Condition-transfer maternal effects modulate inter-locus sexual conflict

Roberto García-Roa[1†], Gonçalo S. Faria[2†], Daniel Noble[3] and Pau Carazo[1*]

1. Ethology lab, Cavanilles Institute of Biodiversity and Evolutionary Biology, University of Valencia, Valencia, Spain.

2. School of Biological Sciences, University of East Anglia, Norwich, United Kingdom.

3. Division of Ecology and Evolution, Research School of Biology, Australian National University, Canberra ACT 2600, Australia.

† authors contributed equally to this manuscript.

* Corresponding author: Pau Carazo, Cavanilles Institute of Biodiversity and Evolutionary Biology, c/ Catedrático José Beltrán 2, 46980, Paterna (Valencia), Spain. Telephone: +34 3544051, e-mail: pau.carazo@uv.es.

Roberto García-Roa http://orcid.org/0000-0002-9568-9191

Pau Carazo http://orcid.org/0000-0002-1525-6522

Running title: Maternal effects curb sexual conflict

Word count: 168 (abstract) and 3553 (main text including all sections); **Figures:** 3;

Keywords: Sexual conflict, sexual selection, maternal effects, population viability, population growth, sexually antagonistic coevolution, evolution.

Abstract

Strong sexual selection frequently favours males that increase their reproductive success by harming females, with potentially negative consequences for natural populations. Understanding what factors modulate conflict between the sexes is hence critical to understand both the evolution of male and female phenotypes and the viability of populations in the wild. Here, we model the evolution of male harm while incorporating male-induced maternal effects on offspring quality. We show that, because male harm can induce condition-transfer maternal effects that reduce the quality of a harming male's own offspring, maternal effects can partially align male and female evolutionary interests and significantly curb the evolution of male harm. These effects are independent of relatedness, the scale of competition, mating system, and whether male harm comes before (i.e. harassment) and/or during/after (i.e. traumatic inseminations or toxic ejaculates) mating, and are particularly salient when maternal effects influence offspring ability to inflict (sons) or resist (daughters) harm. Our results underscore the potential importance of considering maternal effects to unravel 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 (Andersson 1994). This, in turn, can trigger sexually antagonistic coevolution (Parker 1979; Holland and Rice 1998), where sexual strategies in one sex evolve to counteract those of the opposite sex. Sexually antagonistic coevolution leads to inter-locus sexual conflict (i.e. when traits under sexually antagonistic selection depend on different genetic loci in males and females, leading to conflicting evolutionary interest between the sexes) or intra-locus sexual conflict (i.e. when these traits share the same underlying loci in both sexes); both currently recognized as key evolutionary processes shaping male and female adaptations and life-history traits (Arnqvist and Rowe 2005). At a population level, inter-locus sexual conflict frequently leads to adaptations in males that harm females (Chapman et al. 1995; Rice 1996) and reduce population growth, in a process akin to "the tragedy of the commons" (i.e. where selection for selfish competition among males reduces a common finite resource that decreases population growth; Rankin et al. 2011). From male harassment and coercion (Han and Jablonski 2010; Perry and Rowe 2015a) to toxic ejaculates (Wigby and Chapman 2005) and traumatic insemination (Crudgington and Siva-Jothy 2000; Reinhardt et al. 2015), harmful male adaptations are both widespread across the tree of life and extraordinarily diverse in the level of harm they inflict on females, and thus in their potential consequences for population viability. A current priority in evolutionary biology is hence 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 (Rankin 2011; Carazo et al. 2014; Faria et al. 2015; Faria et al. 2020; Lukasiewicz et al. 2017).

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 (Cameron et al. 2003; Pizzari and Snook 2003; Maklakov et al. 2005; Parker 2006; Garcia-Gonzalez and Simmons 2010; Brennan and Prum 2012). On the one hand, manipulative or harmful traits allow males to sire a greater proportion of a female's offspring (e.g. by decreasing female re-mating) 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 (Cameron et al. 2003; Pizzari and Snook 2003; Parker 2006).

Maternal effects can drastically modulate offspring quality (Mousseau and Fox 1998) and are largely mediated by maternal condition (i.e. condition-transfer maternal effects; (Rossiter 1996; Qvarnström and Price 2001; Saino et al. 2005; Bonduriansky and Crean 2018). Male harm can severely impact female condition (Arnqvist and Rowe 2005) 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 (Tregenza et al. 2003; Brommer et al. 2012; Gasparini et al. 2012; Dowling et al. 2014; Carazo et al. 2015; Zajitschek et al. 2018). For example, female guppies (*Poecilia reticulata*) exposed to greater harassment produce smaller daughters and sons with shorter gonopodia (Gasparini et al. 2012), whereby female size is related to fecundity and large gonopodia are favoured in both inter- and intra-sexual selection in this species (Brooks and Caithness 1995; Evans and Pilastro 2011; Gasparini et al. 2012). Furthermore, previous studies have already suggested that maternal effects may have the potential to modulate sexual conflict effects on female offspring (Foerster et al. 2007; Lund-Hansen et al. 2021).

In this study, we develop a mathematical model to formally examine whether 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. In particular, we use a personal-fitness (i.e. neighbour-modulated fitness) kin-selection approach (Hamilton 1964a,b; Taylor and Frank 1996) that incorporates the effects of kin selection, an important factor in the evolution of male harm to females (Faria et al. 2020). Given the potential for maternal effects to bring together the evolutionary interests of females and males, we aim to analyse if such effect happens in conjunction or independently of kin selection. We analyse three scenarios: a) absence of maternal effects on offspring quality, b) presence of maternal effects on offspring fecundity (females) and competitiveness (males), and c) presence of maternal effects on offspring ability to inflict (males) and resist (females) harm (i.e. sexual selection quality).

Methods

1.1 Model without maternal effects

We use a neighbour-modulated fitness approach - an optimisation function to calculate the optimal level of harm (see (Hamilton 1964a,b; Taylor and Frank 1996)). We consider an infinite population divided into social groups (Wright 1931) containing n_f females and n_m males. We follow the approach developed by Faria et al. (2020). Specifically, males invest in a harming trait that increases their personal reproductive success relative to other males but reduces the overall fecundity (number of offspring) of the females in the social group. 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 social group (see below and Supplementary Material for mathematical formulation). Male harm happens within "social groups" because this simulates what typically happens in nature,

whereby males compete for female access within mating patches. This includes both proper social groups and, more commonly, temporal aggregations (e.g. leks, local mating aggregations around resources etc.). In addition, this approach allows us to control for kin and demographic effects which have been shown to be important modulators of male harm (Faria et al. 2020). We consider two different types of populations differing in their mating system: a) monogamic females, where females mate with only one male while males compete to gain access to the females (Figure 1), and b) polygamic females, where all males mate with all females in the social group (Figure 1). We acknowledge this is a simplification, but our focus was to examine the two extreme contexts in which the two types of harm modelled (i.e. prior or during/after mating) seem to be highly beneficial to males (i.e. harassing females when mating success is most important and inflicting mating harm when sperm competition is most important).

Accordingly, a focal female's fecundity $f_f(x',y) = 1 - k\,x' - (1 - k)y$ is a function of the level of harm that the male she mates with inflicts (x') and of the average level of male harm males present in the social group (y), with k determining the degree to which harm comes during mating ($k = 1$) or before mating as a result of male competition to access females ($k = 0$). At one extreme ($k = 1$), harm comes exclusively from the male that mates with the female, as harm is linked to mating (as in traumatic insemination) and mating harm makes the female unavailable for further mating events. In other words, mating happens exclusively during mating and such extreme traumatic insemination effectively makes females monogamous, such as is the case with genital ablation in some spiders (Mouginot et al. 2015). At the other extreme ($k = 0$), harm comes from all males in the social group, which aims to simulate male harm happening exclusively via the harassment of females during pre-copulatory male-male competition (i.e. irrespective of which male mates with the female). Importantly, in our model we consider a continuum, such that a population may lie at any

point between those two extremes. Our aim is to understand when one type of harm may be favoured over the other, but we stress that harassment and traumatic insemination need not be traded-off between each other in nature. Note that, when considering a polygamic population, the variable k disappears because all males are mating with all females and thus all males harm females irrespective of whether this happens during or after mating (therefore, $f_f(y) = 1 - y$).

A focal male's competitiveness for mating success $f_m(x) = 1 + x$ is a function of the level of harm expressed by that focal male (x). His actual mating success depends on how much the other males in the social group invest into harm $f_m(y) = 1 + y$ and, therefore, the relative mating success of the focal male over the other males in the social group is $f_m(x)/f_m(y)$. After mating, each female produces a number of offspring proportional to her fecundity $f_f(x',y)$, with an even sex ratio. Adults then die, and juvenile females and males compete for reproductive resources, with a proportion a of this competition occurring locally with social group mates and a proportion $1 - a$ occurring globally with unrelated individuals (Figure 1). Thus, a takes into account all elements that contribute to determine the scale of competition, from local to global. In other words, a specifies the likelihood of a focal male juvenile competing with other local males in the patch for future breeding opportunities, which occurs when neither disperse to other patches (Faria et al. 2020). Finally, n_f females and n_m males survive at random within each social group to adulthood, returning the population to the beginning of the lifecycle. Our model thus assumes competition for n_f and n_m reproductive spots (i.e. the number of females and males that compete successfully and reproduce; see Faria et al. 2015).

1.2 Model with maternal effects

As in section 1.1 (Figure 1), we consider an infinite diploid population divided into social groups (Wright 1931) containing n_f females and n_m males following a similar life-cycle. We now also assume that there are two types of individuals: low-quality individuals; and high-quality individuals. High-quality individuals are assumed to be no different than the individuals present in the model without maternal effects. We consider two possible scenarios: 1) quality affects an individual's fecundity (females) and competitiveness (males); or 2) quality affects an individual's ability to inflict (males) and resist (females) harm. Thus, in our model maternal effects affect F1 female and male quality differently, with low-quality individuals being produced in proportion to the harm that their mother received, which altogether impacts parental female and male fitness (see Supplementary Material for details).

