Social Evolution in Class-Structured Populations

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Dedicated to my parents, Isabel and Domingos Rodrigues,

my brother Alexandre, and my sister Cristiana.

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Declaration

The work presented in this thesis has been composed by myself, and it results from my own research, except when stated otherwise. This work has not been submitted for any other degree or professional qualification.

All chapters were a result of my own work. Chapter 2 is an essay that I wrote. Chapters 3-5 result from my own work and were supervised by Andy Gardner, who is also a co-author. These chapters are now published: chapter 3, "Rodrigues, A. M. M., and A. Gardner. 2012. Evolution of helping and harming in heterogeneous populations. *Evolution* 66, 2065-2079"; chapter 4, "Rodrigues, A. M. M., and A. Gardner. 2013. Evolution of helping and harming in heterogeneous in viscous populations when group size varies. *The American Naturalist* 181, 609-622"; and chapter 5, ": Rodrigues, A. M. M., and A. Gardner. 2013. Evolution of helping and harming in heterogeneous groups. *Evolution* 67, 2284-2298". Chapter 6 is a collaborative work with Prof Rufus A. Johnstone. He and I designed the paper. I wrote the first draft of the manuscript, and both authors contributed to revisions. Chapter 7 is a collaborative work with Dr Tiffany B. Taylor. She and I designed the paper. I wrote the first draft of the manuscript, and both authors contributed to revisions. Appendix 4 is the result of a collaborative work with Dr Tiffany B. Taylor, and it was supervised by Andy Gardner and Angus Buckling. Dr Taylor did the empirical work, and I did the theoretical work. This paper is now in press in the *Journal of Evolutionary Biology*. Appendix 5 is the result of a collaborative work with Siobhan O'Brien and Angus Buckling. O'Brien did the empirical work, and I did the theoretical work. This paper is now in press in the journal *Proceedings of the Royal Society B*.

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Abstract

Inclusive fitness theory concerns the study of social traits. Often, individuals differ in their phenotype (e.g. size, weight, nutritional state) independently of their genetic make up, that is, individuals differ in their quality. Individuals can then be classified into different "classes" according to their quality, which enable us to understand social evolution in class-structured populations. This is important because individuals in natural populations often differ in quality, either because of intrinsic factors (e.g. size), or extrinsic factors (e.g. resource availability). My thesis concerns the evolution of social traits in class-structured populations. In chapter 1, I make a brief introduction to my thesis, providing the abstract of each chapter. In chapter 2, I outline a general theory of individual quality, where I show how individual quality impacts social evolution in two fundamental ways. In chapter 3, I show that resource heterogeneity greatly influences the evolution of conditional social behaviour. In chapter 4, I show that temporal group-size heterogeneity promotes the evolution of both conditional helping and harming. In chapter 5, I analyse the effect of individual quality on kin selection. I find that individual quality has an important impact in kin selection, which can lead to extreme forms of social behaviour. In chapter 6, I show that stable environments promote the evolution of negative density-dependent dispersal, while unstable environments promote the evolution of positive density-dependent dispersal. In chapter 7, I show that budding and low local quality promote the evolution of dispersal and cooperation.

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Chapter 1. Introduction

1.1. Thesis outline

Charles R. Darwin's *Origin of Species* is largely about the evolution of traits that affect the fitness of their bearers but not the fitness of their bearers' neighbours (Darwin 1859). Traits that do affect the fitness of their bearers' neighbours are called social and explaining their evolution was one of Darwin's special difficulties. When discussing these traits he focused on the sterility of neuters in insect-communities. Darwin was puzzled by the fact that these neuters differ widely from their reproductive counterparts, and by the fact that they could pass on their caste-specific traits, given that neuters do not reproduce.

A fully developed theory that explains social traits arrived more than a century after the publication of Darwin's *Origin*. William D. Hamilton in his 1964 article *The genetical evolution of social behaviour* provided a fully developed theory that explains the evolution of social traits. He based his theory on a conveniently simple model that highlighted the main components of his theory. He made the crucial definition of inclusive fitness as the target of natural selection. However, his theory left implicit several of its complexities. Despite this, Hamilton was able to extrapolate from his simple model, trough verbal arguments, to get some of the complexities of the theory just about right.

Hamilton (1964) argued that non-genetic differences among individuals (i.e. non-genetic phenotypic differences or quality) could influence the costs and benefits of social behaviours, such that some individuals could have a higher tendency to express cooperation than others. In particular he reasoned that: first, an individual that is no longer able to reproduce cannot "cause any further self-effects"; and second, an individual in such condition "may be expected to be entirely altruistic". In chapter 2, I explore the consequences of phenotypic variation to kin selection.

Central to Hamilton's inclusive fitness theory is relatedness, which is expected to be an important correlate of cooperative traits. In his theory, Hamilton went a step further and connected "vagrancy" (dispersal or degree of population viscosity) with relatedness. This allowed him to connect aspects of species ecology with relatedness, and consequently connect ecology and social behaviour. He suggested that low dispersal could lead to high relatedness, and therefore to more cooperation. He also pointed out that high population viscosity could lead to competition among related individuals. However, he considered that this would not be sufficient to stop the evolution of cooperation.

Richard D. Alexander (1974) revisited Hamilton's (1964) argument about the link between population viscosity and cooperation (reviewed by Frank 2013). Contrary to Hamilton's idea, Alexander suggested that kin competition could be sufficiently strong to completely negate the kin selected benefits of population viscosity. Several theoretical models have now shown that this may, in fact, be the case (e.g. Bulmer 1986; Taylor 1992a; Wilson et al. 1992). Most of these models assume homogeneous environments. In chapters 3 and 4, I investigate how dispersal (degree of populations viscosity) mediates the evolution of social traits when populations are heterogeneous.

In chapter 5, I bring population viscosity and non-genetic phenotypic variation (i.e. quality) together in a single theoretical framework. I study how this phenotypic variation among group members and population viscosity simultaneously mediate kin selection.

Hamilton (1964) suggested that kin selection mediated the evolution of vagrancy (i.e. dispersal). Classic dispersal theory predicted that meaningful levels of dispersal would only evolve in temporally variable environments (Gadgil 1971; Van Valen 1971; McPeek and Holt 1992). Hamilton and May (1977) showed that stable and saturated environments would also be conducive to the evolution of significant levels of dispersal. In chapter 6, I investigate how variable local density and kin competition influence the evolution of unconditional and conditional dispersal.

Hamilton (1964) suggested that stable environments would lead to lower dispersal rates, and therefore they should promote the evolution of cooperation. By contrast, he suggested that unstable environments would select for higher dispersal rates, and therefore they would disfavour the evolution of cooperation. In chapter 7, I study the interaction between dispersal and cooperation.

A common thread of my thesis is that individuals may differ in quality. These differences arise because individuals may differ in size, weight, nutritional status, and in many other ways, independently of their genetic make up. In these cases, we can use class-structured population models to analyse the action of natural selection. Individuals are classified into "classes" according to their quality (e.g. size, weight), and they are then further defined by their quality-specific survival and fecundity. A fundamental theoretical framework to analyse kin selection in class-structured populations already exists (Charlesworth 1994; Taylor and Frank 1996; Frank 1998). However, it has been relatively neglected. Here I develop this theoretical framework to tackle the biological problems described above.

Finally in chapter 8, I provide a general discussion of my thesis.

1.2. Chapter abstracts

Chapter 2: Non-genetic phenotypic differences among individuals are thought to mediate the expression of kin selected traits. In this essay I develop a conceptual framework to understand how phenotypic differences between individuals may impact the expression of social traits.

Chapter 3: There has been much interest in understanding how demographic factors can mediate social evolution in viscous populations. Here we examine the impact of heterogeneity in patch quality – i.e. the availability of reproductive resources for each breeder – upon the evolution of helping and harming behaviours. We find that, owing to a cancellation of relatedness and kin competition effects, the evolution of obligate and facultative helping and harming is not influenced by the degree of viscosity in populations characterized by either spatial or temporal heterogeneity in patch quality. However, facultative helping and harming may be favoured when there is both spatial and temporal heterogeneity in patch quality, with helping and harming being favoured in both high-quality and low-quality patches. We highlight the prospects of using kin selection theory to explain within-population variation in social behaviour, and point to the need for further theoretical and empirical investigation of this topic.

Chapter 4: Recent years have seen a huge interest in understanding how demographic factors mediate the evolution of social behaviour in viscous populations. Here we study the impact of variation in group size upon the evolution of helping and harming behaviour. Although variation in group size influences the degree of relatedness and the degree of competition between group mates, we find that these effects often exactly cancel so as to give no net impact of variation in group size upon the evolution of helping and harming. Specifically: (1) obligate helping and harming are never mediated by variation in group size; (2) facultative helping and harming are not mediated by variation in group size when this variation is spatial only; (3) facultative helping and harming are mediated by variation in group size only when this variation is temporal or both spatial and temporal; (4) when there is an effect of variation in group size, facultative helping is favoured in big groups and facultative harming is favoured in little groups. Moreover, we find that spatial and temporal heterogeneity in individual fecundity may interact with patch-size heterogeneity to change these predictions, promoting the evolution of harming in big patches and helping in little patches.

Chapter 5: Social groups are often composed of individuals that differ in many respects. Theoretical studies on the evolution of helping and harming behaviours have largely focused upon genetic differences between individuals. However, non-genetic variation between group members is widespread in natural populations, and may mediate differences in individuals' social behaviour. Here, we develop a framework to study how variation in individual quality mediates the evolution of unconditional and conditional social traits. We investigate the scope for the evolution of social traits that are conditional on the quality of the actor and/or recipients. We find that asymmetries in individual quality can lead to the evolution of plastic traits with different individuals expressing helping and harming traits within the same group. In this context, population viscosity can mediate the evolution of social traits, and local competition can promote both helping and harming behaviours. Furthermore, asymmetries in individual quality can lead to the evolution of competition-like traits between clonal individuals. Overall, we highlight the importance of asymmetries in individual quality, including differences in reproductive value and the ability to engage in successful social interactions, in mediating the evolution of helping and harming behaviours.

Chapter 6: Inbreeding, habitat heterogeneity and local competition may all play a role in the evolution of costly dispersal. Here we focus on how local density in a heterogeneous habitat influences the evolution of unconditional and of conditional (density-dependent) dispersal strategies. We find that habitat heterogeneity tends to promote the evolution of higher unconditional dispersal rates. Allowing for conditional dispersal strategies, we find that in stable environments habitat heterogeneity selects for negative density-dependent dispersal, while in temporally variable environments, habitat heterogeneity selects for positive density-dependent dispersal. In general, dispersal of individuals out of low-density patches is much more sensitive to habitat heterogeneity than is dispersal out of high-density patches.

Chapter 7: Limited dispersal increases the genetic structure of populations and can, therefore, be a key factor mediating the evolution of cooperation. In addition, limited dispersal also increases local competition, which is an important factor mediating the evolution of dispersal. This suggests a strong interaction between the evolution of dispersal and cooperation, and yet theoretical models seeking to understand the interplay between these two traits in viscous populations, owing to limited dispersal, are still scarce. Here, we study the evolution of dispersal and how this, in turn, affects the evolution of cooperation. We consider a viscous population with empty patches where individuals form buds during dispersal and competition. We find a positive correlation between dispersal and cooperation. In particular, we discover that unstable environments, where local habitat quality is low, empty patches, and high dispersal cost favour the evolution of dispersal and cooperation. Cooperation is favoured because high dispersal rates decrease local competition, and at the same time high probability of local patch extinction further reduces the importance of local kin competition.

Chapter 8: Here I provide a general discussion of my thesis. In this discussion I revisit Hamilton's theory of how phenotypic differences among individuals affect the costs and benefits of Hamilton's rule, and how population viscosity mediates the evolution of cooperation. I connect these original ideas of Hamilton to the work I have developed in this thesis. I show that my work clarifies some aspects of Hamilton's original ideas in a novel way.

Chapter 2. Individual quality and the evolution of social behaviour.

An animal whose reproduction is definitely finished cannot cause any further self-effects … the behaviour of a post-reproductive animal may be expected to be entirely altruistic, the smallest degree of relationship with the average neighbour being sufficient to favour the selection of a giving trait.

Hamilton (1964, p. 21)

… the self sacrificing tendencies of parental care should follow a course inverse to that of reproductive value.

Hamilton (1966, p. 23)

2.1. Introduction

Inclusive fitness theory explains why individuals sacrifice some of their Darwinian fitness to increase the Darwinian fitness of others (Hamilton 1964, 1970). Hamilton's inclusive fitness theory predicts that individuals are more willing to help others when the beneficiaries of their actions are close relatives, thus relatedness among interacting individuals plays a pivotal role (Hamilton 1964, 1970). Several factors mediate relatedness, and consequently the evolution of cooperation. For example, the mating system should be an important factor mediating the evolution of cooperation, as monogamy should increase relatedness among group members relative to other mating systems (Boomsma 2007, 2009). Comparative studies in insects, birds, and mammals support this hypothesis (Hughes et al. 2008; Cornwallis et al. 2010; Lukas and Clutton-Brock 2012). More generally, inclusive fitness theory provides an explanation for any type of social behaviour, including the evolution of conflict. In general an individual is selected to act such that resources are transferred from less related individuals to more related individuals. This line of reasoning has been used to successfully predict patterns of conflict within families (Trivers 1974; Haig 2002).

Alongside with relatedness, costs and benefits of cooperation also play a key role in inclusive fitness theory, and like relatedness several factors mediate these costs and benefits (Alexander 1974; West-Eberhard 1975; West et al. 2007a). Non-genetic variation in phenotype (i.e. quality) is one of such factors. Although Hamilton was aware that differences in quality could lead to the differential expression of social behaviour, he did not develop a comprehensive theory of how this could occur (Hamilton 1964, 1987). Notwithstanding, he made two important remarks about this issue: first, he suggested that individuals that were unlikely to reproduce again would be highly altruistic (Hamilton 1964; see introductory quote); and second, he suggested that individuals of lower reproductive value should engage more in cooperation (Hamilton 1966; see introductory quote).

Here I suggest that while reproductive value sometimes does influence the expression of social behaviour, this is not always the case. I first review the literature that has considered how differences in quality influence the evolution of social behaviour. I then suggest that quality that influence the expression of social behaviour can fall into two categories, one being reproductive value and the other what we can call social (or somatic) value. I use this theoretical framework to interpret some recent findings about the conditional expression of social behaviour.

2.2. Kin selection and quality

Kin selection concerns the evolution of traits that affect not only the fitness of the actors but also the fitness of the actors' social partners (Hamilton 1963, 1996; Maynard Smith 1964). Hamilton's rule, $-c+br > 0$, is the condition for the evolution of these traits (Hamilton 1963, 1964, 1970; Charnov 1977). The behaviour evolves if the benefit b provided to the recipients times the relatedness r between actor and recipients is greater than the cost c to the actor. Behaviours are classified according to the sign of the cost c and of the benefit b (Table 1; Hamilton 1964; West et al. 2007b). For example, a behaviour is altruistic if the cost c and the benefit b are both positive. Note that costs and benefits must be measured in units of fitness.

Actor	Recipient	
	+	−
−	Altruism	Spite
+	Cooperation	Selfishness

Table 1 | Classification of social behaviours.

Hamilton's rule emphasises the role of relatedness in the evolution of social behaviour, which was extensively developed by Hamilton. He connected relatedness with dispersal (i.e. degree of population viscosity), a major life-history trait (Hamilton 1964). This effectively connected his theory of inclusive fitness with ecology and the evolution of movement. He also connected relatedness with other important traits such as those involved in recognition systems (Hamilton 1964). By comparison, the ecological variables in Hamilton's rule, i.e. costs and benefits, were much less developed by Hamilton. However, he did notice that differences in phenotypes would influence costs and benefits. He noted that post-reproductive individuals would suffer lower costs (Hamilton 1964; see introductory quote), and, to the best of my knowledge, he was the first to propose a link between altruism and reproductive value (Hamilton 1966; see introductory quote). The concept of reproductive value was developed by R. A. Fisher (1930). It was introduced in the context of age-structured populations, and it measures the relative genetic contribution of an individual to future populations (Fisher 1930; Grafen 2006).

The use of reproductive value in kin selection theory has then followed different paths, perhaps with different origins. One idea that emerged early on was that helping should go from low reproductive value individuals to high reproductive value individuals (e.g. West-Eberhard 1975). This idea was often expressed verbally or given in the form of a Hamilton's rule, such as

$$-v_A + rv_B > 0 \tag{1}$$

where: v_A is the reproductive value of the actor; v_B is the reproductive value of the beneficiary; and r is the relatedness between actor and beneficiary. This idea was mostly used to explain patterns of social behaviour in age-structured populations (e.g. Hrdy and Hrdy 1976; Chapais and Schulman 1980; Combes ad Altmann 2001). For example, in hanuman langurs, females form hierarchies where dominant individuals monopolise reproduction. Empirical evidence suggests that dominance occurrs at intermediate ages, which is usually the pattern of reproductive value in many species. Hrdy and Hrdy (1976) suggested that reproductive value explained the social system of female hanuman langurs. In particular, explained why older females conceded their dominant roles to their daughters to become helpers, and, in particular, to defend the group against non-group members.

These ideas about the relationship between reproductive value and kin selection were largely based on verbal arguments, but rigorous models of this idea were still lacking. Charlesworth (1980b) first pointed out the necessity of testing these verbal arguments with rigorous population genetic models. The first formal model of these verbal arguments was developed by Charlesworth and Charnov (1981). They showed that reproductive value would mediate kin selection if the behaviour affected the survival component of fitness, and that the reproductive values in Hamilton's rule were those of the actor and of the recipient in the time step after the action. Thus, if a behaviour was enacted at time t, Hamilton's rule would be given by:

$$-Cv_{\mathrm{A},t+1} + rBv_{\mathrm{R},t+1} > 0. \tag{2}$$

where: C is the marginal survival cost; $v_{\mathrm{A},t+1}$ is the reproductive value of the actor at time t+1; B is the marginal survival benefit; $v_{\mathrm{B},t+1}$ is the reproductive value of the beneficiary at time t+1; and r is the relatedness between actor and beneficiary. By contrast, if the behaviour affected the fecundity component of fitness, kin selection was not mediate by reproductive values. Thus, Hamilton's rule would be given by:

$$-C + rB > 0. \tag{3}$$

More generally, a behaviour may affect different components of fitness or may even affect both components simultaneously. For example, it may affect the survival of the actor while affecting the fecundity of the beneficiary (see Table 2). Overall, Charlesworth and Charnov's (1981) results emphasised the need for formal models and that the relationship between reproductive value and kin selection was more complex than previously thought.

Actor	Recipient	
	Survival	Fecundity
Survival	$-Cv_{A,t+1} + rBv_{R,t+1} > 0$	$-Cv_{A,t+1} + rB > 0$
Fecundity	$-C + rBv_{R,t+1} > 0$	$-C + rB > 0$

Table 2 | Hamilton's rule for different combinations of marginal fitness effects in actor and recipient. Marginal effects affect either survival or fecundity, assuming that offspring's reproductive value is one (following Charlesworth and Charnov 1981).

At the same time, empiricists were developing more complex verbal models that could account for complex patterns of sociality in natural populations. Notably, Rubenstein (1982) developed a verbal argument advocating that in some cases helping should be directed to low reproductive value beneficiaries. Charlesworth and Charnov's (1981) paper represents an important development in understanding how differences in reproductive value drive kin selection, however it was not able to fully address the problems raised by empiricists. One way to address these issues is to further develop equations (2) and (3) in making the marginal cost and benefit quality-dependent (or role-dependent). Let us consider social interactions enacted by individuals X and Y, where the beneficiary is Z. When X is the actor, one can write

$$-C_{X}v_{X,t+1} + rB_{XZ}v_{Z,t+1} > 0, \text{ and} \tag{4}$$

$$-C_{X} + rB_{XZ} > 0, \tag{5}$$

for behaviours affecting survival and fecundity, respectively. Likewise, when Y is the actor, one can write

$$-C_{Y}v_{Y,t+1} + rB_{YZ}v_{Z,t+1} > 0, \text{ and} \tag{6}$$

$$-C_{Y} + rB_{YZ} > 0, \tag{7}$$

for behaviours affecting survival and fecundity, respectively. If individuals differ in marginal costs and benefits (C and B) we say that they have different social (somatic) value (see chapter 5). Note that inequalities (4-7) have more degrees of freedom than, for example, inequality (1). Furthermore, these inequalities follow from a logical extension of previous theory, and therefore they are formally sound (see also chapter 5). Below, I explore how these concepts may help us understanding social evolution.

2.3. Biological examples

In this section I will focus on three biological systems. I will provide some links between inequalities (4-7) and these biological systems.

2.3.1. The social lives of aphids

The aphid *Quadrartus yoshinomiyai* is a gall-forming aphid that has a host alternating life-cycle. Galls are formed in its primary hosts, the tree *Distylium racemosum*, and each gall starts with a single fundatrix (i.e. a single breeding female). Foundresses reproduce parthenogenetically. Aphids go through several developmental stages. Juveniles can develop to become wingless or winged adults. At the end of the season in its primary host, winged adults disperse to the secondary hosts. Here, sexual morphs are produced, and after sexual reproduction, winged females return to the primary host (Uematsu et al. 2007). Predators such as ladybug larvae are a major threat to the colony's reproductive success (Uematsu et al. 2007, 2010), thus it comes as no surprise that aphids have evolved defensive behaviours against these predators. However, unlike eusocial bees, ants or wasps, altruistic defence of the colony is not performed by morphologically differentiated workers that commit early in development to a non-reproductive life. Instead, defence of the colony is maintained by juveniles and elders with no or small morphological differences (Uematsu et al. 2007, 2010, 2013). Reproductive value in aphids may well follow a dome shape, which is characteristic of many other species. This reproductive value pattern matches the level of age-dependent selfishness observed in aphid colonies, where selfish traits are more frequent at intermediate ages and less frequent at extreme ages. Thus, reproductive value may offer an explanation for the age-dependent altruism observed in aphid colonies.

2.3.2. Explosive backpacks

Termites live in social groups where there is division of labour between reproductives and workers (Korb 2008). In some species there is a pronounced dimorphism between reproductives and workers, and workers enjoy little or no direct reproductive success (Korb 2008). Workers' sterility means that they have low or null reproductive value. If reproductive value is a factor in Hamilton's rule, then this would predict that all workers were equally likely to perform altruistic tasks. Consequently, when two tasks are required in the colony, all workers should be equally likely to enact it. However, in the species *Neocapritermes taracua* this is not the case (Šobotník et al. 2012). Thus, Hamilton's rule should not be equal for all workers. Workers exhibit an association between age, mandible sharpness, and task. Younger workers have sharper mandibles and perform maintenance behaviours. By contrast, older workers have worn out mandibles and perform defensive suicidal behaviours (Šobotník et al. 2012). This suggests that workers are adjusting their social behaviour not according to their reproductive value but according to their somatic or phenotypic condition. What could possibly be the Hamilton's rule that workers are "using" to adjust their behaviour adaptively? Because relatedness and reproductive value are identical across the workers caste, they may well use a rule such as: 'If my mandibles are worn out, I will no longer be an efficient maintenance worker, therefore I should become a defensive worker instead'.

2.3.3. Naked mole rats

Naked mole rats usually live in colonies where only one or few queens and kings reproduce (Jarvis 1981). Several life trajectories have been identified. In the species *Heterocephalus glaber*, "frequent workers" grow more slowly, they do not reproduce, and they perform tasks such as food acquisition or tunnel maintenance (Jarvis 1981; Lacey and Sherman 1991). Fast growing individuals represent another life trajectory. These individuals engage less in tasks such as food acquisition or tunnel maintenance (work less), and they can be characterised as "nonworkers" (Jarvis 1981; Lacey and

Sherman 1991). Two hypotheses are compatible with these two different trajectories. First, there is some underlying factor determining the trajectory of these two sets of individuals; or second, there is competition between individuals, and those that win become "nonworkers", while the others become "frequent workers". These helpers / workers have then less chances of ever becoming reproductives, and therefore they have lower reproductive value. By contrast, the bigger "nonworkers" individuals have greater chances of becoming reproductives, and therefore they have greater reproductive value. One would then expect that risky and costly tasks would be performed exclusively by "frequent workers". This is indeed the case, but only for a sub-set of the tasks needed in the colony. "Frequent workers" care for offspring, dig tunnels, and forage for food. However, when a predator (e.g. a snake) threatens the colony, it is the bigger and higher reproductive value individuals that step in to defend the colony (Lacey and Sherman 1991). This would suggest that some factor other than reproductive value influences the behavioural decisions of individuals. An obvious factor would be size. Because "nonworkers" individuals are bigger than "frequent workers", they may stop the predator more easily than smaller individuals. In this particular case, it seems that combining reproductive value with social (somatic) value may be required to fully understand the social system of naked mole rats.

2.3.4. Elaboration of social adaptations

The examples described above suggest that low reproductive value causes individuals to be more altruistic. However, this does not mean that associations between low reproductive value and altruism imply that low reproductive value is the cause of individuals' altruism. In some cases low reproductive value may just be the result of high relatedness among groups members. In these cases, irreversible low reproductive value can then lead to more elaborate altruistic behaviours, but their underlying cause relies on high relatedness and not on low reproductive value.

A recent study has shown that diseased workers of the honeybee species *Apis mellifera* leave their nest, which seems to be an altruistic behaviour to avoid the spread of the infection to other members of the colony (Rueppell et al. 2010). Another study in the ant *Temnothorax unifasciatus* has arrived to a similar conclusion (Heinz and Walter 2010). One could reason that diseased individuals were unlikely to recover leading to low reproductive value, and subsequently to altruistic suicide. However, one has to remember that ant and honeybee workers are already reproductive altruists, which occurs as soon as they develop into workers. Thus, their infectious state does not alter their reproductive value. Instead, their infectious state may greatly undermine their indirect fitness, which is likely to be the selective force behind their altruistic suicide. Thus, in these cases, altruistic suicide due to infection is likely to be a derived trait of eusociality. As such, it is not a change in the reproductive value resulting from infection that drives the evolution of the behaviour. It would be interesting to study whether these types of behaviour ever evolved in different contexts (e.g. Refardt et al. 2013).

2.4. Discussion

Relatedness is a key factor in social evolution as it explains a wide range of phenomena. It explains why monogamy is ancestral to eusocial insects, and cooperative breeders (Boomsma 2007; Hughes et al. 2008; Cornwallis et al. 2010; Lukas and Clutton-Brock 2012). It explains why offspring try to get more resources than those a mother is willing to give to them (Trivers 1974; Haig 2002). However, in many other cases relatedness is not sufficient to explain certain aspects of social behaviour. In these cases, differences in quality may be key to explaining the expression of social behaviour. Hamilton was aware that differences in quality could mediate the evolution of social behaviour, however, he did not pursue this line of research comprehensively.

The biological examples provided above suggest that early work on how quality mediates kin selection can be expanded to explicitly take into account several aspects of organisms' biology. In the first example (aphids), juveniles (J) and elders (E) may have identical reproductive value ($v_J = v_E$), which is lower than that of adults (A). Thus, $v_J = v_E < v_A$. This may lower the cost of altruism for juveniles and elders, and therefore it may explain why juveniles and elders behave more altruistically than adults. In addition, and for the same reason, the benefits of altruism should be higher if adults are the main recipients of altruism, which further favours the division of labour between juveniles and elders, on the one hand, and adults, on the other hand.

In the second example (termites), the reproductive value of juvenile and elder workers is presumably zero or small but similar ($v_J = v_E$), however they still exhibit (somatic) division of labour. It may be that juveniles are more efficient at nursing and / or foraging than elders, and therefore, for these particular tasks, juveniles provide higher benefits than elders ($B_J > B_E$). This may explain why juvenile workers perform these particular tasks, while elder workers go on to fend predators off. Note that additional factors may also play a role. For example, the death of a good worker may entail an additional inclusive fitness cost for the worker, and this should further prevent the evolution of altruistic suicide in juvenile workers.

In the third example (naked mole rats), the reproductive value of nonworkers (N) should be higher than that of frequent workers (F). Thus, $v_N > v_F$. Despite this, nonworkers seem to be more active defending the colony against predators than frequent workers. This may be explained by the greater size of nonworkers, which may improve their efficiency at fighting predators off. Thus, $B_F > B_N$ for this particular task. An alternative explanation (not mutually exclusive) is that nonworkers (N) have more to lose in terms of future direct reproduction, if a predator threatens the survival of the colony.

To conclude, quality influences kin selection in many different ways. Expanding Hamilton's rule to describe explicitly key aspects of organisms' biology may be a useful way to conceptualise in a systematic and synthetic manner these complexities. Future work should expand the range of models that presently exist to help making further progress in this promising area of research.

Chapter 3. Evolution of helping and harming in heterogeneous populations.

3.1. Introduction

One of the major challenges for evolutionary biology is to explain the evolution of cooperative behaviour (Maynard Smith and Szathmáry 1995; Hamilton 1996; West et al. 2007a). Natural selection favours those individuals who achieve greater personal reproductive success, relative to the other individuals in their population (Darwin 1859; Fisher 1930; Price 1970). All else being equal, a behaviour that improves the reproductive success of another individual will reduce the actor's relative fitness, and hence it will be disfavoured by natural selection. However, all else need not be equal. First, the actor may derive a direct-fitness benefit, owing to mechanisms such as reciprocity (Trivers 1971) or by-product mutualism (West-Eberhard 1975). Such cooperation is termed "mutually-beneficial" (West et al. 2007b). Second, the actor may derive an indirect-fitness benefit, as a consequence of being genetically related to the recipients of the cooperative behaviour (Hamilton 1964). Such cooperation is termed "altruistic" (Hamilton 1964; West et al. 2007b).

One way in which relatedness may arise between social partners is if dispersal rates are low, such that individuals will tend to be genealogically close to their neighbours ('viscous' populations; Hamilton 1964, 1971a). As this mechanism does not require discrimination of one's genetic relatives, it appears to provide a very general solution to the problem of altruistic cooperation. However, a problem with this solution is that limited dispersal may also exacerbate competition among social partners (Hamilton 1971a; Queller 1992; West et al. 2002; Griffin et al. 2004). In the simplest scenario of an inelastic (i.e. saturated), viscous population, the relatedness and competition effects of limited dispersal exactly cancel, so that there is no net impact of the rate of dispersal upon the evolution of indiscriminate helping (Taylor 1992a, 1992b; Kümmerli et al. 2009).

This has stimulated the development of a large body of theoretical – and, increasingly, empirical – research, examining factors that may decouple the relatedness and competition effects of dispersal. For example: population elasticity (Taylor 1992b; Alizon & Taylor 2008), overlapping generations (Taylor & Irwin 2000; Irwin & Taylor 2001), budding dispersal (Gardner & West 2006; Lehmann et al. 2006b; Kümmerli et al. 2009), life-cycle and timing of the social behaviour (Taylor 1992a; Lehmann and Rousset 2010), behaviours mediating patch extinction probabilities (Lehmann et al 2006b), variable group size (Grafen 2007), trans-generational altruism (Lehmann 2007, 2010), dispersal-dependent social behaviour (El Mouden and Gardner 2008), and sex-biased dispersal (Johnstone and Cant 2008; Gardner 2010).

All this work has concerned populations in which resources are evenly distributed among patches – that is, patches do not vary in their intrinsic quality. However, all natural populations are characterised by some degree of resource heterogeneity, and the impact of this variation has been of great interest to ecologists and evolutionary biologists (Levins 1968; Begon et al 2006). The potential for resource heterogeneity to mediate the evolution of helping behaviours has been considered by Frank (1996, 2003, 2010), suggesting that individuals with greater resources will be favoured to make greater investments into helping. However, Frank's models do not place the evolution of social behaviour within an explicit viscous population setting, and so cannot address the issue of whether population viscosity drives the evolution of social behaviour.

Here, we investigate the impact of heterogeneity in resource availability on the evolution of indiscriminate helping in viscous populations. We consider scenarios in which different patches have different resource availability at any given time and / or the same patch has different resource availabilities at different times, (i.e. spatial and / or temporal heterogeneity; cf Cohen and Levin 1991), and we describe the genetic structuring of the population that results from this variation, and its impact upon local competition for resources. In particular, we examine how relatedness and resource competition arise as a consequence of these demographic factors, how they become intertwined or decoupled within patches and across whole populations, and how they interact in mediating the evolution of helping and harming behaviours that are either obligate or facultatively-adjusted to the availability of resources.

3.2. Model and Analysis

3.2.1. Model

We assume an infinite island model (Wright 1931), with social interactions among patchmates (Taylor 1992a). Each patch contains n haploid, asexual individuals. We consider two types of patches: high-quality patches that have high resource availability, and low-quality patches that have low resource availability. Individuals in high-quality patches each produce a large number $F_{\mathrm{H}} = f(x,y)$ of offspring, and individuals in low-quality patches each produce a large number $F_{\mathrm{L}} = s\, f(x,y)$ of offspring, where x is the individual's investment in the social behaviour, y is the average investment among that individual's social partners, and $0 \leq s \leq 1$. Following reproduction, all adults die, and each surviving offspring disperses with probability m to a random patch in the population, or else stays in the native patch with probability $1-m$. Subsequent to dispersal, n offspring are chosen at random to become reproductively mature in each patch, with the remainder dying. Finally, patches may undergo changes in their resource availability: high-quality patches remain high-quality with probability α or else become low-quality with probability $1-\alpha$; low-quality patches remain low-quality with probability β, or else become high-quality with probability $1-\beta$. Model notation is summarized in Table 1.

Symbol	Meaning
H	High-quality patch
L	Low-quality patch
O	Obligate social behaviour
T	Corresponding to Taylor's (1992) analysis
P	Primary recipient
S	Secondary recipient
A_{X}	Potential for helping in condition X
α	Probability that a high-quality patch remains a high-quality patch
β	Probability that a low-quality patch remains a low-quality patch
c_{X}	Class reproductive value in condition X
F_{X}	Fecundity of a breeding female in condition X
$\tilde{h}$	Probability that an individual chosen at random is native to the patch
h_{X}	Probability of philopatry in condition X

m	Migration rate
n	Patch size
π_X	History of resource quality in condition X
p	Frequency of high-quality patches in the population
r_X	'Others-only' relatedness of an individual in condition X
R_X	'Whole-group' relatedness of an individual in condition X
s	Quality ratio
τ	Temporal coefficient of correlation
v_X	Individual reproductive value of an individual in condition X
x	Level of helping of a focal actor
y	Average level of the neighbour's social behaviour
z^*	Candidate evolutionary stable strategy for the social behaviour

Table 1. A summary of model notation.

3.2.2. Helping and harming

We classify social behaviours according to their impact upon fecundity. Specifically, the fecundity cost of the behaviour is given by $-C \equiv \partial f/\partial x$ and the fecundity benefit of the behaviour is given by $B \equiv \partial f/\partial y$, and we define helping behaviours as those involving $B > 0$ and harming behaviours as those involving $B < 0$. We employ the Taylor-Frank neighbour-modulated fitness approach to kin selection analysis (Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al 2007) to determine the direction of selection acting upon the social trait, assuming that the population is at ecological equilibrium (see Appendices A-E for details). The condition for the evolution of obligate helping and harming is given by

$$c_{\mathrm{H}}\left(-C+r_{\mathrm{P|H}}B-\left(B-C\right)r_{\mathrm{S|H}}v_{\mathrm{S|H}}\right)+c_{\mathrm{L}}\left(-C+r_{\mathrm{P|L}}B-\left(B-C\right)r_{\mathrm{S|L}}v_{\mathrm{S|L}}\right)>0, \tag{1}$$

where: c_{H} and c_{L} denote the class reproductive values for individuals in high-quality and low-quality patches, respectively (Fisher 1930; Taylor 1990, 1996; Grafen 2006); $r_{\mathrm{P|H}}$ and $r_{\mathrm{P|L}}$ denote the relatedness of the actor to her patchmates, i.e. the ‘primary’ recipients (cf. West and Gardner 2010), for actors in high-quality and low-quality patches, respectively; $r_{\mathrm{S|H}}$ and $r_{\mathrm{S|L}}$ denote the relatedness of the actor to those individuals who will compete for resources with the extra offspring produced in the actor’s patch, i.e. the ‘secondary’ recipients (cf. West and Gardner 2010), for actors in high-quality and low-quality patches, respectively; and $v_{\mathrm{S|H}}$ and $v_{\mathrm{S|L}}$ denote the individual reproductive values of the secondary recipients, expressed relative to that of the actor and her patchmates, for actors in high-quality and low-quality patches, respectively (see Appendices C and E for details).

The conditions for the evolution of facultative helping and harming are given by

$$-C+r_{\mathrm{P|H}}B-\left(B-C\right)r_{\mathrm{S|H}}v_{\mathrm{S|H}}>0, \text{ and} \tag{2}$$

$$-C+r_{\mathrm{P|L}}B-\left(B-C\right)r_{\mathrm{S|L}}v_{\mathrm{S|L}}>0, \tag{3}$$

for individuals in high-quality patches and individuals in low-quality patches, respectively (see Appendix D for details).

Thus, an increase in investment into the social behaviour impacts upon the actor's inclusive fitness in three ways. First, she suffers a personal cost C. Second, her primary recipients receive a benefit B, and this is weighted by the genetic relatedness r_P of the actor to these individuals. Third, the net increase B-C in fecundity leads to a decrease of B-C in the survival of the secondary recipients, and this is weighted by the genetic relatedness r_S of the actor to these individuals, and their relative reproductive value v_S. Note that this third term in inequality (2) and (3) can be rearranged in the form $\tilde{\mathrm{h}}(B\text{-}C)$ $\tilde{\mathrm{h}}Rv_S$, where $\tilde{\mathrm{h}}$ is the probability that an offspring chosen at random after dispersal is native to the patch, this can be interpreted as follows: of the (B-C) additional offspring created due to the social behaviour, a fraction $\tilde{\mathrm{h}}$ stays in the local patch and wins a breeding site, this displaces other native offspring with probability $\tilde{\mathrm{h}}$, which have on average a relatedness R and an expected relative reproductive value v_S to the actor. The product of relatedness and relative reproductive value is sometimes termed "life-for-life" relatedness (Hamilton 1972), and this describes how well the recipient transmits copies of the actor's genes to future generations, relative to the actor's ability to do this herself (Williams 1996, p181).

Setting the LHS of each of these inequalities (1)-(3) equal to zero, we obtain the conditions for the actor to 'break even', neither increasing nor decreasing her inclusive fitness. Rearranging these conditions into the form $C/B < A$, the quantity A represents the potential for helping (Gardner 2010) and its additive inverse -A represents the potential for harming. If $A > 0$ there is potential for helping to be favoured, and if $A < 0$ there is potential for harming to be favoured, provided the cost is sufficiently small. From inequality (1), the potential for obligate helping is

$$A_{\mathrm{O}} = \frac{c_{\mathrm{H}}(r_{\mathrm{P|H}} - r_{\mathrm{S|H}}v_{\mathrm{S|H}}) + c_{\mathrm{L}}(r_{\mathrm{P|L}} - r_{\mathrm{S|L}}v_{\mathrm{S|L}})}{c_{\mathrm{H}}(1 - r_{\mathrm{S|H}}v_{\mathrm{S|H}}) + c_{\mathrm{L}}(1 - r_{\mathrm{S|L}}v_{\mathrm{S|L}})}\ . \tag{4}$$

From inequalities (2) and (3), the potentials for facultative helping are given by

$$A_{\mathrm{H}} = \frac{r_{\mathrm{P|H}} - r_{\mathrm{S|H}}v_{\mathrm{S|H}}}{1 - r_{\mathrm{S|H}}v_{\mathrm{S|H}}}\text{, and} \tag{5}$$

$$A_{\mathrm{L}} = \frac{r_{\mathrm{P|L}} - r_{\mathrm{S|L}}v_{\mathrm{S|L}}}{1 - r_{\mathrm{S|L}}v_{\mathrm{S|L}}}\ , \tag{6}$$

for actors in high-quality and low-quality patches, respectively.

3.3. Results and Discussion

3.3.1. Spatial heterogeneity

We first consider populations in which resource availability varies across patches within generations but not across generations within patches (i.e., spatial heterogeneity only). We derive the following results:

Result 1 | Spatial heterogeneity has no impact on the potential for obligate helping and harming, which is zero (A_{O}=0) over the whole range of parameter values (see Appendix E for details). This extends Taylor's (1992a) cancellation result for homogeneous populations to obligate social behaviours in spatially heterogeneous populations.

Result 2 | Spatial heterogeneity has no impact on the potential for facultative helping and harming, which is zero ($A_H = A_L = 0$) over the whole range of parameter values (see Appendix E for details). This extends Result 1 to facultative social behaviours.

Why does Taylor's (1992a) result for homogenous populations and obligate helping and harming extend to spatially heterogeneous populations with potentially facultative helping and harming? To understand this, we first consider the potential for facultative helping in high-quality patches, which is given by equation (5), i.e. $A_H = (r_{P|H}-r_{S|H}v_{S|H})/(1-r_{S|H}v_{S|H})$. Note that the relatedness of the actor to her primary recipients $r_{P|H}$ is equal to the product of philopatry $h_H = (1-m)^2/(1-m+m(p+(1-p)s))^2$ for a high-quality patch – where p is the frequency of high-quality patches at equilibrium (see Appendix E for details) – and whole-group relatedness R_H for a high-quality patch, because in the absence of temporal heterogeneity the actor's patch was of high-quality in the previous generation. Also, the relatedness of the actor to her secondary recipients $r_{S|H}$ is equal to the product of philopatry h_H for a high-quality patch and whole-group relatedness R_H for a high-quality patch, because in the absence of temporal heterogeneity the actor's patch remains high-quality in the next generation. Moreover, the reproductive value of a secondary recipient – i.e. an adult in the actor's patch in the next generation – is equal to the reproductive value of the actor, i.e. $v_{S|H} = 1$. This is because both individuals are inhabitants of high-quality patches.

Hence, as in Taylor (1992a), the actor places equal value upon her primary and secondary recipients: $r_{P|H} = r_{S|H}v_{S|H}$ (Figure 1), and consequently the potential for facultative helping is zero, $A_H = 0$ (see Appendix E for details). The same is true for facultative helping in low-quality patches: $h_L = ((1-m)s)^2/((1-m)s+m(p+(1-p)s))^2$ and $v_{S|L} = 1$, hence $r_{P|L} = r_{S|L}v_{S|L}$, and $A_L = 0$ (Figure 1; see Appendix E for details). As a consequence of this cancellation effect in both high-quality and low-quality patches, the potential for obligate helping is also zero, $A_O = 0$ (see Appendix for details). Since the potential for harming is given by the additive inverse of the potential for helping – i.e., -A – the above cancellation also applies to the evolution of obligate and facultative harming behaviours.

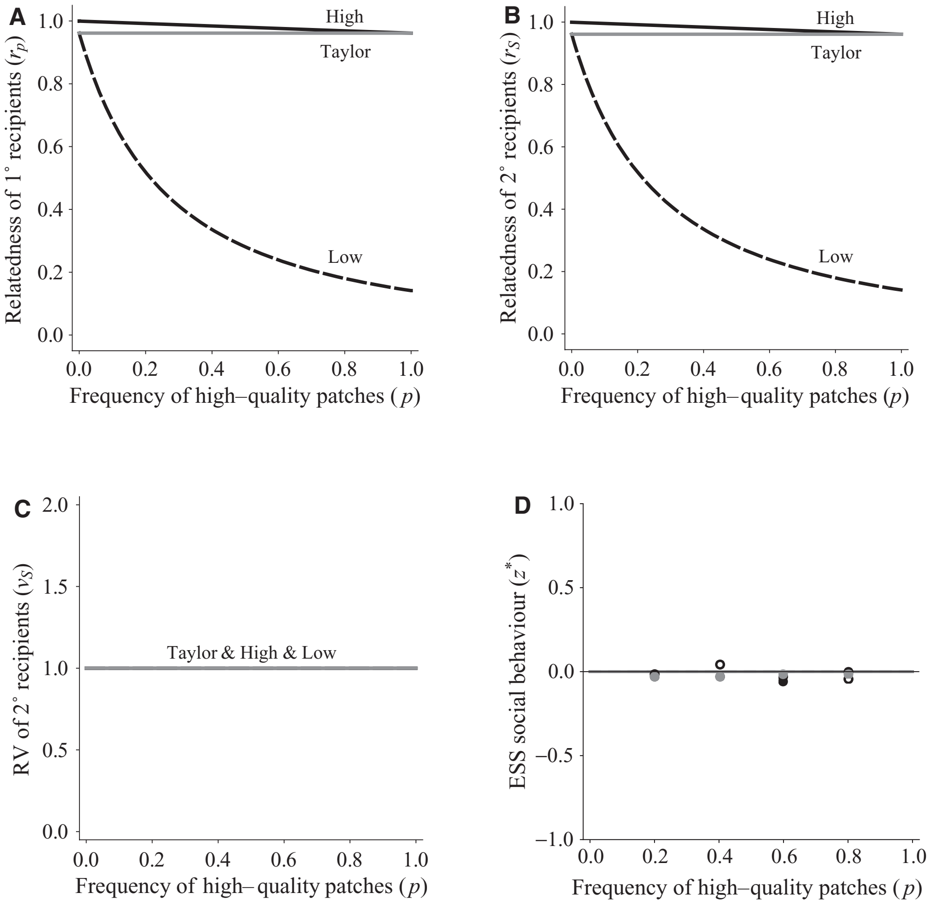

Figure 1 | Impact of spatial heterogeneity upon relatedness, reproductive value, and the evolution of helping and harming. (a) The relatedness of primary recipients (r_P) as a function of the frequency of high-quality patches (p) is shown for high-quality patches ($r_{P|H}$; solid line), low-quality patches ($r_{P|L}$; dashed line) and Taylor's (1992) reference model ($r_{P|T}$; grey line). Relatedness of primary recipients is higher in high-quality patches owing to higher philopatry, and lower in low-quality patches owing to lower philopatry, relative to the reference model ($r_{\mathrm{P|L}} < r_{\mathrm{P|T}} < r_{\mathrm{P|H}}$). (b) The relatedness of secondary recipients (r_{S}) as a function of the frequency of high-quality patches (p). Relatedness of secondary recipients is higher in high-quality patches owing to higher philopatry, and lower in low-quality patches owing to lower philopatry, relative to the reference model ($r_{\mathrm{S|L}} < r_{\mathrm{S|T}} < r_{\mathrm{S|H}}$). Importantly, primary and secondary recipients are equally related ($r_{\mathrm{P|H}} = r_{\mathrm{S|H}}$, $r_{\mathrm{P|L}} = r_{\mathrm{S|L}}$). (c) The relative reproductive value of secondary recipients (v_{S}) is constant with respect to the frequency of high-quality patches (p), and is equal to that of the reference model ($v_{\mathrm{S|H}} = v_{\mathrm{S|L}} = v_{\mathrm{S|T}} = 1$). (d) An illustration of how the equilibrium level of helping or harming depends upon the frequency of high-quality patches (p; see Appendix F for details). Analytical results (lines) and illustrative simulation data (circles) are shown for obligate behaviour (grey line, solid grey circles), facultative behaviour in low-quality patches (dashed line, open circles) and facultative behaviour in high-quality patches (black line, solid black circles). In all panels, we assume $n = 2$, $s = 0.01$, $m = 0.01$, and $\tau = 1.00$.

3.3.2. Temporal heterogeneity

We next consider populations in which resource availability varies across generations within patches but not across patches within generations (i.e., temporal heterogeneity only). We assume that all patches become high-quality with probability p, or become low-quality with probability 1-p. We derive the following results:

Result 3 | Temporal heterogeneity has no impact on the potential for obligate helping and harming, which is zero ($A_{\mathrm{O}} = 0$) over the whole range of parameter values (see Appendix H for details). This extends Taylor's (1992a) cancellation result for homogeneous populations to obligate social behaviours in temporally heterogeneous populations.

Result 4 | Temporal heterogeneity has no impact on the potential for facultative helping and harming, which is zero ($A_H = A_L = 0$) over the whole range of parameter values (see Appendix H for details). This extends Result 3 to facultative social behaviours.

Owing to the absence of spatial heterogeneity, the probability of philopatry is $h_H = h_L = h = (1\text{-}m)^2$ for both high-quality and low-quality patches, whole-group relatedness is $R_H = R_L = R = 1/(n\text{-}(n\text{-}1)(1\text{-}m)^2)$ for both high-quality and low-quality patches, and the relative reproductive value of secondary recipients is $v_{S|H} = v_{S|L} = v_S = 1$ for actors in both high-quality and low-quality patches i.e. the same as in Taylor's (1992) model. Hence, the actor places equal value upon her primary and secondary recipients: $r_{P|H} = r_{S|H}v_{S|H} = r_{P|L} = r_{S|L}v_{S|L}$ (Figure 2), and consequently the potential for helping is zero, $A_H = A_L = A_O = 0$ (see Appendix H for details). Again, a parallel argument can be made for the potential for harming, $-A$.

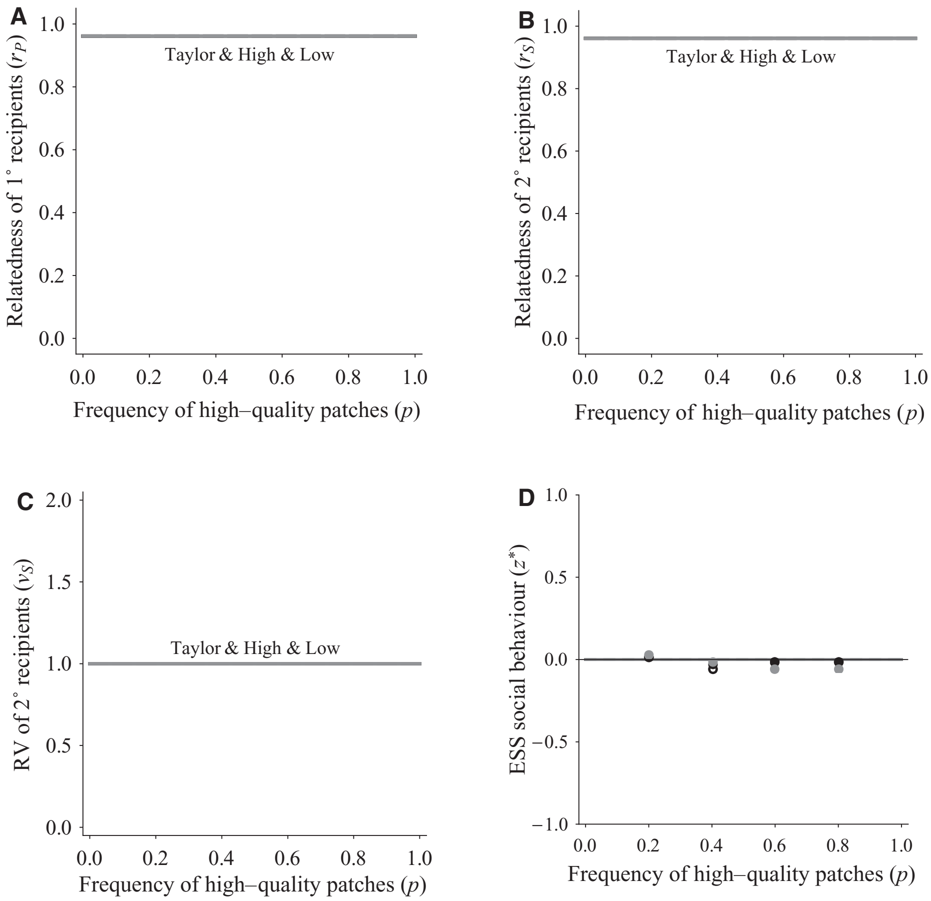

Figure 2 | Impact of temporal heterogeneity upon relatedness, reproductive value and the evolution of helping and harming. (a) The relatedness of primary recipients (r_P) as a function of the frequency of high-quality patches (p) is shown for high-quality patches ($r_{P|H}$; solid line), low-quality patches ($r_{P|L}$; dashed line) and Taylor's (1992) reference model ($r_{P|T}$; grey line). Relatedness of primary recipients in high-quality and low-quality patches is equal to that for the reference model ($r_{P|H} = r_{P|L} = r_{P|T}$). (b) The relatedness of secondary recipients (r_S) as a function of the frequency of high-quality patches (p). Relatedness of secondary recipients in high-quality and low-quality patches is equal to that for the reference model ($r_{S|H} = r_{S|L} = r_{S|T}$). Importantly, primary and secondary recipients are equally related ($r_{P|H} = r_{S|H} = r_{P|L} = r_{S|L}$). (c) The relative reproductive value of secondary recipients (v_S) is constant with respect to the frequency of high-quality patches (p), and is equal to that of the reference model ($v_{\mathrm{S|H}} = v_{\mathrm{S|L}} = v_{\mathrm{S|T}} = 1$). (d) An illustration of how the equilibrium level of helping or harming depends upon the frequency of high-quality patches (p; see Appendix F for details). Analytical results (lines) and illustrative simulation data (circles) are shown for obligate behaviour (grey line, solid grey circles), facultative behaviour in low-quality patches (dashed line, open circles) and facultative behaviour in high-quality patches (black line, solid black circles). In all panels, we assume $n = 2$, $s = 0.01$, $m = 0.01$.

3.3.3. Spatial and temporal heterogeneity

We next consider populations in which resource availability varies both across patches within generations and across generations within patches (i.e., both spatial and temporal heterogeneity). We derive the following results:

Result 5 | Spatial and temporal heterogeneity has no impact on the potential for obligate helping and harming, which is zero ($A_{\mathrm{O}} = 0$) over the whole range of parameter values (see Appendix G for details). This extends Taylor's (1992a) cancellation result to obligate social behaviours in spatially and temporally heterogeneous populations.

Result 6 | Spatial and temporal heterogeneity has an impact on the potential for facultative helping and harming, which may be nonzero ($A_{\mathrm{H}} \neq 0$ and $A_{\mathrm{L}} \neq 0$) depending upon parameter values (see Appendices C and E for details). Thus, selection may favour helping and harming that is facultatively adjusted according to the quality of the actor's patch, in spatially and temporally heterogeneous populations.

Why does Taylor's (1992a) result for homogenous populations and obligate helping and harming collapse when we consider populations that are both spatially and temporally heterogeneous with facultative helping and harming? Focusing our attention upon facultative helping in high-quality patches, we find that the potential for this behaviour to be favoured by natural selection is given by equation (5), i.e. $A_{\mathrm{H}} = (r_{\mathrm{P|H}}-r_{\mathrm{S|H}}v_{\mathrm{S|H}})/(1-r_{\mathrm{S|H}}v_{\mathrm{S|H}})$. If resource availability is heterogeneous through space and time, then the actor's high-quality patch may have been either high or low-quality in the previous generation, and so her relatedness to the primary recipients $r_{\mathrm{P|H}}$ is a weighted average of the product of philopatry $h_{\mathrm{H}}=(1-m)^2/(1-m+m(p+(1-p)s))^2$ and whole-group relatedness R_{H} for high-quality patches and the product of philopatry $h_{\mathrm{L}}=((1-m)s)^2/((1-m)s+m(p+(1-p)s))^2$ and whole-group relatedness R_{L} for low-quality patches (see Appendix E for details). In contrast, her relatedness $r_{\mathrm{S|H}}$ to the secondary recipients is the product of the probability of philopatry h_{H} and whole-group relatedness R_{H} for her high-quality patch. Because we have $h_{\mathrm{H}} \geq h_{\mathrm{L}}$ we have that $\underline{r}_{\mathrm{P|H}} \leq r_{\mathrm{S|H}}$ (see Appendix E for details). Moreover, the reproductive value of her secondary recipients – i.e. adults in her patch in the next generation – is not equal to her own reproductive value. Whilst she has the reproductive value of an individual in a high-quality patch, theirs is a weighted average of the reproductive value of an individual in a high-quality patch and that of an individual in a low-quality patch. Hence, $v_{\mathrm{S|H}} \leq 1$ (see Appendix C for details).

Consequently, the actor may place different values upon her primary and secondary recipients: $r_{\mathrm{P|H}} \neq r_{\mathrm{S|H}}v_{\mathrm{S|H}}$ (Figure 3; see Appendix for details). Hence, the potential for facultative helping in high-quality patches may be nonzero, $A_{\mathrm{H}} \neq 0$. The same is true for facultative helping in low-quality patches: $r_{\mathrm{P|L}} \geq r_{\mathrm{S|L}}$ and $v_{\mathrm{S|L}} \geq 1$, so $r_{\mathrm{P|L}} \neq r_{\mathrm{S|L}}v_{\mathrm{S|L}}$ and $A_{\mathrm{L}} \neq 0$ (Figure 3; see Appendices C and E for details). We find that there is potential for helping in high-quality patches and potential for harming in low-quality patches ($A_{\mathrm{H}} > 0, A_{\mathrm{L}} < 0$) when high-quality patches are relatively rare (low p), and potential for harming in high-quality patches and potential for helping in low-quality patches ($A_{\mathrm{H}} < 0, A_{\mathrm{L}} > 0$) when high-quality patches are relatively common (high p; Figure 4). For example, in high-quality patches the asymmetry in relatedness ($\underline{r}_{\mathrm{P|H}} < r_{\mathrm{S|H}}$) favours harming, as the actor values secondary recipients more than primary recipients. By contrast, the asymmetry in reproductive value ($v_{\mathrm{S|H}} < 1$) favours helping, as the actor values primary recipients more than secondary recipients. The asymmetry in relatedness is largest when the frequency of high-quality patches (p) is intermediate, whilst the asymmetry in reproductive value is largest when the frequency of high-quality patches (p) is low. Thus, helping is more likely to evolve when high-quality patches are relatively rare (Figure 4).

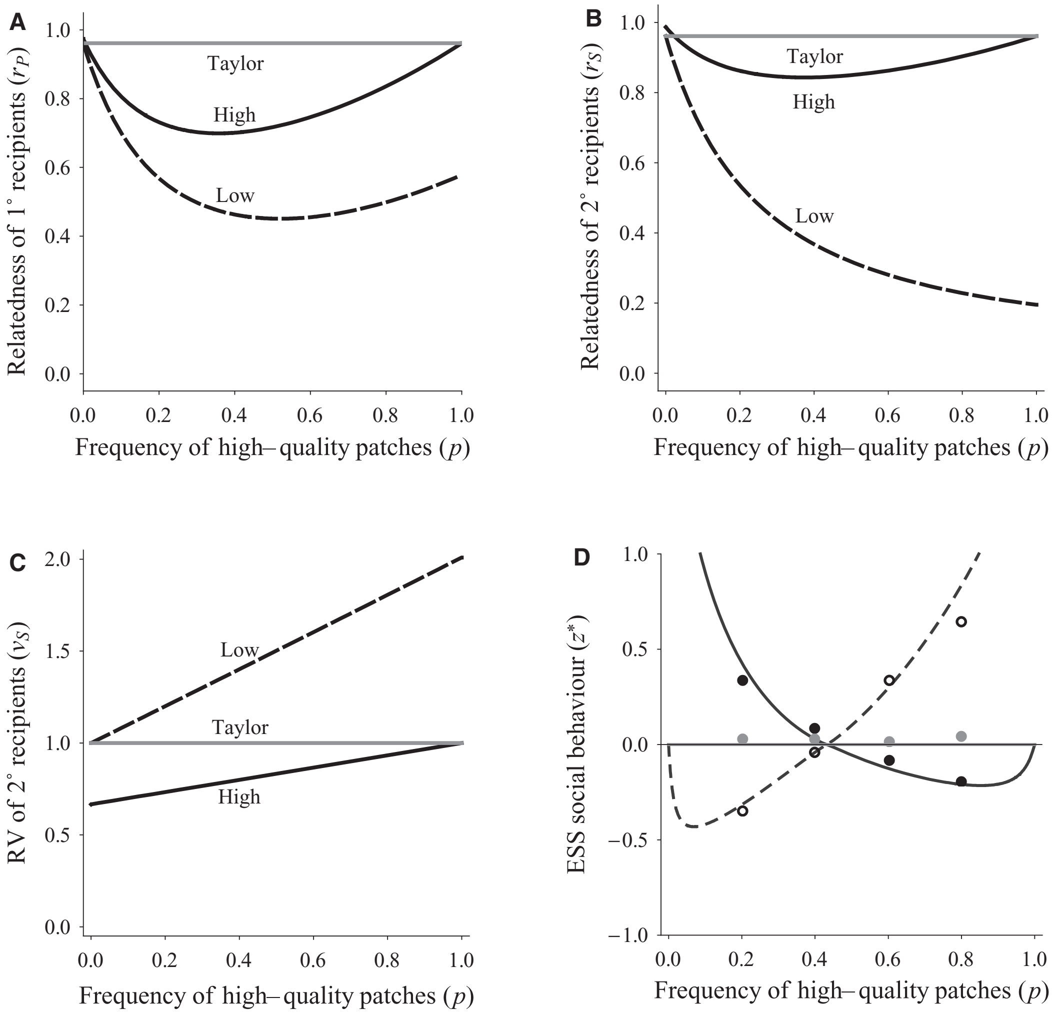

Figure 3 | Impact of spatial and temporal heterogeneity upon relatedness ,and reproductive value and the evolution of helping and harming. (a) The relatedness of primary recipients (r_{P}) as a function of the frequency of high-quality patches (p) is shown for high-quality patches ($r_{\mathrm{P|H}}$; solid line), low-quality patches ($r_{\mathrm{P|L}}$; dashed line) and Taylor's (1992) reference model ($r_{\mathrm{P|T}}$; grey line). Relatedness of primary recipients may be higher or lower in high-quality patches, relative to the reference model, and is lower in low-quality patches, relative to the reference model ($r_{\mathrm{P|L}} < r_{\mathrm{P|H}}, r_{\mathrm{P|T}}$). (b) The relatedness of secondary recipients (r_{S}) as a function of the frequency of high-quality patches (p). Relatedness of secondary recipients may be higher or lower in high-quality patches, relative to the reference model, and is lower in low-quality patches, relative to the reference model ($r_{\mathrm{S|L}} < r_{\mathrm{S|H}}, r_{\mathrm{S|T}}$). Importantly, primary and secondary recipients may be unequally related ($r_{\mathrm{P|H}} \neq r_{\mathrm{S|H}}, r_{\mathrm{P|L}} \neq r_{\mathrm{S|L}}$). (c) The relative reproductive value of secondary recipients (v_{S}) as a function of patch frequency (p). In high-quality patches the relative reproductive value of secondary recipients is less than or equal to that of the reference model, and in low-quality patches the relative reproductive value is greater than or equal to that of the reference model ($v_{\mathrm{S|H}} \leq v_{\mathrm{S|T}} \leq v_{\mathrm{S|L}}$). (d) An illustration of how the equilibrium level of helping or harming depends upon the frequency of high-quality patches (p; see Appendix F for details). Analytical results (lines) and illustrative simulation data (circles) are shown for obligate behaviour (grey line, solid grey circles), facultative behaviour in low-quality patches (dashed line, open circles) and facultative behaviour in high-quality patches (black line, solid black circles). In all panels, numerical examples are given for $n = 2$, $s = 0.01$, $m = 0.01$, and $\tau = 0.10$.

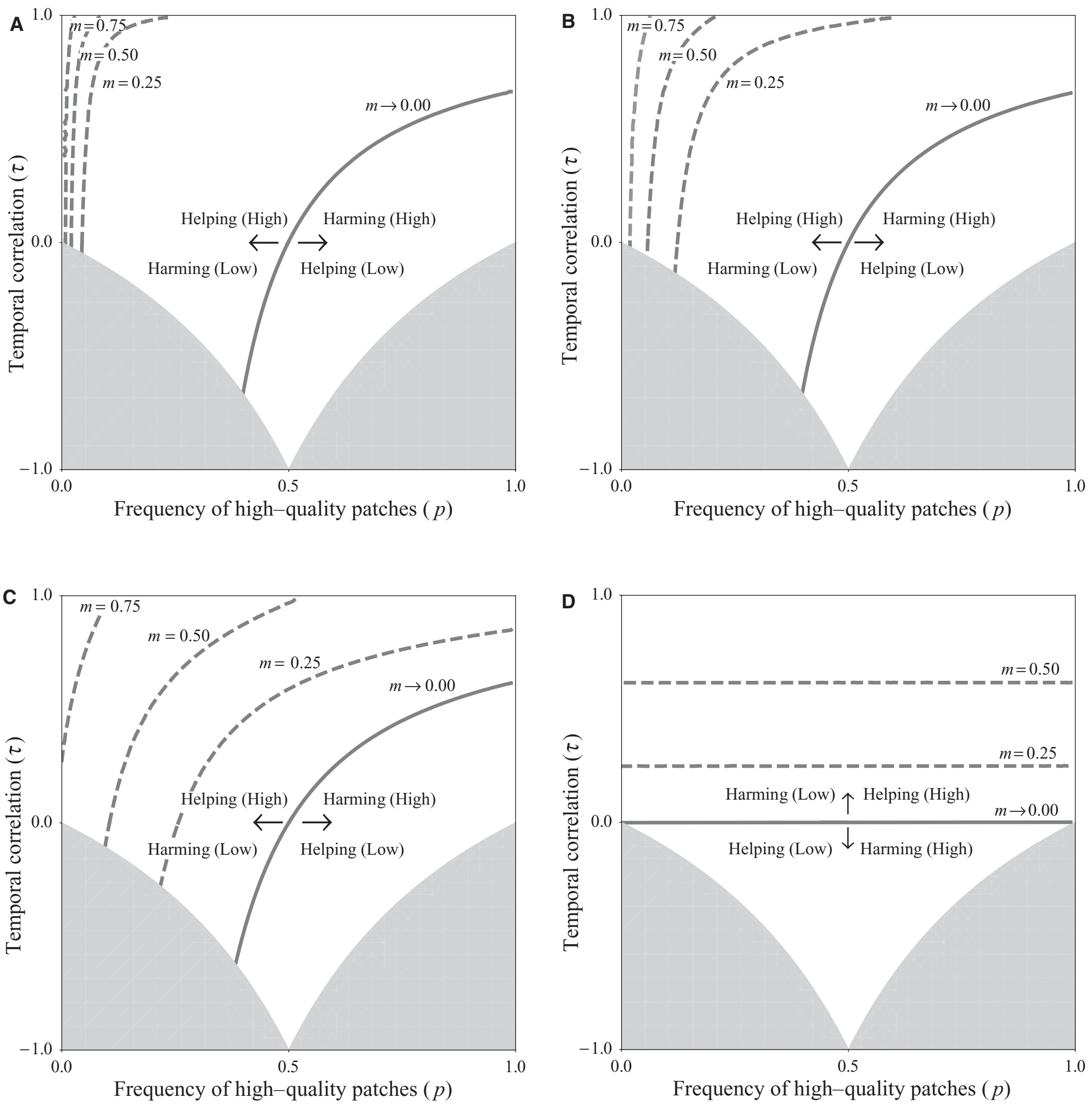

Figure 4 | Impact of spatial and temporal heterogeneity upon the potential for facultative helping and harming. The continuous line represents a null potential for helping and harming ($A_H = A_L = 0$) for varying migration rates ($m \rightarrow 0.00, m = 0.25, 0.50$ & 0.75). (a,b,c) When high-quality patches are relatively rare (low p), there is potential for helping in high-quality patches ($A_H > 0$) and potential for harming in low-quality patches ($A_L < 0$). When high-quality patches are relatively common (high p), there is potential for harming in high-quality patches ($A_H < 0$) and potential for helping in low-quality patches ($A_L > 0$). (d) As the asymmetry in fecundity becomes negligible ($s \rightarrow 1$) the regions where helping or harming are favoured become invariant in relation to the frequency of high-quality patches (p). Note that, in this case, selection is vanishing weak, and when $s = 1$ it vanishes altogether, recovering Taylor's (1992) result. The shaded region represents impossible parameter combinations (see Appendix A for details). Numerical examples are given for $n = 2$, (a) $s = 0.001$, (b) $s = 0.01$, (c) $s = 0.1$, and (d) $s \rightarrow 1$.

In addition, as the environment becomes more staid (i.e. temporal correlation increases) the asymmetry in reproductive value becomes more important than the asymmetry in relatedness. As a result, staid environments are favourable to the evolution of facultative helping in high-quality patches and to the evolution of facultative harming in low-quality patches. By contrast, as the population becomes more mixed (i.e. migration rate increases) the asymmetry in relatedness becomes more important than the asymmetry in reproductive value. As a result, mixed populations are favourable to the evolution of facultative harming in high-quality patches and to the evolution of facultative helping in low-quality patches (Figure 4).

However, whilst actors in high-quality patches place unequal value upon their primary and secondary recipients, and actors in low-quality patches place unequal value upon their primary and secondary recipients, we find that actors place equal value upon their primary and secondary recipients when we take an average over actors in high-quality and low-quality patches. As the ecological dynamics is determined by a time-homogeneous Markov chain, the transition probabilities that mediate the value of primary recipients, are identical to the transition probabilities that mediate the value of secondary recipients (see Appendix G for details). Hence, whilst there is nonzero potential for facultative helping and harming in high-quality and low-quality patches ($A_\mathrm{H} \neq 0, A_\mathrm{L} \neq 0$), there is zero potential for obligate helping and harming ($A_\mathrm{O} = 0$) in populations characterized by both spatial and temporal heterogeneity.

3.4. Conclusions

Population viscosity has been suggested to represent a very general mechanism by which kin selection can drive the evolution of cooperation at all levels of biological organization. However, the seminal result of Taylor (1992a) reveals that, in the simplest scenario of a homogeneous, inelastic island model of population structure, the cooperation-promoting effects of increased genetic relatedness exactly cancel with the cooperation-inhibiting effects of increased kin competition, so that population viscosity has no net effect on the evolution of cooperation. Here we have shown that this result extends to heterogeneous populations, where certain patches have high resource availability and their residents enjoy high fecundity, and other patches have low resource availability and their residents suffer low fecundity. The potential for obligate helping – and harming – is zero irrespective of the degree of population viscosity, in populations characterized by spatial and/or temporal heterogeneity in resource availability. Only when social behaviour is facultatively adjusted according to the actor's patch type and there is both spatial and temporal heterogeneity in resource availability does a nonzero potential for helping or harming arise.

Previous work on social evolution in unsaturated populations (e.g. Alizon and Taylor 2008; Wild et al. 2009; Lion and Gandon 2010) is superficially similar to the present saturated-population analysis, because empty breeding sites can be thought of as a type of resource that varies in its availability between patches. However, empty sites promote helping by easing competition for resources, owing to a feedback from the social behaviour to the number of breeders per patch, and need not impact at all on the genetic relatedness of social partners (Wild et al. 2009; but see Alizon and Taylor 2008 and Lion and Gandon 2010). In contrast, the present model requires that each breeding spot is won at the expense of some other individual's chance of reproductive success, and helping behaviours may instead evolve owing to the way in which resource heterogeneity impacts upon the genetic structure of populations. Moreover, whilst the availability of empty sites depends upon the social behaviour exhibited by a patch's residents, the present model has assumed that resource availability is determined independently of the genetic strategies of the residents. The possibility of incorporating feedback, such that the probability of a patch transitioning from low to high-quality depends upon the social behaviour of its residents, represents an interesting avenue for future exploration.

Empirical data on the relationship between spatiotemporal heterogeneity in resource availability and social behaviour are limited. Recent comparative studies have considered the relationship between temporal heterogeneity and cooperative breeding in birds, finding that greater seasonality is associated with a greater degree of sociality (Rubenstein and Lovette 2007; Cockburn and Russell 2011; Jetz and Rubenstein 2011). We suggest that future comparative studies should look for an interaction between spatial and temporal heterogeneity in resource availability. Moreover, there is ample scope for experimentally testing the predictions of our model using experimental evolution methods. The island model of population structure is very amenable to implementation in laboratory studies of microbial social behaviour (Griffin et al. 2004; Kümmerli et al. 2009), and this experimental methodology could readily be extended to incorporate patch heterogeneity in space and time. More generally, we emphasize the potential for kin selection theory to explain variation within as well as between populations, and we suggest that future empirical studies consider the relationship between local environments and local sociality in addition to the relationship between global environments and whole-population measures of sociality.

Finally, we note some implications for the response of organisms' social behaviours to climate change and other relatively fast, anthropogenic impacts upon the natural environment. The main results of our analysis have concerned populations that are at ecological equilibrium, and adaptive evolutionary change may be relatively slow to track ecological upheavals. Hence, we predict that rapid degradation of the environment may be accompanied by maladaptive facultative changes in organisms' social behaviours. For example, over much of the range of parameter values, our model predicts that facultative harming behaviour is favoured in low-quality patches. Consequently, habitat deterioration might lead to an increase in mutually costly harming behaviours within ecologically threatened species, further contributing to their extinction risk. Such indirect consequences of habitat deterioration may pose an extra obstacle for conservation efforts.

3.5. Appendix

3.5.1. Appendix A – Patch dynamics

Given the assumptions described in the main text, transitions in patch quality can be represented as a matrix as follows

$$\mathbf{P} = \begin{pmatrix} \alpha & 1-\beta \\ 1-\alpha & \beta \end{pmatrix}. \tag{A1}$$

At equilibrium, the fraction of patches that are in the high-quality state is

$$p = \frac{1-\beta}{2-(\alpha+\beta)}, \tag{A2}$$

assuming $\alpha, \beta < 1$. Denoting patch quality at time t by T_t (equal to 1 for H and equal to 0 for L), the correlation coefficient describing the statistical association between a patch's quality at times t and t+1 is $\tau = \mathrm{cov}(T_t, T_{t+1})/\sqrt{(\mathrm{var}(T_t)\mathrm{var}(T_{t+1}))}$, or

$$\tau = \alpha - (1-\beta), \tag{A3}$$

at ecological equilibrium. Note that when temporal correlation is negative ($\tau < 0$), it is no longer independent of the frequency of high-quality patches (p). For example, when $\tau = -1$, the frequency of high-quality patches must be equal to ½. Otherwise we would not have a stable distribution of high-quality patches over generations. Note also that this environmental change may be regarded as analogous to an extra phase of dispersal (cf Débarre & Gandon 2011), albeit a form of 'budding' dispersal (Gardner & West 2006) where all patchmates 'travel' together to the new environment.

3.5.2. Appendix B – Reproductive success

In a high-quality patch, an offspring of a focal breeding female stays in the native patch with probability 1-*m*. The offspring then competes with other offspring and wins a breeding site with probability

$$E^{\bullet}_{\mathrm{H}\to\mathrm{H}} = \frac{f(x_{\mathrm{H}}, y_{\mathrm{H}})}{(1-m)f(Y_H, Y_H) + m(pf(z_{\mathrm{H}}, z_{\mathrm{H}}) + (1-p)f(z_{\mathrm{L}}, z_{\mathrm{L}})s)}, \tag{A4}$$

where Y_{H} is the average trait value in the focal patch, and z_{H} and z_{L} are the population average trait values in high and low-quality patches, respectively. An offspring of the same focal mother migrates to a high-quality patch with probability *mp*. The offspring then competes with other offspring and wins a breeding site with probability

$$E^{\circ}_{\mathrm{H}\to\mathrm{H}} = \frac{f(x_{\mathrm{H}}, y_{\mathrm{H}})}{(1-m)f(z_{\mathrm{H}}, z_{\mathrm{H}}) + m(pf(z_{\mathrm{H}}, z_{\mathrm{H}}) + (1-p)f(z_{\mathrm{L}}, z_{\mathrm{L}})s)}. \tag{A5}$$

Finally an offspring of the same focal mother migrates to a low-quality patch with probability $m(1\text{-}p)$. The offspring then competes with other offspring and wins a breeding site with probability

$$E^{\circ}_{\mathrm{H}\to\mathrm{L}} = \frac{f(x_{\mathrm{H}}, y_{\mathrm{H}})}{(1-m)f(z_{\mathrm{L}}, z_{\mathrm{L}})s + m(pf(z_{\mathrm{H}}, z_{\mathrm{H}}) + (1-p)f(z_{\mathrm{L}}, z_{\mathrm{L}})s)}. \tag{A6}$$

We also derive corresponding expressions for a focal mother in a low-quality patch. These expressions define a matrix **E** which is given by

$$\mathbf{E} = \begin{pmatrix} E_{\mathrm{H}\to\mathrm{H}} & E_{\mathrm{L}\to\mathrm{H}} \\ E_{\mathrm{H}\to\mathrm{L}} & E_{\mathrm{L}\to\mathrm{L}} \end{pmatrix} = \begin{pmatrix} (1-m)E^{\bullet}_{\mathrm{H}\to\mathrm{H}} + mpE^{\circ}_{\mathrm{H}\to\mathrm{H}} & mpE^{\circ}_{\mathrm{L}\to\mathrm{H}} \\ m(1-p)E^{\circ}_{\mathrm{H}\to\mathrm{L}} & (1-m)E^{\bullet}_{\mathrm{L}\to\mathrm{L}} + m(1-p)E^{\circ}_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}. \tag{A7}$$

Matrix **P** and **E** combine to define the reproductive success matrix, which is given by

$$\mathbf{w} = \begin{pmatrix} w_{\mathrm{H}\to\mathrm{H}} & w_{\mathrm{L}\to\mathrm{H}} \\ w_{\mathrm{H}\to\mathrm{L}} & w_{\mathrm{L}\to\mathrm{L}} \end{pmatrix} = \mathbf{PE}. \tag{A8}$$

In the context of class-structured populations, the reproductive success of an adult breeding female must take into account the relative contribution of individuals in different classes for gene frequency change (Fisher 1930; Price 1970; Taylor 1990; Grafen 2006). Thus the reproductive success of a focal individual in a high-quality patch is given by

$$W_\mathrm{H} = w_{\mathrm{H}\to\mathrm{H}} + \frac{v_\mathrm{L}}{v_\mathrm{H}} w_{\mathrm{H}\to\mathrm{L}}, \tag{A9}$$

where v_H and v_L are the reproductive value of individuals in high-quality and low-quality patches, respectively. Likewise, the reproductive success of a focal adult breeding female in a low-quality patch is

$$W_\mathrm{L} = w_{\mathrm{L}\to\mathrm{L}} + \frac{v_\mathrm{H}}{v_\mathrm{L}} w_{\mathrm{L}\to\mathrm{H}}. \tag{A10}$$

The expected reproductive success of an individual is given by the class specific reproductive success (W_H and W_L), weighted by the respective class reproductive values (c_H and c_L), which is given by

$$W = c_\mathrm{H} W_\mathrm{H} + c_\mathrm{L} W_\mathrm{L}. \tag{A11}$$

Class reproductive value are defined as the product of the frequency of individuals in a given patch quality-type and those individuals' reproductive value ($c_H = pv_H$; $c_L = (1-p)v_L$).

3.5.3. Appendix C – Reproductive value

Individuals reproductive values are given by the left-eigenvector of matrix **w** (defined in equation A8) assuming a neutral population (Taylor 1990; Grafen 2006), and are normalized such that $c_H + c_L = 1$ (Taylor 1990; Taylor et al 2007). Hence, a gene drawn at random from the population has an expected reproductive success W_H with probability c_H, and an expected reproductive success W_L with probability c_L. If there is no heterogeneity in the population (i.e. $s = 1$) then there are no differences in the reproductive values ($v_H = v_L$). However, as heterogeneity emerges in the population (i.e. $0 < s < 1$), it is easy to show that $v_H > v_L$, because individuals in high-quality patches have higher fecundity than individuals in low-quality patches. Reproductive values of secondary recipients are given by: $v_{S|H} = (\alpha v_H+(1-\alpha)v_L)/v_H$ and $v_{S|L} = (\beta v_L+(1-\beta)v_H)/v_L$. Note that, because $v_H \geq v_L$, we have $v_{S|H} \leq 1$ and $v_{S|L} \geq 1$.

3.5.4. Appendix D – Hamilton's rule and the potential for helping and harming

To determine how selection acts on the trait of interest we use the Taylor and Frank approach (Taylor and Frank 1996; Frank 1998; Taylor et al 2007). We assume that the genetic variation is segregating at a locus responsible for the social behaviour. We denote the genic value for a focal gene drawn from this locus by g (Price 1970; Falconer 1981).

Assuming a vanishing small genetic variation, the direction of natural selection acting on the social trait is given by

$$\frac{\mathrm{d}W}{\mathrm{d}g} = c_{\mathrm{H}}\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} + c_{\mathrm{L}}\frac{\mathrm{d}W_{\mathrm{L}}}{\mathrm{d}g}. \tag{A12}$$

The derivative in the first term of the RHS of this equation can be expanded as follows:

$$\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} = \frac{\partial W_{\mathrm{H}}}{\partial x_{\mathrm{H}}}\frac{\mathrm{d}x_{\mathrm{H}}}{\mathrm{d}g_{\mathrm{H}}}\frac{\mathrm{d}g_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial y_{\mathrm{H}}}\frac{\mathrm{d}y_{\mathrm{H}}}{\mathrm{d}g'_{\mathrm{H}}}\frac{\mathrm{d}g'_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial Y_{\mathrm{H}}}\frac{\mathrm{d}Y_{\mathrm{H}}}{\mathrm{d}G_{\mathrm{H}}}\frac{\mathrm{d}G_{\mathrm{H}}}{\mathrm{d}g}, \tag{A13}$$

where g'_H denotes social partners average genetic value excluding self, and G_{H} denotes social partners average genetic value including self, genetic values being the average of an individual's genic values. All the derivatives and partial derivatives are evaluated at $x = y = Y = z$ (Taylor and Frank 1996; Frank 1997, 1998; Taylor et al 2007). The partial derivatives give the marginal effects of the mutant phenotypes. The correlations between the phenotype and the genetic breeding values represent the genotype-to-phenotype mapping defined as $\mathrm{d}x_{\mathrm{H}}/\mathrm{d}g_{\mathrm{H}} = \mathrm{d}y_{\mathrm{H}}/\mathrm{d}g'_H = \mathrm{d}Y_{\mathrm{H}}/\mathrm{d}G_{\mathrm{H}} = \gamma_{\mathrm{H}}$, and this is arbitrarily set to unity ($\gamma_{\mathrm{H}} = 1$). The correlations between breeding values are coefficients of consanguinity. These correlations when normalized to the actor's coefficient of consanguinity give the kin selection coefficient of relatedness. The same expansion can be made for the direction of selection acting on low-quality patches ($\mathrm{d}W_{\mathrm{L}}/\mathrm{d}g$). Note that selection acting on the social trait not only depends upon the direction of selection ($\mathrm{d}W_{\mathrm{X}}/\mathrm{d}g$), but also upon the class-reproductive values (c_{X}). As a consequence, we may have $\mathrm{d}W_{\mathrm{X}}/\mathrm{d}g \neq 0$, but $c_{\mathrm{X}} = 0$, in which case selection acting on the trait in the context X is null.

For obligate social behaviours the condition for the evolution of higher breeding values is given by dW/d$g > 0$. For facultative social behaviour the condition for the evolution of higher breeding values in high-quality patches and low-quality patches is given by dW_{H}/d$g > 0$ and dW_{L}/d$g > 0$, respectively. This gives the inequalities (1)-(3) in the main text.

3.5.5. Appendix E – Relatedness

Here we derive the kin selection coefficients of relatedness for haploid populations. We assume vanishingly small genetic variation in the population. To determine the average relatedness structure in the population we define a recursion equation (Wright 1969; Taylor 1992a; Rousset 2004). In a neutral and equilibrium population, the relatedness of a focal offspring to her sisters is 1 with probability $1/n$. The relatedness to her native patchmates is the relatedness among their mothers, which is given by r. These are native to the focal patch with probability h, which is the probability that we choose at random two offspring that have not dispersed. Therefore among offspring, relatedness is given by

$$Q = h\left(\frac{1}{n} + \left(1 - \frac{1}{n}\right)r\right). \tag{A14}$$

A patch in a high-quality state, was a high-quality patch in the previous generation with probability π_{H}, and was a low-quality patch with probability 1-π_{H}. A low-quality patch was a low-quality patch in the previous generation with probability π_{L}, and a high-quality patch with probability 1-π_{L}. The recursion equations are given by

$$\begin{cases} r'_{\mathrm{H}} = \pi_{\mathrm{H}} Q_{\mathrm{H}} + (1-\pi_{\mathrm{H}}) Q_{\mathrm{L}} \\ r'_{\mathrm{L}} = \left(1-\pi_{\mathrm{L}}\right) Q_{\mathrm{H}} + \pi_{\mathrm{L}} Q_{\mathrm{L}} \end{cases}, \tag{A15}$$

Solving the system of equations (A15) for equilibrium, $r'_{\mathrm{H}} = r_{\mathrm{H}}$ and $r'_{\mathrm{L}} = r_{\mathrm{L}}$, we get the kin selection coefficients of relatedness, which are given by

$$r_{\mathrm{H}} = \frac{\pi_{\mathrm{H}} h_{\mathrm{H}} \left(n - h_{\mathrm{L}}(n-1)\right) + (1-\pi_{\mathrm{H}}) h_{\mathrm{L}} n + \left(1-\pi_{\mathrm{L}}\right) h_{\mathrm{H}} h_{\mathrm{L}} (n-1)}{\left(n - \pi_{\mathrm{H}} h_{\mathrm{H}} (n-1)\right)\left(n - h_{\mathrm{L}}(n-1)\right) + \left(1-\pi_{\mathrm{L}}\right) h_{\mathrm{L}} (n-1)\left(n - h_{\mathrm{H}}(n-1)\right)}, \text{ and} \tag{A16}$$

$$r_{\mathrm{L}} = \frac{h_{\mathrm{L}} \left(n - \pi_{\mathrm{H}} h_{\mathrm{H}} (n-1)\right) - (1-\pi_{\mathrm{L}})\left(\left(h_{\mathrm{H}} + h_{\mathrm{L}}\right) n - h_{\mathrm{H}} h_{\mathrm{L}} (n-1)\right)}{\left(n - \pi_{\mathrm{H}} h_{\mathrm{H}} (n-1)\right)\left(n - h_{\mathrm{L}}(n-1)\right) + (1-\pi_{\mathrm{L}}) h_{\mathrm{L}} (n-1)\left(n - h_{\mathrm{H}}(n-1)\right)}. \tag{A17}$$

for high-quality patches and low-quality patches, respectively. ‘Whole-patch’ relatedness coefficients are determined by substituting these values into the expression $R=1/n+(1-1/n)r$, for the corresponding patch quality types. Relatedness of primary recipients is given by $r_{\mathrm{P|H}} = r_{\mathrm{H}}$ and $r_{\mathrm{P|L}} = r_{\mathrm{L}}$ for high-quality patches and low-quality patches, respectively. Relatedness of secondary recipients is given by $r_{\mathrm{S|H}} = h_{\mathrm{H}} R_{\mathrm{H}}$ and $r_{\mathrm{S|L}} = h_{\mathrm{L}} R_{\mathrm{L}}$ for high-quality patches and low-quality patches, respectively. Let us denote the denominator of (A16) and (A17) as D. After some algebra it is easy to show that $r_{\mathrm{P|H}}-r_{\mathrm{S|H}} = -(h_{\mathrm{H}}-h_{\mathrm{L}})n(1-\alpha)/D$ and that $r_{\mathrm{P|L}}-r_{\mathrm{S||L}} = (h_{\mathrm{H}}-h_{\mathrm{L}})n(1-\alpha)/D$. Thus, because $h_{\mathrm{H}} \geq h_{\mathrm{L}}$, we have $r_{\mathrm{P|H}} \leq r_{\mathrm{S|H}}$ and $r_{\mathrm{P|L}} \geq r_{\mathrm{S|L}}$.

In spatially heterogeneous populations ($\tau = 1$), a high-quality patch was always a high-quality patch in the previous generation ($\pi_{\mathrm{H}} = 1$), and a low-quality patch was always a low-quality patch in the previous generation ($\pi_{\mathrm{L}} = 1$). Hence, the coefficients of relatedness of equations (A16) and (A17) reduce to $r_H = h_H/(n-(n-1)h_H)$ and $r_L = h_L/(n-(n-1)h_L)$. This gives the following identity between others-only and whole-group relatedness: $r_H = h_H R_H$ and $r_L = h_H R_L$. As a consequence in spatial heterogeneous populations the relatedness of primary recipients is equal to the relatedness of secondary recipients (see main text). Note also that $r_H-r_L = (h_H-h_L)n/((n-(n-1)h_H)(n-(n-1)h_L))$. Since $h_H \geq h_L$, relatedness in high-quality patches is always larger or equal to relatedness in low-quality patches ($r_H \geq r_L$).

3.5.6. Appendix F – Evolutionarily stable strategy and simulations

We wrote the code for the simulations in the language C++ (see Supporting Information). The protocol of the simulations closely follows the specifications of the model assumptions. However, instead of an infinite number of patches we now a finite number of patches that we set to 5000. Each patch accommodates n=2 breeding individuals. Thus, the total population size is 1000. Each individual's genotype is represented as a real number on the interval [-1,1], where positive values indicate level of investment into helping and negative values indicate level of investment into harming. At initialization, all genotypes are set to 0. In every subsequent generation we choose 10 individuals at random for mutation: we add to their genotypic value a random Gaussian-distributed quantity with mean 0 and variance 0.015. To make quantitative predictions about the candidate evolutionarily stable strategy (ESS; Maynard Smith and Price 1973) for the social behaviour (z^*) one needs to specify the marginal fecundity cost and the marginal fecundity benefits. The marginal fecundity cost is assumed to decrease monotonically with the investment in the social behaviour, such that $-C = \partial f(x,y)/\partial x = -k$, where k is a scaling factor. The marginal fecundity benefits are assumed to have diminishing returns with the investment in the social behaviour such that $B = \partial f(x,y)/\partial y = \mathrm{sign}(y)k\eta\mu|y|^{(\mu-1)}$, where η is a scaling factor, and μ controls the rate at which the benefits saturate with the investment in the social behaviour. In the analytical model, a candidate ESS is found when the ratio between the marginal fecundity cost and the marginal fecundity benefits exactly matches the potential for helping ($C/B = A$). The candidate ESS is then given by $z^* = \mathrm{sign}(A)(\eta\mu|A|)^{1/(1-\mu)}$. In the simulation model, we let the population evolve up to 500,000 generations, and we compute the average genotypic value across all individuals in the final generation. In Figures 1-3 we provide illustrative numerical solutions for z^*, along with illustrative simulation data. Here, we assumed $\eta = 20$, $\mu = 0.1$ and $k = 0.015$.

