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Nice natives and mean migrants: the evolution of dispersal-dependent social behaviour in viscous populations

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Abstract

There has been much interest in the evolution of social behaviour in viscous populations. While low dispersal increases the relatedness of neighbours, which tends to promote the evolution of indiscriminate helping behaviour, it can also increase competition between neighbours, which tends to inhibit the evolution of helping and may even favour harming behaviour. In the simplest scenario, these two effects exactly cancel, so that dispersal rate has no impact on the evolution of helping or harming. Here, we show that dispersal rate does matter when individuals can adjust their social behaviour conditional on whether they have dispersed or whether they have remained close to their place of origin. We find that nondispersing individuals are weakly favoured to indiscriminately help their neighbours, whereas dispersing individuals are more readily favoured to indiscriminately harm their neighbours.

Introduction

Cooperation abounds in nature, and poses one of the greatest problems for evolutionary biology. If natural selection is the survival of the fittest, then how can individuals be favoured to promote the reproductive success of others? (Hamilton 1996; West *et al*. 2007a). Despite this apparent problem, cooperation occurs at all levels of biological organization from within eukaryotic cells, to multi-cellular organisms to complex human and animal societies (Leigh 1977; Buss 1987; Maynard Smith and Szathmary, 1995).

One explanation for cooperation is provided by kin selection (Hamilton 1963, 1964, 1970; Maynard Smith 1964). Because relatives share genes in common, an individual who improves the reproductive success of her relatives may increase her overall genetic contribution to future generations, and genes that promote cooperation may increase in frequency. Hamilton (1964) suggested two major mechanisms for ensuring that cooperative social behaviours be directed primarily towards high relatedness social partners. First, he suggested that individuals might be able to recognize and act preferentially towards their genealogical kin (kin discrimination). However, this mechanism may require complicated cognitive faculties, and may be inherently unstable (Crozier 1986; Rousset & Roze 2007); so, it appears to be rather limited in its application. Second, he proposed that if individuals do not disperse far from their place of origin (population viscosity), then neighbours would tend to be genealogical relatives, and hence even indiscriminate cooperation may be favoured (see also Wright 1945). This requires no cognitive function, nor indeed any perception of the external world, and hence viscosity appears to provide a very general explanation for cooperation at all levels of biological organization (West, *et al*. 2007b).

However, later work showed that cooperation is not so readily favoured in viscous populations. Although reduced dispersal increases the relatedness of social partners, which acts to favour the evolution of cooperation, it also increases competition between social partners for space and resources, which acts to disfavour the evolution of cooperation (reviewed by Queller 1992 and West *et al*. 2002). In the simplest scenario these two effects exactly cancel, so that there is no net impact of the dispersal rate on the evolution of cooperation (Taylor, 1992a, b; see also Wilson et al. 1992). Experimental and observational data support these predictions showing that local competition can reduce or completely negate any effect of limited dispersal on selection for cooperation (West *et al*. 2001; Griffin *et al*. 2004; R. Kümmerli,

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A. Gardner, S.A. West and A.S. Griffin, unpublished data). Indiscriminate cooperation is favoured in a purely viscous population only as readily as it is favoured in a fully dispersing population.

In addition to disfavouring cooperation, localized competition may also favour harming behaviour (reviewed by Gardner & West 2004). However, Taylor's (1992a) model, which implicitly describes the evolution of indiscriminate harming as well as helping behaviour in viscous populations, again predicts that there is no net impact of dispersal rate. While limited dispersal increases competition between neighbours, promoting the evolution of harming, it also increases the relatedness of neighbours, which inhibits the evolution of harming, and these two effects exactly cancel. Neither indiscriminate helping nor indiscriminate harming are affected by the dispersal rate in this simplest scenario.

Taylor's (1992a) simple model provides a useful benchmark upon which to rest and conceptualize more elaborate (and potentially more realistic) models in which the relationships between dispersal, relatedness and competition are altered so that limited dispersal can make an impact on helping and harming behaviour. Analytical extensions of the basic model that have been shown to promote the evolution of indiscriminate helping behaviour include the inclusion of overlapping generations (Taylor & Irwin 2000; Irwin & Taylor 2001; Grafen 2007; Lehmann *et al*., 2007a, b; Taylor, *et al*. 2007), trans-generational social behaviour and niche construction (Lehmann 2007), elastic population structure (Taylor 1992b; van Baalen & Rand 1998; Mitteldorf & Wilson 2000; Alizon & Taylor 2008), group (or 'budding') dispersal (Gardner & West 2006; Lehmann *et al*. 2006), sex-biased dispersal (Johnstone & Cant, 2008) and kin discrimination (Perrin & Lehmann 2001). The evolution of harming has received much less attention (e.g. Gardner *et al*. 2004, 2007; Lehmann et al., 2007a, b; Johnstone & Cant, 2008). A major challenge is to find minimal additions to the basic model that allow social behaviour to be promoted by population viscosity, without invoking unrealistic cognitive and other faculties that will reduce the generality of the mechanism.

In this article, we consider that individuals can assess their dispersal status, i.e. whether they have dispersed or whether they have remained close to their place of origin, and we ask whether they will be favoured to adjust their social behaviour conditional upon this information. Although dispersers and nondispersers do not differ in the resource competition they experience with neighbours, they can differ with respect to their relatedness to their neighbours. Specifically, nondispersers are generally predicted to be more highly related to their neighbours and dispersers will be less related to their neighbours. Thus, we predict that helping will more readily be favoured among nondispersers and harming more readily favoured among dispersers, relative to the case where social behaviour is not adjusted according to dispersal status. This mechanism does not require that individuals assess any aspect of their social partners directly, nor indeed necessarily any aspect of their external environment. Thus, it appears to be rather general in its application.

Models and analyses

Basic model

Our analysis is based on the infinite island model analysed by Taylor (1992a); see also Wright (1931). A summary of the model notation used in this article is provided in Table 1. We consider an infinite population of asexual haploid individuals organized into patches of n individuals, and initially we assume that no social interaction takes place. Individuals produce a large number $K >> 1$ of offspring, then die, and each offspring disperses to another, randomly chosen, patch with

Table 1 A summary of symbols used in the analysis.

