Evolution of haploid selection in predominantly diploid organisms

Sarah P. Otto[a,1], Michael F. Scott[b], and Simone Immler[c]

[a]Department of Zoology & Biodiversity Research Centre, University of British Columbia, Vancouver, BC, Canada V6T 1Z4; [b]Department of Botany & Biodiversity Research Centre, University of British Columbia, Vancouver, BC, Canada V6T 1Z4; and [c]Department of Ecology and Genetics, Evolutionary Biology, Uppsala University, 752 36 Uppsala, Sweden

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Diploid organisms manipulate the extent to which their haploid gametes experience selection. Animals typically produce sperm with a diploid complement of most proteins and RNA, limiting selection on the haploid genotype. Plants, however, exhibit extensive expression in pollen, with actively transcribed haploid genomes. Here we analyze models that track the evolution of genes that modify the strength of haploid selection to predict when evolution intensifies and when it dampens the "selective arena" within which male gametes compete for fertilization. Considering deleterious mutations, evolution leads diploid mothers to strengthen selection among haploid sperm/pollen, because this reduces the mutation load inherited by their diploid offspring. If, however, selection acts in opposite directions in haploids and diploids ("ploidally antagonistic selection"), mothers evolve to reduce haploid selection to avoid selectively amplifying alleles harmful to their offspring. Consequently, with maternal control, selection in the haploid phase either is maximized or reaches an intermediate state, depending on the deleterious mutation rate relative to the extent of ploidally antagonistic selection. By contrast, evolution generally leads diploid fathers to mask mutations in their gametes to the maximum extent possible, whenever masking (e.g., through transcript sharing) increases the average fitness of a father's gametes. We discuss the implications of this maternal–paternal conflict over the extent of haploid selection and describe empirical studies needed to refine our understanding of haploid selection among seemingly diploid organisms.

pollen competition | antagonistic selection | haploid selection | sperm competition | evolutionary theory

Although the sexual cycle necessitates the existence of both a haploid (postmeiotic) and a diploid (postsyngamy) phase, it does not necessitate selection in both phases. In predominantly diploid organisms, parents can modify the extent of selection experienced at the haploid phase via a number of mechanisms. The goal of this study is to investigate, using mathematical models, how evolution is predicted to shape the "selective arena" within which haploids compete for fertilization, given that diploid mothers and fathers may modify this arena.

That diploid organisms have evolved to modify the extent of haploid selection is evidenced by the variety of mechanisms that effectively dampen or intensify selection among gametes and gametophytes. In mammalian egg development, cell divisions arrest early in life, and eggs remain dormant until ovulation; because meiosis II is triggered only when the egg is fertilized by a sperm, the haploid phase of selection is largely avoided by mammalian egg cells. Sperm in many animals also appear to circumvent selection in the haploid phase, but by an entirely different route. Diploid spermatagonia actively divide, producing spermatocytes that undergo meiosis throughout the lifespan of reproductively mature males. During spermatogenesis, however, protein and RNA products are shared among cells across cytoplasmic bridges ("syncytium") into the late postmeiotic stages, so that cells carry a nearly diploid complement of genes (1, 2). Furthermore, although haploid sperm actively swim to reach an egg, their genomes are compactly packaged, restricting haploid expression to a few genes (2). Among plants, angiosperms have evolved relatively little scope for selection among female gametophytes, as few haploid eggs are produced within an ovule. By contrast, gymnosperms have many more haploid eggs per ovule, increasing the opportunity for selection among female gametophytes (3).

Among male gametophytes, haploid pollen are thought to experience extensive selection, as pollen tubes compete for access to ovules (4–6). In turn, the strength of this selection can be modulated by the maternal plant by altering style length (7–9), delaying stigma receptivity (10, 11) and/or delaying pollen tube growth in the pistil (12). Paternal plants could also potentially alter the extent to which pollen are supplied with diploid transcripts, thereby modifying the extent of selection on the haploid genome. Although allele-specific expression data from single pollen grains are lacking, some pollen components are known to be effectively diploid (provisioned by the pollen parent). Particularly noteworthy are flavanoids involved in pollen tube growth (13) and a related enzyme, UDP-flavonoid-glucosyltransferase (14), as well as RNA polymerases required for transcription (15). Furthermore, the difference between sporophytic and gametophytic self-incompatibility systems stems from pollen being phenotypically diploid vs. haploid, respectively (16) (see also ref. 17 for evidence of diploid sporophytic effects on pollen–style incompatibilities between species of tomato).

That selection acts during the haploid phase of predominantly diploid organisms has been confirmed experimentally in a number of studies (18). One of the earliest experiments found that placing pollen farther from the ovaries on the stigma of the

Significance

Predominantly diploid organisms shape the extent to which their haploid gametes and gametophytes experience selection. Although animals are thought to experience only mild selection in the haploid stage, plants often experience strong haploid selection. When should parents limit exposure of gametes to haploid selection and when should they strengthen this selection? We develop mathematical models that consider the "selective arena" within which male gametes or gametophytes (sperm or pollen) compete for fertilization, examining how the intensity of this selective arena evolves when controlled by the mother or the father. These models predict substantial variation in the outcome, depending on whether mothers or fathers exert more control over the selective arena, with mothers often favoring stronger haploid selection than fathers.

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[1]To whom correspondence should be addressed. Email: otto@zoology.ubc.ca.

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carnation *Dianthus chinensis* resulted in faster germination and greater seed weight among the progeny (4). Other studies have documented that fathers differ in siring success when pollen from multiple donors are directly placed on the stigma, with faster pollen tube growth often increasing fertilization success (e.g., refs. 19–24). Furthermore, competition can be manipulated by varying the amount of pollen deposited, which significantly affects siring proportions in some species (e.g., *Cucurbita pepo*) but not others (9, 25). In dioecious *Rumex*, the amount of pollen deposited alters the relative success of X- and Y-bearing gametophytes and hence the resulting sex ratio (26, 27). A recent genomic analysis of fixed and polymorphic sites in *Arabidopsis thaliana* found evidence of both stronger purifying selection and more extensive positive selection among pollen-expressed genes than among randomly chosen genes (28). Indeed, genome-wide expression data indicate that a substantial fraction of genes are expressed in male gametophytes [70% in *A. thaliana* (29) and 67–71% in *Capsella grandiflora* (30)], providing broad genomic potential for selection during the haploid phase in plants. Furthermore, expression data indicate a high degree of overlap between genes expressed in the haploid and diploid phases. In *Arabidopsis*, for example, ∼90% of genes expressed in the gametophyte are also expressed in the sporophyte (29), and over 70 genes are known to be selectively important to both phases (31).

In this study, we explore models that allow the strength of haploid selection to evolve, with maternal or paternal genes modulating selection experienced by gametes. Because the scope for selection among male gametes is generally greater, we limit our attention to selective differences among pollen or sperm, although selection among ovules can be studied similarly. To be concise, we use botanical terms (e.g., pollen for the haploid phase), but the models apply to any organism with a dominant diploid phase where the diploid mother can exert control over the nature of selection in the haploid phase. We also loosely use "gamete" to refer to the haploid phase, including both gametophytes (pollen) and gametes (sperm).

We begin by exploring the evolution of maternal control over the selective arena faced by gametes. We then explore paternal control over the ratio of diploid vs. haploid gene products supplied to the gametes. Finally, we explore the coevolutionary outcome of male and female control over gametic selection. The models developed allow us to quantify these various evolutionary pressures and refine our understanding of when evolution is likely to lead to the expansion or cessation of haploid selection among predominantly diploid organisms.

Theoretical Background

Most previous models exploring the evolution of haploidy and diploidy assume an alternation of generations with free-living haploid and diploid phases, where expanding one phase reduces the other phase (see review by ref. 32). These models have shown that purging of deleterious mutations favors expansion of the haploid phase when recombination is infrequent, but that diploids are favored because they better mask mutations from selection when recombination is common (33, 34). With ploidally antagonistic selection, however, evolution favors the expansion of whichever ploidy phase gains the greatest fitness advantage, on average, from the conflicting selection pressures (35). By contrast, the models developed here assume that the organism is predominantly diploid and that the extent of selection in the diploid phase does not vary. Instead, it is only the strength of selection in the haploid phase that evolves, in response to genes carried by the diploid mother or father.

