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Sexual conflict in viscous populations: The effect of the timing of dispersal

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ABSTRACT

In recent years, there has been increasing theoretical and empirical examination of how sexual conflict can arise between males and females. However, much this work has implicitly assumed that interactions take place in panmictic populations with complete dispersal, where interactions are between unrelated individuals. Here, we examine the consequences of limited dispersal and population structure for the evolution of a male phenotype that is associated with the males pre- and post-copulatory reproductive success, using an inclusive-fitness based analysis applied to group-structured populations. We show that: (i) the sex-specific timing of the dispersal phase of the life cycle can drive the evolution of sexual conflict; (ii) the inclusive fitness of a female in this conflict is determined solely by direct (i.e. personal) effects on its own competitive ability. Our analysis is supported by results from individual-based simulations of multi-level selection. Our results support the suggestion that kin selection can influence the evolution of sexual conflict, but reveal that such a role might be more complex than previously appreciated when sex-specific life histories are taken into consideration. We discuss the implications of our results for sexual conflict in various species of insects, but focus primarily on dipteran flies of the family Sepsidae.

1. Introduction

Reproductive decisions that benefit an individual often reduce the fitness of its partner(s) (Trivers, 1972; Arnqvist and Rowe, 2005; Parker, 2006). For example, male dung flies (*Scatophagus stercoraria*) appear willing to pay the cost associated with fighting one another in order to realize the benefit of ready access to a female, but these fights can result in damage to (or even death of) females (Parker, 1979). In these cases, it seems unlikely that any benefit fighting might confer upon the female would be sufficient to balance the severe fitness costs she incurs. One is inclined, therefore, to conclude that the male fighting in this species is subject to sexual conflict. In polyandrous species, males continue to compete with each other after copulation through the competition of their ejaculates for the fertilization of a set of eggs, and this extends the potential for sexual conflict to a post-copulatory level. Parker and Pizzari (2010) have argued that the possibility of interactions between these levels demands the development of models that link them. In particular, trade-offs between mating success (e.g. the success that follows male–male competition over access to mating opportunities) and traits that confer an advantage in sperm competition could provide a strong connection between pre- and post-copulatory occurrences of sexual conflict (Parker and Pizzari, 2010).

More subtle instances of conflict can arise, not because males and females balance personal (i.e. direct) fitness costs and benefits differently, but rather because males and females balance inclusive fitness costs and benefits differently. The sex-specific patterns of dispersal that are observed in many species of animal (Greenwood, 1980), for example, can help kin selection drive sexual conflict between mates over levels of parental investment (reviewed in West, 2009). More recently, Rankin (2011) has shown that kin selection is also able to shape the evolution of sexual conflict over those harmful male behaviours – like fighting among male dung flies – associated with competition for mates. Specifically, Rankin (2011) has shown that when males and females are related, that this can reduce selection for harming behaviours by males, because it incurs an indirect fitness cost. Along similar lines, Eldakar et al. (2009, 2010) have reported that movement in group-structured populations of the water strider, *Aquarius remigis*, affects the costs and benefits of harmful male behaviour in such a way that less harmful males persist.

Overall, the work cited above has drawn attention to the important part that the related effects of group structure and genetic kinship might play in resolving the sexual conflict that accompanies sexually selected behaviours. Nevertheless, the work neglects the possibility of pre- and post-copulatory male–male

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competition—the kind of male–male competition Parker and Pizzari (2010) argue, we should not ignore.

Our central goals here are (1) to investigate a model for the evolution of sexual conflict in a population in which relatives can interact (i.e. a viscous population), and (2) to investigate a scenario where there is both pre- and post-copulatory male–male competition. In doing so, we extend previous work by allowing the timing of dispersal to differ between males and females, by considering both diploid and haplodiploid genetic systems, and by allowing for varying levels of female promiscuity.

To meet our goals, we apply the inclusive-fitness approach introduced by Hamilton (1964), and extended by Taylor (1989, 1988a,b, 1996). This approach has been previously used to examine the consequences of sex-specific timing of dispersal for a different social trait: the sex ratio (Wild and Taylor, 2005; Wild and West, 2009).

Overall, we show how the sex-specific timing of dispersal can drive sexual conflict when male–male competition occurs both before and after copulation. By considering both positive (helpful) and negative (harmful) effects, we also show that the outcome of sexual conflict can be determined by the effect that male phenotype exerts on female competitive ability. Our conclusions are supported by results from computer simulation of multi-level selection, indicating that the inclusive-fitness approach we employ continues to make useful predictions when key assumptions (most importantly weak selection) are violated. Our conclusions also appear to be reflected in the biology of sepsid dung flies (Diptera: Sepsidae), suggesting a role for kin selection (or group structure) in the mediation of sexual conflict found in that family.

2. The basic inclusive-fitness model

2.1. Social-evolutionary roles

We are interested in the evolution of some continuous male phenotype that influences (a) its own ability to compete for mates, (b) the competitive ability of its female mates, and (c) the ability of its sperm to compete for fertilization.

As mentioned above, we use the inclusive-fitness approach to study the evolution of the male phenotype, and so we place a male in the social-evolutionary role of 'actor'. To study the possibility for social-evolutionary conflict over the level at which male phenotype is expressed, we also fix attention on an individual whose genotype determines male phenotype. In social-evolutionary terms this focal individual is said to have 'control of the actor's behaviour'. In this paper, we give a male control of its phenotype in order to establish the 'male perspective' in a potential conflict situation, and we give a female control of male phenotype to establish the 'female perspective'. In both cases, our inclusive-fitness model will consider how a small change in a male actor's phenotype, in turn, changes the reproductive value of the 'recipients' that are genetically related to the individual with control (Fig. 1).

Models of evolution routinely invoke assumptions about an individual's genotype affecting the way in which its own phenotype is expressed, and so the male-control scenario will likely not strike the reader as odd. Less familiar is the assumption that one individual's genotype affects the phenotypic expression of another (but see for e.g. Taylor, 1988a; Wild and West, 2009). The reader may, therefore, feel relatively uncomfortable with the female-control scenario. To establish a biological context for the female-control scenario we could posit the existence of an intermediary female phenotype that (i) is controlled by the female's own genes, and that (ii) elicits a plastic phenotypic response in *potential* male mates just prior to male–male competition for inseminations. For example, female genotype could control female colouration or receptivity which could, in turn, mean that potential male mates compete more/less aggressively for copulations. Greater investment in competition for mates may imply diminished male condition (e.g. caused by fighting, see Parker, 1979), or that there is less energy available for sperm production. Consequently, more intense mate-competition could compromise eventual male sperm-competition ability following any mating a male manages to secure.

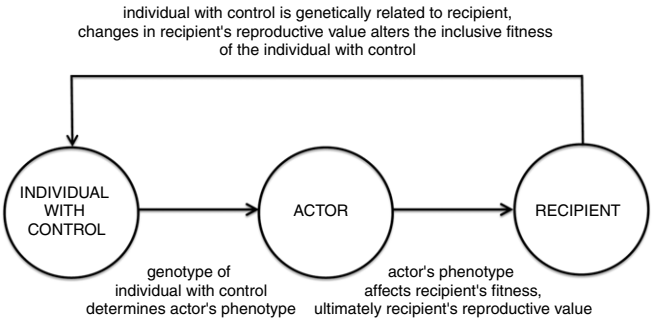


Fig. 1. Social evolutionary roles in the inclusive-fitness model and their relation to one another. Our approach is to calculate the change in the inclusive fitness of the 'individual with control' that occurs as the result of deviant phenotypic expression on the part of the 'actor'. Deviant phenotypic expression is due to the deviant genotype of the 'individual with control'. Inclusive-fitness changes are mediated by changes in the reproductive value of 'recipients' that are genetically related to the 'individual with control'.

We postpone the discussion of conflict to a later section. For now, we focus on building our basic inclusive-fitness model—one that adopts the perspective of an arbitrary individual with control.

2.2. The inclusive fitness effect of changing male phenotype

Consider a sexual population with discrete, non-overlapping generations. We assume, first, that this population is at demographic equilibrium, and we let u_m and u_f denote the density of males and females, respectively, at the beginning of each generation (Table 1 explains all notation used in the main text). Next, we assume that the level of phenotypic expression for each male in the population is determined prior to male–male competition. Finally, we assume that male phenotype is always expressed at some 'normal' or wild-type level, with the sole exception of the deviant or 'mutant' expression exhibited by one particular male (the 'actor' mentioned above). Again, though the male actor may have control over the expression of its phenotype, this may not always be the case.

As previously indicated, the consequences of phenotypic change are to be observed at three distinct points in the life cycle, and so we expect phenotypic change to have multiplicative effects on a recipient's reproductive value. When the phenotypic change shown by the deviant actor is small, however, the overall multiplicative effects can be approximated by simply adding the effects (a)–(c) when these happen in isolation (this is the principle behind the Product Rule for differentiation that one encounters in an introductory course in calculus). That said, it is still important to note that, even though the phenotypic change has effects at multiple points in time, the fitness consequences of phenotypic change must be evaluated beginning with that point in time when the phenotype is determined (i.e. prior to competition for mates).

2.2.1. Effect (a), male's ability to compete for mates

To begin, we consider what happens to the inclusive fitness of the individual with control (i.e. the individual whose perspective we adopt) when the deviant phenotype of the actor affects only its own ability to secure mates. We suppose that each female mates with exactly M males, so that Mu_f/u_m expresses the normal level of male mating success. Let $s_a(\delta)$ measure the mating success of

Table 1
Summary and explanation of notation found in the main text.

Symbol	Explanation
c_i	Total reproductive value of sex-i.
δ	Difference between mutant phenotype and normal (i.e. wild-type) phenotype (reflects the strength of selection).
Δw_X	Change in the inclusive fitness of the individual with control of actor's phenotype due to $X = a, bf, bm, c$ of the life cycle.
Δw	Overall change in the inclusive fitness of the individual with control.
Δw^i	$= \Delta w$, but emphasizes the fact that sex i has control.
ϵ	Weight given to ζ^m in determination of z.
h	Variable that indicates haplodiploidy; $= 0$ if diploid, $= 1$ if haplodipliod.
$k_X(z)$	Instantaneous rate at which $s_X(\delta)$ changes in a neutral population; $= s'_X(0)$.
K_i	Probability that a sex-i individual competing on a given patch is native to that patch.
M	Number of mates selected by each female.
N	Size of groups of females that breed alongside one another (i.e. patch size).
r_a	Relatedness between the individual with control of actor's phenotype and the actor itself
$\bar{r}_a$	Relatedness between the individual with control and the average individual that competes for mates alongside the actor.
r_{bf}	Relatedness between the individual with control and the actor's average mate.
$\bar{r}_{bf}$	Relatedness between the individual with control and the average female competitor of the actor's average mate.
r_{bm}	Relatedness between the individual with control and the average male mated to the actor's mate.
$\bar{r}_{bm}$	Relatedness between the individual with control and the average male mated to the average female that competes against the actor's mate.
$R_{i\to\bar{j}}$	Relatedness between a focal sex-i individual and the average sex-j individual born on the same patch.
$s_X(\delta)$	Competitive success of a mutant or an individual affected by a mutant's phenotype measured relative to that of a normal individual and for stage $X = a, b, c$ of the model life cycle.
u_i	Density of sex-i individuals at demographic equilibrium.
z	Normal (i.e wild-type) level of male phenotypic expression, that eventually describes male sperm-competition ability. Sometimes referred to as 'realized phenotype' (see Section 5).
z^*	Evolutionary equilibrium value of z.
$z^{*,i}$	$= z^*$, but emphasizes that sex i has control.
ζ^i	Sex-specific phenotype assumed to underlie determination of z when modelling the outcome of conflict.

a mutant male relative to that of a normal male, where δ denotes the extent of the mutant's phenotypic deviation. In other words, $s_a(\delta) = (\text{mutant success})/(\text{normal success})$, and so if δ is small the change in mating success of a mutant actor is approximately

$$\left.\begin{aligned}&(\text{mutant success}) - (\text{normal success})\\ &\quad = s_a(\delta)(Mu_f/u_m) - (Mu_f/u_m)\\ &\quad \approx (1 + \delta s'_a(0))(Mu_f/u_m) - (Mu_f/u_m)\\ &\quad = \delta k_a(z)(Mu_f/u_m),\end{aligned}\right\} \tag{1}$$

where $z > 0$ denotes the normal level of phenotypic expression and $k_a(z) = s'_a(0)$ (writing k_a as a function of z reminds us that, in general, $s'_a(0)$ will depend on the normal phenotype). We continue our calculation by assuming that each of the actor's mates enjoys normal competitive success $(1/u_f)$, and that the actor itself enjoys normal fertilization success $(1/M)$. Therefore, the actor changes the total number of offspring it produces by $\delta k_a(z) Mu_f/u_m \times 1/u_f \times 1/M = \delta k_a(z)/u_m$. If $c_m = 1$ is the reproductive value of the male contribution to offspring (Fisher, 1930; Price, 1970), then the change in actor's reproductive value is given by

$$\frac{c_m \delta k_a(z)}{u_m} = \frac{\delta k_a(z)}{u_m}. \tag{2}$$

The individual that controls the actor's level of phenotypic expression has a genetic interest in only a fraction, r_a, of the change in Eq. (2). Here, r_a is the coefficient of relatedness between the individual with control and the actor. The immediate change in the inclusive fitness of the individual with control of the actor's phenotype, then, is expressed as $r_a \delta k_a(z)/u_m$. Because reproductive value is conserved, the change given in Eq. (2) must be exactly compensated (Grafen and Archetti, 2008, see Discussion)—and when it is paid to (or paid by) a relative of the focal individual, this compensation must also appear in the inclusive fitness calculation. If $\bar{r}_a$ denotes the coefficient of relatedness between the individual with control and the actor's average competitor for mates, we can express the focal individual's 'genetic share' of the compensation as $\bar{r}_a \delta k_a(z)/u_m$, and we express the net change to the inclusive fitness of the individual with control as $\delta \Delta w_a$, where

$$\Delta w_a = (r_a - \bar{r}_a)\frac{k_a(z)}{u_m}. \tag{3}$$

2.3. Effect (b), female mates' competitive ability

Next, we consider what happens to the inclusive fitness of the individual with control when the deviant phenotype of the actor affects only its mate's ability to compete for the resources necessary for reproduction. As before, we interpret $1/u_f$ as the normal competitive success of a female. Given that δ/M represents the phenotypic deviation experienced by each of the actor's mates, we use $s_b(\delta/M)$ to denote the competitive success of female mated to a mutant (e.g. each of the actor's mates), relative to that of a normal female. In other words, $s_b(\delta/M) = (\text{mutant success})/(\text{normal success})$. We then approximate the change in the success enjoyed by each of the actor's mates as $(\text{mutant success}) - (\text{normal success}) \approx (\delta/M)s'_b(0)(1/u_f) = \delta k_b(z)/Mu_f$, where $k_b(z) = s'_b(0)$.