Focusing on the first scenario: a focal low-quality female's fecundity is a function of the level of harm of the male that she mates with and of the average level of male harm present in the social group minus a cost s due to her low quality $f'_f(x',y) = 1 - k\,x' - (1-k)y - s$ (for a monogamic female) and $f'_f(y) = 1 - y - s$ (for a polygamic female); 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 $f'_m(x) = 1 + x - s$; a focal high-quality female's fecundity is a function of the level of harm of the male that she mates with and of the average level of male harm present in the social group $f^h_f(x',y) = 1 - k\,x' - (1-k)y$ (for a monogamic female) and $f^h_f(y) = 1 - y$ (for a polygamic female); and a focal high-quality male's competitiveness for mating success is a function of the level of harm expressed by that focal male $f^h_m(x) = 1 + x$.

Focusing on the second scenario, a focal low-quality female's fecundity is a function of the level of harm of the male that she mates with and of the average level of male harm present in the social group multiplied by $1 + h$ due to her low quality $f'_f(x',y) = 1 - (k\,x' + (1-k)y)(1+h)$ (for a monogamic female) and $f'_f(y) = 1 - y(1+h)$ (for a polygamic female);

where h denotes the extra harm caused by reduced female resistance due to her low quality. 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 $f^{h}_{m}(x) = 1 + x(1 - t)$, where t denotes the reduction in harm caused by the mala's low quality. A focal high-quality female's fecundity is a function of the level of harm of the male that she mates with and of the average level of male harm present in the social group $f^{h}_{f}(x',y) = 1 - k\,x' - (1 - k)y$ (for a monogamic female) and $f^{h}_{f}(y) = 1 - y$ (for a polygamic female). Lastly, a focal high-quality male's competitiveness for mating success is a function of the level of harm expressed by that focal male $f^{h}_{m}(x) = 1 + x$. Competition in the social group then follows the same logic described above for section 1.1 (Figure 1).

Results

Model – Following Taylor-Frank approach (see Supplementary Material for details) and assuming that $n_f = n_m$ (i.e. the number of females and males are the same in each social group), the optimal fitness equations (optimum level of harm; see Supplementary Material for details) in a population where females are monogamic are:

$$\left(\frac{1}{1+z^*} - k\frac{1}{1-z^*}\right)(1 - r_{mm}) - \frac{1}{1-z^*}(r_{fm} + r_{mm})(1 - a) = 0, \quad (1)$$

for the model without maternal effects;

$$\left(\frac{1}{1+z^*-sz^*} - k\frac{1}{1-z^*-sz^*}\right)(1 - r_{mm}) - \frac{1}{1-z^*-sz^*}(r_{fm} + r_{mm})(1 - a) = 0, \quad (2)$$

for the model with maternal effects on fecundity (females) and competitiveness (males); and

$$\left(\frac{1-tz^*}{1+z^*(1-tz^*)} - k\frac{1+hz^*}{1-z^*(1+hz^*)}\right)(1-r_{\mathrm{mm}}) - \frac{1+hz^*}{1-z^*(1+hz^*)}(r_{\mathrm{fm}}+r_{\mathrm{mm}})(1-a) = 0, \quad (3)$$

for the model with maternal effects on the ability to inflict (males) and resist harm (females), where: z^* is the optimal level of harm favoured by natural selection; r_{mm} is the relatedness between males in a social group; and r_{fm} is the relatedness between females and males in a social group. Regardless of the model considered, the inclusive fitness interpretation is the same. Specifically, a male increases his mating success by investing into harming (first term). As k increases, harming is increasingly done during mating and this imposes a further cost on the focal male's mating success as it reduces the potential number of offspring that he has with a female (first term). Both are weighted by the relatedness between the focal male and local males, given that an increase in focal male's mating success leads to a corresponding loss of mating success by the other males. This translates into an inclusive fitness loss if the focal male is related to them ($1 - r_{\mathrm{mm}}$). Harming also reduces the overall fecundity of local females, which also decreases the number of offspring produced by local females and males (second term). Such reduction in fecundity can lead to an inclusive fitness loss if the focal individual is related to both local females and males ($r_{\mathrm{fm}} + r_{\mathrm{mm}}$). Finally, such inclusive fitness loss is weighted by the scale of competition ($1 - a$), that is, how much individuals compete with local social group mates for reproductive resources (Taylor 1992).

The conditions for harm to be favoured in a polygamic population are essentially the same, with the exception that the term being multiplied by k disappears. Accordingly, the inclusive fitness interpretation is the same as above, except that the males do not pay a direct cost for harming the females. The optimal level of harm can now be obtained by solving equations (1–3) to z^*.

While the results are similar across the different models, there are relevant quantitative differences (Figure 2; Figure 3). Importantly, the harm benefits are smaller, and

costs are higher, when maternal effects are present, and more so when maternal effects influence the ability of offspring to inflict (males) or resist (females) harm (Figure 2; Figure 3). This happens regardless of harm coming before (i.e. male harassment) and/or during (i.e. traumatic insemination) mating (Figure 2a), relatedness levels (Figure 2b-c; Figure 3a-b), or polygamy (Figure 2; Figure 3). The exception is when local competition is high (Figure 2d; Figure 3c). When local competition is high, maternal effects on fecundity reduce the level of harm more than maternal effects on sexual quality. Note that these results extrapolate generally to any processes that, like maternal effects, affect male/female offspring quality.

Discussion

We found that maternal effects reduce the optimal level of male harm, especially when harm curtails offspring quality during sexual selection (Figures 2 and 3). The latter, however, can change with the scale of competition (i.e. as a approaches 1 and competition becomes increasingly local; Figures 2D and 3D), leading to maternal effects on fecundity reducing the level of harm more than maternal effects on sexual quality. The reason for such difference between maternal effects on fecundity vs. sexual quality is due to the former only reducing daughter fecundity, but not sons' chances of reproduction. In contrast, maternal effects on the sexual quality of sons seem particularly disruptive to a male's long-term fitness. As local competition increases, competition occurs between males of similar quality, thus reducing the importance of such effects and increasing harm to levels above those for maternal effects on fecundity. Regardless, maternal effects consistently reduce harm for different types of male harm (i.e. male harassment and traumatic insemination), mating systems, across different levels of relatedness, and levels of local competition (Figures 2 and 3). It is worth noting that, for simplicity, we focused on monogamous vs. completely polygamous scenarios (i.e. we did not explore intermediate levels of polygamy), but our results were very robust across these

two extreme scenarios, strongly suggesting that they can be generally extrapolated across mating systems. As previously shown, relatedness can shape the level of harm under each one of the different models (Figures 2B and 3A for male-male relatedness and 2C and 2B for female-male relatedness), but our results also show that relatedness (and particularly so female-male relatedness; Figures 2C and 3B) modulates the degree to which maternal effects curtail harm as well as the relative importance of maternal effects on offspring fecundity vs. sexual quality.

Differences in the optimal level of male harm across different populations are therefore not only predicted to reflect differences in relatedness and the scale of competition, leading to the kin selection effects previously described in the literature (Faria et al. 2020), but also in the biology of male harm and its impact on 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 lower whenever harm induces condition-transfer maternal effects on offspring quality, in a manner that is proportional to these effects. Previous studies had already suggested that maternal effects may have the potential to partially compensate for sexual conflict effects on female offspring (Foerster et al. 2007; Lund-Hansen et al. 2021). Here, we show that male harm-mediated maternal effects indeed have the potential to shape sexual conflict evolution.

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 (relative to quantity) 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 (i.e. in species that favour investment in offspring survival vs. quantity). 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. To assess these theoretical predictions, we conducted a systematic search in the literature to identify studies reporting solid quantitative or qualitative evidence of male harm to females and, for these species, collected data on three proxies of maternal effects: parental care, extended gestation and extended maternal provision (see Supplementary Material for details). Our aim was to perform a meta-analysis of this association across the tree of life but, unfortunately, the resulting dataset did not encompass enough variation in the key variables of interest related to maternal effects (e.g. maternal provisioning, gestation period, parental care, level of male harm, etc.) to address a formal analysis. Namely, we found very little scope for maternal effects in the relatively few species for which male harm has been well studied (see Supplementary Material and accompanying data for details), and thus not enough co-variation between both variables. However, there are some general qualitative trends in the data that are worth discussing.

Overall, we found evidence of male harm for 87 species across the tree of life (see Supplementary Figure 1 and accompanying Supplementary Material data). Male harm seems particularly widespread, intense and sophisticated in insects, which include the best-known cases of sexually antagonistic coevolution driven by male harm (Perry and Rowe 2015b) along with many instances of traumatic insemination (Crudgington and Siva-Jothy 2000; Arnqvist et al. 2005; Siva-Jothy 2006; Tatarnic et al. 2014; Reinhardt et al. 2015), including toxic ejaculates (Wigby and Chapman 2005) and extreme coercion (Han and Jablonski 2010). Furthermore, indirect evidence based on the description of male genitalia (and the fitness

312 consequences of similar structures in other species) suggests adaptations for traumatic
313 insemination may occur in as many as ca.1400 species more (see Supplementary Material for
314 details). Albeit there are many obvious exceptions (e.g. eusocial insects), insects are very
315 frequently under strong r-selection, oviparous and normally lack extended maternal provision
316 and parental care. Gastropods, where traumatic insemination also seems common, seem to
317 follow a similar pattern (see Supplementary Figures 1-2 and Supplementary Material data). In
318 contrast, male harm appears to be relatively rare or weak in vertebrates, especially so in taxa
319 with widespread parental care and prolonged gestation such as birds and mammals (see
320 Supplementary Figures 1-2 and Supplementary Material data). As a matter of fact, well-
321 studied cases of male harm reported so far in vertebrates consist exclusively in collateral
322 damage to females (i.e. harassment and/or coercive mating), as opposed to traumatic
323 insemination adaptations, where male fitness benefits derive from harming females *per se*
324 (i.e. direct damage; Aloise King et al. 2013).

