Motherly love curbs harm: maternal effects modulate sexual conflict

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Running title: Maternal effects curb sexual conflict

Article Type: Letter

Word count: 150 (abstract) and 4375 (main text); **Figures:** 3; **References:** 54.

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

Statement of authorship: PC and RG-R conceived this study. GF, DN, RG-R & PC designed the study. GF developed the mathematical models and Figure 1. DN PC & RG-R performed the systematic literature search. DN explored the data for potential meta-analysis. PC prepared Figures 2 and 3. PC wrote the manuscript with contributions by GF, RG-R and DN.

Data accessibility statement: Should the manuscript be accepted, the data supporting the results presented will be deposited in a public repository and the data DOI will be included at the end of the article.

Abstract

Strong sexual selection frequently favours males that increase their reproductive success by harming females, with potentially negative consequences for population growth. Understanding what factors modulate conflict between the sexes is hence critical to unravel the evolution and viability of populations in the wild. 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, because male harm can induce 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 population structure and the type of male harm, 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 (Andersson 1994). This, in turn, can trigger sexually antagonistic coevolution (Parker 1979; Holland & Rice 1998) 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 (Arnqvist & Rowe 2005). At a population level, it 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" (Rankin *et al.* 2011). From male harassment and coercion (Han & Jablonski 2010; Perry & Rowe 2015a) to toxic ejaculates (Wigby & Chapman 2005) and traumatic insemination (Crudgington & Siva-Jothy 2000; Reinhardt *et al.* 2015) , 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 (Rankin 2011; Carazo *et al.* 2014; Faria *et al.* 2015; 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 & Snook 2003; Maklakov *et al.* 2005; Parker 2006; Garcia-Gonzalez & Simmons 2010; Brennan & Prum 2012). 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

65 fecundity. On the other hand, females may obtain indirect genetic benefits by mating with
66 particularly harmful or manipulative males because their own male offspring will inherit
67 these genes, albeit theoretical and empirical evidence shows indirect genetic benefits are
68 generally weaker than direct benefits (Cameron *et al.* 2003; Pizzari & Snook 2003; Parker
69 2006). However, such studies do not consider maternal effects as an important factor in the
70 fitness payoff of male harm evolution. Maternal effects can drastically modulate offspring
71 quality (Mousseau & Fox 1998) and are largely mediated by maternal condition (Rossiter
72 1996; Saino *et al.* 2005). Male harm can severely impact female condition (Arnqvist & Rowe
73 2005) and, although its transgenerational effects have only been studied in a handful of
74 species, it can induce maternal effects that reduce the quality of a male's own offspring
75 (Tregenza *et al.* 2003; Brommer *et al.* 2012; Gasparini *et al.* 2012; Dowling *et al.* 2014;
76 Carazo *et al.* 2015; Zajitschek *et al.* 2018). For example, female guppies (*Poecilia reticulata*)
77 exposed to greater harassment produce smaller daughters and sons with shorter gonopodia
78 (Gasparini *et al.* 2012). We thus posit that male harm can induce maternal effects that reduce
79 the quality of a harming male's own offspring and, in doing so, bring together male and
80 female interests and abate sexual conflict independently from kin selection.

81 In order to test this idea, we use a personal-fitness kin-selection approach (Hamilton
82 1964a,b; Taylor & Frank 1996) to investigate the potential for maternal effects to bring
83 together male and female interest and, therefore, be an important factor in the evolution of
84 male harm to females. We analyse three scenarios: a) absence of maternal effects on
85 offspring quality, b) presence of maternal effects on the offspring fecundity (females) and
86 competitiveness (males), and c) presence of maternal effects on offspring's ability to inflict
87 (males) and resist (females) harm (i.e. sexual selection quality). We apply this approach to
88 cases in which males harm females while competing for access to matings (male harassment)
89 and to cases in which males harm females as a result of male-male post-copulatory

competition (traumatic inseminations). Finally, to assess whether theoretical predictions fit with available data, 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: a) parental care, b) extended gestation and c) extended maternal provision.