3.5.7. Appendix G – Cancellation of obligate social behaviour

The life-for-life relatedness of primary recipients is $\varrho_{\mathrm{P|H}} = r_{\mathrm{P|H}}$ and $\varrho_{\mathrm{P|L}} = r_{\mathrm{P|L}}$, for high-quality and low-quality patches, respectively. The life-for-life relatedness of secondary recipients is $\varrho_{\mathrm{S|H}} = r_{\mathrm{S|H}}v_{\mathrm{S|H}}$ and $\varrho_{\mathrm{S|L}} = r_{\mathrm{S|L}}v_{\mathrm{S|L}}$. For obligate behaviours, the life-for-life coefficients of primary recipients is given by

$$\rho_{\mathrm{P}} = c_{\mathrm{H}}\rho_{\mathrm{P|H}} + c_{\mathrm{L}}\rho_{\mathrm{P|L}}\ , \tag{A18}$$

and the life-for-life relatedness of a secondary recipient is given by

$$\rho_{\mathrm{S}} = c_{\mathrm{H}}\rho_{\mathrm{S|H}} + c_{\mathrm{L}}\rho_{\mathrm{S|L}}\ . \tag{A19}$$

In Taylor's (1992a) model these are $\varrho_{\mathrm{P}} = r = hR$, and $\varrho_{\mathrm{S}} = aR$, where a is the proportion of competition that occurs locally (scale of competition; Frank 1998; Gardner & West 2006; Van Dyken 2010). As $h = a$, we have $\varrho_{\mathrm{P}} = \varrho_{\mathrm{S}}$, as actors value primary recipients as much as secondary recipients there is no selection for social behaviours. In our model we find that same symmetry: $h_{\mathrm{H}} = a_{\mathrm{H}}$, and $h_{\mathrm{L}} = a_{\mathrm{L}}$. As the ecological dynamics is determined by a time-homogeneous Markov chain, we find additional symmetries. To see how these explain the cancellation result for obligate behaviour let us denote the different probabilities (P) in the life-for-life relatedness as follows: $\mathrm{P}(X_{t-1} = \mathrm{H} \mid X_t = \mathrm{H}) = \pi_{\mathrm{H}}$, $\mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{H}) = 1\text{-}\pi_{\mathrm{H}}$, $\mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{L}) = \pi_{\mathrm{H}}$, $\mathrm{P}(X_{t-1} = \mathrm{H} \mid X_t = \mathrm{L}) = 1\text{-}\pi_{\mathrm{L}}$, $\mathrm{P}(X_{t+1} = \mathrm{H} \mid X_t = \mathrm{H}) = \alpha$, $\mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{H}) = 1\text{-}\alpha$, $\mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{L}) = \beta$, $\mathrm{P}(X_{t+1} = \mathrm{H} \mid X_t = \mathrm{L}) = 1\text{-}\beta$, $\mathrm{P}(X_t = \mathrm{H}) = p$, and $\mathrm{P}(X_t = \mathrm{H}) = 1\text{-}p$. Using this notation, the life-for-life coefficients of relatedness for obligate behaviour can be written as follows:

$$\begin{aligned}\rho_{\mathrm{P}} = \quad & \mathrm{P}(X_t = \mathrm{H})(\mathrm{P}(X_{t-1} = \mathrm{H} \mid X_t = \mathrm{H}) h_{\mathrm{H}} R_{\mathrm{H}} v_{\mathrm{H}} + \mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{H}) h_{\mathrm{L}} R_{\mathrm{L}} v_{\mathrm{H}}) \\ & + \mathrm{P}(X_t = \mathrm{L})(\mathrm{P}(X_{t-1} = \mathrm{H} \mid X_t = \mathrm{L}) h_{\mathrm{H}} R_{\mathrm{H}} v_{\mathrm{L}} + \mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{L}) h_{\mathrm{L}} R_{\mathrm{L}} v_{\mathrm{L}})\end{aligned} \text{, and} \tag{A20}$$

$$\begin{aligned}\rho_{\mathrm{S}} = \quad & \mathrm{P}(X_t = \mathrm{H})(\mathrm{P}(X_{t+1} = \mathrm{H} \mid X_t = \mathrm{H}) h_{\mathrm{H}} R_{\mathrm{H}} v_{\mathrm{H}} + \mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{H}) h_{\mathrm{H}} R_{\mathrm{H}} v_{\mathrm{L}}) \\ & + \mathrm{P}(X_t = \mathrm{L})(\mathrm{P}(X_{t+1} = \mathrm{H} \mid X_t = \mathrm{L}) h_{\mathrm{L}} R_{\mathrm{L}} v_{\mathrm{H}} + \mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{L}) h_{\mathrm{L}} R_{\mathrm{L}} v_{\mathrm{L}})\end{aligned} \; . \tag{A21}$$

We can show that life-for-life relatedness of primary recipients is equal to the life-for-life relatedness of secondary recipients. For example, first note that from Bayes' theorem we have that $\mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{H}) = \mathrm{P}(X_t = \mathrm{H} \mid X_{t-1} = \mathrm{L})\mathrm{P}(X_{t-1} = \mathrm{L})/\mathrm{P}(X_t = \mathrm{H})$; second as the ecological dynamics is described by a time-homogeneous Markov chain we have $\mathrm{P}(X_t = \mathrm{H} \mid X_{t-1} = \mathrm{L}) = \mathrm{P}(X_{t+1} = \mathrm{H} \mid X_t = \mathrm{L})$, as a result we have $\mathrm{P}(X_t = \mathrm{H})\mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{H}) = \mathrm{P}(X_t = \mathrm{L})\mathrm{P}(X_{t+1} = \mathrm{H} \mid X_t = \mathrm{L})$. This can be shown to hold for all other terms in the life-for-life coefficients of relatedness. Thus, as in Taylor's (1992a) model of obligate social behaviour in homogeneous populations, we find that the average life-for-life relatedness of primary recipients is equal to the average life-for-life relatedness of secondary recipients ($\varrho_P = \varrho_S$). This identity prevents the evolution of social behaviours.

3.5.8. Appendix H – Temporal heterogeneity

Here we assume that within a generation, all patches have the same quality-state, i.e. the population is in the high-quality state or the population is in the low-quality state. Independently of the current state, the population becomes high-quality with probability p or becomes low-quality with probability 1-p. The matrix **P** is now defined by setting $\alpha = p$ and $\beta = 1\text{-}p$. As before we solve the matrix for its right-eigenvector, and this gives $u_H = p$ and $u_L = 1\text{-}p$. However the interpretation is now different: u_H and u_L represent the expected fraction of generations that the population, on the long run, spends on the high-quality state and on the low-quality state, respectively. We also need to determine the contribution of high-quality individuals and low-quality individuals to the future genetic composition of the population, i.e. the individual reproductive values. The reproductive value of a high-quality individual is determined as follows: the fecundity of focal high-quality individual is 1; with probability p her offspring reproduce as high-quality individuals, in which case they have reproductive value v_H; with probability 1-p her offspring reproduce as low-quality individual, in which case they have reproductive value v_L; hence, at equilibrium, we get the following identity: $v_H = pv_H+(1\text{-}p)v_L$. Therefore, reproductive values are identical and can be normalized such that $v_H = v_L = 1$. Class reproductive values are given by: $c_H = u_H\ v_H = p$ and $c_L = u_L\ v_L = 1\text{-}p$. Because within each generation there is no heterogeneity, and because reproductive values are identical, the class-specific fitness expressions are identical (W_H and W_L), and are given by

$$W_{\mathrm{X}} = \frac{(1-m)f(x_{\mathrm{X}}, y_{\mathrm{X}})}{(1-m)f(Y_{\mathrm{X}}, Y_{\mathrm{X}}) + mf(z_{\mathrm{X}}, z_{\mathrm{X}})} + \frac{mf(x_{\mathrm{X}}, y_{\mathrm{X}})}{f(z_{\mathrm{X}}, z_{\mathrm{X}})}. \tag{A22}$$

As before we use the Taylor-Frank approach (Taylor and Frank 1996) to determine the direction of natural selection acting on the social traits. Relatedness coefficients are again given by equation (A15) with $\pi_{\mathrm{H}} = 1-\pi_{\mathrm{L}} = p$, $\pi_{\mathrm{L}} = 1-\pi_{\mathrm{H}} = 1-p$, and $h_{\mathrm{H}} = h_{\mathrm{L}} = (1-m)^2$. As a result $r_{\mathrm{H}} = r_{\mathrm{L}} = r_{\mathrm{P|H}} = r_{\mathrm{P|L}} = hR_{\mathrm{H}} = hR_{\mathrm{L}} = hR = h/(n-(n-1)h)$. Relatedness of secondary recipients is given by $r_{\mathrm{S|H}} = r_{\mathrm{S|L}} = hR_{\mathrm{H}} = hR_{\mathrm{L}} = hR$. Hence, $r_{\mathrm{P|H}} = r_{\mathrm{S|H}}$ and $r_{\mathrm{P|L}} = r_{\mathrm{S|L}}$. Specific patch dynamics are given for concreteness and clarity. However, the various measures of value – i.e. relatedness and reproductive value – are independent of the particular dynamics and the correlation between population states over generations.

Chapter 4. Evolution of helping and harming in viscous populations when group size varies.

4.1. Introduction

Explaining cooperative behaviour has been a central challenge to evolutionary biologists over the last few decades (Maynard Smith and Szathmáry 1995; Hamilton 1996; West et al. 2007a; Bourke 2011). Natural selection favours those individuals who achieve higher relative fitness (Darwin 1859; Fisher 1930; Price 1970). But cooperative behaviours that improve the fitness of others will, all else being equal, decrease the relative fitness of the actor. This problem can be solved if the actor receives a direct fitness benefit as a consequence of her cooperative behaviour. For example, the immediate cost of the actor's behaviour may be compensated later in her life by reciprocation from others (Trivers 1971). Such cooperation is termed "mutually-beneficial" (West et al. 2007b). Alternatively, the problem can be solved if the actor receives an indirect fitness benefit, owing to her genetic relatives benefitting from her cooperative behaviour (Hamilton 1963, 1964). Such cooperation is termed "altruistic" (Hamilton 1964; West et al. 2007b).

Altruistic cooperation requires that interacting individuals share genes in common. Hamilton (1964, 1971a) suggested three general mechanisms by which this can occur. First, individuals might be able to identify which of their social partners are genealogically close kin, and preferentially direct their altruism to these individuals.

Second, a “greenbeard” gene may provide its bearer the ability to directly identify which of their social partners also carry the same gene, independently of their genealogical relationship (Hamilton 1964; Dawkins 1976; Gardner & West 2010). Third, if dispersal is limited, then neighbouring individuals are likely to be genealogically close kin, such that even indiscriminate cooperation will tend to occur between individuals who share genes in common. As this third mechanism does not require any discrimination mechanism, it could represent a very general principle that explains the evolution of cooperation at all levels of biological organization (Maynard Smith and Szathmáry 1995; Bourke 2011).

However, limited dispersal can also lead to intensified competition for resources among kin (Hamilton 1964; Queller 1992; West et al. 2002). In the simplest scenario of an infinite island population, the kin-competition effects of limited dispersal exactly cancel its relatedness-enhancing effects, such that population viscosity has no net impact upon the evolution of indiscriminate cooperation (Taylor 1992a). This striking result has stimulated the development of a large body of theoretical – and, to a lesser extent, empirical – research, examining what additional factors may decouple the relatedness and competition effects of limited dispersal, such that indiscriminate helping may be favoured in viscous populations. Some of these factors include: population elasticity (Taylor 1992b), overlapping generations (Taylor & Irwin 2000; Irwin & Taylor 2001), budding dispersal (Gardner & West 2006; Lehmann et al. 2006b; Kümmerli et al. 2009), organism’s life-cycle and timing of the social behaviour (Taylor 1992a; Lehmann and Rousset 2010), behaviours mediating patch extinction probabilities (Lehmann et al 2006), trans-generational altruism (Lehmann 2007, 2010), dispersal-dependent social behaviour (El Mouden and Gardner 2008), empty sites (Alizon and Taylor 2008), sex-biased dispersal (Johnstone and Cant 2008; Gardner 2010), reproductive skew (Johnstone 2008), age structure (Johnstone and Cant 2010), and heterogeneity in resource availability (Rodrigues and Gardner 2012).

All this work has concerned populations in which the size of viable groups is fixed. However, natural populations commonly exhibit some degree of group size heterogeneity, and the impact of this variation has been of great interest to ecologists and evolutionary biologists (Levins 1968; McPeek and Holt 1992; Begon et al. 2006). This is important to the evolution of kin selected traits because patch size is likely to have an impact not only upon the relatedness among social partners but also upon local competition, two important factors that jointly mediate the evolution of social traits in viscous populations.

Here, we investigate the impact of heterogeneity in group size on the evolution of indiscriminate helping and harming in viscous populations. Specifically, we consider scenarios in which different groups have different sizes at any given time (spatial heterogeneity) and / or the same group has different sizes at different times (temporal heterogeneity), where group size is extrinsically controlled and not driven by the group's past or present social behaviour. We first consider cases where fecundity is equal across all patch types, and we then relax this assumption by considering cases where fecundity depends on patch type. Moreover, we consider scenarios where individuals obligately express their social behaviour independently of their patch type and scenarios where individuals may facultatively adjust their social behaviour according to their patch type. We determine the impact of this heterogeneity upon the genetic relatedness and intensity of competition between group mates, and examine how these interact and impact upon the evolution of helping and harming behaviours.

4.2. Model and Analysis

4.2.1. Model

We assume an infinite island population (Wright 1931), with two types of patches: big patches with n_B breeding sites, and little patches with n_L breeding sites (without loss of generality, we assume $n_B \geq n_L$). We consider that individuals are haploid and asexual, and that each individual breeding in a big patch has a very large fecundity $F_B = f(x,y)$, and each individual breeding in a little patch has a very large fecundity $F_L = \sigma f(x,y)$, where: x is the individual's investment into the social behaviour, y is the average level of investment among the individual's group mates; and $0 < \sigma < \infty$ is the "reproductive factor" describing fecundity differences between individuals breeding in little versus big patches. Note that if $\sigma = 1$, individuals breeding in little patches have the same fecundity of those breeding in big patches; if $\sigma < 1$, individuals breeding in little patches have less fecundity than those breeding in big patches; and if $\sigma > 1$, individuals breeding in little patches have greater fecundity than those breeding in big patches. The average investment strategy in the population is represented by z. After reproduction, a fraction 1-m of the offspring stay in their natal patch while the remaining fraction m disperse to random patches in the population. After dispersal, patches may undergo changes in their size according to a Markov process: big patches remain big with probability α and become little with probability 1-α, whereas little patches remain little with probability β and become big with probability 1-β (see Appendix A for details). Subsequently, offspring compete for breeding places, with all non-breeding individuals perishing. Table 1 provides a summary of model notation.

Table 1. A summary of model notation.

Symbol	Meaning
B	Big patch
L	Little patch
O	Obligate social behaviour
T	Reference to quantities in Taylor's (1992) model
P	Primary recipient
S	Secondary recipient
A_{X}	Potential for helping in condition X
α	Probability that a big patch remains big
β	Probability that a little patch remains little
c_{X}	Class reproductive value in condition X
F_{X}	Fecundity of a breeding female in condition X
h_{X}	Probability of co-philopatry in condition X
m	Migration rate
n_{X}	Patch size in condition X
π_{X}	History of density state in condition X
p	Frequency of big patches in the population
r_{X}	'Others-only' relatedness of an individual in condition X
R_{X}	'Whole-group' relatedness of an individual in condition X
σ	Fecundity factor
τ	Temporal coefficient of correlation
v_{X}	Reproductive value of an individual in condition X
υ_{X}	Relative reproductive value of patch in condition X
x	Level of helping of a focal actor

y	Average level of the neighbour's social behaviour

4.2.2. Helping and harming

We classify social behaviours according to their impact upon fecundity. The impact upon the actor's fecundity is $-C \equiv \partial f(x,y)/\partial x$ and the impact upon the patchmates' fecundity is $B \equiv \partial f(x,y)/\partial y$ where, assuming vanishing variation in investment strategies, we evaluate derivatives at $x = y = z$ (Taylor and Frank 1996). Helping behaviours are those that improve the fecundity of patchmates ($B > 0$) and harming behaviours are those that reduce the fecundity of patchmates ($B < 0$; Lehmann et al. 2006a; West and Gardner 2010). We employ the neighbour-modulated approach to kin selection analysis (Hamilton 1964; Taylor 1996; Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al. 2007) to determine the direction of natural selection acting upon the social trait (see Appendices B-E for details). The condition for natural selection to favour the evolution of obligate social behaviour is given by

$$c_{\mathrm{B}}\left(-C + r_{\mathrm{P|B}}B - (B-C)r_{\mathrm{S|B}}\upsilon_{\mathrm{S|B}}\right) + c_{\mathrm{L}}\left(-C + r_{\mathrm{P|L}}B - (B-C)r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}}\right) > 0\,, \tag{1}$$

where: c_{B} and c_{L} are the class reproductive values of individuals breeding within big and little patches, respectively (Fisher 1930; Taylor 1990; Grafen 2006); $r_{\mathrm{P|B}}$ and $r_{\mathrm{P|L}}$ are the relatednesses of the actor to her group mates – "primary recipients" – in big patches and little patches, respectively; $r_{\mathrm{S|B}}$ and $r_{\mathrm{S|L}}$ are the relatednesses of the actor to those offspring competing for breeding sites with the offspring born in her patch – "secondary recipients" – in big patches and little patches, respectively; and, $\upsilon_{\mathrm{S|B}}$ and $\upsilon_{\mathrm{S|L}}$ are the reproductive values of secondary recipients, expressed relative to those of primary recipients, in big patches and little patches, respectively (see Appendices D and E for details). Note that we are defining actor, primary recipients and secondary recipients in terms of the three selection pressures that arise from the actor's social behaviour, namely: the cost to the actor, the benefit to group mates, and the kin competition effect. Also note that a focal individual may experience, either directly or else through her offspring, more than one of these selection pressures during her lifetime.

The conditions for natural selection to favour the evolution of facultative social behaviour are given by

$$-C + r_{\mathrm{P|B}}B - (B-C)r_{\mathrm{S|B}}\upsilon_{\mathrm{S|B}} > 0 \text{, and} \tag{2}$$

$$-C + r_{\mathrm{P|L}}B - (B-C)r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}} > 0 \text{,} \tag{3}$$

for actors in big patches and little patches, respectively.

The left-hand side (LHS) of each of these conditions (1)-(3) can be interpreted as the inclusive fitness effect of the social behaviour (Hamilton 1963, 1964, 1970). First, the behaviour imposes a cost *C* upon the actor's own fecundity. Secondly, it provides a benefit *B* to the fecundity of the actor's group mates, and this benefit is weighted by the relatedness of these primary recipients r_{P}. Finally, the behaviour leads to an increase of *B*-*C* in the number of offspring born in the patch, and hence it excludes an equal number of secondary recipients who compete with these extra offspring for breeding opportunities.

Moreover, this effect is weighted by the relatedness of secondary recipients r_S and also by the relative reproductive value of secondary recipients υ_S.

Transforming the inequalities (1)-(3) into equalities, and rearranging into the form $C/B = A$, we obtain the "potential for helping" A (Rodrigues and Gardner 2012; see also Gardner 2010). This represents the valuation that an actor places upon the fecundity of the primary recipients, relative to her own fecundity. Thus, if $A > 0$ the actor is selectively favoured to help her patchmates, provided the personal cost to herself is sufficiently small, whereas if $A < 0$ the actor is selectively favoured to harm her patchmates, provided the personal cost to herself is sufficiently small. We may define the additive inverse of the potential for helping (i.e. $-A$) as the "potential for harming" (Rodrigues and Gardner 2012).

From inequality (1), the potential for obligate helping is given by

$$A_O = \frac{c_B(r_{P|B} - r_{S|B}\upsilon_{S|B}) + c_L(r_{P|L} - r_{S|L}\upsilon_{S|L})}{c_B(1 - r_{S|B}\upsilon_{S|B}) + c_L(1 - r_{S|L}\upsilon_{S|L})}. \quad (4)$$

From inequalities (2) and (3), the potential for facultative helping is given by

$$A_B = \frac{r_{P|B} - r_{S|B}\upsilon_{S|B}}{1 - r_{S|B}\upsilon_{S|B}}, \text{ and} \quad (5)$$

$$A_L = \frac{r_{P|L} - r_{S|L}\upsilon_{S|L}}{1 - r_{S|L}\upsilon_{S|L}}, \quad (6)$$

for big patches and little patches, respectively.

4.3. Results and Analysis

In the next two sections we derive the main results of our model. We begin by considering that the fecundity of individuals is independent of patch size ($\sigma = 1$). We then relax this assumption to consider cases where the individuals' fecundity depends upon the patch they are in ($0 < \sigma < \infty$; see Table 2 for a summary of the results).

Table 2. A summary of model results

	Group-size heterogeneity and homogeneous fecundity			Group-size heterogeneity and variable fecundity		
	Spatial	Temporal	Spatial and Temporal	Spatial	Temporal	Spatial and Temporal
Obligate	Cancel	Cancel	Cancel	✗	✗	✗
Facultative (Big patches)	Cancel	Helping	Helping	✗	✗	✓
Facultative (Little patches)	Cancel	Harming	Harming	✗	✗	✓

Note – Results for group-size heterogeneity with homogeneous fecundity ($\sigma = 1$) for obligate and facultative behaviour and for different group-size heterogeneity assumptions (spatial only, temporal only, and spatial and temporal). Impact of variable fecundity on the results derived for group-size heterogeneity and homogeneous fecundity (✗: does not impact; ✓: does impact). Obligate trait expression and spatial heterogeneity always leads to the cancellation result. Variable fecundity only plays a role in the evolution of social behaviour when populations are spatially and temporally heterogeneous, in which case it can change the predictions of the homogeneous fecundity model.

4.3.1. Group-size heterogeneity

Spatial heterogeneity -- We first consider a population in which group size varies between patches within generations but not within patches between generations (i.e. spatial heterogeneity only). We derive the following results:

Result 1 | Spatial heterogeneity in group size has no impact upon the evolution of obligate social behaviour ($A_O = 0$), irrespective of parameter values (see Appendix D for details). This extends Taylor's (1992a) result for homogeneous populations to populations with spatially heterogeneity in group size.

Result 2 | Spatial heterogeneity in group size has no impact upon the evolution of facultative social behaviour ($A_B = A_L = 0$), irrespective of parameter values (see Appendix D for details). This extends Result 1 for obligate social behaviour to facultative social behaviour.

In Taylor's (1992a) model, the relatedness of primary recipients is given by $r_{P|T} = h_T R_T$, where: h_T is the probability that two offspring, chosen at random from a patch, are both philopatric to that patch after the dispersal phase; and R_T is the relatedness of these two offspring. The relatedness of secondary recipients is also given by $r_{S|T} = h_T R_T$. As a consequence, the relatedness of primary recipients is equal to that of secondary recipients

$r_{P|T} = r_{S|T}$. Moreover, because Taylor's (1992a) model is of a homogeneous population, all recipients have the same reproductive value in a neutral population, and hence $\upsilon_{S|T} = 1$. Since actors place the same value upon primary and secondary recipients irrespective of population viscosity ($r_{P|T} = r_{S|T}\upsilon_{S|T}$; Figure 1), selection acting upon the social behaviour is null ($A_T = 0$).

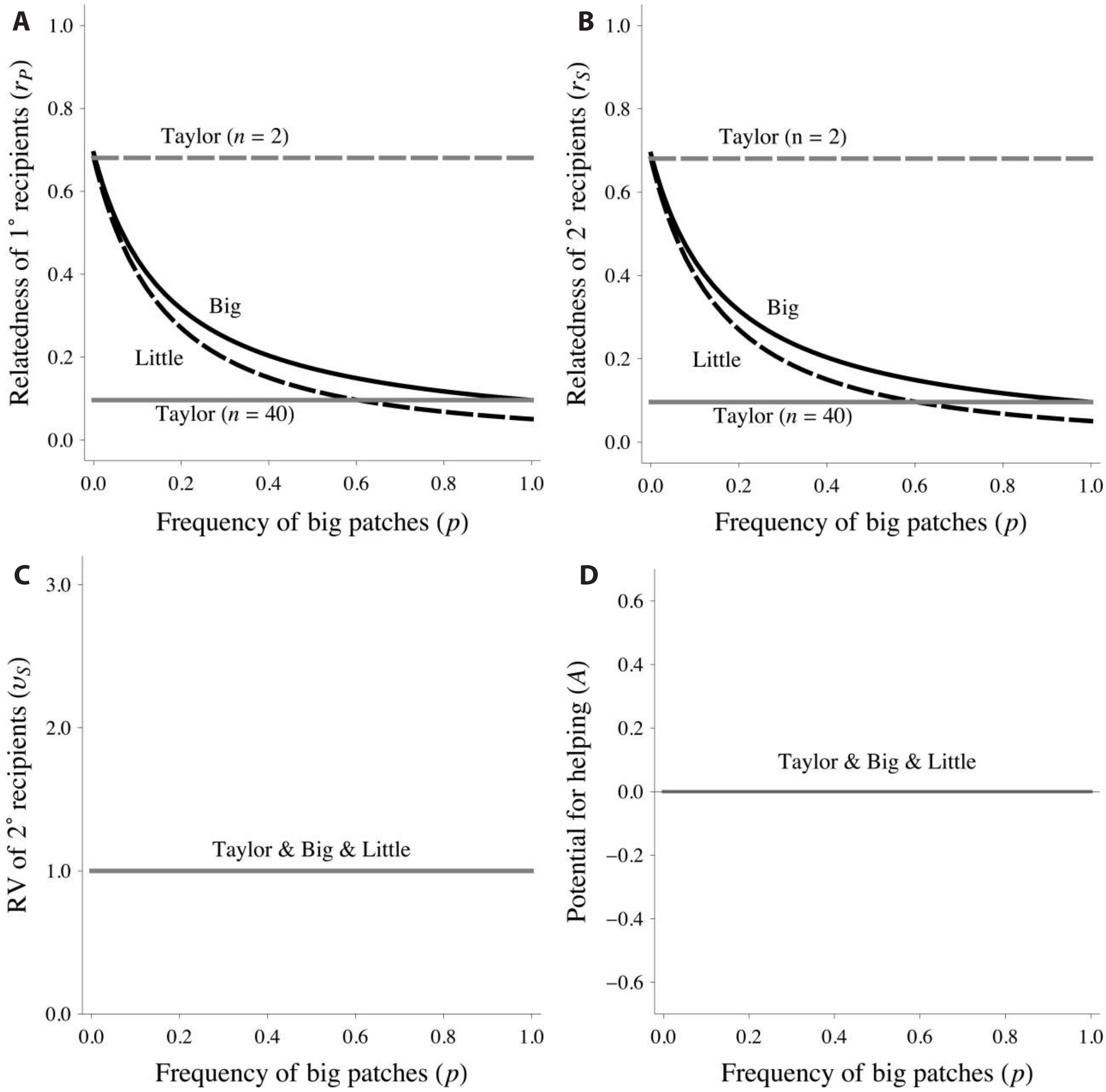

Figure 1 | Impact of spatial heterogeneity in group size upon relatedness, reproductive value and potential for helping. (a) The relatedness of primary recipients (r_P) as a function of the frequency of big patches (p) is shown for big patches ($r_{P|B}$; black solid line), little patches ($r_{\mathrm{P|L}}$; black dashed line) and Taylor's (1992) reference model ($r_{\mathrm{P|T}}$; grey lines). The relatedness of primary recipients is higher in big patches owing to higher co-philopatry, and lower in little patches owing to lower co-philopatry, relative to the reference model with equivalent group sizes ($r_{\mathrm{P|B}} > r_{\mathrm{P|T}}$ and $r_{\mathrm{P|L}} < r_{\mathrm{P|T}}$). (b) The relatedness of secondary recipients (r_{S}) as a function of the frequency of big patches (p). The relatedness of secondary recipients is higher in big patches owing to higher co-philopatry, and lower in little patches owing to lower co-philopatry, relative to the reference model with equivalent group sizes ($r_{\mathrm{S|B}} > r_{\mathrm{S|T}}$ and $r_{\mathrm{S|L}} < r_{\mathrm{S|T}}$). Importantly, primary and secondary recipients are equally related ($r_{\mathrm{P|B}} = r_{\mathrm{S|B}}$, $r_{\mathrm{P|L}} = r_{\mathrm{S|L}}$). (c) The relative reproductive value of secondary recipients (υ_{S}) is constant with respect to the frequency of big patches (p), and is equal to that of the reference model ($\upsilon_{\mathrm{S|B}} = \upsilon_{\mathrm{S|L}} = \upsilon_{\mathrm{S|T}} = 1$). (d) The potential for facultative helping (A_{B} and A_{L}) is constant with respect to the frequency of big patches (p), and is equal to that of the reference model ($A_{\mathrm{B}} = A_{\mathrm{L}} = A_{\mathrm{T}} = 0$). In all panels, we assume $n_{\mathrm{B}} = 40$, $n_{\mathrm{L}} = 2$, $m = 0.10$, and $\tau = 1.00$.

Turning to our model of a spatially heterogeneous population, and focusing upon facultative helping in big patches, we find that the relatedness of primary recipients is given by $r_{\mathrm{P|B}} = h_{\mathrm{B}}R_{\mathrm{B}}$, where: h_{B} is the probability that two offspring, chosen at random from a big patch, are both philopatric to that patch after the dispersal phase; and R_{B} is the relatedness of these two offspring. The relatedness of secondary recipients is also given by $r_{\mathrm{S|B}} = h_{\mathrm{B}}R_{\mathrm{B}}$. As a consequence, the relatedness of primary recipients is equal to that of secondary recipients, $r_{\mathrm{P|B}} = r_{\mathrm{S|B}}$. In addition, the reproductive value of primary recipients is equal to the reproductive value of secondary recipients, $\upsilon_{\mathrm{S|B}} = 1$. Since actors attribute the same value to primary and secondary recipients ($r_{\mathrm{P|B}} = r_{\mathrm{S|B}}\upsilon_{\mathrm{S|B}}$; Figure 1), selection acting upon the social behaviour is null ($A_{\mathrm{B}} = 0$). The same argument can be made in relation to little patches (namely: $r_{\mathrm{P|L}} = h_{\mathrm{L}}R_{\mathrm{L}}$, $r_{\mathrm{S|L}} = h_{\mathrm{L}}R_{\mathrm{L}}$, $r_{\mathrm{P|L}} = r_{\mathrm{S|L}}$, $v_{\mathrm{S|L}} = 1$, $r_{\mathrm{P|L}} = r_{\mathrm{S|L}}v_{\mathrm{S|L}}$ and $A_{\mathrm{L}} = 0$; Figure 1). Finally, selection acting upon obligate social behaviour is determined by selection acting on each size-type patch weighted by the respective class reproductive values (see LHS of inequality (1)). As we have just seen, selection acting on each size-type patch is null ($A_{\mathrm{B}} = 0$ and $A_{\mathrm{L}} = 0$). Therefore selection acting upon obligate social behaviour is also null ($A_{\mathrm{O}} = 0$). Full mathematical details are given in the Appendix F.

Temporal heterogeneity -- We next consider a population in which group size varies within patches between generations but not between patches within generations (i.e. temporal heterogeneity only). In particular, we assume that all patches in the population become big with probability p, or little with probability $1\text{-}p$, in every generation. We derive the following results:

Result 3 | Temporal heterogeneity in group size has no impact upon the evolution of obligate social behaviours ($A_{\mathrm{O}} = 0$), irrespective of parameter values (see Appendix G for details). This extends Taylor's (1992a) cancellation result for homogeneous populations to populations with temporal heterogeneity in group size.

Result 4 | Temporal heterogeneity in group size does have an impact upon the evolution of facultative social behaviours ($A_{\mathrm{B}} \neq 0$ and $A_{\mathrm{L}} \neq 0$). Specifically, selection favours helping in patches with more breeders ($A_{\mathrm{B}} \geq 0$) and harming in patches with fewer breeders ($A_{\mathrm{L}} \leq 0$; see Appendix G for details).

Why does Taylor's (1992a) result no longer hold for facultative social behaviour in temporally heterogeneous populations? Focusing upon helping in big patches only, we find that the relatedness of primary recipients is given by $r_{\mathrm{P|B}} = ph_{\mathrm{B}}R_{\mathrm{B}}+(1\text{-}p)h_{\mathrm{L}}R_{\mathrm{L}}$, where: p

is the probability that the patch was big in the previous generation; h_H is the probability that two offspring, chosen at random from a big patch, are both philopatric to that patch after the dispersal phase; R_B is the relatedness of these two offspring; $1\text{-}p$ is the probability that the patch was little in the previous generation; h_L is the probability that two offspring, chosen at random from a little patch, are both philopatric to that patch after the dispersal phase; and R_L is the relatedness of these two offspring. The relatedness of secondary recipients is $r_{S|B} = h_B R_B$. Note that $h_B = h_L = (1\text{-}m)^2$ and, because there are more breeders in big patches than in little patches, relatedness is higher among the offspring born in the former and lower among the offspring born in the latter patch type, i.e. $R_B < R_L$.

Consequently, the relatedness of primary recipients is larger than the relatedness of secondary recipients, i.e. $r_{P|B} > r_{S|B}$. In addition, because within each generation all individuals are identical, the reproductive value of primary recipients is equal to the reproductive value of secondary recipients, i.e. $v_{P|B} = v_{S|B}$. As actors place more value upon primary recipients than they place upon secondary recipients, selection favours helping behaviours in big patches ($A_B > 0$; Figure 2). A similar argument can be made in relation to little patches. However, as primary recipients are now less valuable for actors than secondary recipients, selection favours harming behaviours in little patches: $r_{P|L} = ph_B R_B + (1\text{-}p)h_L R_L$, $r_{S|L} = h_L R_L$, $h_B = h_L = (1\text{-}m)^2$, $R_B < R_L$, $v_{S|L} = 1$ and $A_L < 0$ (Figure 2; see Appendix G for details). In other words, when patches increase in size (i.e. after a bottleneck), helping is favoured; by contrast, when patches decrease in size, harming is favoured.

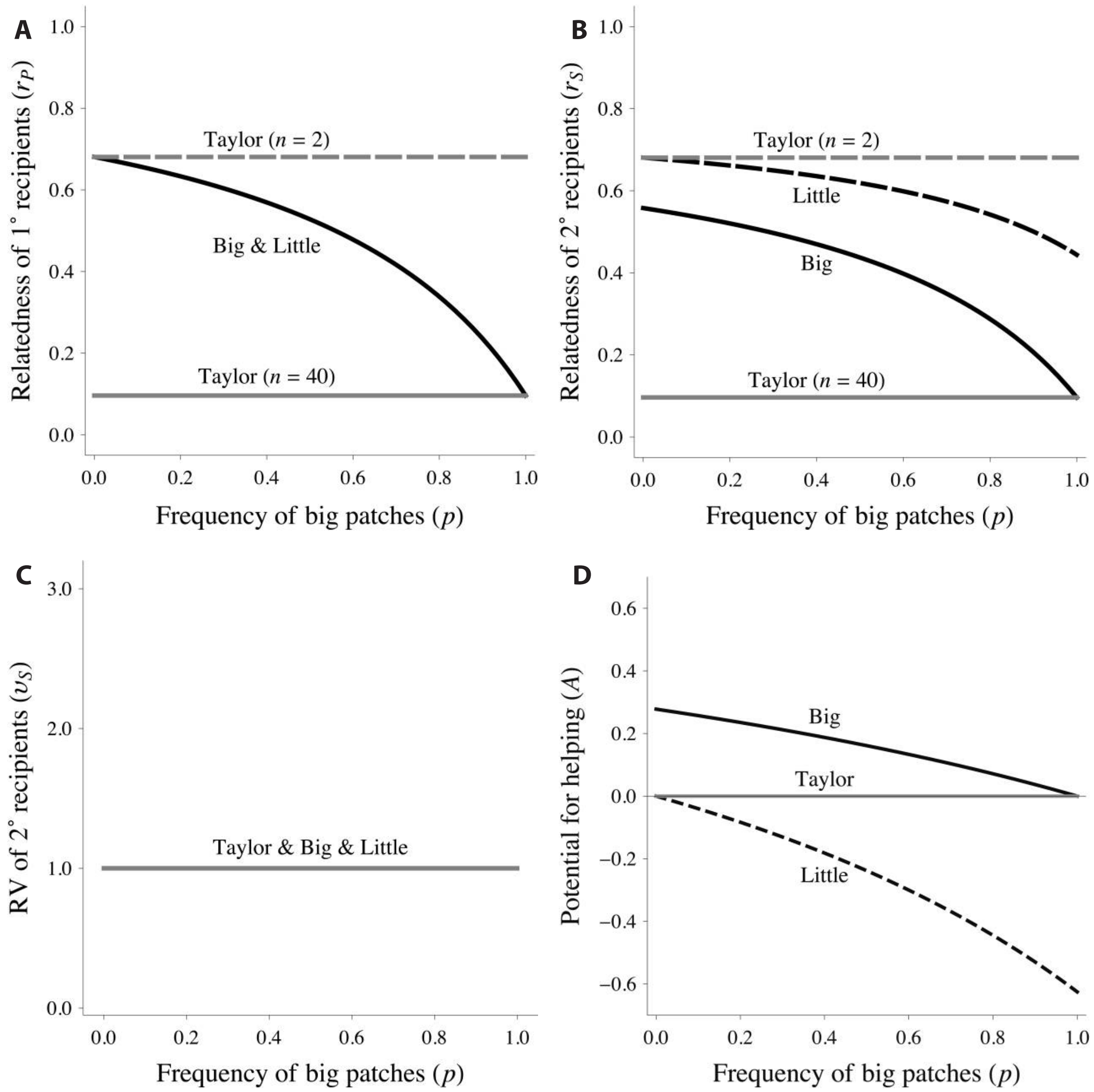

Figure 2 | Impact of temporal heterogeneity in group size upon relatedness, reproductive value, and potential for helping. (a) The relatedness of primary recipients (r_{P}) as a function of the frequency of big patches (p) is shown for big patches ($r_{\mathrm{P|B}}$; black solid line), little patches ($r_{\mathrm{P|L}}$; black dashed line) and Taylor's (1992) reference model ($r_{\mathrm{P|T}}$; grey lines). The relatedness of primary recipients is higher in big patches owing to higher co-philopatry, and lower in little patches owing to lower co-philopatry, relative to the reference model with equivalent group sizes ($r_{\mathrm{P|B}} > r_{\mathrm{P|T}}$ and $r_{\mathrm{P|L}} < r_{\mathrm{P|T}}$). (b) The relatedness of secondary recipients (r_{S}) as a function of the frequency of big patches (p).

The relatedness of secondary recipients is higher in big patches owing to higher co-philopatry, and lower in little patches owing to lower co-philopatry, relative to the reference model with equivalent group sizes ($r_{\mathrm{S|B}} > r_{\mathrm{S|T}}$ and $r_{\mathrm{S|L}} < r_{\mathrm{S|T}}$). Importantly, in big patches primary recipients are more related to the actor than secondary recipients, while in little patches primary recipients are less related to the actor than secondary recipients ($r_{\mathrm{P|B}} > r_{\mathrm{S|B}}$, $r_{\mathrm{P|L}} < r_{\mathrm{S|L}}$). (c) The relative reproductive value of secondary recipients (υ_{S}) is constant with respect to the frequency of big patches (p), and is equal to that of the reference model ($\upsilon_{\mathrm{S|B}} = \upsilon_{\mathrm{S|L}} = \upsilon_{\mathrm{S|T}} = 1$). (d) The potential for facultative helping (A_{B} and A_{L}) as a function of the frequency of big patches (p). In big patches there is potential for helping ($A_{\mathrm{B}} > 0$), while in little patches there is potential for harming ($A_{\mathrm{L}} < 0$). In all panels, we assume $n_{\mathrm{B}} = 40$, $n_{\mathrm{L}} = 2$, $m = 0.10$.

Turning to obligate social behaviour, relatednesses are averaged over patch types, weighting by the correspondent class reproductive values, i.e. $r_{\mathrm{P|O}} = c_{\mathrm{B}}r_{\mathrm{P|B}}+c_{\mathrm{L}}r_{\mathrm{P|L}}$. Because the relatedness of primary recipients in big patches is equal to that of secondary recipients in little patches ($r_{\mathrm{P|B}} = r_{\mathrm{P|L}}$), and the class reproductive values sum to unity ($c_{\mathrm{B}}+c_{\mathrm{L}} = 1$), the relatedness of primary recipients is given by $r_{\mathrm{P|O}} = r_{\mathrm{P|B}} = r_{\mathrm{P|L}} = ph_{\mathrm{B}}R_{\mathrm{B}}+(1\text{-}p)h_{\mathrm{L}}R_{\mathrm{L}}$. The relatedness of secondary recipients is given by $r_{\mathrm{S|O}} = c_{\mathrm{B}}h_{\mathrm{B}}R_{\mathrm{B}}+c_{\mathrm{L}}h_{\mathrm{L}}R_{\mathrm{L}}$. Because class reproductive values are given by $c_{\mathrm{B}} = p$ and $c_{\mathrm{L}} = 1\text{-}p$, we find that the relatedness of primary recipients is equal to that of secondary recipients ($r_{\mathrm{P|O}} = r_{\mathrm{S|O}}$). Consequently, selection acting upon obligate social behaviour is null ($A_{\mathrm{O}} = 0$; see Appendix G for details).

Spatial and temporal heterogeneity -- Finally, we consider a population characterized by heterogeneity in group size both between patches within generations and within patches between generations (i.e. spatial and temporal heterogeneity). We derive the following results:

Result 5 | Spatial and temporal heterogeneity in group size have no impact upon the evolution of obligate social behaviour ($A_O = 0$), irrespective of parameter values (see Appendix F for details). This extends Taylor's (1992a) cancellation result for homogeneous populations to obligate social behaviours in spatially and temporally heterogeneous populations.

Result 6 | Spatial and temporal heterogeneity in group size does influence the evolution of facultative social behaviours ($A_B \neq 0$ and $A_L \neq 0$). Specifically, selection favours helping in patches with more breeders ($A_B \geq 0$) and harming in patches with fewer breeders ($A_L \leq 0$; see Appendix F for details).

Why does Taylor's (1992a) result no longer hold when we consider facultative helping and harming in populations that are both spatially and temporally heterogeneous with respect to group size? Focusing our attention upon facultative helping in big patches, we find that the potential for this to be favoured is given by equation (5), i.e. $A_B = (r_{P|B}- r_{S|B}v_{S|B})/(1- r_{S|B}v_{S|B})$. If patch quality is heterogeneous in space and time, then the actor's big patch may have been either big or little in the previous generation, and so her relatedness to primary recipients $r_{P|B}$ is a weighted average of the product of co-philopatry h_B and whole-group relatedness R_B for big patches and the product of co-philopatry h_L and whole-group relatedness R_L for little patches (see Appendix D for details). In contrast, her relatedness $r_{S|B}$ to the secondary recipients is the product of the probability of co-philopatry h_B and whole-group relatedness R_B for her big patch (see Appendix D for details). Moreover, the reproductive value of her secondary recipients – i.e. adults in her patch in the next generation – need not be equal to her own reproductive value. Whilst she has the reproductive value of an individual in a big patch, theirs is a weighted average of the reproductive values for individuals in big and little patches. Hence, $v_{\mathrm{S|B}} < 1$ (see Appendix E for details).

Consequently, the actor may place different values upon her primary and secondary recipients: $r_{\mathrm{P|B}} \neq r_{\mathrm{S|B}} v_{\mathrm{S|B}}$ (Figure 3; see Appendix F for details). Hence, the potential for facultative helping in big patches may be nonzero, $A_{\mathrm{B}} \neq 0$. The same is true for facultative helping in little patches: $r_{\mathrm{P|L}} \neq r_{\mathrm{S|L}}$ and $v_{\mathrm{S|L}} > 1$, so $r_{\mathrm{P|L}} \neq r_{\mathrm{S|L}} v_{\mathrm{S|L}}$ and $A_{\mathrm{L}} \neq 0$ (Figure 3; see Appendix F for details). We find that there is potential for helping in big patches and potential for harming in little patches ($A_{\mathrm{B}} > 0$, $A_{\mathrm{L}} < 0$; Figure 4).

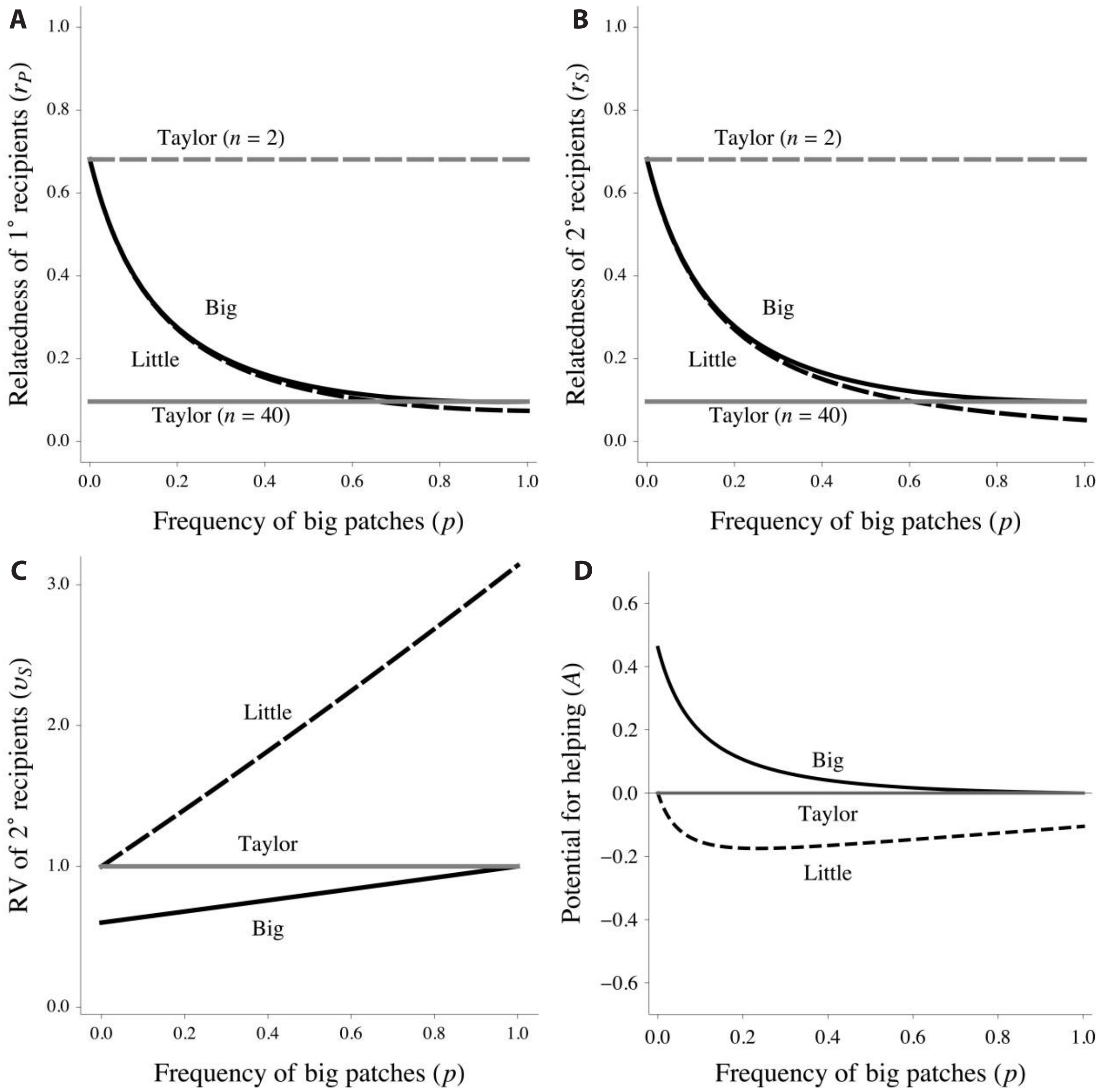

Figure 3 | Impact of spatial and temporal heterogeneity in group size upon relatedness, reproductive value and potential for helping. (a) The relatedness of primary recipients (r_P) as a function of the frequency of big patches (p) is shown for big patches ($r_{\mathrm{P|B}}$; black solid line), little patches ($r_{\mathrm{P|L}}$; black dashed line) and Taylor's (1992) reference model ($r_{\mathrm{P|T}}$; grey lines). The relatedness of primary recipients is higher in big patches owing to higher co-philopatry, and lower in little patches owing to lower co-philopatry, relative to the reference model with equivalent group sizes ($r_{\mathrm{P|B}} > r_{\mathrm{P|T}}$ and $r_{\mathrm{P|L}} < r_{\mathrm{P|T}}$). (b) The relatedness of secondary recipients (r_S) as a function of the frequency of big patches (p). The relatedness of secondary recipients is higher in big patches owing to higher co-philopatry, and lower in little patches owing to lower co-philopatry, relative to the reference model with equivalent group sizes ($r_{\mathrm{S|B}} > r_{\mathrm{S|T}}$ and $r_{\mathrm{S|L}} < r_{\mathrm{S|T}}$). Importantly, in big patches primary recipients are less related to the actor than secondary recipients, while in little patches primary recipients are more related to the actor than secondary recipients ($r_{\mathrm{P|B}} < r_{\mathrm{S|B}}$, $r_{\mathrm{P|L}} > r_{\mathrm{S|L}}$). (c) The relative reproductive value of secondary recipients (υ_{S}) as a function of the frequency of big patches (p). In big patches the relative reproductive value of secondary recipients is less than or equal to that of the reference model, and in little patches the relative reproductive value is greater than or equal to that of the reference model ($\upsilon_{\mathrm{S|B}} \leq \upsilon_{\mathrm{S|T}}$ and $\upsilon_{\mathrm{S|T}} \leq \upsilon_{\mathrm{S|L}}$). (d) The potential for facultative helping (A_{B} and A_{L}) as a function of the frequency of big patches (p). In big patches there is potential for helping ($A_{\mathrm{B}} > 0$), while in little patches there is potential for harming ($A_{\mathrm{L}} < 0$). By numerical analysis, these results were confirmed to hold over the entire range of possible parameter state space. In all panels, numerical examples are given for $n_{\mathrm{B}} = 40$, $n_{\mathrm{L}} = 2$, $m = 0.1$, and $\tau = ½$.

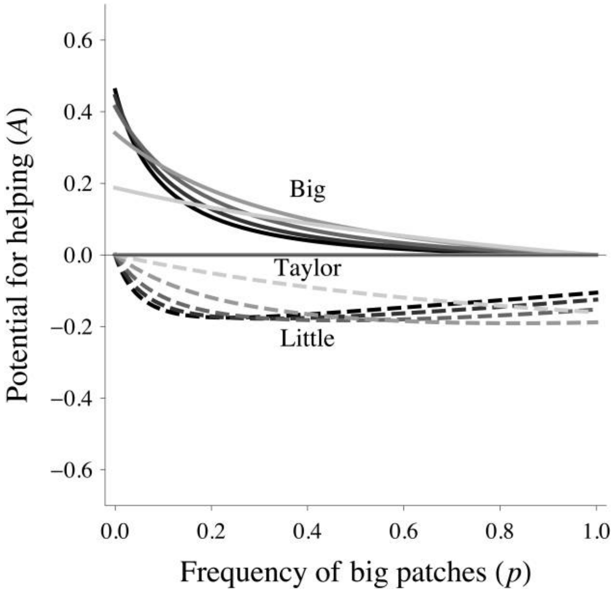

Figure 4 | Impact of spatial and temporal heterogeneity in group size upon the potential for helping. The potential for facultative helping (A_B and A_L) as a function of the frequency of high-quality patches (p), for varying sizes of big patches (n_B = 40, 30, 20, 10, and 4). Darker lines represent larger sizes of big patches, while lighter lines represent smaller sizes of big patches. There is always potential for helping in big patch, while there is always potential for harming in little patches. Parameter values: $n_L = 2$, $m = 0.1$, $\tau = ½$.

Although actors in big patches place different values upon their primary and secondary recipients, and this is also true of actors in little patches, we find that, taking an average over both patch types, actors place equal value upon their primary and secondary recipients. This owes to the properties of the ecological dynamics that determines how patch size changes across generations. Specifically, this is characterised by a time-homogeneous Markov chain, such that the backward processes that determine the value of primary recipients are identical to the forward processes that determine the value of secondary recipients (see Appendix F for details). Hence, whilst there is nonzero potential for facultative helping and harming in big and little patches ($A_B \neq 0$, $A_L \neq 0$), there is zero potential for obligate helping and harming ($A_O = 0$) in populations characterized by both spatial and temporal heterogeneity.

4.3.2 Group-size heterogeneity with variable fecundity

Here we consider that a group's size may impact upon its constituents' fecundity ($0 < \sigma < \infty$.). This enables us to study how the interaction between patch-size heterogeneity and variable fecundity mediates the evolution of obligate and facultative helping and harming. For example, several studies have shown that increasing a group's size may decrease the fitness of its constituent members, for example owing to infectious diseases (e.g. Rifkin et al. 2012). By contrast, other studies have shown that increasing a group's size may increase the fitness of its constituent members, for example owing to a decrease in predation rates or owing to more efficient foraging (e.g. Hamilton 1971b). We derive the following results:

Result 7 | We find that introducing patch productivity heterogeneity has no impact upon the results derived for patch size heterogeneity when: (1) individuals express obligate behaviour; (2) there is spatial heterogeneity only; and (3) there is temporal heterogeneity only.

Results 8 | We find that introducing patch productivity heterogeneity does have an impact upon the results derived for patch size heterogeneity when individuals express facultative behaviour and there is both spatial and temporal heterogeneity.

Why doesn't variable fecundity have and impact on the results obtained for the evolution of helping and harming in a group-size heterogeneous population with homogeneous fecundity when we assume: (1) obligate behaviour, (2) group-size spatial heterogeneity only; and (3) group-size temporal heterogeneity only? Focusing our attention upon cases with obligate behaviour and either spatial or temporal heterogeneity (but not both), we find that the cancellation result obtained for a group-size heterogeneous population (i.e. our Results 1,2,3, and 5) continues to hold, for the same reason, irrespective of variation in fecundity. This is because, whilst variable fecundity alters both the relatedness coefficient and the reproductive value of each recipient, the relationship between the overall value (i.e. life-for-life relatedness) of primary and secondary recipients remains unaltered, and therefore the cancellation result continues to hold.

Why does variable fecundity influence the consequences of group-size heterogeneity in the model with both spatial and temporal heterogeneity? We find that if breeders in little patches have low fecundity (low σ), then facultative helping can be favoured. This is because secondary recipients are usually unrelated immigrants, and therefore the relatedness of secondary recipients is close to zero, making the kin competition effect negligible. By contrast, facultative harming is favoured in big patches. This is because secondary recipients are usually philopatric to the patch, which intensifies the kin competition effect, favouring harming behaviour. By contrast, if breeders in little patches have high fecundity (high σ), then we recover the same qualitative result derived for patch-size heterogeneity with homogeneous fecundity; namely, helping is favoured in big patches whereas harming is favoured in little patches (Figure 5).

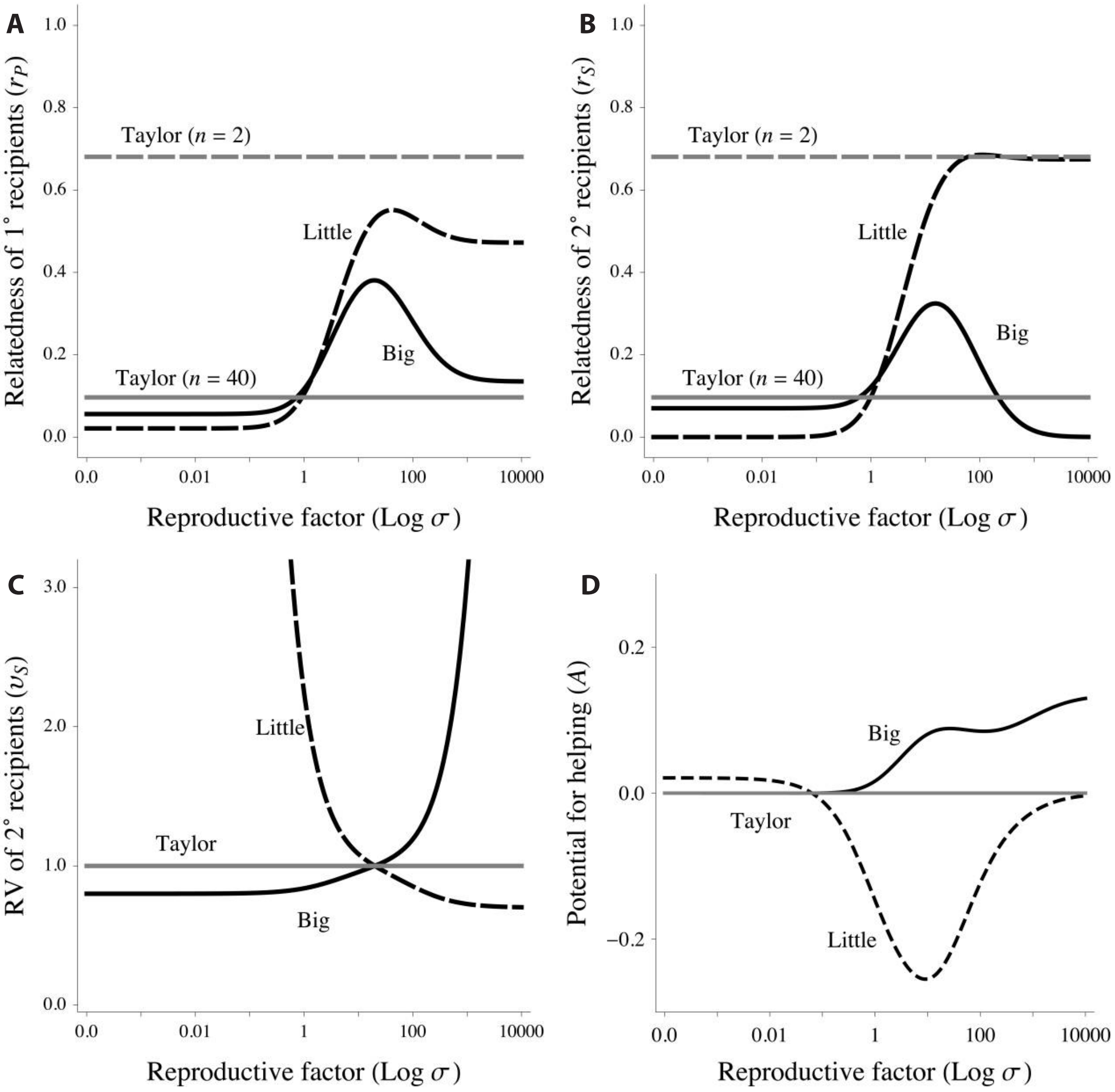

Figure 5 | Impact of spatial and temporal heterogeneity in group size and variable fecundity upon relatedness, reproductive value and potential for helping. (a) The relatedness of primary recipients (r_P) as a function of the reproductive factor (σ) is shown for big patches ($r_{P|B}$; black solid line), little patches ($r_{P|L}$; black dashed line) and Taylor's (1992) reference model ($r_{P|T}$; grey lines). For smaller values of the reproductive factor and considering big patches, the relatedness of primary recipients is less than that of the reference model ($r_{P|B} < r_{P|T}$); while for larger values of the reproductive factor, the relatedness of primary recipients is greater than that of the reference model ($r_{P|B} > r_{P|T}$). In little patches, the relatedness of primary recipients is always less than that of the reference model ($r_{\mathrm{P|L}} < r_{\mathrm{P|T}}$). (b) The relatedness of secondary recipients (r_{S}) as a function of the reproductive factor (σ). For smaller values of the reproductive factor and considering big patches, the relatedness of secondary recipients is less than that of the reference model ($r_{\mathrm{S|B}} < r_{\mathrm{S|T}}$); while for larger values of the reproductive factor, the relatedness of secondary recipients is greater than that of the reference model ($r_{\mathrm{S|B}} > r_{\mathrm{S|T}}$). In little patches, the relatedness of secondary recipients is less than that of the reference model for a wide range of the reproductive factor ($r_{\mathrm{S|L}} < r_{\mathrm{S|T}}$). Importantly, in big patches and for smaller values of the reproductive factor, primary recipients are less related to the actor than secondary recipients ($r_{\mathrm{P|B}} < r_{\mathrm{S|B}}$), while for larger values of the reproductive factor, primary recipients are more related to the actor than secondary recipients ($r_{\mathrm{P|B}} > r_{\mathrm{S|B}}$); by contrast, in little patches, primary recipients are more related to the actor than secondary recipients for smaller values of the reproductive factor ($r_{\mathrm{P|L}} > r_{\mathrm{S|L}}$), while for larger values of the reproductive factor, primary recipients are less related to the actor than secondary recipients ($r_{\mathrm{P|L}} < r_{\mathrm{S|L}}$). (c) The relative reproductive value of secondary recipients (v_{S}) as a function of the reproductive factor (σ). In big patches the relative reproductive value of secondary recipients is less than that of the reference model for smaller values of the reproductive factor ($v_{\mathrm{S|B}} < v_{\mathrm{S|T}}$), while it is greater than that of the reference model for larger values of the reproductive factor ($v_{\mathrm{S|B}} > v_{\mathrm{S|T}}$). In little patches the relative reproductive value is greater than that of the reference model for smaller values of the reproductive factor ($v_{\mathrm{S|T}} < v_{\mathrm{S|L}}$), while it is less than that of the reference model for larger values of the reproductive factor ($v_{\mathrm{S|T}} > v_{\mathrm{S|L}}$). (d) The potential for facultative helping (A_{B} and A_{L}) as a function of the reproductive factor (σ). In big patches, there is potential for harming for smaller values of the reproductive factor ($A_{\mathrm{B}} < 0$), while there is potential for helping for larger values of the reproductive factor ($A_{\mathrm{B}} > 0$). In little patches, there is potential for helping for smaller values of the reproductive factor ($A_{\mathrm{L}} > 0$), while there is potential for harming for larger values of the reproductive factor ($A_L < 0$). In all panels, numerical examples are given for $n_B = 40$, $n_L = 2$, $m = 0.1$, $p = 0.6$, and $\tau = ½$.

4.4. Discussion

Population viscosity has been proposed as a general mechanism that promotes interactions between closely related individuals, and may therefore favour the evolution of kin-selected traits like altruistic cooperation (Hamilton 1964, 1971a). However, it is now well recognized that population viscosity also promotes competition between kin. Here we have shown that Taylor's (1992a) seminal result, that the relatedness and competition effects of viscosity exactly cancel in homogeneous populations, extends to populations where groups vary in size owing to extrinsic factors. Specifically, population viscosity has no impact upon the evolution of obligate helping or harming in populations characterized by spatial and / or temporal heterogeneity in group size. Moreover, if individuals can facultatively adjust their behaviour conditionally upon their group's size, there is no selection for either helping or harming when there is only spatial heterogeneity in group size. However, we have shown that facultative helping is favoured in big groups and facultative harming is favoured in little groups when there is either temporal only or both temporal and spatial heterogeneity in group size. More generally we have shown that Taylor's (1992a) cancellation result for obligate behaviour is robust across a wide range of ecologically realistic scenarios, and we have clarified why this cancellation occurs. Specifically, the proper weights of each selection pressure are the life-for-life relatedness coefficients, which are given in terms recipients' relatednesses and reproductive values, both of which emerge from the specific demographic dynamics of the population (see appendix F for details).

Here we considered that differences in patch size may manifest as differences in the number of breeders that the patches are able to support. Rodrigues and Gardner (2012) also studied how population heterogeneity in patch quality mediates the evolution of social behaviour, when differences in patch quality manifest as differences in the fecundity of the individuals breeding in the patches. In line with the present results, Rodrigues and Gardner (2012) found that spatial and / or temporal heterogeneity have no impact upon the evolution of obligate helping or harming. However, in contrast to the present results, Rodrigues and Gardner (2012) found that both spatial and temporal heterogeneity were required in order for facultative helping and harming to be favoured. Thus, an alteration in a particular assumption as to how patch quality translates into improved productivity – i.e. more breeders having the same fecundity rather than the same number of breeders having greater fecundity – leads to a less stringent condition for population viscosity to promote the evolution of social behaviour. Furthermore, we have explained why variable fecundity influences the results of patch-size heterogeneity and homogeneous fecundity when the spatial and temporal treatment is considered, but not when the temporal treatment alone is considered. This clarifies the importance of understanding how ecological and genetic factors impact upon life-for-life coefficients of relatedness.

Grafen (2007) also studied the evolution of obligate helping in a population characterised by heterogeneity in group size and limited dispersal. However, his model assumed global competition for reproductive resources, and hence the absence of kin competition. Consequently, he found that obligate helping readily evolved in the context of his model. Here we have considered that limited dispersal determines both the genetic structure of the population and the scale of competition. This emphasizes the importance of demography for mediating the costs and benefits of social behaviour as well as the genetic relatedness of social partners. Lehmann et al. (2006b) studied the evolution of social traits in a metapopulation characterized by patch extinctions, and we recover a similar scenario by considering that there are no breeders in little patches ($n_L = 0$). In this special case of our model it is meaningless to distinguish obligate versus facultative behaviour, as all breeders are in big patches, and consequently there is no avenue for helping or harming to be favoured in this scenario. Lehmann et al.'s model considered that transitions between occupied and empty patches are governed by social interaction, allowing helping behaviours to be favoured. This contrasts with our results, which do not require this mechanism for helping and harming to be favoured. Our results also show that environmental heterogeneity favours plastic social responses to local environments, and therefore supports suggestions that plasticity and varying ecological factors can play a role in promoting social behaviour in viscous populations (e.g. Kelly 1992; Queller 1992). More generally, future work should explore how extrinsic factors (e.g. climate change) and intrinsic factors (e.g. social behaviour) interact to drive patch heterogeneity and mediate the evolution of helping and harming.

Our model also relates to the 'bottleneck' hypothesis in the evolution of multicellularity, which suggests that a unicellular bottleneck, followed by a growth cycle in the absence of migration, has been fundamental for the evolution of multicellular organisms (Dawkins 1982; Maynard Smith 1988; Maynard Smith and Szathmáry 1995; Grosberg and Strathmann 1998, 2007; Bourke 2011). These characteristics of the life-cycle already assume pre-adaptations, which can be social traits themselves. Our model predicts that facultative helping is favoured among group mates after the group has passed through a bottleneck and has grown to full size, and it also predicts that facultative harming is favoured before the group has grown to full size (see our Result 4). Therefore, less stringent bottlenecks may have been important to produce pre-adaptations before the full onset of highly complex cooperative societies (e.g. multicellular organisms or eusocial insects). Although some empirical work has been done in this area (Brockhurst 2007; Brockhurst et al. 2007), the kin competition effects of population viscosity in the context of bottlenecks have so far been neglected. Experimental protocols that test the effect of population viscosity in social evolution are already available (Griffin et al. 2004; Kümmerli et al. 2009), and these could be extended to incorporate the effects of spatial and temporal patch size heterogeneity.

Our results show that heterogeneity in patch size and individual fecundity impacts upon the value of each selective force in populations characterized by some degree of viscosity. This happens because: first, it changes the genetic structure of the population and consequently the relatedness of each recipient; and second, it changes the reproductive value of each recipient. Therefore, heterogeneity in patch size and individual fecundity is likely to mediate the evolution of social traits other than those considered here. For example, sex ratio (i.e. the fraction of offspring that are male) is a classic social trait (Hamilton 1967; Charnov 1982; West 2009). Selection pressures acting on sex ratio – including those relating to kin competition – depend both on the relatedness and on the reproductive value of the different recipients (males versus females). Thus, extending our framework to study sex ratio evolution is a promising line of future research.

4.5. Appendix

4.5.1. A – Patch dynamics

We use the transition probabilities in patch quality, given in the main text, to define a transition matrix:

$$\mathbf{P} = \begin{pmatrix} \alpha & 1-\beta \\ 1-\alpha & \beta \end{pmatrix}. \tag{A1}$$

At equilibrium the proportion of big patches is given by

$$p = \frac{1-\beta}{2-(\alpha+\beta)} , \tag{A2}$$

with $\alpha < 1$ and $\beta < 1$. We define a random variable X_t that characterizes a patch's quality (B = 1, L = 0) in any given generation t. The coefficient of temporal correlation between any two generations is given by $\tau = \mathrm{cov}(X_t, X_{t+1})/(\sqrt{\mathrm{var}}(X_t)\mathrm{var}(X_{t+1}))$. In terms of model parameters this is

$$\tau = \alpha - (1-\beta) , \tag{A3}$$

at ecological equilibrium. We find that the temporal correlation τ and the frequency of big patches p cannot be varied totally independently of each other when the temporal correlation is negative ($\tau < 0$). For example, when $\tau = -1$, the frequency of big patches is constrained to be $p = 1/2$.

4.5.2. B – Reproductive success

We follow the life-cycle described in the main text, however, we make the additional assumptions that a mother in a big patch has a large number $F_{\mathrm{B}} = f(x_{\mathrm{B}}, y_{\mathrm{B}})$ of offspring, while a mother in a little patch has a large number $F_{\mathrm{L}} = \sigma f(x_{\mathrm{L}}, y_{\mathrm{L}})$, where $0 < \sigma < \infty$ is the reproductive factor. This recovers the model of Rodrigues and Gardner (2012) as a special case, and hence facilitates connections between the two models. An offspring of a focal breeding mother in a big patch remains in her natal patch with probability 1-m, and subsequently wins a breeding site with probability

$$E^{\bullet}_{\mathrm{B}\to\mathrm{B}} = \frac{f\left(x_{\mathrm{B}}, y_{\mathrm{B}}\right)}{(1-m) f\left(x_{\mathrm{B}}, Y_{\mathrm{B}}\right) n_{\mathrm{B}} + m\left(p f\left(z_{\mathrm{B}}, z_{\mathrm{B}}\right) n_{\mathrm{B}} + (1-p)\sigma f\left(z_{\mathrm{L}}, z_{\mathrm{L}}\right) n_{\mathrm{L}}\right)}, \tag{A4}$$

where Y_{B} is the average level of the social behaviour in the focal patch, and z_{B} and z_{L} are the average levels of the social behaviour across the population in big and little patches, respectively. Conversely, with probability m the offspring migrates to a random patch in the population. With probability p she reaches a big patch, and subsequently wins a breeding site with probability

$$E^{\circ}_{\mathrm{B}\to\mathrm{B}} = \frac{f\left(x_{\mathrm{B}}, y_{\mathrm{B}}\right)}{(1-m) f\left(z_{\mathrm{B}}, z_{\mathrm{B}}\right) n_{\mathrm{B}} + m\left(p f\left(z_{\mathrm{B}}, z_{\mathrm{B}}\right) n_{\mathrm{B}} + (1-p)\sigma f\left(z_{\mathrm{L}}, z_{\mathrm{L}}\right) n_{\mathrm{L}}\right)}. \tag{A5}$$

With probability 1-p she reaches a little patch, and subsequently wins a breeding site with probability

$$E^{\circ}_{\mathrm{B}\to\mathrm{L}} = \frac{f\left(x_{\mathrm{B}}, y_{\mathrm{B}}\right)}{(1-m)\sigma f\left(z_{\mathrm{L}}, z_{\mathrm{L}}\right) n_{\mathrm{L}} + m\left(p f\left(z_{\mathrm{B}}, z_{\mathrm{B}}\right) n_{\mathrm{B}} + (1-p)\sigma f\left(z_{\mathrm{L}}, z_{\mathrm{L}}\right) n_{\mathrm{L}}\right)}. \tag{A6}$$

We derive the same expressions for a mother in a little patch. We may then define a matrix

$$\mathbf{E}=\begin{pmatrix} E_{\mathrm{B}\to\mathrm{B}} & E_{\mathrm{L}\to\mathrm{B}} \\ E_{\mathrm{B}\to\mathrm{L}} & E_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}=\begin{pmatrix} (1-m)E^{\bullet}_{\mathrm{B}\to\mathrm{B}}+mpE^{\mathrm{o}}_{\mathrm{B}\to\mathrm{B}} & mpE^{\mathrm{o}}_{\mathrm{L}\to\mathrm{B}} \\ m(1-p)E^{\mathrm{o}}_{\mathrm{B}\to\mathrm{L}} & (1-m)E^{\bullet}_{\mathrm{L}\to\mathrm{L}}+m(1-p)E^{\mathrm{o}}_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}. \quad \text{(A7)}$$

We define a diagonal matrix **n** where the first element of the diagonal is n_{B} and the second is n_{L}. The matrix that gives the reproductive success of a focal individual for each patch type, in relation to the patch type where their offspring are going to reproduce is then given by

$$\mathbf{w}=\begin{pmatrix} w_{\mathrm{B}\to\mathrm{B}} & w_{\mathrm{L}\to\mathrm{B}} \\ w_{\mathrm{B}\to\mathrm{L}} & w_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}=\mathbf{nPE}\,. \quad \text{(A8)}$$

Thus, the reproductive success of a focal individual is given by

$$W_{\mathrm{B}}=w_{\mathrm{B}\to\mathrm{B}}+\frac{v_{\mathrm{B}}}{v_{\mathrm{L}}}w_{\mathrm{B}\to\mathrm{L}}\text{, and} \quad \text{(A9)}$$

$$W_{\mathrm{L}}=w_{\mathrm{L}\to\mathrm{L}}+\frac{v_{\mathrm{L}}}{v_{\mathrm{B}}}w_{\mathrm{L}\to\mathrm{B}}\,, \quad \text{(A10)}$$

for big and little patches, respectively. The successful offspring must be weighted by their reproductive values (Fisher 1930; Taylor 1990; Grafen 2006). The reproductive value of offspring that reproduce in big patches is v_B, while the reproductive value of offspring that reproduce in little patches is v_L. The reproductive success of a random focal individual in the population is therefore given by

$$W = c_B W_B + c_L W_L \,, \tag{A11}$$

were c_B and c_L are the class reproductive values of big patch and little patches, respectively. Class reproductive values are the product between the frequency of individuals in a given class and their individual reproductive value: $c_B = u_B v_B$ and $c_L = u_L v_L$ for big and little patches, respectively. Class reproductive values are normalized such that $c_B + c_L = 1$ (Taylor 1990, 1996; Taylor et al. 2007).

4.5.3. C – Hamilton’s rule and potential for helping and harming

We assume that the social behaviour is under the control of a single locus. Drawing a gene at random from this locus from a focal individual, we denote its genic value by g. Assuming vanishingly small genetic variation in the population, the direction of selection acting upon the social behaviour is given by

$$\frac{\mathrm{d}W}{\mathrm{d}g} = c_B \frac{\mathrm{d}W_B}{\mathrm{d}g} + c_L \frac{\mathrm{d}W_L}{\mathrm{d}g} \,. \tag{A12}$$

If dW/d$g > 0$, then selection favours an increase in the social behaviour, and if dW/d$g < 0$, then selection favours a decrease in the social behaviour. In the RHS of equation (A12), the derivative in the first term can be expanded as follows:

$$\frac{\mathrm{d}W_{\mathrm{B}}}{\mathrm{d}g} = \frac{\partial W_{\mathrm{B}}}{\partial x_{\mathrm{B}}} \frac{dx_{\mathrm{B}}}{dg_{\mathrm{B}}} \frac{dg_{\mathrm{B}}}{dg} + \frac{\partial W_{\mathrm{B}}}{\partial y_{\mathrm{B}}} \frac{dy_{\mathrm{B}}}{dg'_{\mathrm{B}}} \frac{dg'_{\mathrm{B}}}{dg} + \frac{\partial W_{\mathrm{B}}}{\partial Y_{\mathrm{B}}} \frac{dY_{\mathrm{B}}}{dG_{\mathrm{B}}} \frac{dG_{\mathrm{B}}}{dg} . \tag{A13}$$

All the derivatives are evaluated as $x_{\mathrm{B}} = y_{\mathrm{B}} = Y_{\mathrm{B}} = z_{\mathrm{B}} = z_{\mathrm{L}}$ (Taylor and Frank 1996; Frank 1998). The associations between reproductive success and phenotypes are the effects of the mutant phenotype upon the vital rates of the different recipients. The associations between phenotypes and genotype are the genotype-phenotype mapping and these can be set to unity: dx_{B}/dg_{B} = dy_{B}/dg'_{B}= Y_{B}/dG_{B} = γ_{B} = 1. The associations between genotypes and genic values are the coefficients of consanguinity of the different recipients in relation to the actor. We normalize these coefficients of consanguinity, which give the coefficients of relatedness of the different recipients (Bulmer 1994). These are derived in the next section.

4.5.4. D – Relatedness

We derive the kin selection coefficients of relatedness assuming a neutral population (Wright 1969; Taylor 1992; Rousset 2004). In a focal patch after dispersal, two offspring chosen at random are philopatric to the patch with probability h. With probability $1/n$ they are siblings and have relatedness 1. With probability 1-($1/n$) they are not siblings and they have relatedness r. Hence, relatedness among two offspring chosen at random in a focal patch is given by

$$Q_{\mathrm{B}} = h_{\mathrm{B}}\left(\frac{1}{n_{\mathrm{B}}} + \left(1 - \frac{1}{n_{\mathrm{B}}}\right) r_{\mathrm{B}}\right), \text{ and} \tag{A14}$$

$$Q_{\mathrm{L}} = h_{\mathrm{L}}\left(\frac{1}{n_{\mathrm{L}}} + \left(1 - \frac{1}{n_{\mathrm{L}}}\right) r_{\mathrm{L}}\right), \tag{A15}$$

for big patches and little patches, respectively. A focal big patch was big in the previous generation with probability π_{B}, in which case relatedness among patch mates is Q_{B}. The same patch was little in the previous generation with probability 1-π_{B}, in which case relatedness among patch mates is Q_{L}. A focal little patch was little in the previous generation with probability π_{L}, in which case relatedness among patch mates is Q_{L}. The same patch was big with probability 1-π_{L}, in which case relatedness among patches mates is Q_{B}. This means that relatedness among two patch mates in the next generation is given by

$$\begin{cases} r'_{\mathrm{B}} = \pi_{\mathrm{B}} Q_{\mathrm{B}} + (1 - \pi_{\mathrm{B}}) Q_{\mathrm{L}} \\ r'_{\mathrm{L}} = (1 - \pi_{\mathrm{L}}) Q_{\mathrm{B}} + \pi_{\mathrm{L}} Q_{\mathrm{L}} \end{cases}. \tag{A16}$$

At equilibrium $r'_{\mathrm{B}} = r_{\mathrm{B}}$ and $r'_{\mathrm{L}} = r_{\mathrm{L}}$, and we can solve the equation system (A16) to obtain the 'others-only' kin selection coefficients of relatedness among patches. These are given by

$$r_{\mathrm{B}} = \frac{h_{\mathrm{L}} n_{\mathrm{B}}(1 - \pi_{\mathrm{B}}) + h_{\mathrm{B}}(n_{\mathrm{L}}\pi_{\mathrm{B}} + h_{\mathrm{L}}(n_{\mathrm{L}} - 1)(1 - \pi_{\mathrm{B}} - \pi_{\mathrm{L}}))}{n_{\mathrm{B}}(n_{\mathrm{L}} - (n_{\mathrm{L}} - 1) h_{\mathrm{L}} \pi_{\mathrm{L}}) - h_{\mathrm{B}}(n_{\mathrm{B}} - 1)(n_{\mathrm{L}}\pi_{\mathrm{B}} + h_{\mathrm{L}}(n_{\mathrm{L}} - 1)(1 - \pi_{\mathrm{B}} - \pi_{\mathrm{L}}))}, \text{ and} \tag{A17}$$

$$r_{\mathrm{L}} = \frac{h_{\mathrm{L}} n_{\mathrm{B}} \pi_{\mathrm{L}} + h_{\mathrm{B}}(n_{\mathrm{L}}(1-\pi_{\mathrm{L}}) + h_{\mathrm{L}}(n_{\mathrm{B}}-1)(1-\pi_{\mathrm{B}}-\pi_{\mathrm{L}}))}{n_{\mathrm{B}}(n_{\mathrm{L}} - (n_{\mathrm{L}}-1)h_{\mathrm{L}}\pi_{\mathrm{L}}) - h_{\mathrm{B}}(n_{\mathrm{B}}-1)(n_{\mathrm{L}}\pi_{\mathrm{B}} + h_{\mathrm{L}}(n_{\mathrm{L}}-1)(1-\pi_{\mathrm{B}}-\pi_{\mathrm{L}}))}, \tag{A18}$$

for big and little patches, respectively. The ‘whole-group’ coefficients of relatedness are given by $R = 1/n+(1-(1/n))r$. Relatedness of a focal actor in relation to a primary recipient is equal to the relatedness among patch mates. Hence $r_{\mathrm{P|B}} = r_{\mathrm{B}}$ and $r_{\mathrm{P|L}} = r_{\mathrm{L}}$, for big and little patches, respectively. Relatedness of an actor in relation to a secondary recipient depends on the probability of co-philopatry and the “whole-group” relatedness. This is given by $r_{\mathrm{S|B}} = h_{\mathrm{B}}R_{\mathrm{B}}$ and $r_{\mathrm{S|L}} = h_{\mathrm{L}}R_{\mathrm{L}}$ for big and little patches, respectively.

In spatially heterogeneous populations, patches do not undergo changes in size, hence $\pi_{\mathrm{B}} = 1$ and $\pi_{\mathrm{L}} = 1$. This means that relatedness of primary recipients in big patches is $r_{\mathrm{P|B}} = h_{\mathrm{B}}R_{\mathrm{B}}$. Likewise, relatedness of primary recipients in little patches is $r_{\mathrm{P|L}} = h_{\mathrm{L}}R_{\mathrm{L}}$.

4.5.5. E – Reproductive value

The reproductive values of individuals are given by the left-eigenvector of matrix **w** (Taylor 1990; Grafen 2006). The individual reproductive value is the compound value of the number of offspring times their reproductive value. Multiplying individual reproductive value by group size gives the patch reproductive values: $\upsilon_{\mathrm{B}} = n_{\mathrm{B}}v_{\mathrm{B}}$ and $\upsilon_{\mathrm{L}} = n_{\mathrm{L}}v_{\mathrm{L}}$ for big and little patches, respectively. Relative patch reproductive values are given by

$$\upsilon_{\mathrm{S|B}} = \frac{\alpha\upsilon_{\mathrm{B}} + (1-\alpha)\upsilon_{\mathrm{L}}}{\upsilon_{\mathrm{B}}}, \text{ and} \tag{A19}$$

$$\upsilon_{\mathrm{S|L}} = \frac{\beta \upsilon_{\mathrm{L}} + (1-\beta)\upsilon_{\mathrm{B}}}{\upsilon_{\mathrm{L}}}, \tag{A20}$$

for big and little patches, respectively.

4.5.6. F – Cancellation of obligate helping and harming

Taylor’s (1992a) cancellation result occurs because, in his model, the relatedness of primary recipients is equal to that of secondary recipients, $r_{\mathrm{P|T}} = r_{\mathrm{S|T}}$. This occurs because the probability of co-philopatry used in the derivation of relatedness h_{T}, is equal to the scale of competition $h_{\mathrm{T}} = a_{\mathrm{T}}$ (scale of competition; Frank 1998). This identity still holds for fluctuating environments, $h_{\mathrm{B}} = a_{\mathrm{B}}$ and $h_{\mathrm{L}} = a_{\mathrm{L}}$. We find additional symmetries that lead to the cancellation result for obligate social behaviour. We can write the different quantities in Hamilton’s rule using this notation, such that: $\pi_{\mathrm{B}} = \mathrm{P}(X_{t\text{-}1} = \mathrm{B} \mid X_{\mathrm{t}} = \mathrm{B})$, $1\text{-}\pi_{\mathrm{B}} = \mathrm{P}(X_{\mathrm{t\text{-}1}} = \mathrm{L} \mid X_{\mathrm{t}} = \mathrm{B})$, $\pi_{\mathrm{L}} = \mathrm{P}(X_{\mathrm{t\text{-}1}} = \mathrm{L} \mid X_{\mathrm{t}} = \mathrm{L})$, $1\text{-}\ \pi_{\mathrm{L}} = \mathrm{P}(X_{\mathrm{t\text{-}1}} = \mathrm{B} \mid X_{\mathrm{t}} = \mathrm{L})$, $\alpha = \mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{B})$, $1\text{-}\alpha = \mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{B})$, $\beta = \mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{L})$, and $1\text{-}\beta = \mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{L})$. As a result, we can write the life-for-life relatedness (Hamilton 1972; Bulmer 1994) as

$$\begin{aligned}\rho_{\mathrm{P}} = \quad & p_{\mathrm{B}}(\mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{B}) h_{\mathrm{B}} R_{\mathrm{B}} \upsilon_{\mathrm{B}} + \mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{B}) h_{\mathrm{L}} R_{\mathrm{L}} \upsilon_{\mathrm{B}}) \\ & + p_{\mathrm{L}}(\mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{L}) h_{\mathrm{B}} R_{\mathrm{B}} \upsilon_{\mathrm{L}} + \mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{L}) h_{\mathrm{L}} R_{\mathrm{L}} \upsilon_{\mathrm{L}})\end{aligned}, \text{ and} \tag{A21}$$

$$\begin{aligned}\rho_{\mathrm{S}} = \quad & p_{\mathrm{B}}(\mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{B}) h_{\mathrm{B}} R_{\mathrm{B}} \upsilon_{\mathrm{B}} + \mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{B}) h_{\mathrm{B}} R_{\mathrm{B}} \upsilon_{\mathrm{L}}) \\ & + p_{\mathrm{L}}(\mathrm{P}(X_{t+1} = \mathrm{L} \mid X_t = \mathrm{L}) h_{\mathrm{L}} R_{\mathrm{L}} \upsilon_{\mathrm{L}} + \mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{L}) h_{\mathrm{L}} R_{\mathrm{L}} \upsilon_{\mathrm{B}})\end{aligned}, \tag{A22}$$

We can prove that the life-for-life relatedness coefficients cancel each other in a pairwise way. For example, first note that at equilibrium $\forall t$:$\mathrm{P}(X_{\mathrm{t}} = \mathrm{B}) = p_{\mathrm{B}} = p$ & $\mathrm{P}(X_{\mathrm{t}} = \mathrm{L}) = p_{\mathrm{L}} =$

1-p; second, from Bayes' theorem we find that $\mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{B}) = \mathrm{P}(X_t = \mathrm{B} \mid X_{t-1} = \mathrm{L}) p_\mathrm{L}/p_\mathrm{B}$; third, as the patch dynamics is described by a time-homogeneous Markov chain, we have $\mathrm{P}(X_t = \mathrm{B} \mid X_{t-1} = \mathrm{L}) = \mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{L})$; as a consequence, we find that $p_\mathrm{B}\mathrm{P}(X_{t-1} = \mathrm{L} \mid X_t = \mathrm{B}) = p_\mathrm{L}\mathrm{P}(X_{t+1} = \mathrm{B} \mid X_t = \mathrm{L})$. We can generalise this result for all pairwise terms in equations (A14) and (A15). As a result, the value of primary recipients is equal to the value of secondary recipients $\rho_\mathrm{P} = \rho_\mathrm{S}$, and therefore obligate social behaviour is never favoured.

4.5.7. G – Temporal heterogeneity

We assume that all patches become big with probability p, and become little with probability 1-p, independently of the present state of the population (cf. Iizuka 2001; Rousset 2004 Ch. 10). As a consequence, at any given generation, the probability that patches are in the big state is p, while the probability that patches are in the little state is 1-p. Hence, over time, the expected number of individuals in the big state is $u_\mathrm{B} = pn_\mathrm{B}$, while the expected number of individuals in the little state is $u_\mathrm{L} = (1-p)n_\mathrm{L}$. Given that the population is in the big state, the probability that it remains big is p, in which case the fecundity of a focal individual in a neutral population is 1. With probability 1-p the population changes to the little state, in which case the fecundity of a focal individual in a neutral population is $n_\mathrm{L}/n_\mathrm{B}$. Hence, at equilibrium, the reproductive value of a focal individual in a big patch is $v_\mathrm{B} = pv_\mathrm{B}+(1-p)(n_\mathrm{L}/n_\mathrm{B})v_\mathrm{L}$. This means that $v_\mathrm{B} = (n_\mathrm{L}/n_\mathrm{B})v_\mathrm{L}$. We can normalize the reproductive values such that $v_\mathrm{B} = 1/n_\mathrm{B}$ and $v_\mathrm{L} = 1/n_\mathrm{L}$. Note that the probability that the population was in the big state in a previous generation is independent of the present state of the population, such that $\pi_\mathrm{B} = 1-\pi_\mathrm{L} = p$ and $1-\pi_\mathrm{B} = \pi_\mathrm{L} = 1-p$. As a consequence relatedness coefficients obey to the following identities: $r_\mathrm{B} = phR_\mathrm{B}+(1-p)hR_\mathrm{L}$, and $r_\mathrm{L} = phR_\mathrm{B}+(1-p)hR_\mathrm{L}$. Hence, $r_\mathrm{B} = r_\mathrm{L}$ and $r_{\mathrm{P}\mid\mathrm{B}} = r_{\mathrm{P}\mid\mathrm{L}}$.

Chapter 5. Evolution of helping and harming in heterogeneous groups.

The worst pathologies of the kin-selection criterion arise when genes for social behavior are unconditionally expressed – i.e. expressed by every individual of a given genotype… there are numerous asymmetries that make conditional expression the natural course … Conditionality, although mentioned, was insufficiently emphasized in my previous work.

Hamilton (1987, p433)

5.1. Introduction

Understanding cooperation is a major challenge for evolutionary biology (Hamilton 1996; Sachs et al. 2004; West et al. 2007a; Bourke 2011). Hamilton's (1963, 1964, 1970, 1972, 1975) theory of inclusive fitness provides a general explanation for the adaptive evolution of cooperation. The idea is that individuals can increase their genetic representation in future populations not only by improving their own reproductive success, but also by improving the reproductive success of their genetic relatives. The key result of inclusive fitness theory is Hamilton's rule, which states that natural selection will favour an increase in any trait if $-c + rb > 0$, where c is the direct fitness cost to the actor, b is the benefit enjoyed by the recipient, and r is the genetic relatedness of recipient and actor (Hamilton 1963, 1964, 1970; Charnov 1977). Relatedness has often taken central stage in the literature on inclusive fitness (Lehmann and Keller 2006; Bourke 2011), and is understood to be an important driver of a diversity of evolutionary phenomena, including eusociality (Boomsma 2007, 2009; Hughes et al. 2008), cooperative breeding (Cornwallis et al. 2010), multicellularity (Dawkins 1982; Maynard Smith 1988; Maynard Smith and Szathmáry 1995; Grosberg and Strathmann 1998, 2007), sex allocation (Hamilton 1967; Charnov 1982; West 2009), virulence (Frank 1996), parent-offspring conflict (Trivers 1974) and genomic imprinting (Haig 2002).