325 The absence of adaptations for direct harm in mammals is particularly salient given
326 the strength of male-male competition in many species within this group (Andersson 1994).
327 Furthermore, although harassment is widely interpreted as an inherently costly male
328 phenotype for females, it does not necessarily translate into a reduction in female fitness. For
329 example, female resistance to male harassment has been suggested to function as a form of
330 mate choice as a way of screening high quality males (Cordero and Eberhard 2003). Thus, the
331 mere existence of male harassment and/or coercion does not necessarily imply fitness costs to
332 females. Different forms of sexual harassment and/or coercion to females have been reported
333 for a number of vertebrates (and are probably common; Clutton-Brock and Parker 1995),
334 such that in these cases intense courtship is assumed to be harmful for females. However,
335 direct evidence that such harassment reduces female fitness is limited (Magurran and
336 Ojanguren 2007; Makowicz and Schlupp 2013; Iglesias-Carrasco et al. 2019). For example,

337 forced copulations are common in waterfowls (reported for at least 55 species; McKinney et
338 al. 1983; McKinney and Evarts 1998), where they are frequently accompanied by male
339 harassment behaviour that can occasionally result in injuries and even the death of the female
340 (McKinney et al. 1983). Nonetheless, evidence that such behaviour has net harmful effects on
341 females is more restricted (see Adler 2010; Supplementary Figure 1). In short, available data
342 may seem to be generally aligned with the prediction of a relationship between the
343 importance of maternal effects within broad taxonomic groups and evidence of costly male
344 harm, particularly in the case of direct harm to females. However, it is important to stress that
345 this cannot be taken as preliminary evidence in support of this hypothesis, because existing
346 studies do not span enough co-variation in the scope of maternal effects and male harm to
347 perform a formal analysis.

348 To conclude, in this study we aim to bring attention to male-induced maternal effects
349 as a potentially important factor in the evolution of sexual conflict. Similarly to relatedness
350 (Rankin 2011; Carazo et al. 2014; Faria et al. 2015, 2020; Lukasiewicz et al. 2017), we show
351 that maternal effects can align the evolutionary interests of males and females and abate
352 conflict over sexual strategies. Such effects could be important to understand sexual conflict
353 evolution in nature for two main reasons. The first is the existence of variation in condition-
354 transfer maternal effects that can impinge on offspring quality, both across and within taxa
355 (Royle et al. 2012; Bonduriansky and Crean 2017). The second is the well-established fact
356 that male harm can have a dramatic impact on female condition (Arnqvist and Rowe 2005).
357 We thus suggest that future empirical studies should aim to test the general ideas we lay out
358 here (see above) arising from the interplay between maternal effects and male harm, which
359 could further our understanding of sexual conflict.

360

361 **CONFLICT OF INTEREST**

The authors declare no competing interests.

FUNDING

RGR was supported by a postdoctoral grant (FJC2018-037058-I) funded by MCIN/AEI/ 10.13039/501100011033 and by a Marie Sklodowska Curie Fellowship (HORIZON-MSCA-2021-PF-01 101061275). PC was supported by a research grant (PID2020-118027GB-I00) funded by MCIN/AEI/ 10.13039/501100011033 and a research grant AICO/2021/113 from Generalitat Valenciana. DWAN was supported by an Australian Research Council (ARC) Future Fellowship (FT220100276).

Data accessibility statement: Analyses reported in this article can be reproduced using the data provided by Carazo (2023)

References

Adler M. 2010. Sexual conflict in waterfowl: why do females resist extrapair copulations? Behav Ecol, 21: 182–192.

Aloise King ED, Banks PB, Brooks RC. 2013. Sexual conflict in mammals: consequences for mating systems and life history: Sexual conflict in mammals. Mammal Rev, 43: 47–58.

Andersson M. 1994. Sexual Selection. Princeton University Press, Princeton.

Arnqvist G, Nilsson T, Katvala M. 2005. Mating rate and fitness in female bean weevils. Behav Ecol, 16: 123–127.

Arnqvist G, Rowe C. 2005. Sexual Conflict. Princeton University Press, Princeton.

Bonduriansky R, Crean AJ. 2018. What are parental condition-transfer effects and how can they be detected?. Methods Ecol Evol, 9: 450-456.

Brennan PLR, Prum RO. 2012. The limits of sexual conflict in the narrow sense: new insights from waterfowl biology. Philos Trans R Soc B Biol Sci, 367: 2324–2338.

Brommer JE, Fricke C, Edward DA, Chapman T. 2012. Interactions between genotype and sexual conflict environment influence transgenerational fitness in *Drosophila melanogaster*. Evolution, 66: 517–31.

Brooks R, Caithness N. 1999. Intersexual and intrasexual selection, sneak copulation and male ornamentation in guppies (*Poecilia reticulata*). S Afr J Zool, 34: 48–52.

Cameron E, Day T, Rowe L. 2003. Sexual conflict and indirect benefits. J Evol Biol, 16: 1055–1060.

Carazo P, Perry JC, Johnson F, Pizzari T, Wigby S. 2015. Related male *Drosophila melanogaster* reared together as larvae fight less and sire longer lived daughters. Ecol. Evol, 5: 2787–2797.

Carazo P, Tan CKW, Allen F, Wigby S, Pizzari T. 2014. Within-group male relatedness reduces harm to females in *Drosophila*. Nature, 505: 672–675.

Carazo, Pau; García-Roa, Roberto; Faria, Gonçalo; Noble, Daniel (2021). Data from: Condition-transfer maternal effects modulate inter-locus sexual conflict [Dataset]. Dryad. https://doi.org/10.5061/dryad.cnp5hqc53

Chapman T, Liddle LF, Kalb JM, Wolfner MF, Partridge L. 1995. Cost of mating in *Drosophila melanogaster* females is mediated by male accessory gland products. Nature, 373: 241.

Clutton-Brock TH, Parker GA. 1995. Sexual coercion in animal societies. Anim Behav, 49: 1345–1365.

Cordero C, Eberhard WG. 2003. Female choice of sexually antagonistic male adaptations: a critical review of some current research. J Evol Biol, 16: 1–6.

Crudgington HS, Siva-Jothy M.T. 2000. Genital damage, kicking and early death - The battle of the sexes takes a sinister turn in the bean weevil. Nature, 407: 855–856.

Dowling DK, Williams BR, Garcia-Gonzalez F. 2014. Maternal sexual interactions affect offspring survival and ageing. J Evol Biol, 27: 88–97.

Evans JP, Pilastro A, Schlupp I. 2011. Ecology and evolution of poeciliid fishes. University of Chicago Press.

Faria GS, Gardner A, Carazo P. 2020. Kin discrimination and demography modulate patterns of sexual conflict. Nat Ecol Evol, 4: 1141–1148.

Faria GS, Varela SA, Gardner A. 2015. Sex-biased dispersal, kin selection and the evolution of sexual conflict. J Evol Biol, 28: 1901–1910.

Foerster K, Coulson T, Sheldon BC, Pemberton JM, Clutton-Brock TH, Kruuk LEB. 2007. Sexually antagonistic genetic variation for fitness in red deer. Nature, 447: 1107–1110.

Garcia-Gonzalez F, Simmons LW. 2010. Male-induced costs of mating for females compensated by offspring viability benefits in an insect. J Evol Biol, 23: 2066–75.

Gasparini C, Devigili A, Pilastro A. 2012. Cross-generational effects of sexual harassment on female fitness in the guppy: costs of sexual harassment in the guppy. Evolution, 66: 532–543.

Hamilton, W.D. 1964a. The genetical evolution of social behaviour. I. J Theor Biol, 7: 1–16.

Hamilton, W.D. 1964b. The genetical evolution of social behaviour. II. J Theor Biol, 7: 17–52.

Han CS, Jablonski PG. 2010. Male water striders attract predators to intimidate females into copulation. Nat Commu., 1: 52.

Holland B, Rice WR. 1998. Perspective: Chase-Away Sexual Selection: Antagonistic Seduction Versus Resistance. Evolution, 52: 1–7.

Iglesias-Carrasco M, Fox RJ, Vega-Trejo R., Jennions MD, Head ML. 2019. An experimental test for body size-dependent effects of male harassment and an elevated copulation rate on female lifetime fecundity and offspring performance. J Evol Biol, 32: 1262–1273.

Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gøtzsche PC, Ioannidis JPA, ... Mother D. 2009. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. PLOS Med, 6: e1000100.

Lukasiewicz A, Szubert-Kruszynska A, Radwan J. 2017. Kin selection promotes female productivity and cooperation between the sexes. Sci Adv, 3: 31602262.

Lund-Hansen KK, Olito C, Morrow EH, Abbott JK. 2021. Sexually antagonistic coevolution between the sex chromosomes of *Drosophila melanogaster*. Proc Natl Acad Sci, 118: e2003359118.

Magurran A, Ojanguren A. 2007. Male harassment reduces short-term female fitness in guppies. Behaviour, 144: 503–514.

Maklakov AA, Bilde T, Lubin Y. 2005. Sexual conflict in the wild: elevated mating rate reduces female lifetime reproductive success. Am Nat, 165 Suppl 5: S38-45.