The change in female success affects a normal number of female mates (Mu_f/u_m, the number of mates secured by each normal actor) and their female competitors, altering the reproductive value of each by $c_f = (1 + h)c_m = (1 + h)$, where $h = 1$ for haplodiploids and $h = 0$ for diploids (Fisher, 1930; Price, 1970). Let r_{bf} and $\bar{r}_{bf}$ denote the coefficient of relatedness between the individual with control of the actor's phenotype and the actor's average mate and that average mate's (female) competitor, respectively. From the perspective of the individual with control, then, the inclusive fitness change is $\delta \Delta w_{bf}$, where

$$\begin{aligned}\Delta w_{bf} &= (r_{bf} - \bar{r}_{bf})c_f \frac{k_b(z)}{Mu_f}\frac{Mu_f}{u_m}\\ &= (r_{bf} - \bar{r}_{bf})\frac{k_b(z)(1+h)}{u_m}.\end{aligned} \tag{4}$$

The change in female success also affects a normal number of sperm competitors (all M males mated with each of the actor's Mu_f/u_m mates, for a total of M^2u_f/u_m males affected), as well as a normal number of males (M^2u_f/u_m) mated to female competitors. Each of these males enjoys normal fertilization success $(1/M)$ and, if successful in this regard, each changes its reproductive value by an amount $c_m = 1$. Let r_{bm} denote the coefficient of relatedness between the individual with control of the actor's phenotype and the average sperm competitor mated to the actor's mate (this may include relatedness to self, see Table 2), and let $\bar{r}_{bm}$ denote the coefficient of relatedness between the focal individual and the average male mated to the average female that competes against the actor's mate. Again, from the perspective of the individual with control, the inclusive fitness change is $\delta \Delta w_{bm}$, where

$$\begin{aligned}\Delta w_{bm} &= (r_{bm} - \bar{r}_{bm})c_m \frac{k_b(z)}{Mu_f}\frac{M^2u_f}{u_m}\frac{1}{M}\\ &= (r_{bm} - \bar{r}_{bm})\frac{k_b(z)}{u_m}.\end{aligned} \tag{5}$$

Table 2
Coefficients of relatedness introduced during inclusive-fitness model development, expressed in terms of the relatedness between offspring born on the same patch. We consider a life history in which all dispersal occurs prior to mating (DDM). We also consider a life history in which male dispersal occurs prior to mating and female dispersal occurs after mating (DMD). Detailed explanations of coefficients can be found in Appendix A. In all cases, an individual that disperses successfully competes in a randomly selected social group.

Life history	Coefficient	Male control, Δw^m	Female control, Δw^f
DDM	r_a	1	$K_m K_f R_{f\to\tilde{m}}$
	$\bar{r}_a$	$K_m^2 R_{m\to\tilde{m}}$	$K_m K_f R_{f\to\tilde{m}}$
	r_{bf}	$K_m K_f R_{m\to\tilde{f}}$	$K_f^2 R_{f\to\tilde{f}}$
	$\bar{r}_{bf}$	$K_m K_f R_{m\to\tilde{f}}$	$K_f^2 R_{f\to\tilde{f}}$
	r_{bm}	$r_a/M + \bar{r}_a(M-1)/M$	$K_m K_f R_{f\to\tilde{m}}$
	$\bar{r}_{bm}$	$K_m^2 R_{m\to\tilde{m}}$	$K_m K_f R_{f\to\tilde{m}}$
DMD	r_a	1	$K_m R_{f\to\tilde{m}}$
	$\bar{r}_a$	$K_m^2 R_{m\to\tilde{m}}$	$K_m R_{f\to\tilde{m}}$
	r_{bf}	$K_m R_{m\to\tilde{f}}$	$R_{f\to\tilde{f}}$
	$\bar{r}_{bf}$	$K_m K_f^2 R_{m\to\tilde{f}}$	$K_f^2 R_{f\to\tilde{f}}$
	r_{bm}	$r_a/M + \bar{r}_a(M-1)/M$	$K_m R_{f\to\tilde{m}}$
	$\bar{r}_{bm}$	$(K_m K_f)^2 R_{m\to\tilde{m}}$	$K_m K_f^2 R_{f\to\tilde{m}}$

2.4. Effect (c), male's sperm competition ability

Finally, we consider what happens to the inclusive fitness of the individual with control when the deviant phenotype of the actor affects only the actor's sperm competition ability. We interpret $1/M$ as normal fertilization success and we use $s_c(\delta)$ to denote the mutant fertilization success, measured relative to $1/M$. As before, $s_c(\delta) =$ (mutant success)/(normal success), and the change in fertilization success in this case is, (mutant success) − (normal success) $\approx \delta s_c'(0)(1/M) = \delta k_c(z)/M$, where $k_c(z) = s_c'(0)$.

The change in fertilization success arises in a normal number of matings (Mu_f/u_m). Since our calculations assume females involved in these matings enjoy normal competitive success ($1/u_f$) we express the change in reproductive value as, $(Mu_f/u_m)(1/u_f)c_m\delta k_c(z)/M = \delta k_c(z)/u_m$. Recall that r_a is the coefficient of relatedness between the actor and the individual with control, and r_{bm} is the coefficient of relatedness between the individual with control and the average sperm competitor mated to the actor's mate. Using these coefficients of relatedness, we express the change in the inclusive fitness of the individual with control as $\delta\Delta w_c$, where

$$\Delta w_c = (r_a - r_{bm})\frac{k_c(z)}{u_m}. \tag{6}$$

2.5. The inclusive fitness effect of a deviant actor's phenotype

To calculate the overall change in the inclusive fitness of the individual with control of the actor's phenotype we simply multiply each of the per-actor effects (a)–(c) by the number of actors (this number is proportional to u_m) and sum to get $\delta\Delta w$, where

$$\left.\begin{aligned}\Delta w &= \Delta w_a u_m + \Delta w_{bf} u_m + \Delta w_{bm} u_m + \Delta w_c u_m\\ &= (r_a - \bar{r}_a)k_a(z) + [(r_{bf} - \bar{r}_{bf})(1+h)\\ &\quad + (r_{bm} - \bar{r}_{bm})]k_b(z) + (r_a - r_{bm})k_c(z)\end{aligned}\right\}. \tag{7}$$

Note that we are justified in adding various inclusive-fitness effects in Eq. (7), because each has been recorded as a change in reproductive value—the 'common currency' of social evolutionary analysis (Frank, 1998).

It will often be necessary to be clear about which class of individual (male or female) is being assigned control of the male phenotype. When necessary, then, we will furnish Δw with a superscript $i = m$ (for male control), or $i = f$ (for female control). Thus, Δw^m will contain coefficients of relatedness that express the degree of kinship between individuals affected by the actor's behaviour and the actor itself, whereas Δw^f will contain coefficients of relatedness that express the degree of kinship between individuals affected by the actor's behaviour and the female with control.

2.6. The standard approach to analysis

The standard kin-selection analysis focuses on the sign of Δw, because it, in turn, reflects the sign of the selection gradient (Taylor, 1996). When Δw is positive, selection favours an increase in z, and when Δw is negative, selection favours a decrease in z. When Δw vanishes the population is at equilibrium with respect to the evolution of z. Therefore, if $z = z^*$ is the average level of phenotypic expression in a population at evolutionary equilibrium, then z^* satisfies the 'equilibrium condition',

$$\Delta w|_{z=z^*} = 0. \tag{8}$$

We can classify populations at evolutionary equilibrium according to the convergence stability of z^* (Christiansen, 1991). If z^* is convergence stable, then selection will tend to reduce the size of any small perturbation of the equilibrium population—a situation that is implied by the 'convergence stability condition',

$$\left.\frac{d\Delta w}{dz}\right|_{z=z^*} < 0. \tag{9}$$

(Taylor, 1989, 1996). Unfortunately, there is no analogue of condition (9) that implies Maynard Smith's (1982) well-known 'evolutionary stability' concept (=non-invasibility of z^*) holds; and so, we cannot use Δw to identify evolutionarily stable strategies or ESSs (Taylor, 1989). That said, the equilibrium condition (8) is still a necessary part of evolutionary stability (Taylor, 1989, 1996), and so any convergence-stable value of z^* we identify could be reasonably called a 'candidate ESS'.

The actual value taken by z^* may depend on which class of individual (male or female) is assumed to have control. Therefore we write $z^{*,i}$ to indicate when z^* is calculated from the sex-i perspective (i.e. calculated under the assumption of sex-i control).

It is important to note that selection does not always lead a population towards an equilibrium phenotype. Phenotypes on or near the boundary of a set can also be favoured in the long-term by selection. Phenotypes on or near the boundary of a set are favoured by selection when the direction of the selection gradient near said boundary points towards it. In a one-dimensional space, this means that $\Delta w < 0$ near the lower endpoint of a set, and $\Delta w > 0$ near the upper endpoint of a set.

2.7. A 'reference phenotype'

To make further analysis more straightforward we must decide what the phenotype z actually represents. Without loss of generality we can simply choose male phenotype to be any one of (a) mate-competition ability, (b) the effect a male has on competitive ability of its mates, or (c) sperm-competition ability as a 'reference phenotype'. In this case, we will assume that the male phenotype in question is actually sperm-competition ability. Thus, z represents normal (i.e. wild-type) sperm-competition ability, $s_c(\delta) = ((z+\delta)/Mz)/(1/M)$, and $k_c(z) = s_c(0) = 1/z$.

3. Population structure and life-history assumptions

To make progress we need to introduce assumptions about population structure and life-history. We assume that the population is arranged into social groups (i.e. patches) that consist of exactly N

inseminated females. We will also assume that a given social group is repopulated by a new set of N inseminated females each generation. These females may have been born locally or non-locally, and may have been inseminated by males born locally or non-locally. The kind of group structure described here is reflected natural populations where sexual conflict is known to occur [e.g. groups of water striders inhabit pools separated by riffles in streams (Krupa and Sih, 1993; Eldakar et al., 2009, 2010), bed bugs (Heteroptera: Cimidae), as their name suggests, aggregate in 'refugia' (Reinhardt and Siva-Jothy, 2007), and mating/oviposition in dung flies usually occurs in groups centred around animal droppings or carrion (Parker, 1972a,b; Eberhard, 1999; Parker and Pizzari, 2010)].

Continuing with our additional assumptions, we could suppose that males disperse between social groups prior to mating, while inseminated females disperse after mating. Alternatively, we might suppose that both males and females disperse prior to mating, so that mating occurs on the same site where offspring are produced. Taylor (1994) and Wild and Taylor (2004) have called the former situation the 'DMD life history' (males *D*isperse, then *M*ating occurs, then inseminated females *D*isperse, hence the acronym DMD), and have called the latter situation the 'DDM life history' (males *D*isperse and females *D*isperse, both before *M*ating occurs, hence the acronym DDM). Both DMD and DDM life histories are investigated below. Note that the situation where females disperse before mating and males disperse after mating is not of interest, here, since it can actually be thought of as a special case of DDM. Note also that although the model life histories are idealized (they are, after all, model life histories), they still provide a contrast between two biologically relevant extremes. As we will show, in the DMD scenario the various effects of male–male competition are separated by a major life-history event, namely female dispersal. No such separation occurs in the DDM scenario.

Appendix A reviews all assumptions about the DDM and DMD life histories, and presents an alternative approach to the inclusive-fitness model based on the approach developed by Taylor and Frank (1996). Appended results show that the alternative modelling approach and the inclusive-fitness approach that led to Eq. (7) yield equivalent mathematical descriptions of kin selection (see also Taylor et al., 2007).

Table 2 presents brief expressions for coefficients of relatedness under both the DDM and DMD life histories (see Appendix B for details of calculations) in terms of K_i, the probability that a sex-i individual found competing in a given group is native to that group, and $R_{i\to\tilde{j}}$, the relatedness between a sex-i individual with control of an actor's phenotype, and the average sex-j individual born in its same group. Even though relatedness sometimes accounts for an individual's genetic similarity to itself [what Pepper (2000) has termed 'whole group relatedness'], genetic similarity between different individuals [Pepper's (2000) 'others-only relatedness' and the similarity measure that appears in Hamilton's (1964) rule] contributes substantially to the size of coefficients in the scenarios we consider.

4. Individual-based simulation

To verify predictions of the inclusive-fitness model presented above, we constructed an individual-based simulation of both the DMD and DDM scenarios described above. Briefly, we simulate male–male competition for mates, and female–female competition for resources in a hierarchical manner, allowing competition to occur first among social groups, then among individuals within the winning group. Given the assumptions already stated above, there was no need to simulate male–male sperm competition in a hierarchical manner. Additional details and complete Matlab simulation code have been appended Appendix C, but the reader should note that simulations allow for large phenotypic differences among individuals (i.e. they permit selection that is not weak).

5. Modelling conflict and possible outcomes

To model conflict and its outcome we follow the approach pursued by Wild and Taylor (2005) and suppose that the realized male phenotype, z, is a weighted average of an underlying male phenotype, ζ^m, that is under male control (hence the superscript), and an underlying female phenotype, ζ^f, that is under female control. i.e., we suppose

$$z = \epsilon\zeta^m + (1-\epsilon)\zeta^f. \tag{10}$$

We will assume that, although males (resp. females) carry genes for ζ^f (resp. ζ^m), these genes remain silent. As an example, we might think of realized sperm-competition ability as the eventual result of how aggressively a male pursues its potential mate, and how receptive its potential mate is to being pursued. From a practical perspective, treating z as a weighted average of male and female phenotypes allows us to investigate possible power imbalances between the sexes (Birkhead and Parker, 1997).

Provided ζ^m and ζ^f can vary independently (an assumption we now make), the sign of Δw^i will be the same as the sign of the selection gradient acting on ζ^i. The sexes will be in conflict, then, when Δw^m and Δw^f have opposing signs, i.e. when the evolutionary interests of males and females do not coincide (Birkhead and Parker, 1997). The outcome of a conflict will be determined by the direction in which selection takes the population-average values of ζ^m and ζ^f, respectively.

6. Main results

6.1. Sex-specific timing of dispersal can drive conflict

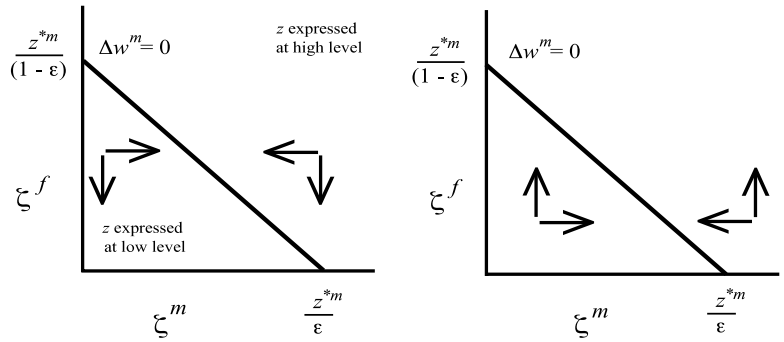
Substituting relatedness coefficients from Table 2 into Eq. (7), we find that, under DDM, females are indifferent to the level at which male phenotype is expressed ($\Delta w^f = 0$), so any inclusive-fitness change is negligible. We predict no conflict between male and female perspectives under DDM, then, because the sign of Δw^m and the sign of Δw^f do not oppose one another. We will discuss female indifference under DDM later, and turn much of our focus to the DMD life history where conflict between the sexes exists.

6.2. Effect on female's competitive ability, alone, can determine outcome of conflict

Male and female perspectives come into conflict under DMD. We find that, while the male perspective under DMD may be moderate (in the sense that the male may 'prefer' an equilibrium level of expression, z^{*m}), the female perspective under DMD is always extreme, provided $k_b'(z)$ does not change sign (an assumption we now make). When the control is given to a female, we have $\Delta w^f \propto k_b(z)$. Consequently, if a male's phenotype has a negative (resp. positive) effect on its mates' competitive ability, then a female always 'prefers' a reduction (resp. increase) in z.

The trade-off between pre- and post-copulatory male success suggests that selection in the male-control inclusive-fitness model will establish a convergence-stable equilibrium, z^{*m}. Fig. 2 depicts the coevolutionary outcome of this conflict in this case. When increased z harms female competitive ability, conflict occurs at relatively low levels of expression z, and selection on ζ^m and ζ^f promotes the male perspective (Fig. 2(a)). By contrast, when increased z helps female competitive ability, conflict occurs at relatively high levels of expression z, and selection on ζ^m and ζ^f promotes the female perspective (Fig. 2(b)).

The outcome of the conflict, here, is analogous to 'the loudest voice prevails' rule for genomic imprinting (Greenwood-Lee et al., 2001; Wilkins and Haig, 2003; Van Cleve et al., 2010). Since there is



(a) Female harmed $k_b(z) < 0$. (b) Female helped $k_b(z) > 0$.