One mechanism that can give rise to relatedness between social partners is population viscosity, whereby individuals do not move far from their place of origin during their lives (Hamilton 1964, 1971). In such scenarios, even indiscriminate cooperation towards one's neighbours is likely to involve significant genetic relatedness between interactants. However, limited dispersal may also increase resource competition among social partners, and this tends to inhibit the evolution of cooperation (Hamilton 1971; Taylor 1992a; Queller 1992; Frank 1998; West et al. 2002). In the simplest models of population viscosity these two effects exactly cancel, giving no net effect of dispersal on the evolution of cooperation (Taylor 1992a,b; Kümmerli et al. 2009). This result has motivated a large body of theoretical (and, increasingly, empirical) research on factors that may decouple relatedness and competition to allow viscosity to promote the evolution of helping and harming behaviours (reviewed by West et al. 2002; and Lehmann and Rousset 2010).

Most of this literature has assumed that populations are homogeneous in individual quality, other than differences in sex (but see Frank 1996, 2003, 2010; Johnstone 2008; and Rodrigues and Gardner 2012, 2013a). However, within-group heterogeneities are likely to be important for the evolution of social behaviour, especially in determining which individuals enact which behaviours (Hamilton 1964; Alexander 1974; Michener and Brothers 1974; West-Eberhard 1975; Craig 1983; Alexander et al. 1991; Pamilo 1991; Crozier and Pamilo 1995; West-Eberhard 2003; Bourke 2007; West et al. 2007a). In social amoebae, larger cells tend to develop as reproductive spores rather than altruistic stalk (Leach et al. 1973). In brewer's yeast, damaged cells appear more predisposed to self-sacrifice (Fabrizio et al. 2004; Herker et al. 2004). In primitively eusocial insects, individuals of better nutritional state are more likely to develop as reproductives (Gadagkar et al. 1988), and workers kill their queen if her fecundity drops below a threshold (Bourke 1994). Thus, a better understanding of how within-group variation in individual quality mediates the evolution of helping and harming is desired.

Here, we investigate how within-group variation in quality influences the evolution of helping and harming behaviours. We consider both unconditional social behaviour and also social behaviour that is conditionally adjusted according to the quality of the actor and/or her social partners. We first perform a general analysis that treats genetic relatedness and local competition as separate parameters that can be varied independently of each other (e.g. Frank 1998). We then consider an explicit viscous population model, in which relatedness and local competition emerge from explicit demographic assumptions (e.g. Taylor 1992a). We use our analyses as a foundation upon which to discuss the role for individual quality to mediate the evolution of social behaviours.

5.2. General Model

5.2.1. Basic model

We assume a population of haploid, asexually-reproducing individuals of which a fraction u_H are high-quality and a fraction u_L are low-quality. Low-quality individuals have a fecundity that is a fraction 1-s of that of high-quality individuals. Offspring quality is assigned at random, the individual being high-quality with probability p and low-quality with probability 1-p, independently of parent quality.

Juveniles engage in social interactions that mediate survival to adulthood. Specifically: high-quality juveniles survive with probability $S_H(x_H,y_H,y_L)$, where x_H is the focal juvenile's phenotype, y_H is the average phenotype among high-quality juveniles in the local neighbourhood, and y_L is the average phenotype among low-quality juveniles in the local neighbourhood; and low-quality juveniles survive with probability $S_L(x_L,y_H,y_L)$, where x_L is the focal juvenile's phenotype. We classify social behaviour according to two factors: first, whether or not it is conditioned on the actor's quality; and, second, whether or not it is conditioned on the recipient's quality.

The marginal survival cost of social behaviour is $\partial S_H(x_H,y_H,y_L)/\partial x_H = -C$ and $\partial S_L(x_L,y_H,y_L)/\partial x_L = -C$ for high-quality and low-quality juveniles, respectively. The marginal survival benefit depends on the type of social interaction. For example, if there is no discrimination of recipients' quality and focusing on a focal high-quality individual, the marginal survival benefit is $\partial S_H(x_H,y_H,y_L)/\partial y_H = u_H B$ and $\partial S_H(x_H,y_H,y_L)/\partial y_L = u_L B$; whereas if there is discrimination of recipients' quality, and high-quality individuals are the sole recipients of the social behaviours, the marginal survival benefit is $\partial S_H(x_H,y_H,y_L)/\partial y_H = B$ and $\partial S_H(x_H,y_H,y_L)/\partial y_L = (u_L/u_H)B$. Similar functions can be derived for all other types of behaviour as well as for low-quality individuals. We assume that the costs and benefits of social actions are small, i.e. $C, B << 1$, and we scale survival probability such that the baseline is one (see Appendix A for details).

After social interaction, juveniles compete for scarce resources, where a fraction *a* of the competition is among juveniles in the local neighbourhood, and a fraction 1-*a* of the competition occurs globally (Frank 1998). Thus, fitness is

$$W_{\mathrm{H}}=\frac{S_{\mathrm{H}}\left(x_{\mathrm{H}},y_{\mathrm{H}},y_{\mathrm{L}}\right)}{a\left(u_{\mathrm{H}}S_{\mathrm{H}}\left(y_{\mathrm{H}},y_{\mathrm{L}}\right)+u_{\mathrm{L}}S_{\mathrm{L}}\left(y_{\mathrm{H}},y_{\mathrm{L}}\right)\right)+(1-a)\left(u_{\mathrm{H}}S_{\mathrm{H}}\left(z_{\mathrm{H}},z_{\mathrm{L}}\right)+u_{\mathrm{L}}S_{\mathrm{L}}\left(z_{\mathrm{H}},z_{\mathrm{L}}\right)\right)}\text{, and} \qquad (1)$$

$$W_{\mathrm{L}}=\frac{S_{\mathrm{L}}\left(x_{\mathrm{L}},y_{\mathrm{H}},y_{\mathrm{L}}\right)}{a\left(u_{\mathrm{H}}S_{\mathrm{H}}\left(y_{\mathrm{H}},y_{\mathrm{L}}\right)+u_{\mathrm{L}}S_{\mathrm{L}}\left(y_{\mathrm{H}},y_{\mathrm{L}}\right)\right)+(1-a)\left(u_{\mathrm{H}}S_{\mathrm{H}}\left(z_{\mathrm{H}},z_{\mathrm{L}}\right)+u_{\mathrm{L}}S_{\mathrm{L}}\left(z_{\mathrm{H}},z_{\mathrm{L}}\right)\right)}\text{,} \qquad (2)$$

for high-quality and low-quality juveniles, respectively (see Appendix B for details). The denominator represents the amount of competition for a focal individual, where z_{H} and z_{L} are the population average phenotypes of high-quality and low-quality juveniles, respectively. Note that, owing to the assumption of small marginal effects and identical baseline survival between the two classes, the expectation of fitness over all high-quality individuals, and the expectation of fitness over all low-quality individuals, is unity. Natural selection favours those heritable traits that are, on average, associated with higher fitness. The appropriate average of fitness is given by $W = c_{\mathrm{H}}W_{\mathrm{H}}+c_{\mathrm{L}}W_{\mathrm{L}}$, where c_{H} and c_{L} are the class-reproductive values for high-quality and low-quality individuals, respectively (Fisher 1930; Taylor 1990; Taylor and Frank 1996; Frank 1998; Grafen 2006; see Appendix C for details). Model notation is summarized in Table 1.

Symbol	Meaning
H	High-quality individual
L	Low-quality individual

B	Both high- and low-quality individuals
F	Corresponding to Frank’s (1998) analysis
T	Corresponding to Taylor’s (1992a) analysis
A	Actor
P	Primary recipient
S	Secondary recipient
a	Scale of competition
A_{Z}	Potential for helping corresponding to reference model Z
$A_{\mathrm{X\|Y,Z}}$	Potential for helping where actor is in condition X, and primary recipients are in condition Y, for reference model Z
p	Probability that an offspring becomes a high-quality juvenile
c_{X}	Class reproductive value in condition X ($c_{\mathrm{X}} = u_{\mathrm{X}}v_{\mathrm{X}}$)
F_{X}	Fecundity of a breeding female in condition X ($F_{\mathrm{L}} = (1\text{-}s)F_{\mathrm{H}}$)
h_{X}	Probability of co-philopatry in condition X
m	Migration rate
n	Group-size
n_{e}	Effective group-size
ϱ_{X}	Life-for-life relatedness (or value) of an individual in condition X ($\varrho_{\mathrm{X}} = \upsilon_{\mathrm{X}}r_{\mathrm{X}}$)
r_{X}	‘Others-only’ relatedness of an individual in condition X
s	Reproductive asymmetry
u_{X}	Frequency of individuals in condition X
v_{X}	Reproductive value of an individual in condition X
υ_{X}	Relative reproductive value of an individual in condition X ($\upsilon_{\mathrm{X}} = v_{\mathrm{X}}/v_{\mathrm{A}}$)

x	Average social behaviour of focal actor
y	Average social behaviour of juveniles in focal patch
z	Average social behaviour of juveniles in population
z^*	Convergence stable strategy for the social behaviour

Table 1 | A summary of model notation.

5.2.2. Evolution of helping and harming

We classify social behaviours according to their impact upon the survival of group mates (Lehmann et al. 2006a; West and Gardner 2010). Specifically, helping behaviours as those where $B > 0$, and harming behaviours as those where $B < 0$. We use the neighbour-modulated-fitness approach to kin-selection analysis (Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al. 2007) to determine the direction of selection acting upon social traits (see Appendices B-D for details). This yields a form of Hamilton's (1963, 1964, 1970, 1972) rule:

$$-v_{\mathrm{A}}C + v_{\mathrm{P}}r_{\mathrm{P}}B - (B - C)v_{\mathrm{S}}r_{\mathrm{S}} > 0\ . \qquad (3)$$

This Hamilton's rule comprises three additive terms, each of which can be associated with an individual or set of individuals affected by the behaviour of the actor. The first term concerns effects of the behaviour on the actor; the second term concerns the immediate effects of the behaviour on social partners, i.e. primary recipients; and the third term concerns the kin competition effects of the behaviour on social partners, i.e. secondary recipients.

Thus, inequality (3) can be read as follows: v_A is the actor's reproductive value (Fisher 1930; Taylor 1990, 1996; Grafen 2006); v_P is the reproductive value of the primary recipients; v_S is the reproductive value of the secondary recipients; $r_P = r$ is the relatedness between actor and primary recipients (Hamilton 1964, 1970; Grafen 1985); $r_S = ar$ is the relatedness between actor and secondary recipients (Hamilton 1964, 1970; Grafen 1985), where a is the scale of competition (Frank 1998), i.e. the proportion of secondary recipients who are also primary recipients. Note that if the scale of competition is one ($a = 1$), then relatedness of secondary recipients is equal to that of primary recipients ($r_S = r_P = r$), and that if the scale of competition is zero ($a = 0$), then the relatedness of secondary recipients is zero ($r_S = 0$). Also, because juvenile quality is assigned at random, relatedness is independent of actor and recipient class.

Consequently, an actor's social behaviour has a three-fold impact upon her own inclusive fitness. First, the actor pays a survival cost C, and this is weighted by actor's reproductive value v_A. Second, primary recipients receive a survival benefit B, and this is weighted by primary recipient relatedness r_P and reproductive value v_P. Third, owing to the impact of the social behaviour upon local competition, secondary recipients suffer a cost B-C, and this is weighted by secondary recipient relatedness r_S and reproductive value v_S,

Inequality (3) can be normalized with respect to the actor's reproductive value, to derive what we call relative reproductive values (υ). Note that in this case actor's relative reproductive value is one. Thus, the weights of the marginal survival effects on primary and secondary recipients are now the product of relative reproductive value and relatedness, i.e. "life-for-life relatedness" (ϱ; Hamilton 1972; Bulmer 1994). Specifically, life-for-life relatedness of primary recipients is $\varrho_P = \upsilon_P r$, and life-for-life relatedness of secondary recipients is $\varrho_S = \upsilon_S ar$, where $\upsilon_P = v_P/v_A$ and $\upsilon_S = v_S/v_A$ are the relative reproductive values of primary and secondary recipients, respectively. Life-for-life relatedness describes how much an actor values the recipients of her actions, taking into account not only the extent to which they share genes in common (r), but also their relative capacity to transmit genes to future generations (υ; see Appendix C for details).

Setting the left-hand side (LHS) of inequality (3) equal to zero, we may find the condition where the actor is indifferent with regards to helping/harming slightly more versus slightly less. This condition can be re-arranged into the form $C/B = A$, where A defines the potential for helping (Rodrigues and Gardner 2012; cf Gardner 2010) and $-A$ defines the potential for harming (Rodrigues and Gardner 2012). Assuming that the life-for-life relatedness of a secondary recipient is less than that of the actor ($\varrho_S < 1$), then $A = (\varrho_P - \varrho_S)/(1-\varrho_S)$. Consequently: if the life-for-life relatedness of a primary recipient exceeds that of a secondary recipient ($\varrho_P > \varrho_S$), there is potential for helping ($A > 0$); whereas if the life-for-life relatedness of a secondary recipient exceeds that of a primary recipients ($\varrho_P < \varrho_S$), there is potential for harming. In contrast, if the life-for-life relatedness of a secondary recipient is greater than that of the actor ($\varrho_S \geq 1$), actors are favoured to invest all of their resources into helping or harming, because any cost incurred by an actor is fully compensated by benefits to secondary recipients. This may be better understood if one re-writes Hamilton's rule (inequality 3) in the form $-(1-\varrho_S)C + (\varrho_P - \varrho_S)B > 0$: if $\varrho_S \geq 1$ then $-(1-\varrho_S)C > 0$, which means that the actor's inclusive fitness increases with increasing personal survival cost, even in the absence of any survival benefit to primary recipients. Whether the actor is selected to invest into helping or harming depends upon the value of primary versus secondary recipients ($\varrho_P > \varrho_S$ versus $\varrho_P < \varrho_S$). We establish a convention of setting $A = \infty$ if $\varrho_P > \varrho_S$, and $A = -\infty$ if $\varrho_P < \varrho_S$. Thus, we have

$$\begin{cases} A = (\rho_\text{P} - \rho_\text{S}) / (1 - \rho_\text{S}) & ,\text{if } \rho_\text{S} < 1 \\ A = \infty & ,\text{if } \rho_\text{S} \geq 1 \text{ and } \rho_\text{P} > \rho_\text{S} \\ A = -\infty & ,\text{if } \rho_\text{S} \geq 1 \text{ and } \rho_\text{P} < \rho_\text{S} \end{cases} , \tag{4}$$

where the last two lines define the region of parameter space where actors are selected to invest all of their resources into social interactions. This effect was foreshadowed by Dawkins (1976, p130): "As soon as a runt becomes so small and weak that his expectation of life is reduced to the point where benefit to him due to parental investment is less than half the benefit that the same investment could potentially confer on the other babies, the runt should die gracefully and willingly… he should give up and preferably let himself be eaten by his litter-mates or his parents". We term the portion of parameter space in which the potential for helping has infinite magnitude the "Dawkins' Runt" region. Potential for helping for the different types of behaviour is given in Table 2.

Actors	Recipients		
	Both	High	Low
Both	$\frac{r - ar}{1 - ar}$	$\frac{v_\text{H} r - ar}{1 - ar}$	$\frac{v_\text{L} r - ar}{1 - ar}$
High	$\frac{r - ar}{v_\text{H} - ar}$	$\frac{v_\text{H} r - ar}{v_\text{H} - ar}$	$\frac{v_\text{L} r - ar}{v_\text{H} - ar}$
Low	$\frac{r - ar}{v_\text{L} - ar}$	$\frac{v_\text{H} r - ar}{v_\text{L} - ar}$	$\frac{v_\text{L} r - ar}{v_\text{L} - ar}$

Table 2 | The potential for helping (A) in the general model is shown for different combinations of the actor's quality and the primary recipients' quality

5.3. Island Model

5.3.1. Model

Here we elaborate upon the model presented in the previous section, making the demography of the population more explicit. We assume that the population is subdivided into an infinite number of social groups with n adults each, i.e. Wright's (1931) island model. Groups are composed of n_{H} high-quality adults and n_{L} low-quality adults, where n_{H} and n_{L} are random variables that satisfy the constraint $n = n_{\mathrm{H}}+n_{\mathrm{L}}$. Both high- and low-quality individuals produce a large number of offspring, denoted F_{H} and $F_{\mathrm{L}} = (1-s)F_{\mathrm{H}}$, respectively. After reproduction, all adults die, and juveniles engage in social interactions that mediate their survival to adulthood. After social interaction, each juvenile disperses with probability m to a random group or else, with probability $1-m$, remains in the natal group.

5.3.2. Evolution of helping and harming

As in the previous section, we perform a neighbour-modulated fitness analysis (Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al. 2007; see Appendices B-D for details), to determine the direction of selection acting on the social trait. This yields a Hamilton's rule that takes the same form as given for the previous model (Inequality 3). However, we find that the scale of competition a is now given by the probability of co-philopatry h, i.e. the probability that two individuals sampled at random are philopatric to the group. Hence, $a = h = (1-m)^2$. Moreover, relatedness is given by

$$r = \frac{1}{n_{\mathrm{e}} - (n_{\mathrm{e}} - 1)h} \quad , \tag{5}$$

where n_e is the effective group-size (see Appendix E for details). Reproductive asymmetry decreases effective group-size: $n_e < n$ for $s > 0$. As a result, relatedness is always greater than that of Taylor's (1992a) model ($r > r_T$; Figure 1). We find that relatedness is highest when reproductive asymmetry is high (high s) and migration rate is low (low m), and attains a maximum at an intermediate value p^* of the frequency of high-quality individuals, where $p^* \leq ½$. Furthermore, we find that p^* decreases with increasing reproductive asymmetry. The relatedness of primary recipients is given by $r_P = r$, while that of secondary recipients is given by $r_S = (1\text{-}m)^2 r$. Consequently, the relatedness of primary recipients is always greater than or equal to that of secondary recipients ($r_P \geq r_S$), and the difference between the two is greatest when $p = p^*$. As before, we set the LHS of Hamilton's rule to zero and re-arrange to derive the potential for helping A. These results are given in Table 3.

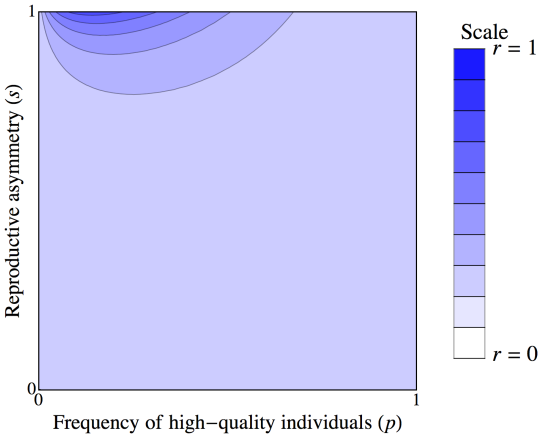

Figure 1 | Relatedness as a function of the frequency of high-quality individuals (p) and reproductive asymmetry (s). Darker shades represent higher relatedness. Parameter values: $m = 0.25$ and $n = 10$.

Actors	Recipients		
	Both	High	Low
Both	$\frac{1}{n_\mathrm{e}}$	$\frac{v_\mathrm{H}-h}{n_\mathrm{e}(1-h)}$	$\frac{v_\mathrm{L}-h}{n_\mathrm{e}(1-h)}$
High	$\frac{1-h}{v_\mathrm{H}n_\mathrm{e}(1-h)+(v_\mathrm{H}-1)h}$	$\frac{v_\mathrm{H}-h}{v_\mathrm{H}n_\mathrm{e}(1-h)+(v_\mathrm{H}-1)h}$	$\frac{v_\mathrm{L}-h}{v_\mathrm{H}n_\mathrm{e}(1-h)+(v_\mathrm{H}-1)h}$
Low	$\frac{1-h}{v_\mathrm{L}n_\mathrm{e}(1-h)+(v_\mathrm{L}-1)h}$	$\frac{v_\mathrm{H}-h}{v_\mathrm{L}n_\mathrm{e}(1-h)+(v_\mathrm{L}-1)h}$	$\frac{v_\mathrm{L}-h}{v_\mathrm{L}n_\mathrm{e}(1-h)+(v_\mathrm{L}-1)h}$

Table 3 | The potential for helping (*A*) in the island model is shown for different combinations of the actor's quality and the primary recipients' quality.

5.4. Analysis and Results

For each of our two models, we define a 'reference model'. In the general-model analysis, the reference model corresponds to a homogeneous population, where each individual's fecundity is identical, i.e. $s = 0$. This recovers the model of Frank (1998, pp114-115), where the potential for helping is $A_\mathrm{F} = (r\text{-}ar)/(1\text{-}ar)$. Note that under the assumptions of Frank's model, the potential for helping is always non-negative ($A_\mathrm{F} \geq 0$), and is greatest when relatedness is high (high r) and when local competition is low (low a). In the island-model analysis, the reference model also corresponds to a homogeneous population, where each individual's fecundity is identical, i.e. $s = 0$. This recovers Taylor's (1992a) asexual model. In this case, the potential for helping is given by $A_\mathrm{T} = (r_\mathrm{T}\text{-}h_\mathrm{T}r_\mathrm{T})/(1\text{-}h_\mathrm{T}r_\mathrm{T})$, where $h_\mathrm{T} = (1\text{-}m)^2$ is the probability of co-philopatry. As relatedness is given by $r_\mathrm{T} = 1/(n\text{-}(n\text{-}1)h_\mathrm{T})$, the potential for helping is given by $A_\mathrm{T} = 1/n$. Consequently, the potential for helping is independent of the migration rate (m) and decreases with increasing group-size

(n). We use these reference models as a benchmark upon which to compare our results for heterogeneous groups ($s > 0$).

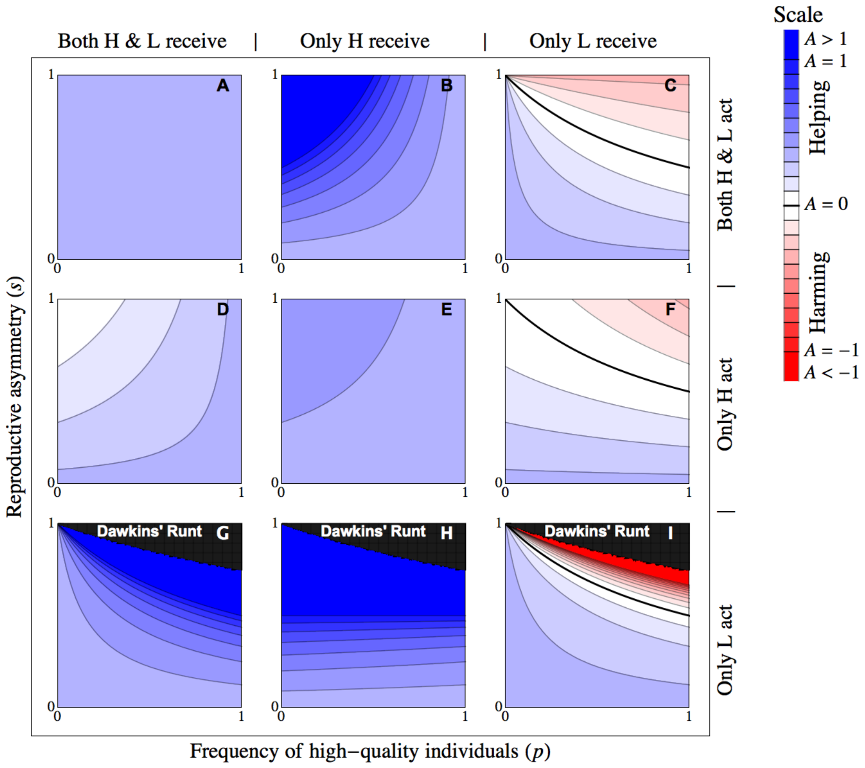

Figure 2 | The potential for helping (A) is shown for different combinations of unconditional and conditional social traits. The potential for helping is given as a function of the frequency of high-quality juveniles (p) and the reproductive asymmetry (s). The potential for helping of Frank's (1998) reference model corresponds to $s = 0$. [A] Unconditional social behaviour: the potential for helping is equal to that of the references model ($A_{\mathrm{B|B,F}} = A_{\mathrm{F}}$). As a result, the potential helping is always non-negative ($A_{\mathrm{B|B,F}} \geq 0$). [B] Behaviour that is unconditional on actor's quality and is directed to high-quality group mates: the potential for helping is always non-negative ($A_{\mathrm{B|H,F}} \geq 0$), and it is always greater than that of the references model ($A_{\mathrm{B|H,F}} > A_{\mathrm{F}}$). [C] Behaviour that is unconditional on actor's quality and is directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{B|L,F} < A_F$); high reproductive asymmetry and high local competition promote harming behaviour ($A_{B|L,F} < 0$). [D] Behaviour expressed by high-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($A_{H|B,F} \geq 0$), and it is always less than that of the reference model ($A_{H|B,F} < A_F$). [E] Behaviour expressed by high-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{H|H,F} \geq 0$), and it is always greater than that of the references model ($A_{H|H,F} > A_F$). [F] Behaviour expressed by high-quality juveniles directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{H|L,F} < A_F$); high reproductive asymmetry and high local competition promote harming behaviour ($A_{H|L,F} < 0$). [G] Behaviour expressed by low-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($\mathrm{A}_{L|B,F} \geq 0$), and it is always greater than that of the reference model ($A_{L|B,F} > A_F$). [H] Behaviour expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{L|H,F} \geq 0$), and it is always greater than that of the reference model ($A_{L|H,F} > A_F$). [I] Behaviour expressed by low-quality juveniles and directed to low-quality group mates: there is potential for helping and potential for harming ($\mathrm{A}_{L|L,F} > 0$ and $\mathrm{A}_{L|L,F} < 0$). The potential for helping is always less than that of the reference model ($A_{L|L,F} < A_F$). [G-I] When secondary recipients' value is greater than or equal to actor's value ($\varrho_S \geq 1$), there is infinite potential for helping ($A_{L|B,F} = \infty$ and $A_{L|H,F} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{L|L,F} = -\infty$, Dawkins' Runt region, panel I). In all cases, relatedness is set to $r = 0.5$ and local competition is set to $a = 0.5$.

We divide our analysis into three sections. In the first section, we consider that actors cannot discriminate recipients' quality. In the second section, we consider that actors can discriminate recipients' quality, and that they direct the social behaviour to high-quality recipients only. In the third section, we consider that actors direct the social behaviour to low-quality recipients only. In each of the three sections, we consider social behaviour that is unconditional or conditional upon actor's quality.

5.4.1. Indiscriminate social behaviour

Individuals often express different phenotypes independently of their genotype and in ways that correlate with their quality. For example, in yeast, programmed cell death (PCD) is mostly expressed by senescent cells (Fabrizio et al. 2004; Herker et al. 2004). In this section we assume that actors cannot discriminate the quality of recipients, hence all individuals in the group are equally likely to be recipients of the social actions, and therefore we focus on unconditional and conditional behaviour on actor's quality.

We find that the potential for unconditional helping is always non-negative ($A_{\mathrm{B|B,F}} \geq 0$ and $A_{\mathrm{B|B,T}} \geq 0$). Furthermore, while in the general model reproductive asymmetry has no impact upon the potential for helping (which is equal to that of Frank's model: $A_{\mathrm{B|B,F}} = A_{\mathrm{F}}$; Figure 2A, Table 2), in the island model reproductive asymmetry does have an impact upon the potential for helping (which is greater than that of Taylor's model: $A_{\mathrm{B|B,T}} > A_{\mathrm{T}}$; Figure 3A, Table 3). In both models, reproductive asymmetry ($s > 0$) does not lead to differences in the reproductive value of actors, primary recipients and secondary recipients ($v_{\mathrm{A}} = v_{\mathrm{P}} = v_{\mathrm{S}}$). However, in the island model, reproductive asymmetry inflates relatedness ($\mathrm{d}r/\mathrm{d}s > 0$), which, in turn, increases the potential for helping ($\mathrm{d}A/\mathrm{d}s > 0$). Importantly, if the reproductive values of actor, primary recipients and secondary recipients are equal, the potential for helping is unaffected by dispersal rate ($\mathrm{d}A_{\mathrm{B|B,T}} /\mathrm{d}m = 0$). This recovers and explains Taylor's (1992a) result. If the reproductive values of actor and recipients are not equal, then the potential for helping may be mediated by dispersal rate ($\mathrm{d}A/\mathrm{d}m \neq 0$).

We now assume that actors can conditionally adjust their behaviour according to their own quality. We start by considering conditionally-adjusted behaviour enacted by high-quality juveniles. We find that the potential for helping is always non-negative ($A_{\mathrm{H|B,F}} \geq 0$ and $A_{\mathrm{H|B,T}} \geq 0$). Moreover, reproductive asymmetry reduces the potential for helping in the general model (for a wide range of parameters), and in the island model ($A_{\mathrm{H|B,F}} < A_{\mathrm{F}}$, and often $A_{\mathrm{H|B,T}} < A_{\mathrm{T}}$; Figures 2D and 3D, and Table 2 and 3). We find that the reproductive value of primary recipients is equal to that of secondary recipients ($v_{\mathrm{P}} = v_{\mathrm{S}}$). The reproductive value of the actor is always greater than that of the primary and secondary recipients ($v_{\mathrm{A}} > v_{\mathrm{P}} = v_{\mathrm{S}}$). This decreases the potential for helping relative to that of the reference models for two reasons: first, it decreases the difference in life-for-life relatedness between primary and secondary recipients (lower ϱ_{P}-ϱ_{S}); second, it increases the difference in life-for-life relatedness between actor and secondary recipient value (higher 1-ϱ_{S}). Turning our attention to the island model, we find that for extreme values of reproductive asymmetry (high s), sufficiently high migration rate (high m) and intermediate frequencies of high-quality juveniles (medium p), there may be a positive effect on the potential for helping, which can be greater than that of the reference model ($A_{\mathrm{H|B,T}} > A_{\mathrm{T}}$). This is because, under these circumstances, the actor is more closely related to primary recipients than to secondary recipients.

We now turn our attention to conditional behaviour of low-quality juveniles. We find that the potential for helping is always non-negative ($A_{\mathrm{L|B,F}} \geq 0$ and $A_{\mathrm{L|B,T}} \geq 0$). Moreover, we find that reproductive asymmetry ($s > 0$) increases the potential for helping ($A_{\mathrm{L|B,F}} > A_{\mathrm{F}}$

and $A_{\mathrm{L|B,T}} > A_{\mathrm{T}}$; Figures 2G and 3G, and Table 2 and 3). We find that while the reproductive value of primary recipients is equal to that of secondary recipients ($v_{\mathrm{P}} = v_{\mathrm{S}}$), the reproductive value of the actor is always less than that of primary and secondary recipients ($v_{\mathrm{A}} < v_{\mathrm{P}} = v_{\mathrm{S}}$). Consequently, actors put more value on their social partners, and this yields a potential for helping that is greater than that of the reference model. Extreme values of reproductive asymmetry (high s), together with high local competition (low m or high a) and sufficiently high frequency of high-quality juveniles (high p), can lead to the life-for-life relatedness of secondary recipients being greater than or equal to that of the actor ($\varrho_{\mathrm{S}} \geq 1$), which favours actors to invest all of their resources into social behaviour ($A_{\mathrm{L|B,F}} = \infty$ and $A_{\mathrm{L|B,T}} = \infty$; Dawkins' Runt region in Figures 2G and 3G).

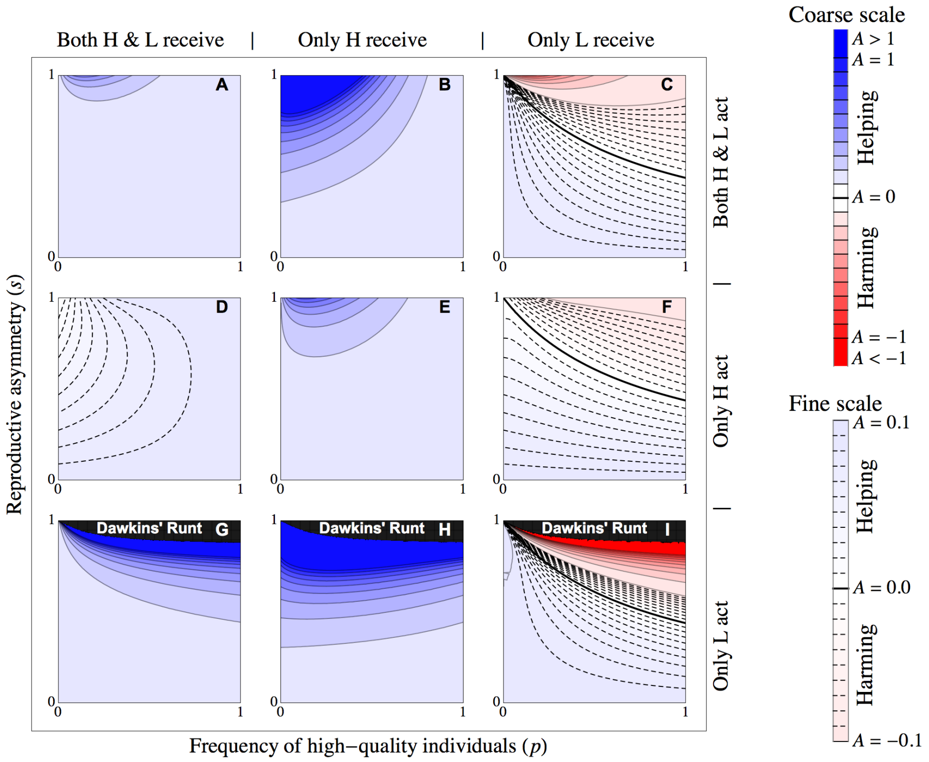

Figure 3 | The potential for helping (A) is shown for different combinations of unconditional and conditional social traits. The potential for helping is given as a function of the frequency of high-quality individuals (p) and reproductive asymmetry (s). The potential for helping of Taylor's (1992a) reference model corresponds to $s = 0$. Darker shades represent higher potential for helping or harming. [A] Unconditional social behaviour: the potential for helping is greater than that of the references model ($A_{\mathrm{B|B,T}} > A_{\mathrm{T}}$). [B] Behaviour that is unconditional on actor's quality and directed to high-quality group mates: the potential for helping is always non-negative ($A_{\mathrm{B|B,T}} \geq 0$), and it is always greater than that of the references model ($A_{\mathrm{B|B,T}} > A_{\mathrm{T}}$). [C] Behaviour that is unconditional on actor's quality and directed to low-quality group mates: the potential for helping is always less than that of the references model ($A_{\mathrm{B|L,T}} < A_{\mathrm{T}}$). High reproductive asymmetry and high local competition promote harming behaviour ($A_{\mathrm{B|L,T}} < 0$). [D] Behaviour expressed by high-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($A_{\mathrm{H|B,T}} \geq 0$), and it is always less than that of the reference model ($A_{\mathrm{H|B,T}} < A_{\mathrm{T}}$). [E] Behaviour expressed by high-quality juveniles and direct to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{H|H,T}} \geq 0$), and it is always greater than that of the reference model ($A_{\mathrm{H|H,T}} > A_{\mathrm{T}}$). [F] Behaviour expressed by high-quality juveniles and directed to low-quality group mates: the potential for helping is always less than that of the references model ($A_{\mathrm{H|L,T}} < A_{\mathrm{T}}$). High reproductive asymmetry and high local competition promote harming behaviour ($A_{\mathrm{H|L,T}} < 0$). [G] Behaviour expressed by low-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|B,T}} \geq 0$), and it is always greater than that of the references model ($A_{\mathrm{L|B,T}} > A_{\mathrm{T}}$). [H] Behaviour expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|H,T}} \geq 0$), and it is always greater than that of the references model ($A_{\mathrm{L|H,T}} \geq A_{\mathrm{T}}$). [I] Behaviour expressed by low-quality juveniles and direct to low-quality group mates: there is potential for helping and potential for harming ($\mathrm{A}_{\mathrm{L|L,T}} > 0$ and $\mathrm{A}_{\mathrm{L|L,T}} < 0$). For a wide range of parameter values, the potential for helping is less than that of the reference model ($A_{\mathrm{L|L,T}} < A_{\mathrm{T}}$). However, if reproductive asymmetry is high and frequency of high-quality juveniles is low, the potential for helping may be greater than that of the reference model ($A_{\mathrm{L|L,T}} > A_{\mathrm{T}}$). [G,H] When secondary recipients' value is greater than or equal to actor's value ($\varrho_{\mathrm{S}} \geq 1$) there is an infinite potential for helping ($A_{\mathrm{L|B,T}} = \infty$ and $A_{\mathrm{L|H,T}} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{\mathrm{L|L,T}} = -\infty$, Dawkins' Runt region, panel I). In all cases, relatedness is set to $n = 10$ and migration rate is set to $m = 0.25$.

5.4.2. Quality discrimination

Individuals often condition their behaviour not only on their own quality, but also on the quality of their social partners. For example, paper-wasp workers appear to directly assess their queen's quality by monitoring her egg production, and behave more selfishly if her fecundity declines (Liebig et al. 2005). In the following two sub-sections, we consider that actors can discriminate the quality of recipients and can direct their behaviour accordingly.

5.4.2.1. High-quality recipients

Here, we assume that actors direct their actions towards high-quality group mates, who are the sole primary recipients of social actions. We begin by considering behaviour that is expressed independently of the actor's quality. We find that the potential for helping is always non-negative ($A_{\mathrm{B|H,F}} \geq 0$ and $A_{\mathrm{B|H,T}} \geq 0$). Furthermore, we find that reproductive asymmetry ($s > 0$) has a positive impact upon the potential for helping ($A_{\mathrm{B|H,T}} > A_{\mathrm{T}}$ and $A_{\mathrm{B|H,F}} > A_{\mathrm{F}}$; Figures 2B and 3B, and Table 2 and 3). This is because, while the reproductive value of the actor is equal to that of secondary recipients ($v_A = v_S$), the reproductive value of primary recipients is greater than that of secondary recipients ($v_P > v_S$).

We now turn our attention to social behaviour that is conditional on the actor's quality. Let us begin by considering that only high-quality juveniles express the behaviour. We find that the potential for helping is always non-negative ($A_{H|H,T} \geq 0$ and $A_{H|H,F} \geq 0$). In addition, reproductive asymmetry ($s > 0$) increases the potential for helping ($A_{H|H,F} > A_F$ and $A_{H|H,T} > A_T$; Figures 2E and 3E, and Table 2 and 3). The reproductive value of primary recipients is greater than that of secondary recipients ($v_P > v_S$), while that of the actor is equal to that of primary recipients ($v_A = v_P$). Thus, while there is a positive impact upon the potential for helping in relation to that of the reference model, the potential for facultative helping of high-quality juveniles is less than the potential for obligate helping ($A_{H|H,F} < A_{B|H,F}$ and $A_{H|H,T} < A_{B|H,T}$)

We now consider that only low-quality juveniles express the behaviour. We find that the potential for helping is always non-negative ($A_{L|H,F} \geq 0$ and $A_{L|H,T} \geq 0$). In addition, reproductive asymmetry ($s > 0$) increases the potential for helping ($A_{L|H,F} > A_F$ and $A_{L|H,T} > A_T$; Figures 2H and 3H, and Table 2 and 3). The reproductive value of primary recipients is greater than that of secondary recipients ($v_P > v_S$). Moreover, the reproductive value of secondary recipients is always greater than that of the actor ($v_S > v_A$). This drives the increase in potential for helping by low-quality juveniles. The life-for-life relatedness of secondary recipients is greater than that of the actor ($\varrho_S \geq 1$) when local competition is high (low m or high a), reproductive asymmetry is high (high s), and frequency of high-quality juveniles is high (high p). This promotes the evolution of unconstrained helping, whereby actors are favoured to invest all of their resources into helping ($A_{\mathrm{L|H,F}} = \infty$ and $A_{\mathrm{L|H,T}} = \infty$; Dawkins' Runt region in Figures 2H and 3H).

5.4.2.2. Low-quality recipients

Here, we consider that individuals can discriminate the quality of recipients, and that low-quality individuals are the sole primary recipients of social actions. We begin by considering behaviour expressed independently of actor's quality. We find that there is both potential for helping and harming ($A_{\mathrm{B|L,F}} > 0$ or $A_{\mathrm{B|L,F}} < 0$ and $A_{\mathrm{B|L,T}} > 0$ or $A_{\mathrm{B|L,T}} < 0$). In addition, we also find that, for a wide range of model parameters, reproductive asymmetry ($s > 0$) decreases the potential for helping ($A_{\mathrm{B|L,F}} < A_{\mathrm{F}}$ and generally $A_{\mathrm{B|L,T}} < A_{\mathrm{T}}$; Figures 2C and 3C, and Table 2 and 3). This occurs because the reproductive value of primary recipients is less than that of secondary recipients ($v_{\mathrm{P}} < v_{\mathrm{S}}$), and the reproductive value of the actor is equal to that of secondary recipients ($v_{\mathrm{A}} = v_{\mathrm{S}}$). However, in the island model, if high-quality juveniles are rare (low p) and migration rate is high (high m), reproductive asymmetry may increase the potential for helping ($A_{\mathrm{B|L,T}} > A_{\mathrm{T}}$). This occurs because relatedness of actors to primary recipients is high, while relatedness to secondary recipients is low. This asymmetry in relatedness offsets the asymmetry in reproductive value.

We now turn our attention to social behaviour that is conditional on actor quality. We first consider behaviour that is expressed by high-quality juveniles only. We find that there is both potential for helping and harming ($A_{\mathrm{H|L,F}} > 0$ or $A_{\mathrm{H|L,F}} < 0$ and $A_{\mathrm{H|L,T}} > 0$ or $A_{\mathrm{H|L,T}} < 0$). In addition, reproductive asymmetry ($s > 0$) decreases the potential for helping ($A_{\mathrm{H|L,F}} < A_{\mathrm{F}}$ and $A_{\mathrm{H|L,T}} < A_{\mathrm{T}}$; Figures 2F and 3F, and Table 2 and 3). We find that the reproductive value of primary recipients is less than that of secondary recipients ($v_{\mathrm{P}} < v_{\mathrm{S}}$)

and that of secondary recipients is always less than that of the actor ($v_S < v_A$). This drives a potential for helping that is always less than that for helping behaviour expressed independently of the actor's quality ($A_{H|L,F} < A_{B|L,F}$ and $A_{H|L,T} < A_{B|L,T}$; if $A_{H|L,F} > 0$ and $A_{H|L,T} > 0$), and a potential for harming that is always less than that for harming behaviour expressed independently of the actor's quality ($A_{H|L,F} > A_{B|L,F}$ and $A_{H|L,T} > A_{B|L,T}$; if $A_{H|L,F} < 0$ and $A_{H|L,T} < 0$). Harming is most favoured when local competition is high (high a or low m), reproductive asymmetry is high (high s), and frequency of high-quality juveniles is high (high p).

We now consider behaviour that is expressed by low-quality juveniles only. We find that there is both potential for helping and harming ($A_{L|L,F} > 0$ or $A_{L|L,F} < 0$ and $A_{L|L,T} > 0$ or $A_{L|L,T} < 0$). We find that reproductive asymmetry ($s > 0$) typically decreases the potential for helping ($A_{L|L,F} < A_F$ and in general $A_{L|L} < A_T$; Figures 2I and 3I, and Table 2 and 3). We find that the reproductive value of primary recipients is always less than that of secondary recipients ($v_P < v_S$). Low frequencies of high-quality individuals (lower p), high reproductive asymmetry (high s) and low local competition (low a or high m) favour high potential for helping. By contrast, high frequencies of high-quality individuals (high p), and high local competition (high a or low m), favour high potential for harming. The life-for-life relatedness of secondary recipients is greater than or equal to that of the actor ($\varrho_S \geq 1$) for higher local competition (high a or low m), higher reproductive asymmetries (high s), and higher frequencies of high-quality individuals (high p). This promotes the evolution of unconstrained helping, whereby actors are favoured to invest all of their resources into harming behaviours ($A_{L|H,F} = -\infty$ and $A_{L|H,T} = -\infty$; Dawkins' Runt region in Figures 2I and 3I).

5.5. Discussion

5.5.1. Helping and harming

We have considered how the impact of within-group variation in individual quality drives the evolution of helping and harming behaviours in structured populations. We have found that the exact cancellation of relatedness and kin competition effects of dispersal, observed in the simplest models of population viscosity (Taylor 1992a,b), need no longer obtain. Specifically, the cancellation result requires that the actor, primary recipients and secondary recipients all have the same (expected) reproductive value and, whilst this is true for unconditional behaviour, it need not be true for behaviour that is conditioned on actor and/or recipient quality. Moreover, we have also shown that low-quality individuals may even be favoured to invest all of their resources into suicidal helping or harming behaviours. This clarifies that heterogeneity in individual quality may be an important factor in social evolution.

5.5.2. Facultative phenotypes

We find that selection operating on social traits that are expressed irrespective of the actor's quality can greatly differ from selection operating on social traits that are conditional on the actor's quality. In particular, the potential for helping or harming unconditional on the actor's quality is always intermediate between the potentials for helping or harming conditional on the actor's quality. Moreover, while the potential for helping of high-quality individuals is always less than the potential for helping that is unconditional on the actor's quality, the potential for helping of low-quality individuals is always greater than the potential for helping that is unconditional on the actor's quality. This is because, all else being equal, a high-quality individual has more to lose in terms of direct genetic contribution to future generations (i.e. reproductive value; Fisher 1930), such that any investment in costly behaviour has a larger negative impact on its fitness than the same costly investment by a low-quality individual. We suggest that these different selection pressures may provide the basis for the evolution of phenotypic divergence between high- and low-quality individuals, leading these to exhibit very different adaptive phenotypes.

A large number of social species exhibit conditional phenotypes within groups, whereby individuals exhibit differences in morphology and / or behaviour independently of their genotypes (Pardi 1948; Wilson 1971; Ross and Matthews 1991; Bourke and Franks 1995; Crozier and Pamilo 1996; Gross 1996; Brockmann 2001; Gadagkar 2001). In agreement with our predictions, there is evidence for helping being associated with low-quality (i.e. poor prospects of future direct fitness), and selfishness being associated with high-quality (i.e. good prospects of future direct fitness). For example, in hanuman langurs, older females exhibit traits associated with helping (e.g. group defence), while younger females exhibit traits associated with selfishness (e.g. harassment of older females). Hrdy & Hrdy (1976) suggest that this owes to differences in relative reproductive value: as older females have lower reproductive value than younger females, they stand to gain more, relative to younger females, from helping younger females than from investing in their own reproduction.

Another example is provided by several species of primitively eusocial wasps, where individuals in better nutritional status are more likely to adopt reproductive roles (Gadagkar et al. 1988, 1991; O’Donnell 1998; Keeping 2002; Hunt et al. 2007, 2010). The idea that individual condition influences social behaviour is also present in the "subfertility hypothesis" (West 1967; West-Eberhard 1969, 1975; Craig 1983), that suggests that individuals with lower reproductive potential should have a higher tendency to become helpers. In our analysis, we have not specified the causes underlying differences in reproductive value. Depending on the specific biological system, these causes could involve multiple factors, such as: differences in ovary development (Pardi 1948), infection status (Shykoff and Schmid-Hempel 1991; O'Donnell 1997), or timing of birth (Queller 1989, 1994).

5.5.3. Quality discrimination

We find that selection operating on social traits that are unconditional on the primary recipients' quality can greatly differ from selection operating in traits that are conditional on the primary recipients' quality. In particular, we find that the potential for helping of traits that are not conditional on recipients' quality is always intermediate between the potentials for helping of traits that are conditional on the recipients' quality. Thus, if actors direct their actions towards high-quality recipients, the potential for helping is always greater than the potential for helping when actors do not discriminate recipients' quality. By contrast, if actors direct their actions towards low-quality recipients, the potential for helping is always lower than the corresponding potential helping when actors do not discriminate recipients' quality. Furthermore, if the social behaviour is directed towards low-quality recipients, harming behaviour can be favoured. These results suggest that natural selection may favour quality discrimination, as this may have important inclusive-fitness consequences to those involved in social interactions.

Previous work has already highlighted the significance of quality discrimination for the evolution of social traits (West-Eberhard 2003, ch 25; Keller and Nonacs 1993). Several empirical studies have proposed a number of quality discrimination mechanisms. For example, female paper wasps can discriminate each others' quality by assessing the number of laid eggs (Liebig et al. 2005) or by use of facial marks or patterns (Tibbetts and Dale 2004; Tibbetts 2006; Tibbetts and Curtis 2007; Cervo et al. 2008), and ponerine ants do so by assessing correlates of quality such as cuticular hydrocarbons (Liebig et al. 2000; D'Ettorre et al. 2004). In the context of social insects, workers monitor their queen's quality, and when the queen's quality starts to decline, workers may shift their behaviour from helping to aggression, which can culminate in worker matricide (Forsyth 1980; Bourke 1994).

5.5.4. Dawkins' Runt

If the differences in the reproductive value of high- and low-quality juveniles are sufficiently large, local competition may drive low-quality juveniles to invest all of their resources into helping. We term an individual that expresses such extreme altruistic behaviour a "Dawkins' Runt" (after Dawkins 1976, p130). This has received some theoretical treatment (e.g. O'Connor 1978; Godfray and Harper 1990), but convincing empirical evidence is lacking. One of the factors that may explain its rarity among traditional organisms is that relatedness is often less than one, which may constrain the evolution of such extreme altruistic behaviour. Moreover, a weak offspring may still be able to recover and derive high reproductive success as an adult. More generally, reproductive value should be an important factor mediating the evolution of family interactions.

Altruistic suicide seems to be more common in unicellular organisms, where recent studies have identified a number of mechanisms that are likely to be suicide programmes.

Several authors have suggested that mechanisms of cell death have evolved as social traits (Lewis 2000; Longo et al. 2005; Buttner et al. 2006; Nedelcu et al. 2011; Reece et al. 2011). For example, in yeast, programmed cell death (PCD) is mostly expressed by senescent cells (Fabrizio et al. 2004; Herker et al. 2004). PCD, in these cases, may be favoured not only because it alleviates local competition, but also because it enriches the environment with additional resources. In the protozoan parasites *Leishmania*, *Trypanosoma cruzi* and *T. brucei*, PCD has been suggested as a mechanism whereby the best cells are chosen to be transmitted to the next host (Debrabant and Nakhasi 2003; Seed and Wenk 2003). Our analysis suggests that local competition for resources may be key to generating the selection pressure for the suicide of low-quality cells.

More generally, we highlight that cooperation can be favoured not only owing to an increase in group productivity, but also owing to an increase in average group quality, that is to say groups that produce fewer but higher-quality individuals may be at an evolutionary advantage in relation to those groups that produce more but lower-quality individuals. Moreover, our framework also predicts that any asymmetry in quality can be reinforced by the action of natural selection. Worse-off cells are selected to give up some of their survival, thereby becoming even more worse-off. This leads to a causal association between helping and survival or, in other words, between kin selected traits and senescence (e.g. Bourke 2007; Ronce and Promislow 2010).

Ronce and Promislow (2010) used a kin selection model of limited dispersal to study the evolution of senescence. They showed that a mother of age x is selected to give up some of her survival if her expected reproductive value v_{x+1} is less than the probability of offspring's co-philopatry $h(x)$ times their reproductive value v_1 ($v_{x+1} < h(x)v_1$; the condition (3.6) of Ronce and Promislow (2010)). If we consider our model for conditional social behaviour, and if we assume that patch size is one ($n = 1$), and that the benefits are zero ($B = 0$), then we find that the selection gradient, as given by Hamilton's rule in inequality (3), is $v_A < hv_S$, which is true when the actor is a low-quality individual: $v_L < hv_S$; and which is equivalent to Ronce and Promislow's (2010) condition (3.6). Note also that this condition says implicitly that the life-for-life relatedness to secondary recipients must be larger than one ($hv_S/v_L = \varrho_S > 1$), which is similar to our definition of the Dawkin's Runt region ($\varrho_S \geq 1$). Studies have suggested that PCD may be associated with benefits provided by the dying cells (i.e. $B > 0$; Herker et al. 2004; Durand et al. 2011). Overall, these findings and studies provide further evidence for an interaction between helping / harming behaviour, kin competition and senescence that represents an interesting avenue for future research.

5.5.5. Individual quality

Here we have focused on one component of individual quality that we defined as an individual's reproductive value (Fisher 1930; Grafen 2006), and which emerges as a weighting factor in Hamilton's rule (Hamilton 1972; Taylor 1990; Taylor & Frank 1996; Frank 1998). In our model, differences in reproductive value emerge because of differences in fecundity between high- and low-quality individuals. These differences in fecundity can result in turn from a number of underlying factors. For example, it has been suggested that ovarian development in paper wasps is positively correlated with fecundity (Pardi 1946). The reproductive value that derives from these traits, which can be said to be intrinsic to the individual, has been called "basal" or "solitary" reproductive value (West-Eberhard 2003, p451), or "inherent reproductive potential" (Röseler 1991, p334).

However, differences in reproductive value can arise for other reasons. For example, an individual can gain a reproductive advantage over group mates by supressing the group mates' reproduction (Vehrencamp 1983a,b). In these cases, differences in reproductive value emerge due to asymmetries in control over social interactions, and this has been called "social reproductive value" (West-Eberhard 2003, p451). Frank (1990, 1998) has analysed natural selection of social traits in terms of three measures of value: marginal value, relatedness and reproductive value. The marginal value criterion states that candidate evolutionary end points are reached when then genetic benefits of extra investment in a fitness component are exactly cancelled by associated genetic costs in a different fitness component (Frank 1990). These marginal values are mediated by relatedness and reproductive value, and implicitly by costs and benefits.

In the context of our model, differences in social skills, which we call "social value", are better captured by asymmetries in costs and benefits affecting either survival or fecundity (i.e. B's and C's; see Appendix F). In the main text, we have assumed that social value is equal among all juveniles (i.e. $C_{\mathrm{H}} = C_{\mathrm{L}}$ and $B_{\mathrm{HH}} = B_{\mathrm{HL}} = B_{\mathrm{LL}} = B_{\mathrm{LH}}$), thus juveniles with high reproductive value are selected to invest less in helping than juveniles with low reproductive value. However, this prediction may be reversed if we also allow differences in social value. For example, juveniles with higher reproductive value may be selected to invest more into helping if they also have higher social value (e.g. suffer a lower cost from expressing helping behaviours, $C_{\mathrm{H}} < C_{\mathrm{L}}$; see Appendix F). In summary, our conceptual framework divides individual quality into reproductive value and social value, both of which interact to mediate selection acting on social traits. Under this conceptual framework, asymmetries in social value may offset asymmetries in reproductive value such that individual quality may be more-or-less uniform among group members, and therefore marked differences among individuals need not lead to differences in social behaviour (see Appendix F).

5.5.6. Social amoebae

The cellular slime mould *Dictyostelium discoideum* has a peculiar life-cycle: when feeding and dividing it lives a relatively solitary life; however, when resources are depleted from the environment, they may initiate aggregation to form a multicellular fruiting body (reviewed by Bonner 2009; Queller et al. 2003). After aggregation, cells either differentiate into stalk cells, dying in the process, or spore cells, which will ultimately disperse to more favourable environments. Thus, stalk-forming cells have been viewed as altruists that benefit the spore-forming cells in the fruiting body (Strassmann et al. 2000).

Most of the research on this topic has been centred on how relatedness mediates the evolution of the altruistic trait (Strassmann et al. 2000; Kuzdzal-Fick et al. 2011). However, several studies have found that stalk-cell differentiation correlates with other factors, such as nutritional status (Leach et al. 1973; Castillo et al. 2011), cell size, or cell-cycle stage (Weijer et al. 1984; McDonald and Durston 1984). These factors do not appear to directly correlate with any characteristic that make cells more efficient stalk cells, and so it is unlikely that a tendency to differentiate as stalk is driven by asymmetries in social value. Rather, these factors may well be correlated with intrinsic differences in the prospects for future reproduction. That is, smaller or food-deprived cells may have lower reproductive value, and hence have less to lose by altruistically developing as stalk.

During development, cells produce a differentiation inducing factor (DIF) that reinforces the predisposition of cells to differentiate into stalk cells (Shaulsky and Loomis 1996). Evidence suggests that the main producers of DIF are cells which already show some predisposition to become spore cells (Kay and Thompson 2001) and, in addition, lower sensitivity to DIF (Thompson and Kay 2000). This has led several authors to interpreted DIF as a competitive trait, whereby fitter cells force weaker cells to differentiate into stalk cells (Atzmony et al. 1997). If this trait has evolved in the context of clonal groups, then standard social evolution theory struggles to explain this “competitive-like” trait. Our results suggest that, owing to reproductive value asymmetries, there is in fact potential for the evolution of competition-like traits which can function as a mechanism for increasing average group quality. Low-quality cells should voluntarily give up their survival, thus sparing high-quality cells from paying the additional costs of inducing others to cooperate. However, constraints at different levels, (e.g. lack of information about relative cell-quality or cells inability to act), may indeed favour the evolution of competition-like traits among genetically identical individuals. This principle may in fact be a general design mechanism of organisms as a way of increasing group average quality in different taxa. This idea is supported by evidence from different species, both unicellular (Khare and Shaulsky 2006) and multicellular (Khare and Shaulsky 2006 ; Sadras and Denison 2009; see also Livnat and Pippenger 2006).

5.5.7. Social wasps

Our results predict that, all else being equal, individuals with lower prospects of future fitness (i.e. low reproductive value) should invest more in helping behaviour, while individuals with higher prospects of future fitness (i.e. high reproductive value) should invest less in helping and behave more selfishly. Several species of social wasps are characterized by reproductive and social dominance hierarchies, where top-ranked females with queen-like physiology and / or behaviour tend to monopolize reproduction, and where low-ranked females with worker-like physiology and / or behaviour tend to have low fecundity (Pardi 1948; West 1967; West-Eberhard 1969; Röseler 1991). Group social dynamics seem to follow the dominance hierarchy, such that if the top-ranked female dies or is lost from the nest it is usually the female just below in rank that assumes the dominant role (Pardi 1948; West-Eberhard 1981; Jeanne 1991). Furthermore, females at the bottom of the hierarchy usually exhibit traits that involve high-risk tasks, whereas high-ranked females are more likely to adopt idle, low-risk behaviours (Gadagkar and Joshi 1983; West-Eberhard 1986; Gadagkar 2001).

Several authors have used verbal arguments based on asymmetries in reproductive value to explain the social structure of these social wasps (West-Eberhard 1981; Jeanne 1991; cf. Field et al. 2006). The basic ideas are: those individuals with lower prospects of future direct fitness should try to derive indirect fitness benefits and therefore adopt riskier cooperative behaviours; those with higher prospects of future reproduction should invest less in riskier cooperative behaviours, and invest more in behavioural or physiological traits associated with their own reproduction; those with the best prospects of future direct fitness should invest their resources in reproductive traits and in other traits that allow them to keep their reproductive dominant role. Although our model does not incorporate all aspects of these groups' biology (e.g. overlapping generations), it yields predictions about how individuals should behave according to the prospects of future fitness.

5.6. Supporting Information to "Evolution of helping and harming in heterogeneous groups"

5.6.1. Appendix A: Survival functions

Here we define the survival functions for each of the unconditional and conditional behaviours. In general, the survival of a focal high-quality juvenile is $S_H(x_H,y_H,y_L) = 1-C_Hx_H+G_{HH}y_H+G_{LH}y_L$, whilst the survival of a focal low-quality juvenile is $S_L(x_L,y_H,y_L) = 1-C_Lx_L+G_{HL}y_H+G_{LL}y_L$, where: the base-line survival is set to one; C_H / C_L is the survival cost paid by the focal high- / low-quality juvenile due to its own investment x_H / x_L in the behaviour; G_{HH} / G_{HL} is the net survival gain for the focal high- / low-quality juvenile due to her high-quality group mates' average investment y_H in the behaviour; finally, G_{LH} / G_{LL} is the net survival gain for the focal high- / low-quality juvenile due to her low-quality group mates' average investment y_L in the behaviour. The particular forms of the net survival gains (G's) depend on who performs the behaviour and on who are the beneficiaries of the social behaviour. Thus, if the actors belong to the class of high-quality juveniles and the recipients belong to the class of low-quality juveniles, then the net survival gains are $G_{HH} = G_{LH} = G_{LL} = 0$, because high-quality juveniles are not beneficiaries and / or low-quality juveniles do not invest in social behaviour ($B_{HH} = B_{LH} = B_{LH} = 0$); while $G_{HL} = (u_H/u_L)B_{HL}$, because low-quality juveniles are beneficiaries and high-quality juveniles are actors. A summary of the net survival gains for each type of unconditional and conditional behaviour is given in Table A1.

Actors	Recipients		
	Both	High	Low
Both	$G_{HH} = u_H B_{HH}$	$G_{HH} = B_{HH}$	$G_{HH} = 0$
	$G_{LH} = u_L B_{LH}$	$G_{LH} = (u_L/u_H) B_{LH}$	$G_{LH} = 0$
	$G_{HL} = u_H B_{HL}$	$G_{HL} = 0$	$G_{HL} = (u_H/u_L) B_{HL}$
	$G_{LL} = u_L B_{LL}$	$G_{LL} = 0$	$G_{LL} = B_{LL}$
High	$G_{HH} = u_H B_{HH}$	$G_{HH} = B_{HH}$	$G_{HH} = 0$
	$G_{LH} = 0$	$G_{LH} = 0$	$G_{LH} = 0$
	$G_{HL} = u_H B_{HL}$	$G_{HL} = 0$	$G_{HL} = (u_H/u_L) B_{HL}$
	$G_{LL} = 0$	$G_{LL} = 0$	$G_{LL} = 0$
Low	$G_{HH} = 0$	$G_{HH} = 0$	$G_{HH} = 0$
	$G_{LH} = u_L B_{LH}$	$G_{LH} = (u_L/u_H) B_{LH}$	$G_{LH} = 0$
	$G_{HL} = 0$	$G_{HL} = 0$	$G_{HL} = 0$
	$G_{LL} = u_L B_{LL}$	$G_{LL} = 0$	$G_{LL} = B_{LL}$

Table A1 | The net survival benefits (G's) are shown for different combinations of the actor's quality and the primary recipients' quality.

In the main text, we assume that survival marginal costs and benefits are independent of actors' and recipients' quality, and therefore they are identical, $C_H = C_L = C$ and $B_{HH} = B_{HL} = B_{LH} = B_{LL} = B$. More generally, individuals may exhibit asymmetries in social value (see Discussion in the main text). In appendix F, we provide an illustrative example where social value among individuals is asymmetric, and therefore C's and B's may depend on the actors' and the recipients' quality.

5.6.2. Appendix B: Reproductive success

Following the life-cycle described in the main text for the island model, we define the class-specific reproductive success of a focal juvenile, that is, the expected number of offspring belonging to a particular class produced by a focal juvenile of a given class. Thus, the reproductive success of a high-quality juvenile that stays in her natal patch via her offspring that become high-quality juveniles is

$$w_{\mathrm{P,H \to H}} = \frac{S_{\mathrm{H}}(x_H, y_{\mathrm{H}}, y_{\mathrm{L}})(1-m)nF_{\mathrm{H}}p}{(1-m)\left(N_{\mathrm{H}}S_{\mathrm{H}}(y_{\mathrm{H}}, y_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(y_{\mathrm{H}}, y_{\mathrm{L}})\right) + m\left(N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})\right)}, \qquad \text{(A1)}$$

where N_{H} and N_{L} is the number of high- and low-quality juveniles, respectively. The reproductive success of a high-quality juvenile that disperses away from her natal patch via her offspring that become high-quality juveniles is

$$w_{\mathrm{D,H \to H}} = \frac{S_{\mathrm{H}}(x_H, y_{\mathrm{H}}, y_{\mathrm{L}})mnF_{\mathrm{H}}p}{N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})}. \qquad \text{(A2)}$$

The sum of these two components of a focal high-quality juvenile's reproductive success gives her reproductive success via her offspring that become high-quality juveniles, hence

$$w_{\mathrm{H \to H}} = w_{\mathrm{P,H \to H}} + w_{\mathrm{D,H \to H}}. \qquad \text{(A3)}$$

We now turn our attention to a focal high-quality juvenile's reproductive success via her offspring that become low-quality juveniles. Thus, the reproductive success of a high-quality juvenile that remains in her natal patch via her offspring that become high-quality juveniles is

$$w_{\mathrm{P,H \to L}} = \frac{S_{\mathrm{H}}(x_{H}, y_{\mathrm{H}}, y_{\mathrm{L}})(1-m)nF_{\mathrm{H}}(1-p)}{(1-m)\left(N_{\mathrm{H}}S_{\mathrm{H}}(y_{\mathrm{H}}, y_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(y_{\mathrm{H}}, y_{\mathrm{L}})\right) + m\left(N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})\right)}. \qquad \text{(A4)}$$

The reproductive success of a high-quality juvenile that disperses away from her natal patch via her offspring that become low-quality juveniles is

$$w_{\mathrm{D,H \to L}} = \frac{S_{\mathrm{H}}(x_{\mathrm{H}}, y_{\mathrm{H}}, y_{\mathrm{L}})mnF_{\mathrm{H}}(1-p)}{N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})}. \qquad \text{(A5)}$$

The sum of these two components of a focal high-quality juvenile's reproductive success gives her reproductive success via her offspring that become low-quality juveniles, hence

$$w_{\mathrm{H \to L}} = w_{\mathrm{P,H \to L}} + w_{\mathrm{D,H \to L}}. \qquad \text{(A6)}$$

Expressions for the reproductive success of a focal low-quality juvenile can be derived in a similar way. The various class-specific reproductive success of a focal individual can be conveniently arranged in a transition matrix. Hence

$$w = \begin{pmatrix} w_{\mathrm{H \to H}} & w_{\mathrm{L \to H}} \\ w_{\mathrm{H \to L}} & w_{\mathrm{L \to L}} \end{pmatrix}. \qquad \text{(A7)}$$

To define the reproductive success of a focal individual we need to weight her class-specific reproductive success by relative reproductive values. Hence, expressions for the reproductive success of a focal individual are

$$W_{\mathrm{H}} = w_{\mathrm{H}\to\mathrm{H}} + \frac{v_{\mathrm{L}}}{v_{\mathrm{H}}} w_{\mathrm{H}\to\mathrm{L}}\text{, and} \tag{A8}$$

$$W_{\mathrm{L}} = w_{\mathrm{L}\to\mathrm{L}} + \frac{v_{\mathrm{H}}}{v_{\mathrm{L}}} w_{\mathrm{L}\to\mathrm{H}}\text{,} \tag{A9}$$

for a focal high- and low-quality juvenile, respectively. The reproductive success of a random individual in the population is given by the class-specific fitness weighted by the correspondent class-reproductive values, as given in the main text (Taylor 1990; Taylor et al. 2007).

For the general model, we pursue an identical approach to that of Frank (1998, p114), see also Boyd (1982), Kelly (1992), and Gardner and West (2006), where the relationship between relatedness and the scale of competition is left undefined. However, we extend this approach to accommodate class-structure. Thus, the reproductive success of a high-quality juvenile via its offspring that become high-quality juveniles is

$$w_{\mathrm{H}\to\mathrm{H}} = \frac{S_{\mathrm{H}}\left(x_{\mathrm{H}}, y_{\mathrm{H}}, y_{\mathrm{L}}\right) F_{\mathrm{H}} p}{a\left(u_{\mathrm{H}} S_{\mathrm{H}}\left(y_{\mathrm{H}}, y_{\mathrm{L}}\right) + u_{\mathrm{L}} S_{\mathrm{L}}\left(y_{\mathrm{H}}, y_{\mathrm{L}}\right)\right) + (1-a)\left(u_{\mathrm{H}} S_{\mathrm{H}}\left(z_{\mathrm{H}}, z_{\mathrm{L}}\right) + u_{\mathrm{L}} S_{\mathrm{L}}\left(z_{\mathrm{H}}, z_{\mathrm{L}}\right)\right)}\,. \tag{A10}$$

The other equations, i.e. $w_{\mathrm{H}\to\mathrm{L}}$, $w_{\mathrm{L}\to\mathrm{H}}$, and $w_{\mathrm{L}\to\mathrm{L}}$, can be defined using the same logic. These are then used to define the transition matrix of the general model, which is analogous to the matrix (A7) of the island model, and to derive the equations (1) and (2) in the main text.

5.6.3. Appendix C: Stable-class frequencies and reproductive value

Class reproductive values are defined as the aggregate reproductive value of all juveniles belonging to a class. Thus they can be expressed as the product of individual reproductive value, v, and the stable-class frequency of juveniles belonging to a class, u, where the population is assumed to be neutral and at equilibrium (Taylor 1990; Taylor 1996; Taylor and Frank 1996). We conveniently set the sum of class-reproductive values to one, $c_H+c_L = 1$. Stable-class frequencies are given by the right-eigenvector of the transition matrix (A7). We find that the stable-class frequency of high-quality juveniles is $u_H = p$, while the frequency of low-quality juveniles is $u_L = 1-p$. Individual reproductive values are given by the elements of the left-eigenvector of the transition matrix (A7) (Taylor 1990; Grafen 2006). We find that the individual reproductive value of a high-quality juvenile is given by $v_H = 1/(p+(1-p)(1-s))$, whilst the individual reproductive value of a low-quality juvenile is given by $v_L = (1-s)/(p+(1-p)(1-s))$. It is important to keep in mind that s is the reproductive asymmetry between high- and low-quality individuals, such that $F_L = (1-s)F_H$. Thus if $s = 0$, $v_H = v_L$. The reproductive value of primary recipients depends on who are the direct beneficiaries of the actors' social actions. Thus, if actors direct their behaviours towards a random group mate, primary recipients' reproductive value is $v_P = u_H v_H+u_L v_L$. If behaviours are directed towards high-quality group mates, primary recipients' reproductive value is $v_P = v_H$. Finally, if behaviours are directed towards low-quality group mates, primary recipients' reproductive value is $v_P = v_L$. As the impact of actors' behaviour on local competition is equally shared among all group members, secondary recipients' reproductive value is always $v_S = u_H v_H + u_L v_L$. We obtain relative reproductive values by dividing recipients' reproductive value by an actor's reproductive value. Assuming facultative social behaviour, and considering a focal high-quality actor, we find that the secondary recipients' relative reproductive value is $\upsilon_{S|H} = v_S/v_H = p+(1-p)(1-s)$. Hence $\partial\upsilon_{S|H}/\partial p = s$, which means that secondary recipients' relative reproductive value always increases with increasing frequency of high-quality individuals. Moreover, we find that $\partial\upsilon_{S|H}/\partial s = -(1-p)$, which means that secondary recipients' relative reproductive value always decreases with increasing reproductive asymmetry. If the actor is a low-quality juvenile, we find that secondary recipients' relative reproductive value is $\upsilon_{S|L} = v_S/v_L = (p+(1-p)(1-s))/(1-s)$. Hence $\partial\upsilon_{S|L}/\partial p = s/(1-s)$, which means that secondary recipients' relative reproductive value always increases with increasing frequency of high-quality individuals. Moreover, we find that $\partial\upsilon_{S|L}/\partial s = p/(1-s)^2$, which means that secondary recipients' relative reproductive value always increases with increasing reproductive asymmetry. As the general model is equivalent to the island model, these reproductive values are also used in the general model.

5.6.4. Appendix D: Hamilton's rule

We use the Taylor-Frank approach (Taylor and Frank 1996; Frank 1997, 1998; Taylor et al. 2007) to derive the direction of selection acting on the social trait. We derive the response to selection of a mutant for the breeding value g; this is given by

$$\frac{\mathrm{d}W}{\mathrm{d}g} = c_H \frac{\mathrm{d}W_H}{\mathrm{d}g} + c_L \frac{\mathrm{d}W_L}{\mathrm{d}g} . \qquad \text{(A11)}$$

Expanding the derivative in the first term on the RHS of equation (A11), we get

$$\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} = \frac{\partial W_{\mathrm{H}}}{\partial x_{\mathrm{H}}}\frac{\mathrm{d}x_{\mathrm{H}}}{\mathrm{d}g_{\mathrm{H}}}\frac{\mathrm{d}g_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial y_{\mathrm{H}}}\frac{\mathrm{d}y_{\mathrm{H}}}{\mathrm{d}g'_{\mathrm{H}}}\frac{\mathrm{d}g'_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial y_{\mathrm{L}}}\frac{\mathrm{d}y_{\mathrm{L}}}{\mathrm{d}g'_{\mathrm{L}}}\frac{\mathrm{d}g'_{\mathrm{L}}}{\mathrm{d}g}, \qquad \text{(A12)}$$

where g_{H} denotes self's breeding value, g'_{H} denotes the high-quality social partners' breeding value, and g'_{L} denotes the low-quality social partners' breeding value. All derivatives and partial derivatives are evaluated at $x_{\mathrm{H}} = y_{\mathrm{H}} = z_{\mathrm{H}}$ and $\mathrm{x}_{\mathrm{L}} = y_{\mathrm{L}} = z_{\mathrm{L}}$ (Taylor and Frank 1996; Frank 1997, 1998; Taylor et al. 2007). The partial derivatives of fitness on phenotype are the marginal survival effects, that is the increment or decrement of survival that occurs owing to the actor's social behaviour. The correlation of the breeding value on the phenotype is the mapping between genotype and phenotype and is given by $\mathrm{d}x_{\mathrm{H}}/\mathrm{d}g_{\mathrm{H}} = \mathrm{d}y_{\mathrm{H}}/\mathrm{d}g'_{\mathrm{H}} = \mathrm{d}y_{\mathrm{L}}/\mathrm{d}g'_{\mathrm{L}} = \gamma_{\mathrm{H}}$, which can be set to one ($\gamma_{\mathrm{H}} = 1$). The correlations between the actor's genic value and the recipients' breeding value are the coefficients of consanguinity. Thus, the correlation between the actor's genic value and the self's breeding value is $\mathrm{d}g_{\mathrm{H}}/\mathrm{d}g$; the correlation between the actor's genic value and the high-quality primary recipients' breeding value is $\mathrm{d}g'_{\mathrm{H}}/\mathrm{d}g$; the correlation between the actor's genic value and the low-quality primary recipients' breeding value is $\mathrm{d}g'_{\mathrm{L}}/\mathrm{d}g$. The kin selection coefficients of relatedness are derived by normalizing the coefficients of consanguinity to the actor's coefficient of consanguinity (Bulmer 1994). An analogous expression can be derived for the direction of selection acting on a focal low-quality juvenile ($\mathrm{d}W_{\mathrm{L}}/\mathrm{d}g$). We find that these various selection gradients (i.e. Hamilton's rule) have an identical mathematical form, which is that of inequality (3) in the main text.

5.6.5. Appendix E: Relatedness

We assume vanishingly small genetic variance in the population. Under these assumptions, the genetic structure of the population at equilibrium is determined using recursion equations (Wright 1969; Taylor 1992; Rousset 2004; Rodrigues and Gardner 2012). Relatedness between two high-quality adults in a patch with j high-quality adults is denoted by $r_{\mathrm{HH},j}$; relatedness between a high-quality adult and a low-quality adult, or between a low-quality adult and a high-quality adult in a patch with j high-quality adults is denoted by $r_{\mathrm{HL},j}$; relatedness between two low-quality adults in a patch with j high-quality adults is denoted by $r_{\mathrm{LL},j}$. With probability $h_{\mathrm{HH},j}$, two high-quality adults are philopatric to the j-type patch. With probability $h_{\mathrm{HL},j}$, two individuals in different classes are philopatric to the j-type patch. With probability $h_{\mathrm{LL},j}$, two low-quality individuals are philopatric to the j-type patch. We find that $h_{\mathrm{HH},j} = h_{\mathrm{HL},j} = h_{\mathrm{LL},j} = h = (1\text{-}m)^2$. Given that two high-quality individuals are philopatric to the patch, the probability that they are derived from high-quality individuals is $Q_{\mathrm{H},j} = (j/(j+(n\text{-}j)(1\text{-}s)))^2$, in which case with probability $1/n_{\mathrm{H},j}$ they are siblings and are related by 1, and with probability $1\text{-}1/n_{\mathrm{H},j}$ they are related by $r_{\mathrm{HH},j}$. Given that two high-quality individuals are philopatric to the patch, with probability $Q_{\mathrm{HL},j} = 2(j(n\text{-}j)(1\text{-}s)/(j+(n\text{-}j)(1\text{-}s))^2)$ they are derived from individuals belonging to different classes, in which case they are related by $r_{\mathrm{HL},j}$. Given that two low-quality individuals are philopatric to the patch, with probability $Q_{\mathrm{L},j} = ((n\text{-}j)(1\text{-}s)/(j+(n\text{-}j)(1\text{-}s)))^2$ they are both derived from low-quality individuals, in which case with probability $1/n_{\mathrm{L},j}$ they are siblings and are related by 1, whereas with probability $1\text{-}1/n_{\mathrm{L},j}$ they are related by $r_{\mathrm{LL},j}$. A similar argument can be made to determine the analogous quantities when individuals belong to different classes, and when individuals belong to the low-quality class. We find that $r_{\mathrm{HH},j} = r_{\mathrm{HL},j} = r_{\mathrm{LL},j} = r_{\mathrm{A}}$, which is given by

$$r_{\mathrm{A}} = \frac{h}{n_{\mathrm{e}} - (n_{\mathrm{e}} - 1)h}\,, \qquad \text{(A13)}$$

where n_{e} is the effective group size. Relatedness among juveniles is given by $r = r_{\mathrm{A}}/h$, which is given by equation (5) in the main text. We find that the effective group-size is given by

$$n_{\mathrm{e}} = \frac{1}{\sum_{j=0}^{n} P_j Q_{\mathrm{H},j} / j + \sum_{j=0}^{n} P_j Q_{\mathrm{L},j} / (n-j)}\,, \qquad \text{(A14)}$$

where $Q_{\mathrm{H},j} = (j/(j+(n\text{-}j)(1\text{-}\underline{s})))^2$, $Q_{\mathrm{L},j} = ((n\text{-}j)(1\text{-}s) /(j+(n\text{-}j)(1\text{-}s)))^2$, and P_{j} the frequency of j-type patches.

5.6.6. Appendix F: Social value

In the main text we have assumed that individuals exhibit no differences in task efficiency (i.e. $C_{\mathrm{H}} = C_{\mathrm{L}}$ and $B_{\mathrm{HH}} = B_{\mathrm{HL}} = B_{\mathrm{LL}} = B_{\mathrm{LL}}$); thus, the marginal costs to the actor and marginal benefits to primary recipients are identical among all juveniles. As a result, differences between the selection pressures acting on high-quality juveniles and those acting on low-quality juveniles were due to differences in reproductive value. However, individuals may exhibit differences in task efficiency. For example, large individuals may be better at defending the group, and therefore they may incur much lower personal costs than smaller individuals who perform the same behaviour. To consider this scenario, we develop the following model. The life-cycle is equal to that of the general model presented in the main text. We consider the scenario where the behaviour is conditional on the actor's quality but not on the recipients' quality. However, in contrast with the general model presented in the main text, we relax the assumption that the costs are independent of the actor's quality. Hence, we may have $C_\mathrm{H} \neq C_\mathrm{L}$. Moreover, we make explicit assumptions about how costs and benefits depend on trait values, i.e. z_H and z_L. We assume that high-quality juveniles perform tasks more efficiently, and therefore they are able to perform a behaviour at a lower cost to themselves. Task efficiency is denoted by ε, with $0 \leq \varepsilon \leq 1$. For example, if $\varepsilon = 0$, then high-quality juveniles perform the social task with the same efficiency of low-quality juveniles; however, if $\varepsilon > 0$, then, high-quality juveniles suffer a lower cost when expressing the social trait than low-quality juveniles. We further assume that the marginal cost of the social trait increases linearly with actor's trait level. Under these assumptions the marginal cots are $C_\mathrm{H} = k(1\text{-}\varepsilon)z_\mathrm{H}$, and $C_\mathrm{L} = kz_\mathrm{L}$, for high- and low-quality juveniles, respectively, where k is assumed to be small, $k << 1$. We assume that the marginal benefits are independent of the recipients' quality, and that they decrease linearly with the group's trait level. Thus $B_\mathrm{HH} = B_\mathrm{HL} = B_\mathrm{LH} = B_\mathrm{LL} = k(1\text{-}z)$, where $z = pz_\mathrm{H}+(1\text{-}p)z_\mathrm{L}$ is the group's trait level. Taking into account that $C_\mathrm{H} \neq C_\mathrm{L} \neq C$, we can use Hamilton's rule (inequality 3 in the main text), to derive the selection gradients acting on the social traits. These are given by:

$$-(1-\varepsilon)z_\mathrm{H} + (1-z)(p+(1-p)(1-s))r - a(p+(1-p)(1-s))(1-z-(1-\varepsilon)z_\mathrm{H})r > 0 \text{ , and} \tag{A15}$$

$$-(1-s)z_\mathrm{L} + (1-z)(p+(1-p)(1-s))r - a(p+(1-p)(1-s))(1-z-z_\mathrm{L})r > 0 \text{ ,} \tag{A16}$$

for high-quality juveniles and low-quality juveniles, respectively. To determine the equilibrium rates of investment in the social trait, z_H^* and z_L^*, we set the LHS of inequalities (A15) and (A16) to zero and solved both equations simultaneously. We define the LHS of inequalities (A15) and (A16) as $J_{\mathrm{H}}(z_{\mathrm{H}},z_{\mathrm{L}})$ and $J_{\mathrm{L}}(z_{\mathrm{H}},z_{\mathrm{L}})$, respectively. We check the equilibria for convergence stability (CS, Eshel and Motro 1981; Christiansen 1991; Taylor 1996) by defining matrix **J**, where the first and second elements of the first row are given by $\partial J_{\mathrm{H}}(z_{\mathrm{H}},z_{\mathrm{L}})/ \partial z_{\mathrm{H}}|_{z\mathrm{H}=z\mathrm{H}*,z\mathrm{L}=z\mathrm{L}*}$, and $\partial J_{\mathrm{H}}(z_{\mathrm{H}},z_{\mathrm{L}})/\partial z_{\mathrm{L}}|_{z\mathrm{H}=z\mathrm{H}*,z\mathrm{L}=z\mathrm{L}*}$, respectively; whilst the first and second elements of the second row are $\partial J_{\mathrm{L}}(z_{\mathrm{H}},z_{\mathrm{L}})/\partial z_{\mathrm{H}}|_{z\mathrm{H}=z\mathrm{H}*,z\mathrm{L}=z\mathrm{L}*}$, and $\partial J_{\mathrm{L}}(z_{\mathrm{H}},z_{\mathrm{L}})/\partial z_{\mathrm{L}}|_{z\mathrm{H}=z\mathrm{H}*,z\mathrm{L}=z\mathrm{L}*}$, respectively. We find that both eigenvalues of matrix **J** are always negative, and therefore $z_{\mathrm{H}}*$ and $z_{\mathrm{L}}*$ are convergence stable (Otto and Day 2007). We find that if the task efficiency of high-quality juveniles is high (high ε) and if local competition is low (low a), then high-quality juveniles are selected to invest more into helping than low-quality juveniles ($z_{\mathrm{H}}* > z_{\mathrm{L}}*$; Figure A1). In the absence of local competition ($a = 0$), if $(1- \varepsilon) = (1-s)$ then high- and low-quality juveniles are selected to invest the same amount of resources in helping ($z_{\mathrm{H}}* = z_{\mathrm{L}}*$; circle in Figure A1). That is the high reproductive value of high-quality individuals ($v_{\mathrm{H}} > v_{\mathrm{L}}$) selects them to invest less in helping, however this is offset by their high social value ($C_{\mathrm{H}} < C_{\mathrm{L}}$), which selects them to invest more in helping.

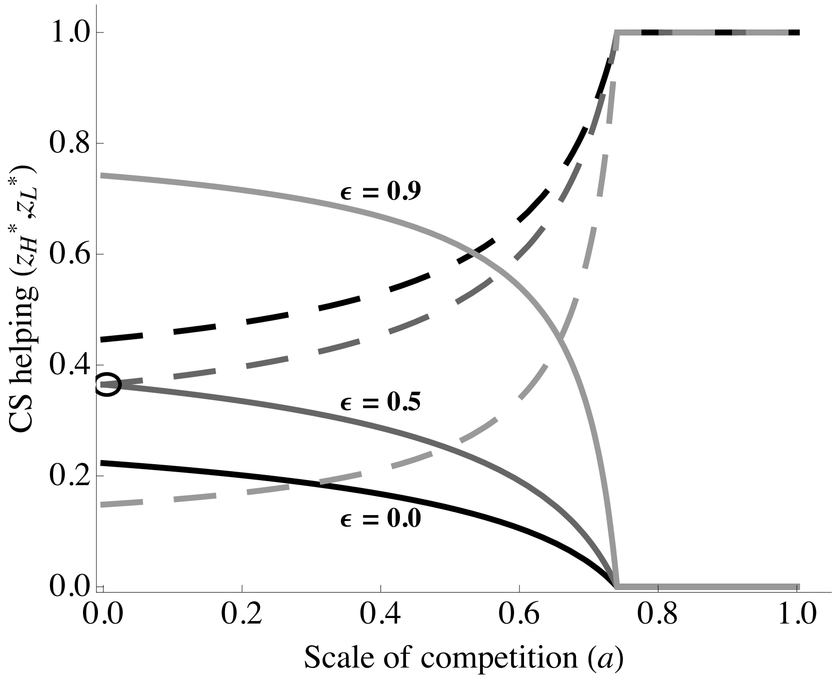

Figure A1 | Convergence stable helping for high-quality individuals (z_H*; solid lines) and low-quality individuals (z_L*; dashed lines), as a function of the scale of competition (a), for various values of social efficiency (ε). If social efficiency of high-quality individuals is equal to the social efficiency of low-quality individuals ($\varepsilon = 0$), high-quality individuals are selected to invest less in helping than low-quality individuals. By contrast, if the social efficiency of high-quality individuals is high (high ε), then they may be selected to invest more in helping than low-quality individuals. Without local competition ($a = 0$), when (1-s) = (1- ε), all individuals invest the same in helping ($z_H^* = z_L^*$, circled point). Parameter values: $p = 0.5$, $s = 0.5$, $r = 0.9$.

5.6.7. Supplementary Figure S1

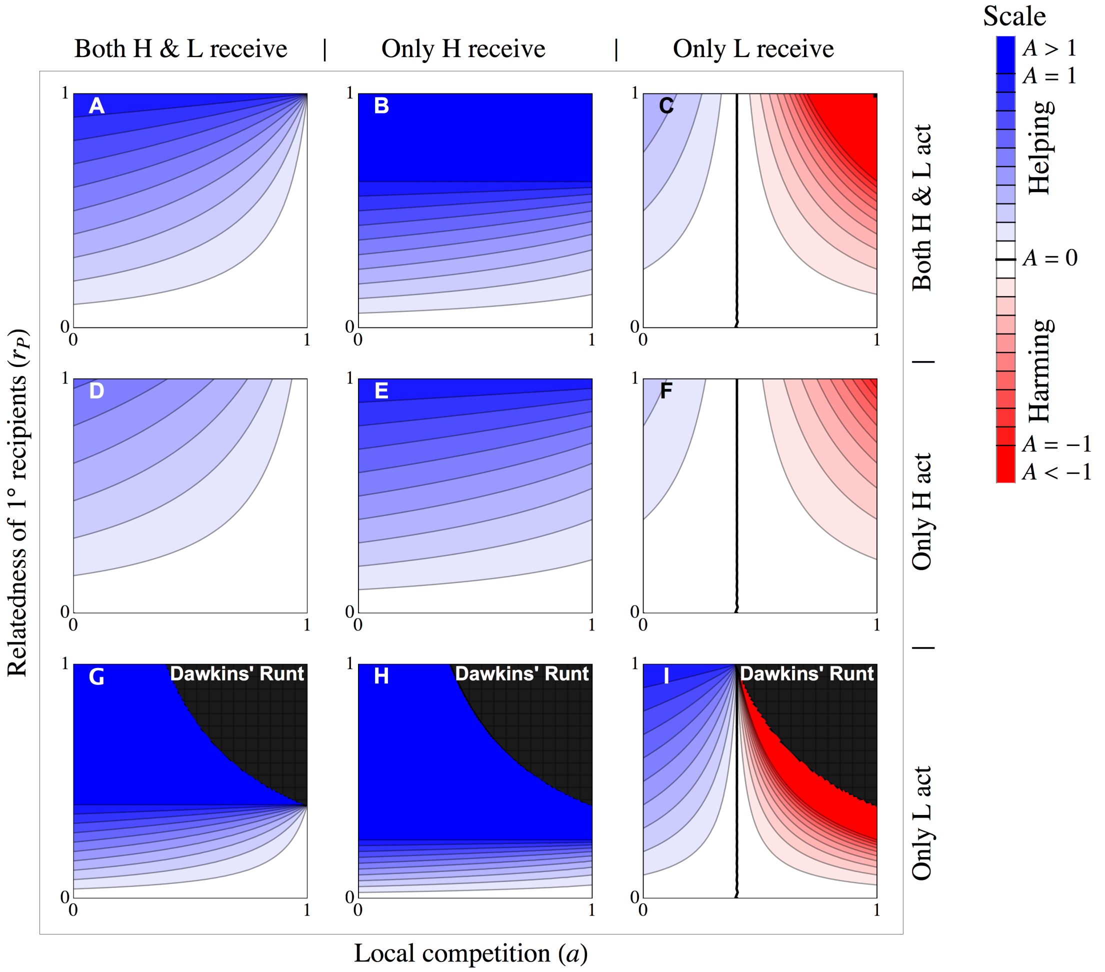

Figure S1 | The potential for helping (A) is shown for unconditional and conditional behaviour. The potential for helping is given as a function of local competition (a) and the relatedness of 1° recipients (r_P). The potential for helping of Frank's (1998) reference model corresponds to $s = 0$. [A] Unconditional social behaviour: the potential for helping is equal to that of the reference model ($A_{B|B,F} = A_F$). As a result, helping is always non-negative ($A_{B|B,F} \geq 0$), and it increases with increasing relatedness of 1° recipients while it decreases with increasing local competition. [B] Behaviour that is unconditional on actor's quality and is directed to high-quality group mates: the potential for helping is always non-negative ($A_{B|H,F} \geq 0$), and it is always greater than that of the reference model

($A_{\mathrm{B|H,F}} > A_{\mathrm{F}}$), and it increases with increasing relatedness of 1° recipients while it decreases with increasing local competition. [C] Behaviour that is unconditional on actor's quality and is directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{\mathrm{B|L,F}} < A_{\mathrm{F}}$); the potential for helping decreases and the potential for harming increases with increasing local competition. Both the potential for helping and harming increase with increasing relatedness of 1° recipients. [D] Behaviour expressed by high-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($A_{\mathrm{H|B,F}} \geq 0$), and it is always less than that of the reference model ($A_{\mathrm{H|B,F}} < A_{\mathrm{F}}$). The potential for helping increases with increasing relatedness of 1° recipients, while it decreases with increasing local competition. [E] Behaviour expressed by high-quality juveniles directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{H|H,F}} \geq 0$), and it is always greater than that of the reference model ($A_{\mathrm{H|H,F}} > A_{\mathrm{F}}$). The potential for helping increases with increasing relatedness of 1° recipients, while it decreases with increasing local competition. [F] Behaviour expressed by high-quality juveniles and directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{\mathrm{H|L,F}} < A_{\mathrm{F}}$); the potential for helping decreases and the potential for harming increases with increasing local competition. Both the potential for helping and harming increase with increasing relatedness of 1° recipients. [G] Behaviour expressed by low-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|B,F}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{L|B,F}} > A_{\mathrm{F}}$). The potential for helping increases with increasing relatedness of 1° recipients. For lower relatedness of 1° recipients, the potential for helping decreases with increasing local competition. For higher relatedness of 1° recipients, by contrast, the potential for helping increases with increasing local competition. [H] Behaviour expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|H,F}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{L|H,F}} > A_{\mathrm{F}}$). The potential for helping increases with increasing relatedness of 1° recipients. For lower relatedness of 1° recipients, the potential for helping decreases with increasing local competition. For higher relatedness of 1° recipients, by contrast, the potential for helping increases with increasing local competition. [I] Behaviour expressed by low-quality juveniles and directed to low-quality group mates: there is potential for helping and potential for harming ($\mathrm{A}_{\mathrm{L|L,F}} > 0$ and $\mathrm{A}_{\mathrm{L|L,F}} < 0$). The potential for helping is always less than that of the reference model ($A_{\mathrm{L|L,F}} < A_{\mathrm{F}}$). The potential for helping decreases and the potential for harming increases with increasing local competition. Both the potential for helping and harming increase with increasing relatedness of 1° recipients. [G-I] When secondary recipients' value is greater than or equal to actor's value ($\varrho_{\mathrm{S}} \geq 1$), there is infinite potential for helping ($A_{\mathrm{L|B,F}} = \infty$ and $A_{\mathrm{L|H,F}} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{\mathrm{L|L,F}} = -\infty$, Dawkins' Runt region, panel I). In all cases, frequency of high-quality juveniles is set to $p = 0.5$, and reproductive asymmetry is set to $s = 0.75$.