Symbol	Definition
a	Fecundity cost of harming for harmer
A	Personal cost function for harming
b	Fecundity benefit of helping for recipients
B	Recipient benefit function for helping
$\tilde{b}$	Fitness effect of social behaviour on recipient
c	Fecundity cost of helping for helper
C	Personal cost function for helping
$\tilde{c}$	Fitness effect of social behaviour on actor
c_X	Reproductive value of class X
d	Fecundity cost of harming for victims
D	Victim cost function for harming
D	Disperser
F	Fecundity of a focal individual
$\bar{F}$	Average fecundity of focal patch
g	Genetic 'breeding' value for social behaviour
i	Number of mutants among a focal individual's patch mates
K	Average number of offspring per individual in the population
m	Dispersal rate
M	Mutant
n	Number of breeding spaces per patch
N	Nondisperser
q_X	Abundance of class X
r	Coefficient of relatedness between patch mates (i.e. not including self)
R	Coefficient of relatedness within a patch (i.e. including self)
t	Generation number
u	Generic nondisperser class
U	Set of all nondisperser classes
v	Generic disperser class
V	Set of all disperser classes
w	Darwinian fitness
x	Helping strategy
$\hat{x}$	Mutant helping strategy
$\bar{x}$	Average helping strategy
y	Harming strategy
$\hat{y}$	Mutant harming strategy
$\bar{y}$	Average harming strategy

probability m or else, with probability $1 - m$, remains in its patch of origin. After dispersal, n offspring are chosen on each patch to mature to adulthood, whereas the rest die, and this returns the population to its original size for the beginning of the next lifecycle.

Assuming that all genetic variation in the population is neutral with respect to reproductive success, we can use the recursion approach of Taylor (1992a) to determine the relatedness structure of the population at equilibrium. We note that the expected relatedness of any individual in generation t to a randomly chosen member of her own patch (including herself) is $R_t = 1/n + ((n-1)/n)r_t$, where $1/n$ is the probability of choosing herself, in which case the relatedness is 1, and $(n-1)/n$ is the probability of choosing a different individual, in which case the relatedness is r_t, the relatedness of two different individuals in the same patch. The only way for two different individuals from the same patch to be related is if neither dispersed, which occurs with probability $(1-m)^2$. In this case, the two individuals will have an expected relatedness equal to the relatedness of two individuals chosen at random from that same patch in the previous generation, R_{t-1}. This allows us to write down a recursion:

$$R_t = \frac{1}{n} + \frac{n-1}{n}(1-m)^2 R_{t-1}, \tag{1}$$

which can be solved for equilibrium ($R_{t-1} = R_t = R$) to yield an equilibrium relatedness of an individual to her group (including herself) of:

$$R = \frac{1}{n-(n-1)(1-m)^2}, \tag{2}$$

as determined by Taylor (1992a). Substituting this into the expression $R = 1/n + ((n-1)/n)r$ and solving for r obtains the equilibrium relatedness between different individuals within the same patch:

$$r = \frac{(1-m)^2}{n-(n-1)(1-m)^2}, \tag{3}$$

i.e. the F_{ST} of Wright (1943). Note that both these 'whole-patch' and 'others-only' relatedness coefficients (R and r; Pepper 2000) are expressed as averages over all individuals, with dispersers and nondispersers taken together. However, dispersing individuals always find themselves in a patch containing no relatives other than themselves, and so restricting attention to dispersers only, the equilibrium relatedness is $R_D = 1/n$ to the whole patch and $r_D = 0$ to the other individuals in the patch. Noting that $R = mR_D + (1-m)R_N$ and $r = mr_D + (1-m)r_N$, where R_N and r_N are the whole-patch and others-only relatedness for nondispersing individuals, we can solve to find that:

$$R_N = \frac{n+(n-1)m(1-m)}{n(n-(n-1)(1-m)^2)}, \tag{4}$$

and:

$$r_N = \frac{1-m}{n-(n-1)(1-m)^2}, \tag{5}$$

for nondispersers. Note that for $0 < m < 1$, we have $R_D < R < R_N$ and $r_D < r < r_N$, i.e. dispersers experience a lower relatedness to their social partners, and nondispersers a higher relatedness to their social partners, than average (Fig. 1). Taylor (1992a) found that unconditional helping behaviour cannot be favoured if it incurs a net fecundity cost for the actor; the result also extends to unconditional harming behaviour, revealing that this is also disfavoured if the actor incurs a net fecundity cost. In the next two sections, we explore whether and when the increased relatedness experienced by nondispersers favours helping behaviour that is expressed only by nondispersers, and whether and when the decreased relatedness experienced by dispersers favours harming behaviour that is expressed only by dispersers.

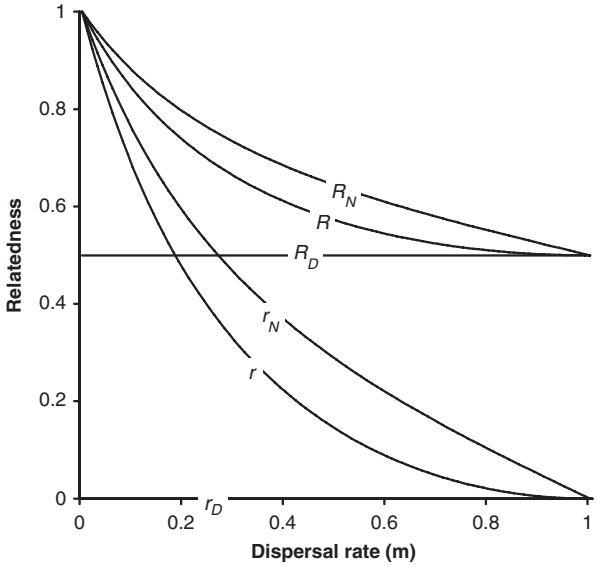

Fig. 1 Relatedness coefficients in viscous populations. Increased dispersal (m) separates kin and hence reduces the relatedness between patchmates (r) and the relatedness of an individual to the patch as a whole (i.e. including itself; R). These relatedness coefficients correspond to Taylor's (1992a) model of unconditional social behaviour, in which neither costly helping nor costly harming is favoured by selection. Our model considers that nondispersing individuals will experience higher than average relatedness to their patch ($r_N > r$ and $R_N > R$), whereas dispersing individuals will experience lower than average relatedness to their patch ($r_D < r$ and $R_D < R$), and thus nondispersers may be favoured to exhibit indiscriminate helping behaviour, and dispersers may be favoured to exhibit indiscriminate harming behaviour. Numerical solutions are calculated assuming a patch size of two individuals ($n = 2$).