Fig. 2. Outcome of sexual conflict over the realized level of phenotypic expression z. The outcome of the conflict is determined by the joint evolution of ζ^m and ζ^f, and arrows indicate the direction of the selection gradient for these underlying, sex-specific traits. In (a) we see that when a female is harmed by increased realized z sexual conflict occurs in the region below the selection nullcline, $\Delta w^m = 0$. Following the direction indicated by the arrows in (a) we see that the male perspective is promoted (selection favours a population in which ζ^m and ζ^f on the line $\Delta w^m = 0$). In (b) we see that when a female is helped by increased realized z sexual conflict occurs in the region above the selection nullcline, $\Delta w^m = 0$. Following the direction indicated by the arrows in (b) we see that the female perspective is promoted in the sense that z is expressed at ever-increasing levels over time.

a natural lower bound on z (namely, zero) the sex that favours the lower level of phenotypic expression can promote its perspective only so far. Since there is no clear upper bound on z the individual that favours the greater level of phenotypic expression (i.e. the 'louder voice') will always be able to promote its perspective, and so its perspective will eventually win out.

6.3. Conflict with linear competitive ability

If a male's ability to compete for mates and a female's ability to compete for reproductive resources, respectively, depend on z in a linear fashion, then $k_a(z)$ and $k_b(z)$ will take reasonably simple forms. When increasing z reduces a female's ability to compete we have

$$\text{female harmed: } k_a(z) = -\frac{1}{1-z} \quad \text{and} \quad k_b(z) = -\frac{1}{1-z}, \tag{11}$$

and when increasing z improves a female's ability to compete we have

$$\text{female helped: } k_a(z) = -\frac{1}{1-z} \quad \text{and} \quad k_b(z) = \frac{1}{z}. \tag{12}$$

Note that, here, we assume that z is both bounded below ($z > 0$) and above ($z < 1$); e.g., z might represent a proportion of some maximum competitive ability.

When increased realized z results in female harm ($k_b(z) < 0$, line (11)), we find the convergence-stable level of phenotypic expression from a male's perspective to be

$$z^{*m} = \frac{(r_a - r_{bm})}{(r_a - \bar{r}_a) + [(r_{bf} - \bar{r}_{bf})(1+h) + (r_{bm} - \bar{r}_{bm})] + (r_a - r_{bm})}. \tag{13}$$

When z improves female competitive ability ($k_b(z) > 0$, line (12)), the convergence-stable level from a male's perspective is

$$z^{*m} = \frac{[(r_{bf} - \bar{r}_{bf})(1+h) + (r_{bm} - \bar{r}_{bm})] + (r_a - r_{bm})}{(r_a - \bar{r}_a) + [(r_{bf} - \bar{r}_{bf})(1+h) + (r_{bm} - \bar{r}_{bm})] + (r_a - r_{bm})}. \tag{14}$$

We can interpret the denominator of Eqs. (13) and (14) as the total (positive) value the actor's genetic lineage places on the fitness changes that coincide with an increase in z. Continuing with this interpretation, both Eqs. (13) and (14) show that z^{*m} is the fraction of this total value that receives a net benefit from increased expression of the realized phenotype.

Fig. 3 presents Eqs. (13) and (14) for the DMD model (the model in which conflict exists) plotted as functions of M and N, for specific values of K_m and K_f. When female competitive ability is reduced by increased male phenotypic expression, we find that z^* increases (and so the extent of sexual conflict increases) with increasing M and N (Fig. 3(a) and (c)). When female competitive ability is improved by increased male phenotypic expression, we find that z^* decreases (and so the extent of sexual conflict increases, as before) with increasing M and N (Fig. 3(b) and (d)). Various combinations of K_m and K_f were investigated (nine pairs made by the set, 0.25, 0.5, 0.75) and all combinations revealed the same qualitative effect of increasing M and N (data not shown).

In the DMD scenario, we still have $\Delta w^f \propto k_b(z)$, so the female perspective is an extreme one: a female prefers either smallest possible z when the realized phenotype causes harm, or largest possible z when the realized phenotype helps. Again, when male and female perspectives conflict we see that the loudest voice prevails (Fig. 4).

6.4. Simulations support inclusive-fitness predictions

We found remarkably close agreement between results of simulations that adopted the male perspective, and results of the inclusive-fitness analysis, even though the key assumption of small δ has been violated in the simulation (Fig. 5). Although there was qualitative agreement between simulations that adopted the female perspective, and the inclusive-fitness analysis, some discrepancy between the two is clear. In Fig. 6(a) and (b) we see that simulations approached, but failed to reach, the boundary phenotype (zero or one) predicted by the inclusive-fitness analysis ($z = 1$ in the case of Fig. 6(a), and $z = 0$ in the case of Fig. 6(b)). The discrepancy, in this case, is likely due to the fact that the simulation incorporates both mutation and a feature that prevents phenotypes from leaving a biologically feasible set; thus boundary phenotypes reflect rather than absorb evolutionary trajectories. Finally, the simulation depicted in Fig. 6(c) supports the prediction of female indifference under a DDM life history, made above, since no clear equilibrium is established even after 5000 simulated generations.

7. Discussion

7.1. Inclusive fitness and sexual conflict

This paper builds on recent work that demonstrates kin selection/group structure can play an important role in the resolution of sexual conflict (Eldakar et al., 2009, 2010; Rankin, 2011). Here, we explicitly explore conflict over the expression of a single phenotype that affects pre- and post-copulatory competitive

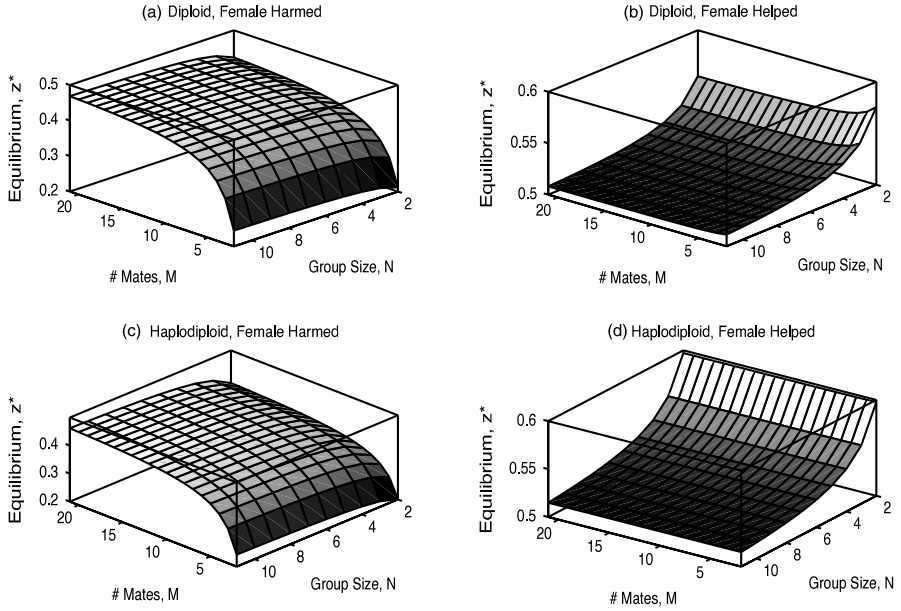
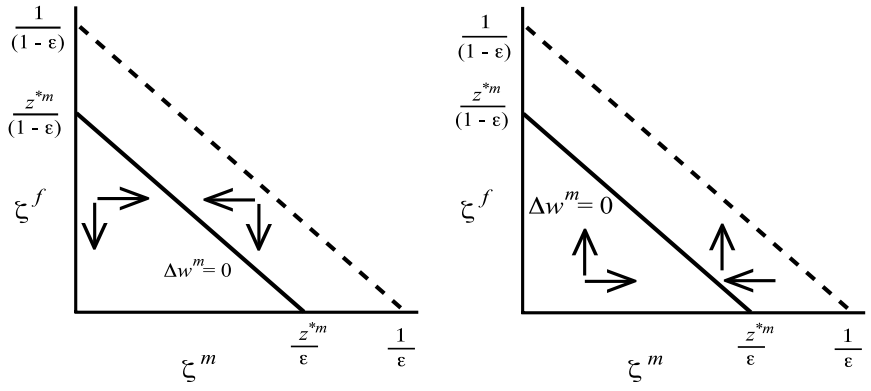


Fig. 3. Relationship between the convergent-stable, equilibrium level of male phenotypic expression (z^{*m}) in the DMD model, number of mates per female (M), and patch size (N). Here, $K_m = 0.25$ and $K_f = 0.5$, for both diploid (a and b) and haplodiploid (c and d) species. In panels (a) and (c) increased z decreases both competitive ability of males and females, $k_a(z) = k_b(z) = -1/(1-z)$. In (b) and (d) increased z decreases the competitive ability of males, but increases that of females, $k_a(z) = -1/(1-z)$, $k_b(z) = 1/z$.



(a) Female harmed $k_b(z) < 0$. (b) Female helped $k_b(z) > 0$.

Fig. 4. Outcome of sexual conflict over the realized level of phenotypic expression z, when this affects male and female competitive ability in a linear fashion. The outcome of the conflict is determined by the joint evolution of ζ^m and ζ^f in the set $\epsilon\zeta^m + (1-\epsilon)\zeta^f \leq 1$. Arrows again indicate the direction of the selection gradient for the underlying, sex-specific traits. In (a) we see that when a female is harmed by increased realized z sexual conflict occurs in the region below the selection nullcline, $\Delta w^m = 0$. Following the direction indicated by the arrows in (a) we see that the male perspective is promoted (selection favours a population in which ζ^m and ζ^f on the line $\Delta w^m = 0$). In (b) we see that when a female is helped by increased realized z sexual conflict occurs in the region above the selection nullcline, $\Delta w^m = 0$. Following the direction indicated by the arrows in (b) we see that the female perspective is promoted in the sense that z tends towards its maximum value of one, over time.

success of males. In our model, the male phenotype in question could result in harm to a female; however, we also consider the possibility that females gain a fitness advantage with increased male phenotypic expression.

Like Rankin (2011), we conclude that changes in relatedness can affect the disparity between the perspectives that frame a sexual conflict. Specifically, given assumptions about marginal costs and benefits (lines (11) and (12)), we find that the stable level of investment in sperm-competition ability (z) reflects the value the focal individual places on those components of its inclusive fitness that benefit from increasing z (as a fraction of total value of all effects). Like Rankin (2011) we also show that the female perspective is unaffected by changes in relatedness; in other words, changes in the disparity between perspectives are due solely to changes in the z^{*m}.

Our results highlight the fact that the existence of sexual conflict is not guaranteed. In particular, given biologically plausible life histories (DDM life histories), a female is predicted to be indifferent to male phenotypic expression, even when such expression reduces the females competitive ability. Where conflict does exist we find that it is the perspective that favours the greater investment in sperm-competition ability (z) that wins out. In other words, 'the loudest voice prevails'.

7.2. Female indifference under DDM

At first glance, female indifference to the level at which male phenotype is expressed in the DDM model might seem puzzling, especially in light of the lack of indifference predicted to occur among females in the DMD model, and by males in general. However, like those of numerous social-evolutionary studies, our results can be understood by considering the scale at which competition occurs (Bulmer and Taylor, 1980; Taylor, 1992; Queller, 1994; Frank, 1998; West et al., 2001, 2002; Griffin

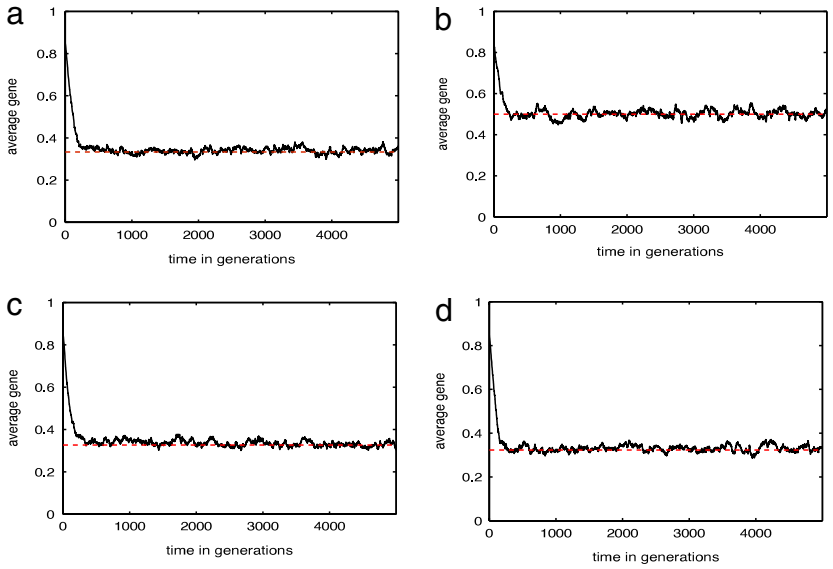


Fig. 5. Results from sample simulations of the evolution of male phenotype when the male perspective is adopted (i.e. when male has control of its own phenotype). In the simulation genes take a value between zero and one, and this value is then used to determine phenotype. We have plotted, here, the value taken by the mean-average gene in the population (the 'average gene') over the course of 5000 simulated generations (solid black line), and predictions generated by the inclusive fitness analysis for comparison (dashed red line, colour online). Panel (a) shows a simulated diploid, DDM life history with $N = 4$, $M = 3$, $Km = 0.5$, $Kf = 0.75$, and $k_b(z) = -1/(1-z)$. Panel (b) shows a simulated haplodiploid DDM life history with $N = 4$, $M = 3$, $K_m = K_f = 0.5$ and $k_b = 1/z$. Panels (c) and (d) show simulated diploid and haplodiploid DMD life histories, respectively, with $N = 4$, $M = 3$, $K_m = 0.5$, $K_f = 0.75$, and $k_b(z) = -1/(1-z)$. In all cases a close correspondence between simulation results and inclusive-fitness based predictions is evident.

et al., 2004; Wild and Taylor, 2004; Gardner and West, 2004, 2006; Gardner et al., 2004; Grafen and Archetti, 2008; Pizzari and Foster, 2008; Kümmerli et al., 2009; Taylor et al., 2011).

To understand why the scale of competition is important here, one needs to recognize first that, at a fundamental level, individuals compete against one another for genetic representation in generations far into the future. In other words, individuals compete for reproductive value. Since we can regard the total reproductive value of the population as being fixed (no individual can be ancestor to more than 100% of a descendant population), an increase in the reproductive value of any individual must be compensated by a decrease in the reproductive value of another. Similarly, a decrease in the reproductive value of any individual will be compensated by a increase in the reproductive value of another (see Grafen and Archetti, 2008). The scale of competition is important, then, because it determines where (or rather by whom) compensation is made. When competition occurs on a global scale, the genetic relatives of the primary recipient rarely compensate for its fitness gains or losses (ultimately, its increase or decrease in reproductive value). When competition occurs on a local scale, though, compensation by kin is quite probable, and this can mean that those fitness changes we expect to observe 'unexpectedly' cancel each other out in the overall analysis (Taylor, 1992; Kümmerli et al., 2009).

Turning back to the results of this study, we see that female indifference in the DDM scenario is due to similar kinds of 'unexpected' cancellations. Under DDM, the scale at which competition occurs means that every fitness gain made by either the actor or the actor's mate is compensated by a fitness change in an individual whose value to the genetic lineage of the female assigned control is identical. For example, (i) the actor that improves its own chances of securing a mate in part (a) of the life cycle, and (ii) the male that actor will displace are, from the perspective of a female, both simply males competing against one another on the same patch; moreover, they are males found on the same patch that she will later find herself, provided she is competitively successful ($r_a = \bar{r}_a = K_m K_f R_{f\to\bar{m}}$, see Table 2). As we see in Table 2, similar 'unexpected' cancellation does not occur when we adopt the male perspective under DDM, simply because the relatedness between the focal individual and the owner of primary fitness change differs from that between the focal individual and the individual that compensates for the primary change. Consequently, the male ends up with a 'preferred' level phenotypic expression.

7.3. The effect of pre- and post-copulatory fitness trade-offs

In the DMD scenario, lack of female indifference is due to the fact that pre-copulatory and post-copulatory fitness changes are separated by female dispersal. Although kin are again involved in compensating for the personal fitness gains made by the actor and its mates, the compensation is of lesser value to the genetic lineage of the female assigned control when it occurs among social groups established after female dispersal. For example, under DMD we are sure that the actor's mate and the focal female were born on the same patch, but there is a $<100\%$ chance the same is true for the female that is displaced by the actor's mate ($r_{bf} = R_{f\to\bar{f}}$, but $\bar{r}_{bf} = K_f^2 R_{f\to\bar{m}}$, see Table 2). Simply put, the female's genetic lineage under has some net stake in the expression of male phenotype under DMD.