We recently considered the combined effects of deleterious mutation load and Fisherian sex ratio selection in dioecious plants with X and Y sex chromosomes (as in *Rumex*) (36). Sex ratio selection can be seen as a form of ploidally antagonistic selection in that haploid selection favoring one chromosome (say the X) causes a female-biased sex ratio that is counterselected among the diploid offspring. Considering only maternal control of selection, we found that a biased sex ratio could be stably maintained because of the advantages to the mother of purging deleterious mutations, even though this skews her offspring's sex ratio. Here, we provide a more general modeling framework within which we explore the evolution of the selective arena in which gametes compete, considering antagonistic selection between ploidy levels and/or sexes, deleterious mutations, and the interplay between maternal and paternal control over haploid selection.

Maternal Control of Haploid Selection

Model. We first consider a model where the diploid mother can evolve to alter the haploid selective arena. For example, in plants, mothers could modify the length, shape, or structure of the style or modify resource provisioning for growing pollen tubes to reduce or enhance fitness differences among pollen (8). Similarly, in animals, females might alter the reproductive tract or the amount of time that sperm are stored to accentuate or attenuate fitness differences among sperm.

Throughout, we focus on organisms with internal fertilization, where males compete for fertilization (polygamy) and females receive sufficient pollen to fertilize all eggs (no pollen limitation). That said, the model may also apply in some circumstances to external fertilization (e.g., if the timing of egg release influences the extent of selection among male gametes). Hermaphrodites are also described by the model, where processes within female tissues (maternal control) or male tissues (paternal control) impact the nature of selection faced by male gametes.

Specifically, we track a modifier gene (**M** with alleles M and m) that alters the strength of selection among haploid pollen at a selected locus (**A** with alleles A and a), with recombination at rate r between the two genes. Fitnesses of male gametes and diploid individuals of sex k are given in Table 1 (distinguishing selection against deleterious mutations from antagonistic selection, using roman letters and Greek letters, respectively). Without loss of generality, we simplify the presentation by assuming that the a allele is the one that is selected against in the haploid phase ($t, \tau > 0$). The term c_{ij} modulates the strength of selection in the haploid phase, depending on the mother's genotype (ij) at the modifier locus. When $c_{ij} = 0$, selection is absent in the haploid phase. When $c_{ij} = 1$, selection is maximized; that is, we assume a fixed point at which female fitness would be reduced significantly by increasing haploid selection further, either because of abnormal floral development or because pollen becomes limiting.

We focus on two main scenarios that maintain polymorphism at locus **A**: (i) mutation–selection balance (MS), where a is unconditionally deleterious ($t, s^k > 0$) but recurs due to mutation at rate μ (back mutations have a negligible effect and are ignored), and (ii) antagonistic selection (AS) with opposing and balanced selection pressures among ploidy levels ($\tau > 0$; $\sigma^k < 0$) and/or among the sexes ($\sigma^{♂}\sigma^{♀} < 0$). Both forms of selection were first observed to maintain polymorphism by Haldane (37, 38). The frequency of a at these polymorphic equilibria is denoted by $\hat{q}_{\mathrm{MS}}$ or

Table 1. Fitness regime

Genotype	Fitness*
Haploid	
A	$v_A = 1$
a	$v_a = 1 - c_{ij}t$
Diploid	($k = ♀$ or $♂$)
AA	$W^k_{AA} = 1$
Aa	$W^k_{Aa} = 1 - h^k s^k$
aa	$W^k_{aa} = 1 - s^k$

*Greek letters τ, σ, and η are used in place of t, s, and h for antagonistic selection.

$\hat{q}_{AS}$, respectively (we use $\hat{q}$ to refer to either one, depending on the form of selection being considered). The model recursions are given in Eq. **S2**.

We then consider the fate of a new allele m that modifies the extent of haploid selection. Allele m spreads if its long-term relative fitness is greater than that of the resident allele, M, that is, if the leading eigenvalue, λ, of the local stability matrix is greater than one. We calculate λ assuming weak selection and loose linkage (r large relative to selection), keeping only leading-order terms [see *Mathematica* package deposited at Dryad (doi:10.5061/dryad.4gn47)].

In all of the models of maternal control considered, the leading eigenvalue is

$$\lambda \approx 1 + \delta q\ \hat{q}(1-\hat{q})\ \delta W. \qquad [1]$$

The term $\delta q\ \hat{q}(1-\hat{q})$ represents the impact that mothers have on the allele frequency of pollen used to fertilize ovules, comparing *Mm* mothers to resident *MM* mothers, where δq is the effective strength of gametic selection against a alleles, which is multiplied by the probability that the a allele is found in the pollen pool ($\hat{q}$) and replaced by the A allele ($1-\hat{q}$). The term δW is the impact of this gametic selection on offspring fitness, calculated as the average fitness effect of replacing an a allele with an A in daughters and in sons, including the impact on the pollen success of sons. With weak selection, subsequent effects on future generations, as well as terms involving the recombination rate, enter only as lower-order terms. We next evaluate Eq. **1** under different forms of selection.

Case 1: Mutation–Selection Balance. Unconditionally deleterious alleles are expected to favor expansion of selection in the haploid phase, as mothers with stronger selective sieves will fertilize ovules with fitter pollen (6, 18). Indeed, assuming very weak mutation rates, we find that all of the terms in Eq. **1** are positive when *Mm* mothers select more strongly among their pollen ($c_{Mm} > c_{MM}$). The effect of the modifier on selection against a in the pollen pool is $\delta q = (c_{Mm} - c_{MM})t$, the equilibrium equals

$$\hat{q}_{MS} \approx \frac{\mu}{(1/2)(h^{\varphi}s^{\varphi} + h^{\sigma}s^{\sigma} + c_{MM}t)}, \qquad [2]$$

and the effect of replacing an a pollen with an A pollen on the mean offspring fitness is

$$\delta W = \frac{1}{2}h^{\varphi}s^{\varphi} + \frac{1}{2}h^{\sigma}s^{\sigma} + \frac{1}{4}c_{MM}t. \qquad [3]$$

The last term in Eq. **3** represents the effect on sons' pollen success and is multiplied by 1/4 because only half the time is the offspring a son and only half of the time would he transmit the allele inherited from his father's pollen. Plugging these terms into Eq. **1**, using $\hat{q}_{MS}(1-\hat{q}_{MS}) \approx \hat{q}_{MS}$, yields equation 10 in ref. 36.

Case 2: Antagonistic Selection. A polymorphic equilibrium can also be maintained by opposing selection pressures in different life phases (ploidally antagonistic selection) or in different sexes (sexually antagonistic selection) (39). In a population fixed for the M allele, the a allele equilibrates at a frequency

$$\hat{q}_{AS} \approx \frac{-\eta^{\varphi}\sigma^{\varphi} - \eta^{\sigma}\sigma^{\sigma} - c_{MM}\tau}{-\sigma^{\varphi}(2\eta^{\varphi}-1) - \sigma^{\sigma}(2\eta^{\sigma}-1)}, \qquad [4]$$

as long as

$$-(1-\eta^{\varphi})\sigma^{\varphi} - (1-\eta^{\sigma})\sigma^{\sigma} < c_{MM}\tau < -\eta^{\varphi}\sigma^{\varphi} - \eta^{\sigma}\sigma^{\sigma}. \qquad [5]$$

At an equilibrium with antagonistic selection, modifiers that cause mothers to reduce expression in the haploid phase ($c_{Mm} < c_{MM}$) are always favored. Again, $\delta q = (c_{Mm} - c_{MM})\tau$, but now the impact of stronger gametic selection on the offspring is negative and can be simplified to $\delta W = -c_{MM}\tau/4$ (Eq. **S16**). Intuitively, mothers are expected to reduce the strength of haploid selection if the main effect of haploid selection is to promote alleles that harm their diploid offspring.