5.6.8. Supplementary Figure S2

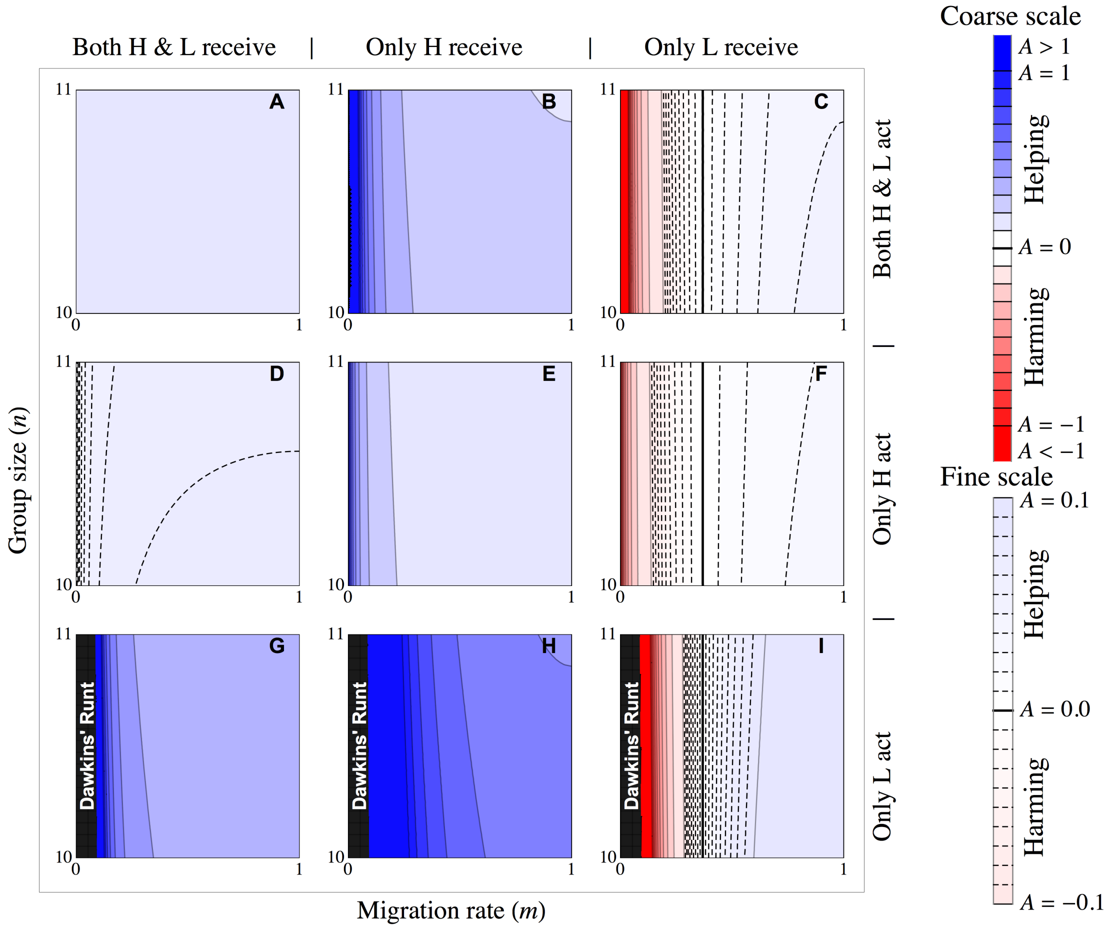

Figure S2 | The potential for helping (A) is shown for unconditional and conditional behaviour. The potential for helping is given as a function of the frequency of high-quality individuals (p) and the reproductive asymmetry (s). The potential for helping of Taylor's (1992a) reference model corresponds to $s = 0$. [A] Unconditional social behaviour: the potential for helping is greater than that of the references model ($A_{\mathrm{B|B,T}} > A_{\mathrm{T}}$). [B] Behaviour that is unconditional on actor's quality and is directed to high-quality group mates: the potential for helping is always non-negative ($A_{\mathrm{B|H,T}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{B|H,T}} > A_{\mathrm{T}}$), and it decreases with increasing group size and increasing migration rate. [C] Behaviour that is unconditional on actor's quality and is directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{\mathrm{B|L,T}} < A_{\mathrm{T}}$); the potential for helping increases and the potential for harming decreases with increasing migration rate. Both, the potential for helping and harming, decrease with increasing group size. [D] Behaviour expressed by high-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($A_{\mathrm{H|B,T}} \geq 0$), and it is always less than that of the reference model ($A_{\mathrm{H|B,T}} < A_{\mathrm{T}}$). The potential for helping decreases with increasing group size increasing migration rate. [E] Behaviour expressed by high-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{H|H,T}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{H|H,T}} > A_{\mathrm{T}}$). [F] Behaviour expressed by high-quality juveniles and directed to low-quality group mates: the potential for helping increases and the potential for harming decreases with increasing migration. Both the potential for helping and harming decrease with increasing group size. [G] Behaviour expressed by low-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|B,T}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{L|B,T}} > A_{\mathrm{T}}$). The potential for helping decreases with increasing group size. The potential for helping decreases with increasing migration rate. [H] Behaviour expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|H,T}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{L|H,T}} \geq A_{\mathrm{T}}$). The potential for helping decreases with increasing group size. The potential for helping decreases with increasing migration rate. [I] Behaviour expressed by low-quality juveniles, and directed to low-quality group mates: there is potential for helping and potential for harming ($\mathrm{A}_{\mathrm{L|L,T}} > 0$ and $\mathrm{A}_{\mathrm{L|L,T}} < 0$).The potential for helping increases and the potential for harming decreases with increasing migration rate. Both the potential for helping and harming decrease with increasing group size. [G-I] When secondary recipients' value is greater than or equal to actor's value ($\varrho_S \geq 1$) there is an infinite potential for helping ($A_{L|B,T} = \infty$ and $A_{L|H,T} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{L|L,T} = -\infty$, Dawkins' Runt region, panel I). In all cases, the frequency of high-quality juveniles is set to $p = 0.5$, and reproductive asymmetry is set to $s = 0.75$.

Chapter 6. Evolution of positive and negative density-dependent dispersal.

6.1. Introduction

Dispersal is a key determinant of fitness and varies markedly both among and within species (Clobert et al. 2001, 2012; Ronce 2007). Three general factors have been suggested to favour the evolution of costly dispersal. First, dispersal may allow individuals to avoid the costs of inbreeding (Bengtsson 1978). Second, it may allow individuals to escape habitats of inferior quality (Gadgil 1971; Van Valen 1971; McPeek & Holt 1992). Third, it may alleviate local competition between non-dispersing kin (Hamilton & May 1977). These two last factors are likely to be important across a wide range of species, since they apply to both sexual and asexual organisms.

Over the last decades much evidence has accumulated that dispersal is a highly plastic trait (Bowler & Benton 2009; Clobert et al. 2009). To give just a few, illustrative examples, in family groups of carrion crows (*Corvus corone corone*) juveniles dispersed less when territories were food-supplemented (Baglione et al. 2006); while in the butterfly *Boloria eunomia*, dispersal rates were raised when patch quality was experimentally decreased but remained lower in untreated patches (Baguette et al. 2011). Similarly, in the common lizard *Lacerta vivipara*, local high density promoted higher levels of dispersal (Léna et al. 1998). There is, however, much variation between species in the way that dispersal responds to factors such as resource availability and population density. In particular, while some studies show positive density-dependent dispersal

(Fonseca & Hart 1996; De Meester & Bonte 2010; Innocent et al. 2010), others show negative density-dependent dispersal (Meylan et al. 2007).

One possible reason why the pattern of density-dependent dispersal might vary from one species to another is that density may be correlated with both local resource availability and local competition, which may exert opposing effects on dispersal. For example, if high density is associated with high local competition, individuals may be selected to disperse more, while if it is more strongly correlated with resource abundance, individuals may be selected to disperse less. While this idea has already received some attention in a verbal argument put forward by Clobert et al. (2004), it is still unclear how all these factors interact to mediate the evolution of dispersal, especially because density may also influence the action of kin selection on dispersal. Although a few models have begun to explore the evolution of dispersal in complex environments that are both spatially (Hastings 1983; Greenwood-Lee & Taylor 2001; Leturque & Rousset 2002; Massol et al. 2010) and, in some cases, also temporally heterogeneous (McPeek & Holt 1992; Gandon & Michalakis 1999; Rousset 2012), there exists no well-developed theoretical framework that can account for the observed diversity in density-dependent dispersal tactics.

Here we use an inclusive fitness approach to model the evolution of both unconditional and conditional (density-dependent) dispersal strategies in response to habitat heterogeneity and local competition. We assume two types of patches - high-quality patches with abundant resources that give rise to a high density of juveniles, and low-quality patches with sparser resources that lead to a lower density of juveniles - and we allow patch quality (and the concomitant density of young) to vary both between patches and over time within a patch. We describe how these different types of environmental heterogeneity influence local kinship, and consider the evolution of both unconditional dispersal rates, and also of conditional dispersal strategies, allowing for dispersal decisions that are density-dependent.

6.2. Model

6.2.1. Life-cycle

We assume an infinite island model (Wright 1931; Hamilton & May 1977; Rodrigues and Gardner 2012) in which each patch contains *n* haploid, asexual breeding individuals. Generations are non-overlapping. We assume that there are two patch types: a fraction *p* of patches are high-quality with abundant resources, while a fraction (1-*p*) are low-quality with more limited resources (note that when patch quality may change from one generation to the next, the equilibrium fraction *p* of high-quality patches depends upon the rates with which patches switch in state, as described below). In every generation, individuals in high-quality patches each produce a large number F_H of young, whereas individuals in low-quality patches each produce a smaller (though still large) number $F_L \equiv (1-\sigma)F_H$ of young, where $0 < \sigma < 1$ specifies the fecundity difference between individuals in high- and low-quality patches. High quality patches are thus characterised by a high density of juveniles prior to dispersal, and low quality patches by a lower density. After reproduction, breeding individuals die. With probability $1-z_H$, a juvenile remains in her high quality (and high density) natal patch, whereas with probability z_H she disperses away from this natal patch, reaching a random patch in the population with probability 1-*c*, where *c* is the cost of dispersal. With probability $1-z_L$, a juvenile remains in her low quality (and low density) natal patch, whereas with probability z_L she disperses away from this natal patch, reaching a random patch in the population with probability 1-c. After dispersal, juveniles on a patch (both native and immigrant) compete for the n available breeding sites. Finally, patches may undergo changes in their quality-state: with probability α a high-quality patch remains high-quality, whereas with probability 1-α it becomes low-quality; with probability β a low-quality patch remains low-quality, whereas with probability 1-β it becomes high-quality (so that the equilibrium fraction of high-quality patches is given by $p = (1- \beta)/(2- \alpha- \beta)$. Model notation is summarised in Table 1.

Symbol	Meaning
H	High-quality patch
J	Juvenile
L	Low-quality patch
U	Unconditional dispersal rate
∅	Reference model
α	Probability that a high-quality patch remains a high-quality patch
β	Probability that a low-quality patch remains a low-quality patch
c	Cost of dispersal
c_X	Class reproductive value of individual in condition X
η_X	Probability that a X-quality patch was a X-quality patch the previous generation
F_X	Fecundity of a breeding female in condition X
h_X	Probability of philopatry in a X-quality patch
ϕ_X	Probability of co-philopatry in a X-quality patch
n	Patch size

p	Frequency of high-quality patches in the population
r_{X}	Relatedness among juveniles in condition X before dispersal
R_{X}	Relatedness among juveniles in condition X after dispersal
σ	Reproductive asymmetry
τ	Temporal correlation between the quality of a patch in successive generations
v_{X}	Expected reproductive value of an juvenile in condition X
υ_{X}	Relative reproductive value of a juvenile who survives dispersal in relation to philopatric juveniles
x_{X}	Dispersal rate of a focal individual in condition X
y_{X}	Average dispersal rate in X-quality patches
z_{X}	Average dispersal rate in the population of X-quality patches
$z_{\o}^*$	Evolutionary stable (ES) dispersal rate in the reference model
z_{X}^*	Evolutionary stable (ES) dispersal rate in X-quality patches

Table 1. A summary of model notation.

We are interested in the evolution of dispersal rates, both when individuals are constrained to adopt an unconditional strategy, such that $z_H = z_L = z_{\mathrm{U}}$, and when they are free to adopt a conditional, density-dependent strategy for which $z_H \neq z_L$. In the appendix, we derive convergently stable unconditional and conditional dispersal rates, using the Taylor-Frank ‘direct fitness’ approach to capture the effects of local kin competition (Taylor & Frank 1996; Frank 1998). Our analysis involves the derivation of patch-state frequencies at demographic equilibrium, followed by expressions for reproductive success, i.e. the expected numbers of surviving offspring on low-quality and high-quality patches produced by parents reproducing on low- and high-quality patches. From these we derive the individual reproductive values of surviving offspring on low-quality and high-quality patches. Together with expressions for the relatedness among individuals on low- and high-quality patches, we can then construct expressions for the selection gradient(s) acting on dispersal, allowing us to identify convergently stable values (at which these gradients are equal to zero; see Appendix for details).

Convergence stability implies that natural selection, assuming vanishingly small variation in the population, drives a population towards the equilibrium value. Evolutionary stability further implies that such an equilibrium, once established, cannot be invaded by a mutant that disperses at a slightly different rate. The analysis of evolutionary stability of social traits in patch-structured populations is not an easy task. In the Supplementary Information, however, we use the approach developed by Metz & Gyllenberg (2001) and Ajar (2003) to confirm that the convergently stable equilibria we have identified are also evolutionarily stable. The method involves computing the total number of emigrants that are descendants of a single successful immigrant (Metz & Gyllenberg 2001; Ajar 2003), where this quantity is denoted by R_m (see Appendix for details).

Below, we explore the impact of the model's parameters on the evolutionarily stable rates of unconditional and conditional dispersal, derived as described above. To provide greater insight into the predictions of the model, however, we summarise our analysis in terms of Hamilton's rule.

6.2.2. Hamilton’s rule

Applying Hamilton’s rule to an individual offspring’s decision to disperse or to remain on its natal patch, we see that conditional dispersal is favoured when

$$-1 + (1-c)\upsilon_{\mathrm{H}} + r_{\mathrm{H}} > 0, \text{ and} \tag{1}$$

$$-1 + (1-c)\upsilon_{\mathrm{L}} + r_{\mathrm{L}} > 0, \tag{2}$$

for individuals on high- and low-quality patches, respectively. The condition for a high-quality patch (inequality 1) features three terms: first, a disperser suffers a cost of one to its fitness because it loses the opportunity to breed in its natal patch; second, a disperser gains a benefit of $(1-c)\upsilon_{\mathrm{H}}$ to its fitness, where υ_{H} is the relative reproductive value of a juvenile (born in a high-quality patch) that competes for breeding sites in a foreign patch compared to one that remains in the home patch; finally, a disperser alleviates local competition between related individuals contesting breeding sites in the focal patch. These local competitors are related to the focal individual by $r_{\mathrm{H}} = h_{\mathrm{H}}R_{\mathrm{H}}$, where h_{H} is the probability of philopatry in a high-quality patch and R_{H} is the relatedness among philopatric juveniles in high-quality patches. The condition for a low-quality patch can be interpreted in a similar way (equation 2; see Appendix for details).

Applying Hamilton’s rule to the evolution of unconditional dispersal, we see that dispersal is favoured when

$$-1 + (1-c)\upsilon_{\mathrm{U}} + c_{\mathrm{H,J}}r_{\mathrm{H}} + c_{\mathrm{L,J}}r_{\mathrm{L}} > 0, \tag{3}$$

where: $c_{\mathrm{H,J}}$ is the total reproductive value of high-quality philopatric juveniles; $c_{\mathrm{L,J}}$ is the total reproductive value of low-quality philopatric juveniles; and v_{U} is the expected relative reproductive value of a juvenile that survives dispersal in relation to the total reproductive value of philopatric juveniles, which is given by $v_{\mathrm{U}} = ((p+(1-p)(1-\sigma))v_{\mathrm{D,J}})/(c_{\mathrm{H,J}}+c_{\mathrm{L,J}})$, with $v_{\mathrm{D,J}} = pv_{\mathrm{H,J}} + (1-p)v_{\mathrm{L,J}}$, where $v_{\mathrm{D,J}}$ is the expected reproductive value of a successful disperser (see Appendix for details).

Candidate equilibrium dispersal rates are those at which the selection gradient on dispersal is zero, i.e for which the left hand sides of inequalities (1-3) are null (implying that an individual obtains equal expected payoffs regardless of whether it chooses to disperse or remain).

6.3. Results and Analysis

When $\sigma = 0$, patches do not vary in resource availability, and the environment is homogeneous (Hamilton & May 1977; Frank 1986; Taylor 1988). Our approach is to use this reference model as a base-line case against which we can compare later results for spatially and temporally heterogeneous populations. If the population is homogeneous, then reproductive values do not differ between patches ($v_{\mathrm{H}} = v_{\mathrm{L}}$, $c_{\mathrm{H}} = c_{\mathrm{L}}$) and neither does local relatedness ($r_{\mathrm{H}} = r_{\mathrm{L}}$). Consequently conditions (1), (2) and (3) for conditional dispersal from high-quality and low-quality patches and for unconditional dispersal all reduce to the same inequality

$$-1+(1-c)v_{\varnothing} + r_{\varnothing} > 0, \quad (4)$$

where $r_{Ø} = h_{Ø}R_{Ø}$ is the relatedness between a disperser and the beneficiaries of reduced competition in this homogeneous case, $h_{Ø} = (1-z)/(1-cz)$ is the probability of philopatry and $R_{Ø} = 1/(n-(n-1)\phi_{Ø})$ is the relatedness among philopatric juveniles on a patch, where the probability of co-philopatry $\phi_{Ø} = h_{Ø}^2$ (Frank 1986; Taylor 1988). Setting the left-hand side of inequality (4) equal to zero, and solving it for the dispersal rate z, we derive the evolutionary stable dispersal rate $z_{Ø}^* = (H+1-2nc)/(H+1-2nc^2)$, where $H = \sqrt{}(1+4n(n-1)c^2)$ (Frank 1986; Taylor 1988; Taylor & Frank 1996; Ajar 2003). This is a decreasing function of the cost of dispersal c and patch size n.

6.3.1. Conditional dispersal

Compare inequalities (1) and (2) for conditional dispersal from high-quality and low-quality patches in a heterogeneous population with inequality (4) for dispersal in a homogeneous population. In all three cases the first term, which represents the cost to a dispersing individual of losing the opportunity to breed in its natal patch, is equal to 1. This is because we have normalized to actor's value. The second term, however, differs between cases. While the probability of surviving dispersal is equal to $1-c$ in both a homogeneous and a heterogeneous population, the relative reproductive value of young who survive dispersal (compared to the value of philopatric young), differs between high- and low-quality patches. Thus, υ_{H} and υ_{L} can be different from $\upsilon_{Ø} = 1$. In addition, the third term, representing the indirect fitness effects of dispersal, may also differ between cases, i.e. r_{H} and r_{L} can be different from $r_{Ø} = h_{Ø}R_{Ø}$. The overall impact of heterogeneity on conditional dispersal thus depends on the balance between direct and indirect fitness effects.

As figure 1 and 2 shows, when the environment is temporally stable, i.e. when the correlation τ between the quality of a patch in successive generations (see Appendix for details) is high, and when the cost of dispersal c is low, selection favours lower rates of dispersal from high-quality patches ($z_H^* < z_L^*$). Conversely, when the environment is unstable, i.e. when τ is low, and when the cost of dispersal is high, selection favours higher rates of dispersal from high-quality patches ($z_H^* > z_L^*$). What drives this reversal?

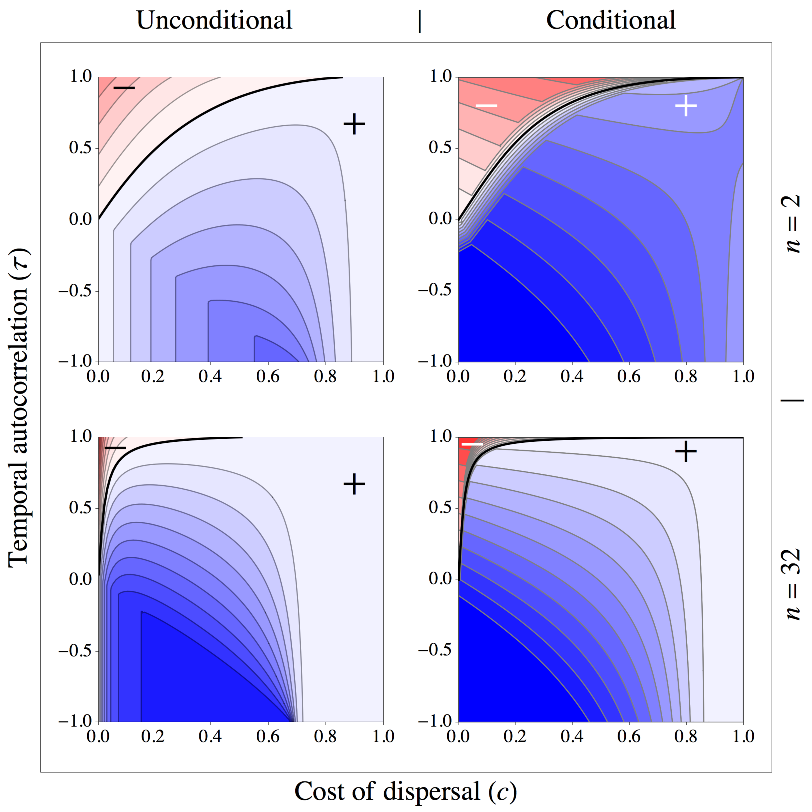

Figure 1 | The difference between the unconditional ES dispersal rate and the ES dispersal rate of the reference model (z_U^* - $z_{\varnothing}^*$), and between the conditional ES dispersal rate from high-quality patches and the conditional ES dispersal rate from low-quality patches (z_H^* - z_L^*), as a function of the cost of dispersal (c) and the temporal autocorrelation (τ) for varying patch size (n). The bold black line is when the unconditional ES dispersal rate is equal to the ES dispersal of the reference model ($z_U^* = z_\varnothing^*$), and when the ES dispersal rate from high-quality patches is equal to the ES dispersal rate form low-quality patches ($z_H^* = z_L^*$). To the left / right of the bold black line we have that: (1) environmental heterogeneity inhibits / promotes dispersal relative to the reference model; (2) the dispersal rate from high-quality patches is less / greater than that from low-quality patches, i.e. negative- / positive-density dependent dispersal. Parameters values: $p = 0.5$.

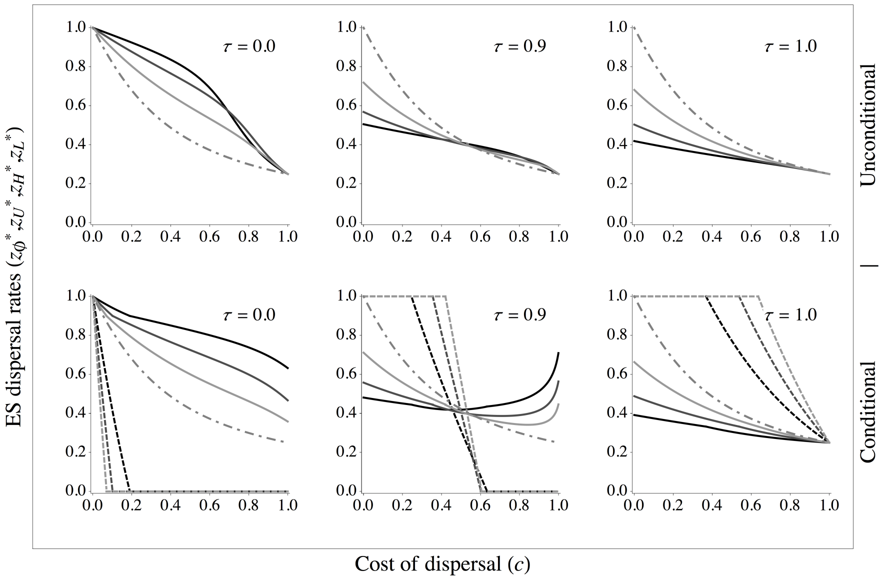

Figure 2 | Unconditional (z_U^*; top row) and conditional (z_H^* - solid lines, z_L^* - dashed lines; bottom row) ES dispersal rates as a function of the cost of dispersal (c) for varying frequencies of high-quality patches ($p = 0.25, 0.50$, and 0.75; from dark to light grey lines), and for varying temporal correlation ($\tau = 0.0, 0.9, 1.0$). The dot-dashed grey line is the ES dispersal rate of the reference model ($z_\varnothing^*$). Parameter values: $n = 2$.

In a temporally stable environment (high τ), offspring born in a high-quality patch are likely to enjoy a high-quality environment as adults if they remain; conversely, offspring born in a low-quality patch are likely to suffer a low-quality environment as adults if they remain. Consequently, the relative reproductive value of offspring that disperse away from a high-quality patch (compared to those that stay) is lower than in a homogeneous population ($\upsilon_H < \upsilon_\varnothing = 1$), while the relative reproductive value of offspring that disperse away from a low-quality patch is greater than in a homogeneous population ($\upsilon_L > \upsilon_\varnothing = 1$). This tends to favour lower rates of dispersal from high-quality patches. The effect is partially offset because the greater fecundity of breeders in high-quality patches leads to more intense competition for local breeding spots, and to greater local relatedness, both of which tend to weaken selection for philopatry. Nevertheless, if τ is large enough, the differences in reproductive value between high- and low-quality patches outweigh the differences in relatedness, so that the outcome is lower rates of dispersal from higher-quality patches ($z_H^* < z_L^*$).

If the environment is temporally unstable (low τ), then the quality of a patch in the current generation is a poor predictor of its quality in the next. Consequently, the advantage of remaining in a high-quality patch (or the cost of remaining in a low-quality patch) is reduced. Instead, the effects of more intense local competition and greater local relatedness described above predominate. The result is that selection favours higher rates of dispersal from higher-quality patches ($z_H^* > z_L^*$).

Intuitively, in a stable environment, a high-quality patch represents an enduring resource that it is worth staying to compete over. By contrast, in an unstable environment, a high-quality patch offers no long-term benefit; rather, it is associated with a short-term increase in local kin competition that it is worth dispersing to escape from.

What about the effect of dispersal costs? As described above, local competition for breeding spots is greater in high-quality patches, where more young are produced, and lower in low-quality patches, where fewer young are produced. This effect tends to favour greater dispersal from high-quality patches (especially when the environment is unstable). As the costs of dispersal decrease, however, greater numbers of surviving immigrants tend to mask local differences in productivity, and equalise the intensity of competition between the two patch types. Consequently, when the cost of dispersal c is low, selection is more likely to favour lower rates of dispersal from high-quality patches.

6.3.2. Unconditional dispersal

Compare inequality (3) for unconditional dispersal with inequality (4) for dispersal in a homogeneous population. Again, in both cases the first term is equal to one. This is because we have normalized to actor's value. The second term, however, differs in relation to the relative reproductive value of a dispersing offspring. Thus, v_{U} can be different from $v_{\varnothing} = 1$. Moreover, the third term, representing the indirect fitness effects of dispersal, may also differ between both cases, i.e. $c_{\mathrm{H,J}}r_{\mathrm{H}}+c_{\mathrm{L,J}}r_{\mathrm{L}}$ can be different from $r_{\varnothing}$. The overall impact of heterogeneity on unconditional dispersal thus depends on the balance between direct and indirect fitness effects.

As figure 1 and 2 shows, when the environment is temporally stable, and when the cost of dispersal is low, selection favours lower rates of unconditional dispersal ($z_{\mathrm{U}}^* < z_{\varnothing}^*$). Conversely, when the environment is unstable, and when the cost of dispersal is high, selection favours higher rates of unconditional dispersal ($z_U^* > z_Ø^*$). What drives this reversal?

We first note that the proportion of dispersers leaving high-quality patches is $p/(p+(1-p)(1-\sigma))$, while the proportion of dispersers arriving to high-quality is p. This means that more individuals leave high-quality patches than arrive to high-quality patches, as $p/(p+(1-p)(1-\sigma)) > p$. In temporally stable environments, the reproductive value of juveniles competing in high-quality patches is higher than the reproductive value of a successful disperse ($v_{H,J} > v_{D,J}$). As a result, more dispersers trade a higher reproductive value for a lower reproductive value (i.e. $v_{H,J}$ for $v_{D,J}$), than trade a lower reproductive for a higher reproductive value (i.e. $v_{L,J}$ for $v_{D,J}$). Therefore, the relative reproductive value of unconditional dispersal is lower than that of a homogeneous population, i.e. $\upsilon_U < \upsilon_Ø = 1$. This tends to favour lower unconditional dispersal rates.

If the environment is unstable (low τ), then the quality of the patches is reversed, and low-quality patches become high-quality patches, whilst high-quality patches become low-quality patches. As a result, while in stable environments more dispersers trade high reproductive value for intermediate reproductive value than the reverse, now more dispersers trade low reproductive value for intermediate reproductive value. As a result, the average reproductive value of a disperser is greater than the average reproductive value of a philopatric juveniles ($\upsilon_U > \upsilon_Ø = 1$). As a result, unstable environments tend to favour higher unconditional dispersal rates ($z_U^* > z_Ø^*$).

What about the effect of dispersal costs? Low cost of dispersal does not favour dispersal when the environment is stable. This is because unconditional dispersal brings about more individuals trading high-quality breeding habitats for low-quality habitats than the reverse. This is beneficial when habitats are likely to switch their quality from one season to the other, however is detrimental when habitats tend to keep their quality from one season to the other.

6.4. Discussion

Our analysis shows that environmental heterogeneity can exert a strong influence on the evolution dispersal strategies, whether these are unconditional or conditional. At the same time, the way in which individuals respond to such heterogeneity depends critically on the temporal stability of the environment. When the environment is highly stable (i.e. when local differences in patch quality persist over multiple generations), selection favours negative density-dependent dispersal (i.e. less dispersal from high quality, high density patches) and, in the unconditional case, rates of dispersal that are lower than in a homogeneous environment. By contrast, when the environment is temporally unstable, unpredictable or seasonal (i.e. when local differences in patch quality may disappear or even be reversed from one generation to the next), selection favours positive density-dependent dispersal (i.e. more dispersal from high quality, high density patches) and, in the unconditional case, rates of dispersal that are higher than in a homogeneous environment.

Temporal stability affects the evolution of dispersal strategies because it influences the relative long-term value of offspring that establish themselves in a patch that is currently high quality vs one that is currently low quality. In a stable environment (where $0 << \tau \leq 1$), high quality patches are likely to remain high quality (and low quality patches to remain low quality) over many generations. Consequently, offspring that establish themselves in a high quality patch are of relatively high value. If one allows for conditional dispersal, this favours lower rates of dispersal from high-quality, high-density patches in the conditional case. In the unconditional case, the fact that the majority of offspring are born on high-quality patches means that selection favours lower dispersal rates overall for the same reason. Suppose, by contrast, that the environment is less temporally stable (so that $\tau << 1$). Then the current quality of a patch is a poor predictor of its future quality, so that the long-term value of offspring is largely unaffected by the quality of the patch in which they establish themselves. At the same time, the high density of juveniles on a high quality patch means that an individual is less likely to claim a breeding spot there. Under these circumstances, selection favours higher rates of dispersal from high-quality, high-density patches in the conditional case, and in the unconditional case, higher rates of dispersal overall.

The other main factor (apart from temporal stability) that determines the response to habitat heterogeneity is the cost of dispersal. Low costs tend to favour more negative (or less positive) density-dependent dispersal, while high costs favour more positive (or less negative) density-dependent dispersal. Since lower costs lead to higher dispersal rates overall, we would therefore expect to see (all other things being equal) more negative density-dependent dispersal in populations with higher dispersal rates, and more positive density-dependent dispersal in populations with lower dispersal rates.

So far, we have focused on whether selection favours positive or negative density-dependence in dispersal. However, our analysis also yields predictions about the strength of density-dependence, i.e. the extent to which dispersal rates should vary with density.

This depends in a more complex way upon habitat heterogeneity, the cost of dispersal and local group size. When group size is large and kin competition is negligible, more stable habitats and higher costs of dispersal favour relatively weaker density-dependence (while less stable habitats and lower costs of dispersal favour stronger density-dependence). By contrast, if group size is small and kin competition effects are relatively important, the picture becomes slightly more complicated. For example, in unstable habitats we may have: strong positive density-dependence when the cost of dispersal is high; no density-dependence when the cost of dispersal is intermediate; and strong negative density-dependence when the cost of dispersal is low. Overall, we expect stronger density-dependence (whether positive or negative) when kin competition effects are present.

How does our model compare to previous analyses of the evolution of conditional (i.e. density dependent) dispersal? Not many studies have addressed this issue explicitly; of those that have, most predict higher rates of dispersal from high-density patches (McPeek & Holt 1992; Ezoe & Iwasa 1997; Travis et al. 1999; Poethke & Hovestadt 2002), while fewer predict the opposite trend (Leturque & Rousset 2003). None, however, account for both patterns as we do, by invoking varying levels of temporal stability in environmental heterogeneity. Indeed, very few models have considered the impact of temporal variation in patch productivity on conditional dispersal (Ezoe & Iwasa 1997). At the same time, of course, other models address many complexities of the dispersal process that we have not considered here. To highlight a few interesting possibilities for future analysis, one might extend the present model to allow for patch selection by dispersing individuals, leading to equilibration of reproductive value between patches, as in the model of Rousset (1999). In addition, one might consider the impact of variation in individual body condition on dispersal strategies, as in the models of Gyllenberg et al. (2011a, 2011b). Lastly, one might also consider the influence of parent-offspring conflict on dispersal, as in the model of Taylor (1988).

Turning to the implications of our model for empirical studies of dispersal, our results suggest that spatial heterogeneity alone is likely to be a poor predictor of conditional dispersal tactics. The cost of dispersal and, to an even greater extent, the temporal stability of the habitat greatly affect the expected unconditional and conditional dispersal rates. In line with this general prediction, empirical studies focusing on spatial heterogeneity have observed contradictory patterns of density-dependent dispersal under natural conditions, with some studies reporting a positive correlation between density and dispersal (e.g. Fonseca & Hart 1996; De Meester & Bonte 2010; Innocent et al. 2010), and others a negative correlation (e.g. Meylan et al. 2007). Experimental manipulations of patch quality have yielded equally confusing results, with some studies reporting an increase in dispersal from patches of reduced quality together with a decrease in dispersal from patches that of elevated quality (e.g. Baguette et al. 2011), while others found that conditional dispersal remained unchanged when patches were elevated in quality (e.g. Matter & Roland 2002). Our results can potentially help to account for this diversity of results, and suggest a need for empirical studies that follow individuals over their entire life–cycle while also tracking environmental changes over several generations, in order to assess temporal stability.

What cues should individuals use to decide whether to disperse or not? Several studies suggest that individuals may respond to: local densities of individuals (Fonseca & Hart 1996; Matthysen 2005; Meylan et al. 2007; Chaput-Bardy et al. 2010; De Meester & Bonte 2010; Innocent et al. 2010); resource availability (Baglione et al. 2006; Baguette et al. 2011); or the presence of kin (Léna et al. 1998). In the context of our model, local density may: (1) correlate with relatedness when the environment is stable; (2) have no correlation with relatedness when the environment is unpredictable; and (3) correlate negatively with relatedness when the environment is seasonal. Moreover, local density may also: (1) correlate with reproductive value in stable environment; (2) have no correlation with adults' reproductive value when the environment is unpredictable; and (3) correlate negatively with reproductive value when the environment is seasonal. Therefore, if individuals use local density alone as a cue to decide whether to disperse or not, they are likely to engage in a sub-optimal dispersal strategy. Our study consequently suggests that individuals may need to combine multiple sources of information, as anticipated by Clobert et al. (2004), if they are to make adaptive dispersal decisions. More generally, it suggests a need to integrate proximate explanations and ultimate explanations of dispersal.

6.5. Supporting Information to "Evolution of positive and negative density-dependent dispersal"

6.5.1. Appendix A – Patch Dynamics

Following the patch dynamics described in the main text (see also Rodrigues & Gardner 2012), we arrange the probabilities that describe how patches change in quality from generation to generation in a matrix, as follows:

$$\mathbf{P} = \begin{pmatrix} \alpha & 1-\beta \\ 1-\alpha & \beta \end{pmatrix}. \qquad \text{(A1)}$$

The right eigenvector of this matrix gives the frequency of high- and low-quality patches at equilibrium. We find that at equilibrium the fraction of high-quality patches is given by

$$p = \frac{1-\beta}{2-\alpha-\beta}, \tag{A2}$$

assuming α, $\beta < 1$. We now want to find the temporal correlation between the quality of patches in successive generations. To this purpose, we define the random variable T_t as the quality of a patch at time t, such that $T_t = 1$ if the patch is high-quality, and $T_t = 0$ if the patch is low-quality. The temporal correlation of patch quality over generations is then given by $\tau = \mathrm{cov}(T_t, T_{t+1})/\sqrt{}(\mathrm{var}(T_t)\mathrm{var}(T_{t+1})) = \alpha - (1-\beta)$.

6.5.2. Appendix B – Reproductive Success

The expected number of surviving offspring in high-quality patches produced by a focal mother in a high-quality patch is given by

$$w_{\mathrm{H}\to\mathrm{H}} = \frac{(1-x_\mathrm{H})\alpha}{1-y_\mathrm{H}+\big(pz_\mathrm{H}+(1-p)z_\mathrm{L}(1-\sigma)\big)(1-c)} + \frac{x_\mathrm{H}(1-c)p\alpha}{1-z_\mathrm{H}+\big(pz_\mathrm{H}+(1-p)z_\mathrm{L}(1-\sigma)\big)(1-c)} + \frac{x_\mathrm{H}(1-c)(1-p)(1-\beta)}{(1-z_\mathrm{L})(1-\sigma)+\big(pz_\mathrm{H}+(1-p)z_\mathrm{L}(1-\sigma)\big)(1-c)}, \tag{A3}$$

where y_H is the average dispersal rate out of the focal patch, z_H is the population average dispersal rate out of high-quality patches, and z_L is the population average dispersal rate out of low-quality patches. The expected number of surviving offspring in low-quality patches produced by a focal mother in a high-quality patch is given by

$$w_{\mathrm{H}\to\mathrm{L}} = \frac{(1-x_{\mathrm{H}})(1-\alpha)}{1-y_{\mathrm{H}}+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)} + \frac{x_{\mathrm{H}}(1-c)p(1-\alpha)}{1-z_{\mathrm{H}}+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)} + \frac{x_{\mathrm{H}}(1-c)(1-p)\beta}{(1-z_{\mathrm{L}})(1-\sigma)+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)}, \tag{A4}$$

The expected number of surviving offspring in low-quality patches produced by a focal mother in a low-quality patch is given by

$$w_{\mathrm{L}\to\mathrm{L}} = \frac{(1-x_{\mathrm{L}})(1-\sigma)\beta}{(1-y_{\mathrm{L}})(1-\sigma)+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)} + \frac{x_{\mathrm{L}}(1-\sigma)(1-c)p(1-\alpha)}{1-z_{\mathrm{H}}+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)} + \frac{x_{\mathrm{L}}(1-\sigma)(1-c)(1-p)\beta}{(1-z_{\mathrm{L}})(1-\sigma)+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)}, \tag{A5}$$

where y_{L} is the average dispersal rate in the focal patch. The expected number of surviving offspring in high-quality patches produced by a focal mother in a low-quality patch is given by is given by

$$w_{\mathrm{L}\to\mathrm{H}} = \frac{(1-x_{\mathrm{L}})(1-\sigma)(1-\beta)}{(1-y_{\mathrm{L}})(1-\sigma)+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)} + \frac{x_{\mathrm{L}}(1-\sigma)(1-c)p\alpha}{1-z_{\mathrm{H}}+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)} + \frac{x_{\mathrm{L}}(1-\sigma)(1-c)(1-p)(1-\beta)}{(1-z_{\mathrm{L}})(1-\sigma)+\big(pz_{\mathrm{H}}+(1-p)z_{\mathrm{L}}(1-\sigma)\big)(1-c)}, \tag{A6}$$

The fitness of a high-quality individual is given by $W_{\mathrm{H}} = w_{\mathrm{H}\to\mathrm{H}}+(v_{\mathrm{L,A}}/v_{\mathrm{H,A}})w_{\mathrm{H}\to\mathrm{L}}$, whilst the fitness of a low-quality individual is given by $W_{\mathrm{L}} = w_{\mathrm{L}\to\mathrm{L}}+(v_{\mathrm{H,A}}/v_{\mathrm{L,A}})w_{\mathrm{L}\to\mathrm{H}}$, where $v_{\mathrm{H,A}}$ and $v_{\mathrm{L,A}}$ are the reproductive values of high- and low-quality adults, respectively. The fitness of a random individual in the population is given by $W = c_{\mathrm{H,A}}W_{\mathrm{H}}+\mathrm{c}_{\mathrm{L,A}}W_{\mathrm{L}}$, where $c_{\mathrm{H,A}}$ is the class-reproductive value of high-quality adults, whereas $c_{\mathrm{L,A}}$ is the class-reproductive value of low-quality adults.

6.5.3. Appendix C – Reproductive Value

If we arrange the class-specific reproductive success values derived in Appendix B in a transition matrix as follows:

$$\mathbf{A} = \begin{pmatrix} w_{\mathrm{H}\to\mathrm{H}} & w_{\mathrm{L}\to\mathrm{H}} \\ w_{\mathrm{H}\to\mathrm{L}} & w_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}. \tag{A7}$$

then the individual reproductive values of high- and low-quality adults ($v_{\mathrm{H,A}}$ and $v_{\mathrm{L,J}}$, respectively) are given by the left-eigenvector of matrix **A** (assuming a neutral populations, i.e. $x_{\mathrm{H}} = y_{\mathrm{H}} = z_{\mathrm{H}}$ and $x_{\mathrm{L}} = y_{\mathrm{L}} = z_{\mathrm{L}}$; Fisher 1930; Taylor 1990; Taylor & Frank 1996). The reproductive value of a juvenile competing in a high-quality patch is given by $v_{\mathrm{H,J}} = \omega_{\mathrm{H}}\, v_{\mathrm{H}}$, whilst the reproductive value of a juvenile competing in a low-quality patch is given by $\mathrm{v}_{\mathrm{L,J}} = \omega_{\mathrm{H}}\, v_{\mathrm{L}}$, where: $\omega_{\mathrm{H}} = 1/(1\text{-}z_{\mathrm{H}}+(pz_{\mathrm{H}}+(1\text{-}p)(1\text{-}\sigma)z_{\mathrm{L}})(1\text{-}c))$ is proportional to the likelihood that a juvenile wins a breeding spot in a high-quality patch; $\omega_{\mathrm{L}} = 1/((1\text{-}z_{\mathrm{L}})(1\text{-}\sigma)+(pz_{\mathrm{H}}+(1\text{-}p)(1\text{-}\sigma)z_{\mathrm{L}})(1\text{-}c))$ is proportional to the likelihood that a juvenile wins a breeding spot in a low-quality patch; $v_{\mathrm{H}} = \alpha\, v_{\mathrm{H,A}}+(1\text{-}\alpha)\, v_{\mathrm{H,A}}$ is the expected reproductive value of a juvenile who has won a breeding spots in a high-quality patch; and $v_{\mathrm{L}} = \beta\, v_{\mathrm{L,A}}+(1\text{-}\beta)v_{\mathrm{L,A}}$ is the expected reproductive value of a juvenile who has won a breeding spot in a low-quality patch. The expected reproductive value of a juvenile that survives dispersal is given by $v_{\mathrm{D,J}} = pv_{\mathrm{H,J}}+(1\text{-}p)v_{\mathrm{L,J}}$.

6.5.4. Appendix D – Selection Gradients

To determine the selection gradient acting on dispersal, we use the Taylor-Frank approach (Taylor & Frank 1996; Frank 1997, 1998; Taylor et al. 2007). We assume vanishingly small genetic variation for the dispersal trait, where its genic value is denoted by g (Falconer 1981). The direction of selection acting on dispersal is then given by

$$\frac{\mathrm{d}W}{\mathrm{d}g} = c_{\mathrm{H,A}}\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} + c_{\mathrm{L,A}}\frac{\mathrm{d}W_{\mathrm{L}}}{\mathrm{d}g}. \tag{A8}$$

The first derivative on the RHS of this equation can be expanded as follows:

$$\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} = \frac{\partial W_{\mathrm{H}}}{\partial x_{\mathrm{H}}}\frac{\mathrm{d}x_{\mathrm{H}}}{\mathrm{d}g_{\mathrm{H}}}\frac{\mathrm{d}g_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial y_{\mathrm{H}}}\frac{\mathrm{d}y_{\mathrm{H}}}{\mathrm{d}G_{\mathrm{H}}}\frac{\mathrm{d}G_{\mathrm{H}}}{\mathrm{d}g}, \tag{A9}$$

where G_{H} denotes social partners' average genetic value, including self, and all partial derivatives are evaluated at $x = y = z$ (Taylor & Frank 1996; Frank 1997, 1998; Taylor et al. 2007). The partial derivatives give the marginal fitness effect of the mutant's phenotype. The correlations between the phenotype and breeding value represent the genotype-to-phenotype mapping defined as $\mathrm{d}x_{\mathrm{H}}/\mathrm{d}g_{\mathrm{H}} = \mathrm{d}y_{\mathrm{H}}/\mathrm{d}G_{\mathrm{H}} = \gamma_{\mathrm{H}}$, where γ_{H} is arbitrarily set to one. The correlations between breeding values are the coefficients of consanguinity that normalized to the actor's coefficient of consanguinity give the kin selection coefficients of relatedness (Bulmer 1994). The direction of selection operating on low-quality patches ($\mathrm{d}W_{\mathrm{L}}/\mathrm{d}g$) can be expanded in a similar way. Higher values of unconditional dispersal evolve if $\mathrm{d}W/\mathrm{d}g > 0$. Higher values of conditional dispersal from high-/low-quality patches evolve if $\mathrm{d}W_{\mathrm{H}}/d\mathrm{g} > 0$/ $\mathrm{d}W_{\mathrm{L}}/d\mathrm{g} > 0$.

6.5.5. Appendix E – Relatedness

Here we determine the relatedness coefficients among adults in high-quality patches ($r_{\mathrm{H,A}}$), and among adults in low-quality patches ($r_{\mathrm{L,A}}$); this is the probability that two different individuals sampled at random in the same patch share a gene in common (Taylor 1992; Rousset 2004; Rodrigues and Gardner 2012). To this purpose, we need to take into account the probability that a patch was of a particular quality in the previous generation given the quality of the patch in the current generation. We denote these probabilities as follows: the probability that a high-quality patch was a high-quality patch in the previous generation is given by $\mathrm{P}(X_{t\text{-}1}{=}\mathrm{H}|X_t{=}\mathrm{H}) = \eta_{\mathrm{H}}$; the probability that a high-quality patch was a low-quality patch in the previous generation is given by $\mathrm{P}(X_{t\text{-}1}{=}\mathrm{L}|X_t{=}\mathrm{H}) = 1\text{-}\eta_{\mathrm{H}}$; the probability that a low-quality patch was a low-quality patch in the previous generation is given by $\mathrm{P}(X_{t\text{-}1}{=}\mathrm{L}|X_t{=}\mathrm{L}) = \eta_{\mathrm{L}}$; finally the probability that a low-quality patch was a high-quality patch in the previous generation is given by $\mathrm{P}(X_{t\text{-}1}{=}\mathrm{H}|X_t{=}\mathrm{L}) = 1\text{-}\eta_{\mathrm{L}}$. The recursion equations are given by

$$\begin{cases} r'_{\mathrm{H,J}} = \eta_{\mathrm{H}}\phi_{\mathrm{H}}\left(\frac{1}{n} + \left(1 - \frac{1}{n}\right) r_{\mathrm{H,J}}\right) + (1 - \eta_{\mathrm{H}})\phi_{\mathrm{L}}\left(\frac{1}{n} + \left(1 - \frac{1}{n}\right) r_{\mathrm{L,J}}\right) \\ r'_{\mathrm{L,J}} = (1 - \eta_{\mathrm{L}})\phi_{\mathrm{H}}\left(\frac{1}{n} + \left(1 - \frac{1}{n}\right) r_{\mathrm{H,J}}\right) + \eta_{\mathrm{L}}\phi_{\mathrm{L}}\left(\frac{1}{n} + \left(1 - \frac{1}{n}\right) r_{\mathrm{L,J}}\right) \end{cases}, \tag{A10}$$

where: $\phi_{\mathrm{H}} = h_{\mathrm{H}}^2$ and $\phi_{\mathrm{L}} = h_{\mathrm{L}}^2$ are the probabilities of co-philopatry for high- and low-quality patches, respectively; and where h_{H} and h_{L} are the fraction of philopatric individuals competing for breeding spots in high- and low-quality patches, respectively. To determine the coefficients of relatedness at equilibrium we solve the system of equations (A11) setting $r_{\mathrm{H,J}}' = r_{\mathrm{H,J}}$ and $r_{\mathrm{L,J}}' = r_{\mathrm{L,J}}$. Note that theses are the coefficients of relatedness among adults. Relatedness coefficients among philopatric juveniles are given by $r_{\mathrm{H}} = 1/n+(1-1/n)r_{\mathrm{H\,J}}$ for high-quality patches, and $r_{\mathrm{L}} = 1/n+(1-1/n)r_{\mathrm{L\,J}}$ for low-quality patches. This is because with probability $1/n$ two randomly sampled juveniles are born of the same mother, while with probability $1-1/n$ two randomly sampled juveniles are born of different mothers, in which case they are related by $r_{\mathrm{H\,J}}$ in high-quality patches or $r_{\mathrm{L\,J}}$ in low-quality patches.

6.5.6. Appendix F – Stability Analysis

Convergence Stability of Candidate Evolutionary Stable Dispersal Rates

In this section we check the convergence stability of the candidate evolutionary stable (cES) dispersal rates. As we have seen in the main text, cES dispersal rates are those that lead to a null selection gradient, where these are defined by the left-hand side of inequalities (1), (2), and (3). If these cES dispersal rates are attainable by the process of natural selection, assuming gradual changes in the wild-type phenotype, they are called "convergence stable" (Christiansen 1991; Eshel 1996; Rousset 2004; Otto & Day 2005; Leimar 2009). Let us define the selection gradients S_{H}, S_{L} and S as the left-hand side of inequalities (1), (2) and (3), respectively. To determine convergence stability, we need to define a transition matrix around the cES dispersal rates. For the unconditional case, this is given by

$$J_{\mathrm{U}} = \left.\frac{\partial S}{\partial z_{\mathrm{U}}}\right|_{z_{\mathrm{U}} = z_{\mathrm{U}}^{*}}. \tag{A11}$$

An unconditional cES dispersal rate is convergence stable if $J_{\mathrm{U}} < 0$. For the conditional case, we have two evolving traits in the population. As a result, we now have a 2×2 transition matrix, which is given by

$$\mathbf{J}_{\mathrm{C}} = \begin{pmatrix} \frac{\partial s_{\mathrm{H}}}{\partial z_{\mathrm{H}}} & \frac{\partial s_{\mathrm{H}}}{\partial z_{\mathrm{L}}} \\ \frac{\partial s_{\mathrm{L}}}{\partial z_{\mathrm{H}}} & \frac{\partial s_{\mathrm{L}}}{\partial z_{\mathrm{L}}} \end{pmatrix}_{z_{\mathrm{H}} = z_{\mathrm{H}}^{*}, z_{\mathrm{L}} = z_{\mathrm{L}}^{*}} . \tag{A12}$$

A cES dispersal rate is convergence stable is both eigenvalues, λ_{H} and λ_{L}, associated with the transition matrix $\mathbf{J}_{\mathrm{C}}$ are negative, that is if $\lambda_{\mathrm{H}} < 0$ and $\lambda_{\mathrm{L}} < 0$. We use analytical and numerical methods to determine the convergence stability of the cES dispersal rates. Our analysis suggests that all cES dispersal rates are convergence stable.

R_{m} and Evolutionary Stable Dispersal Rates

In the main text we used the Taylor-Frank approach (Taylor & Frank 1996; Frank 1998) to determine the (cES) dispersal rates for both the unconditional and conditional case. However, whether the cES dispersal rates are stable or subject to disruptive selection has not been investigated. In this section we develop a method to determine the evolutionary stability (ES; Maynard Smith 1982) of the analytically derived cES dispersal rates. To this purpose, we use the concept of R_{m}, which is the total number of successful emigrants produced by a single immigrant (Metz & Gyllenberg 2001), and which has been used to determine the evolutionary stability of social traits in subdivided homogeneous populations (Ajar 2003). Here, we expand this method to analyse the stability of social traits in subdivided heterogeneous populations in the context of our model. The objective is to keep track of the number of mutants in a patch from one generation to another, and at the same time to keep track of the quality of the patch. This involves a number of intermediate steps. Let us start by determining the probability that a randomly chosen juvenile is a descendant of one of the *j* philopatric mutant mothers in the previous generation. This is given by

$$\pi_{\mathrm{H},j} = \frac{j(1-x_{\mathrm{H}})}{j(1-x_{\mathrm{H}})+(n-j)(1-z_{\mathrm{H}})+n(1-c)\left(z_{\mathrm{H}}p+z_{\mathrm{L}}(1-p)(1-\sigma)\right)}, \text{ and} \tag{A13}$$

$$\pi_{\mathrm{L},j} = \frac{j(1-x_{\mathrm{L}})(1-\sigma)}{j(1-x_{\mathrm{L}})(1-\sigma)+(n-j)(1-z_{\mathrm{L}})(1-\sigma)+n(1-c)\left(z_{\mathrm{H}}p+z_{\mathrm{L}}(1-p)(1-\sigma)\right)}, \tag{A14}$$

for a focal high- and low-quality patch, respectively. Hence, the probability that a patch of *j* mutant mothers gives origin to a patch of *k* mutant mothers in the next generation is

$$a_{\mathrm{H}}(k|j) = C_k^n {\pi_{\mathrm{H},j}}^k \left(1-\pi_{\mathrm{H},j}\right)^{n-k}, \text{ and} \tag{A15}$$

$$a_{\mathrm{L}}(k|j) = C_k^n {\pi_{\mathrm{L},j}}^k \left(1-\pi_{\mathrm{L},j}\right)^{n-k}, \tag{A16}$$

for a focal high- and low-quality patch, respectively. In addition to transitions in the number of mutants, patches may also undergo transitions in their quality. Taking into account these transitions in patch quality, and combining them with the transition probabilities defined in equations (A15) and (A16), we define a transition matrix **M** as follows:

$$\mathbf{M} = \begin{pmatrix} \alpha\left(a_{\mathrm{H}}(k|j)\right)_{n\times n} & (1-\alpha)\left(a_{\mathrm{H}}(k|j)\right)_{n\times n} \\ (1-\beta)\left(a_{\mathrm{L}}(k|j)\right)_{n\times n} & \beta\left(a_{\mathrm{L}}(k|j)\right)_{n\times n} \end{pmatrix}. \tag{A17}$$

This matrix fully describes a patch both in terms of the number of mutants and quality. Thus, we can determine the number of offspring that disperse from each patch. Each generation, mutant mothers produce a number of dispersing offspring that is proportional to x_{H} and (1- σ)x_{L} for mothers in high- and low-quality patches, respectively. These quantities can be arranged in a vector:

$$\mathbf{B} = ((jx_{\mathrm{H}})_n \quad (j(1-\sigma)x_{\mathrm{L}})_n)^{\mathrm{T}}. \tag{A18}$$

The number of successful emigrants produced by *j* mutant mothers in a high-quality patch is denoted by $e_{\mathrm{H,j}}$, whilst the number of successful emigrants produced by *j* mutant mothers in a low-quality patch is denoted by $e_{\mathrm{L,j}}$. Thus, the vector of the number of successful emigrants is then given by:

$$\mathbf{e} = \left(\left(e_{\mathrm{H},j}\right)_n \quad \left(e_{\mathrm{L},j}\right)_n\right)^{\mathrm{T}}. \tag{A19}$$

To determine the number of successful emigrants for each case, we define the following system of equations:

$$\mathbf{e} = \mathbf{B} + \mathbf{Me}, \tag{A20}$$

which can be solved recursively. Having solved this system, we can use the number of successful emigrants produced by a mutant immigrant established in a high-quality patch $e_{\mathrm{H,1}}$, and the number of successful emigrants produced by a mutant immigrant established in a low-quality patch $e_{\mathrm{L,1}}$, to determine the R_{m} for our model. This is given by

$$R_{\mathrm{m}} = \frac{p(1-c)}{1-z_{\mathrm{H}}+(1-c)\left(z_{\mathrm{H}}p+z_{\mathrm{L}}(1-p)(1-\sigma)\right)}\left(\alpha e_{\mathrm{H},1} + (1-\alpha)e_{\mathrm{L},1}\right) + \frac{(1-p)(1-c)}{(1-z_{\mathrm{L}})(1-\sigma)+(1-c)\left(z_{\mathrm{H}}p+z_{\mathrm{L}}(1-p)(1-\sigma)\right)}\left((1-\beta)e_{\mathrm{H},1} + \beta e_{\mathrm{L},1}\right). \tag{A21}$$

We used R_{m} to compute pairwise invasibility plots and numerically check the ES of the cES dispersal rates. Note that we can determine the selection gradient using the growth rate R_{m}. The selection gradient is the first derivative of R_{m} with respect to x around the wild-type strategy z, that is $\partial R_{\mathrm{m}}/\partial x|x=z$. Moreover, we can now find the cES, which is the dispersal rate for which the selection gradient is null. A cES dispersal strategy is evolutionary stable if the following condition is satisfied:

$$\left.\frac{\partial^2 R_{\mathrm{m}}}{\partial x_{\mathrm{U}}{}^2}\right|_{x_{\mathrm{U}} = z_{\mathrm{U}}{}^*, z_{\mathrm{U}} = z_{\mathrm{U}}{}^*} \leq 0. \tag{A22}$$

For conditional cES dispersal rates, we first need to define the transition matrix around the cES dispersal rates. This is given by

$$\left.\begin{pmatrix} \frac{\partial}{\partial x_{\mathrm{H}}}\left(\frac{\partial R_{\mathrm{m}}}{\partial x_{\mathrm{H}}}\right) & \frac{\partial}{\partial x_{\mathrm{L}}}\left(\frac{\partial R_{\mathrm{m}}}{\partial x_{\mathrm{H}}}\right) \\ \frac{\partial}{\partial x_{\mathrm{H}}}\left(\frac{\partial R_{\mathrm{m}}}{\partial x_{\mathrm{L}}}\right) & \frac{\partial}{\partial x_{\mathrm{L}}}\left(\frac{\partial R_{\mathrm{m}}}{\partial x_{\mathrm{L}}}\right) \end{pmatrix}\right|_{x_{\mathrm{H}} = z_{\mathrm{H}} = z_{\mathrm{H}}^*, x_{\mathrm{L}} = z_{\mathrm{L}} = z_{\mathrm{L}}^*}. \tag{A23}$$

The multiple cES dispersal rates are evolutionary stable if both eigenvalues of this matrix are negative, this is if

$$\lambda_{\mathrm{H}} < 0, \text{ and } \lambda_{\mathrm{L}} < 0. \tag{A24}$$

Stability Analysis: Illustrative Example

When patch size is equal to one, we find that the number of emigrants produced by an individual in a high-quality and in a low-quality patch is

$$e_{\mathrm{H},1} = \frac{x_{\mathrm{H}}(1-a_{\mathrm{L}}(1|1)\beta)+x_{\mathrm{L}}(1-\sigma)a_{\mathrm{H}}(1|1)(1-\alpha)}{1-(a_{\mathrm{H}}(1|1)\alpha+a_{\mathrm{L}}(1|1)\beta)-a_{\mathrm{H}}(1|1)a_{\mathrm{L}}(1|1)(1-\alpha-\beta)}, \text{ and} \tag{A25}$$

$$e_{\mathrm{L},1} = \frac{x_{\mathrm{H}}a_{\mathrm{L}}(1|1)(1-\beta)+x_{\mathrm{L}}(1-\sigma)(1-a_{\mathrm{H}}(1|1)\alpha)}{1-(a_{\mathrm{H}}(1|1)\alpha+a_{\mathrm{L}}(1|1)\beta)-a_{\mathrm{H}}(1|1)a_{\mathrm{L}}(1|1)(1-\alpha-\beta)}, \tag{A26}$$

respectively. The growth rate $R_{\mathrm{m}}(x_{\mathrm{H}},x_{\mathrm{L}},z_{\mathrm{H}},z_{\mathrm{L}})$ is defined by substituting equations (A25) and (A26) into equation (A21). To determine the evolutionary stability of the cES dispersal rates, we substitute $R_{\mathrm{m}}(x_{\mathrm{H}},x_{\mathrm{L}},z_{\mathrm{H}},z_{\mathrm{L}})$ into matrix (A23), and check condition (A24) for evolutionary stability. We find that condition (A24) always holds and therefore the cES dispersal rates are evolutionary stable. To analyse the stability of unconditional dispersal rates we set $x_{\mathrm{H}} = x_{\mathrm{L}} = x_{\mathrm{U}}$ and $z_{\mathrm{H}} = z_{\mathrm{L}} = z_{\mathrm{U}}$. Now we substitute the growth rate $R_{\mathrm{m}}(x_{\mathrm{H}},x_{\mathrm{L}},z_{\mathrm{H}},z_{\mathrm{L}})$ into condition (A22) to check evolutionary stability. We find that condition (A22) always holds and therefore the cES dispersal rates are evolutionary stable. As patch size increases these derivations become more cumbersome. However, using a combination of numerical and graphical methods, such as Pairwise Invasibility Plots, it is still possible to check the evolutionary stability of cES dispersal rates.

Chapter 7. Empty patches, budding, and the evolution of dispersal and cooperation.

7.1. Introduction

The evolution of cooperation is a major challenge to evolutionary biologists (Trivers 1985; Maynard Smith and Szathmáry 1995; Hamilton 1996; West et al. 2007a). If natural selection favours those individuals that survive and reproduce better, why should an individual forfeit some of these fitness traits to help other individuals, who are likely to be its competitors? This apparent dilemma is resolved if cooperation occurs among close relatives who, by definition, share a certain proportion of genes in common (Hamilton 1963, 1964, 1970). Hamilton's rule, $-c + rb > 0$, provides the condition for the evolution of cooperation. It says that cooperation evolves if the benefit b provided to the recipient times the relatedness r between actor and recipient is greater than the cost c to the actor. High relatedness is, therefore, key for the evolution of cooperation. Three mechanisms have been suggested to promote high relatedness among interacting individuals: first, kin recognition mechanisms allow individuals to identify genealogically close relatives (Hamilton 1964); second, green beard mechanisms allow individuals to identify close relatives at the cooperative locus (Hamilton 1964; Dawkins 1976); and finally, limited dispersal, whereby individuals tend to remain close to their place of origin, can lead to social groups of close relatives, and may even allow for indiscriminate cooperation (Hamilton 1964, 1971).

Limited dispersal due to its simplicity is likely to be an important mechanism involved in the evolution of cooperation in a wide range of species (Hamilton 1964). However, while limited dispersal may increase the kin selected benefits, it may also increase competition among kin (Hamilton 1971; Alexander 1974). In fact, simple models of limited dispersal show that these two opposing selective forces exactly cancel each other, so that dispersal does not mediate the evolution of cooperation (Taylor 1992a,b). A large body of theoretical work has focused on factors that may decouple these two opposing selective forces, including: population elasticity (Taylor 1992b; Alizon and Taylor 2008), overlapping generations (Taylor and Irwin 2000), budding or group dispersal (Gardner and West 2006; Lehmann et al. 2006b; Kümmerli et al. 2009), trans-generational altruism (Lehmann 2007, 2010), empty sites (Alizon and Taylor 2008), sex-biased dispersal (Johnstone and Cant 2008; Gardner 2010), age-structure (Johnstone and Cant 2010), and behaviour conditional on habitat quality (Rodrigues and Gardner 2012, 2013a), or on group members' quality (Rodrigues and Gardner 2013b). While in most of these models the dispersal rate has been treated as a non-evolving parameter (but see Alizon and Taylor 2008), it is well known that local competition among kin, owing to population viscosity, may mediate the evolution of dispersal (Hamilton and May 1977; reviewed by Ronce 2007). As a result, the evolution of cooperation is likely to depend on how the specific demographic and genetic assumptions of these models mediate the evolution of dispersal.

Here we are concerned with the evolution of dispersal and cooperation and how these traits are mediated by budding dispersal and competition in a viscous population with empty patches. Although both theoretical and empirical studies have identified budding and empty patches as important factors influencing the evolution of dispersal and / or cooperation, they have usually been considered separately. Theoretically, budding has been suggested to mediate the evolution of cooperation (Gardner and West 2006; Lehmann et al. 2006b) as well as dispersal (Gandon and Michalakis 1999). Similarly, empty patches have been associated with the evolution of cooperation (Lehmann et al. 2006b) as well as dispersal (Gandon and Michalakis 1999; see also Gadgil 1971; Van Valen 1971; McPeek and Holt 1992). Empirically, budding has been suggested to occur in a number of organisms, including: unicellular microorganisms (Pfeiffer and Bonhoeffer 2003; Schtickzelle et al. 2009; Fisher et al. 2013), invertebrates (Peeters and Ito 2001; Seppä et al. 2008), birds (Hannon et al. 1985; Sharp et al. 2008) and mammals (McComb et al. 1994; Griffin et al. 2003; Bradley et al. 2007; Nichols et al. 2012); and empty patches have been associated with the evolution of cooperation and / or dispersal in bacteria (Taylor et al. *submitted*), birds (Hannon et al. 1985; Pruett-Jones and Lewis 1990; Komdeur 1992; Komdeur et al. 1995), and other organisms (e.g. *C. elegans*, Friedenberg 2003)

In spite of this wealth of studies, a clear theoretical understanding of how budding and empty patches mediate the evolution of dispersal and cooperation is still lacking. For example, Gardner and West (2006) considered a population characterized by budding where cooperation could either be favoured, when dispersal is high, or disfavoured, when dispersal is low. In a related study, Gandon and Michalakis (1999) showed that budding is likely to have a negative impact on dispersal, even in the presence of empty patches. On the other hand, empty patches seem to promote dispersal in the superb fairy-wren, a bird that exhibits budding dispersal (Pruett-Jones and Lewis 1990), and budding seems to promote cooperation in long-tailed tits (Sharp et al. 2008).

In this paper, we develop a kin selection model to study how budding and empty patches mediate the evolution of dispersal and cooperation. We consider a viscous population characterised by budding dispersal and competition, and by different environmental conditions depending on the degree of habitat saturation and local habitat quality. We describe how these different factors influence different variables such as relatedness, and local competition. We first study the evolution of cooperation assuming that dispersal is a fixed parameter, we then study the evolution of dispersal, and finally we study the joint evolution of dispersal and cooperation.

7.2. Model and Analysis

7.2.1. Life-cycle

We assume a population of asexual haploid individuals subdivided into a large number of patches, each with n breeding females (Wright 1931; Hamilton and May 1977). We assume that a fraction p of these patches are occupied (and therefore fertile), while a fraction 1-p are empty (and therefore unfertile). Before reproduction, females engage in social interactions. In occupied patches, each of the n breeding females has a large number $F(x_C,y_C)$ of offspring, where x_C is the focal mother's investment in cooperation, whilst y_C is the average investment of the focal mother's social partners, including herself, in cooperation. After social interactions and reproduction mothers die, offspring may disperse. A focal offspring disperses to a random patch in the population with probability x_D, while the average offspring dispersal rate in the focal patch is y_D. Average offspring dispersal rate in the population is z_D. Offspring are assumed to disperse in buds/coalitions of n offspring, and each bud survives dispersal with probability 1-c, where c is the cost of dispersal. After reaching a distant patch buds engage in bud competition for the n available breeding sites. Only one bud wins, the remainder die. After bud competition, adult females stay in the patch with probability 1-m, and migrate to another patch with probability m (as in Gardner and West 2006). Following migration, patches may undergo changes in their fertility status. A fertile patch remains fertile with probability α, and becomes unfertile with probability 1-α. An unfertile patch remains unfertile with probability β, and recovers fertility with probability 1-β. After this, the life-cycle begins again (see Table 1 for a summary of model notation).

Table 1 | A summary of the model notation.

Symbol	Meaning
C	Referring to the cooperation trait
D	Referring to the dispersal trait
E	Empty patch
O	Occupied patch
∅	Reference model for dispersal
T	Reference model for cooperation (Taylor 1992a)
A_T	Potential for cooperation in the reference model (1/n)
A	Potential for cooperation (R-$v\varphi R$)/(1-$v\varphi R$)
A^*	Potential for cooperation at equilibrium
a	Quality of a local occupied patch
d	Dispersal rate as a model parameter ($d = z_D$)
α	Probability that a fertile patch remains fertile
β	Probability that an unfertile patch remains unfertile
F	Fecundity of a breeding female
h_O	Probability of philopatry in an occupied patch $(1-z_D)/(1-z_D+pz_D(1-c))$

$h_{\varnothing}$	Probability of philopatry in the reference model $(1-z_D)/(1-z_D+z_D(1-c))$
φ	Scale of competition $(1-z_D)^2/(1-z_D+pz_D(1-c))^2$
φ_m	Probability of co-philopatry in relation to migration $(1-m_I)^2/(1-m_I+pm_I(1-c))^2$
φ_T	Scale of competition in the reference model for cooperation $(1-z_D)^2$
c	Cost of dispersal
m_I	Intrinsic migration rate
m	Migration rate ($m = pm_I(1-c)/(1-m_I+pm_I(1-c))$)
n	Patch size
p	Frequency of occupied patches in the population $(1-\beta)/(2-(\alpha+\beta))$
R	Relatedness between individuals
ω_E	Likelihood of winning in empty patches $1/(pz_D(1-c))$
ω_O	Likelihood of winning in occupied patches $(1-z_D)/(1-z_D+pz_D(1-c))$
v_E	Reproductive value of an individual in an empty patch $\omega_E(1-a)$
v_O	Reproductive value of an individual in an occupied patch $\omega_O a$
v_D	Benefit of dispersal $(1-c)(pv_O+(1-p)v_E)v_O$
υ	Reproductive value of non-dispersing offspring ($pa/(pa+(1-p)(1-a))$)
x_X	Trait level of a focal individual for trait X
y_X	Average trait level of local individuals for trait X
z_X	Average trait level of the population for trait X
$z_{\varnothing}$*	Evolutionary stable (ES) dispersal rate in the reference model
z_D*	Evolutionary stable (ES) dispersal rate

7.2.2. Hamilton's Rule: Cooperation

We first determine how natural selection acts on cooperation. For this purpose, we consider that there is no variation in the dispersal trait, so that, $x_D = y_D = z_D$. We assume that the focal mother's cooperative behaviour has a cost C to her fecundity, so that, $-\partial F(x_C, y_C)/\partial x_C = C$, and provides a benefit B to her social partners, including herself, so that, $\partial F(x_C, y_C)/\partial y_C = B$. We take the Taylor-Frank approach to kin selection (Taylor and Frank 1996; Frank 1998) to determine the direction of selection acting on the cooperative behaviour (see Appendix for details). We find that the condition for the evolution of cooperation, i.e. Hamilton's rule, is given by

$$-C + BR - (B - C)\upsilon\varphi R > 0, \tag{1}$$

where: υ is the reproductive value (Fisher 1930; Grafen 2006) of a non-dispersing offspring, and φ is the scale of competition, that is the probability that two offspring sampled at random after dispersal are both philopatric to the patch. The inclusive fitness interpretation of the left-hand side (LHS) of inequality (1), i.e. the selection gradient, is as follows: an actor pays a cost C to provide a benefit B to her patch mates, including herself, to whom she is related by R; finally, this cooperative behaviour leads to an increase in local productivity, which displaces B-C offspring that are locally born with probability φ, and that have a reproductive value of υ and are related to the actor by R. The reproductive value υ is given by

$$\upsilon = \frac{pa}{pa + (1-p)(1-a)}, \tag{2}$$

where a is the quality of an occupied patch. If $a = 1$, then an occupied fertile patch always remains fertile, while an empty unfertile patch never recovers fertility. By contrast, if $a = 0$, then a fertile patch will certainly become unfertile the next season, while an unfertile patch will certainly become fertile the next season (see Appendix B for details).

Setting the LHS of inequality (1) to zero, we get the condition for which investing a little more into cooperation is exactly equal to investing a little less. Re-arranging this equation we get the potential for cooperation A, which has the form $A = C/B$ (Rodrigues and Gardner 2013a), and which is given by

$$A = \frac{R - \upsilon\varphi R}{1 - \upsilon\varphi R}. \qquad (3)$$

The potential for cooperation is the highest cost-to-benefit ratio that still allows the evolution of cooperation.

7.2.3. Hamilton's Rule: Dispersal

We now determine how natural selection acts on dispersal. We assume that there is no variation in the cooperation trait, so that, $x_C = y_C = z_C$. As before, we take the Taylor-Frank approach to kin selection (Taylor and Frank 1996; Frank 1998) to determine the direction of selection acting on the dispersal rate of juveniles (see Appendix for details). The condition for the evolution of slightly higher dispersal rates, i.e. Hamilton's rule, is given by

$$-1 + \upsilon_D + h_0 R > 0, \qquad (4)$$

where: v_D is the benefit of dispersal; and h_O is the probability of philopatry, that is the probability that an offspring sampled at random is local. . The inclusive fitness interpretation of the left-hand side (LHS) of inequality (4), i.e. the selection gradient, is as follows: a disperser suffers an immediate cost of -1 for not breeding in her natal patch; at the same time she gains a benefit v_D for dispersing and breeding in a distant patch; finally, she alleviates local competition in her natal patch, which benefits a philopatric individual with probability h_O, to whom she is related by R. The benefit of dispersal v_D is given by

$$v_\mathrm{D} = (1-c)\frac{pv_\mathrm{O}+(1-p)v_\mathrm{E}}{v_\mathrm{O}}, \tag{5}$$

where: v_O if the reproductive value of a disperser that arrives to an occupied patch and v_E is the reproductive value of a disperser that arrives to an empty patch. The benefit v_D (equation 5) can be interpreted as follows: with probability 1-c an offspring survives dispersal, in which case (i) with probability p she arrives to a previously occupied patch, where she derives a reproductive value of v_O, and (ii) with probability 1-p she arrives to an empty patch, where she derives a reproductive value of v_E. Note that the benefit of dispersal v_D is normalised to the reproductive value of an offspring competing for a breeding site in an occupied patch v_O. Moreover, $v_\mathrm{E} = \omega_\mathrm{E}(1-a)$ and $v_\mathrm{O} = \omega_\mathrm{O}a$, where $\omega_\mathrm{E} = 1/(pz_\mathrm{D}(1-c))$ and $\omega_\mathrm{O} = (1-z_\mathrm{D})/(1-z_\mathrm{D}+pz_\mathrm{D}(1-c))$ are the likelihoods of winning a contest in an empty and in an occupied patch, respectively.

Our aim is to determine the candidate evolutionary stable (cES) dispersal rate (z^*), that is the dispersal rate that leads to a null selection gradient. Although we can derive analytical expressions for the cES dispersal rates, these are not presented here due to their size and complexity. To determine the evolutionary stability of the cES dispersal rates, we expand the methodology developed by Metz and Gyllenberg (2001) and Ajar (2003). For this purpose, we construct a genetic model, equivalent to our kin selection model, that incorporates budding and empty patches. We use this genetic model, together with individual-based simulations, to check the validity of the analytical result derived from our kin selection model, and to check the convergence stability (Christiansen 1991; Eshel 1996) and evolutionary stability (Maynard Smith and Price 1973; Metz and Gyllenberg 2001; Ajar 2003) of the cES dispersal rates (see Supporting Information for details). Our analysis suggests that all cES dispersal rates are both convergence and evolutionary stable.

7.3. Results

We divide our results into four subsections. In the first subsection we focus on the evolution of cooperation assuming that dispersal is a fixed parameter. In the second subsection, we consider the evolution of dispersal. In the third subsection, we consider the joint evolution of cooperation and dispersal. Finally, we consider the relationship between budding and individuality.

7.3.1. Cooperation

We contrast our results to those of Taylor's (1992a) model. In contrast with our model, Taylor's (1992a) reference model assumes: (1) a saturated habitat (i.e. p =1), and therefore highest local habitat quality (i.e. $a = 1$); (2) a fixed dispersal rate that is treated as a parameter (i.e. $x_D = y_D = z_D = d$), which we also assume in this subsection; and (3) that both dispersal and competition occur at the level of the individual. As a result the potential for cooperation of Taylor's (1992a) model is given by $A_T = (R_T-\varphi_T R_T)/(1-\varphi_T R_T)$, where $\varphi_T = (1\text{-}d)^2$ is the scale of competition (Frank 1998), and where $R_T = 1/(n\text{-}(n\text{-}1)\varphi_T)$ is the relatedness among group members. This gives a potential for cooperation of $A_T = 1/n$, which is independent of the dispersal rate ($dA_T/dd = 0$), and which decreases with increasing group size ($dA_T/dn < 0$). There are some differences between our model and the reference model that are important to highlight. First, while in Taylor's (1992a) model the weight of kin competition is simply the scale of competition φ_T times the relatedness R_T, in our model there is an additional effect of the reproductive value of local offspring υ (see inequality 1). Second, while in Taylor's reference model relatedness decreases with increasing dispersal rate, in our model relatedness is not a function of dispersal. Instead, in our model relatedness R decreases with increasing patch occupancy and increases with increasing cost of dispersal (Figure 1A; see Appendix for details). Finally, while in Taylor's reference model the scale of competition is a function of dispersal only, in our model the scale of competition is also a function of the cost of dispersal c and of patch occupancy p. Specifically, higher cost of dispersal and lower habitat occupancy leads to higher scale of competition (Figure 1C,D). Note that in our model relatedness is given by $R = 1/(n\text{-}(n\text{-}1)\varphi_m)$, where φ_m is the probability of co-philopatry in relation to migration. We assume that the probability of co-philopatry φ_m is $(1\text{-}m_I)/(1\text{-}m_I\text{+}pm_I(1\text{-}c))$, where m_I is the intrinsic migration rate (see Appendix for details).

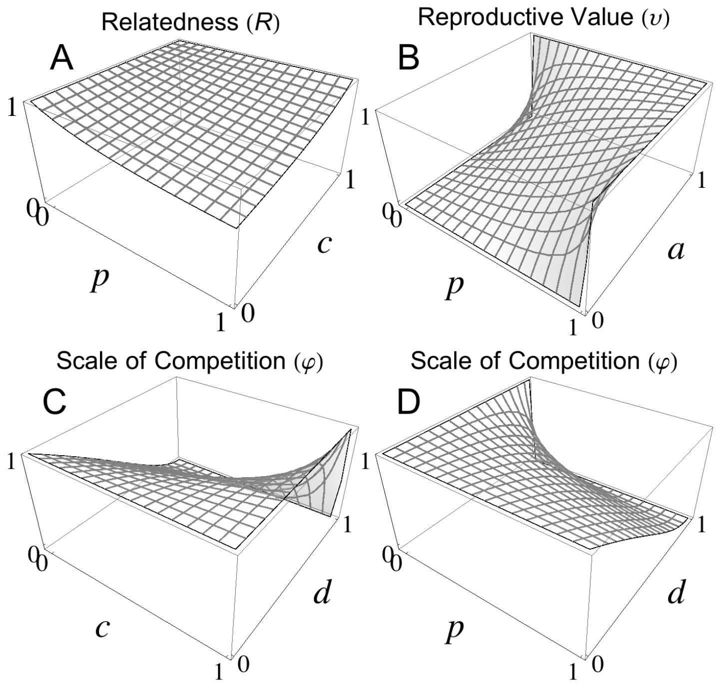

Figure 1 | Relatedness (R), relative reproductive value (υ) and scale of competition (φ) as a function of model parameters. [A] Relatedness decreases with increasing habitat occupancy (p). By contrast, it increases with increasing cost of dispersal (c). [B] The value of local offspring rises when both habitat occupancy (p) rise and local habitat quality (a) rise. [C] The scale of local competition rises when the cost of dispersal (c) rises, and falls when the dispersal rate (d) rises. Parameter values: $p = 0.5$. [D] The scale of competition falls when habitat occupancy (p) rises, and when the dispersal rate (d) falls. Parameter values: $c = 0.5$. [A-D] Parameter values: $n = 2$, $m_\mathrm{I} = 0.3$.

Now, we turn the attention to our model. How does the dispersal rate influence the potential for cooperation? We find that that the potential for cooperation increases with increasing dispersal rate ($\mathrm{d}A/\mathrm{d}d > 0$; Figure 2A,B). The reason is that while the kin selected benefits remain constant with increasing dispersal rate ($\mathrm{d}R/\mathrm{d}d = 0$), the scale of competition (and consequently the weight of the kin competition costs, $\upsilon\varphi R$) decreases with increasing dispersal rate ($\mathrm{d}\varphi/\mathrm{d}z_\mathrm{D} < 0$; Figure 1C,D).

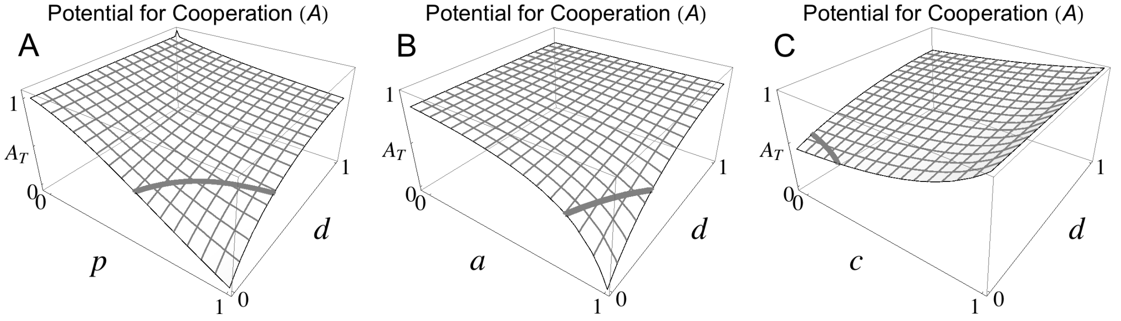

Figure 2 | The potential for cooperation (A) as a function of model parameters. The bold grey curve denotes the potential for cooperation of the reference model ($A_Ø$). [A] The potential for cooperation falls when habitat occupancy (p) rises, and rises when the dispersal rate (d) rises. Parameter values: $a = 0.75$, $c = 0.5$. [B] The potential for cooperation falls when the local patch quality (a) rises. Parameter values: $p = 0.5$, $c = 0.5$. [C] The potential for cooperation rises when the cost of dispersal (c) rises. Parameter values: $p = 0.5$, $a = 0.75$. [A-C] Parameter values: $n = 2$, and $m_I = 0.3$.

How does the cost of dispersal influence the potential for cooperation? First, we focus on cases where occupied patches never become extinct, i.e. $a = 1$. We find that if the dispersal rate is greater / less than the intrinsic migration rate, then the potential for cooperation increases / decreases with increasing cost of dispersal (if $d > m_I$ then $dA/dc > 0$; if $d < m_I$ then $dA/dc < 0$). An intuitive explanation for this rests on the observation that when dispersal is greater than the intrinsic migration rate, then relatedness is relatively greater than the scale of competition. Therefore an increase in the cost of dispersal has a higher impact on relatedness than on the scale of competition, which promotes cooperation. The reverse is true if dispersal is less than the intrinsic migration rate. Now let us focus on cases where occupied patches may become extinct, i.e. lower local habitat quality ($a < 1$). We find that the potential for cooperation, in general, increases with increasing cost of dispersal, and this is independent of the relationship between the dispersal rate and the migration rate ($\mathrm{d}A/\mathrm{d}c > 0$; Figure 2C). If patches may become extinct, offspring that remain in the local patch may not have the opportunity to breed, and therefore their reproductive value is less than one ($\upsilon < 1$; Figure 1B). As a result, the kin competition costs are attenuated. Thus, an increment in the cost of dispersal does not increase kin competition as much as it increases the kin selection benefits. Hence, there is a positive effect on the potential for cooperation if the cost of dispersal increases.

How does habitat occupancy influence the potential for cooperation? We find that if local habitat quality is highest ($a = 1$), the potential for cooperation decreases / increases with increasing habitat occupancy, given that the dispersal rate is greater / less than the intrinsic migration rate (if $d > m_\mathrm{I}$, then $\mathrm{d}A/\mathrm{d}p < 0$; if $d < m_\mathrm{I}$, then $\mathrm{d}A/\mathrm{d}p > 0$). By contrast, if local habitat quality is not highest ($a < 1$), the potential for cooperation always decreases with increasing habitat occupancy ($\mathrm{d}A/\mathrm{d}p < 0$; Figure 2A). Note that this is the same pattern of results we have shown above for the impact of the cost of dispersal on the potential for cooperation. This is because the cost of dispersal and habitat occupancy mediate relatedness and the scale of competition in a similar way. Specifically, both higher cost of dispersal or lower habitat occupancy reduce the number of incoming, which increases the scale of competition and relatedness.

How does local habitat quality influence the potential for cooperation? We find that the potential for cooperation decreases with increasing local habitat quality ($\mathrm{d}A/\mathrm{d}a < 0$; Figure 2B). The reason is that while higher local habit quality does not affect the kin selected benefits, it does increase the kin competition costs. This is because higher local habitat quality increases the reproductive value of offspring competing locally ($\mathrm{d}\upsilon/\mathrm{d}a > 0$), which, in turn, increases the weight of local kin competition (Figure 1B).

How do our results compare with those of Taylor's (1992a) reference model? We find that for a large number of demographic and ecological scenarios budding and empty patches promote the evolution of cooperation ($A > A_T$; Figure 2). Two main reasons explain this. First, budding increases relatedness, even when dispersal is high, and this, in turn, increases the kin selected benefits. Second, local patch extinctions reduces the scale of local competition, even when dispersal is low, and this, in turn, decrease the kin competition costs (Figure 2). This second effect is particularly important when occupied patches are very likely to go extinct, and empty patches are very likely to recover fertility, i.e. when a → 0.

7.3.2. Dispersal

We contrast our results to those of a homogeneous saturated population with individual dispersal ($p = 1$; Hamilton and May 1977; Frank 1986; Taylor 1988). In this case, the condition for the evolution of slightly higher dispersal rates, i.e. Hamilton's rule, is $-1+(1-c)+h_{\varnothing}R_{\varnothing} > 0$, where $h_{\varnothing} = (1-z_D)/(1-z_D+z_D(1-c))$, and $R_{\varnothing} = 1/(n-(n-1)h_{\varnothing}^2)$. The ES dispersal rate is $z_{\varnothing}^* = (H+1-2nc)/(H+1-2nc^2)$, where $H = \sqrt{}(1+4n(n-1)c^2)$ (Frank 1986; Taylor 1988; Taylor and Frank 1996; Ajar 2003). The ES dispersal rate $z_{\varnothing}^*$ is a decreasing function of the cost of dispersal c ($\mathrm{d}z_{\varnothing}^*/\mathrm{d}c < 0$) and of patch size n ($\mathrm{d}z_{\varnothing}^*/\mathrm{d}n < 0$). Note that in this reference model the benefit of dispersal is simply the probability of survival $1-c$, while in our model the benefits of dispersal also depend on habitat occupancy and on the reproductive value in occupied and in empty patches (see equation 5; Figure 3). Furthermore, in our model, the benefit of dispersal v_D may be greater than that of the reference model when the quality of local patches is low (Figure 3B).

