SHORT COMMUNICATION

Sex-biased dispersal, kin selection and the evolution of sexual conflict

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Abstract

There is growing interest in resolving the curious disconnect between the fields of kin selection and sexual selection. Rankin's (2011, *J. Evol. Biol.* **24**, 71–81) theoretical study of the impact of kin selection on the evolution of sexual conflict in viscous populations has been particularly valuable in stimulating empirical research in this area. An important goal of that study was to understand the impact of sex-specific rates of dispersal upon the coevolution of male-harm and female-resistance behaviours. But the fitness functions derived in Rankin's study do not flow from his model's assumptions and, in particular, are not consistent with sex-biased dispersal. Here, we develop new fitness functions that do logically flow from the model's assumptions, to determine the impact of sex-specific patterns of dispersal on the evolution of sexual conflict. Although Rankin's study suggested that increasing male dispersal always promotes the evolution of male harm and that increasing female dispersal always inhibits the evolution of male harm, we find that the opposite can also be true, depending upon parameter values.

Introduction

Kin selection operates when individuals impact upon the reproductive success of their genetic relatives (Hamilton, 1963, 1964, 1970; Maynard Smith, 1964). Because mating success is a component of reproductive success, there is great potential for kin selection to operate in contexts that have traditionally been viewed through the lens of sexual selection (Pizzari & Gardner, 2012). Yet, the vast literatures on kin selection and sexual selection remain curiously disconnected.

Rankin's (2011) theoretical analysis of the role of kin selection in mediating the evolution of sexual conflict in viscous populations is therefore a welcome contribution, which has helped to draw empirical research attention to this important but greatly neglected topic (e.g. Carazo *et al.*, 2014). Sexual conflict occurs when male–male competition favours harmful traits in males that reduce female fitness and when females evolve counteradaptations to make them less vulnerable to such male traits (Parker, 1979; Chapman *et al.*, 2003; Arnqvist & Rowe, 2005; Parker, 2006). The major conceptual aim of Rankin's analysis was to demonstrate that kin selection can operate on such sexual conflict traits. And the main mathematical aim of the analysis was to investigate how sex-specific rates of dispersal impact upon the evolution of male-harm and female-resistance phenotypes. Specifically, Rankin's analysis suggested that male-harm traits are promoted by male dispersal and that they are inhibited by female dispersal.

Although the effects of sex-biased dispersal upon the genetical structure of the population – and hence the relatedness term in Hamilton's (1963, 1964, 1970) rule of kin selection – were carefully tracked in Rankin's analysis, the effects of sex-biased dispersal upon fitness – and hence upon the cost and benefit terms in Hamilton's rule – were neglected. As Rankin's analysis is incomplete, his results can be considered only tentative.

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Accordingly, the role for sex-specific rates of dispersal to mediate the evolution of sexual conflict remains obscure.

Here, we incorporate the impact of sex-biased dispersal upon fitness in Rankin's model, to investigate the overall impact of male dispersal and female dispersal on the evolution of sexual conflict. Following Rankin's analysis, our main focus is on the evolution of male-harm traits: in particular, we derive a threshold condition describing when male harm will evolve, and we determine the level of male harm that is expected to evolve in the absence of female resistance. We then consider the concomitant evolution of female resistance traits as a counteradaptation to male harm, and the resulting coevolutionary dynamics of sexual conflict. Our presentation largely follows that of Rankin's study, to facilitate comparison, but we provide a more thorough investigation of the effects of varying model parameters in the Supporting information.

Analysis

Model

Following Rankin (2011), we consider an infinite population, structured into patches containing n_M diploid males and n_F diploid females, which engage in sexual conflict. We consider that each individual's fecundity is mediated by their own quantitative traits and those of their patchmates. Specifically, the fecundity of a focal male i in patch j is given by $f_{Mij} = 1 + b\, y_{ij}(1 - s\, x_j) - u\, y_{ij}$, where: y_{ij} describes this male's own level of harming; x_j describes the average level of female resistance in patch j; b describes the personal fecundity benefit of harming in the absence of female resistance, in terms of improved mating success; s describes the effectiveness of female resistance in reducing the benefits of male harm; and u describes the personal fecundity cost of harming. And the fecundity of a focal female i in patch j is given by $f_{Fij} = 1 - c\, y_j(1 - h\, x_{ij}) - v\, x_{ij}$, where: x_{ij} describes this female's own level of resistance; y_j describes the average level of male harm in patch j; c describes the personal fecundity cost of being harmed in the absence of female resistance; h describes the effectiveness of female resistance in reducing the female fecundity cost of male harm; and v describes the personal fecundity cost of resistance. Mating occurs within the patch, at random, and with each female mating a large number of times, leading to the production of a large number of offspring in proportion to her fecundity, with an even sex ratio. Adults then die, and each offspring either disperses to a new randomly chosen patch with probability m_M for males and m_F for females or else remains in the natal patch. Following the dispersal phase, n_M males and n_F females are chosen at random within each patch to survive to adulthood, and all other individuals perish, returning the population to the beginning of the lifecycle.