We find that compensation made by social groups established prior to female dispersal in the DMD model [the group of mate competitors in part (a) of the life cycle, and the group of sperm competitors in part (c) of the life cycle] is no more or less valuable to a female's lineage than the personal fitness gains these groups accommodate. As a result, some 'unexpected' cancellations continue to occur in the DMD model when the female perspective is adopted. These cancellations correspond to fitness changes that occur at points in the life cycle where the trade-off between pre- and post-copulatory male competitive ability becomes relevant [points (a) and (c) it the life cycle], and so the trade-off, itself, effectively vanishes from the females inclusive fitness.

Of course changes in the scale of competition also underlie the differences we observed in the male perspective as we

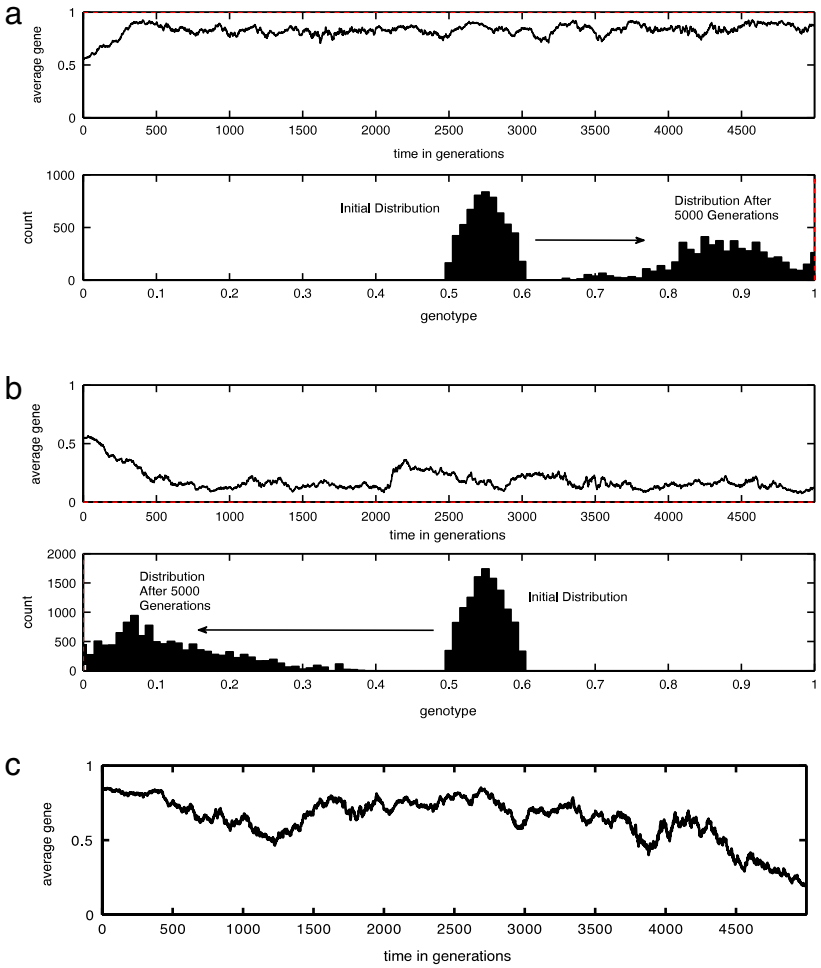


Fig. 6. Results from sample simulations of the evolution of female phenotype when the female perspective is adopted (i.e. when male phenotype is determined by the genotype of its average potential mate). In the simulation genes take a value between zero and one, and this value is then used to determine phenotype. We have plotted, here, the value taken by the mean-average gene in the population (the 'average gene') over the course of 5000 simulated generations (solid black line), and, when appropriate, predictions generated by the inclusive fitness analysis for comparison (dashed red line—colour online). Panel (a) shows a simulated haplodiploid, DMD life history with $N = 2, M = 3, K_m = 0.75, K_f = 0.1$, and $k_b(z) = 1/z$. Panel (b) also shows a simulated haplodiploid, DMD life history, but with $N = 3, M = 4, K_m = 0.75, K_f = 0.2$, and $k_b(z) = -1/(1-z)$. Panels (a) and (b) also compare the initial distribution of genotypes in the population to the distribution observed after 5000 simulated generations. Panel (c) shows a simulated diploid, DDM life history, with $N = 3, M = 3, K_m = 0.75, K_f = 0.1$, and $k_b(z) = 1/z$.

moved from the DDM model to the DMD model. Clearly, then, we would agree with Rankin's (2011) suggestion that the scale at which competition occurs is relevant to sexual conflict discourse. However, we emphasize that when male–male (or indeed female–female) competition occurs at different points in time (e.g. both before and after copulation), the appropriate scale of competition will, in general, shift from time-point to time point. Consequently, we expect theoretical predictions about sexual conflict to be sensitive to the timing of major life-history events like dispersal.

The fact that the timing of life-history events affects the possibility of sexual conflict suggests that existing empirical work could be expanded to include more detailed life-history descriptions. Work with natural populations of water striders (*A. remigis*), for example, has emphasized the role of female dispersal as a mechanism for avoiding male aggression, but has paid less attention to how (or if) dispersal events precede or follow oviposition (e.g. Eldakar et al., 2010). A recent review of bed-bug biology points to a similar gap in our knowledge of the dispersal ecology of these species (see Reinhardt and Siva-Jothy, 2007). Since female bed bugs store sperm for more than a month (Reinhardt and Siva-Jothy, 2007), it is reasonable to expect that dispersal by inseminated females often precedes oviposition in this species. If this is indeed the case, then the DMD model could inform discussions about the adaptive significance of male harm in bed bugs as well—that is, provided empirical studies of the dispersal ecology of bed bugs find DMD to be a reasonable approximation.

Certainly, the models above could be applied to understand the effect of kin selection (or group structure) on the evolution of male harm in water striders and bed bugs. However, it appears that our predictions are already well supported by observations of the dung flies *Sepsis cynipsea* and *S. neocynipsea* (Diptera: Sepsidae). In fact, sepsid life histories reflect many of the main assumptions of the DMD model. First, mating and oviposition in sepsid dung flies typically occurs on or near a pile of animal droppings or carrion (as explained in the previous section) (Parker, 1972a; Eberhard, 1999). This matches our basic assumptions about discrete group structure above.

Second, males engage one another in pre-copulatory struggles that ultimately determine success in gaining access to females in a way that, again, matches assumptions above. Male dung flies engage in violent territorial fights in which they grapple with their fore legs (Parker, 1972a; Eberhard, 1999). Male dung flies will also attempt to displace rivals that have grasped, but not copulated with a female (Parker, 1972a; Ward et al., 1992). Fore-leg strength continues to play a role in competitive success here, as it is the

fore-legs that are responsible for maintaining a hold on a female that withstands attacks from other males, and the evasive manoeuvres of unreceptive females (Ward et al., 1992, see below).

Third, the observations that fresh animal droppings are colonized by both males searching for mates and ovipositing females (Parker, 1972a), along with more recent evidence of sexual behaviour around female feeding sites on plants (Eberhard, 1999) suggests (i) that mating by dung flies does not necessarily occur on the same site as oviposition, (ii) some amount of male dispersal precedes mating, and (iii) that mating and oviposition in sepsid flies is likely separated by the dispersal of inseminated females. Although, the actual the sex-specific timing of the dispersal in dung flies does not match the DMD model exactly, the model itself seems to provide a reasonable caricature of sepsid life history.

Fourth, there is evidence to suggest that post-copulatory competition among males (sperm competition) in dung flies occurs. Female dung flies store sperm, effectively setting the stage for sperm competition (Ward et al., 1992). Sperm competition is, in turn, reflected in the elaborate and complex morphology of male genitals (Ward et al., 1992). Since one unit of energy used to grow/strengthen fore legs (body parts involved in much pre-copulatory male–male competition) is one unit of energy that cannot be allocated to genital growth/development (body part that plays a key role in post-copulatory male–male competition), the assumed the trade off between pre- and post-copulatory competitive success among males is certainly plausible.

Given, that the assumptions of the model match, in a coarse-grained way, the biology of *S. cynipsea* and *S. neocynipsea*, and given the fact that male phenotypes do inflict harm at the time of copulation [females suffer wounds inflicted by a males elaborate genitals (Blanckenhorn et al., 2002)], it is not surprising that females often actively reject a male's attempt at copulation with vigorous shaking [only 40% of male–female pairs actually copulate (Ward et al., 1992)]. In fact, a selective advantage for female reluctance is a main prediction of our DMD inclusive-fitness model. While the DMD model likely fails to capture all aspects of sepsid biology, the model does suggest that kin selection (or group structure) is able to mediate sexual conflict in these species of fly.

7.4. Future theoretical work

We have assumed, here, that each female reproduces only once during its life. In some instances of sexual conflict, though, male phenotypes increase female fecundity in the short-term, and could affect residual female fitness through more complicated female life-history trade-offs (Parker and Pizzari, 2010, and references therein). In general, we suggest that the development of kin-selection models that incorporate longer-term effects on female fitness will be important to developing our understanding of sexual conflict.

We have also assumed, here, that male fertilization success is independent of the order in which mating occurs. However, mating order is known to affect a male's probability of siring offspring (Garefalaki et al., 2010; Raveh et al., 2010, are two recent examples). The effects of mating order could also be investigated, provided one understands how such effects impact male reproductive value.

Given the central role played by compensation it would also be interesting to investigate how our predictions change when local populations can expand or contract to accommodate changes in fitness. While compensation in these 'elastic' populations continues to occur, elasticity itself changes the way in which compensatory fitness changes factor into the inclusive fitness method of accounting (e.g. Alizon and Taylor, 2008).

It is important to note that the models presented above do not include an investigation of the relationship between the sex ratio the extent of male harm. Observations of water striders suggest that skewed sex ratios could represent an additional cost of aggressive male behaviours, as harassed females tend to leave areas with aggressive males for areas where aggression levels are lower (Eldakar et al., 2010). Conditional female movement would not change our models' predictions: the discrete nature of our model life histories and the fact that competition is entirely intrasexual means that the sex ratio cancels out of our calculations. In fact, the lack of relationship between the sex ratio and level of male harm we predict is consistent with laboratory investigations of Drosophila (Wigby and Chapman, 2004). That said, a continuous-time model in which females alter their dispersal rate in response to a varying number of local males (and, by extension, a varying level of local male harm) could, in principle, be used to model the observations of Eldakar et al. (2010), but such an extension is beyond the scope of the present work.

Like the sex ratio, inbreeding depression is thought to have important consequences for sexual conflict (Gay et al., 2011). In the context of our inclusive-fitness model, though, the effects of inbreeding depression would only affect the size of relatedness coefficients and would not change our qualitative conclusions. An increase/decrease in the expression of a harmful male phenotype would not result in more/less inbreeding, because the expression itself would not be preferentially directed towards relatives. Conversely, more/less inbreeding would not change the number of inclusive-fitness costs and benefits under consideration, rather it would just modify the sizes of those that already exist in our calculation.

Acknowledgements

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Appendix A. Direct-fitness model formulation

In this appendix we show how to arrive at Eq. (7) of the main text using the direct-fitness approach of Taylor and Frank (1996), and using the specific assumptions about the DDM and DMD life histories. Recall that both life histories assume a group-structured population in which inseminated females are organized into a large number of habitat patches (i.e. social groups) of identical size and quality.

A.1. DDM life history

Each generation of the DDM model consists of the following series of events, presented in order.

(i) *Natal dispersal*. Each female and each male offspring disperses from its natal patch independently with probability d_f and d_m, respectively. A dispersed offspring fails to find a new patch on which to compete with probability μ, in which case it dies. We make the standard assumption that, should a focal offspring find a new patch on which to compete, that patch will not support any relatives of the focal individual.

(ii) *Male–male competition for mates*. Each female chooses exactly M mates from among the group of males found on its current patch, storing a large amount of sperm from each mate for later use. Males are chosen at random, with replacement, according to their 'competitive weight', $w_m(y) > 0$, where y is a 'male phenotype' (recall our 'reference' male phenotype is sperm-competition ability). We assume $w'_m(y) < 0$, thus the

expression of a male's sperm-competition ability is increased at the expense of its ability to compete for mates. Once all mating has finished, we assume that all males die.

(iii) *Female–female competition for breeding sites*. Each female competes for one of the N breeding sites available on its current patch. Competition among females occurs at random, again according to a 'competitive weight' $w_f(\bar{y}_\times)$ where $\bar{y}_\times$ is some average of male mates' phenotypes (details below). We will assume either $w_f'(\bar{y}_\times) < 0$ or $w_f'(\bar{y}_\times) > 0$, depending on whether we wish to investigate the possibility that increased intensity of male sperm competition has positive (former case) or negative (latter case) implications for female competitive ability. After female–female competition has finished all unsuccessful females are assumed to die.

(iv) *Birth of offspring and sperm competition*. Each female that has secured a breeding site produces a large number of offspring, κ. A fraction of each brood, α is made up of sons, and the remaining fraction, $(1-\alpha)$, is made up of daughters. A male with phenotype y is assumed to successfully father each offspring, independently, with probability $y/M\bar{y}_\times$.

A.1.1. Components of male fitness

Fix attention on one patch (the 'focal patch') and one male on that patch (the 'focal male') just prior to stage (i) of the DDM life history described above. With the focal male and the focal patch in mind, we introduce the following notation:

- y, the phenotype of the focal male;
- $\bar{y}$, the phenotype of the average male born on the focal patch at stage (i) above;
- z, the phenotype of the average male in the population at stage (i) above;
- $z_a = [(1-d_m)\bar{y} + d_m(1-\mu)z]/(1-\mu d_m)$, the phenotype of the average stage-(ii) competitor of the focal male when it competes on its natal patch. We use the subscript 'a', here, to correspond with the use of this subscript in the main text.
- $z_b^L = y/M + z_a(M-1)/M$, the phenotype of the average male that inseminated a female that had, in turn, been inseminated by the focal male on the focal patch (i.e. locally, hence the superscript 'L'). Note that the weak selection or small δ assumption introduced in the main text allows us to define z_b^L using an assumption of selective neutrality.
- $z_b^{NL} = y/M + z(M-1)/M$, the phenotype of the average male that inseminated a female that had, in turn, been inseminated by the focal male on a patch other than focal patch (i.e. non-locally, hence the superscript 'NL').

With notation now defined, we are in a position to develop mathematical expressions for the fitness of the focal male in terms of the variables above.

With probability $(1-d_m)$ the focal male does not disperse from its natal patch. In that case, the competitive ability of the focal male, $w_m(y)$, must be measured relative to, $N\kappa\alpha(1-\mu d_m)w_m(z_a)$, the total competitive effort put forth by males on the focal patch at stage (ii) above. [Although it may appear that we need w_m to be linear for this calculation to hold, our assumption of weak selection means that even non-linear w_m can be treated as if they are linear.] It follows that, when competing on its natal patch, the focal male wins each mating with probability $w_m(y)/N\kappa\alpha(1-\mu d_m)w_m(z_a)$. Since there are $MN\kappa(1-\alpha)(1-\mu d_f)$ mating opportunities on the focal patch, the focal male, itself, expects

$$\mathrm{E}\{\#\text{matings} \mid \text{no male dispersal}\} = \frac{1-\alpha}{\alpha}\frac{M(1-\mu d_f)w_m(y)}{(1-\mu d_m)w_m(z_a)} \tag{A.1}$$

local inseminations.

With probability $d_m(1-\mu)$ the focal male successfully disperses from its natal patch to a new one. In this case, $w_m(y)$ is measured relative to $N\kappa\alpha(1-\mu d_m)w_m(z)$. There are again $MN\kappa(1-\alpha)(1-\mu d_f)$ mating opportunities on the new, non-focal patch, so the focal male expects

$$\mathrm{E}\{\#\text{matings} \mid \text{male dispersal}\} = \frac{1-\alpha}{\alpha}\frac{M(1-\mu d_f)w_m(y)}{(1-\mu d_m)w_m(z)} \tag{A.2}$$

non-local inseminations.

