Motherly love curbs harm: maternal effects can modulate sexual conflict

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Abstract

Strong sexual selection frequently favours males that increase their reproductive success by harming females, with potentially negative consequences for the growth of populations. Understanding what factors may resolve this reproductive "tragedy of the commons" is a key question in evolutionary biology. Studies addressing the evolution of sexual conflict have so far considered direct effects on male and female reproductive success along with indirect genetic benefits (e.g. good genes) to females. Here, we model the evolution of male harm while incorporating male-induced maternal effects on offspring quality. We show that maternal effects can partially align male and female evolutionary interests, fostering cooperation between the sexes and significantly reducing optimal levels of male harm. This finding fits broadly with available evidence across the tree of life, opening a novel avenue to understand the evolution of sexual conflict.

Introduction

Strong sexual selection frequently leads to scenarios where male and female evolutionary interests misalign – known in the literature as sexual conflict[1]. This, in turn, can trigger sexually antagonistic coevolution[2,3] where sexual strategies in one sex evolve to counteract those of the opposite sex. Sexually antagonistic coevolution is currently recognized as one of the key evolutionary processes shaping male and female adaptations and life-history traits[4]. At a population level, it frequently leads to adaptations in males that harm females[5,6] and reduce population growth, in a process akin to "the tragedy of the commons"[7]. From male harassment and coercion[8,9] to toxic ejaculates[10] and traumatic insemination[11,12] , harmful male adaptations are both widespread across the tree of life and extraordinarily diverse in the levels of harm they inflict on females, and thus in their potential consequences for population viability. A current priority in evolutionary biology is to identify factors that modulate sexual conflict and explain the diversity of male harm adaptations observed in nature. For example, recent research shows that, by aligning the interests of males and females, kin selection has the potential to modulate the evolution of male harm to females[13–16]. We posit here that male harm can induce maternal effects that reduce the quality of a harming male's own offspring and, in doing so, bring together male and female interests and abate sexual conflict.

Studies seeking to explain the evolution of antagonistic or harmful male adaptations have focused on direct costs (to females) and benefits (to males), as well as the potential indirect genetic benefits to females through their male offspring[17–22]. On the one hand, manipulative or harmful traits allow males to sire a greater proportion of a female's offspring at the expense of that female's overall fecundity. On the other hand, females may obtain indirect genetic benefits by mating with particularly harmful or manipulative males because their own male offspring will inherit these genes, albeit theoretical and empirical evidence shows indirect genetic benefits are generally weaker than direct benefits[17–19]. We contend male-induced maternal effects are an overlooked yet potentially important factor in the fitness payoff of male harm evolution.

Maternal effects can drastically modulate offspring quality[23] and are largely mediated by maternal condition[24,25]. In turn, male harm can severely impact female condition[4] and, although its transgenerational effects have only been studied in a handful of species, it can induce maternal effects that reduce the quality of a male's own offspring[26–31]. For example, female guppies (*Poecilia reticulata*) exposed to greater harassment produce smaller daughters and sons with shorter gonopodia[30]. We suggest this type of effects will reduce the

fitness returns of male harm to females because any fitness gains in terms of direct reproductive success (i.e. number of offspring sired vs. non-harmful males) will be partially cancelled-out by maternally-induced effects on offspring quality (i.e. decrease in mean offspring quality vs. non-harmful males). To formally test this hypothesis, we use a personal-fitness kin-selection approach[32–34] to model optimal levels of male harm to females in three contexts (see methods for details): a) in the absence of maternal effects on offspring quality[35], b) in the presence of maternal effects on the fecundity of offspring, and c) in the presence of maternal effects on offspring's ability to inflict (males) or resist (females) harm (i.e. sexual selection quality).

Results and discussion

We find that maternal effects reduce the optimal level of male harm, especially when harm curtails offspring quality during sexual selection (Figure 1). Importantly, these results are consistent across different levels of dispersal (Figure 1; see methods for details). As such, while kin selection can still shape the level of harm under each one of the different models, the reduction of sexual conflict through induced maternal effects is independent of kin selection. Differences in the optimal level of male harm across different populations are therefore not only predicted to reflect demographic differences leading to kin selection but also differences in how male harm impacts offspring quality. For example, differences in harm may arise due to intra-specific differences in local ecological conditions that may compromise female condition, making it more vulnerable to male harm (e.g. food availability), or due to inter-specific differences in the importance of maternal effects across taxa. Generally, our model predicts that sexual conflict via male harm will be disfavoured whenever harm induces maternal effects on offspring quality, in a manner that is proportional to these effects. We thus identify male harm-mediated maternal effects as a previously unrecognized factor shaping sexual conflict evolution, a realization that may have far-reaching taxonomic implications.

