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Kin discrimination and demography modulate patterns of sexual conflict

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Recent years have seen an explosion of interest in the overlap between kin selection and sexual selection, particularly concerning how kin selection can put the brakes on harmful sexual conflict. However, there remains a significant disconnect between theory and empirical research. Whilst empirical work has focused on kin-discriminating behaviour, theoretical models have assumed indiscriminating behaviour. Additionally, theoretical work makes particular demographic assumptions that constrain the relationship between genetic relatedness and the scale of competition, and it is not clear that these assumptions reflect the natural setting in which sexual conflict has been empirically studied. Here, we plug this gap between current theoretical and empirical understanding by developing a mathematical model of sexual conflict that incorporates kin discrimination and different patterns of dispersal. We find that kin discrimination and group dispersal inhibit harmful male behaviours at an individual level, but kin discrimination intensifies sexual conflict at the population level.

Since their inception, the theories of kin selection and sexual selection have been subjected to intense research within evolutionary biology, but traditionally they have had surprisingly little interaction with one another[1–3]. Recent years have seen an explosion of interest in the interplay between these two processes[3], both theoretically and empirically, with a particular focus on how kin selection can shape the evolution of sexual conflict[4–16]. Specifically, one widespread consequence of sexual conflict is the evolution of male traits that inflict harm upon females[17]. Such harming behaviour not only reduces a female's fitness but can also have pronounced repercussions for the population as a whole, in an outcome akin to the 'tragedy of the commons'[4,18–23]. Kin selection might curb the evolution of this harmful behaviour by aligning the interests of different individuals, which implies that reproductive cooperation may not be limited to a few highly social species[14,24], as currently surmised.

However, there is a major disconnect between theory and empirical research on this topic (Table 1). On the one hand, theory has focused on population viscosity as the driver of kin selection[25,26], whereby limited dispersal of individuals means that social partners tend to be genetically related and hence indiscriminate altruistic behaviour may evolve[3–5,8–10]. On the other hand, empirical research has overwhelmingly focused on kin discrimination[25,26], whereby individuals are capable of identifying their genealogical relatives and adjusting their behaviour accordingly[6,7,11–14,16]. This disconnect implies that current theoretical models cannot make predictions as to how sexual conflict evolves when individuals are capable of kin discrimination. Moreover, current theoretical work[4,9] makes very particular assumptions about dispersal patterns, such that genetic relatedness and resource competition become tied together in a potentially artificial way. Specifically this work has assumed purely viscous populations, in which reduced dispersal increases relatedness of neighbours but also intensifies competition between kin, with these two factors having opposite effects on sexual conflict. Consequently, it remains unclear how kin selection will act to modulate sexual conflict—if at all—in ecological scenarios where relatedness and competition are not so tightly intertwined.

Here we bridge the gap between theoretical and empirical understanding of the impact of kin selection on sexual conflict, by incorporating these key empirical aspects into a new theoretical model of male harming behaviour. First, we incorporate kin-discriminating behaviour and contrast its evolution with indiscriminate harming. Second, we explore how kin selection modulates sexual conflict under different patterns of dispersal where relatedness and competition are not intertwined. Specifically, we (1) develop an 'open model'[27,28] that describes relatedness and competition in general terms, thereby capturing the essential selective forces that shape sexual conflict; (2) explore a range of 'closed model'[27,28] demographic scenarios to investigate how sexual conflict evolves in empirically relevant systems; and (3) compare the impact of harmful male behaviour at the population level under different dispersal patterns and in the presence or absence of kin discrimination. Our overall aim is to provide a mathematical framework that delivers both concrete theoretical predictions and improved conceptual understanding as to how sexual conflict evolves in empirically relevant scenarios.

The role of relatedness and the scale of competition

We consider a population divided into social groups, with each group comprising males and females who are interacting with each other in fitness-modulating ways. Our focus is on male harming behaviour: to the extent that a male harms a female, he increases his share of the paternity of her offspring but reduces the overall number of offspring that she is able to produce—and, accordingly, reduces the overall fecundity of females and males within his group. Following reproduction, all adults die and juveniles 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, juveniles mature to adulthood, returning the population to the beginning of the life cycle.

Mathematically, we may express a male's competitiveness for mating success as being proportional to $f_m(y)$, where y is his investment into harming, and we may express a female's fecundity as being

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proportional to $f_f(Y)$, where Y is the harm that she experiences. Following the standard tragedy-of-the-commons approach[4], we assume that the harm experienced by a female is equal to the average level of harming among the males in her patch (see Supplementary Information and Extended Data Fig. 4 for the consequences of relaxing this assumption to cover cases where females are harmed by only a subset of the males in her social group). Accordingly, a female's relative fitness is $W_f = f_f(Y)/(af_f(Y) + (1-a)f_f(\bar{Y}))$, and a male's relative fitness is $W_m = (f_m(y)/f_m(Y))(f_f(Y)/(af_f(Y) + (1-a)f_f(\bar{Y})))$, where $\bar{Y}$ is the average level of harm in the population. Note that a male's fitness is modulated not only by his own competitiveness for mates but also by the fecundity of the females in his group, and the fecundity of the local females is dependent on the average level of harm exhibited by the males in the group—including the focal male himself. In other words, the focal male's harming phenotype, y, directly impacts on the level of harming, Y, suffered by the females in his group. Performing a kin selection analysis[29] (see Supplementary Information), we find that natural selection favours an increase in male harm when $B(1-r_{mm}) - C(1-a)(r_{fm} + r_{mm}) > 0$, where $B = (\partial f_m(y)/\partial y|_{y=Y=\bar{Y}})/f_m(\bar{Y})$ is the benefit for a male of harming females, $C = -(\partial f_f(Y)/\partial Y|_{Y=\bar{Y}})/f_f(\bar{Y})$ is the cost for a female of being harmed, r_{fm} is the relatedness between local adult females and local adult males, and r_{mm} is the relatedness between local adult males (see Supplementary Information).

A male who invests more into harming seizes a greater share of the overall mating success of the males with whom he competes, yielding a direct-fitness benefit B. However, the corresponding loss of mating success by local males (who are related to the actor by r_{mm}) yields an inclusive-fitness loss Br_{mm}. Harming also reduces the overall number of offspring produced by local females (who are related to the actor by r_{fm}) and local males by C, yielding inclusive-fitness losses of Cr_{fm} and Cr_{mm}. Owing to local competition (a), this translates into a loss of only $C(1-a)$ surviving offspring for both local females and local males. Harm, therefore, affects males in two different ways: directly through sexual competition between males (Br_{mm}), and indirectly through reduction of the overall number of offspring produced by harmed females (Cr_{mm}). Consequently, relatedness between males has a greater potential to shape harm in the population than relatedness between females and males, particularly under localized competition (larger a; Fig. 1).

The above condition captures the selective forces modulating the evolution of harm—in particular, the role of demography (a, r_{mm} and r_{fm}) and the details as to how harm translates into fecundity (B and C). We now narrow our focus directly onto demography by rearranging this condition into the form $C/B < A$, where $A = (1-r_{mm})/((1-a)(r_{fm} + r_{mm}))$ is the potential[30] for harm associated with the particular demographic context. That is, a higher A means that the condition for an increase in harming to be favoured is less stringent. Accordingly, harm is more likely to be favoured with lower relatedness (r_{mm} and r_{fm} closer to 0; Fig. 1a,b) and with more local competition (a closer to 1; Fig. 1c,d).

Our result is consistent with much of the work on kin selection and sexual conflict (Table 1), both theoretically and empirically. Within the theoretical literature, there has been a particular focus on population viscosity as the driver of kin selection[3–5,8–10]. That is, limited dispersal results in individuals being genetically related to their social partners. Łukasiewicz et al.'s[15] experimental-evolution study of bulb-mites found that increased relatedness is associated with a reduction in male harm. The coefficients of relatedness (r_{mm} and r_{fm}) appearing in the above condition capture the results of Łukasiewicz et al.[15], with their increase yielding a lower potential for harm (Fig. 1a,b). However, theory predicts that limited dispersal does not necessarily inhibit male harm: whilst limited dispersal does increase relatedness (higher r), which tends to disfavour harm, it also intensifies local competition (higher a), which tends to favour harm[9,27,31–34], and this too is captured by our condition (Fig. 1c,d).

There is, therefore, entanglement between relatedness and scale of competition when harming behaviour is expressed indiscriminately in the context of a purely viscous population. However, most of the empirical research on this topic has focused on the role of kin discrimination in modulating sexual conflict (Table 1). Specifically, individuals identify which of their social partners are kin and adjust their behaviour accordingly. Mathematically, this is equivalent to varying the relatedness coefficients while holding the scale of competition constant, with the potential for harm decreasing as the relatedness coefficients increase (Fig. 1a.b). Kin discrimination, therefore, effectively disentangles relatedness and scale of competition.

Another possible way to disentangle relatedness and scale of competition involves holding relatedness constant while varying the scale of competition (Fig. 1c,d), which is expected to result in a reduction in harm as the scale of competition becomes more global (lower a). This can be achieved when competition occurs between groups of individuals, such that low-harming, high-fecundity groups are able to competitively displace their high-harming, low-fecundity rivals. This has been explored in populations of water-striders, where male aggression is disfavoured when there is between-group competition[35,36]. Outwith sexual conflict, the effect of varying the scale of competition while holding relatedness fixed has been explored through dispersal of groups of relatives—budding dispersal[37–40]—which is one possible scenario that, mathematically, leads to the same result.

A predictive model for empirical systems

Our open model suggests that kin discrimination and budding dispersal can be important mechanisms in defining how sexual conflict will evolve. Nevertheless, while this model generalizes much of the work that has been done to date in the context of sexual conflict and kin selection (Table 1), it fails to offer concrete predictions on how harmful male traits will evolve in biological systems, particularly given how demographic factors can affect both relatedness coefficients and the scale of competition.

To gain predictive power, we explicitly define the fecundity of a focal female as $f_f = 1 - Y$ and the competitiveness for mating success of a focal male as $f_m = 1 + y^\beta$, where Y is the level of harm present in the focal patch, y is the level of harm of a focal male and β determines the marginal benefit of harming females (see Supplementary Information). Throughout this section, we consider an infinite population divided into patches[41] containing three adult females and three adult males (see Fig. 2 for an illustration). Each female mates a large number of times, and each time with a randomly and independently chosen male from her group—with the probability that she mates with a particular male being proportional to his relative competitiveness for mating success. Females always disperse to new patches, while males disperse to a new patch with probability d_m. This is close to the conditions explored in empirical studies[6,7,11–14,16] and allows us to disregard the potential effect of inbreeding depression, which would require its own study to do it justice. Moreover, while most of those empirical studies have one adult female interacting with three adult males, their experimental populations (and most wild populations of those species) do not have male-biased sex ratios, so here we assume a 3:3 sex ratio (but note, this does not qualitatively change our results—Extended Data Fig. 1).

