Condition-transfer maternal effects modulate sexual conflict

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Abstract

Strong sexual selection frequently favours males that increase their reproductive success by harming females, with potentially negative consequences for population growth. 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 population structure, 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 & 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; 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 & 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

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 & Snook 2003; Parker 2006).

Maternal effects can drastically modulate offspring quality (Mousseau & Fox 1998) and are largely mediated by maternal condition (i.e. condition-transfer maternal effects; Rossiter 1996; Saino *et al.* 2005; (Bonduriansky & Crean 2018). Male harm can severely impact female condition (Arnqvist & 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). 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 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 independently from kin selection.

In order to test this idea, we use a personal-fitness kin-selection approach (Hamilton 1964a,b; Taylor & Frank 1996) that incorporates the effects of kin selection, an important factor in the evolution of male harm to females (Faria et al. 2020). In particular, 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)

85 presence of maternal effects on the offspring fecundity (females) and competitiveness
86 (males), and c) presence of maternal effects on offspring's ability to inflict (males) and resist
87 (females) harm (i.e. sexual selection quality). Finally, to assess whether theoretical
88 predictions fit with available data, we conducted a systematic search in the literature to
89 identify studies reporting solid quantitative or qualitative evidence of male harm to females
90 and, for these species, collected data on three proxies of maternal effects: parental care,
91 extended gestation and extended maternal provision.

92

93 **Methods**

94 *1.1 Model without maternal effects*

95 We consider an infinite diploid population divided into social groups (Wright 1931)
96 containing n_f females and n_m males. We follow the approach developed by Faria et al. (2020).
97 Specifically, males invest in a harming trait that increases their personal reproductive success
98 relative to other males but reduces the overall fecundity of the females in the social group.
99 Each male's reproductive success is directly proportional to his competitiveness for mating
100 success and inversely proportional to the average competitiveness for mating success of the
101 males in his social group. We consider two different populations with two different mating
102 systems: monogamic females, where females mate with only one male while males compete
103 to gain access to the females (Figure 1); polygamic females, where females mate with all
104 males in the social group and all males mate with all females in the social group (Figure 1).

105 Accordingly, a focal female's fecundity $f_f(x',y) = 1 - k\,x' - (1 - k)y$ is a function of
106 the level of harm of the male that she mates with (x') and of the average level of male harm
107 males present in the social group (y), with k determining the degree to which harm comes
108 during mating ($k = 1$) or before mating as a result of male competition to access females ($k =$
109 0). At one extreme ($k = 1$), the harm comes exclusively from the male that mates with the

female, for instance circumstances where traumatic insemination by the male makes the female unavailable for further mating events. At the other extreme ($k = 0$), the harm comes from all males in the social group, for instance circumstances where males harass females during the process of competing with the other males. Importantly, we consider a continuum and population may lie in any point between those two extremes. Note that, when considering a polygamic population, the variable k disappears because of the underlying assumption that all males are mating with all females (being, 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 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 (Figure 1). 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.

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. Good-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);

135 or 2) quality affects an individual's ability to inflict (males) and resist (females) harm. Low-
136 quality individuals are produced in proportion to the harm that their mother received (see SM
137 for details).

138 Focusing on the first scenario: a focal low-quality female's fecundity is a function of
139 the level of harm of the male that she mates with and of the average level of male harm
140 present in the social group minus a cost s due to her low quality $f^{\dagger}_{f}(x',y) = 1 - k\,x' - (1 - k)y -$
141 s (for a monogamic female) and $f^{\dagger}_{f}(y) = 1 - y - s$ (for a polygamic female); a focal low-quality
142 male's competitiveness for mating success is a function of the level of harm expressed by that
143 focal male minus a cost s due to his low quality $f^{\dagger}_{m}(x) = 1 + x - s$; a focal high-quality
144 female's fecundity is a function of the level of harm of the male that she mates with and of
145 the average level of male harm present in the social group $f^{\hbar}_{f}(x',y) = 1 - k\,x' - (1 - k)y$ (for a
146 monogamic female) and $f^{\hbar}_{f}(y) = 1 - y$ (for a polygamic female); and a focal high-quality
147 male's competitiveness for mating success is a function of the level of harm expressed by that
148 focal male $f^{\hbar}_{m}(x) = 1 + x$.