The overarching prediction that stems from our results is that, all else being equal, we might expect lower levels of male harm to females in taxa where maternal effects on offspring quality are higher, more amenable to changes in maternal condition, and/or in which offspring quality loads heavily on parental fitness. Specifically, we would predict generally lower levels of male harm in species with prolonged gestation (e.g. viviparous/ovoviviparous vs. oviparous), in species with extended maternal provisioning (e.g. matrotrophic vs. lecithotrophic), in species with (vs. without) parental care, and at large in

species that are under strong K- (vs. r-) selection. Identifying maternal effects as a potential modulator of sexual conflict thus gives rise to specific predictions, about where male harm might have evolved and how intense we might expect it to be, that are in principle applicable across a wide range of taxa. Available evidence, collected following a systematic literature search (see methods for details), seems to fit reasonably well with these predictions (Figure 2). We found male harm to be particularly widespread, intense and sophisticated in insects, which include the best-known cases of sexually antagonistic coevolution driven by male harm[36] along with many instances of traumatic insemination[11,12,37–39] , toxic ejaculates[10] and extreme coercion[9]. Furthermore, indirect evidence based on the description of male genitalia (and the fitness consequences of similar structures in other species) suggests adaptations for traumatic insemination may occur in as many as 1425 species more (Figure 2B; see supplementary materials for details). In accordance to predictions, insects are typically under strong r-selection, oviparous and normally lack extended maternal provision and parental care (Figure 2A and supplementary materials). Gastropods, where traumatic insemination also seems common, follow a very similar pattern.

In contrast, male harm appears to be relatively rare or weak in vertebrates, especially so in taxa with widespread parental care and prolonged gestation such as birds and mammals (Figure 2A). As a matter of fact, well-studied cases of male harm reported so far in vertebrates consist exclusively in collateral damage to females (i.e. harassment and/or coercive matings), as opposed to adaptations for harming females *per se*[40] (i.e. direct damage). The absence of adaptations for direct harm in mammals is perhaps particularly salient given the strength of male-male competition in many species within this group[1]. Furthermore, although harassment is widely interpreted as an inherently costly male phenotype for females, it does not necessarily translate into a reduction in female fitness. For example, female resistance to male harassment has been suggested to participate in mate choice as a way of screening high quality males[41]. Thus, the mere existence of male harassment and/or coercion is far from a litmus test for male harm. Different forms of sexual harassment and/or coercion to females have been reported for a number of vertebrates (and are probably common[42]), but direct evidence that such harassment reduces female fitness is limited[43–45]. In short, there does seem to be a relationship between the overall scope for maternal effects within broad taxonomic groups and reports of male harm. This is of course tentative at this stage as phylogenetic signal within groups will be strong due to other factors. Looking specifically at the 73 species for which we found good evidence of male harm to

females across all taxa, we found little scope for maternal effects in cases of indirect harm (harassment) and less so in cases of direct harm (Figure 2C; see supplementary material for details), which again seems to echo our predictions.

To conclude, we bring attention to male-induced maternal effects as a factor in the evolution of sexual conflict. Similarly to relatedness in kin-selection models[13–16,35] , in this study we show that maternal effects can bring together the interests of males and females and abate conflict over sexual strategies. We contend that such effects are likely to be important to understand sexual conflict evolution in nature for two main reasons. First, due to the existence of substantial variation in condition-dependent maternal effects that can impinge on offspring quality, both across and within taxa[46]. Second, due to the well-established fact that male harm can have a dramatic impact on female condition[4]. Available evidence seems consonant with the idea that maternal effects may have at least partly modulated the evolution of male harm at a broad taxonomic level. We suggest future empirical and comparative studies should aim to test predictions arising from the interplay between maternal effects and male harm, an exciting novel research avenue that could significantly further our understanding of sexual conflict.

Methods

1.1 Model without maternal effects

We consider an infinite diploid population divided into patches[47] containing n_f females and n_m males. We follow the approach developed by Faria et al.[35]. Specifically, males invest in a harming trait that increases their personal reproductive success relative to other males but reduces the overall fecundity of the females in the patch. Each male's reproductive success is directly proportional to his competitiveness for mating success and inversely proportional to the average competitiveness for mating success of the males in his patch. Accordingly, a focal female's fecundity is a function of the average level of male harm present in her patch $f_f(Y) = 1 - Y$ and a focal male's competitiveness for mating success is a function of the level of harm expressed by that focal male $f_m(y) = 1 + y$, where: y is the level of harm expressed by the focal male; and Y is the average level of harm present in the focal patch. Mating follows, and each female produces a large number of offspring with an even sex ratio and in direct proportion to her fecundity. Adults then die, and juvenile females disperse with probability d_f and juvenile males disperse with probability d_m. Following dispersal, n_f females and n_m males survive at random within each patch to adulthood, returning the population to the beginning of the lifecycle.