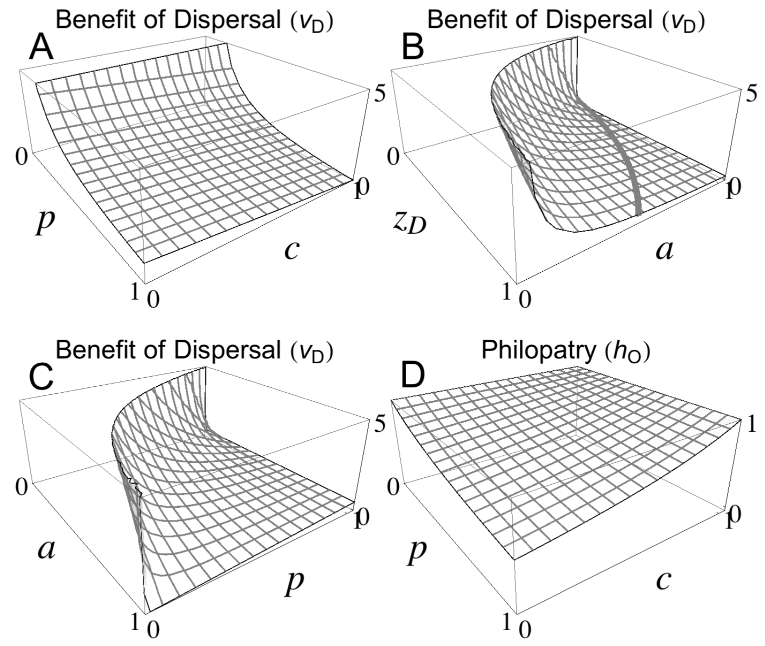

Figure 3 | The benefit of dispersal (v_D) and the probability of philopatry (h_O) as a function of model parameters and the population dispersal rate. [A] The benefit of dispersal falls, when both habitat occupancy (p) and the cost of dispersal (c) rises. Parameter values: $z_D = 0.5$, and $a = 0.5$. [B] The benefit of dispersal falls, when both the population dispersal rate (z_D) and the local habitat quality (a) rise. Parameter values: $p = 0.5$, and $c = 0.5$. [C] The benefit of dispersal always falls, when local habitat quality (a) rises. By contrast, the benefit of dispersal falls with rising habitat occupancy (p) when local quality (a) is lower, but it rises with rising habitat occupancy when local quality is higher. Parameter values: $a = 0.5$, and $c = 0.5$. [D] The probability of philopatry rises, when habitat occupancy (p) rises, while it falls when the cost of dispersal rises (c). Parameter values: $z_D = 0.5$ [A-D] Parameter values: $n = 2$, and $m_I = 0.3$.

Now, we turn the attention to our model. How does the cost of dispersal influence the ES dispersal rate? We find that in cases where occupied patches never become extinct, i.e. when local habitat quality is highest ($a = 1$), the ES dispersal rate decreases with increasing cost of dispersal ($\mathrm{d}z_\mathrm{D}^*/\mathrm{d}c < 0$; Figure 4A). By contrast, when occupied patch can become extinct, i.e. when local habitat quality is not one ($a < 1$), dispersal may increase or decrease with increasing cost of dispersal ($\mathrm{d}z_\mathrm{D}^*/\mathrm{d}c > 0$ or $\mathrm{d}z_\mathrm{D}^*/\mathrm{d}c < 0$; Figure 4A). Specifically, the ES dispersal rate usually decreases at low cost of dispersal, while it usually increases at high cost of dispersal. How can we explain these results? Let us note that an increase in the cost of dispersal has two effects: first, it has a cost to the direct fitness component of the focal individual; and second, it also has a benefit to the indirect fitness component of the focal individual, as it increases both relatedness and philopatry (Figures 1A, and 3C). When local habitat quality is highest, the first effect is always stronger than the second and the ES dispersal rate always decreases with increasing cost of dispersal. However, this is not the case if local habitat quality is not highest. Under this scenario, at low cost of dispersal, the first effect is usually stronger than the second, and as a result an increment in the cost of dispersal decreases the ES dispersal rate. By contrast, at high cost of dispersal, the first effect is weaker than the second effect, and as a result an increment in the cost of dispersal increases the ES dispersal rate.

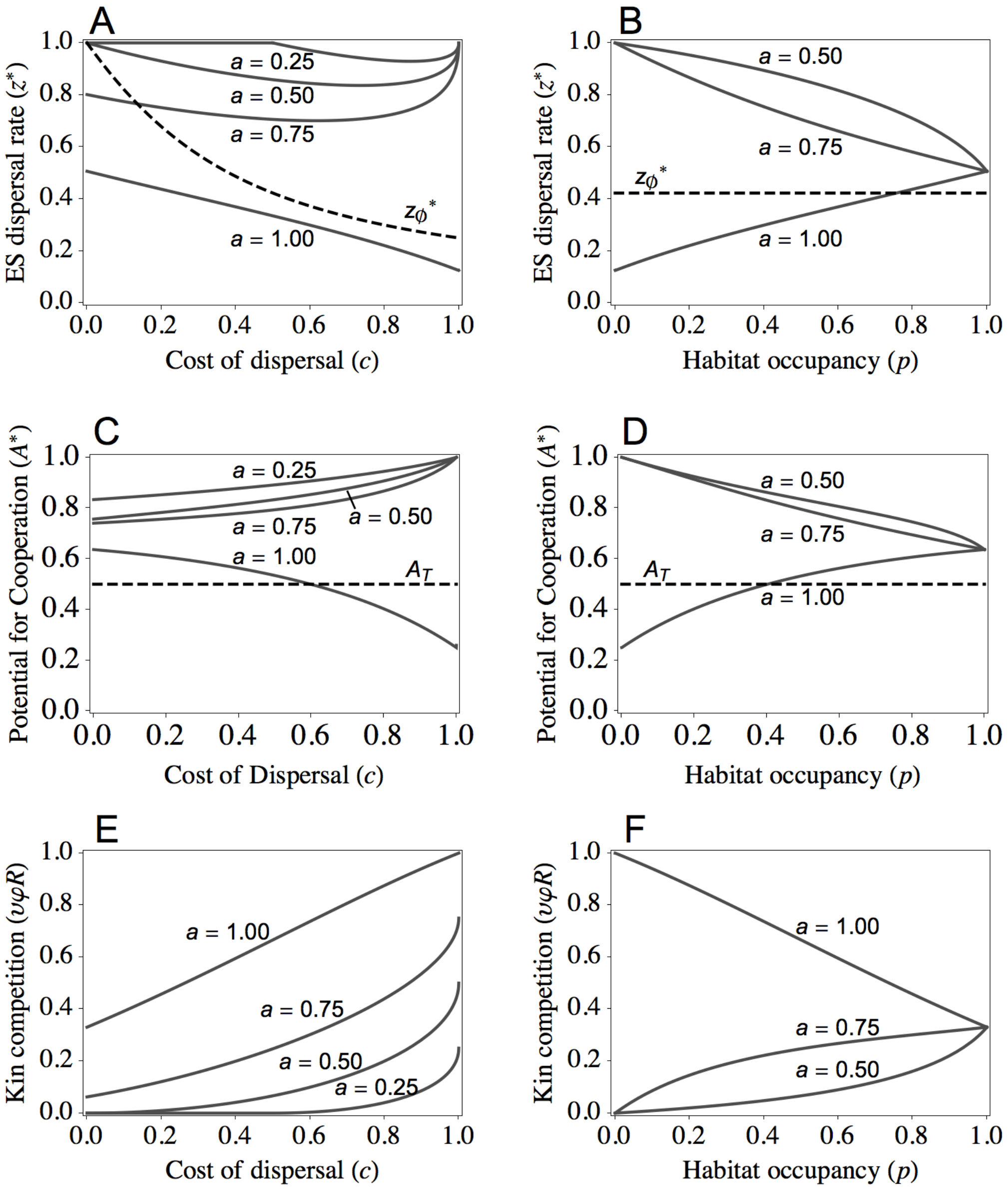

Figure 4 | The ES dispersal rate (z^*), the potential for cooperation (A^*) and the weight of kin competition ($v\phi R$) as a function of the cost of dispersal (c) and habitat occupancy (p), for varying local habitat quality (a). [A] When local habitat quality is equal to one, the ES dispersal rate (z^*) always decreases with increasing cost of dispersal. Otherwise, the ES dispersal rate decreases with increasing cost of dispersal when the cost of dispersal is lower, while it increases with increasing cost of dispersal when the cost of dispersal is higher. [B] When local habitat quality is one, the potential for cooperation decreases with increasing cost of dispersal. Otherwise, the potential for cooperation increases with increasing cost of dispersal. [C] The weight of kin competition increases with increasing cost of dispersal and with increasing local habitat quality. [B,D] When local habitat quality is one, both the ES dispersal rate and the potential for cooperation increase with increasing habitat occupancy. Otherwise, they decrease with increasing habitat occupancy. [F] When local habitat quality is one, the weight of kin competition decreases with increasing habitat occupancy. Otherwise, it increases with increasing habitat occupancy. [A,C, and E] Parameter values: $p = 0.5$. [B, D, and F] Parameter values: $c = 0.5$. [A-F] Parameter values: $n = 2$, $m_\mathrm{I} = 0.3$.

How does habitat occupancy influence the ES dispersal rates? First, we focus on cases where occupied patches never become extinct, i.e. local habitat quality is one ($a = 1$). In these cases, we find that the ES dispersal rate increases with increasing habitat occupancy ($\mathrm{d}z_\mathrm{D}^*/\mathrm{d}p > 0$; Figure 4B). By contrast, when occupied patches may become extinct, i.e. local habitat quality is less than one ($a < 1$), the ES dispersal rate decreases with increasing habitat occupancy ($\mathrm{d}z_\mathrm{D}^*/\mathrm{d}p < 0$; Figure 4B). How can we explain these results? The reason is that when local habitat quality is one, empty patches do not recover their fertility, and therefore individuals that settle in these patches do not breed. As a result, more empty patches (where the possibility of breeding is low) in the population represent an additional cost to dispersers, which disfavours dispersal. As the quality of empty patches increases (and the quality of occupied patches decreases), breeding in these patches becomes more likely (and the likelihood of breeding locally decreases). Consequently, when local quality is lower, dispersal increases with increasing frequency of empty patches.

How does local habitat quality influence the evolution of dispersal? We find that the ES dispersal rate decreases as the local habitat quality increases ($\mathrm{d}z_{\mathrm{D}}^*/\mathrm{d}a < 0$; Figure 4A,B). The reason is that when local quality increases, the quality of empty patches decreases, and therefore the benefit of dispersal decreases (Figure 3B), which, in turn, disfavours the evolution of dispersal.

How do our results compare with those of the reference model? We find that for a large number of demographic and ecological scenarios budding and empty patches promote the evolution of dispersal ($z^* > z_{\varnothing}^*$; Figure 4A,B). Scenarios where dispersal is disfavoured occur when empty patches are unlikely to recover fertility, and therefore, as we have seen before, represent a considerable cost for dispersers (Figure 4A,B).

7.3.3. Dispersal and Cooperation

Here we determine the potential for cooperation A^* assuming that the population reached its ES dispersal rate z^*.

How does the cost of dispersal influence the potential for cooperation? We find that if occupied patches never become extinct, i.e. local habitat quality is highest ($a = 1$), the potential for cooperation decreases with increasing cost of dispersal ($\mathrm{d}A^*/\mathrm{d}c < 0$; Figure 4C). By contrast, if occupied patches may become extinct, i.e. local habitat quality is less than one ($a < 1$), the potential for cooperation increases with increasing cost of dispersal ($\mathrm{d}A^*/\mathrm{d}c > 0$; Figure 4B). This is because when local habitat quality is highest, the ES dispersal rate decreases with increasing cost of dispersal, which increases the scale of competition (Figure 1D), which then increases the kin competition costs (Figure 4A). This, in turn, reduces the potential for cooperation. When local habitat quality is less than one, cooperation is favoured because of two factors. First, lower habitat quality decreases the kin competition costs but not the kin selected benefits. As a result, the kin selected benefits are more sensitive to an increase in the cost of dispersal than the kin competition costs, and therefore cooperation is favoured. Second, high cost of dispersal can favour the evolution of dispersal, which reduces the kin competition costs, which then favours the evolution of cooperation.

How does habitat occupancy mediate the potential for cooperation? We find that when local habitat quality is highest ($a = 1$) the potential for cooperation decreases with increasing habitat occupancy ($\mathrm{d}A^*/\mathrm{d}p > 0$; Figure 4D). By contrast, when local habitat quality is less than one ($a < 1$) the converse relationship is observed ($\mathrm{d}A^*/\mathrm{d}p < 0$; Figure 4D). When local quality is highest ($a = 1$), higher habitat occupancy favours dispersal (Figure 4B), which reduces the weight of kin competition (Figure 4F), and this, in turn, promotes the evolution of cooperation. When local quality is less than one ($a < 1$), higher habitat occupancy disfavours dispersal (Figure 4B), which increases the weight of kin competition (Figure 4F), which then supresses the evolution of cooperation.

How does local habitat quality influence the potential for cooperation? We find that the potential for cooperation decreases as the local habitat quality increases ($\mathrm{d}A^*/\mathrm{d}a < 0$; Figure 4C,D). Higher local quality disfavours dispersal (Figure 4A,B), which increases the weight of kin competition (Figure 4E,F), and this, in turn, supresses the evolution of cooperation.

What is the relationship between dispersal and cooperation? Often we find a positive association between dispersal and cooperation ($\mathrm{d}z_{\mathrm{D}}^*/\mathrm{d}A^* > 0$; compare Figure 4A,B with

Figure 4C,D). Exceptions to this scenario occur when the cost of dispersal is low. In this case, while dispersal decreases with increasing cost of dispersal, the potential for cooperation increases with increasing cost of dispersal, and therefore a negative association between dispersal and cooperation is observed.

7.3.4. Budding and Individuality

In the previous sections we have assumed that some migration between groups exists, and therefore groups do not become clonal. Here we explore some of the consequences of having no genetic exchange, i.e. migration, between groups ($m \to 0$). Under this scenario, and if we assume a saturated population, i.e. $p = 1$, we recover Taylor's (1992a) result for the evolution of cooperation when there is a single breeder per patch. In this case, the potential for cooperation is one ($A = A_T = 1$), and is independent of the dispersal rate. Similarly, under the same scenario, we also recover Hamilton and May's (1977) result for the evolution of dispersal, where the ES dispersal rate is $z^* = z_{\varnothing}^* = 1/(1+c)$.

7.4. Discussion

Here, we have studied how budding and empty patches drive the evolution of cooperation and dispersal in a viscous population. We have shown that budding and empty patches can lead to a positive association between dispersal and cooperation. This is because high dispersal reduces local kin competition without reducing within-group relatedness, and this favours the evolution of cooperation. In addition, conditions that favour the evolution of dispersal also favour the evolution of cooperation. For example, low local habitat quality favours cooperation not only because it favours dispersal, which reduces local kin competition, but also because nondispersers have lower breeding success, which further reduces local kin competition.

How does budding and empty patches influence the evolution of cooperation? Gardner and West (2006) developed a model of budding dispersal. In this model budding decouples the kin-selected benefit from the kin competition costs. As a result, if groups are not clonal, then budding favours the evolution of cooperation when dispersal is high, because high dispersal reduces kin competition and does not destroy kin groups. Conversely, when dispersal is low, kin competition increases and, as a result, cooperation is relatively disfavoured. We have shown that habitat variation may alter this result, that is cooperation can be favoured even when dispersal is low. This is because whilst low local habitat quality does not destroy kin groups, it greatly reduces kin competition because degradation of local conditions reduces the costs associated with kin competition.

How does budding and empty patches influence the evolution of dispersal? Gandon and Michalakis (1999) developed a model where they studied the effect of individual dispersal, budding dispersal, and patch extinctions on the evolution of dispersal. They found that dispersal is favoured as extinction events become more frequent. This is because local extinctions create empty patches, and thus, colonisation opportunities, where competition is low, and dispersal permits escape from local extinction events. In several respects, this result contrasts with ours. In their model higher fraction of empty patches in the population always lead to higher dispersal. In our model, by contrast, higher fraction of empty patches does not always lead to higher dispersal. For example, if empty patches rarely recover fertility (and the environment becomes less variable temporally), then dispersal is disfavoured. This is in line with previous results showing that spatial heterogeneity only disfavours the evolution of dispersal (Hastings 1983; Holt 1985; McPeek and Holt 1992). Gandon and Michalakis (1999) also found that budding dispersal had, in general, a negative effect on the evolution of dispersal, because it exports kin competition to distant patches. As we assume that competition occurs between coalitions/buds, rather than between individuals, kin competition does not occur in distant patches, and therefore, in our model, budding favours dispersal.

How does budding and empty patches influence the joint evolution of dispersal and cooperation? Perrin and Lehmann (2001) have studied the evolution of dispersal and the evolution of cooperation that is expressed by local individuals. They found a negative correlation between dispersal and cooperation, and, in particular, that high cost of dispersal leads to low dispersal, which then leads to high cooperation because of high relatedness. By contrast, in our model, high cost of dispersal can lead to high dispersal, which then leads to high cooperation because of low local competition. Le Galliard et al. (2005) also studied the evolution of dispersal and cooperation, finding a positive correlation between dispersal and cooperation. We also find a positive correlation between dispersal and cooperation, but for different reasons. While in Le Galliard et al. (2005) the evolution of altruism drives the evolution of dispersal, in our case, low local habitat quality promotes dispersal, and this eases local competition, which favours the evolution of cooperation.

The interaction between dispersal and cooperation in response to various ecological factors has attracted widespread interest in the context of the evolution of cooperative breeding. Cooperative breeders often display delayed dispersal of offspring. Therefore, theories for the evolution of cooperative breeding have highlighted the importance of heterogeneity in habitat quality to the evolution of delayed dispersal and how this can favour the evolution of cooperation (reviewed in Solomon and French 1997; and Koenig and Dickinson 2004). Thus, a negative association between dispersal and cooperation is at the core of these hypotheses, which contrasts with our prediction of a positive relationship between dispersal and cooperation. These opposing predictions are not necessarily incompatible. Instead, they emerge from different model assumptions. For example, in these hypotheses, limited dispersal is assumed to increase within-group relatedness to a greater extent than it increases kin competition. By contrast, in our model, dispersal has no influence on within-group relatedness, owing to budding, but it does have a large impact on kin competition. This highlights how fine demographic and ecological details may have considerable impact on the selective forces acting on dispersal and cooperation.

Reflecting on the results gained in this study, it is interesting to consider the parallels that may be important for vector-borne diseases, such as malaria. Malaria often exists within a host as a mixed-genotype infection, i.e. they are frequently dispersing to occupied patches (Read et al. 2002). But kinship patterns observed within an infected host suggest that relatedness within the mosquito vector (i.e. during dispersal) is high (Nkhoma et al. 2012). Moreover, it seems that parasites are able to discriminate between related and non-related malaria parasites (Reece et al. 2008), suggesting kin structure to be important in the success of the parasitic lifecycle. If relatedness within the vector is high, then relatedness is not destroyed by dispersal, and a type of budding dispersal is established. In addition, theoretical work has shown that both high competition within a mixed-genotype infection (Read et al. 2002) and long distance transmission mechanisms (such as those that are vector-borne; Boots and Sasaki 1999) will select for more virulent pathogens.

The consequence of this higher virulence is a more unstable patch (because the host dies more quickly), and our work predicts this will also select for a higher dispersal rate. Therefore, in this example at least, we find a nice comparison whereby a budding group remains competitive when entering a colonised patch, however, the intense within patch competition reduces patch stability and subsequently selects for high dispersal.

7.5. Appendix

7.5.1. A. Patch Dynamics

Here we follow the ecological dynamic of patches as described in the main text. This ecological dynamic can be described by a transition matrix, which is given by

$$\mathbf{P} = \begin{pmatrix} \alpha & 1-\beta \\ 1-\alpha & \beta \end{pmatrix}. \tag{A1}$$

At any given breeding season, the frequency of occupied patches is given by the right-eigenvector of matrix **P**, and this is

$$p = \frac{1-\beta}{2-(\alpha+\beta)}, \tag{A2}$$

when α or β is less than one.

7.5.2. B. Reproductive Success

Here we define the fitness function of a focal individual for the life-cycle described in the main text. It is useful to transform the two variables that describe how patches change their quality from one breeding season to the other, i.e. α and β, into a single variable a, where a is defined as

$$a = \frac{\alpha}{\alpha + 1 - \beta}\,. \tag{A3}$$

This is the probability that an occupied patch remains fertile relative to the probability that an empty patch recovers to become fertile. Note that: if occupied patches are more likely to be fertile in the next season than empty patches, then $a > 0.5$; if both are equally likely to be fertile in the next season, then $a = 0.5$; and if occupied patches are less likely to become fertile in the next season than empty patches, then $a < 0.5$. Thus, we define a as the quality of occupied patches relative to empty patches. Given this definition, the fitness function of a focal individual is given by

$$\begin{aligned} w(x_\mathrm{D}, y_\mathrm{D}, z_\mathrm{D}, x_\mathrm{C}, y_\mathrm{C}, z_\mathrm{C}) = \frac{(1-x_\mathrm{D})F(x_\mathrm{C},y_\mathrm{C})a}{(1-y_\mathrm{D})F(y_\mathrm{C},y_\mathrm{C})+pz_\mathrm{D}F(z_\mathrm{C},z_\mathrm{C})(1-c)} + \\ x_\mathrm{D}F(x_\mathrm{C}, y_\mathrm{C})(1-c)\left(\frac{pa}{(1-z_\mathrm{D})F(y_\mathrm{C},y_\mathrm{C})+pz_\mathrm{D}F(y_\mathrm{C},y_\mathrm{C})(1-c)} + \frac{(1-p)(1-a)}{pz_\mathrm{D}F(y_\mathrm{C},y_\mathrm{C})(1-c)}\right)\end{aligned}. \tag{A4}$$

7.5.3. C. Hamilton’s Rule: Cooperation and Dispersal

To determine the selection gradient for the trait of interest, we use the Taylor-Frank approach to kin selection (Taylor and Frank 1996; Frank 1998; Taylor et al. 2007). We assume that the genetic variation is vanishingly small and is segregating at a locus responsible for the behaviour. We denote the breeding value (Falconer 1989) for the locus of interest as g_D and g_C for the dispersal behaviour and the cooperation behaviour, respectively. The selection gradient for the trait of interest is

$$\frac{\mathrm{d}w}{\mathrm{d}g} = \frac{\partial w}{\partial x}\frac{\mathrm{d}x}{\mathrm{d}g_x}\frac{\mathrm{d}g_x}{\mathrm{d}g} + \frac{\partial w}{\partial y}\frac{\mathrm{d}y}{\mathrm{d}g_y}\frac{\mathrm{d}g_y}{\mathrm{d}g}, \tag{A5}$$

where: g_x is the breeding value of the focal individual; g_y is the breeding value of its social partners, $\partial w/\partial x$ and $\partial w/\partial y$ are the marginal fitness effects of the mutant behaviour; $\mathrm{d}x/\mathrm{d}g_\mathrm{x}$ and $\mathrm{d}y/\mathrm{d}g_\mathrm{y}$ are the genotype-to-phenotype mapping, which we arbitrarily set to 1. All the derivatives and partial-derivatives are evaluated at $x = y = z$ (Taylor and Frank 1996; Frank 1998; Taylor et al. 2007). The correlations between breeding values are the coefficients of consanguinity that when normalized to the actor's coefficient of consanguinity give the kin selection relatedness coefficients (Bulmer 1994). Higher breeding values evolve when the selection gradient, as given in A4, is positive (i.e. $\mathrm{d}w/\mathrm{d}g > 0$).

7.5.4. D. Relatedness

We now derive the kin selection relatedness coefficients. We assume a neutral population and we define a recursion equation (Taylor 1992a; Rousset 2004; Rodrigues and Gardner 2012). The probability that a presently occupied patch was occupied in the previous season is P(O|O), and the probability that a presently occupied patch was empty in the previous season is P(E|O). The probability of co-philopatry is φ_m, and this is the probability that two individuals chosen at random from a group are offspring of mothers that belonged to the same group in the previous breeding season. The probability that two offspring chosen at random are siblings is $1/n$. Finally, the probability that two offspring chosen at random are offspring of two different mothers that were groups mates in the previous season is $(n-1)/n$, in which case they are related by r. These probabilities can be added together to define a recursion equation describing changes in relatedness from one breeding season to the other, which, at equilibrium, is given by:

$$r = P(0|0)\varphi_{\mathrm{m}}\left(\frac{1}{n}+\frac{n-1}{n}r\right) + P(\mathrm{E}|0)\varphi_{\mathrm{m}}\left(\frac{1}{n}+\frac{n-1}{n}r\right). \tag{A6}$$

We solve this equation to find relatedness at equilibrium, which is

$$r = \frac{\varphi_{\mathrm{m}}}{n-(n-1)\varphi_{\mathrm{m}}}. \tag{A7}$$

This is the “other-only” relatedness coefficient (Pepper 2000). “Whole-group” relatedness is given by $R = 1/n+(1-1/n)r$ (Pepper 2000), which gives

$$R = \frac{1}{n-(n-1)\varphi_{\mathrm{m}}}. \tag{A8}$$

7.6. Supporting Information to “Empty patches, budding, and the evolution of dispersal and cooperation”

7.6.1. Genetic model

Here we define a genetic model corresponding to the kin selection model described in the main text. We use this genetic model to assess the stability of the candidate evolutionary stable (cES) dispersal rates derived from the kin selection model. Our genetic model follows the methodology developed by Metz and Gillenberg (2001) and Ajar (2003), and, at the same time, extends it to accommodate budding dispersal and competition. We define classes of patches according to their number of mutants j. Our goal is to determine the number of k-patches, i.e. patches with k mutants, produced by a j-patch. To this purpose, we define a matrix **A** describing reproduction, dispersal, and competition events, and a matrix **M** describing the migration of adults between patches, before they reproduce. The transition (fitness) matrix **w**, describing the rate at which j-patch produce k-patches, is given by the inner product of these two matrices, i.e. $\mathbf{w} = \mathbf{M} \bullet \mathbf{A}$.

We consider a focal patch with j mutant individuals. Each of the j mutant individuals gives birth to F offspring, of which a fraction $1\text{-}x_{\mathrm{D}}$ remain in the focal patch. Likewise, each of the $n\text{-}j$ wild-type individuals gives birth to F offspring, of which a fraction $1\text{-}z_{\mathrm{D}}$ remain in the focal patch. Hence, the total number offspring that do not disperse, i.e. philopatric offspring, is $j(1\text{-}x_{\mathrm{D}})F+(n\text{-}j)(1\text{-}z_{\mathrm{D}})F$. These offspring associate in groups of n individuals each, and therefore the expected number of philopatric groups that compete locally is $j(1\text{-}x_{\mathrm{D}})F+(n\text{-}j)(1\text{-}z_{\mathrm{D}})F/n$. These groups compete with successful migrant groups from occupied patches, which is given by $pnz_{\mathrm{D}}(1\text{-}c)F/n$. Hence, the probability that a philopatric group wins the contest for the focal patch, and that the patch remains fertile is

$$G_{j,\mathrm{P}} = \frac{j(1-x_{\mathrm{D}})+(n-j)(1-z_{\mathrm{D}})}{j(1-x_{\mathrm{D}})+(n-j)(1-z_{\mathrm{D}})+p\,n\,z_{\mathrm{D}}(1-c)}\alpha. \tag{S1}$$

Applying the same logic as before, the expected number of dispersing groups produced by a focal patch that survive dispersal is $(jx_{\mathrm{D}}F+(n\text{-}j)z_{\mathrm{D}}F)(1\text{-}c)/n$. With probability p, these dispersing groups arrive to a distant occupied fertile patch, and therefore they compete with $n(1\text{-}z_{\mathrm{D}})F/n$ local groups, and with $pnz_{\mathrm{D}}(1\text{-}c)F/n$ migrant groups. With probability α the patch of arrival remains fertile. With probability $1\text{-}p$, these dispersing groups arrive to a distant empty unfertile patch, and therefore they compete with $pnz_{\mathrm{D}}(1\text{-}c)F/n$ migrant groups. The patch of arrival recovers fertility with probability 1-β. Thus, the expected number of contests for fertile patches that are won by the dispersing groups is

$$G_{j,\mathrm{D}} = (jx_{\mathrm{D}} + (n-j)z_{\mathrm{D}})(1-c)\left(p\frac{\alpha}{n(1-z_{\mathrm{D}})+p\,n\,z_{\mathrm{D}}(1-c)} + (1-p)\frac{(1-\beta)}{p\,n\,z_{\mathrm{D}}(1-c)}\right). \tag{S2}$$

We now determine the expected number of mutants in philopatric and dispersing groups. A focal patch with j mutant individuals gives origin to philopatric groups with i mutants with probability

$$W_{j\to i,\mathrm{P}} = \binom{n}{i}\left(\frac{j(1-x_{\mathrm{D}})}{j(1-x_{\mathrm{D}})+(n-j)(1-z_{\mathrm{D}})}\right)^{i}\left(1-\frac{j(1-x_{\mathrm{D}})}{j(1-x_{\mathrm{D}})+(n-j)(1-z_{\mathrm{D}})}\right)^{n-i}. \tag{S3}$$

Combining equation (S1) with equation (S3), we obtain the probability that a focal patch with j mutants gives origin to a patch with i mutants, which is then given by

$$\pi_{j\to i,\mathrm{P}} = G_{j,\mathrm{P}}W_{j\to i,\mathrm{P}}\ . \tag{S4}$$

A focal patch with j mutant individuals produce dispersing groups with i mutants with probability

$$W_{j\to i,D} = \binom{n}{i}\left(\frac{jx_{\mathrm{D}}}{jx_{\mathrm{D}}+(n-j)z_{\mathrm{D}}}\right)^{i}\left(1-\frac{jx_{\mathrm{D}}}{jx_{\mathrm{D}}+(n-j)z_{\mathrm{D}}}\right)^{n-i}. \tag{S5}$$

Combining equation (S2) with equation (S5), we obtain the probability that a focal patch with j mutants gives origin to a distant patch with i mutants, which is then given by

$$\pi_{j\rightarrow i,\mathrm{D}} = G_{j,\mathrm{D}} W_{j\rightarrow i,\mathrm{D}} \ . \qquad \text{(S6)}$$

Following competition, breeding females may exchange groups. Breeding females remain in their patch with probability 1-m, where m is the effective migration which is defined as $m = pm_{\mathrm{I}}(1\text{-}c)/(1\text{-}m_{\mathrm{I}}+ pm_{\mathrm{I}}(1\text{-}c))$. We define the migration matrix **M** of order $n\times n$, whose elements m_{ik} are the rates at which a patch with i mutants gives origin to a patch with k mutants after migration. To illustrate how the elements of the migration matrix are defined, let us consider a population where each patch has two breeders. If a patch has a single mutant, then with probability 1-m the mutant female remains in her patch, otherwise she migrates to a patch where she will be the only mutant. Thus, a patch with a single mutant always gives rise to a patch with a single mutant, and therefore $m_{11} = 1$. From this, it follows that $m_{12} = 0$. If the patch has two mutants, then with probability $(1-m)^2$ both mutants remain in the local patch. Otherwise, at least one mutant disperses, in which case they give rise to two patches with a single mutant. Therefore, the transition matrix when patch size is 2 is given by

$$\mathbf{M} = \begin{pmatrix} 1 & 2(1-(1-m)^2) \\ 0 & (1-m)^2 \end{pmatrix}. \qquad \text{(S7)}$$

The fitness matrix is then defined by the inner product of matrix **M** and matrix **A**, such that, $\mathbf{w}(x_{\mathrm{D}},z_{\mathrm{D}}) = \mathbf{M}\bullet\mathbf{A}$.

7.6.2. Evolutionary stability of dispersal rates

Here we determine the convergence stability (CS; Christiansen 1991; Eshel 1996; Taylor 1989) and evolutionary stability (ES; Maynard Smith 1982; Metz and Gyllenberg 2001;

Ajar 2003; Rousset 2004) of candidate evolutionary stable (cES) dispersal rates. Our first goal is to define the appropriate measure of fitness, which we then use to determine the cES dispersal rates and their stability. Ajar (2003), following Metz and Gyllenberg (2001), defined fitness as the total number of successful emigrants descendent from a single successful immigrant. This is also the long-term contribution to future populations of a single mutant. Fisher (1930) defined the long-term genetic contribution to future populations of an individual as her reproductive value (see also Grafen 2006). The individual reproductive value of each class is given by the elements of the left-eigenvector associated with the leading eigenvalue of the fitness matrix (Leslie 1948; Goodman 1968; Charlesworth 1980a; Taylor 1990; Caswell 2001). Thus, in our model, the reproductive value of a mutant in a patch with a single mutant is given by the first element $e_1(x_\mathrm{D},z_\mathrm{D})$ of the left eigenvector of matrix $\mathbf{w}(x_\mathrm{D},z_\mathrm{D})$, as defined in the previous section, and this is equivalent to the fitness measure defined by Metz and Gyllenberg (2001) and Ajar (2003). Thus, $R_\mathrm{m}(x_\mathrm{D},z_\mathrm{D}) = e(x_\mathrm{D},z_\mathrm{D})$. cES dispersal rates are those that lead to a null selection gradient, i.e. $\mathrm{d}R_\mathrm{m}(x_\mathrm{D},z_\mathrm{D})/\mathrm{d}x_\mathrm{D} = 0$, with $x_\mathrm{D} = z_\mathrm{D}$. A cES dispersal rate is convergence stable if

$$\frac{\partial}{\partial z_\mathrm{D}}\left(\left.\frac{\partial R_\mathrm{m}(x_\mathrm{D},z_\mathrm{D})}{\partial x_\mathrm{D}}\right|_{x_\mathrm{D}=z_\mathrm{D}}\right)\Bigg|_{z_\mathrm{D}=z_\mathrm{D}^*} < 0. \tag{S8}$$

Finally, a cES dispersal rate is evolutionary stable (ES) if

$$\left.\frac{\partial^2 R_\mathrm{m}(x_\mathrm{D},z_\mathrm{D})}{\partial {x_\mathrm{D}}^2}\right|_{x_\mathrm{D}=z_\mathrm{D}=z_\mathrm{D}^*} < 0. \tag{S9}$$

7.6.3. Pairwise Invasibility Plots

The cES dispersal rates and their stability can be graphically assessed by Pairwise Invasibility Plots (PIPs; e.g. Ajar 2003; Rousset 2004). The solid diagonal line in Figure S3 and S4 is where the variant dispersal rate x_D is equal to the resident dispersal rate z_D and therefore where the selection gradient is null, $\partial R_m(x_D,z_D)/\partial x_D = 0$ with $x_D = z_D$. The solid curve is where other combinations of variant and resident dispersal rates also lead to a null selection gradient. Thus, the point where the solid diagonal line and the solid curve intersect is a cES dispersal rate. The cES dispersal rate is convergence stable (CS) if the region just above the solid line to the left of the cES dispersal rate is shaded and if the region just below the solid line to the right of the cES dispersal rate is also shaded. Moreover, the cES dispersal rate is evolutionary stable (ES) if the regions along the vertical line that passes trough the cES dispersal rate are not shaded. We have explored, both analytically and numerically, the condition (S8) for convergence stability and condition (S9) for evolutionary stability, and we have not found any instance where the cES dispersal rate is not convergent and evolutionary stable. Figures shows some illustrative examples of PIPs.

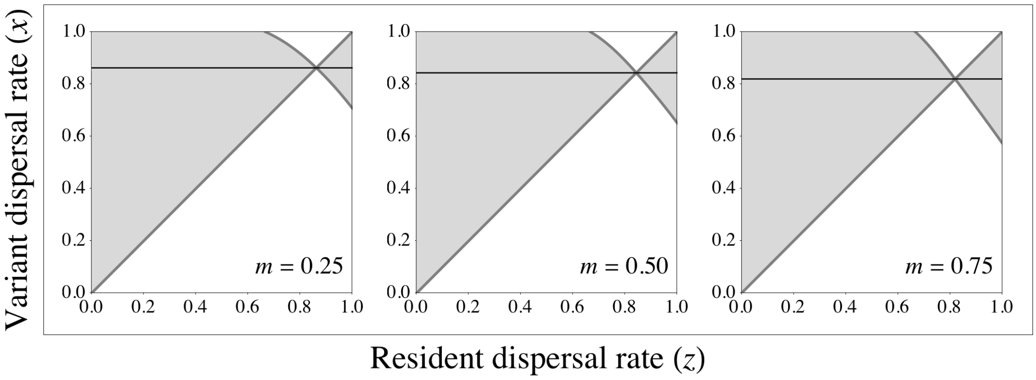

Figure S1 | Pairwise Invasibility Plots (PIPs) for varying intrinsic migration rate. The black line represents the analytical predictions. Parameter values: $n = 2$, $p = 0.50$, $a = 0.50$, $c = 0.50$.

7.6.4. Individual based simulations

We check the analytical results derived from the kin selection model described in the main text using individual-based simulations. We adjust the computer protocol develop by Rodrigues and Gardner (2012) to our model. We consider a population of 1000 patches with 2 breeding females each. The genotype of each individual is a number between 0 and 1, and every generation random mutations can slightly change the genotype of an individual. We run the simulations for a very large number of generations until the average phenotypic value stabilises.

Chapter 8. Discussion.

At the end of each chapter, I have provided a discussion of the results, where I have linked the findings of each chapter to the wider literature. Here, I will first summarise the findings of each chapter. Then, I will revisit Hamilton's (1964) original arguments on how population viscosity and phenotypic variation between individuals mediate the evolution of kin selected traits. I will connect the results of my chapters to these original arguments. I will also focus on key developments that arose from Hamilton's (1964) discussion of these topics. For recent reviews on some aspects of these topics see Frank (1998, 2013), and West et al. (2007a) and references therein.

8.1. Thesis summary

Habitat heterogeneity and phenotypic differences among individuals may both mediate the evolution of social traits, such as helping, harming and dispersal.

- In chapter 2, I analysed the action of kin selection when individuals condition their behaviour on their phenotype. I reviewed the literature on kin selection and individual phenotypic variation and developed a theoretical framework to understand how phenotypic variation among individuals mediates kin selection. I conclude that separating reproductive value from somatic/social value may be a useful way to understand how phenotypic differences drive kin selection.

- In chapter 3, I studied how heterogeneity in resource availability impacts on the evolution of social behaviour in a viscous population. I have considered both the unconditional and conditional expression of this trait, and I have found that while the unconditional expression of social behaviour is not favoured (i.e. not mediated by population viscosity), the conditional expression of social behaviour is favoured. In particular, conditional expression of helping and harming is favoured when variation in resource availability occurs across space and time, but not when this variation occurs across space only, or time only.

- In chapter 4, I studied how variation in group size impacts on the evolution of social behaviour in a viscous population. As in chapter 3, I have considered both unconditional and conditional expression of this trait, and found that while unconditional expression of cooperation is not favoured (i.e. not mediated by population viscosity), conditional expression of cooperation or harming is favoured. In contrast to the results of chapter 3, I have found that temporal variation in group size alone selects for the evolution of conditional social behaviour.

- In chapter 5, I have analysed the action of kin selection when individuals vary in quality. I have studied the influence of non-genetic phenotypic variation in reproductive capacity and local competition on kin selection. I have shown that kin selected traits are greatly influenced by both reproductive capacity and local competition. This can lead to extreme social behaviour, such as altruistic suicide.

- In chapter 6, I have considered the impact of local variation in density and competition on the evolution of dispersal. I showed that positive density-dependent dispersal is favoured in relatively stable habitats, while negative density-dependent dispersal is favoured in relatively unstable habitats.

- In chapter 7, I have investigated the influence of local habitat quality and budding on the evolution of dispersal, and how dispersal in turn influences the evolution of cooperation. I found that budding and variable environments may interact synergistically in mediating the evolution of cooperation.

8.2. Population viscosity and quality

8.2.1. Hamilton's theory of population viscosity

Hamilton (1964) developed a theory of how dispersal may affect cooperation. He argued that a concentration of relatives would form due to "a general inability or disinclination of the organisms to move far from their places of birth", and that this could lead to "find giving-traits commonest and most highly developed in the species with the most viscous populations whereas uninhibited competition should characterise species with the most freely mixing population". Hamilton noted that under these circumstances competition among kin may occur, and citing Haldane (1923) he acknowledged that "competition between sibs" has a negative impact on "the progress of gene-frequency change", which "would be slower than normal". Thus, although Hamilton thought that kin competition due to low dispersal would have a negative impact on gene-frequency change, he did not think that this would prevent the evolution of cooperation. I refer to this idea about how dispersal influences the evolution of cooperation Hamilton's theory of population viscosity.

8.2.2. Moths, quality, and kin competition

With this theory of population viscosity Hamilton analysed two kin selection problems. In the first problem, he analysed the evolution of post-reproductive behaviour of moths. In the second problem, he analysed the evolution of social behaviour in wasps. Let us first consider the evolution of post-reproductive behaviour of moths. Following Blest (1963), Hamilton considered the post-reproductive (i.e. individuals with low prospects of future reproduction) behaviour of aposematic and cryptic moth species. As Blest observed, in aposematic species post-reproductives should prolong their life. By contrast, in cryptic species post-reproductives should die quickly. Hamilton used his inclusive fitness theory to make the crucial remark that low future reproduction leads to a lower cost of cooperation. This would explain the altruistic behaviour of post-reproductive individuals. Thus, he connected differences in phenotypes with differences in the cost of social behaviour. Differences in phenotype explained why post-reproductive individuals expressed the altruistic behaviour, however, if this behaviour was indeed altruistic, it did not explain how individuals would reap indirect fitness benefits. Hamilton used his theory of population viscosity to provide a hypothesis for this problem. Specifically, if the behaviour was to evolve by kin selection, moth populations should be viscous.

However, Hamilton's theory of population viscosity suffers from a slight problem because he neglected the extent to which kin competition could prevent the evolution of cooperation. This was highlighted by Richard D. Alexander in his review of social behaviour (Alexander 1974; reviewed in Frank 2013). Alexander argued that kin competition could prevent the evolution of cooperation altogether. Thus, Hamilton's theory for the evolution of post-reproductive behaviour of moths is not complete, because it does not include the effects of kin competition. In fact, my model in chapter 5 provides a mathematical description of Hamilton's hypothesis, which in addition includes the effects of kin competition. I show that if behaviour is conditional on low fecundity, then cooperation can evolve even when the population is viscous and therefore when kin competition effects are taken into account. Thus, although Hamilton has neglected kin competition effects in his hypothesis, his prediction still holds, and therefore is still a valid explanation for the evolution of altruism in moth species.

8.2.3. Social wasps, environmental heterogeneity, and dispersal

Hamilton also applied his theory of population viscosity to the evolution of cooperation in *Polistes* wasps. To this purpose, he added a new proposition to his theory of population viscosity: he hypothesised that dispersal would be mediated by climate. In particular, he suggested that temperate regions due to their seasonality would promote the evolution of dispersal. This would lead to lower population viscosity, which would lead to lower relatedness among individuals. This, in turn, would disfavour the evolution of cooperation. By contrast, in tropical regions the environment is more stable, as a result individuals tend to stay near their place of birth, and therefore more cooperation would occur. He complemented this argument with aspects of behavioural and functional biology, such as the existence of kin recognition or the flight capacity of individuals.

With this more sophisticated theory of population viscosity, Hamilton attempted to explain behavioural differences between tropical and temperate *Polistes*. He noted that species that lived in tropical climate usually founded nests alone, whilst those in temperate climates founded nests in associations. He also noted that in these associations, the readiness of potential queens to accept auxiliary roles should be considered an altruistic trait, and therefore required explanation. Because associations occurred in tropical but not in temperate climate, he suggested that tropical climate should select for lower dispersal rates (due to its constancy), and therefore lead to a more viscous population favouring the evolution of cooperation.

Let us remember once more that the basic Hamilton's theory of population viscosity neglected the potential costs of kin competition. In chapter 3 and 4, I showed that environmental heterogeneity would not have any effect on the evolution of unconditional cooperation. However, environmental heterogeneity may select for conditional social behaviour, and therefore it can promote not only the evolution of cooperation, but also the evolution of harming. It would be interesting to investigate if populations living in unstable environments exhibit more conditional behaviour than populations living in stable environments.

8.2.4. Budding, dispersal, and cooperation

Interestingly, Hamilton noted that in some species colony reproduction was by swarming with several queens in each swarm. This would undermine his theory of how population viscosity mediates the evolution of cooperation in *Polistes* wasps, as some of these species are located in temperate climates. To overcome this problem, Hamilton further developed his theory of population viscosity. He suggested that in some cases queens in each swarm could be sisters. He gave the example of the North American species *P. annularis*, where after hibernation, queens return to their "old nest" before swarming to found a new nest. As a result, even if swarms dispersed widely their relatedness would be kept high, which would favour cooperation. This is in fact one of the first examples of budding being described in the context of inclusive fitness theory. This life-cycle roughly corresponds to the life-cycle described in chapter 7. Moreover, the results of chapter 7 are consistent with the prediction of Hamilton, despite the fact that he does not take into account the potential costs of kin competition. Thus, chapter 7 provides a formal model of how budding, environmental heterogeneity, and dispersal mediate cooperation, while also taking into account a neglected aspect of Hamilton's original theory, namely the negative effects of kin competition.

8.3. Conclusion

To conclude, kin selection acts on individuals that often differ phenotypically because of intrinsic non-genetic factors or environmental factors, i.e. differ in their quality. Substantial empirical evidence suggests that quality is important for the evolution of social behaviour. My thesis presents a novel way of looking at how quality variation among individuals mediates social evolution. It also suggests the development of theoretical models that take phenotypic and environmental heterogeneity into account. In particular, the development of general models and models tailored for specific biological systems constitute a promising line of research. At the same time, my thesis suggests that empirical studies that connect phenotypes and proximate mechanisms to long-term fitness would greatly contribute for the successful development of further theoretical work.

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Appendix

This Appendix contains the publications resulting from my DPhil:

Section 1. Rodrigues, A. M. M., and A. Gardner. 2012. Evolution of helping and harming in heterogeneous populations. *Evolution* 66, 2065-2079.

Section 2. Rodrigues, A. M. M., and A. Gardner. 2013. Evolution of helping and harming in viscous populations when group size varies. *The American Naturalist* 181, 609-622.

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EVOLUTION OF HELPING AND HARMING IN HETEROGENEOUS POPULATIONS

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There has been much interest in understanding how demographic factors can mediate social evolution in viscous populations. Here, we examine the impact of heterogeneity in patch quality—that is, the availability of reproductive resources for each breeder—upon the evolution of helping and harming behaviors. We find that, owing to a cancellation of relatedness and kin competition effects, the evolution of obligate and facultative helping and harming is not influenced by the degree of viscosity in populations characterized by either spatial or temporal heterogeneity in patch quality. However, facultative helping and harming may be favored when there is both spatial and temporal heterogeneity in patch quality, with helping and harming being favored in both high-quality and low-quality patches. We highlight the prospect for using kin selection theory to explain within-population variation in social behavior, and point to the need for further theoretical and empirical investigation of this topic.

KEY WORDS: **dispersal, facultative behavior, kin selection, obligate behavior, reproductive value, scale of competition.**

One of the major challenges for evolutionary biology is to explain the evolution of cooperative behavior (Maynard Smith and Szathmáry 1995; Hamilton 1996; West et al. 2007a). Natural selection favors those individuals who achieve greater personal reproductive success, relative to the other individuals in their population (Darwin 1859; Fisher 1930; Price 1970). All else being equal, a cooperative behavior that improves the reproductive success of another individual will reduce the actor's relative fitness, and hence it will be disfavored by natural selection. However, all else need not be equal. First, the actor may also derive a direct-fitness benefit, owing to mechanisms such as reciprocity (Trivers 1971) or by-product mutualism (West-Eberhard 1975). Such cooperation is termed "mutually beneficial" (West et al. 2007b). Second, the actor may derive an indirect-fitness benefit, as a consequence of being genetically related to the recipients of the cooperative behavior (Hamilton 1964). Such cooperation is termed "altruistic" (Hamilton 1964; West et al. 2007b).

One way in which relatedness may arise between social partners is if dispersal rates are low, such that individuals will tend to be genealogically close to their neighbors ("viscous" populations; Hamilton 1964, 1971). As this mechanism does not require discrimination of one's genetic relatives, it appears to provide a very general solution to the problem of altruistic cooperation. However, a problem with this idea is that limited dispersal may also exacerbate competition among social partners (Hamilton 1971; Queller 1992; West et al. 2002; Griffin et al. 2004). In the simplest scenario of an inelastic (i.e., saturated), viscous population, the relatedness and competition effects of limited dispersal exactly cancel, so that there is no net impact of the rate of dispersal upon the evolution of indiscriminate helping (Taylor 1992a, b; Kümmerli et al. 2009).

This has stimulated the development of a large body of theoretical—and, increasingly, empirical—research, examining factors that may decouple the relatedness and competition effects of dispersal. For example: population elasticity (Taylor 1992b; Alizon and Taylor 2008), overlapping generations (Taylor and Irwin 2000; Irwin and Taylor 2001), budding dispersal (Gardner and West 2006; Lehmann et al. 2006; Kümmerli et al. 2009), life-cycle and timing of the social behavior (Taylor 1992a; Lehmann and Rousset 2010), behaviors mediating patch extinction probabilities (Lehmann et al. 2006), variable group size (Grafen 2007), trans-generational altruism (Lehmann 2007, 2010), dispersal-dependent social behavior (El Mouden and Gardner 2008), and sex-biased dispersal (Johnstone and Cant 2008; Gardner 2010).

All this work has concerned populations in which resources are evenly distributed among patches—that is, patches do not vary in their intrinsic quality. However, all natural populations are characterized by some degree of resource heterogeneity, and the impact of this variation has been of great interest to ecologists and evolutionary biologists (Levins 1968; Begon et al. 2006). The potential for resource heterogeneity to mediate the evolution of helping behaviors has been considered by Frank (1996, 2003, 2010), suggesting that individuals with greater resources will be favored to make greater investments into helping. However, Frank's models do not place the evolution of social behavior within an explicit viscous population setting, and so cannot address the issue of whether population viscosity drives the evolution of social behavior.

Here, we investigate the impact of heterogeneity in resource availability on the evolution of indiscriminate helping in viscous populations. We consider scenarios in which different patches have different resource availability at any given time and/or the same patch has different resource availabilities at different times, (i.e., spatial and/or temporal heterogeneity; cf Cohen and Levin 1991), and we describe the genetic structuring of the population that results from this variation, and its impact upon local competition for resources. In particular, we examine how relatedness and resource competition arise as a consequence of these demographic factors, how they become intertwined or decoupled within patches and across whole populations, and how they interact in mediating the evolution of helping and harming behaviors that are either obligate or facultatively adjusted to the availability of resources.

Table 1. A summary of model notation.

Symbol	Meaning
H	High-quality patch
L	Low-quality patch
O	Obligate social behavior
T	Corresponding to Taylor's (1992) analysis
P	Primary recipient
S	Secondary recipient
A_X	Potential for helping in condition X
α	Probability that a high-quality patch remains a high-quality patch
β	Probability that a low-quality patch remains a low-quality patch
c_X	Class-reproductive value in condition X
F_X	Fecundity of a breeding female in condition X
$\tilde{h}$	Probability that an individual chosen at random is native to the patch
h_X	Probability of philopatry in condition X
m	Migration rate
n	Patch size
π_X	History of resource quality in condition X
p	Frequency of high-quality patches in the population
r_X	"Others-only" relatedness of an individual in condition X
R_X	"Whole-group" relatedness of an individual in condition X
s	Quality ratio
τ	Temporal coefficient of correlation
v_X	Individual reproductive value of an individual in condition X
x	Level of helping of a focal actor
y	Average level of the neighbor's social behavior
z^*	Candidate evolutionary stable strategy for the social behavior

Model and Analysis

MODEL

We assume an infinite island model (Wright 1931), with social interactions among patchmates (Taylor 1992a). Each patch contains n haploid, asexual individuals. We consider two types of patches: high-quality patches that have high resource availability, and low-quality patches that have low resource availability. Individuals in high-quality patches each produce a large number $F_H = f(x, y)$ of offspring, and individuals in low-quality patches each produce a large number $F_L = s\,f(x, y)$ of offspring, where x is the individual's investment in the social behavior, y is the average investment among that individual's social partners, and $0 \leq s \leq 1$. Following reproduction, all adults die, and each surviving offspring disperses with probability m to a random patch in the population, or else stays in the native patch with probability $1 - m$. Subsequent to dispersal, n offspring are chosen at random to become reproductively mature in each patch, with the remainder dying. Finally, patches may undergo changes in their resource availability: high-quality patches remain high quality with probability α or else become low quality with probability $1 - \alpha$; low-quality patches remain low quality with probability β, or else become high quality with probability $1 - \beta$. Model notation is summarized in Table 1.

HELPING AND HARMING

We classify social behaviors according to their impact upon fecundity. Specifically, the fecundity cost of the behavior is given by $-C \equiv \partial f/\partial x$ and the fecundity benefit of the behavior is given by $B \equiv \partial f/\partial y$, and we define helping behaviors as those involving $B > 0$ and harming behaviors as those involving $B < 0$. We employ the Taylor–Frank neighbor-modulated fitness approach to kin selection analysis (Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al. 2007) to determine the direction of selection acting upon the social trait, assuming that the population is at ecological equilibrium (see Appendices A–E for details). The condition for the evolution of obligate helping and harming is given by

$$\begin{aligned} &c_{\mathrm{H}}(-C + r_{\mathrm{P|H}}B - (B - C)r_{\mathrm{S|H}}\upsilon_{\mathrm{S|H}}) \\ &\quad + c_{\mathrm{L}}(-C + r_{\mathrm{P|L}}B - (B - C)r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}}) > 0, \end{aligned} \tag{1}$$

where c_{H} and c_{L} denote the class-reproductive values for individuals in high-quality and low-quality patches, respectively (Fisher 1930; Taylor 1990, 1996; Grafen 2006); $r_{\mathrm{P|H}}$ and $r_{\mathrm{P|L}}$ denote the relatedness of the actor to her patchmates (the "primary" recipients of West and Gardner 2010), for actors in high-quality and low-quality patches, respectively; $r_{\mathrm{S|H}}$ and $r_{\mathrm{S|L}}$ denote the relatedness of the actor to those individuals who will compete for resources with the extra offspring produced in the actor's patch (the "secondary" recipients of West and Gardner 2010), for actors in high-quality and low-quality patches, respectively; and $\upsilon_{\mathrm{S|H}}$ and $\upsilon_{\mathrm{S|L}}$ denote the individual reproductive values of the secondary recipients, expressed relative to that of the actor and her patchmates, for actors in high-quality and low-quality patches, respectively (see Appendices C and E for details).

The conditions for the evolution of facultative helping and harming are given by

$$-C + r_{\mathrm{P|H}}\,B - (B - C)\,r_{\mathrm{S|H}}\,\upsilon_{\mathrm{S|H}} > 0, \tag{2}$$

and

$$-C + r_{\mathrm{P|L}}\,B - (B - C)\,r_{\mathrm{S|L}}\,\upsilon_{\mathrm{S|L}} > 0, \tag{3}$$

for individuals in high-quality patches and individuals in low-quality patches, respectively (see Appendix D for details).

Thus, an increase in investment into the social behavior impacts upon the actor's inclusive fitness in three ways. First, she suffers a personal cost C. Second, her primary recipients receive a benefit B, and this is weighted by the genetic relatedness r_{P} of the actor to these individuals. Third, the net increase $B - C$ in fecundity leads to a decrease of $B - C$ in the survival of the secondary recipients, and this is weighted by the genetic relatedness r_{S} of the actor to these individuals, and their relative reproductive value υ_{S}. Note that this third term in inequality (2) and (3) can be rearranged in the form $\tilde{h}(B - C)\ \tilde{h}\upsilon\ R_{\mathrm{S}}$, where $\tilde{h}$ is the probability that an offspring chosen at random after dispersal is native to the patch, this can be interpreted as follows: of the ($B - C$) additional offspring created due to the social behavior, a fraction $\tilde{h}$ stays in the local patch and wins a breeding site, displacing other native offspring which have on average a relatedness R and an expected relative reproductive value υ_{S} to the actor. The product of relatedness and relative reproductive value is sometimes termed "life-for-life" relatedness (Hamilton 1972), and this describes how well the recipient transmits copies of the actor's genes to future generations, relative to the actor's ability to do this herself (cf. Williams 1996, p. 181).

Setting the LHS of each of these inequalities (1)–(3) equal to zero, we obtain the conditions for the actor to "break even," neither increasing nor decreasing her inclusive fitness. Rearranging these conditions into the form $C/B = A$, the quantity A represents the potential for helping (Gardner 2010) and its additive inverse $-A$ represents the potential for harming. If $A > 0$ there is potential for helping to be favored, and if $A < 0$ there is potential for harming to be favored, provided the cost is sufficiently small. From inequality (1), the potential for obligate helping is

$$A_0 = \frac{c_{\mathrm{H}}(r_{\mathrm{P|H}} - r_{\mathrm{S|H}}\upsilon_{\mathrm{S|H}}) + c_{\mathrm{L}}(r_{\mathrm{P|L}} - r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}})}{c_{\mathrm{H}}(1 - r_{\mathrm{S|H}}\upsilon_{\mathrm{S|H}}) + c_{\mathrm{L}}(1 - r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}})}. \tag{4}$$

From inequalities (2) and (3), the potentials for facultative helping are given by

$$A_{\mathrm{H}} = \frac{r_{\mathrm{P|H}} - r_{\mathrm{S|H}}\upsilon_{\mathrm{S|H}}}{1 - r_{\mathrm{S|H}}\upsilon_{\mathrm{S|H}}} \tag{5}$$

and

$$A_{\mathrm{L}} = \frac{r_{\mathrm{P|L}} - r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}}}{1 - r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}}}, \tag{6}$$

for actors in high-quality and low-quality patches, respectively.

Results and Discussion

SPATIAL HETEROGENEITY

We first consider populations in which resource availability varies across patches within generations but not across generations within patches (i.e., spatial heterogeneity only). We derive the following results:

Result 1

Spatial heterogeneity has no impact on the potential for obligate helping and harming, which is zero ($A_{\mathrm{O}} = 0$) over the whole range of parameter values (see Appendix E for details). This extends Taylor's (1992a) cancellation result for homogeneous populations to obligate social behaviors in spatially heterogeneous populations.

Result 2

Spatial heterogeneity has no impact on the potential for facultative helping and harming, which is zero ($A_{\mathrm{H}} = A_{\mathrm{L}} = 0$) over the whole range of parameter values (see Appendix E for details). This extends Result 1 to facultative social behaviors.

Why does Taylor's (1992a) result for homogenous populations and obligate helping and harming extend to spatially heterogeneous populations with potentially facultative helping and harming? To understand this, we first consider the potential for facultative helping in high-quality patches, which is given by equation (5), that is, $A_H = (r_{P|H} - r_{S|H} v_{S|H})/(1 - r_{S|H} v_{S|H})$. Note that the relatedness of the actor to her primary recipients $r_{P|H}$ is equal to the product of philopatry $h_H = (1 - m)^2/(1 - m + m(p + (1 - p)s))^2$ for a high-quality patch—where p is the frequency of high-quality patches at equilibrium (see Appendix E for details)—and whole-group relatedness (Pepper 2000) R_H for a high-quality patch, because in the absence of temporal heterogeneity the actor's patch was of high quality in the previous generation. Also, the relatedness of the actor to her secondary recipients $r_{S|H}$ is equal to the product of philopatry h_H for a high-quality patch and whole-group relatedness R_H for a high-quality patch, because in the absence of temporal heterogeneity, the actor's patch remains high quality in the next generation. Moreover, the reproductive value of a secondary recipient—that is, an adult in the actor's patch in the next generation—is equal to the reproductive value of the actor, that is, $v_{S|H} = 1$. This is because both individuals are inhabitants of high-quality patches.

Hence, as in Taylor (1992a), the actor places equal value upon her primary and secondary recipients: $r_{P|H} = r_{S|H} v_{S|H}$ (Fig. 1), and consequently the potential for facultative helping is zero, $A_H = 0$ (see Appendix E for details). The same is true for facultative helping in low-quality patches: $h_L = ((1 - m)s)^2/((1 - m)s + m(p + (1 - p)s))^2$ and $v_{S|L} = 1$, hence $r_{P|L} = r_{S|L} v_{S|L}$, and $A_L = 0$ (Fig. 1; see Appendix E for details). As a consequence of this cancellation effect in both high-quality and low-quality patches, the potential for obligate helping is also zero, $A_O = 0$ (see Appendix for details). Since the potential for harming is given by the additive inverse of the potential for helping—that is, $-A$—the above cancellation also applies to the evolution of obligate and facultative harming behaviors.

TEMPORAL HETEROGENEITY

We next consider populations in which resource availability varies across generations within patches but not across patches within generations (i.e., temporal heterogeneity only). We assume that all patches become high quality with probability p, or become low quality with probability $1 - p$. We derive the following results:

Result 3

Temporal heterogeneity has no impact on the potential for obligate helping and harming, which is zero ($A_O = 0$) over the whole range of parameter values (see Appendix H for details). This extends Taylor's (1992a) cancellation result for homogeneous populations to obligate social behaviors in temporally heterogeneous populations.

Result 4

Temporal heterogeneity has no impact on the potential for facultative helping and harming, which is zero ($A_H = A_L = 0$) over the whole range of parameter values (see Appendix H for details). This extends Result 3 to facultative social behaviors.

Owing to the absence of spatial heterogeneity, the probability of philopatry is $h_H = h_L = h = (1 - m)^2$ for both high-quality and low-quality patches, whole-group relatedness is $R_H = R_L = R = 1/(n - (n - 1)(1 - m)^2)$ for both high-quality and low-quality patches, and the relative reproductive value of secondary recipients is $v_{S|H} = v_{S|L} = v_S = 1$ for actors in both high-quality and low-quality patches, that is, the same as in Taylor's (1992) model. Hence, the actor places equal value upon her primary and secondary recipients: $r_{P|H} = r_{S|H} v_{S|H} = r_{P|L} = r_{S|L} v_{S|L}$ (Fig. 2), and consequently the potential for helping is zero, $A_H = A_L = A_O = 0$ (see Appendix H for details). Again, a parallel argument can be made for the potential for harming, $-A$.

SPATIAL AND TEMPORAL HETEROGENEITY

We next consider populations in which resource availability varies both across patches within generations and across generations within patches (i.e., both spatial and temporal heterogeneity). We derive the following results:

Result 5

Spatial and temporal heterogeneity has no impact on the potential for obligate helping and harming, which is zero ($A_O = 0$) over the whole range of parameter values (see Appendix G for details). This extends Taylor's (1992a) cancellation result to obligate social behaviors in spatially and temporally heterogeneous populations.

Result 6

Spatial and temporal heterogeneity has an impact on the potential for facultative helping and harming, which may be nonzero ($A_H \neq 0$ and $A_L \neq 0$) depending upon parameter values (see Appendices C and E for details). Thus, selection may favor helping and harming that is facultatively adjusted according to the quality of the actor's patch, in spatially and temporally heterogeneous populations.

Why does Taylor's (1992a) result for homogenous populations and obligate helping and harming collapse when we consider populations that are both spatially and temporally heterogeneous with facultative helping and harming? Focusing our attention upon facultative helping in high-quality patches, we find that the potential for this behavior to be favored by natural selection is given by equation (5), that is, $A_H = (r_{P|H} - r_{S|H} v_{S|H})/(1 - r_{S|H} v_{S|H})$. If resource availability is heterogeneous through space and time, then the actor's high-quality patch may have been either high or low quality in the previous generation, and so her

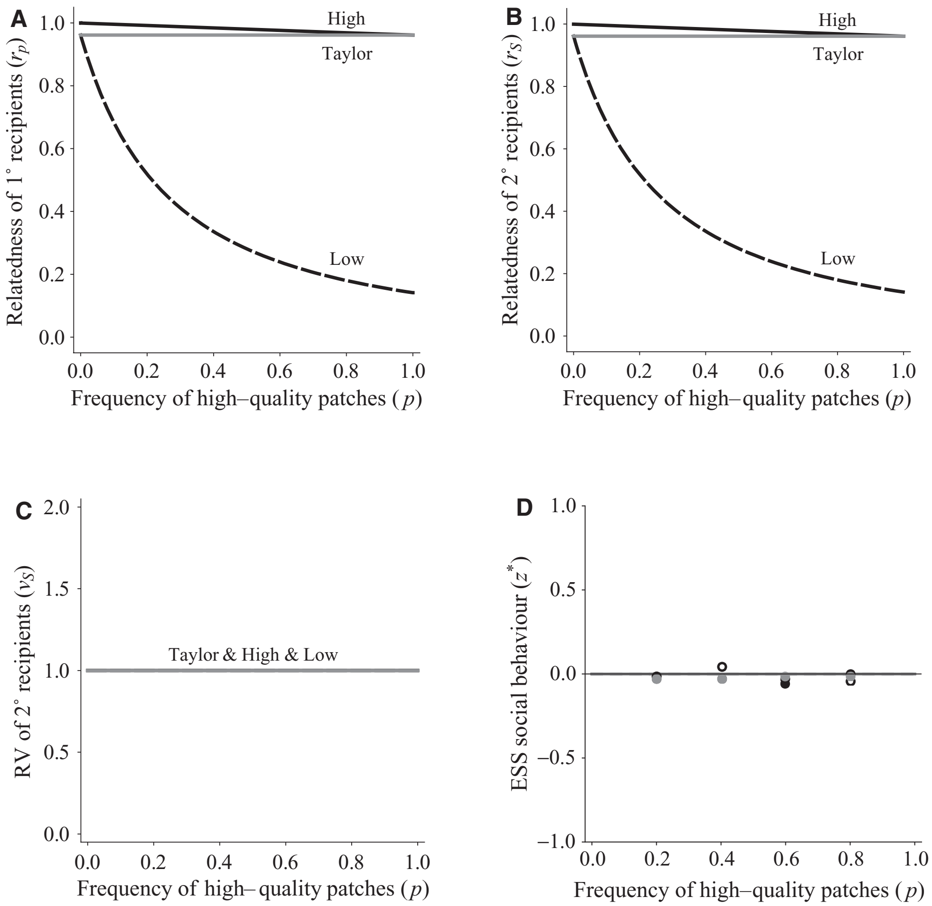

Figure 1. Impact of spatial heterogeneity upon relatedness, reproductive value, and the evolution of helping and harming. (A) The relatedness of primary recipients (r_P) as a function of the frequency of high-quality patches (p) is shown for high-quality patches ($r_{P|H}$; solid line), low-quality patches ($r_{P|L}$; dashed line), and Taylor's (1992) reference model ($r_{P|T}$; gray line). Relatedness of primary recipients is higher in high-quality patches owing to higher philopatry, and lower in low-quality patches owing to lower philopatry, relative to the reference model ($r_{P|L} < r_{P|T} < r_{P|H}$). (B) The relatedness of secondary recipients (r_S) as a function of the frequency of high-quality patches (p). Relatedness of secondary recipients is higher in high-quality patches owing to higher philopatry, and lower in low-quality patches owing to lower philopatry, relative to the reference model ($r_{S|L} < r_{S|T} < r_{S|H}$). Importantly, primary and secondary recipients are equally related ($r_{P|H} = r_{S|H}$, $r_{P|L} = r_{S|L}$). (C) The relative reproductive value of secondary recipients (v_S) is constant with respect to the frequency of high-quality patches (p), and is equal to that of the reference model ($v_{S|H} = v_{S|L} = v_{S|T} = 1$). (D) An illustration of how the equilibrium level of helping or harming depends upon the frequency of high-quality patches (p; see Appendix F for details). Analytical results (lines) and illustrative simulation data (circles) are shown for obligate behavior (gray line, solid gray circles), facultative behavior in low-quality patches (dashed line, open circles), and facultative behavior in high-quality patches (black line, solid black circles). In all panels, we assume $n = 2$, $s = 0.01$, $m = 0.01$, and $\tau = 1.00$.

relatedness to the primary recipients $r_{\mathrm{P|H}}$ is a weighted average of the product of philopatry $h_{\mathrm{H}} = (1 - m)^2/(1 - m + m(p + (1 - p)s))^2$ and whole-group relatedness R_{H} for high-quality patches and the product of philopatry $h_{\mathrm{L}} = ((1 - m)s)^2/((1 - m)s + m(p + (1 - p)s))^2$ and whole-group relatedness R_{L} for low-quality patches (see Appendix E for details). In contrast, her relatedness $r_{\mathrm{S|H}}$ to the secondary recipients is the product of the probability of philopatry h_{H} and whole-group relatedness R_{H} for her high-quality patch. Because we have $h_{\mathrm{H}} \geq h_{\mathrm{L}}$ we have that $r_{\mathrm{P|H}} \leq r_{\mathrm{S|H}}$ (see Appendix E for details). Moreover, the reproductive value of her secondary recipients—that is, adults in her patch in the next generation—is not equal to her own reproductive value. Although she has the reproductive value of an individual in a high-quality patch, theirs is a weighted average of the reproductive value of an individual in a high-quality patch and that of an individual in a low-quality patch. Hence, $v_{\mathrm{S|H}} \leq 1$ (see Appendix C for details).

Consequently, the actor may place different values upon her primary and secondary recipients: $r_{\mathrm{P|H}} \neq r_{\mathrm{S|H}} v_{\mathrm{S|H}}$ (Fig. 3; see Appendix for details). Hence, the potential for facultative helping in high-quality patches may be nonzero, $A_{\mathrm{H}} \neq 0$. The same is true for facultative helping in low-quality patches: $r_{\mathrm{P|L}} \geq r_{\mathrm{S|L}}$

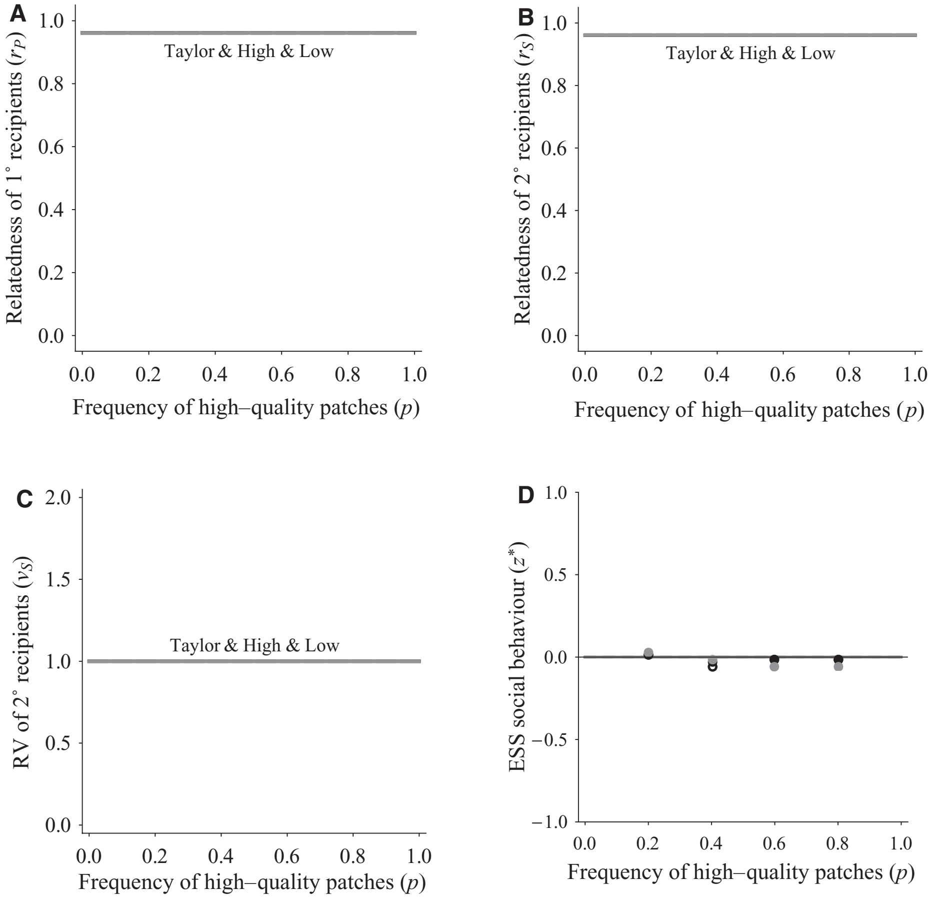

Figure 2. Impact of temporal heterogeneity upon relatedness, reproductive value, and the evolution of helping and harming. (A) The relatedness of primary recipients (r_P) as a function of the frequency of high-quality patches (p) is shown for high-quality patches ($r_{P|H}$; solid line), low-quality patches ($r_{P|L}$; dashed line), and Taylor's (1992) reference model ($r_{P|T}$; gray line). Relatedness of primary recipients in high-quality and low-quality patches is equal to that for the reference model ($r_{P|H} = r_{P|L} = r_{P|T}$). (B) The relatedness of secondary recipients (r_S) as a function of the frequency of high-quality patches (p). Relatedness of secondary recipients in high-quality and low-quality patches is equal to that for the reference model ($r_{S|H} = r_{S|L} = r_{S|T}$). Importantly, primary and secondary recipients are equally related ($r_{P|H} = r_{S|H} = r_{P|L} = r_{S|L}$). (C) The relative reproductive value of secondary recipients (v_S) is constant with respect to the frequency of high-quality patches (p), and is equal to that of the reference model ($v_{S|H} = v_{S|L} = v_{S|T} = 1$). (D) An illustration of how the equilibrium level of helping or harming depends upon the frequency of high-quality patches (p; see Appendix F for details). Analytical results (lines) and illustrative simulation data (circles) are shown for obligate behavior (gray line, solid gray circles), facultative behavior in low-quality patches (dashed line, open circles), and facultative behavior in high-quality patches (black line, solid black circles). In all panels, we assume $n = 2$, $s = 0.01$, $m = 0.01$.

and $v_{S|L} \geq 1$, so $r_{P|L} \neq r_{S|L} v_{S|L}$ and $A_L \neq 0$ (Fig. 3; see Appendices C and E for details). We find that there is potential for helping in high-quality patches and potential for harming in low-quality patches ($A_H > 0$, $A_L < 0$) when high-quality patches are relatively rare (low p), and potential for harming in high-quality patches and potential for helping in low-quality patches ($A_H < 0$, $A_L > 0$) when high-quality patches are relatively common (high p; Fig. 4). For example, in high-quality patches, the asymmetry in relatedness ($r_{P|H} < r_{S|H}$) favors harming, as the actor values secondary recipients more than primary recipients. By contrast, the asymmetry in reproductive value ($v_{S|H} < 1$) favors helping, as the actor values primary recipients more than secondary recipients. The asymmetry in relatedness is largest when the frequency of high-quality patches (p) is intermediate, whereas the asymmetry in reproductive value is largest when the frequency of high-quality patches (p) is low. Thus, helping is more likely to evolve when high-quality patches are relatively rare (Fig. 4).

In addition, as the environment becomes more staid (temporal correlation increases) the asymmetry in reproductive value becomes more important than the asymmetry in relatedness. As a result, staid environments are favorable to the evolution of facultative helping in high-quality patches and to the evolution of facultative harming in low-quality patches. By contrast, as the population becomes more mixed (i.e., migration

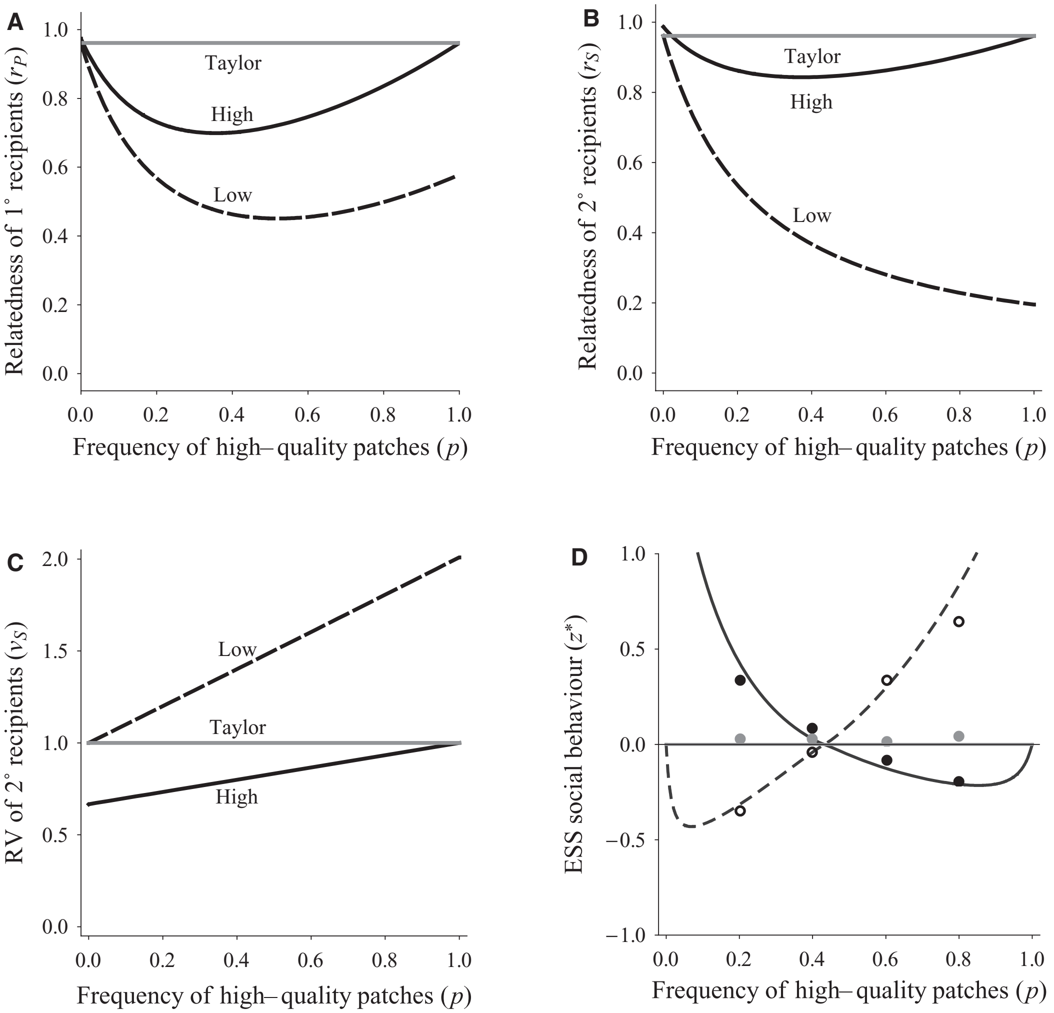

Figure 3. Impact of spatial and temporal heterogeneity upon relatedness, and reproductive value and the evolution of helping and harming. (A) The relatedness of primary recipients (r_P) as a function of the frequency of high-quality patches (p) is shown for high-quality patches ($r_{P|H}$; solid line), low-quality patches ($r_{P|L}$; dashed line), and Taylor's (1992) reference model ($r_{P|T}$; gray line). Relatedness of primary recipients may be higher or lower in high-quality patches, relative to the reference model, and is lower in low-quality patches, relative to the reference model ($r_{P|L} < r_{P|H}, r_{P|T}$). (B) The relatedness of secondary recipients (r_S) as a function of the frequency of high-quality patches (p). Relatedness of secondary recipients may be higher or lower in high-quality patches, relative to the reference model, and is lower in low-quality patches, relative to the reference model ($r_{S|L} < r_{S|H}, r_{S|T}$). Importantly, primary and secondary recipients may be unequally related ($r_{P|H} \neq r_{S|H}, r_{P|L} \neq r_{S|L}$). (C) The relative reproductive value of secondary recipients (v_S) as a function of patch frequency (p). In high-quality patches, the relative reproductive value of secondary recipients is less than or equal to that of the reference model; and in low-quality patches, the relative reproductive value is greater than or equal to that of the reference model ($v_{S|H} \leq v_{S|T} \leq v_{S|L}$). (D) An illustration of how the equilibrium level of helping or harming depends upon the frequency of high-quality patches (p; see Appendix F for details). Analytical results (lines) and illustrative simulation data (circles) are shown for obligate behavior (gray line, solid gray circles), facultative behavior in low-quality patches (dashed line, open circles), and facultative behavior in high-quality patches (black line, solid black circles). In all panels, numerical examples are given for $n = 2$, $s = 0.01$, $m = 0.01$, and $\tau = 0.10$.

rate increases), the asymmetry in relatedness becomes more important than the asymmetry in reproductive value. As a result, mixed populations are favorable to the evolution of facultative harming in high-quality patches and to the evolution of facultative helping in low-quality patches (Fig. 4).

However, although actors in high-quality patches place unequal value upon their primary and secondary recipients, and actors in low-quality patches place unequal value upon their primary and secondary recipients, we find that actors place equal value upon their primary and secondary recipients when we take an average over actors in high-quality and low-quality patches. As the ecological dynamics is determined by a time-homogeneous Markov chain, the transition probabilities that mediate the value of primary recipients are identical to the transition probabilities

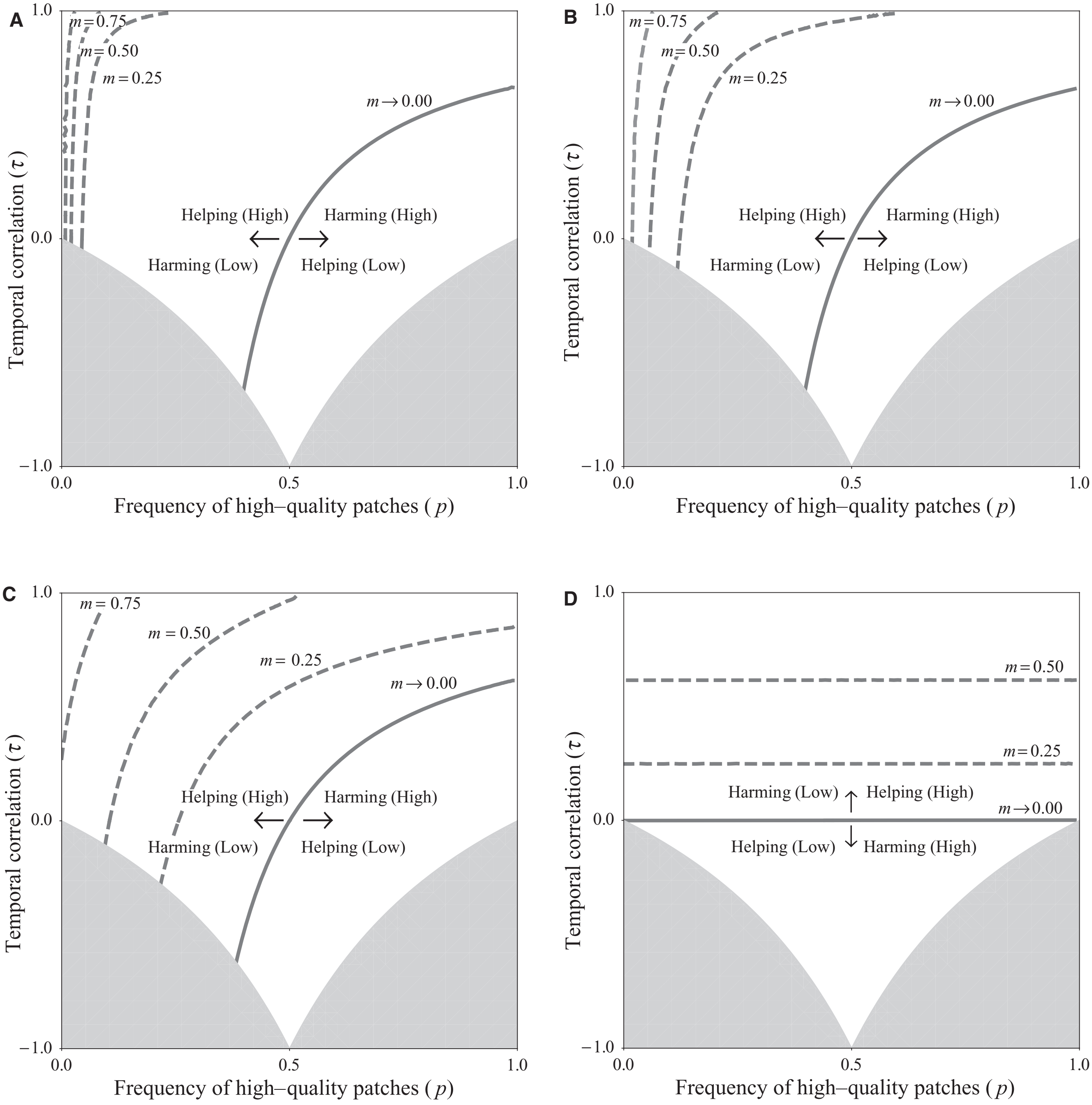

Figure 4. Impact of spatial and temporal heterogeneity upon the potential for facultative helping and harming. The continuous line represents a null potential for helping and harming ($A_H = A_L = 0$) for varying migration rates ($m \to 0.00$, $m = 0.25$, 0.50, and 0.75). (A, B, C) When high-quality patches are relatively rare (low p), there is potential for helping in high-quality patches ($A_H > 0$) and potential for harming in low-quality patches ($A_L < 0$). When high-quality patches are relatively common (high p), there is potential for harming in high-quality patches ($A_H < 0$) and potential for helping in low-quality patches ($A_L > 0$). (D) As the asymmetry in fecundity becomes negligible ($s \to 1$), the regions where helping or harming are favored become invariant in relation to the frequency of high-quality patches (p). Note that, in this case, selection is vanishing weak, and when $s = 1$ it vanishes altogether, recovering Taylor's (1992) result. The shaded region represents impossible parameter combinations (see Appendix A for details). Numerical examples are given for $n = 2$, (A) $s = 0.001$, (B) $s = 0.01$, (C) $s = 0.1$, and (D) $s \to 1$.

that mediate the value of secondary recipients (see Appendix G for details). Hence, although there is nonzero potential for facultative helping and harming in high-quality and low-quality patches ($A_{\mathrm{H}} \neq 0, A_{\mathrm{L}} \neq 0$), there is zero potential for obligate helping and harming ($A_{\mathrm{O}} = 0$) in populations characterized by both spatial and temporal heterogeneity.

Conclusions

Population viscosity has been suggested to represent a very general mechanism by which kin selection can drive the evolution of cooperation at all levels of biological organization. However, the seminal result of Taylor (1992a) reveals that, in the simplest scenario of a homogeneous, inelastic island model of population structure, the cooperation-promoting effects of increased genetic relatedness exactly cancel with the cooperation-inhibiting effects of increased kin competition, so that population viscosity has no net effect on the evolution of cooperation. Here, we have shown that this result extends to heterogeneous populations, where certain patches have high resource availability and their residents enjoy high fecundity, and other patches have low resource availability and their residents suffer low fecundity. The potential for obligate helping—and harming—is zero irrespective of the degree of population viscosity, in populations characterized by spatial and/or temporal heterogeneity in resource availability. Only when social behavior is facultatively adjusted according to the actor's patch type and there is both spatial and temporal heterogeneity in resource availability does a nonzero potential for helping or harming arise.

Previous work on social evolution in unsaturated populations (e.g., Alizon and Taylor 2008; Wild et al. 2009; Lion and Gandon 2010) is superficially similar to the present saturated-population analysis, because empty breeding sites can be thought of as a type of resource that varies in its availability between patches. However, empty sites promote helping by easing competition for resources, owing to a feedback from the social behavior to the number of breeders per patch, and need not impact at all on the genetic relatedness of social partners (Wild et al. 2009; but see Alizon and Taylor 2008 and Lion and Gandon 2010). In contrast, the present model requires that each breeding spot is won at the expense of some other individual's chance of reproductive success, and helping behaviors may instead evolve owing to the way in which resource heterogeneity impacts upon the genetic structure of populations. Moreover, although the availability of empty sites in such models depends upon the social behavior exhibited by a patch's residents, the present model has assumed that resource availability is determined independently of the genetic strategies of the residents. The possibility of incorporating feedback, such that the probability of a patch transitioning from low to high quality depends upon the social behavior of its residents, represents an interesting avenue for future exploration.

Empirical data on the relationship between spatiotemporal heterogeneity in resource availability and social behavior are limited. Recent comparative studies have considered the relationship between temporal heterogeneity and cooperative breeding in birds, finding that greater seasonality is associated with a greater degree of sociality (Rubenstein and Lovette 2007; Cockburn and Russell 2011; Jetz and Rubenstein 2011). We suggest that future comparative studies should look for an interaction between spatial and temporal heterogeneity in resource availability. Moreover, there is ample scope for experimentally testing the predictions of our model using experimental evolution methods. The island model of population structure is very amenable to implementation in laboratory studies of microbial social behavior (Griffin et al. 2004; Kümmerli et al. 2009), and this experimental methodology could readily be extended to incorporate patch heterogeneity in space and time. More generally, we emphasize the potential for kin selection theory to explain variation within as well as between populations, and we suggest that future empirical studies consider the relationship between local environments and local sociality in addition to the relationship between global environments and whole-population measures of sociality.

Finally, we note some implications for the response of organisms' social behaviors to climate change and other relatively fast, anthropogenic impacts upon the natural environment. The main results of our analysis have concerned populations that are at ecological equilibrium, and adaptive evolutionary change may be relatively slow to track ecological upheavals. Hence, we predict that rapid degradation of the environment may be accompanied by maladaptive facultative changes in organisms' social behaviors. For example, over much of the range of parameter values, our model predicts that facultative harming behavior is favored in low-quality patches. Consequently, habitat deterioration might lead to an increase in mutually costly harming behaviors within ecologically threatened species, further contributing to their extinction risk. Such indirect consequences of habitat deterioration may pose an extra obstacle for conservation efforts.