Success described by Eqs. (A.1) and (A.2) is diminished by competition among females during stage (iii) of the DDM life history. When the focal male has not dispersed, the competitive ability of each of its mates is $w_f(z_b^L)$, and their ability is measured relative to $N\kappa(1-\alpha)(1-\mu d_f)w_f(z_a)$, the total competitive ability of females inseminated by males competing on the focal patch (weak selection justifies the use of z_a as the argument of w_f in this total). It follows that each female inseminated by the focal male on the focal patch wins one of the N available sites with probability

$$\Pr\{\text{female success} \mid \text{no male dispersal}\} = \frac{w_f(z_b^L)}{\kappa(1-\alpha)(1-\mu d_f)w_f(z_a)}. \tag{A.3}$$

When the focal male has dispersed, the competitive ability of each of its mates, now $w_f(z_b^{NL})$, is measured relative to $N\kappa(1-\alpha)(1-\mu d_f)w_f(z)$. In this case, each female inseminated by the focal male on the non-focal patch wins one of the N sites for which it competes with probability

$$\Pr\{\text{female success} \mid \text{male dispersal}\} = \frac{w_f(z_b^{NL})}{\kappa(1-\alpha)(1-\mu d_f)w_f(z)}. \tag{A.4}$$

After stage (iv), the focal male that does not disperse expects to be credited with

$$\mathrm{E}\{\#\text{sons per mate} \mid \text{no male dispersal}\} = \frac{\kappa\alpha y}{Mz_b^L} \tag{A.5}$$

sons, and

$$\mathrm{E}\{\#\text{daughters per mate} \mid \text{no male dispersal}\} = \frac{\kappa(1-\alpha)y}{Mz_b^L} \tag{A.6}$$

daughters for every one of its competitively successful mates. When the focal male does disperse, it expects to be credited with

$$\mathrm{E}\{\#\text{sons per mate} \mid \text{male dispersal}\} = \frac{\kappa\alpha y}{Mz_b^{NL}} \tag{A.7}$$

sons, and

$$\mathrm{E}\{\#\text{daughters per mate} \mid \text{male dispersal}\} = \frac{\kappa(1-\alpha)y}{Mz_b^{NL}} \tag{A.8}$$

daughters for every one of its competitively successful mates.

We now combine Eqs. (A.1)–(A.8) to produce expressions for the male and female components of male fitness, respectively. Let a_{ij} denote the expected number of sex-i offspring ($i = m, f$) produced by a sex-j focal individual ($j = m, f$), weighted by genetic contribution to the offspring in question. Since we have been considering a focal male so far, we are immediately interested in deriving a_{mm} and a_{fm} for both diploid ($h = 0$) and haplodiploid ($h = 1$) species.

We can express fitness components in words as,

$$\begin{aligned} a_{mm} = {} & (1/2)(1-h)\Pr\{\text{no male dispersal}\} \\ & \times \mathrm{E}\{\#\text{matings} \mid \text{no male dispersal}\} \\ & \times \Pr\{\text{female success} \mid \text{no male dispersal}\} \end{aligned}$$

$$
\begin{aligned}
&\times \mathrm{E}\{\#\text{sons per mate} \mid \text{no male dispersal}\}\\
&+ (1/2)(1-h)\,\Pr\{\text{male dispersal}\}\\
&\times \mathrm{E}\{\#\text{matings} \mid \text{male dispersal}\}\\
&\times \Pr\{\text{female success} \mid \text{male dispersal}\}\\
&\times \mathrm{E}\{\#\text{sons per mate} \mid \text{male dispersal}\},
\end{aligned}
$$

and

$$
\begin{aligned}
a_{fm} = {} & (1/2)\,\Pr\{\text{no male dispersal}\}\\
&\times \mathrm{E}\{\#\text{matings} \mid \text{no male dispersal}\}\\
&\times \Pr\{\text{female success} \mid \text{no male dispersal}\}\\
&\times \mathrm{E}\{\#\text{daughters per mate} \mid \text{no male dispersal}\}\\
&+ (1/2)\,\Pr\{\text{male dispersal}\}\\
&\times \mathrm{E}\{\#\text{matings} \mid \text{male dispersal}\}\\
&\times \Pr\{\text{female success} \mid \text{male dispersal}\}\\
&\times \mathrm{E}\{\#\text{daughters per mate} \mid \text{male dispersal}\}.
\end{aligned}
$$

Alternatively, using K_i from the main text in place of the expression $(1-d_i)/(1-\mu d_i)$ we can write

$$
\left.
\begin{aligned}
a_{mm} = {} & \frac{(1-h)K_m}{2}\frac{w_m(y)}{w_m(z_a)}\frac{w_f(z_b^L)}{w_f(z_a)}\frac{y}{z_b^L}\\
&+ \frac{(1-h)(1-K_m)}{2}\frac{w_m(y)}{w_m(z)}\frac{w_f(z_b^{NL})}{w_f(z)}\frac{y}{z_b^{NL}},\\
a_{fm} = {} & \frac{K_m}{2}\frac{1-\alpha}{\alpha}\frac{w_m(y)}{w_m(z_a)}\frac{w_f(z_b^L)}{w_f(z_a)}\frac{y}{z_b^L}\\
&+ \frac{1-K_m}{2}\frac{1-\alpha}{\alpha}\frac{w_m(y)}{w_m(z)}\frac{w_f(z_b^{NL})}{w_f(z)}\frac{y}{z_b^{NL}}
\end{aligned}
\right\}. \tag{A.9}
$$

A.1.2. Components of female fitness

Now fix attention on one patch, and one *female* on that patch (the 'focal female') just prior to the start of stage (i) of the DDM life history. Regardless of whether it disperses or not, the focal female chooses its mates in the same way and from the same pool as its competitors. It follows that the focal female expects to secure one of the N sites available on the patch on which it competes with probability

$$
\left.
\begin{aligned}
\Pr\{\text{female success}\} = {} & \frac{1-d_m}{\kappa(1-\alpha)(1-\mu d_f)}\\
&+ \frac{(1-\mu)d_m}{\kappa(1-\alpha)(1-\mu d_f)}\\
= {} & \frac{1}{\kappa(1-\alpha)}
\end{aligned}
\right\}. \tag{A.10}
$$

Once a site is secured, the focal female produces

$$
\mathrm{E}\{\#\text{sons} \mid \text{success}\} = \kappa\alpha \tag{A.11}
$$

sons, and

$$
\mathrm{E}\{\#\text{sons} \mid \text{success}\} = \kappa(1-\alpha) \tag{A.12}
$$

daughters. For the focal female, then, we have

$$
\left.
\begin{aligned}
a_{mf} = {} & \Pr\{\text{female success}\}\mathrm{E}\{\#\text{sons} \mid \text{success}\}\\
= {} & \frac{(1+h)}{2}\frac{\alpha}{1-\alpha},\\
a_{ff} = {} & \Pr\{\text{female success}\}\mathrm{E}\{\#\text{daughters} \mid \text{success}\}\\
= {} & \frac{1}{2}
\end{aligned}
\right\}. \tag{A.13}
$$

A.1.3. Combining components of fitness

We combine the various components of fitness, above, using neutral measures of individual reproductive value and the neutral distribution of sexes (Taylor and Frank, 1996). In the absence of selection the sex-specific components of fitness can be stored in the 2×2 matrix,

$$
\begin{aligned}
\mathbf{A}^{\circ} &= \begin{bmatrix} a_{mm}^{\circ} & a_{mf}^{\circ} \\ a_{fm}^{\circ} & a_{ff}^{\circ} \end{bmatrix}\\
&= \frac{1}{2}\begin{bmatrix} 1-h & (1+h)\alpha/(1-\alpha) \\ (1-\alpha)/\alpha & 1 \end{bmatrix}
\end{aligned} \tag{A.14}
$$

where the superscript '$\circ$' is a reminder that we have set all phenotypes equal to the population average value, z.

It is easy to check that the dominant eigenvalue of $\mathbf{A}^{\circ}$ is equal to one, and so a monomorphic model population is neither growing nor shrinking in the long-run, as expected. It is also easy to check that the dominant right eigenvector of $\mathbf{A}^{\circ}$ is,

$$
\mathbf{u} = \begin{bmatrix} u_m \\ u_f \end{bmatrix} = \begin{bmatrix} \alpha \\ 1-\alpha \end{bmatrix}. \tag{A.15}
$$

The entries of $\mathbf{u}$ give the frequency with which we encounter males and females, respectively, at the very beginning of stage (i) of the DDM life history.

The dominant left eigenvector of $\mathbf{A}^{\circ}$ is

$$
\mathbf{v} = \begin{bmatrix} v_m \\ v_f \end{bmatrix} = \begin{bmatrix} 1/\alpha \\ (1+h)/(1-\alpha) \end{bmatrix}. \tag{A.16}
$$

The entries of $\mathbf{v}$ are proportional to the reproductive value of an individual male and female, respectively, in a neutral population at the beginning of stage (i) of the DDM life history. The novice may find it useful to observe that the reproductive value of a sex-i parent, defined as c_i in the main text, is the total reproductive value of its offspring weighted by parental genetic contribution. In a neutral population, a male parent is credited with $1/M$ offspring from each of M broods, and a female is credited with all offspring from one brood, thus for the same constant of proportionality

$$
\left.
\begin{aligned}
c_m &\propto \alpha v_m(1-h)/2 + (1-\alpha)v_f/2 = 1,\\
c_f &\propto \alpha v_m(1+h)/2 + (1-\alpha)v_f/2 = 1+h,
\end{aligned}
\right\} \tag{A.17}
$$

as noted in the main text.

A.1.4. Expressions for recipient fitness

Following Taylor and Frank (1996) we can define the fitness of a focal male (in social evolutionary parlance, a 'male recipient') and a focal female (a 'female recipient') as

$$
\left.
\begin{aligned}
W_m &= (v_m a_{mm} + v_f a_{fm})/v_m,\\
W_f &= (v_m a_{mf} + v_f a_{ff})/v_f,
\end{aligned}
\right\} \tag{A.18}
$$

respectively.

A.2. DMD life history

Each generation of the DMD model consists of its own series of events. These are presented in very briefly (in order) as many of the details are not changed from the DDM case.

(i) *Natal dispersal of males*. As described above.
(ii) *Male–male competition for mates*. As described above.
(iii) *Dispersal of inseminated females*. As described above, except now when a female disperses, it carries with it the sperm collected from M males.
(iv) *Female–female competition for breeding sites*. As described above.
(v) *Birth of offspring and sperm competition*. As described above.

A.2.1. Components of male fitness

We again fix attention on one patch and one male born on that patch. The expected number of mates won by the focal male, conditioned upon its dispersal status are given now by

$$\mathrm{E}\{\#\text{matings} \mid \text{no male dispersal}\}, = \frac{1-\alpha}{\alpha}\frac{M w_m(y)}{(1-\mu d_m) w_m(z_a)} \tag{A.19}$$

and

$$\mathrm{E}\{\#\text{matings} \mid \text{male dispersal}\} = \frac{1-\alpha}{\alpha}\frac{M w_m(y)}{(1-\mu d_m) w_m(z)}. \tag{A.20}$$

Given that the focal male does not disperse, each of its mates competes successfully for a breeding site with probability

$$\Pr\{\text{female success} \mid \text{no male dispersal}\} = \frac{1}{\kappa(1-\alpha)}\left(\frac{K_f w_f(z_b^L)}{w_f(z_c^L)} + \frac{(1-K_f) w_f(z_b^L)}{w_f(z)}\right) \tag{A.21}$$

where

$$z_c^L = K_f z_a + (1-K_f) z \tag{A.22}$$

is the average phenotype of the mate of a stage-(iv) female competitor of the mate of the focal male on the focal patch, in the absence of selection. Given that the focal male does disperse,

$$\Pr\{\text{female success} \mid \text{male dispersal}\} = \frac{1}{\kappa(1-\alpha)}\frac{w_f(z_b^{NL})}{w_f(z)}. \tag{A.23}$$

For each mate of the focal male that competes successfully, the focal male expects certain numbers sons and daughters. The appropriate expectations are still given by Eqs. (A.5)–(A.8).

Following the calculations from the previous section set out in words, we now have

$$\left.\begin{aligned} a_{mm} &= \frac{(1-h)K_m}{2}\frac{w_m(y)}{w_m(z_a)}\left(\frac{K_f w_f(z_b^L)}{w_f(z_c^L)} + \frac{(1-K_f) w_f(z_b^L)}{w_f(z)}\right)\frac{y}{z_b^L} \\ &\quad + \frac{(1-h)(1-K_m)}{2}\frac{w_m(y)}{w_m(z)}\frac{w_f(z_b^{NL})}{w_f(z)}\frac{y}{z_b^{NL}}, \\ a_{fm} &= \frac{1-\alpha}{\alpha}\frac{K_m}{2}\frac{w_m(y)}{w_m(z_a)}\left(\frac{K_f w_f(z_b^L)}{w_f(z_c^L)} + \frac{(1-K_f) w_f(z_b^L)}{w_f(z)}\right)\frac{y}{z_b^L} \\ &\quad + \frac{1-\alpha}{\alpha}\frac{1-K_m}{2}\frac{w_m(y)}{w_m(z)}\frac{w_f(z_b^{NL})}{w_f(z)}\frac{y}{z_b^{NL}} \end{aligned}\right\}. \tag{A.24}$$

A.2.2. Components of female fitness

Now we consider a focal female on the focal patch at the very beginning of stage (i) of the DMD life history. The focal female mates with the average male found on the focal patch following stage (i). It follows that the focal female competes successfully for a breeding site with probability

$$\Pr\{\text{female successful} \mid \text{no female dispersal}\} = \frac{1}{\kappa(1-\alpha)}\frac{w_f(z_a)}{(1-\mu d_f) w_f(z_c^L)} \tag{A.25}$$

when it does not disperse (probability $1-d_f$), and with probability

$$\Pr\{\text{female successful} \mid \text{no female dispersal}\} = \frac{1}{\kappa(1-\alpha)}\frac{w_f(z_a)}{(1-\mu d_f) w_f(z)} \tag{A.26}$$

when it does disperse (probability d_f). Overall, then

$$\Pr\{\text{female successful}\} = \frac{1}{\kappa(1-\alpha)}\left(\frac{K_f w_f(z_a)}{w_f(z_c^L)} + \frac{(1-K_f) w_f(z_a)}{w_f(z)}\right) \tag{A.27}$$

and substituting Eqs. (A.11), (A.12) and (A.27) into Eq. (A.13) we find

$$\left.\begin{aligned} a_{mf} &= \frac{\alpha}{1-\alpha}\frac{1+h}{2}\left(\frac{K_f w_f(z_a)}{w_f(z_c^L)} + \frac{(1-K_f) w_f(z_a)}{w_f(z)}\right), \\ a_{ff} &= \frac{1}{2}\left(\frac{K_f w_f(z_a)}{w_f(z_c^L)} + \frac{(1-K_f) w_f(z_a)}{w_f(z)}\right) \end{aligned}\right\}. \tag{A.28}$$

A.2.3. Combining components of fitness

We combine the various components of fitness as in the previous section using the neutral distribution and reproductive values of the sexes. Since the matrix $\mathbf{A}^\circ$ we use in this section is identical to that used in the DDM case (Eq. (A.14)), the requisite distribution and reproductive values are again given by the elements of the vectors $\mathbf{u}$ and $\mathbf{v}$ in Eqs. (A.15) and (A.16), respectively. Expressions of recipient fitness for the DMD model are also constructed as they were for DDM (Eq. (A.18)), but using formulae for a_{ij} found in Eqs. (A.24) and (A.28).