Absence of kin discrimination and budding dispersal. First, let us focus on the level of harm in the absence of kin discrimination and budding dispersal. An increase in the level of harm is favoured when:

$$\frac{(1-r_{mm})(\bar{Y}^{\beta-1}\beta)}{1+\bar{Y}^\beta} - r_{mm}(1-a)\frac{1}{1-\bar{Y}} > 0, \quad (1)$$

where $\bar{Y}$ is the average level of harm in the population, $a = (1-d_m)^2/2$ is the scale of competition and $r_{mm} = 1/3 + (2/3)(1-d_m)^2 r$ is the

Table 1 | Literature on the impact of kin selection on the evolution of sexual conflict

Authors	Approach	Kin selection mechanism	Notes
Rankin[4]	Theoretical—mathematical model	Population viscosity	Rankin's model cannot be used to study sex-biased dispersal due to a mathematical error in his analysis. The results can be captured by our model when dispersal is not sex-biased.
Wild et al.[5]	Theoretical—mathematical model	Population viscosity	Insofar as there is a conflict between females and males, our model captures their results.
Pizzari & Gardner[3]	Theoretical—verbal model	Population viscosity Kin discrimination	The verbal models dedicated to sexual conflict between females and males are captured by our model.
Carazo et al.[6]	Empirical—facultative adjustment of behaviour	Kin discrimination	Males of *Drosophila melanogaster* can discriminate between genealogically related and unrelated males, increasing harm to females when interacting with unrelated males. Our model yields the same qualitative result as this experimental study.
Chippindale et al.[7]	Empirical—facultative adjustment of behaviour	Kin discrimination	Replication of Carazo et al.[6]. They were unable to replicate the same patterns, and therefore our model does not yield the same qualitative results.
Pizzari et al.[8]	Theoretical—mathematical model	Population viscosity	Insofar as there is a conflict between females and males, our model captures their results.
Faria et al.[9]	Theoretical—mathematical model	Population viscosity	Extends Rankin's[4] result to sex-biased dispersal. Captured by our model.
Hollis et al.[11]	Empirical—facultative adjustment of behaviour	Kin discrimination	Extension of Carazo et al.[6]. They found familiarity between males to be important for them to reduce the harm they express. Our model yields the same qualitative result as this experimental study.
Martin & Long[12]	Empirical—facultative adjustment of behaviour	Kin discrimination	Replication of Carazo et al.[6] with high relatedness (that is, inbreed lines, $r > 0.5$). They are unable to replicate the same patterns, and therefore our model does not yield the same qualitative results.
Faria et al.[10]	Theoretical—mathematical model	Population viscosity	Maternal-origin and paternal-origin genes have different levels of relatedness, generating an intragenomic conflict between the two classes of genes. This result is consistent with our model.
Tan et al.[13]	Empirical—facultative adjustment of behaviour	Kin discrimination	Males of *Gallus gallus* can discriminate between genealogically related and unrelated males, increasing harm to females when interacting with unrelated males. Our model yields the same qualitative result as this experimental study.
Le Page et al.[14]	Empirical—facultative adjustment of behaviour	Kin discrimination	Extension of Carazo et al.[6]. They found that both familiarity and genealogical relatedness is necessary for males to recognize genealogical related males and, therefore, reduce the harm that they express. Our model yields the same qualitative result as this experimental study.
Łukasiewicz et al.[15]	Empirical—experimental evolution	Population viscosity	Males of *Rhizoglyphus robini* reduce harm to females when evolving in populations with higher levels of genetic relatedness. Our model yields the same qualitative result as this experimental study.
Lymbery & Simmons[16]	Empirical—facultative adjustment of behaviour	Kin discrimination	Males of *Callosobruchus maculatus* can discriminate between genealogically related and unrelated males, increasing harm to females when interacting with unrelated males. Familiarity between males is necessary. Our model yields the same qualitative result as this experimental study.
Berg et al.[47]	Empirical—facultative adjustment of behaviour	Kin discrimination	Replication of Lymbery & Simmons[16]. Male relatedness improved female survival but did not affect lifetime reproductive success. Our model is consistent with their result for survival but does not yield the same qualitative result for lifetime reproductive success, for which they did not replicate the same pattern as Lymbery & Simmons[16].

relatedness between males in a patch (where $r = 1/(5 + d_m(2 - d_m))$ is the relatedness between individuals born in the same patch; see Supplementary Information for details). Note that a specifies the likelihood of a focal male juvenile competing with other males in the patch for future breeding opportunities, which occurs when neither disperses to other patches.

We can use inequality (1) to calculate the optimal level of harm for indiscriminating males (Fig. 3a; see Supplementary Information for details). We find that, as male dispersal decreases, relatedness increases (larger r_{mm}), which promotes harm. This, however, is counteracted by the scale of competition (a), which becomes more local and inhibits harm. Nevertheless, the presence of female-biased

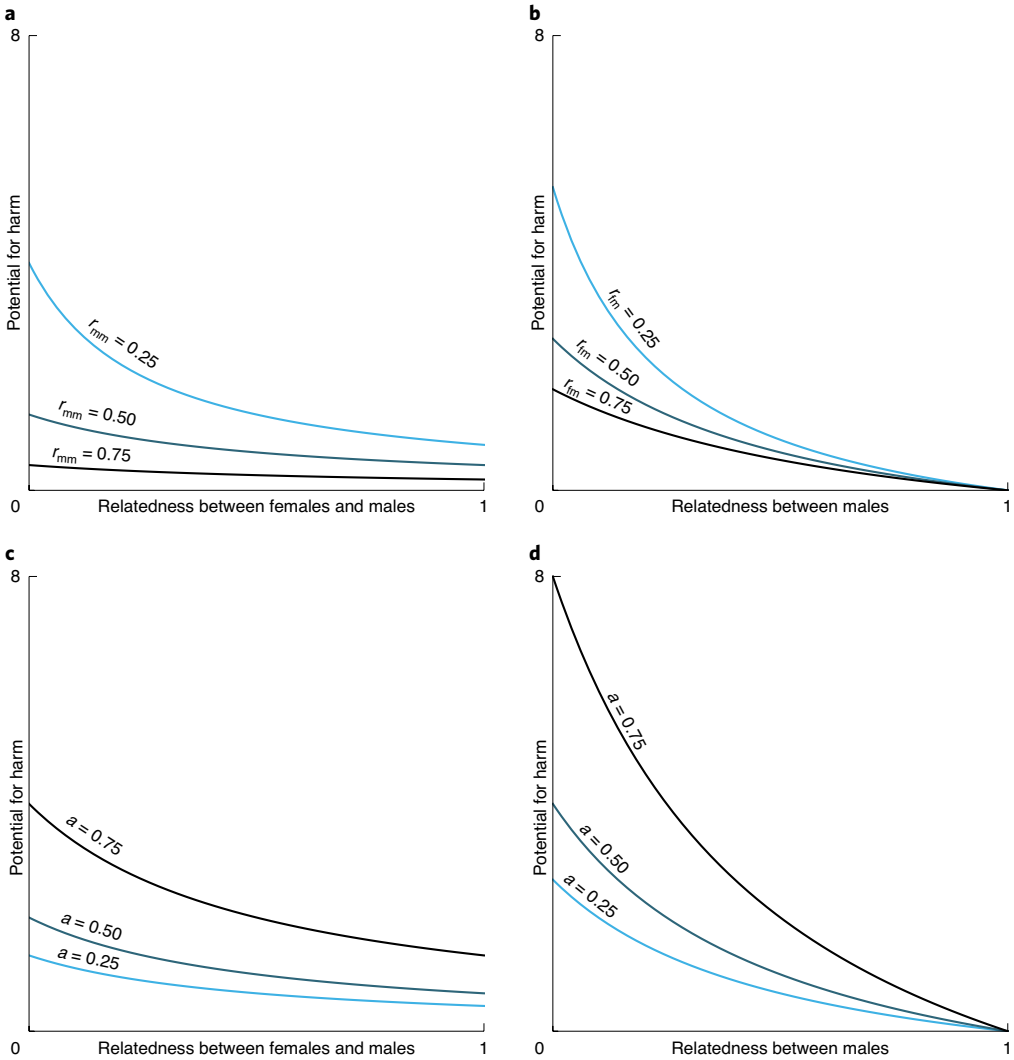

Fig. 1 | Potential for harm to evolve as a function of relatedness and scale of competition. a–d, The harm to which females are subjected by males is expected to increase as relatedness decreases (**a**,**b**) and as the intensity of local competition increases (**c**,**d**). **a**,**b**, The scale of competition: $a = 0.25$. **c**, The relatedness between males: $r_{mm} = 0.50$. **d**, The relatedness between females and males: $r_{fm} = 0.50$.

dispersal ($d_f = 1$) means that the two effects do not exactly cancel out[9]. That is, whilst dispersal by each sex has a symmetrical impact on the scale of competition, they can have asymmetrical impact on relatedness if the sexes experience different reproductive skews. Accordingly, decreased male dispersal favours a higher level of harm due to increased kin competition (Fig. 3a).

Presence of kin discrimination and absence of budding dispersal. We now consider the consequences of kin discrimination. Specifically, we assume that individuals who were born on the same patch are able to recognize each other later in life. This has been termed 'familiarity'[14,42–44], and is thought to represent the most common cue for kin recognition in nature[43–45] and to be required even when direct 'genetic' kin recognition is present[14,44]. In our model, for all $0 < d_m < 1$, a focal male is either interacting with (1) two familiar males ($r_{2mm} = 1/3 + (2/3)r$), (2) one familiar and one unfamiliar male ($r_{1mm} = 1/3 + (1/3)r$) or (3) two unfamiliar males ($r_{0mm} = 1/3$; see Supplementary Information). Accordingly, a male is expected to show a low level of harm when interacting with two familiar males, an intermediate level of harm when interacting with one familiar and one unfamiliar male, and a high level of harm when interacting with two unfamiliar males (Fig. 3a; for simulation results see Extended Data Fig. 2a).

Male dispersal continues to affect relatedness, but only through its impact on relatedness of familiar males ($r = 1/(5 + d_m(2 - d_m))$; see Supplementary Information). In contrast, its impact on kin competition remains unchanged ($a = (1 - d_m)^2/2$) and, accordingly, increased male dispersal reduces harm by making the competition more global (lower a). This suggests that, in kin-discriminating species, an experimental-evolution regime that increases relatedness through reduced dispersal need not necessarily lead to a reduction in harm (Fig. 3a; for simulation results see Extended Data Fig. 2a). Specifically, a male interacting with familiar males may still exhibit lower harm in comparison to one interacting with unfamiliar males, but the harm expressed within each treatment may actually be higher with reduced dispersal due to an increase in local competition (larger a).

Absence of kin discrimination and presence of budding dispersal. It is also possible to maintain a constant scale of competition (fixed a) while varying relatedness between social partners through budding dispersal[39,40]. As before, and without considering kin discrimination, an increase in harm is favoured whenever the right-side of inequality (1) is positive. However, now kin competition depends on budding dispersal ($a = (1 - d_B)^2$, with d_B being the

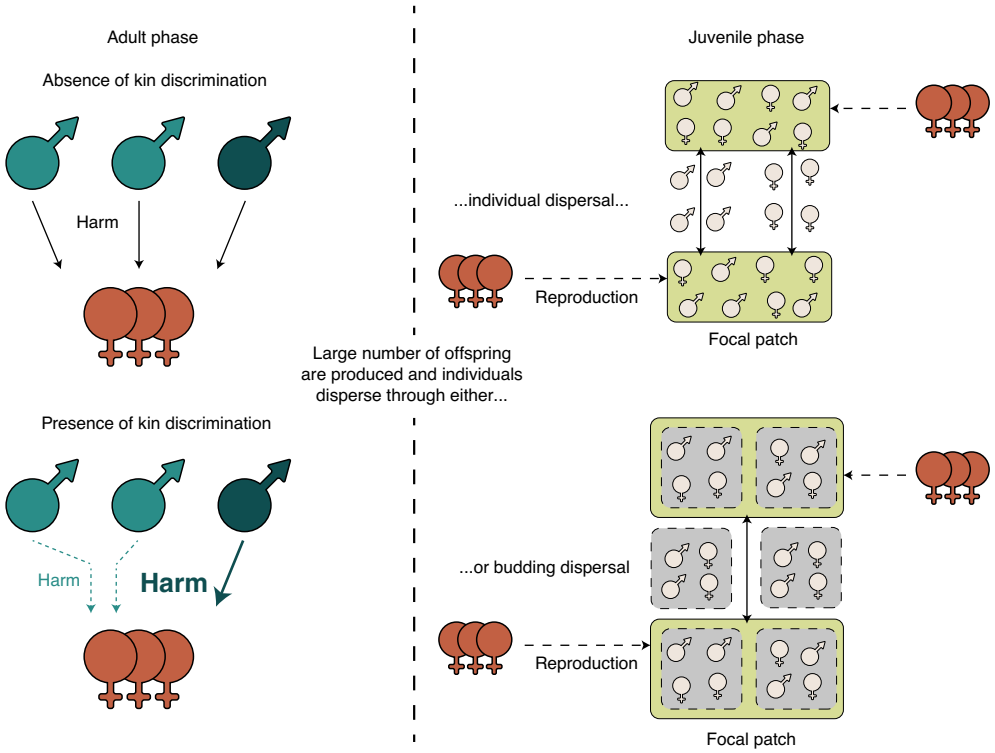

Fig. 2 | Kin selection model of sexual conflict. During the adult phase of the model, males can harm females. In the absence of kin discrimination, all males exhibit the same level of harm. In the presence of kin discrimination, males that recognize other males as being related reduce the level of harm. In contrast, males that recognize other males as being unrelated increase the level of harm. During the juvenile phase of the model, individuals can either disperse from their patch individually—with juvenile females and juvenile males competing with other juvenile females and juvenile males, respectively—or in groups, with groups competing with other groups.

probability of a group dispersing to a new patch; see Supplementary Information). Following budding dispersal, males can still disperse between groups but without affecting the scale of competition (a)—only relatedness between males ($r_{mm} = 1/3 + (2/3)(1 - d_m)^2 r$; see Supplementary Information for details). If we consider full budding dispersal ($d_B = 1$), then competition occurs exclusively at a global scale ($a = 0$) with increased male dispersal leading to reduced relatedness and, therefore, a higher level of harm (Fig. 3b). Compared to a scenario where only individual dispersal is present, budding dispersal generally leads to lower levels of harm (Fig. 3b). The exception is when there is full male dispersal ($d_m = 1$), in which case the level of harm is the same in both scenarios (Fig. 3b).