Methods

1.1 Model without maternal effects

We consider an infinite diploid 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 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. Accordingly, a focal female's fecundity $f_f(y',Y) = 1 - k\,y' - (1 - k)Y$ is a function of the level of harm of the male that she mates with (y') 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 ($k = 1$; i.e., traumatic insemination) or before mating as a result of male competition to access females ($k = 0$; i.e., male harassment). A focal male's competitiveness for mating success $f_m(y) = 1 + y$ is a function of the level of harm expressed by that focal male (y). After mating, 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 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. Finally, n_f females and n_m males survive at random

115 within each social group to adulthood, returning the population to the beginning of the
116 lifecycle.
117 Female's relative fitness in the context of the present model is given by:
118
119 $$W_{\mathrm{f}} = f_{\mathrm{f}}(y', Y)\left(\frac{1}{a f_{\mathrm{f}}(Y', Y) + (1-a) f_{\mathrm{f}}(\bar{Y})}\right), \quad (1)$$
120
121 where $f_{\mathrm{f}}(Y', Y) = 1 - k\,Y' - (1-k)Y$ is the average fecundity of local females, Y' is the level of
122 harm of the males that the local females mate with, and $f_{\mathrm{f}}(\bar{Y}) = 1 - \bar{Y}$ is the average fecundity
123 of females in the population, which is a function of the average level of harm present in the
124 population ($\bar{Y}$). Specifically, the focal female produces a number of offspring proportional to
125 her fecundity $f_{\mathrm{f}}(y', Y)$. Each of her offspring then competes for breeding opportunities in
126 proportion to $a\,f_{\mathrm{f}}(Y', Y) + (1-a) f_{\mathrm{f}}(\bar{Y})$. Male's relative fitness in the context of the present
127 model is given by:
128
129 $$W_{\mathrm{m}} = f_{\mathrm{m}}(y, Y)\left(\frac{1}{a f_{\mathrm{f}}(Y'', Y) + (1-a) f_{\mathrm{f}}(\bar{Y})}\right), \quad (2)$$
130
131 where $f_{\mathrm{m}}(y, Y)$ is the mating success of the focal male which is a function of the focal male's
132 level of male harm (y) and the average level of male harm in the social group (Y; see
133 Supplementary Material for details and for full derivation), and $f_{\mathrm{f}}(Y'', Y) = 1 - k\,(Y'') - (1 -$
134 $k)Y$ is how male harm is affecting the average female fecundity in the social group (where Y''
135 is the average level of harm of the males that get to mate with the females in the social group;
136 see Supplementary Material for details and for full derivation). Competition in social group
137 then follows the same logic described above for female's relative fitness.
138
139 *1.2 Model with maternal effects*

As in section 1.1, 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. 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. Low-quality individuals are produced in proportion to the harm that their mother received.

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 males present in the social group minus a cost s due to her low quality $f_{lf}(y',Y) = 1 - k\,y' - (1 - k)Y - s$; 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 level of harm of the male that she mates with and of the average level of male harm males present in the social group $f_{hf}(y',Y) = 1 - k\,y' - (1 - k)Y$; and a focal high-quality male's competitiveness for mating success is a function of the level of harm expressed by that focal male $f_{hm}(y) = 1 + y$.

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 males present in the social group multiplied by $1 + h$ due to her low quality $f_{lf}(y',Y) = 1 - (k\,y' + (1 - k)Y)(1 + h)$; 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 $f_{lm}(y) = 1 + y(1 - t)$; 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 males present in the social group $f_{hf}(y',Y) = 1 - k\,y' - (1 - k)Y$; and a focal high-quality male's

competitiveness for mating success is a function of the level of harm expressed by that focal male $f_{hm}(y) = 1 + y$.

Regardless of the scenario considered, female's relative fitness is then given by:

$$W_f = \bar{f}_f(y', Y)\left(\frac{1}{a\bar{f}_f(Y', Y) + (1-a)\bar{f}_f(\bar{Y})}\right), \quad (3)$$