454 Makowicz AM, Schlupp I. 2013. The direct costs of living in a sexually harassing
455 environment. Anim Behav, 85: 569–577.
456 McKinney F, Derrickson SR, Mineau P. 1983. Forced Copulation in Waterfowl. Behaviour,
457 86: 250–293.
458 McKinney F, Evarts S. 1998. Sexual Coercion in Waterfowl and Other Birds. Ornithol
459 Monogr, 163–195.
460 Mouginot P, Prügel J, Thom U, Steinhoff POM, Kupryjanowicz J, Uhl G. 2015. Securing
461 Paternity by Mutilating Female Genitalia in Spiders. Curr Biol, 25: 2980–2984.
462 Mousseau T, Fox CW. 1998. The adaptive significance of maternal effects. Trends Ecol
463 Evol, 13: 403–407.
464 Nakata K. 2016. Female genital mutilation and monandry in an orb-web spider. Biol Lett, 12:
465 20150912.
466 Parker, G.A. 1979. Sexual selection and sexual conflict. In: Sexual selection and reproductive
467 competition in insects (eds. Blum, M.S. and Blum, N.A.). Academic Press, New York,
468 USA, pp. 123–166.
469 Parker G.A. 2006. Sexual conflict over mating and fertilization: an overview. Phil Trans Roy
470 Soc, 361.
471 Perry JC, Rowe L. 2015a. The Evolution of Sexually Antagonistic Phenotypes. Cold Spring
472 Harb Perspect Biol, 7: a017558.
473 Perry JC, Rowe L. 2015b. The Evolution of Sexually Antagonistic Phenotypes. Cold Spring
474 Harb Perspect Biol, 7: a017558.
475 Pizzari T, Snook RR. 2003. Perspective: Sexual Conflict and Sexual Selection: Chasing
476 Away Paradigm Shifts. Evolution, 57: 1223–1236.
477 Qvarnström A, Price TD. 2001. Maternal effects, paternal effects and sexual selection.
478 Trends Ecol Evol, 16: 95–100.

Rankin DJ. 2011. Kin selection and the evolution of sexual conflict. J Evol Biol, 24: 71–81.

Rankin DJ, Dieckmann U, Kokko H. 2011. Sexual conflict and the tragedy of the commons. Am Nat, 177: 780–91.

Reinhardt K, Anthes N, Lange R. 2015. Copulatory Wounding and Traumatic Insemination. Cold Spring Harb Perspect Biol, 7: a017582.

Řezáč M. 2009. The spider *Harpactea sadistica*: co-evolution of traumatic insemination and complex female genital morphology in spiders. Proc R Soc B Biol Sci, 276: 2697–2701.

Rice WR. 1996. Sexually antagonistic male adaptation triggered by experimental arrest of female evolution. Nature, 381: 232–4.

Rossiter M. 1996. Incidence and consequences of inherited environmental effects. Annu Rev Ecol Syst, 27: 451–476.

Royle NJ, Smiseth PT, Kölliker M. 2012. The evolution of parental care. 1st edn. Oxford University Press.

Saino N, Romano M, Ferrari RP, Martinelli R, Møller AP. 2005. Stressed mothers lay eggs with high corticosterone levels which produce low-quality offspring. J Exp Zoolog A Comp Exp Biol, 303A: 998–1006.

Siva-Jothy MT. 2006. Trauma, disease and collateral damage: conflict in cimicids. Philos Trans R Soc B Biol Sci, 361: 269–275.

Tatarnic NJ, Cassis G, Siva-Jothy, MT. 2014. Traumatic Insemination in Terrestrial Arthropods. Annu Rev Entomol, 59: 245–261.

Taylor PD. 1992. Altruism in viscous populations- an inclusive fitness model. Evol Ecol, 6: 352–356.

Taylor PD, Frank SA. 1996. How to make a kin selection model. J Theor Biol, 180: 27–37.

504 Tregenza T, Wedell N, Hosken DJ, Ward PI. 2003. Maternal effects on offspring depend on
505 female mating pattern and offspring environment in yellow ding-flies. Evolution, 57:
506 297–304.
507 Wigby S, Chapman T. 2005. Sex peptide causes mating costs in female *Drosophila*
508 *melanogaster*. Curr Biol, 15: 316–321.
509 Wright S. 1931. Evolution in mendelian populations. Genetics, 16: 97–159.
510 Zajitschek SRK, Dowling DK, Head ML, Rodriguez-Exposito E, Garcia-Gonzalez, F. 2018.
511 Transgenerational effects of maternal sexual interactions in seed beetles. Heredity,
512 121: 282–291.

Figure 1 | Graphical representation of the theoretical model.

During the adult phase, and assuming monogamy, only one adult male is successful in mating with one adult female (that r_{mm} and r_{fm} represent male-male and female-male relatedness, respectively; k reflects the extent to which harm is inflicted during mating (k = 1) or before mating (k = 0) as a result of male-male competition for females). In this case, male harm to females depends on the value of k, with harm only coming from the male that mates if $k = 1$ and with non-mating males in the social group also harming females if $k < 1$. Assuming polygamy, all adult males mate with all adult females, with all males harming all females in the process. During the juvenile phase, females and males compete for reproductive opportunities. Competition occurs between local individuals in proportion to a, with competition occurring between local and non-local individuals in proportion to $1 - a$.

Figure 2 | Optimal level of harm favoured in monogamic females.

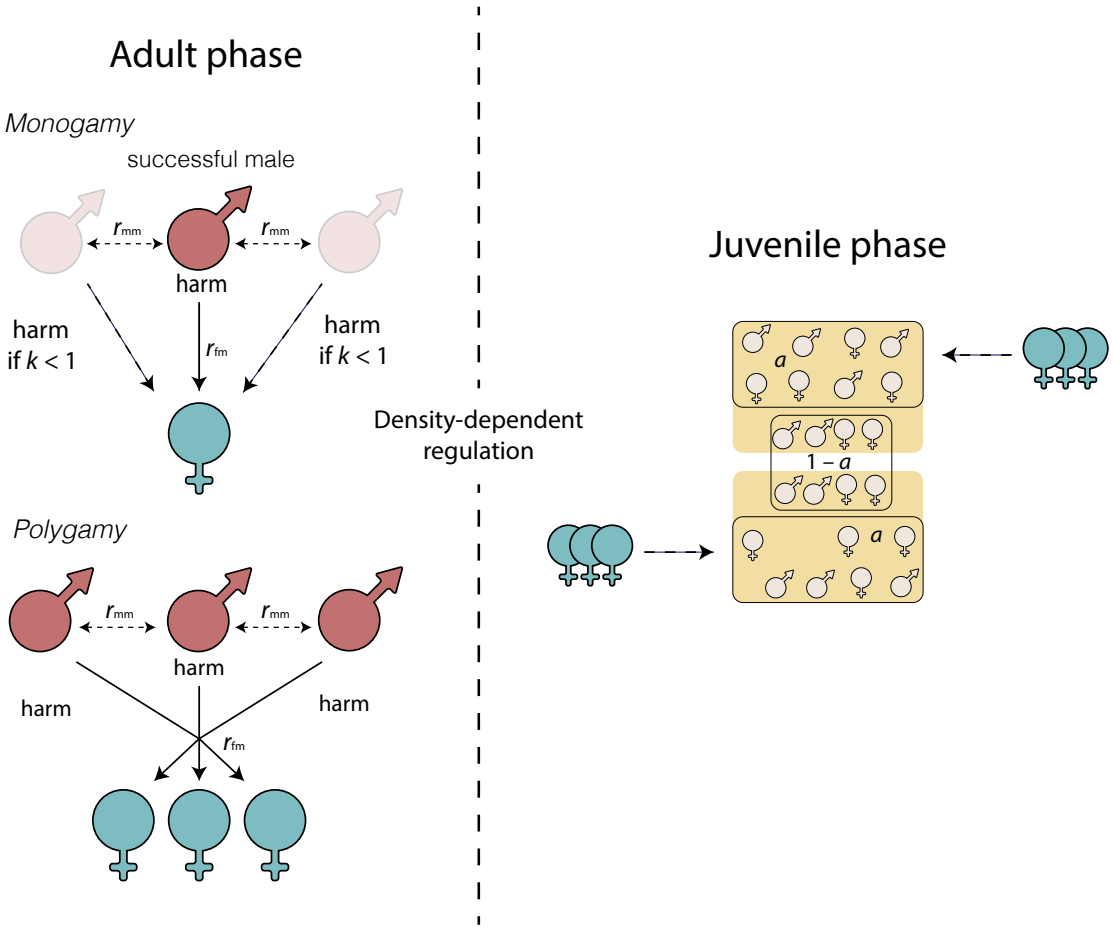
The level of harm that is favoured by natural selection depends on the absence or presence of maternal effects in monogamic females. Accordingly, absence of maternal effects leads to higher levels of harm than in the presence of maternal effects, more so when they affect the individuals' sexual quality. Such effect is present regardless of k (i.e. the degree to which harm occurs before and/or during mating) (A), of the levels of relatedness between individuals in the social group of a population of monogamic females (B and C). When local competition is high, maternal effects still lead to lower levels of harm, but maternal effects on fecundity can lead to lower levels of harm when compared to maternal effects on sexual quality (D). For all panels, the following parameters were used: number of males $n_m = 3$; number of females $n_f = 3$; fecundity and competitiveness cost $s = 0.5$; sexual cost for females $h = 0.5$; and sexual cost for males $t = 0.5$. In a and d: relatedness between males $r_{mm} = 0.15$; and relatedness between females and males $r_{fm} = 0.15$. In b, c, and d: harm exclusive from

sexual partners $k = 0$. In a, b, and c: level of local competition $a = 0.5$. In b: relatedness between females and males $r_{fm} = 0.15$. In c: relatedness between males $r_{mm} = 0.15$.

Figure 3 | Optimal level of harm favoured in polygamic females.

