1+ρ)/2 | a<sup>2</sup> ρ |
| mMP1’           | a(1+ρ)/2    | a               | a ρ             | a(1+ρ)/2    | a ρ         |
| mMP2’           | a ρ<sub>P</sub> | a ρ<sub>MP</sub>| a ρ<sub>PP</sub>| a<sup>2</sup> ρ | a<sup>2</sup> ρ |
| mPM1’           | a(1+ρ)/2    | a               | a ρ             | a(1+ρ)/2    | a ρ         |
| mPM2’           | a ρ<sub>M</sub> | a ρ<sub>MM</sub>| a ρ<sub>PM</sub>| a<sup>2</sup>(1+ρ)/2 | a<sup>2</sup> ρ |
| mPP1’           | a(1+ρ)/2    | a               | a ρ             | a(1+ρ)/2    | a ρ         |
| mPP2’           | a ρ<sub>P</sub> | a ρ<sub>MP</sub>| a ρ<sub>PP</sub>| a<sup>2</sup> ρ | a<sup>2</sup> ρ |

Table A3.1.1. Consanguinities for the female PGE invasion analysis: autosomal genes. The five autosomal actor classes are the female’s autosomal genes (A), her maternal-origin autosomal genes (A<sub>Mat</sub>), her paternal-origin autosomal genes (A<sub>Pat</sub>), her mother’s autosomal genes (A<sub>Mo</sub>) and her father’s autosomal genes (A<sub>Fa</sub>). Shown here are their consanguinities ρ<sub>PSTUv</sub> to each of the recipient classes sTUv among the focal female’s offspring, and the consanguinities p<sub>fSTUv</sub> to each of the recipient classes mTUv’, among the males competing for mates with the focal female’s sons.

*fitness can be expressed relative to the average of their class, by W<sub>sTU</sub>(ϕ,ϕ′,μ,μ′,α,β) = W<sub>sTU</sub>(ϕ,ϕ′,μ,μ′,α,β)/ W<sub>sTU</sub>(α,β).*  

vanishingly rare in the population, the average fitness among all individuals of a particular class is given by  W<sub>sTU</sub>(α,β) = W<sub>sTU</sub>(0,0,0,α,β), and each individual’s

Gene fitness – There are 6 classes of gene, because individuals of each of the two female classes carry only paternal-origin genes, whereas individuals of each of the two male classes carry separate maternal-origin and paternal-origin genes. As before, gene classes are denoted in the form sTUv, where v ∈ {1,2} according to
PGE1 in females

Reproductive values and corresponding system of 6 linear equations may be written in linear algebraic form \( \mathbb{P} \) probability.

from genes of class generation.

For example, genes of class reproductive value for a gene in a male is given by \( W_{mPP2} (f_1) = W_{f1} (\phi, \mu, \mu', \alpha, \beta) = W_{mPP2} (f_1) = W_{mPP2} (\phi, \mu, \mu', \alpha, \beta) \).

Table A3.1.2. Consanguinities for the male PGE analysis: autosomal genes. The five autosomal actor classes are the male's autosomal genes (A), his maternal-origin autosomal genes (A_{mat}), his paternal-origin autosomal genes (A_{pat}), his mother's autosomal genes (A_{mom}), and his father's autosomal genes (A_{dad}). Shown here are their consanguinities \( q_{STUV} \) to each of the recipient classes \( \alpha_{STUV} \) among the male's offspring.

whether the gene is of maternal (\( v = 1 \)) or paternal (\( v = 2 \)) origin. The relative fitness of a gene in a female is given by \( W_{f1} (\phi, \mu, \mu', \alpha, \beta) = W_{f1}(\phi, \mu, \mu', \alpha, \beta) = W_{mPP2}(\phi, \mu, \mu', \alpha, \beta) \).

Reproductive value – As before, each gene’s class in any generation may provide some information and some uncertainty about that gene’s origin in the previous generation. For example, genes of class f-P2 in the present generation are derived from genes of class mPU2 in the previous generation, where \( U = M \) or \( P \) with equal probability. Consequently, the f-P2 gene derived from a mPM2 gene with probability \( q_{f1-P2,mPM2} = 1/2 \) and from a mPP2 gene with probability \( q_{f1-P2,mPP2} = 1/2 \). The corresponding system of 6 linear equations may be written in linear algebraic form as \( c = c \cdot G \),

where \( c = \{ c_{f1-M2}, c_{f1-P2}, c_{mPM2}, c_{mPP1}, c_{mPP2} \} \) is the vector of class reproductive values and \( G \) is the gene-flow matrix. Solving obtains \( c_{STUV} = 1/6 \) for all gene classes.

PGE in females – Following the same procedure as before, the condition for increase in female PGE is \( c_{f1-M2} \).

\[
\begin{array}{cccc}
\text{Recipient} & A & A_{mat} & A_{pat} & A_{mot} & A_{fat} \\
\text{Class} & & & & & \\
\hline
fMM1 & \rho_m & \rho_{MM} & \rho_p & \rho_{MP} & a(1+\rho)/2 & a \rho \\
fMM2 & (1+\rho)/2 & 1 & \rho & (1+\rho)/2 & \rho & \\
fMP1 & \rho_m & \rho_{MM} & \rho_p & a(1+\rho)/2 & a \rho \\
fMP2 & (1+\rho)/2 & \rho & 1 & (1+\rho)/2 & a \rho \\
fPM1 & \rho_p & \rho_{PM} & \rho_p & a \rho & \rho \\
fPM2 & (1+\rho)/2 & 1 & \rho & (1+\rho)/2 & a \rho \\
fPP1 & \rho_p & \rho_{PM} & \rho_p & a \rho & a \rho \\
fPP2 & (1+\rho)/2 & \rho & 1 & (1+\rho)/2 & a \rho \\
mMM1 & \rho_m & \rho_{MM} & \rho_p & a(1+\rho)/2 & a \rho \\
mMM2 & (1+\rho)/2 & 1 & \rho & (1+\rho)/2 & \rho & \\
mMP1 & \rho_m & \rho_{MM} & \rho_p & a(1+\rho)/2 & a \rho \\
mMP2 & (1+\rho)/2 & \rho & 1 & (1+\rho)/2 & a \rho \\
mPP1 & \rho_p & \rho_{PM} & \rho_p & a \rho & \rho \\
mPP2 & (1+\rho)/2 & \rho & 1 & (1+\rho)/2 & a \rho \\
\end{array}
\]
\[
\left( dW_{mPM1}/d\phi \right) \times (d\phi/dG) \times (dG'/dg_{mPM1}) + c_{mPM2} \left( (dW_{mPM2}/d\phi) \times (d\phi/dG) \times dG/dg_{mPM2} \right) + (dW_{mPM2}/d\phi) \times (d\phi/dG') \times (dG'/dg_{mPM2}) + c_{mPP1} \left( (dW_{mPP1}/d\phi) \times (d\phi/dG) \times (dG'/dg_{mPP1}) \right) + c_{mPP2} \left( (dW_{mPP2}/d\phi) \times (d\phi/dG) \times (dG'/dg_{mPP2}) + (dW_{mPP2}/d\phi) \times (d\phi/dG') \times (dG'/dg_{mPP2}) \right) > 0,
\]
where \( \phi \) and \( G \) are the consanguinity between the focal class-\( s \)TUv gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier; and \( dG'/dg_{mTUv} = p_{mTUv} \) is the consanguinity between the focal class-\( s \)TUv gene and the genes, residing at the same locus, who control PGE in the females who contribute offspring to the focal individual’s mating group.

\[ PGE \text{ in males} \quad - \text{Similarly, the condition for increase in male PGE is } c_{1-M2} \left( (dW_{1-M2}/d\mu) \times (d\mu/dG) \times (dG/dg_{1-M2}) \right) + c_{1-P2} \left( (dW_{1-P2}/d\mu) \times (d\mu/dG) \times (dG/dg_{1-P2}) \right) + c_{mPM1} \left( (dW_{mPM1}/d\mu) \times (d\mu/dG) \times (dG/dg_{mPM1}) \right) + c_{mPM2} \left( (dW_{mPM2}/d\mu) \times (d\mu/dG) \times (dG/dg_{mPM2}) \right) + c_{mPP1} \left( (dW_{mPP1}/d\mu) \times (d\mu/dG) \times (dG/dg_{mPP1}) \right) + c_{mPP2} \left( (dW_{mPP2}/d\mu) \times (d\mu/dG) \times (dG/dg_{mPP2}) \right) > 0, \]
where \( d\mu/dG = 1 \), and \( dG/dg_{sTUv} = q_{sTUv} \) is the consanguinity between the focal class-\( s \)TUv gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier.

\[ \text{Consanguinity} \quad - \text{The coefficient of inbreeding for Z-linked genes is } \rho^{**} = a((1/2) \times \rho^{**} + (1/2) \times \psi^{**}). \text{ That is: with probability } a \text{ the male and female share the same mother; so with probability } \frac{1}{2} \text{ we pick the paternal-origin gene from the female and the maternal-origin gene from the male and hence their consanguinity is simply the consanguinity of mating partners, } \rho^{**}; \text{ and with probability } \frac{1}{2} \text{ we pick the paternal-origin genes from both individuals, in which case their consanguinity is that of two males in the same mating group, or } \psi^{**}. \text{ Note that } \psi^{**} = a((1/4) \times 1 + (1/2) \times \rho^{**} + (1/4) \times \psi^{**}). \text{ That is, with probability } a \text{ the two males share the same mother; so with probability } \frac{1}{4} \text{ we pick the maternal-origin genes from both males, so their consanguinity is simply the mother’s consanguinity to herself, i.e. } 1; \text{ with probability } \frac{1}{2} \text{ we pick the maternal-origin gene from one male and the paternal-origin gene from the other, in which case their consanguinity is that between mating partners, } \rho^{**}; \text{ and with probability } \frac{1}{4} \text{ we pick the paternal-origin genes from both males, so their consanguinity is that of two males in the same mating group, } \psi^{**}. \text{ Simultaneously solving both equations obtains } \rho^{**} = a^2/(8-a(6+a)) \text{ and } \psi^{**} = a(2-a)/(8-a(6+a)). \text{ From these coefficients can be defined other consanguinities between mating partners: } \rho^{**}_M = a \rho^{**}, \text{ from the perspective of the male’s maternal-origin gene; and } \rho^{**}_P = a \psi^{**}, \text{ from the perspective of the male’s paternal-origin gene. These coefficients define all the consanguinities needed to solve the model (listed in Tables A3.2.1 & A3.2.2).} \]

\[ \text{Potential for PGE} \quad - \text{The potentials for female and male PGE are calculated as described above. These are illustrated in Figure 3 of the main text.} \]
Survival

1. linked genes. Thus, class isnotated in the form $s \in \{f, m\}$. If the individual is female, then all of her W-linked genes are maternal in origin (and came from her maternal grandmother). And if the individual is male, he has no W-linked genes. Thus, class is noted in the form $s \in \{f, m\}$.

Survival – The probabilities of survival for each class are $S(s, \mu, \alpha, \beta) = (1/2)(1-\phi)(1-\mu) + \phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$ and $S_m(s, \mu, \alpha, \beta) = (1/2)(1-\phi)(1-\mu) + (1/2)(1-\phi)\mu(1-\beta)$. 

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<thead>
<tr>
<th>Actor Class</th>
<th>Recipient Class</th>
<th>$Z$</th>
<th>$Z_{\text{Mot}}$</th>
<th>$Z_{\text{Fat}}$</th>
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</tbody>
</table>

Table A3.2.1. Consanguinities for the female PGE invasion analysis: Z-linked genes. The three Z-linked actor classes are the female’s Z-linked genes ($Z$), her mother’s Z-linked genes ($Z_{\text{Mot}}$) and her father’s Z-linked genes ($Z_{\text{Fat}}$). Shown here are their consanguinities $p_{sTUv}$ to each of the recipient classes $sTUv$ among the focal female’s offspring, and the consanguinities $p'_{sTUv}$ to each of the recipient classes $mTUv'$, among the males competing for mates with the focal female’s sons.