149 Focusing on the second scenario: a focal low-quality female's fecundity is a function
150 of the level of harm of the male that she mates with and of the average level of male harm
151 present in the social group multiplied by $1 + h$ due to her low quality $f^{\dagger}_{f}(x',y) = 1 - (k\,x' + (1$
152 $- k)y)(1 + h)$ (for a monogamic female) and $f^{\dagger}_{f}(y) = 1 - y(1 + h)$ (for a polygamic female); a
153 focal low-quality male's competitiveness for mating success is a function of the level of harm
154 expressed by that focal male multiplied by $1 - t$ due to his low quality $f^{\dagger}_{m}(x) = 1 + x(1 - t)$; a
155 focal high-quality female's fecundity is a function of the level of harm of the male that she
156 mates with and of the average level of male harm present in the social group $f^{\hbar}_{f}(x',y) = 1 - k$
157 $x' - (1 - k)y$ (for a monogamic female) and $f^{\hbar}_{f}(y) = 1 - y$ (for a polygamic female); and a focal
158 high-quality male's competitiveness for mating success is a function of the level of harm

expressed by that focal male $f^{\text{h}}_{\text{m}}(x) = 1 + x$. 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 SM for details) and assuming that $n_{\text{f}} = n_{\text{m}}$ (i.e. the number of females and males are the same in each social group), the optimal fitness equations (see SM for details) in a population where females are monogamic are:

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

for the model without maternal effects;

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

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

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

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

183 the focal male's mating success as it reduces the potential offspring that he has with a female
184 (first term). Both are weighted by the relatedness between the focal male and local males,
185 given that an increase in focal male's mating success leads to a corresponding loss of mating
186 success by the other males. This translates into an inclusive fitness loss if the focal male is
187 related to them ($1 - r_{mm}$). Harming also reduces the overall fecundity of local females, which
188 also decreases the number of offspring produced by local females and males (second term).
189 Such reduction in fecundity can lead to an inclusive fitness loss if the focal individual is
190 related to both local females and males ($r_{fm} + r_{mm}$). Finally, such inclusive fitness loss is
191 weighted by local competition ($1 - a$), that is, how much individuals compete with local
192 social group mates for reproductive resources (Taylor 1992).

193 The conditions for harm to be favoured in a polygamic population are essentially the
194 same, with the exception that the term being multiplied by k disappears. Accordingly, the
195 inclusive fitness interpretation is the same as above, except that the males do not pay a direct
196 cost for harming the females.

197 The optimal level of harm can now be obtained by solving equations (1–3) to z^*
198 (Figure 2; Figure 3). While the results are similar across the different models, there are
199 important quantitative differences. Specifically, the harm benefits are smaller, and costs are
200 higher, when maternal effects are present, and more so when maternal effects influence the
201 ability to inflict (males) or resist (females) harm (Figure 2; Figure 3). This happens regardless
202 of harm coming before (i.e. male harassment) and/or during (i.e. traumatic insemination) the
203 mating act (Figure 2a), relatedness levels (Figure 2b-c; Figure 3a-b), or polygamy (Figure 2;
204 Figure 3). The exception is when local competition is high (Figure 2d; Figure 3c). When local
205 competition is high, maternal effects on fecundity reduce the level of harm more than
206 maternal effects on sexual quality.

207 **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). This, however, can change if the level of local competition is high, leading to maternal effects on fecundity reducing the level of harm more than maternal effects on sexual quality. 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 (Figure 2; Figure 3). While relatedness 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 relatedness.

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 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

233 which offspring quality (relative to quantity) loads heavily on parental fitness. Specifically,
234 we would predict generally lower levels of male harm in species with prolonged gestation
235 (e.g. viviparous/ovoviviparous vs. oviparous), in species with extended maternal provisioning
236 (e.g. matrotrophic vs. lecithotrophic), in species with (vs. without) parental care, and at large
237 in species that are under strong K- (vs. r-) selection. Identifying maternal effects as a
238 potential modulator of sexual conflict thus gives rise to specific predictions about where male
239 harm might have evolved and how intense we might expect it to be. We conducted a
240 systematic literature search with the hope of formally testing this association across the tree
241 of life, but currently available data is, unfortunately, not amenable for formal analysis (see
242 SM).

243 Male harm appears to be particularly widespread, intense and sophisticated in insects,
244 which include the best-known cases of sexually antagonistic coevolution driven by male
245 harm (Perry & Rowe 2015b) along with many instances of traumatic insemination
246 (Crudgington & Siva-Jothy 2000; Arnqvist *et al.* 2005; Siva-Jothy 2006; Tatarnic *et al.* 2014;
247 Reinhardt *et al.* 2015), including toxic ejaculates (Wigby & Chapman 2005) and extreme
248 coercion (Han & Jablonski 2010). Furthermore, indirect evidence based on the description of
249 male genitalia (and the fitness consequences of similar structures in other species) suggests
250 adaptations for traumatic insemination may occur in as many as ca.1400 species more (see
251 SM for details). Insects are typically under strong r-selection, oviparous and normally lack
252 extended maternal provision and parental care, and gastropods, where traumatic insemination
253 also seems common, follow a very similar pattern (see SM). In contrast, male harm appears
254 to be relatively rare or weak in vertebrates, especially so in taxa with widespread parental
255 care and prolonged gestation such as birds and mammals (see SM). As a matter of fact, well-
256 studied cases of male harm reported so far in vertebrates consist exclusively in collateral
257 damage to females (i.e. harassment and/or coercive mating), as opposed to traumatic

insemination adaptations aimed to harming females *per se* (i.e. direct damage; Aloise King *et al.* 2013).