The level of male harm that is favoured by natural selection depends on the absence or presence of maternal effects in polygamic females. Accordingly, absence of maternal effects leads to higher levels of harm than in the presence of maternal effects, more so when male harm affects the individuals' sexual quality. Such effect is present regardless of the levels of relatedness between individuals in the social group (A and B). When local competition is high, maternal effects still lead to lower levels of harm but maternal effects on fecundity can lead to lower levels of harm when compared to maternal effects on sexual quality (C). For all panels, the following parameters were used: number of males $n_m = 3$; number of females $n_f = 3$; fecundity and competitiveness cost $s = 0.5$; sexual cost for females $h = 0.5$; and sexual cost for males $t = 0.5$. In A and B: level of local competition $a = 0.5$. In A: relatedness between females and males $r_{fm} = 0.15$. In B: relatedness between males $r_{mm} = 0.15$. In C: relatedness between males $r_{mm} = 0.15$; and relatedness between females and males $r_{fm} = 0.15$.

554 **Figure 1.**



555

556

Figure 2.

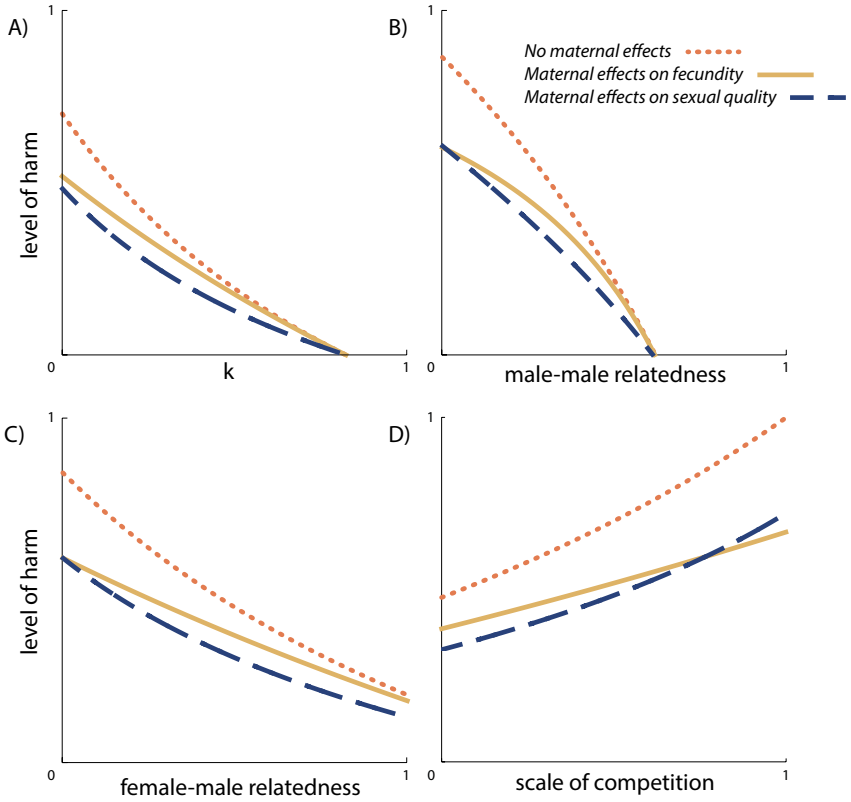
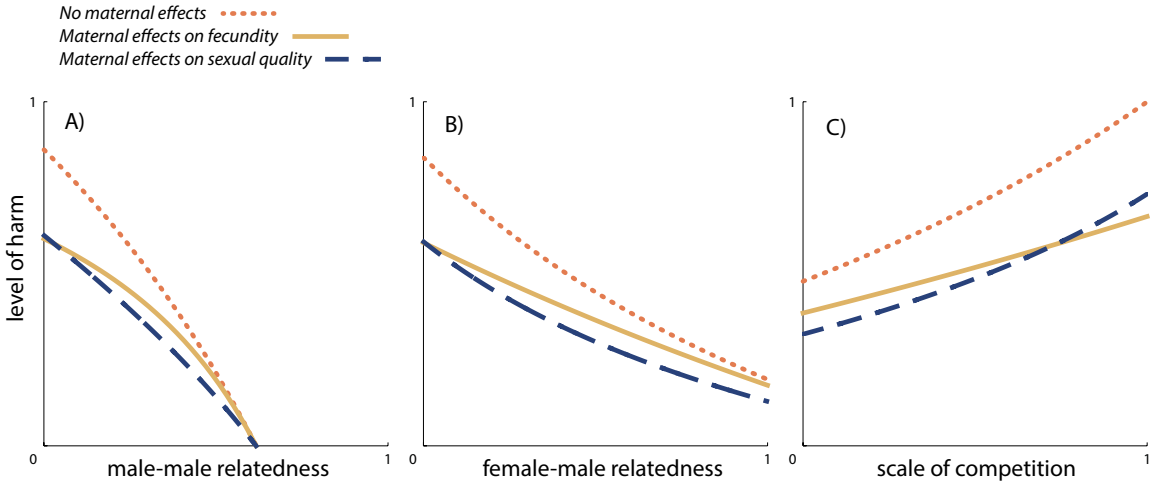

A)
B)
C)
D)
level of harm
level of harm
k
male-male relatedness
female-male relatedness
scale of competition
0
1
No maternal effects
Maternal effects on fecundity
Maternal effects on sexual quality

Figure 3.


No maternal effects
Maternal effects on fecundity
Maternal effects on sexual quality
A)
B)
C)
level of harm
male-male relatedness
female-male relatedness
scale of competition
0
1

Electronic supplementary material

1.1 Model without maternal effects

Following the model without maternal effects of the main text, monogamic female's relative fitness is:

$$W_{\mathrm{f}} = f_{\mathrm{f}}(x', y)\left(\frac{1}{a f_{\mathrm{f}}(y', y) + (1-a) f_{\mathrm{f}}(z)}\right), \quad \text{(A1)}$$

where $f_{\mathrm{f}}(y', y) = 1 - k\,y' - (1 - k)y$ is the average fecundity of local females, y' is the level of harm of the males that the local females mate with, and $f_{\mathrm{f}}(z) = 1 - z$ is the average fecundity of females in the population, which is a function of the average level of harm present in the population (z). Specifically, the focal female produces a number of offspring proportional to her fecundity $f_{\mathrm{f}}(x', y)$. Each of her offspring then competes for breeding opportunities in proportion to $a\,f_{\mathrm{f}}(y', y) + (1 - a) f_{\mathrm{f}}(z)$. Polygamic female's relative fitness in the context of the model of the main text is:

$$W_{\mathrm{f}} = f_{\mathrm{f}}(y)\left(\frac{1}{a f_{\mathrm{f}}(y) + (1-a) f_{\mathrm{f}}(z)}\right), \quad \text{(A2)}$$

A similar logic to the one described above for monogamic females applies. In the context of the present model, male's relative fitness in a monogamic population (monogamic females) is

$$W_{\mathrm{m}} = f_{\mathrm{m}}(x, y)\left(\frac{1}{a f_{\mathrm{f}}(\chi', y) + (1-a) f_{\mathrm{f}}(z)}\right), \quad \text{(A3)}$$

where:

$$f_{\mathrm{m}}(x,y) = \sum_{\mu=1}^{n_{\mathrm{f}}} \left(\frac{n_{\mathrm{f}}!}{\mu!(n_{\mathrm{f}}-\mu)} \left(\frac{f_m(x)}{n_{\mathrm{m}} f_m(y)}\right)^{\mu} \left(1 - \frac{f_m(x)}{n_{\mathrm{m}} f_m(y)}\right)^{n_{\mathrm{f}}-\mu} \mu f_{\mathrm{f}}(x,y)\right); \quad \text{(A4)}$$

$$\chi' = \frac{\mu}{n_{\mathrm{f}}} x + \frac{n_{\mathrm{f}}-\mu}{n_{\mathrm{f}}} y''; \quad \text{(A5)}$$

$f_{\mathrm{f}}(\chi',y) = 1 - k\,(\chi') - (1-k)y$; χ' is the average level of harm of the males that get to mate with the females in the social group; μ is the number of females that the focal male is able to mate with; $f_{\mathrm{f}}(x,y) = 1 - k\,x - (1-k)y$ is the fecundity of the female that the focal male mates with; and y'' is the harm of the average male in the social group excluding the focal male. Accordingly, $f_{\mathrm{f}}(\chi',y)$, which defines how male harm is affecting the average female fecundity in the social group, ranges from $1 - k\,x - (1-k)y$, if the focal male gets to mate with all the females, to $1 - k\,y'' - (1-k)y$, if the focal male does not get to mate with any of the females in the social group.

Polygamic male's relative fitness in the context of the present model is:

$$W_{\mathrm{m}} = \frac{f_{\mathrm{m}}(x)}{f_{\mathrm{m}}(y)} f_{\mathrm{f}}(y) \left(\frac{1}{a f_{\mathrm{f}}(y) + (1-a) f_{\mathrm{f}}(z)}\right). \quad \text{(A6)}$$

Specifically, a focal male compete with the other males in the social group and his success is determined by his level of harm and the level of harm of local males. Then, depending how successful the male is, he gets a share of the offspring produced by the females in the social group $f_{\mathrm{f}}(y)$. Competition in the social group then follows the logic described above for female's relative fitness.