where: $\bar{f}_f(y', Y) = (1 - Y_0)f_{hf}(y', Y) + Y_0 f_{lf}(y', Y)$; $\bar{f}_f(Y', Y) = (1 - Y_0)f_{hf}(Y', Y) + Y_0 f_{lf}(Y', Y)$; $\bar{f}_f(\bar{Y}) = (1 - \bar{Y}_0)f_{hf}(\bar{Y}) + \bar{Y}_0 f_{lf}(\bar{Y})$; and Y_0 and $\bar{Y}$ are the levels of harm present in the social group and population in the previous generation. If we are considering the first scenario, then: $f_{hf}(Y', Y) = 1 - k\,Y' - (1 - k)Y$; $f_{lf}(Y', Y) = 1 - k\,Y' - (1 - k)Y - s$; $f_{hf}(\bar{Y}) = 1 - \bar{Y}$; and $f_{lf}(\bar{Y}) = 1 - \bar{Y} - s$. If we are considering the second scenario, then: $f_{hf}(Y', Y) = 1 - k\,Y' - (1 - k)Y$; $f_{lf}(Y', Y) = 1 - (k\,Y' + (1 - k)Y)(1 + h)$; $f_{hf}(\bar{Y}) = 1 - \bar{Y}$; and $f_{lf}(\bar{Y}) = 1 - \bar{Y}(1 + h)$. Accordingly, low-quality females are produced with probability Y_0 and $\bar{Y}_0$, depending if it is a female in the social group or the average female in the population, and high-quality females are produced with probability $1 - Y_0$ and $1 - \bar{Y}_0$, again depending if it is a female in the social group or the average female in the population. Male's relative fitness is given by:

$$W_m = \bar{f}_m(y, Y)\left(\frac{1}{a\bar{f}_f(Y'', Y) + (1-a)\bar{f}_f(\bar{Y})}\right), \quad (4)$$

where: $\bar{f}_m(y, Y)$ is the mating success of the focal male which is a function of the focal male's level of male harm (y) and the average level of male harm in the social group (Y) and considers that low-quality males are produced in proportion to Y_0 and high-quality males are produced in proportion to $1 - Y_0$ (see Supplementary Material for details and for full derivation); and $\bar{f}_f(Y'', Y) = (1 - Y_0)f_{hf}(Y'', Y) + Y_0 f_{lf}(Y'', Y)$. If we are considering the first

scenario, then: $f_{hf}(Y'',Y) = 1 - k\,Y'' - (1-k)Y$; and $f_{lf}(Y'',Y) = 1 - k\,Y'' - (1-k)Y - s$. If we are considering the second scenario, then: $f_{hf}(Y'',Y) = 1 - k\,Y'' - (1-k)Y$; and $f_{lf}(Y'',Y) = 1 - (k\,Y'' + (1-k)Y)(1+h)$. Competition in social group then follows the same logic described above for female's relative fitness.

1.3 Model analysis

The method to analyse the models above is the same regardless of the model that we are considering. Following Taylor-Frank approach (see SM for details), natural selection favours an increase in the level of harm that males express when:

$$\frac{1}{2} r_{\mathrm{fm}} \left(\frac{\partial W_{\mathrm{f}}}{\partial y'} + \frac{\partial W_{\mathrm{f}}}{\partial Y} + \frac{\partial W_{\mathrm{f}}}{\partial Y'} \right) + \frac{1}{2} \left(\frac{\partial W_{\mathrm{m}}}{\partial y} + r_{\mathrm{mm}} \left(\frac{\partial W_{\mathrm{m}}}{\partial Y} + \frac{\partial W_{\mathrm{m}}}{\partial Y''} \right) \right) > 0 \qquad (5)$$

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 $y = y' = Y = Y' = Y'' = Y_0 = \bar{Y}_0 = \bar{Y} = \bar{Y}^*$; and $\bar{Y}^*$ is the optimal level of harm in the population.

1.4 Systematic review

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

213 adaptations may not be expected to impose high fitness costs in females over most
214 evolutionary time (Reinhardt *et al.* 2015). We thus opted to include both cases where the
215 consequences of male harm were measured in terms of female fitness (i.e. quantitative
216 evidence; see SM) and cases in which lifetime/reproductive fitness costs to females were not
217 studied but male adaptations involved produced measurable harm to females (i.e. injuries),
218 such as in traumatic insemination via genital ablation or copulatory wounding, or in cases
219 where male harassment regularly leads to female injuries and occasional deaths (i.e.
220 qualitative evidence).

