Harmful Mating Tactics in Hermaphrodites

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Submitted July 3, 2008; Accepted November 19, 2008; Electronically published March 20, 2009

ABSTRACT: While empirical data suggest that sperm competition and multiple mating both contribute to the evolution of harmful mating tactics in hermaphrodites, a precise understanding of their interaction is lacking. We therefore formulate a game-theoretical model of mating behavior in hermaphrodites, where harmful mating tactics confer an advantage in sperm competition while simultaneously reducing the mating partner's survival. The model predicts evolutionarily stable values of resource allocation between sexual functions and the degree of harmful mating. Our analysis provides support for the empirical observation that harmful mating is associated with multiply mating species in which sperm precedence strongly favors the first mate. The model also shows that this criterion becomes less important as harmful mating tactics become more efficient. As harmful tactics make sperm displacement more effective, a consequence is a more female-biased resource allocation. Provided that fertilized egg production is not limited by availability of sperm, a more female-biased allocation should increase the number of offspring produced, but the model instead shows that harmful mating tactics more than countercompensate, leading to reduced fitness. Hermaphrodites that use harmful mating tactics may therefore be at a disadvantage when competing with other species for a limited resource.

Keywords: hermaphrodites, harmful mating tactics, sex allocation, sperm competition.

Introduction

Sexual reproduction between males and females is frequently a discordant affair because of a conflict between the interests of males and females. This means that if an adaptation to increase fitness arises in one sex, then a counteradaptation may arise in the other, leading to a potential, perpetual coevolutionary arms race (Arnqvist and Rowe 2005). In hermaphrodites, these conflicts are further complicated because an individual can take both roles at the same time while simultaneously making fine adjustments to the resources allocated to each sex (Michiels 1998; Arnqvist and Rowe 2005).

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Am. Nat. 2009. Vol. 173, pp. 000–000.
0003-0147/2009/17305-50577$15.00.
DOI: 10.1086/597377

There has been some debate as to precise definitions, but the origin and maintenance of harmful male adaptations is generally understood from two main perspectives. First, the "adaptive harm" hypothesis (Johnstone and Keller 2000; Morrow et al. 2003; Lessells 2005) defines a general scenario in which a male harming a female causes a change in behavior so as to directly increase paternity (e.g., reduced probability of remating). An alternative perspective is that of "collateral harm," where harm evolves as a negative pleiotropic side effect of a trait that benefits the male function (Parker 1979), such as increased efficiency of allosperm displacement.

Examples of male harming behavior include the seminal "toxins" of *Drosophila* fruit flies (Chapman et al. 1995), genital spines that prolong copulation (Lloyd 1979), hypodermic insemination to moderate female choice (Michiels and Newman 1998), and "love" darts to hormonally influence fertilization (Koene and Chase 1998; Landolfa et al. 2001). The aim of all these tactics appears to be to increase the sperm precedence of the harming male, though the tactics also appear to cause physical damage to the female. In extreme circumstances, some tactics have been observed to increase probability of the female's death following mating (Crudgington and Siva-Jothy 2000; Blanckenhorn et al. 2002).

Michiels and Koene (2006) have shown that collateral harm may evolve in both gonochorists and hermaphrodites when linked to sperm precedence. However, their model was limited in that individuals could not mate more than twice, and resource allocation between male and female functions was fixed for hermaphrodites. Since another model of sperm competition in hermaphrodites (Charnov 1996; Greeff and Michiels 1999) showed that the most successful resource allocation strategy is often not an equal division of resources between male and female functions, further refinements are clearly required.

We therefore extended a previous sperm competition model (Charnov 1996; Greeff and Michiels 1999)—one that in the absence of harm predicts that the resource allocation to male and female function can tend to equality in hermaphrodites—to include mating tactics that cause collateral harm. These tactics increase sperm precedence

for the sperm donor but reduce the survival probability of the sperm recipient. We formulate fitness equations analytically and search numerically for evolutionarily stable pairs of values for resource allocation to male function and degree of harmful mating tactics. This mathematical approach allows predictions that would be difficult to obtain simply by intuition.

