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THE EVOLUTION AND SUPPRESSION OF MALE SUICIDE UNDER PATERNAL GENOME ELIMINATION

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Different genetic systems can be both the cause and the consequence of genetic conflict over the transmission of genes, obscuring their evolutionary origin. For instance, with paternal genome elimination (PGE), found in some insects and mites, both sexes develop from fertilized eggs, but in males the paternally derived chromosomes are either lost (embryonic PGE) or deactivated (germline PGE) during embryogenesis and not transmitted to the next generation. Evolution of germline PGE requires two transitions: (1) elimination of the paternal genome during spermatogenesis; (2) deactivation of the paternal genome early in development. Hypotheses for the evolution of PGE have mainly focused on the first transition. However, maternal genes seem to be responsible for the deactivation and here we investigate if maternal suppression could have evolved in response to paternally expressed male suicide genes. We show that sibling competition can cause such genes to spread quickly and that inbreeding is necessary to prevent fixation of male suicide, and subsequent population extinction. Once male-suicide has evolved, maternally expressed suppressor genes can invade in the population. Our results highlight the rich opportunity for genetic conflict in asymmetric genetic systems and the counterintuitive phenotypes that can evolve as a result.

KEY WORDS: **Genetic system, genomic conflict, male-killing, paternal genome elimination, scale insect.**

It is now known that there is a great diversity of genetic and sex-determining systems across taxa, resulting in differences in reproductive mode, ploidy levels between the sexes and the mechanisms of sex determination (Norton et al. 1993; Normark 2003; Uller et al. 2007). Furthermore, these differences can occur between closely related taxa (such as scale insects: Ross et al. 2010). However, the evolutionary significance of this variation is poorly understood. Recently the role of conflict between different genetic entities on the evolution of novel genetic and sex determination systems has gained widespread attention (Hurst 1995; Normark 2004a, 2006; Uller et al. 2007; Ross et al. 2010). These genetic conflicts can arise both within genomes (for instance between driving sex chromosomes and autosomes: [Burt and Trivers 2006]) or between genomes (for instance between hosts and symbionts: [Wernegreen 2004; Werren et al. 2008]). In this article, we consider the role of intragenomic conflict on the evolution of one particular system: paternal genome elimination (PGE).

PGE is found in several taxa among insects and mites (Nur 1980; Norton et al. 1993; Normark 2003). PGE can be roughly divided into two classes. The first is embryonic PGE, in which the paternal genome is eliminated early during male embryonic development, rendering males haploid (Brown 1965; Nur 1980;

Normark 2003). This system is found in some armored scale insects (Hemiptera: Diaspididae) (Nur 1980) and in some Pytoseiid mites (Acari: Phytoseiidae) (Cruickshank and Thomas 1999). The second is germline PGE, in which the paternal genome remains present in males, but is eliminated from the germline during or just before spermatogenesis and is therefore not transmitted, making males effectively haploid in terms of their transmission genetics (Schrader 1921; Brown and Nelson-Rees 1961; Nur 1980; Normark 2003). This system is found in most scale insects (Hemiptera: Coccoidea) (Nur 1980), in sciarid flies (Diptera: Sciaridae) (Goday and Esteban 2001) and in the coffee berry borer beetle, *Hypothenemus hampei* (Coleoptera: Scolytidae) (Borsa and Kjellberg 1996).

Although the evolutionary relationship between the two systems is unresolved in some taxa, it is clear at least in scale insects that embryonic PGE has evolved from germline PGE (Nur 1980; Morse and Normark 2006; Ross et al. 2010). Interestingly, in species with germline PGE, even though the paternal genome is present in all tissues, it is deactivated in most. In one scale insect (the mealybug *Planococcus citri*) this deactivation has been shown to be induced by the maternal genome (Chandra 1962; Nur 1962; Brown and Nur 1964). Therefore, the evolution of germline PGE consists of two important evolutionary transitions: (1) the elimination of the paternal genome from the germline; (2) the deactivation of the paternal genome early in development. Explanations for the evolution of PGE have in general focused only on the first of the two transitions. The hypotheses of Brown (1964) and Bull (1979) assume that maternal chromosome drive has led to the evolution of PGE and therefore focus only on the first transition. Similarly, the hypothesis of Haig (1993a) considers the role of X-chromosomal drive in the evolution of PGE and again focuses exclusively on the first evolutionary transition. These three models all consider intra-genomic conflicts. In contrast, the fourth hypothesis, formulated by Normark (2004a), assumes the involvement of male-killing endosymbionts. He argued that to kill males (which do not transmit the endosymbionts) the endosymbionts destroy male-determining sperm when they fertilize the oocytes. However once the host evolves haploid male viability, this leads to a similar type of maternal chromosome drive as in the models of Brown, Bull and Haig.