Presence of kin discrimination and budding dispersal. Adding kin discrimination into this model recovers the previous result, where males interacting with familiar individuals manifest a lower level of harm, except that now male dispersal does not have an appreciable effect on harm (Fig. 3c; for simulation results see Extended Data Fig. 2b). This again suggests that, in species with kin discrimination, increased relatedness through limited dispersal may not be relevant in reducing the level of harm. Limited dispersal still affects the likelihood of individuals finding and interacting with related individuals, but its role in reducing the harm manifested by the males in those encounters may be restricted.

Discussion

We have developed kin selection models to formally investigate how relatedness and the scale of competition modulate sexual conflict between females and males. Using open models, we have generalized and synthesized the theoretical and empirical work done in the fields of kin selection and sexual conflict (Table 2). Using closed models, we have derived concrete predictions that can be tested in future empirical studies (Table 2). Both approaches highlight the role of kin discrimination and budding dispersal in modulating the extent to which males harm females. Accordingly, through the disentanglement of relatedness and kin competition, kin discrimination and budding dispersal increase the scope for kin selection to curb the evolution of harming behaviour. These findings apply both when harm is inflicted upon a female by her mates and her unsuccessful suitors (for example, sexual harassment) and when it occurs exclusively during mating (for example, toxic ejaculates or traumatic insemination). Overall levels of male harm are predicted to be lower in the latter case (Table 2; see Supplementary Information for details), but our main qualitative conclusions hold in both scenarios.

Compared to population viscosity, kin discrimination allows for a finer-grained adjustment of social behaviour in response to genetic relatedness. Through kin discrimination, individuals facultatively assess their relatedness to social partners and behave accordingly[26,29]. Kin discrimination strongly contrasts with population viscosity, where an individual's behaviour is selected according to the average relatedness of the actor to their social partners[29]. Nevertheless, population viscosity has been considered an important mechanism in the kin selection literature due to its simplicity. Unlike kin discrimination, it does not require behavioural plasticity reliant on complex (and probably costly) cognitive processes, meaning that it can occur even in simple organisms[26]. Here, we draw attention to another possible cost associated with kin discrimination.

When harming females, males gain a relative advantage concerning other males by siring more offspring from those females. However, this benefit is only relative, with the total number of offspring produced by these females being smaller than it would

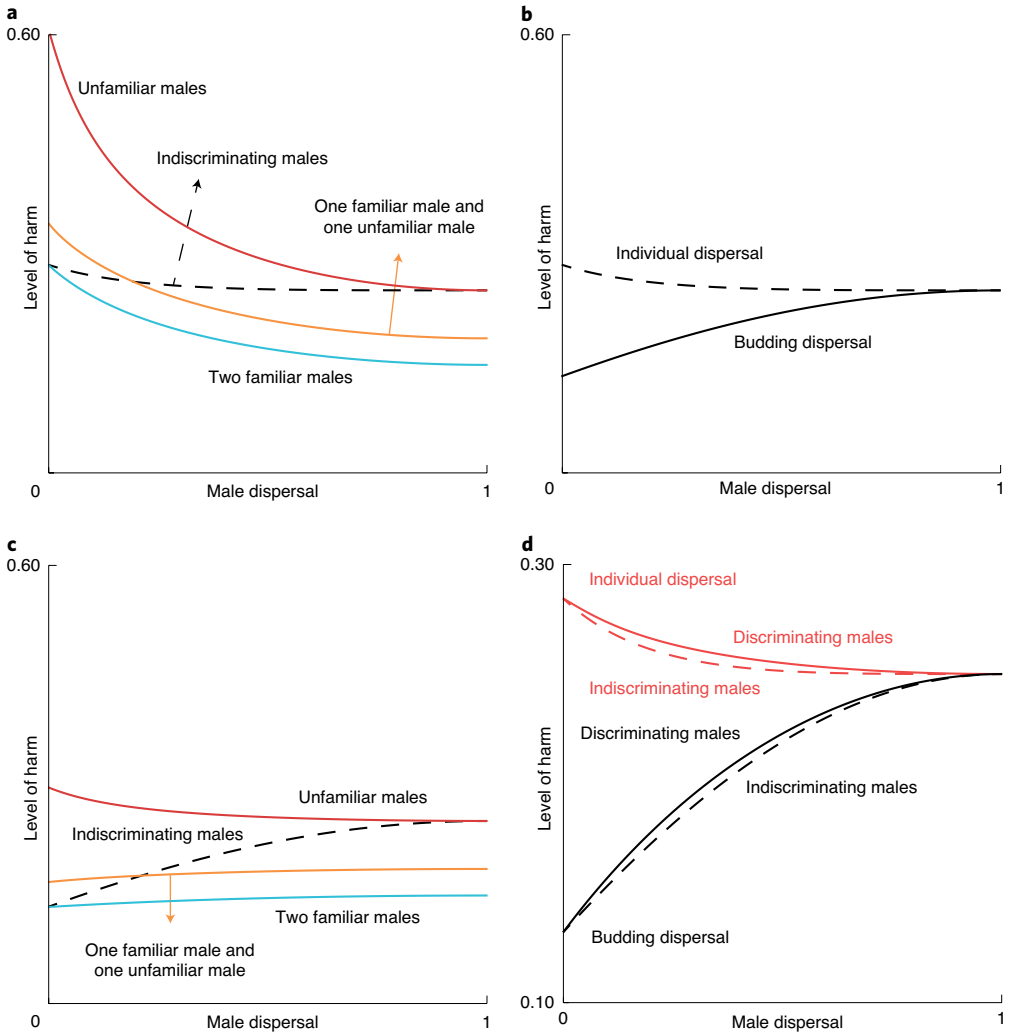

Fig. 3 | Optimal level of harm as a function of male dispersal (d_m) and the average of harm in the population. Level of harm versus male dispersal. **a**, In the presence of kin discrimination and absence of budding dispersal, the optimal level of harm expressed by males decreases as male dispersal (d_m) increases for both indiscriminating and discriminating males, but the decrease is more pronounced when kin discrimination is present. **b**, In the absence of kin discrimination and presence of budding dispersal, the optimal level of harm expressed by males increases as male d_m increases. **c**, In the presence of kin discrimination and budding dispersal, the optimal level of harm for discriminating males decreases if males are interacting only with unfamiliar males, and increases if males are interacting with familiar males. For indiscriminating males, the optimal level of harm increases as d_m increases. Regardless of the absence (**a**) or presence (**b**) of budding dispersal, males interacting with unfamiliar males express a high level of harm, males interacting with one familiar male and one unfamiliar male express an intermediate level of harm, and males interacting with two familiar males express a low level of harm. **d**, The resultant average harm in the population is higher when individuals are capable of kin discrimination than when compared to its absence, and lower in the presence of budding dispersal when compared to individual dispersal. The following parameters were used: marginal benefit of harm: $\beta = 0.5$; female dispersal rate: $d_f = 1$; number of females: $n_f = 3$; and number of males: $n_m = 3$. **b**–**d**, Budding $d_B = 1$.

be in the absence of harm. Such a cost describes what is known as a tragedy of the commons, whereby individual competition for resources—here, offspring provided by females—reduces the average productivity of the whole group[4,9,18,19,45]. This prompts the question as to which mechanism—kin discrimination or population viscosity—is worse for the population as a whole.

Kin recognition makes an actor more altruistic to those that he recognizes as kin but makes him less altruistic to those that he does not recognise as kin. Therefore, it is not clear how kin recognition should impact the overall level of altruism in the group. Faria & Gardner[45] show that kin discrimination increases selfishness in the group whenever the optimal value of the trait under study changes convexly with relatedness. This is the case in our model, with the level of harm expressed by males being a convex function of relatedness (Extended Data Fig. 3). As a consequence, kin discrimination leads to higher average harm in the population when compared to its absence, both when organisms disperse individually or in groups (Fig. 3d). As male dispersal approaches 0 or 1, the proportion of patches comprising either only familiar or only unfamiliar males, respectively, increases. At this point, presence versus absence of kin discrimination is irrelevant (Fig. 3d) because males experience only one type of social condition.

Therefore, kin discrimination may lead to a decrease in the overall productivity of a population and consequently increase sexual conflict between males and females (Table 2). If kin discrimination is also associated with cognitive costs, this suggests that kin discrimination should be more prevalent in species with intermediate dispersal levels[46]. Specifically, with low dispersal, individuals are likely to interact with kin, making kin discrimination redundant, while, with high dispersal, individuals are unlikely to encounter kin,

Table 2 | Main conclusions of our study

Kin selection approach	Conclusions regarding the evolution of sexual conflict
Open and closed models	Relatedness and kin competition are often entangled. Increased relatedness, in the absence of changes in kin competition, leads to lower levels of harmful phenotypes. Increased kin competition, in the absence of changes in relatedness, leads to higher levels of harmful phenotypes.
Open model	Relatedness can change independently through kin discrimination. Kin competition can change independently through group competition for reproductive patches.
Closed model	Decreased dispersal may increase the level of harmful phenotypes through increased kin competition when individuals are capable of kin discrimination. Dispersal may have little effect on the level of harmful phenotypes in the presence of kin discrimination and group competition for reproductive patches.
Populational consequences	Kin discrimination can lead to increased sexual conflict at the population level and, therefore, decreased population productivity.

which again makes kin discrimination unnecessary. Intermediate levels of dispersal, however, are also when there is a higher difference between population viscosity and kin discrimination in terms of productivity. This creates an interesting trade-off—the demographic conditions that make kin discrimination advantageous for the individuals are also those where kin discrimination is more costly for the population. Far from resolving the negative consequences of sexual conflict at the population level, our results suggest that kin discrimination actually contributes to setting the scene for an evolutionary tragedy of the commons. Presence of cognitive costs may erode the trade-off by making the evolution of kin discrimination too costly in the first place, but this is beyond the scope of our model. Nevertheless, it could be an interesting avenue for future theoretical and empirical research.

Reporting Summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

Code availability

Code used for the simulations is available at https://github.com/GSFaria-wasp/Sexual-conflict.git.