1.2 Model with maternal effects

Regardless of the scenario considered, a monogamic high-quality female's relative fitness is:

$$W^{\mathrm{h}}{}_{\mathrm{f}} = f^{\mathrm{h}}{}_{\mathrm{f}}(x', y)\left(\frac{1}{a(\bar{f}_{\mathrm{f}}(y', y)) + (1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A7)}$$

a monogamic low-quality female's relative fitness is:

$$W^{\mathrm{l}}{}_{\mathrm{f}} = f^{\mathrm{l}}{}_{\mathrm{f}}(x', y)\left(\frac{1}{a(\bar{f}_{\mathrm{f}}(y', y)) + (1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A8)}$$

a polygamic good-quality female's relative fitness is:

$$W^{\mathrm{h}}{}_{\mathrm{f}} = f^{\mathrm{h}}{}_{\mathrm{f}}(y)\left(\frac{1}{a\bar{f}_{\mathrm{f}}(y) + (1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A9)}$$

and a polygamic low-quality female's relative fitness is:

$$W^{\mathrm{l}}{}_{\mathrm{f}} = f^{\mathrm{l}}{}_{\mathrm{f}}(y)\left(\frac{1}{a\bar{f}_{\mathrm{f}}(y) + (1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A10)}$$

where: $\bar{f}_{\mathrm{f}}(y',y) = (1-\omega)f^{\mathrm{h}}{}_{\mathrm{f}}(y',y) + \omega f^{\mathrm{l}}{}_{\mathrm{f}}(y',y)$; $\bar{f}_{\mathrm{f}}(y) = (1-\omega)f^{\mathrm{h}}{}_{\mathrm{f}}(y) + \omega f^{\mathrm{l}}{}_{\mathrm{f}}(y)$; $\bar{f}_{\mathrm{f}}(z) = (1-\Omega)f^{\mathrm{h}}{}_{\mathrm{f}}(z) + \Omega f^{\mathrm{l}}{}_{\mathrm{f}}(z)$; and ω and Ω are the levels of harm present in the social group and population in the previous generation. If we are considering the first scenario, then: $f^{\mathrm{h}}{}_{\mathrm{f}}(y',y) = 1 - k\,y' - (1-k)y$; $f^{\mathrm{l}}{}_{\mathrm{f}}(y',y) = 1 - k\,y' - (1-k)y - s$; $f^{\mathrm{h}}{}_{\mathrm{f}}(z) = 1 - z$; and $f^{\mathrm{l}}{}_{\mathrm{f}}(z) = 1 - z - s$. If we are considering the second scenario, then: $f^{\mathrm{h}}{}_{\mathrm{f}}(y',y) = 1 - k\,y' - (1-k)y$; $f^{\mathrm{l}}{}_{\mathrm{f}}(y',y) = 1 - (k\,y' + (1-k)y)(1+h)$; $f^{\mathrm{h}}{}_{\mathrm{f}}(z) = 1 - z$; and $f^{\mathrm{l}}{}_{\mathrm{f}}(z) = 1 - z(1+h)$. Accordingly, low-quality females are produced in proportion ω and Ω, depending if it is a female in the social group or the average female in the population, and high-quality females are produced in proportion $1 - \omega$ and $1 -$

641 Ω, again depending if it is a female in the social group or the average female in the

642 population. A good-quality male's relative fitness in a monogamic population is:

643

644 $$W^{\mathrm{h}}{}_{\mathrm{m}} = f^{\mathrm{h}}{}_{\mathrm{m}}(x,y)\left(\frac{1}{a\bar{f}_{\mathrm{f}}(\chi',y)+(1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A11)}$$

645

646 a low-quality male's relative fitness in a monogamic population is:

647

648 $$W^{\mathrm{l}}{}_{\mathrm{m}} = f^{\mathrm{l}}{}_{\mathrm{m}}(x,y)\left(\frac{1}{a\bar{f}_{\mathrm{f}}(\chi',y)+(1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A12)}$$

649

650 a good-quality male's relative fitness in a polygamic population is:

651

652 $$W^{\mathrm{h}}{}_{\mathrm{m}} = \frac{f^{\mathrm{h}}{}_{\mathrm{m}}(x)}{\bar{f}_{\mathrm{m}}(y)}\bar{f}_{\mathrm{f}}(y)\left(\frac{1}{a\bar{f}_{\mathrm{f}}(y)+(1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A13)}$$

653

654 and a low-quality male's relative fitness in a polygamic population is:

655

656 $$W^{\mathrm{l}}{}_{\mathrm{m}} = \frac{f^{\mathrm{l}}{}_{\mathrm{m}}(x)}{\bar{f}_{\mathrm{m}}(y)}\bar{f}_{\mathrm{f}}(y)\left(\frac{1}{a\bar{f}_{\mathrm{f}}(y)+(1-a)\bar{f}_{\mathrm{f}}(z)}\right); \quad \text{(A14)}$$

657

658 where:

659

660 $$f^{\mathrm{h}}{}_{\mathrm{m}}(x,y) = \sum_{\mu=1}^{n_{\mathrm{f}}}\left(\frac{n_{\mathrm{f}}!}{\mu!(n_{\mathrm{f}}-\mu)}\left(\frac{f^{\mathrm{h}}{}_{\mathrm{m}}(x)}{n_{\mathrm{m}}\bar{f}_{\mathrm{m}}(y)}\right)^{\mu}\left(1-\frac{f^{\mathrm{h}}{}_{\mathrm{m}}(x)}{n_{\mathrm{m}}\bar{f}_{\mathrm{m}}(y)}\right)^{n_{\mathrm{f}}-\mu}\mu_{\mathrm{f}}\bar{f}_{\mathrm{f}}(x,y)\right); \quad \text{(A15)}$$

661

662 $$f^{\mathrm{l}}{}_{\mathrm{m}}(x,y) = \sum_{\mu=1}^{n_{\mathrm{f}}}\left(\frac{n_{\mathrm{f}}!}{\mu!(n_{\mathrm{f}}-\mu)}\left(\frac{f^{\mathrm{l}}{}_{\mathrm{m}}(x)}{n_{\mathrm{m}}\bar{f}_{\mathrm{m}}(y)}\right)^{\mu}\left(1-\frac{f^{\mathrm{l}}{}_{\mathrm{m}}(x)}{n_{\mathrm{m}}\bar{f}_{\mathrm{m}}(y)}\right)^{n_{\mathrm{f}}-\mu}\mu_{\mathrm{f}}\bar{f}_{\mathrm{f}}(x,y)\right); \quad \text{(A16)}$$

663

$\bar{f}_{\mathrm{f}}(\chi',y) = (1-\omega)f^{\mathrm{h}}_{\mathrm{f}}(\chi',y) + \omega f^{\mathrm{l}}_{\mathrm{f}}(\chi',y)$; $\bar{f}_{\mathrm{m}}(y) = (1-\omega)f^{\mathrm{h}}_{\mathrm{m}}(y) + \omega f^{\mathrm{l}}_{\mathrm{m}}(y)$; and $\bar{f}_{\mathrm{f}}(x,y) = (1-\omega)f^{\mathrm{h}}_{\mathrm{f}}(x,y) + \omega f^{\mathrm{l}}_{\mathrm{f}}(x,y)$. If we are considering the first scenario, then: $f_{\mathrm{hf}}(\chi',y) = 1 - k\chi' - (1-k)y$; $f_{\mathrm{lf}}(\chi',y) = 1 - k\,\chi' - (1-k)y - s$; $f_{\mathrm{hf}}(x,y) = 1 - k\,x - (1-k)y$; $f_{\mathrm{lf}}(x,y) = 1 - k\,x - (1-k)y - s$; $f_{\mathrm{hm}}(y) = 1 + y$; $f_{\mathrm{lm}}(y) = 1 + y - s$; and $\chi' = (\mu/n_{\mathrm{f}})x + ((n_{\mathrm{f}}-\mu)/n_{\mathrm{f}})y''$. If we are considering the second scenario, then: $f_{\mathrm{hf}}(\chi',y) = 1 - k\,\chi' - (1-k)y$; $f_{\mathrm{lf}}(\chi',y) = 1 - (k\,\chi' - (1-k)y)(1+h)$; $f_{\mathrm{hf}}(x,y) = 1 - k\,x - (1-k)y$; $f_{\mathrm{lf}}(x,y) = 1 - (k\,x - (1-k)y)(1+h)$; $f_{\mathrm{hm}}(y) = 1 + y$; $f_{\mathrm{lm}}(y) = 1 + y(1-t)$; $\chi' = (\mu/n_{\mathrm{f}})x + ((n_{\mathrm{f}}-\mu)/n_{\mathrm{f}})((1-\omega)y'' + \omega(y''(1-t))$ if the focal male is a good-quality male; and $\chi' = (\mu/n_{\mathrm{f}})x(1-t) + ((n_{\mathrm{f}}-\mu)/n_{\mathrm{f}})((1-\omega)y'' + \omega(y''(1-t))$ if the focal male is a low-quality male. Accordingly, low-quality individuals are produced in proportion ω and Ω, depending on if it is an individual in the social group or the average individual in the population, and high-quality individuals are produced in proportion $1-\omega$ and $1-\Omega$, again depending on if it is an individual in the social group or the average individual in the population.

2 Taylor-Frank Approach

We assume that natural selection favours genes associated with greater individual relative fitness (Fisher 1930; Price 1970). Assuming vanishingly little genetic variation, this condition may be expressed using the mathematics of differential calculus: $\mathrm{d}W/\mathrm{d}g > 0$, 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 (Taylor 1996). The appropriate measure of relative fitness is a class-reproductive-value-weighted average taken across females and males. Furthermore, when maternal effects are present, two other classes of individuals exist, specifically good-quality and low-quality individuals.