The absence of adaptations for direct harm in mammals is perhaps particularly salient given the strength of male-male competition in many species within this group (Andersson 1994). Furthermore, although harassment is widely interpreted as an inherently costly male phenotype for females, it does not necessarily translate into a reduction in female fitness. For example, female resistance to male harassment has been suggested to participate in mate choice as a way of screening high quality males (Cordero & Eberhard 2003). Thus, the mere existence of male harassment and/or coercion does not necessarily imply fitness costs to females. Different forms of sexual harassment and/or coercion to females have been reported for a number of vertebrates (and are probably common; Clutton-Brock & Parker 1995), but direct evidence that such harassment reduces female fitness is limited (Magurran & Ojanguren 2007; Makowicz & Schlupp 2013; Iglesias-Carrasco *et al.* 2019). For example, forced copulations are common in waterfowls (e.g. reported for at least 55 species; McKinney *et al.* 1983; McKinney & Evarts 1998), where they are frequently accompanied by male harassment behaviour that can occasionally result in injuries and even the death of the female (McKinney *et al.* 1983), but evidence that such behaviour actually harm females is more restricted (see Adler 2010; Figure S1). In short, there may seem to be a tenuous relationship between the overall scope for maternal effects within broad taxonomic groups and reports of male harm, particularly when species exhibit male adaptations for direct harm to females. However, available evidence clearly do not span enough co-variation in the scope of maternal effects and male harm to draw any conclusions. For example, across the 87 species for which we found good evidence of male harm to females across all taxa, we found very little variation in general proxies for maternal effects, particularly so in cases of direct harm (Figure S1; see also SM).

To conclude, in this study we aim to bring attention to male-induced maternal effects as a potentially important 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), we show that maternal effects can bring together the interests of males and females and abate conflict over sexual strategies. Such effects could be important to understand sexual conflict evolution in nature for two main reasons. First, due to the existence of substantial variation in condition-transfer maternal effects that can impinge on offspring quality, both across and within taxa (Royle *et al.* 2012; Bonduriansky & Crean 2017). Second, due to the well-established fact that male harm can have a dramatic impact on female condition (Arnqvist & Rowe 2005). We thus suggest that future empirical studies should aim to test the predictions we lay out here arising from the interplay between maternal effects and male harm, which could further our understanding of sexual conflict.

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307 the systematic literature search. DN explored the data for potential meta-analysis. PC prepared
308 Figures 2 and 3. PC and GSF wrote the manuscript with contributions by RG-R and DN.
309
310 **Data accessibility statement:** Should the manuscript be accepted, all data supporting the
311 results presented will be deposited in a public repository and the data DOI will be included at
312 the end of the article.

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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. Male harm to females depends on the value of k, with harm only coming from the successful male if $k = 1$ and coming from all the males in the social group 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 $1 - a$.