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References

1. Cronin, H. *The Ant and the Peacock: Altruism and Sexual Selection from Darwin to Today* (Cambridge Univ. Press, 1993).
2. Boomsma, J. J. Kin selection versus sexual selection: why the ends do not meet. *Curr. Biol.* **17**, R673–R683 (2007).
3. Pizzari, T. & Gardner, A. The sociobiology of sex: inclusive fitness consequences of inter-sexual interactions. *Phil. Trans. R. Soc. Lond. B* **367**, 2314–2323 (2012).
4. Rankin, D. J. Kin selection and the evolution of sexual conflict. *J. Evol. Biol.* **24**, 71–81 (2011).
5. Wild, G., Pizzari, T. & West, S. A. Sexual conflict in viscous populations: the effect of the timing of dispersal. *Theor. Popul. Biol.* **80**, 298–316 (2011).
6. Carazo, P., Tan, C. K. W., Allen, F., Wigby, S. & Pizzari, T. Within group male relatedness reduces harm to females in *Drosophila*. *Nature* **505**, 672–675 (2014).
7. Chippindale, A. K., Berggren, M., Alpern, J. H. M. & Montgomerie, R. Does kin selection moderate sexual conflict in *Drosophila*? *Proc. R. Soc. Lond. B* **282**, 120151417 (2015).
8. Pizzari, T., Biernaskie, J. M. & Carazo, P. Inclusive fitness and sexual conflict: how population structure can modulate the battle of the sexes. *BioEssays* **37**, 155–166 (2015).
9. Faria, G. S., Varela, S. A. M. & Gardner, A. Sex-biased dispersal, kin selection and the evolution of sexual conflict. *J. Evol. Biol.* **28**, 1901–1910 (2015).
10. Faria, G. S., Varela, S. A. M. & Gardner, A. Sexual selection modulates genetic conflicts and patterns of genomic imprinting. *Evolution* **71**, 526–540 (2017).
11. Hollis, B. T., Kawecki, T. J. & Keller, L. No evidence that within-group male relatedness reduces harm to females in *Drosophila*. *Ecol. Evol.* **5**, 979–983 (2015).
12. Martin, E. S. & Long, T. A. F. Are flies kind to kin? The role of intra- and inter-sexual relatedness in mediating reproductive conflict. *Proc. R. Soc. Lond. B* **282**, 20151991 (2015).
13. Tan, C. K. W. et al. The contrasting role of male relatedness in different mechanisms of sexual selection in red junglefowl. *Evolution* **71**, 403–420 (2017).
14. Le Page, S. et al. Male relatedness and familiarity are required to modulate male-induced harm to females in *Drosophila*. *Proc. R. Soc. Lond. B* **284**, 20170441 (2017).
15. Łukasiewicz, A., Szubert-Kruszyńska, A. & Radwan, J. Kin selection promotes female productivity and cooperation between the sexes. *Sci. Adv.* **3**, e1602262 (2017).
16. Lymbery, S. J. & Simmons, L. W. Males harm females less when competing with familiar relatives. *Proc. R. Soc. Lond. B* **284**, 20171984 (2017).
17. Pitnick, S. & García-González, F. Harm to females increases with male body size in *Drosophila melanogaster*. *Proc. R. Soc. Lond. B* **269**, 1821–1828 (2002).
18. Lloyd, W. F. *Two Lectures on the Checks to Population* (Oxford Univ. Press, 1833).
19. Hardin, G. The tragedy of the commons. *Science* **162**, 1243–1248 (1968).
20. Holland, B. & Rice, W. R. Experimental removal of sexual selection reverses intersexual antagonistic coevolution and removes a reproductive load. *Proc. Natl Acad. Sci. USA* **96**, 5083–5088 (1999).
21. Le Galliard, J. F., Fitze, P. S., Ferrière, R. & Clobert, J. Sex ratio bias, male aggression, and population collapse in lizards. *Proc. Natl Acad. Sci. USA* **102**, 18231–18236 (2005).
22. Arnqvist, G. & Tuda, M. Sexual conflict and the gender load: correlated evolution between population fitness and sexual dimorphism in seed beetles. *Proc. R. Soc. Lond. B* **277**, 1345–1352 (2010).
23. Berger, D. et al. Intralocus sexual conflict and the tragedy of the commons in seed beetles. *Am. Nat.* **188**, R98–R112 (2016).
24. Lymbery, S. J., Tomkins, J. L. & Simmons, L. W. Male responses to sperm competition when rivals vary in number and familiarity. *Proc. R. Soc. Lond. B* **286**, 20182589 (2019).
25. Hamilton, W. D. The evolution of altruistic behaviour. *Am. Nat.* **97**, 354–356 (1963).
26. Hamilton, W. D. The genetical evolution of social behaviour. I & II. *J. Theor. Biol.* **7**, 1–52 (1964).
27. Gardner, A. & West, S. A. Demography, altruism, and the benefits of budding. *J. Evol. Biol.* **17**, 1707–1716 (2006).
28. Cooper, G. A., Levin, S. R., Wild, G. & West, S. A. Modeling relatedness and demography in social evolution. *Evol. Lett.* **2**, 260–271 (2018).
29. Frank, S. A. *Foundations of Social Evolution* (Princeton Univ. Press, 1998).
30. Gardner, A. Sex-biased dispersal of adults mediates the evolution of altruism among juveniles. *J. Theor. Biol.* **262**, 339–345 (2010).
31. Taylor, P. D. Altruism in viscous populations – an inclusive fitness approach. *Evol. Ecol.* **6**, 352–356 (1992).
32. Taylor, P. D. Inclusive fitness in a heterogeneous environment. *Proc. R Soc. Lond. B* **249**, 299–302 (1992).
33. Queller, D. C. Does population viscosity promote kin selection? *Trends Ecol. Evol.* **7**, 322–324 (1992).
34. West, S. A., Pen, I. & Griffin, A. S. Cooperation and competition between relatives. *Science* **296**, 72–75 (2002).
35. Eldakar, O. T., Dlugos, M. J., Pepper, J. W. & Wilson, D. S. Population structure mediates sexual conflict in water striders. *Science* **326**, 816 (2009).
36. Eldakar, O. T., Wilson, D. S., Dlugos, M. J. & Pepper, J. W. The role of multilevel selection in the evolution of sexual conflict in the water strider *Aquarius remiges*. *Evolution* **64**, 3183–3189 (2010).

37. Haldane, J. B. S. *The Causes of Evolution* (Princeton Univ. Press, 1932).
38. Goodnight, K. F. The effect of stochastic variation on kin selection in a budding-viscous population. *Am. Nat.* **140**, 1028–1040 (1992).
39. Lehmann, L., Perrin, N. & Rousset, F. Population demography and the evolution of helping behaviors. *Evolution* **60**, 1137–1151 (2006).
40. Gardner, A., Arce, A. & Alpedrinha, J. Budding dispersal and the sex ration. *J. Evol. Biol.* **22**, 1036–1045 (2009).
41. Wright, S. Evolution in Mendelian populations. *Genetics* **16**, 97–159 (1931).
42. Penn, D. J. & Frommen, J. G. in *Animal Behaviour: Evolution and Mechanisms* (ed. Kapperler, P.) 55–85 (Springer, 2010).
43. Bradbury, J. W. & Vehrencamp, S. L. *Principles of Animal Communication* (Sinauer Associates, 2011).
44. Holmes, W. G. & Sherman, P. W. Kin recognition in animals: the prevalence of nepotism among animals raises basic questions about how and why they distinguish relatives from unrelated individuals. *Am. Sci.* **1**, 46–55 (1983).
45. Faria, G. S. & Gardner, A. Does kin discrimination promote cooperation? *Biol. Lett.* **16**, 20190742 (2020).
46. Griffin, A. S. & West, S. A. Kin discrimination and the benefit of helping in cooperatively breeding vertebrates. *Science* **302**, 634–636 (2003).
47. Berg, E. C., Lind, M. I., Monahan, S., Bricout, S. & Maklakov, A. A. Kin but less than kind: within-group male relatedness does not increase female fitness in seed beetles. *Proc. R. Soc. B* **286**, 20191664 (2019).

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Author contributions

G.S.F., A.G. and P.C. conceived the study. G.S.F. led the theoretical analysis with input from A.G. G.S.F., A.G. and P.C. wrote the paper.

Competing interests

The authors declare no competing interests.

Additional information

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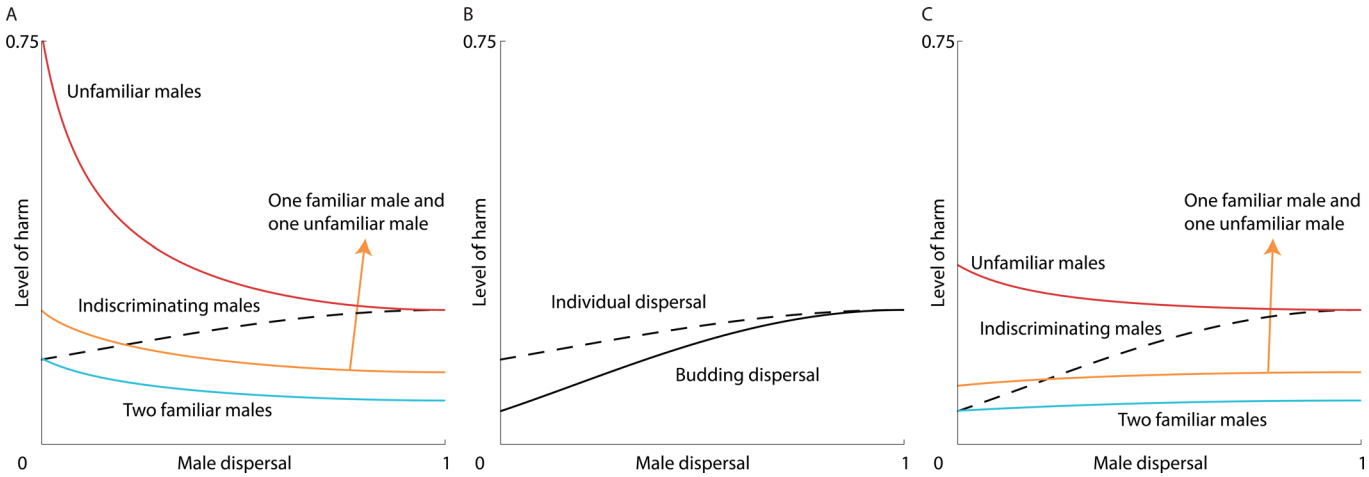
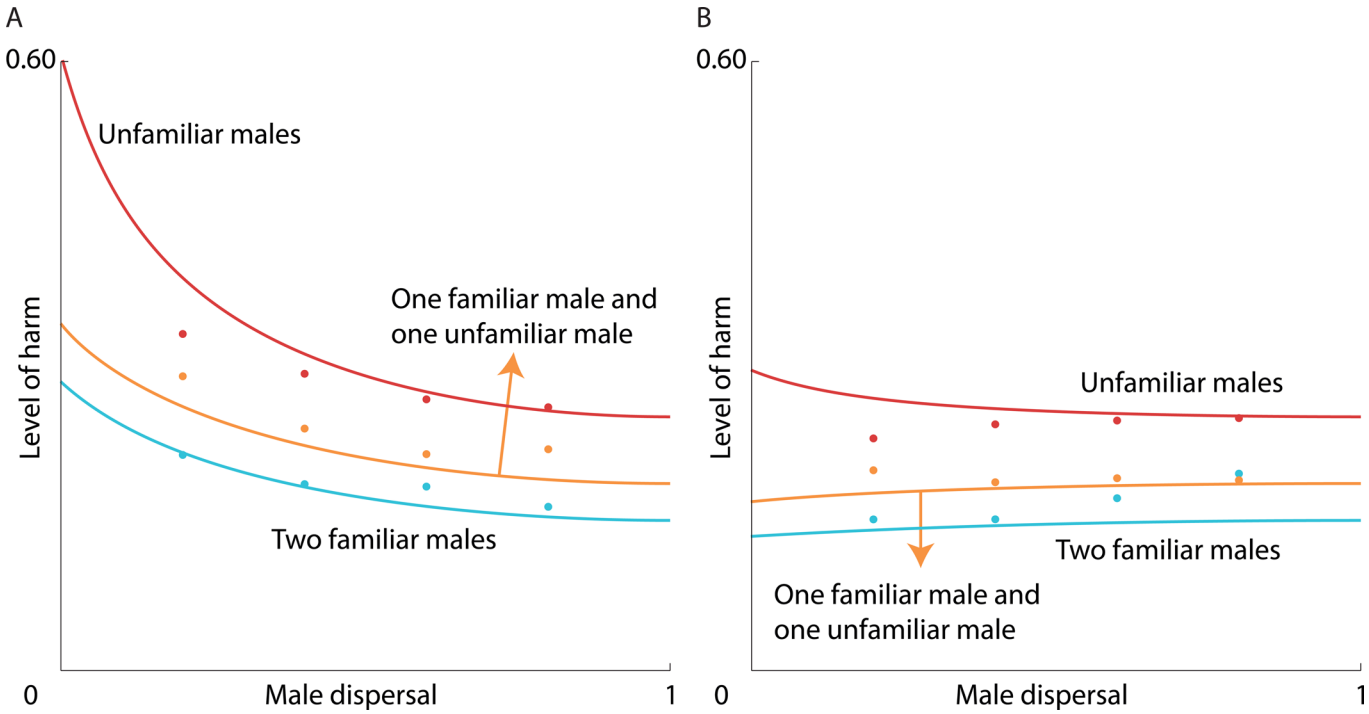
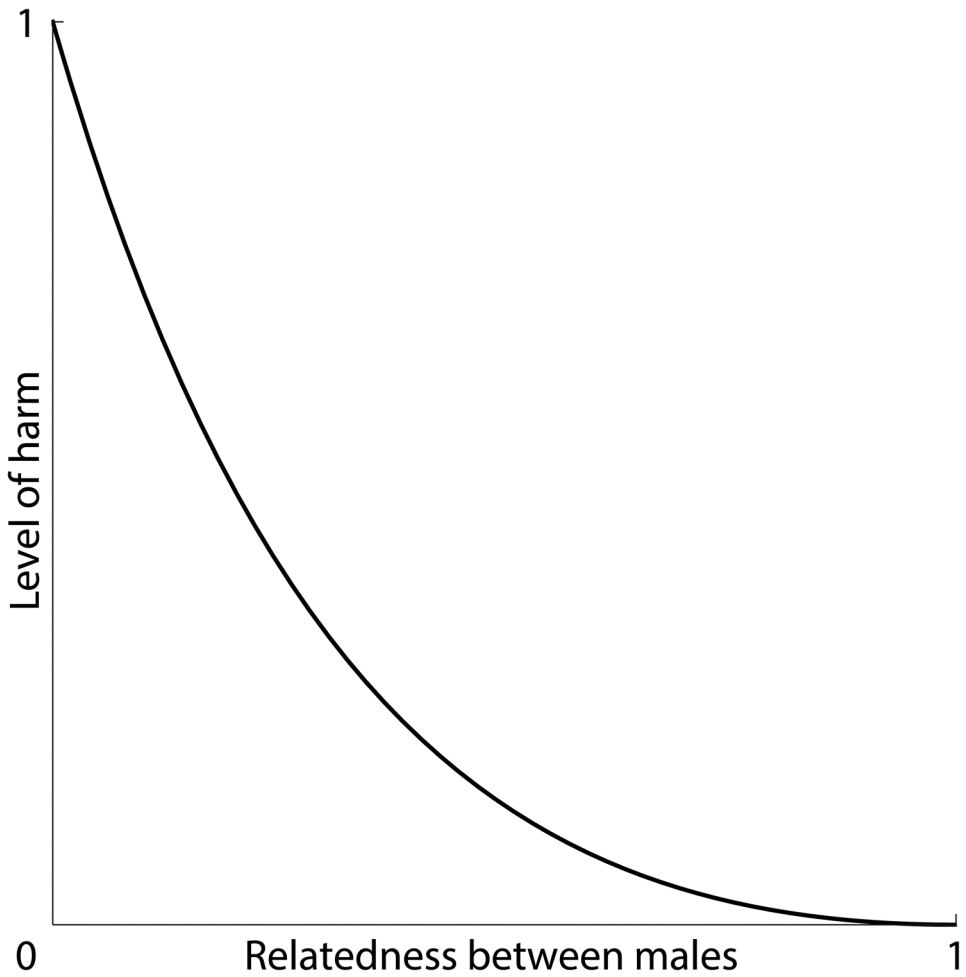
Correspondence and requests for materials should be addressed to G.S.F.