Charnov's Infinity Model

Charnov (1996) considered a hermaphrodite over an interval of time yielding one mating. Each hermaphrodite has a total resource R at its disposal per mating. This resource is divided such that a fraction r is allocated to the male function (sperm production), leaving a fraction $1 - r$ to the female function (egg production). Sperm competition is modeled by a function $\phi(r)$, which represents the fraction of sperm an individual displaces in its mating partner's sperm store. Charnov (1996) considered a mutant X with resource allocation $\hat{r}$ in a population with resource allocation r. On mating, the mutant displaces a fraction of sperm, $\phi(\hat{r})$, in the stores of its partner, Y. When Y next lays eggs, X fathers a fraction $\phi(\hat{r})$. Y goes on to mate again, almost certainly with a wild type, as the mutant is rare. This latest mate displaces a fraction $\phi(\hat{r})$ of Y's sperm stores, leaving a fraction $1 - \phi(r)$ from previous mates. When Y lays eggs again, X fathers an additional fraction $\phi(\hat{r})[1 - \phi(r)]$. This process continues as Y remates an infinite number of times. Thus, the fitness of this mutant, X, from one mating is given by

$$W^{(\mathrm{CM})} = R(1 - \hat{r}) + R(1 - r)\phi(\hat{r})\{1 + [1 - \phi(r)] + [1 - \phi(r)]^2 + \ldots\}. \quad (1)$$

The first term in equation (1) represents the fitness from the mutant's female function, and the second term represents fitness from the mutant's male function. Equation (1) can be summed as a geometric series, and the evolutionarily stable strategy (ESS) for resource allocation can be calculated.

Charnov tested three different variations of the sperm displacement function $\phi(r)$ and found that the exact form of the sperm displacement function did not affect the ESS for resource allocation. The sperm displacement functions used by Charnov were characterized by the parameter

$$\delta = Rc/\mu, \quad (2)$$

where c is a constant that converts resource to the number of sperm deposited in the recipient's sperm stores and μ is the total sperm remaining in the recipient's sperm stores from previous mating partners.

One sperm displacement function considered by Charnov (1996) was the case where new sperm is mixed with the stored sperm and a fair sample of this mixture is stored, $\phi_1(r) = \delta r/(\delta r + 1)$. Another sperm displacement function considered by Charnov (1996) represents the case in which sperm flows smoothly into the sperm stores with constant mixing and flushing of new and old sperm, $\phi_2(r) = 1 - e^{-\delta r}$. The sperm stored from previous mating partners (μ) was assumed to be constant in the investigations of Charnov (1996) and Greeff and Michiels (1999) and is assumed to be constant in our model.

Finite Number of Matings Model

Greeff and Michiels (1999) modified the model of Charnov (1996) by considering a hermaphrodite that took part in T reproductive bouts. It was assumed that the population had nonoverlapping generations and that reproductive bouts were synchronous across the population. Following Charnov (1996), their model assumed that an individual oviposits after each mating and that resource allocation is fixed for an individual's lifetime. Lifetime reproductive success (the total number of offspring produced) was used as a measure of fitness.

Greeff and Michiels (1999) considered a mutant with resource allocation $\hat{r}$ in a population with resource allocation r. The mutant's fitness from female function is $TR(1 - \hat{r})$. The fitness from the mutant's male function is given by

$$w_{\mathrm{m}}^{(\mathrm{GM})} = R(1 - r)\phi(\hat{r}) \begin{bmatrix} 1 + [1 - \phi(r)] + [1 - \phi(r)]^2 + \ldots + [1 - \phi(r)]^{T-1} + \\ 1 + [1 - \phi(r)] + [1 - \phi(r)]^2 + \ldots + [1 - \phi(r)]^{T-2} + \\ \vdots \\ 1 \end{bmatrix}. \quad (3)$$

Starting with the first mating, each next line in equation (3) represents male fitness achieved through each successive mating by the mutant. Equation (3) can be summed as a geometric series. Greeff and Michiels (1999) found the ESS resource allocation by numerically searching for a strategy that cannot be invaded by mutants. For simplicity, Greeff and Michiels (1999), like Charnov (1996) before them, used the approximation that the spermatheca already contains sperm at the first mating. They pointed out that this approximation should become more accurate with increasing T.