Evolution of helping in nondispersers

We now consider a vanishingly rare genetic mutation that causes the bearer to express indiscriminate, others-only helping behaviour that incurs a relative personal fecundity cost c and gives a relative fecundity benefit b that is shared equally by her $n - 1$ patch mates; these fecundity effects are expressed relative to the baseline fecundity, which is set equal to 1, and we assume b, $c << 1$. Crucially, we assume that this helping behaviour is expressed conditionally with respect to the individual's own dispersal status; in particular, we assume that only nondispersers, who have remained in their natal patch, exhibit helping behaviour.

In order for the mutation to be favoured by selection it must, on average, increase the expected fitness of its bearers, i.e. the number of surviving offspring that the individual produces over her lifetime (Price 1970). There are two routes by which a gene may impact on the fitness of its bearer: first, by having a direct impact on that individual's personal reproductive success, and second, by having an indirect impact on the individual's personal reproductive success due to its presence among relatives who socially interact with her (neighbour-modulated fitness; Hamilton 1963, 1964). There are neither direct nor indirect effects of the gene on carriers that have dispersed away from their patch of origin, as they do not express the helping behaviour and they do not encounter any other individuals carrying the mutant gene. So, the condition for the gene to be favoured is if it leads, through direct and/or indirect effects, to an increase in the expected number of surviving offspring (i.e. Darwinian fitness) of nondispersers (see Appendix for mathematical details). The average fitness in the population is 1, because the population size remains constant from generation to generation, and dispersers and nondispersers have the same expected fitness; so, a condition for the mutant gene to be favoured by natural selection is that the expected fitness of a disperser carrying the gene exceeds 1.

We now proceed to calculate the expected fitness of a nondisperser who is carrying the mutant gene, and this is a function of her fecundity and the intensity of competition experienced by her offspring for one of the n breeding spaces within whichever patch they find themselves in after the dispersal event. The relative (to the population average) fecundity of a mutant nondisperser that shares her patch with i other mutant nondispersers is:

$$F_{\mathrm{MN},i} = 1 - c + \frac{i}{n-1}b. \tag{6}$$

Thus, the focal individual produces a total of $KF_{\mathrm{MN},i}$ offspring, of which $mKF_{\mathrm{MN},i}$ disperse, whereas $(1 - m)$ $KF_{\mathrm{MN},i}$ remain in the natal patch.

We consider first the fate of the $mKF_{\mathrm{MN},i}$ dispersing offspring of the focal mutant individual. Each arrives in a separate patch, where they compete with $(1 - m)nK$ locals and mnK other dispersers. As there are n breeding spaces within the patch, the probability that a particular dispersing offspring of the focal mutant survives competition to find a breeding space is $n/(1 + (1 - m)$ $nK + mnK) \approx 1/K$. Thus, the expected number of survivors among the dispersing offspring of the focal mutant is $mKF_{\mathrm{MN},i}/K = mF_{\mathrm{MN},i,}$.

We now consider the fate of the $(1 - m)KF_{\mathrm{MN},i}$ nondispersing offspring of the focal mutant individual, who compete for a breeding space on their natal patch. The competitors include $(1 - m)K\bar{F}_{\mathrm{MN},i}$ nondispersing locals plus $mKnF$ incoming migrants, where $\bar{F}_{\mathrm{MN},i}$ is the average relative fecundity of parents in the focal patch, which is:

$$\bar{F}_{\mathrm{MN},i} = 1 + \frac{i+1}{n}(b - c). \tag{7}$$

Again, there are n breeding spaces available, and so the expected number of successful nondispersing offspring due to the focal mutant individual is

$$\frac{(1-m)KF_{\mathrm{MN},i}}{(1-m)K\bar{F}_{\mathrm{MN},i} + mK}$$

which, to first order in b and c, is:

$$(1-m)\left(1 - c + \frac{i}{n-1}b - (b-c)\left(\frac{i+1}{n}\right)(1-m)\right). \tag{8}$$

By adding the expected number of surviving dispersing and nondispersing offspring we can find the expected fitness of the focal mutant nondisperser, which is:

$$w_{\mathrm{MN},i} = 1 - c + \frac{i}{n-1}b - (1-m)^2(b-c)\frac{i+1}{n}. \tag{9}$$

This expected fitness is conditional on there being i other (nondispersing) mutants in the focal individual's patch. As outlined above, the condition for invasion of the mutant gene is $w_{\mathrm{MN}} > 1$, where w_{MN} is the average fitness of all nondispersing mutant individuals, i.e. $w_{\mathrm{MN}} = E_{\mathrm{MN}}(w_{\mathrm{MN},i})$. This average fitness obtains:

$$w_{\mathrm{MN}} = 1 - c + bE_{\mathrm{MN}}\left(\frac{i}{n-1}\right) - (1-m)^2(b-c)E_{\mathrm{MN}}\left(\frac{i+1}{n}\right). \tag{10}$$

Note that $i/(n - 1)$ is the proportion of (nondisperser) mutants among the social partners of the focal individual, and so the average of this quantity, taken over all nondispersing mutants in the population, is equal to r_{N}, the others-only relatedness experienced by nondispersers. This makes use of the assumption that the mutant is vanishingly rare in the population. Similarly, $(i + 1)/n$ is the proportion of (nondisperser) mutants among all the individuals in the focal individual's patch, including herself; so, averaging this quantity over all nondisperser

mutants in the population obtains R_N, the whole-group relatedness experienced by nondispersers. Making this substitution, we obtain a condition, in the form of Hamilton's rule, for when the helping mutant will invade from rarity:

$$-c + br_N - (1-m)^2(b-c)R_N > 0. \qquad (11)$$

Because we have assumed weak selection, this condition for increase is frequency independent (Rousset 2004, p. 80), and therefore it also describes the progress of the mutant gene when it is no longer rare. Although condition 11 has been derived using a neighbour-modulated fitness approach to kin selection, it readily yields an inclusive fitness interpretation. Helping by nondispersers is favoured when the sum of three components, equal to the inclusive fitness effect of their helping, is positive. First, their help leads to a personal loss of c offspring. Second, their help leads to b extra offspring for the other individuals on the patch, and these are valued at r_N, the relatedness to patch mates. Third, the net increase of $b - c$ offspring on the patch leads to enhanced competition for locals, to the extent that these extra offspring do not disperse and nor do the other locals [i.e. $(1 - m)^2$], and this leads to the competitive exclusion of local offspring (including own offspring) which are valued by R_N, the whole-group relatedness appropriate for nondispersers.