Natural selection – Natural selection favours any gene associated with greater individual relative fitness[48,49]. Assuming vanishingly little genetic variation, this condition many be expressed using the mathematics of differential calculus: $dW/dg > 0$, where g is the genic value of a gene picked at random from the population and W is the relative fitness of the individual carrying this gene[50]. The appropriate measure of relative fitness is a class-reproductive-value-weighted average taken across females and males, i.e. $W = ½W_f + ½W_m$, where W_f is the relative fitness of the female carrying the gene and W_m is the relative fitness of the male carrying the gene[34,50]. Female's relative fitness in the context of the present model is given by:

$$W_f = f_f(Y)\left(\frac{1}{2}\left(\frac{1-d_f}{(1-d_f)f_f(Y)+d_f f_f(\bar{Y})}+\frac{d_f}{f_f(\bar{Y})}\right)+\frac{1}{2}\left(\frac{1-d_m}{(1-d_m)f_f(Y)+d_m f_f(\bar{Y})}+\frac{d_m}{f_f(\bar{Y})}\right)\right), \quad \text{(A1)}$$

where $f_f(\bar{Y}) = 1 - \bar{Y}$ is the average fecundity of females in the population, which is a function of the average level of harm present in the population ($\bar{Y}$). Male's relative fitness in the context of the present model is given by:

$$W_m = \frac{f_m(y)}{f_m(Y)} f_f(Y)\left(\frac{1}{2}\left(\frac{1-d_f}{(1-d_f)f_f(Y)+d_f f_f(\bar{Y})}+\frac{d_f}{f_f(\bar{Y})}\right)+\frac{1}{2}\left(\frac{1-d_m}{(1-d_m)f_f(Y)+d_m f_f(\bar{Y})}+\frac{d_m}{f_f(\bar{Y})}\right)\right), \quad \text{(A2)}$$

where $f_m(Y) = 1 + Y$ is the average competitiveness for mating success of local males which is a function of the average level of male harm in the patch (Y). Following the approach of Taylor & Frank[34] for a class-structured population, we may write $dW/dg = ½\,(dW_f/dg_f) + ½\,(dW_m/dg_m) = ½\,((\partial W_f/\partial y)(dy/dG_f)(dG_f/dg_f) + (\partial W_f/\partial Y)(dY/dG_f')(dG_f'/dg_f) + (\partial W_f/\partial Y)(dY/dG_m')(dG_m'/dg_f)) + ½\,((\partial W_m/\partial y)(dy/dG_m)(dG_m/dg_m) + (\partial W_m/\partial Y)(dY/dG_m')(dG_m'/dg_m) + (\partial W_m/\partial Y)(dY/dG_f')(dG_f'/dg_m))$, where: g_f is the genic value of a gene picked at random from a female in the population; g_m is the genic value of a gene picked at random from a male in the population; G_f is the focal female's breeding value; G_f' is the average breeding value of local females; G_m is the focal male's breeding value; G_m' is the average breeding value of local males; $dy/dG_f = dY/dG_f' = \gamma_f$ is the mapping between genotype and phenotype in the females; $dG_f/dg_f = p_f$ is the consanguinity of the gene in the focal female to the female herself; $dG_f'/dg_f = p_{ff}$ is the consanguinity of the gene in the focal female with a randomly-chosen local female; $dG_m'/dg_f = p_{fm}$ is the consanguinity of the gene

in the focal female with a randomly-chosen local male; $dy/dG_m = dY/dG_m' = \gamma_m$ is the mapping between genotype and phenotype in the males; $dG_m/dg_m = p_m$ is the consanguinity of the gene in the focal male to the male himself; $dG_m'/dg_m = p_{mm}$ is the consanguinity of the gene in the focal male with a randomly-chosen local male; and $dG_f'/dg_m = p_{mf}$ is the consanguinity of the gene in the focal male with a randomly-chosen local female. The consanguinity between a gene to its carrier is the same no matter the sex that we are considering and, therefore, $p_f = p_m = p$. We divide all the terms of the right side of the equation by p to get the kin-selection coefficient of relatedness (see below[51]).

We assume that harm is only expressed by the males and that their genes are in full control of the phenotype. Accordingly, $\gamma_f = 0$ and $\gamma_m = 1$. Therefore, natural selection favours an increase in the level of harm that males express when:

$$\frac{1}{2}\left(\frac{\partial W_f}{\partial Y} r_{fm}\right) + \frac{1}{2}\left(\frac{\partial W_m}{\partial y} + \frac{\partial W_m}{\partial Y} r_{mm}\right) > 0, \quad \text{(A3)}$$

where: r_{fm} is the relatedness between a focal female with a randomly-chosen local male; r_{mm} is the relatedness between a focal male with a randomly-chosen local male; the derivatives are evaluated at $y = Y = \bar{Y} = \bar{Y}^*$; and $\bar{Y}^*$ is the optimal level of harm in the population. Setting the left-side of the equation equal to 0 and calculating the derivatives yields the marginal fitness equation for this model:

$$\frac{1-r_{mm}}{1+\bar{Y}^*} - \frac{1}{1-\bar{Y}^*}(r_{fm} + r_{mm})(1 - a) = 0, \quad \text{(A4)}$$

where $a = ((1 - d_f)^2 + (1 - d_m)^2)/2$.