Herrick and Seger (1999) were the first to note that once the elimination of the paternal genome from the male germline has evolved, this leads to other evolutionary conflicts of interest between paternal and maternal genes in males. Specifically, they argued that there would be selection on the paternal genome to evolve mechanisms to prevent this elimination. The paternal genome might have several options for doing so. For instance, it could completely block PGE, by restoring a fair meiosis and resisting the elimination during spermatogenesis. Alternatively individual chromosomes might occasionally be able to swap place with a maternal homologue and thereby gain access to the sperm. Herrick and Seger (1999) also argued that these attempts by paternally inherited genes to regain transmission will select for a counter response by the maternal genes. They argue that one way for the maternal genome to prevent counter adaptation by the paternal genome is to deactivate the paternal genome. In a verbal model, they propose that continuing coevolution between the maternal and paternal genes in males might have lead to the gradual deactivation of the paternal genome, starting with genes or chromosomes in germline cells, as these might be more "powerful" in affecting their own transmission, but gradually spreading to the soma as well. They also argued that this maternal–paternal coevolution might have caused the evolution of the different types of PGE in which the paternal genome is eliminated from the germline progressively earlier (reviewed by Ross et al. 2010).

However, although there will be strong selection on the paternal genes to regain access to the germline and thereby gain direct fitness, this might be hard to achieve. In species with PGE, meiosis and spermatogenesis are modified so that even if paternal chromosomes avoid elimination this might not necessary lead to successful transmission, as it will often lead to diploid or nonfunctional sperm. Furthermore, "normal" meiosis and spermatogenesis might not have taken place in PGE species for millions of generations and the resulting loss of necessary genes might hinder the restoration of normal diplodiploidy (Nur 1970; Herrick and Seger 1999).

There might however be another way in which paternal genes can increase their fitness. Although males do not transmit their paternal genes to the next generation and therefore the paternal genome in males does not have any direct fitness, paternal genes can obtain indirect fitness by enhancing survival or reproduction of sisters or other relatives. This leads to a situation within a sibgroup where paternal genes in males can favor their sisters reproduction at the expense of their own (Normark 2001). Specifically, we argue that paternal genes may be selected to commit suicide, if the surviving sisters can use the newly available resources and increase their fitness. This is then an intragenomic version of the well-known argument for male-killing by maternally transmitted endosymbionts (Hurst 1991).

The first aim of this article is to investigate theoretically under what conditions a paternally expressed suicide gene could invade a population. We will test how population substructure and resulting levels of sib-mating will affect (1) if a suicide gene can invade and (2) what level of male-killing is expected under different levels of inbreeding. Once a male suicide gene has invaded in the population, this will have strong effects on the population sex ratio. We therefore also explore if the presence of a paternally expressed male suicide gene selects for biased primary sex ratios. Finally, the invasion of a paternally expressed male suicide gene is expected to impose a strong selection pressure on the maternal

genes in males to suppress the suicide phenotype. We therefore also model the spread of a maternally expressed suppressor gene, once a male-suicide gene is present, and discuss if this could have led to the deactivation of the paternal genome in males.

INCLUSIVE FITNESS MODEL FOR SUICIDE EVOLUTION

To understand if paternal suicide genes could evolve in taxa with PGE, we need to consider the life history of those taxa. Normark (2004a) pointed out that most taxa with PGE not only have strong levels of sib-competition (which would increase the selection pressure for male suicide) but also high levels of sib-mating and inbreeding. At first glance, one might expect inbreeding to counteract the spread of paternal male suicide as it can lead to increased relatedness between the maternal and paternal genome of individual. However, inbreeding also increases relatedness between sibs, which might promote male suicide. To make matters even more complicated, a life history with inbreeding and sib-competition may select for female-biased sex ratios, thus increasing the reproductive value of individual males, which might be an additional obstacle to the evolution of male suicide. Clearly, a formal model is required to investigate the balance of these opposing effects.

We consider the fate of a partially suicidal gene that is expressed in males by the paternally inherited half of their genome. We allow for some degree of inbreeding by assuming that the population is subdivided in standard-sized patches of n mated females whose offspring mate randomly on their natal patch followed by dispersal of newly mated females to random patches according to a standard "island model" of dispersal.

Offspring mortality occurs in two subsequent "rounds". In round one—the male suicide round—some males may die during early development as a result of the action of a paternally inherited gene. The resources accumulated by (or not exploited by) dead males can be partially recycled and enhance the survival of their sibs during the second round of offspring mortality. Specifically, we assume that a focal male commits suicide with probability x, while x_b is the average suicide probability among all males in the focal brood and x_p is the patch-level suicide probability of males during round one. In the second round, individual male and female survivors of round one will survive an additional round with (nonsex specific) probability

$$y_b = y_0 + (1 - y_0)bsx_b. \tag{1}$$

Here $0 < y_0 \leq 1$ is a baseline level of survival in case no male sibs were killed during round one, and the second term on the right represents the (linear) increase in survival with the amount of resources made available by deceased male sibs. Parameter $0 \leq b \leq 1$ is a measure of recycling efficiency and $0 \leq s \leq 1$ is the brood sex ratio (proportion males). Thus, minimal survival in phase two equals y_0, whereas survival approaches unity in case the brood consists almost entirely of suicidal males that are recycled with maximal efficiency($x_b \approx 1$, $b \approx 1$ and $s \approx 1$). In what follows, for the easy interpretation of the derived formulas, we assume $y_0 = 1/2$, but this has no qualitative effect on the conclusions.