Condition 11 is expressed in terms of fecundity effects, but can be re-written as $-\tilde{c} + \tilde{b}r_N > 0$, in terms of the direct ($-\tilde{c}$) and indirect ($\tilde{b}$) fitness effects of helping. The direct cost to the actor is $\tilde{c} = c + (1-m)^2(b-c)/n$ and the benefit to the recipient is $\tilde{b} = b(1-m)^2 (b-c)(n-1)/n$; both cost and benefit are positive under the assumption $b > c$, and so the helping behaviour is altruistic ($\tilde{c}$ and $\tilde{b} > 0$; Hamilton 1964).

Note that the relatedness coefficients can be written in the form of eqns 4 and 5; making this substitution into inequality 11 obtains an invasion condition that is expressed wholly in terms of model parameters (n, m, b and c):

$$b/c > \frac{n^2(2-m) - (n-1)(1-m)^3}{1 + m(2n - (n-1)(3-m)m - 3)}. \qquad (12)$$

The above analysis has determined when a mutation that incrementally increases (or decreases, if we change the sign of b and c) the level of indiscriminate helping exhibited by nondispersers will invade from rarity under the action of natural selection. The RHS of inequality 12, which we may denote $f(n,m)$, presents a quantity that must be exceeded by the benefit/cost ratio of helping in order for nondispersing individuals to be favoured to indiscriminately help their patch mates. We find that as n increases, the RHS of the inequality becomes larger ($\partial f/\partial n > 0$), i.e. helping is less favoured as the number of individuals per patch increases (Fig. 2a). We find that helping is most readily favoured at intermediate migration rates (there is some threshold $0 < m^* < 1$ such that $\partial f/\partial m < 0$ for $m < m^*$ and $\partial f/\partial m > 0$ for $m > m^*$; Fig. 2a). At the extreme of a very viscous population, $m \to 0$, the condition for helping to be favoured in nondispersers is $b/c > 1 + (2n-1)n$. However, the selection pressure here is vanishingly small (LHS of eqn $11 \to 0$ as $m \to 0$), and so helping is a nearly neutral character in extremely viscous populations and is not expected to be elaborated by natural selection. At the other extreme of a fully mixing population, costly helping is never favoured ($f \to \infty$ as $m \to 1$). Numerical investigation reveals that

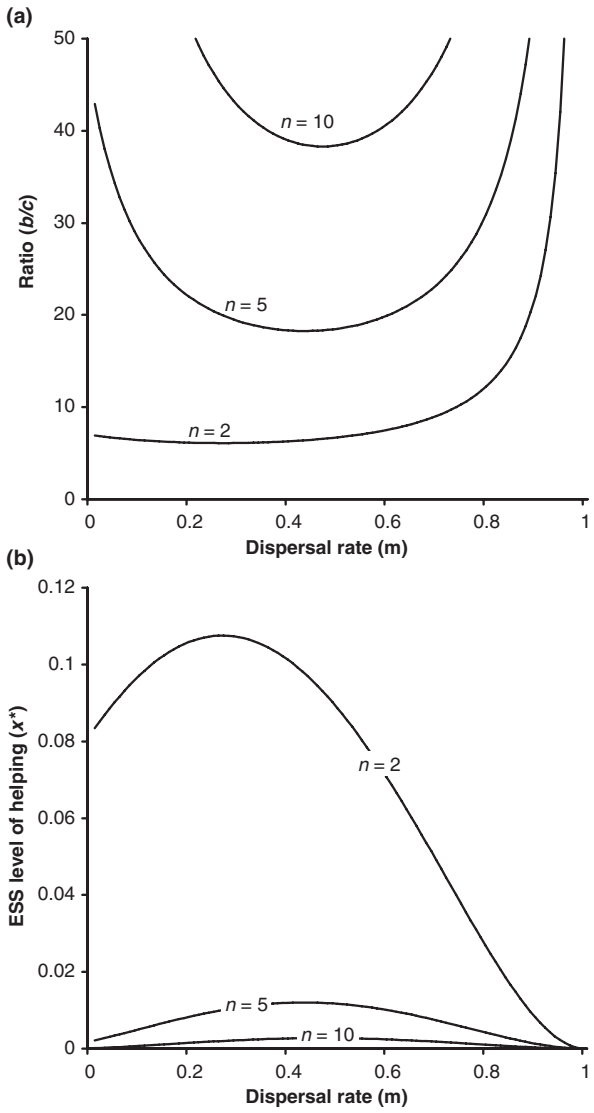

Fig. 2 Indiscriminate helping by nondispersers. (a) The benefit/cost ratio (b/c) below which a mutant gene increasing the level of helping is favoured by selection, for n = 2, 5, 10 individuals per patch, and a range of dispersal rates $0 < m < 1$. (b) The ESS level of helping, assuming $C(x) = kx$ and $B(x) = k\alpha x^{\beta}$ and $\alpha = 4$, $\beta = 0.5$, $k \to 0$, n = 2, 5, 10 and $0 < m < 1$.

the optimum migration rate for helping (i.e. $m = m^*$) is less than 0.5, but converges upon 0.5 as $n \to \infty$, and the condition for helping to evolve at this optimum is approximately $b/c > 4n - 2$.

We have determined when a small increase or decrease in helping by nondispersers will be favoured by natural selection, and we can use this to determine the absolute level of helping that is expected to evolve, i.e. the evolutionarily stable strategy (ESS; Maynard Smith & Price 1973). If we assume that expressing a level x of helping incurs a personal fecundity cost $C(x)$ and an others-only fecundity benefit $B(x)$ (where B, $C << 1$; see Appendix), then the incremental increases in cost and benefit that result from adopting a mutant strategy $\hat{x}$, rather than the resident strategy $\bar{x}$ are $C(\hat{x}) - C(\bar{x}) = C'(\bar{x})(\hat{x} - \bar{x}) = c$ and $B(\hat{x}) - B(\bar{x}) = B'(\bar{x})(\hat{x} - \bar{x}) = b$, respectively. Substituting these into the invasion condition 12, we find that an incremental increase in the helping strategy of nondispersers ($\hat{x} > \bar{x}$) is favoured when the condition is satisfied, an incremental decrease in helping ($\hat{x} < \bar{x}$) is favoured when the reversed condition is satisfied, and a candidate (non-zero) evolutionarily stable strategy (x^*; i.e. a resident strategy that is uninvadable by any mutant strategy $\hat{x}$) is implicitly defined by changing the invasion condition to an equation:

$$\frac{B'(x^*)}{C'(x^*)} = \frac{n^2(2-m) - (n-1)(1-m)^3}{1 + m(2n - (n-1)(3-m)m - 3)}. \qquad (13)$$

Strictly, this condition applies only if the ESS takes a positive value. An explicit solution for the ESS level of helping x^* requires explicit functional forms for $C(x)$ and $B(x)$. Numerical solutions for an illustrative example are given in Fig. 2b.