221 We conducted a first literature search on 03/04/20 using the Scopus, PubMed and
222 Web of Science (WoS) databases with the search terms "sexual conflict" & "male harm" OR
223 "sexual conflict" & "female harm" for animal taxa. Overall, very few papers were found with
224 these search strings (73 total: Scopus = 31, PubMed = 15 and WoS = 27). After removing
225 duplicates only 36 papers were relevant, and we exported them to Rayyan. We conducted a
226 second literature search on 03/04/20 using the Scopus, PubMed and Web of Science (WoS)
227 databases with the search terms: "sexual conflict" & "female fitness" OR "sexual conflict" &
228 "female productivity" OR "sexual conflict" & "female fecundity" OR "sexual conflict" &
229 "female reproductive success". We found a total of 694 papers (Scopus = 250, PubMed = 144
230 and WoS = 300). After removing 373 duplicates, we exported 321 to Rayyan. We conducted
231 a final search on the 7/04/20 using the search terms: "sexual conflict" & harassment. We
232 found a total of 414 items (Scopus = 175, PubMed = 50 and WoS = 189). After removing 175
233 duplicates, we exported 239 to Rayyan. In Rayyan, we checked for duplicates within the
234 complete database comprising all the papers located via these three searches and removed 69
235 duplicates, leading to 527 unique studies for more detailed screening. Based on the title and
236 abstract we excluded 347 papers that clearly did not report adaptations for male harm, leaving
237 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 27 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 87 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 2 were taken from key references provided in the supplementary materials.

Results

Model– Using the analysis described above for our mathematical model, we calculate the marginal fitness equation for the evolution of male harm by setting the left-side of the inequality (5) equal to 0 and performing the derivatives. Assuming that $n_f = n_m$ (i.e. the number of females and males are the same in each social group), we obtain:

$$\left(\frac{1}{1+\bar{Y}^*}-\frac{k}{1-\bar{Y}^*}\right)(1-r_{mm})-\frac{1}{1-\bar{Y}^*}(r_{fm}+r_{mm})(1-a)=0, \quad (6)$$

for the model without maternal effects;

$$\left(\frac{1}{1+\bar{Y}^*-s\bar{Y}^*}-\frac{k}{1-\bar{Y}^*-s\bar{Y}^*}\right)(1-r_{mm})-\frac{1}{1-\bar{Y}^*-s\bar{Y}^*}(r_{fm}+r_{mm})(1-a)=0, \quad (7)$$

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

$$\left(\frac{1-t\bar{Y}^*}{1+\bar{Y}^*(1-t\bar{Y}^*)}-\frac{k(1+h\bar{Y}^*)}{1-\bar{Y}^*(1+h\bar{Y}^*)}\right)(1-r_{\mathrm{mm}})-\frac{1+h\bar{Y}^*}{1-\bar{Y}^*(1+h\bar{Y}^*)}(r_{\mathrm{fm}}+r_{\mathrm{mm}})(1-a)=0. \quad (8)$$

for the model with maternal effects on the ability to inflict (males) and resist harm (females). 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 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 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 local competition ($1 - a$), that is, how much individuals compete with local social group mates for reproductive resources (Taylor 1992).

While the results are similar across the different models, there are important quantitative differences. Specifically, the harm benefits are smaller, and costs are higher, when maternal effects are present, and more so when maternal effects influence the ability to inflict (males) or resist (females) harm (Figure 1). This happens regardless of harm coming before (i.e. male harassment) or during (i.e. traumatic insemination) the mating act (Figure 1A), or relatedness levels (Figure B&C).

Systematic literature search– In total, we obtained evidence of male harm to females for a total of 87 species (see Figures 2 and 3, and SM for details); 51 insects, 7 arachnids, 7 birds, 6 fish, 5 mammals, 4 gastropods, 2 amphibians, 2 reptiles, 1 shark, 1 crustacean, and 1

nematode. For most of these species (48) studies reported qualitative evidence of male harm (i.e. some evidence of harm to females), while quantitative evidence (i.e. estimation of the degree to which female fitness decreases with male harm) was only reported for 39 species (see SM for details). Overall, the overwhelming majority of these 87 species exhibited little scope for maternal effects. Namely, some form of parental care has been described for only 18 of these species (11 of which are vertebrates), while only 11 are viviparous (all vertebrates) and only 5 (all vertebrates) exhibit some form of extended maternal provision (vs. lecithotrophy). Such restricted variability, particularly across invertebrates, precluded a formal meta-analysis to explore the relationship between the existence and/or intensity of male harm and the scope for maternal effects.

Discussion

We found 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 for both types of male harm (i.e. male harassment and traumatic insemination) and across different levels of dispersal (Figure 1). 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 the kin selection effects previously described in the literature (Faria *et al.* 2020), but also differences 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 disfavoured whenever harm induces maternal effects on offspring quality, in a manner

312 that is proportional to these effects. We thus identify male harm-mediated maternal effects as
313 a previously unrecognized factor shaping sexual conflict evolution, a realization that may
314 have far-reaching taxonomic implications.