Fitness

To investigate how kin selection acts upon male-harm and female-resistance traits, Rankin (2011, eqn 6) assumed that female fitness is given by $W_{Fij} = f_{Fij}[\{(1 - \bar{m})/((1 - \bar{m})f_{Fj} + \bar{m}f_F)\} + \bar{m}/f_F]$, where: f_{Fj} is the average fecundity of all the females in patch j; f_F is the average fecundity of all the females in the population; and $\bar{m} = (m_F + m_M)/2$ is the average of two sex-specific rates of dispersal. However, the correct female fitness function emerging from the assumptions of Rankin's model is as follows:

$$W_{Fij} = f_{Fij}\left(\frac{1}{2}\left(\frac{1 - m_F}{(1 - m_F)f_{Fj} + m_F f_F} + \frac{m_F}{f_F}\right) + \frac{1}{2}\left(\frac{1 - m_M}{(1 - m_M)f_{Fj} + m_M f_F} + \frac{m_M}{f_F}\right)\right). \tag{1}$$

That is, the focal female produces a number of offspring in proportion to her fecundity f_{Fij}, and of these, a fraction (½) are female and a fraction (½) are male (more generally, these quantities refer to each sex's reproductive value at birth; Fisher, 1930; Gardner, 2014; Grafen, 2014). Each daughter either remains in the focal patch with probability $1 - m_F$, in which case she competes for breeding opportunities with a number of other females proportional to $(1 - m_F)f_{Fj} + m_F f_F$, or she disperses to a new patch with probability m_F, in which case she competes for breeding opportunities with a number of other females proportional to f_F. Similarly, each son either remains in the focal patch with probability $1 - m_M$, in which case he competes for breeding opportunities with a number of other males proportional to $(1 - m_M) f_{Fj} + m_M f_F$, or he disperses to a new patch with probability m_M, in which case he competes for breeding opportunities with a number of other males proportional to f_F.

Our eqn (1) is equivalent to Rankin's eqn (6) if there is no sex bias in dispersal (i.e. $m_F = m_M = \bar{m}$). However, the two equations are distinct if there is sex-biased dispersal (i.e. $m_F \neq m_M$). In particular, because the model assumes a fixed number of adult females and a fixed number of adult males breeding within each patch, the probability that a particular female wins a breeding position depends only on the number of females competing for breeding opportunities on that patch and does not depend on the number of males competing for breeding opportunities on that patch, and the probability that a particular male wins a breeding position depends only on the number of males competing for breeding opportunities on that patch and does not depend on the number of females competing for breeding opportunities on that patch. This within-sex aspect of competition for breeding opportunities is reflected in our eqn (1) but not in Rankin's eqn (6).

Similarly, Although Rankin (2011) assumed that male fitness is given by $W_{Mij} = [f_{Mij}/f_{Mj}]f_{Fj}[\{(1 - \bar{m})/((1 - \bar{m})f_{Fj} + \bar{m}f_F)\} + \bar{m}/f_F]$, where f_{Mj} is the average

fecundity of all the males in patch j, the correct male fitness function emerging from the assumptions of Rankin's model is as follows:

$$W_{Mij} = \frac{f_{Mij}}{f_{Mj}} f_{Fj} \left(\frac{1}{2} \left(\frac{1-m_F}{(1-m_F)f_{Fj}+m_F f_F} + \frac{m_F}{f_F} \right) \right.$$
$$\left. + \frac{1}{2} \left(\frac{1-m_M}{(1-m_M)f_{Fj}+m_M f_F} + \frac{m_M}{f_F} \right) \right). \quad (2)$$

That is: the total number of offspring fathered by all of the males in his patch is equal to the total number of offspring produced by females in his patch and is proportional to f_{Fj}; he fathers a fraction f_{Mij}/f_{Mj} of these offspring; and the opportunity for these offspring to obtain breeding positions is exactly as calculated for eqn (1). Again, our eqn (2) is equivalent to Rankin's eqn (5) in the absence of any sex bias in dispersal (i.e. $m_F = m_M = \bar{m}$), but the two equations are distinct if there is any sex bias in dispersal (i.e. $m_F \neq m_M$) and for the same reason explained above.

Relatedness

Also required for a kin selection analysis of sexual conflict is calculation of the appropriate kin selection coefficients of genetic relatedness. The three coefficients that are required for the present analysis were calculated by Rankin (2011) to be as follows:

$$r_{mm} = \frac{1}{n_M} + \frac{n_M - 1}{n_M}(1-m_M)^2 r \quad (3)$$

for the relatedness between two adult males drawn at random from the same patch, with replacement;

$$r_{ff} = \frac{1}{n_F} + \frac{n_F - 1}{n_F}(1-m_F)^2 r \quad (4)$$

for the relatedness between two adult females drawn at random from the same patch, with replacement; and

$$r_{fm} = (1-m_F) + (1-m_M)r \quad (5)$$

for the relatedness between an adult female and an adult male drawn at random from the same patch.

These three coefficients of relatedness are each expressed in terms of the relatedness between two random juveniles, born on the same patch, which Rankin gives as $r = (n_F + n_M)/[(1 - m_F)^2 n_F + (1 - m_M)^2 n_M + (4 - m_F - m_M)(m_F + m_M)n_F n_M]$ in his eqn (14), but which we find to be given by the following (see Appendix for derivation):

$$r = \frac{n_F + n_M}{(1-m_F)^2 n_M + (1-m_M)^2 n_F + (4-m_F-m_M)(m_F+m_M)n_F n_M}. \quad (6)$$

Although our eqn (6) is equivalent to Rankin's eqn (14) when there is no sex bias in dispersal (i.e.

$m_F = m_M$), the two expressions are distinct if there is sex-biased dispersal (i.e. $m_F \neq m_M$). This particular error in Rankin's analysis appears to be purely typographical and does not carry through to his results.