In agreement with Charnov (1996), Greeff and Michiels (1999) found that sperm displacement functions $\phi_1(r)$ and

$\phi_2(r)$ gave similar results in their analysis. However, they found that $\phi_1(r)$ often yielded equations solvable using standard analytical techniques, and they used $\phi_1(r)$ in their model.

Harmful Mating Model

Here we extend the sperm competition model by Greeff and Michiels (1999) to include a harmful mating tactic that promotes the sperm precedence of the sperm donor at the expense of the sperm recipient.

We found that sperm displacement functions $\phi_1(r)$ and $\phi_2(r)$ gave similar results in the analysis of our model. However, $\phi_1(r)$ yielded equations that were much simpler to solve; thus, we have used the sperm displacement function $\phi_1(r) = \delta r/(\delta r + 1)$ as a basis in the model described below.

A tactic that increases the sperm precedence of the donor should increase the total fraction of sperm that an individual can displace in the sperm stores of the recipient. We characterize this increase using the parameter s, where the sperm displacement function becomes

$$\phi(r, s) = \frac{r(\delta + s)}{r(\delta + s) + 1}. \tag{4}$$

A hermaphrodite with a larger value of s will displace more sperm and thus father more offspring. An s value of zero returns ϕ to the Charnov (1996) formulation. The additive relation between δ and s proposed in equation (4) is the simplest form that captures this interaction. For example, a multiplicative relationship would be reasonable for the large δ and s limit but not for $\delta \to 0$, which corresponds to no sperm displacement.

Our model considers the case where harm to the sperm recipient is a side effect of a tactic that promotes a donor's sperm. An example of this in nature is the increased probability of death for females following mating (Crudgington and Siva-Jothy 2000; Blanckenhorn et al. 2002). In our model, when a harmful mating tactic is employed by a sperm donor, there is an associated probability of death for the sperm recipient. This possible death occurs between mating and laying eggs.

When harmful mating tactics are not used (when $s = 0$), the probability of survival of a sperm recipient is unity. It is expected that an increase in the ability to displace sperm using a harmful mating tactic, s, should result in reduced probability of survival for the sperm recipient. The probability of survival, $\sigma(s)$, is a decreasing function of s. An appropriate function relating the survival probability of sperm recipient to parameter s was selected to be

$$\sigma(s) = \left(\frac{1}{1 + s}\right)^m. \tag{5}$$

The relationship between survival probability and s is tuned by a severity parameter m. Increasing m increases the probability of death for any given value of $s > 0$ (see fig. 1). The parameter m therefore characterizes the severity of the harmful mating tactic, with larger m corresponding to greater severity. An example of this is the acceleration of harm with respect to increased dose observed for a number of different toxins (Bryan and Shimkin 1943; Murphy and Cheever 1968). We chose equation (5) for mathematical convenience, but our results below do not depend on the precise form of the survival probability.

A hermaphrodite's life in our model proceeds in the following way: it mates in male and female function, it experiences a probability of death due to harm it receives during mating, and if it survives it lays eggs. This cycle is repeated a maximum of T times. Consider a rare mutant using the strategy $(\hat{r}, \hat{s})$ in a population using the strategy (r, s). If the maximum number of matings is $T = 2$, the fitness of the rare mutant due to its female function is given by

$$w_f(\hat{r}, \hat{s}, r, s) = \sigma(1 - \hat{r})R + \sigma^2(1 - \hat{r})R. \tag{6}$$

For ease of notation, the survival probabilities are written as $\hat{\sigma} = \sigma(\hat{s})$ and $\sigma = \sigma(s)$. The first term in equation (6) is the product of the probability that the mutant survives the first mating with a wild type to lay eggs and the resource the mutant has allocated to egg production. The second term is the product of the probability that the