<table>
<thead>
<tr>
<th>Actor Class</th>
<th>Recipient Class</th>
<th>$Z$</th>
<th>$Z_{\text{Mat}}$</th>
<th>$Z_{\text{Pat}}$</th>
<th>$Z_{\text{Mot}}$</th>
<th>$Z_{\text{Fat}}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>f-M2</td>
<td>$(1+\rho_0^{-})/2$</td>
<td>1</td>
<td>$\rho_0^{-}$</td>
<td>1</td>
<td>$\rho_0^{-}$</td>
<td></td>
</tr>
<tr>
<td>f-P2</td>
<td>$(1+\rho_0^{-})/2$</td>
<td>$\rho_0^{-}$</td>
<td>1</td>
<td>$\rho_0^{-}$</td>
<td>$(1+\rho_0^{-})/2$</td>
<td></td>
</tr>
<tr>
<td>mPM1</td>
<td>$\rho_0^{-}$</td>
<td>$\rho_0^{-}, M$</td>
<td>$\rho_0^{-}, P$</td>
<td>$a \rho_0^{-}$</td>
<td>$a \rho_0^{-}$</td>
<td></td>
</tr>
<tr>
<td>mPM2</td>
<td>$(1+\rho_0^{-})/2$</td>
<td>1</td>
<td>$\rho_0^{-}$</td>
<td>1</td>
<td>$\rho_0^{-}$</td>
<td></td>
</tr>
<tr>
<td>mPP1</td>
<td>$\rho_0^{-}$</td>
<td>$\rho_0^{-}, M$</td>
<td>$\rho_0^{-}, P$</td>
<td>$a \rho_0^{-}$</td>
<td>$a \rho_0^{-}$</td>
<td></td>
</tr>
<tr>
<td>mPP2</td>
<td>$(1+\rho_0^{-})/2$</td>
<td>$\rho_0^{-}$</td>
<td>1</td>
<td>$\rho_0^{-}$</td>
<td>$(1+\rho_0^{-})/2$</td>
<td></td>
</tr>
</tbody>
</table>

Table A3.2.2. Consanguinities for the male PGE invasion analysis: Z-linked genes. The five Z-linked actor classes are the male’s Z-linked genes ($Z$), his maternal-origin Z-linked genes ($Z_{\text{Mat}}$), his paternal-origin Z-linked genes ($Z_{\text{Pat}}$), his mother’s Z-linked genes ($Z_{\text{Mot}}$) and his father’s Z-linked genes ($Z_{\text{Fat}}$). Shown here are their consanguinities $q_{sTUv}$ to each of the recipient classes $sTUv$ among the male’s offspring.

3.3. W-linked genes

Classes – There are 2 classes of individual, classified according to their sex $s \in \{f, m\}$. If the individual is female, then all of her W-linked genes are maternal in origin (and came from her maternal grandmother). And if the individual is male, he has no W-linked genes. Thus, class is noted in the form $s \in \{f, m\}$. 

Survival – The probabilities of survival for each class are $S(s, \mu, \alpha, \beta) = (1/2)(1-\phi)(1-\mu) + \phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi \mu (1-\alpha)(1-\beta)$ and $S_m(s, \mu, \alpha, \beta) = (1/2)(1-\phi)(1-\mu) + (1/2)(1-\phi)\mu(1-\beta)$. 

20
Reproductive success – Contingent upon survival, expected reproductive success is 
\( R(\phi',\mu',\alpha,\beta) = 1 \) for females and \( R_m(\phi',\mu',\alpha,\beta) = (1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta) \) for males, 
where \( z(\phi',\mu',\alpha,\beta) = S_m(\phi',\mu',\alpha,\beta)/(S_l(\phi',\mu',\alpha,\beta) + S_m(\phi',\mu',\alpha,\beta)) \).

Fitness – Expected fitness is given by the product of survival to maturity and 
expected reproductive success contingent upon surviving to maturity, i.e. 
\( w_5(\phi,\phi',\mu,\mu',\alpha,\beta) = S_l(\phi,\mu,\alpha,\beta)xR_m(\phi',\mu',\alpha,\beta) \). As before, because PGE is vanishingly 
rare in the population, the average fitness among all individuals of a particular class 
is given by \( \tilde{w}_5(\alpha,\beta) = w_5(0,0,0,0,\alpha,\beta) \), and each individual’s fitness can be expressed 
relative to the average of their class, by \( W_5(\phi,\phi',\mu,\mu',\alpha,\beta) = w_5(\phi,\phi',\mu,\mu',\alpha,\beta)/\tilde{w}_5(\alpha,\beta) \).

Gene fitness – There is only one class of W-linked gene, because females carry only 
one class of W-linked gene and males carry no W-linked genes. Accordingly, the 
relative fitness of a W-linked gene in a female is simply \( W_5(\phi,\phi',\mu,\mu',\alpha,\beta) \).

Reproductive value – Since there is only one class of W-linked gene, all reproductive 
value belongs to this class: \( r_l = 1 \).

PGE in females – Following the same procedure as before, the condition for increase 
in female PGE is \( c_l ((dW_l/d\phi) \times (d\phi/dG) \times (dG/dg_l)) > 0 \). Once again: \( d\phi/dG \) is an 
arbitrary mapping between genic value and phenotypic value, and can be set to 1; 
and \( dG/dg_l = p_l \) is the consanguinity between the focal gene and the genes, residing 
at the same locus, who control PGE in the mother of the focal gene’s carrier.

PGE in males – Similarly, the condition for increase in male PGE is \( c_l ((dW_l/d\mu) \times 
(d\mu/dG) \times (dG/dg_l)) > 0 \), where \( d\mu/dG = 1 \) and \( dG/dg_l = q_l \) is the consanguinity 
between the focal class-sTUV gene and the genes, residing at the same locus, who 
control PGE in the father of the focal gene’s carrier.

Consanguinity – Coefficients of inbreeding are undefined for W-linked genes, 
because males do not carry any such genes. Only three consanguinities feature in 
the W-linkage analysis: between a female’s W-genes and her mother’s W-genes; 
between a female’s W-genes and her maternal grandmother’s W-genes; and 
between a female’s W-genes and her paternal grandmother’s W-genes (listed in 
Tables A3.3.1 & A3.3.2).

Potential for PGE – The potentials for female and male PGE are calculated as 
described above. These are illustrated in Figure 3 of the main text.
Table A3.3.1. Consanguinities for the female PGE invasion analysis: W-linked genes. The two W-linked actor classes are the female’s own W-linked genes (W) and the female’s mother’s W-linked genes (WMot). Shown here are their consanguinity p_t to the single recipient class f among the focal female’s daughters.

<table>
<thead>
<tr>
<th>Recipient Class</th>
<th>Actor Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>f</td>
<td>W W_WMot</td>
</tr>
</tbody>
</table>

Table A3.3.2. Consanguinities for the male PGE invasion analysis: W-linked genes. The only W-linked actor class is the male’s mother’s W-linked genes (WMot). Shown here are their consanguinity q_t to the recipient class f among the focal male’s daughters.

<table>
<thead>
<tr>
<th>Recipient Class</th>
<th>Actor Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>f</td>
<td>W_WMot</td>
</tr>
</tbody>
</table>

4. MGE in females and/or males under ZW inheritance: invasion analysis

4.1. Autosomal genes

Classes – There are 8 classes of individual, classified according to their sex s ∈ {f,m}, the grandparent of origin of their maternal-origin gene T ∈ {M,P} and the grandparent of origin of their paternal-origin gene U ∈ {M,P}. Each class is notated in the form sTU, i.e. fMM, fMP, fPM, fPP, mMM, mMP, mPM and mPP.

Survival – Denoting the probability that a zygote’s mother exhibited MGE by φ, the probability that the zygote’s father exhibited MGE by µ, the viability cost associated with female MGE by α and the viability cost associated with male MGE by β, the probability that each potential zygote survives to maturity is: 

\[ S_{MM}(\phi,\mu,\alpha,\beta) = \frac{(1/8)(1-\phi)(1-\mu)}{\sum_{T=(M,P),U=(M,P)} S_{TU}(\phi',\mu',\alpha,\beta)} \]

Reproductive success – Upon survival to maturity, every female achieves a unit of expected reproductive success, and every male achieves \((1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta))\) units of expected reproductive success, where: 

\[ z(\phi',\mu',\alpha,\beta) = \frac{(\sum_{T=(M,P),U=(M,P)} S_{TU}(\phi',\mu',\alpha,\beta))}{(\sum_{s=(f,m),T=(M,P),U=(M,P)} S_{TU}(\phi',\mu',\alpha,\beta))} \]

is the sex ratio (proportion male) in his mating group; \(\phi',\mu'\) is the level of MGE among the females contributing offspring to the mating group; and \(\mu'\) is the level of MGE among the fathers contributing offspring to the mating group. Thus, contingent upon survival, expected
reproductive success is $R_{TU}(\phi',\mu',\alpha,\beta) = 1$ for females and $R_{mTU}(\phi',\mu',\alpha,\beta) = (1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta)$ for males.

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. $w_{TU}(\phi,\mu,\alpha,\beta) = S_{TU}(\phi,\mu,\alpha,\beta) \times R_{TU}(\phi,\mu,\alpha,\beta)$. Because MGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by $\bar{w}_{TU}(\alpha,\beta) = w_{TU}(0,0,0,\alpha,\beta)$, and each individual's fitness can be expressed relative to the average of their class, by $W_{TU}(\phi,\mu,\mu',\alpha,\beta) = w_{TU}(\phi,\mu,\mu',\alpha,\beta)/\bar{w}_{TU}(\alpha,\beta)$.

**Gene fitness** – There are 16 classes of gene, because every class of individual carries one maternal-origin gene and one paternal-origin gene. Thus, gene classes are denoted in the form $sTUv$, where $v \in \{1,2\}$ according to whether the gene is of maternal $(v = 1)$ or paternal $(v = 2)$ origin. The fitness of a gene, defined in terms of its probability of being transmitted to a potential (rather than a realised) zygote, is proportional to the fitness of the individual who carries it. Consequently, the relative fitness of a gene is equal to the relative fitness of its carrier: $W_{TU1}(\phi,\mu,\mu',\alpha,\beta) = W_{TU2}(\phi,\mu,\mu',\alpha,\beta) = W(\phi,\mu,\mu',\alpha,\beta)$.

**Reproductive value** – The flow of autosomal genes between classes is exactly the same under XY/XO and ZW inheritance, so the class reproductive values are identical to those derived in section 2.1.1, i.e. $c_{SUv} = 1/16$ for all gene classes.

**MGE in females** – The condition for increase in female MGE is

$$
\sum_{s \in \{f,m\}, T \in \{M,P\}, U \in \{M,P\}, v \in \{1,2\}} \frac{c_{SUv}}{(dW_{TUv}/d\phi) \times (d\phi/dG) \times (dG/dg_{SUv}) + (dW_{TUv}/d\phi') \times (d\phi'/dG') \times (dG'/dg_{SUv})} > 0,
$$

where $G$ is the average genic value among those genes at the same locus whose expression controls MGE in the focal individual’s mother, and $G'$ is the average genic value among those genes at the same locus whose expression controls MGE in the females contributing offspring to the focal individual’s mating group. Here: $d\phi/dG = d\phi'/dG' = 1$; $dG/dg_{SUv} = p_{SUv}$ is the consanguinity between the focal class-$sTUv$ gene and the genes, residing at the same locus, who control MGE in the mother of the focal gene’s carrier; and $dG'/dg_{SUv} = p_{SUv}'$ is the consanguinity between the focal class-$sTUv$ gene and the genes, residing at the same locus, who control MGE in the females who contribute offspring to the focal individual’s mating group. Note that, because $dW_{TUv}/d\phi' = 0$ for all classes of genes in females, we need only calculate consanguinities $p_{mSUv}$ for classes of genes in males. Again, the consanguinities will depend upon which class or classes of genes control MGE in females -- see the Consanguinity section below.