We want to calculate the inclusive fitness effect of a small change in the suicidal tendencies of the focal gene, and for this we need to consider how the fitness of females and males depend on x, x_b, and x_p. We assume the fitness of a female depends only on her brood-level x_b (i.e., the mean suicide rate of her brothers) mediated by its effect on round two survival of females:

$$W_f = y_b. \tag{2}$$

The fitness of a focal male is his probability of survival $(1 - x_b)y_b$ across both rounds times his expected number of mates $(1 - s)/[(1 - x_p)s]$:

$$W_m = (1 - x)y_b \frac{1 - s}{(1 - x_p)s}. \tag{3}$$

The inclusive fitness effect of a small change in x can then be calculated according to a standard method (Taylor and Frank 1996; Pen 2006) as

$$\Delta W_{IF} = s\frac{\partial W_m}{\partial x}r + 2(1 - s)\frac{\partial W_f}{\partial x_b}r_f + s\frac{\partial W_m}{\partial x_b}r_{m_b} + s\frac{\partial W_m}{\partial x_p}r_{m_p}. \tag{4}$$

The right-hand side is evaluated at $x = x_b = x_p$. The marginal fitness effects (the partial derivatives) for each sex are multiplied by the frequency of each sex, as dictated by the sex ratio s. Female fitness is additionally multiplied by 2 because in haplodiploids the reproductive value of a daughter is twice that of a son in terms of passing on genes to future generations (Hamilton 1979; Bulmer 1994). The various r-parameters are different coefficients of relatedness from the viewpoint of the controlling gene, in this case the paternally inherited x-gene in a focal male. Specifically, the coefficient r is the relatedness of the maternal genome to the paternal genome in the focal male, and it equals the inbreeding coefficient f, because f is by definition the probability that an individual's maternally and paternally inherited genes are identical by descent. The coefficient r_f is the relatedness of a sister to the controlling gene in the focal male, and this equals $r_f = \frac{1}{2} + \frac{1}{2}f$, the mean of the relatedness of the sister's paternal genes to the controlling gene (a relatedness of 1, because fathers are effectively haploid) and the relatedness of her maternal genes to the controlling gene (by definition, f). Similarly, $r_{m_b} = f$ is the relatedness of a brother's maternal genome to the paternal genome of the focal male, and $r_{m_p} = (1/n)f$ is the relatedness of a random male competitor from the focal patch to the paternal genome of the focal male.

Replacing the coefficients of relatedness in (4) with the derived expressions in terms of inbreeding coefficients gives

$$\Delta W_{\mathrm{IF}} = s\frac{\partial W_{\mathrm{m}}}{\partial x} f + (1-s)\frac{\partial W_{\mathrm{f}}}{\partial x_{\mathrm{b}}}(1+f) + s\frac{\partial W_{\mathrm{m}}}{\partial x_{\mathrm{b}}} f + s\frac{\partial W_{\mathrm{m}}}{\partial x_{\mathrm{p}}} f/n. \tag{5}$$

From inspecting the definitions of W_{m} and W_{f}, it is clear that all partial derivatives on the right-hand side of (5) are positive except for the first one ($\partial W_{\mathrm{m}}/\partial x$). Therefore, if there is no inbreeding ($f = 0$), only a single positive term remains, and suicide (x) of males will evolve to its maximal value (i.e., all males commit suicide). Therefore some minimum level of inbreeding (i.e., $f > 0$) is required for selection against 100% male suicide.

The equilibrium suicide rate is found by calculating the derivatives in (5), evaluating them at $x = x_{\mathrm{b}} = x_{\mathrm{p}} = x^*$, setting the right-hand side equal to zero and solving for x^*:

$$x^* = \frac{n(1+2f) - (n-1)f/(bs)}{n + (3n-1)f}. \tag{6}$$

or $x^* = 0$ if the right-hand side is negative (i.e., there is no male suicide). Note that $x^* = 1$ when $f = 0$, that is, in the absence of inbreeding, selection favors 100% male suicide, which would cause population extinction.