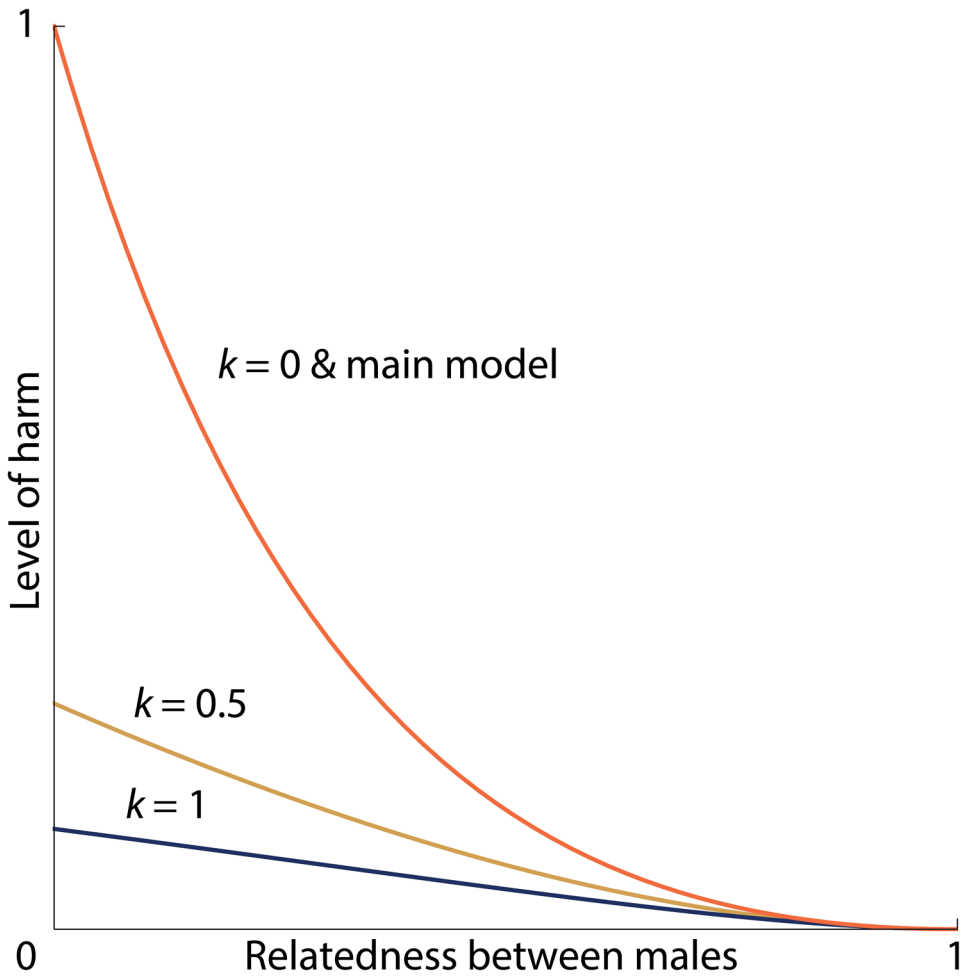
Extended Data Fig. 1 | Optimal level of harm as a function of male dispersal (d_m). In the presence of kin discrimination and absence of budding dispersal (**A**), the optimal level of harm that males express decreases as male dispersal (d_m) increases for discriminating males and increases as male dispersal (d_m) increases for indiscriminating males. In the absence of kin discrimination and presence of budding dispersal (**B**), the optimal level of harm that males express increases as male dispersal (d_m) increases. In the presence of kin discrimination and budding dispersal (**C**), the optimal level of harm for discriminating males decreases if males are interacting only with unfamiliar males and increases if males are interacting with familiar males as male dispersal (d_m) increases. For indiscriminating males, the optimal level of harm that males express increases as male dispersal (d_m) increases. Regardless of absence (**A**) or presence of budding dispersal (**B**), males interacting with unfamiliar males express higher level of harm, males interacting with one familiar male and one unfamiliar male express intermediate level of harm, and males interacting with two familiar males express lower level of harm. For all panels, the following parameters were used: marginal benefit of harm $\beta = 0.5$; female dispersal rate $d_f = 1$; number of females $n_f = 1$; and number of males $n_m = 3$. Additionally, in (**B-C**) budding dispersal rate $d_B = 1$.



Extended Data Fig. 2 | Optimal level of harm in the absence (A) and in the presence (B) of budding dispersal as a function of male dispersal (d_m) for discriminating males. In absence of budding dispersal (**A**), the optimal level of harm that males express decreases as male dispersal (d_m) increases. In the presence of budding dispersal (**B**), as male dispersal (d_m) increases, the optimal level of harm that males express decreases if males are interacting only with unfamiliar males and increases if males are interacting with familiar males. Regardless of absence (**A**) or presence of budding dispersal (**B**), males interacting unfamiliar males express higher level of harm, males interacting with one familiar male and one unfamiliar male express intermediate level of harm, and males interacting with two familiar males express lower level of harm. In both panels (**A-B**), the following parameters were used: marginal benefit of harm $\beta = 0.5$; female dispersal rate $d_f = 1$; number of females $n_f = 3$; and number of males $n_m = 3$. Additionally, in (**B**) budding dispersal rate $d_B = 1$. Dots represent the simulations results, with the following additional parameters used: mutation rate of 0.01; population of 4000 patches; number of generations 5×10^4. Each dot is the average of the last 1×10^4 generations.



Extended Data Fig. 3 | Level of harm as a function of relatedness between males. In the absence of kin discrimination, the level of harm that males express changes convexly with relatedness. The following parameters were used: marginal benefit of harm $\beta = 0.5$; female dispersal rate $d_f = 1$; male dispersal rate $d_m = 0.5$; and relatedness between females and males $r_{fm} = 0$.



Extended Data Fig. 4 | Comparison of different assumptions and how they differ from the main model. When the level of harm that males express affect all the females in the patch ($k=0$), the model is exactly the same as our main model. When harm that the females are subjected to comes half from the male that they mate with and half from the other males ($k=0.5$), the model differs from our main model, with lower levels of harm. When harm that the females are subjected comes exclusively from the male that they mate with ($k=1$), the model differs from our main model, with lower levels of harm. The following parameters were used: marginal benefit of harm $\beta = 0.5$; female dispersal rate $d_f=1$; number of females $n_f=3$; number of males $n_m=3$; male dispersal rate $d_m=0.5$; and relatedness between females and males $r_{fm}=0$.

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Supplementary information

1. Open model

We consider a population divided into social groups. In each social group, there are females and males that interact with each other. During those interactions, males can 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 relative reproductive success is directly proportional to his fecundity and inversely proportional to the average fecundity of the males in the social group. A female's fecundity is $f_f(Y)$, and a male's competitiveness for mating success is $f_m(y)$. The former is a function of the average level of male harm present in the social group (Y) and the latter is a function of the level of harm expressed by the focal male (y). Mating follows, with each female producing a large number of offspring, with an even sex ratio and in direct proportion to her fecundity. Adults then die, and juveniles of both sexes compete with local juveniles in proportion to a, with a defining the scale of competition (Frank 1998; Gardner 2010). Females and males then survive at random within each social group to adulthood, returning the population to the beginning of the lifecycle.

Natural selection – Natural selection favours any gene associated with greater individual relative fitness (Fisher 1930; Price 1970). Assuming vanishingly little genetic variation, this condition may be expressed using the mathematics of differential calculus: $dW/dg > 0$, 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 population is divided into females and males, meaning that the appropriate measure of relative fitness is a class-reproductive-value-weighted average taken across females and males, i.e. $W = ½W_f + ½W_m$, where W_f is the relative fitness of the female carrying the gene and W_m is the relative fitness of the male carrying the gene (Taylor 1996; Taylor & Frank 1996). Female's relative fitness in the context of the present model is given by:

$$W_f = \frac{f_f(Y)}{af_f(Y)+(1-a)f_f(\bar{Y})}, \quad \text{(A1)}$$

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

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

where $f_m(Y)$ is the average competitiveness for mating success of local males which is a function of the average level of male harm in the social group (Y). Following the approach of Taylor & Frank (1996) for a class-structured population, we may write $dW/dg = ½ (dW_f/dg_f) + ½ (dW_m/dg_m) = ½ ((\partial W_f/\partial y)(dy/dG_f)(dG_f/dg_f) + (\partial W_f/\partial Y)(dY/dG_f')(dG_f'/dg_f) + (\partial W_f/\partial Y)(dY/dG_m')(dG_m'/dg_f)) + ½ ((\partial W_m/\partial y)(dy/dG_m)(dG_m/dg_m) + (\partial W_m/\partial Y)(dY/dG_m')(dG_m'/dg_m) + (\partial W_m/\partial Y)(dY/dG_f')(dG_f'/dg_m))$, where: g_f is the genic value of a gene picked at random from a female in the population; g_m is the genic value of a gene picked at random from a male in the population; G_f is the focal female's breeding value; G_f' is the average breeding value of local females; G_m is the focal male's breeding value; G_m' is the average breeding value of local males; y is the level of harm of the focal male; Y is the average level of harm of local males; $dy/dG_f = dY/dG_f' = \gamma_f$ is the mapping between genotype

and phenotype in the females; $dG_f/dg_f = p_f$ is the consanguinity of the gene in the focal female to the female herself; $dG_f'/dg_f = p_{ff}$ is the consanguinity of the gene in the focal female with a randomly-chosen local female (including the focal female herself); $dG_m'/dg_f = p_{fm}$ is the consanguinity of the gene in the focal female with a randomly-chosen local male; $dy/dG_m = dY/dG_m' = \gamma_m$ is the mapping between genotype and phenotype in the males; $dG_m/dg_m = p_m$ is the consanguinity of the gene in the focal male to the male himself; $dG_m'/dg_m = p_{mm}$ is the consanguinity of the gene in the focal male with a randomly-chosen local male (including the focal male himself); and $dG_f'/dg_m = p_{mf}$ is the consanguinity of the gene in the focal male with a randomly-chosen local female. The consanguinity between a gene to its carrier is the same no matter the sex that we are considering and, therefore, $p_f = p_m = p$. We divide all terms of the right side of the equation by p to get the kin-selection coefficient of relatedness (see below; Bulmer 1994).