**MGE in males** – Similarly, the condition for increase in male MGE is

$$
\sum_{s \in \{f,m\}, T \in \{M,P\}, U \in \{M,P\}, v \in \{1,2\}} \frac{c_{SUv}}{(dW_{TUv}/d\mu) \times (d\mu/dG) \times (dG/dg_{SUv})} > 0,
$$

where $G$ is the average genic value among those genes at the same locus whose expression controls MGE in the focal individual’s father. Here, $d\mu/dG = 1$, and $dG/dg_{SUv} = q_{SUv}$ is the consanguinity between the focal class-$sTUv$ gene and the genes, residing at the same
locus, who control MGE in the father of the focal gene’s carrier. Again, the consanguinities will depend upon which class or classes of genes control MGE in males -- see the Consanguinity section below.

Consanguinity – The inheritance of autosomal genes is exactly the same under XY/XO and ZW inheritance so, just as in section 2.1: the consanguinity of mating partners is \( \rho = a/(8-7\alpha) \). Moreover, as before, we have \( \rho_M = a((1/2) \times (1+\rho) + (1/2) \times \rho) \), \( \rho_P = a \rho \), \( \rho_{MP} = a \rho \), \( \rho_{PM} = a \rho \), \( \rho_{PP} = a \rho \), and \( \rho_{MM} = a (1+\rho)/2 \), \( \rho_{MP} = a \rho \), \( \rho_{PM} = a \rho \), and \( \rho_{PP} = a \rho \). These coefficients define all the consanguinities needed to solve the model (listed in Tables A3.1.1 & A3.1.2).

Potential for MGE – The potential for female MGE and the potential for male MGE are calculated in the same way as before, and these are illustrated in Figure 3 of the main text.

4.2. Z-linked genes

Classes – There are 4 classes of individual, classified according to their sex \( s \in \{f,m\} \) and the grandparent of origin of their paternal-origin gene \( U \in \{M,P\} \). If the individual is female, she has no maternal-origin Z-linked genes. And if the individual is male, then all of his maternal-origin Z-linked genes are derived from his maternal grandfather. Thus, each class is notated in the form \( sTU \), where \( T \in \{M,-\} \), i.e. \( fM, fP, mP \) and \( mPP \).

Survival – The probabilities of survival for each class are:

\[
S_{sTU}(\phi,\mu,\alpha,\beta) = \frac{1}{4}(1-\phi)(1-\mu); S_{fP}(\phi,\mu,\alpha,\beta) = \frac{1}{4}(1-\phi)(1-\mu) + (1/2)(1-\phi)(1-\mu); S_{mPP}(\phi,\mu,\alpha,\beta) = \frac{1}{4}(1-\phi)(1-\mu) + (1/2)(1-\phi)(1-\mu)(1-\alpha) + (1/2)(1-\phi)(1-\mu) \]

Reproductive success – Contingent upon survival, expected reproductive success is

\[
R_{STU}(\phi',\mu',\alpha,\beta) = 1 \text{ for females and } R_{mTU}(\phi',\mu',\alpha,\beta) = (1-z(\phi',\mu',\alpha,\beta))/z(\phi',\mu',\alpha,\beta) \text{ for males, where } z(\phi',\mu',\alpha,\beta) = (S_{mPM}(\phi',\mu',\alpha,\beta) + S_{mPP}(\phi',\mu',\alpha,\beta))/S_{fM}(\phi',\mu',\alpha,\beta) + S_{fP}(\phi',\mu',\alpha,\beta) + S_{mPP}(\phi',\mu',\alpha,\beta) + S_{mPM}(\phi',\mu',\alpha,\beta)).
\]

Fitness – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e.

\[
w_{STU}(\phi,\phi',\mu,\mu',\alpha,\beta) = S_{STU}(\phi,\mu,\alpha,\beta) \times R_{STU}(\phi',\mu',\alpha,\beta). \]

As before, because MGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by

\[
\bar{w}_{sTU}(\alpha,\beta) = w_{sTU}(0,0,0,0,\alpha,\beta), \text{ and each individual's fitness can be expressed relative to the average of their class, by } W_{sTU}(\phi,\phi',\mu,\mu',\alpha,\beta) = w_{sTU}(\phi,\phi',\mu,\mu',\alpha,\beta)/\bar{w}_{sTU}(\alpha,\beta).
\]

Gene fitness – There are 6 classes of gene, because individuals of each of the two female classes carry only paternal-origin genes, whereas individuals of each of the two male classes carry separate maternal-origin and paternal-origin genes. As
before, gene classes are denoted in the form sTUv, where v ∈ {1,2} according to whether the gene is of maternal (v = 1) or paternal (v = 2) origin. The relative fitness of a gene in a female is given by \( W_{r-fU}((\phi,\mu,\gamma,\alpha,\beta)) = W_{r-u}((\phi,\mu,\gamma,\alpha,\beta)) \) and the relative fitness of a gene in a male is given by \( W_{r-mU}((\phi,\mu,\gamma,\alpha,\beta)) = W_{r-mU}((\phi,\mu,\gamma,\alpha,\beta)) \).

Reproductive value — The flow of Z-linked genes between classes is exactly the same as in section 3.2. Accordingly, \( c_{sTUv} = 1/6 \) for all gene classes.

MGE in females — Following the same procedure as before, the condition for increase in female MGE is \( c_{sTUv} = 1/6 \) for all gene classes.

MGE in males — Similarly, the condition for increase in male MGE is \( c_{sTUv} = 1/6 \) for all gene classes.

Consanguinity — The coefficients of consanguinity are exactly the same as described in section 3.2 (listed in Tables A3.2.1 & A3.2.2).

Potential for MGE — The potentials for female and male MGE are calculated as described above. These are illustrated in Figure 3 of the main text.

4.3. W-linked genes

Classes — There are 2 classes of individual, classified according to their sex \( s \in \{f,m\} \). If the individual is female, then all of her W-linked genes are maternal in origin (and came from her maternal grandmother). And if the individual is male, he has no W-linked genes. Thus, class is noted in the form \( s \in \{f,m\} \).
Survival – The probabilities of survival for each class are 
\[ S_t(\phi, \mu, \alpha, \beta) = \frac{1}{2}(1-\phi)(1-\mu) + (1/2)(1-\phi)\mu(1-\beta) \]
and 
\[ S_m(\phi, \mu, \alpha, \beta) = \frac{1}{2}(1-\phi)(1-\mu) + \phi(1-\mu)(1-\alpha) + (1/2)(1-\phi)\mu(1-\beta) + \phi\mu(1-\alpha)(1-\beta). \]

Reproductive success – Contingent upon survival, expected reproductive success is 
\[ R_t(\phi', \mu', \alpha, \beta) = 1 \] for females and 
\[ R_m(\phi', \mu', \alpha, \beta) = \frac{1-\phi(\phi', \mu', \alpha, \beta)}{1-\phi(\phi', \mu', \alpha, \beta)} \] for males, 
where 
\[ z(\phi', \mu', \alpha, \beta) = \frac{S_m(\phi', \mu', \alpha, \beta)}{S_t(\phi', \mu', \alpha, \beta)} = \frac{S_m(\phi', \mu', \alpha, \beta) + S_m(\phi', \mu', \alpha, \beta)}{S_t(\phi', \mu', \alpha, \beta)}. \]

Fitness – Expected fitness is given by the product of survival to maturity and 
expected reproductive success contingent upon surviving to maturity, i.e.
\[ w_s(\phi, \phi', \mu, \mu', \alpha, \beta) = S_s(\phi, \mu, \alpha, \beta) \times R_s(\phi', \mu', \alpha, \beta). \] As before, because MGE is vanishingly rare in the population, the average fitness among all individuals of a particular class is given by 
\[ \bar{w}_s(\alpha, \beta) = w_s(0,0,0,0,\alpha, \beta), \] and each individual’s fitness can be expressed relative to the average of their class, by 
\[ W_s(\phi, \phi', \mu, \mu', \alpha, \beta) = w_s(\phi, \phi', \mu, \mu', \alpha, \beta) / \bar{w}_s(\alpha, \beta). \]

Gene fitness – There is only one class of W-linked gene, because females carry only one class of W-linked gene and males carry no W-linked genes. Accordingly, the relative fitness of a W-linked gene in a female is simply 
\[ W_f(\phi, \phi', \mu, \mu', \alpha, \beta). \]

Reproductive value – Since there is only one class of W-linked gene, all reproductive value belongs to this class: \[ c_t = 1. \]

MGE in females – Following the same procedure as before, the condition for increase in female MGE is 
\[ c_t \left( (dW_t/d\phi) \times (d\phi/dG) \times (dG/dq_f) \right) > 0. \] Once again: \( d\phi/dG \) is an arbitrary mapping between genic value and phenotypic value, and can be set to 1; and 
\( dG/dq_f = p_t \) is the consanguinity between the focal gene and the genes, residing at the same locus, who control MGE in the mother of the focal gene’s carrier.

MGE in males – Similarly, the condition for increase in male MGE is 
\[ c_t \left( (dW_t/d\mu) \times (d\mu/dG) \times (dG/dq_f) \right) > 0, \] where 
\( d\mu/dG = 1 \) and 
\( dG/dq_f = q_t \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control MGE in the father of the focal gene’s carrier.

Consanguinity – The consanguinities are exactly the same as those derived in section 3.3 (listed in Tables A3.3.1 & A3.3.2).

Potential for MGE – The potentials for female and male MGE are calculated as described above. These are listed in illustrated in Figure 3 of the main text.

5. PGE in males under XY/XO inheritance: equilibrium analysis

5.1. Autosomal genes

Classes – There are 8 classes of individual, classified according to their sex \( s \in \{f,m\}, \) the grandparent of origin of their maternal-origin gene \( T \in \{M,P\} \) and the
grandparent of origin of their paternal-origin gene $U \in \{M, P\}$. Each class is noted in the form $sTU$, i.e. $fMM$, $fMP$, $fPM$, $fPP$, $mMM$, $mMP$, $mPM$ and $mPP$.

**Survival** – The zygote survival functions are obtained by substituting $\phi = \beta = 0$ into those of section 1.1. This yields $S_{MM}(\mu) = (1/8)(1-\mu) + (1/2)\mu$; $S_{MP}(\mu) = (1/8)(1-\mu)$; $S_{PM}(\mu) = (1/8)(1-\mu) + (1/2)\mu$; $S_{PP}(\mu) = (1/8)(1-\mu)$; $S_{mMM}(\mu) = (1/8)(1-\mu)$; $S_{mMP}(\mu) = (1/8)(1-\mu)$; $S_{mPM}(\mu) = (1/8)(1-\mu)$; and $S_{mPP}(\mu) = (1/8)(1-\mu)$.

**Reproductive success** – Upon survival to maturity, every female achieves a unit of expected reproductive success, and every male achieves $(1-z(\mu'/\mu))$ units of expected reproductive success, where: $z(\mu') = (\sum_{sTU} S_{sTU}(\mu')) / (\sum_{sTU} S_{sTU}(\mu'))$ is the sex ratio (proportion male) in his mating group and $\mu'$ is the level of PGE among the fathers contributing offspring to the mating group. Thus, contingent upon survival, expected reproductive success is $R_{sTU}(\mu') = 1$ for females and $R_{mTU}(\mu') = (1-z(\mu'/\mu))$ for males.

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. $w_{sTU}(\mu, \mu') = S_{sTU}(\mu) \times R_{sTU}(\mu')$. The average fitness among all individuals of a particular class is given by $\bar{w}_{sTU}(\mu) = w_{sTU}(\mu, \mu)$, and each individual’s fitness can be expressed relative to the average of their class, by $W_{sTU}(\mu, \mu') = w_{sTU}(\mu, \mu') / \bar{w}_{sTU}(\mu)$.