Evolution of male harm

Performing a kin selection analysis of the fitness functions given by eqns (1) and (2), using the 'personal fitness' or 'neighbour-modulated fitness' approach (Hamilton, 1964; Taylor, 1996; Frank, 1997, 1998; Rousset, 2004; Taylor *et al.*, 2007; see Appendix for details), analogous to the one performed by Rankin (2011), we find that natural selection favours the emergence of male harm (i.e. for y to increase from zero) when:

$$b(1-sx) - u > \frac{c(1-hx)}{(1-vx)}$$
$$\times \frac{\left(\frac{r_{mm}+r_{fm}}{2}\right)4\left(1 - \frac{(1-m_F)^2+(1-m_M)^2}{2}\right)}{2(1-r_{mm})}. \quad (7)$$

Note that although expression (7) could be further simplified, for example by cancelling the common factor 2 from the numerator and denominator on the right hand side, we present it in this form to facilitate comparison with Rankin's expression (11). Inspection reveals that only difference between our expression (7) and Rankin's expression (11) is that we write 4 $\{1 - [(1 - m_F)^2 + (1 - m_M)^2]/2\}$, where he writes $(4 - m_F - m_M)(m_F + m_M)$, and this confirms the nature of Rankin's error. Specifically, our term can be rewritten as $4(1 - a)$, where $a = [(1 - m_F)^2 + (1 - m_M)^2]/2$ is the 'scale of competition', i.e. the probability that any two individuals competing for breeding opportunities within a patch are native to that patch (Frank, 1998; Gardner, 2010). That is: a female competes only with other females for access to the fixed number of female breeding opportunities, she is a native with probability $1 - m_F$, and her competitor is a native with probability $1 - m_F$, giving a combined probability of $(1 - m_F)^2$; a male competes only with other males for access to the fixed number of male breeding opportunities, he is a native with probability $1 - m_M$, and his competitor is a native with probability $1 - m_M$, giving a combined probability of $(1 - m_M)^2$; and the average of these two effects, taken across individuals of both sexes, is as follows: $[(1 - m_F)^2 + (1 - m_M)^2]/2$. In contrast, Rankin's term rearranges as $4(1 - a')$, where $a' = [1 - (m_F + m_M)/2]^2$ is an incorrect formulation of the scale of competition, which appears to suggest that males and females are competing with each other for the same, unisex breeding positions (which contradicts Rankin's model assumption of a fixed number of females and a fixed number of males breeding in every patch). The proper expression for the scale of competition for sex-biased dispersal previously appeared

in the two studies upon which Rankin's population model is explicitly based: in the form $[(1 - d_f)^2 + (1 - d_m)^2]/2$, where d_f and d_m are simply alternative notation for m_F and m_M, in Gardner (2010); and in the form $(h_f^2 + h_m^2)/2$, where h_f and h_m are exactly equivalent to $1 - m_F$ and $1 - m_M$ in the absence of any cost of dispersal, in Johnstone & Cant (2008).

Again, although our expression (7) is equivalent to Rankin's expression (11) when there is no sex bias in dispersal (i.e. $m_F = m_M$), the two expressions are distinct if there is sex-biased dispersal (i.e. $m_F \neq m_M$). As Rankin noted, in the absence of relatedness ($r_{mm} = r_{fm} = 0$), this expression reduces to $b > u/(1 - sx)$, such that the cost c that a female experiences upon being subjected to harm is irrelevant to the evolution of the male trait.

Female resistance to male harm is not needed if there is no male harm, so it is informative to consider expression (7) in the absence of female resistance ($x = 0$), and this yields the following:

$$b - u > c \times \frac{\left(\frac{r_{mm} + r_{fm}}{2}\right) 4 \left(1 - \frac{(1 - m_F)^2 + (1 - m_M)^2}{2}\right)}{2(1 - r_{mm})}. \quad (8)$$

Our expression (8) is equivalent to Rankin's expression (12) when there is no sex bias in dispersal (i.e. $m_F = m_M$), but, again, the two expressions are distinct if there is sex-biased dispersal (i.e. $m_F \neq m_M$). As was noted by Rankin, increasing the relatedness among males (higher r_{mm}) and/or increasing relatedness between females and males (higher r_{fm}) leads to a more stringent condition for male harm to emerge from rarity.

Expression (8) may be rearranged into a form that makes clearer the selective forces operating upon the male-harm trait as follows:

$$b - u - (b - u)r_{mm} - c(r_{mm} + r_{fm})(1 - a) > 0, \quad (9)$$

where $a = ((1 - m_F)^2 + (1 - m_M)^2)/2$ is the scale of competition (see above). Expression (9) is readily interpreted in terms of a male actor's inclusive fitness. A male who increases his investment into harming seizes a greater share of the overall mating success of the males in his patch, yielding an inclusive fitness benefit $(b - u)$. However, the corresponding loss of mating success experienced by local males yields an inclusive fitness loss $(b - u)r_{mm}$. Moreover, harming reduces the overall fecundity of local females – decreasing the number of offspring produced by local females by c and decreasing the number of offspring produced by local males by c – but, owing to local competition, this translates into a loss of only $c(1 - a)$ surviving offspring for local females, and the same for local males, and this leads to an inclusive fitness loss of $c(r_{mm} + r_{fm})(1 - a)$.