Accordingly, in the absence of maternal effects and assuming monogamy, the absolute fitness of an individual is w_i, where i = f when the focal individual is a female and i = m when the focal individual is a male. Relative fitness is, therefore, $W_i = ½(w_f/\overline{w}_f) + ½(w_m/\overline{w}_m)$ where $\overline{w}_i$ is the average absolute fitness of the sex i in the population. Reproductive value weights are identical for females and males (therefore, ½). Female relative fitness is $W_f = w_f/\overline{w}_f$ and male relative fitness is $W_m = w_m/\overline{w}_m$. Following the approach of Taylor & Frank (1996) for a class-structured population, and in the context of the model present in the main text without maternal effects, we may write $dW_i/dg = ½ (dW_f/dg_f) + ½ (dW_m/dg_m) = ½ ((\partial W_f/\partial x')(dx'/dG_m')(dG_m'/dg_f) + (\partial W_f/\partial y)(dy/dG_m')(dG_m'/dg_f) + (\partial W_f/\partial y')(dy'/dG_m')(dG_m'/dg_f)) + ½ ((\partial W_m/\partial x)(dx/dG_m)(dG_m/dg_m) + (\partial W_m/\partial y)(dy/dG_m')(dG_m'/dg_m) + (\partial W_m/\partial \chi')(d\chi'/dG_m')(dG_m'/dg_m))$, 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_m is the focal male's breeding value; G_m' is the average breeding value of local males; $dx/dG_m' = dx'/dG_m' = dy/dG_m' = dy'/dG_m = d\chi'/dG_m' = \gamma_m$ is the mapping between genotype and phenotype in the males (females do not express the gene); $dG_m'/dg_f = p_{fm}$ is the consanguinity of the gene in the focal female with a randomly-chosen local male; $dG_m/dg_m = p_m$ is the consanguinity of the gene in the focal male to the male himself; and $dG_m'/dg_m = p_{mm}$ is the consanguinity of the gene in the focal male with a randomly-chosen local male. We divide all the terms of the right side of the equation by p_m to get the kin-selection coefficient of relatedness (Bulmer 1994). We assume that harm is only expressed by the males and that their genes are in full control of the phenotype. Accordingly, $\gamma_m = 1$. Therefore, natural selection favours an increase in the level of harm that males express when:

$$\frac{1}{2} r_{fm} \left(\frac{\partial W_f}{\partial x'} + \frac{\partial W_f}{\partial y} + \frac{\partial W_f}{\partial y'} \right) + \frac{1}{2} \left(\frac{\partial W_m}{\partial x} + r_{mm} \left(\frac{\partial W_m}{\partial y} + \frac{\partial W_m}{\partial \chi'} \right) \right) > 0. \quad \text{(A17)}$$

Equalling inequality A17 to 0, evaluating the derivatives at $x = x' = y = y' = \chi' = z = z^*$, with z^* being the optimal level of harm in the population, returns the optimal fitness equation for the model present in the main text.

In the presence of maternal effects and assuming monogamy, the absolute fitness of an individual is $w^{\mathrm{k}}_{\mathrm{i}}$, where i = f when the focal individual is a female, i = m when the focal individual is a male, k = h when the focal individual is a good-quality individual, and k = l when the focal individual is a low-quality individual. Relative fitness is, therefore, $W_{\mathrm{i}} = ½((1-\omega)(w^{\mathrm{h}}_{\mathrm{f}}/\bar{w}_{\mathrm{f}}) + \omega(w^{\mathrm{l}}_{\mathrm{f}}/\bar{w}_{\mathrm{f}})) + ½((1-\omega)(w^{\mathrm{h}}_{\mathrm{m}}/\bar{w}_{\mathrm{m}}) + \omega(w^{\mathrm{l}}_{\mathrm{m}}/\bar{w}_{\mathrm{m}}))$. Following the approach of Taylor & Frank (1996) for a class-structured population, and in the context of the model present in the main text without maternal effects, we may write $\mathrm{d}W_{\mathrm{i}}/\mathrm{d}g = ½\,(\mathrm{d}W^{\mathrm{k}}_{\mathrm{f}}/\mathrm{d}g_{\mathrm{f}}) + ½\,(\mathrm{d}W^{\mathrm{k}}_{\mathrm{m}}/\mathrm{d}g_{\mathrm{m}}) = ½((1-\omega)\,((\partial W^{\mathrm{h}}_{\mathrm{f}}/\partial x')(\mathrm{d}x'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{h}}_{\mathrm{f}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{h}}_{\mathrm{f}}/\partial y')(\mathrm{d}y'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}})) + \omega((\partial W^{\mathrm{l}}_{\mathrm{f}}/\partial x')(\mathrm{d}x'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{l}}_{\mathrm{f}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{l}}_{\mathrm{f}}/\partial y')(\mathrm{d}y'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}))) + ½\,((1-\omega)((\partial W^{\mathrm{h}}_{\mathrm{m}}/\partial x)(\mathrm{d}x/\mathrm{d}G_{\mathrm{m}})(\mathrm{d}G_{\mathrm{m}}/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{h}}_{\mathrm{m}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{h}}_{\mathrm{m}}/\partial \chi')(\mathrm{d}\chi'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}})) + \omega((\partial W^{\mathrm{l}}_{\mathrm{m}}/\partial x)(\mathrm{d}x/\mathrm{d}G_{\mathrm{m}})(\mathrm{d}G_{\mathrm{m}}/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{l}}_{\mathrm{m}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{l}}_{\mathrm{m}}/\partial \chi')(\mathrm{d}\chi'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}})))$. The terms are similar to the ones presented above for the model that does not consider maternal effects. We divide all the terms of the right side of the equation by p_{m} to get the kin-selection coefficient of relatedness (Bulmer 1994). We assume that harm is only expressed by the males and that their genes are in full control of the phenotype. Therefore, natural selection favours an increase in the level of harm that males express when:

$$\frac{1}{2}r_{\mathrm{fm}}((1-\omega)\left(\frac{\partial W_{\mathrm{f}}^{\mathrm{h}}}{\partial x'}+\frac{\partial W_{\mathrm{f}}^{\mathrm{h}}}{\partial y}+\frac{\partial W_{\mathrm{f}}^{\mathrm{h}}}{\partial y'}\right)+\omega\left(\frac{\partial W_{\mathrm{f}}^{\mathrm{l}}}{\partial x'}+\frac{\partial W_{\mathrm{f}}^{\mathrm{l}}}{\partial y}+\frac{\partial W_{\mathrm{f}}^{\mathrm{l}}}{\partial y'}\right))+\frac{1}{2}\Bigg((1-\omega)\frac{\partial W_{\mathrm{m}}^{\mathrm{h}}}{\partial x}+\omega\frac{\partial W_{\mathrm{m}}^{\mathrm{l}}}{\partial x}+$$

$$r_{\mathrm{mm}}((1-\omega)\left(\frac{\partial W_{\mathrm{m}}^{\mathrm{h}}}{\partial y}+\frac{\partial W_{\mathrm{m}}^{\mathrm{h}}}{\partial \chi'}\right)+\omega\left(\frac{\partial W_{\mathrm{m}}^{\mathrm{l}}}{\partial y}+\frac{\partial W_{\mathrm{m}}^{\mathrm{l}}}{\partial \chi'}\right))\Bigg)>0. \quad \text{(A18)}$$

Equalling inequality A18 to 0, evaluating the derivatives at $x = x' = y = y' = \chi' = z = \omega = \Omega = z^*$, with z^* being the optimal level of harm in the population, returns the optimal fitness equation for the model present in the main text.

The models that consider polygamy are a subset of the models presented above. Specifically, in the absence of maternal effects, natural selection favours an increase in the level of harm that males express when

$$\frac{1}{2}r_{\mathrm{fm}}\left(\frac{\partial W_{\mathrm{f}}}{\partial y}\right)+\frac{1}{2}\left(\frac{\partial W_{\mathrm{m}}}{\partial x}+r_{\mathrm{mm}}(\frac{\partial W_{\mathrm{m}}}{\partial y})\right)>0, \quad \text{(A19)}$$

and in the presence of maternal effects, natural selection favours an increase in the level of harm that males express when

$$\frac{1}{2}r_{\mathrm{fm}}((1-\omega)\frac{\partial W_{\mathrm{f}}^{\mathrm{h}}}{\partial y}+\omega\frac{\partial W_{\mathrm{f}}^{\mathrm{l}}}{\partial y})+\frac{1}{2}\left((1-\omega)\frac{\partial W_{\mathrm{m}}^{\mathrm{h}}}{\partial x}+\omega\frac{\partial W_{\mathrm{m}}^{\mathrm{l}}}{\partial x}+r_{\mathrm{mm}}((1-\omega)\frac{\partial W_{\mathrm{m}}^{\mathrm{h}}}{\partial y}+\omega\frac{\partial W_{\mathrm{m}}^{\mathrm{l}}}{\partial y})\right)>$$

$$0. \quad \text{(A20)}$$

Then we can use a similar approach to the one described above for monogamy to obtain the optimal fitness equations present in the main text.

3 References

761

762 Bulmer, M. G. Theoretical evolutionary ecology. MA: Sinauer Associates, Sunderland

763 (1994).

764

765 Fisher, R. A. The genetical theory of natural selection. Claredon Press, Oxford (1930).

766 Price, G. R. Selection and covariance. *Nature* **227**, 520-521 (1970).

767 Taylor, P. D. Inclusive fitness arguments in genetic models of behaviour. *J. Math. Biol.* **34**,

768 654-674 (1996)

769 Taylor, P.D. & Frank, S.A. 1996. How to make a kin selection model. J. Theor. Biol. 180:

770 27–37.

2. Systematic review

Methods– We conducted a systematic review of the existing literature following the PRISMA protocol (Liberati *et al.* 2009). 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 (Reinhardt *et al.* 2015). We thus opted to include both cases where the consequences of male harm were measured in terms of female fitness (i.e. quantitative evidence; see SM) and 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 (i.e. qualitative evidence).