**Gene fitness** – There are 16 classes of gene, because every class of individual carries one maternal-origin gene and one paternal-origin gene. Thus, gene classes are denoted in the form $sTUv$, where $v \in \{1,2\}$ according to whether the gene is of maternal ($v = 1$) or paternal ($v = 2$) origin. The fitness of a gene, defined in terms of its probability of being transmitted to a potential (rather than a realised) zygote, is proportional to the fitness of the individual who carries it. Consequently, the relative fitness of a gene is equal to the relative fitness of its carrier: $W_{sTU1}(\mu, \mu', \mu) = W_{sTU2}(\mu, \mu', \mu)$.

**Reproductive value** – The flow of genes between classes determines each class’s reproductive value. Each gene’s class in any generation may provide some information and some uncertainty about that gene’s origin in the previous generation. For example, genes of class $fMM1$ in the present generation are derived from genes of class $fTU1$ in the previous generation, where $T = M$ or $P$ and $U = M$ or $P$. The effects of PGE in a male are accounted for in the survival of his potential offspring: for example, his potential male offspring are eliminated in the event that he undergoes PGE. So the class $fTU1$ donating genes to the class $fMM1$ involves $T = M$ or $P$ with equal probability, because there is no bias regarding the source of their maternal genes, but $U = M$ or $P$ with unequal probability, because there is possible bias regarding the source of their paternal genes. Specifically, $U = M$ with probability $\bar{w}_{fTU1}(\mu) = (1+3\mu)/(2+2\mu)$, and $U = P$ with probability $\bar{w}_{fTU1}(\mu) = (1-\mu)/(2+2\mu)$. Accordingly, $\phi_{fMM1-fMM1} = (1+3\mu)/(4+4\mu)$, $\phi_{fMM1-fPM1} = (1+3\mu)/(4+4\mu)$, $\phi_{fMM1-fPP1} = (1-\mu)/(4+4\mu)$.
and $φ_{\text{mem1-ppp}} = (1 - \bar{µ})/(4 + 4 \bar{µ})$. Note that, making the substitution $\bar{µ} = 0$, all of these gene-flow coefficients are equal to $\frac{1}{4}$, as in section 1.1. Also note that, if the donor class involves genes in males, then there is no bias, because no male is the product of a PGE father (all PGE events lead to the production of daughters). This defines a system of 16 linear equations, which can be written as $c = cG$, where $c = \{c_{\text{MM1}}, c_{\text{MM2}}, c_{\text{MP1}}, …, c_{\text{MPP2}}\}$ is the vector of class reproductive values and $G$ is the gene-flow matrix. Solving (i.e. finding the left eigenvector of $G$ corresponding to the eigenvalue 1) obtains $c_{\text{MM1}} = (1 + 3 \bar{µ})/(16 + 8 \bar{µ})$, $c_{\text{MM2}} = (1 + \bar{µ})/(16 + 8 \bar{µ})$ and $c_{\text{MP1}} = (1 - \bar{µ})/(16 + 8 \bar{µ})$. In the absence of male PGE ($\bar{µ} = 0$), all gene classes have reproductive value $c_{\text{MM1}} = 1/16$; that is, a gene chosen at random from the distant future has an equal chance of tracing back to any of the 16 gene classes in the present generation. In the extreme of full male PGE ($\bar{µ} = 1$), $c_{\text{MM1}} = 1/6$, $c_{\text{MM2}} = 0$, $c_{\text{MP1}} = 1/12$ and $c_{\text{MP2}} = 0$; that is, twothird of the third reproductive value of the population belongs to genes in females and only one third belongs to genes in males, just as under haplodiploidy.

**PGE in males** – The condition for increase in male PGE is $\sum_{s=(L,M),t=(M,P),u=(M,P),v=1,2} f_{\text{mmm},tu,v}(dW_{\text{cm},tu,v}/d\mu) \times (d\mu/dG) \times (dG/dg_{\text{cm},tu,v}) + (dW_{\text{mm},tu,v}/d\mu) \times (d\mu/dG') \times (dg'/dg_{\text{mm},tu,v}) > 0$, where $G$ is the average genic value among those genes at the same locus whose expression controls PGE in the focal individual’s father, and $G'$ is the average genic value among those genes at the same locus whose expression controls PGE in the males contributing offspring to the focal individual’s mating group. Here: $d\mu/dG = d\mu'/dG' = 1$; $dG/dg_{\text{mm},tu,v} = q_{\text{mm},tu,v}$ is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier; and $dg'/dg_{\text{mm},tu,v} = q_{\text{mm},tu,v}$ is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the males who contribute offspring to the focal individual’s mating group. Note that, because $dW_{\text{mm},tu,v}/d\mu' = 0$ for all classes of genes in females, we need only calculate consanguinities $q_{\text{mm},tu,v}$ for classes of genes in males. The consanguinities will depend upon which class or classes of genes control PGE in males -- see the Consanguinity section below, and Table A5.1.1, for details.

**Consanguinity** – The coefficient of inbreeding is $\rho = a((1/4) \times ((1/2) + (1/2) \times (\theta \rho_M + (1 - \theta)\rho)) + (1/4) \times (\theta \rho_M + (1 - \theta)\rho) + (1/4) \times (\theta \rho_M + (1 - \theta)\rho) + (1/4) \times (\theta \psi_M + (1 - \theta)\psi))$, where $\theta = 2 \bar{µ}/(1 + \bar{µ})$ is the probability that a focal adult female derived from an egg that was fertilized by a PGE sperm. Note that whilst the consanguinity of mating partners is $\rho$, the consanguinity of the genes that they transmit to their offspring is $\theta \rho_M + (1 - \theta)\rho$, because with probability $\theta$ the male exhibits PGE and so only transmits his maternal-origin genes, and with probability $1 - \theta$ the male does not exhibit PGE and hence transmits maternal-origin and paternal-origin genes with equal probability. The conditional consanguinities of mating partners are: $\rho_M = a((1/2)((1/2) + (1/2)(\theta \rho_M + (1 - \theta)\rho)) + (1/2)(\theta \rho_M + (1 - \theta)\rho))$, from the perspective of the male’s maternal-origin gene; $\rho_M = a((1/2)((1/2) + (1/2)(\theta \rho_M + (1 - \theta)\rho)) + (1/2)\rho)$, from the perspective of the female’s maternal-origin gene; $\rho_F = a((1/2)\rho + (1/2)(\theta \psi_M + (1-}
from the perspective of the male’s paternal-origin gene; $\rho_p = a((1/2)(\theta \rho_M + (1-\theta)\rho) + (1/2)(\theta \rho_M + (1-\theta)\psi))$, from the perspective of the female’s paternal-origin gene; $\rho_{MP} = a((1/2) + (1/2)(\theta \rho_M + (1-\theta)\rho))$, from the perspective of both mating partners’ maternal-origin genes; $\rho_{MP} = a\psi$, from the perspective of the female’s paternal-origin gene and the male’s paternal-origin gene; and $\rho_{PP} = a(\theta \psi_M + (1-\theta)\psi)$, from the perspective of both mating partners’ maternal-origin genes. The consanguinity of two males in the same mating group is $\psi = a((1/4)((1/2) + (1/2)(\theta \rho_M + (1-\theta)\rho)) + (1/2) + (1/2)\psi)$, and the conditional consanguinities between males are: $\psi_{MP} = a((1/2)((1/2) + (1/2)(\theta \rho_M + (1-\theta)\rho)) + (1/2) + (1/2)\psi)$, from the perspective of one male’s maternal-origin gene; $\psi_{MP} = a((1/2) + (1/2)(\theta \rho_M + (1-\theta)\rho))$, from the perspective of both males’ maternal-origin genes; $\psi_{MP} = a\psi$, from the perspective of both males’ paternal-origin genes. The consanguinity of two females in the same mating group is $\zeta = a((1/4)((1/2) + (1/2)(\theta \rho_M + (1-\theta)\rho)) + (1/2) + (1/2)(\theta \rho_M + (1-\theta)\rho)) + (1/2)(\theta \rho_M + (1-\theta)\rho) + (1/4)(\theta \rho_M + (1-\theta)\rho)$, from the perspective of one female’s maternal-origin gene; $\zeta_P = a((1/2)(\theta \rho_M + (1-\theta)\rho) + (1/2)(\theta \rho_M + (1-\theta)\rho))$, from the perspective of one female’s paternal-origin gene; $\zeta_{MP} = a((1/2) + (1/2)(\theta \rho_M + (1-\theta)\rho))$, from the perspective of both females’ maternal-origin genes; $\zeta_{MP} = a(\theta \rho_M + (1-\theta)\rho)$, from the perspective of one female’s maternal-origin gene and the other’s paternal-origin gene; and $\zeta_{PP} = a(\theta \rho_M + (1-\theta)\rho)$, from the perspective of both females’ paternal-origin genes. Simultaneously solving these equations yields $\rho = a(2+2+2\rho)(\rho_M - a(2+2\rho)(\rho_M) + a(16+16+16\rho)\mu)/(16(1+\mu) - a(16+16+16\rho)\mu)$, and each of the other consanguinities. And these in turn define all the consanguinities needed to solve the model (listed in Table A5.1.1).

**Equilibrium level of PGE** – If the condition for increase in PGE is not satisfied at $\overline{\mu} = 0$, then PGE cannot invade the population from rarity, and its equilibrium level is $g^* = 0$. If the condition for increase in PGE is satisfied at $\overline{\mu} = 1$, then non-PGE cannot invade from rarity, and hence the equilibrium level of PGE is $g^* = 1$. Setting the LHS of the condition equal to zero, and solving for $\overline{\mu} = g^*$, obtains the intermediate equilibrium level of PGE. The equilibrium level of PGE is illustrated in Figure 4 of the main text.

5.2. X-linked genes

**Classes** – There are 4 classes of individual, classified according to their sex $s \in \{F,M\}$ and the grandparent of origin of their maternal-origin gene $T \in \{M,P\}$. If the individual is female, then all of her paternal-origin X-linked genes are derived from
### Table A5.1.1. Consanguinities for the male PGE equilibrium analysis: autosomal genes

The five autosomal actor classes are the male’s autosomal genes (A), his maternal-origin autosomal genes (A\text{Mat}), his paternal-origin autosomal genes (A\text{Pat}), his mother’s autosomal genes (A\text{Mot}) and his father’s autosomal genes (A\text{Fat}). Shown here are their consanguinities \( q_{sTUv} \) to each of the recipient classes \( sTUv \) among the males competing for mates with the focal male’s sons. Here, \( \gamma = (\theta \rho \cdot \gamma + (1-\theta)\rho) \) and \( \eta = (\theta \psi \cdot \eta + (1-\theta)\psi) \).