The inbreeding coefficient f depends on patch size n, and can be considered a "fast variable" relative to the speed of evolution, whose quasi-equilibrium value can be calculated from a standard recursion equation (see Taylor 1988):

$$f = 1/(4n-3). \tag{7}$$

Plugging the resulting f into (6) gives the main result

$$x^* = \frac{n(4n-1) - (n-1)/(bs)}{4n^2 - 1}. \tag{8}$$

or $x^* = 0$, whichever is larger. From inspection, it is clear that—all else being equal—for sufficiently small b-values there will be no selection for suicide. A female-biased sex ratio (small s) also leads to lower suicide rates, and finally, x^* increases with n.

Some examples of x^* for varying values of b and n are shown in Figure 1. For the brood sex ratio s we took the equilibrium value under maternal control, and we show in the Appendix 1 how this is calculated. In addition to the analytical solutions, we also show results of individual-based simulations to verify the stability of the equilibria (see Appendix 2; C++ code is available on request). It is clear from Figure 1 that male suicide is straightforward to evolve. It is also interesting that primary sex ratios can be male-biased, in contrast to the sex ratios in standard LMC models (West 2009).

To confirm our prediction that under no inbreeding the evolution of male suicide can lead to population extinction in Figure 2 we show simulation results where we assume a single large random-mating population ($n = 10{,}000$) and show that male suicide quickly evolves to 100% and that this drives the population extinct. Further details on this simulation can be found in the Appendix 2.

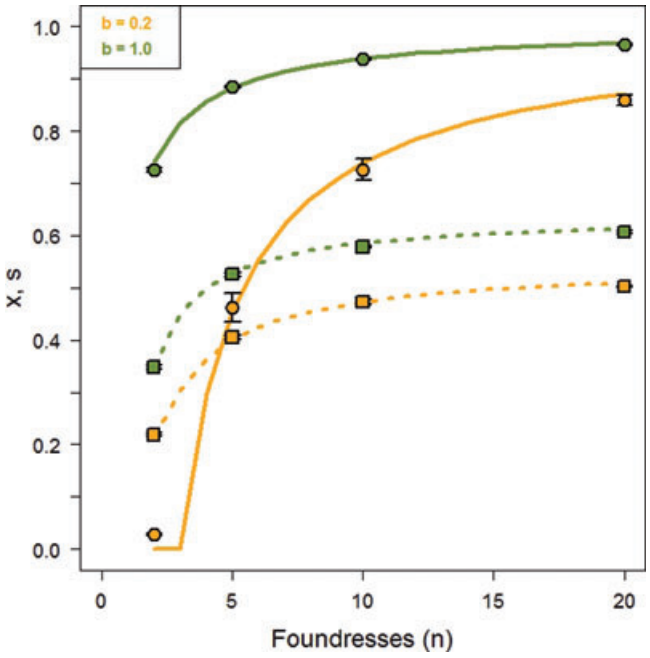

Figure 1. Male suicide can evolve and generate male-biased equilibrium sex ratios. Equilibrium levels of male suicide rates *x* and brood sex ratios *s* (proportion male), as a function of number of females (foundresses, *n*) per local patch. Solid curves represent male survival as predicted by the analytical model for two values of *b*, the efficiency of recycling killed males into resources for sibs. Dashed curves represent coevolved sex ratios as predicted by the analytical model. Note that male-biased sex ratios arise for some parameter combinations. The individual-based simulation results are presented by symbols representing averages (±1 standard deviation) of 10 replicates (circles: male survival; squares: sex ratios). The simulations fit the analytical predictions quite closely.

COUNTER-EVOLUTION OF MATERNALLY INHERITED SUICIDE-SUPPRESSORS

In the previous section, we have shown that under PGE, a paternally expressed gene is able to evolve male suicide, as long as sibs can benefit sufficiently from recycled resources. Here, we explore if suppression expressed by maternally inherited genes can evolve, once male suicide is present. We use an individual-based simulation approach, where we allow a maternally expressed suppressor gene z to evolve simultaneously with x. This locus determines the probability of expression of x. We would first like to see if a maternal suppressor (z) is able to invade, under what conditions it will invade, and if it will lead to partial or complete suppression. We would also like to see how fast such a maternally expressed suppression gene will spread and if it will go to fixation. Finally, we explore how the efficiency (b) with which the resources that