Net Selection on Modifier. We now consider how the above forces would scale up from one selected locus to the entire genome. Assuming that all selected loci act independently on the modifier (i.e., loci are loosely linked and nonepistatic), we can approximate the net strength of selection acting on a modifier as the sum of indirect selective forces arising from each locus ($S_{net} = \lambda_{net} - 1 = \Sigma(\lambda_l - 1)$ where the sum is over all loci, including L_{MS} loci subject to purifying selection and L_{AS} loci subject to antagonistic selection). For simplicity, we assume constant selection coefficients and mutation rates across loci subject to a particular form of selection, but more precise results could be obtained by summing over the distributions of effects, if known.

The net selection on a modifier is then

$$S_{net} = (c_{Mm} - c_{MM})\left\{t\,\Psi\,\mu\,L_{MS} - \frac{c_{MM}\tau^2\hat{q}_{AS}(1-\hat{q}_{AS})}{4}\,L_{AS}\right\} \qquad [6]$$

with

$$\Psi = \frac{h^{\varphi}s^{\varphi} + h^{\sigma}s^{\sigma} + \frac{1}{2}c_{MM}t}{h^{\varphi}s^{\varphi} + h^{\sigma}s^{\sigma} + c_{MM}t}.$$

Note that genes selected only in the pollen phase favor strengthening haploid selection (contributing solely to the L_{MS} term with $s^{\varphi} = s^{\sigma} = 0$), because of the increased pollen fitness experienced by sons.

For sufficiently high deleterious mutation rates across the genome, the term in braces in Eq. **6** is positive for $c_{MM} \approx 1$, so selection will maintain organisms at this maximum level of haploid selection. Defining $\Upsilon = \mu L_{MS}/L_{AS}$ as the genome-wide deleterious mutation rate per antagonistic locus, maximal haploid selection is maintained whenever $\Upsilon > \Upsilon^*$, where

$$\Upsilon^* = \left(\frac{\tau^2\,\hat{q}_{AS}\,(1-\hat{q}_{AS})}{4\,t\,\Psi}\right)_{c_{MM}=1}. \qquad [7]$$

Conversely, for sufficiently low deleterious mutation rates, one might expect antagonistic selection to drive c_{ij} to zero, representing the complete suppression of haploid selection. This does not occur, however, when selection is ploidally antagonistic. As haploid selection weakens, the allele favored in diploids rises in frequency and eventually fixes. Specifically, c_{ij} evolves toward a minimum c_{min}, where $c_{min}\tau$ reaches the bottom of the interval in Eq. **5**, after which polymorphism is lost. At this point, antagonistic selection is resolved in favor of the diploid-beneficial allele and no longer exerts selection on the modifier ($\lambda = 1$ when $\hat{q}_{AS} = 1$). That said, as the extent of haploid selection (c_{MM}) declines, different sets of loci might exhibit variation, with polymorphism switching to loci experiencing stronger haploid selection. Alternatively, with sexually antagonistic selection, polymorphism can be maintained even with the complete loss of haploid selection (as long as the interval in Eq. **5** spans zero). Under these circumstances, evolution can lead to the complete cessation of haploid selection.

The evolutionarily stable strategy (ESS) for the level of haploid selection, $c*$ occurs when $S_{net} = 0$. Because $\hat{q}_{AS}$ is a function of c_{MM}, Eq. **6** is a quartic function of c_{MM} whose solutions represent potential evolutionarily stable strategies. These solutions are plotted in Fig. 1 for various values of the deleterious mutation rate per antagonistic locus (Υ) and the strength of antagonistic

selection in haploids (τ). According to Eq. **6**, if we multiply all of the selection coefficients ($s^{\varphi}, s^{\sigma}, t, \sigma^{\varphi}, \sigma^{\sigma}, \tau$) by a constant factor, the ESS value $c*$ would remain the same as long as Υ was also multiplied by the same factor. Thus, for a given Υ, mutation–selection balance becomes the more dominant force with weaker selection, favoring the evolution of more extensive gametic selection, whereas antagonistic selection dominates with stronger selection.

Sex Linkage. So far, we have considered only autosomal loci. Sex chromosomes are thought to accumulate sexually antagonistic loci (40) and have been found empirically to exhibit disproportionately high levels of sexually antagonistic genetic variation (41, 42). In *SI Text* we extend the above treatment to sex linkage. The evolution of the haploid selective arena is then driven, in the first place, to equalize the birth sex ratio, *BSR*. For example, if Y-bearing gametes have low fitness, a modifier that increases the strength of haploid selection will increase the proportion of female offspring and will thus spread when the resident sex ratio is male biased ($BSR > 1/2$). Thus, heightened or weakened gametic selection may be favored, in a manner that brings the birth sex ratio closer to 50:50.

With a nearly even sex ratio ($BSR \approx 1/2$), mothers would again evolve to alter gametic selection according to Eq. **1** (see *SI Text* for δq and δW). Although a sex-linked antagonistic locus tends to select for reduced gametic selection, as was found for an autosomal locus, increased gametic selection can be favored when selection is sexually antagonistic and the haploid-beneficial allele is also female beneficial (*SI Text*). In such cases, we would expect maximal gametic selection, regardless of the deleterious mutation rate.

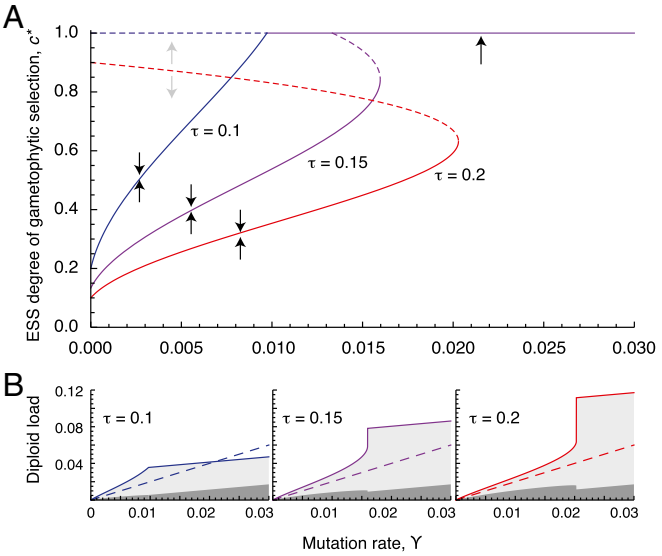

Fig. 1. Evolution of the selective arena experienced by haploids. (*A*) When the degree of gametic selection is maternally controlled, evolution drives the level of selection in the haploid phase toward the ESS, $c*$, given by the solid curves (dashed curves are repelling). As the deleterious mutation rate per antagonistic locus (Υ) rises along the x axis, selection favors higher levels of gametic selection among mothers. On the other hand, as ploidally antagonistic selection becomes stronger in the haploid phase (larger τ), mothers evolve to reduce the degree of gametic selection. (*B*) The resulting effects on the fitness load experienced by diploid offspring at the ESS. Diploid mean fitness is reduced both by recurrent mutations ("mutation load" in dark gray) and by ploidally antagonistic selection (light gray) as mutation rate Υ increases. Because gametic selection eliminates deleterious mutations, the mutation load (dark gray) is much lower than it would be in the absence of gametic selection (dashed line). Nevertheless, the total diploid load (ploidally antagonistic and mutation load) is often higher at the ESS, because the degree of gametic selection is also shaped by fitness variation in haploids. Other parameters: $t = 0.1$, $s^k = 0.2$, $h^k = 0.1$, $\sigma^k = -0.1$, $\eta^k = 0.9$, for both sexes.

Paternal Control of Haploid Selection

We now consider a model where fathers can modify the phenotype of their gametes and hence control the degree of haploid selection. In animals, such control can occur during gametogenesis, depending on the extent of sharing of RNAs, proteins, and cellular signals across the syncytium connecting haploid sperm. In plants, paternal control can be exerted by supplying the pollen grain with more diploid gene products from the microsporophyte. We continue to assume that selection occurs after the gametes are released from the father.