<table>
<thead>
<tr>
<th>Recipient Class</th>
<th>Actor Class</th>
<th>A</th>
<th>A\text{Mat}</th>
<th>A\text{Pat}</th>
<th>A\text{Mot}</th>
<th>A\text{Fat}</th>
</tr>
</thead>
<tbody>
<tr>
<td>fMM1</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MP} )</td>
<td>( a \gamma )</td>
<td>( a \rho )</td>
<td></td>
</tr>
<tr>
<td>fMM2</td>
<td>( (1+\rho)/2 )</td>
<td>1</td>
<td>( \rho )</td>
<td>( (1+\gamma)/2 )</td>
<td>( \rho )</td>
<td></td>
</tr>
<tr>
<td>fMP1</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MP} )</td>
<td>( a(1+\gamma)/2 )</td>
<td>( a \rho )</td>
<td></td>
</tr>
<tr>
<td>fMP2</td>
<td>( (1+\rho)/2 )</td>
<td>( \rho )</td>
<td>1</td>
<td>( \rho )</td>
<td>( (1+\rho)/2 )</td>
<td></td>
</tr>
<tr>
<td>fPM1</td>
<td>( \rho \text{PM} )</td>
<td>( \rho \text{PM} )</td>
<td>( \rho \text{PP} )</td>
<td>( a \gamma )</td>
<td>( a \eta )</td>
<td></td>
</tr>
<tr>
<td>fPM2</td>
<td>( (1+\rho)/2 )</td>
<td>1</td>
<td>( \rho )</td>
<td>( (1+\gamma)/2 )</td>
<td>( \rho )</td>
<td></td>
</tr>
<tr>
<td>fPP1</td>
<td>( \rho \text{PP} )</td>
<td>( \rho \text{PP} )</td>
<td>( \rho \text{PP} )</td>
<td>( a \gamma )</td>
<td>( a \eta )</td>
<td></td>
</tr>
<tr>
<td>fPP2</td>
<td>( (1+\rho)/2 )</td>
<td>( \rho )</td>
<td>1</td>
<td>( \rho )</td>
<td>( (1+\rho)/2 )</td>
<td></td>
</tr>
<tr>
<td>mMM1</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MP} )</td>
<td>( a(1+\gamma)/2 )</td>
<td>( a \rho )</td>
<td></td>
</tr>
<tr>
<td>mMM2</td>
<td>( (1+\rho)/2 )</td>
<td>( \rho )</td>
<td>1</td>
<td>( (1+\gamma)/2 )</td>
<td>( \rho )</td>
<td></td>
</tr>
<tr>
<td>mMP1</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MM} )</td>
<td>( \rho \text{MP} )</td>
<td>( a(1+\gamma)/2 )</td>
<td>( a \rho )</td>
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</tr>
<tr>
<td>mMP2</td>
<td>( (1+\rho)/2 )</td>
<td>( \rho )</td>
<td>1</td>
<td>( (1+\gamma)/2 )</td>
<td>( \rho )</td>
<td></td>
</tr>
<tr>
<td>mPP1</td>
<td>( \rho \text{PP} )</td>
<td>( \rho \text{PP} )</td>
<td>( \rho \text{PP} )</td>
<td>( a \gamma )</td>
<td>( a \eta )</td>
<td></td>
</tr>
<tr>
<td>mPP2</td>
<td>( (1+\rho)/2 )</td>
<td>( \rho )</td>
<td>1</td>
<td>( (1+\rho)/2 )</td>
<td>( \rho )</td>
<td></td>
</tr>
<tr>
<td>mMM1’</td>
<td>( a \rho \text{MM} )</td>
<td>( a \rho \text{MM} )</td>
<td>( a \rho \text{MP} )</td>
<td>( a^2(1+\gamma)/2 )</td>
<td>( a^2 \rho )</td>
<td></td>
</tr>
<tr>
<td>mMM2’</td>
<td>( a \psi \text{MM} )</td>
<td>( a \psi \text{MM} )</td>
<td>( a \psi \text{MP} )</td>
<td>( a \psi \text{MM} )</td>
<td>( a \psi \text{MP} )</td>
<td></td>
</tr>
<tr>
<td>mMP1’</td>
<td>( a \rho \text{MM} )</td>
<td>( a \rho \text{MM} )</td>
<td>( a \rho \text{MP} )</td>
<td>( a^2(1+\gamma)/2 )</td>
<td>( a^2 \rho )</td>
<td></td>
</tr>
<tr>
<td>mMP2’</td>
<td>( a \psi \text{MM} )</td>
<td>( a \psi \text{MM} )</td>
<td>( a \psi \text{MP} )</td>
<td>( a \psi \text{MM} )</td>
<td>( a \psi \text{MP} )</td>
<td></td>
</tr>
<tr>
<td>mPP1’</td>
<td>( a \rho \text{PP} )</td>
<td>( a \rho \text{PP} )</td>
<td>( a \rho \text{PP} )</td>
<td>( a \rho \text{PP} )</td>
<td>( a^2 \eta )</td>
<td></td>
</tr>
<tr>
<td>mPP2’</td>
<td>( a \psi \text{PP} )</td>
<td>( a \psi \text{PP} )</td>
<td>( a \psi \text{PP} )</td>
<td>( a \psi \text{PP} )</td>
<td>( a \psi \text{PP} )</td>
<td></td>
</tr>
</tbody>
</table>

\( \phi \mu, \alpha, \beta \) and \( \phi \mu, \alpha, \beta \) are the probabilities of survival for each class.

Survival – The probabilities of survival for each class are: \( S_{fMM}(\phi \mu, \alpha, \beta) = (1/4)(1-\mu) + (1/2)\mu \); \( S_{fPM}(\phi \mu, \alpha, \beta) = (1/4)(1-\mu) + (1/2)\mu \); \( S_{mMM}(\phi \mu, \alpha, \beta) = (1/4)(1-\mu) \); and \( S_{mMP}(\phi \mu, \alpha, \beta) = (1/4)(1-\mu) \).
Reproductive success – Contingent upon survival, expected reproductive success is \( R_{TU}(\mu) = 1 \) for females and \( R_{mTU}(\mu) = (1 - z(\mu))/z(\mu) \) for males, where \( z(\mu) = (S_{mm}(\mu') + S_{mp}(\mu'))/(S_{MM}(\mu') + S_{MP}(\mu') + S_{mm}(\mu') + S_{mp}(\mu')) \).

Fitness – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. \( \bar{w}_{STU}(\mu, \mu') = S_{STU}(\mu) \times R_{TU}(\mu') \). Average fitness among all individuals of a particular class is given by \( \bar{w}_{STU}(\bar{\mu}) = \bar{w}_{STU}(\bar{\mu}, \bar{\mu}) \), and each individual’s fitness can be expressed relative to the average of their class, by \( W_{STU}(\mu, \mu', \bar{\mu}) = \bar{w}_{STU}(\mu, \mu')/\bar{w}_{STU}(\bar{\mu}) \).

Gene fitness – There are 6 classes of gene, because individuals of each of the two female classes carry separate maternal-origin and paternal-origin genes, whereas individuals of each of the two male classes carry only maternal-origin genes. As before, gene classes are denoted in the form sTUv, where \( v \in \{1, 2\} \) according to whether the gene is of maternal \((v = 1)\) or paternal \((v = 2)\) origin. The relative fitness of a gene in a female is given by \( W_{fTP1}(\mu, \mu', \bar{\mu}) = W_{fTP2}(\mu, \mu', \bar{\mu}) = W_{fTP}(\mu, \mu', \bar{\mu}) \) and the relative fitness of a gene in a male is given by \( W_{mT-1}(\mu, \mu', \bar{\mu}) = W_{mT}(\mu, \mu', \bar{\mu}) \).

Reproductive value – PGE in males does not impact upon the gene-flow coefficients for X-linked genes, because males can only transmit maternal-origin X-linked genes anyway. Accordingly, all of the non-zero gene-flow coefficients are equal to \( \frac{1}{2} \), and reproductive value is \( c_{STUV} = 1/6 \) for all gene classes, as in section A1.2.

PGE in males – As before, the condition for increase in male PGE is \( c_{MM1} \)

\[
((d W_{MM1}/d \mu) \times (d \mu/d G) \times (d G/d g_{MM1})) + c_{MM2} \left((d W_{MM2}/d \mu) \times (d \mu/d G) \times (d G/d g_{MM2})\right) + c_{IP1} \left((d W_{IP1}/d \mu) \times (d \mu/d G) \times (d G/d g_{IP1})\right) + c_{IP2} \left((d W_{IP2}/d \mu) \times (d \mu/d G) \times (d G/d g_{IP2})\right) + c_{mM-1} \left((d W_{mM-1}/d \mu) \times (d \mu/d G) \times (d G/d g_{mM-1})\right) + (d W_{mM-1}/d \mu') \times (d \mu'/d G') \times (d G'/d g_{mM-1}) + c_{mP-1} \left((d W_{mP-1}/d \mu) \times (d \mu/d G) \times (d G/d g_{mP-1})\right) + (d W_{mP-1}/d \mu') \times (d \mu'/d G') \times (d G'/d g_{mP-1})) > 0,
\]

where: \( d \mu/d G = d \mu'/d G' = 1; \) \( d G'/d g_{TUv} \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier; and \( d G'/d g_{mTUv} = g_{mTUv} \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the males who contribute offspring to the focal individual’s mating group.

Consanguinity – Again, PGE in males does not impact upon the transmission of X-linked genes, because males can only transmit maternal-origin X-linked genes anyway. Accordingly, the coefficients of consanguinity are exactly the same as those given in Table A1.2.2.

Equilibrium level of PGE – If the condition for increase in PGE is not satisfied at \( \bar{\mu} = 0 \), then PGE cannot invade the population from rarity, and its equilibrium level is \( g^* = 0 \). If the condition for increase in PGE is satisfied at \( \bar{\mu} = 1 \), then non-PGE cannot invade from rarity, and hence the equilibrium level of PGE is \( g^* = 1 \). Setting the LHS of the condition equal to zero, and solving for \( \bar{\mu} = g^* \), obtains the intermediate

31
equilibrium level of PGE. The equilibrium level of PGE is illustrated in Figure 4 of the main text.

5.3. Y-linked genes

Classes – There are 2 classes of individual, classified according to their sex $s \in \{f,m\}$. If the individual is female, then she has no Y-linked genes. And if the individual is male, then all of his Y-linked genes are paternal in origin (and came from his paternal grandfather). Thus, class is notated in the form $s \in \{f,m\}$.

Survival – The probabilities of survival for each class are $S_f(\mu) = (1/2)(1-\mu) + \mu$ and $S_m(\mu) = (1/2)(1-\mu)$.

Reproductive success – Contingent upon survival, expected reproductive success is $R_f(\mu') = 1$ for females and $R_m(\mu') = (1-z(\mu'))/z(\mu')$ for males, where $z(\mu') = S_m(\mu')/(S(\mu') + S_m(\mu'))$.

Fitness – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. $w_s(\mu,\mu') = S_s(\mu)R_s(\mu')$. The average fitness among all individuals of a particular class is given by $\bar{w}_s(\mu) = w_s(\mu, \mu, \mu)$, and each individual’s fitness can be expressed relative to the average of their class, by $W_s(\mu,\mu', \mu) = w_s(\mu,\mu', \mu)/\bar{w}_s(\mu, \mu, \mu)$.

Gene fitness – There is only one class of Y-linked gene, because females carry no Y-linked genes and males carry one class of Y-linked genes. Accordingly, the relative fitness of a Y-linked gene in a male is simply $W_m(\mu,\mu', \mu)$.

Reproductive value – Since there is only one class of Y-linked gene, all reproductive value belongs to this class: $c_m = 1$.

PGE in males – As before, the condition for increase in male PGE is $c_m ((dW_m/d\mu) \times (d\mu/dG) \times (dG/dg_m)) + (dW_m/d\mu') \times (d\mu'/dG') \times (dG'/dg_m)) > 0$, where: $d\mu/dG = d\mu'/dG' = 1$; $dG/dg_m = q_m$ is the consanguinity between the focal class-sTUV gene and the genes, residing at the same locus, who control PGE in the father of the focal gene’s carrier; and $dG'/dg_m = q'_m$ is the consanguinity between the focal class-sTUV gene and the genes, residing at the same locus, who control PGE in the males who contribute offspring to the focal individual’s mating group.

Consanguinity – PGE in males does not impact upon consanguinity of Y-linked genes, which are exactly the same as those listed in Table A1.3.2.

Equilibrium level of PGE – If the condition for increase in PGE is not satisfied at $\mu = 0$, then PGE cannot invade the population from rarity, and its equilibrium level is $g^* = 0$. If the condition for increase in PGE is satisfied at $\mu = 1$, then non-PGE cannot invade from rarity, and hence the equilibrium level of PGE is $g^* = 1$. Setting the LHS...
of the condition equal to zero, and solving for $\bar{\mu} = g^*$, obtains the intermediate equilibrium level of PGE. The equilibrium level of PGE is illustrated in Figure 4 of the main text.

6. PGE in females under ZW inheritance: equilibrium analysis

6.1. Autosomal genes

*Classes* – There are 8 classes of individual, classified according to their sex $s \in \{f,m\}$, the grandparent of origin of their maternal-origin gene $T \in \{M,P\}$ and the grandparent of origin of their paternal-origin gene $U \in \{M,P\}$. Each class is notated in the form $sTUV$, i.e. $fMM$, $fMP$, $fPM$, $fPP$, $mMM$, $mMP$, $mPM$ and $mPP$.