Relatedness – The relatedness between a genic actor in the focal female with a randomly-chosen male in her patch is approximately given by:

$$r_{fm} = (1 - d_f)(1 - d_m)r, \quad \text{(A5)}$$

and they are only related if they are both local $(1 - d_f)(1 - d_m)$ and, if so, their relatedness is given by the relatedness through the genic actor between two different juveniles born in the

same patch (r). The relatedness between a genic actor in the focal male and a randomly-chosen male in his patch (including the focal male himself):

$$r_{\mathrm{mm}} = \frac{1}{n_{\mathrm{m}}} + \frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}}(1-d_{\mathrm{m}})^2 r, \qquad \text{(A6)}$$

where: with probability $1/n_{\mathrm{m}}$ the randomly-chosen male is the focal male himself, in which case relatedness is 1; and with probability $(n_{\mathrm{m}} - 1)/n_{\mathrm{m}}$ is a different male, in which case they are only related if they are both locals $(1 - d_{\mathrm{m}})^2$ and, if so, their relatedness is given by the relatedness through the genic actor between two different juveniles born in the same patch (r).

Relatedness through the genic actor between two different juveniles born in the same patch is then given by $r = p'/p$, where p' is the consanguinity through the genic actor between two individuals born in the same patch and is defined by picking the genic actor from the focal individual and a random gene from the other individual and calculating the probability that the two are identical by descent[51]. Assuming that consanguinities are at their neutral-equilibrium values, appropriate if selection is weak[52], we write:

$$p' = \frac{1}{4}\left(\frac{1}{n_{\mathrm{f}}}p + \frac{n_{\mathrm{f}}-1}{n_{\mathrm{f}}}(1-d_{\mathrm{f}})^2 p'\right) + \frac{1}{4}\left(\frac{1}{n_{\mathrm{m}}}p + \frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}}(1-d_{\mathrm{m}})^2 p'\right) + \frac{1}{2}(1-d_{\mathrm{f}})(1-d_{\mathrm{m}})p', \qquad \text{(A7)}$$

where: with probability of ¼ we may have drawn the maternal-origin genes from both individuals, in which case with probability of $1/n_{\mathrm{f}}$ they share the same mother (and they have consanguinity of p) and with probability of $(n_{\mathrm{f}} - 1)/n_{\mathrm{f}}$ they have different mothers (and they will only have consanguinity if both mothers are local, giving a consanguinity of $(1 - d_{\mathrm{f}})^2 p'$); with probability of ¼ we may have drawn the paternal-origin genes from both individuals, in which case with probability of $1/n_{\mathrm{m}}$ they share the same father (and they have consanguinity of p) and with probability of $(n_{\mathrm{m}} - 1)/n_{\mathrm{m}}$ they have different fathers (and they will only have consanguinity if both fathers are local, giving a consanguinity of $(1 - d_{\mathrm{m}})^2 p'$); and with probability ½ we have drawn the maternal-origin gene from one and the paternal-origin gene from the other and they will only have consanguinity if both these parents are local (giving a consanguinity of $(1 - d_{\mathrm{f}})(1 - d_{\mathrm{m}})p'$). Rearranging, we get:

$$p' = \frac{n_f + n_m}{(1-d_m)^2 n_f + (1-d_f)^2 n_m + (4-d_f-d_m)(d_f+d_m) n_f n_m} p, \quad \text{(A8)}$$

and the relatedness between two random individuals born in the same patch is then given by $r = p'/p$ (Bulmer 1994). Rearranging, we obtain:

$$r = \frac{n_f + n_m}{(1-d_m)^2 n_f + (1-d_f)^2 n_m + (4-d_f-d_m)(d_f+d_m) n_f n_m}. \quad \text{(A9)}$$

We can replace equation (A9) into the equations (A5–A6) to obtain the different coefficients of relatedness. All of these approximations become exact in the limit of vanishingly weak selection[52].

Optimal level of harm – The optimal level of harm can now be calculated by solving equation (A4) to $\bar{Y}^*$:

$$\bar{Y}^* = \frac{1 - r_{fm}(1-a) - r_{mm}(2-a)}{1 + r_{fm}(1-a) - r_{mm} a}. \quad \text{(A10)}$$

Replacing the relatedness coefficients into equation (A10) then results in the optimal level of harm represented in Figure 1 of the main text (no maternal effects line).