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

796 and WoS = 300). After removing 373 duplicates, we exported 321 to Rayyan. We conducted
797 a final search on the 7/04/20 using the search terms: “sexual conflict” & harassment. We
798 found a total of 414 items (Scopus = 175, PubMed = 50 and WoS = 189). After removing 175
799 duplicates, we exported 239 to Rayyan. In Rayyan, we checked for duplicates within the
800 complete database comprising all the papers located via these three searches and removed 69
801 duplicates, leading to 527 unique studies for more detailed screening. Based on the title and
802 abstract we excluded 347 papers that clearly did not report adaptations for male harm, leaving
803 a total of 180 papers for in-depth screening.
804 We carefully screened these papers and excluded papers that did not comply with our
805 selection criteria described above. In the process of screening, we added 27 more papers
806 through forward and backward searches of citations and references, leading to a total final
807 sample of selected studies reporting male harm adaptations for a total of 87 different species
808 (see SM for complete list). Finally, for all these species we performed specific searches on
809 their mode of reproduction (i.e. ovipary, vivipary or ovovivipary), evidence for parental care
810 and evidence of extended maternal provisioning (i.e. matrotrophy, including brooding).
811 Similar data for broad taxonomic groups taxa included in Figure 2 were taken from key
812 references provided in the supplementary materials.
813
814 ***Results***– In total, we obtained evidence of male harm to females for a total of 87
815 species (see Figure S1 and S2 and accompanying data); 51 insects, 7 arachnids, 7 birds, 6
816 fish, 5 mammals, 4 gastropods, 2 amphibians, 2 reptiles, 1 shark, 1 crustacean, and 1
817 nematode. For most of these species (48) studies reported qualitative evidence of male harm
818 (i.e. some evidence of harm to females), while quantitative evidence (i.e. estimation of the
819 degree to which female fitness decreases with male harm) was only reported for 39 species
820 (see accompanying data for details). Overall, the overwhelming majority of these 87 species

821 exhibited little scope for maternal effects. Namely, some form of parental care has been
822 described for only 18 of these species (11 of which are vertebrates), while only 11 are
823 viviparous (all vertebrates) and only 5 (all vertebrates) exhibit some form of extended
824 maternal provision (vs. lecithotrophy). Such restricted variability, particularly across
825 invertebrates, precluded a formal meta-analysis to explore the relationship between the
826 existence and/or intensity of male harm and the scope for maternal effects. It is important to
827 note that the evidence described above is obviously correlative, and hence inadequate to
828 sustain a causal relationship. Furthermore, the lack of variation in the collected proxies for
829 the scope of maternal effects prevented a formal meta-analysis relating these variables with
830 the level of male harm (i.e. drop in female fitness due to male harm). Similarly, we wish to
831 stress that our measure of the scope for maternal effects is unavoidably coarse. For example,
832 species that have long gestational periods can exhibit complex adaptations to buffer offspring
833 from effects of mothers (e.g. placentas, or ability to metabolize maternal hormones), so
834 longer gestational periods do not necessarily equal "more" maternal effects. Finally,
835 phylogenetic signal within groups is expected to be strong due to other factors (e.g. specific
836 maternal effects adaptations, sperm competition levels or the opportunity for selection). Thus,
837 we stress that conclusions to this respect must be taken as completely preliminary, at best.
838
839 **Figure S1 | Distribution of male harm adaptations across the tree of life.**
840 Results from taxa that encompass the 87 species for which we found evidence of male harm.
841 Outward-facing circles in the red shaded area represent the presence of direct (red) and/or
842 collateral (orange) adaptations leading to male harm of females (i.e. one for each species).
843 Inner-facing circles in the green shaded area reflect evidence in the literature of parental care,
844 extended gestation (viviparity/ovoviviparity vs. oviparity) and extended maternal
845 provisioning (matrotophy/placentotrophy/brooding vs. lecithotrophy) in the taxon. The size

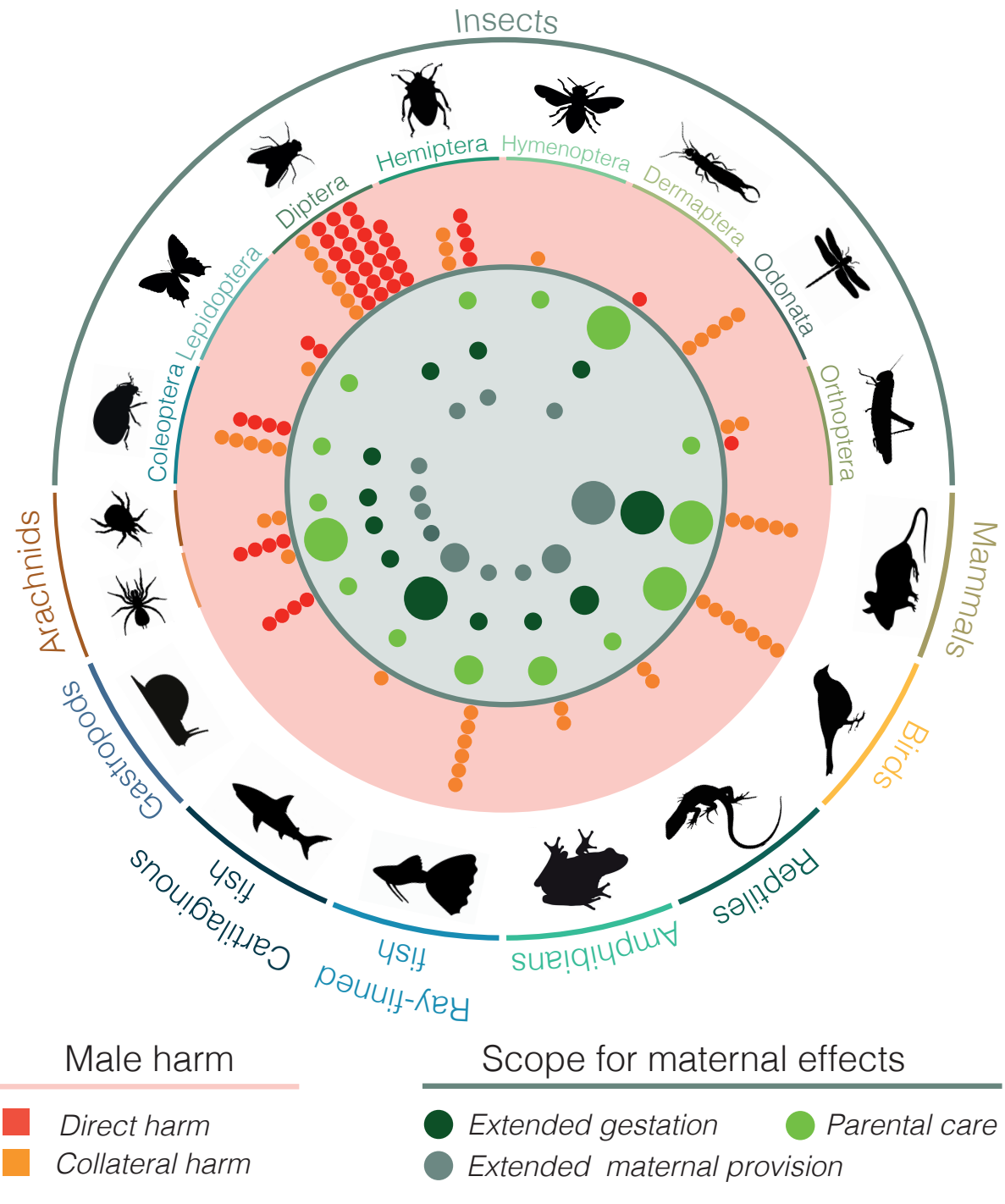
of inward-facing green circles illustrates how widespread these strategies are according to the literature: large circles denote a widespread strategy (i.e. adopted by most or all known species), medium-size circles represent a common strategy (i.e. more than 5% but less than 50% of known species) and small-sizes circles represent a rare strategy (i.e. < 5% known species). We excluded two species (i.e. *Caenorhabditis ramnei* – Nematode– and *Idotea balthica* –Isopod–) from the figure due to non-available data for maternal effects (*I. balthica*) or the type of male harm (*C. ramnei*). See accompanying data for details.

Figure S2 | Male harm and scope for maternal effects

A) Percentage of the 87 species for which parental care (light green), extended gestation (dark green) and extended maternal provision (grey), respectively from left to right, has been reported in the literature. B) Summary of indirect evidence (i.e. mostly based on descriptions of male genitalia; see methods and SM) 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 (Řezáč 2009; Mouginot *et al.* 2015; Nakata 2016). 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 (Řezáč 2009; Mouginot *et al.* 2015; Nakata 2016). The numbers beside red bars represent the number of species within the group that are estimated to exhibit such male harm adaptations. Green-shaded circles reflect evidence in the literature of parental care, extended gestation and extended maternal provisioning in the taxon; respectively, from left to right. See accompanying data for details.

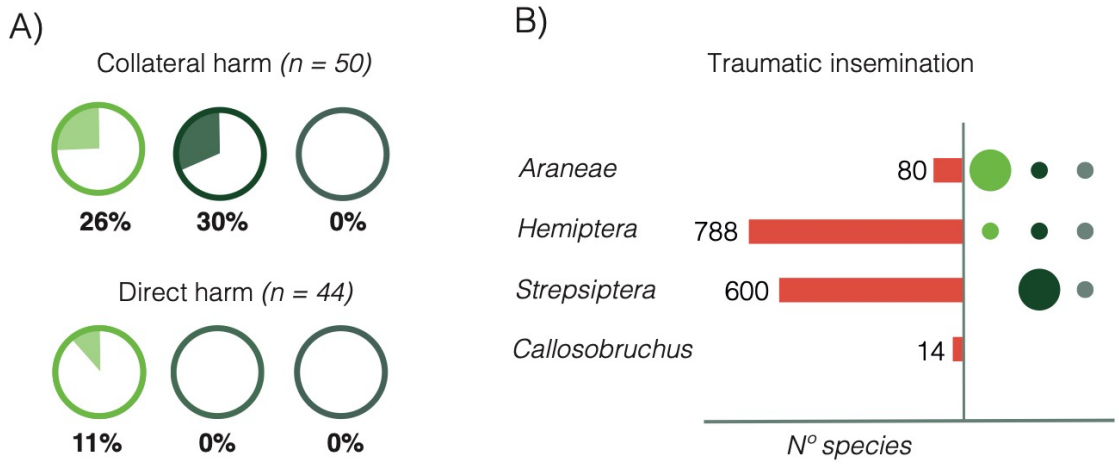
868 **Figure S1**.

869



870

871 **Figure S2**.



872

873

874