The evolution of harming in dispersers

We now investigate the evolutionary success of a vanishingly rare mutant gene that causes bearers to express harming behaviour upon dispersal to a new patch, and which confers a relative personal fecundity cost a and a further relative fecundity cost d that is shared equally among the $n - 1$ other individuals on the patch. Again we express a and d relative to baseline fecundity, and we assume that a, $d << 1$. This mutant gene will spread if, on average, it increases fitness of its bearers (whether they are dispersers or nondispersers), either due to direct or indirect fitness effects. There are no direct or indirect effects of the mutant gene on nondispersing mutants as these do not express the gene nor do they encounter dispersing mutants who do express the harming behaviour. Therefore, the mutant gene can only invade from rarity if it increases the average fitness of mutant dispersers (see Appendix for mathematical details). The relative fecundity of a mutant disperser is simply:

$$F_{MD} = 1 - a. \qquad (14)$$

This focal individual produces a total of KF_{MD} offspring, of which mKF_{MD} disperse to a new patch and $(1 - m)KF_{MD}$ remain in the natal patch. Using the same approach as in the previous section, the probability that any one of the dispersing offspring of the focal mutant will survive the competition and find a breeding space is $1/K$. This means that the expected number of dispersing offspring due to the focal individual is $mF_{MD} = m(1 - a)$.

We now consider the fate of the $(1 - m)KF_{MD}$ nondispersing offspring of the focal mutant disperser, which remain on the natal patch to compete for breeding spaces. The competitors consist of $(1 - m)K\bar{F}_{MD}$ nondispersing locals, plus mKn incoming migrants, where $\bar{F}_{MD}$ is the average relative fecundity of parents in the focal patch and is given by:

$$\bar{F}_{MD} = 1 - \frac{1}{n}(a + d). \qquad (15)$$

Again, there are n breeding spaces available, and so the expected number of successful nondispersing offspring due to the focal mutant individual is $\frac{n(1-m)KF_{MD}}{(1-m)K\bar{F}_{MD}n + mKn}$, which is, to first order in a and d, equal to:

$$(1 - m)(1 - a) - (1 - m)^2(a + d)\frac{1}{n}. \qquad (16)$$

By adding the expected numbers of dispersing and nondispersing offspring together we can find the average fitness of a mutant disperser, and this is:

$$w_{MD} = 1 - a + (a + d)(1 - m)^2\frac{1}{n}. \qquad (17)$$

Note that $1/n$ is equal to R_D, the relatedness coefficient for the whole patch the mutant is found in (for dispersing mutants, r_D is 0). In order for the mutant gene to invade from rarity, it must provide a fitness advantage, i.e. $w_{MD} > 1$. This gives an inequality in the form of Hamilton's rule:

$$-a + (a + d)(1 - m)^2\frac{1}{n} > 0 \qquad (18)$$

Again, we can derive an inclusive fitness interpretation. Harming by dispersers is favoured when the sum of three components, equal to the inclusive fitness effect of harming, is positive. First, the harming act leads to a personal loss of a offspring. Second, the act will inflict a cost d on fellow patch mates; but, as these are related by $r_D = 0$, this component of inclusive fitness is zero. Third, the net decrease of $a+d$ offspring on the patch leads to decreased competition for locals, to the extent that dispersal does not occur, and hence an effective increase in local offspring (including the mutants own offspring) that would otherwise have been competitively excluded. These are valued by $R_D = 1/n$, the whole-group relatedness for dispersing mutants.

Note that condition 18 can be recovered from condition 11 by substituting $a = c$ and $d = -b$ and

replacing the relatedness coefficients for nondispersers (r_N and R_N) with those for dispersers (r_D and R_D). As we did for condition 11, condition 18 can be re-expressed in terms of fitness effects, i.e. $-\tilde{c}+\tilde{b}r_D$, where $\tilde{c}$ and $\tilde{b}$ are the direct fitness cost and indirect fitness benefit of harming. We find that $\tilde{c}=a-(1-m)^2(a+d)/n$ and $\tilde{b}=-d+(1-m)^2(a+d)(n-1)/n$; both cost and benefit are negative when condition 18 is satisfied so harming, whenever it is favoured by selection, is selfish ($\tilde{c}$ and $\tilde{b}<0$; Hamilton 1964).

We now rearrange inequality 18 to give a condition:

$$d/a > \frac{n}{(1-m)^2} - 1, \tag{19}$$

which can be used to determine when a mutation that incrementally increases (or decreases) the level of indiscriminate harming exhibited by dispersers will invade from rarity under the action of natural selection. It is helpful to denote the RHS of condition 19 by $g(n,m)$. We find that, as with the helping analysis considered in the previous section, harming by dispersers is progressively more difficult to evolve as the number of individuals per patch increases (i.e. $\partial g/\partial n > 0$; Fig. 3a). Moreover, harming is less easily favoured with increasing dispersal rates (i.e. $\partial g/\partial m > 0$; Fig. 3a): at the extreme of a very viscous population ($m \to 0$) harming is favoured whenever the ratio of the harm inflicted on patch mates to harm inflicted upon self is greater than the number of individuals the disperser shares a patch with (i.e. $d/a > n-1$; Hamilton 1971), and at the opposite extreme of a fully mixing population harming will not evolve under any circumstances ($g \to \infty$ as $m \to 1$). We note that, if the dispersal rate is tuned so as to be most favourable to harming (i.e. $m \to 0$) then harming by dispersers evolves more easily than does helping by nondispersers in the corresponding best-case scenario for the latter to evolve (i.e. $m = m^*$; and $n-1 < 4n-2$).