Specifically, we allow the provisioning of diploid gene products to be adjusted, depending on the father's genotype (ij) at a modifier locus (**M**), with gametes exhibiting a proportion p_{ij} of gene products reflecting their own haploid genome and $1-p_{ij}$ reflecting the diploid genome of the father. For homozygous AA (or aa) fathers, we assume that such provisioning has no effect on the gamete, as all transcripts involve the A (or a) allele. Fathers that are Aa, however, produce gametes whose fitnesses are functions of the provisioning: $g_A(p_{ij})$ or $g_a(p_{ij})$, depending on whether the gamete carries allele A or a, respectively. These functions can take any form, although we assume that allele a is selected against in haploids ($g_A(p_{ij}) \geq g_a(p_{ij})$) and that complete provisioning causes all gametes of Aa fathers to have equal fitness ($g_A(0) = g_a(0)$).

We then track the spread of a new modifier allele m introduced into a population at either (i) mutation–selection balance or (ii) an equilibrium under antagonistic selection (see *SI Text* for recursions and equilibrium frequencies). In either case, the new modifier allele spreads at rate

$$\lambda = 1 + (\bar{g}_{Mm} - \bar{g}_{MM})\,\hat{q}(1-\hat{q}), \qquad [8]$$

where $\bar{g}_{ij} = (g_A(p_{ij}) + g_a(p_{ij}))/2$ is the average fitness of gametes produced by an Aa father with modifier genotype ij. Thus, assuming weak selection, fathers evolve solely to increase the fertilization success of their gametes, regardless of the subsequent impacts on offspring fitness. Again, the net effect of selection summed over all loci that are expressed in the haploid stage would be $S_{\text{net}} = \lambda_{\text{net}} - 1 = \Sigma(\lambda_l - 1)$.

As a result, modifiers that reduce the degree of haploid expression ($p_{Mm} < p_{MM}$) spread whenever mixing of gene products increases the average fitness of gametes, e.g., by protecting them from poorly functioning alleles. Essentially, when $\bar{g}_{Mm} > \bar{g}_{MM}$, diploid fathers that better mask their own gametes have higher siring success. This condition requires that the fitter allele is partially dominant to the less fit allele with respect to gamete fitness when both gene products are present within the gamete. Although this need not always be true, empirical evidence suggests that such dominance is common for diploids (43), and we assume this pattern carries over to gene mixing in haploids, for loci subject to both mutation–selection balance and antagonistic selection (see *Mathematica* package for explorations of other cases).

Coevolution of Maternal and Paternal Control

Whereas mothers typically evolve to ensure some degree of selection among pollen (Fig. 1), fathers evolve to minimize this selection. Selection on the fathers to mask their gametes is, however, stronger than that acting on mothers, the former being proportional to the strength of selection (Eq. **8**) and the latter to the square of selection (assuming no sex linkage or an unbiased sex ratio). This asymmetry occurs because the evolution of maternal control requires that mothers change the efficacy of selection among pollen and that this change has subsequent fitness consequences to their diploid offspring (both δq and δW in Eq. **1** being proportional to selection). As a result, our analysis predicts an overwhelming paternal drive to provision gametes with

diploid gene products to the maximum degree possible (i.e., the minimum amount of haploid expression, $p_{\min}$). This is particularly true if there are few genes expressed in the haploid stage. In this case, selection summed across the genome, S_{net}, may be sufficient to dominate drift with paternal control but not with maternal control (i.e., if $4NS_{\text{net}}$ falls substantially below one for maternal but not paternal control, for a given population size N).

Assuming haploid selection is substantial, however, paternal provisioning is limited, both because it ends before females receive gametes and because homozygous fathers cannot mask their alleles. Hence females may continue to evolve control over the selective arena even in the face of provisioning.

We then explored how mothers subsequently evolve when fathers provision gametes with diploid gene products. We approached this question in two ways. In the first approach, we asked how mothers might further adjust the strength of selection among the gametes received (adjusting c_{ij}). In this case, paternal provisioning was held constant at $p_{\min}$ and the fitnesses of gametes were adjusted for pollen derived from *Aa* males. In all other respects, the model is identical to that considered before with maternally controlled selection. The results are summarized in *SI Text*. In response to paternal provisioning, females evolve to an even higher ESS level of gametic selection c^* for a given genome-wide mutation rate per antagonistic locus Υ (Fig. 2*A*).

In the second approach, we asked how mothers might alter the selective arena in such a way that paternally derived gene products are depleted before ovule fertilization (altering $p_{\min}$). In this case, the relative fitness of gametes from homozygous *AA* or *aa* fathers is not affected by the mother, only the fitness of gametes produced by *Aa* fathers (*SI Text*). In this case, mothers evolve to deplete paternal provisions to the maximum extent possible ($c^* = 1$, causing pollen to fully express their haploid genome) when the deleterious mutation rate is sufficiently high per antagonistic locus ($\Upsilon > 0.013$ in Fig. 2*B*). Below this mutation rate, females evolve to an ESS that allows paternal provisioning (lower c^*). In this case, females also benefit from some paternal provisioning of diploid gene products because this reduces the fitness costs to offspring of ploidally antagonistic selection.

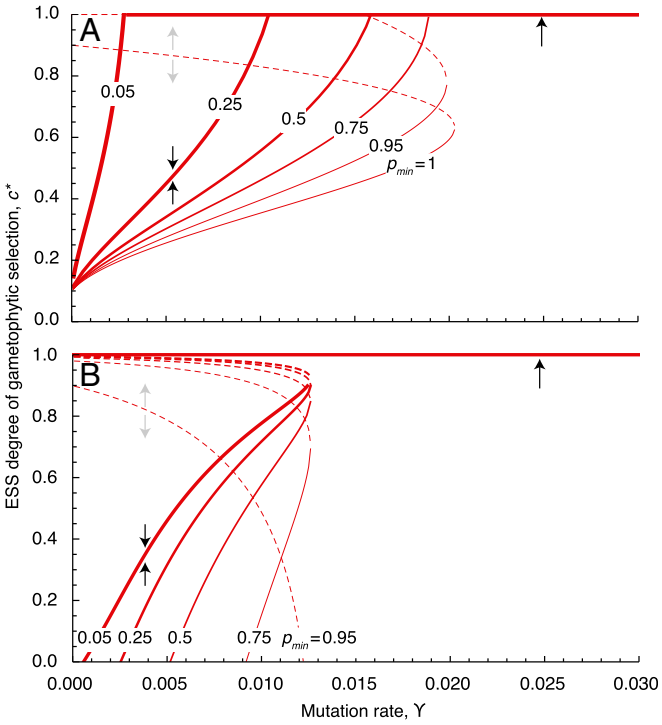

Fig. 2. Evolution of maternal control when fathers provision. Paternal control selects for reduced haploid expression to the minimum level possible ($p_{\min}$). (*A*) When fathers provide a lower proportion of haploid gene products (lower $p_{\min}$), mothers respond by evolving higher levels of gametic selection, with the ESS level of gametic selection shown by solid curves. (No paternal provisioning, $p_{\min} = 1$, corresponds to the red curve in Fig. 1*A*.) (*B*) Alternatively, when mothers can manipulate the impact of paternal diploid transcripts on fertilization (e.g., by delaying fertilization), mothers evolve to maximize haploid expression when the mutation rate is sufficiently high (ESS $c^* = 1$, solid curves), but not when Υ is low. The fitness of gamete type *I* from *Aa* fathers was set to $g_I(p_{\min}, c_{ij}) = 1 - c_{ij}t\,(1-(1-\iota_I^x)^{1/x})$ in *A* and to $g_I(p_{\min}, c_{ij}) = 1 - t\,(1-(1-\iota_I^x)^{1/x})$ in *B*, where ι_I measures the extent to which a gamete carrying allele *I* from a heterozygous father has a fitness similar to that of an *a* gamete from an *aa* father ($\iota_I = 1$) vs. an *A* gamete from an *AA* father ($\iota_I = 0$). Functions were chosen to correspond to a dominance coefficient of 0.1 in gametes with an equal abundance of *A* and *a* gene products: $x = 2.25$, $\iota_a = 1 - \iota_A$, and $\iota_A = (1 - p_{\min})/2$ (*A*) or $\iota_A = (1 - c_{ij})(1 - p_{\min})/2$ (*B*). Other parameters: $t = 0.1$, $s^k = 0.2$, $h^k = 0.1$, $\tau = 0.2$, $\sigma^k = -0.1$, $\eta^k = 0.9$, for both sexes.