1.2 Model with maternal effects on fecundity

As in section 1.1, we consider an infinite diploid population divided into patches[47] containing n_f females and n_m males. We now assume that there are two types of individuals: low-quality individuals; and high-quality individuals. Specifically, low-quality individuals are produced in proportion to the harm that their mother received. Low-quality individuals have a lower fecundity or lower competitiveness for mating success when compared to high-quality individuals. As before, males invest in a harming trait, with its effect on female's fecundity and male's competitiveness for mating success being the same as in the previous section. Accordingly, a focal low-quality female's fecundity is a function of the average level of male harm present in her patch minus a cost s due to her low quality $f_{lf}(Y) = 1 - Y - s$, a focal low-quality male's competitiveness for mating success is a function of the level of harm expressed by that focal male minus a cost s due to his low quality $f_{lm}(y) = 1 + y - s$, a focal high-quality female's fecundity is a function of the average level of male harm present in her

patch $f_{hf}(Y) = 1 - Y$, and a focal high-quality male's competitiveness for mating success is a function of the level of harm expressed by that focal male $f_{hm}(y) = 1 + y$. Mating follows, and each female produces a large number of offspring with an even sex ratio and in direct proportion to her fecundity. Adults then die, and juvenile females disperse with probability d_f and juvenile males disperse with probability d_m. Following dispersal, n_f females and n_m males survive at random within each patch to adulthood, returning the population to the beginning of the lifecycle.

Natural selection – We now follow the same approach as in the section 1.1. Female's relative fitness in the context of the present model is given by:

$$W_f = \bar{f}_f(Y)\left(\frac{1}{2}\left(\frac{1-d_f}{(1-d_f)\bar{f}_f(Y)+d_f\bar{f}_f(\bar{Y})}+\frac{d_f}{\bar{f}_f(\bar{Y})}\right)+\frac{1}{2}\left(\frac{1-d_m}{(1-d_m)\bar{f}_f(Y)+d_m\bar{f}_f(\bar{Y})}+\frac{d_m}{f_f(\bar{Y})}\right)\right), \quad \text{(A11)}$$

where: $\bar{f}_f(Y) = (1 - Y')f_{hf}(Y) + Y'f_{lf}(Y)$; $\bar{f}_f(\bar{Y}) = (1 - \bar{Y}')f_{hf}(\bar{Y}) + \bar{Y}'f_{lf}(\bar{Y})$; Y' is the average level of harm that the mother of the average individual in the focal patch was exposed to; and $\bar{Y}'$ is the average level of harm that the mother of the average individual in the population was exposed to. Accordingly, low-quality females are produced with probability Y' and $\bar{Y}'$, depending if it is the average female in the focal patch or the average female in the population, and high-quality females are produced with probability $(1 - Y')$ and $(1 - \bar{Y}')$, again depending if it is the average female in the focal patch or the average female in the population. Male's relative fitness in the context of the present model is given by:

$$W_m = \frac{\bar{f}_m(y)}{\bar{f}_m(Y)}\bar{f}_f(Y)\left(\frac{1}{2}\left(\frac{1-d_f}{(1-d_f)\bar{f}_f(Y)+d_f\bar{f}_f(\bar{Y})}+\frac{d_f}{\bar{f}_f(\bar{Y})}\right)+\frac{1}{2}\left(\frac{1-d_m}{(1-d_m)\bar{f}_f(Y)+d_m\bar{f}_f(\bar{Y})}+\frac{d_m}{\bar{f}_f(\bar{Y})}\right)\right), \quad \text{(A12)}$$

where: $\bar{f}_m(y) = (1 - Y')f_{hm}(y) + Y'f_{lm}(y)$; and $\bar{f}_f(Y) = (1 - Y')f_{hm}(Y) + Y'f_{lm}(Y)$. Using the same logic as above, low-quality males are produced with probability Y', and high-quality males are produced with probability $(1 - Y')$. We now follow the same procedure as in section 1.1. Accordingly, evaluating the derivatives of the inequality (A3) at at $y = Y = Y' = \bar{Y} = \bar{Y}' = \bar{Y}^*$ while using the fitness equations (A11–12) and setting the left-side of the inequality to 0 yields the marginal fitness equation for this model:

$$\frac{1-r_{\mathrm{mm}}}{1+\bar{Y}^*(1-s)} - \frac{1}{1-\bar{Y}^*(1+s)}(r_{\mathrm{fm}} + r_{\mathrm{mm}})(1-a) = 0. \quad \text{(A13)}$$

Optimal level of harm – The optimal level of harm can now be calculated by solving equation (A13) to $\bar{Y}^*$:

$$\bar{Y}^* = \frac{1-r_{\mathrm{fm}}(1-a)-r_{\mathrm{mm}}(2-a)}{1+r_{\mathrm{fm}}(1-a)(1-s)-r_{\mathrm{mm}}a(1-s)-s(2r_{\mathrm{mm}}-1)}. \quad \text{(A14)}$$

We are assuming a similar population structure as in section 1.1, meaning that we can still use the coefficients of relatedness represented by equations (A5–6) into equation (A14). This then results in the optimal level of harm represented in Figure 1 of the main text (maternal effects on fecundity line).