315 The overarching prediction that stems from our results is that, all else being equal, we
316 might expect lower levels of male harm to females in taxa where maternal effects on
317 offspring quality are higher, more amenable to changes in maternal condition, and/or in
318 which offspring quality (relative to quantity) loads heavily on parental fitness. Specifically,
319 we would predict generally lower levels of male harm in species with prolonged gestation
320 (e.g. viviparous/ovoviviparous vs. oviparous), in species with extended maternal provisioning
321 (e.g. matrotrophic vs. lecithotrophic), in species with (vs. without) parental care, and at large
322 in species that are under strong K- (vs. r-) selection. Identifying maternal effects as a
323 potential modulator of sexual conflict thus gives rise to specific predictions about where male
324 harm might have evolved and how intense we might expect it to be. Available evidence,
325 collected following a systematic literature search, seems to fit reasonably well with these
326 predictions (Figures 2 and 3). We found male harm to be particularly widespread, intense and
327 sophisticated in insects, which include the best-known cases of sexually antagonistic
328 coevolution driven by male harm (Perry & Rowe 2015b) along with many instances of
329 traumatic insemination (Crudgington & Siva-Jothy 2000; Arnqvist *et al.* 2005; Siva-Jothy
330 2006; Tatarnic *et al.* 2014; Reinhardt *et al.* 2015) , including toxic ejaculates (Wigby &
331 Chapman 2005) and extreme coercion (Han & Jablonski 2010). Furthermore, indirect
332 evidence based on the description of male genitalia (and the fitness consequences of similar
333 structures in other species) suggests adaptations for traumatic insemination may occur in as
334 many as ca.1400 species more (Figure 3; see SM for details). In accordance to predictions,
335 insects are typically under strong r-selection, oviparous and normally lack extended maternal
336 provision and parental care (Figure 2 and SM). Gastropods, where traumatic insemination

337 also seems common, follow a very similar pattern. Actually, there is an almost complete lack
338 of variation in the scope of maternal effects across these taxa.

339 In contrast, male harm appears to be relatively rare or weak in vertebrates, especially
340 so in taxa with widespread parental care and prolonged gestation such as birds and mammals
341 (Figure 2). As a matter of fact, well-studied cases of male harm reported so far in vertebrates
342 consist exclusively in collateral damage to females (i.e. harassment and/or coercive mating),
343 as opposed to traumatic insemination adaptations aimed to harming females *per se* (i.e. direct
344 damage; Aloise King *et al.* 2013). The absence of adaptations for direct harm in mammals is
345 perhaps particularly salient given the strength of male-male competition in many species
346 within this group (Andersson 1994). Furthermore, although harassment is widely interpreted
347 as an inherently costly male phenotype for females, it does not necessarily translate into a
348 reduction in female fitness. For example, female resistance to male harassment has been
349 suggested to participate in mate choice as a way of screening high quality males (Cordero &
350 Eberhard 2003). Thus, the mere existence of male harassment and/or coercion is far from a
351 litmus test for male harm. Different forms of sexual harassment and/or coercion to females
352 have been reported for a number of vertebrates (and are probably common; Clutton-Brock &
353 Parker 1995), but direct evidence that such harassment reduces female fitness is limited
354 (Magurran & Ojanguren 2007; Makowicz & Schlupp 2013; Iglesias-Carrasco *et al.* 2019).
355 For example, forced copulations are common in waterfowls (e.g. reported for at least 55
356 species; McKinney *et al.* 1983; McKinney & Evarts 1998), where they are frequently
357 accompanied by male harassment behaviour that can occasionally result in injuries and even
358 the death of the female (McKinney *et al.* 1983), but evidence that such behaviour actually
359 harm females is more restricted (see Adler 2010; Figure 2).

360 In short, there does seem to be a relationship between the overall scope for maternal
361 effects within broad taxonomic groups and reports of male harm, particularly when species

exhibit male adaptations for direct harm to females. Looking specifically at the 87 species for which we found good evidence of male harm to females across all taxa, we found little scope for maternal effects overall, and particularly so in cases of direct harm (Figure 2; see also SM), which again seems to echo our predictions. Unfortunately, the lack of variation in the collected proxies for the scope of maternal effects prevented a formal meta-analysis relating these variables with the level of male harm (i.e. drop in female fitness due to male harm). Thus, we stress that conclusions to this respect are necessarily tentative and preliminary at this stage, as phylogenetic signal within groups is expected to be strong due to other factors (e.g. sperm competition levels or the opportunity for selection).