Discussion

Despite representing a small fraction of the life cycle of multicellular animals and plants, the haploid phase can have a disproportionately large impact on selection because alleles are directly exposed to selection and because gametes are often in abundance and compete strongly for fertilization.

Nevertheless, organisms vary greatly in the extent to which haploid gametes are exposed to or protected from selection. Here we have explored theoretical models to provide insights into the evolution of the selective arena faced by haploid gametes in predominantly diploid organisms.

It has long been recognized that genes that intensify haploid selection are favored because this purges deleterious mutations from the gametes used for fertilization (6, 18). When haploid selection is under maternal control, we confirm that this mechanism favors the spread of genes that strengthen haploid selection. On the other hand, if the very mutations that are favored in haploids are selected against in diploids, then this purging among haploids will decrease the fitness of their diploid offspring. This "ploidally antagonistic selection" (39) places a brake on the expansion of selection in the haploid phase. Our analysis predicts the balance between purifying and ploidally antagonistic selection when the extent of haploid selection is under maternal control (Fig. 1).

Among fathers, the evolutionary pressures are entirely different. Fathers evolve to mask mutations carried by their haploid gametes (e.g., by provisioning with diploid gene products), thereby making their sperm more competitive. This is true for both deleterious mutations and ploidally antagonistic alleles, as long as transcript mixing raises the average fitness of their gametes (as expected when less fit alleles are at least partially recessive). Along these lines, ref. 6 suggested that males might evolve to mask selfish genetic elements that reduce fertility.

Whereas fathers are predicted to reduce exposure of their gametes to haploid selection, maternal control acts after fertilization, allowing counterselection by females. Indeed, we find that females continue to evolve high levels of gametic selection when deleterious mutations are common (Fig. 2).

The models developed allow us to quantify these various evolutionary pressures and refine our understanding of when evolution is likely to lead to the expansion or cessation of haploid selection among predominantly diploid organisms.

Conventional wisdom suggests two extremes: haploid selection being extensive among plant pollen and largely absent among animal sperm. To some extent, this pattern might reflect differences in the scope for ploidally antagonistic selection. The pollen tubes of plants must undergo cell growth to migrate through the style (6), requiring basic metabolic functions that may be beneficial in both ploidy phases. By contrast, in animals, sperm are selected primarily to be actively mobile, thus favoring alleles that may have antagonistic effects on the diploid phase. Our analysis also indicates that evolutionary forces acting maternally

to expand the scope of selection will be overwhelmed by drift before those acting paternally, suggesting that, once haploid selection becomes sufficiently weakened, it may be difficult to reevolve.

Nevertheless, these broad strokes likely mask substantial variation within groups in the strength of gametic selection. In animals, evidence is accumulating of selection based on the haploid genotype of sperm at a number of loci [sperm adhesion molecule *Spam1* (44); segregation distortion responder *Rsp* (45); the *t*-locus responder *Tcr* (46); and likely *Catsper1*, a voltage-gated calcium channel located in the plasma membrane of the sperm tail (47)]. In addition, preliminary evidence in Atlantic salmon (*Salmo salar*) suggests that variation in sperm phenotype at the intraejaculate level impacts performance in the resulting offspring (48). The efficacy of transcript sharing across the syncytium during postmeiotic sperm maturation has been measured only in a few species, and the extent of haploid expression among sperm deserves further empirical work in a broader array of species.

Given that mothers may evolve to strengthen gametic selection by delaying fertilization, it would be particularly valuable to study haploid expression and selection in animals whose sperm must remain active for extended periods (e.g., with long-term sperm storage or broadcast spawning).

Although we have assumed polygamy, we would expect that the evolutionary pressures acting upon fathers and mothers would become more closely aligned with inbreeding and/or monogamy, because fathers that are guaranteed mating success will then be selected, like mothers, to increase the fertilization success of gametes that improve offspring fitness. Comparisons of paternal gametic provisioning would thus be valuable, contrasting the degree of haploid gene expression in related species with different mating systems.

We also predict that species that experience pollen limitation should exhibit weaker gametic selection. Although we did not explicitly model pollen limitation, the maximum value that c_{ij} can reach before negatively impacting female fertility is lower in species that regularly experience pollen limitation. Again, comparative analyses that test these predictions promise to shed light on the role of haploid selection among predominantly diploid organisms.

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Supporting Information

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SI Text

A *Mathematica* file detailing the analyses performed in this paper has been deposited in the Dryad database, datadryad.org (doi:10.5061/dryad.4gn47).

Recursions with Maternal Control of Haploid Selection. To develop the recursions, we start by censusing the population at the haploid phase, immediately after meiosis. The life cycle then proceeds through receipt of gametes by a female, selection among gametes at a strength regulated by the female, fertilization, and selection among the resulting diploid individuals, followed by meiosis, at which point mutation at rate μ and recombination at rate r occur. Let the frequency of haploid genotype i in individuals of sex k be x_i^k ($i = 1, 2, 3, 4$ for *MA*, *Ma*, *mA*, and *ma*). Because the degree of pollen competition and hence the pollen available to fertilize ovules depends on the mother's genotype at the **M** locus, we keep track of ovules according to whether they are located within a homozygous (*MM* or *mm*) or a heterozygous mother (*Mm*). Thus, $x_{1,\mathrm{hom}}^{\female}$ represents an *MA* ovule carried by a homozygous mother, who must have been *MM*. Similarly, $x_{3,\mathrm{het}}^{\female}$ represents an *mA* ovule carried by a heterozygous mother (*Mm*).

We assume that females receive pollen from multiple individuals, in proportion to their frequency in the total pollen pool, and that all ovules are fertilized (no pollen limitation). Hence, we normalize by the pollen pool each female is expected to receive, accounting for selection among the pollen grains. The mean fitness among pollen received by a mother whose diploid genotype is ij at the **M** locus is then

$$\bar{v}_{ij} = x_1^{\male} + x_3^{\male} + (1 - c_{ij}t)\left(x_2^{\male} + x_4^{\male}\right). \qquad \textbf{[S1]}$$

This assumes that c_{ij} influences the success of *A*-bearing vs. *a*-bearing pollen but pays no attention to whether the pollen is X or Y bearing (for sex-linked antagonistic selection, see *Maternal Control with Sex Linkage*).