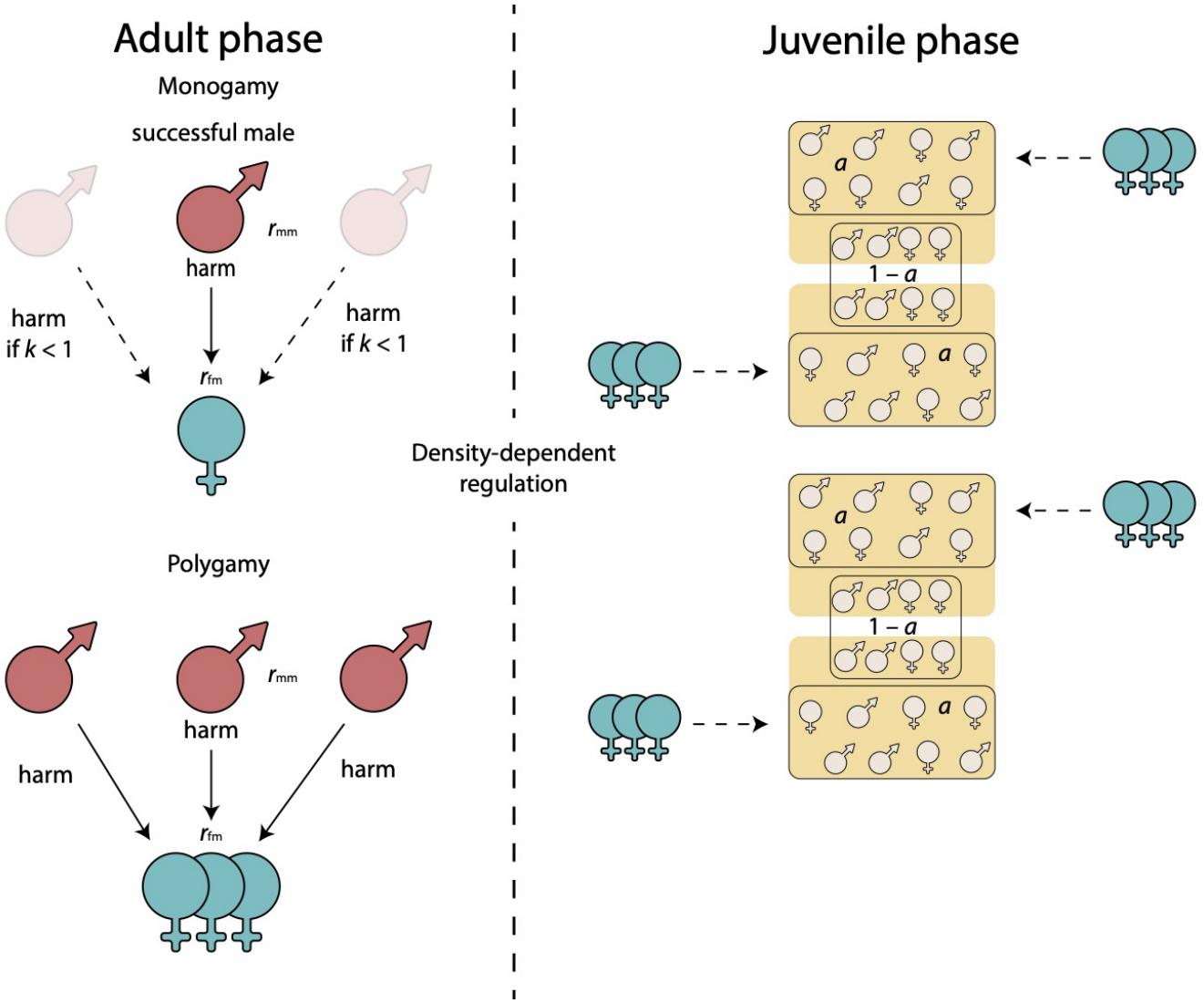
Figure 2 | Optimal level of harm favoured in monogamic females.

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 if harming 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&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 affect the individuals' sexual quality. Such effect is present regardless of the levels of relatedness between individuals in the social group (a&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 $r_{fm} = 0.15$.

476 **Figure 1**



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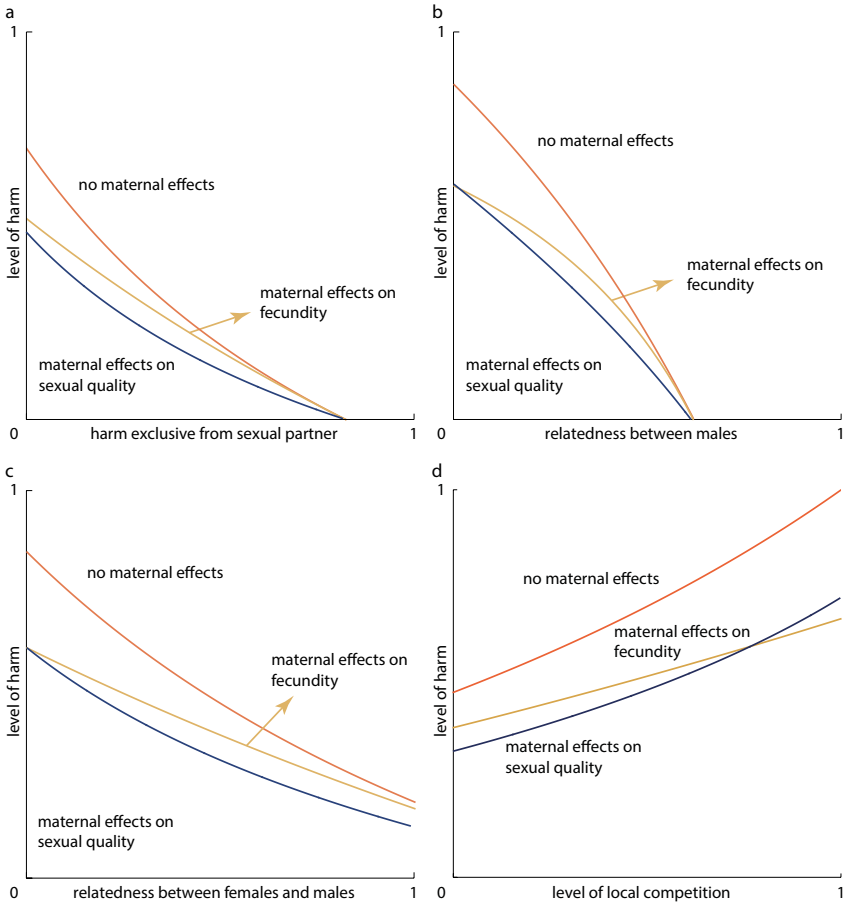
479 **Figure 2**