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Appendix A

PATCH DYNAMICS

Given the assumptions described in the main text, transitions in patch quality can be represented as a matrix as follows:

$$\mathrm{P} = \begin{pmatrix} \alpha & 1-\beta \\ 1-\alpha & \beta \end{pmatrix}. \tag{A1}$$

At equilibrium, the fraction of patches that are in the high-quality state is

$$p = \frac{1-\beta}{2-(\alpha+\beta)}, \tag{A2}$$

assuming $\alpha, \beta < 1$. Denoting patch quality at time t by T_t (equal to 1 for H and equal to 0 for L), the correlation coefficient describing the statistical association between a patch's quality at times t and $t+1$ is $\tau = \mathrm{cov}(T_t, T_{t+1})/\surd(\mathrm{var}(T_t)\mathrm{var}(T_{t+1}))$, or

$$\tau = \alpha - (1-\beta), \tag{A3}$$

at ecological equilibrium. Note that when temporal correlation is negative ($\tau < 0$), it is no longer independent of the frequency of high-quality patches (p). For example, when $\tau = -1$, the frequency of high-quality patches must be equal to ½. Otherwise we would not have a stable distribution of high-quality patches over generations. Note also that this environmental change may be regarded as analogous to an extra phase of dispersal (cf Débarre and Gandon 2011), albeit a form of "budding" dispersal (Gardner and West 2006) where all patchmates "travel" together to the new environment.

Appendix B

REPRODUCTIVE SUCCESS

In a high-quality patch, each offspring of a focal breeding female stays in the native patch with probability $1-m$. The expected number of her non-dispersing offspring that successfully win a breeding site in the patch is given by $(1-m)\, E^{\bullet}_{\mathrm{H}\to\mathrm{H}}$, where

$$E^{\bullet}_{\mathrm{H}\to\mathrm{H}} = \frac{f(x_{\mathrm{H}}, y_{\mathrm{H}})}{(1-m)f(Y_H, Y_H) + m(pf(z_{\mathrm{H}}, z_{\mathrm{H}}) + (1-p)f(z_{\mathrm{L}}, z_{\mathrm{L}})s)}, \tag{A4}$$

where Y_{H} is the average trait value in the focal patch, and z_{H} and z_{L} are the population average trait values in high- and low-quality patches, respectively. Each offspring of the same focal mother migrates to a high-quality patch with probability mp. The expected number of her offspring that disperse to a high-quality patch and successfully win a breeding site in the patch is given by $m\,p\, E^{\circ}_{\mathrm{H}\to\mathrm{H}}$, where:

$$E^{\circ}_{\mathrm{H}\to\mathrm{H}} = \frac{f(x_{\mathrm{H}}, y_{\mathrm{H}})}{(1-m)f(z_{\mathrm{H}}, z_{\mathrm{H}}) + m(pf(z_{\mathrm{H}}, z_{\mathrm{H}}) + (1-p)f(z_{\mathrm{L}}, z_{\mathrm{L}})s)}. \tag{A5}$$

Finally, each offspring of the same focal mother migrates to a low-quality patch with probability $m(1-p)$. The expected number of her offspring that disperse to a low-quality patch and successfully win a breeding site in the patch is given by $m\,(1-p)\, E^{\circ}_{\mathrm{H}\to\mathrm{L}}$, where:

$$E^{\circ}_{\mathrm{H}\to\mathrm{L}} = \frac{f(x_{\mathrm{H}}, y_{\mathrm{H}})}{(1-m)f(z_{\mathrm{L}}, z_{\mathrm{L}})s + m(pf(z_{\mathrm{H}}, z_{\mathrm{H}}) + (1-p)f(z_{\mathrm{L}}, z_{\mathrm{L}})s)}. \tag{A6}$$

We also derive corresponding expressions for a focal mother in a low-quality patch. These expressions define a matrix $\mathbf{E}$ which is given by

$$\mathbf{E} = \begin{pmatrix} E_{\mathrm{H}\to\mathrm{H}} & E_{\mathrm{L}\to\mathrm{H}} \\ E_{\mathrm{H}\to\mathrm{L}} & E_{\mathrm{L}\to\mathrm{L}} \end{pmatrix} = \begin{pmatrix} (1-m)E^{\bullet}_{\mathrm{H}\to\mathrm{H}} + mpE^{\circ}_{\mathrm{H}\to\mathrm{H}} & mpE^{\circ}_{\mathrm{L}\to\mathrm{H}} \\ m(1-p)E^{\circ}_{H\to L} & (1-m)E^{\bullet}_{L\to L} + (1-p)E^{\circ}_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}. \tag{A7}$$

Matrices $\mathbf{P}$ and $\mathbf{E}$ combine to define the reproductive success matrix, which is given by

$$\mathbf{w} = \begin{pmatrix} w_{\mathrm{H}\to\mathrm{H}} & w_{\mathrm{L}\to\mathrm{H}} \\ w_{\mathrm{H}\to\mathrm{L}} & w_{\mathrm{L}\to\mathrm{L}} \end{pmatrix} = \mathbf{PE}. \tag{A8}$$

In the context of class-structured populations, the reproductive success of an adult breeding female must take into account the relative contribution of individuals in different classes for gene frequency change (Fisher 1930; Price 1970; Taylor 1990; Grafen 2006). Thus, the reproductive success of a focal individual in a high-quality patch is given by

$$W_{\mathrm{H}} = w_{\mathrm{H}\to\mathrm{H}} + \frac{v_{\mathrm{L}}}{v_{\mathrm{H}}} w_{\mathrm{H}\to\mathrm{L}}, \tag{A9}$$

where v_{H} and v_{L} are the reproductive value of individuals in high-quality and low-quality patches, respectively. Likewise, the reproductive success of a focal adult breeding female in a low-quality patch is

$$W_{\mathrm{L}} = w_{\mathrm{L}\to\mathrm{L}} + \frac{v_{\mathrm{H}}}{v_{\mathrm{L}}} w_{\mathrm{L}\to\mathrm{H}}. \tag{A10}$$

The expected reproductive success of an individual is given by the class-specific reproductive success (W_{H} and W_{L}), weighted by the respective class-reproductive values

(c_{H} and c_{L}), which is given by

$$W = c_{\mathrm{H}}W_{\mathrm{H}} + c_{\mathrm{L}}W_{\mathrm{L}}. \tag{A11}$$

Class-reproductive value is defined as the product of the frequency of individuals in a given patch quality-type and those individuals' reproductive value ($c_{\mathrm{H}} = pv_{\mathrm{H}}$; $c_{\mathrm{L}} = (1-p)v_{\mathrm{L}}$).

Appendix C

REPRODUCTIVE VALUE

Individuals reproductive values are given by the left-eigenvector of matrix **w** (defined in eq. A8) assuming a neutral population (Taylor 1990; Grafen 2006), and are normalized such that $c_{\mathrm{H}} + c_{\mathrm{L}} = 1$ (Taylor 1990; Taylor et al. 2007). Hence, a gene drawn at random from the population has an expected reproductive success W_{H} with probability c_{H}, and an expected reproductive success W_{L} with probability c_{L}. If there is no heterogeneity in the population (i.e., $s = 1$) then there are no differences in the reproductive values ($v_{\mathrm{H}} = v_{\mathrm{L}}$). However, as heterogeneity emerges in the population (i.e., $0 < s < 1$), it is easy to show that $v_{\mathrm{H}} > v_{\mathrm{L}}$, because individuals in high-quality patches have higher fecundity than individuals in low-quality patches. Reproductive values of secondary recipients are given by: $v_{\mathrm{S|H}} = (\alpha v_{\mathrm{H}} + (1-\alpha)v_{\mathrm{L}})/v_{\mathrm{H}}$ and $v_{\mathrm{S|L}} = (\beta v_{\mathrm{L}} + (1-\beta)v_{\mathrm{H}})/v_{\mathrm{L}}$. Note that, because $v_{\mathrm{H}} \geq v_{\mathrm{L}}$, we have $v_{\mathrm{S|H}} \leq 1$ and $v_{\mathrm{S|L}} \geq 1$.

Appendix D

HAMILTON'S RULE AND THE POTENTIAL FOR HELPING AND HARMING

To determine how selection acts on the trait of interest, we use the Taylor-Frank approach (Taylor and Frank 1996; Frank 1998; Taylor et al. 2007). We assume that the genetic variation is segregating at a locus responsible for the social behavior. We denote the genic value for a focal gene drawn from this locus by g (Price 1970; Falconer 1981). Assuming a vanishingly small genetic variation, the direction of natural selection acting on the social trait is given by

$$\frac{\mathrm{d}W}{\mathrm{d}g} = c_{\mathrm{H}}\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} + c_{\mathrm{L}}\frac{\mathrm{d}W}{\mathrm{d}g}. \tag{A12}$$

The derivative in the first term of the RHS of this equation can be expanded as follows:

$$\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} = \frac{\partial W_{\mathrm{H}}}{\partial x_{\mathrm{H}}}\frac{\mathrm{d}x_{\mathrm{H}}}{\mathrm{d}g_{\mathrm{H}}}\frac{\mathrm{d}g_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial y_{\mathrm{H}}}\frac{\mathrm{d}y_{\mathrm{H}}}{\mathrm{d}g'_{\mathrm{H}}}\frac{\mathrm{d}g'_{H}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial Y_{\mathrm{H}}}\frac{\mathrm{d}Y_{\mathrm{H}}}{\mathrm{d}G_{\mathrm{H}}}\frac{\mathrm{d}G_{\mathrm{H}}}{\mathrm{d}g}, \tag{A13}$$

where g'_{H} denotes social partners' average genetic value excluding self, and G_{H} denotes social partners' average genetic value including self, genetic values being the average of an individual's genic values. All the derivatives and partial derivatives are evaluated at $x = y = Y = z$ (Taylor and Frank 1996; Frank 1997, 1998; Taylor et al. 2007). The partial derivatives give the marginal effects of the mutant phenotypes. The correlations between the phenotype and the genetic breeding values represent the genotype-to-phenotype mapping defined as $\mathrm{d}x_{\mathrm{H}}/\mathrm{d}g_{\mathrm{H}} = \mathrm{d}y_{\mathrm{H}}/\mathrm{d}\,g'_{H} = \mathrm{d}Y_{\mathrm{H}}/\mathrm{d}G_{\mathrm{H}} = \gamma_{\mathrm{H}}$, and this is arbitrarily set to unity ($\gamma_{\mathrm{H}} = 1$). The correlations between breeding values are coefficients of consanguinity. These correlations when normalized to the actor's coefficient of consanguinity give the kin selection coefficient of relatedness (Bulmer 1994). The same expansion can be made for the direction of selection acting on low-quality patches ($\mathrm{d}W_{\mathrm{L}}/\mathrm{d}g$). Note that selection acting on the social trait not only depends upon the direction of selection ($\mathrm{d}W_{\mathrm{X}}/\mathrm{d}g$), but also upon the class-reproductive values (c_{X}). As a consequence, we may have $\mathrm{d}W_{\mathrm{X}}/\mathrm{d}g \neq 0$, but $c_{\mathrm{X}} = 0$, in which case selection acting on the trait in the context X is null.

For obligate social behaviors, the condition for the evolution of higher breeding values is given by $\mathrm{d}W/\mathrm{d}g > 0$. For facultative social behavior, the condition for the evolution of higher breeding values in high-quality patches and low-quality patches is given by $\mathrm{d}W_{\mathrm{H}}/\mathrm{d}g > 0$ and $\mathrm{d}W_{\mathrm{L}}/\mathrm{d}g > 0$, respectively. This gives the inequalities (1)–(3) in the main text.

Appendix E

RELATEDNESS

Here, we derive the kin selection coefficients of relatedness for haploid populations. We assume vanishingly small genetic variation in the population. To determine the average relatedness structure in the population, we define a recursion equation (Wright 1969; Taylor 1992a; Rousset 2004). In a neutral and equilibrium population, the relatedness of a focal offspring to her sisters is 1 with probability $1/n$. The relatedness to her native patchmates is the relatedness among their mothers, which is given by r. These are native to the focal patch with probability h, which is the probability that we choose at random two offspring that have not dispersed. Therefore among offspring, relatedness is given by

$$Q = h\left(\frac{1}{n} + \left(1 - \frac{1}{n}\right)r\right). \tag{A14}$$

A patch in a high-quality state was a high-quality patch in the previous generation with probability π_{H}, and was a low-quality patch with probability $1 - \pi_{\mathrm{H}}$. A low-quality patch was a low-quality patch in the previous generation with probability π_{L}, and a high-quality patch with probability $1 - \pi_{\mathrm{L}}$. The recursion equations are given by

$$\begin{cases} r'_{\mathrm{H}} = \pi_{\mathrm{H}} Q_{\mathrm{H}} + (1 - \pi_{\mathrm{H}}) Q_{\mathrm{L}} \\ r'_{\mathrm{H}} = (1 - \pi_{\mathrm{L}}) Q_{\mathrm{H}} + \pi_{\mathrm{L}} Q_{\mathrm{L}} \end{cases}, \tag{A15}$$

Solving the system of equation (A15) for equilibrium, $r'_{\mathrm{H}} = r_{\mathrm{H}}$ and $r'_{\mathrm{L}} = r_{\mathrm{L}}$, we get the kin selection coefficients of relatedness, which are given by

$$r_{\mathrm{H}} = \frac{\pi_{\mathrm{H}} h_{\mathrm{H}}(n - h_{\mathrm{L}}(n-1)) + (1-\pi_{\mathrm{H}}) h_{\mathrm{L}} n + (1-\pi_{\mathrm{L}})(n-1)}{(n - \pi_{\mathrm{H}} h_{\mathrm{H}}(n-1))(n - h_{\mathrm{L}}(n-1)) + (1-\pi_{\mathrm{L}}) h_{\mathrm{L}}(n-1)(n - h_{\mathrm{H}}(n-1))}, \tag{A16}$$

and

$$r_{\mathrm{L}} = \frac{h_{\mathrm{L}}(n - \pi_{\mathrm{H}} h_{\mathrm{H}}(n-1)) + (1-\pi_{\mathrm{L}})((h_{\mathrm{H}} + h_{\mathrm{L}})n - h_{\mathrm{H}} h_{\mathrm{L}}(n-1))}{(n - \pi_{\mathrm{H}} h_{\mathrm{H}}(n-1))(n - h_{\mathrm{L}}(n-1)) + (1-\pi_{\mathrm{L}}) h_{\mathrm{L}}(n-1)(n - h_{\mathrm{H}}(n-1))} \tag{A17}$$

for high-quality patches and low-quality patches, respectively. “Whole-patch” relatedness coefficients are determined by substituting these values into the expression $R = 1/n + (1 - 1/n)r$, for the corresponding patch quality types. Relatedness of primary recipients is given by $r_{\mathrm{P|H}} = r_{\mathrm{H}}$ and $r_{\mathrm{P|L}} = r_{\mathrm{L}}$ for high-quality patches and low-quality patches, respectively. Relatedness of secondary recipients is given by $r_{\mathrm{S|H}} = h_{\mathrm{H}} R_{\mathrm{H}}$ and $r_{\mathrm{S|L}} = h_{\mathrm{L}} R_{\mathrm{L}}$ for high-quality patches and low-quality patches, respectively. Let us denote the denominator of (A16) and (A17) as D. After some algebra, it is easy to show that $r_{\mathrm{P|H}} - r_{\mathrm{S|H}} = -(h_{\mathrm{H}} - h_{\mathrm{L}})n(1 - \alpha)/D$ and that $r_{\mathrm{P|L}} - r_{\mathrm{S\|L}} = (h_{\mathrm{H}} - h_{\mathrm{L}})n(1 - \alpha)/D$. Thus, because $h_{\mathrm{H}} \geq h_{\mathrm{L}}$, we have $r_{\mathrm{P|H}} \leq r_{\mathrm{S|H}}$ and $r_{\mathrm{P|L}} \geq r_{\mathrm{S|L}}$.

In spatially heterogeneous populations ($\tau = 1$), a high-quality patch was always a high-quality patch in the previous generation ($\pi_{\mathrm{H}} = 1$), and a low-quality patch was always a low-quality patch in the previous generation ($\pi_{\mathrm{L}} = 1$). Hence, the coefficients of relatedness of equations (A16) and (A17) reduce to $r_{\mathrm{H}} = h_{\mathrm{H}}/(n - (n-1)h_{\mathrm{H}})$ and $r_{\mathrm{L}} = h_{\mathrm{L}}/(n - (n-1)h_{\mathrm{L}})$. This gives the following identity between others-only and whole-group relatedness: $r_{\mathrm{H}} = h_{\mathrm{H}} R_{\mathrm{H}}$ and $r_{\mathrm{L}} = h_{\mathrm{H}} R_{\mathrm{L}}$. As a consequence in spatial heterogeneous populations, the relatedness of primary recipients is equal to the relatedness of secondary recipients (see main text). Note also that $r_{\mathrm{H}} - r_{\mathrm{L}} = (h_{\mathrm{H}} - h_{\mathrm{L}})n/((n - (n-1)h_{\mathrm{H}})(n - (n-1)h_{\mathrm{L}}))$. Since $h_{\mathrm{H}} \geq h_{\mathrm{L}}$, relatedness in high-quality patches is always larger or equal to relatedness in low-quality patches ($r_{\mathrm{H}} \geq r_{\mathrm{L}}$).

Appendix F

EVOLUTIONARILY STABLE STRATEGY AND SIMULATIONS

We wrote the code for the simulations in the language C++ (see Supporting Information). The protocol of the simulations closely follows the specifications of the model assumptions. However, instead of an infinite number of patches we now have a finite number of patches that we set to 5000. Each patch accommodates $n = 2$ breeding individuals. Thus, the total population size is 10,000. Each individual’s genotype is represented as a real number on the interval $[-1,1]$, where positive values indicate level of investment into helping and negative values indicate level of investment into harming. At initialization, all genotypes are set to 0. In every subsequent generation, we choose 10 individuals at random for mutation: we add to their genotypic value a random Gaussian-distributed quantity with mean 0 and variance 0.015. To make quantitative predictions about the candidate evolutionarily stable strategy (ESS; Maynard Smith and Price 1973) for the social behavior (z^*), one needs to specify the marginal fecundity cost and the marginal fecundity benefits. The marginal fecundity cost is assumed to decrease monotonically with the investment in the social behavior, such that $-C = \partial f(x,y)/\partial x = -k$, where k is a scaling factor. The marginal fecundity benefits are assumed to have diminishing returns with the investment in the social behavior such that $B = \partial f(x,y)/\partial y = \mathrm{sign}(y) k \eta \mu |y|^{(\mu - 1)}$, where η is a scaling factor, and μ controls the rate at which the benefits saturate with the investment in the social behavior. In the analytical model, a candidate ESS is found when the ratio between the marginal fecundity cost and the marginal fecundity benefits exactly matches the potential for helping ($C/B = A$). The candidate ESS is then given by $z^* = \mathrm{sign}(A)(\eta\mu|A|)^{1/(1-\mu)}$. In the simulation model, we let the population evolve up to 500,000 generations, and we compute the average genotypic value across all individuals in the final generation. In Figures 1–3, we provide illustrative numerical solutions for z^*, along with illustrative simulation data. Here, we assumed $\eta = 20$, $\mu = 0.1$, and $k = 0.015$.

Appendix G

CANCELLATION OF OBLIGATE SOCIAL BEHAVIOR

The life-for-life relatedness of primary recipients is $\rho_{\mathrm{P|H}} = r_{\mathrm{P|H}}$ and $\rho_{\mathrm{P|L}} = r_{\mathrm{P|L}}$ for high-quality and low-quality patches, respectively. The life-for-life relatedness of secondary recipients is $\rho_{\mathrm{S|H}} = r_{\mathrm{S|H}} v_{\mathrm{S|H}}$ and $\rho_{\mathrm{S|L}} = r_{\mathrm{S|L}} v_{\mathrm{S|L}}$. For obligate behaviors, the life-for-life coefficients of primary recipients is given by

$$\rho_{\mathrm{P}} = c_{\mathrm{H}} \rho_{\mathrm{P|H}} + c_{\mathrm{L}} \rho_{\mathrm{P|L}}, \tag{A18}$$

and the life-for-life relatedness of a secondary recipient is given by

$$\rho_{\mathrm{S}} = c_{\mathrm{H}} \rho_{\mathrm{S|H}} + c_{\mathrm{L}} \rho_{\mathrm{S|L}}. \tag{A19}$$

In Taylor’s (1992a) model, these are $\rho_{\mathrm{P}} = r = hR$, and $\rho_{\mathrm{S}} = aR$, where a is the proportion of competition that occurs locally (scale of competition; Frank 1998; Gardner and West 2006; Van Dyken 2010). As $h = a$, we have $\rho_P = \rho_S$, as actors value primary recipients as much as secondary recipients there is no selection for social behaviors. In our model, we find that same symmetry: $h_H = a_H$, and $h_L = a_L$. As the ecological dynamics is determined by a time-homogeneous Markov chain, we find additional symmetries. To see how these explain the cancellation result for obligate behavior, let us denote the different probabilities (P) in the life-for-life relatedness as follows: $P(X_{t-1} = H \mid X_t = H) = \pi_H$, $P(X_{t-1} = L \mid X_t = H) = 1 - \pi_H$, $P(X_{t-1} = L \mid X_t = L) = \pi_L$, $P(X_{t-1} = H \mid X_t = L) = 1 - \pi_L$, $P(X_{t+1} = H \mid X_t = H) = \alpha$, $P(X_{t+1} = L \mid X_t = H) = 1 - \alpha$, $P(X_{t+1} = L \mid X_t = L) = \beta$, $P(X_{t+1} = H \mid X_t = L) = 1 - \beta$, $P(X_t = H) = p$, and $P(X_t = H) = 1 - p$. Using this notation, the life-for-life coefficients of relatedness for obligate behavior can be written as follows:

$$\begin{aligned}\rho_P = {} & P(X_t = H)(P(X_{t-1} = H|X_t = H)h_H R_H v_H \\ & + P(X_{t-1} = L|X_t = H)h_L R_L v_H) \\ & + P(X_t = L)(P(X_{t-1} = H|X_t = L)h_H R_H v_L \\ & + P(X_{t-1} = L|X_t = L)h_L R_L v_L)\end{aligned}, \tag{A20}$$

and

$$\begin{aligned}\rho_S = {} & P(X_t = H)(P(X_{t+1} = H|X_t = H)h_H R_H v_H \\ & + P(X_{t+1} = L|X_t = H)h_H R_H v_L) \\ & + P(X_t = L)(P(X_{t+1} = H|X_t = L)h_L R_L v_H \\ & + P(X_{t+1} = L|X_t = L)h_L R_L v_L).\end{aligned} \tag{A21}$$

We can show that life-for-life relatedness of primary recipients is equal to the life-for-life relatedness of secondary recipients. For example, first note that from Bayes' theorem, we have that $P(X_{t-1} = L \mid X_t = H) = P(X_t = H \mid X_{t-1} = L)P(X_{t-1} = L)/P(X_t = H)$; second as the ecological dynamics is described by a time-homogeneous Markov chain, we have $P(X_t = H \mid X_{t-1} = L) = P(X_{t+1} = H \mid X_t = L)$, as a result we have $P(X_t = H)P(X_{t-1} = L \mid X_t = H) = P(X_t = L)P(X_{t+1} = H \mid X_t = L)$. This can be shown to hold for all other terms in the life-for-life coefficients of relatedness. Thus, as in Taylor's (1992a) model of obligate social behavior in homogeneous populations, we find that the average life-for-life relatedness of primary recipients is equal to the average life-for-life relatedness of secondary recipients ($\rho_P = \rho_S$). This identity prevents the evolution of social behaviors.

Appendix H

TEMPORAL HETEROGENEITY

Here, we assume that within a generation, all patches have the same quality-state, that is, the population is in the high-quality state or the population is in the low-quality state. Independently of the current state, the population becomes high quality with probability p or becomes low quality with probability $1 - p$. The matrix $\mathbf{P}$ is now defined by setting $\alpha = p$ and $\beta = 1 - p$. As before, we solve the matrix for its right-eigenvector, and this gives $u_H = p$ and $u_L = 1 - p$. However, the interpretation is now different: u_H and u_L represent the expected fraction of generations that the population, on the long run, spends on the high-quality state and on the low-quality state, respectively. We also need to determine the contribution of high-quality individuals and low-quality individuals to the future genetic composition of the population, that is, the individual reproductive values. The reproductive value of a high-quality individual is determined as follows: the fecundity of focal high-quality individual is 1; with probability p her offspring reproduce as high-quality individuals, in which case they have reproductive value v_H; with probability $1 - p$ her offspring reproduce as low-quality individual, in which case they have reproductive value v_L; hence, at equilibrium, we get the following identity: $v_H = pv_H + (1 - p)v_L$. Therefore, reproductive values are identical and can be normalized such that $v_H = v_L = 1$. Class-reproductive values are given by: $c_H = u_H\, v_H = p$ and $c_L = u_L\, v_L = 1 - p$. Because within each generation there is no heterogeneity, and because reproductive values are identical, the class-specific fitness expressions are identical (W_H and W_L), and are given by

$$W_X = \frac{(1-m)f(x_X, y_X)}{(1-m)f(Y_X, Y_X) + mf(z_X, z_X)} + \frac{mf(x_X, y_X)}{f(z_X, z_X)}. \tag{A22}$$

As before, we use the Taylor–Frank approach (Taylor and Frank 1996) to determine the direction of natural selection acting on the social traits. Relatedness coefficients are again given by equation (A15) with $\pi_H = 1 - \pi_L = p$, $\pi_L = 1 - \pi_H = 1 - p$, and $h_H = h_L = (1 - m)^2$. As a result, $r_H = r_L = r_{P|H} = r_{P|L} = hR_H = hR_L = hR = h/(n - (n - 1)h)$. Relatedness of secondary recipients is given by $r_{S|H} = r_{S|L} = hR_H = hR_L = hR$. Hence, $r_{P|H} = r_{S|H}$ and $r_{P|L} = r_{S|L}$. Specific patch dynamics are given for concreteness and clarity. However, the various measures of value—that is, relatedness and reproductive value—are independent of the particular dynamics and the correlation between population states over generations.

Supporting Information

The following supporting information is available for this article:

Supporting Text. Simulation code.

Supporting Information may be found in the online version of this article.

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Evolution of Helping and Harming in Viscous Populations When Group Size Varies

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ABSTRACT: Recent years have seen huge interest in understanding how demographic factors mediate the evolution of social behavior in viscous populations. Here we study the impact of variation in group size on the evolution of helping and harming behavior. Although variation in group size influences the degree of relatedness and the degree of competition between groupmates, we find that these effects often exactly cancel, so as to give no net impact of variation in group size on the evolution of helping and harming. Specifically, (1) obligate helping and harming are never mediated by variation in group size, (2) facultative helping and harming are not mediated by variation in group size when this variation is spatial only, (3) facultative helping and harming are mediated by variation in group size only when this variation is temporal or both spatial and temporal, and (4) when there is an effect of variation in group size, facultative helping is favored in big groups and facultative harming is favored in little groups. Moreover, we find that spatial and temporal heterogeneity in individual fecundity may interact with patch-size heterogeneity to change these predictions, promoting the evolution of harming in big patches and of helping in little patches.

Keywords: class structure, conditional behavior, inclusive fitness, kin selection, phenotypic plasticity, reproductive value.

Introduction

Explaining cooperative behavior has been a central challenge to evolutionary biologists over the past few decades (Maynard Smith and Szathmáry 1995; Hamilton 1996; West et al. 2007*a*; Bourke 2011). Natural selection favors those individuals who achieve higher relative fitness (Darwin 1859; Fisher 1930; Price 1970). But cooperative behaviors that improve the fitness of others will, all else being equal, decrease the relative fitness of the actor. This problem can be solved if the actor receives a direct fitness benefit as a consequence of her cooperative behavior. For example, the immediate cost of the actor's behavior may be compensated for later in her life by reciprocation from others (Trivers 1971). Such cooperation is termed "mutually beneficial" (West et al. 2007*b*). Alternatively, the problem can be solved if the actor receives an indirect fitness benefit, through her genetic relatives benefiting from her cooperative behavior (Hamilton 1963, 1964). Such cooperation is termed "altruistic" (Hamilton 1964; West et al. 2007*b*).

Altruistic cooperation requires that interacting individuals have genes in common. Hamilton (1964, 1971*b*) suggested three general mechanisms by which this can occur. First, individuals might be able to identify which of their social partners are genealogically close kin and preferentially direct their altruism to these individuals. Second, a "greenbeard" gene may provide its bearer the ability to directly identify which of their social partners also carry the same gene, independently of their genealogical relationship (Hamilton 1964; Dawkins 1976; Gardner and West 2010). Third, if dispersal is limited, then neighboring individuals are likely to be genealogically close kin, such that even indiscriminate cooperation will tend to occur between individuals who have genes in common. As this third mechanism does not require any discrimination mechanism, it could represent a very general principle that explains the evolution of cooperation at all levels of biological organization (Maynard Smith and Szathmáry 1995; Bourke 2011).

However, limited dispersal can also lead to intensified competition for resources among kin (Hamilton 1964; Queller 1992; West et al. 2002). In the simplest scenario of an infinite island population, the kin-competition effects of limited dispersal exactly cancel its relatedness-enhancing effects, such that population viscosity has no net impact on the evolution of indiscriminate cooperation (Taylor 1992*a*). This striking result has stimulated the development of a large body of theoretical—and, to a lesser extent, empirical—research examining what additional factors may decouple the relatedness and competition ef-

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fects of limited dispersal, so that indiscriminate helping may be favored in viscous populations. Some of these factors include population elasticity (Taylor 1992*b*), overlapping generations (Taylor and Irwin 2000; Irwin and Taylor 2001), budding dispersal (Gardner and West 2006; Lehmann et al. 2006*b*; Kümmerli et al. 2009), an organism's life cycle and timing of social behavior (Taylor 1992*a*; Lehmann and Rousset 2010), behaviors mediating patch-extinction probabilities (Lehmann et al. 2006*b*), transgenerational altruism (Lehmann 2007, 2010), dispersal-dependent social behavior (El Mouden and Gardner 2008), empty sites (Alizon and Taylor 2008), sex-biased dispersal (Johnstone and Cant 2008; Gardner 2010), reproductive skew (Johnstone 2008), age structure (Johnstone and Cant 2010), and heterogeneity in resource availability (Rodrigues and Gardner 2012).

All this work has concerned populations in which the size of viable groups is fixed. However, natural populations commonly exhibit some degree of group-size heterogeneity, and the impact of this variation has been of great interest to ecologists and evolutionary biologists (Levins 1968; McPeek and Holt 1992; Begon et al. 2006). This is important to the evolution of kin-selected traits because patch size is likely to have an impact not only on the relatedness among social partners but also on local competition, two important factors that jointly mediate the evolution of social traits in viscous populations.

Here we investigate the impact of heterogeneity in group size on the evolution of indiscriminate helping and harming in viscous populations. Specifically, we consider scenarios in which different groups have different sizes at any given time (spatial heterogeneity) and/or the same group has different sizes at different times (temporal heterogeneity), where group size is extrinsically controlled and not driven by the group's past or present social behavior. We first consider cases where fecundity is equal across all patch types, and we then relax this assumption by considering cases where fecundity depends on patch type. Moreover, we consider scenarios where individuals obligately express their social behavior independently of their patch type and scenarios where individuals may facultatively adjust their social behavior according to their patch type. We determine the impact of this heterogeneity on the genetic relatedness and intensity of competition between groupmates and examine how these interact and affect the evolution of helping and harming behaviors.

Model and Analysis

Model

We assume an infinite island population (Wright 1931) with two types of patches: big patches with n_B breeding sites and little patches with n_L breeding sites (without loss of generality, we assume $n_B \geq n_L$). We consider that individuals are haploid and asexual, that each individual breeding in a big patch has a very high fecundity $F_B = f(x, y)$, and that each individual breeding in a little patch has a very high fecundity $F_L = \sigma f(x, y)$, where x is the individual's investment in the social behavior, y is the average level of investment among the individual's groupmates, and $0 < \sigma < \infty$ is the "reproductive factor" describing fecundity differences between individuals breeding in little versus big patches. Note that if $\sigma = 1$, individuals breeding in little patches have the same fecundity as those breeding in big patches; if $\sigma < 1$, individuals breeding in little patches have less fecundity than those breeding in big patches; and if $\sigma > 1$, individuals breeding in little patches have greater fecundity than those breeding in big patches. The average investment strategy in the population is represented by z. After reproduction, a fraction $1 - m$ of the offspring stay in their natal patch, while the remaining fraction m disperse to random patches in the population. After dispersal, patches may undergo changes in their size according to a Markov process: big patches remain big with probability α and become little with probability $1 - \alpha$, whereas little patches remain little with probability β and become big with probability $1 - \beta$ (see "Patch Dynamics," available online, for details). Subsequently, offspring compete for breeding places, with all nonbreeding individuals perishing. Table 1 provides a summary of model notation.

Helping and Harming

We classify social behaviors according to their impact on fecundity. The impact on the actor's fecundity is $-C \equiv \partial f(x, y)/\partial x$, and the impact on the patchmates' fecundity is $B \equiv \partial f(x, y)/\partial y$, where, assuming vanishing variation in investment strategies, we evaluate derivatives at $x = y = z$ (Taylor and Frank 1996). Helping behaviors are those that improve the fecundity of patchmates ($B > 0$), and harming behaviors are those that reduce the fecundity of patchmates ($B < 0$; Lehmann et al. 2006*a*; West and Gardner 2010). We employ the neighbor-modulated approach to kin-selection analysis (Hamilton 1964; Taylor 1996; Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al. 2007) to determine the direction of natural selection acting on the social trait (see sections "Reproductive Success" through "Reproductive Value" in the appendix, available online, for details). The condition for natural selection to favor the evolution of obligate social behavior is given by

Table 1: Summary of model notation

Symbol	Meaning
B	Big patch
L	Little patch
O	Obligate social behavior
T	Reference to quantities in Taylor's (1992*a*) model
P	Primary recipient
S	Secondary recipient
A_X	Potential for helping in condition X
α	Probability that a big patch remains big
β	Probability that a little patch remains little
c_X	Class reproductive value in condition X
F_X	Fecundity of a breeding female in condition X
h_X	Probability of cophilopatry in condition X
m	Migration rate
n_X	Patch size in condition X
π_X	History of density state in condition X
p	Frequency of big patches in the population
r_X	"Other-only" relatedness of an individual in condition X
R_X	"Whole-group" relatedness of an individual in condition X
σ	Fecundity factor
τ	Temporal coefficient of correlation
v_X	Reproductive value of an individual in condition X
υ_X	Relative reproductive value of patch in condition X
x	Level of helping of a focal actor
y	Average level of the neighbor's social behavior

$$c_{\mathrm{B}}[-C + r_{\mathrm{P|B}}B - (B - C)r_{\mathrm{S|B}}\upsilon_{\mathrm{S|B}}] + c_{\mathrm{L}}[-C + r_{\mathrm{P|L}}B - (B - C)r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}}] > 0, \qquad (1)$$

where c_{B} and c_{L} are the class reproductive values of individuals breeding within big and little patches, respectively (Fisher 1930; Taylor 1990; Grafen 2006); $r_{\mathrm{P|B}}$ and $r_{\mathrm{P|L}}$ are the relatednesses of the actor to her groupmates—"primary recipients"—in big patches and little patches, respectively; $r_{\mathrm{S|B}}$ and $r_{\mathrm{S|L}}$ are the relatednesses of the actor to those offspring competing for breeding sites with the offspring born in her patch—"secondary recipients"—in big patches and little patches, respectively; and $\upsilon_{\mathrm{S|B}}$ and $\upsilon_{\mathrm{S|L}}$ are the reproductive values of secondary recipients, relative to those of primary recipients, in big patches and little patches, respectively (see "Relatedness" and "Reproductive Value," available online, for details). Note that we are defining actor, primary recipients, and secondary recipients in terms of the three selection pressures that arise from the actor's social behavior, namely, the cost to the actor, the benefit to groupmates, and the kin-competition effect. Also note that a focal individual may experience, either directly or through her offspring, more than one of these selection pressures during her lifetime. The conditions for natural selection to favor the evolution of facultative social behavior are given by

$$-C + r_{\mathrm{P|B}}B - (B - C)r_{\mathrm{S|B}}\upsilon_{\mathrm{S|B}} > 0 \qquad (2)$$

and

$$-C + r_{\mathrm{P|L}}B - (B - C)r_{\mathrm{S|L}}\upsilon_{\mathrm{S|L}} > 0 \qquad (3)$$

for actors in big patches and little patches, respectively.

The left-hand side (LHS) of conditions (1)–(3) can be interpreted as the inclusive fitness effect of the social behavior (Hamilton 1963, 1964, 1970). First, the behavior imposes a cost C on the actor's own fecundity. Second, it provides a benefit B to the fecundity of the actor's groupmates, and this benefit is weighted by the relatedness of these primary recipients r_{P}. Finally, the behavior leads to an increase of $B - C$ in the number of offspring born in the patch, and hence it excludes an equal number of secondary recipients who compete with these extra offspring for breeding opportunities. Moreover, this effect is weighted by the relatedness of secondary recipients r_{S} and also by the relative reproductive value of secondary recipients υ_{S}.

Transforming inequalities (1)–(3) into equalities and rearranging into the form $C/B = A$, we obtain the "potential for helping" A (Rodrigues and Gardner 2012; see also Gardner 2010). This represents the valuation that an actor places on the fecundity of the primary recipients, relative to her own fecundity. Thus, if $A > 0$, the actor is selectively favored to help her patchmates, provided that the personal cost to herself is sufficiently small, whereas if $A < 0$, the actor is selectively favored to harm her patchmates, provided that the personal cost to herself is sufficiently small. We may define the additive inverse of the potential for helping (i.e., $-A$) as the "potential for harming" (Rodrigues and Gardner 2012).

From inequality (1), the potential for obligate helping is given by

$$A_O = \frac{c_B(r_{P|B} - r_{S|B}v_{S|B}) + c_L(r_{P|L} - r_{S|L}v_{S|L})}{c_B(1 - r_{S|B}v_{S|B}) + c_L(1 - r_{S|L}v_{S|L})}. \tag{4}$$

From inequalities (2) and (3), the potential for facultative helping is given by

$$A_B = \frac{r_{P|B} - r_{S|B}v_{S|B}}{1 - r_{S|B}v_{S|B}} \tag{5}$$

and

$$A_L = \frac{r_{P|L} - r_{S|L}v_{S|L}}{1 - r_{S|L}v_{S|L}} \tag{6}$$

for big patches and little patches, respectively.

Results and Analysis

In the next two sections, we derive the main results of our model. We begin by considering that the fecundity of individuals is independent of patch size ($\sigma = 1$). We then relax this assumption to consider cases where the individuals' fecundity depends on the patch they are in ($0 < \sigma < \infty$; see table 2 for a summary of the results).

Group-Size Heterogeneity

Spatial Heterogeneity. We first consider a population in which group size varies between patches within generations but not within patches between generations (i.e., spatial heterogeneity only). We derive the following results:

Result 1. Spatial heterogeneity in group size has no impact on the evolution of obligate social behavior ($A_O = 0$), irrespective of parameter values (see "Relatedness" for details). This extends Taylor's (1992*a*) result for homogeneous populations to populations with spatial heterogeneity in group size.

Result 2. Spatial heterogeneity in group size has no impact on the evolution of facultative social behavior ($A_B = A_L = 0$), irrespective of parameter values (see "Relatedness" for details). This extends result 1 for obligate social behavior to facultative social behavior.

In Taylor's (1992*a*) model, the relatedness of primary recipients is given by $r_{P|T} = h_T R_T$, where h_T is the probability that two offspring chosen at random from a patch are both philopatric to that patch after the dispersal phase and R_T is the relatedness of these two offspring. The relatedness of secondary recipients is also given by $r_{S|T} = h_T R_T$. As a consequence, the relatedness of primary recipients is equal to that of secondary recipients, $r_{P|T} = r_{S|T}$. Moreover, because Taylor's (1992*a*) model is of a homogeneous population, all recipients have the same reproductive value in a neutral population, and hence $v_{S|T} = 1$. Since actors place the same value on primary and secondary recipients irrespective of population viscosity ($r_{P|T} = r_{S|T}v_{S|T}$; fig. 1), selection acting on the social behavior is null ($A_T = 0$).

Turning to our model of a spatially heterogeneous population and focusing on facultative helping in big patches, we find that the relatedness of primary recipients is given by $r_{P|B} = h_B R_B$, where h_B is the probability that two offspring chosen at random from a big patch are both philopatric to that patch after the dispersal phase and R_B is the relatedness of these two offspring. The relatedness of secondary recipients is also given by $r_{S|B} = h_B R_B$. As a consequence, the relatedness of primary recipients is equal to that of secondary recipients, $r_{P|B} = r_{S|B}$. In addition, the reproductive value of primary recipients is equal to the reproductive value of secondary recipients, $v_{S|B} = 1$. Since actors attribute the same value to primary and secondary recipients ($r_{P|B} = r_{S|B}v_{S|B}$; fig. 1), selection acting

Table 2: Summary of model results

	Group-size heterogeneity with homogeneous fecundity			Group-size heterogeneity with variable fecundity		
	Spatial	Temporal	Spatial and temporal	Spatial	Temporal	Spatial and temporal
Obligate	Cancel	Cancel	Cancel	−	−	−
Facultative (big patches)	Cancel	Helping	Helping	−	−	+
Facultative (little patches)	Cancel	Harming	Harming	−	−	+

Note: Results for group-size heterogeneity with homogeneous fecundity ($\sigma = 1$) for obligate and facultative behavior and for different group-size heterogeneity assumptions (spatial only, temporal only, and spatial and temporal) and impact of variable fecundity on the results derived for group-size heterogeneity with homogeneous fecundity (minus sign: has no impact; plus sign: has an impact). Obligate trait expression and spatial heterogeneity always lead to the cancellation result. Variable fecundity plays a role in the evolution of social behavior only when populations are spatially and temporally heterogeneous, in which case it can change the predictions of the homogeneous-fecundity model.

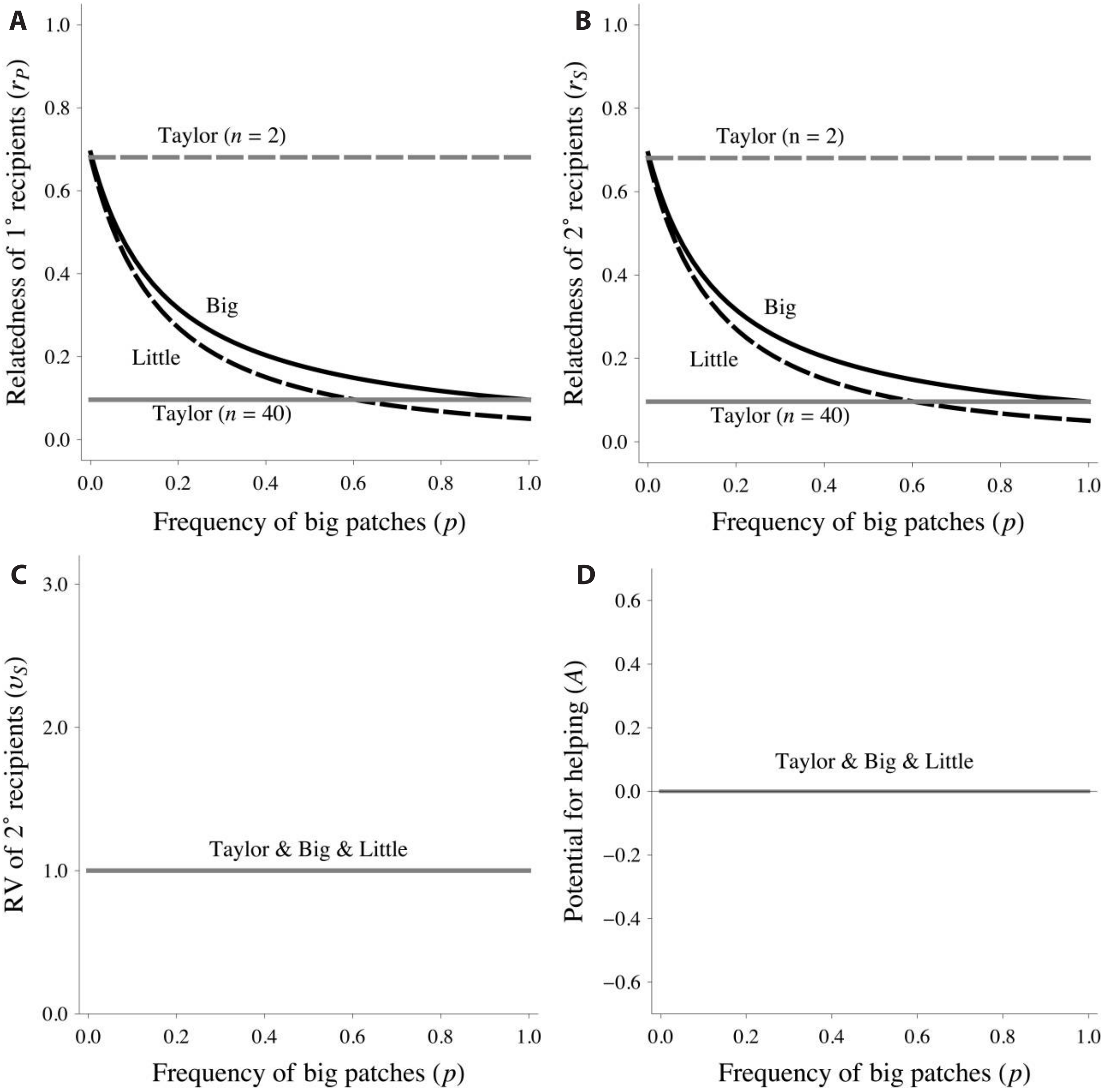

Figure 1: Impact of spatial heterogeneity in group size on relatedness, reproductive value, and potential for helping. *A*, Relatedness of primary (1°) recipients (r_P) as a function of the frequency of big patches (p) for big patches ($r_{P|B}$; solid black line), little patches ($r_{P|L}$; dashed black line), and Taylor's (1992*a*) reference model ($r_{P|T}$; gray lines). The relatedness of primary recipients is higher in big patches, because of higher cophilopatry, and lower in little patches, because of lower cophilopatry, than that in the reference model with equivalent group sizes ($r_{P|B} > r_{P|T}$ and $r_{P|L} < r_{P|T}$). *B*, Relatedness of secondary (2°) recipients (r_S) as a function of the frequency of big patches (p). The relatedness of secondary recipients is higher in big patches, because of higher cophilopatry, and lower in little patches, because of lower cophilopatry, than that in the reference model with equivalent group sizes ($r_{S|B} > r_{S|T}$ and $r_{S|L} < r_{S|T}$). Importantly, primary and secondary recipients are equally related ($r_{P|B} = r_{S|B}$ and $r_{P|L} = r_{S|L}$). *C*, The relative reproductive value (RV) of secondary recipients (υ_S) is constant with respect to the frequency of big patches (p) and is equal to that of the reference model ($\upsilon_{S|B} = \upsilon_{S|L} = \upsilon_{S|T} = 1$). *D*, The potential for facultative helping (A_B and A_L) is constant with respect to the frequency of big patches (p) and is equal to that of the reference model ($A_B = A_L = A_T = 0$). In all plots, we assume $n_B = 40$, $n_L = 2$, $m = 0.10$, and $\tau = 1.00$.

on the social behavior is null ($A_B = 0$). The same argument can be made in relation to little patches (namely, $r_{P|L} = h_L R_L$, $r_{S|L} = h_L R_L$, $r_{P|L} = r_{S|L}$, $v_{S|L} = 1$, $r_{P|L} = r_{S|L} v_{S|L}$, and $A_L = 0$; fig. 1). Finally, selection acting on obligate social behavior is determined by selection acting on each size-type patch weighted by the respective class reproductive values (see the LHS of inequality [1]). As we have just seen, selection acting on each size-type patch is null ($A_B = 0$ and $A_L = 0$). Therefore, selection acting on obligate social behavior is also null ($A_O = 0$). Full mathematical details are given in "Cancellation of Obligate Helping and Harming," available online.

Temporal Heterogeneity. We next consider a population in which group size varies within patches between generations but not between patches within generations (i.e., temporal heterogeneity only). In particular, we assume that all patches in the population become big with probability p or little with probability $1 - p$ in every generation. We derive the following results:

Result 3. Temporal heterogeneity in group size has no impact on the evolution of obligate social behavior ($A_O = 0$), irrespective of parameter values (see "Temporal Heterogeneity" in the appendix, available online, for details). This extends Taylor's (1992*a*) cancellation result for homogeneous populations to populations with temporal heterogeneity in group size.

Result 4. Temporal heterogeneity in group size does have an impact on the evolution of facultative social behavior ($A_B \neq 0$ and $A_L \neq 0$). Specifically, selection favors helping in patches with more breeders ($A_B \geq 0$) and harming in patches with fewer breeders ($A_L \leq 0$; see "Temporal Heterogeneity" in the appendix for details).

Why does Taylor's (1992*a*) result no longer hold for facultative social behavior in temporally heterogeneous populations? Focusing on helping in big patches only, we find that the relatedness of primary recipients is given by $r_{P|B} = p h_B R_B + (1 - p) h_L R_L$, where p is the probability that the patch was big in the previous generation, h_B is the probability that two offspring chosen at random from a big patch are both philopatric to that patch after the dispersal phase, R_B is the relatedness of these two offspring, $1 - p$ is the probability that the patch was little in the previous generation, h_L is the probability that two offspring chosen at random from a little patch are both philopatric to that patch after the dispersal phase, and R_L is the relatedness of these two offspring. The relatedness of secondary recipients is $r_{S|B} = h_B R_B$. Note that $h_B = h_L = (1 - m)^2$ and that because there are more breeders in big patches than in little patches, relatedness is higher among the offspring born in the former and lower among the offspring born in the latter patch type; that is, $R_B < R_L$.

Consequently, the relatedness of primary recipients is greater than the relatedness of secondary recipients; that is, $r_{P|B} > r_{S|B}$. In addition, because within each generation all individuals are identical, the reproductive value of primary recipients is equal to the reproductive value of secondary recipients; that is, $v_{P|B} = v_{S|B}$. Since actors place more value on primary recipients than on secondary recipients, selection favors helping behavior in big patches ($A_B > 0$; fig. 2). A similar argument can be made in relation to little patches. However, because primary recipients are now less valuable than secondary recipients for actors, selection favors harming behavior in little patches: $r_{P|L} = p h_B R_B + (1 - p) h_L R_L$, $r_{S|L} = h_L R_L$, $h_B = h_L = (1 - m)^2$, $R_B < R_L$, $v_{S|L} = 1$, and $A_L < 0$ (fig. 2; see "Temporal Heterogeneity" in the appendix for details). In other words, when patches increase in size (i.e., after a bottleneck), helping is favored; by contrast, when patches decrease in size, harming is favored.

When we turn to obligate social behavior, relatednesses are averaged over patch types, weighted by the corresponding class reproductive values; that is, $r_{P|O} = c_B r_{P|B} + c_L r_{P|L}$. Because the relatedness of primary recipients in big patches is equal to that of primary recipients in little patches ($r_{P|B} = r_{P|L}$) and the class reproductive values sum to unity ($c_B + c_L = 1$), the relatedness of primary recipients is given by $r_{P|O} = r_{P|B} = r_{P|L} = p h_B R_B + (1 - p) h_L R_L$. The relatedness of secondary recipients is given by $r_{S|O} = c_B h_B R_B + c_L h_L R_L$. Because class reproductive values are given by $c_B = p$ and $c_L = 1 - p$, we find that the relatedness of primary recipients is equal to that of secondary recipients ($r_{P|O} = r_{S|O}$). Consequently, selection acting on obligate social behavior is null ($A_O = 0$; see "Temporal Heterogeneity" in the appendix for details).

Spatial and Temporal Heterogeneity. Finally, we consider a population characterized by heterogeneity in group size both between patches within generations and within patches between generations (i.e., spatial and temporal heterogeneity). We derive the following results:

Result 5. Spatial and temporal heterogeneity in group size has no impact on the evolution of obligate social behavior ($A_O = 0$), irrespective of parameter values (see "Cancellation of Obligate Helping and Harming" for details). This extends Taylor's (1992*a*) cancellation result for homogeneous populations to obligate social behaviors in spatially and temporally heterogeneous populations.

Result 6. Spatial and temporal heterogeneity in group size does influence the evolution of facultative social behaviors ($A_B \neq 0$ and $A_L \neq 0$). Specifically, selection favors helping in patches with more breeders ($A_B \geq 0$) and harming in patches with fewer breeders ($A_L \leq 0$; see "Cancellation of Obligate Helping and Harming" for details).

Why does Taylor's (1992*a*) result no longer hold when

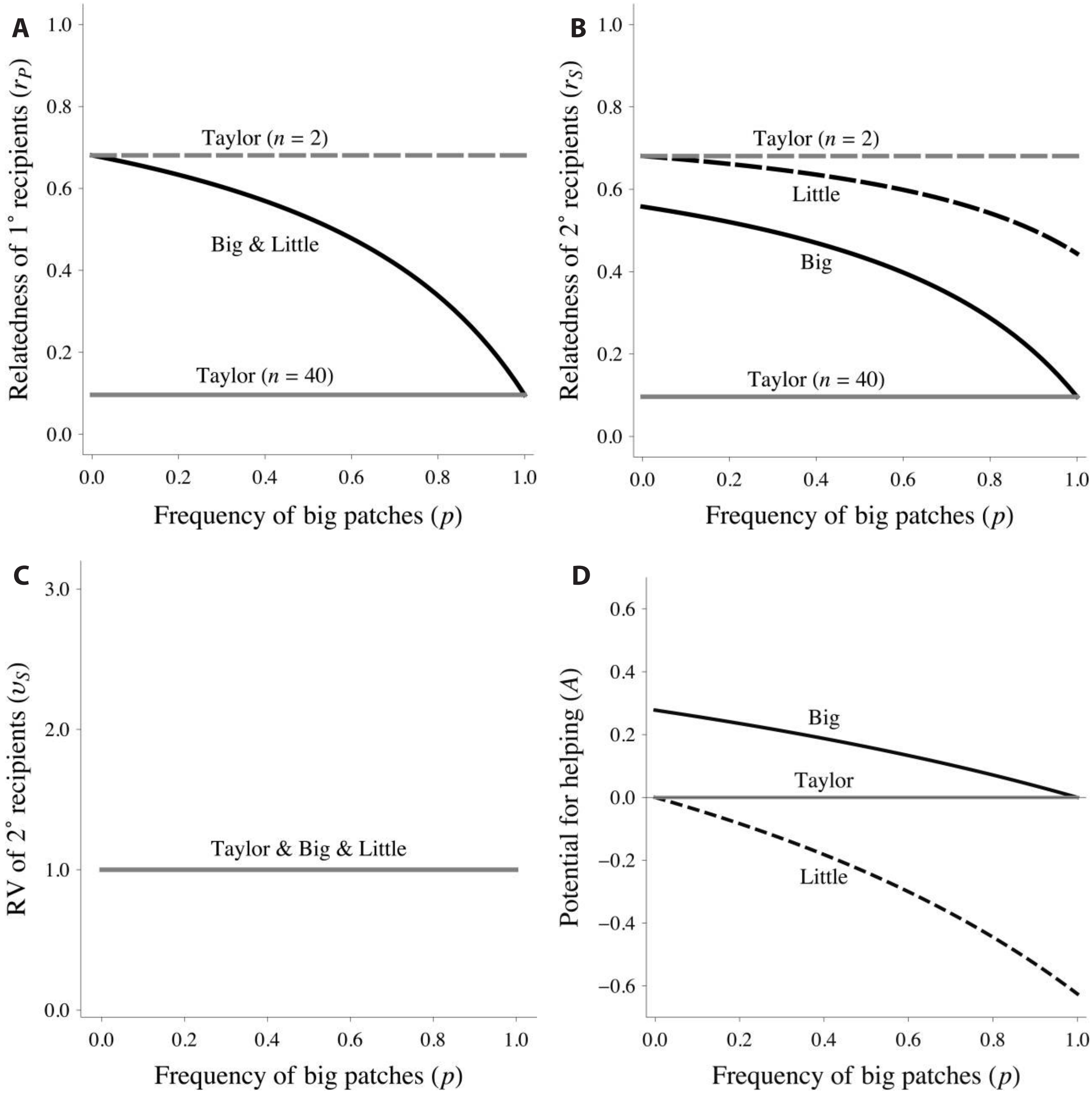

Figure 2: Impact of temporal heterogeneity in group size on relatedness, reproductive value, and potential for helping. *A*, Relatedness of primary (1°) recipients (r_P) as a function of the frequency of big patches (p) for big patches ($r_{P|B}$; solid black line), little patches ($r_{P|L}$; dashed black line), and Taylor's (1992*a*) reference model ($r_{P|T}$; gray lines). The relatedness of primary recipients is higher in big patches, because of higher cophilopatry, and lower in little patches, because of lower cophilopatry, than that of the reference model with equivalent group sizes ($r_{P|B} > r_{P|T}$ and $r_{P|L} < r_{P|T}$). *B*, Relatedness of secondary (2°) recipients (r_S) as a function of the frequency of big patches (p). The relatedness of secondary recipients is higher in big patches, because of higher cophilopatry, and lower in little patches, because of lower cophilopatry, than that of the reference model with equivalent group sizes ($r_{S|B} > r_{S|T}$ and $r_{S|L} < r_{S|T}$). Importantly, in big patches primary recipients are more related to the actor than are secondary recipients, while in little patches primary recipients are less related to the actor than are secondary recipients ($r_{P|B} > r_{S|B}$ and $r_{P|L} < r_{S|L}$). *C*, The relative reproductive value (RV) of secondary recipients (v_S) is constant with respect to the frequency of big patches (p) and is equal to that of the reference model ($v_{S|B} = v_{S|L} = v_{S|T} = 1$). *D*, Potential for facultative helping (A_B and A_L) as a function of the frequency of big patches (p). In big patches there is potential for helping ($A_B > 0$), while in little patches there is potential for harming ($A_L < 0$). In all plots, we assume $n_B = 40$, $n_L = 2$, and $m = 0.10$.

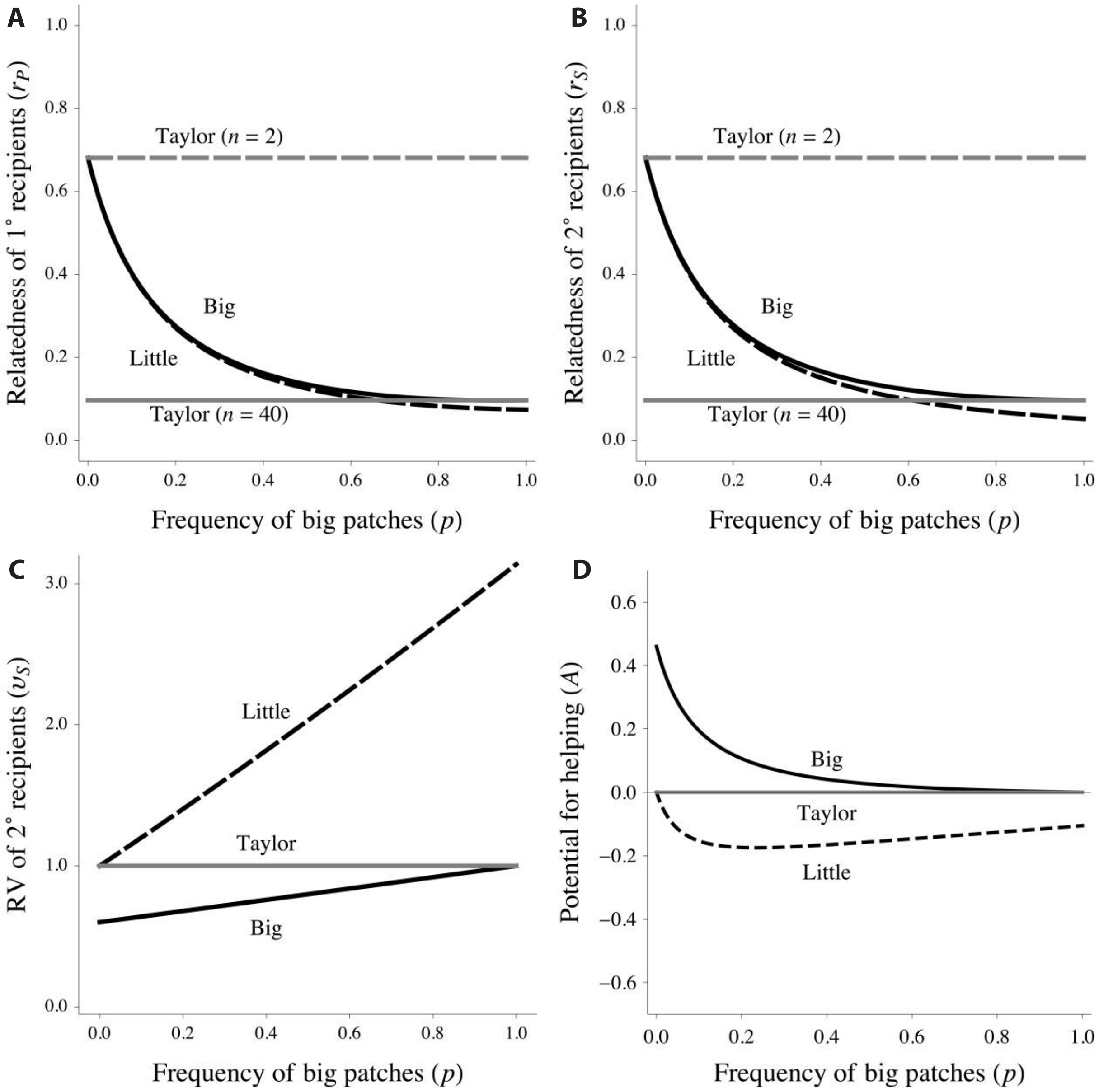

Figure 3: Impact of spatial and temporal heterogeneity in group size on relatedness, reproductive value, and potential for helping. *A*, Relatedness of primary (1°) recipients (r_{P}) as a function of the frequency of big patches (p) for big patches ($r_{\mathrm{P|B}}$; solid black line), little patches ($r_{\mathrm{P|L}}$; dashed black line), and Taylor's (1992*a*) reference model ($r_{\mathrm{P|T}}$; gray lines). The relatedness of primary recipients is higher in big patches, because of higher cophilopatry, and lower in little patches, because of lower cophilopatry, than that of the reference model with equivalent group sizes ($r_{\mathrm{P|B}} > r_{\mathrm{P|T}}$ and $r_{\mathrm{P|L}} < r_{\mathrm{P|T}}$). *B*, Relatedness of secondary (2°) recipients (r_{S}) as a function of the frequency of big patches (p). The relatedness of secondary recipients is higher in big patches, because of higher cophilopatry, and lower in little patches, because of lower cophilopatry, than that of the reference model with equivalent group sizes ($r_{\mathrm{S|B}} > r_{\mathrm{S|T}}$ and $r_{\mathrm{S|L}} < r_{\mathrm{S|T}}$). Importantly, in big patches primary recipients are less related to the actor than are secondary recipients, while in little patches primary recipients are more related to the actor than are secondary recipients ($r_{\mathrm{P|B}} < r_{\mathrm{S|B}}$ and $r_{\mathrm{P|L}} > r_{\mathrm{S|L}}$). *C*, Relative reproductive value (RV) of secondary recipients (v_{S}) as a function of the frequency of big patches (p). In big patches the relative reproductive value of secondary recipients is less than or equal to that of the reference model, and in little patches the relative reproductive value is greater than or equal to that of the reference model ($v_{\mathrm{S|B}} \leq v_{\mathrm{S|T}}$ and $v_{\mathrm{S|T}} \leq v_{\mathrm{S|L}}$). *D*, Potential for facultative helping (A_{B} and A_{L}) as a function of the frequency of big patches (p). In big patches there is potential for helping ($A_{\mathrm{B}} > 0$), while in little patches there is potential for harming ($A_{\mathrm{L}} < 0$). By numerical analysis, these results were confirmed to hold over the entire range of possible parameter state space. In all plots, numerical examples are given for $n_{\mathrm{B}} = 40$, $n_{\mathrm{L}} = 2$, $m = 0.1$, and $\tau = 1/2$.

we consider facultative helping and harming in populations that are both spatially and temporally heterogeneous with respect to group size? Focusing our attention on facultative helping in big patches, we find that the potential for this to be favored is given by equation (5); that is, $A_\mathrm{B} = (r_{\mathrm{P}|\mathrm{B}} - r_{\mathrm{S}|\mathrm{B}}v_{\mathrm{S}|\mathrm{B}})/(1 - r_{\mathrm{S}|\mathrm{B}}v_{\mathrm{S}|\mathrm{B}})$. If patch quality is heterogeneous in space and time, then the actor's big patch may have been either big or little in the previous generation, and so her relatedness to primary recipients $r_{\mathrm{P}|\mathrm{B}}$ is a weighted average of the product of the probability of cophilopatry h_B and whole-group relatedness R_B for big patches and the product of the probability of cophilopatry h_L and whole-group relatedness R_L for little patches (see "Relatedness" for details). In contrast, her relatedness $r_{\mathrm{S}|\mathrm{B}}$ to the secondary recipients is the product of the probability of cophilopatry h_B and whole-group relatedness R_B for her big patch (see "Relatedness" for details). Moreover, the reproductive value of her secondary recipients—that is, adults in her patch in the next generation—need not be equal to her own reproductive value. While she has the reproductive value of an individual in a big patch, theirs is a weighted average of the reproductive values for individuals in big and little patches. Hence, $v_{\mathrm{S}|\mathrm{B}} < 1$ (see "Reproductive Value" for details).

Consequently, the actor may place different values on her primary and secondary recipients: $r_{\mathrm{P}|\mathrm{B}} \neq r_{\mathrm{S}|\mathrm{B}}v_{\mathrm{S}|\mathrm{B}}$ (fig. 3; see "Cancellation of Obligate Helping and Harming" for details). Hence, the potential for facultative helping in big patches may be nonzero, $A_\mathrm{B} \neq 0$. The same is true for facultative helping in little patches: $r_{\mathrm{P}|\mathrm{L}} \neq r_{\mathrm{S}|\mathrm{L}}$ and $v_{\mathrm{S}|\mathrm{L}} > 1$, so $r_{\mathrm{P}|\mathrm{L}} \neq r_{\mathrm{S}|\mathrm{L}}v_{\mathrm{S}|\mathrm{L}}$ and $A_\mathrm{L} \neq 0$ (fig. 3; see "Cancellation of Obligate Helping and Harming" for details). We find that there is potential for helping in big patches and potential for harming in little patches ($A_\mathrm{B} > 0$ and $A_\mathrm{L} < 0$; fig. 4).

Although actors in big patches place different values on their primary and secondary recipients and this is also true of actors in little patches, we find that, on average over both patch types, actors place equal value on their primary and secondary recipients. This owes to the properties of the ecological dynamics that determines how patch size changes across generations. Specifically, this is characterized by a time-homogeneous Markov chain, such that the backward processes that determine the value of primary recipients are identical to the forward processes that determine the value of secondary recipients (see "Cancellation of Obligate Helping and Harming" for details). Hence, while there is nonzero potential for facultative helping and harming in big and little patches ($A_\mathrm{B} \neq 0$ and $A_\mathrm{L} \neq 0$), there is zero potential for obligate helping and harming ($A_\mathrm{O} = 0$) in populations characterized by both spatial and temporal heterogeneity.

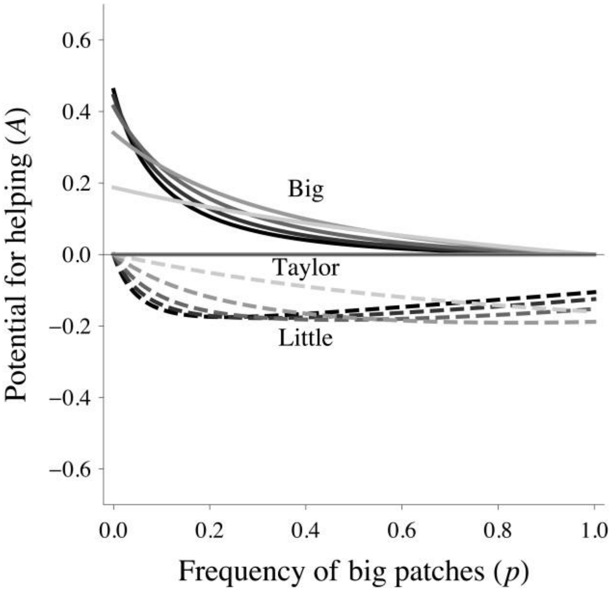

Figure 4: Impact of spatial and temporal heterogeneity in group size on the potential for helping: potential for facultative helping (A_B and A_L) as a function of the frequency of high-quality patches (p), for varying sizes of big patches ($n_\mathrm{B} = 40, 30, 20, 10$, and 4). Darker lines represent larger patch sizes, while lighter lines represent smaller patch sizes. There is always potential for helping in big patches, while there is always potential for harming in little patches. Parameter values: $n_\mathrm{L} = 2$, $m = 0.1$, and $\tau = 1/2$.

Group-Size Heterogeneity with Variable Fecundity

Here we consider that a group's size may affect its constituents' fecundity ($0 < \sigma < \infty$). This enables us to study how the interaction between patch-size heterogeneity and variable fecundity mediates the evolution of obligate and facultative helping and harming. For example, several studies have shown that increasing a group's size may decrease the fitness of its constituent members, for example, because of infectious diseases (e.g., Rifkin et al. 2012). By contrast, other studies have shown that increasing a group's size may increase the fitness of its constituent members, for example, because of a decrease in predation rates or more efficient foraging (e.g., Hamilton 1971*a*). We derive the following results:

Result 7. We find that introducing patch productivity heterogeneity has no impact on the results derived for patch-size heterogeneity when (1) individuals express obligate behavior and/or (2) there is spatial heterogeneity only or (3) there is temporal heterogeneity only.

Result 8. We find that introducing patch productivity heterogeneity does have an impact on the results derived for patch-size heterogeneity when individuals express facultative behavior and there is both spatial and temporal heterogeneity.

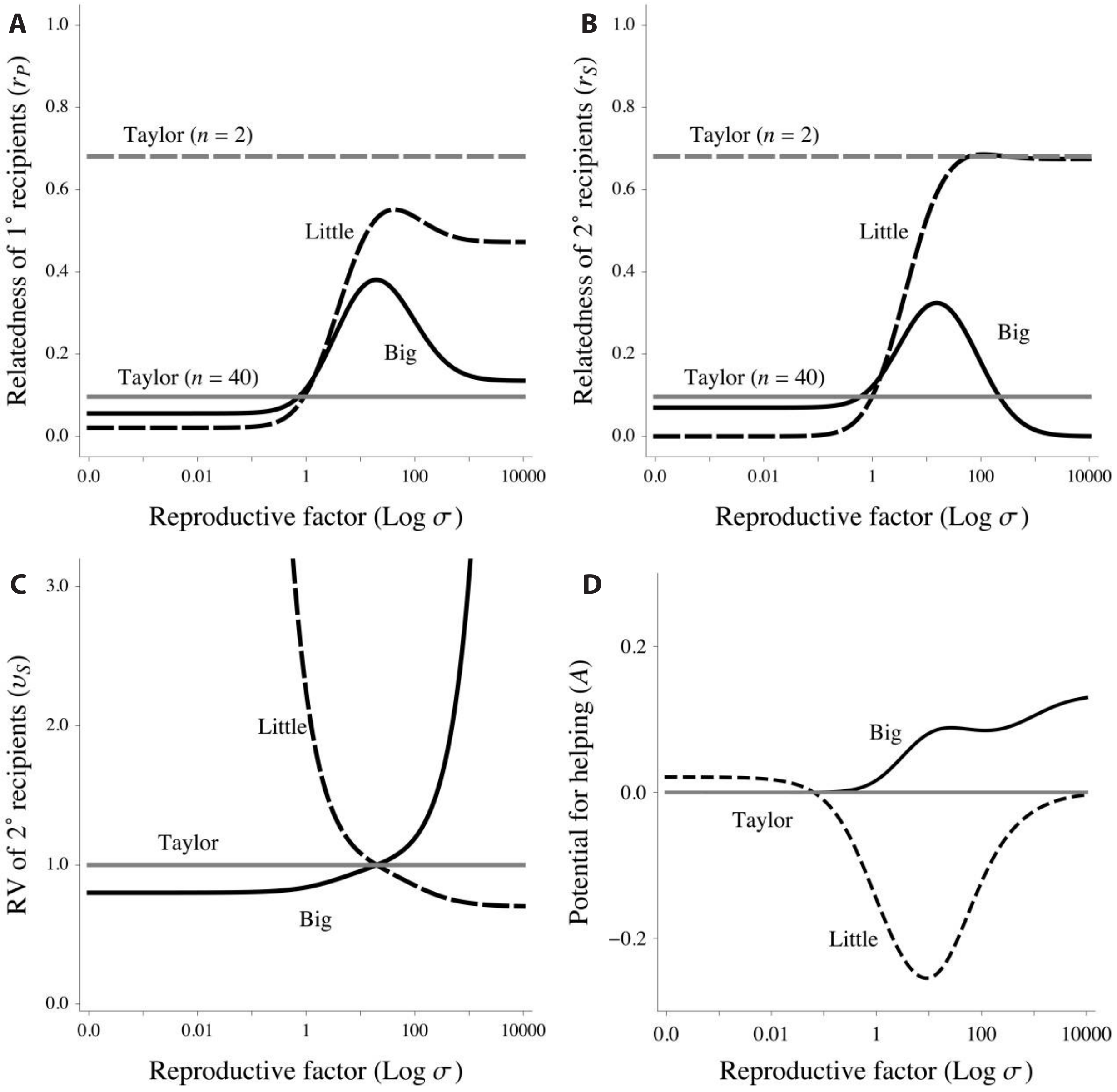

Figure 5: Impact of spatial and temporal heterogeneity in group size and variable fecundity on relatedness, reproductive value, and potential for helping. *A*, Relatedness of primary (1°) recipients (r_P) as a function of the reproductive factor (σ) for big patches ($r_{P|B}$; solid black line), little patches ($r_{P|L}$; dashed black line), and Taylor's (1992*a*) reference model ($r_{P|T}$; gray lines). In big patches, for lower values of the reproductive factor, the relatedness of primary recipients is less than that of the reference model ($r_{P|B} < r_{P|T}$), while for higher values of the reproductive factor, the relatedness of primary recipients is greater than that of the reference model ($r_{P|B} > r_{P|T}$). In little patches, the relatedness of primary recipients is always less than that of the reference model ($r_{P|L} < r_{P|T}$). *B*, Relatedness of secondary (2°) recipients (r_S) as a function of the reproductive factor (σ). In big patches, for lower values of the reproductive factor, the relatedness of secondary recipients is less than that of the reference model ($r_{S|B} < r_{S|T}$), while for higher values of the reproductive factor, the relatedness of secondary recipients is greater than that of the reference model ($r_{S|B} > r_{S|T}$). In little patches, the relatedness of secondary recipients is less than that of the reference model for a wide range of the reproductive factor ($r_{S|L} < r_{S|T}$). Importantly, in big patches and for lower values of the reproductive factor, primary recipients are less related to the actor than are secondary recipients ($r_{P|B} < r_{S|B}$), while for higher values of the reproductive factor, primary recipients are more related to the actor than are secondary recipients ($r_{P|B} > r_{S|B}$); by contrast, in little patches, primary recipients are more related to the actor than are secondary recipients for smaller values of the reproductive factor ($r_{P|L} > r_{S|L}$), while for higher values of the reproductive factor, primary recipients are less related to the actor than are secondary recipients ($r_{P|L} < r_{S|L}$). *C*, Relative reproductive value (RV) of secondary recipients (υ_S) as a function of the reproductive factor (σ). In big patches, the relative reproductive value of secondary recipients is less than that of the reference model for smaller values of the reproductive factor ($\upsilon_{S|B} < \upsilon_{S|T}$), while it is greater than that of the reference model for higher values of the reproductive factor ($\upsilon_{S|B} > \upsilon_{S|T}$). In little patches, the relative

Why does variable fecundity have no impact on the results obtained for the evolution of helping and harming in a group-size-heterogeneous population with homogeneous fecundity when we assume (1) obligate behavior and/or (2) group-size spatial heterogeneity only or (3) group-size temporal heterogeneity only? We find that the cancellation result obtained for a group-size-heterogeneous population (i.e., our results 1–3 and 5) continues to hold, for the same reason, irrespective of variation in fecundity. This is because, while variable fecundity alters both the relatedness coefficient and the reproductive value of each recipient, the relationship between the overall value (i.e., life-for-life relatedness) of primary and secondary recipients remains unaltered, and therefore the cancellation result continues to hold.

Why does variable fecundity influence the consequences of group-size heterogeneity in the model with both spatial and temporal heterogeneity? We find that if breeders in little patches have low fecundity (low σ), then facultative helping can be favored. This is because secondary recipients are usually unrelated immigrants, and therefore the relatedness of secondary recipients is close to 0, making the kin-competition effect negligible. By contrast, facultative harming is favored in big patches. This is because secondary recipients are usually philopatric to the patch, which intensifies the kin-competition effect, favoring harming behavior. By contrast, if breeders in little patches have high fecundity (high σ), then we recover the same qualitative result derived for patch-size heterogeneity with homogeneous fecundity, namely, that helping is favored in big patches and harming is favored in little patches (fig. 5).

Discussion

Population viscosity has been proposed as a general mechanism that promotes interactions between closely related individuals and may therefore favor the evolution of kin-selected traits such as altruistic cooperation (Hamilton 1964, 1971*b*). However, it is now well recognized that population viscosity also promotes competition between kin. Here we have shown that Taylor's (1992*a*) seminal result, that the relatedness and competition effects of viscosity exactly cancel in homogeneous populations, extends to populations where groups vary in size because of extrinsic factors. Specifically, population viscosity has no impact on the evolution of obligate helping or harming in populations characterized by spatial and/or temporal heterogeneity in group size. Moreover, if individuals can facultatively adjust their behavior conditionally on their group's size, then there is no selection for either helping or harming when there is only spatial heterogeneity in group size. However, we have shown that facultative helping is favored in big groups and that facultative harming is favored in little groups when there is either only temporal or both temporal and spatial heterogeneity in group size. More generally, we have shown that Taylor's (1992*a*) cancellation result for obligate behavior is robust across a wide range of ecologically realistic scenarios, and we have clarified why this cancellation occurs. Specifically, the proper weights of each selection pressure are the life-for-life relatedness coefficients, which are given in terms of recipients' relatednesses and reproductive values, both of which emerge from the specific demographic dynamics of the population (see "Cancellation of Obligate Helping and Harming" for details).

Here we considered that differences in patch size may manifest themselves as differences in the number of breeders that the patches are able to support. Rodrigues and Gardner (2012) also studied how population heterogeneity in patch quality mediates the evolution of social behavior when differences in patch quality are manifested as differences in the fecundity of the individuals breeding in the patches. In line with our results here, Rodrigues and Gardner (2012) found that spatial and/or temporal heterogeneity has no impact on the evolution of obligate helping or harming. However, in contrast to our results, Rodrigues and Gardner (2012) found that both spatial and temporal heterogeneity were required in order for facultative helping and harming to be favored. Thus, an alteration in a particular assumption as to how patch quality translates into improved productivity—that is, more breeders having the same fecundity rather than the same number of breeders having greater fecundity—leads to a less stringent condition for population viscosity to promote the evolution of social behavior. Furthermore, we have explained why variable fecundity influences the results of patch-size heterogeneity and homogeneous fecundity when the spatial and temporal treatment is considered, but not when the temporal treatment alone is considered. This clarifies the

reproductive value is greater than that of the reference model for lower values of the reproductive factor ($v_{S|T} < v_{S|L}$), while it is less than that of the reference model for higher values of the reproductive factor ($v_{S|T} > v_{S|L}$). *D*, Potential for facultative helping (A_B and A_L) as a function of the reproductive factor (σ). In big patches there is potential for harming for lower values of the reproductive factor ($A_B < 0$), while there is potential for helping for higher values of the reproductive factor ($A_B > 0$). In little patches there is potential for helping for lower values of the reproductive factor ($A_L > 0$), while there is potential for harming for higher values of the reproductive factor ($A_L < 0$). In all plots, numerical examples are given for $n_B = 40$, $n_L = 2$, $m = 0.1$, $p = 0.6$, and $\tau = 1/2$.

importance of understanding how ecological and genetic factors affect life-for-life coefficients of relatedness.

Grafen (2007) also studied the evolution of obligate helping in a population characterized by heterogeneity in group size and limited dispersal. However, his model assumed global competition for reproductive resources and hence the absence of kin competition. Consequently, he found that obligate helping readily evolved in the context of his model. Here we have considered that limited dispersal determines both the genetic structure of the population and the scale of competition. This emphasizes the importance of demography for mediating the costs and benefits of social behavior as well as the genetic relatedness of social partners. Lehmann et al. (2006*b*) studied the evolution of social traits in a metapopulation characterized by patch extinctions, and we recover a similar scenario by considering that there are no breeders in little patches ($n_L = 0$). In this special case of our model, it is meaningless to distinguish obligate from facultative behavior, as all breeders are in big patches, and consequently there is no avenue for helping or harming to be favored in this scenario. The Lehmann et al. model considered that transitions between occupied and empty patches are governed by social interaction, allowing helping behaviors to be favored. This contrasts with our results, which do not require this mechanism for helping and harming to be favored. Our results also show that environmental heterogeneity favors plastic social responses to local environments and therefore support the idea that plasticity and varying ecological factors can play a role in promoting social behavior in viscous populations (e.g., Kelly 1992; Queller 1992). More generally, future work should explore how extrinsic factors (e.g., climate change) and intrinsic factors (e.g., social behavior) interact to drive patch heterogeneity and mediate the evolution of helping and harming.

Our model also relates to the "bottleneck" hypothesis in the evolution of multicellularity, which suggests that a unicellular bottleneck, followed by a growth cycle in the absence of migration, has been fundamental for the evolution of multicellular organisms (Dawkins 1982; Maynard Smith 1988; Maynard Smith and Szathmáry 1995; Grosberg and Strathmann 1998, 2007; Bourke 2011). These characteristics of the life cycle already assume preadaptations, which can be social traits themselves. Our model predicts that facultative helping is favored among groupmates after the group has passed through a bottleneck and has grown to full size, and it also predicts that facultative harming is favored before the group has grown to full size (see result 4). Therefore, less stringent bottlenecks may have been important to produce preadaptations before the full onset of highly complex cooperative societies (e.g., multicellular organisms or eusocial insects). Although some empirical work has been done in this area (Brockhurst 2007; Brockhurst et al. 2007), the kin-competition effects of population viscosity in the context of bottlenecks have so far been neglected. Experimental protocols that test the effect of population viscosity in social evolution are already available (Griffin et al. 2004; Kümmerli et al. 2009), and these could be extended to incorporate the effects of spatial and temporal patch-size heterogeneity.

Our results show that heterogeneity in patch size and individual fecundity affects the value of each selective force in populations characterized by some degree of viscosity. This happens because (1) it changes the genetic structure of the population and consequently the relatedness of each recipient and (2) it changes the reproductive value of each recipient. Therefore, heterogeneity in patch size and individual fecundity is likely to mediate the evolution of social traits other than those considered here. For example, sex ratio (i.e., the fraction of offspring that are male) is a classic social trait (Hamilton 1967; Charnov 1982; West 2009). Selection pressures acting on sex ratio—including those relating to kin competition—depend on both the relatedness and the reproductive value of the different recipients (males vs. females). Thus, extending our framework to study sex ratio evolution is a promising line of future research.

Acknowledgments

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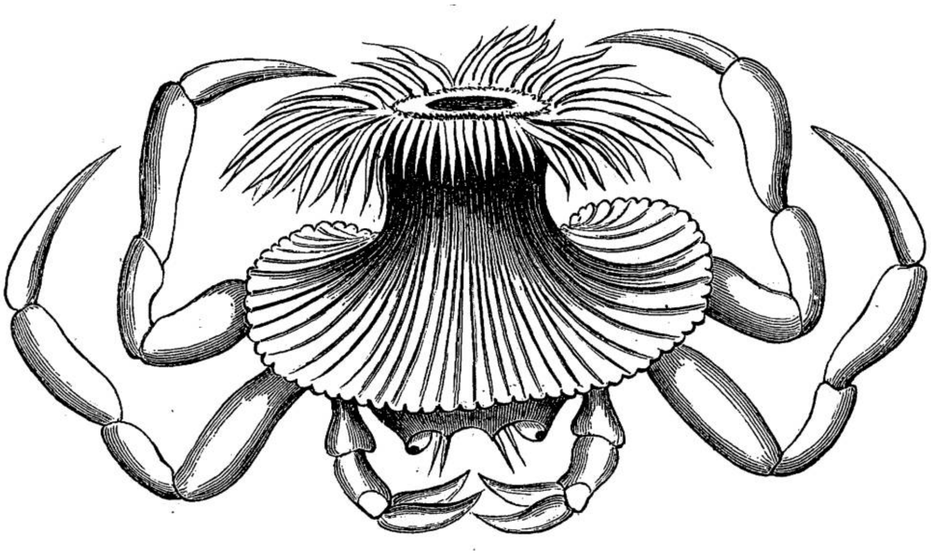

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Appendix from A. M. M. Rodrigues and A. Gardner, “Evolution of Helping and Harming in Viscous Populations When Group Size Varies”

(Am. Nat., vol. 181, no. 5, p. 609)

Patch Dynamics

We use the transition probabilities in patch quality, given in the main text, to define a transition matrix,

$$\mathbf{P} = \begin{pmatrix} \alpha & 1-\beta \\ 1-\alpha & \beta \end{pmatrix}. \tag{A1}$$

At equilibrium, the proportion of big patches is given by

$$p = \frac{1-\beta}{2-(\alpha+\beta)}, \tag{A2}$$

with $\alpha < 1$ and $\beta < 1$. We define a random variable X_t that characterizes a patch’s quality ($B = 1$, $L = 0$) in any given generation t. The coefficient of temporal correlation between any two generations is given by $\tau = \mathrm{Cov}\,(X_t, X_{t+1})/(\mathrm{Var}\,(X_t)\,\mathrm{Var}\,(X_{t+1}))^{1/2}$. In terms of model parameters, this is

$$\tau = \alpha - (1-\beta) \tag{A3}$$

at ecological equilibrium. We find that the temporal correlation τ and the frequency of big patches p cannot be varied totally independently of each other when the temporal correlation is negative ($\tau < 0$). For example, when $\tau = -1$, the frequency of big patches is constrained to be $p = 1/2$.

Reproductive Success

We follow the life cycle described in the main text; however, we make the additional assumptions that a mother in a big patch has a large number $F_{\mathrm{B}} = f(x_{\mathrm{B}}, y_{\mathrm{B}})$ of offspring, while a mother in a little patch has a large number $F_{\mathrm{L}} = \sigma f(x_{\mathrm{L}}, y_{\mathrm{L}})$, where $0 < \sigma < \infty$ is the reproductive factor. This recovers the model of Rodrigues and Gardner (2012) as a special case and hence facilitates connections between the two models. An offspring of a focal breeding mother in a big patch remains in her natal patch with probability $1 - m$ and subsequently wins a breeding site with probability

$$E^{\bullet}_{\mathrm{B}\to\mathrm{B}} = \frac{f(x_{\mathrm{B}}, y_{\mathrm{B}})}{(1-m)f(x_{\mathrm{B}}, Y_{\mathrm{B}})n_{\mathrm{B}} + m[pf(z_{\mathrm{B}}, z_{\mathrm{B}})n_{\mathrm{B}} + (1-p)\sigma f(z_{\mathrm{L}}, z_{\mathrm{L}})n_{\mathrm{L}}]}, \tag{A4}$$

where Y_{B} is the average level of the social behavior in the focal patch and z_{B} and z_{L} are the average levels of the social behavior across the population in big and little patches, respectively. Conversely, with probability m the offspring migrates to a random patch in the population. With probability p she reaches a big patch, and she subsequently wins a breeding site with probability

$$E^{\circ}_{\mathrm{B}\to\mathrm{B}} = \frac{f(x_{\mathrm{B}}, y_{\mathrm{B}})}{(1-m)f(z_{\mathrm{B}}, z_{\mathrm{B}})n_{\mathrm{B}} + m[pf(z_{\mathrm{B}}, z_{\mathrm{B}})n_{\mathrm{B}} + (1-p)\sigma f(z_{\mathrm{L}}, z_{\mathrm{L}})n_{\mathrm{L}}]}. \tag{A5}$$

With probability $1 - p$ she reaches a little patch, and she subsequently wins a breeding site with probability

$$E^{\circ}_{\mathrm{B}\to\mathrm{L}} = \frac{f(x_{\mathrm{B}}, y_{\mathrm{B}})}{(1-m)\sigma f(z_{\mathrm{L}}, z_{\mathrm{L}})n_{\mathrm{L}} + m[pf(z_{\mathrm{B}}, z_{\mathrm{B}})n_{\mathrm{B}} + (1-p)\sigma f(z_{\mathrm{L}}, z_{\mathrm{L}})n_{\mathrm{L}}]}. \tag{A6}$$

We derive the same expressions for a mother in a little patch. We may then define a matrix

$$\mathbf{E} = \begin{pmatrix} E_{\mathrm{B}\to\mathrm{B}} & E_{\mathrm{L}\to\mathrm{B}} \\ E_{\mathrm{B}\to\mathrm{L}} & E_{\mathrm{L}\to\mathrm{L}} \end{pmatrix} = \begin{pmatrix} (1-m)E^{\bullet}_{\mathrm{B}\to\mathrm{B}} + mpE^{\circ}_{\mathrm{B}\to\mathrm{B}} & mpE^{\circ}_{\mathrm{L}\to\mathrm{B}} \\ m(1-p)E^{\circ}_{\mathrm{B}\to\mathrm{L}} & (1-m)E^{\bullet}_{\mathrm{L}\to\mathrm{L}} + m(1-p)E^{\circ}_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}. \tag{A7}$$

We define a diagonal matrix **n** where the first element of the diagonal is n_{B} and the second is n_{L}. The matrix that gives the reproductive success of a focal individual for each patch type, in relation to the patch type where their offspring are going to reproduce, is then given by

$$\mathbf{w} = \begin{pmatrix} w_{\mathrm{B}\to\mathrm{B}} & w_{\mathrm{L}\to\mathrm{B}} \\ w_{\mathrm{B}\to\mathrm{L}} & w_{\mathrm{L}\to\mathrm{L}} \end{pmatrix} = \mathbf{nPE}. \tag{A8}$$

Thus, the reproductive success of a focal individual is given by

$$W_{\mathrm{B}} = w_{\mathrm{B}\to\mathrm{B}} + \frac{v_{\mathrm{B}}}{v_{\mathrm{L}}} w_{\mathrm{B}\to\mathrm{L}} \tag{A9}$$

and

$$W_{\mathrm{L}} = w_{\mathrm{L}\to\mathrm{L}} + \frac{v_{\mathrm{L}}}{v_{\mathrm{B}}} w_{\mathrm{L}\to\mathrm{B}} \tag{A10}$$

for big and little patches, respectively. The successful offspring must be weighted by their reproductive values (Fisher 1930; Taylor 1990; Grafen 2006). The reproductive value of offspring that reproduce in big patches is v_{B}, while the reproductive value of offspring that reproduce in little patches is v_{L}. The reproductive success of a random focal individual in the population is therefore given by

$$W = c_{\mathrm{B}} W_{\mathrm{B}} + c_{\mathrm{L}} W_{\mathrm{L}}, \tag{A11}$$

where c_{B} and c_{L} are the class reproductive values of big and little patches, respectively. Class reproductive values are the product of the frequency of individuals in a given class and their individual reproductive values: $c_{\mathrm{B}} = u_{\mathrm{B}} v_{\mathrm{B}}$ and $c_{\mathrm{L}} = u_{\mathrm{L}} v_{\mathrm{L}}$ for big and little patches, respectively. Class reproductive values are normalized such that $c_{\mathrm{B}} + c_{\mathrm{L}} = 1$ (Taylor 1990, 1996; Taylor et al. 2007).