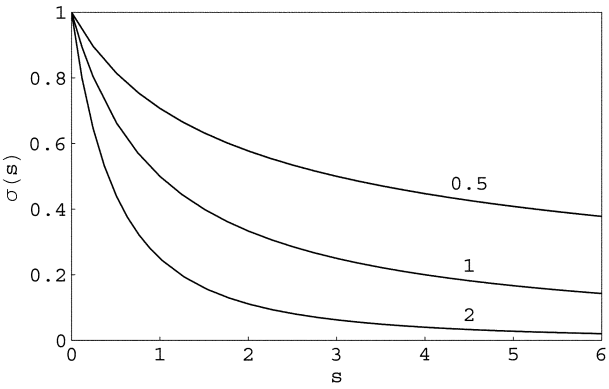

Figure 1: Survival probability of sperm recipient, σ, versus s. The parameter s is a measure of the increased ability to displace sperm in the mating partner's sperm stores (see eq. [4]). Plots are shown for $m = 0.5$, 1, and 2, where m characterizes the severity of the harmful mating tactic (see eq. [5]).

mutant survives the first and second matings with wild types to lay a second clutch of eggs and the resource the female has allocated to egg production.

The fitness from the female function of a rare mutant hermaphrodite using strategy $(\hat{r}, \hat{s})$ in a population with strategy (r, s) is generalized for T matings by the equation

$$w_{\mathrm{f}}(\hat{r}, \hat{s}, r, s) = R(1 - \hat{r}) \sum_{i=1}^{T} \sigma^{i}. \tag{7}$$

The fitness of the same mutant due to its male function, for $T = 2$, is given by

$$w_{\mathrm{m}}(\hat{r}, \hat{s}, r, s) =$$

$$\hat{\sigma}\hat{\phi}R(1 - r) + \hat{\sigma}\sigma\hat{\phi}(1 - \phi)R(1 - r) + \sigma\hat{\sigma}\hat{\phi}R(1 - r), \tag{8}$$

where $\hat{\phi} = \phi(\hat{r}, \hat{s})$ and $\phi = \phi(r, s)$. The first term in equation (8) is from eggs produced by the sperm recipient (wild type) immediately after mating with the mutant. This is a product of the probability that the wild type survives the mating, the fraction of sperm the mutant displaces in the wild type's sperm stores, and the number of eggs produced by the wild type. The second term in equation (8) is from offspring produced by the wild type after it engages in a second mating with another wild type. Thus, in order to lay eggs, the wild type needs to have survived its first and second matings. The second mating will leave a fraction $\phi(\hat{r})[1 - \phi(r)]$ of the mutant's sperm in the wild type's sperm stores. The third term in equation (8) is from the second mating of the mutant. The mutant needs to have survived its first mating in female function in order to engage in a second mating. Thus, the third term is equal to the first term multiplied by the probability that the mutant lives to mate a second time. Following the models of Charnov (1996) and Greeff and Michiels (1999), we approximate that there is sperm stored at the first mating. This approximation should be accurate for large numbers of matings or large values of δ, where mating with an individual with no sperm stored becomes less significant. For small numbers of matings and small values of δ, the approximation becomes less accurate; the impact of this is discussed later.

The fitness from the male function of a rare mutant using strategy $(\hat{r}, \hat{s})$ in a population using strategy (r, s) is generalized for T matings by the equation

$$w_{\mathrm{m}}(\hat{r}, \hat{s}, r, s) = R(1 - r)\hat{\phi}\hat{\sigma} \sum_{i=1}^{T} \left[\sigma^{i-1} \sum_{j=0}^{T-i} \sigma^{j}(1 - \phi)^{j} \right]. \tag{9}$$

The second sum in equation (9) is over the mutant's mating partner going on to mate $T - i$ times with others in the population, weighted by the probability that the mating partner survives to take part in the jth mating. The first sum in equation (9) sums the T matings of the rare mutant in question, weighted by the probability that the mutant survives to take part in the ith mating. Equation (9) can be summed as a geometric series.