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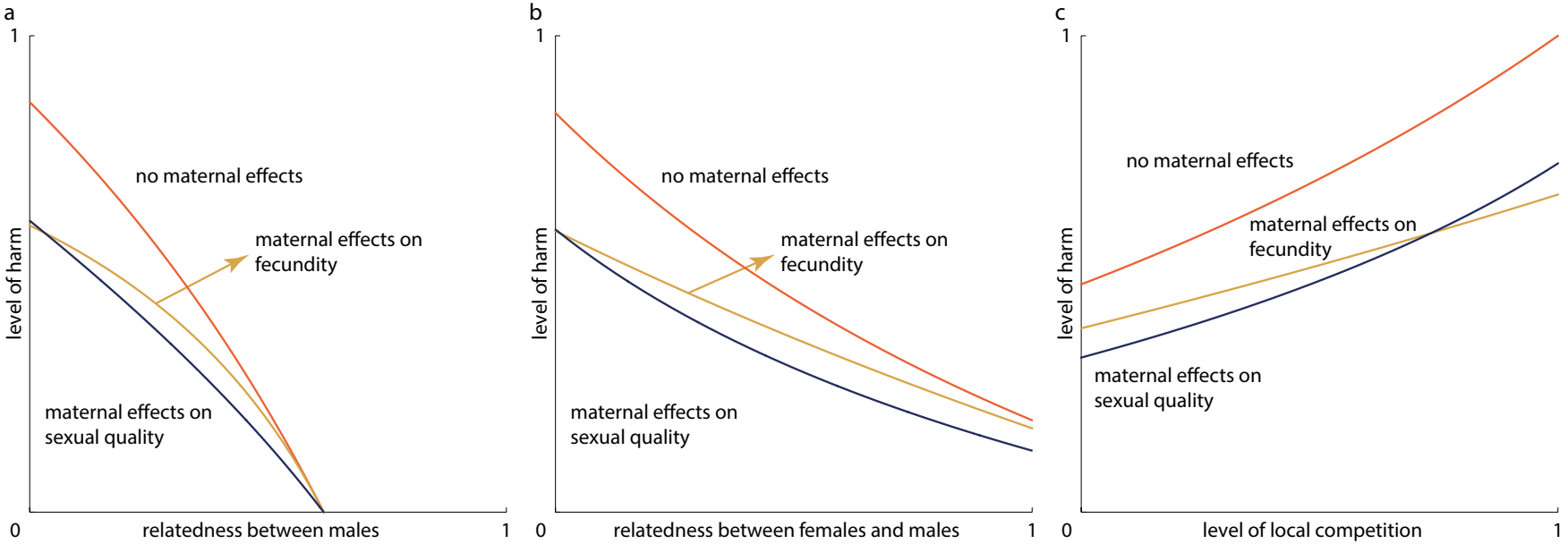
483 **Figure 3**



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485 **Electronic supplementary material**

486 **1.1 Model without maternal effects**

487

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

490

491 $$W_{\mathrm{f}} = f_{\mathrm{f}}(x', y)\left(\frac{1}{a f_{\mathrm{f}}(y', y) + (1-a) f_{\mathrm{f}}(z)}\right), \quad \text{(A1)}$$

492

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

500

501 $$W_{\mathrm{f}} = f_{\mathrm{f}}(y)\left(\frac{1}{a f_{\mathrm{f}}(y) + (1-a) f_{\mathrm{f}}(z)}\right), \quad \text{(A2)}$$

502

503 A similar logic to the one described above for monogamic female applies. Male's relative
504 fitness in a monogamic population and in the context of the present model is

505

506 $$W_{\mathrm{m}} = f_{\mathrm{m}}(x, y)\left(\frac{1}{a f_{\mathrm{f}}(\chi', y) + (1-a) f_{\mathrm{f}}(z)}\right), \quad \text{(A3)}$$

507

508 where:

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

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

$f_{\mathrm{f}}(\chi',y) = 1 - k\,(\chi') - (1-k)y$; 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 $1 - \Omega$, again depending if it is a female in the social group or the average female in the population. A good-quality male's relative fitness in a monogamic population is:

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

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

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

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

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

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

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

where:

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

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

$\bar{f}_{\mathrm{f}}(\chi',y) = (1-\omega)f^{\mathrm{h}}{}_{\mathrm{f}}(\chi',y) + \omega f^{\mathrm{l}}{}_{\mathrm{f}}(\chi',y)$; $\bar{f}_{\mathrm{m}}(y) = (1-\omega)f^{\mathrm{h}}{}_{\mathrm{m}}(y) + \omega f^{\mathrm{l}}{}_{\mathrm{m}}(y)$; 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: $f_{\mathrm{hf}}(\chi',y) = 1 - k\chi' - (1-$