Expression (8) describes the condition for nonzero male harm to be favoured by natural selection, in terms of an adult male's relatedness to the local population of females and males in his patch. Equations (3) and (5) describe these relatedness coefficients in terms of the demographic parameters of the population, and so substituting these equations into expression (8), we recover a demographically explicit condition for nonzero male harm to be favoured by natural selection:

$$b - u > c$$
$$\frac{[(m_F(2 - m_F) + m_M(2 - m_M))(m_F^2(n_F - 1)}{} $$
$$\frac{- m_M(2 + n_F + m_M(n_M - 1) - 3n_M) - 2(n_F + n_M)}{}$$
$$\frac{+ m_F(2 + n_F(m_M - 3) + n_M(1 - m_M)))]}{[2(n_M - 1)(m_F(2 - m_F) - m_M(2 - m_M))}$$
$$\frac{}{- n_F(4 - m_F - m_M)(m_F + m_M))]}. \quad (10)$$

Our expression (10) is equivalent to Rankin's expression (15) when there is no sex bias in dispersal (i.e. $m_F = m_M$), but, once more, the two expressions are distinct if there is sex-biased dispersal (i.e. $m_F \neq m_M$). Following Rankin, we illustrate this condition in Fig. 1, under the simplification of an even operational sex ratio ($n_F = n_M$). In contrast to Rankin's suggestion that increased female dispersal inhibits the evolution of male harm and increased male dispersal promotes the evolution of male harm, we find that increased female dispersal may promote or inhibit the evolution of male harm, depending upon parameter values (Fig. 1a), and increased male dispersal may also promote or inhibit the evolution of male harm, depending upon parameter values (Fig. 1b). For instance, inspecting Fig. 1a, an increase in the rate of female dispersal (increasing m_F) acts to promote male harm (i.e. decreases the threshold male benefit/female-harm ratio) when there is no male dispersal ($m_M = 0$), but it acts to inhibit male harm (i.e. increases the threshold male benefit/female-harm ratio) when there is complete male dispersal ($m_M = 1$); inspecting Fig. 1b, an increase in the rate of male dispersal (increasing m_M) acts to promote male harm (i.e. decreases the threshold male benefit/female-harm ratio) when there is no female dispersal ($m_F = 0$), but it acts to inhibit male harm (i.e. increases the threshold male benefit/female-harm ratio) when there is complete female dispersal ($m_F = 1$). We also explore the consequences of varying the parameters n_F and n_M in the Supporting information (Fig. S1), finding that increasing n_F and/or n_M acts to promote male harm, owing to the concomitant reduction in genetic relatedness.

Following Rankin, we also investigate the stable level of male harm that evolves in the absence of female resistance, and we find that this is given by the following (see Appendix for details):

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$$y^* = \frac{[2(b-u)(1-r_{mm}) - c(r_{mm} + r_{fm})}{(m_F(2-m_F) + m_M(2-m_M))]}{[(b-u)c(2(1-r_{mm}) + (r_{mm} + r_{fm})}{(m_F(2-m_F) + m_M(2-m_M)))]}. \qquad (11)$$

Once again, our eqn (11) is equivalent to Rankin's eqn (17) when there is no sex bias in dispersal (i.e. $m_F = m_M$), but the two expressions are distinct if there is sex-biased dispersal (i.e. $m_F \neq m_M$). Following Ran-