1.3 Model with maternal effects on sexual traits

As in section 1.1 and 1.2, we consider an infinite diploid population divided into patches[47] containing n_{f} females and n_{m} males. We assume that there are two types of individuals: low-quality individuals; and high-quality individuals. As in section 1.2, low-quality individuals are produced in proportion to the harm that their mother received. Now, however, low-quality individuals differ from high-quality individuals on their sexual traits. That is, low-quality females differ from high-quality females on their ability to deal with the harm that they receive from males while low-quality males differ from high-quality males on their ability to harm females. As before, males invest in a harming trait, with its effect on female's fecundity and male's competitiveness for mating success being the same as in the previous sections. Accordingly, a focal low-quality female's fecundity is a function of the average level of male harm present in her patch multiplied by $(1 + h)$ due to her low quality $f_{\mathrm{lf}}(Y) = 1 - Y(1 + h)$, a focal low-quality male's competitiveness for mating success is a function of the level of harm expressed by that focal male multiplied by $(1 - t)$ due to his low quality $f_{\mathrm{lm}}(y) = 1 + y(1 - t)$, a focal high-quality female's fecundity is a function of the average level of male harm present in her patch $f_{\mathrm{hf}}(Y) = 1 - Y$, and a focal high-quality male's competitiveness for mating success is a function of the level of harm expressed by that focal male $f_{\mathrm{hm}}(y) = 1 + y$. Mating follows, and each female produces a large number of offspring with an even sex ratio and in direct proportion to her fecundity. Adults then die, and juvenile females disperse with probability d_{f} and juvenile males disperse with probability d_{m}. Following dispersal, n_{f} females

and n_m males survive at random within each patch to adulthood, returning the population to the beginning of the lifecycle.

Natural selection – We now follow the same approach as in the section 1.1 and 1.2. Female's and male's relative fitness in the context of the present model is the same as in section 1.2, with the only differences being in the female's fecundity and male's competitiveness for mating success (see above). Because of those difference, the resulting marginal fitness equation is also different (see section 1.2 for the procedure to obtain it):

$$\frac{(1-r_{mm})(1-t\bar{Y}^*)}{1+\bar{Y}^*(1-t\bar{Y}^*)} - \frac{1+h\bar{Y}^*}{1-\bar{Y}^*(1+h\bar{Y}^*)}(r_{fm}+r_{mm})(1-a) = 0. \quad \text{(A15)}$$

Optimal level of harm – The optimal level of harm can now be calculated by solving equation (A15) to $\bar{Y}^*$. Note that three solutions are found, but only one follows the assumptions of the model. We can then use that solution to represent the optimal level of harm as in Figure 1 of the main text (maternal effects on sexual traits line). Unfortunately, the analytical solution is too lengthy to be explicitly represented here.

1.4 Systematic review

We conducted a systematic review of the existing literature following the PRISMA protocol[53] as closely as possible. Specifically, we looked for studies that described adaptations leading to male harm to females, consisting of male adaptations involving direct trauma to females. We only qualified extracted phenotypic traits when it was clear from the reported paper, or the raw data, that the trait had a direct negative impact on female lifetime reproductive success and/or (in the absence of this measures) because male adaptations inflicted obvious injuries to females. Due to the co-evolution of female resistance and male harm, harmful male adaptations may not be expected to impose high fitness costs in females over most evolutionary time[12]. We thus opted to included cases in which lifetime/reproductive fitness costs to females were not studied but male adaptations involved produced measurable harm to females (i.e. injuries), such as in traumatic insemination via genital ablation or copulatory wounding, or in cases where male harassment regularly leads to female injuries and occasional deaths. We conducted a first literature search on 03/04/20 using the Scopus, PubMed and Web of Science (WoS) databases with the search terms "sexual conflict" & "male harm" OR "sexual conflict" & "female harm" for animal taxa. Overall, very few

papers were found with these search strings (73 total: Scopus = 31, PubMed = 15 and WoS = 27). After removing duplicates only 36 papers were relevant, and we exported them to Rayyan. We conducted a second literature search on 03/04/20 using the Scopus, PubMed and Web of Science (WoS) databases with the search terms: “sexual conflict” & “female fitness” OR “sexual conflict” & “female productivity” OR “sexual conflict” & “female fecundity” OR “sexual conflict” & “female reproductive success”. We found a total of 694 papers (Scopus = 250, PubMed = 144 and WoS = 300). After removing 373 duplicates, we exported 321 to Rayyan. We conducted a final search on the 7/04/20 using the search terms: “sexual conflict” & harassment. We found a total of 414 items (Scopus = 175, PubMed = 50 and WoS = 189). After removing 175 duplicates, we exported 239 to Rayyan. In Rayyan, we checked for duplicates within the complete database comprising all the papers located via these three searches and removed 69 duplicates, leading to 527 unique studies for more detailed screening. Based on the title and abstract we excluded 347 papers that clearly did not report adaptations for male harm, leaving a total of 180 papers for in-depth screening. We carefully screened these papers and excluded papers that did not comply with our selection criteria described above. In the process of screening, we added more papers through forward and backward searches of citations and references, leading to a total final sample of selected studies reporting male harm adaptations for a total of 73 different species (see supplementary materials for complete list). Finally, for all these species we performed specific searches on their mode of reproduction (i.e. ovipary, vivipary or ovovivipary), evidence for parental care and evidence of extended maternal provisioning (i.e. matrotrophy, including brooding). Similar data for broad taxonomic groups taxa included in Figure 2A were taken from key references provided in the supplementary materials.