$k)y$; $f_{lf}(\chi',y) = 1 - k\,\chi' - (1-k)y - s$; $f_{hf}(x,y) = 1 - k\,x - (1-k)y$; $f_{lf}(x,y) = 1 - k\,x - (1-k)y - s$; $f_{hm}(y) = 1 + y$; $f_{lm}(y) = 1 + y - s$; and $\chi' = (\mu/n_f)x + ((n_f - \mu)/n_f)y''$. If we are considering the second scenario, then: $f_{hf}(\chi',y) = 1 - k\,\chi' - (1-k)y$; $f_{lf}(\chi',y) = 1 - (k\,\chi' - (1-k)y)(1+h)$; $f_{hf}(x,y) = 1 - k\,x - (1-k)y$; $f_{lf}(x,y) = 1 - (k\,x - (1-k)y)(1+h)$; $f_{hm}(y) = 1 + y$; $f_{lm}(y) = 1 + y(1-t)$; $\chi' = (\mu/n_f)x + ((n_f - \mu)/n_f)((1-\omega)y'' + \omega(y''(1-t))$ if the focal male is a good-quality male; and $\chi' = (\mu/n_f)x(1-t) + ((n_f - \mu)/n_f)((1-\omega)y'' + \omega(y''(1-t))$ 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

Natural selection favours any gene associated with greater individual relative fitness (Fisher 1930; Price 1970). Assuming vanishingly little genetic variation, this condition many be expressed using the mathematics of differential calculus: $dW/dg > 0$, where g is the genic value of a gene picked at random from the population and W is the relative fitness of the individual carrying this gene (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. 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, $W_{\mathrm{i}} = \frac{1}{2}((1-\omega)(w^{\mathrm{h}}_{\mathrm{f}}/\overline{w}_{\mathrm{f}}) + \omega(w^{\mathrm{l}}_{\mathrm{f}}/\overline{w}_{\mathrm{f}})) + \frac{1}{2}((1-\omega)(w^{\mathrm{h}}_{\mathrm{m}}/\overline{w}_{\mathrm{m}}) + \omega(w^{\mathrm{l}}_{\mathrm{m}}/\overline{w}_{\mathrm{m}}))$. 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 $\mathrm{d}W_{\mathrm{i}}/\mathrm{d}g = \frac{1}{2}(\mathrm{d}W^{\mathrm{k}}_{\mathrm{f}}/\mathrm{d}g_{\mathrm{f}}) + \frac{1}{2}(\mathrm{d}W^{\mathrm{k}}_{\mathrm{m}}/\mathrm{d}g_{\mathrm{m}}) = \frac{1}{2}((1-\omega)((\partial W^{\mathrm{h}}_{\mathrm{f}}/\partial x')(\mathrm{d}x'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{h}}_{\mathrm{f}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{h}}_{\mathrm{f}}/\partial y')(\mathrm{d}y'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}})) + \omega((\partial W^{\mathrm{l}}_{\mathrm{f}}/\partial x')(\mathrm{d}x'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{l}}_{\mathrm{f}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}) + (\partial W^{\mathrm{l}}_{\mathrm{f}}/\partial y')(\mathrm{d}y'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{f}}))) + \frac{1}{2}((1-\omega)((\partial W^{\mathrm{h}}_{\mathrm{m}}/\partial x)(\mathrm{d}x/\mathrm{d}G_{\mathrm{m}})(\mathrm{d}G_{\mathrm{m}}/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{h}}_{\mathrm{m}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{h}}_{\mathrm{m}}/\partial \chi')(\mathrm{d}\chi'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}})) + \omega((\partial W^{\mathrm{l}}_{\mathrm{m}}/\partial x)(\mathrm{d}x/\mathrm{d}G_{\mathrm{m}})(\mathrm{d}G_{\mathrm{m}}/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{l}}_{\mathrm{m}}/\partial y)(\mathrm{d}y/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}}) + (\partial W^{\mathrm{l}}_{\mathrm{m}}/\partial \chi')(\mathrm{d}\chi'/\mathrm{d}G_{\mathrm{m}}')(\mathrm{d}G_{\mathrm{m}}'/\mathrm{d}g_{\mathrm{m}})))$. 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 selections 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

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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