kin, we illustrate the stable level of male harm in Fig. 2, under the simplification of an even operational sex ratio ($n_F = n_M$). As before, our results are qualitatively and quantitatively different from those of Rankin, in that increasing the rate of female dispersal may either increase or decrease the stable level of male harm (Fig. 2a), and increasing the rate of male dispersal may either increase or decrease the stable level of male harm (Fig. 2b). We also explore the consequences of varying the parameters n_F and n_M in the Supporting
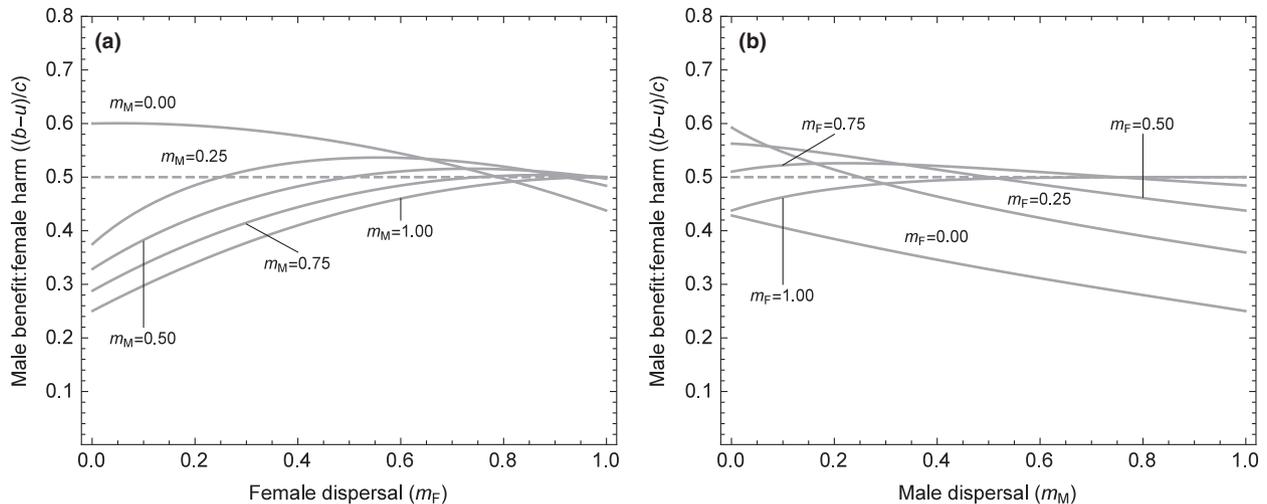
Fig. 1 Invasion threshold for male harm y, in the absence of female resistance ($x = 0$), as a function of (a) female dispersal rate m_F and (b) male dispersal rate m_M, assuming three females and three males in each patch ($n_F = n_M = 3$). In contrast to Rankin's (2011) fig. 1, we find that increasing female dispersal may promote or inhibit male harming and that increasing male dispersal may promote or inhibit male harming, depending upon parameter values.
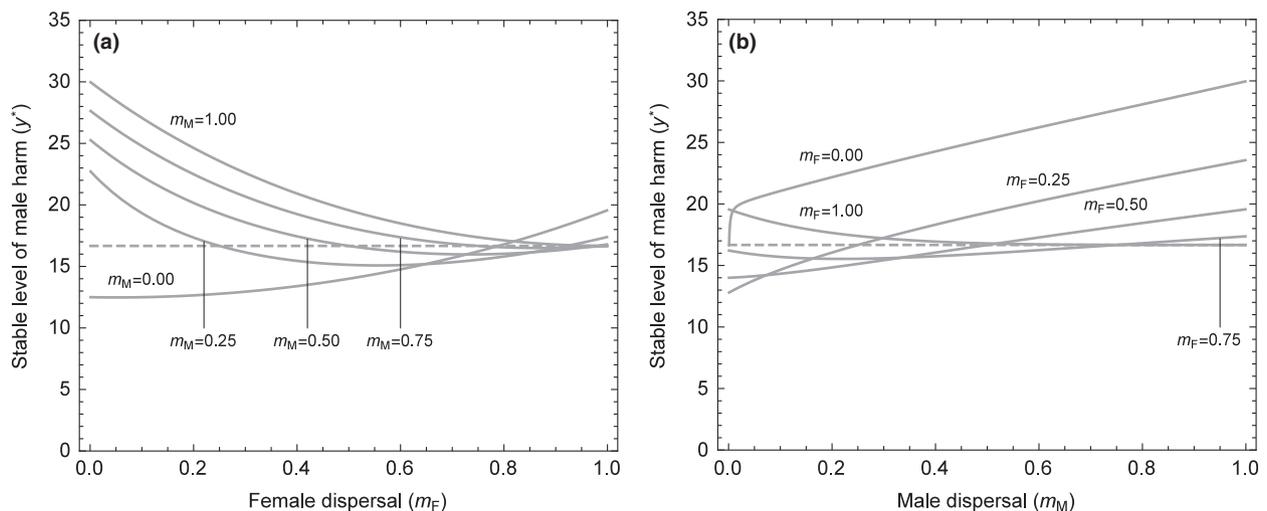
Fig. 2 Stable level of male harm y*, in the absence of female resistance ($x = 0$), as a function of (a) female dispersal rate m_F and (b) male dispersal rate m_M, assuming three females and three males in each patch ($n_F = n_M = 3$). In contrast to Rankin's (2011) fig. 2, we find that increasing female dispersal may promote or inhibit male harming and that increasing male dispersal may promote or inhibit male harming, depending upon parameter values. Other parameter values are $b = 0.05$, $u = 0.05$ and $c = 0.02$.

information (Fig. S2), again finding that increasing n_F and/or n_M acts to promote male harm owing to reduced relatedness. Finally, we perform numerical simulations to confirm our analytical results (Figs S3 and S4).

Evolution of female resistance

Performing a kin selection analysis of the fitness functions given by eqns (1) and (2), analogous to the one performed by Rankin, we find that natural selection favours the emergence of female resistance to male harm (i.e. for x to increase from zero) when:

$$\frac{hcy - v}{1 - cy} \left[1 - ar_{ff} + (1-a)r_{fm} \right] > 0, \qquad (12)$$

which, so long as $cy < 1$, is equivalent to $hcy > v$ and which recovers Rankin's eqn (18); that is, female resistance is favoured so long as it improves female fecundity, and the relatedness structure of the population serves only to scale the strength of natural selection. In particular, female resistance is not favoured in the absence of male harm ($y = 0$), and the condition for female resistance to be favoured becomes less stringent as the level of male harm increases (higher y).

Assuming equal genetic variance in the male and female traits – such that the response to selection is proportional to marginal fitness, with the same constant of proportionality for both male and female traits (see Appendix for more details) – we may follow Rankin's approach to investigate the coevolutionary dynamics of male harm and female resistance. Although quantitatively different, here our results are qualitatively the same as Rankin's: in general, stable limit cycles emerge, with low female resistance favouring an increase in male harm, which favours an increase in female resistance, which favours reduced male harm, which then favours reduced female resistance, etc. (Fig. 3). We also explore the consequences of varying the relative evolvability of female and male traits – which has a quantitative but not a qualitative impact – and provide numerical simulation confirmation of these coevolutionary dynamics, in the Supporting information (Fig. S5).