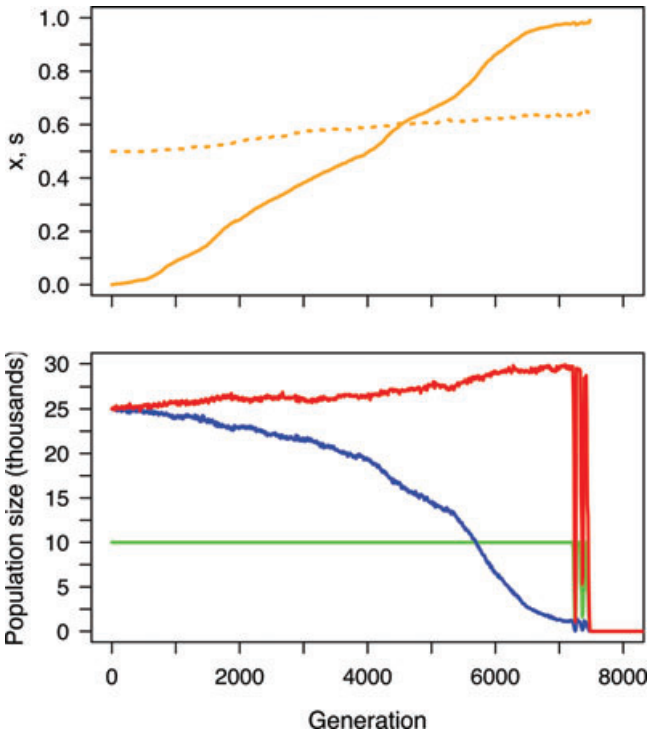

Figure 2. Under the absence of inbreeding, male suicide can lead to population extinction due to the resulting lack of males. Simulation results for the evolution of male suicide in a large undivided population. The top panel shows the value of the suicide gene *x* (solid line) and the sex allocation gene *s* (dashed line). The bottom panel shows the number of reproducing females (in green or light grey [in printed version]) and the number of surviving sons (in blue or dark grey) and daughters (in red or medium grey). Further parameter values are given in the Appendix 2.

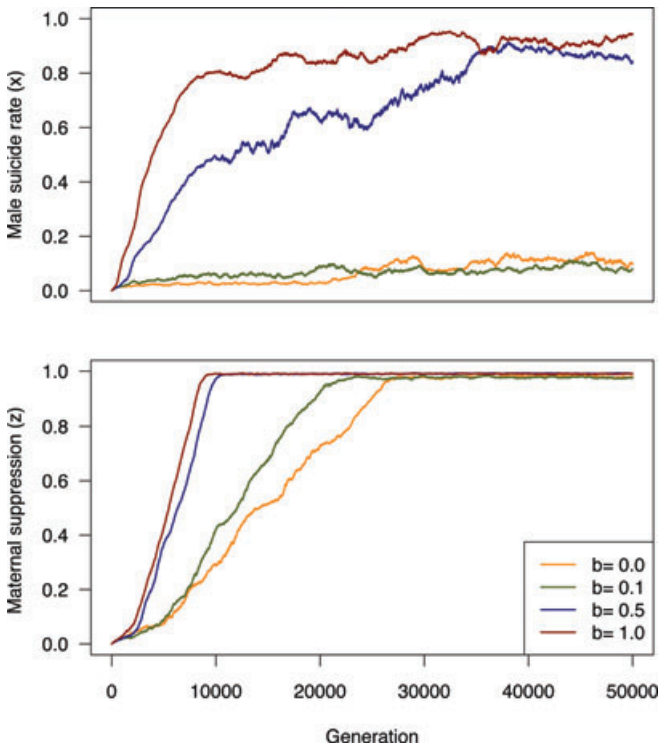

Figure 3. Maternal suppression of male suicide can evolve, even when there is rather little suicide. Simulation results for the evolution of a maternally expressed suicide-suppressor. The top panel shows the value *z* of the suicide suppressor. The bottom panel shows the value of *x*, when *x* and *z* are allowed to evolve simultaneously (and the expression of *x* is determined by *z*). In both panels, results are shown for different levels of the recycling efficiency ($b = 0, 0.1, 0.5, 1.0$). Further parameter values are given in the Appendix 2.

become available after male-killing can be used by the male's siblings affects the evolution of maternal suicide suppression. Simulation results are shown for a local mate competition scenario with four foundresses per patch (Fig. 3; see Appendix 2 for details) and four different recycling efficiencies (b). These results first of all show that a maternally expressed suppression gene can invade under all the conditions that were considered and that it leads to complete suppression of the paternally expressed suicide gene. Second, they show that although the suppression gene spreads to fixation under all conditions, the recycling efficiency rate *b* affects how fast *z* spreads and becomes fixed, with a faster spread at higher recycling efficiencies.

Discussion

Asymmetric genetic systems, in which transmission is unequal for different genetic entities or elements, are a rich evolutionary playground for strange and seemingly counter-intuitive phenotypes (Burt and Trivers 2006; Normark 2006). We have shown that in species with one such asymmetric system, PGE, if paternal genes are expressed in males then the evolution of genes causing male suicide is possible, as long as sibs can profit from the recycled resources of killed males. In the absence of inbreeding, our model predicts the evolution of a rate of 100% male suicide, which will lead to population extinction (Fig. 2), while increasing levels of inbreeding limits the extent of male suicide or may even prevent it altogether. As male suicide evolves, coevolution of the sex ratio may occur, and this can lead to male-biased primary sex ratios, as males may benefit their sibs when they commit suicide. This is surprising as predictions of male-biased population sex ratios are rare under the standard sex ratio models, and the population structure modeled here, would normally predict strongly female-biased sex ratio (according to local mate competition theory (Hamilton 1967). We have also shown that once these male suicide genes have evolved, a maternally expressed suppressor can evolve and that this results in a complete suppression of the paternally derived suicide genes.