714 and WoS = 300). After removing 373 duplicates, we exported 321 to Rayyan. We conducted
715 a final search on the 7/04/20 using the search terms: “sexual conflict” & harassment. We
716 found a total of 414 items (Scopus = 175, PubMed = 50 and WoS = 189). After removing 175
717 duplicates, we exported 239 to Rayyan. In Rayyan, we checked for duplicates within the
718 complete database comprising all the papers located via these three searches and removed 69
719 duplicates, leading to 527 unique studies for more detailed screening. Based on the title and
720 abstract we excluded 347 papers that clearly did not report adaptations for male harm, leaving
721 a total of 180 papers for in-depth screening.
722 We carefully screened these papers and excluded papers that did not comply with our
723 selection criteria described above. In the process of screening, we added 27 more papers
724 through forward and backward searches of citations and references, leading to a total final
725 sample of selected studies reporting male harm adaptations for a total of 87 different species
726 (see SM for complete list). Finally, for all these species we performed specific searches on
727 their mode of reproduction (i.e. ovipary, vivipary or ovovivipary), evidence for parental care
728 and evidence of extended maternal provisioning (i.e. matrotrophy, including brooding).
729 Similar data for broad taxonomic groups taxa included in Figure 2 were taken from key
730 references provided in the supplementary materials.
731
732 ***Results***– In total, we obtained evidence of male harm to females for a total of 87
733 species (see Figures 2 and 3, and SM for details); 51 insects, 7 arachnids, 7 birds, 6 fish, 5
734 mammals, 4 gastropods, 2 amphibians, 2 reptiles, 1 shark, 1 crustacean, and 1 nematode. For
735 most of these species (48) studies reported qualitative evidence of male harm (i.e. some
736 evidence of harm to females), while quantitative evidence (i.e. estimation of the degree to
737 which female fitness decreases with male harm) was only reported for 39 species (see SM for
738 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. Iit is important to note that the evidence described above is obviously correlative, and hence inadequate to sustain a causal relationship. Furthermore, 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). Similarly, we wish to stress that our measure of the scope for maternal effects is unavoidably coarse. For example, species that have long gestational periods can exhibit complex adaptations to buffer offspring from effects of mothers (e.g., placentas, or ability to metabolize maternal hormones), so longer gestational periods do not necessarily equal "more" maternal effects. Finally, phylogenetic signal within groups is expected to be strong due to other factors (e.g. specific maternal effects adaptations, sperm competition levels or the opportunity for selection). Thus, we stress that conclusions to this respect must be taken as completely preliminary, at best.

Figure S1 | Distribution of male harm adaptations across the tree of life.
Results from taxa that encompass the 87 species for which we found evidence of male harm. Outward-facing circles in the red shaded area represent the presence of direct (red) and/or collateral (orange) adaptations leading to male harm of females (i.e. one for each species). Inner-facing circles in the green shaded area reflect evidence in the literature of parental care, extended gestation (viviparity/ovoviviparity vs. oviparity) and extended maternal provisioning (matrotophy/placentotrophy/brooding vs. lecithotrophy) in the taxon. The size

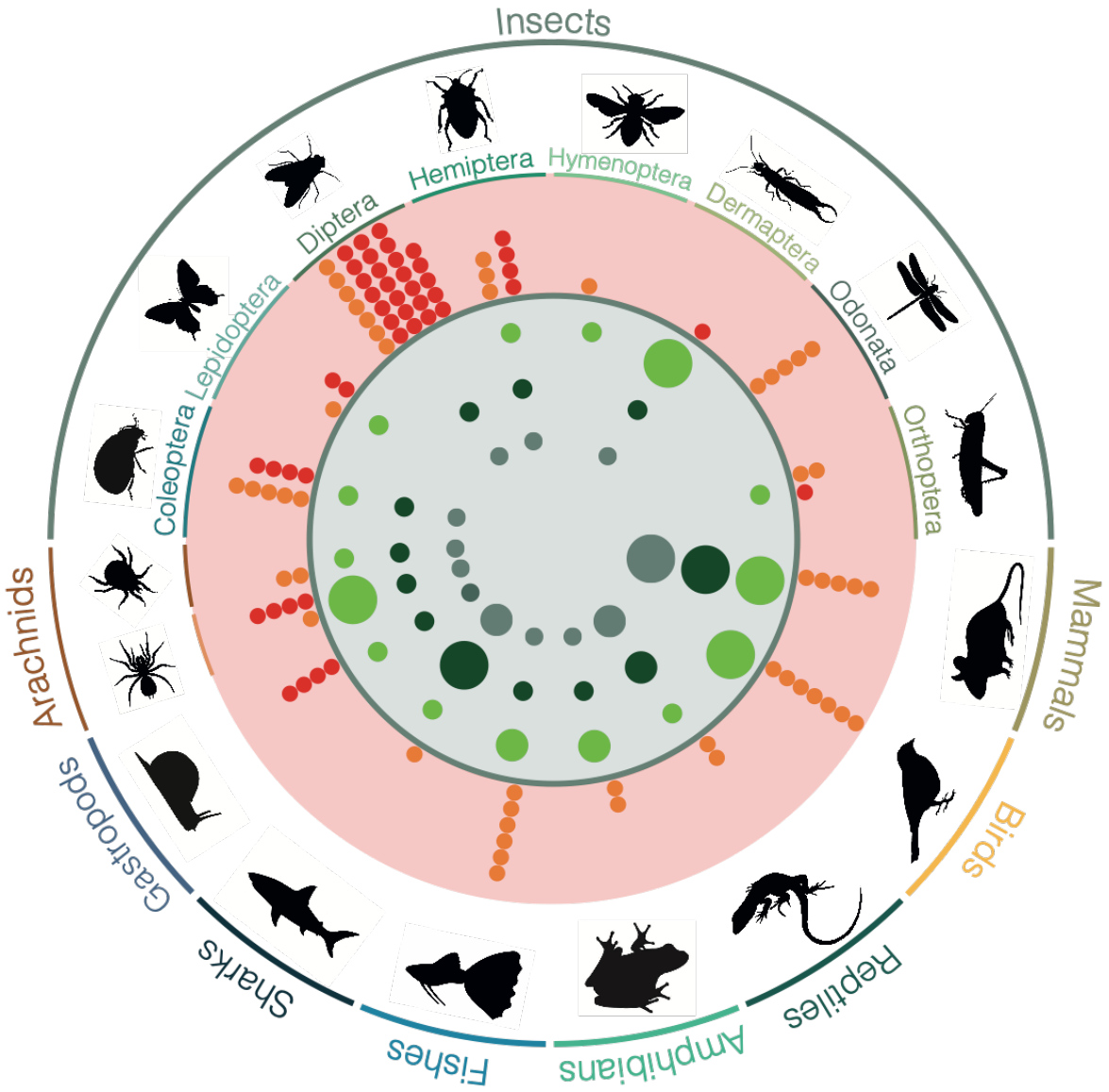
764 of inward-facing green circles illustrates how widespread these strategies are according to the
765 literature: large circles denote a widespread strategy (i.e. adopted by most or all known
766 species), medium-size circles represent a common strategy (i.e. more than 5% but less than
767 50% of known species) and small-sizes circles represent a rare strategy (i.e. < 5% known
768 species). We excluded two species (i.e. *Caenorhabditis ramnei* – Nematode– and *Idotea*
769 *balthica* –Isopod–) from the figure due to non-available data for maternal effects (*Idotea*
770 *balthica*) or the type of male harm (*Caenorhabditis ramnei*). See accompanying data for
771 details.