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

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

where: r_{fm} is the relatedness between a focal female with a randomly-chosen local male; and r_{mm} is the relatedness between a focal male with a randomly-chosen local male. Evaluating the derivatives at $y = Y = \bar{Y}$, we obtain:

$$\frac{(1-r_{mm}) f_m(\bar{Y})'}{f_m(\bar{Y})} + (1-a)(r_{fm} + r_{mm}) \frac{f_f(\bar{Y})'}{f_f(\bar{Y})} > 0, \quad \text{(A4)}$$

where: $f_m(\bar{Y})$ is the average competitiveness for mating success of males in the population which is a function of the average level of male harm in the population ($\bar{Y}$); $f_m(\bar{Y})'$ is the derivative of the average competitiveness for mating success of males in the population; $f_f(\bar{Y})'$ is the derivative of the average fecundity of females in the population. $f_m(\bar{Y})'/f_m(\bar{Y}) := B$ is the overall benefit for males of expressing harm and $f_f(\bar{Y})'/f_f(\bar{Y}) := C$ is the overall cost for females of receiving harm and, therefore, we can change (A4) to:

$$B(1 - r_{mm}) - C(1-a)(r_{fm} + r_{mm}) > 0. \quad \text{(A5)}$$

Expression (A5) is readily interpreted in inclusive fitness terms: a male who invests into harming seizes a greater share of the overall mating success of the males that he competes with, yielding a direct fitness benefit B; however, the corresponding loss of mating success by local males yields an inclusive fitness loss Br_{mm}; harming reduces the overall number of offspring produced by local females by Cr_{fm} and by local males by Cr_{mm}; owing to local competition, this translates into a loss of only $C(1 - a)$ surviving offspring for both local females and local males; and this gives a total of $C(1 - a)(r_{fm} + r_{mm})$ of inclusive fitness loss. Rearranging (A5), we obtain:

$$\frac{C}{B} < \frac{(1-r_{mm})}{(1-a)(r_{fm}+r_{mm})}. \quad \text{(A6)}$$

Expression (A6) represents the potential for harm to evolve. For any given benefit B and cost C of harm – and with the scale of competition a remaining constant –, increased relatedness between social partners always decrease the potential for harm to evolve (Figure 1A&B).

Consider that both the scale of competition (a) and relatedness between social partners (r_{fm} and r_{mm}) depend on female dispersal and male dispersal rates (see below). In that case, relatedness and the scale of competition cannot vary independently unless other considerations are made. One possible consideration is kin discrimination. That is, if individuals can discriminate the context that they are in, then males can adjust the level of harm accordingly (Figure 1A&B) without necessarily changing the scale of competition.

It is also possible to maintain the relatedness coefficients constant while varying the scale of competition (Figure 1C&D), with harm being reduced as the scale of competition becomes more global (a closer to 0). An example of such scenario is when competition occurs between groups of related individuals, with local groups competing with each other in proportion to a. Mathematically, such approach is equivalent to dispersal of groups of related individuals, known in the literature as budding dispersal (Goodnight 1992; Gardner & West 2006; Lehmann et al. 2006; Gardner et al. 2009).

2.1 Closed model – absence of kin discrimination and absence of budding dispersal

We consider an infinite diploid population divided into patches containing n_f females and n_m males. As in section 1, males invest in a harming trait that increases their personal reproductive success relative to other males but reduces the overall fecundity of the females in the patch. Each male's relative reproductive success is directly proportional to his competitiveness for mating success and inversely proportional to the average competitiveness for mating success of the males in his patch. A female's fecundity is f_f, and a male's competitiveness for mating success is f_m. Following 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 females disperse with probability d_f and males disperse with probability d_m. Following dispersal, n_f females and n_m males survive at random within each patch to adulthood, returning the population to the beginning of the lifecycle.

Fecundity and competitiveness for mating success – We now use particular functions to define how harm affects both females' and males' competitiveness for mating success. Accordingly, a focal female's fecundity is:

$$f_f(Y) = 1 - Y; \tag{A7}$$

and a focal male's competitiveness for mating success is:

$$f_m(y) = 1 + y^{\beta}, \tag{A8}$$

with β being the marginal benefit of harm, that is, it defines how the benefits of harm increase with the increase of the level of harm itself ($0 < \beta < 1$, close to 0 the benefits grow exponentially, close to 1 the benefits grow linearly). Accordingly, the average female fecundity in the focal patch is $f_f(Y)$,the average female fecundity in the population is $f_f(\bar{Y})$, the average male competitiveness for mating success in the focal patch is $f_m(Y)$, and the average male fecundity in the population is $f_m(\bar{Y})$.

Fitness – From Faria et al. (2015, 2017), female fitness in the context of the present model is given by:

$$W_{\mathrm{f}} = f_{\mathrm{f}}(Y)\left(\frac{1}{2}\left(\frac{1-d_{\mathrm{f}}}{(1-d_{\mathrm{f}})f_{\mathrm{f}}(Y)+d_{\mathrm{f}}f_{\mathrm{f}}(\bar{Y})}+\frac{d_{\mathrm{f}}}{f_{\mathrm{f}}(\bar{Y})}\right)+\frac{1}{2}\left(\frac{1-d_{\mathrm{m}}}{(1-d_{\mathrm{m}})f_{\mathrm{f}}(Y)+d_{\mathrm{m}}f_{\mathrm{f}}(\bar{Y})}+\frac{d_{\mathrm{m}}}{f_{\mathrm{f}}(\bar{Y})}\right)\right), \quad \text{(A9)}$$

and male fitness in the context of the present model is given by:

$$W_{\mathrm{m}} = \frac{f_m(y)}{f_m(Y)}f_{\mathrm{f}}(Y)\left(\frac{1}{2}\left(\frac{1-d_{\mathrm{f}}}{(1-d_{\mathrm{f}})f_{\mathrm{f}}(Y)+d_{\mathrm{f}}f_{\mathrm{f}}(\bar{Y})}+\frac{d_{\mathrm{f}}}{f_{\mathrm{f}}(\bar{Y})}\right)+\frac{1}{2}\left(\frac{1-d_{\mathrm{m}}}{(1-d_{\mathrm{m}})f_{\mathrm{f}}(Y)+d_{\mathrm{m}}f_{\mathrm{f}}(\bar{Y})}+\frac{d_{\mathrm{m}}}{f_{\mathrm{f}}(\bar{Y})}\right)\right). \quad \text{(A10)}$$

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

$$r_{\mathrm{fm}} = (1-d_{\mathrm{f}})(1-d_{\mathrm{m}})r, \quad \text{(A11)}$$

and they are only related if they are both local $(1 - d_{\mathrm{f}})(1 - d_{\mathrm{m}})$ and, if so, their relatedness is defined by the relatedness through the genic actor in the focal female (r). For the relatedness between a genic actor in the focal male and a randomly-chosen male in his patch (including the focal male himself):

$$r_{\mathrm{mm}} = \frac{1}{n_{\mathrm{m}}}+\frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}}(1-d_{\mathrm{m}})^2 r, \quad \text{(A12)}$$

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

Relatedness through the genic actor between two different juveniles born in the same patch is then given by $r = p'/p$, where p' is the consanguinity through the genic actor between two individuals born in the same patch and is defined by picking the genic actor from the focal individual and a random gene from the other individual and calculating the probability that the two are identical by descent (Bulmer 1994). Assuming that consanguinities are at their neutral-equilibrium values, appropriate if selection is weak (Gardner et al. 2011), we write:

$$p' = \frac{1}{4}\left(\frac{1}{n_{\mathrm{f}}}p+\frac{n_{\mathrm{f}}-1}{n_{\mathrm{f}}}(1-d_{\mathrm{f}})^2 p'\right)+\frac{1}{4}\left(\frac{1}{n_{\mathrm{m}}}p+\frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}}(1-d_{\mathrm{m}})^2 p'\right)+\frac{1}{2}(1-d_{\mathrm{f}})(1-d_{\mathrm{m}})p', \quad \text{(A13)}$$

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

$$p' = \frac{n_{\mathrm{f}}+n_{\mathrm{m}}}{(1-d_{\mathrm{m}})^2 n_{\mathrm{f}}+(1-d_{\mathrm{f}})^2 n_{\mathrm{m}}+(4-d_{\mathrm{f}}-d_{\mathrm{m}})(d_{\mathrm{f}}+d_{\mathrm{m}})n_{\mathrm{f}}n_{\mathrm{m}}}p, \quad \text{(A14)}$$

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

$$r = \frac{n_{\mathrm{f}}+n_{\mathrm{m}}}{(1-d_{\mathrm{m}})^2 n_{\mathrm{f}}+(1-d_{\mathrm{f}})^2 n_{\mathrm{m}}+(4-d_{\mathrm{f}}-d_{\mathrm{m}})(d_{\mathrm{f}}+d_{\mathrm{m}})n_{\mathrm{f}}n_{\mathrm{m}}}. \quad \text{(A15)}$$

We can replace equation (A15) into the equations (A11–A12) to obtain the different coefficients of relatedness.

Marginal fitness equations and optimal levels of male harm – We can now use the same strategy used in section 1 (see above) with the fecundity and fitness equations defined above (A7–A10) and with the coefficients of relatedness explicitly represented by individuals' dispersal rates (A11–A12). Following the same strategy of section 1, we now obtain the derivatives of the left side of the inequality (A3) and the following marginal fitness equation for this model:

$$\frac{(1-r_{\mathrm{mm}})(\bar{Y}^{*\beta-1}\beta)}{1+\bar{Y}^{*\beta}} - (1-a)(r_{\mathrm{fm}}+r_{\mathrm{mm}})\frac{1}{1-\bar{Y}^{*}} = 0, \quad \text{(A16)}$$

where $\bar{Y}^*$ is the optimal level of male harm and $a = ((1 - d_f)_2 + (1 - d_m)_2)/2$. Now we can solve equation (A16) for $\bar{Y}^*$ to get the optimal level of harm. This solution can then be used to plot the dashed black line of Figure 3A-B, Figure 3D, and Extended Data Figure 1A-B. Note that two solutions are found, but only one makes sense given the assumptions of the model.

2.2 Closed model – presence of kin discrimination and absence of budding dispersal

For illustrative purposes and due to the inherent complexity of incorporating kin discrimination based on familiarity into a mathematical model, here we make additional assumptions regarding the population demography, with every other detail of the lifecycle of the population remaining the same as above. Specifically, we now assume that each patch of the population contains $n_f = 3$ females and $n_m = 3$ males. Moreover, we assume full female dispersal $d_f = 1$. Nonetheless, our results and conclusions should hold for any demographic conditions being considered.