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 (Rankin 2011; Carazo *et al.* 2014; Faria *et al.* 2015, 2020; Lukasiewicz *et al.* 2017), maternal effects can bring together the interests of males and females and abate conflict over sexual strategies. 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 (Royle *et al.* 2012). Second, due to the well-established fact that male harm can have a dramatic impact on female condition (Arnqvist & Rowe 2005). Available evidence seems consonant with the idea that maternal effects 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.

Acknowledgements

We thank Tobias Uller for insightful comments on an earlier version of this paper and Andy Gardner for helpful comments on an earlier version of the theoretical model. P.C. was supported by a Plan Nacional I+D+i "Excelencia" grant (CGL2017-89052-P) and a "Ramón y Cajal" Research Fellowship (RYC-2013-12998) by the Spanish Government. R.G-R. was supported by a "Juan de la Cierva Formación" Research Fellowship (FJC2018-037058-I) by the Spanish Government. G.S.F acknowledges IAST funding from the French National Research Agency (ANR) under the Investments for the Future (Investissements d'Avenir) program, grant ANR-17-EURE-0010.

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519 **Figure 1 | Optimal level of harm favoured in different models and as a function of male**
520 **dispersal (d_m).**
521 The level of harm that is favoured by natural selection depends on the absence or presence of
522 maternal effects. Accordingly, absence of maternal effects leads to higher levels of harm than
523 in the presence of maternal effects, more so when they affect the individuals' sexual quality.
524 Such effect is present regardless if harming occurs before or during mating (A) and of the
525 levels of relatedness between individuals in the social group (B&C). For all panels, the
526 following parameters were used: scale of competition $a = 0.50$; number of males $n_m = 3$;
527 number of females $n_f = 3$; fecundity and competitiveness cost $s = 0.5$; sexual cost for females
528 $h = 0.5$; and sexual cost for males $t = 0.5$. In A: relatedness between males $r_{mm} = 0.15$; and
529 relatedness between females and males $r_{fm} = 0.15$. In B and C: harm exclusive from sexual
530 partners $k = 0$.

531

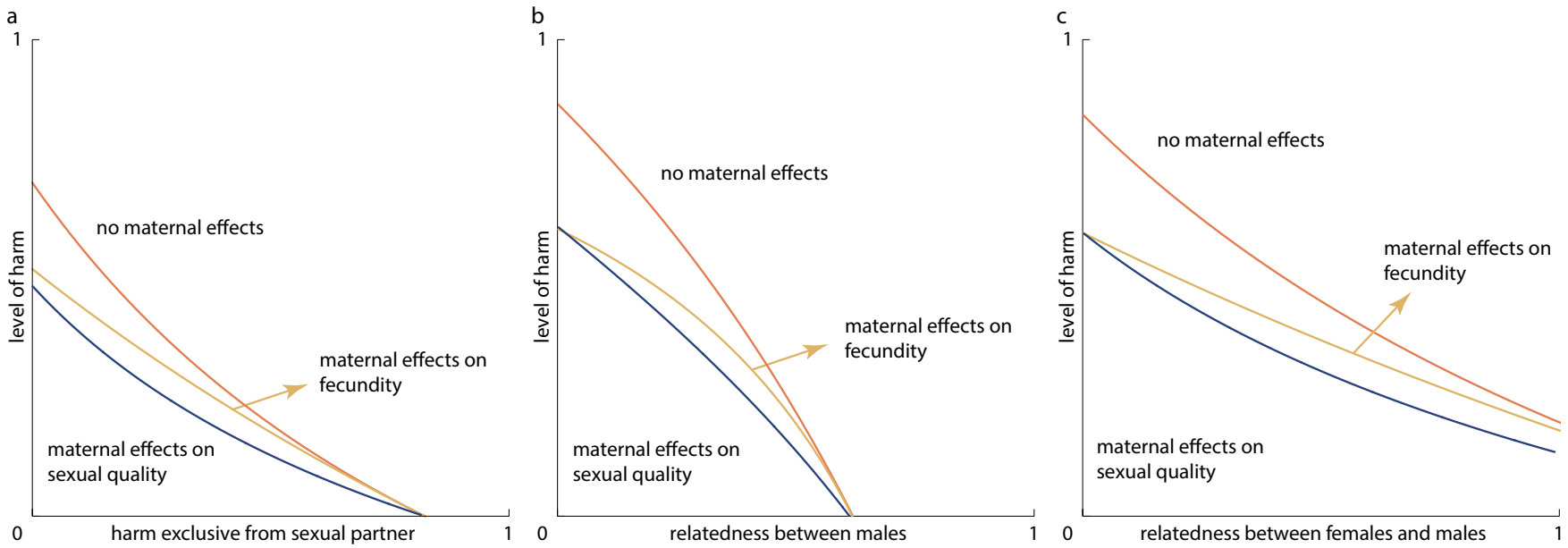
532 **Figure 2 | Distribution of male harm adaptations across the tree of life.**
533 Results from taxa that encompass the 87 species for which we found evidence of male harm.
534 Outward-facing circles in the red shaded area represent the presence of direct (red) and/or
535 collateral (orange) adaptations leading to male harm of females (i.e. one for each species).
536 Triangles denote the existence of substantial evidence suggesting this strategy is widespread
537 in at least some taxa within the group (see supplementary material for details). Inner-facing
538 circles in the green shaded area reflect evidence in the literature of parental care, extended
539 gestation (viviparity/ovoviviparity vs. oviparity) and extended maternal provisioning
540 (matrotophy/placentotrophy/brooding vs. lecithotrophy) in the taxon. The size of inward-
541 facing green circles illustrates how widespread these strategies are according to the literature:
542 large circles denote a widespread strategy (i.e. adopted by most or all known species),
543 medium-size circles represent a common strategy (i.e. more than 5% but less than 50% of