A.3. The direct fitness analysis

To analyse the functions W_j developed in the previous subsections, we treat the various local phenotypes on which these W_j functions depend, in turn, as functions of the g_j genotypic value of the sex-j recipient in question (Taylor and Frank, 1996). For DDM, then, we write

$$\left.\begin{aligned} W_m &\equiv W_m(y(g_m), z_a(g_m), z_b^L(g_m), z_b^{NL}(g_m)), \\ W_f &\equiv \text{constant}, \end{aligned}\right\} \tag{A.29}$$

and for DMD we write

$$\left.\begin{aligned} W_m &\equiv W_m(y(g_m), z_a(g_m), z_b^L(g_m), \\ &\qquad z_b^{NL}(g_m), z_c^L(g_m)), \\ W_f &\equiv W_m(z_a(g_f), z_c^L(g_f)) \end{aligned}\right\}. \tag{A.30}$$

We calculate marginal recipient fitness by taking an ordinary derivative of W_j with respect to g_j and then putting neutral coefficients of relatedness in the place derivatives of phenotypes. The weak selection assumption means that we evaluate our calculations by setting all phenotypes equal to the global average value, z.

For DDM the direct-fitness procedure yields,

$$\left.\begin{aligned} dW_m/dg_m|_z &= r_y \partial_y W_m|_z + r_{z_a}\partial_{z_a} W_m|_z \\ &\quad + r_{z_b^L}\partial_{z_b^L} W_m|_z + r_{z_b^{NL}}\partial_{z_b^{NL}} W_m|_z, \\ dW_f/dg_f|_z &= 0, \end{aligned}\right\} \tag{A.31}$$

where rs are relatedness coefficients expressed from the perspective of the individual whose genotype controls the focal male's phenotype, i.e. 'the individual with control' (see Taylor, 1988a, and Appendix B). Note that the individual with control could be the focal male itself, but it does not have to be. For the sake of clarity we will define the following, rather involved, list of relatedness coefficients:

- r_y is the relatedness between the focal male and the individual with control of the focal male's phenotype;

- r_{z_a} is the relatedness between the focal male and the individual with control of the average male competitor's phenotype when the focal male competes on the focal patch;
- $r_{z_b^L}$ is the relatedness between the focal male and the individual with control of the phenotype of the average male that inseminated a female that had, in turn, been inseminated by the focal male on the focal patch;
- $r_{z_b^{NL}}$ is the relatedness between the focal male and the individual with control of the phenotype of the average male that inseminated a female that had, in turn, been inseminated by the focal male on a patch other than the focal patch.

For DMD, the same procedure yields

$$\left.\begin{aligned} dW_m/dg_m|_z &= r_y\partial_y W_m|_z + r_{z_a}\partial_{z_a}W_m|_z \\ &\quad + r_{z_b^L}\partial_{z_b^L}W_m|_z + r_{z_b^{NL}}\partial_{z_b^{NL}}W_m|_z + r_{z_c^L}\partial_{z_c^L}W_m|_z \\ dW_f/dg_f|_z &= \rho_{z_a}\partial_{z_a}W_f|_z + \rho_{z_c^L}\partial_{z_c^L}W_f|_z, \end{aligned}\right\} \quad (A.32)$$

where

- $r_{z_c^L}$ is the relatedness between the focal male and the individual with control of the phenotype of the average mate of a female competitor of the mate of the focal male that had been inseminated on the focal patch and remained there;
- ρ_{z_a} is the relatedness between the focal female and the individual with control of the phenotype of its average mate;
- $\rho_{z_c^L}$ is the relatedness between the focal female and the individual with control of the phenotype of the average male that inseminated a female that had, in turn, remained to compete on the focal patch.

To express the overall inclusive-fitness effect, denoted Δw in the main text, we sum the ordinary derivatives dW_j/dg_j after first weighting these by the total reproductive value measures c_j in Eq. (A.17) (Taylor and Frank, 1996). For the DDM model the appropriate weighted sum is as follows:

$$\left.\begin{aligned} \Delta w &= \frac{w_m'(z)}{w_m(z)}(r_y - K_m r_{z_a}) \\ &\quad + \frac{w_f'(z)}{w_f(z)}(K_m r_{z_b^L} + (1-K_m)r_{z_b^{NL}} - K_m r_{z_a}) \\ &\quad + \frac{1}{z}(r_y - K_m r_{z_b^L} - (1-K_m)r_{z_b^{NL}}), \end{aligned}\right\} \quad (A.33)$$

and for the DMD model it is

$$\left.\begin{aligned} \Delta w &= \frac{w_m'(z)}{w_m(z)}(r_y - K_m r_{z_a}) \\ &\quad + \frac{w_f'(z)}{w_f(z)}(K_m(r_{z_b^L} - K_f r_{z_c^L}) + (1-K_m)r_{z_b^{NL}}) \\ &\quad + \frac{1}{z}(r_y - K_m r_{z_b^L} - (1-K_m)r_{z_b^{NL}}) \\ &\quad + (1+h)\frac{w_f'(z)}{w_f(z)}(\rho_{z_a} - K_f\rho_{z_c^L}) \end{aligned}\right\}. \quad (A.34)$$

The equivalence between Eqs. (A.33) and (A.34), and Eq. (7) of the main text becomes evident when we note that in the main text contains the definitions, $s_a(\delta) = w_m(z+\delta)/w_m(z)$, $s_b(\delta) = w_f(z+\delta)/w_f(z)$, and $s_c(\delta) = (z+\delta)/z$. Thus, $k_a(z) = s_a'(0) = w_m'(z)/w_m(z)$, $k_b(z) = s_b'(0) = w_f'(z)/w_f(z)$, and $k_c(z) = s_b'(0) = 1/z$. In addition,

- $r_y - K_m r_{z_a}$ corresponds to $r_a - \bar{r}_a$ in equation of the main text for both DDM and DMD;
- $K_m r_{z_b^L} + (1-K_m)r_{z_b^{NL}} - K_m r_{z_a}$ corresponds to $r_{bm} - \bar{r}_{bm}$ in equation of the main text for the DDM model;
- $K_m(r_{z_b^L} - K_f r_{z_c^L}) + (1-K_m)r_{z_b^{NL}}$ corresponds to $r_{bm} - \bar{r}_{bm}$ in equation of the main text for the DMD model;
- $r_y - K_m r_{z_b^L} - (1-K_m)r_{z_b^{NL}}$ corresponds to $r_a - r_{bm}$ in equation of the main text for both DDM and DMD;
- $\rho_{z_a} - K_f\rho_{z_c^L}$ corresponds to $r_{bf} - \bar{r}_{bf}$ in equation of the main text for the DMD model (this difference does not appear in the DDM).

The correspondences listed above will be made more explicit in the next appendix.

Appendix B. Calculation of relatedness coefficients

B.1. Preliminary notes and definitions

We measure relatedness, here, using probabilities of identity-by-descent, i.e. coefficients of consanguinity, or CCs (Michod and Hamilton, 1980). To be clear, the CC between individual X and individual Y is the probability that a uniform random gene from X and a uniform random gene from Y are identical by descent.

As has been done in other models of social evolutionary conflict (e.g. Taylor, 1988a; Wild and Taylor, 2005) we define relatedness by making reference to a 'recipient' and an 'actor'. The actor affects the fitness of the recipient and the relatedness between them is actual expressed in terms of the CC between the individual whose genotype determines actor's phenotype (i.e. the 'individual with control' or 'the individual whose perspective we adopt'), and the recipient. It is important to note that, because the main text investigates the model using the actor-centred inclusive-fitness approach, the 'focal individual' in the main text is *not* a recipient, but rather an individual with control of an actor's phenotype.

Let $G_{X\to Y}$ denote the CC between X, the individual with control, and recipient individual, Y. The coefficient of relatedness between X and Y is written, $R_{X\to Y} = G_{X\to Y}/G_{X\to X}$, and as the reader will see, these are approximated to zeroth order in the strength of selection, δ. There is a symmetry to CCs and that is not emphasized by the notation $G_{X\to Y}$, and it will sometimes be necessary to exploit that symmetry. On occasion, then, CCs will be denoted using the calligraphic letter, $\mathcal{G}$; this calligraphic notation will be used to emphasize the symmetric nature of CCs.

In the following subsections we explain how the various relatedness coefficients in Appendix A and the main text are calculated. Because direct-fitness (Appendix A) and inclusive-fitness (main text) accounting each focus on different individuals (the recipient of a social interaction in the former case, and the individual with control of the actor's phenotype in the latter case), it is more straightforward to explain the calculation of coefficients in Appendix A and the main text separately. The calculation of CCs required by both direct-fitness and inclusive fitness relatedness is done in a single section at the end of this appendix.

B.2. Relatedness for the direct-fitness model

B.2.1. Male control

We first give a male control of its own phenotype. In this case, it is easy to see that relatedness between the focal male recipient and the individual that controls its phenotype is simply the male's relatedness to itself, thus

$$\text{DDM and DMD: } r_y = 1. \quad (B.1)$$

Let $G_{\bar{m}\to m}$ denote the CC between the focal male, $Y = m$, and the average male born on the focal patch, $X = \bar{m}$ (equivalently, $\mathcal{G}_{mm}$, the CC between two males chosen uniformly at random with replacement from the same patch at the beginning of stage (i) of either the DDM of DMD model). The constant $K_m = (1-d_m)/(1-\mu d_m)$ is the conditional probability that any male competes on its natal patch, given that it has survived to compete for mates.

Consequently, the CC between a focal male and its average mate competitor, given that the focal male competes on its natal patch is $K_m G_{\bar{m}\to m}$, thus

$$\text{DDM and DMD: } r_{z_a} = \frac{K_m G_{\bar{m}\to m}}{G_{\bar{m}\to\bar{m}}} = \frac{K_m \mathcal{G}_{mm}}{\mathcal{G}_{m\circlearrowleft}} = K_m R_{\bar{m}\to m}, \tag{B.2}$$

where $\mathcal{G}_{m\circlearrowleft}$ is the alternative way of denoting the CC between a male and itself.

A focal male that has successfully competed on its natal patch is related to the average male that successfully inseminated its mate either because the average male is the focal male itself (probability $1/M$), or because the average male is another male (probability $(M-1)/M$) competing on the focal male's natal patch. It follows that

$$\text{DDM and DMD: } r_{z_b^L} = \frac{1}{M} r_y + \frac{M-1}{M} r_{z_a}. \tag{B.3}$$

A focal male that has successfully competed on a patch other than its natal patch is related to the average male that successfully inseminated its mate only because that average male is the focal male itself (probability $1/M$). It follows that

$$\text{DDM and DMD: } r_{z_b^{NL}} = \frac{1}{M} r_y. \tag{B.4}$$

Consider a focal male that mated successfully on the focal patch, and whose mate (the focal female mate) also remained to compete on the focal patch. In the DMD scenario the focal male is related to the average male mated to the female competitor of the focal female mate only if both the average male and the female competitor are both native to the focal patch (probability $K_m K_f$). It follows that

$$\text{DMD: } r_{z_c^L} = K_m K_f R_{\bar{m}\to m}. \tag{B.5}$$

Let $G_{\bar{m}\to f}$ denote the CC between a focal female, $Y = f$, and the average male born on the focal patch, $X = \bar{m}$ (equivalently, $\mathcal{G}_{mf} = \mathcal{G}_{fm}$, the CC between a male and a female chosen uniformly at random from the same patch at the beginning of stage (i) of either the DDM or DMD model). Now, consider a focal female and its average mate. In the DMD scenario, the female is sure to mate on the focal patch. It follows that the CC between the focal female and its average mate is $K_m G_{\bar{m}\to f}$, and so

$$\text{DMD: } \rho_{z_a} = \frac{K_m G_{\bar{m}\to f}}{G_{\bar{m}\to\bar{m}}} = \frac{K_m \mathcal{G}_{mf}}{\mathcal{G}_{m\circlearrowleft}} = K_m R_{\bar{m}\to f}. \tag{B.6}$$

Given that the focal female competes on its natal patch, it is related to the mate of its average female competitor, only in the case where both the female competitor and its mates are native to the focal patch (probability $K_m K_f$). It follows that

$$\text{DMD: } \rho_{z_c^L} = K_m K_f R_{\bar{m}\to f}. \tag{B.7}$$

B.2.2. Female control

We now give control of male phenotype to a female. To handle this case correctly, control must be given to the female prior to competition for mates, because it is the mate-competition stage of the life cycle where the fitness effects of male phenotype are first observed. We suppose that male phenotype is determined just prior to mate competition. To make the biology underlying the female-control model more straightforward, we posit a male phenotypic response to the average (i.e. uniform random) female genotype on the patch on which the male is to compete. For example, a male may respond to some average intermediary female phenotype like colour, or receptivity.

A focal male that survives to compete for mates does so on its natal patch with probability K_m. In the DDM scenario a fraction K_f of the females found on the focal patch are native, whereas in the DMD scenario all females found on the focal patch are native. If $G_{\bar{f}\to m}$ denotes the CC between a focal male, $Y = m$, and the average female born on the focal patch, $X = \bar{f}$ (equivalently, $\mathcal{G}_{mf} = \mathcal{G}_{fm}$), then

$$\text{DDM: } r_y = \frac{K_m K_f G_{\bar{f}\to m}}{G_{\bar{f}\to\bar{f}}} = \frac{K_m K_f \mathcal{G}_{fm}}{\mathcal{G}_{f\circlearrowleft}} = K_m K_f R_{\bar{f}\to m}, \tag{B.8}$$

and

$$\text{DMD: } r_y = \frac{K_m G_{\bar{f}\to m}}{G_{\bar{f}\to\bar{f}}} = \frac{K_m \mathcal{G}_{fm}}{\mathcal{G}_{f\circlearrowleft}} = K_m R_{\bar{f}\to m}. \tag{B.9}$$

Consider the average mate competitor of the focal male. Given that the focal male competes on its natal patch, both its phenotype and the phenotype of its competitor will have been determined by a female born on the focal patch with probability K_f in DDM, and with probability one in DMD. It follows that

$$\text{DDM: } r_{z_a} = K_f R_{\bar{f}\to m}, \tag{B.10}$$

and

$$\text{DMD: } r_{z_a} = R_{\bar{f}\to m}. \tag{B.11}$$

Similarly, given that the focal male competes on its natal patch, all males that inseminated the focal male's mate will have had their phenotype determined by a local female either with probability K_f (DDM), or with probability one (DMD). It follows that

$$\begin{aligned} &\text{DDM: } r_{z_b^L} = K_f R_{\bar{f}\to m}, \\ &\text{DMD: } r_{z_b^L} = R_{\bar{f}\to m}. \end{aligned} \tag{B.12}$$

When a focal male does not compete on its natal patch its phenotype as well as those of all other males with whom it competes will have been determined by a foreign female, thus

$$\text{DDM or DMD: } r_{z_b^{NL}} = 0. \tag{B.13}$$

Consider a focal male that mated successfully on the focal patch and whose mate (the focal female mate) also remained to compete on the focal patch. In the DMD model, a competitor of the focal female mate was native to the patch with probability K_f, in which case the phenotype of that competitor's male mate was determined by a local female with probability one. It follows that

$$\text{DMD: } r_{z_c^L} = K_f R_{\bar{f}\to m}. \tag{B.14}$$

Now we shift focus to a female recipient. In the DMD scenario the phenotype of the average mate of the focal female is determined by the genotype of a native female with probability one. If $G_{\bar{f}\to f}$ is the CC between the focal female and the average female born on the same patch (equivalently, $\mathcal{G}_{ff}$, the CC between two females chosen uniformly at random with replacement from the same patch at the beginning of stage (i) of either the DDM of DMD model), then

$$\text{DMD: } \rho_{z_a} = \frac{G_{\bar{f}\to f}}{G_{\bar{f}\to\bar{f}}} = \frac{\mathcal{G}_{ff}}{\mathcal{G}_{f\circlearrowleft}} = R_{\bar{f}\to f}. \tag{B.15}$$

Again in the DMD scenario, when a focal female competes on its natal patch it competes against another native female with probability K_f. It follows that

$$\text{DMD: } \rho_{z_c^L} = K_f R_{\bar{f}\to f}. \tag{B.16}$$

B.3. Relatedness for the inclusive-fitness model

B.3.1. Male control

In this section we fix attention on a male *in the role of actor*, and we assume that it has control of its own phenotype. Following the usage in the main text, we will refer to this male as the 'focal male'.