Hamilton's Rule and Potential for Helping and Harming

We assume that the social behavior is under the control of a single locus. Drawing a gene at random from this locus from a focal individual, we denote its genic value by g. Assuming vanishingly small genetic variation in the population, the direction of selection acting on the social behavior is given by

$$\frac{dW}{dg} = c_{\mathrm{B}} \frac{dW_{\mathrm{B}}}{dg} + c_{\mathrm{L}} \frac{dW_{\mathrm{L}}}{dg}. \tag{A12}$$

If $dW/dg > 0$, then selection favors an increase in the social behavior, and if $dW/dg < 0$, then selection favors a decrease in the social behavior. In the right-hand side of equation (A12), the derivative in the first term can be expanded as follows:

$$\frac{dW_{\mathrm{B}}}{dg} = \frac{\partial W_{\mathrm{B}}}{\partial x_{\mathrm{B}}} \frac{dx_{\mathrm{B}}}{dg_{\mathrm{B}}} \frac{dg_{\mathrm{B}}}{dg} + \frac{\partial W_{\mathrm{B}}}{\partial y_{\mathrm{B}}} \frac{dy_{\mathrm{B}}}{dg'_{\mathrm{B}}} \frac{dg'_{\mathrm{B}}}{dg} + \frac{\partial W_{\mathrm{B}}}{\partial Y_{\mathrm{B}}} \frac{dY_{\mathrm{B}}}{dG_{\mathrm{B}}} \frac{dG_{\mathrm{B}}}{dg}. \tag{A13}$$

All the derivatives are evaluated as $x_{\mathrm{B}} = y_{\mathrm{B}} = Y_{\mathrm{B}} = z_{\mathrm{B}} = z_{\mathrm{L}}$ (Taylor and Frank 1996; Frank 1998). The associations between reproductive success and phenotypes are the effects of the mutant phenotype on the vital rates of the different recipients. The associations between phenotypes and genotype γ constitute the genotype-phenotype mapping, and these can be set to unity: $dx_{\mathrm{B}}/dg_{\mathrm{B}} = dy_{\mathrm{B}}/dg'_{\mathrm{B}} = Y_{\mathrm{B}}/dG_{\mathrm{B}} = \gamma_{\mathrm{B}} = 1$. The associations between genotypes and genic values are the coefficients of consanguinity of the different recipients in relation to the actor. We normalize these coefficients of consanguinity, which give the coefficients of relatedness of the different recipients (Bulmer 1994). These are derived in the next section.

Relatedness

We derive the kin-selection coefficients of relatedness, assuming a neutral population (Wright 1969; Taylor 1992; Rousset 2004). In a focal patch after dispersal, two offspring chosen at random are philopatric to the patch with probability h.

With probability $1/n$ they are siblings and have relatedness 1. With probability $1 - (1/n)$ they are not siblings and they have relatedness r. Hence, relatedness among two offspring chosen at random in a focal patch is given by

$$Q_{\mathrm{B}} = h_{\mathrm{B}}\left[\frac{1}{n_{\mathrm{B}}} + \left(1 - \frac{1}{n_{B}}\right)r_{\mathrm{B}}\right] \tag{A14}$$

and

$$Q_{\mathrm{L}} = h_{\mathrm{L}}\left[\frac{1}{n_{\mathrm{L}}} + \left(1 - \frac{1}{n_{\mathrm{L}}}\right)r_{\mathrm{L}}\right] \tag{A15}$$

for big and little patches, respectively. A focal big patch was big in the previous generation with probability π_{B}, in which case relatedness among patchmates is Q_{B}. The same patch was little in the previous generation with probability $1 - \pi_{\mathrm{B}}$, in which case relatedness among patchmates is Q_{L}. A focal little patch was little in the previous generation with probability π_{L}, in which case relatedness among patchmates is Q_{L}. The same patch was big with probability $1 - \pi_{\mathrm{L}}$, in which case relatedness among patchmates is Q_{B}. This means that relatedness among two patchmates in the next generation is given by

$$\begin{aligned} r'_{\mathrm{B}} &= \pi_{\mathrm{B}}Q_{\mathrm{B}} + (1 - \pi_{\mathrm{B}})Q_{\mathrm{L}}, \\ r'_{\mathrm{L}} &= (1 - \pi_{\mathrm{L}})Q_{\mathrm{B}} + \pi_{\mathrm{L}}Q_{\mathrm{L}}. \end{aligned} \tag{A16}$$

At equilibrium, $r'_{\mathrm{B}} = r_{\mathrm{B}}$ and $r'_{\mathrm{L}} = r_{\mathrm{L}}$, and we can solve equation system (A16) to obtain the "other-only" kin-selection coefficients of relatedness (Pepper 2000). These are given by

$$r_{\mathrm{B}} = \frac{h_{\mathrm{L}}n_{\mathrm{B}}(1 - \pi_{\mathrm{B}}) + h_{\mathrm{B}}[n_{\mathrm{L}}\pi_{\mathrm{B}} + h_{\mathrm{L}}(n_{\mathrm{L}} - 1)(1 - \pi_{\mathrm{B}} - \pi_{\mathrm{L}})]}{n_{\mathrm{B}}[n_{\mathrm{L}} - (n_{\mathrm{L}} - 1)h_{\mathrm{L}}\pi_{\mathrm{L}}] - h_{\mathrm{B}}(n_{\mathrm{B}} - 1)[n_{\mathrm{L}}\pi_{\mathrm{B}} + h_{\mathrm{L}}(n_{\mathrm{L}} - 1)(1 - \pi_{\mathrm{B}} - \pi_{\mathrm{L}})]} \tag{A17}$$

and

$$r_{\mathrm{L}} = \frac{h_{\mathrm{L}}n_{\mathrm{B}}\pi_{\mathrm{L}} + h_{\mathrm{B}}[n_{\mathrm{L}}(1 - \pi_{\mathrm{L}}) + h_{\mathrm{L}}(n_{\mathrm{B}} - 1)(1 - \pi_{\mathrm{B}} - \pi_{\mathrm{L}})]}{n_{\mathrm{B}}[n_{\mathrm{L}} - (n_{\mathrm{L}} - 1)h_{\mathrm{L}}\pi_{\mathrm{L}}] - h_{\mathrm{B}}(n_{\mathrm{B}} - 1)[n_{\mathrm{L}}\pi_{\mathrm{B}} + h_{\mathrm{L}}(n_{\mathrm{L}} - 1)(1 - \pi_{\mathrm{B}} - \pi_{\mathrm{L}})]}, \tag{A18}$$

for big and little patches, respectively. The "whole-group" coefficients of relatedness (Pepper 2000) are given by $R = 1/n + [1 - (1/n)]r$. Relatedness of a focal actor in relation to a primary recipient is equal to the relatedness among patchmates. Hence, $r_{\mathrm{P|B}} = r_{\mathrm{B}}$ and $r_{\mathrm{P|L}} = r_{\mathrm{L}}$ for big and little patches, respectively. Relatedness of an actor in relation to a secondary recipient depends on the probability of cophilopatry and the "whole-group" relatedness. This is given by $r_{\mathrm{S|B}} = h_{\mathrm{B}}R_{\mathrm{B}}$ and $r_{\mathrm{S|L}} = h_{\mathrm{L}}R_{\mathrm{L}}$ for big and little patches, respectively.

In spatially heterogeneous populations, patches do not undergo changes in size; hence, $\pi_{\mathrm{B}} = 1$ and $\pi_{\mathrm{L}} = 1$. This means that relatedness of primary recipients in big patches is $r_{\mathrm{P|B}} = h_{\mathrm{B}}R_{\mathrm{B}}$. Likewise, relatedness of primary recipients in little patches is $r_{\mathrm{P|L}} = h_{\mathrm{L}}R_{\mathrm{L}}$.

Reproductive Value

The reproductive values of individuals are given by the left eigenvector of matrix **w** (Taylor 1990; Grafen 2006). The individual reproductive value is the compound value of the number of offspring times their reproductive value. Multiplying individual reproductive value by group size gives the patch reproductive values: $v_{\mathrm{B}} = n_{\mathrm{B}}\nu_{\mathrm{B}}$ and $v_{\mathrm{L}} = n_{\mathrm{L}}\nu_{\mathrm{L}}$ for big and little patches, respectively. Relative patch reproductive values are given by

$$v_{\mathrm{S|B}} = \frac{\alpha v_{\mathrm{B}} + (1 - \alpha)v_{\mathrm{L}}}{v_{\mathrm{B}}} \tag{A19}$$

and

$$v_{\mathrm{S|L}} = \frac{\beta v_{\mathrm{L}} + (1 - \beta)v_{\mathrm{B}}}{v_{\mathrm{L}}}, \tag{A20}$$

for big and little patches, respectively.

Cancellation of Obligate Helping and Harming

Taylor's (1992*a*) cancellation result occurs because, in his model, the relatedness of primary recipients is equal to that of secondary recipients, $r_{\mathrm{P|T}} = r_{\mathrm{S|T}}$. This occurs because the probability of cophilopatry used in the derivation of relatedness, h_{T}, is equal to the scale of competition: $h_{\mathrm{T}} = a_{\mathrm{T}}$ (Frank 1998). This identity still holds for fluctuating environments: $h_{\mathrm{B}} = a_{\mathrm{B}}$ and $h_{\mathrm{L}} = a_{\mathrm{L}}$. We find additional symmetries that lead to the cancellation result for obligate social behavior. We can write the different quantities in Hamilton's rule using this notation, such that $\pi_{\mathrm{B}} = P(X_{t-1} = \mathrm{B}|X_t = \mathrm{B})$, $1 - \pi_{\mathrm{B}} = P(X_{t-1} = \mathrm{L}|X_t = \mathrm{B})$, $\pi_{\mathrm{L}} = P(X_{t-1} = \mathrm{L}|X_t = \mathrm{L})$, $1 - \pi_{\mathrm{L}} = P(X_{t-1} = \mathrm{B}|X_t = \mathrm{L})$, $\alpha = P(X_{t+1} = \mathrm{B}|X_t = \mathrm{B})$, $1 - \alpha = P(X_{t+1} = \mathrm{L}|X_t = \mathrm{B})$, $\beta = P(X_{t+1} = \mathrm{L}|X_t = \mathrm{L})$, and $1 - \beta = P(X_{t+1} = \mathrm{B}|X_t = \mathrm{L})$. As a result, we can write the life-for-life relatedness (Hamilton 1972; Bulmer 1994) as

$$\begin{aligned}\rho_{\mathrm{P}} &= p_{\mathrm{B}}(P(X_{t+1} = \mathrm{B}|X_t = \mathrm{B})h_{\mathrm{B}}R_{\mathrm{B}}v_B + P(X_{t+1} = \mathrm{L}|X_t = \mathrm{B})h_{\mathrm{L}}R_{\mathrm{L}}v_{\mathrm{B}})\\ &\quad + p_{\mathrm{L}}(P(X_{t+1} = \mathrm{B}|X_t = \mathrm{L})h_{\mathrm{B}}R_{\mathrm{B}}v_{\mathrm{L}} + P(X_{t+1} = \mathrm{L}|X_t = \mathrm{L})h_{\mathrm{L}}R_{\mathrm{L}}v_{\mathrm{L}}), \qquad \text{(A21)}\\ \rho_{\mathrm{S}} &= p_{\mathrm{B}}(P(X_{t+1} = \mathrm{B}|X_t = \mathrm{B})h_{\mathrm{B}}R_{\mathrm{B}}v_{\mathrm{B}} + P(X_{t+1} = \mathrm{L}|X_t = \mathrm{B})h_{\mathrm{B}}R_{\mathrm{B}}v_{\mathrm{L}})\\ &\quad + p_{\mathrm{L}}(P(X_{t+1} = \mathrm{L}|X_t = \mathrm{L})h_{\mathrm{L}}R_{\mathrm{L}}v_{\mathrm{L}} + P(X_{t+1} = \mathrm{B}|X_t = \mathrm{L})h_{\mathrm{L}}R_{\mathrm{L}}v_{\mathrm{B}}). \qquad \text{(A22)}\end{aligned}$$

We can prove that the life-for-life relatedness coefficients cancel each other in a pairwise way. For example, first note that at equilibrium, $\forall t$: $P(X_t = \mathrm{B}) = p_{\mathrm{B}} = p$ and $P(X_t = \mathrm{L}) = p_{\mathrm{L}} = 1 - p$; second, from Bayes's theorem we find that $P(X_{t-1} = \mathrm{L}|X_t = \mathrm{B}) = P(X_t = \mathrm{B}|X_{t-1} = \mathrm{L})p_{\mathrm{L}}/p_{\mathrm{B}}$; third, because the patch dynamics is described by a time-homogeneous Markov chain, we have $P(X_t = \mathrm{B}|X_{t-1} = \mathrm{L}) = P(X_{t+1} = \mathrm{B}|X_t = \mathrm{L})$; as a consequence, we find that $p_{\mathrm{B}}P(X_{t-1} = \mathrm{L}|X_t = \mathrm{B}) = p_{\mathrm{L}}P(X_{t+1} = \mathrm{B}|X_t = \mathrm{L})$. We can generalize this result for all pairwise terms in equations (A14) and (A15). As a result, the value of primary recipients is equal to the value of secondary recipients, $\rho_{\mathrm{P}} = \rho_{\mathrm{S}}$, and therefore obligate social behavior is never favored.

Temporal Heterogeneity

We assume that all patches become big with probability p or become little with probability $1 - p$, independently of the present state of the population (cf. Iizuka 2001; Rousset 2004, ch. 10). As a consequence, at any given generation, the probability that patches are in the big state is p, while the probability that patches are in the little state is $1 - p$. Hence, over time, the expected number of individuals in the big state is $u_{\mathrm{B}} = pn_{\mathrm{B}}$, while the expected number of individuals in the little state is $u_{\mathrm{L}} = (1 - p)n_{\mathrm{L}}$. Given that the population is in the big state, the probability that it remains big is p, in which case the fecundity of a focal individual in a neutral population is 1. With probability $1 - p$, the population changes to the little state, in which case the fecundity of a focal individual in a neutral population is $n_{\mathrm{L}}/n_{\mathrm{B}}$. Hence, at equilibrium, the reproductive value of a focal individual in a big patch is $v_{\mathrm{B}} = pv_{\mathrm{B}} + (1 - p)(n_{\mathrm{L}}/n_{\mathrm{B}})v_{\mathrm{L}}$. This means that $v_{\mathrm{B}} = (n_{\mathrm{L}}/n_{\mathrm{B}})v_{\mathrm{L}}$. We can normalize the reproductive values such that $v_{\mathrm{B}} = 1/n_{\mathrm{B}}$ and $v_{\mathrm{L}} = 1/n_{\mathrm{L}}$. Note that the probability that the population was in the big state in a previous generation is independent of the present state of the population, such that $\pi_B = 1 - \pi_{\mathrm{L}} = p$ and $1 - \pi_{\mathrm{B}} = \pi_{\mathrm{L}} = 1 - p$. As a consequence, relatedness coefficients obey the following identities: $r_{\mathrm{B}} = phR_{\mathrm{B}} + (1 - p)hR_{\mathrm{L}}$ and $r_{\mathrm{L}} = phR_{\mathrm{B}} + (1 - p)hR_{\mathrm{L}}$. Hence, $r_{\mathrm{B}} = r_{\mathrm{L}}$ and $r_{\mathrm{P|B}} = r_{\mathrm{P|L}}$.

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EVOLUTION OF HELPING AND HARMING IN HETEROGENEOUS GROUPS

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Social groups are often composed of individuals who differ in many respects. Theoretical studies on the evolution of helping and harming behaviors have largely focused upon genetic differences between individuals. However, nongenetic variation between group members is widespread in natural populations, and may mediate differences in individuals' social behavior. Here, we develop a framework to study how variation in individual quality mediates the evolution of unconditional and conditional social traits. We investigate the scope for the evolution of social traits that are conditional on the quality of the actor and/or recipients. We find that asymmetries in individual quality can lead to the evolution of plastic traits with different individuals expressing helping and harming traits within the same group. In this context, population viscosity can mediate the evolution of social traits, and local competition can promote both helping and harming behaviors. Furthermore, asymmetries in individual quality can lead to the evolution of competition-like traits between clonal individuals. Overall, we highlight the importance of asymmetries in individual quality, including differences in reproductive value and the ability to engage in successful social interactions, in mediating the evolution of helping and harming behaviors.

KEY WORDS: **Individual variation, kin selection, nongenetic differences, quality discrimination, reproductive value, social value.**

> *The worst pathologies of the kin-selection criterion arise when genes for social behavior are unconditionally expressed—i.e. expressed by every individual of a given genotype ... there are numerous asymmetries that make conditional expression the natural course ... Conditionality, although mentioned, was insufficiently emphasized in my previous work.*
>
> Hamilton (1987, p. 433)

Understanding cooperation is a major challenge for evolutionary biology (Hamilton 1996; Sachs et al. 2004; West et al. 2007; Bourke 2011). Hamilton's (1963, 1964, 1970, 1972, 1975) theory of inclusive fitness provides a general explanation for the adaptive evolution of cooperation. The idea is that individuals can increase their genetic representation in future populations not only by improving their own reproductive success, but also by improving the reproductive success of their genetic relatives. The key result of inclusive fitness theory is Hamilton's rule, which states that natural selection will favor an increase in any trait if $-c + rb > 0$, where c is the direct fitness cost to the actor, b is the benefit enjoyed by the recipient, and r is the genetic relatedness between actor and recipient (Hamilton 1963, 1964, 1970; Charnov 1977). Relatedness has often taken central stage in the literature on inclusive fitness (Lehmann and Keller 2006; Bourke 2011), and is understood to be an important driver of a diversity of evolutionary phenomena, including eusociality (Boomsma 2007, 2009; Hughes et al. 2008), cooperative breeding (Cornwallis et al. 2010), multicellularity (Dawkins 1982; Maynard Smith 1988; Maynard Smith and Szathmáry 1995; Grosberg and Strathmann 1998, 2007), sex allocation (Hamilton 1967; Charnov 1982; West 2009), virulence (Frank 1996b), parent–offspring conflict (Trivers 1974), and genomic imprinting (Haig 2002).

One mechanism that can give rise to relatedness between social partners is population viscosity, whereby individuals do not move far from their place of origin during their lives (Hamilton 1964, 1971). In such scenarios, even indiscriminate cooperation toward one's neighbors is likely to involve significant genetic

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relatedness between interactants. However, limited dispersal may also increase resource competition among social partners, and this tends to inhibit the evolution of cooperation (Hamilton 1971; Taylor 1992a; Queller 1992; Frank 1998; West et al. 2002). In the simplest models of population viscosity these two effects exactly cancel, giving no net effect of dispersal on the evolution of cooperation (Taylor 1992a, b; Kümmerli et al. 2009). This result has motivated a large body of theoretical (and, increasingly, empirical) research on factors that may decouple relatedness and competition to allow viscosity to promote the evolution of helping and harming behaviors (reviewed by West et al. 2002; Lehmann and Rousset 2010).

Most of this literature has assumed that populations are homogeneous in individual quality, other than differences relating to sex (but see Frank 1996a, 2003, 2010; Johnstone 2008; Rodrigues and Gardner 2012, 2013). However, within-group heterogeneities are likely to be important for the evolution of social behavior, especially in determining which individuals enact which behaviors (Hamilton 1964; Alexander 1974; Michener and Brothers 1974; West-Eberhard 1975; Milinski 1978; Rubenstein 1982; Craig 1983; Alexander et al. 1991; Pamilo 1991; Crozier and Pamilo 1996; West-Eberhard 2003; Bourke 2007; West et al. 2007). In social amoebae, larger cells tend to develop as reproductive spores rather than altruistic stalk (Leach et al. 1973). In brewer's yeast, damaged cells appear more predisposed to self-sacrifice (Fabrizio et al. 2004; Herker et al. 2004). In primitively eusocial insects, individuals of better nutritional state are more likely to develop as reproductives (Gadagkar et al. 1988), and workers kill their queen if her fecundity drops below a threshold (Bourke 1994). Thus, a better understanding of how within-group variation in individual quality mediates the evolution of helping and harming is desired.

Here, we investigate how within-group variation in quality influences the evolution of helping and harming behaviors. We consider both unconditional social behavior and also social behavior that is conditionally adjusted according to the quality of the actor and/or her social partners. We first perform a general analysis that treats genetic relatedness and local competition as separate parameters that can be varied independently of each other (e.g., Frank 1998). We then consider an explicit viscous population model, in which relatedness and local competition emerge from explicit demographic assumptions (e.g., Taylor 1992a). We use our analyses as a foundation upon which to discuss the role of individual quality in mediating the evolution of social behaviors.

General Model

BASIC MODEL

We assume a population of haploid, asexually reproducing individuals of which a fraction u_H is high quality and a fraction u_L is low quality. Low-quality individuals have a fecundity that is a fraction $1 - s$ of that of high-quality individuals. Offspring quality is assigned at random, the individual being high quality with probability p and low quality with probability $1 - p$, independently of parent quality.

Juveniles engage in social interactions that mediate survival to adulthood. Specifically, high-quality juveniles survive with probability $S_H(x_H,y_H,y_L)$, where x_H is the focal juvenile's phenotype, y_H is the average phenotype among high-quality juveniles in the local neighborhood, and y_L is the average phenotype among low-quality juveniles in the local neighborhood; and low-quality juveniles survive with probability $S_L(x_L,y_H,y_L)$, where x_L is the focal juvenile's phenotype. We classify social behavior according to two factors: first, whether it is conditioned on the actor's quality and second, whether it is conditioned on the recipient's quality.

The marginal survival cost of social behavior is $\partial S_H(x_H,y_H,y_L)/\partial x_H = -C$ and $\partial S_L(x_L,y_H,y_L)/\partial x_L = -C$ for high-quality and low-quality juveniles, respectively. The marginal survival benefit depends on the type of social interaction. For example, if there is no discrimination of recipients' quality and focusing on a focal high-quality individual, the marginal survival benefit is $\partial S_H(x_H,y_H,y_L)/\partial y_H = u_H B$ and $\partial S_H(x_H,y_H,y_L)/\partial y_L = u_L B$; whereas, if there is discrimination of recipients' quality, and high-quality individuals are the sole recipients of the social behaviors, the marginal survival benefit is $\partial S_H(x_H,y_H,y_L)/\partial y_H = B$ and $\partial S_H(x_H,y_H,y_L)/\partial y_L = (u_L/u_H)B$. Similar functions can be derived for all other types of behavior as well as for low-quality individuals. We assume that the costs and benefits of social actions are small, that is C, $B << 1$, and we scale survival probability such that the baseline is one (see Appendix S1 for details).

After social interactions, juveniles compete for scarce resources, where a fraction a of the competition is among juveniles in the local neighborhood, and a fraction $1 - a$ of the competition occurs globally (Frank 1998). Thus, fitness is

$$W_H = \frac{S_H(x_H, y_H, y_L)}{\left(\begin{array}{l} a(u_H S_H(y_H, y_L) + u_L S_L(y_H, y_L)) \\ \quad + (1-a)(u_H S_H(z_H, z_L) + u_L S_L(z_H, z_L)) \end{array}\right)} \tag{1}$$

and

$$W_L = \frac{S_L(x_L, y_H, y_L)}{\left(\begin{array}{l} a(u_H S_H(y_H, y_L) + u_L S_L(y_H, y_L)) \\ \quad + (1-a)(u_H S_H(z_H, z_L) + u_L S_L(z_H, z_L)) \end{array}\right)}, \tag{2}$$

for high-quality and low-quality juveniles, respectively (see Appendix S2 for details). The denominator represents the amount of competition for a focal individual, where z_H and z_L are the population average phenotypes of high-quality and low-quality juveniles, respectively. Note that, owing to the assumption of small marginal effects and identical baseline survival between the two classes, the expectation of fitness over all high-quality

Table 1. A summary of model notation.

Symbol	Meaning
H	High-quality individual
L	Low-quality individual
B	Both high- and low-quality individuals
F	Corresponding to Frank's (1998) analysis
T	Corresponding to Taylor's (1992a) analysis
A	Actor
P	Primary recipient
S	Secondary recipient
a	Scale of competition
A_Z	Potential for helping corresponding to reference model Z
$A_{X\|Y,Z}$	Potential for helping where actor is in condition X, and primary recipients are in condition Y, for reference model Z
p	Probability that an offspring becomes a high-quality juvenile
c_X	Class reproductive value in condition X ($c_X = u_X v_X$)
F_X	Fecundity of a breeding female in condition X ($F_L = (1 - s)F_H$)
h_X	Probability of co-philopatry in condition X
m	Migration rate
n	Group-size
n_e	Effective group-size
ρ_X	Life-for-life relatedness (or value) of an individual in condition X ($\rho_X = \upsilon_X r_X$)
r_X	"Others-only" relatedness of an individual in condition X
s	Reproductive asymmetry
u_X	Frequency of individuals in condition X
v_X	Reproductive value of an individual in condition X
υ_X	Relative reproductive value of an individual in condition X ($\upsilon_X = v_X/v_A$)
x	Average social behavior of focal actor
y	Average social behavior of juveniles in focal patch
z	Average social behavior of juveniles in population
z^*	Convergence stable strategy for the social behavior

individuals, and the expectation of fitness over all low-quality individuals, is unity. Natural selection favors those heritable traits that are, on average, associated with higher fitness. The appropriate average of fitness is given by $W = c_H W_H + c_L W_L$, where c_H and c_L are the class-reproductive values for high-quality and low-quality individuals, respectively (Fisher 1930; Price and Smith 1972; Taylor 1990; Taylor and Frank 1996; Frank 1998; Grafen 2006; see Appendix S3 for details). Model notation is summarized in Table 1.

EVOLUTION OF HELPING AND HARMING

We classify social behaviors according to their impact upon the survival of group mates (Lehmann et al. 2006; West and Gardner 2010). Specifically, helping behaviors are those where $B > 0$, and harming behaviors as those where $B < 0$. We use the neighbor-modulated fitness approach to kin-selection analysis (Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al. 2007) to determine the direction of selection acting upon social traits (see Appendices S2–S4 for details). This yields a form of Hamilton's (1963, 1964, 1970, 1972) rule:

$$-v_A C + v_P r_P B - (B - C) v_S r_S > 0. \qquad (3)$$

This Hamilton's rule comprises three additive terms, each of which can be associated with an individual or set of individuals affected by the behavior of the actor. The first term concerns effects of the behavior on the actor; the second term concerns the immediate effects of the behavior on social partners, that is, primary recipients; and the third term concerns the kin competition effects of the behavior on social partners, that is, secondary recipients.

Thus, inequality (3) can be read as follows: v_A is the actor's reproductive value (Fisher 1930; Taylor 1990, 1996; Grafen 2006); v_P is the reproductive value of the primary recipients; v_S is the reproductive value of the secondary recipients; $r_P = r$ is the relatedness between actor and primary recipients (Hamilton 1964, 1970; Grafen 1985); $r_S = ar$ is the relatedness between actor and secondary recipients (Hamilton 1964, 1970; Grafen 1985), where a is the scale of competition (Frank 1998), that is the proportion of secondary recipients who are also primary recipients. Note that if the scale of competition is one ($a = 1$), then relatedness of secondary recipients is equal to that of primary recipients ($r_S = r_P = r$), and that if the scale of competition is zero ($a = 0$), then the relatedness of secondary recipients is zero ($r_S = 0$). Also, because juvenile quality is assigned at random, relatedness is independent of actor and recipient class.

Consequently, an actor's social behavior has a three-fold impact upon her own inclusive fitness. First, the actor pays a survival cost C, and this is weighted by actor's reproductive value v_A. Second, primary recipients receive a survival benefit B, and this is weighted by primary recipient relatedness r_P and reproductive value v_P. Third, owing to the impact of the social behavior upon local competition, secondary recipients suffer a cost $B - C$, and this is weighted by secondary recipient relatedness r_S and reproductive value v_S,

Inequality (3) can be normalized with respect to the actor's reproductive value, to derive what we call relative reproductive values (υ). Note that in this case actor's relative reproductive value is one. Thus, the weights of the marginal survival effects on primary and secondary recipients are now the product of relative reproductive value and relatedness, that is "life-for-life relatedness" (ρ; Hamilton 1972; Bulmer 1994). Specifically, the life-for-life relatedness of primary recipients is $\rho_P = \upsilon_P r$, and the life-for-life relatedness of secondary recipients is $\rho_S = \upsilon_S ar$, where υ_P

$= v_P/v_A$ and $\upsilon_S = v_S/v_A$ are the relative reproductive values of primary and secondary recipients, respectively. Life-for-life relatedness describes how much an actor values the recipients of her actions, taking into account not only the extent to which they share genes in common (r), but also their relative capacity to transmit genes to future generations (υ; see Appendix S3 for details).

Setting the left-hand side (LHS) of inequality (3) equal to 0, we find the condition where the actor is indifferent with regards to helping/harming slightly more versus slightly less. This condition can be re-arranged into the form $C/B = A$, where A defines the potential for helping (Rodrigues and Gardner 2012; cf. Gardner 2010) and $-A$ defines the potential for harming (Rodrigues and Gardner 2012). Assuming that the life-for-life relatedness of a secondary recipient is less than that of the actor ($\rho_S < 1$), then $A = (\rho_P - \rho_S)/(1 - \rho_S)$. Consequently: if the life-for-life relatedness of a primary recipient exceeds that of a secondary recipient ($\rho_P > \rho_S$), there is potential for helping ($A > 0$); whereas, if the life-for-life relatedness of a secondary recipient exceeds that of a primary recipients ($\rho_P < \rho_S$), there is potential for harming. In contrast, if the life-for-life relatedness of a secondary recipient is greater than that of the actor ($\rho_S \geq 1$), actors are favored to invest all of their resources into helping or harming, because any cost incurred by an actor is fully compensated by benefits to secondary recipients. This may be better understood if one rewrites Hamilton's rule (inequality 3) in the form $-(1 - \rho_S)C + (\rho_P - \rho_S)B > 0$: if $\rho_S \geq 1$ then $-(1 - \rho_S)C > 0$, which means that the actor's inclusive fitness increases with increasing personal survival cost, even in the absence of any survival benefit to primary recipients. Whether the actor is selected to invest into helping or harming depends upon the value of primary versus secondary recipients ($\rho_P > \rho_S$ vs. $\rho_P < \rho_S$). We establish a convention of setting $A = \infty$ if $\rho_P > \rho_S$, and $A = -\infty$ if $\rho_P < \rho_S$. Thus, we have

$$\begin{cases} A = (\rho_P - \rho_S)/(1 - \rho_S), & \text{if } \rho_S < 1, \\ A = \infty, & \text{if } \rho_S \geq 1 \text{ and } \rho_P > \rho_S, \\ A = -\infty, & \text{if } \rho_S \geq 1 \text{ and } \rho_P < \rho_S, \end{cases} \tag{4}$$

where the last two lines define the region of parameter space where actors are selected to invest all of their resources into social interactions. This effect was foreshadowed by Dawkins (1976, p. 130): "As soon as a runt becomes so small and weak that his expectation of life is reduced to the point where benefit to him due to parental investment is less than half the benefit that the same investment could potentially confer on the other babies, the runt should die gracefully and willingly … he should give up and preferably let himself be eaten by his litter-mates or his parents." We term the portion of parameter space in which the potential for helping has infinite magnitude the "Dawkins' Runt" region. The potential for helping for the different types of behavior is given in Table 2.

Table 2. The potential for helping (A) in the general model is shown for different combinations of the actor's quality and the primary recipients' quality.

	Recipients		
Actors	Both	High	Low
Both	$\frac{r - ar}{1 - ar}$	$\frac{v_H r - ar}{1 - ar}$	$\frac{v_L r - ar}{1 - ar}$
High	$\frac{r - ar}{v_H - ar}$	$\frac{v_H r - ar}{v_H - ar}$	$\frac{v_L r - ar}{v_H - ar}$
Low	$\frac{r - ar}{v_L - ar}$	$\frac{v_H r - ar}{v_L - ar}$	$\frac{v_L r - ar}{v_L - ar}$

Island Model

MODEL

Here we elaborate upon the model presented in the previous section, making the demography of the population more explicit. We assume that the population is subdivided into an infinite number of social groups with n adults each, that is Wright's (1931) island model. Groups are composed of n_H high-quality adults and n_L low-quality adults, where n_H and n_L are random variables that satisfy the constraint $n = n_H + n_L$. Both high- and low-quality individuals produce a large number of offspring, denoted F_H and $F_L = (1 - s)F_H$, respectively. After reproduction, all adults die, and juveniles engage in social interactions that mediate their survival to adulthood. After social interactions, each juvenile disperses with probability m to a random group or else, with probability $1 - m$, remains in the natal group.

EVOLUTION OF HELPING AND HARMING

As in the previous section, we perform a neighbor-modulated fitness analysis (Taylor and Frank 1996; Frank 1997, 1998; Rousset 2004; Taylor et al. 2007; see Appendices S2–S4 for details) to determine the direction of selection acting on the social trait. This yields a Hamilton's rule that takes the same form as given for the previous model (Inequality 3). However, we find that the scale of competition a is now given by the probability of co-philopatry h, that is the probability that two individuals sampled at random are philopatric to the group. Hence, $a = h = (1 - m)^2$. Moreover, relatedness is given by

$$r = \frac{1}{n_e - (n_e - 1)h}, \tag{5}$$

where n_e is effective group-size (see Appendix S5 for details). Reproductive asymmetry decreases effective group-size: $n_e < n$ for $s > 0$. As a result, relatedness is always greater than that of Taylor's (1992a) model ($r > r_T$; Fig. 1). We find that relatedness is highest when reproductive asymmetry is high (high s) and migration rate is low (low m), and attains a maximum at an intermediate

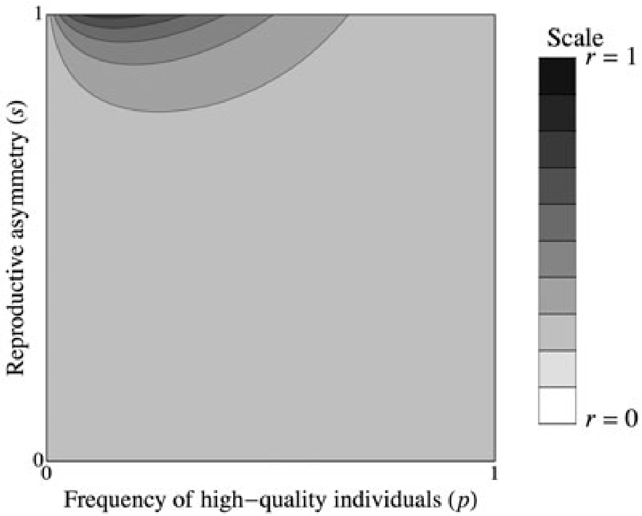

Figure 1. Relatedness as a function of the frequency of high-quality individuals (p) and reproductive asymmetry (s). Darker shades represent higher relatedness. Parameter values: $m = 0.25$ and $n = 10$.

value p^* of the frequency of high-quality individuals, where $p^* \leq \frac{1}{2}$. Furthermore, we find that p^* decreases with increasing reproductive asymmetry. The relatedness of primary recipients is given by $r_P = r$ whereas that of secondary recipients is given by $r_S = (1 - m)^2 r$. Consequently, the relatedness of primary recipients is always greater than or equal to that of secondary recipients ($r_P \geq r_S$), and the difference between the two is greatest when $p = p^*$. As before, we set the LHS of Hamilton's rule to 0 and rearrange to derive the potential for helping A. These results are given in Table 3.

Analysis and Results

For each of our two models, we define a "reference model." In the general-model analysis, the reference model corresponds to a homogeneous population, where each individual's fecundity is identical, that is $s = 0$. This recovers the model of Frank (1998, pp. 114–115), where the potential for helping is $A_F = (r - ar)/(1 - ar)$. Note that under the assumptions of Frank's model, the potential for helping is always nonnegative ($A_F \geq 0$), and is greatest when relatedness is high (high r) and when local competition is low (low a). In the island-model analysis, the reference model also corresponds to a homogeneous population, where each individual's fecundity is identical, that is $s = 0$. This recovers Taylor's (1992a) asexual model. In this case, the potential for helping is given by $A_T = (r_T - h_T r_T)/(1 - h_T r_T)$, where $h_T = (1 - m)^2$ is the probability of co-philopatry. As relatedness is given by $r_T = 1/(n - (n - 1)h_T)$, the potential for helping is given by $A_T = 1/n$. Consequently, the potential for helping is independent of the migration rate (m) and decreases with increasing group size (n). We use these reference models as a benchmark upon which to compare our results for heterogeneous groups ($s > 0$).

We divide our analysis into three sections. In the first section, we consider that actors cannot discriminate recipients' quality. In the second section, we consider that actors can discriminate recipients' quality, and that they direct the social behavior to high-quality recipients only. In the third section, we consider that actors direct the social behavior to low-quality recipients only. In each of the three sections, we consider social behavior that is either unconditional or conditional upon actor's quality.

INDISCRIMINATE SOCIAL BEHAVIOR

Individuals often express different phenotypes independently of their genotype and in ways that correlate with their quality. For example, in yeast, programmed cell death (PCD) is mostly expressed by senescent cells (Fabrizio et al. 2004; Herker et al. 2004). In this section, we assume that actors cannot discriminate the quality of recipients, hence all individuals in the group are equally likely to be recipients of the social actions, and therefore we focus on unconditional and conditional behavior on actor's quality.

We find that the potential for unconditional helping is always nonnegative ($A_{B|B,F} \geq 0$ and $A_{B|B,T} \geq 0$). Furthermore, although in the general model reproductive asymmetry has no impact upon the potential for helping (which is equal to that of Frank's model:

Table 3. The potential for helping (A) in the island model is shown for different combinations of the actor's quality and the primary recipients' quality.

	Recipients		
Actors	Both	High	Low
Both	$\frac{1}{n_e}$	$\frac{v_H - h}{n_e(1-h)}$	$\frac{v_L - h}{n_e(1-h)}$
High	$\frac{1-h}{v_H n_e(1-h) + (v_H - 1)h}$	$\frac{v_H - h}{v_H n_e(1-h) + (v_H - 1)h}$	$\frac{v_L - h}{v_L n_e(1-h) + (v_L - 1)h}$
Low	$\frac{v_H - h}{v_L n_e(1-h) + (v_L - 1)h}$	$\frac{v_H - h}{v_L n_e(1-h) + (v_L - 1)h}$	$\frac{v_L - h}{v_L n_e(1-h) + (v_L - 1)h}$

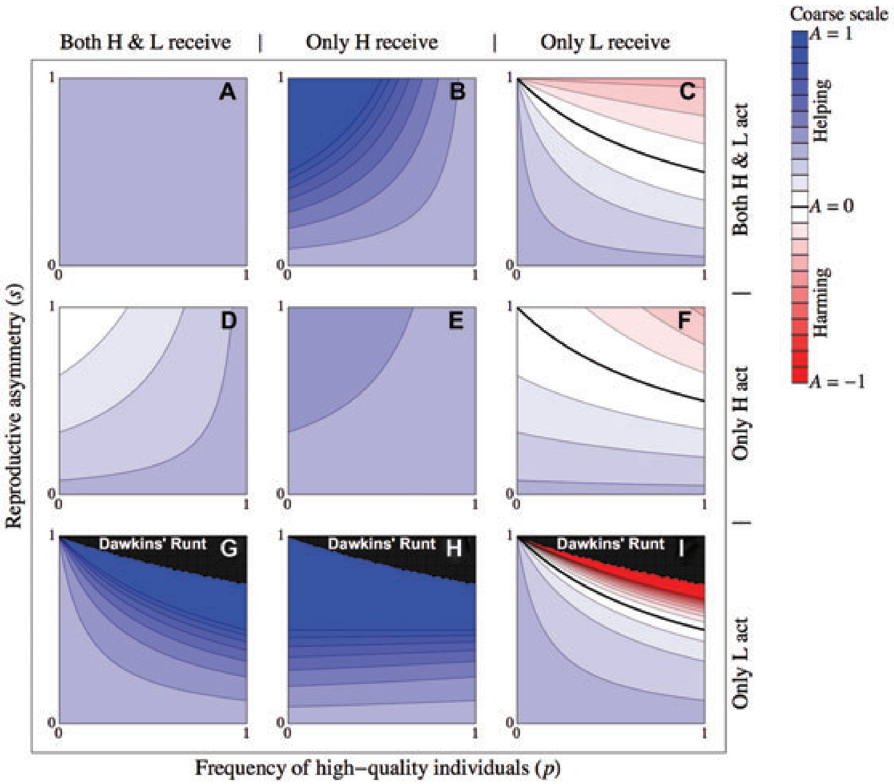

Figure 2. The potential for helping (A) is shown for different combinations of unconditional and conditional social traits. The potential for helping is given as a function of the frequency of high-quality juveniles (p) and the reproductive asymmetry (s). The potential for helping of Frank's (1998) reference model corresponds to $s = 0$. (A) Unconditional social behavior: the potential for helping is equal to that of the reference model ($A_{B|B,F} = A_F$). As a result, the potential for helping is always nonnegative ($A_{B|B,F} \geq 0$). (B) Behavior that is unconditional on actor's quality and is directed to high-quality group mates: the potential for helping is always nonnegative ($A_{B|H,F} \geq 0$), and it is always greater than that of the reference model ($A_{B|H,F} > A_F$). (C) Behavior that is unconditional on actor's quality and is directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{B|L,F} < A_F$); high reproductive asymmetry and high local competition promote harming behavior ($A_{B|L,F} < 0$). (D) Behavior expressed by high-quality juveniles and directed to all group mates: the potential for helping is always nonnegative ($A_{H|B,F} \geq 0$), and it is always less than that of the reference model ($A_{H|B,F} < A_F$). (E) Behavior expressed by high-quality juveniles and directed to high-quality group mates: the potential for helping is always nonnegative ($A_{H|H,F} \geq 0$), and it is always greater than that of the references model ($A_{H|H,F} > A_F$). (F) Behavior expressed by high-quality juveniles directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{H|L,F} < A_F$); high reproductive asymmetry and high local competition promote harming behavior ($A_{H|L,F} < 0$). (G) Behavior expressed by low-quality juveniles and directed to all group mates: the potential for helping is always nonnegative ($A_{L|B,F} \geq 0$), and it is always greater than that of the reference model ($A_{L|B,F} > A_F$). (H) Behavior expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always nonnegative ($A_{L|H,F} \geq 0$), and it is always greater than that of the reference model ($A_{L|H,F} > A_F$). (I) Behavior expressed by low-quality juveniles and directed to low-quality group mates: there is potential for helping and potential for harming ($A_{L|L,F} > 0$ and $A_{L|L,F} < 0$). The potential for helping is always less than that of the reference model ($A_{L|L,F} < A_F$). (G–I) When secondary recipients' value is greater than or equal to actor's value ($\rho_S \geq 1$), there is infinite potential for helping ($A_{L|B,F} = \infty$ and $A_{L|H,F} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{L|L,F} = -\infty$, Dawkins' Runt region, panel I). In all cases, relatedness is set to $r = 0.5$ and local competition is set to $a = 0.5$.

$A_{B|B,F} = A_F$; Fig. 2A, Table 2), in the island model reproductive asymmetry does have an impact upon the potential for helping (which is greater than that of Taylor's model: $A_{B|B,T} > A_T$; Fig. 3A, Table 3). In both models, reproductive asymmetry ($s > 0$) does not lead to differences in the reproductive value of actors, primary recipients and secondary recipients ($v_A = v_P = v_S$). However, in the island model, reproductive asymmetry inflates relatedness ($\mathrm{d}r/\mathrm{d}s > 0$), which, in turn, increases the potential for helping ($\mathrm{d}A/\mathrm{d}s > 0$). Importantly, if the reproductive values of actor, primary recipients, and secondary recipients are equal, the potential for helping is unaffected by the dispersal rate ($\mathrm{d}A_{B|B,T}/\mathrm{d}m = 0$). This recovers and explains Taylor's (1992a) result. If the reproductive values of actor and recipients are not equal, then the potential for helping may be mediated by dispersal rate ($\mathrm{d}A/\mathrm{d}m \neq 0$).

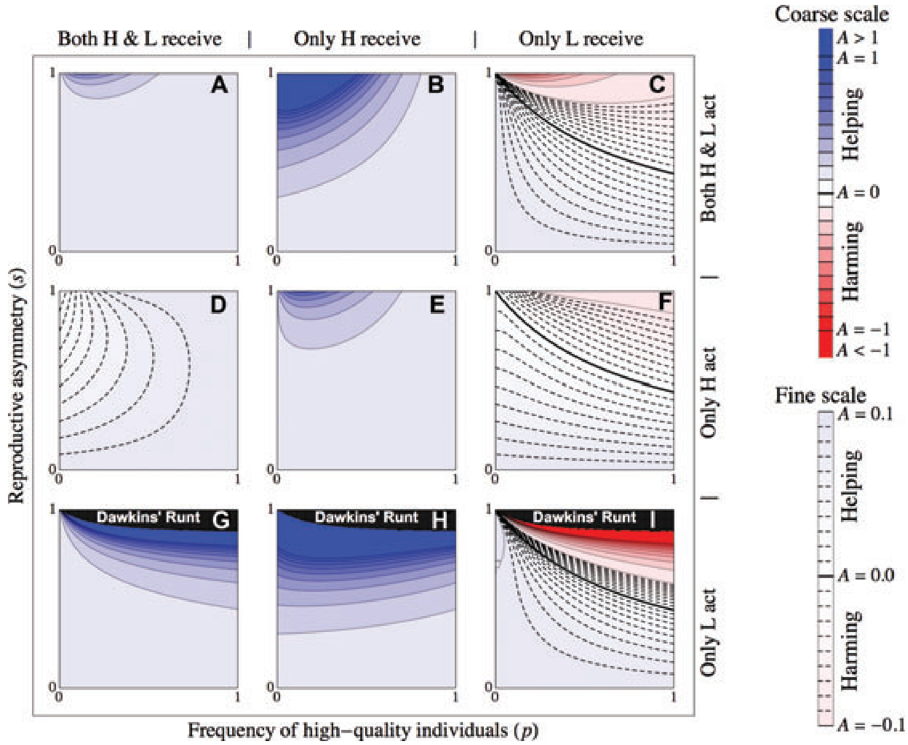

Figure 3. The potential for helping (A) is shown for different combinations of unconditional and conditional social traits. The potential for helping is given as a function of the frequency of high-quality individuals (p) and reproductive asymmetry (s). The potential for helping of Taylor's (1992a) reference model corresponds to $s = 0$. Darker shades represent higher potential for helping or harming. (A) Unconditional social behavior: the potential for helping is greater than that of the reference model ($A_{B|B,T} > A_T$). (B) Behavior that is unconditional on actor's quality and directed to high-quality group mates: the potential for helping is always nonnegative ($A_{B|H,T} \geq 0$), and it is always greater than that of the reference model ($A_{B|H,T} > A_T$). (C) Behavior that is unconditional on actor's quality and directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{B|L,T} < A_T$). High reproductive asymmetry and high local competition promote harming behavior ($A_{B|L,T} < 0$). (D) Behavior expressed by high-quality juveniles and directed to all group mates: the potential for helping is always nonnegative ($A_{H|B,T} \geq 0$), and it is always less than that of the reference model ($A_{H|B,T} < A_T$). (E) Behavior expressed by high-quality juveniles and direct to high-quality group mates: the potential for helping is always nonnegative ($A_{H|H,T} \geq 0$), and it is always greater than that of the reference model ($A_{H|H,T} > A_T$). (F) Behavior expressed by high-quality juveniles and directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{H|L,T} < A_T$). High reproductive asymmetry and high local competition promote harming behavior ($A_{H|L,T} < 0$). (G) Behavior expressed by low-quality juveniles and directed to all group mates: the potential for helping is always nonnegative ($A_{L|B,T} \geq 0$), and it is always greater than that of the reference model ($A_{L|B,T} > A_T$). (H) Behavior expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always nonnegative ($A_{L|H,T} \geq 0$), and it is always greater than that of the reference model ($A_{L|H,T} \geq A_T$). (I) Behavior expressed by low-quality juveniles and directed to low-quality group mates: there is potential for helping and potential for harming ($A_{L|L,T} > 0$ and $A_{L|L,T} < 0$). For a wide range of parameter values, the potential for helping is less than that of the reference model ($A_{L|L,T} < A_T$). However, if reproductive asymmetry is high and frequency of high-quality juveniles is low, the potential for helping may be greater than that of the reference model ($A_{L|L,T} > A_T$). (G-I) When secondary recipients' value is greater than or equal to actor's value ($\rho_S \geq 1$) there is an infinite potential for helping ($A_{L|B,T} = \infty$ and $A_{L|H,T} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{L|L,T} = -\infty$, Dawkins' Runt region, panel I). In all cases, group size is set to $n = 10$ and migration rate is set to $m = 0.25$.

We now assume that actors can conditionally adjust their behavior according to their own quality. We start by considering conditionally adjusted behavior enacted by high-quality juveniles. We find that the potential for helping is always nonnegative ($A_{H|B,F} \geq 0$ and $A_{H|B,T} \geq 0$). Moreover, reproductive asymmetry reduces the potential for helping in the general model, and, for a wide range of parameters, in the island model ($A_{H|B,F} < A_F$, and often $A_{H|B,T} < A_T$; Figs. 2D, 3D, Tables 2 and 3). We find that the reproductive value of primary recipients is equal to that of secondary recipients ($v_P = v_S$). The reproductive value of the actor is always greater than that of the primary and secondary recipients ($v_A > v_P = v_S$). This decreases the potential for helping relative to that of the reference models for two reasons: first, it decreases the difference in life-for-life relatedness between primary and secondary recipients (lower $\rho_P - \rho_S$); second, it increases the difference in life-for-life relatedness between actor and secondary recipients (higher $1 - \rho_S$). Turning our attention to the island model, we find that for extreme values of reproductive asymmetry (high s), sufficiently high migration rate (high m) and intermediate frequency of high-quality juveniles (medium p), there may be a positive effect on the potential for helping, which can be greater than that of the reference model ($A_{H|B,T} > A_T$). This is because, under these circumstances, the actor is more closely related to primary recipients than to secondary recipients.

We now turn our attention to conditional behavior of low-quality juveniles. We find that the potential for helping is always nonnegative ($A_{L|B,F} \geq 0$ and $A_{L|B,T} \geq 0$). Moreover, we find that reproductive asymmetry ($s > 0$) increases the potential for helping ($A_{L|B,F} > A_F$ and $A_{L|B,T} > A_T$; Figs. 2G, 3G, Tables 2, 3). We find that although the reproductive value of primary recipients is equal to that of secondary recipients ($v_P = v_S$), the reproductive value of the actor is always less than that of primary and secondary recipients ($v_A < v_P = v_S$). Consequently, actors put more value on their social partners, and this yields a potential for helping that is greater than that of the reference model. Extreme values of reproductive asymmetry (high s), together with high local competition (low m or high a) and sufficiently high frequency of high-quality juveniles (high p), can lead to the life-for-life relatedness of secondary recipients being greater than or equal to that of the actor ($\rho_S \geq 1$), which favors actors to invest all of their resources into social behavior ($A_{L|B,F} = \infty$ and $A_{L|B,T} = \infty$; Dawkins' Runt region in Figs. 2G, 3G).

QUALITY DISCRIMINATION

Individuals often condition their behavior not only on their own quality, but also on the quality of their social partners. For example, paper-wasp workers appear to directly assess their queen's quality by monitoring her egg production, and behave more selfishly if her fecundity declines (Liebig et al. 2005). In the following two subsections, we consider that actors can discriminate the quality of recipients and can direct their behavior accordingly.

High-quality recipients

Here, we assume that actors direct their actions toward high-quality group mates, who are the sole primary recipients of their social actions. We begin by considering behavior that is expressed independently of the actor's quality. We find that the potential for helping is always nonnegative ($A_{B|H,F} \geq 0$ and $A_{B|H,T} \geq 0$). Furthermore, we find that reproductive asymmetry ($s > 0$) has a positive impact upon the potential for helping ($A_{B|H,T} > A_T$ and $A_{B|H,F} > A_F$; Figs. 2B, 3B, Tables 2, 3). This is because, although the reproductive value of the actor is equal to that of secondary recipients ($v_A = v_S$), the reproductive value of primary recipients is greater than that of secondary recipients ($v_P > v_S$).

We now turn our attention to social behavior that is conditional on the actor's quality. Let us begin by considering that only high-quality juveniles express the behavior. We find that the potential for helping is always nonnegative ($A_{H|H,T} \geq 0$ and $A_{H|H,F} \geq 0$). In addition, reproductive asymmetry ($s > 0$) increases the potential for helping ($A_{H|H,F} > A_F$ and $A_{H|H,T} > A_T$; Figs. 2E, 3E, Tables 2, 3). The reproductive value of primary recipients is greater than that of secondary recipients ($v_P > v_S$) whereas that of the actor is equal to that of primary recipients ($v_A = v_P$). Thus, although there is a positive impact upon the potential for helping in relation to that of the reference model, the potential for conditional helping by high-quality juveniles is less than the potential for helping that is unconditional on the actor's quality ($A_{H|H,F} < A_{B|H,F}$ and $A_{H|H,T} < A_{B|H,T}$).

We now consider that only low-quality juveniles express the behavior. We find that the potential for helping is always nonnegative ($A_{L|H,F} \geq 0$ and $A_{L|H,T} \geq 0$). In addition, reproductive asymmetry ($s > 0$) increases the potential for helping ($A_{L|H,F} > A_F$ and $A_{L|H,T} > A_T$; Figs. 2H, 3H, Tables 2, 3). The reproductive value of primary recipients is greater than that of secondary recipients ($v_P > v_S$). Moreover, the reproductive value of secondary recipients is always greater than that of the actor ($v_S > v_A$). This drives the increase in potential for helping by low-quality juveniles. The life-for-life relatedness of secondary recipients is greater than that of the actor ($\rho_S \geq 1$) when local competition is high (low m or high a), reproductive asymmetry is high (high s), and frequency of high-quality juveniles is high (high p). This promotes the evolution of unconstrained helping, whereby actors are favored to invest all of their resources into helping ($A_{L|H,F} = \infty$ and $A_{L|H,T} = \infty$; Dawkins' Runt region in Figures 2H, 2H).

Low-quality recipients

Here, we consider that individuals can discriminate the quality of recipients, and that low-quality individuals are the sole primary recipients of their social actions. We begin by considering behavior expressed independently of actor's quality. We find that there is both potential for helping and harming ($A_{B|L,F} > 0$ or $A_{B|L,F} < 0$ and $A_{B|L,T} > 0$ or $A_{B|L,T} < 0$). In addition, we also find that, for a wide range of model parameters, reproductive asymmetry ($s > 0$) decreases the potential for helping ($A_{B|L,F} < A_F$ and generally $A_{B|L,T} < A_T$; Figs. 2C, 3C, Tables 2, 3). This occurs because the reproductive value of primary recipients is less than that of secondary recipients ($v_P < v_S$), and the reproductive value of the actor is equal to that of secondary recipients ($v_A = v_S$). However, in the island model, if high-quality juveniles are rare (low p) and migration rate is high (high m), reproductive asymmetry may increase the potential for helping ($A_{B|L,T} > A_T$). This occurs because relatedness of actors to primary recipients is high whereas relatedness to secondary recipients is low. This asymmetry in relatedness offsets the asymmetry in reproductive value.

We now turn our attention to social behavior that is conditional on actor quality. We first consider behavior that is expressed by high-quality juveniles only. We find that there is both potential for helping and harming ($A_{\mathrm{H|L,F}} > 0$ or $A_{\mathrm{H|L,F}} < 0$ and $A_{\mathrm{H|L,T}} > 0$ or $A_{\mathrm{H|L,T}} < 0$). In addition, reproductive asymmetry ($s > 0$) decreases the potential for helping ($A_{\mathrm{H|L,F}} < A_{\mathrm{F}}$ and $A_{\mathrm{H|L,T}} < A_{\mathrm{T}}$; Figs. 2F, 3F, Tables 2, 3). We find that the reproductive value of primary recipients is less than that of secondary recipients ($v_{\mathrm{P}} < v_{\mathrm{S}}$) and that of secondary recipients is always less than that of the actor ($v_{\mathrm{S}} < v_{\mathrm{A}}$). This drives a potential for helping that is always less than that for helping behavior expressed independently of the actor's quality ($A_{\mathrm{H|L,F}} < A_{\mathrm{B|L,F}}$ and $A_{\mathrm{H|L,T}} < A_{\mathrm{B|L,T}}$; if $A_{\mathrm{H|L,F}} > 0$ and $A_{\mathrm{H|L,T}} > 0$), and a potential for harming that is always less than that for harming behavior expressed independently of the actor's quality ($A_{\mathrm{H|L,F}} > A_{\mathrm{B|L,F}}$ and $A_{\mathrm{H|L,T}} > A_{\mathrm{B|L,T}}$; if $A_{\mathrm{H|L,F}} < 0$ and $A_{\mathrm{H|L,T}} < 0$). Harming is most favored when local competition is high (high a or low m), reproductive asymmetry is high (high s), and frequency of high-quality juveniles is high (high p).

We now consider behavior that is expressed by low-quality juveniles only. We find that there is both potential for helping and harming ($A_{\mathrm{L|L,F}} > 0$ or $A_{\mathrm{L|L,F}} < 0$ and $A_{\mathrm{L|L,T}} > 0$ or $A_{\mathrm{L|L,T}} < 0$). We find that reproductive asymmetry ($s > 0$) typically decreases the potential for helping ($A_{\mathrm{L|L,F}} < A_{\mathrm{F}}$ and in general $A_{\mathrm{L|L,T}} < A_{\mathrm{T}}$; Figs. 2I, 3I, Tables 2, 3). We find that the reproductive value of primary recipients is always less than that of secondary recipients ($v_{\mathrm{P}} < v_{\mathrm{S}}$). Low frequency of high-quality individuals (low p), high reproductive asymmetry (high s), and low local competition (low a or high m) favor high potential for helping. By contrast, high frequency of high-quality individuals (high p), and high local competition (high a or low m), favor high potential for harming. The life-for-life relatedness of secondary recipients is greater than or equal to that of the actor ($\rho_{\mathrm{S}} \geq 1$) for higher local competition (higher a or lower m), higher reproductive asymmetries (higher s), and higher frequency of high-quality individuals (higher p). This promotes the evolution of unconstrained helping, whereby actors are favored to invest all of their resources into harming behaviors ($A_{\mathrm{L|H,F}} = -\infty$ and $A_{\mathrm{L|H,T}} = -\infty$; Dawkins' Runt region in Figs. 2I, 3I).

Discussion

HELPING AND HARMING

We have considered how the impact of within-group variation in individual quality drives the evolution of helping and harming behaviors in structured populations. We have found that the exact cancellation of relatedness and kin competition effects of dispersal, observed in the simplest models of population viscosity (Taylor 1992a, b), need no longer obtain. Specifically, the cancellation result requires that the actor, primary recipients, and secondary recipients all have the same (expected) reproductive value and, although this is true for unconditional behavior, it need not be true for behavior that is conditioned on actor and/or recipient quality. Moreover, we have also shown that low-quality individuals may even be favored to invest all of their resources into suicidal helping or harming behaviors. This clarifies that heterogeneity in individual quality may be an important factor in social evolution.

CONDITIONAL PHENOTYPES

We find that selection operating on social traits that are expressed irrespective of the actor's quality can greatly differ from selection operating on social traits that are conditional on the actor's quality. In particular, the potential for helping or harming unconditional on the actor's quality is always intermediate between the potentials for helping or harming conditional on the actor's quality. Moreover, although the potential for helping of high-quality individuals is always less than the potential for helping that is unconditional on the actor's quality, the potential for helping of low-quality individuals is always greater than the potential for helping that is unconditional on the actor's quality. This is because, all else being equal, a high-quality individual has more to lose in terms of direct genetic contribution to future generations (i.e., reproductive value; Fisher 1930), such that any investment in costly behavior has a larger negative impact on its fitness than the same costly investment by a low-quality individual. We suggest that these different selection pressures may provide the basis for the evolution of phenotypic divergence between high- and low-quality individuals, leading these to exhibit very different adaptive phenotypes.

A large number of social species exhibit conditional phenotypes within groups, whereby individuals exhibit differences in morphology and/or behavior independently of their genotypes (Pardi 1948; Wilson 1971; Ross and Matthews 1991; Bourke and Franks 1995; Crozier and Pamilo 1996; Gross 1996; Brockmann 2001; Gadagkar 2001). In agreement with our predictions, there is evidence for helping being associated with low quality (i.e., poor prospects of direct fitness), and selfishness being associated with high quality (i.e., good prospects of direct fitness). For example, in hanuman langurs, older females exhibit traits associated with helping (e.g., group defense) whereas younger females exhibit traits associated with selfishness (e.g., harassment of older females). Hrdy and Hrdy (1976) suggest that this owes to differences in relative reproductive value: as older females have lower reproductive value than younger females, they stand to gain more, relative to younger females, from helping younger females than from investing in their own reproduction.

Another example is provided by several species of primitively eusocial wasps, where individuals in better nutritional status are more likely to adopt reproductive roles (Gadagkar et al.

1988, 1991; O'Donnell 1998; Keeping 2002; Hunt et al. 2007, 2010). The idea that individual condition influences social behavior is also present in the "subfertility hypothesis" (West 1967; West-Eberhard 1969, 1975; Craig 1983), which suggests that individuals with lower reproductive potential should have a higher tendency to become helpers. In our analysis, we have not specified the causes underlying differences in reproductive value. Depending on the specific biological system, these causes could involve multiple factors, such as differences in ovary development (Pardi 1948), infection status (Shykoff and Schmid-Hempel 1991; O'Donnell 1997), or timing of birth (Queller 1989, 1994).

QUALITY DISCRIMINATION

We find that selection operating on social traits that are unconditional on the primary recipients' quality can greatly differ from selection operating in traits that are conditional on the primary recipients' quality. In particular, we find that the potential for helping of traits that are not conditional on recipients' quality is always intermediate between the potentials for helping of traits that are conditional on the recipients' quality. Thus, if actors direct their actions toward high-quality recipients, the potential for helping is always greater than the potential for helping when actors do not discriminate recipients' quality. By contrast, if actors direct their actions toward low-quality recipients, the potential for helping is always lower than the corresponding potential for helping when actors do not discriminate recipients' quality. Furthermore, if the social behavior is directed toward low-quality recipients, harming behavior can be favored. These results suggest that natural selection may favor quality discrimination, as this may have important inclusive-fitness consequences to those involved in social interactions.

Previous work has already highlighted the significance of quality discrimination for the evolution of social traits (West-Eberhard 2003, ch. 25; Keller and Nonacs 1993). Several empirical studies have proposed a number of quality discrimination mechanisms. For example, female paper wasps can discriminate each others' quality by assessing the number of laid eggs (Liebig et al. 2005) or by use of facial marks or patterns (Tibbetts and Dale 2004; Tibbetts 2006; Tibbetts and Curtis 2007; Cervo et al. 2008), and ponerine ants do so by assessing correlates of quality such as cuticular hydrocarbons (Liebig et al. 2000; D'Ettorre et al. 2004). In the context of social insects, workers monitor their queen's quality, and when the queen's quality starts to decline, workers may shift their behavior from helping to aggression, which can culminate in worker matricide (Forsyth 1980; Bourke 1994).

DAWKINS' RUNT

If the differences in the reproductive value of high- and low-quality juveniles are sufficiently large, local competition may drive low-quality juveniles to invest all of their resources into helping. We term an individual that expresses such extreme altruistic behavior a "Dawkins' Runt" (after Dawkins 1976, p. 130). This phenomenon has received some theoretical treatment (e.g., O'Connor 1978; Godfray and Harper 1990), but convincing empirical evidence is lacking. One of the factors that may explain its rarity among traditional organisms is that relatedness is often less than one, which may constrain the evolution of such extreme altruistic behavior. Moreover, a weak offspring may still be able to recover and derive high reproductive success as an adult. More generally, reproductive value should be an important factor mediating the evolution of family interactions.

Altruistic suicide seems to be more common in unicellular organisms, where recent studies have identified a number of mechanisms that are likely to be suicide programmes. Several authors have suggested that mechanisms of cell death have evolved as social traits (Lewis 2000; Longo et al. 2005; Buttner et al. 2006; Nedelcu et al. 2010; Reece et al. 2011). For example, in yeast, PCD is mostly expressed by senescent cells (Fabrizio et al. 2004; Herker et al. 2004). PCD, in these cases, may be favored not only because it alleviates local competition, but also because it enriches the environment with additional resources. In the protozoan parasites *Leishmania*, *Trypanosoma cruzi*, and *Trypanosoma brucei*, PCD has been suggested as a mechanism whereby the best cells are chosen to be transmitted to the next host (Debrabant and Nakhasi 2003; Seed and Wenk 2003). Our analysis suggests that local competition for resources may be key to generating the selection pressure for the suicide of low-quality cells.

More generally, we highlight that cooperation can be favored not only owing to an increase in group productivity, but also owing to an increase in average group quality, that is to say groups that produce fewer but higher-quality individuals may be at an evolutionary advantage in relation to those groups that produce more but lower-quality individuals. Moreover, our framework also predicts that any asymmetry in quality can be reinforced by the action of natural selection. Worse-off cells are selected to give up some of their survival, thereby becoming even more worse-off. This leads to a causal association between helping and survival or, in other words, between kin-selected traits and senescence (e.g., Bourke 2007; Ronce and Promislow 2010).

Ronce and Promislow (2010) used a kin selection model of limited dispersal to study the evolution of senescence. They showed that a mother of age x is selected to give up some of her survival if her expected reproductive value v_{x+1} is less than the probability of offspring's co-philopatry $h(x)$ times their reproductive value v_1 ($v_{x+1} < h(x)v_1$; the condition (3.6) of Ronce and Promislow (2010)). If we consider our model for conditional social behavior, and if we assume that patch size is one ($n = 1$), and that the benefits are zero ($B = 0$), then we find that the selection gradient, as given by Hamilton's rule in inequality (3), is $v_A < hv_S$, which is true when the actor is a low-quality individual: $v_L < hv_S$; and which is equivalent to Ronce and Promislow's (2010) condition (3.6). Note also that this condition says implicitly that the life-for-life relatedness to secondary recipients must be larger than one ($hv_S/v_L = \rho_S > 1$), which is similar to our definition of the Dawkin's Runt region ($\rho_S \geq 1$). Interestingly, empirical studies have suggested that PCD may be associated with benefits provided by the dying cells (i.e., $B > 0$; Herker et al. 2004; Durand et al. 2011). Overall, these findings and studies provide further evidence for an interaction between helping/harming behavior, kin competition and senescence that represents an interesting avenue for future research.

INDIVIDUAL QUALITY

Here we have focused on one component of individual quality that we defined as an individual's reproductive value (Fisher 1930; Grafen 2006), and which emerges as a weighting factor in Hamilton's rule (Hamilton 1972; Taylor 1990; Taylor and Frank 1996; Frank 1998). In our model, differences in reproductive value emerge because of differences in fecundity between high- and low-quality individuals. These differences in fecundity can result in turn from a number of underlying factors. For example, it has been suggested that ovarian development in paper wasps is positively correlated with fecundity (Pardi 1948). The reproductive value that derives from these traits, which can be said to be intrinsic to the individual, has been called "basal" or "solitary" reproductive value (West-Eberhard 2003, p. 451), or "inherent reproductive potential" (Röseler 1991, p. 334).

However, differences in reproductive value can arise for other reasons. For example, an individual can gain a reproductive advantage over group mates by suppressing the group mates' reproduction (Vehrencamp 1983a, b). In these cases, differences in reproductive value emerge due to asymmetries in control over social interactions, and this has been called "social reproductive value" (West-Eberhard 2003, p. 451). Frank (1990, 1998) has analyzed natural selection of social traits in terms of three measures of value: marginal value, relatedness, and reproductive value. The marginal value criterion states that candidate evolutionary end points are reached when the genetic benefits of extra investment in a fitness component are exactly cancelled by associated genetic costs in a different fitness component (Frank 1990). These marginal values are mediated by relatedness and reproductive value, and implicitly by costs and benefits.

In the context of our model, differences in social skills, which we call "social value," are better captured by asymmetries in costs and benefits affecting either survival or fecundity (i.e., B's and C's; see Appendix S6). In the main text, we have assumed that social value is equal among all juveniles (i.e., $C_H = C_L$ and $B_{HH} = B_{HL} = B_{LL} = B_{LH}$), thus juveniles with high reproductive value are selected to invest less in helping than juveniles with low reproductive value. However, this prediction may be reversed if we also allow differences in social value. For example, juveniles with higher reproductive value may be selected to invest more into helping if they also have higher social value (e.g., suffer a lower cost from expressing helping behaviors, $C_H < C_L$; see Appendix S6). In summary, our conceptual framework divides individual quality into reproductive value and social value, both of which interact to mediate selection acting on social traits. Under this conceptual framework, asymmetries in social value may offset asymmetries in reproductive value such that individual quality may be more-or-less uniform among group members, and therefore marked differences among individuals need not lead to differences in social behavior (see Appendix S6).

SOCIAL AMOEBAE

The cellular slime mould *Dictyostelium discoideum* has a peculiar life cycle: when feeding and dividing it lives a relatively solitary life; however, when local resources are depleted, they may initiate aggregation to form a multicellular fruiting body (reviewed by Bonner 2009; Queller et al. 2003). After aggregation, cells either differentiate into stalk cells, dying in the process, or spore cells, which will ultimately disperse to more favorable environments. Thus, stalk-forming cells have been viewed as altruists that benefit the spore-forming cells in the fruiting body (Strassmann et al. 2000).

Most of the research on this topic has been centered on how relatedness mediates the evolution of the altruistic trait (Strassmann et al. 2000; Kuzdzal-Fick et al. 2011). However, several studies have found that stalk-cell differentiation correlates with other factors, such as nutritional status (Leach et al. 1973; Castillo et al. 2011), cell size, or cell-cycle stage (Weijer et al. 1984; McDonald and Durston 1984). These factors do not appear to directly correlate with any characteristic that make cells more efficient stalk cells, and so it is unlikely that a tendency to differentiate as stalk is driven by asymmetries in social value. Rather, these factors may well be correlated with intrinsic differences in their reproductive value. That is, smaller or food-deprived cells may have lower reproductive value, and hence have less to lose by altruistically developing as stalk.

During development, cells produce a differentiation-inducing factor (DIF) that reinforces the predisposition of cells to differentiate into stalk cells (Shaulsky and Loomis 1996). Evidence suggests that the main producers of DIF are cells who already show some predisposition to become spore cells (Kay and Thompson 2001) as well as lower sensitivity to DIF (Thompson and Kay 2000). This has led several authors to interpreted DIF as a competitive trait, whereby fitter cells force weaker cells to differentiate into stalk cells (Atzmony et al. 1997). If this trait has evolved in the context of clonal groups, then standard social evolution theory struggles to explain this "competitive-like" trait. Our results suggest that, owing to reproductive value asymmetries, there is in fact potential for the evolution of competition-like traits, which can function as a mechanism for increasing average group quality. Low-quality cells should voluntarily give up their survival, thus sparing high-quality cells from paying the additional costs of inducing others to cooperate. However, constraints at different levels (e.g., lack of information about relative cell-quality or cells inability to act) may indeed favor the evolution of competition-like traits among genetically identical individuals. This principle may in fact be a general design mechanism of organisms as a way of increasing group average quality in different taxa. This idea is supported by evidence from different species, both unicellular (Khare and Shaulsky 2006) and multicellular (Khare and Shaulsky 2006; Sadras and Denison 2009; see also Livnat and Pippenger 2006).

SOCIAL WASPS

Our results predict that, all else being equal, individuals with lower prospects of direct fitness (i.e., lower reproductive value) should invest more in helping behavior, whereas individuals with higher prospects of direct fitness (i.e., higher reproductive value) should invest less in helping and behave more selfishly. Several species of social wasps are characterized by reproductive and social dominance hierarchies, where top-ranked females with queen-like physiology and/or behavior tend to monopolize reproduction, and where low-ranked females with worker-like physiology and/or behavior tend to have low fecundity (Pardi 1948; West 1967; West-Eberhard 1969; Röseler 1991). Group social dynamics seem to follow the dominance hierarchy, such that if the top-ranked female dies or is lost from the nest it is usually the female just below in rank that assumes the dominant role (Pardi 1948; West-Eberhard 1981; Jeanne 1991). Furthermore, females at the bottom of the hierarchy usually exhibit traits that involve high-risk tasks, whereas high-ranked females are more likely to adopt idle, low-risk behaviors (Gadagkar and Joshi 1983; West-Eberhard 1986; Gadagkar 2001).

Several authors have used verbal arguments based on asymmetries in reproductive value to explain the social structure of these social wasps (West-Eberhard 1981; Jeanne 1991; cf. Field et al. 2006). The basic ideas are: those individuals with lower reproductive value should try to derive indirect fitness benefits and therefore adopt riskier cooperative behaviors; those with higher reproductive value should invest less in riskier cooperative behaviors, and invest more in behavioral or physiological traits associated with their own reproduction; those with the best prospects of future direct fitness should invest their resources in reproductive traits and in other traits that allow them to keep their reproductive dominant role. Although our model does not incorporate all aspects of these groups' biology (e.g., overlapping generations), it yields predictions about how individuals should behave according to their own reproductive value as well as that of their social partners.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Appendix S1. Survival functions.
Appendix S2. Reproductive success.
Appendix S3. Stable-class frequencies and reproductive value.
Appendix S4. Hamilton's rule.
Appendix S5. Relatedness.
Appendix S6. Social value.
Figure S1. Potential for helping and local competition.
Figure S2. Potential for helping and migration rate.

Supporting Information to “Evolution of helping and harming in heterogeneous groups”£

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Content:

Appendix A: Survival functions

Here we define the survival functions for each of the unconditional and conditional behaviours. In general, the survival of a focal high-quality juvenile is $S_H(x_H,y_H,y_L)$ = 1-C_Hx_H+$G_{HH}y_H$+$G_{LH}y_L$, whilst the survival of a focal low-quality juvenile is $S_L(x_L,y_H,y_L)$ = 1-C_Lx_L+$G_{HL}y_H$+$G_{LL}y_L$, where: the base-line survival is set to one; C_H / C_L is the survival cost paid by the focal high- / low-quality juvenile due to its own investment x_H / x_L in the behaviour; G_{HH} / G_{HL} is the net survival gain for the focal high- / low-quality juvenile due to her high-quality group mates’ average investment y_H in the behaviour; finally, G_{LH} / G_{LL} is the net survival gain for the focal high- / low-quality juvenile due to her low-quality group mates’ average investment y_L in the behaviour. The particular forms of the net survival gains (G’s) depend on who performs the behaviour and on who are the beneficiaries of the social behaviour. Thus, if the actors belong to the class of high-quality juveniles and the recipients belong to the class of low-quality juveniles, then the net survival gains are G_{HH} = G_{LH} = G_{LL} = 0, because high-quality juveniles are not beneficiaries and / or low-quality juveniles do not invest in social behaviour (B_{HH} = B_{LH} = B_{LH} = 0); while G_{HL} = $(u_H/u_L)B_{HL}$, because low-quality juveniles are beneficiaries and high-quality juveniles are actors. A summary of the net survival gains for each type of unconditional and conditional behaviour is given in Table A1.

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Actors	Recipients		
	Both	High	Low
Both	$G_{\mathrm{HH}} = u_{\mathrm{H}}B_{\mathrm{HH}}$ $G_{\mathrm{LH}} = u_{\mathrm{L}}B_{\mathrm{LH}}$ $G_{\mathrm{HL}} = u_{\mathrm{H}}B_{\mathrm{HL}}$ $G_{\mathrm{LL}} = u_{\mathrm{L}}B_{\mathrm{LL}}$	$G_{\mathrm{HH}} = B_{\mathrm{HH}}$ $G_{\mathrm{LH}} = (u_{\mathrm{L}}/u_{\mathrm{H}})B_{\mathrm{LH}}$ $G_{\mathrm{HL}} = 0$ $G_{\mathrm{LL}} = 0$	$G_{\mathrm{HH}} = 0$ $G_{\mathrm{LH}} = 0$ $G_{\mathrm{HL}} = (u_{\mathrm{H}}/u_{\mathrm{L}})B_{\mathrm{HL}}$ $G_{\mathrm{LL}} = B_{\mathrm{LL}}$
High	$G_{\mathrm{HH}} = u_{\mathrm{H}}B_{\mathrm{HH}}$ $G_{\mathrm{LH}} = 0$ $G_{\mathrm{HL}} = u_{\mathrm{H}}B_{\mathrm{HL}}$ $G_{\mathrm{LL}} = 0$	$G_{\mathrm{HH}} = B_{\mathrm{HH}}$ $G_{\mathrm{LH}} = 0$ $G_{\mathrm{HL}} = 0$ $G_{\mathrm{LL}} = 0$	$G_{\mathrm{HH}} = 0$ $G_{\mathrm{LH}} = 0$ $G_{\mathrm{HL}} = (u_{\mathrm{H}}/u_{\mathrm{L}})B_{\mathrm{HL}}$ $G_{\mathrm{LL}} = 0$
Low	$G_{\mathrm{HH}} = 0$ $G_{\mathrm{LH}} = u_{\mathrm{L}}B_{\mathrm{LH}}$ $G_{\mathrm{HL}} = 0$ $G_{\mathrm{LL}} = u_{\mathrm{L}}B_{\mathrm{LL}}$	$G_{\mathrm{HH}} = 0$ $G_{\mathrm{LH}} = (u_{\mathrm{L}}/u_{\mathrm{H}})B_{\mathrm{LH}}$ $G_{\mathrm{HL}} = 0$ $G_{\mathrm{LL}} = 0$	$G_{\mathrm{HH}} = 0$ $G_{\mathrm{LH}} = 0$ $G_{\mathrm{HL}} = 0$ $G_{\mathrm{LL}} = B_{\mathrm{LL}}$

Table A1 | The net survival benefits (G's) are shown for different combinations of the actor's quality and the primary recipients' quality.

In the main text, we assume that survival marginal costs and benefits are independent of actors' and recipients' quality, and therefore they are identical, $C_{\mathrm{H}} = C_{\mathrm{L}} = C$ and $B_{\mathrm{HH}} = B_{\mathrm{HL}} = B_{\mathrm{LH}} = B_{\mathrm{LL}} = B$. More generally, individuals may exhibit asymmetries in social value (see Discussion in the main text). In appendix F, we provide an illustrative example where social value among individuals is asymmetric, and therefore C's and B's may depend on the actors' and the recipients' quality.

Appendix B: Reproductive success

Following the life-cycle described in the main text for the island model, we define the class-specific reproductive success of a focal juvenile, that is, the expected number of offspring belonging to a particular class produced by a focal juvenile of a given class. Thus, the reproductive success of a high-quality juvenile that stays in her natal patch via her offspring that become high-quality juveniles is

$$w_{\mathrm{P,H\to H}} = \frac{S_{\mathrm{H}}(x_H, y_{\mathrm{H}}, y_{\mathrm{L}})(1-m)nF_{\mathrm{H}}p}{(1-m)\big(N_{\mathrm{H}}S_{\mathrm{H}}(y_{\mathrm{H}}, y_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(y_{\mathrm{H}}, y_{\mathrm{L}})\big) + m\big(N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})\big)}, \tag{A1}$$

where N_{H} and N_{L} is the number of high- and low-quality juveniles, respectively. The reproductive success of a high-quality juvenile that disperses away from her natal patch via her offspring that become high-quality juveniles is

$$w_{\mathrm{D,H\to H}} = \frac{S_{\mathrm{H}}(x_H, y_{\mathrm{H}}, y_{\mathrm{L}})mnF_{\mathrm{H}}p}{N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})}. \tag{A2}$$

The sum of these two components of a focal high-quality juvenile's reproductive success gives her reproductive success via her offspring that become high-quality juveniles, hence

$$w_{\mathrm{H}\to\mathrm{H}} = w_{\mathrm{P,H}\to\mathrm{H}} + w_{\mathrm{D,H}\to\mathrm{H}}\,. \tag{A3}$$

We now turn our attention to a focal high-quality juvenile's reproductive success via her offspring that become low-quality juveniles. Thus, the reproductive success of a high-quality juvenile that remains in her natal patch via her offspring that become high-quality juveniles is

$$w_{\mathrm{P,H}\to\mathrm{L}} = \frac{S_{\mathrm{H}}(x_{H}, y_{\mathrm{H}}, y_{\mathrm{L}})(1-m)nF_{\mathrm{H}}(1-p)}{(1-m)\big(N_{\mathrm{H}}S_{\mathrm{H}}(y_{\mathrm{H}}, y_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(y_{\mathrm{H}}, y_{\mathrm{L}})\big) + m\big(N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})\big)}\,. \tag{A4}$$

The reproductive success of a high-quality juvenile that disperses away from her natal patch via her offspring that become low-quality juveniles is

$$w_{\mathrm{D,H}\to\mathrm{L}} = \frac{S_{\mathrm{H}}(x_{\mathrm{H}}, y_{\mathrm{H}}, y_{\mathrm{L}})mnF_{\mathrm{H}}(1-p)}{N_{\mathrm{H}}S_{\mathrm{H}}(z_{\mathrm{H}}, z_{\mathrm{L}}) + N_{\mathrm{L}}S_{\mathrm{L}}(z_{\mathrm{H}}, z_{\mathrm{L}})}\,. \tag{A5}$$

The sum of these two components of a focal high-quality juvenile's reproductive success gives her reproductive success via her offspring that become low-quality juveniles, hence

$$w_{\mathrm{H}\to\mathrm{L}} = w_{\mathrm{P,H}\to\mathrm{L}} + w_{\mathrm{D,H}\to\mathrm{L}}\,. \tag{A6}$$

Expressions for the reproductive success of a focal low-quality juvenile can be derived in a similar way. The various class-specific reproductive success of a focal individual can be conveniently arranged in a transition matrix. Hence

$$w = \begin{pmatrix} w_{\mathrm{H}\to\mathrm{H}} & w_{\mathrm{L}\to\mathrm{H}} \\ w_{\mathrm{H}\to\mathrm{L}} & w_{\mathrm{L}\to\mathrm{L}} \end{pmatrix}. \tag{A7}$$

To define the reproductive success of a focal individual we need to weight her class-specific reproductive success by relative reproductive values. Hence, expressions for the reproductive success of a focal individual are

$$W_{\mathrm{H}} = w_{\mathrm{H}\to\mathrm{H}} + \frac{v_{\mathrm{L}}}{v_{\mathrm{H}}} w_{\mathrm{H}\to\mathrm{L}}\text{, and} \tag{A8}$$

$$W_{\mathrm{L}} = w_{\mathrm{L}\to\mathrm{L}} + \frac{v_{\mathrm{H}}}{v_{\mathrm{L}}} w_{\mathrm{L}\to\mathrm{H}}\,, \tag{A9}$$

for a focal high- and low-quality juvenile, respectively. The reproductive success of a random individual in the population is given by the class-specific fitness weighted by the correspondent class-reproductive values, as given in the main text (Taylor 1990; Taylor et al. 2007).

For the general model, we pursue an identical approach to that of Frank (1998, p114), see also Boyd (1982), Kelly (1992), and Gardner and West (2006), where the relationship between relatedness and the scale of competition is left undefined. However, we extend this approach to accommodate class-structure. Thus, the reproductive success of a high-quality juvenile via its offspring that become high-quality juveniles is

$$w_{\mathrm{H}\to\mathrm{H}} = \frac{S_{\mathrm{H}}\left(x_{\mathrm{H}}, y_{\mathrm{H}}, y_{\mathrm{L}}\right) F_{\mathrm{H}} p}{a\left(u_{\mathrm{H}} S_{\mathrm{H}}\left(y_{\mathrm{H}}, y_{\mathrm{L}}\right) + u_{\mathrm{L}} S_{\mathrm{L}}\left(y_{\mathrm{H}}, y_{\mathrm{L}}\right)\right) + (1-a)\left(u_{\mathrm{H}} S_{\mathrm{H}}\left(z_{\mathrm{H}}, z_{\mathrm{L}}\right) + u_{\mathrm{L}} S_{\mathrm{L}}\left(z_{\mathrm{H}}, z_{\mathrm{L}}\right)\right)}. \quad \text{(A10)}$$

The other equations, i.e. $w_{\mathrm{H}\to\mathrm{L}}$, $w_{\mathrm{L}\to\mathrm{H}}$, and $w_{\mathrm{L}\to\mathrm{L}}$, can be defined using the same logic. These are then used to define the transition matrix of the general model, which is analogous to the matrix (A7) of the island model, and to derive the equations (1) and (2) in the main text.

Appendix C: Stable-class frequencies and reproductive value

Class reproductive values are defined as the aggregate reproductive value of all juveniles belonging to a class. Thus they can be expressed as the product of individual reproductive value, v, and the stable-class frequency of juveniles belonging to a class, u, where the population is assumed to be neutral and at equilibrium (Taylor 1990; Taylor 1996; Taylor and Frank 1996). We conveniently set the sum of class-reproductive values to one, $c_{\mathrm{H}}+c_{\mathrm{L}} = 1$. Stable-class frequencies are given by the right-eigenvector of the transition matrix (A7). We find that the stable-class frequency of high-quality juveniles is $u_{\mathrm{H}} = p$, while the frequency of low-quality juveniles is $u_{\mathrm{L}} = 1\text{-}p$. Individual reproductive values are given by the elements of the left-eigenvector of the transition matrix (A7) (Taylor 1990; Grafen 2006). We find that the individual reproductive value of a high-quality juvenile is given by $v_{\mathrm{H}} = 1/(p+(1\text{-}p)(1\text{-}s))$, whilst the individual reproductive value of a low-quality juvenile is given by $v_{\mathrm{L}} = (1\text{-}s)/(p+(1\text{-}p)(1\text{-}s))$. It is important to keep in mind that s is the reproductive asymmetry between high- and low-quality individuals, such that $F_{\mathrm{L}} = (1\text{-}s)F_{\mathrm{H}}$. Thus if $s = 0$, $v_{\mathrm{H}} = v_{\mathrm{L}}$. The reproductive value of primary recipients depends on who are the direct beneficiaries of the actors' social actions. Thus, if actors direct their behaviours towards a random group mate, primary recipients' reproductive value is $v_{\mathrm{P}} = u_{\mathrm{H}}v_{\mathrm{H}}+u_{\mathrm{L}}v_{\mathrm{L}}$. If behaviours are directed towards high-quality group mates, primary recipients' reproductive value is $v_{\mathrm{P}} = v_{\mathrm{H}}$. Finally, if behaviours are directed towards low-quality group mates, primary recipients' reproductive value is $v_{\mathrm{P}} = v_{\mathrm{L}}$. As the impact of actors' behaviour on local competition is equally shared among all group members, secondary recipients' reproductive value is always $v_{\mathrm{S}} = u_{\mathrm{H}}v_{\mathrm{H}}+u_{\mathrm{L}}v_{\mathrm{L}}$. We obtain relative reproductive values by dividing recipients' reproductive value by an actor's reproductive value. Assuming facultative social behaviour, and considering a focal high-quality actor, we find that the secondary recipients' relative reproductive value is $\upsilon_{\mathrm{S|H}} = v_{\mathrm{S}}/v_{\mathrm{H}} = p+(1\text{-}p)(1\text{-}s)$. Hence $\partial\upsilon_{\mathrm{S|H}}/\partial p = s$, which means that secondary recipients' relative reproductive value always increases with increasing frequency of high-quality individuals. Moreover, we find that $\partial\upsilon_{\mathrm{S|H}}/\partial s = \text{-}(1\text{-}p)$, which means that secondary recipients' relative reproductive value always decreases with increasing reproductive asymmetry. If the actor is a low-quality juvenile, we find that secondary recipients' relative reproductive value is $\upsilon_{\mathrm{S|L}} = v_{\mathrm{S}}/v_{\mathrm{L}} = (p+(1\text{-}p)(1\text{-}s))/(1\text{-}s)$.

Hence $\partial v_{S|L}/\partial p = s/(1\text{-}s)$, which means that secondary recipients' relative reproductive value always increases with increasing frequency of high-quality individuals. Moreover, we find that $\partial v_{S|L}/\partial s = p/(1\text{-}s)^2$, which means that secondary recipients' relative reproductive value always increases with increasing reproductive asymmetry. As the general model is equivalent to the island model, these reproductive values are also used in the general model.