After fertilization of ovules with successful pollen, the resulting diploid offspring undergo selection followed by gamete production with recombination (mutation is handled in the next step). These gametes are labeled according to whether they were produced by heterozygotes or homozygotes at the **M** locus. The following recursions apply for gametes produced by either diploid sons ($k = \male$) or diploid daughters ($k = \female$). The modifier, however, does not act in diploid sons, so we can collapse together $x_{i,\mathrm{hom}}^{\male}$ and $x_{i,\mathrm{het}}^{\male}$, the sum of which gives the male gamete frequencies, $x_i^{\male}$:

$$\begin{aligned}
x_{1,\mathrm{hom}}^{k'} &= \frac{W_{AA}^k}{\bar{W}^k}\left(\frac{x_1^{\male}}{\bar{v}_{MM}}x_{1,\mathrm{hom}}^{\female} + \frac{x_1^{\male}}{\bar{v}_{Mm}}x_{1,\mathrm{het}}^{\female}\right) \\
&\quad + \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left(\frac{x_1^{\male}}{\bar{v}_{MM}}x_{2,\mathrm{hom}}^{\female} + \frac{x_1^{\male}}{\bar{v}_{Mm}}x_{2,\mathrm{het}}^{\female} + \frac{x_2^{\male}(1 - c_{MM}t)}{\bar{v}_{MM}}x_{1,\mathrm{hom}}^{\female}\right. \\
&\quad \left. + \frac{x_2^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{1,\mathrm{het}}^{\female}\right) \\
x_{1,\mathrm{het}}^{k'} &= \frac{1}{2}\frac{W_{AA}^k}{\bar{W}^k}\left(\frac{x_1^{\male}}{\bar{v}_{mm}}x_{3,\mathrm{hom}}^{\female} + \frac{x_1^{\male}}{\bar{v}_{Mm}}x_{3,\mathrm{het}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{MM}}x_{1,\mathrm{hom}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{Mm}}x_{1,\mathrm{het}}^{\female}\right) \\
&\quad + \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left((1 - r)\,\chi_A + r\,\chi_B\right)
\end{aligned}$$

$$\begin{aligned}
x_{2,\mathrm{hom}}^{k'} &= \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left(\frac{x_1^{\male}}{\bar{v}_{MM}}x_{2,\mathrm{hom}}^{\female} + \frac{x_1^{\male}}{\bar{v}_{Mm}}x_{2,\mathrm{het}}^{\female} + \frac{x_2^{\male}(1 - c_{MM}t)}{\bar{v}_{MM}}x_{1,\mathrm{hom}}^{\female}\right. \\
&\quad \left. + \frac{x_2^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{1,\mathrm{het}}^{\female}\right) \\
&\quad + \frac{W_{aa}^k}{\bar{W}^k}\left(\frac{x_2^{\male}(1 - c_{MM}t)}{\bar{v}_{MM}}x_{2,\mathrm{hom}}^{\female} + \frac{x_2^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{2,\mathrm{het}}^{\female}\right) \\
x_{2,\mathrm{het}}^{k'} &= \frac{1}{2}\frac{W_{aa}^k}{\bar{W}^k}\left(\frac{x_2^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{4,\mathrm{het}}^{\female} + \frac{x_2^{\male}(1 - c_{mm}t)}{\bar{v}_{mm}}x_{4,\mathrm{hom}}^{\female}\right. \\
&\quad \left. + \frac{x_4^{\male}(1 - c_{MM}t)}{\bar{v}_{MM}}x_{2,\mathrm{hom}}^{\female} + \frac{x_4^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{2,\mathrm{het}}^{\female}\right) \\
&\quad + \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left(r\,\chi_A + (1 - r)\,\chi_B\right) \\
x_{3,\mathrm{hom}}^{k'} &= \frac{W_{AA}^k}{\bar{W}^k}\left(\frac{x_3^{\male}}{\bar{v}_{mm}}x_{3,\mathrm{hom}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{Mm}}x_{3,\mathrm{het}}^{\female}\right) \\
&\quad + \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left(\frac{x_3^{\male}}{\bar{v}_{mm}}x_{4,\mathrm{hom}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{Mm}}x_{4,\mathrm{het}}^{\female} + \frac{x_4^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{3,\mathrm{het}}^{\female}\right. \\
&\quad \left. + \frac{x_4^{\male}(1 - c_{mm}t)}{\bar{v}_{mm}}x_{3,\mathrm{hom}}^{\female}\right) \\
x_{3,\mathrm{het}}^{k'} &= \frac{1}{2}\frac{W_{AA}^k}{\bar{W}^k}\left(\frac{x_1^{\male}}{\bar{v}_{mm}}x_{3,\mathrm{hom}}^{\female} + \frac{x_1^{\male}}{\bar{v}_{Mm}}x_{3,\mathrm{het}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{MM}}x_{1,\mathrm{hom}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{Mm}}x_{1,\mathrm{het}}^{\female}\right) \\
&\quad + \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left(r\,\chi_A + (1 - r)\,\chi_B\right) \\
x_{4,\mathrm{hom}}^{k'} &= \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left(\frac{x_3^{\male}}{\bar{v}_{mm}}x_{4,\mathrm{hom}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{Mm}}x_{4,\mathrm{het}}^{\female} + \frac{x_4^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{3,\mathrm{het}}^{\female}\right. \\
&\quad \left. + \frac{x_4^{\male}(1 - c_{mm}t)}{\bar{v}_{mm}}x_{3,\mathrm{hom}}^{\female}\right) \\
&\quad + \frac{W_{aa}^k}{\bar{W}^k}\left(\frac{x_4^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{4,\mathrm{het}}^{\female} + \frac{x_4^{\male}(1 - c_{mm}t)}{\bar{v}_{mm}}x_{4,\mathrm{hom}}^{\female}\right) \\
x_{4,\mathrm{het}}^{k'} &= \frac{1}{2}\frac{W_{aa}^k}{\bar{W}^k}\left(\frac{x_2^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{4,\mathrm{het}}^{\female} + \frac{x_2^{\male}(1 - c_{mm}t)}{\bar{v}_{mm}}x_{4,\mathrm{hom}}^{\female}\right. \\
&\quad \left. + \frac{x_4^{\male}(1 - c_{MM}t)}{\bar{v}_{MM}}x_{2,\mathrm{hom}}^{\female} + \frac{x_4^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{2,\mathrm{het}}^{\female}\right) \\
&\quad + \frac{1}{2}\frac{W_{Aa}^k}{\bar{W}^k}\left((1 - r)\,\chi_A + r\,\chi_B\right).
\end{aligned} \qquad \textbf{[S2]}$$

Here, $\bar{W}^k$ represents the mean fitness among diploids of sex k, and χ_A and χ_B describe the crosses affected by recombination:

$$\chi_A = \frac{x_1^{\male}}{\bar{v}_{mm}}x_{4,\mathrm{hom}}^{\female} + \frac{x_1^{\male}}{\bar{v}_{Mm}}x_{4,\mathrm{het}}^{\female} + \frac{x_4^{\male}(1 - c_{MM}t)}{\bar{v}_{MM}}x_{1,\mathrm{hom}}^{\female} + \frac{x_4^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{1,\mathrm{het}}^{\female}$$

$$\chi_B = \frac{x_2^{\male}(1 - c_{Mm}t)}{\bar{v}_{Mm}}x_{3,\mathrm{het}}^{\female} + \frac{x_2^{\male}(1 - c_{mm}t)}{\bar{v}_{mm}}x_{3,\mathrm{hom}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{MM}}x_{2,\mathrm{hom}}^{\female} + \frac{x_3^{\male}}{\bar{v}_{Mm}}x_{2,\mathrm{het}}^{\female}.$$

Finally, we account for mutation, such that the frequencies of haploid gametes at the next census equal

$$\begin{aligned} x''_{1,\mathrm{hom}} &= (1-\mu)\, x'_{1,\mathrm{hom}} \\ x''_{2,\mathrm{hom}} &= \mu\, x'_{1,\mathrm{hom}} + x'_{2,\mathrm{hom}} \\ x''_{3,\mathrm{hom}} &= (1-\mu)\, x'_{3,\mathrm{hom}} \\ x''_{4,\mathrm{hom}} &= \mu\, x'_{3,\mathrm{hom}} + x'_{4,\mathrm{hom}}. \end{aligned} \qquad \textbf{[S3]}$$

Eqs. **S3** apply for offspring that are homozygous at the **M** locus. Similar equations for gametes produced by offspring that are heterozygous at the **M** locus are obtained by replacing hom with het.

Using these recursion equations, we perform a perturbation analysis, assuming weak selection (order ϵ) and even weaker mutation rates per site (order ϵ^2). We first calculate the frequency of allele a at equilibrium (Eqs. **2** and **4**). We then determine the asymptotic fitness of allele m, while rare, within a resident population of M alleles (Eq. **1**), as given by the leading eigenvalue λ of the local stability matrix evaluated at an equilibrium. Recombination does not enter the eigenvalue, to leading order, unless linkage is assumed tight (r also of order ϵ). The *Mathematica* package provides a complete derivation (available at Dryad).