*Survival* – The zygote survival functions are obtained by substituting $\mu = \alpha = 0$ into those of section 3.1. This yields: $s_{MM}(\phi) = (1/8)(1-\phi) + (1/2)\phi$; $s_{MP}(\phi) = (1/8)(1-\phi) + (1/2)\phi$; $s_{PM}(\phi) = (1/8)(1-\phi) + (1/2)\phi$; $s_{PP}(\phi) = (1/8)(1-\phi)$; $s_{mMM}(\phi) = (1/8)(1-\phi)$; $s_{mMP}(\phi) = (1/8)(1-\phi)$; $s_{mPM}(\phi) = (1/8)(1-\phi)$; and $s_{mPP}(\phi) = (1/8)(1-\phi)$.

*Reproductive success* – Upon survival to maturity, every female achieves a unit of expected reproductive success, and every male achieves $(1-z(\phi'))/z(\phi')$ units of expected reproductive success, where: $z(\phi') = (\sum_{T=\{M,P\},U=\{M,P\}} s_{TU}(\phi'))/ (\sum_{T=\{f,m\},U=\{M,P\}} s_{TU}(\phi'))$ is the sex ratio (proportion male) in his mating group. Thus, contingent upon survival, expected reproductive success is $R_{TU}(\phi') = 1$ for females and $R_{mTU}(\phi') = (1-z(\phi'))/z(\phi')$ for males.

*Fitness* – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. $w_{sTU}(\phi,\phi') = s_{TU}(\phi) \times R_{TU}(\phi')$. The average fitness among all individuals of a particular class is given by $\bar{w}_{sTU}(\phi') = w_{sTU}(\phi,\phi') \overline{\phi}$, where $\overline{\phi}$ is the average level of female PGE in the population. Each individual’s fitness can be expressed relative to the average of their class, by $W_{sTU}(\phi,\phi', \overline{\phi}) = w_{sTU}(\phi,\phi')/ \bar{w}_{sTU}(\phi')$.

*Gene fitness* – There are 16 classes of gene, because every class of individual carries one maternal-origin gene and one paternal-origin gene. Thus, genes are denoted in the form $sTUv$, where $v \in \{1,2\}$ according to whether the gene is of maternal ($v = 1$) or paternal ($v = 2$) origin. The fitness of a gene, defined in terms of its probability of being transmitted to a potential (rather than a realised) zygote, is proportional to the fitness of the individual who carries it. Consequently, the relative fitness of a gene is equal to the relative fitness of its carrier: $W_{sTU2}(\phi,\phi', \overline{\phi}) = W_{sTU1}(\phi,\phi', \overline{\phi})$.

*Reproductive value* – The flow of genes between classes determines each class’s reproductive value. Each gene’s class in any generation may provide some information and some uncertainty about that gene’s origin in the previous generation. For example, genes of class $fMM1$ in the present generation are derived
from genes of class fTU1 in the previous generation, where \( T = M \) or \( P \) and \( U = M \) or \( P \). The effects of PGE in a female are accounted for in the survival of her potential offspring; for example, her potential male offspring are eliminated in the event that she undergoes PGE. So the class fTU1 donating genes to the class fMM1 involves \( T = P \) with unequal probability, because there is possible bias regarding the source of the latter class's maternal-origin genes, and \( U = M \) or \( P \) with equal probability, because there is no bias regarding the source of their paternal genes. Specifically, \( T = M \) with probability \( \tilde{w}_{fMU1}/(\tilde{w}_{fMU1} + \tilde{w}_{fPU1}) = (1+3 \phi)/(2+2 \phi) \), and \( T = P \) with probability \( \tilde{w}_{fPU1}/(\tilde{w}_{fMU1} + \tilde{w}_{fPU1}) = (1- \phi)/(2+2 \phi) \). Accordingly, \( \varphi_{fMM1-fMU1} = (1+3 \phi)/(4+4 \phi) \), \( \varphi_{fMM1-fPM1} = (1 \phi)/(4+4 \phi) \), \( \varphi_{fMM1-fPM1} = (1- \phi)/(4+4 \phi) \) and \( \varphi_{fMM1-fPP1} = (1- \phi)/(4+4 \phi) \). Note that, making the substitution \( \phi = 0 \), all of these gene-flow coefficients are equal to \( \frac{1}{4} \), as in section 3.1. Also note that, if the donor class involves genes in males, then there is no bias, because no male is the product of a PGE mother (all PGE events lead to the production of daughters). This defines a system of 16 linear equations, which can be written as \( c = c \cdot G \), where \( c = \{c_{fMM1-fMM2}, c_{fMM1-fPM1}, \ldots, c_{fMM1-fPP1}\} \) is the vector of class reproductive values and \( G \) is the geneflow matrix. Solving (i.e., finding the left eigenvector of \( G \) corresponding to the eigenvalue 1) obtains \( c_{fMU1} = (1+3 \phi)/(16-8 \phi) \), \( c_{fMU2} = (1- \phi)/(16-8 \phi) \), \( c_{fPU1} = (1- \phi)/(8(2- \phi)) \), \( c_{fPU2} = (1- \phi)/(8(2- \phi)) \) and \( c_{fPM1} = (1- \phi)/(8(2- \phi)) \). In the absence of female PGE ( \( \phi = 0 \) ), all gene classes have reproductive value \( c_{fTU1} = 1/16 \). In the extreme of full female PGE ( \( \phi = 1 \) ), \( c_{fMU1} = \frac{1}{2} \), \( c_{fMU2} = 0 \), \( c_{fPU1} = 0 \), \( c_{fPU2} = 0 \) and \( c_{fPM1} = 0 \), i.e. all of the population’s reproductive value belongs to maternal grandmaternal genes in females (classes fMM1 and fMP1), and no gene in any male has any reproductive value.

**PGE in females** – The condition for increase in female PGE is \( \sum_{c \in \{M,P,U\}} \sum_{(M,P) \in \{M,P\}} \sum_{v \in \{1,2\}} c_{fTUv} \left( (dW_{fTUv}/d\phi) \times (d\phi/dG) \times (dG/d\varphi_{fTUv}) \right) > 0 \), where \( G \) is the average genic value among those genes at the same locus whose expression controls PGE in the focal individual’s mother, and \( G' \) is the average genic value among those genes at the same locus whose expression controls PGE in the females contributing offspring to the focal individual’s mating group. Here: \( d\phi/dG = d\phi'/dG' = 1 \); \( dG/d\varphi_{fTUv} = p_{fTUv} \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier; and \( dG'/d\varphi_{fTUv} = p'_{fTUv} \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the females who contribute offspring to the focal individual’s mating group.

**Consanguinity** – The coefficient of inbreeding is \( \rho = a((1/4)(1/2) + (1/2)(1/2)) \), where \( \lambda = 2 \phi/(1+ \phi) \) is the probability that a focal adult female derived from a PGE egg. The conditional consanguinities of mating partners are: \( \rho_{M} = a((1/2)(1/2) + (1/2)(1/2)) \), \( \rho_{U} = a((1/2)(1/2) + (1/2)(1/2)) \), \( \rho_{P} = a((1/2)(1/2) + (1/2)(1/2)) \), from the perspective of the female’s maternal-origin gene; \( \rho_{M} = a((1/2)(1/2) + (1/2)(1/2)) \), \( \rho_{M} = a((1/2)(1/2) + (1/2)(1/2)) \), \( \rho_{P} = a((1/2)(1/2) + (1/2)(1/2)) \), from the perspective of the male’s maternal-origin gene; \( \rho_{P} = a((1/2)(1/2) + (1/2)(1/2)) \).
female’s paternal-origin gene; $\rho_P = a((1/2)((\lambda \rho_M + (1-\lambda)\rho) + (1/2)\psi)$, from the perspective of the male’s paternal-origin gene; $\rho_{PP} = a((1/2) + (1/2)(\lambda \rho_M + (1-\lambda)\rho))$, from the perspective of both mating partners’ maternal-origin genes; $\rho_{MP} = a(\lambda \rho_M + (1-\lambda)\rho)$, from the perspective of the female’s maternal-origin gene and the male’s paternal-origin gene; $\rho_{PM} = a\rho$, from the perspective of the female’s paternal-origin gene and the male’s maternal-origin gene; and $\rho_{PP} = a\psi$, from the perspective of both mating partners’ paternal-origin genes. The consanguinity of two males in the same mating group is $\psi = a((1/4)((1/2) + (1/2)\times(\lambda \rho_M + (1-\lambda)\rho)) + (1/2)\rho + (1/4)\psi)$, and the conditional consanguinities between males are: $\psi_{M} = a((1/2)((1/2) + (1/2)\times(\lambda \rho_M + (1-\lambda)\rho)) + (1/2)\rho)$, from the perspective of one male’s maternal-origin gene; $\psi_{P} = a((1/2)\rho + (1/2)\psi)$, from the perspective of one male’s paternal-origin gene; $\psi_{MM} = a((1/2) + (1/2)(\lambda \rho_M + (1-\lambda)\rho))$, from the perspective of both males’ maternal-origin genes; $\psi_{MP} = a\rho$, from the perspective of both males’ paternal-origin genes and the other’s paternal-origin gene; and $\psi_{PP} = a\psi$, from the perspective of both males’ maternal-origin genes and the other’s maternal-origin gene; and the conditional consanguinities between females are: $\zeta_{M} = a((1/2)((\lambda^2 + (1-\lambda^2)((1/2) + (1/2)(\lambda \rho_M + (1-\lambda)\rho)) + (1/2)(\lambda \rho_M + (1-\lambda)\rho))$, from the perspective of one female’s maternal-origin gene; $\zeta_{P} = a((1/2)(\lambda \rho_M + (1-\lambda)\rho) + (1/2)\psi)$, from the perspective of one female’s paternal-origin gene; $\zeta_{MM} = a((\lambda^2 + (1-\lambda^2)((1/2) + (1/2)(\lambda \rho_M + (1-\lambda)\rho)())$, from the perspective of both females’ maternal-origin genes; $\zeta_{MP} = a(\lambda \rho_M + (1-\lambda)\rho)$, from the perspective of one female’s maternal-origin gene and the other’s paternal-origin gene; and $\zeta_{PP} = a\psi$, from the perspective of both females’ paternal-origin genes. Simultaneously solving these equations yields $\rho = a(4 + (4a^2)\phi)/(32(1 + \phi) - a(28 + (2a(26 + 3a))\phi))$, and each of the other consanguinities. And these in turn define all the consanguinities needed to solve the model (listed in Table A6.1.1).

Equilibrium level of PGE – If the condition for increase in PGE is not satisfied at $\bar{\phi} = 0$, then PGE cannot invade the population from rarity, and its equilibrium level is $g^* = 0$. If the condition for increase in PGE is satisfied at $\bar{\phi} = 1$, then non-PGE cannot invade from rarity, and hence the equilibrium level of PGE is $g^* = 1$. Setting the LHS of the condition equal to zero, and solving for $\bar{\phi} = g^*$, obtains the intermediate equilibrium level of PGE. The equilibrium level of PGE is illustrated in Figure 4 of the main text.

6.2. Z-linked genes

Classes – There are 4 classes of individual, classified according to their sex $s \in \{f, m\}$ and the grandparent of origin of their paternal-origin gene $U \in \{M, P\}$. If the individual is female, she has no paternal-origin Z-linked genes. And if the individual is male, then all of his paternal-origin Z-linked genes are derived from his maternal grandfather. Thus, each class is notated in the form $.STU$, where $T \in \{M, -, \}$, i.e. f-M, f-P, mPM and mPP.
<table>
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<tr>
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<th>APat</th>
<th>AMot</th>
<th>AFat</th>
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<tr>
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<td>a\rho_{PM}</td>
<td>a^{2}(1+\nu)/2</td>
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<tr>
<td>mPP1'</td>
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<tr>
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<td>a\rho_{-P}</td>
<td>a\rho_{MP}</td>
<td>a\rho_{PP}</td>
<td>a\rho_{PP}</td>
<td>a\rho_{PP}</td>
<td>a^{2}\rho</td>
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Table A6.1.1. Consanguinities for the female PGE equilibrium analysis: autosomal genes. The five autosomal actor classes are the female’s autosomal genes (A), her maternal-origin autosomal genes (AMat), her paternal-origin autosomal genes (APat), her mother’s autosomal genes (AMot) and her father’s autosomal genes (AFat). Shown here are their consanguinities \rho_{sTUv} to each of the recipient classes sTUv among the focal female’s offspring, and the consanguinities \rho_{sTUv}' to each of the recipient classes mTUv', among the males competing for mates with the focal female’s sons. Here, \nu = (\lambda \rho_{MP} + (1-\lambda)\rho).