Again, we can locate a candidate ESS to examine the absolute level of harming that we expect to evolve among dispersing individuals. We assume that expressing a level y of harming incurs a personal fecundity cost $A(y)$ and an others-only fecundity cost of $D(y)$ (where A, $D << 1$; see Appendix), hence the incremental increases in personal cost and damage to victims that results from adopting the mutant strategy $\hat{y}$ rather than the resident strategy $\bar{y}$ are given by $D(\hat{y})-D(\bar{y})=D'(\bar{y})(\hat{y}-\bar{y})=d$ and $A(\hat{y})-A(\bar{y})=A'(\bar{y})(\hat{y}-\bar{y})=a$ respectively. Substituting these into invasion condition 18, we find that an incremental increase in the harming strategy of nondispersers ($\hat{y}>\bar{y}$) is favoured when the condition is satisfied, an incremental decrease in harming ($\hat{y}<y$) is favoured when the condition is reversed, and a candidate ESS (y^*; i.e. a resident strategy that is uninvadable by any mutant strategy y) is implicitly defined by changing the invasion condition to an equation:

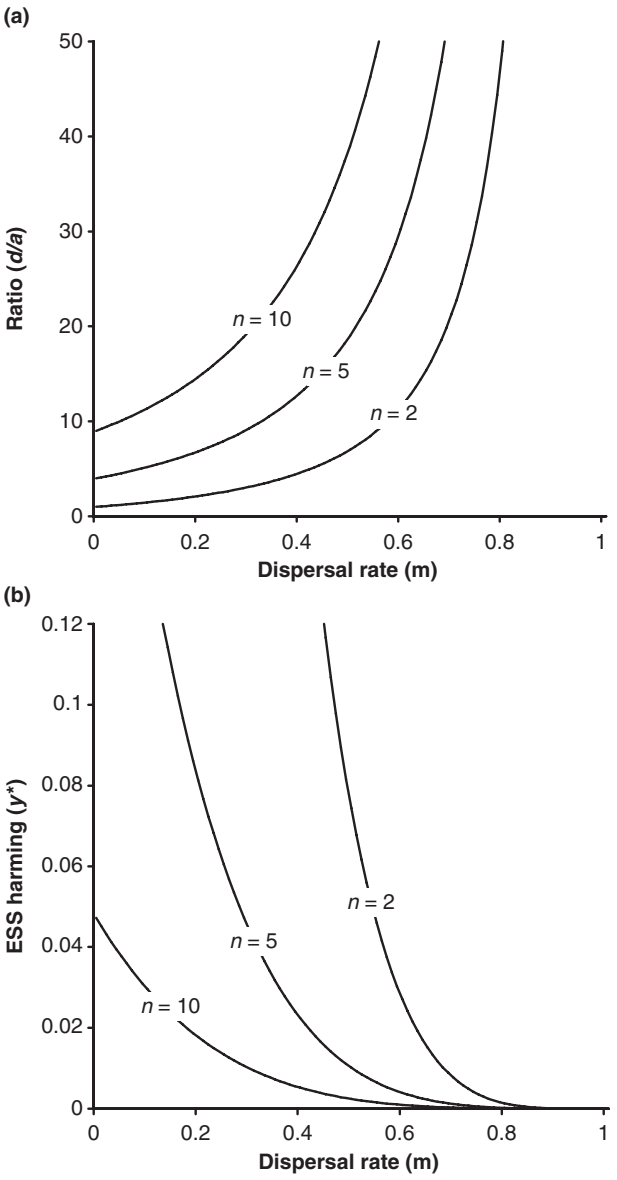

Fig. 3 Indiscriminate harming by dispersers. (a) The harm/cost ratio (d/a) below which a mutant gene increasing the level of harming is favoured by selection, for n = 2, 5, 10 individuals per patch, and a range of dispersal rates $0 < m < 1$. (b) The ESS level of harming, assuming $A(y) = ky$ and $D(y) = k\alpha y^{\beta}$ and $\alpha = 4$, $\beta = 0.5$, $k \to 0$, n = 2, 5, 10 and $0 < m < 1$.

$$\frac{D'(y^*)}{A'(y*)} = \frac{n}{(1-m)^2} - 1. \tag{20}$$

Again, this applies strictly to ESSs that take intermediate values. An explicit solution for the ESS level of helping y^* requires explicit functional forms for $D(y)$ and $A(y)$. A numerical solution for an illustrative example is given in Fig. 3b. Note that, although we have modelled helping and harming behaviours as separate traits, an

alternative (but equivalent) analysis could treat this social behaviour as a single trait varying from extreme conflict to extreme cooperation.

Discussion

Hamilton (1964, 1971) suggested that indiscriminate helping behaviour should be commonest in viscous populations, because these were associated with greater relatedness between neighbouring individuals. However, more recent work has emphasized the potential for kin competition to inhibit helping behaviour in viscous populations (reviewed by Queller 1992 and West et al. 2002), and in the simplest scenario of an inelastic island model there is no net impact of dispersal rate upon the evolution of helping (Taylor 1992a). There is much interest in determining which minimal modifications to the basic model can be made in order to allow social behaviour to flourish. We have examined the possibility that social behaviour is adjusted conditionally upon the dispersal status of the actor, and have shown that this allows both helping and harming behaviour to evolve in viscous populations. Because no external information need be assessed by the individual – for example, its dispersal status may be inferred according to whether it is a winged or wingless morph or whether it experienced a period of cooling or desiccation associated with aerial dispersion – and hence there is no reliance on complex cognitive function, this appears to be a rather general mechanism for the evolution of social behaviours in viscous populations.

In the simplest case of an inelastic island model with dispersal-independent social behaviour (Taylor 1992a), the effect of increased relatedness associated with population viscosity is exactly cancelled by the effect of increased competition between social partners. Thus, both helping and harming behaviours evolve no more readily in viscous populations than they do in fully mixing populations. By contrast, dispersal-dependent social behaviour decouples relatedness and competition so that, although dispersers and nondispersers experience the same level of competition with their social partners, they differ in their genetic relatedness. In particular, nondispersers are more related than average to their social partners and dispersers are less related than average to their social partners, and this can allow for the evolution of indiscriminate helping by nondispersers and indiscriminate harming by dispersers. In the special cases of a completely viscous or completely mixing population, individuals are either all nondispersers or all dispersers, and so at these extremes our analysis recovers Taylor's (1992a) results and here we predict that neither costly helping nor costly harming will evolve (for example, although dispersers are expected to invest more into harming as the population becomes more viscous, the frequency of dispersers is decreasing towards zero). Thus, we predict that costly helping and harming behaviours are most abundant at intermediate population viscosity.

We have found a basic asymmetry between helping and harming evolution such that, over all, helping is relatively weakly favoured and harming is relatively strongly favoured in viscous populations. This is because zero relatedness (on average) is guaranteed by one's own dispersal, irrespective of the population level of dispersal, whereas the relatedness achieved by not dispersing is nonzero only insofar as one's social partners are also nondispersers. In a highly viscous population, conditions are favourable to the evolution of harming by dispersers, because they experience strong local competition and zero relatedness to their social partners. Conversely, although the low competition experienced in highly dispersive populations is conducive to helping, even those individuals not dispersing will have low relatedness to their social partners, who will often be immigrants. So, although the level of harming exhibited by a disperser is predicted to increase with increasing population viscosity, the level of helping by a nondisperser is predicted to maximize at intermediate population viscosity. Thus, if a sample of species were collected that had relatively low viscosity, then we would support Hamilton's (1964) prediction that, within this sample, there will be a positive correlation between viscosity and the level of helping. However, a sample of species with relatively high population viscosities might show the reverse trend.