Maternal Control with Sex Linkage. Here we treat the case where the antagonistic locus **A** is completely linked to the sex-determining region. Fitnesses continue to be given by Table 1, with additional fitnesses of $1-\tau_Y$ for gametes bearing a Y chromosome and fitnesses of W_{X_AY} and W_{X_aY} for A- and a-bearing male diploids.

As shown by ref. 39, selection on a Y-linked locus does not maintain a polymorphism so we focus on an X-linked locus (**A** with alleles A and a). On the Y chromosomes, we assume that this locus is either fixed for a particular allele or absent altogether. We allow for the possibility that the ratio of X- to Y-bearing pollen may be biased due to segregation distortion, with the fraction of Y-bearing gametes being α. Again, c_{ij} represents the extent of selection during the haploid phase for a mother of genotype ij at the modifier locus **M**, which lies anywhere in the genome (recombining with **A** at rate r). The recursions and detailed derivations are presented in the *Mathematica* package.

With weak selection, the frequency of allele a is nearly equal on the X chromosome in males and females and given by

$$\hat{q}_X \approx \frac{-2\eta^{\varphi}\sigma^{\varphi} - \sigma^{\vartheta} - c_{MM}\tau}{2\sigma^{\varphi}(1-2\eta^{\varphi})}. \qquad \textbf{[S4]}$$

This equilibrium exists and is stable if the following condition is met,

$$-2\left(1-\eta^{\varphi}\right)\sigma^{\varphi} - \sigma^{\vartheta} < c_{MM}\tau < -2\eta^{\varphi}\sigma^{\varphi} - \sigma^{\vartheta}, \qquad \textbf{[S5]}$$

where again we assume that the a allele is selected against in haploids ($\tau > 0$).

Given that allele a has reached equilibrium frequency $\hat{q}_X$ (Eq. **S4**), the relative fitness of a rare mutant (m) with maternal control is given, to leading order, by

$$\lambda = 1 - ((c_{Mm} - c_{MM})(\hat{q}_X\,\tau - \tau_Y)/2)(BSR - 1/2), \qquad \textbf{[S6]}$$

where the birth sex ratio, BSR, is the fraction of offspring that are male and the term $(\hat{q}_X\,\tau - \tau_Y)$ represents the effect of gametic selection on the sex ratio (the average difference in gametic fitness between X-bearing and Y-bearing gametes).

Selection on the modifier primarily acts to equalize the sex ratio among offspring (Eq. **S6**), where the fraction of diploid offspring that are male is

$$BSR = \frac{\alpha(1-c_{MM}\tau_Y)}{\alpha(1-c_{MM}\tau_Y) + (1-\alpha)((1-\hat{q}_X) + \hat{q}_X(1-c_{MM}\tau))}. \qquad \textbf{[S7]}$$

For example, if Y-bearing gametes have low fitness ($\tau_Y > \hat{q}_X\,\tau$), a modifier that increases the strength of haploid selection ($c_{Mm} > c_{MM}$) will increase the proportion of female offspring and will thus spread when the resident sex ratio is male biased ($BSR > 1/2$). Thus, heightened or weakened gametic selection may be favored, in a manner that brings the birth sex ratio closer to 50:50. Eq. **S6** is equivalent to a weak selection approximation of equation 5 in ref. 36, where the latter assumed no polymorphism ($\hat{q}_X = 0$).

If we assume that the sex ratio at birth is near 1:1 ($BSR \approx 1/2$), then we can find the (lower-order) indirect fitness effects on the modifier coming from altering allele frequencies at locus **A** in offspring. The leading eigenvalue is then described by Eq. **1** with $\hat{q} = \hat{q}_X$. The term δq describing the effect of the modifier on gametic selection against a is now $(c_{Mm} - c_{MM})\tau/2$, where the $1/2$ enters because only half of the pollen bear the X chromosome with the A/a polymorphism. The impact on offspring fitness of exchanging an a-bearing pollen with an A-bearing one becomes

$$\delta W = \frac{1}{2}\Omega + \frac{1}{2}\omega\frac{2r}{3-r}. \qquad \textbf{[S8]}$$

The two terms in Eq. **S8** refer to the fitness impact of gametic selection on daughters; there is no effect in sons, who inherited the Y from the pollen, not the A or the a allele. The first term Ω is the difference in fitness of daughters born from A-bearing pollen rather than a-bearing pollen:

$$\Omega = \left((1-\hat{q}_X) + \hat{q}_X(1-\eta^{\varphi}\sigma^{\varphi})\right) - \left((1-\hat{q}_X)(1-\eta^{\varphi}\sigma^{\varphi}) + \hat{q}_X(1-\sigma^{\varphi})\right). \qquad \textbf{[S9]}$$

In addition, when these daughters produce gametes, recombination can associate the modifier (inherited from the ovule) with alleles inherited from pollen. This tends to pair modifiers that increase the strength of gametic selection with the haploid-beneficial allele A. The expected number of generations that the modifier is then associated with the A allele is given by the term $2r/(3-r)$ (*Mathematica* package). Consequently, the fitness difference between the diploid offspring of A-bearing and a-bearing ovules is given by

$$\omega = \frac{1}{2}\Omega + \frac{1}{2}(W_{X_AY} - W_{X_aY}), \qquad \textbf{[S10]}$$

where 1/2 of the offspring are female (first term) and 1/2 are male (second term).

If selection is ploidally antagonistic, but not sexually antagonistic, then both Ω and ω will be negative (recall that we have assumed that the a allele is selected against in haploids and so must be favored in diploids), favoring modifiers that reduce the strength of gametic selection. With sexually antagonistic selection, however, either increased or decreased gametic selection can be favored. Given that conditions [**S5**] must be satisfied for a polymorphism to be present, a necessary (but not sufficient) condition for increased gametic selection to be favored is that the haploid-beneficial allele is also female beneficial. Overall, the evolution of gametic selection is not expected to perturb the sex ratio substantially from 1:1 given only weak selection at one locus **A**, because selection to restore a 1:1 sex ratio (Eq. **S6**) is of larger order and dominates the dynamics when gametic fitnesses differ among A-bearing and a-bearing X chromosomes and Y chromosomes.

Recursions with Paternal Control of Haploid Selection. The recursions for paternal control follow the same life cycle as described

for maternal control with an autosomal modifier. The key difference is that the fitness of a male gamete depends on his father's genotype at both the **M** locus and **A** locus, rather than the mother's genotype. Specifically, if the father is homozygous ***AA***, his gametes are assumed to have fitness 1, whereas if he is *aa*, his gametes have fitness $1-\hat{c}\,t$, assuming that mothers limit selection among gametes by a fixed amount $\hat{c}$. Only if the father is heterozygous *Aa* does the modifier affect fitness, provisioning the gametes with the gene products from both *A* and *a* alleles, with the extent of mixing regulated by the father's modifier genotype *ij* and leading to gamete fitnesses of $g_A(p_{ij})$ and $g_a(p_{ij})$, respectively.