Discussion

An important aim of Rankin's (2011) analysis of the evolution of sexual conflict in genetically structured populations was to determine the impact of sex-specific rates of dispersal upon the evolution of male-harm and female-resistance phenotypes. However, that analysis did not correctly take into account the impact of sex-biased dispersal upon female and male fitness. Here, we have remedied this problem, allowing fitness expressions to emerge directly from the assumptions of Rankin's model, and we have explored the consequences of implementing these new fitness functions for the evolution of
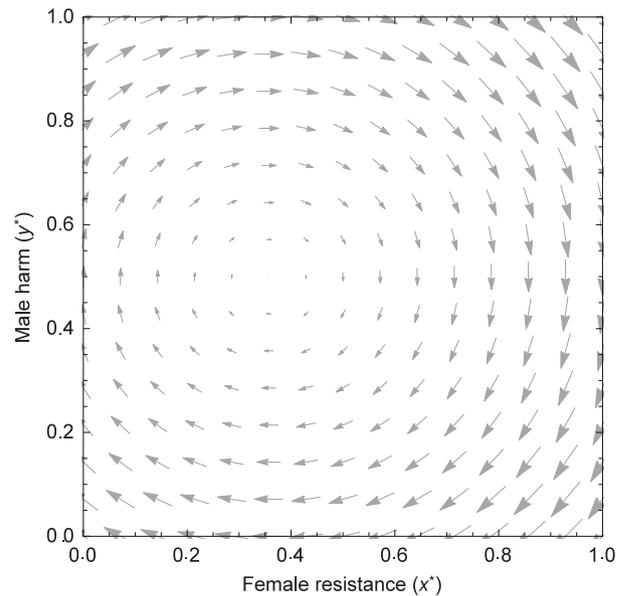
Fig. 3 Cyclical coevolutionary dynamics of male harm y and female resistance x, assuming $n_F = n_M = 3$, $b = 0.05$, $u = 0.03$, $c = 0.02$, $v = 0.01$, $s = 0.75$, $h = 1$, $m_F = 0.25$, $m_F = 0.25$ and $k = 1$ in line with Rankin's (2011) fig. 3. The arrows indicate the direction and relative magnitude of the response to natural selection, that is a product of marginal fitness and additive genetic variance. Owing to weakness of selection, actual generation-to-generation changes are vanishingly small.

sexual conflict. In contrast to Rankin's prediction that increased female dispersal always inhibits the evolution of male harm and increased male dispersal always promotes the evolution of male harm, we have found no straightforward relationship between sex-biased dispersal and the evolution of male harm, as the increased dispersal of either sex may promote or inhibit the evolution of male harm, depending upon parameter values.

Broadly speaking, we find that a higher rate of female dispersal may inhibit the evolution of male harm when the rate of male dispersal is also high (i.e. relatively unbiased dispersal), but it may promote the evolution of male harm if the rate of male dispersal is low (i.e. relatively female-biased dispersal). Similarly, a higher rate of male dispersal may promote the evolution of male harm when the rate of female dispersal is low (i.e. relatively male-biased dispersal), but it may inhibit the evolution of male harm when the rate of female dispersal is also high (i.e. relatively unbiased dispersal). In other words, male harming is most favoured when dispersal is strongly sex-biased, irrespective of the direction of this bias. This mirrors Gardner's (2010) result that selfishness is maximized when one sex always disperses and the other sex never disperses.

Our study serves to highlight that care must be taken in ensuring that appropriate fitness functions are subjected to kin selection analysis. The Taylor–Frank approach to kin selection analysis (Taylor, 1996; Taylor

& Frank 1996; Frank, 1997, 1998; Taylor *et al.*, 2007) achieves great flexibility by not being prescriptive as to how fitness functions are to be developed, allowing the modeller to choose their own preferred framework. For example, the fitness function may be derived from explicit consideration of ecological processes, as has been done in the present study (see Gardner *et al.* 2007 for an example of a more detailed derivation of a fitness function from explicit population dynamics); or, alternatively, the fitness function may be developed in a more free-form way, without reference to an explicit population model, enabling greater generality (e.g. eqn 7.1 of Frank, 1998). But it is important to ensure that, whatever approach is taken, the analysis is not self-contradictory. The problem with the fitness functions assumed by Rankin (2011) is that they implicitly assume that individuals of both sexes compete with each other for breeding opportunities, whereas other aspects of Rankin's model assume that only individuals of the same sex compete with each other for breeding opportunities. Accordingly, Rankin's error is not methodological but rather one of overspecification.

Despite the qualitative difference in our results, the present analysis confirms several of the broader conceptual points made by Rankin (2011). Importantly, Rankin highlighted that the relationship between dispersal and male harm is complicated by the fact that dispersal tends to reduce relatedness within mating groups – which tends to promote male harm – but it also tends to relax local competition for reproductive resources – which tends to inhibit male harm. In scenarios where both sexes disperse at the same rate, these opposing effects of dispersal may often cancel each other out, such that there is negligible impact of dispersal rate on the evolution of social traits (Bulmer, 1986; Frank, 1986; Taylor, 1992a). The possibility of sex-biased dispersal complicates matters further, as it may decouple the impact of dispersal upon relatedness versus local competition (Gardner, 2010). Given these complexities, it is unsurprising that we have found no straightforward relationship between sex-specific rates of dispersal and levels of male aggression towards females. Moreover, we emphasize that, although increased relatedness will tend to inhibit male harm, it may be correlated – via demographic factors such as limited dispersal – with confounding local competition effects that may tend to promote male harm, leading to potentially misleading correlations in empirical studies and results that may appear to conflict with those of other theoretical studies.