The implications of this simple mechanism for the evolution of indiscriminate helping by nondispersers were also considered by Perrin & Lehmann (2001). However, in that previous study, the authors evaluated fitness by considering the fecundity of actors averaged over the disperser and nondisperser classes (their eqn 5a), instead of averaging the fitness component obtained by disperser and nondisperser, with the result that the fitness consequences for disperser and nondisperser were not properly separated. This led to an underestimation of the intensity of kin competition (Lehmann 2003, pp. 14–16; L. Lehmann, personal communication). Thus, the results of the present analysis on the evolution of helping are quantitatively and also qualitatively different from those obtained previously. For example, Perrin & Lehmann's (2001) analysis predicted that the evolutionarily stable level of helping should increase monotonically with population viscosity, whereas we have described a dome-shaped relationship, with maximal helping at intermediate viscosity. In addition, Perrin & Lehmann (2001) did not consider the evolution of indiscriminate harming by dispersing individuals.

Our model of dispersal-dependent social behaviour may provide insight into the evolution of social parasitism in aphids, whereby individuals disperse to new colonies and, upon arrival, downregulate their usual

altruistic colony-defence function and upregulate selfish, personal reproduction (Abbot et al. 2001; Foster 2002). Although social parasitism is known in several taxa, this might usually be attributed to direct discrimination of own versus alien colonies. However, aphids are presently understood to lack the faculty for kin recognition that would permit them to directly assess genetic relatedness of their neighbours, and so it may be the simple act of dispersal that provides the cue for this facultative change in their social behaviour (Foster 2002). The present analysis is also conceptually related to a study of dispersal-dependent sex allocation by Taylor & Crespi (1994), which predicted (and empirically tested and confirmed in a species of thrips) that dispersing and nondispersing individuals would allocate differentially to sons vs. daughters due to the correlation between dispersal status and relatedness to neighbours. In particular, Taylor & Crespi's (1994) diploidy model predicted that nondispersers should exhibit a slight bias towards daughters, and dispersers a strong bias towards sons. This is analogous to our result that nondispersers are weakly favoured to help and dispersers are strongly favoured to harm, and indeed in both cases the pattern is due to the different relatedness experienced by dispersers and nondispersers.

A correlation between dispersal status and social behaviour can have important consequences for population demography, suggesting avenues for future research directions. By modifying the fitness differences between dispersers and nondispersers, this association can drive selection for changes in the rate and pattern of dispersal. Perrin & Lehmann (2001) considered the role for indiscriminate helping by nondispersers to mediate the evolution of dispersal, and the subsequent feedback on the helping trait itself. As with our analysis, Perrin & Lehmann (2001) found that helping was only weakly favoured in viscous populations, and hence the co-evolution of helping and dispersal did not have a dramatic impact on either trait. However, the role for harming by dispersers to mediate the evolution of population viscosity remains to be considered, and our analysis suggests potential for rather stronger effects. An association between dispersal status and social behaviour might also impact upon inter-specific interactions, influencing the distribution and abundance of competing species. For example, Duckworth and Badyaev (2007) have described a positive correlation between dispersal and aggression in blue birds that appears to mediate species range expansions.

More generally, we have highlighted that the kin selection coefficient of relatedness describes the conditional information that an individual has with regard to the genetic similarity of its social partners, rather than their actual genetic similarity (e.g. Frank 1998, ch. 6). Typical extensions of Taylor's (1992a) simple island model that promote the evolution of social behaviour in viscous populations do so by decoupling the genetic structure of the population from the intensity of local competition; for example, by introducing 'elasticity' so that cooperative patches can grow in size, localized competition is reduced while genetic structuring remains high. By contrast, the degree of genetic structure and the intensity of localized competition described in our model is exactly as occurs in Taylor's (1992a) model, and the decoupling of relatedness and localized competition occurs through the use of conditional information: whether or not the individual dispersed.

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Appendix

Natural selection and social behaviour

We consider a locus G controlling social behaviour of one type of individual (i.e. nondisperser or disperser), and we denote an individual's genetic 'breeding' value (Price 1970; Falconer 1981; Grafen 1985) for the behaviour as g. The change in the population average breeding value is given by:

$$\Delta\bar{g} = \sum_{u\in U} c_u \ \mathrm{cov}_u(w/\bar{w}_u, g) + \sum_{v\in V} c_v \ \mathrm{cov}_v(w/\bar{w}_v, g), \quad \text{(A1)}$$

where U is the set of all nondisperser classes (defined according to the number of nondispersers among their social partners), V is the set of all disperser classes (also defined according to the number of nondispersers among their social partners), c_u and c_v are the class reproductive values of nondisperser class u and disperser class v respectively; $\bar{w}_u$ and $\bar{w}_v$ are the average fitnesses for individuals of class u and v respectively; and cov is the statistical covariance taken over individuals of the indicated set (Price 1970; Taylor 1996).

Natural selection and social behaviour in nondispersers

Consider first that the gene affects social behaviour in nondispersers only. We can expand the selection component due to dispersers as:

$$\sum_{v\in V} c_v \ \mathrm{cov}_v(w/\bar{w}_v, g) = \sum_{v\in V} c_v\beta_v(w/\bar{w}_v, g) \ \mathrm{var}_v(g), \quad \text{(A2)}$$

where β is a least-squares linear regression, and var is the statistical variance, both taken over the indicated class of

individuals. Expanding the regression into partial regressions, we have:

$$\begin{aligned}\beta_v(w/\bar{w}_v, g) = {} & \beta_v(w/\bar{w}_v, g|g'_N \;\&\; g'_D) \\ & + \beta_v(w/\bar{w}_v, g'_N|g \;\&\; g'_D)\beta_v(g'_N, g) \\ & + \beta_v(w/\bar{w}_v, g'_D|g'_N \;\&\; g)\beta_v(g'_D, g), \end{aligned} \quad \text{(A3)}$$

where g'_N and g'_D are the average breeding values of the individual's nondisperser and disperser social partners respectively. Note that, because the gene is not expressed in dispersers, $\beta_v(w/\bar{w}_v, g|g'_N \;\&\; g'_D) = \beta_v(w/\bar{w}_v, g'_D|g \;\&\; g'_N) = 0$. Also, because the focal individual is a disperser, it is unrelated to the other individuals on its patch, and so $\beta_v(g'_N, g) = \beta_v(g'_D, g) = 0$. Hence, $\beta_v(w/\bar{w}_v, g) = 0$ and so, from eqns (A1) and (A2), selection on the gene is given by:

$$\Delta\bar{g} = \sum_{u\in U} c_u \operatorname{cov}_u(w/\bar{w}_u, g). \quad \text{(A4)}$$

Thus, if the gene is expressed only in nondispersers, selection for or against the gene is determined solely by the fitness consequences for nondispersers.