**Survival** – The probabilities of survival for each class are: \(S_{sTU}(\phi) = (1/4)(1-\phi) + (1/2)\phi\); \(S_{sTU}(\phi) = (1/4)(1-\phi) + (1/2)\phi\); \(S_{sTU}(\phi) = (1/4)(1-\phi)\); and \(S_{sTU}(\phi) = (1/4)(1-\phi)\).

**Reproductive success** – Contingent upon survival, expected reproductive success is \(R_{sTU}(\phi') = 1\) for females and \(R_{sTU}(\phi') = (1-z(\phi'))/z(\phi')\) for males, where \(z(\phi') = (S_{sTU}(\phi') + S_{sTU}(\phi'))/(S_{sTU}(\phi') + S_{sTU}(\phi') + S_{sTU}(\phi') + S_{sTU}(\phi')).\)

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. \(w_{sTU}(\phi,\phi')\).
= S_{STU}(\phi) \times R_{STU}(\phi')$. The average fitness among all individuals of a particular class is given by $\bar{w}_{STU}(\bar{\phi}) = w_{STU}(\phi, \bar{\phi})$. Each individual’s fitness can be expressed relative to the average of their class, by $w_{STU}(\phi, \bar{\phi}) = w_{STU}(\phi, \bar{\phi}) / \bar{w}_{STU}(\bar{\phi})$.

**Gene fitness** – There are 6 classes of gene, because individuals of each of the two female classes carry only paternal-origin genes, whereas individuals of each of the two male classes carry separate maternal-origin and paternal-origin genes. As before, gene classes are denoted in the form sTUv, where $v \in \{1,2\}$ according to whether the gene is of maternal ($v = 1$) or paternal ($v = 2$) origin. The relative fitness of a gene in a female is given by $W_{i-U2}(\phi, \bar{\phi}) = W_{i-U}(\phi, \bar{\phi})$ and the relative fitness of a gene in a male is given by $W_{mPU2}(\phi, \bar{\phi}) = W_{mPU}(\phi, \bar{\phi})$.

**Reproductive value** – PGE in females does not impact upon the gene-flow coefficients for Z-linked genes, because females can only transmit paternal-origin Z-linked genes anway. Accordingly, all of the non-zero gene-flow coefficients are equal to 1/2, and reproductive value is $c_{STUV} = 1/6$ for all gene classes, as in section A3.2.

**PGE in females** – Following the same procedure as before, the condition for increase in female PGE is $c_{f-M2} (\{dW_{f-M2}/d\phi\} \times (d\phi/dG) \times (dG/dg_{f-M2})) + c_{f-P2} (\{dW_{f-P2}/d\phi\} \times (d\phi/dG) \times (dG/dg_{f-P2})) + (dW_{mPM1}/d\phi) \times (d\phi'/dG') \times (dG'/dg_{mPM1}) + c_{mPM2} (\{dW_{mPM2}/d\phi\} \times (d\phi'/dG') \times (dG'/dg_{mPM2})) + c_{mPP1} (\{dW_{mPP1}/d\phi\} \times (d\phi'/dG') \times (dG'/dg_{mPP1})) + (dW_{mPP2}/d\phi) \times (d\phi'/dG') \times (dG'/dg_{mPP2}) \times (d\phi'/dG') \times (dG'/dg_{mPP2}) > 0$, where: $d\phi'/dG = d\phi'/dG' = 1$; $dG'/dg_{STUV} = p_{STUV}$ is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier; and $dG'/dg_{mTUv} = p_{mTUv}$ is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, who control PGE in the females who contribute offspring to the focal individual’s mating group.

**Consanguinity** – Again, PGE in females does not impact upon the transmission of Z-linked genes, because females can only transmit maternal-origin Z-linked genes anway. Accordingly, the coefficients of consanguinity are exactly the same as those given in Table A3.2.1.

**Equilibrium level of PGE** – If the condition for increase in PGE is not satisfied at $\bar{\phi} = 0$, then PGE cannot invade the population from rarity, and its equilibrium level is $g^* = 0$. If the condition for increase in PGE is satisfied at $\bar{\phi} = 1$, then non-PGE cannot invade from rarity, and hence the equilibrium level of PGE is $g^* = 1$. Setting the LHS of the condition equal to zero, and solving for $\bar{\phi} = g^*$, obtains the intermediate equilibrium level of PGE. The equilibrium level of PGE is illustrated in Figure 4 of the main text.
6.3. W-linked genes

Classes – There are 2 classes of individual, classified according to their sex \( s \in \{f,m\} \). If the individual is female, then all of her W-linked genes are maternal in origin (and came from her maternal grandmother). And if the individual is male, he has no W-linked genes. Thus, class is notated in the form \( s \in \{f,m\} \).

Survival – The probabilities of survival for each class are \( S_f(\phi) = (1/2)(1-\phi) + \phi \) and \( S_m(\phi) = (1/2)(1-\phi) \).

Reproductive success – Contingent upon survival, expected reproductive success is \( R_f(\phi') = 1 \) for females and \( R_m(\phi') = (1-z(\phi'))/z(\phi') \) for males, where \( z(\phi') = S_m(\phi')/(S(\phi') + S_m(\phi')) \).

Fitness – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e. \( w_{STU}(\phi, \phi', \phi_f) = S_{STU}(\phi) \times R_{STU}(\phi') \). The average fitness among all individuals of a particular class is given by \( \bar{w}_{STU}(\phi_f) = w_{STU}(\phi, \phi_f) \). Each individual’s fitness can be expressed relative to the average of their class, by \( \bar{w}_{STU}(\phi, \phi_f) = w_{STU}(\phi, \phi_f)/\bar{w}_{STU}(\phi_f) \).

Gene fitness – There is only one class of W-linked gene, because females carry only one class of W-linked gene and males carry no W-linked genes. Accordingly, the relative fitness of a W-linked gene in a female is simply \( W_f(\phi, \phi_f) \).

Reproductive value – Since there is only one class of W-linked gene, all reproductive value belongs to this class: \( c_f = 1 \).

PGE in females – Following the same procedure as before, the condition for increase in female PGE is \( c_f ((dW_f/d\phi) \times (d\phi/dG) \times (dG/dg_f)) > 0 \). Once again: \( d\phi/dG \) is an arbitrary mapping between genic value and phenotypic value, and can be set to 1; and \( dG/dg_f = p_f \) is the consanguinity between the focal gene and the genes, residing at the same locus, who control PGE in the mother of the focal gene’s carrier.

Consanguinity – PGE in females does not impact upon consanguinity of W-linked genes, which are exactly the same as those listed in Table A3.3.1.

Equilibrium level of PGE – If the condition for increase in PGE is not satisfied at \( \bar{\phi} = 0 \), then PGE cannot invade the population from rarity, and its equilibrium level is \( g^* = 0 \). If the condition for increase in PGE is satisfied at \( \bar{\phi} = 1 \), then non-PGE cannot invade from rarity, and hence the equilibrium level of PGE is \( g^* = 1 \). Setting the LHS of the condition equal to zero, and solving for \( \bar{\phi} = g^* \), obtains the intermediate equilibrium level of PGE. The equilibrium level of PGE is illustrated in Figure 4 of the main text.
7. PGE in males under XY/XO inheritance: equilibrium analysis with a new mode of sex determination

7.1. Autosomal genes

**Classes** – There are 8 classes of individual, classified according to their sex \( s \in \{ f, m \} \), the grandparent of origin of their maternal-origin gene \( T \in \{ M, P \} \) and the grandparent of origin of their paternal-origin gene \( U \in \{ M, P \} \). Each class is notated in the form \( sTU \), i.e. \( fMM, fMP, fPM, fPP, mMM, mMP, mPM \) and \( mPP \).

**Survival** – If sex allocation \( z \) is determined independently of genotype, then the zygote survival functions are

\[
S_{fMM}(\mu, z) = (1-z)((1/4)(1-\mu) + (1/2)\mu); \quad S_{fMP}(\mu, z) = (1-z)((1/4)(1-\mu) + (1/2)\mu); \quad S_{fPM}(\mu, z) = (1-z)((1/4)(1-\mu) + (1/2)\mu);
\]

\[
S_{mMM}(\mu, z) = z((1/4)(1-\mu) + (1/2)\mu); \quad S_{mMP}(\mu, z) = z((1/4)(1-\mu) + (1/2)\mu); \quad S_{mPM}(\mu, z) = z((1/4)(1-\mu) + (1/2)\mu); \quad S_{mPP}(\mu, z) = z(1/4)(1-\mu).
\]

**Reproductive success** – Upon survival to maturity, every female achieves a unit of expected reproductive success, and every male achieves \((1-z')/z'\) units of expected reproductive success, where \( z' \) is the sex ratio of the group in which they compete for mates. Thus, contingent upon survival, expected reproductive success is \( R_{fTU}(z') = 1 \) for females and \( R_{mTU}(z') = (1-z')/z' \) for males.

**Fitness** – Expected fitness is given by the product of survival to maturity and expected reproductive success contingent upon surviving to maturity, i.e.

\[
w_{fTU}(\mu, z, z') = S_{fTU}(\mu, z) \times R_{fTU}(z').
\]

The average fitness among all individuals of a particular class is given by \( w_{fTU}(\overline{\mu}, \overline{z}) = w_{fTU}(\overline{\mu}, \overline{z}) \), and each individual’s fitness can be expressed relative to the average of their class, by \( W_{fTU}(\mu, z, z', \overline{z}) = w_{fTU}(\mu, z, z')/w_{fTU}(\overline{\mu}, \overline{z}) \).

**Gene fitness** – There are 16 classes of gene, because every class of individual carries one maternal-origin gene and one paternal-origin gene. Thus, gene classes are denoted in the form \( sTU_{v} \), where \( v \in \{ 1, 2 \} \) according to whether the gene is of maternal \( (v = 1) \) or paternal \( (v = 2) \) origin. The fitness of a gene, defined in terms of its probability of being transmitted to a potential (rather than a realised) zygote, is proportional to the fitness of the individual who carries it. Consequently, the relative fitness of a gene is equal to the relative fitness of its carrier:

\[
W_{fTU_{1}}(\mu, z, z', \overline{z}) = W_{fTU_{2}}(\mu, z, z', \overline{z}) = W_{fTU}(\mu, z, z', \overline{z}).
\]

**Reproductive value** – The flow of genes between classes determines each class’s reproductive value. Each gene’s class in any generation may provide some information and some uncertainty about that gene’s origin in the previous generation. For example, genes of class \( fMM1 \) in the present generation are derived from genes of class \( fTU1 \) in the previous generation, where \( T = M \) or \( P \) and \( U = M \) or \( P \). The effects of PGE in a male are accounted for in the survival of his potential offspring: for example, his potential male offspring are eliminated in the event that
he undergoes PGE. So the class ITU1 donating genes to the class IMM1 involves T = M or P with equal probability, because there is no bias regarding the source of their maternal genes, but U = M or P with unequal probability, because there is possible bias regarding the source of their paternal genes. Specifically, U = M with probability \( \bar{w}_{TM1} / (\bar{w}_{TM1} + \bar{w}_{TP1}) = (1 + \mu) / 2 \), and U = P with probability \( \bar{w}_{TP1} / (\bar{w}_{TM1} + \bar{w}_{TP1}) = (1 - \mu) / 2 \). Accordingly, \( \phi_{IM1-IM1} = (1 + \mu) / 4 \), \( \phi_{IM1-IP1} = (1 + \mu) / 4 \), \( \phi_{MM1-IM1} = (1 - \mu) / 4 \) and \( \phi_{MM1-IP1} = (1 - \mu) / 4 \). Note that, making the substitution \( \mu = 0 \), all of these gene-flow coefficients are equal to \( \frac{1}{4} \), as in section 1.1. This defines a system of 16 linear equations, which can be written as \( c = c \cdot G \), where \( c = \{ c_{MM1,MM2,CM1,\ldots,c_{MM2}} \} \) is the vector of class reproductive values and \( G \) is the gene-flow matrix. Solving (i.e. finding the left eigenvector of \( G \) corresponding to the eigenvalue 1) obtains \( c_{TMV} = (1 + \mu^2) / (16 + 8 \mu) \), \( c_{TPV} = (1 - \mu^2) / (16 + 8 \mu) \), \( c_{MM1} = (1 + \mu^2) / (16 + 8 \mu) \), \( c_{MM2} = (1 - \mu^2) / (16 + 8 \mu) \), \( c_{MP1} = (1 - \mu^2) / (16 + 8 \mu) \), and \( c_{MP2} = (1 + \mu^2) / (16 + 8 \mu) \).