772 **Figure S2 | Male harm and scope for maternal effects**

773 A) Percentage of the 87 species for which parental care (light green), extended gestation
774 (dark green) and extended maternal provision (grey), respectively from left to right, has been
775 reported in the literature. B) Summary of indirect evidence (i.e. mostly based on descriptions
776 of male genitalia; see methods and SM) for male adaptations that may be directly harmful to
777 females (i.e. traumatic insemination, including genital wounding and ablation). External
778 genital mutilation is common in some spiders, where maternal care in the form of egg-
779 protection is widespread (Řezáč 2009; Mouginot *et al.* 2015; Nakata 2016). External genital
780 mutilation likely evolved in response to sperm competition by preventing females from
781 effectively re-mating. However, there is no evidence to date that it reduces female fitness and
782 it does not affect receptivity to male mating attempts (Řezáč 2009; Mouginot *et al.* 2015;
783 Nakata 2016). The numbers beside red bars represent the number of species within the group
784 that are estimated to exhibit such male harm adaptations. Green-shaded circles reflect
785 evidence in the literature of parental care, extended gestation and extended maternal
786 provisioning in the taxon; respectively, from left to right. See accompanying data for details.

787 **Figure S1**.

788



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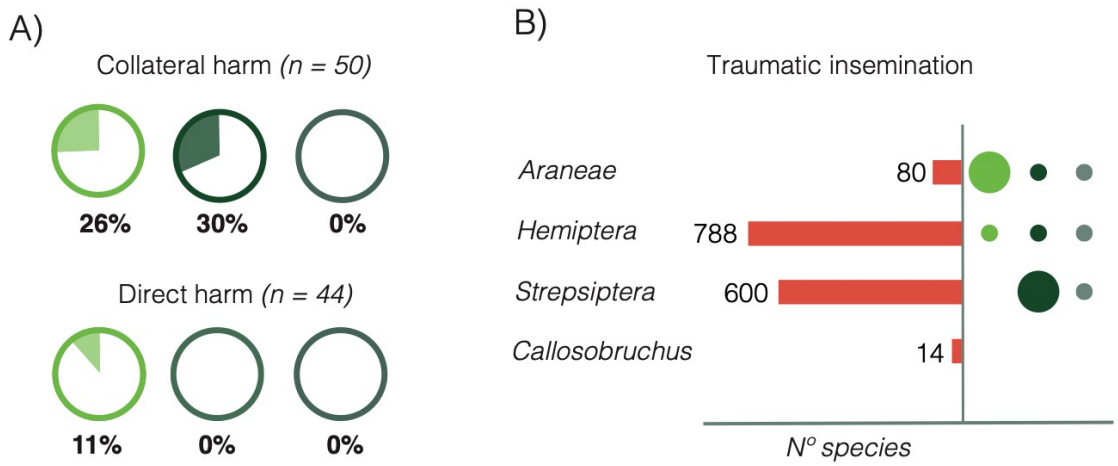
790 **Figure S2**.



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