We must thus keep track of the frequency of male gametes from each type of father separately, with hom or het standing for homozygous or heterozygous fathers at the modifier **M** locus and HOM or HET standing for homozygous or heterozygous fathers at the trait **A** locus. Thus, $x^{\male}_{1,\mathrm{hom,HOM}}$ represents an *MA* gamete produced by a homozygous father at both loci, who must therefore have been *MMAA*. Similarly, $x^{\male}_{3,\mathrm{het,HET}}$ represents an *mA* gamete carried by a heterozygous father at both loci (*MmAa*). We assume that the modifier acts only in fathers and no longer keep track of the maternal origin of a gamete ($x^{\female}_{i,\mathrm{hom}}+x^{\female}_{i,\mathrm{het}}=x^{\female}_{i}$). We again assume that all ovules are fertilized, with males contributing pollen into a single pool such that pollen from all males compete against each other. Recursion equations are equivalent to those in the model for maternal control but keeping track of the paternal origin of the pollen instead of the maternal origin of the ovule and adjusting the fitnesses of male gametes. For example,

$$x^{k'}_{1,\mathrm{hom,HOM}}=\frac{W^k_{AA}}{\overline{W}^k}\,\frac{x^{\male}_{1,\mathrm{hom,HOM}}+x^{\male}_{1,\mathrm{hom,HET}}\,g_A(p_{MM})+x^{\male}_{1,\mathrm{het,HOM}}+x^{\male}_{1,\mathrm{het,HET}}\,g_A(p_{Mm})}{\bar{v}}x^{\female}_1$$
$$x^{k'}_{1,\mathrm{hom,HET}}=\frac{W^k_{Aa}}{\overline{W}^k}\left(\frac{x^{\male}_{1,\mathrm{hom,HOM}}+x^{\male}_{1,\mathrm{hom,HET}}\,g_A(p_{MM})+x^{\male}_{1,\mathrm{het,HOM}}+x^{\male}_{1,\mathrm{het,HET}}\,g_A(p_{Mm})}{\bar{v}}x^{\female}_2\right.$$
$$\left.+\frac{x^{\male}_{2,\mathrm{hom,HOM}}(1-\hat{c}\,t)+x^{\male}_{2,\mathrm{hom,HET}}\,g_a(p_{MM})+x^{\male}_{2,\mathrm{het,HOM}}(1-\hat{c}\,t)+x^{\male}_{2,\mathrm{het,HET}}\,g_a(p_{Mm})}{\bar{v}}\,x^{\female}_1\right), \qquad \textbf{[S11]}$$

where $\bar{v}$ is the average gamete fitness (equivalent for all females). The full recursions and analyses are given in the *Mathematica* package.

At loci subject to mutation–selection balance, the equilibrium frequency of allele *a* while *M* is fixed is

$$\hat{q}_{\mathrm{MS}}=\frac{\mu}{(1/2)(h^{\female}s^{\female}+h^{\male}s^{\male}+(1-g_a(p_{MM})))}. \qquad \textbf{[S12]}$$

With antagonistic selection [replacing $g_l(p_{ij})$ with $\gamma_l(p_{ij})$], the equilibrium frequency of allele *a* is instead

$$\hat{q}_{\mathrm{AS}}=\frac{-\eta^{\female}\sigma^{\female}-\eta^{\male}\sigma^{\male}+(\gamma_a(p_{MM})-1)}{-\sigma^{\female}(2\eta^{\female}-1)-\sigma^{\male}(2\eta^{\male}-1)+2(\overline{\gamma}_{MM}-G)}, \qquad \textbf{[S13]}$$

where $\overline{\gamma}_{MM}=(\gamma_A(p_{ij})+\gamma_a(p_{ij}))/2$ and G represents the average fitness of gametes coming from an *AA* and an *aa* father. In calculating gamete fitness, we assume that mothers may be controlling the extent of gametic selection to some degree, $\hat{c}$, but this does not depend on the genotype at the current modifier, so $G=1/2+(1-\hat{c}\tau)/2$.

Results with Maternal Control of Haploid Selection When Fathers Provision. When fathers produce gametes whose fitness reflects the gamete's haploid genome only to some degree ($p_{\min}$), females of genotype *ij* can still alter the selective arena experienced by gametes, modifying the strength of this selection by a factor c_{ij}. For pollen-bearing allele *l* from *Aa* fathers, gametic fitness then becomes $g_l(p_{\min},c_{ij})$. Selective differences among pollen grains remain, however, both because of the gametes produced by homozygous fathers (according to Table 1) and because the gametes produced by heterozygous fathers may differ in fitness, depending on the allele that they carry (if $p_{\min}>0$).

The recursions are similar to Eqs. **S3** but now account for the paternal origin of pollen (*Mathematica* package). The equilibria are again described by Eqs. **S12** and **S13**, recognizing that the functions $g_l()$ and $\gamma_l()$ are now evaluated in a population where fathers provision at rate $p_{\min}$ and *MM* mothers alter gametic selection by an amount $\hat{c}=c_{MM}$.

The spread of a modifier allele *m* that alters the extent of gametic selection continues to be described by Eq. **1**. For both mutation–selection balance ($\hat{q}=\hat{q}_{\mathrm{MS}}$) and antagonistic selection ($\hat{q}=\hat{q}_{\mathrm{AS}}$, replacing roman with Greek letters), the change in selection against gametes bearing allele *a* caused by the modifier is

$$\delta q=\bar{t}_{Mm}-\bar{t}_{MM}, \qquad \textbf{[S14]}$$

where $\bar{t}_{ij}$ measures the fitness difference between *A*-bearing pollen and *a*-bearing pollen (the two terms in braces):

$$\bar{t}_{ij}=\left\{(1-\hat{q})+\hat{q}\;g_A\left(p_{\min},c_{ij}\right)\right\}-\left\{(1-\hat{q})\;g_a\left(p_{\min},c_{ij}\right)+\hat{q}\;\left(1-c_{ij}t\right)\right\}. \qquad \textbf{[S15]}$$

The modifier's effect on gametic selection reduces to $\delta q=(c_{Mm}-c_{MM})\,t$ when there is no paternal provisioning ($g_A(p_{\min},c_{ij})=1$, $g_a(p_{\min},c_{ij})=(1-c_{ij}t)$). The impact on the offspring caused by replacing an *a*-bearing pollen with an *A*-bearing pollen allele is then

$$\delta W=\sum_k\frac{1}{2}\left\{\left((1-\hat{q})+\hat{q}(1-h^ks^k)\right)-\left((1-\hat{q})(1-h^ks^k)+\hat{q}(1-s^k)\right)\right\}$$
$$+\frac{1}{4}\,\bar{t}_{MM}+\frac{1}{4}\left((1-\hat{q})\;(1-g_A(p_{\min},c_{MM}))\right.$$
$$\left.+\hat{q}\;(g_a(p_{\min},c_{MM})-(1-c_{MM})t)\right). \qquad \textbf{[S16]}$$

The first line of Eq. **S16** measures the effect on the fitness of offspring themselves, summing over daughters ($k=\female$) and sons ($k=\male$). The remaining lines measure the effect on fitness via sons'

fertility, with the first term $\bar{\imath}_{MM}/4$ measuring the benefit to sons produced by a pollen grain that bears allele A rather than a and the last term measuring the fertility benefit to sons produced by an ovule whose allele will be better masked by paternal provisioning of gametes in the son, because the son's other allele (the one inherited from the pollen) is more likely to be A than a.

At mutation–selection balance, $\hat{q}_{\mathrm{MS}}$ is small and can be ignored in δW, which reduces to Eq. **3** in the absence of paternal provisioning (last term is zero). With antagonistic selection, δW can be rewritten using the equilibrium value for $\hat{q}_{\mathrm{AS}}$ as $\delta W = -\frac{1}{4}(\gamma_A(p_{\min}, c_{MM}) - \gamma_a(p_{\min}, c_{MM}))$, which reduces to $-\frac{1}{4}c_{MM}\tau$ in the absence of paternal provisioning. Derivations are given in the *Mathematica* package.

Results with Maternal Control of Paternal Provisioning. Alternatively, mothers may be able to alter the selective arena in such a way that paternal gene products are depleted, and gametes must transcribe their own genotype. Now let $g_l(p_{\min}, c_{ij})$ represent the fitness among ij mothers of pollen-bearing allele l from Aa fathers when mothers alter the extent to which the fitness of pollen reflects the gamete's haploid genome (i.e., altering $p_{\min}$). With this revised definition of the extent of gamete masking, the spread of a modifier continues to be described by Eq. **1** with Eqs. **S14**–**S16**, except that the modifier does not affect selection experienced by a gametes from aa fathers so that the term $(1 - c_{ij}t)$ in Eq. **S15** becomes $(1 - \hat{c}\ t)$ according to the average degree of gametic selection in the population, $\hat{c}$ (assumed equal to 1 in Fig. 2).