Notice that in equations (7) and (9), fertilized egg production is not limited by ability to get sperm but by resource allocated to eggs. Also, male reproductive success is limited by ability to gain access to eggs (these are the assumptions behind Bateman's principle; Bateman 1948; Charnov 1979). A consequence of these assumptions is that if there is no competing sperm, males can fertilize eggs with vanishingly small amounts of sperm. Under these conditions, this can act as a selection pressure for unrealistically low allocation to male function. This assumption was also used by Charnov (1996) and Greeff and Michiels (1999).

The total fitness $W(\hat{r}, \hat{s}, r, s)$ of a mutant individual using strategy $(\hat{r}, \hat{s})$ in a population using strategy (r, s) is given by the sum of its fitness from male function and female function (eqq. [7], [9]), $W = w_{\mathrm{f}} + w_{\mathrm{m}}$.

Candidate ESSs were determined by solving the equilibrium equations (10) and (11) simultaneously subject to the constraints $0 \le r \le 1$ and $s \ge 0$. These equations were solved numerically using Mathematica. These candidates were then tested for ESS and convergent stability (Eshel 1983; Abrams et al. 1993; Brown et al. 2007):

$$\left. \frac{\partial W}{\partial \hat{r}} \right|_{\hat{s}=s, \hat{r}=r} = 0, \tag{10}$$

$$\left. \frac{\partial W}{\partial \hat{s}} \right|_{\hat{s}=s, \hat{r}=r} = 0. \tag{11}$$

Results and Discussion

The stability of harmful mating tactics was investigated by varying the parameters T (number of matings), δ (measure of ability to displace sperm without harmful mating tactic; see eq. [2]), and m (severity of harmful mating tactic). Our analysis showed three possible outcomes: not using harmful mating tactics is the ESS, using harmful mating tactics is the ESS, and there is no viable ESS. A phase diagram for these outcomes is plotted in figure 2.

In the region of figure 2 marked "no harm," there is a unique boundary ESS ($r^* > 0$, $s^* = 0$) that also shows convergent stability. Harmful mating tactics cannot evolve

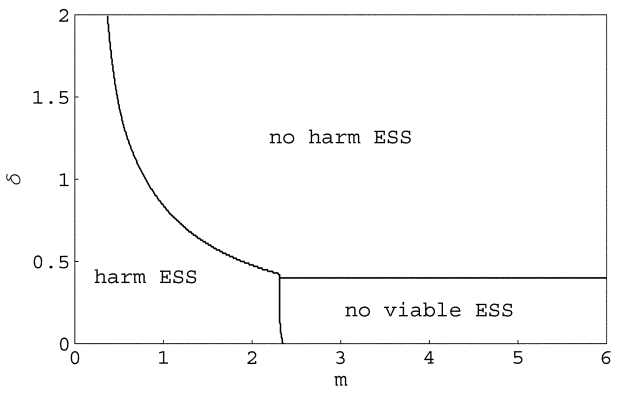

Figure 2: For number of matings $T = 4$, the ESS phase diagram for parameters m, the severity of the harmful mating tactic, and δ, a measure of the ability to displace sperm in the mating partner's sperm stores when harmful mating tactics are not used (see eq. [2]). In the region marked "no harm," not using harmful mating tactics is the ESS ($r^* > 0$, $s^* = 0$). In the region marked "harm," using harmful mating tactics is the ESS ($r^* > 0$, $s^* > 0$). In the region marked "no viable ESS," $r^* \to 0$, so sperm competition ceases.

in this region. In the region of figure 2 marked "harm," boundary points ($s = 0$) are not an ESS; however, there exists a unique ESS ($r^* > 0$, $s^* > 0$) that shows convergent stability. In this region, rare mutants that use harmful mating tactics will always invade a resident population where harm is absent. One possible biological consequence of these inferences is that polymorphism for "harm"/"no harm" is an unstable condition and so will tend not to persist.