As discussed earlier, the evolution of PGE will lead to conflict between maternally and paternally inherited genes in males. It has previously been noted that PGE results in selection on

the paternal genes to resist their elimination from the germline in males. However, only two cases of reversal from PGE back to normal diplodiploidy have been observed (in the scale insect genera *Lachnodius* and *Stictococcus*) (Nur 1980) and both evolved from germline PGE. So although this shows that reversal is possible, it is rare. Our results show that in cases in which paternal genes cannot—for whatever reason—defeat PGE, they may still obtain indirect fitness benefits by evolving a male-killing phenotype.

Our results also suggest that the evolution of paternally expressed suicide genes could trigger the evolution of maternal suppression of the paternal genome set to silence suicide genes. Although (partial) paternal genome deactivation in males has been shown in all taxa with germline PGE, the mechanism of suppression has been mainly studied in mealybugs. In these species, it has been shown that DNA methylation plays an important role in the deactivation. The paternal genome is found to be hypomethylated in both sexes and several histone proteins have been shown to be involved in the deactivation (Bongiorni et al. 1999; Bongiorni et al. 2007). When the expression of these histone proteins is blocked, this results in the reactivation of the paternal genome (Bongiorni et al. 2007). These results agree with earlier observations of individuals with artificially constructed haploid embryos that lacked the maternal genome in which the paternal genome became active (Brown and Nur 1964), suggesting maternally expressed suppression.

It has been argued earlier that conflict over transmission through sperm could have led to the evolution of maternal deactivation of the paternal genes to stop paternal attempts to regain transmission (i.e., "policing" PGE itself; Herrick and Seger 1999). However although the deactivation of the paternal genome in males would indeed prevent those attempts, it will presumably come with a considerable fitness cost for the male. Furthermore it is hard to reconcile with the observation that in mealybugs although the paternal genome is deactivated in most tissues it is active in the testis, the very place where it is eliminated. If the paternal genes are deactivated to prevent them from fighting their elimination, we would expect them to be repressed most strongly in tissue where they might have most power to affect their transmission.

The alternative explanations for the deactivation of the paternal genome will be difficult to distinguish, and currently little has been done to experimentally manipulate maternal deactivation of paternal chromosomes in these species, and so the phenotypes that would result are unknown. If maternal deactivation is preventing paternally driven male suicide, then male death (including failed embryos) may be the result of such manipulations. However, such phenotypes are inherently hard to study, especially in terms of confirming the cause of the embryonic (or later stage) mortality. To test if paternally expressed suicide genes have indeed evolved and that the suppression of the paternal genome has evolved in response it may be helpful to focus on systems in which the suppression is incomplete, or where the extent of male suicide is incomplete in the absence of maternal suppression.

In addition to wrestling over control of paternal gene expression in males, there are other possible outcomes or ways to avoid male suicide. In sciarid flies only certain paternal chromosomes are lost during embryogenesis, whereas the others remain active in the soma (Haig 1993b). This might make *Sciara* particularly susceptible to the evolution of paternally expressed male suicide genes. However, many species of sciarid flies are completely monogenic (i.e., females produce broods of one offspring sex only, thus exhibiting "split sex ratios" [Haig 1993b]) or have monogenic strains. This will presumably eliminate selection in favor of male suicide as males do not have sisters to channel indirect benefits. Simulations confirm (results not shown) that a monogenic population cannot be invaded by paternally inherited alleles that cause male suicide. Whether the converse also holds true—that monogeny is an adaptation to male suicide—remains an interesting speculation. Monogeny appears to be quite rare, having been found mostly in dipteran species with PGE: Sciarids and Cecidomyids (Haig 1993b; Dorchin and Freidberg 2004).

Currently, no direct evidence for paternally expressed male suicide is available for species with PGE. However many species are poorly studied and male-suicide will be hard to observe as it might it might only reveal itself as female-biased sex ratios, which could be easily overlooked or interpreted as facultative sex ratio adjustment. Furthermore, observing male-suicide might be difficult as once such a phenotype evolves there will be strong selection on maternal genes, for example by the suppression of the paternal genome, or by producing split sex ratios. Additionally, if such suppression does not evolve quickly enough it might lead to population extinction. Comparative approaches to testing the correlates of PGE might help us make progress though. Interestingly, one such study has recently shown that each of the two origins of embryonic PGE in scale insects is associated with an increase in net diversification rate, possibly indicating a reduced extinction rate as a result of suppressing paternal gene expression (Andersen 2009).