Fecundity and competitiveness for mating success – Males have now the capacity of discriminating between familiar and unfamiliar males, changing their behaviour accordingly. By familiar, we mean that males recognise individuals that they grew up with throughout their lifecycle as familiar. Under the demographic conditions that we assume, three types of patch exist: patch type-2, where the focal male recognises the two other males as being familiar; patch type-1, where the focal male recognises one male as being familiar and one male as being unfamiliar; and patch type-0, where the focal male recognises the two other males as being unfamiliar. Because males change their behaviour depending on the type of patch that they are in, females' fecundity is also affected. According, a focal female in a patch type-2 has fecundity of:

$$f_{\mathrm{f2}}(Y_2, Y_1, Y_0) = 1 - Y_2, \quad \text{(A17)}$$

where Y_2 is the average level of harm of males that interact with two familiar males in the focal patch, Y_1 is the average level of harm of males that interact with one familiar male and one unfamiliar male in the focal patch, and Y_0 is the average level of harm of males that

interact with two unfamiliar males in the focal patch. A focal female in a patch type-1 has fecundity of:

$$f_{\mathrm{f1}}(Y_2, Y_1, Y_0) = 1 - \frac{2Y_1 + Y_0}{3}. \quad \text{(A18)}$$

Finally, a female in a patch type-0 has fecundity of:

$$f_{\mathrm{f0}}(Y_2, Y_1, Y_0) = 1 - Y_0. \quad \text{(A19)}$$

Similarly, a focal male interacting with two familiar males has competitiveness for mating success of:

$$f_{\mathrm{m2}}(y_2) = 1 + y_2^{\beta}, \quad \text{(A20)}$$

where y_2 is the level of harm of a focal male interacting with two familiar males. A focal male interacting with one familiar male and one unfamiliar male has competitiveness for mating success of:

$$f_{\mathrm{m1}}(y_1) = 1 + y_1^{\beta}, \quad \text{(A21)}$$

where y_1 is the level of harm of a focal male interacting with one familiar male and one unfamiliar male. Finally, a focal male interacting with two unfamiliar males has competitiveness for mating success of:

$$f_{\mathrm{m0}}(y_0) = 1 + y_0^{\beta}, \quad \text{(A22)}$$

where y_0 is the level of harm of a focal male interacting with two unfamiliar males. Accordingly, a focal male interacting with two familiar males has a relative competitiveness for mating success of:

$$\phi_{m22} = \frac{f_{\mathrm{m2}}(y_2)}{f_{\mathrm{m2}}(Y_2)} f_{\mathrm{f2}}(Y_2, Y_1, Y_0). \quad \text{(A23)}$$

A focal male interacting with one familiar male and with one unfamiliar male has a relative competitiveness for mating success of:

$$\phi_{m11} = \frac{f_{\mathrm{m1}}(y_1)}{2f_{\mathrm{m1}}(Y_1) + f_{\mathrm{m0}}(y_0)} f_{\mathrm{f1}}(Y_2, Y_1, Y_0). \quad \text{(A24)}$$

A focal male interacting with two unfamiliar males that are familiar to each other has a relative competitiveness for mating success of:

$$\phi_{m01} = \frac{f_{\mathrm{m0}}(y_0)}{2f_{\mathrm{m1}}(Y_1) + f_{\mathrm{m0}}(y_0)} f_{\mathrm{f1}}(Y_2, Y_1, Y_0). \quad \text{(A25)}$$

Finally, a focal male interacting with two unfamiliar males that are also unfamiliar to each other has a relative competitiveness for mating success of:

$$\phi_{m00} = \frac{f_{\mathrm{m0}}(y_0)}{f_{\mathrm{m0}}(Y_0)} f_{\mathrm{f0}}(Y_2, Y_1, Y_0). \quad \text{(A26)}$$

Fitness – Due to the presence of different types of patches, equations (A9–A10) can no longer be used. That is, females in different patches will necessarily have different fitness, meaning that we cannot treat females as a single uniform class. The same is true for males. Specifically, we need to take into consideration the frequency of the different patches in the population. A female's relative fitness is now represented by:

$$W_{\mathrm{f}} = \sum_k q_{\mathrm{f}k} f_{\mathrm{f}k}(Y_2, Y_1, Y_0) \left(\frac{1}{2} \left(\frac{1-d_{\mathrm{f}}}{(1-d_{\mathrm{f}}) f_{\mathrm{f}k}(Y_2,Y_1,Y_0) + d_{\mathrm{f}} f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} + \frac{d_{\mathrm{f}}}{f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} \right) + \right.$$
$$\left. \frac{1}{2} \left(\frac{1-d_{\mathrm{m}}}{(1-d_{\mathrm{m}}) f_{\mathrm{f}k}(Y_2,Y_1,Y_0) + d_{\mathrm{m}} f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} + \frac{d_{\mathrm{m}}}{f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} \right) \right), \quad \text{(A27)}$$

where $d_{\mathrm{f}} = 1$, $k \in \{2, 1, 0\}$ is the type of patch that the females are in, $q_{\mathrm{f}k}$ is the frequency of those type of patches in the population, $\bar{Y}_2$ is the average level of harm of males that interact with two familiar males in the population, $\bar{Y}_1$ is the average level of harm of males that interact with one familiar male and one unfamiliar male in the population, $\bar{Y}_0$ is the average level of harm of males that interact with two unfamiliar males in the population, and $f_{\mathrm{f}}(\bar{Y}_2, \bar{Y}_1, \bar{Y}_0) = \sum_k q_{\mathrm{f}k} f_{\mathrm{f}k}(\bar{Y}_2, \bar{Y}_1, \bar{Y}_0)$ is the average female fecundity in the population taken across the different patch types. For $k = 2$, $q_{\mathrm{f}2} = (1 - d_{\mathrm{m}})^3$, for $k = 1$, $q_{\mathrm{f}1} = 3(1 - d_{\mathrm{m}})^2(d_{\mathrm{m}})$, and for $k = 0$, $q_{\mathrm{f}0} = 3(1 - d_{\mathrm{m}})(d_{\mathrm{m}})^2 + (d_{\mathrm{m}})^3$. Similarly, a male's relative fitness is now represented by:

$$W_{\mathrm{m}} = \sum_j \sum_k q_{\mathrm{m}jk} \phi_{\mathrm{m}jk} \left(\frac{1}{2} \left(\frac{1-d_{\mathrm{f}}}{(1-d_{\mathrm{f}}) f_{\mathrm{f}k}(Y_2,Y_1,Y_0) + d_{\mathrm{f}} f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} + \frac{d_{\mathrm{f}}}{f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} \right) + \right.$$
$$\left. \frac{1}{2} \left(\frac{1-d_{\mathrm{m}}}{(1-d_{\mathrm{m}}) f_{\mathrm{f}k}(Y_2,Y_1,Y_0) + d_{\mathrm{m}} f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} + \frac{d_{\mathrm{m}}}{f_{\mathrm{f}}(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)} \right) \right), \quad \text{(A28)}$$

where $d_{\mathrm{f}} = 1$, $j \in \{2, 1, 0\}$ represents how many males the focal male recognizes as being familiar and $k \in \{2, 1, 0\}$ the type of patch that they are in. Therefore, the possible combinations are $jk \in \{\{2,2\},\{1,1\},\{0,1\},\{0,0\}\}$ and $q_{\mathrm{m}jk}$ is the frequency of those different scenarios that males can find themselves in. Accordingly, for $j = 2$ and $k = 2$, $q_{\mathrm{m}22} = (1 - d_{\mathrm{m}})^3$, for $j = 1$ and $k = 1$, $q_{\mathrm{m}11} = 2(1 - d_{\mathrm{m}})^2(d_{\mathrm{m}})$, for $j = 0$ and $k = 1$, $q_{\mathrm{m}01} = (d_{\mathrm{m}})(1 - d_{\mathrm{m}})^2$, and for $j = 0$ and $k = 0$, $q_{\mathrm{m}00} = 3(d_{\mathrm{m}})^2(1 - d_{\mathrm{m}}) + (d_{\mathrm{m}})^3$.

Relatedness – We now do not need to consider that females may be local given that we are assuming full female dispersal $d_{\mathrm{f}} = 1$. Accordingly, we only need to focus on how related the males are to each other. For the relatedness between a genic actor in a focal male that recognises two other males as being familiar and a randomly-chosen male (including the focal male himself):

$$r_{2\mathrm{mm}} = \frac{1}{n_{\mathrm{m}}} + \frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}} r, \quad \text{(A29)}$$

where $n_{\mathrm{m}} = 3$. Therefore, with probability 1/3 the randomly-chosen male is the focal male himself, in which case relatedness is 1; and with probability 2/3 it is a different male, in which case their relatedness is defined by the relatedness through the genic actor in the focal male (r). For the relatedness between a genic actor in a focal male that recognises one male as being familiar and another one as being unfamiliar with a randomly-chosen male (including the focal male himself):

$$r_{1\mathrm{mm}} = \frac{1}{n_\mathrm{m}} + \frac{n_\mathrm{m}-2}{n_\mathrm{m}} r, \tag{A30}$$

where $n_\mathrm{m} = 3$. Therefore, with probability 1/3 the randomly-chosen male is the focal male himself, in which case relatedness is 1; and with probability 1/3 is the male that he recognises as being a familiar male, in which case their relatedness is defined by the relatedness through the genic actor in the focal male (r). For the relatedness between a genic actor in the focal male that recognises two males as being unfamiliar with a randomly-chosen male (including the focal male himself):

$$r_{0\mathrm{mm}} = \frac{1}{n_\mathrm{m}} + \frac{n_\mathrm{m}-3}{n_\mathrm{m}} r, \tag{A31}$$

where $n_\mathrm{m} = 3$. Therefore, with probability 1/3 the randomly-chosen male is the focal male himself, in which case relatedness is 1; and he is unrelated to the other two males.

Relatedness through the genic actor between two different juveniles born in the same patch is the same as in section 2.1, that is, $r = p'/p$, where p' is the consanguinity through the genic actor between two individuals born in the same patch and is defined by picking the genic actor from the focal individual and a random gene from the other individual and calculating the probability that the two are identical by descent (Bulmer 1994). Assuming that consanguinities are at their neutral-equilibrium values, appropriate if selection is weak (Gardner et al. 2011), we can still use equation (A15) with the relatedness coefficients (A29–A31), provided that we use the demographic conditions that we are assuming here ($n_\mathrm{f} = 3$, $n_\mathrm{m} = 3$, and $d_\mathrm{f} = 1$).

Marginal fitness equations – We now have three marginal fitness equations: one for males that interact with two familiar males; one for males that interact with one familiar male and one unfamiliar male; and one for males that interact with two unfamiliar males. Solving this system of equations gives us the optimal levels of harm that males should express in each situation that they happen to be in:

$$\frac{1}{2}\left(\frac{\partial W_\mathrm{m}}{\partial y_2} + \frac{\partial W_\mathrm{m}}{\partial Y_2} r_{2\mathrm{mm}}\right) = 0; \tag{A32}$$

$$\frac{1}{2}\left(\frac{\partial W_\mathrm{m}}{\partial y_1} + \frac{\partial W_\mathrm{m}}{\partial Y_1} r_{1\mathrm{mm}}\right) = 0; \tag{A33}$$

$$\frac{1}{2}\left(\frac{\partial W_\mathrm{m}}{\partial y_0} + \frac{\partial W_\mathrm{m}}{\partial Y_0} r_{0\mathrm{mm}}\right) = 0. \tag{A34}$$

Evaluating the derivatives using equation (A28) at $y_2 = Y_2 = \bar{Y}_2^*$, $y_1 = Y_1 = \bar{Y}_1^*$, and $y_0 = Y_0 = \bar{Y}_0^*$ and solving the resultant system of equations for $\{\bar{Y}_2^*, \bar{Y}_1^*, \bar{Y}_0^*\}$ yields the optimal level of harm for the different types of males. These solutions can then be used to plot the dashed coloured lines represented in Figure 3A, Extended Data Figure 1A, and Extended Data Figure 2A with the values there described.

2.3 Closed model – Presence of budding dispersal

We now consider the possibility of budding dispersal (Goodnight 1992; Gardner & West 2006; Lehmann et al. 2006; Gardner et al. 2009). Specifically, juveniles form groups and

each group disperse with probability d_B. Density-dependent regulation of the population occurs at this stage and between groups, with a single group surviving in each patch. This is followed by individual dispersal, with females dispersing with probability d_f and males dispersing with probability d_m. Every other detail of the lifecycle of the population remains the same as in the absence/presence of kin discrimination (see above), depending on the model being considered.