544 known species) and small-sizes circles represent a rare strategy (i.e. < 5% known species).
545 We excluded two species (i.e. *Caenorhabditis ramnei* – Nematode– and *Idotea balthica* –
546 Isopod–; see SM) from the figure due to non-available data for maternal effects (*Idotea*
547 *balthica*) or the type of male harm (*Caenorhabditis ramnei*).

548 **Figure 3 | Male harm and scope for maternal effects**
549 A) Percentage of the 87 species for which parental care (light green), extended gestation
550 (dark green) and extended maternal provision (grey), respectively from left to right, has been
551 reported in the literature. B) Summary of indirect evidence (i.e. mostly based on descriptions
552 of male genitalia; see methods and SM) for male adaptations that may be directly harmful to
553 females (i.e. traumatic insemination, including genital wounding and ablation). External
554 genital mutilation is common in some spiders, where maternal care in the form of egg-
555 protection is widespread(Řezáč 2009; Mouginot *et al.* 2015; Nakata 2016). External genital
556 mutilation likely evolved in response to sperm competition by preventing females from
557 effectively re-mating. However, there is no evidence to date that it reduces female fitness and
558 it does not affect receptivity to male mating attempts(Řezáč 2009; Mouginot *et al.* 2015;
559 Nakata 2016). The numbers beside red bars represent the number of species within the group
560 that are estimated to exhibit such male harm adaptations. Green-shaded circles reflect
561 evidence in the literature of parental care, extended gestation and extended maternal
562 provisioning in the taxon; respectively, from left to right.