Following mating with an individual using the ESS, s^*, the probability an individual survives, $\sigma(s^*)$, can be found by substituting the ESS s^* and the model parameter m into equation (5). Figure 3 plots the survival probability per mating in a population using the ESS, $\sigma(s^*)$, as a function of the model parameter δ for two different values of T, the parameter denoting the maximum number of matings. The survival probability is unity when "no harm" is an ESS ($s^* = 0$). When $\delta \sim 0.8$, there are discontinuities in the curves and survival probability drops rapidly. This is because the use of a harmful mating tactic is now an ESS ($s^* > 0$). As δ decreases, a hermaphrodite's ability to displace sperm without the use of the harmful mating tactic decreases. It then becomes necessary for a hermaphrodite to escalate the use of the harmful mating tactic to increase sperm displacement. This occurs at the expense of its mating partner's probability of survival. An interesting outcome is that the parameter T has little effect on the ESS level of harmful mating tactic, s^*, possibly because the mean life span, given by $[1 - \sigma(s)]^{-1}$, does not allow most individuals to complete T matings when harmful mating tactics are present.

For finite numbers of matings, Greeff and Michiels (1999) observed that decreasing δ resulted in an increased resource allocation to male function r^*. They noticed an exception for small δ and T when $r^* \to 0$. This was attributed to the poor ability to displace rival sperm from the receiver's sperm storage organ. The ESS resource allocation (r^*) from our analysis is plotted as a function of δ in figure 4. A discontinuity is again observed when $\delta \sim 0.8$ and coincides with s^* becoming greater than zero. It was observed that as δ tends to zero, the ESS resource allocation for different T converges. This coincides with similar values of s^* for different T and with the mean life span of individuals becoming shorter than that required to complete T matings.

Figure 4 shows that harmful mating tactics should lead to a distinctly female-biased resource allocation strategy, contrasting markedly with the conclusions of Greeff and Michiels (1999), who found that for large numbers of matings T and small δ, the resources allocated between male and female function tend to approach equality ($r^* \to 0.5$). One of the main conclusions of their article may therefore only apply to hermaphrodites where harmful mating tactics are absent. This contrast can be understood by looking at what happens to the ability to displace sperm, measured by $\delta + s^*$, when the use of harmful mating tactics becomes stable. Once the use of harmful mating tactics become stable, decreasing the parameter δ actually results in an increase in the potential amount of sperm that can be displaced, $\delta + s^*$. The intuitive explanation for this is that if sperm displacement is made more efficient by harmful mating tactics, then the ESS returns to a more

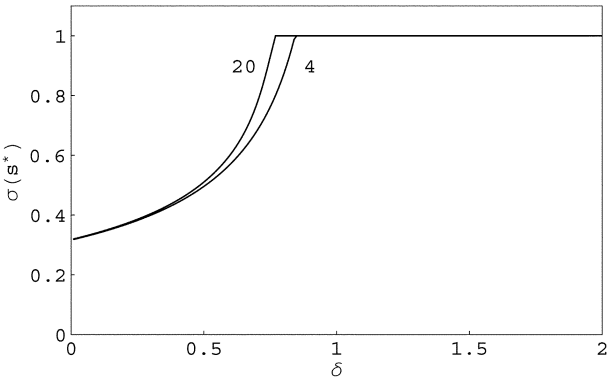

Figure 3: Survival probability in a population using the ESS level of harm, $\sigma(s^*)$, versus δ, a measure of the ability to displace sperm in the mating partner's sperm stores when harmful mating tactics are not used (see eq. [2]). Plots are shown for number of matings $T = 4$ and 20. For all plots, the severity parameter of the harmful mating tactic is $m = 1$.