Competing interests

The authors declare no competing interests.

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Author contributions

PC and RG-R conceived this study. GF, PC & RG-R designed the study. GF developed the mathematical models and Figure 1. PC & RG-R performed the systematic literature search. PC prepared Figure 2. PC wrote the manuscript with contributions by GF and RG-R.

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Figure 1 | Optimal level of harm favoured in different models and as a function of male dispersal (d_m). The level of harm that is favoured by natural selection depends on the absence or presence of maternal effects. Accordingly, absence of maternal effects leads to higher levels of harm than in the presence of maternal effects, regardless of those effects being on the fecundity or on the sexual quality of the individuals. For all panels, the following parameters were used: female dispersal rate $d_f = 0.5$, number of females $n_f = 3$; number of males $n_m = 3$; fecundity cost $s = 0.5$; sexual cost for females $h = 0.5$; and sexual cost for males $t = 0.5$.

Figure 2 | Distribution of male harm adaptations across the tree of life. A) Results from taxa that encompass the 73 species for which we found evidence of male harm. Outward-facing circles in the red shaded area represent the presence of direct (red) and/or collateral (orange) adaptations leading to male harm of females (i.e. one for each species). Triangles denote the existence of substantial evidence suggesting this strategy is widespread in at least some taxa within the group (see supplementary material for details). Inner-facing circles in the green shaded area reflect evidence in the literature of parental care, extended gestation (viviparity/ovoviviparity vs. oviparity) and extended maternal provisioning (matrotophy/placentotrophy/brooding vs. lecithotrophy) in the taxon. The size of inward-facing green circles illustrates how widespread these strategies are according to the literature: large circles denote a widespread strategy (i.e. adopted by most or all known species), medium-size circles represent a common strategy (i.e. more than 5% but less than 50% of known species) and small-sizes circles represent a rare strategy (i.e. < 5% known species). B) Summary of indirect evidence (i.e. mostly based on descriptions of male genitalia; see supplementary material for details) for male adaptations that may be directly harmful to females (i.e. traumatic insemination, including genital wounding and ablation). External genital mutilation is common in some spiders, where maternal care in the form of egg-protection is widespread[54–56]. External genital mutilation likely evolved in response to sperm competition by preventing females from effectively re-mating. However, there is no evidence to date that it reduces female fitness and it does not affect receptivity to male mating attempts[54–56]. The numbers beside red bars represent the number of species within the group that are estimated to exhibit such male harm adaptations. Like in A) green-shaded circles reflect evidence in the literature of parental care, extended gestation and extended maternal provisioning in the taxon. C) Percentage of the 73 species in A) for which parental care, extended gestation and extended maternal provision has been reported in the literature.