Carazo *et al.* (2014) provide the only empirical study explicitly testing Rankin's (2011) predictions on the role of kin selection in sexual conflict. They found that triplets of unrelated males exhibited more competitive behaviour in the presence of a single unrelated female than did triplets of related males (in particular, brothers) in the presence of a single unrelated female and that females in the former treatment suffered reduced lifetime reproductive success than those in the latter treatment. This result is in broad agreement with Rankin's and our general prediction that increased male–male relatedness *per se* reduces the incentive for harming behaviour. However, as Carazo *et al.*'s (2014) study considered facultative behaviour, presumably in response to kin recognition, rather than obligate evolutionary adaptation to different ecological and demographic scenarios, it does not provide a direct test of either Rankin's or our theoretical predictions. Such a direct test could be achieved using evolutionary experimental methods, whereby a laboratory population of a suitably amenable species is subjected to different ecological and demographic treatments – including variable dispersal rates (i.e. m_F and m_M) and operational sex ratios (i.e. n_F and n_M) – for multiple generations, analogous to Macke *et al.*'s (2014) elegant study of relatedness-mediated conflict between mating partners over sex allocation in spider mites.

Although we have focused upon correcting a number of errors in Rankin's (2011) theoretical study, more generally our study is supportive of Rankin's main conceptual point, which was to emphasize the potential role for kin selection to modulate the evolution of sexual conflict. Despite kin selection and sexual selection both originating with Charles Darwin – in *The Origin of Species* (Darwin, 1859) and *The Descent of Man* (Darwin, 1871), respectively – these two topics have developed more or less independently ever since. Yet there is huge scope for interactions between genetic relatives to impact upon each other's mating success and, accordingly, for individuals who are striving to maximize their inclusive fitness to take into account both their own mating success and also that of their relatives. Our reanalysis of Rankin's (2011) model is not intended to halt this exciting crossover of ideas between subdisciplines, but rather to establish these links in rigorous theoretical terms.

Indeed, there is a growing theoretical and empirical literature on kin selection in viscous populations, which has explored not only the consequences of sex-biased dispersal (Johnstone & Cant, 2008; Gardner, 2010; Rankin, 2011; the present study), but also – for example – the impact of budding dispersal (Gardner & West, 2006; Lehmann *et al.*, 2006; Gardner *et al.*, 2009; Kümmerli *et al.*, 2009), population elasticity (Taylor, 1992b; Boots & Mealor, 2007; Alizon & Taylor, 2008; Wild *et al.*, 2009), ploidy (Taylor, 1988; Yeh & Gardner, 2012), dispersal-dependent behaviour (Perrin & Lehmann, 2001; El Mouden & Gardner, 2008; Wild & Fernandes, 2009), variation in resource availability (Rodrigues & Gardner, 2012, 2013, 2015; Rodrigues & Johnstone, 2014), overlapping generations (Taylor & Irwin, 2000; Irwin & Taylor, 2001), birth–death versus death–birth demographies (Grafen & Archetti, 2008) and reproductive skew (Johnstone, 2008). Many of these ecological, demographic and genetic factors will have application to the biology of sexual selection and

sexual conflict, and this represents an exciting avenue for future theoretical developments and empirical research.

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Appendix

Natural selection

Natural selection favours an increase in an allele's frequency if carriers of that allele are, on average, fitter than noncarriers. In the context of separate female and male classes, this correct measure of fitness is as follows: $W = c_F W_F + c_M W_M$, where c_F and c_M are the class reproductive values of females and males, respectively (and are given by $c_F = c_M = \frac{1}{2}$ under diploidy), and W_F and W_M are the relative fitnesses of a focal female and a focal male, respectively (and are given by $W_F = W_{Fij}$ and $W_M = W_{Mij}$; that is, we have simply dispensed with the unnecessary indices). Accordingly, natural selection will act to increase the average genetic 'breeding' value for any phenotype of interest if $dW/dg > 0$, where g is the genic value associated with a gene picked at random from the population. Using the chain rule, and considering that the phenotype is male harm, then we may write $dW/dg = c_F[(\partial W_F/\partial y'_M) \times (dy'_M/dG'_M) \times (dG'_M/dg_F)] + c_M[(\partial W_M/\partial y_M) \times (dy_M/dG_M) \times (dG_M/dg_M) + (\partial W_M/\partial y'_M) \times (dy'_M/dG'_M) \times (dG'_M/dg_M)]$, where: y_M is a focal male's own level of harm (equivalent to y_{ij} in Rankin's notation); y'_M is the average level of male harm in the focal individual's patch (equivalent to y_j); G_M is a focal male's breeding value for the harming trait; and G'_M is the average breeding value for harm among the males in the focal individual's patch. We may denote: the genotype–phenotype map by $dy_M/dG_M = dy'_M/dG'_M = \psi$; the consanguinity of a male to himself by $dG_M/dg_M = p_m$ (Bulmer, 1994); the consanguinity of a male to a random male on his patch (including himself) by

$dG'_M/dg_M = p_{mm}$; and the consanguinity of a male to a random female on his patch by $dG'_M/dg_F = p_{fm}$. Finally, we may express the relatedness of a male to a random male on his patch (including himself) by $r_{mm} = p_{mm}/p_m$ (Bulmer, 1994) and the relatedness of a male to a random female on his patch by $r_{fm} = p_{fm}/p_m$. This yields the condition for increase:

$$\frac{u - b(1-sx)}{uy - b(1-sx)y - 1} + \left(\frac{b - u - bsx}{uy - b(1-sx)y - 1} \right. $$
$$\left. - \frac{c((2-m_F)m_F + (2-m_M)m_M)(1-hx)}{2(1 - vx - c(y-hxy))} \right) r_{mm}$$
$$- \frac{c((2-m_F)m_F + (2-m_M)m_M)(1-hx)}{2(1 - vx - c(y-hxy))} r_{fm} > 0. \tag{A1}$$

In addition to describing the direction of selection, expression (A1) also provides information about the magnitude of the response to selection. In particular, for a given amount of genetic variance in the male harm trait, the change in the population average of the trait owing to natural selection is proportional to the left hand side of expression (A1). Moreover, setting the left hand side of expression (A1) equal to zero, and solving for y, yields the stable level of harming y^* which, for $x = 0$, is given by eqn (11) of the main text.