Since r_a denotes the relatedness between the focal male actor and the individual with control of its phenotype, it is easy to see that

$$\text{DDM and DMD: } r_a = 1. \tag{B.17}$$

In both the DDM and DMD scenarios, the focal male actor is related to its average competitor for mates only when both focal male and competitor are competing on their natal patch (probability K_m^2). If $G_{m\to\bar{m}}$ is the CC between the focal male actor, $X = m$, and the average male born on the focal patch, $Y = \bar{m}$ (equivalently, $\mathcal{G}_{mm}$), then

$$\begin{aligned}\text{DDM and DMD: } \bar{r}_a &= \frac{K_m^2 G_{m\to\bar{m}}}{G_{m\to m}} = \frac{K_m^2 \mathcal{G}_{mm}}{\mathcal{G}_{m\circlearrowleft}} \\ &= K_m^2 R_{m\to\bar{m}}.\end{aligned} \tag{B.18}$$

The focal male actor competes successfully on its natal patch with probability K_m. In the DDM scenario the focal male finds a native female on its natal patch with probability K_f, and in the DMD scenario the same probability is one. If $G_{m\to\bar{f}}$ is the CC between the focal male actor, $X = m$, and the average female born on the focal patch, $Y = \bar{f}$ (equivalently, $\mathcal{G}_{mf} = \mathcal{G}_{fm}$), then the relatedness between the focal male and any of its mates is either

$$\begin{aligned}\text{DDM: } r_{bf} &= \frac{K_m K_f G_{m\to\bar{f}}}{G_{m\to m}} = \frac{K_m K_f \mathcal{G}_{mf}}{\mathcal{G}_{m\circlearrowleft}} \\ &= K_m K_f R_{m\to\bar{f}},\end{aligned} \tag{B.19}$$

or

$$\text{DMD: } r_{bf} = \frac{K_m G_{m\to\bar{f}}}{G_{m\to m}} = \frac{K_m \mathcal{G}_{mf}}{\mathcal{G}_{m\circlearrowleft}} = K_m R_{m\to\bar{f}}. \tag{B.20}$$

In the DDM scenario, the focal male is only related to the female competitor of one of its own mates, when the focal male has secured its mate on its natal patch (probability K_m), and when the competitor of the focal male's mate has remained to compete on its natal patch (probability K_f). It follows that

$$\begin{aligned}\text{DDM: } \bar{r}_{bf} &= \frac{K_m K_f G_{m\to\bar{f}}}{G_{m\to m}} = \frac{K_m K_f \mathcal{G}_{mf}}{\mathcal{G}_{m\circlearrowleft}} \\ &= K_m K_f R_{m\to\bar{f}}.\end{aligned} \tag{B.21}$$

In the DMD scenario, in order to be related to the female competitor of one of its own mates the focal must have secured the mate on its natal patch (probability K_m), and both the mate and its competitor must have been born on the same patch as the focal male (probability K_f^2). It follows that

$$\begin{aligned}\text{DMD: } \bar{r}_{bf} &= \frac{K_m K_f^2 G_{m\to\bar{f}}}{G_{m\to m}} = \frac{K_m K_f^2 \mathcal{G}_{mf}}{\mathcal{G}_{m\circlearrowleft}} \\ &= K_m K_f^2 R_{m\to\bar{f}}.\end{aligned} \tag{B.22}$$

Consider the average (i.e. uniform random) male mated to a female that has been inseminated by the focal male actor. The focal male is related to this average male either because the average male *is* the focal male (probability $1/M$), or because the average male is not the focal male but was born on the same patch as the focal male (probability $K_m^2(M-1)/M$). It follows that

$$\text{DDM and DMD: } r_{bm} = \frac{1}{M} + \frac{M-1}{M} K_m^2 R_{m\to\bar{m}}. \tag{B.23}$$

Consider a female inseminated by the focal male (i.e. the 'focal mate'). In the DDM scenario, the focal male is related to the average male that inseminated a female competitor of the focal mate only when both the focal male and that average male have been competing on their natal patch (probability K_m^2). It follows that

$$\text{DDM: } \bar{r}_{bm} = K_m^2 R_{m\to\bar{m}}. \tag{B.24}$$

In the DMD scenario, the focal male is related to the average male that inseminated a female competitor of the focal mate only when both the focal male and that average male, *and* both the focal mate and its competitor have been competing on their natal patch (probability $(K_m K_f)^2$). Thus,

$$\text{DMD: } \bar{r}_{bm} = (K_m K_f)^2 R_{m\to\bar{m}}. \tag{B.25}$$

B.3.2. Female control

Now we give control to a female. Recall that, in order to help make our calculations clear, we assume a female control, means that the phenotype of a male actor is determined just prior to mate competition by the average (i.e. uniform random) female on its current patch.

Fix attention on a female (i.e., the 'focal female with control'). In the DDM model, each male phenotype determined by the focal female is exhibited by a male that is related to the focal female only when both are competing on the female's natal patch (probability $K_m K_f$). The same can be said for the relationship between the focal female average competitor of the male whose phenotype the focal female determined. If $G_{f\to\bar{m}}$ is the CC between the focal female and the average male born on the same patch (equivalently, $\mathcal{G}_{fm} = \mathcal{G}_{mf}$), then

$$\begin{aligned}\text{DDM: } r_a = \bar{r}_a &= \frac{K_m K_f G_{f\to\bar{m}}}{G_{f\to f}} = \frac{K_m K_f \mathcal{G}_{fm}}{\mathcal{G}_{f\circlearrowleft}} \\ &= K_m K_f R_{f\to\bar{m}}.\end{aligned} \tag{B.26}$$

In the DMD model, each male phenotype determined by the focal female is exhibited by a male that is related to the focal female only when the male competing on the female's natal patch (probability K_m). Again, the same can be said for the relationship between the focal female average competitor of the male whose phenotype the focal female determined, thus

$$\text{DMD: } r_a = \bar{r}_a = K_m R_{f\to\bar{m}}. \tag{B.27}$$

Consider a female mate of the male whose phenotype was determined by the focal female. In the DDM model, the focal female and this female mate are related only when both are competing on their natal patch (probability K_f^2). In the DDM case, the focal female is said female mate with probability one. If $G_{f\to\bar{f}}$ is the CC between the focal female, $X = f$, and the average female born on the same patch, $Y = \bar{f}$ (equivalently, $\mathcal{G}_{ff}$), then

$$\text{DDM: } r_{bf} = \frac{K_f^2 G_{f\to\bar{f}}}{G_{f\to f}} = \frac{K_f^2 \mathcal{G}_{ff}}{\mathcal{G}_{f\circlearrowleft}} = K_f^2 R_{f\to\bar{f}}, \tag{B.28}$$

and

$$\text{DMD: } r_{bf} = R_{f\to\bar{f}}. \tag{B.29}$$

Consider a female mate of the male whose phenotype was determined by the focal female, and consider a competitor of said female mate. In the DDM model the focal female is as related to the female mate as it is to that female mate's competitor, so $r_{bf} = \bar{r}_{bf}$ in that case. In the DMD model the focal female is as related to the female mate as it is to that female mate's competitor, only when the mate and its competitor compete against one another on their natal patch (probability K_f^2). For both models, then,

$$\text{DDM and DMD: } \bar{r}_{bf} = K_f^2 R_{f\to\bar{f}}. \tag{B.30}$$

Because male phenotype is determined just prior to mate-competition, there is no distinction between a male whose phenotype is determined by the focal female and a male who competes successfully on the same patch as a male whose phenotype is determined by the focal female. Thus $\bar{r}_{bm} = r_a$, and so

Table B.3
Comparison between relatedness terms found in the direct-fitness models of Appendix A and the inclusive-fitness model in the main text (Eq. (7)).

Marginal fitness	Relatedness terms from model	Male control	Female control
Direct fitness, DDM			
$\frac{w'_m(z)}{w_m(z)}$	$r_y - K_m r_{z_a}$	$1 - K_m^2 R_{\bar{m}\to m}$	$K_m K_f R_{\bar{f}\to m} - K_m K_f R_{\bar{f}\to m} = 0$
$\frac{w'_f(z)}{w_f(z)}$	$K_m r_{z_b^L} + (1-K_m) r_{z_b^{NL}} - K_m r_{z_a}$	$\frac{1}{M} + \frac{M-1}{M} K_m^2 R_{\bar{m}\to m} - K_m^2 R_{\bar{m}\to m}$	$K_m K_f R_{\bar{f}\to m} - K_m K_f R_{\bar{f}\to m} = 0$
$\frac{1}{z}$	$r_y - K_m r_{z_b^L} - (1-K_m) r_{z_b^{NL}}$	$1 - \frac{1}{M} - \frac{M-1}{M} K_m^2 R_{\bar{m}\to m}$	$K_m K_f R_{\bar{f}\to m} - K_m K_f R_{\bar{f}\to m} = 0$
Direct fitness, DMD			
$\frac{w'_m(z)}{w_m(z)}$	$r_y - K_m r_{z_a}$	$1 - K_m^2 R_{\bar{m}\to m}$	$K_m R_{\bar{f}\to m} - K_m R_{\bar{f}\to m} = 0$
$\frac{w'_f(z)}{w_f(z)}$	$K_m (r_{z_b^L} - K_f r_{z_c^L}) + (1-K_m) r_{z_b^{NL}}$	$\frac{1}{M} + \frac{M-1}{M} K_m^2 R_{\bar{m}\to m} - (K_m K_f)^2 R_{\bar{m}\to m}$	$K_m R_{\bar{f}\to m} + K_m K_f^2 R_{\bar{f}\to m}$
$\frac{1}{z}$	$r_y - K_m r_{z_b^L} - (1-K_m) r_{z_b^{NL}}$	$1 - \frac{1}{M} - \frac{M-1}{M} K_m^2 R_{\bar{m}\to m}$	$K_m R_{\bar{f}\to m} - K_m R_{\bar{f}\to m} = 0$
$(1+h)\frac{w'_f(z)}{w_f(z)}$	$\rho_{z_a} - K_f \rho_{z_c^L}$	$K_m R_{\bar{m}\to f} - K_m K_f^2 R_{\bar{m}\to f}$	$R_{\bar{f}\to f} - K_f^2 R_{\bar{f}\to f}$
Inclusive fitness, DDM			
$k_a(z)$	$r_a - \bar{r}_a$	$1 - K_m^2 R_{m\to\bar{m}}$	$K_m K_f R_{f\to\bar{m}} - K_m K_f R_{f\to\bar{m}} = 0$
$k_b(z)$	$r_{bm} - \bar{r}_{bm}$	$\frac{1}{M} + \frac{M-1}{M} K_m^2 R_{m\to\bar{m}} - K_m^2 R_{m\to\bar{m}}$	$K_m K_f R_{f\to\bar{m}} - K_m K_f R_{f\to\bar{m}} = 0$
$k_c(z)$	$r_a - r_{bm}$	$1 - \frac{1}{M} - \frac{M-1}{M} K_m^2 R_{m\to\bar{m}}$	$K_m K_f R_{f\to\bar{m}} - K_m K_f R_{f\to\bar{m}} = 0$
$(1+h)k_b(z)$	$r_{bf} - \bar{r}_{bf}$	$K_m K_f R_{m\to\bar{f}} - K_m K_f R_{m\to\bar{f}} = 0$	$K_f^2 R_{f\to\bar{f}} - K_f^2 R_{f\to\bar{f}} = 0$
Inclusive fitness, DMD			
$k_a(z)$	$r_a - \bar{r}_a$	$1 - K_m^2 R_{m\to\bar{m}}$	$K_m R_{f\to\bar{m}} - K_m R_{f\to\bar{m}} = 0$
$k_b(z)$	$r_{bm} - \bar{r}_{bm}$	$\frac{1}{M} + \frac{M-1}{M} K_m^2 R_{m\to\bar{m}} - (K_m K_f)^2 R_{m\to\bar{m}}$	$K_m R_{f\to\bar{m}} + K_m K_f^2 R_{f\to\bar{m}}$
$k_c(z)$	$r_a - r_{bm}$	$1 - \frac{1}{M} - \frac{M-1}{M} K_m^2 R_{m\to\bar{m}}$	$K_m R_{f\to\bar{m}} - K_m R_{f\to\bar{m}} = 0$
$(1+h)k_b(z)$	$r_{bf} - \bar{r}_{bf}$	$K_m R_{m\to\bar{f}} - K_m K_f^2 R_{m\to\bar{f}}$	$R_{f\to\bar{f}} - K_f^2 R_{f\to\bar{f}}$

$$\text{DDM: } r_{bm} = K_m K_f R_{f\to\bar{m}}, \tag{B.31}$$

and

$$\text{DMD: } r_{bm} = K_m R_{f\to\bar{m}}. \tag{B.32}$$

In the DDM model a focal female is related to a male mate of a female competitor only when the focal female, itself, and the male mate are competing (or have competed) on their natal patch (probability $K_m K_f$), thus

$$\text{DDM: } \bar{r}_{bm} = K_m K_f R_{f\to\bar{m}}. \tag{B.33}$$

In the DMD model a focal female is related to a male mate of a female competitor only when the focal female, the male mate, and the female competitor are competing (or have competed) on their natal patch (probability $K_m K_f^2$), thus

$$\text{DDM: } \bar{r}_{bm} = K_m K_f^2 R_{f\to\bar{m}}. \tag{B.34}$$

B.3.3. Comparison between direct- and inclusive-fitness results

Table B.3 summarizes the marginal fitness terms and the associated relatedness terms found in the direct-fitness models (Eqs. (A.33) and (A.34)) as well as those found in the inclusive fitness model (Eq. (7) main text). As we have noted above,

$$\left.\begin{aligned} R_{m\to\bar{m}} &= \frac{\mathcal{G}_{mm}}{\mathcal{G}_{m\circlearrowleft}} = R_{\bar{m}\to m}, \\ R_{f\to\bar{f}} &= \frac{\mathcal{G}_{ff}}{\mathcal{G}_{f\circlearrowleft}} = R_{\bar{f}\to f}, \\ R_{f\to\bar{m}} &= \frac{\mathcal{G}_{fm}}{\mathcal{G}_{f\circlearrowleft}} = \frac{\mathcal{G}_{mf}}{\mathcal{G}_{f\circlearrowleft}} = R_{\bar{f}\to m}, \\ R_{m\to\bar{f}} &= \frac{\mathcal{G}_{mf}}{\mathcal{G}_{m\circlearrowleft}} = \frac{\mathcal{G}_{fm}}{\mathcal{G}_{m\circlearrowleft}} = R_{\bar{m}\to f}, \end{aligned}\right\} \tag{B.35}$$

and so the information in Table B.3 shows that the direct-fitness and inclusive-fitness models agree.

B.4. Calculation of CCs

The calculation of CCs used in this paper have appeared elsewhere (Wild and Taylor, 2004, 2005; Pen, 2006; Wild and West, 2009), but we present them again for the reader's convenience.