Figure 1

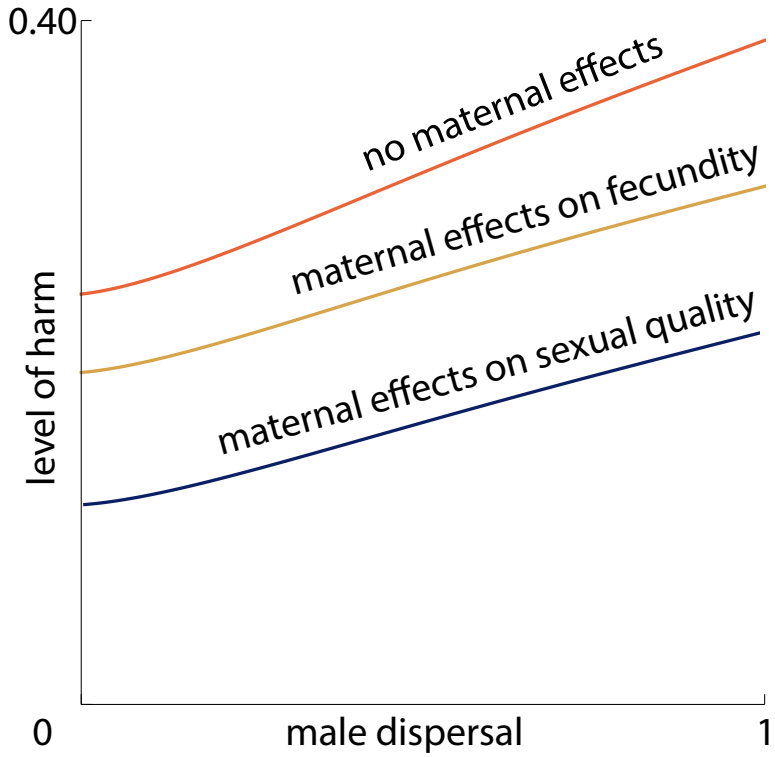
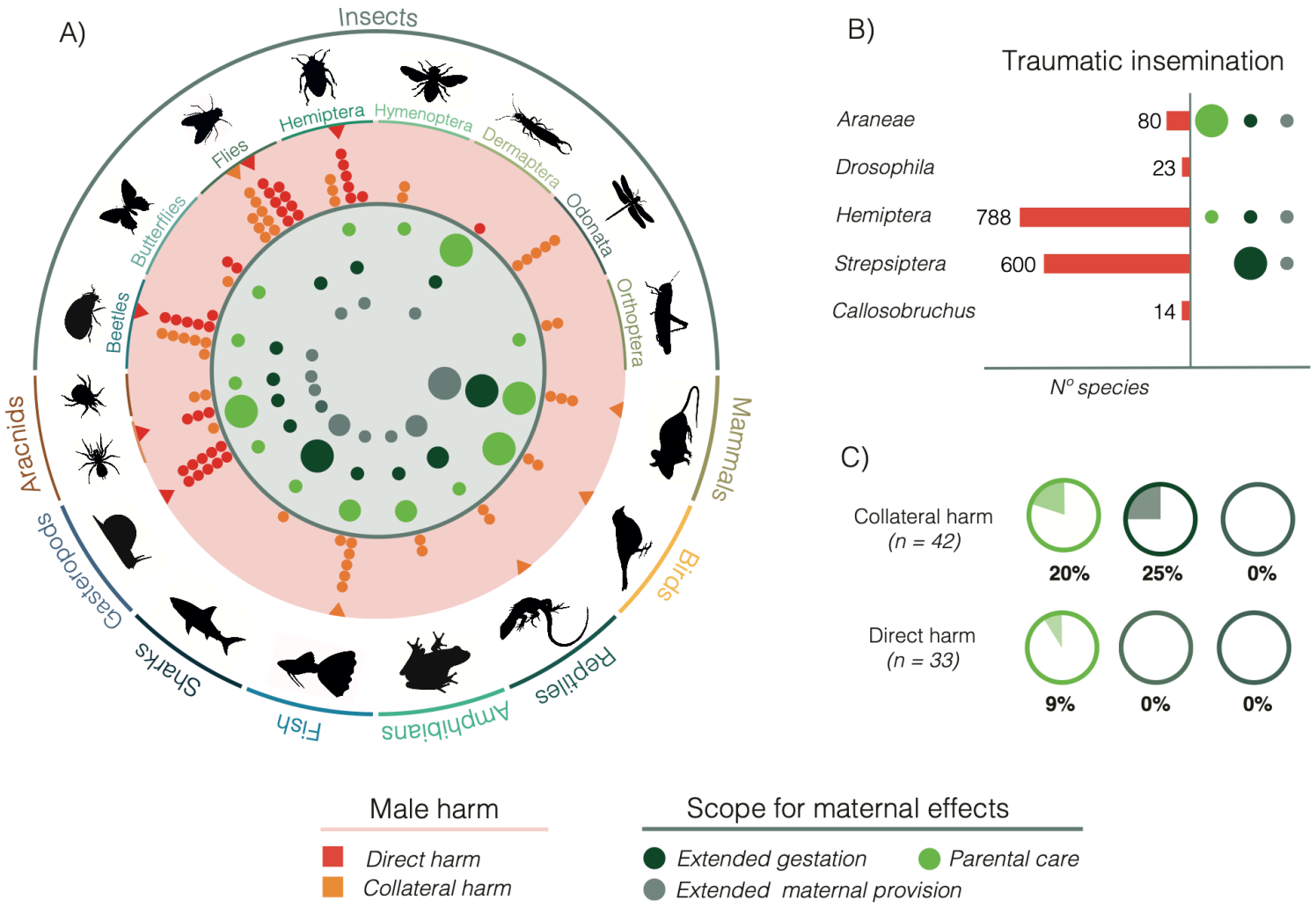

0.40
level of harm
no maternal effects
maternal effects on fecundity
maternal effects on sexual quality
0
male dispersal
1

Figure 2


A)
Insects
Hemiptera
Hymenoptera
Dermaptera
Odonata
Orthoptera
Flies
Butterflies
Beetles
Aracnids
Gasteropods
Sharks
Fish
Amphibians
Reptiles
Birds
Mammals
B)
Traumatic insemination
Araneae
Drosophila
Hemiptera
Strepsiptera
Callosobruchus
80
23
788
600
14
Nº species
C)
Collateral harm
(n = 42)
20%
25%
0%
Direct harm
(n = 33)
9%
0%
0%
Male harm
Direct harm
Collateral harm
Scope for maternal effects
Extended gestation
Parental care
Extended maternal provision