The evolution of suicidal phenotypes might seem counterintuitive, but there are ample examples in other contexts. Perhaps best known are those induced by endosymbiotic bacteria that either kill their male host (and thereby themselves) to benefit related endosymbionts in females: "male-killing" (Hurst 1991, 1995) or that kill early embryos resulting from crosses between an infected male and uninfected female: "cytoplasmic-incompatibility" (Wade and Stevens 1985; Werren et al. 2008). Similar transmission genetics impose similar selection on mitochondria. Although mitochondria have not been found to induce male suicide, they have been linked to reduced male fitness, especially reducing sperm function in a number of taxa (Wade and

Brandvain 2009). Additionally mitochondria have been found to induce the sterility of male function in hermaphroditic plants (Saumitou-Laprade et al. 1994). Finally mitochondria have recently been found to play a crucial role in apoptosis (programmed cell death: [Blackstone and Green 1999]), although the evolutionary significance of this finding is not well understood. Wade and Brandvain (2009) recently showed that although mitochondria cannot obtain any direct fitness through males, either under inbreeding or in situations in which males help their sisters, they can obtain indirect fitness. This might explain why there is selection against mitochondrial mutations that have a deleterious effect on male fitness under these conditions. However, as our model shows, under conditions of sib competition, such a mutation might spread.

Other genetic entities that under certain conditions could be selected to induce suicide are the polar bodies. These cells form during meiosis and contain the three haploid genome sets that do not form the final germ cell. In most species these cells quickly degenerate although in some taxa they persist, for instance forming the endosperm in plants (Haig 1986). Similarly, in some scale insects the maternally derived polar bodies fuse with an embryonic cell to form the organ in which the endosymbiotic bacteria reside (Brown 1965; Tremblay and Caltagirone 1973; Normark 2001, 2004b). This inclusion of the maternally derived polar bodies in an embryo might increase genomic conflicts within the individual as it creates tissue that contains both maternal and embryonic genes (Normark 2001; Burt and Trivers 2006) (Normark 2004b). With sibling-competition, the interests of the embryo- and polar body-derived genes might not coincide as some polar body genes might be absent from the embryo but present in its siblings and so in line with the previous argument for the evolution of paternally expressed male-killing, the genes derived from the maternal polar bodies might also be selected to evolve suicide (Normark 2001). Therefore some of the variation in bacteriome formation found in mealybugs and armored scale insects might have evolved through selection on chromosomes outside the bacteriome to limit the expression of suicidal genes. For example, Brown (1965) showed that in some armored scale insect species the bacteriome contains three condensed haploid genomes. He suggested that these are the chromosomes from the polar bodies that, although present, have been deactivated (Normark 2001). If this is indeed the case it shows an interesting similarity with the fate of the paternal genome in the soma of males with PGE.

An important assumption underpinning our models is that there is competition among siblings and that the resources that become available through the death of a male can be used by its sisters. There is evidence of sibling competition in a species of mite with PGE (Nagelkerke and Sabelis 1998), whereas scale insects (where PGE is the most common genetic system) have evolved several reproductive adaptations that lead to intensive and prolonged contact between siblings. For example vivipary and ovoviviparity are common among scale insects and many taxa have evolved an ovisac or a marsipium in which their offspring develop (Gullan and Kosztarab 1997). Moreover, scale insects are also often sedentary and settle close to the place they were born, typically forming large colonies on host plants. Due to these factors strong sibling-competition might be expected (Normark 2001, 2004a).

However, the flip-side of an ecology that promotes sibling competition is that it might also promote sib-mating. Recently it has in fact been noted that PGE often evolves in species with mating systems that lead to high levels of sib-mating (Hamilton 1993; Normark 2004a). Our results show that while under PGE paternal suicide genes can invade, inbreeding leads to a lower level of suicide. It is therefore tempting to suggest that inbreeding might be required to prevent population extinction (due to fixation of paternally expressed suicide genes) and perhaps this is why PGE is observed primarily in species with high levels of sib-mating. However, it will be difficult to disentangle the opposing effects of sib-competition and sib-mating in promoting or preventing male suicide.

In this article we have presented the possibility that in species with PGE intragenomic male killing can evolve. The conditions that are required for the evolution of intragenomic male killing to evolve are similar to those required for intergenomic, endosymbiont induced male killing (Hurst 1991). Furthermore, most taxa with PGE harbor endosymbiotic bacteria (Normark 2004a), with which they often have an intimate and obligate association. This suggests that in many of these taxa both the endosymbiont and the paternal genome in males could be selected to induce male killing and this therefore raises the tantalizing possibility that inter- and intragenomic suicidal interests may interact to facilitate male-killing.

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Appendix 1: Sex Ratio Coevolution

Here, we derive an inclusive fitness model for the coevolution of brood ratios under maternal control in a subdivided population of patches with n females each.

A focal mother produces a brood sex ratio s_b (proportion sons), while the patch-level mean sex ratio is s_p. Her fitness through daughters is then given by

$$W_f = (1 - s_b)y_b. \quad \text{(A1)}$$

Note that $y_b = y_0 + (1 - y_0)bs_b x_b$ depends on the brood sex ratio, and this is where our model differs from the standard models of sex ratio evolution in subdivided populations (West 2009). Also note that for $x_b = 0$ our model reduces to the standard models.