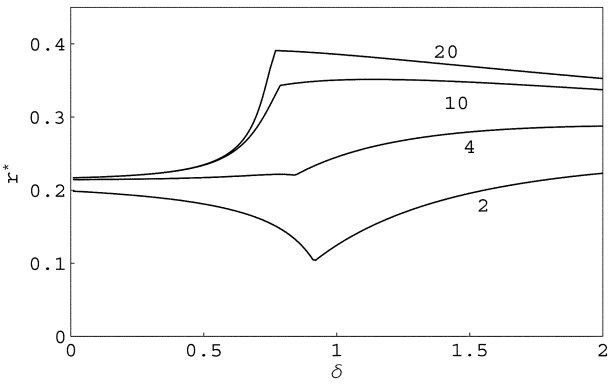

Figure 4: ESS resource allocation, r^*, versus δ, a measure of the ability to displace sperm in the mating partner's sperm stores when harmful mating tactics are not used (see eq. [2]). Plots are shown for number of matings $T = 2$, 4, 10, and 20. For all plots, the severity parameter of the harmful mating tactic is $m = 1$.

female-biased resource allocation strategy. In addition, increased mortality rates due to harmful mating should reduce r^*, as mortality decreases the return from male function (Charnov 1996).

Calculating the sperm displacement function $\phi(\delta, r^*, s^*)$ from the ESS values r^* and s^* for a given set of model parameters (δ, T, m) gives the fraction of offspring fathered by the last male mating partner. Close to the boundary between "harm" and "no harm" in figure 2, $\phi(\delta, r^*, s^*)$ is typically around 0.1–0.3, marking the largest values for which harmful mating can invade a population. Deep inside the region where harmful mating tactics are an ESS (small m, small δ), $\phi(\delta, r^*, s^*)$ increased to values of 0.3–0.6. This may therefore indicate that harmful mating tactics are likely to initially evolve in populations where the first mate gains the most fertilization but can lead to last-male sperm precedence. Decreasing the severity parameter of the harmful mating tactic (decreasing m) was found to have a marked influence on sperm displacement, following the invasion of harmful mating tactics (fig. 5). In comparison, the parameter T was found to have a very small effect, with large T resulting in larger values of $\phi(\delta, r^*, s^*)$ (not shown). This is a result of r^* and s^* not varying much with T, as explained above.

If ability to displace sperm in a partner is low (if $\delta + s$ is small), many future egg-laying events are required by a sperm donor's partner to compensate for this high investment; otherwise, investment in the female function would yield higher fitness. However, if the subsequent number of egg-laying events is not sufficient to make investment in the male function profitable, the ESS for resource allocation to male function can tend to zero. If this occurs, then it follows that $\phi(\delta, r^*, s^*)$ tends to zero; thus, sperm competition ceases. This unrealistically low allocation to male function is a consequence of the approximations used in constructing our model. When displacing rival sperm is difficult (when δ is small) and the number of matings is small, fitness due to mating with an individual with no sperm stored should become more significant. Our model approximates that there is sperm stored at the first mating and that production of fertilized eggs is not limited by availability of sperm. Thus, when T and $\delta + s$ are both small, the individual is better off investing all in female function and $r^* \to 0$. This marks the limit in the validity of our approximations.

The ESS for resource allocated to the male function tends to zero on the boundary of the "no viable ESS" region (fig. 2) and is equal to zero inside this region. This is because the return in paternity due to a finite number of matings, T, is insufficient, and a hermaphrodite is better off investing all in female function. Increasing T shifts the boundary of the "no viable ESS" region in figure 2 to lower values of δ and higher values of m. In the limit that T tends to infinity, the "no viable ESS" region disappears for a plot equivalent to figure 2. The limit $r^* \to 0$ was also observed in figure 2 of Greeff and Michiels (1999). They observed that for finite T, resource allocation to male function tends to zero for small δ and small T. In the limit that T tends to infinity (Charnov 1996), r^* tends to 0.5 for small δ.