B.4.1. Diploidy

Let $\mathcal{G} = \mathcal{G}_{ij}$ be the CC between two offspring born on the same patch. At equilibrium, $\mathcal{G}$ must satisfy,

$$\begin{aligned} \mathcal{G} = {} & \frac{1}{N}(\text{CC between individuals with same mother}) \\ & + \frac{N-1}{N}(\text{CC between individuals with} \\ & \text{different mothers})\mathcal{G}. \end{aligned} \tag{B.36}$$

Conditioning on the parents from which the uniform random alleles chosen have originated we determine the CC between individuals with the same mother in the DDM scenario to be

$$\begin{aligned} \text{DDM: } & \frac{1}{4}\left(\frac{1+K_m K_f \mathcal{G}}{2}\right) + \frac{1}{2}(K_m K_f \mathcal{G}) \\ & + \frac{1}{4}\left(\frac{1}{M}\frac{1+K_m K_f \mathcal{G}}{2} + \frac{M-1}{M} K_m^2 \mathcal{G}\right). \end{aligned} \tag{B.37}$$

For DMD the CC between individuals with the same mother is

$$\begin{aligned} \text{DMD: } & \frac{1}{4}\left(\frac{1+K_m \mathcal{G}}{2}\right) + \frac{1}{2}(K_m \mathcal{G}) \\ & + \frac{1}{4}\left(\frac{1}{M}\frac{1+K_m \mathcal{G}}{2} + \frac{M-1}{M} K_m^2 \mathcal{G}\right). \end{aligned} \tag{B.38}$$

Again conditioning on the parents from which the uniform random alleles have originated we determine the CC between individuals different mothers to be

$$\text{DDM: } \frac{1}{4}K_f^2 \mathcal{G} + \frac{1}{2}K_m K_f \mathcal{G} + K_m^2 \mathcal{G} = \frac{1}{4}\mathcal{G}(K_f + K_m)^2, \tag{B.39}$$

and

$$\text{DMD: } \frac{1}{4}K_f^2 \mathcal{G} + \frac{1}{2}K_m K_f^2 \mathcal{G} + (K_f K_m)^2 \mathcal{G} = \frac{1}{4}\mathcal{G}K_f^2(1+K_m)^2. \tag{B.40}$$

For any sex i, $\mathcal{G}_{i\circlearrowleft} = (1 + K_m K_f \mathcal{G})/2$ for DDM and $\mathcal{G}_{i\circlearrowleft} = (1 + K_m \mathcal{G})/2$ for DMD. Thus, substituting either Eqs. (B.37) and (B.39), or (B.38) and (B.40) into (B.36) and solving for $\mathcal{G}$ gives us all building blocks necessary to formulate an expressions for the coefficients $R_{i\to\bar{j}}$ or $R_{\bar{i}\to j}$ introduced above.

B.4.2. Haplodiploidy

To calculated CCs in a haplodiploid system we introduce $\mathcal{H}_{ij} = \mathcal{H}_{ji}$ the CC between a uniform random sex-i individual (or its genetic contribution to offspring, e.g. egg, sperm) and a uniform random sex-j individual (or its genetic contribution to offspring) chosen from among the N sites in a given patch.

Another simple conditioning argument shows that, at equilibrium, the CCs for the DDM model must satisfy

$$\text{DDM:}\quad \left\{\begin{array}{rcl} \mathcal{H}_{ff} &=& \dfrac{1}{N}\left(\dfrac{1+K_mK_f(\mathcal{H}_{ff}+\mathcal{H}_{fm})/2}{2}\right) \\ && +\dfrac{N-1}{N}K_f^2\left(\dfrac{1}{4}\mathcal{H}_{ff}+\dfrac{1}{2}\mathcal{H}_{fm}+\dfrac{1}{4}\mathcal{H}_{mm}\right) \\ \mathcal{H}_{fm} &=& K_mK_f\left(\dfrac{1}{2}\mathcal{H}_{ff}+\dfrac{1}{2}\mathcal{H}_{fm}\right) \\ \mathcal{H}_{ff} &=& \dfrac{1}{NM}+\dfrac{NM-1}{NM}K_m^2\mathcal{H}_{ff} \end{array}\right\}, \tag{B.41}$$

and the CCs for the DMD model must satisfy

$$\text{DMD:}\quad \left\{\begin{array}{rcl} \mathcal{H}_{ff} &=& \dfrac{1}{N}\left(\dfrac{1+K_m(\mathcal{H}_{ff}+\mathcal{H}_{fm})/2}{2}\right) \\ && +\dfrac{N-1}{N}K_f^2\left(\dfrac{1}{4}\mathcal{H}_{ff}+\dfrac{1}{2}\mathcal{H}_{fm}+\dfrac{1}{4}\mathcal{H}_{mm}\right) \\ \mathcal{H}_{fm} &=& \left(\dfrac{1}{N}K_m+\dfrac{N-1}{N}K_mK_f^2\right) \\ && \times\left(\dfrac{1}{2}\mathcal{H}_{ff}+\dfrac{1}{2}\mathcal{H}_{fm}\right) \\ \mathcal{H}_{ff} &=& \dfrac{1}{N}\dfrac{1}{M}+\dfrac{1}{N}\dfrac{M-1}{M}K_m^2\mathcal{H}_{ff} \\ && +\dfrac{N-1}{N}(K_mK_f)^2\mathcal{H}_{ff} \end{array}\right\}. \tag{B.42}$$

Solutions to systems (B.41) or (B.42) are substituted into the following formulae for the CC between individuals born on the same patch:

$$\left.\begin{array}{l} \mathcal{G}_{ff} = \dfrac{1}{4}\mathcal{H}_{ff}+\dfrac{1}{2}\mathcal{H}_{fm}+\dfrac{1}{4}\mathcal{H}_{mm} \\ \mathcal{G}_{fm} = \dfrac{1}{2}\mathcal{H}_{ff}+\dfrac{1}{2}\mathcal{H}_{fm} \\ \mathcal{G}_{mm} = \mathcal{H}_{ff} \end{array}\right\}. \tag{B.43}$$

The CCs in line (B.43) can now be used along with $\mathcal{G}_{m\circlearrowleft} = 1$, $\mathcal{G}_{f\circlearrowleft} = (1+K_mK_f\mathcal{G}_{fm})/2$ (DDM), and $\mathcal{G}_{f\circlearrowleft} = (1+K_m\mathcal{G}_{fm})/2$ (DMD) to construct the relatedness coefficients introduced above.

Appendix C. Description of simulations

In this appendix we provide a brief description of the simulations used to generate results presented in the main text. We also provide Matlab scripts that can be used to generate results like those in Figs. 5 and 6 (files DMDSim.m and DDMSim.m, attached). We do not claim that the scripts provided are written in the most efficient way.

In our simulation of the DMD and DDM life histories, a population of the offspring produced by every inseminated female in each of P groups (i.e. P patches) is randomly initialized, and the first of T simulated generations is begun.

At the beginning of each simulated generation the phenotypes of male offspring are determined. When the male perspective is adopted (male control), phenotypes are determined by the males own genotype. By contrast, when the female perspective is adopted (female control), a males phenotype is determined by the genotype of its average potential mate, conditioned on the event that the male itself survives to compete for mates.

Once individual phenotypes have been determined male–male competition occurs for the M mating opportunities offered by each female offspring found in the population. As indicated in the main text, we simulate mate competition in a hierarchical fashion by first allowing among-group competition for mates to occur. During the among-group competitive stage, we award a mating to a male from the local group with probability K_m; with probability $1-K_m$ the winner is chosen from any patch in the population by conducting a loaded raffle with patch-average w_m acting as weights. Once the winning group has been established, within-group competition to occur among its members, and the exact winner is determined by a loaded raffle with individuals' w_m acting as weights.

Female–female competition for reproductive resources (here, breeding sites) also occurs in a hierarchical fashion similar to that described above. Once a winning female has been determined, sperm competition occurs among its male partners; once all winning females have been determined the genotypes of the new cohort of offspring are mutated, and a new generation is begun.

The key difference between the DMD and DDM simulations, of course, has to do with the timing of female dispersal. Unlike our DMD computer model, our DDM model redistributes females prior to male–male competition for mates by mixing up simulated broods of offspring in a random manner.

Appendix D. Supplementary data

Supplementary material related to this article can be found online at doi:10.1016/j.tpb.2011.09.002.

References

Alizon, S., Taylor, P.D., 2008. Empty sites can promote altruistic behaviour. Evolution 62, 1335–1344.

Arnqvist, G., Rowe, L., 2005. Sexual Conflict. Princeton University Press, Princeton, NJ.

Birkhead, T.R., Parker, G.A., 1997. Sperm competition and mating systems. In: Krebs, J.R., Davies, N.B. (Eds.), Behavioural Ecology: An Evolutionary Approach, 4th ed.. Blackwell, Oxford, UK, pp. 121–145.

Blanckenhorn, W.U., Hosken, D.J., Martin, O.Y., Reim, C., Teuschl, Y., Ward, P.I., 2002. The costs of copulating in the dung fly *Sepsis cynipsea*. Behavioral Ecology 13, 353–358.

Bulmer, M.G., Taylor, P.D., 1980. Dispersal and the sex ratio. Nature 284, 448–449.

Christiansen, F.B., 1991. On the conditions for evolutionary stability for a continuously varying character. American Naturalist 138, 37–50.

Eberhard, W.G., 1999. Mating systems of sepsid flies and sexual behaviour away from oviposition sites by *Sepsis neocynipsea* (Diptera: Sepsidae). Journal of the Kansas Entomological Society 72, 129–130.

Eldakar, O.T., Dlugos, M.J., Holt, G.P., Wilson, D.S., Pepper, J.W., 2010. Population structure influences sexual conflict in wild populations of water striders. Behaviour 147, 1615–1631.

Eldakar, O.T., Dlugos, Pepper, M.J., J.W., Wilson, D.S., 2009. Population structure mediates sexual conflict in water striders. Science 326, 816.

Fisher, R.A., 1930. The Genetical Theory of Natural Selection. Clarendon, Oxford, UK.

Frank, S.A., 1998. Foundations of Social Evolution. Princeton University Press, Princeton, NJ.

Gardner, A., West, S.A., 2004. Spite and the scale of local competition. Journal of Evolutionary Biology 17, 1195–1203.

Gardner, A., West, S.A., 2006. Demography, altruism, and the benefits of budding. Journal of Evolutionary Biology 19, 1707–1716.

Gardner, A., West, S.A., Buckling, A., 2004. Bacteriocins, spite and virulence. Proceedings of the Royal Society of London B 271, 1529–1535.

Garefalaki, M.E., Triantafyllidis, A., Abatzopoulos, T.J., Staikou, A., 2010. The outcome of sperm competition is affected by behavioural and anatomical reproductive traits in a simultaneously hermaphroditic land snail. Journal of Evolutionary Biology 23, 966–976.

Gay, L., Hoseken, D.J., Eady, P., Vasudev, R., Tregenza, T., 2011. The evolution of harm-effect of sexual conflicts and population size. Evolution 65, 725–737.

Grafen, A., Archetti, M., 2008. Natural selection of altruism in inelastic viscous homogeneous population. Journal of Theoretical Biology 252, 694–710.

Greenwood, P.J., 1980. Mating systems, philopatry and dispersal in birds and mammals. Animal Behaviour 28, 1140–1162.

Greenwood-Lee, J., Taylor, P.D., Haig, D., 2001. The inclusive fitness dynamics of genomic imprinting. Selection 2, 101–116.

Griffin, A.S., West, S.A., Buckling, A., 2004. Cooperation and competition in pathogenic bacteria. Nature 430, 510–513.

Hamilton, W.D., 1964. The genetical evolution of social behaviour, I and II. Journal of Theoretical Biology 7, 1–52.
Krupa, J.J., Sih, A., 1993. Experimental studies on water strider mating dynamics: spatial variation in density and sex ratio. Behavioral Ecology and Sociobiology 33, 107–120.
Kümmerli, R., Gardner, A., West, S.A., Griffin, A.S., 2009. Limited dispersal, budding dispersal and cooperation: an experimental study. Evolution 63, 939–949.
Maynard Smith, J., 1982. Evolution and the Theory of Games. Cambridge University Press, Cambridge, UK.
Michod, R.E., Hamilton, W.D., 1980. Coefficients of relatedness in sociobiology. Nature 288, 694–697.
Parker, G.A., 2006. Sexual conflict over mating and fertilization. Philosophical Transactions of the Royal Society B 361, 235–259.
Parker, G.A., 1979. Sexual selection and sexual conflict. In: Blum, M.S., Blum, N.A. (Eds.), Reproductive Competition and Sexual Selection in Insects. Academic Press, New York, NY, pp. 123–166.
Parker, G.A., 1972a. Reproductive behaviour of *Sepsis cynipsea* (L.) (Diptera: Sepsidae). I. a preliminary analysis of the reproductive strategy and its associated behaviour patterns. Behaviour 41, 172–206.
Parker, G.A., 1972b. Reproductive behaviour of *Sepsis cynipsea* (L.) (Diptera: Sepsidae). II. the significance of the precopulatory passive phase and emigration. Behaviour 41, 242–250.
Parker, G.A., Pizzari, T., 2010. Sperm competition and ejaculate economics. Biological Reviews 85, 897–934.
Pen, I., 2006. When boys want to be girls: effects of mating system and dispersal on parent-offspring sex ratio conflict. Evolutionary Ecology Research 8, 103–113.
Pepper, J.W., 2000. Relatedness in trait group models of social evolution. Journal of Theoretical Biology 206, 355–368.
Pizzari, T., Foster, K.R., 2008. Sperm sociality: cooperation, altruism and spite. PLoS Biology 6, e310.
Price, G.R., 1970. Selection and covariance. Nature 227, 520–521.
Queller, D.C., 1994. Genetic relatedness in viscous populations. Evolutionary Ecology 8, 70–73.
Rankin, D.J., 2011. Kin selection and the evolution of sexual conflict. Journal of Evolutionary Biology 24, 71–81.
Raveh, S., Heg, D., Dobson, F.S., Coltman, D.W., Gorrell, J.C., Balmer, A., Neuhaus, P., 2010. Mating order and reproductive success in male columbian ground squirrels (*Urocitellus columbianus*). Behavioral Ecology 21, 537–547.
Reinhardt, K., Siva-Jothy, M.T., 2007. Biology of the bed bugs (Cimidae). Annual Review of Entomology 52, 351–374.
Taylor, P.D., 1992. Altruism in viscous populations—an inclusive fitness approach. Evolutionary Ecology 6, 352–356.
Taylor, P.D., 1994. Sex ratio in a stepping-stone population with sex-specific dispersal. Theoretical Population Biology 45, 203–218.
Taylor, P.D., 1996. Inclusive fitness arguments in genetic models of behaviour. Journal of Mathematical Biology 34, 654–674.
Taylor, P.D., 1988a. An inclusive fitness model for dispersal of offspring. Journal of Theoretical Biology 130, 363–378.
Taylor, P.D., 1988b. Inclusive fitness models with two sexes. Theoretical Population Biology 34, 145–168.
Taylor, P.D., 1989. Evolutionary stability in one-parameter models under weak selection. Theoretical Population Biology 36, 125–143.
Taylor, P.D., Frank, S.A., 1996. How to make a kin selection model. Journal of Theoretical Biology 180, 27–37.
Taylor, P.D., Wild, G., Gardner, A., 2007. Direct or inclusive fitness: how shall we model kin selection? Journal of Evolutionary Biology 20, 301–309.
Taylor, P.D., Lillicrap, T., Cownden, D., 2011. Inclusive fitness analysis on mathematical groups. Evolution 65, 849–859.
Trivers, R.L., 1972. Parental investment and sexual selection. In: Campbell, B. (Ed.), Sexual Selection and the Descent of Man. Aldine, Chicago, IL, pp. 136–179.
Van Cleve, J., Feldman, M.W., Lehmann, L., 2010. How demography, life history, and kinship shape the evolution of genomic imprinting. American Naturalist 176, 440–455.
Ward, P.I., Hemmi, J., Röösli, T., 1992. Sexual conflict in the dung fly *Sepsis cynipsea*. Functional Ecology 6, 649–653.
West, S.A., 2009. Sex Allocation. Princeton University Press, Princeton, NJ.
West, S.A., Murray, M.G., Machado, C.A., Griffin, A.S., Herre, E.A., 2001. Testing hamiltons rule with competition between relatives. Nature 409, 510–513.
West, S.A., Pen, I., Griffin, A.S., 2002. Cooperation and competition between relatives. Science 296, 72–75.
Wigby, S., Chapman, T., 2004. Female resistance to male harm evolves in response to manipulation of sexual conflict. Evolution 58, 1028–1037.
Wild, G., Taylor, P.D., 2004. Kin selection models for the co-evolution of the sex ratio and sex-specific dispersal. Evolutionary Ecology Research 6, 481–502.
Wild, G., Taylor, P.D., 2005. A kin-selection approach to the resolution of a sex-ratio conflict between mates. Journal of Theoretical Biology 236, 126–136. doi:10.1016/j.jtbi.2005.03.003.
Wild, G., West, S.A., 2009. Genomic imprinting and sex allocation. American Naturalist 173, E1–E14.
Wilkins, J.F., Haig, D., 2003. What good is genomic imprinting: the function of parent specific gene expression. Nature Reviews Genetics 4, 1–10.