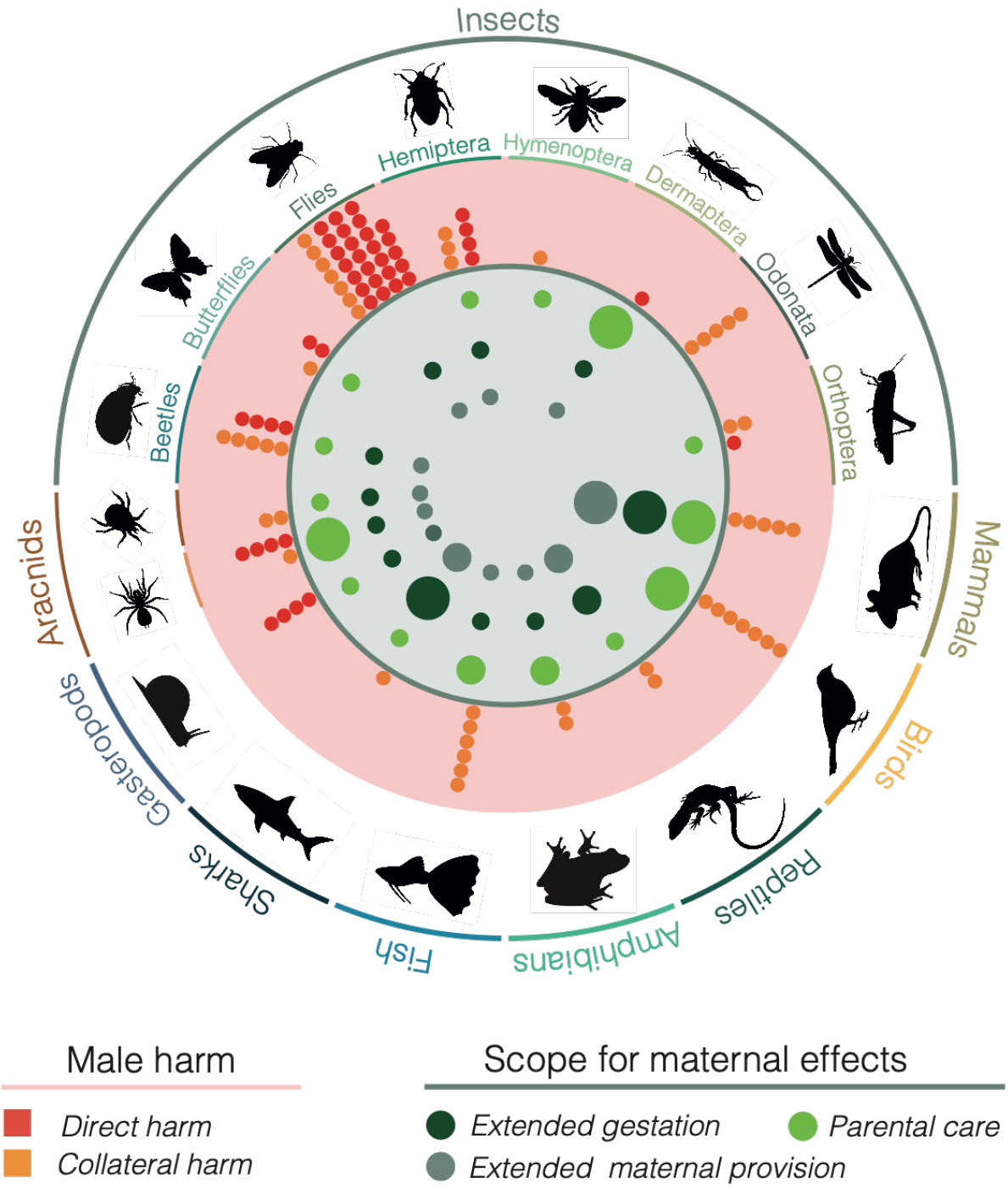
563 **Figure 1**

564



565

566 **Figure 2**



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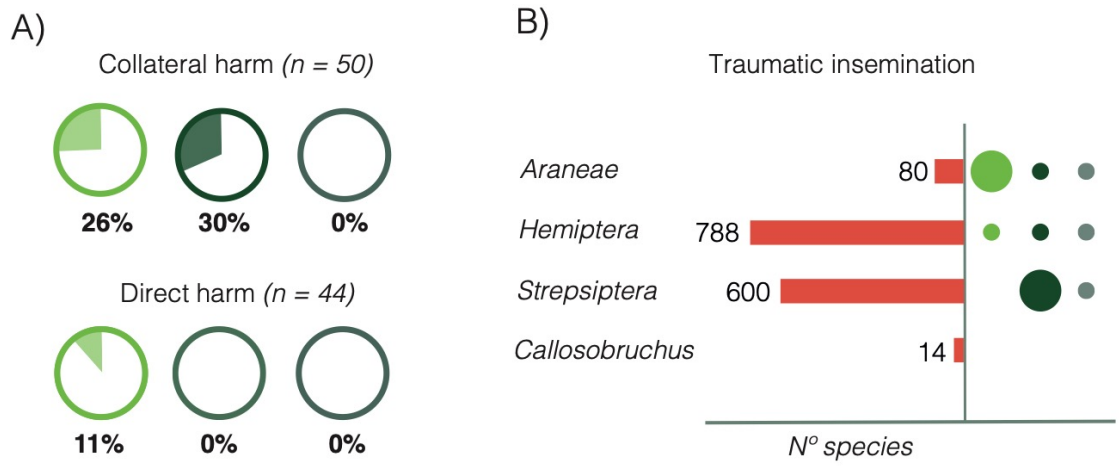
568 **Figure 3**

569



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