Natural selection and social behaviour in dispersers

Alternatively, consider that the gene affects social behaviour in dispersers only. We can expand the selection component due to nondispersers

$$\sum_{u\in U} c_u \operatorname{cov}_u(w/\bar{w}_u, g) = \sum_{u\in U} c_u \beta_u(w/\bar{w}_u, g) \operatorname{var}_u(g), \quad \text{(A5)}$$

and,expanding the regression into partial regressions, we have:

$$\begin{aligned}\beta_u(w/\bar{w}_u, g) = {} & \beta_u(w/\bar{w}_u, g|g'_N \;\&\; g'_D) \\ & + \beta_u(w/\bar{w}_u, g'_N|g \;\&\; g'_D)\beta_u(g'_N, g) \\ & + \beta_u(w/\bar{w}_u, g'_D|g'_N \;\&\; g)\beta_u(g'_D, g), \end{aligned} \quad \text{(A6)}$$

where g'_N and g'_D are once again the average breeding values of the individual's nondisperser and disperser social partners respectively. Note that, because the gene is not expressed in nondispersers, $\beta_u(w/\bar{w}_u, g|g'_N \;\&\; g'_D) = \beta_u(w/\bar{w}_u, g'_N|g \;\&\; g'_D) = 0$. Moreover, because the focal individual is unrelated to its disperser social partners, $\beta_u(g'_D, g) = 0$. Hence, $\beta_u(w/\bar{w}_u, g) = 0$ and so, from eqns A1 and A5, selection on the gene is given by:

$$\Delta\bar{g} = \sum_{v\in V} c_v \operatorname{cov}_v(w/\bar{w}_v, g). \quad \text{(A7)}$$

Thus, if the gene is expressed only by dispersers, selection for or against the gene is determined solely by the fitness consequences for dispersers.

Invasion condition for nondisperser social behaviour

We now determine the invasion condition for a gene affecting social behaviour of nondispersers, and we do so under the assumptions that: (1) the gene has vanishingly small impact on the phenotype; and (2) social behaviours in general have vanishingly small impact upon fecundity. Because the population is not growing in size, we can write the fitness of a focal nondisperser individual $j \in J_u$ in class u as $w_{uj} = 1 + \delta w_{uj}$, the average fitness of individuals of this class as $\bar{w}_u = 1 + \delta\bar{w}_u$, and the reproductive value of this class as $c_u = q_u + \delta c_u$, where q_u is the proportion of all individuals in the population that belong to class u. These three deviation terms are all vanishingly small quantities (δw_{uj}, $\delta\bar{w}_u$, $\delta c_u \ll 1$).

Taking eqn A4, and making the individual notation more explicit, the action of selection is given by:

$$\begin{aligned}\Delta\bar{g} &= \sum_{u\in U}(q_u + \delta c_u)\operatorname{cov}_u(w_{uj}/\bar{w}_u, g_{uj}) \\ &= \sum_{u\in U} q_u \operatorname{cov}_u(w_{uj}/\bar{w}_u, g_{uj}) + \sum_{u\in U}\delta c_u \operatorname{cov}_u(w_{uj}/\bar{w}_u, g_{uj}). \end{aligned} \quad \text{(A8)}$$

Due to the weak-selection assumption, $\operatorname{cov}_u(w_{uj}/\bar{w}_u, g_{uj}) \ll 1$, and so the second term of eqn A8 is negligible. Using $w_{uj} = 1 + \delta w_{uj}$ and $\bar{w}_u = 1 + \delta\bar{w}_u$, and discarding higher order terms of δ, we can rewrite eqn A8 as:

$$\begin{aligned}\Delta\bar{g} &= \sum_{u\in U} q_u \operatorname{cov}_u(w_{uj} - \delta\bar{w}_u, g_{uj}) \\ &= \sum_{u\in U} q_u \operatorname{cov}_u(w_{uj}, g_{uj}) \\ &= q_N \sum_{u\in U}\frac{q_u}{q_N}\left(\sum_{j\in J_u}\frac{q_{uj}}{q_u} w_{uj}g_{uj} - w_u g_u\right), \end{aligned} \quad \text{(A9)}$$

where $q_N = \sum_U q_u = 1 - m$ is the total fraction of individuals in the population that are nondispersers. Making the standard assumption that gene frequencies are equal in all classes, i.e. $g_u = \bar{g}$ for all $u \in U$, this obtains:

$$\begin{aligned}\Delta\bar{g} &= (1-m)\left(\sum_{u\in U}\sum_{j\in J_u}\frac{q_{uj}}{q_N} w_{uj}g_{uj} - \bar{w}_N\bar{g}\right) \\ &= (1-m)\operatorname{cov}_N(w_{uj}, g_{uj}), \end{aligned} \quad \text{(A10)}$$

where cov_N is a covariance taken over the set of all nondisperser individuals in the population. Natural selection acts to increase the average genetic breeding value of the population when $\Delta\bar{g} > 0$. In order to track the invasion success of a rare variant gene, we may assign it a value of $g = 1$ and all its alleles a value of $g = 0$, in which case we can write $\Delta\bar{g} = (1-m)\bar{g}(w_{MN} - \bar{w}_N)$, where w_{MN} is the average fitness of all the nondisperser carriers of the variant gene. Hence, the variant gene invades if, on average, it increases the fitness of its nondisperser carriers above that of the average nondisperser individual ($w_{MN} > \bar{w}_N$).

Invasion condition for disperser social behaviour

The action of selection upon genes for disperser social behaviour (eqn A7) has the same form as that for nondisperser social behaviour (eqn A4), and an invasion condition can be determined by following the procedure of the last section and simply switching the nondisperser notation to the corresponding disperser notation (i.e. u to v, U to V and N to D). This reveals that a rare genetic variant that alters the social behaviour of disperser individuals is favoured if the average fitness of its disperser carriers is greater than the average fitness of all dispersers in the population ($w_{\mathrm{MD}} > \bar{w}_{\mathrm{D}}$).