The fitness of a population using a particular ESS strategy (r^*, s^*) can be calculated by substituting $\hat{r} = r = r^*$ and $\hat{s} = s = s^*$ into the equation for total fitness, W. This gives the fitness in units of the resource available per individual per mating, R. If a species is competing with other species for the same resource under density-dependent

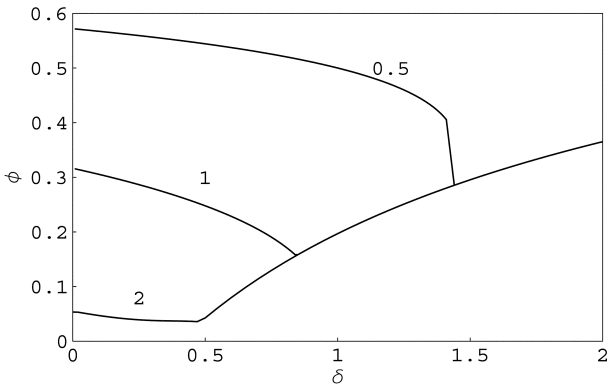

Figure 5: ESS sperm displacement, $\phi(\delta, r^*, s^*)$, versus δ, a measure of the ability to displace sperm in the mating partner's sperm stores when harmful mating tactics are not used (see eq. [2]). Plots are shown for the severity parameter of harmful mating tactic $m = 0.5$, 1, and 2. For all plots, the number of matings is $T = 4$.

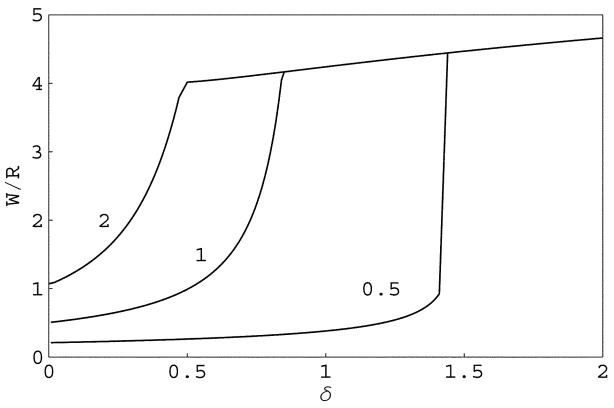

Figure 6: Fitness (W) calculated for resident population at ESS, (r^*, s^*), versus δ, a measure of the ability to displace sperm in the mating partner's sperm stores when harmful mating tactics are not used (see eq. [2]). Fitness is in units of resource per mating, R. Plots are shown for the severity parameter of harmful mating tactic $m = 0.5$, 1, and 2. For all plots, the number of matings is $T = 4$.

selection, then the growth rate of each will ultimately determine success or extinction.

Our model assumes the number of eggs to be the limiting factor in population growth; thus, greater allocation to female function would be expected to increase the fitness of the population. Perhaps surprisingly, the model instead shows that harmful mating tactics more than countercompensate. Thus, although resource allocation may be female biased, this heavy bias makes little difference to the overall fitness of a population once harm invades, especially when δ is small or, similarly, when the severity parameter for the harmful mating tactic is low ($m < 0.5$; fig. 6). Nonetheless, the fitness per mating when harm is present is considerably less than when harm is absent (fig. 6). In consequence, species that compete under density-dependent selection may be at a disadvantage when harmful mating tactics are present.

Conclusions

The majority of the inferences from the model are reassuring in that they are in keeping with what might be expected based on intuition. We found that it is likely that harmful mating in hermaphrodites is associated with species in which sperm precedence strongly favors the first mate. Our model predicts that this criterion becomes less important as harmful mating tactics become more efficient. This was explicitly pointed out by Arnqvist and Rowe (2005) and Morrow et al. (2003). Harmful mating tactics enable the sperm donor to increase its sperm precedence and can lead to last-male sperm precedence for very efficient harmful mating tactics.

In contrast to the conclusions of previous work that did not include harmful mating (Greeff and Michiels 1999), the model indicates that when harm is also considered, hermaphrodites may again return to a female-biased resource allocation strategy, largely because sperm displacement is made more efficient by harmful tactics. Perhaps the most surprising inference is that harmful mating tactics more than countercompensate in these circumstances, leading to a reduced fitness. Hermaphrodites that use harmful mating tactics may therefore be at a disadvantage when competing with other species for a limited resource.

Acknowledgments

We would like to thank the Royal Society, the Engineering and Physical Sciences Research Council, and the University of Nottingham for financial support.

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