**PGE in males** – The condition for increase in male PGE is \( \sum_{s=(LM),T=(M,P),U=(M,P),v=(1,2)} c_{STUV} dW_{STUV} / d\mu \times (d\mu / dG) \times (dG / d_{STUV}) > 0 \), where \( G \) is the average genic value among those genes at the same locus whose expression controls PGE in the focal individual’s father. Here: \( d\mu / dG = 1 \) and \( dG / d_{STUV} = q_{STUV} \) is the consanguinity between the focal class-sTUv gene and the genes, residing at the same locus, which control PGE in the father of the focal gene’s carrier. The consanguinities will depend upon which class or classes of genes control PGE in males -- see the Consanguinity section below, and Table A7.1.1, for details.

**Consanguinity** – Note that there are no sex differences in genes carried (but there are sex differences in genes transmitted), so \( \psi = \zeta = \rho \). The coefficient of inbreeding is \( \rho = a((1 + \mu^2) / (16 + 8 \mu)) + (1 - \mu^2) \rho \), from the perspective of one partner’s maternal-origin gene; \( \rho_{P} = a((1 - \mu^2) / (16 + 8 \mu)) + (1 - \mu^2) \rho \), from the perspective of one partner’s paternal-origin gene; \( \rho_{MM} = a((1 + \mu^2) / (16 + 8 \mu)) + (1 - \mu^2) \rho \), from the perspective of both partners’ maternal-origin genes; \( \rho_{MP} = a((1 - \mu^2) / (16 + 8 \mu)) + (1 - \mu^2) \rho \), from the perspective of both partners’ paternal-origin genes; \( \rho_{MP} = a((1 + \mu^2) / (16 + 8 \mu)) + (1 - \mu^2) \rho \), from the perspective of one partner’s maternal-origin gene and the other partner’s paternal-origin gene; and \( \rho_{PP} = a((1 + \mu^2) / (16 + 8 \mu)) + (1 - \mu^2) \rho \), from the perspective of both partners’ paternal-origin genes. Simultaneously solving these equations yields \( \rho = a(2 + a(3 - \mu^2) / (16 - a(4 - a(3 - \mu)(1 - \mu) - 4 \mu) \mu)) \), and each of the other consanguinities. And these in turn define all the consanguinities needed to solve the model (listed in Table A7.1.1).
the frequency of GE is impact upon the GE phenotype. If several genes of the same type (e.g. unimprinted autosomal variety), those that are beneficial are favoured by natural selection if \( \mu > 0 \), while those that are disfavoured by natural selection if \( \mu < 0 \). The corresponding selective change in the frequency of GE is \( \mu \times \text{var}(g) \times \text{var}(g) \times \text{var}(g) \), where \( \text{var}(g) \) is the additive genetic variance in GE contributed by that gene position. If several genes of the same type (e.g. unimprinted autosomal genes) impact upon the GE phenotype, then the corresponding selective change in the frequency of GE is \( \mu \times \text{var}(g) \times \text{var}(g) \), where \( \text{var}(g) \) is the total additive genetic

<table>
<thead>
<tr>
<th>Recipient Class</th>
<th>Actor Class</th>
<th>( A_{\text{Mat}} )</th>
<th>( A_{\text{Pat}} )</th>
<th>( A_{\text{Mot}} )</th>
<th>( A_{\text{Fat}} )</th>
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<tr>
<td>fMM1</td>
<td>( \rho_{\text{mm}} )</td>
<td>( \rho_{\text{mm}} )</td>
<td>( \rho_{\text{mp}} )</td>
<td>( a (1+\sigma)/2 )</td>
<td>( a \rho )</td>
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<tr>
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<td>( \sigma )</td>
<td>( (1+\sigma)/2 )</td>
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<tr>
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<tr>
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<tr>
<td>fPP2</td>
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<td>( \sigma )</td>
<td>( (1+\sigma)/2 )</td>
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<tr>
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<td>( \rho_{\text{mm}} )</td>
<td>( \rho_{\text{mp}} )</td>
<td>( a(1+\sigma)/2 )</td>
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<tr>
<td>mMM2</td>
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<td>( \sigma )</td>
<td>( (1+\sigma)/2 )</td>
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Table A7.1.1. Consanguinities for the male PGE equilibrium analysis: autosomal genes. The five autosomal actor classes are the male’s autosomal genes (A), his maternal-origin autosomal genes (A_{Mat}), his paternal-origin autosomal genes (A_{Pat}), his mother’s autosomal genes (A_{Mot}) and his father’s autosomal genes (A_{Fat}). Shown here are their consanguinities \( q_{su} \) to each of the recipient classes sTu among the male’s offspring, and the consanguinities \( q_{tu} \) to each of the recipient classes mTu+, among the males competing for mates with the focal male’s sons. Here, \( \sigma = (\mu \rho_{\text{Mat}} + (1-\mu)\rho) \).

Equilibrium level of PGE – If the condition for increase in PGE is not satisfied at \( \bar{\mu} = 0 \), then PGE cannot invade the population from rarity, and its equilibrium level is \( g^* = 0 \). If the condition for increase in PGE is satisfied at \( \bar{\mu} = 1 \), then non-PGE cannot invade from rarity, and hence the equilibrium level of PGE is \( g^* = 1 \). Setting the LHS of the condition equal to zero, and solving for \( \bar{\mu} = g^* \), obtains the intermediate equilibrium level of PGE.

8. Combined action of multiple genic actors with conflicting interests

8.1. Selection under conflict

Total selective change – As outlined in the above sections, a gene that promotes GE is favoured by natural selection if \( dW/dg > 0 \) and disfavoured by natural selection if \( dW/dg < 0 \). The corresponding selective change in the frequency of GE is \( (dW/dg) \times \text{var}(g) \), where \( \text{var}(g) \) is the additive genetic variance in GE contributed by that gene position. If several genes of the same type (e.g. unimprinted autosomal genes) impact upon the GE phenotype, then the corresponding selective change in the frequency of GE is \( (dW/dg) \times \text{var}(g) \), where \( \text{var}(g) \) is the total additive genetic
variance in GE contributed by those gene positions. And if several genes of different types (e.g. unimprinted autosomal genes and imprinted maternal-origin genes and paternal-origin genes) impact upon the GE phenotype, then the corresponding selective change in the frequency of GE is \( \sum_{T} (dW/dg)_t \pi_t \text{var}(g) \), where \( T \) is the set of all types of gene position impacting upon the GE phenotype, \( \text{var}(g) \) is the total additive genetic variance in GE contributed by those gene positions, \( (dW/dg)_t \) is the marginal fitness associated with gene positions of type \( t \), and \( \pi_t \) is the proportion of the total additive genetic variance that is contributed by gene positions of type \( t \).

**Condition for increase** – The condition for natural selection to favour an increase in the frequency of GE is \( \sum_{T} (dW/dg)_t \pi_t > 0 \), where the LHS of the condition represents a weighted average of the interests of the different genic types.

**Equilibrium point** – Note that \( (dW/dg)_t \leq \sum_{T} (dW/dg)_t \pi_t \leq (dW/dg)_t^* \), where \( t^* \in T \) is the type of gene position least-strongly favouring (or most-strongly disfavouring) GE – i.e. \( (dW/dg)_t \leq \sum_{T} (dW/dg)_t \pi_t \leq (dW/dg)_t^* \), for all \( t \in T \) – and \( t^* \in T \) is the type of gene position most-strongly favouring (or least-strongly disfavouring) GE – i.e. \( (dW/dg)_t \geq (dW/dg)_t^* \), for all \( t \in T \). Hence, \( \sum_{T} (dW/dg)_t \pi_t = 0 \) when \( (dW/dg)_t^* = 0 \), and \( \sum_{T} (dW/dg)_t \pi_t \leq 0 \) when \( (dW/dg)_t^* = 0 \), such that the equilibrium level of GE under conflict is bounded by the equilibria favoured by the extremist parties.

### 8.2. The parliament of genes

*Interests of maternal-origin and paternal-origin genes cancel* – The marginal fitness for a maternal-origin gene at a diploid locus may be expressed as \( (dW/dg)_M = -c + \sum_{i=1}^{U} b_i p_M, \) i.e. its inclusive-fitness effect, where \(-c\) is the impact an increase in gene expression has on its own replicative success, \( b_i \) is the impact on the replicative success of its social partner \( i \in I \), and \( p_M \) is its consanguinity with this social partner. The marginal fitness for the corresponding paternal-origin gene may be expressed as \( (dW/dg)_P = -c + \sum_{i=1}^{U} b_i p_P, \) where \( p_P \) need not be equal to \( p_M \) on account of social partners being more related through one parent than through the other. This asymmetry in consanguinity coefficients, and hence inclusive-fitness effects, is the reason for intragenomic conflict between maternal-origin and paternal-origin genes. If maternal-origin and paternal-origin genes contribute equally to additive genetic variance \( \pi_M = \pi_P = \pi \), then \( \sum_{(M,P)} (dW/dg)_i \pi_i > 0 \) is equivalent to \(-c + \sum_{i=1}^{U} b_i p_i > 0 \) where \( p_i = (p_M + p_P)/2 \) is the consanguinity that has not been conditioned upon parent of origin, and this inequality is equivalent to \((dW/dg)_U > 0 \), where \( U \) denotes the corresponding genes that lack information as to their parent of origin. For example, considering the evolution of PGE in males under XY/XO sex determination (section 1 above, and SM2 section 1), the marginal fitness for maternal-origin autosomal genes is \( (dW/dg)_M = (4-8\beta-a(4-5\beta-a(4-2a(1-\beta)-3\beta)))/(2(8-7a)) \) and the marginal fitness for paternal-origin autosomal genes is \( (dW/dg)_P = -(4-a(4- \beta+a(2a(1-\beta)-\beta)))/(2(8-7a)) \) and the average of these two exactly recovers the marginal fitness for unimprinted autosomal genes, \( (dW/dg)_U = (a^2(1-\beta)-2\beta+a\beta)/(8-7a) \). Accordingly, the combined action of multiple maternal-origin and paternal-
origin genes is expected to give an equilibrium level of GE that approximately coincides with that favoured by their unimprinted counterparts.

*Majority party dominates the conflict* – The weighted-average marginal fitness \[ \sum_{t \in T} (dW/dg)^t \pi_t \rightarrow (dW/dg), \pi_t \rightarrow 1. \] That is, the interests of the majority party dominate the conflict. If the individual’s own autosomal genes (including unimprinted genes and imprinted genes of maternal-origin and paternal-origin; see above) contribute most of the additive genetic variance in GE, for example on account of their numerical superiority over the sex chromosomes and also because the individual’s own genome is necessarily closely involved with the process of GE whereas its parents’ genomes can act only at a distance and so have less control, then the majority interest will be equivalent to that of the individual’s autosomal genes lacking information as to their parent of origin.