Similarly, considering that the phenotype is female resistance, then we may write $dW/dg = c_F\{(\partial W_F/\partial x_F) \times (dx_F/dG_F) \times (dG_F/dg_F) + (\partial W_F/\partial x'_F) \times (dx'_F/dG'_F) \times (dG'_F/dg_F)\} + c_M\{(\partial W_M/\partial x'_F) \times (dx'_F/dG'_F) \times (dG'_F/dg_M)\}$, where: x_F is a focal female's own level of resistance (equivalent to x_{ij} in Rankin's notation); x'_F is the average level of female resistance in the focal individual's patch (equivalent to x_j); G_F is a focal female's breeding value for the resistance trait; and G'_F is the average breeding value for resistance among the females in the focal individual's patch. We may denote: the genotype–phenotype map by $dx_F/dG_F = dx'_F/dG'_F = \xi$; the consanguinity of a female to herself by $dG_F/dg_F = p_f$ (which is equal to p_m); and the consanguinity of a female to a random female on her patch (including herself) by $dG'_F/dg_F = p_{ff}$. Finally, we may express the relatedness of a female to a random female on her patch (including herself) by $r_{ff} = p_{ff}/p_f$. This yields the condition for increase:

$$\frac{v - chy}{vx + c(y-hxy) - 1}$$
$$- \frac{(2 - (2-m_F)m_F - (2-m_M)m_M)(v-chy)}{2(vx + c(y-hxy) - 1)} r_{ff}$$
$$+ \frac{((2-m_F)m_F + (+2-m_M)m_M)(v-chy)}{2(vx + c(y-hxy) - 1)} r_{mf} > 0. \tag{A2}$$

Again, expression (A2) provides information about the magnitude of the response to selection. For a given amount of genetic variance in the female-resistance trait, the change in the population average of the trait owing to natural selection is proportional to the left hand side of expression (A2). Moreover, if the genetic variance for the female-resistance trait is k times that for the male-resistance trait, then the constant of proportionality for corresponding to expression (A2) is k times that corresponding to expression (A1).

Relatedness

The consanguinity p' of two offspring that are born on the same patch is defined by drawing a gene at random from one of the two offspring and a gene at random (from the same locus) from the other offspring, and calculating the probability that these two genes are identical by descent (Bulmer, 1994). Assuming that consanguinities take their equilibrium values, we may write

$$p' = \frac{1}{4}\left(\frac{1}{n_F}p + \frac{n_F - 1}{n_F}(1 - m_F)^2 p'\right)$$
$$+ \frac{1}{4}\left(\frac{1}{n_M}p + \frac{n_M - 1}{n_M}(1 - m_M)^2 p'\right)$$
$$+ \frac{1}{2}(1 - m_F)(1 - m_M)p', \tag{A3}$$

where p is the consanguinity of an individual to itself. That is: with probability ¼, we have drawn the maternal gene from both individuals, in which case with probability $1/n_F$ they share the same mother giving consanguinity p, or else with probability $(n_F - 1)/n_F$ they have different mothers who are consanguineous only to the extent that both are locals, giving consanguinity $(1 - m_F)^2 p'$; with probability ¼, we have drawn the paternal gene from both individuals, in which case with probability $1/n_M$ they share the same father giving consanguinity p, or else with probability $(n_M - 1)/n_M$ they have different fathers who are consanguineous only to the extent that both are locals, giving consan-

guinity $(1 - m_M)^2 p'$; and with probability ½, we have drawn the maternal gene from one and the paternal gene from the other, in which case they are consanguineous only to the extent that both of these parents are locals, giving consanguinity $(1 - m_F)(1 - m_M)p'$. Rearranging, we have $p' = [(n_F + n_M)/((1 - m_F)^2 n_M + (1 - m_M)^2 n_F + (4 - m_F - m_M)(m_F + m_M)n_F n_M)]p$, and dividing both sides by p yields the coefficient of relatedness ($r = p'/p$; Bulmer, 1994), given by eqn (6) of the main text.

Supporting information

Additional Supporting Information may be found in the online version of this article:
Appendix S1. Simulation code.
Figure S1 Invasion threshold for male harm y, in the absence of female resistance ($x = 0$), as a function of (a) number of females n_F and (b) number of males n_M, assuming equal male and female dispersal in each patch ($m_F = m_M = 0.5$).
Figure S2 Stable level of male harm $y*$, in the absence of female resistance ($x = 0$), as a function of (a) number of females n_F and (b) number of males n_M, assuming equal male and female dispersal in each patch ($m_F = m_M = 0.5$).
Figure S3 Stable level of male harm $y*$, in the absence of female resistance ($x = 0$), as a function of female dispersal rate m_F, assuming three females and three males in each patch ($n_F = n_M = 3$).
Figure S4 Stable level of male harm $y*$, in the absence of female resistance ($x = 0$), as a function of female dispersal rate m_M, assuming three females and three males in each patch ($n_F = n_M = 3$).
Figure S5 Coevolutionary dynamics of male harm y and female resistance x, assuming $n_F = n_M = 3$, $b = 0.05$, $u = 0.03$, $c = 0.02$, $v = 0.01$, $s = 0.75$, $h = 1$, $m_M = 0.25$ and $m_F = 0.25$.