Accordingly, in the absence of kin discrimination, female fitness is now given by:

$$W_f = f_f(Y)\left(\frac{1-d_B}{(1-d_B)f_f(Y)+d_B f_f(\bar{Y})}+\frac{d_B}{f_f(\bar{Y})}\right), \quad \text{(A35)}$$

and male fitness is now given by:

$$W_m = \frac{f_m(y)}{F_m(Y)} f_f(Y)\left(\frac{1-d_B}{(1-d_B)f_f(Y)+d_B f_f(\bar{Y})}+\frac{d_B}{f_f(\bar{Y})}\right). \quad \text{(A36)}$$

Following the same strategy as in section 2.1 (see above), we obtain the same equation (A16), except that now $a = (1 - d_B)^2$. Solving the equation for $\bar{Y}^*$ obtains the optimal level of harm in the absence of kin discrimination and in the presence of budding dispersal. This solution can then be used to plot the solid black line of Figure 3B, Figure 3D, and Extended Data Figure 1B and the dashed black line of Figure 3C, and Extended Data Figure 1C. Two solutions are found, but only one makes sense given the assumptions of the model.

Similarly, in the presence of kin discrimination, female fitness is now given by:

$$W_f = \sum_k q_{fk} f_{fk}(Y_2, Y_1, Y_0)\left(\frac{1-d_B}{(1-d_B)f_{fk}(Y_2,Y_1,Y_0)+d_B f_f(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)}+\frac{d_B}{f_f(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)}\right), \quad \text{(A37)}$$

and male fitness is now given by:

$$W_m = \sum_j \sum_k q_{mjk}\phi_{mjk}\left(\frac{1-d_B}{(1-d_B)f_{fk}(Y_2,Y_1,Y_0)+d_B f_f(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)}+\frac{d_B}{f_f(\bar{Y}_2,\bar{Y}_1,\bar{Y}_0)}\right). \quad \text{(A38)}$$

We can now use these equations (A37–A38) with the marginal fitness equations (A32–A34) of section 2.2 and, following the same strategy, this yields the optimal level of harm for the different types of males in the presence of budding dispersal. These solutions can then be used to plot the solid coloured lines represented in Figure 3C, Extended Data Figure 1C, and Extended Data Figure 2C with the values there described.

3. Within-group variation in harm experienced by females

So far we have assumed that females receive harm equally from all the males in their social group. If harm is inflicted during mating *per se*, then this is consistent with the assumption – ultimately deriving from the model of Rankin (2011) – that each female mates a large number of times, with her mate being chosen at random from her social group and independently for each mating. Alternatively, harm may be conceptualised as occurring during a mating attempt, such that every time the female is mated she is not only being harmed by her successful mate but also by the other males who are unsuccessfully trying to mate with her as well (e.g. via male harassment). More generally, it may be that a female mates a smaller number of times or with a smaller number of males, and that she experiences a level of harm

that is relatively more dependent on the harming phenotypes of those males with whom she actually mates (e.g. toxic ejaculates) than on the harming phenotypes of other males in her social group. Here, we explore a model in which females mate monogamously and experience a level of harm that is more heavily dependent upon the harm of their mate than by the other males in her social group. Specifically, the harm that a female experiences is $ky' + (1 - k)Y$, where y' is the level of harming employed by her mate, Y is the average level of harming employed by all the males in her social group, and $0 \leq k \leq 1$. Aside from the assumption that females mate monogamously, all other assumptions match the closed model of indiscriminate harming investigated in the main text.

Fecundity and competitiveness for mating success – The fecundity of a focal female is:

$$f_{\mathrm{f}}(y', Y) = 1 - k\, y' - (1 - k)Y. \tag{A39}$$

As before, the competitiveness for mating success of a focal male is:

$$f_{\mathrm{m}}(y) = 1 + y^{\beta}. \tag{A40}$$

Accordingly, the average female fecundity in the focal patch is $f_{\mathrm{f}}(Y', Y)$, and the average female fecundity in the population is $f_{\mathrm{f}}(\bar{Y})$, where Y' is the average level of harming employed by the mate of the average female in the focal patch. The average competitiveness for mating success of a male in the focal patch is $f_{\mathrm{m}}(Y)$, and the average competitiveness for mating success of a male in the population is $f_{\mathrm{m}}(\bar{Y})$.

Fitness – Female fitness in the context of the present model is given by:

$$W_{\mathrm{f}} = f_{\mathrm{f}}(y', Y)\left(\frac{1}{2}\left(\frac{1-d_{\mathrm{f}}}{(1-d_{\mathrm{f}})f_{\mathrm{f}}(Y',Y)+d_{\mathrm{f}}f_{\mathrm{f}}(\bar{Y})} + \frac{d_{\mathrm{f}}}{f_{\mathrm{f}}(\bar{Y})}\right) + \frac{1}{2}\left(\frac{1-d_{\mathrm{m}}}{(1-d_{\mathrm{m}})f_{\mathrm{f}}(Y',Y)+d_{\mathrm{m}}f_{\mathrm{f}}(\bar{Y})} + \frac{d_{\mathrm{m}}}{f_{\mathrm{f}}(\bar{Y})}\right)\right), \tag{A41}$$

and male fitness in the context of the present model is given by:

$$W_{\mathrm{m}} = \sum_{\mu=1}^{n_{\mathrm{f}}}\left(\frac{n_{\mathrm{f}}!}{\mu!(n_{\mathrm{f}}-\mu)}\left(\frac{1+y^{\beta}}{n_{\mathrm{m}}(1+Y^{\beta})}\right)^{\mu}\left(1 - \frac{1+y^{\beta}}{n_{\mathrm{m}}(1+Y^{\beta})}\right)^{n_{\mathrm{f}}-\mu} \mu_{\mathrm{f}} f_{\mathrm{f}}(y', Y)\left(\frac{1}{2}\left(\frac{1-d_{\mathrm{f}}}{(1-d_{\mathrm{f}})f_{\mathrm{f}}(Y_{m,\mu},Y)+d_{\mathrm{f}}f_{\mathrm{f}}(\bar{Y})} + \frac{d_{\mathrm{f}}}{f_{\mathrm{f}}(\bar{Y})}\right) + \frac{1}{2}\left(\frac{1-d_{\mathrm{m}}}{(1-d_{\mathrm{m}})f_{\mathrm{f}}(Y_{m,\mu},Y)+d_{\mathrm{m}}f_{\mathrm{f}}(\bar{Y})} + \frac{d_{\mathrm{m}}}{f_{\mathrm{f}}(\bar{Y})}\right)\right)\right), \tag{A42}$$

where:

and

$$Y_{m,\mu} = \frac{\mu}{n_{\mathrm{f}}} y + \frac{n_{\mathrm{f}}-\mu}{n_{\mathrm{f}}} Y''. \tag{A43}$$

Specifically, μ is the number of matings that the focal male achieves, which is a binomially distributed random number. Accordingly, the first three terms of equation (A42) correspond to the binomial probability of achieving μ matings. The fitness that the focal male achieves depends on $f_{\mathrm{f}}(Y_{m,\mu}, Y)$, which defines how male harm is affecting the average female fecundity in the focal patch. Correspondently, it ranges from $1 - k\, y - (1 - k)Y$, 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, with *Y''* being the harm of the average male in the focal patch excluding the focal male.

Marginal fitness equations – Following the approach of Taylor & Frank (1996) for a class-structured population (see section 1), we have:

$$\frac{1}{2}\left(\left(\frac{\partial W_{\mathrm{f}}}{\partial Y}+\frac{\partial W_{\mathrm{f}}}{\partial Y_{\mathrm{m},\mu}}\right)r_{\mathrm{fm}}\right)+\frac{1}{2}\left(\frac{\partial W_{\mathrm{m}}}{\partial y}+\frac{\partial W_{\mathrm{m}}}{\partial y'}+\left(\frac{\partial W_{\mathrm{m}}}{\partial Y}+\frac{\partial W_{\mathrm{m}}}{\partial Y_{\mathrm{m},\mu}}\right)r_{\mathrm{mm}}\right)>0, \tag{A44}$$

where the derivatives are evaluated at $y = y' = Y = Y_{\mathrm{m},\mu} = \bar{Y} = \bar{Y}^*$. The only difference from section 1 is that now we need to consider both the harm imposed onto the females by males in the patch regardless if they mate with the females or not (Y) and by males that do mate with them (y' and $Y_{\mathrm{m},\mu}$). Evaluating the derivatives and setting the left-side of the equation equal to zero yields:

$$\frac{n_{\mathrm{f}}}{n_{\mathrm{m}}}\left(\frac{(1-r_{\mathrm{mm}})\bar{Y}^{*\beta-1}\beta}{1+\bar{Y}^{*\beta}}-k\frac{1-r_{\mathrm{mm}}}{1-\bar{Y}^{*}}\right)+(1-a)\left(\frac{n_{\mathrm{m}}r_{\mathrm{fm}}+n_{\mathrm{f}}r_{\mathrm{mm}}}{n_{\mathrm{m}}(1-\bar{Y}^{*})}-k\frac{r_{\mathrm{fm}}}{1-\bar{Y}^{*}}\right)=0, \tag{A45}$$

where $a = ((1 - d_{\mathrm{f}})_2 + (1 - d_{\mathrm{m}})_2)/2$. If $n_{\mathrm{f}} = n_{\mathrm{m}} = 3$ and $k = 0$, that is, similar assumptions to the ones used in the previous models, then equation (A45) reduces to equation (A16). That is, if we express the optimal level of male harm as a function of relatedness (r_{mm} and r_{fm}) and scale of competition (a) *per se*, then the $k = 0$ case exactly recovers the scenario described in the main text in which females are harmed equally by all males in their social group. However, note that the present model assumes monogamous females whereas the models of the main text assume promiscuous females, and this leads to differences in relatedness and hence a divergence in model results when expressed at a more proximate level in terms of explicit demographic parameters such as dispersal rate. Increasing the value of k above zero, such that a female's fecundity is more heavily dependent upon the male with whom she mates, and hence a male's reproductive success is more negatively impacted by the harm he inflicts upon his mates, we find that natural selection favours a reduced level of male harm, but that the main qualitative result – that higher relatedness tends to inhibit the evolution of male harm – remains unchanged (Extended Data Figure 4).

4. Individual-based simulations

We run individual-based simulations where we consider an initial population of 4000 patches in which each patch contains three males and three females ($n_{\mathrm{m}} = n_{\mathrm{f}} = 3$). Each individual has a probability of being a parent to the individuals of the next generation and that probability is given by their fecundity. However, rather than giving rise to offspring, we jump straight to new adults. Each of the new adults has a probability of being from a specific patch, being this dependent on the dispersal rates of each sex as well on the fecundity present in each patch. Males' fecundity is dependent on the harm that they express, being the level of harm dependent on males interacting with two familiar males, one familiar and one unfamiliar male, and two unfamiliar males. Regardless, the level of harm expressed is controlled by the sum of the two genes. Females' fecundity decreases as the average of harm present in the patch increases. Then, we assign to each one of the adults a mother and a father from the patch where they came from (which may be different from the patch where the individual is now, if she has dispersed). Specifically, the higher the fecundity of a female in the previous generation, the higher her probability of being a mother of an adult in the current generation. Similarly, the higher the competitiveness for mating success of a male in the previous

generation, the higher his probability of being a father of an adult in the current generation. The gene value, transmitted from the parents to the adults of the next generation, may change due to mutations (either increasing or decreasing) which add up to the original value of the gene. The range of that change varies between –0.01 and 0.01. In both cases, we are using a uniform distribution to modulate the mutational changes that occur in the traits considered. The only constraint to the values of the harm expressed is that they cannot decrease below 0. For all the simulations, this happens with a probability of 10^{-2}. The simulations were run for $5x10^4$ generations.

References

Bulmer, M. G. Theoretical evolutionary ecology. MA: Sinauer Associates, Sunderland (1994).

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

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

Gardner, A., West, S. A. & Wild, G. The genetical theory of kin selection. *J. Evol. Biol.* **24**, 1020-1043 (2011).

Taylor, P. D. Inclusive fitness arguments in genetic models of behaviour. *J. Math. Biol.* **34**, 654-674 (1996)