A focal mother's fitness (number of mated females) through sons is given by

$$W_m = s_b(1 - x_b)y_b \frac{1 - s_p}{s_p}. \quad \text{(A2)}$$

The inclusive fitness effect of a small change in the mother's sex ratio is then obtained according a standard direct fitness method (Taylor and Frank 1996):

$$\Delta W_{IF} = 2\frac{\partial W_f}{\partial s_b} r_{f_b} + \frac{\partial W_m}{\partial s_b} r_{m_b} + \frac{\partial W_m}{\partial s_p} r_{m_p}. \quad \text{(A3)}$$

Note that female fitness is multiplied by two to account for their double reproductive value compared to males in haplodiploids. The relatedness coefficients are as follows. The relatedness of daughters to their mother is given by

$$r_{f_b} = \frac{1 + 3f}{2 + 2f} \quad \text{(A4)}$$

Relatedness of sons to their mother: $r_{m_b} = 1$; relatedness of random male to mother:

$$r_{m_p} = 1/n \quad \text{(A5)}$$

Analytical solutions of (A3) are easily available but rather uninformative. In the case of $x_b = 0$ they reduce to well-known results (Hamilton 1979; Taylor and Bulmer 1980; West 2009).

In the scenario of coevolving suicide rates and sex ratios, equations (5) and (A3) must be solved simultaneously. Note that (8) is no longer an explicit solution of (5), because the s in (5) now depends on x. We did not analytically check for stability of solutions but relied on the individual-based simulations to verify stability properties.

Appendix 2: Details of Individual-Based Simulation Models

PATERNALLY EXPRESSED MALE SUICIDE

The simulations work with a population of diploid individuals, sub-divided into n_P standard-sized patches, each founded by n mated females. Each female lays a clutch of $k = 50$ offspring with a binomial sex ratio determined a single additive gene locus. The early survival of male offspring is determined by an additional unlinked single gene locus x that is paternally expressed. The survival of the remaining offspring is influenced by (1) the number of male sibs that have died; and (2) the efficiency b of reallocation of dead sibs. Specifically, survival y_b follows:

$$y_b = 0.5 + 0.5b\frac{k - k'}{k - 1},$$

where $k'$is the number of surviving siblings after male suicide. Note that $0.5 \leq y_b \leq 1$.

The surviving offspring mate with a random individual from the same patch. When there are no males in a patch all females are unable to mate and the patch will go extinct. After mating females disperse with probability d. The dispersing females are randomly assigned to a patch until the n breeding positions on a patch are occupied.

Alleles were mutated with a rate of 0.01 per generation, and given that a mutation occurred, the mutation step size was drawn from a normal distribution with mean zero and standard deviation 0.01 (see Table A1). More realistic lower mutation rates (e.g., 10^{-6}) did not affect the evolutionary trajectories, but did slow down the simulations considerably.

Table A1. Overview, description, and values of the parameters used in the simulations. The numbers in brackets in the third column show which parameter values have been used in each simulation and correspond with those in the Appendix 2 (simulation 1: Paternally expressed male suicide, results shown in Fig. 1, simulation 2: Maternal suppression, results shown in Fig. 2 and simulation 3: Polar body induced male suicide, results shown in Fig. 3).

Parameter	Description	Value used in simulation
n_P	Number of patches	2500 (1,3), 1 (2)
N	Number of mated females per patch	4 (1,3), 10,000 (2)
K	Clutch size	10 (1,2,3)
s	Sex ratio	evolving (1,2,3)
b	Efficiency reallocation of dead sons	1.0 (1) 0, 0.1, 0.5, 1.0 (2)
		0.5 (3)
x	Male suicide rates	evolving (1,2,3)
z	Suppressor gene (maternally expressed)	evolving (2)
μ	Mutation probability	0.01 (1,2,3)
σ	Standard deviation mutation size	0.01 (1,2,3)

EXTINCTION UNDER RANDOM MATING

In this simulation we test if male suicide can lead to population extinction when there is no inbreeding (under random

mating). The simulation is similar to the one described above but with two important differences. First of all in this simulation we assume one large random-mating population (instead of a subdivided population as previously assumed). Second here we make an additional assumption on the number of females a male can successfully inseminate, with a maximum of 20 females per male. Each female in the population is randomly assigned a mate, however when her mate has already had 100 previous mating, the female remain uninseminated and will fail to produce offspring. See Table A1 for the parameter values used in this simulation.

MATERNAL SUPPRESSION

This simulation explores the evolution of a gene that suppresses the paternally inherited suicide genes. The simulation is identical to described above, except an additional independently segregating gene coding for maternally inherited suppression, which determines the probability of expression of x.