Appendix D: Hamilton's rule

We use the Taylor-Frank approach (Taylor and Frank 1996; Frank 1997, 1998; Taylor et al. 2007) to derive the direction of selection acting on the social trait. We derive the response to selection of a mutant for the breeding value *g*; this is given by

$$\frac{\mathrm{d}W}{\mathrm{d}g} = c_{\mathrm{H}}\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} + c_{\mathrm{L}}\frac{\mathrm{d}W_{\mathrm{L}}}{\mathrm{d}g}. \tag{A11}$$

Expanding the derivative in the first term on the RHS of equation (A11), we get

$$\frac{\mathrm{d}W_{\mathrm{H}}}{\mathrm{d}g} = \frac{\partial W_{\mathrm{H}}}{\partial x_{\mathrm{H}}}\frac{\mathrm{d}x_{\mathrm{H}}}{\mathrm{d}g_{\mathrm{H}}}\frac{\mathrm{d}g_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial y_{\mathrm{H}}}\frac{\mathrm{d}y_{\mathrm{H}}}{\mathrm{d}g'_{\mathrm{H}}}\frac{\mathrm{d}g'_{\mathrm{H}}}{\mathrm{d}g} + \frac{\partial W_{\mathrm{H}}}{\partial y_{\mathrm{L}}}\frac{\mathrm{d}y_{\mathrm{L}}}{\mathrm{d}g'_{\mathrm{L}}}\frac{\mathrm{d}g'_{\mathrm{L}}}{\mathrm{d}g}, \tag{A12}$$

where g_{H} denotes self's breeding value, g'_{H} denotes the high-quality social partners' breeding value, and g'_{L} denotes the low-quality social partners' breeding value. All derivatives and partial derivatives are evaluated at $x_{\mathrm{H}} = y_{\mathrm{H}} = z_{\mathrm{H}}$ and $\mathrm{x}_{\mathrm{L}} = y_{\mathrm{L}} = z_{\mathrm{L}}$ (Taylor and Frank 1996; Frank 1997, 1998; Taylor et al. 2007). The partial derivatives of fitness on phenotype are the marginal survival effects, that is the increment or decrement of survival that occurs owing to the actor's social behaviour. The correlation of the breeding value on the phenotype is the mapping between genotype and phenotype and is given by $\mathrm{d}x_{\mathrm{H}}/\mathrm{d}g_{\mathrm{H}} = \mathrm{d}y_{\mathrm{H}}/\mathrm{d}g'_{\mathrm{H}} = \mathrm{d}y_{\mathrm{L}}/\mathrm{d}g'_{\mathrm{L}} = \gamma_{\mathrm{H}}$, which can be set to one ($\gamma_{\mathrm{H}} = 1$). The correlations between the actor's genic value and the recipients' breeding value are the coefficients of consanguinity. Thus, the correlation between the actor's genic value and the self's breeding value is $\mathrm{d}g_{\mathrm{H}}/\mathrm{d}g$; the correlation between the actor's genic value and the high-quality primary recipients' breeding value is $\mathrm{d}g'_{\mathrm{H}}/\mathrm{d}g$; the correlation between the actor's genic value and the low-quality primary recipients' breeding value is $\mathrm{d}g'_{\mathrm{L}}/\mathrm{d}g$. The kin selection coefficients of relatedness are derived by normalizing the coefficients of consanguinity to the actor's coefficient of consanguinity (Bulmer 1994). An analogous expression can be derived for the direction of selection acting on a focal low-quality juvenile ($\mathrm{d}W_{\mathrm{L}}/\mathrm{d}g$). We find that these various selection gradients (i.e. Hamilton's rule) have an identical mathematical form, which is that of inequality (3) in the main text.

Appendix E: Relatedness

We assume vanishingly small genetic variance in the population. Under these assumptions, the genetic structure of the population at equilibrium is determined using recursion equations (Wright 1969; Taylor 1992; Rousset 2004; Rodrigues and Gardner 2012). Relatedness between two high-quality adults in a patch with j high-quality adults is denoted by $r_{\mathrm{HH},j}$; relatedness between a high-quality adult and a low-quality adult, or between a low-quality adult and a high-quality adult in a patch with j high-quality adults is denoted by $r_{\mathrm{HL},j}$; relatedness between two low-quality adults in a patch with j high-quality adults is denoted by $r_{\mathrm{LL},j}$. With probability $h_{\mathrm{HH},j}$, two high-quality adults are philopatric to the j-type patch. With probability $h_{\mathrm{HL},j}$, two individuals in different classes are philopatric to the j-type patch. With probability $h_{\mathrm{LL},j}$, two low-quality individuals are philopatric to the j-type patch. We find that $h_{\mathrm{HH},j} = h_{\mathrm{HL},j} = h_{\mathrm{LL},j} = h = (1\text{-}m)^2$. Given that two high-quality individuals are philopatric to the patch, the probability that they are derived from high-quality individuals is $Q_{\mathrm{H},j} = (j/(j+(n\text{-}j)(1\text{-}s)))^2$, in which case with probability $1/n_{\mathrm{H},j}$ they are siblings and are related by 1, and with probability $1\text{-}1/n_{\mathrm{H},j}$ they are related by $r_{\mathrm{HH},j}$. Given that two high-quality individuals are philopatric to the patch, with probability $Q_{\mathrm{HL},j} = 2(j(n\text{-}j)(1\text{-}s)/(\ j+(n\text{-}j)(1\text{-}s))^2)$ they are derived from individuals belonging to different classes, in which case they are related by $r_{\mathrm{HL},j}$. Given that two low-quality individuals are philopatric to the patch, with probability $Q_{\mathrm{L},j} = ((n\text{-}j)(1\text{-}s)/(j+(n\text{-}j)(1\text{-}s)))^2$ they are both derived from low-quality individuals, in which case with probability $1/n_{\mathrm{L},j}$ they are siblings and are related by 1, whereas with probability $1\text{-}1/n_{\mathrm{L},j}$ they are related by $r_{\mathrm{LL},j}$. A similar argument can be made to determine the analogous quantities when individuals belong to different classes, and when individuals belong to the low-quality class. We find that $r_{\mathrm{HH},j} = r_{\mathrm{HL},j} = r_{\mathrm{LL},j} = r_{\mathrm{A}}$, which is given by

$$r_{\mathrm{A}} = \frac{h}{n_{\mathrm{e}} - (n_{\mathrm{e}} - 1)h}, \tag{A13}$$

where n_{e} is the effective group size. Relatedness among juveniles is given by $r = r_{\mathrm{A}}/h$, which is given by equation (5) in the main text. We find that the effective group-size is given by

$$n_{\mathrm{e}} = \frac{1}{\sum_{j=0}^{n} P_j Q_{\mathrm{H},j} / j + \sum_{j=0}^{n} P_j Q_{\mathrm{L},j} / (n-j)}, \tag{A14}$$

where $Q_{\mathrm{H},j} = (j/(j+(n\text{-}j)(1\text{-}\underline{s})))^2$, $Q_{\mathrm{L},j} = ((n\text{-}j)(1\text{-}s)\ /(j+(n\text{-}j)(1\text{-}s)))^2$, and P_{j} the frequency of j-type patches.

Appendix F: Social value

In the main text we have assumed that individuals exhibit no differences in task efficiency (i.e. $C_{\mathrm{H}} = C_{\mathrm{L}}$ and $B_{\mathrm{HH}} = B_{\mathrm{HL}} = B_{\mathrm{LL}} = B_{\mathrm{LL}}$); thus, the marginal costs to the actor and marginal benefits to primary recipients are identical among all juveniles. As a result, differences between the selection pressures acting on high-quality juveniles and those acting on low-quality juveniles were due to differences in reproductive value. However, individuals may exhibit differences in task efficiency. For example, large individuals may be better at defending the group, and therefore they may incur much lower personal costs than smaller individuals who perform the same behaviour. To consider this scenario, we develop the following model. The life-cycle is equal to that of the general model presented in the main text. We consider the scenario where the behaviour is conditional on the actor's quality but not on the recipients' quality. However, in contrast with the general model presented in the main text, we relax the assumption that the costs are independent of the actor's quality. Hence, we may have $C_H \neq C_L$. Moreover, we make explicit assumptions about how costs and benefits depend on trait values, i.e. z_H and z_L. We assume that high-quality juveniles perform tasks more efficiently, and therefore they are able to perform a behaviour at a lower cost to themselves. Task efficiency is denoted by ε, with $0 \leq \varepsilon \leq 1$. For example, if $\varepsilon = 0$, then high-quality juveniles perform the social task with the same efficiency of low-quality juveniles; however, if $\varepsilon > 0$, then, high-quality juveniles suffer a lower cost when expressing the social trait than low-quality juveniles. We further assume that the marginal cost of the social trait increases linearly with actor's trait level. Under these assumptions the marginal cots are $C_H = k(1-\varepsilon)z_H$, and $C_L = kz_L$, for high- and low-quality juveniles, respectively, where k is assumed to be small, $k << 1$. We assume that the marginal benefits are independent of the recipients' quality, and that they decrease linearly with the group's trait level. Thus $B_{HH} = B_{HL} = B_{LH} = B_{LL} = k(1-z)$, where $z = pz_H+(1-p)z_L$ is the group's trait level. Taking into account that $C_H \neq C_L \neq C$, we can use Hamilton's rule (inequality 3 in the main text), to derive the selection gradients acting on the social traits. These are given by:

$$-(1-\varepsilon)z_H + (1-z)(p+(1-p)(1-s))r - a(p+(1-p)(1-s))(1-z-(1-\varepsilon)z_H)r > 0 \text{, and} \tag{A15}$$

$$-(1-s)z_L + (1-z)(p+(1-p)(1-s))r - a(p+(1-p)(1-s))(1-z-z_L)r > 0 \text{,} \tag{A16}$$

for high-quality juveniles and low-quality juveniles, respectively. To determine the equilibrium rates of investment in the social trait, z_H^* and z_L^*, we set the LHS of inequalities (A15) and (A16) to zero and solved both equations simultaneously. We define the LHS of inequalities (A15) and (A16) as $J_H(z_H,z_L)$ and $J_L(z_H,z_L)$, respectively. We check the equilibria for convergence stability (CS, Eshel and Motro 1981; Christiansen 1991; Taylor 1996) by defining matrix **J**, where the first and second elements of the first row are given by $\partial J_H(z_H,z_L)/\partial z_H|_{zH=zH^*,zL=zL^*}$, and $\partial J_H(z_H,z_L)/\partial z_L|_{zH=zH^*,zL=zL^*}$, respectively; whilst the first and second elements of the second row are $\partial J_L(z_H,z_L)/\partial z_H|_{zH=zH^*,zL=zL^*}$, and $\partial J_L(z_H,z_L)/\partial z_L|_{zH=zH^*,zL=zL^*}$, respectively. We find that both eigenvalues of matrix **J** are always negative, and therefore z_H^* and z_L^* are convergence stable (Otto and Day 2007). We find that if the task efficiency of high-quality juveniles is high (high ε) and if local competition is low (low a), then high-quality juveniles are selected to invest more into helping than low-quality juveniles ($z_H^* > z_L^*$; Figure A1). In the absence of local competition ($a = 0$), if $(1- \varepsilon) = (1-s)$ then high- and low-quality juveniles are selected to invest the same amount of resources in helping ($z_H^* = z_L^*$; circle in Figure A1). That is the high reproductive value of high-quality individuals ($v_H > v_L$) selects them to invest less in helping, however this is offset by their high social value ($C_H < C_L$), which selects them to invest more in helping.

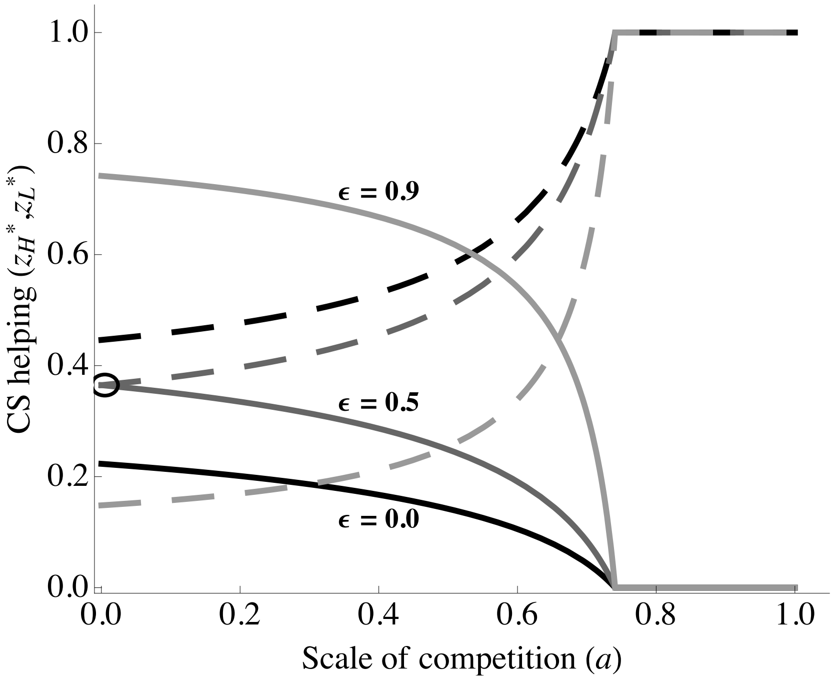

Figure A1 | Convergence stable helping for high-quality individuals (z_H*; solid lines) and low-quality individuals (z_L*; dashed lines), as a function of the scale of competition (a), for various values of social efficiency (ε). If social efficiency of high-quality individuals is equal to the social efficiency of low-quality individuals (ε = 0), high-quality individuals are selected to invest less in helping than low-quality individuals. By contrast, if the social efficiency of high-quality individuals is high (high ε), then they may be selected to invest more in helping than low-quality individuals. Without local competition (a = 0), when (1-s) = (1- ε), all individuals invest the same in helping (z_H* = z_L*, circled point). Parameter values: p = 0.5, s = 0.5, r = 0.9.

Supplementary Figure S1

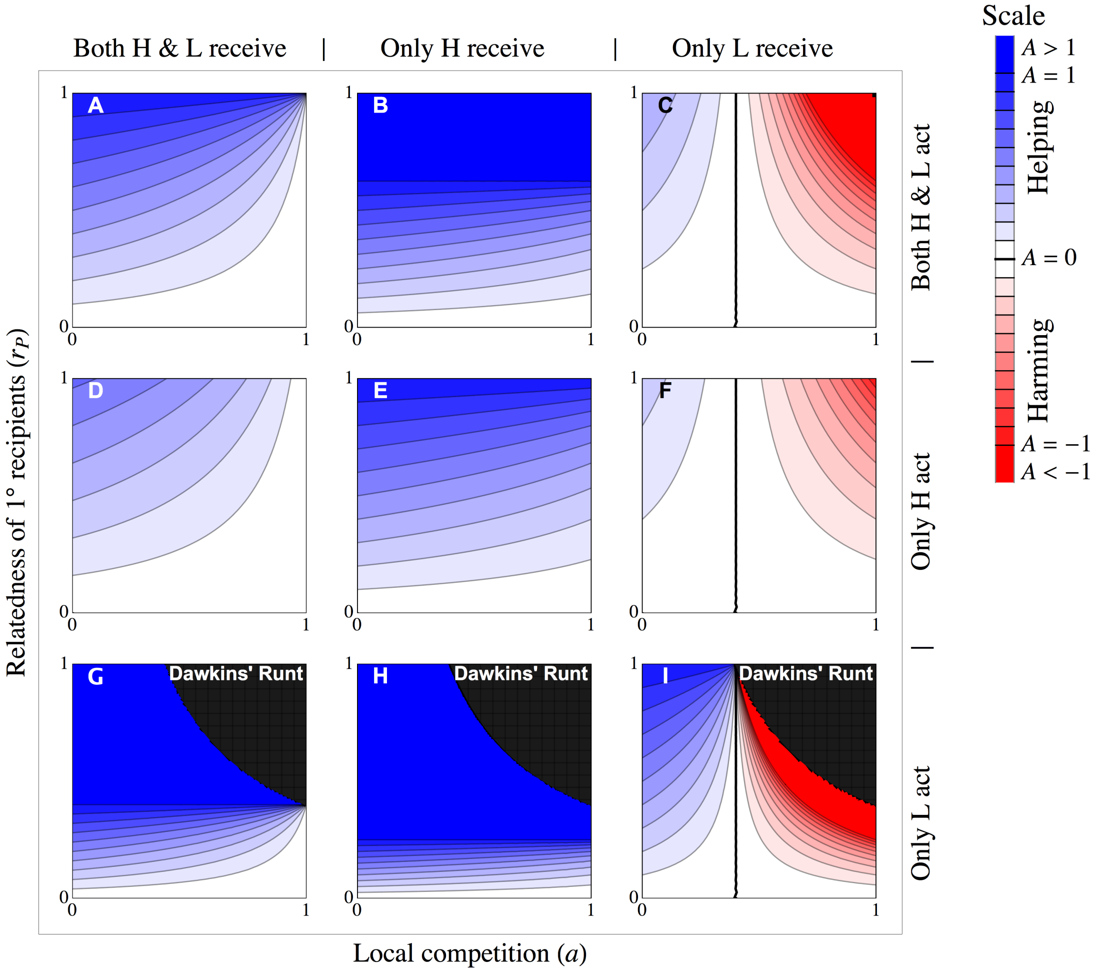

Figure S1 | The potential for helping (A) is shown for unconditional and conditional behaviour. The potential for helping is given as a function of local competition (a) and the relatedness of 1° recipients (r_P). The potential for helping of Frank's (1998) reference model corresponds to $s = 0$. [A] Unconditional social behaviour: the potential for helping is equal to that of the reference model ($A_{B|B,F} = A_F$). As a result, helping is always non-negative ($A_{B|B,F} \geq 0$), and it increases with increasing relatedness of 1° recipients while it decreases with increasing local competition. [B] Behaviour that is unconditional on actor's quality and is directed to high-quality group mates: the potential for helping is always non-negative ($A_{B|H,F} \geq 0$), and it is always greater than that of the reference model ($A_{B|H,F} > A_F$), and it increases with increasing relatedness of 1° recipients while it decreases with increasing local competition. [C] Behaviour that is unconditional on actor's quality and is directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{B|L,F} < A_F$); the potential for helping decreases and the potential for harming increases with increasing local competition. Both the potential for helping and harming increase with increasing relatedness of 1° recipients. [D] Behaviour expressed by high-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($A_{H|B,F} \geq 0$), and it is always less than that of the reference model ($A_{H|B,F} < A_F$). The potential for helping increases with increasing relatedness of 1° recipients, while it decreases with increasing local competition. [E] Behaviour expressed by high-quality juveniles directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{H|H,F}} \geq 0$), and it is always greater than that of the reference model ($A_{\mathrm{H|H,F}} > A_{\mathrm{F}}$). The potential for helping increases with increasing relatedness of 1° recipients, while it decreases with increasing local competition. [F] Behaviour expressed by high-quality juveniles and directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{\mathrm{H|L,F}} < A_{\mathrm{F}}$); the potential for helping decreases and the potential for harming increases with increasing local competition. Both the potential for helping and harming increase with increasing relatedness of 1° recipients. [G] Behaviour expressed by low-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|B,F}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{L|B,F}} > A_{\mathrm{F}}$). The potential for helping increases with increasing relatedness of 1° recipients. For lower relatedness of 1° recipients, the potential for helping decreases with increasing local competition. For higher relatedness of 1° recipients, by contrast, the potential for helping increases with increasing local competition. [H] Behaviour expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($\mathrm{A}_{\mathrm{L|H,F}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{L|H,F}} > A_{\mathrm{F}}$). The potential for helping increases with increasing relatedness of 1° recipients. For lower relatedness of 1° recipients, the potential for helping decreases with increasing local competition. For higher relatedness of 1° recipients, by contrast, the potential for helping increases with increasing local competition. [I] Behaviour expressed by low-quality juveniles and directed to low-quality group mates: there is potential for helping and potential for harming ($\mathrm{A}_{\mathrm{L|L,F}} > 0$ and $\mathrm{A}_{\mathrm{L|L,F}} < 0$). The potential for helping is always less than that of the reference model ($A_{\mathrm{L|L,F}} < A_{\mathrm{F}}$). The potential for helping decreases and the potential for harming increases with increasing local competition. Both the potential for helping and harming increase with increasing relatedness of 1° recipients. [G-I] When secondary recipients' value is greater than or equal to actor's value ($\rho_{\mathrm{S}} \geq 1$), there is infinite potential for helping ($A_{\mathrm{L|B,F}} = \infty$ and $A_{\mathrm{L|H,F}} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{\mathrm{L|L,F}} = -\infty$, Dawkins' Runt region, panel I). In all cases, frequency of high-quality juveniles is set to $p = 0.5$, and reproductive asymmetry is set to $s = 0.75$.

Supplementary Figure S2

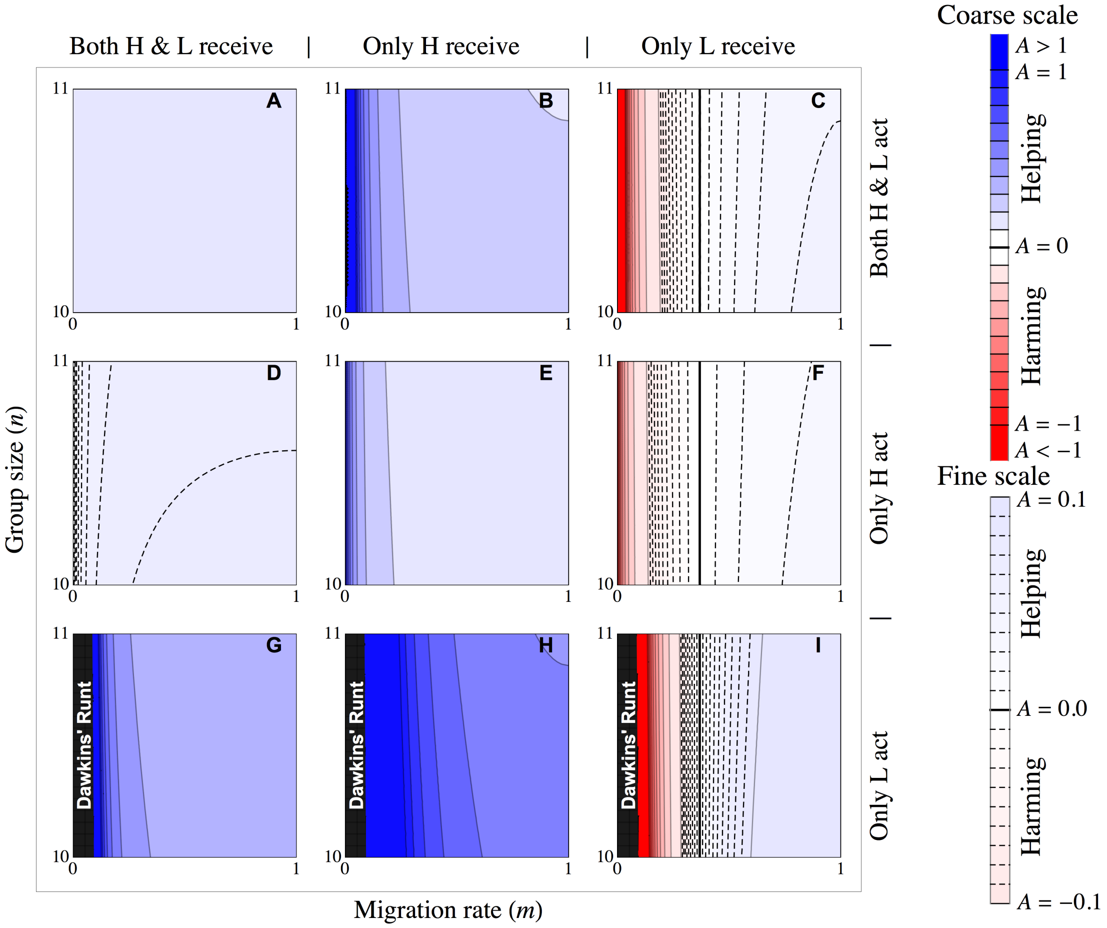

Figure S2 | The potential for helping (*A*) is shown for unconditional and conditional behaviour. The potential for helping is given as a function of the frequency of high-quality individuals (*p*) and the reproductive asymmetry (*s*). The potential for helping of Taylor's (1992a) reference model corresponds to $s = 0$. [A] Unconditional social behaviour: the potential for helping is greater than that of the references model ($A_{\mathrm{B|B,T}} > A_{\mathrm{T}}$). [B] Behaviour that is unconditional on actor's quality and is directed to high-quality group mates: the potential for helping is always non-negative ($A_{\mathrm{B|H,T}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{B|H,T}} > A_{\mathrm{T}}$), and it decreases with increasing group size and increasing migration rate. [C] Behaviour that is unconditional on actor's quality and is directed to low-quality group mates: the potential for helping is always less than that of the reference model ($A_{\mathrm{B|L,T}} < A_{\mathrm{T}}$); the potential for helping increases and the potential for harming decreases with increasing migration rate. Both, the potential for helping and harming, decrease with increasing group size. [D] Behaviour expressed by high-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($A_{\mathrm{H|B,T}} \geq 0$), and it is always less than that of the reference model ($A_{\mathrm{H|B,T}} < A_{\mathrm{T}}$). The potential for helping decreases with increasing group size increasing migration rate. [E] Behaviour expressed by high-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($A_{\mathrm{H|H,T}} \geq 0$), and this is always greater than that of the reference model ($A_{\mathrm{H|H,T}} > A_{\mathrm{T}}$). [F] Behaviour expressed by high-quality juveniles and directed to low-quality group mates: the potential for helping increases and the potential for harming decreases with increasing migration. Both the potential for helping and harming decrease with increasing group size. [G] Behaviour expressed by low-quality juveniles and directed to all group mates: the potential for helping is always non-negative ($A_{L|B,T} \geq 0$), and this is always greater than that of the reference model ($A_{L|B,T} > A_T$). The potential for helping decreases with increasing group size. The potential for helping decreases with increasing migration rate. [H] Behaviour expressed by low-quality juveniles and directed to high-quality group mates: the potential for helping is always non-negative ($A_{L|H,T} \geq 0$), and this is always greater than that of the reference model ($A_{L|H,T} \geq A_T$). The potential for helping decreases with increasing group size. The potential for helping decreases with increasing migration rate. [I] Behaviour expressed by low-quality juveniles, and directed to low-quality group mates: there is potential for helping and potential for harming ($A_{L|L,T} > 0$ and $A_{L|L,T} < 0$).The potential for helping increases and the potential for harming decreases with increasing migration rate. Both the potential for helping and harming decrease with increasing group size. [G-I] When secondary recipients' value is greater than or equal to actor's value ($\rho_S \geq 1$) there is an infinite potential for helping ($A_{L|B,T} = \infty$ and $A_{L|H,T} = \infty$, Dawkins' Runt region, panels G and H) and infinite potential for harming ($A_{L|L,T} = -\infty$, Dawkins' Runt region, panel I). In all cases, the frequency of high-quality juveniles is set to p = 0.5, and reproductive asymmetry is set to s = 0.75.

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The social evolution of dispersal with public goods cooperation

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kin selection;
neighbour-modulated fitness.

Abstract

Selection can favour the evolution of individually costly dispersal if this alleviates competition between relatives. However, conditions that favour altruistic dispersal also mediate selection for other social behaviours, such as public goods cooperation, which in turn is likely to mediate dispersal evolution. Here, we investigate – both experimentally (using bacteria) and theoretically – how social habitat heterogeneity (i.e. the distribution of public goods cooperators and cheats) affects the evolution of dispersal. In addition to recovering the well-known theoretical result that the optimal level of dispersal increases with genetic relatedness of patch mates, we find both mathematically and experimentally that dispersal is always favoured when average patch occupancy is low, but when average patch occupancy is high, the presence of public goods cheats greatly alters selection for dispersal. Specifically, when public goods cheats are localized to the home patch, higher dispersal rates are favoured, but when cheats are present throughout available patches, lower dispersal rates are favoured. These results highlight the importance of other social traits in driving dispersal evolution.

Introduction

Understanding dispersal is a major aim of evolutionary ecology (Weins, 2001). Theoretical work suggests that dispersal provides possible benefits when the environment varies in time (Van Valen, 1971; McPeek & Holt, 1992) and if it reduces inbreeding depression (Bengtsson, 1978) and kin competition (Hamilton & May, 1977). Conversely, dispersal can be opposed by obvious costs, such as increased mortality or reduced reproduction (Rousset & Gandon, 2002; Bonte *et al.*, 2012). The indirect fitness benefits of dispersal, that arise owing to it alleviating kin competition, highlight that dispersal is often a social trait and that it can be favoured by kin selection even in the context of severe fitness penalties for the dispersing individuals (Hamilton & May, 1977; Comins *et al.*, 1980; Taylor & Frank, 1996; Gandon & Michalakis, 1999; Taylor & Buckling, 2010; Wei *et al.*, 2011). The evolution of altruistic traits often requires high relatedness, and in terms of dispersal, this would mean a tendency for individuals who share the same 'dispersal alleles' to be associated in space. However, dispersal itself is likely to reduce relatedness, which in turn can reduce selection for dispersal (Taylor, 1988; Gandon, 1999; Gandon & Michalakis, 1999; Ronce, 2007).

The change in population structure resulting from dispersal is likely to have particularly important consequences for the evolution of dispersal rates when other social traits have important fitness consequences (Perrin & Lehmann, 2001 and references therein; Le Galliard *et al.*, 2005). High relatedness, as well as selecting for elevated dispersal rates, also selects for other forms of altruism, where individuals pay a cost for the benefit of the group as a whole (Hamilton, 1964). In contrast to dispersal, where leaving the group is often an altruistic act, many altruistic traits require individuals to stay in groups. This has led to the development of theoretical models that address coevolution between social behaviours and dispersal behaviour (Koella, 2000; Perrin & Lehmann, 2001; Le Galliard *et al.*, 2005). A

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key prediction from these models is that selection for dispersal will be reduced when other cooperative behaviours have important direct or indirect fitness benefits.

We investigate how selection for dispersal in bacteria is influenced when individuals are cooperating (or not) by the production of public goods. Wild-type bacteria produce numerous extracellular molecules, such as tissue degrading enzymes, iron-scavenging siderophores and sticky polymers, to protect surface-growing bacteria (biofilms), which are individually costly but benefit the group as a whole (West *et al.*, 2007). Such behaviours are readily exploitable by nonproducing cheats; hence, dispersal might be selected against in public good producing bacteria if it increases the chance of encountering exploiting cheats. Conversely, selection for dispersal may be beneficial if it allows escape from social exploitation or if it promotes individuals to move from saturated to empty patches. We investigate how the evolution of dispersal is affected by public goods production and exploitation when exploiters are locally (i.e. exhibit low patch occupancy) or widely (i.e. exhibit high patch occupancy) distributed. We define patch occupancy as the fraction of patches that are occupied. We explore this result experimentally and theoretically, with the aim to link empirical data with general theory of the evolution of dispersal.

We use a well-studied bacteria model for social evolution, the opportunistic bacterial pathogen, *Pseudomonas aeruginosa. Pseudomonas aeruginosa* possesses a range of motility mechanisms, which trade-off against each other, to move in different environments (Bardy *et al.*, 2003; Taylor & Buckling, 2011). We manipulate dispersal behaviour using two genetically modified pili mutants of *P. aeruginosa,* which in a semi-solid agar plate show disparate dispersal behaviours: one acts as a 'disperser' and is able to quickly colonize the plate due to the ability to move freely through the substrate; the other acts as a 'nondisperser' and is unable to colonize the plate as quickly due to restricted movement (details of genotypes are given in Materials and methods). Consistent with theoretical results, our previous work using these mutants has shown that conditions of high relatedness favour the disperser (Taylor & Buckling, 2010).

Pseudomonas aeruginosa produces numerous public goods but, for simplicity, we focus upon a single trait: extracellular iron-chelating siderophores (Ratledge & Dover, 2000; West & Buckling, 2003). Iron is vital for bacterial growth; however, most iron in the natural environment exists in the insoluble ferric form and must be reduced via reactions initiated by siderophore molecules to be utilized. Under conditions of iron limitation, clonal populations of siderophore producers reach much higher densities (and lead to more severe infections) than isogenic mutants that do not produce the primary siderophore, pyoverdin (West & Buckling, 2003). However, nonproducing mutants can exploit the pyoverdin of producers and hence outcompete them, when in direct competition as a result of the metabolic cost of pyoverdin production (Griffin *et al.*, 2004).

Materials and methods

Strain details and growth conditions

Two transposon mutants defective in type IV pili and generated from a wild-type strain of *P. aeruginosa* (PAO1) were used as dispersing phenotypes: PilA acts as the disperser as it is absent of pili (surface organelles which aid motility on hard surfaces), and PilU acts as the nondisperser as it is able to express but unable to retract pili (hyperpiliated) (D'Argenio *et al.*, 2001). These mutants are isogenic, and hence, siderophore production does not differ between strains. A soft agar medium creates conditions whereby PilA is able to move freely through the substrate, whereas the drag caused by permanently extruded pili causes PilU to become stuck. A third mutant defective in siderophore production (PAO1Δ*pvdD pchEF*; Ghysels *et al.*, 2004) was used as the 'cheat'. This strain is unable to grow in isolation in an iron-limited environment and requires access to a siderophore-producing strain to grow. The dispersing phenotype of the cheat is intermediate between PilA and PilU; therefore, PilA is able to disperse beyond the range of cheats, but PilU is not. For comparison, a wild-type PAO1 strain was used as the 'cooperator' (its dispersing phenotype is equivalent to the cheats, but they produce siderophores and therefore do not impose a social cost). Bacterial cultures were grown overnight at 37 °C in 6 mL Casamino acid media (CAA) shaken at 0.9 g. Cells were then pelleted and resuspended in M9 buffer solution twice to ensure that all nutrients were removed from the media.

Any free iron which potentially remained in resuspended cultures was removed by the addition of an iron chelator (100 μg mL^{-1} of human apo-transferrin and 20 mM $NaHCO_3$; Sigma; Meyer *et al.*, 1996; Griffin *et al.*, 2004). Bacteria were left to grow and disperse for 72 h.

Treatment conditions

Our simple factorial experimental design involved determining the fitness (growth) of cooperating dispersers (PilA) and nondispersers (PilU) in the presence of one of two competitors – cheats (exploiter) or cooperators (WT) – which were distributed throughout the plate (high-occupancy treatment) or only present in the inoculation site (low-occupancy treatment). This resulted in eight experimental treatments (Fig. 1). A total of 25 mL of iron-limited CAA agar (0.6% w/v agar) was poured into 20-cm-diameter Petri dishes and allowed to dry in the laminar flow hood for 20 min.

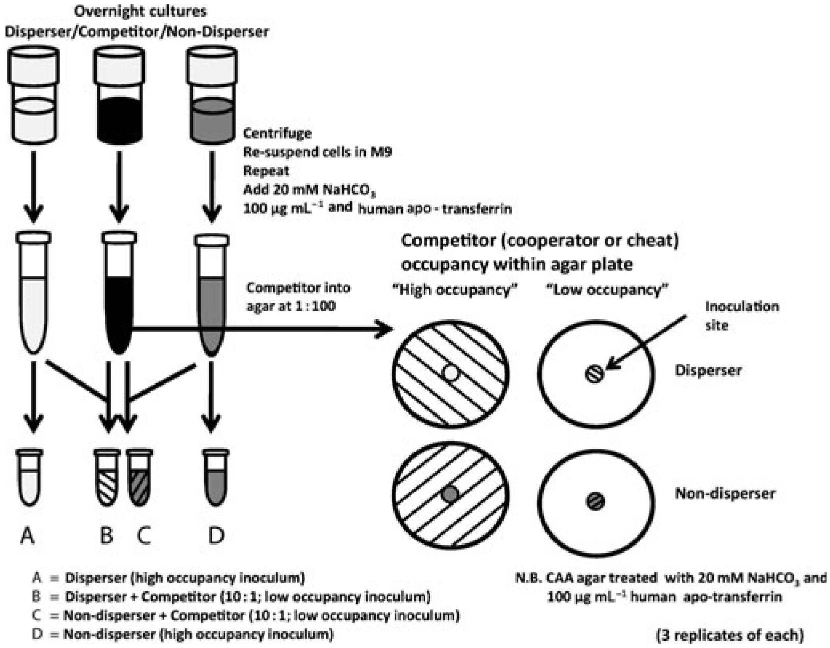

Fig. 1 Methods for setting up the eight treatment groups: competition is either low or high occupancy; the competitor is either a cooperator or a cheat; and the dispersing variant is either a disperser (PilA) or a nondisperser (PilU). Under the high-occupancy treatment, agar is supplemented with a competitor strain (either a cooperator or cheat), which will compete for space and resources. The disperser or nondisperser is inoculated into the centre of each plate. Under high-occupancy treatment, this inoculum is pure, but under low-occupancy treatment, this inoculum is mixed with one of the competitor strains at a concentration of 10 : 1.

Agar plates used for high-occupancy treatment were supplemented with 250 μL (approximately 10^9 cells) overnight competitor culture (either cheat or cooperator), and the other half remained bacteria free. Inoculum was prepared as follows: (i) low-occupancy treatment: competitors (cheats or cooperators) were mixed in an Eppendorf with each of the dispersal variants (disperser or nondisperser) at 1 : 10; (ii) high-occupancy treatment: pure disperser (PilA) and nondisperser (PilU) cultures were used as the inoculum, and 2.5 μL of inoculum (approximately 10^7 cells) was pipetted into the centre of the agar plate of the corresponding treatment group (i.e. low-occupancy or high-occupancy treatment). Each treatment was replicated three times.

It was necessary to address whether pleiotropic effects from the deleted PilA and PilU genes could lead to one of the dispersing variants being an intrinsically worse competitor than the other, due to costs incurred from pili production (or lack of). Cooperator and cheat strains were acting as environmental factors to dispersing strains, and their competitive behaviour was not the focus of this study, but is considered elsewhere (e.g. Jiricny *et al.*, 2010; Kümmerli *et al.*, 2009a). We compared growth rates (to test the relative fitness of the disperser (PilA) with the nondisperser (PilU)) under conditions where motility would confer little or no advantage. We grew the dispersal variants together (approximately 1.2×10^7 cells of disperser and nondisperser) at 37 °C in 6 mL liquid KB shaken at 0.9 g and found growth rates to be equivalent (Wilcoxon, $P = 0.993$). In addition, we also competed the genotypes where bacteria were evenly inoculated throughout soft agar; hence, moving from one colonized 'patch' would simply result in entering another. Again, we found no significant difference in relative fitness between the two strains (Wilcoxon, $P = 0.14$).

Data collection

Methods were as in Taylor & Buckling (2010). Samples were taken using a 1-mL pipette (Finn pipette), at regular 5-mm intervals along the radius of the colony. The samples were then washed in M9 buffer (12.8 g L^{-1} Na_2HPO_4, 3 g L^{-1} $KHPO_4$, 0.5 g L^{-1} NaCl, 1 g L^{-1} NH_4Cl), diluted to an appropriate dilution to allow colony differentiation and plated to count colony-forming units (CFUs). The relative fitness is always measured between dispersers (PilA) and nondispersers (PilU), and determined by calculating the ratio of the total number of each cell type across corresponding plates. The nature of the experimental design means that in some treatments, there are competitors present, and in others, they are not. However, the fully factorial design of the experiment means that all combinations are comparable.

Visual differentiation was made between siderophore-producing (green) and nonproducing colonies (white) on KB agar. In plates where all strains were cooperators, differentiation could be made between dispersal variants by the colony morphology: on hard agar, the wild-type cooperator (which has fully functional pili) can move more efficiently than the dispersal variants and therefore produces larger colonies than the dispersing or nondispersing phenotypes. The relative fitness of dispersers (PilA) vs. nondispersers (PilU) was determined by dividing the cell density of dispersers by the cell density of nondispersers across randomly paired plates within the same treatment group (for example: disperser vs. nondisperser under treatment, competitor = cheat; competitor distribution = high occupancy). All analyses and figures were produced on PASW Statistics 18 (SPSS; part of IBM UK Ltd, Middlesex, UK).

Theoretical model

To link the empirical data with the existing general theory of dispersal evolution, we developed a theoretical model to investigate the dynamics between public goods cooperation and kin competition in the context of evolving dispersal behaviour. Our model is relatively simple, as we are mostly interested in qualitative predictions, but it nevertheless captures the main aspects of our experimental design and enables generalization to other populations. We derived an expression for the personal fitness of a cooperator exhibiting a rare genetic variant dispersal strategy. We assume that only cooperators are able to disperse (with varying probability), and therefore, the genetic variation for dispersal is only in cooperators. Thus, this genetic variation at the dispersal locus does not correlate with genetic variation at the cooperation locus (because cooperation is constant among cooperators). We then employed a neighbour-modulated fitness approach to kin selection analysis (Taylor, 1996; Taylor & Frank, 1996; Frank, 1997, 1998; Rousset, 2004; Taylor *et al.*, 2007) to identify the evolutionary equilibrium rate of dispersal, which we then checked for convergence stability (Eshel, 1983; Taylor, 1996).

We assume an infinite metapopulation with three types of patches that vary in their quality (where patch quality is defined by their carrying capacity). Each patch type occurs at a given fixed frequency in the population, which is independent of trait value. A proportion α of patches can contain both cooperator and cheat bacterial cells ('occupied patches'), a proportion β of patches are empty ('vacant patches') and a proportion $\gamma = 1\text{-}(\alpha+\beta)$ of patches contain only cheats ('cheat patches'). Under this patch arrangement, the only way to escape social cheats is by dispersing to another patch. As in the experimental design, we assume that these quantities are fixed parameters.

We denote the genetic relatedness (with respect to the genes for dispersal) among cooperators within patches by r. Each cooperator disperses with independent probability z to a random patch or else remains in their natal patch with probability $1\text{-}z$. Following dispersal, bacteria reproduce asexually, with cooperators reaching carrying capacities of aK in occupied patches, $(1\text{-}a)K$ in vacant patches and 0 in cheat patches. Thus, by varying the value of the parameter a between 0 and 1, we are able to investigate the whole range of possibilities for the impact of patch occupancy upon carrying capacity, from occupied patches having vastly lower carrying capacity ($a \to 0$) to vastly greater carrying capacity ($a \to 1$), than vacant patches. The parameter K acts as a scaling factor, to reflect that there may be numerous bacterial cells in a patch. Note, that the carrying capacity aK includes the cooperators already present in the patch and any new migrants that enter. This parameter defines the carrying capacity of the focal strain only, rather than that of the total bacterial population. That is, we allow for occupancy (relative to vacancy) to either deteriorate a patch, resulting in a decreased carrying capacity ($a < 1/2$), or improve a patch, resulting in an increased carrying capacity ($a > 1/2$). A simple biological interpretation of this difference is that social cheats reduce carrying capacity in the former, and the presence of public goods cooperators increases carrying capacity in the latter (Griffin *et al.*, 2004). For example, (i) migration to empty patches might have a phenotypic effect on cooperators that reduce their ability to cooperate (as in the 'benefits of philopatry' hypothesis; Stacey & Ligon, 1987, 1991), or (ii) empty patches may be intrinsically inferior habitats (as in the 'habitat saturation' hypothesis; Emlen, 1982). In other words, cheats effectively act as an environmental hazard which will reduce the fitness of cooperators who share their space (cf. Frank, 2010) – the relative density of cooperators to cheats will determine carrying capacity of the patch and as such the fitness cost imposed. We assume that cheats do not disperse because further growth is impossible for a cooperator in a pure cheat patch, and cheats are unable to survive in the absence of cooperators. A key assumption in this model is that cheats do not disperse, whereas cheats within the experimental setting exhibit intermediate dispersal. However, the important factor is that dispersers are able to escape cheats, whereas nondispersers cannot, and this was also the case in the experiments. Cooperators can occupy a patch by themselves if they disperse to empty patches, and when $a = 0.5$ and $\gamma = 0$, then our model behaves as if all patches were cooperators only. Given these assumptions, cooperators can only exhibit meaningful growth in initially occupied patches and in vacant patches. Taken together, these two types of patches make up a fraction $\alpha + \beta$ of the total population. Thus, it is convenient to define the proportion of habitable patches that are occupied as $p = \alpha/(\alpha+\beta)$. Full details are given in the Data S1.

Results

Experimental

We measured the relative fitness of the dispersers (PilA) compared with the nondispersers (PilU) for each of the eight treatments (resulting in four average relative fitness values, because each comparable treatment for dispersers and nondispersers would be randomly paired, and the relative fitness value between the treatments calculated) (Fig. 2). The effect of high-occupancy vs. low-occupancy conditions on the relative fitness of the disperser depended on whether the dispersers were competing with cheats or cooperators (Two-way ANOVA, interaction between Treatment*Competitor; $F_{1,8} = 17.215$; $P = 0.003$). Specifically, it is better to disperse when cheats occupy patches at a low rate (One-sample *t*-test, test value = 1: $t_2 = 14.28$; $P = 0.01$), but better to remain sessile when cheats are at high occupancy and distributed throughout the environment (One-sample *t*-test, test value = 1: $t_2 = 374.71$; $P < 0.001$). By contrast, the disperser maintains a fitness advantage across both treatment groups in the presence of cooperators (One-sample *t*-test, test value = 1: low occupancy, $t_2 = 7.79$; $P = 0.032$; high occupancy, $t_2 = 41.42$; $P = 0.002$).

Theoretical

We use our model assumptions to determine the convergence stable (CS; Eshel & Motro, 1981; Eshel, 1983; Christiansen, 1991; Taylor, 1996) dispersal strategy, z^* (see Theoretical model, Materials and methods and Data S1). An important special case of our model is where we assume the absence of vacant patches ($\beta = 0$, and hence $p = 1$) and clonal relatedness among the cooperators in each patch ($r = 1$): this is equivalent to the model of Hamilton & May (1977), and here, we recover their key result, $z^* = 1/(1 + c)$, where the cost of dispersal (c) is simply the probability of landing upon a cheat patch (i.e. $c = \gamma$). In this classic model, increasingly costly dispersal favours a lower rate of dispersal. However, a surprisingly high rate of dispersal is nonetheless favoured despite even extreme costs (e.g. $z^* \to 1/2$ as $c \to 1$).

More generally, analysis of our model reveals that relatedness (r), costs of dispersal ($c = \gamma$), the proportion of habitable patches that are occupied ($p = \alpha/(\alpha+\beta)$) and the relative carrying capacity of occupied patches (a) can interact to mediate the evolution of dispersal. Increasing genetic relatedness of cooperators (with respect to the dispersal genes they carry) within patches always increases the CS rate of dispersal ($\mathrm{d}z^*/\mathrm{d}r > 0$). This is because the indirect fitness benefits of dispersal, owing to the relaxation of resource competition for one's kin, scale with the relatedness of patch mates. When occupation deteriorates patches ($a \leq 1/2$), then increasing the proportion of habitable patches that are occupied (p) always decreases the CS rate of dispersal ($\mathrm{d}z^*/\mathrm{d}p < 0$). When occupation improves patches ($a > 1/2$), the CS rate of dispersal may be either an increasing or a decreasing function of the proportion of habitable patches that are occupied (p). This is because, even if occupation improves patches, dispersers may still be better off if they land on an unoccupied patch, as there are more individuals competing for resources on occupied patches. Hence, the direct fitness of a disperser may increase or decrease with the proportion of occupied patches (higher p), depending upon whether the improvement or competition effect dominates. In contrast, the indirect fitness of a disperser always decreases with increasing abundance of occupied patches (higher p), because it is increasingly likely that the freed up opportunities for reproduction in her natal patch will be won by immigrants rather than her nondispersing kin. Increasing the relative carrying capacity of occupied patches (a) always reduces the CS rate of dispersal ($\mathrm{d}z^*/\mathrm{d}a < 0$). This is because any direct benefit of dispersal owes to the individual finding herself in a patch that is better than the one that she dispersed away from.

The relationship between the cost of dispersal (c) and the CS rate of dispersal (z^*) is qualitatively affected by both the proportion of habitable patches that are occupied (p) and relatedness (r). Increasing mortality cost of dispersal always reduces the direct fitness effect of dispersal and always increases the indirect fitness effect of dispersal. In Hamilton & May's (1977) classic model, the former effect always outweighs the latter, such that increasing mortality cost always reduces the CS rate of dispersal. However, this is not true in our more elaborate model. Consequently, whereas the CS rate of dispersal is

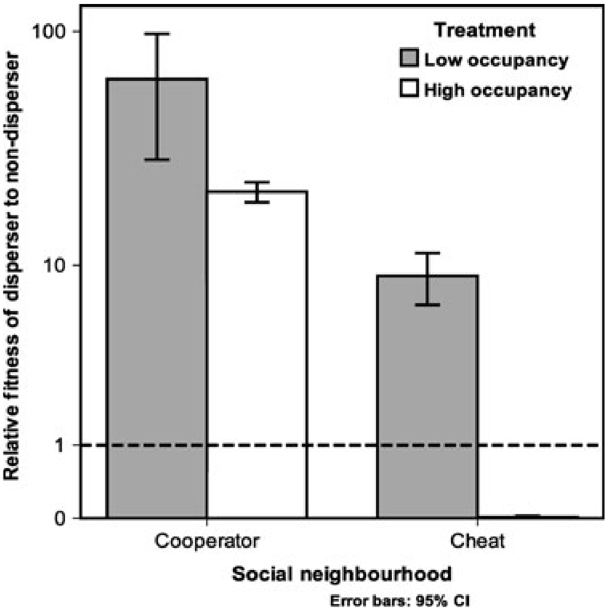

Fig. 2 Average fitness of dispersers (PilA) relative to nondispersers (PilU) in environments which vary in competitor type which form the social neighbourhood (dispersal variants are competing with either cooperators or cheats), and the structure of the social neighbourhood, with competitors distributed throughout the agar (high occupancy) or located only within the colonization patch (low occupancy). Error bars represent ± 95% CI, and the dashed line indicates a relative fitness of 1, that is, when the fitness of the disperser and the nondisperser are equivalent.

sometimes monotonically decreasing with increasing mortality cost for some parameter values, for others, it may be a U-shaped function of the mortality cost (see also Gandon & Michalakis, 1999; Ronce, 2007). These results are summarized in Fig. 3.

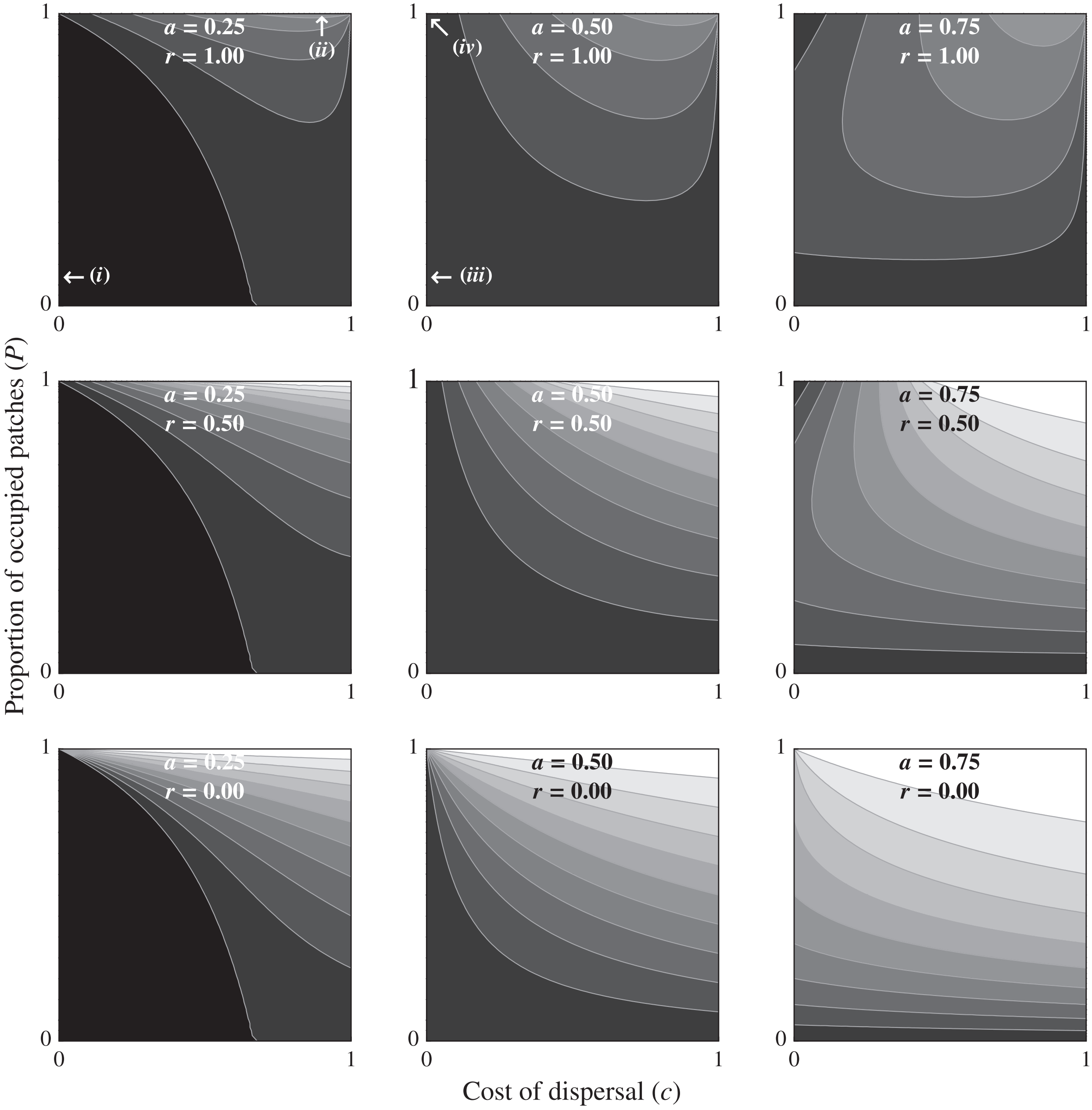

Fig. 3 Contour plots of the convergence stable (CS) rate of dispersal (z^*), as a function of the cost of dispersal (c; abscissae), the proportion of habitable patches that are occupied (p; ordinates), the relative carrying capacity of occupied patches (a; columns) and the coefficient of genetic relatedness (r; rows). The scale varies from $z^* = 0$ (no dispersal, white) to $z^* = 1$ (full dispersal, black). The CS rate of dispersal (z^*) decreases as the carrying capacity of the home patch (a) or the proportion of habitable patches that are occupied (p) increases, and decreases as the genetic relatedness within patches (r) increases. The relation between the CS rate of dispersal (z^*) and the cost of dispersal (c) is more complicated, and is mediated by relatedness (r). For relatively low relatedness ($r = 0.00, 0.50$), the CS rate of dispersal (z^*) decreases monotonically with the cost of dispersal (c). For relatively high relatedness ($r = 1.00$), the CS probability of dispersal (z^*) is a U-shaped function of the cost of dispersal (c). The arrows indicate the points in the parameter space that correspond to the four treatments of Fig. 1, hence: (i) low patch occupancy and cheats as social neighbourhood; (ii) high patch occupancy and cheats as social neighbourhood; (iii) low patch occupancy population and cooperators as social neighbourhood; (iv) high patch occupancy population and cooperators as social neighbourhood.

We now summarize the model results to qualitatively answer our main question: how does the presence of public goods cheats affect the evolution of dispersal under high patch occupancy and low patch occupation? Under low-occupancy conditions (β is high, α is low, and hence, p is low), dispersal will always evolve to be high (Fig. 2), because there are no costs of dispersal, only benefits, regardless of whether cheats are present (low a) or absent (high a) in occupied patches. Under high-occupancy conditions (β is low, and hence, p is high) in the absence of cheats (high a), dispersal also evolves to a high level, despite surrounding patches affording equal growth as the home patch, as a result of indirect fitness benefits: dispersal alleviates local kin competition (Hamilton & May, 1977). By contrast, high-occupancy conditions with a high frequency of cheat patches (high γ and low α, with $c < 1$) result in the evolution of lower dispersal, because the home patch where there are cooperators as well as cheats is a better environment than the surrounding pure cheat patches (Fig. 3).

Discussion

In this study, we investigated how patch occupation and social habitat heterogeneity affect the evolution of dispersal. We compared the relative cell densities of dispersing and nondispersing isogenic strains of the bacterium *P. aeruginosa* when cheats/cooperators were locally and globally present. We found that dispersal was always favoured when there were vacant patches available to colonize (low patch occupancy), but when patches were saturated (high patch occupancy), the presence of public goods cheats greatly altered selection for dispersal. Specifically, when public goods cheats were localized to the home patch, higher dispersal rates were favoured, but when cheats were present throughout available patches, lower dispersal rates were favoured. These results are qualitatively consistent with our theoretical model.

The results can be explained by the dispersing morph adopting a more risky strategy, depleting the numbers in the inoculation site and exposing itself to potential unknown costs within the environment (in this case, the cheats). This strategy pays off when the dispersers find themselves in high nutrient, virgin territory beyond the threat of the cheats (as in the low-occupancy treatment group), but dispersal is costly when there is a strong probability that cheats wait beyond the home site. On the other hand, the nondispersers will not expose themselves to external threats and will therefore dominate the home site – but not beyond – in all treatment groups. This was particularly apparent when cheats were at high patch occupancy: dispersers did not appear to grow beyond the range of the inoculation site, whereas nondispersers were able to slowly migrate beyond this area. Presumably, this is because the dispersers diminished their numbers in the inoculation site as cells dispersed from the colonizing group, whereas the nondispersers maintained relatively high numbers in the inoculation site (because they are not losing cells to dispersal events). We can rule out the possibility that density dependence alone can explain the results given above due to the fact that dispersers reach a relatively higher density (i.e. are more fit) when cheat occupation is low and limited to the home patch. If the correlation between fitness and relatedness (at the dispersal loci) were entirely due to density dependence, dispersers should be even more fit when cooperators are locally confined to the home patch, because under these conditions, there will be a higher density of dispersing cooperators.

Our theoretical model also investigated the interaction between relatedness (with respect to dispersal strategy), the presence of public goods cheats and patch occupancy on the evolution of dispersal (Fig. 3). Relatedness was not manipulated in our experiment, experimental populations were isogenic, consisting of either dispersers or nondispersers, and this therefore corresponds with a theoretical scenario in which $r = 1$. However, by allowing relatedness to vary in our theoretical model, we were able to dissect the direct vs. indirect fitness mediators of the evolution of dispersal and, moreover, facilitate connections with the wider theoretical literature on the social evolution of dispersal. To understand this model effectively, it is important to clarify that although relatedness will often depend strongly on dispersal, it will not always. For example: if patches are founded by a single cell, there will be clonal relatedness within patches irrespective of the rate of dispersal; alternatively, budding dispersal can also allow for scenarios where dispersal is complete and there is clonal relatedness (Gandon & Michalakis, 1999). However, low relatedness generally means that direct benefits will drive the evolution of dispersal, such that patch occupancy determines the fitness of dispersing phenotypes: dispersal is favoured under low patch occupancy, regardless of the presence of cheats (as in the models of Van Valen, 1971; McPeek & Holt, 1992; Greenwood-Lee & Taylor, 2001; Leturque & Rousset, 2002). Under high relatedness, indirect benefits also drive the dispersal patterns, such that dispersal can be favoured under higher values of patch occupancy, because dispersal alleviates kin competition in the patch of origin (as in the models of Hamilton & May, 1977; Frank, 1986 and Gandon & Michalakis, 1999). Our model emphasizes the relative impact of differences in an individual's social environment and patch occupancy upon the evolution of dispersal. Our results are also in line with those of Le Galliard *et al.* (2003, 2005), who have analysed how altruism and mobility interact. They suggested that cooperators can either exhibit high mobility, owing to high local kin competition and relatively low cost of mobility, or exhibit low mobility, owing to high cost of mobility and relatively low local kin competition. This is identical to our conclusions. However, whereas in their model, the cost of mobility is due to an environmental factor, and in our case, the analogous cost of dispersal is due to the presence of cheats in the environment.

Whereas the combination of theory and empirical work we present here helps us to both interpret and generalize the results from our simple experiment, it is important to emphasize the key limitations of our study. First, we did not allow dispersal phenotypes to evolve as a result of mutations generated *de novo* during the course of the experiment, but instead relied on measuring the fitness of defined mutants. Gene knockouts can be associated with large pleiotropic effects that can impact relative fitness between the strains. However, controls in shaken liquid and soft agar showed no significant difference in growth rate between the two strains when motility was unlikely to confer any advantage, and therefore, any fitness differences observed are likely the result of the dispersal phenotypes.

Second, a number of our theoretical model assumptions may limit generality, and alternative assumptions would change model predictions. We assumed that cheats do not disperse, whereas cheats within the experimental setting have intermediate dispersal. If cheats were dispersers, we would expect contrasting results. However, the key assumption in the model is that dispersers are able to escape cheats, whereas nondispersers cannot, and this was also the case in the experiments. This provides a mechanism for cooperators to escape cheat exploitation. Also, we defined relatedness only with respect to the dispersal strategy and not with respect to public good production, because our analysis concerns the evolution of the former rather than the latter trait. More complex models, which consider co-evolution of dispersal and cheating, would need to compute relatedness for both of these traits. However, using a combined empirical and theoretical approach clarifies and expands results from the empirical study alone. This allows the results to be understood in terms of direct and indirect fitness benefits that shape the evolution of dispersal by breaking down the factors of relatedness and patch quality.

Third, the social habitat in our experimental setup was continuous, whereas the model defines a patch-structured habitat. This limitation of the model allows greater analytical tractability and is more true to the experimental set-up. Lattice models differ from island models in that they take into account the geographic distance between subpopulations and individuals and might therefore provide a better approximation to a continuous habitat set-up (Rousset, 2004). However, previous studies of kin competition in genetically structured populations show that patch-structured populations (Taylor, 1992a) and lattice-structured populations (Taylor, 1992b) yield qualitatively similar predictions. Moreover, a comparative analysis of the evolution of dispersal in a homogeneous population under different structures, ranging from patch-structured habitats to several variations of lattice-structured habitats, shows that although these different habitat structures give slightly different quantitative results, the qualitative results are similar (Gandon & Rousset, 1999). A particular major challenge for future studies, both theoretically and empirically, is to consider populations where habitat structure co-evolves with traits to bridge the gap between laboratory and natural settings (e.g. Jessup *et al.*, 2004; Johnson & Stinchcombe, 2007; Lion & van Baalen, 2008; Lehmann & Rousset, 2010).

Fourth, our theoretical results are given in terms of evolutionary endpoints, whereas the experimental results concern evolving populations that have not yet settled to equilibrium. This is a common limitation of experimental evolutionary studies (Buckling *et al.*, 2009; Kümmerli *et al.*, 2009b; Kawecki *et al.*, 2012). Indeed, this limitation is true of any application of comparative statics to biological populations. However, this approach remains one of the most successful in advancing our understanding of the selective forces underlying the adaptive evolution of organisms (Grafen, 1984, 1991; Frank, 1998 Ch 12; West, 2009).

Microbes engage in many collective actions, and this usually requires the maintenance of a kin-structured environment (Czárán & Hoekstra, 2009). For pathogens, maintaining social behaviours – many of which are important virulence factors (Rumbaugh *et al.*, 2009) – will also aid transmission by ensuring the host is inoculated with an infective dose (Hall-Stoodley & Stoodley, 2005). *Agrobacterium tumefaciens* is a plant pathogen that requires highly social groups to elicit a successful pathogenic attack on a host, and it has recently been proposed that for transmission to be effective, dispersal would have to occur via budding (Platt *et al.*, 2012). What is less well explored is the additional mechanism this behaviour might provide to escaping harmful invaders such as predators (Matz & Kjelleberg, 2005), toxin producers (Majeed *et al.*, 2011), parasites (Wilson & Sherman, 2010) or, indeed, cheats (Velicer, 2003).

The theoretical model offers generalization of our results beyond the microbial world. There has been much interest in investigating the role of kin competition and habitat saturation in driving the evolution of dispersal, and hence, there are many empirical examples that demonstrate increased dispersal driven by kin competition [such as in voles (Bollinger *et al.*, 1993) and insects (Kasuya, 2000)] and decreased dispersal driven by local patch occupancy [as seen in kangaroo rats (Jones, 1988), and black kites (Forero *et al.*, 2002)]. However, here, we are assuming not only the number of occupants, but also the nature of the occupants (cooperators or cheats), will influence the evolution of dispersal, and there is evidence that kin are often more favourable neighbours than nonkin. For example, many animals appear to show kin-biased habitat choice [for example in the crow (Baglione *et al.*, 2003) and the lizard (Sinervo & Clobert, 2003)], and in addition, it has been shown that neighbour type (kin vs. nonkin) can also impose selection on important fitness traits. For example, in the Townsend's vole (*Microtus townsendii*), females tend to nest nearer to kin and those nesting in close proximity with kin have higher survival than those nesting near nonkin (Lambin & Krebs, 1993). Highly social organisms, such as the eusocial hymenoptera, can overcome conflicts between the benefits of dispersal and costs resulting from the breakdown of cooperation via budding dispersal – where groups from a larger colony will disperse together to ensure the founding colony will maintain high relatedness (Ross & Keller, 1995; Gardner & West, 2006; Kümmerli *et al.*, 2009b). Here, we suggest that dispersal can also provide a benefit to cooperative groups if it offers the opportunity to run-away from invading cheats; however, this risky strategy only pays off if population structure is such that escape is possible. A greater knowledge of the relative costs and benefits of associating with kin and nonkin helps to explain this result in more detail and adds to the body of work that describes mechanisms for the observed diversity of natural dispersal behaviours.

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Supporting information

Additional Supporting Information may be found in the online version of this article:
Data S1 Full details of theoretical model.

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Supporting Information

Data S1 Full details of theoretical model.

An individual’s Darwinian fitness is her expected number of surviving descendants, relative to the average of her class. Following the assumptions outlined in the main text, the Darwinian fitness of a cooperative bacterium with dispersal strategy (i.e. rate of dispersal) x, in a patch where the average dispersal strategy among cooperators is y, and in a population where the average dispersal strategy among cooperators is z, is given by:

$$w=(1-x)\frac{aK}{1-y+p(1-c)z}+x(1-c)\left(p\frac{aK}{1-z+p(1-c)z}+(1-p)\frac{(1-a)K}{p(1-c)z}\right),$$

(A1)

where $p = \alpha/(\alpha+\beta)$ is the proportion of habitable patches that are occupied rather than vacant, and $c = \gamma$ is the cost of dispersal (i.e. the probability of landing on a cheat patch, and achieving no reproductive success as a result).

We use a neighbour-modulated fitness approach to analyse how kin selection acts upon dispersal (Taylor, 1996; Taylor & Frank, 1996; Frank, 1997; Frank, 1998; Rousset, 2004; Taylor et al., 2007). The condition for natural selection to favour an increase in dispersal is $\mathrm{d}w/\mathrm{d}x = \partial w/\partial x + r\, \partial w/\partial y > 0$, where $r = \mathrm{d}y/\mathrm{d}x$ is the kin-selection coefficient of relatedness and all derivatives are evaluated at $x = y = z$. This condition is Hamilton’s rule, $-C + rB > 0$ (Hamilton, 1963; Hamilton, 1964; Hamilton, 1970; Taylor & Frank, 1996; Frank, 1998). The equilibrium rate of dispersal is found by solving $\mathrm{d}w/\mathrm{d}x|_{x=y=z=z^*} = 0$ (upon the assumption that $0 < z^* < 1$), and this is given by:

$$z^* = \frac{((1-a)(2-p)+(2a-1)p)t - rap}{2((2a-1)pt^2+(1-a)t^2-rap)} - \frac{\sqrt{(2(1-a)(1-p)t+ap(t-r))^2-4(1-a)(1-p)((2a-1)pt^2+(1-a)t^2-rap)}}{2((2a-1)pt^2+(1-a)t^2-rap)},$$

(A2)

where $t = 1-(1-c)p$. This can then be checked for convergence stability (CS; Eshel & Motro, 1981; Christiansen, 1991; Taylor, 1996). Defining $J(z) \equiv \mathrm{d}w/\mathrm{d}x|_{x=y=z}$, we have:

$$\left.\frac{\mathrm{d}J}{\mathrm{d}z}\right|_{z=z^*} = -\left(\frac{(1-c)pra}{(1-z+pz(1-c))^2} + \frac{(1-p)(1-a)}{pz^2}\right). \qquad \text{(A3)}$$

The RHS of equation (A3) is negative for all parameter values, and therefore the equilibrium level of dispersal z^* is always convergence stable (Eshel & Motro, 1981; Christiansen, 1991; Taylor, 1996). Note that the classic model of Hamilton and May (1977) is recovered as a special case of our model. This is done by assuming that all habitable patches are occupied ($p = 1$) and that there is clonal relatedness among co-operators within patches ($r = 1$), prior to dispersal. In this scenario, we recover the usual result: $z^*=1/(1+c)$. Hamilton and May (1977) emphasized that the evolution of dispersal involves a tension between the direct fitness cost ($C > 0$) for the individual disperser and the indirect fitness benefit ($rB > 0$) that she receives as a consequence of the improved reproductive success of her genetical relatives.

In Hamilton and May's (1977) model, an individual who survives dispersal and lands upon a habitable patch is (on average) no better off than if she had stayed in her natal patch. Hence, dispersal is always associated with a direct fitness cost owing to the mortality cost of dispersal ($C = c > 0$). But, by dispersing, the individual frees up breeding opportunities for her non-dispersing relatives, leading to an indirect fitness benefit ($rB = (1-z)/(1-cz) > 0$). Note that this benefit increases with the mortality cost of dispersal ($\partial(rB)/\partial c>0$), as a lower rate of disperser survival increases the extent to which the disperser's patch-mates – rather than immigrants arriving at her natal patch – benefit from her sacrifice. However, in Hamilton and May's (1977) model, the impact of increased mortality cost on direct fitness is always greater than its impact on indirect fitness, so the net effect of increasing the mortality cost is to reduce the convergence stable level of dispersal ($\mathrm{d}z^*/\mathrm{d}c < 0$).

In contrast, in our generalization of Hamilton and May's (1977) model, there is a more complicated relationship between the mortality cost (c) and the convergence stable level of dispersal (z^*). Owing to the presence of unoccupied habitable patches ($p < 1$), individuals who survive dispersal and land on a habitable patch find themselves – on average – better off than if they had stayed in their natal patch. This reduces the direct fitness cost of dispersal, and may even provide a direct fitness benefit to the disperser. As a consequence, the negative impact of the mortality cost on the direct fitness effect of dispersal ($\partial C/\partial c$) may be less than the positive impact of the mortality cost on the indirect fitness effect of dispersal ($\partial(rB)/\partial c$). This means that, for some parameter values, the convergence stable level of dispersal increases with the mortality cost of dispersal ($\mathrm{d}z^*/\mathrm{d}c > 0$; see Figure 3).

The evolution of dispersal is also mediated by the proportion of habitable patches that are occupied (p). Increasing this proportion always decreases the indirect fitness benefit of dispersal (i.e. $\partial(rB)/\partial p = -ar(1-c)z(1-z)/(1-(1-(1-c)p)z)^2 < 0$). However, increasing this proportion may either increase or decrease the direct fitness cost of dispersal (i.e. $\partial C/\partial p = (1\text{-}2a)(1\text{-}c) + (1\text{-}a)(1\text{-}p)(1\text{-}z)/(p^2 z) + (1\text{-}a)(1\text{-}z)/(p\ z)$). If $a \leq ½$, then this impact on the direct fitness cost is guaranteed to be positive, and hence for all $a \leq ½$ the CS rate of dispersal is a monotonically decreasing function of the proportion of habitable patches that are habitable (Figure 2). If $a > ½$, then increasing this proportion may increase or decrease the CS rate of dispersal (Figure 2). In addition, the evolution of dispersal is also directly affected by the relative carrying capacity of occupied patches ($0 < a < 1$). If $a < ½$, then occupied patches support a lower carrying capacity than do vacant patches, and if $a > ½$ then the opposite is true. As vacant patches can only be reached by dispersers, a lower value of a makes vacant patches – and hence dispersal – more attractive, whereas a higher value of a makes vacant patches – and hence dispersal – less attractive. Hence, as the relative carrying capacity of occupied patches increases, the convergence stable rate of dispersal decreases ($\mathrm{d}z^*/\mathrm{d}a < 0$; Figure 3).

Supporting Information References:

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A5. The evolution of bacterial mutation rates under simultaneous selection by inter-specific and social parasitism.

A5.1. Abstract

Many bacterial populations harbour substantial numbers of hypermutable bacteria, in spite of hypermutation being associated with deleterious mutations. One reason for the persistence of hypermutators is the provision of novel mutations, enabling rapid adaptation to continually changing environments, such as coevolving virulent parasites. However, hypermutation also increases the rate at which intra-specific parasites (social cheats) are generated. Inter-specific and intra-specific parasitism are therefore likely to impose conflicting selection pressure on mutation rate. Here, we combine theory and experiments to investigate how simultaneous selection from inter- and intra-specific parasitism affects the evolution of bacterial mutation rates in the plant-colonizing bacterium *Pseudomonas fluorescens*. Both our theoretical and experimental results suggest that phage presence increases, and selection for public goods cooperation (the production of iron-scavenging siderophores) decreases selection for mutator bacteria. Moreover, phages imposed a much greater growth cost than social cheating, and when both selection pressures were imposed simultaneously, selection for cooperation did not affect mutation rate evolution. Given the ubiquity of infectious phages in the natural environment and clinical infections, our results suggest that phages are likely to be more important than social interactions in determining mutation rate evolution.

A5.2. Introduction

One hypothesis for the evolution of sexual reproduction is an increased ability to evade parasites: the "Parasite Red Queen" hypothesis [1-8]. Parasites become adapted to the most frequent host genotypes, and host sex can result in novel genotypes that escape infection by the most common parasites. However, parasites can quickly adapt, and so there is continual selection for genotypic diversity in the presence of parasites. However, another form of parasitism, social parasitism, may itself be a cost of sexual reproduction. Specifically, the genetic diversity produced by sex means that interacting individuals are less likely to share the same alleles for cooperation. This reduction in relatedness reduces the indirect benefits required to select for cooperative behaviour. This idea that "sex is an antisocial force in evolution" was first proposed by Wilson [9], and has been explored both theoretically and comparatively [10-14], demonstrating that highly cooperative societies such as those of the eusocial insects or cooperative breeders are likely to be preceded by strict lifetime monogamy (rather than promiscuity), whereby relatedness between interacting individuals is predictably high. Sexual reproduction can therefore create a tradeoff between resistance to inter- and intra-specific parasitism [15, 16].

The evolution of mutation rates in obligatory asexual species may be affected by similar conflicting selection pressures from inter- and intra-specific parasitism. Many bacterial populations harbour hypermutable bacterial strains (10-1000 fold increase in genomic mutation rate [17-20]; with the frequency of hypermutators sometimes in excess of 30% [21] in clinical infections. Hypermutators increase in frequency through hitch-hiking with beneficial mutations, hence continual changing selection pressures tend to result in increases in mutator frequencies [18, 22, 23]. Antagonistic coevolution with viruses creates continual selection for novel resistant phenotypes, and has been shown to confer a large selective advantage on hypermutator bacteria *in vitro* [24, 25]. However, hypermutators have been shown to be selected against when cooperative behaviours are advantageous (specifically, the production of siderophores: extracellular iron-scavenging public goods). This is because hypermutators generate non-producing cheats more readily, reducing relatedness in populations founded by mutators [26-28]. Beyond bacteria, selection for cooperation can keep relatedness high through processes such as kin recognition [29, 30] reproductive skew [31] and within-group segregation [32].

Here, we combine theory and experiments to investigate how simultaneous selection from inter- and intra-specific parasitism affects the evolution of bacterial mutation rates. We use the well-studied plant-colonizing bacterium *Pseudomonas fluorescens* SBW25, which undergoes extensive reciprocal evolution of defense and counter-defense with an obligatory killing bacteriophage, ϕ2, in both nutrient media [33] and soil [34]. For the cooperative trait, we focus on the extracellular production of iron-scavenging siderophores, which are individually costly to produce but can be used by neighbouring conspecifics. As a result, non-producing cheats can readily invade but reduce iron-dependent population growth rate in iron-limited environments in both *P. fluorescens* SBW25 [35] and the closely related bacterium, *P. aeruginosa* [36]. We experimentally manipulated these opposing selection pressures by competing *P. fluorescens* SBW25 and an isogenic mutator in iron-limited or iron-rich media (only the former conditions require siderophore production), and in the presence or absence of phages, in experimental metapopulations. Our experimental design simulated global competition, whereby all patches within a metapopulation were mixed and clones from this mixture were used to inoculate new patches. In doing so, genotypes from the more successful patches were overrepresented in subsequent generations, while those from less successful patches were underrepresented. Consequently, any phenotype that reduced group productivity was selected against. To ensure selection for cooperation, we regularly established new patches in the metapopulations with single clones (high relatedness) [27, 36]. This design allowed the benefits of diversity to be realised by hypermutators, but also kept selection for cooperation high.

A5.3. Theoretical model and analysis

We develop a simple mathematical model to make qualitative predictions about the evolution of bacterial mutation rates μ in the presence of social cheats and parasites. We assume that higher mutation rates both increases bacterial resistance to parasites and the local abundance of social cheats. We consider a metapopulation composed of a large number of patches, each of which is colonised by a single bacterial strain with mutation rate μ, with $0 \leq \mu \leq 1$. All strains are assumed to be functional for the cooperative trait, and cheats arise by loss-of-function mutations. While cheats impose a growth cost on cooperative lineages, we assume that cheats are poor colonisers of new patches, and therefore evolutionarily unviable. We assume that the fitness w of a bacterial strain with mutation rate μ is a function of its survival S and its growth G, such that $w(\mu) = SG$.

Bacterial survival decreases as parasite density π increases, while it increases as bacterial hosts evolve resistance to parasites. For simplicity of analysis we assume that the probability that bacterial hosts acquire resistance alleles is equal to their mutation rate

μ , and that parasite-induced mortality decreases linearly with resistance. As a result, the survival cost imposed by parasites on their bacterial hosts is (1- μ)π, such that the survival of a strain with mutation rate μ is S = 1-(1- μ)π. Note that in absence of parasites (π = 0), or when there is full resistance (μ = 1), survival is assumed to be 1.

We assume that the costs associated with social cheats depend on three factors: i) the local abundance of cheats; ii) the intrinsic cost of cheats to cooperators; and iii) parasite negative density-dependent regulation of hosts' cooperative growth. We assume that the local abundance of cheats is equal to the mutation rate μ of the local bacterial strain. Cheats are assumed to impose an intrinsic cost c on cooperative growth. In addition, cooperative growth may be subject to density-dependent regulation by parasites, which reduces the impact of cheats on cooperative growth. We assume that parasite density-dependent regulation of hosts' cooperative growth is given by $\alpha\ \pi$, where $0 \leq \alpha \leq 1$ is the degree of parasite density-dependent regulation. Putting all these factors together, we define the overall growth cost of cheats to cooperative growth as (1- $\alpha\pi$)$c\,\mu$, and the growth of a focal lineage as G = 1-(1- $\alpha\pi$)$c\,\mu$. Note that if: i) cheats are absent (μ = 0); or ii) cheats do not impose an intrinsic cost on cooperators (c = 0); or iii) there is full density dependent regulation ($\alpha = \pi = 1$), growth is one, G = 1. The fitness of a focal strain with mutation rate μ is then given by

$$w(\mu) = \overbrace{(1 - \underbrace{(1-\mu)\pi}_{\text{Cost of parasites}})}^{\text{Survival}}\overbrace{(1 - \underbrace{(1-\alpha\pi)c\mu}_{\text{Cost of social cheats}})}^{\text{Growth}}. \qquad (1)$$

Our objective is to determine the evolutionary stable (ES) mutation rate (μ*; [37,38]). We find that the marginal fitness benefit of a slight increase in the mutation rate is given by $B(\mu) = \pi(1-(1-\alpha\pi)c\mu) = \pi G$. Thus, the marginal benefit increases with increasing parasite density π, as long as there is some growth. The marginal fitness cost of a slight increase in the mutation rate is given by $C(\mu) = (1-(1-\mu)\pi)(1-\alpha\pi)c\mu = S(1-\alpha\pi)c$. Thus, as long as there is some survival, the growth cost is low if: i) there is strong parasite density-dependent regulation (high $\alpha\pi$); and ii) the intrinsic cost of cheats is low (low c). The ES mutation rate is the value μ* for which the marginal cost exactly cancels the marginal benefit of a slight increase in the mutation rate, i.e. $B(\mu^*) = C(\mu^*)$. This is given by

$$\mu^* = \frac{1}{2}\left(\frac{1}{c(1-\alpha\pi)} - \frac{1-\pi}{\pi}\right). \qquad (2)$$

We find that the ES mutation rate μ* decreases with increasing intrinsic cost of cheats ($\partial\mu^*/\partial c < 0$). By contrast, the ES mutation rate μ* increases with increasing degree of parasite negative density-dependent regulation of hosts ($\partial\mu^*/\partial\alpha > 0$), and with increasing parasite density ($\partial\mu^*/\partial\pi > 0$; see Figure 1).

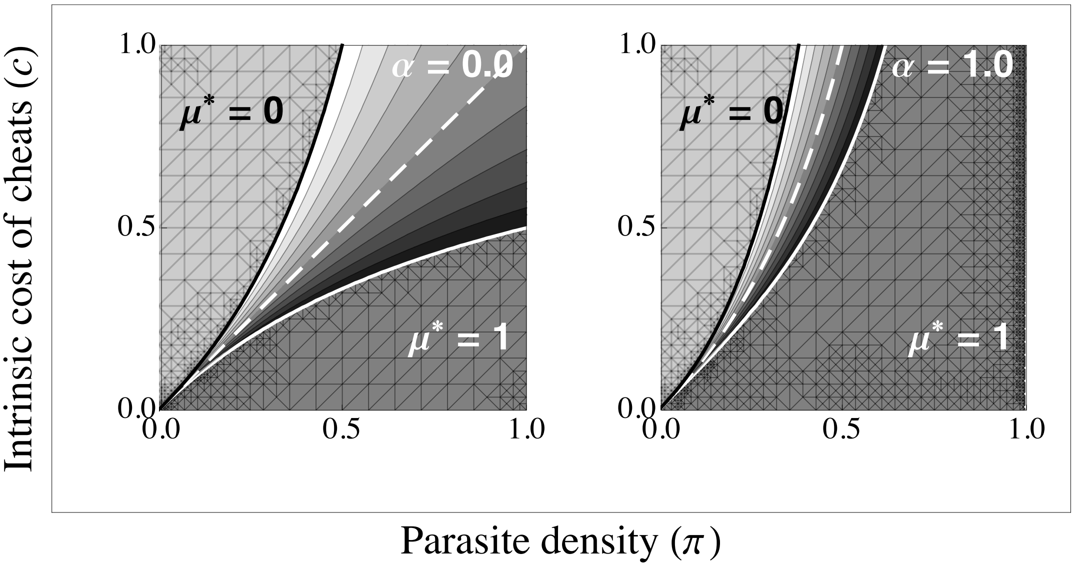

Figure 1: The ES mutation rate (μ^*) as a function of parasite density (π) and the intrinsic cost of cheats (c), for various levels of parasite density-dependent regulation of hosts (α). If there is no parasite density-dependent regulation ($\alpha = 0$), then when parasite density is low and the cost of cheats is high, selection favours low mutation rates ($c \geq \pi/(1+\pi)$; $\mu^* = 0$). If parasite density is equal to the intrinsic cost of cheats, then intermediate levels of mutation rates are favoured ($\pi = c$; $\mu^* = 0.5$; dashed white line). If parasite density is high relative to the cost of cheats, then selection favours high mutation rates ($c \leq \pi/(1-\pi)$; $\mu^* = 1$). If there is strong parasite density-dependent regulation ($\alpha = 1$), the ES mutation rate becomes less sensitive to changes in the cost of cheats (c).

A5.4. Experimental materials and methods

i) Strains

The *P. fluorescens* strain SBW25 [39] was used as a wildtype, and the strain SBW25Δ*mutL* which has a mutation in the mismatch repair gene *mutL,* was used as a hypermutator. SBW25Δ*mutL* exhibits a spontaneous mutation rate 100-fold times higher than SBW25, and is resistant to the antibiotic gentamicin [24], allowing it to be distinguished from the wildtype. We used the lytic phage phi 2 (φ2) [33].

ii) Experimental design

To ensure that each population was colonised by a single strain (i.e relatedness = 1) at the first transfer into medium, each bacterial culture was grown in 6ml King's Medium B (KB; (10 g glycerol, 20 g proteose peptone no. 3, 1.5 g $K_2HPO_4 \bullet 3H_2O$, 1.5 g $MgSO_4 \bullet 7H_2O$, per litre), for 24 hours at 27°C, when it was diluted with M9 buffer and grown for 36 hours on KB agar at 27°C. A single colony could then be selected and further grown in KB medium at 27°C for 24 hours. This single-strain culture could then be used to establish populations.

We manipulated phage and iron availability in a fully factorial experiment, setting up each of our 4 treatments (a: iron + phage, b: iron only, c: phage only, d: no iron, no phage) in 96 well plates. We used 6 replicates for each treatment, with each replicate consisting of a metapopulation made up of 6 patches (wells). Each well was inoculated with a total of 190μl KB broth, with 3 wells of each replicate initially inoculated with approximately 10^7 colony forming units (CFUs) of clonal wildtype bacteria, and 3 wells inoculated with approximately 10^7 CFUs of clonal mutator bacteria. Iron limitation was created by the addition of 100μg/ml human apotransferrin and 20mM sodium hydrogen carbonate ($NaHCO_3$) to standard KB medium immediately before use [see 35]; $NaHCO_3$ was also added to non-iron limited treatments. Approximately 10^5 phages were added to phage treatments in 10µl KB, and the same volume of KB media added to non-phage wells. Plates were incubated at 27°C under static conditions.

Every second day, aliquots (2µl) from all 4 treatments were transferred to new KB medium (+/- Fe) using a sterile pin replicator. Every 4 transfers (8 days), 100µl from each of the 6 wells within a replicate was mixed, simulating global competition [36] and plated out on KB and KB + Gentamicin agar to assess frequencies of mutator and wildtype bacteria (see below). Phage was isolated from this mixture each time, by adding 10% chloroform and centrifuging at 13000rpm, which lysed and pelleted bacteria. Six colonies from each KB agar plate were randomly selected, grown independently for 24 hours in 180 µl KB medium at 27°C, and each was subsequently used to inoculate a new well within a treatment (10^7 CFUs), to ensure high relatedness, and hence the potential for selection for cooperation, was continually re-established. For phage treatments, both sympatric (10µl) phages and ancestral (10µl) phages were added (to ensure continual selection for phage resistance where sympatric phages had reached low densities); equivalent volumes of phage-free KB media were added to no-phage treatments. Mixing and plating were performed 5 times in total, for approximately 250 generations of bacterial growth (carrying out 6 transfers instead of 4 between mixing after the 3rd assay).

iii) Assays

The relative fitness of mutator and wildtype strains was assessed by plating a mixture of all patches within a replicate onto both KB agar (for an estimate of total density) and KB agar supplemented with 10µg/ml gentamicin (on which only gentamicin resistant mutator bacteria can grow) over 36 hours, resulting in a KB and KB+gentamicin plate for each replicate, and 6 plates of each type per treatment. Colony types were also categorised by colour into either siderophore producers (yellow/green) or non-producers (white), based on the fact that the primary siderophore of *P. fluorescens*, pyoverdine, is yellow/green. As the amount of colony pigmentation varied considerably, we classified a colony showing any yellow/green pigmentation after 36 hours as a producer [see 26]. We carried out an additional experiment to verify that the appearance of milky white colonies indeed represented the evolution of siderophore negative cheats. We selected 19 wildtype and 19 milky colonies at random at the final timepoint, and inoculated each colony into iron-limited KB media. Populations were grown at 27°C for 24 hours, after which, fluorescence of 180ul of culture was measured at 460nm, following excitation at 400nm using a Biotek Synergy 2 spectrophotometer (standard protocol for measuring pyoverdine production [40, 41]). Milky colonies produced pyoverdine at significantly lower levels than wildtype (student's t test, $T_{18.017} = 15.7631$, $p < 0.0001$) and so any milky colonies that emerged during the experiment were labelled siderophore cheats.

iv) Costs of cheating and phage parasitism

To directly assess the relative cost of cheats and parasites on the growth of wildtype bacteria, wildtype SBW25 and an isogenic cheat strain (SBW25Δ*pvdL*, with the primary siderophore, pyoverdine, knocked out, [42]) were grown in isolation in the presence and absence of phages (phi 2 (φ2)), with 6 replicates per treatment. All treatments were carried out in iron-limited KB broth to facilitate selection for cooperation under starting conditions described above. After 24 hours at 27°C, the abundance of cheat and wildtype genotypes was assessed by plating each treatment on KB agar, incubating for 36 hours, and counting the colonies on each plate as before. We also confirmed previous work [35] that cheats have a growth rate advantage in the presence of wildtype bacteria under these conditions by competing 50:50 mixtures of cheats and wildtype.

v) Statistical analysis

R software [43] was employed for all statistical analyses. Maximum likelihood analysis was carried out using Linear Mixed Effects Revised models with arcsine square root (proportion of mutators/cheats) as a response variable, and iron, phage and time, including 2-way interactions, as explanatory variables. A third model with cheat frequency as the response variable was employed to investigate any affect mutator frequency might have on the appearance of cheats, and the extent to which this was affected by iron limitation. Iron and phage presence/absence were fitted as factors and time as a continuous variable, assigning 'population' as a random factor as follows:

*mutator frequency ~ iron*phage +iron*time + phage*time+* 1/*population*
*cheat frequency ~ iron*phage +iron*time + phage*time+* 1/*population*
*cheat frequency~ mutator frequency*iron +* 1/*population*,

where * represents an interaction and separate terms. To assess the relative costs of cheating and parasitism, student's t-tests were used to compare fitness of wildtype and cheat strains after 24 hours growth in the presence and absence of phage.

A5.5. Experimental results

i) Costs of cheats and phages

To establish the relative magnitude of costs of cheating and phage parasitism on bacterial growth, we measured the densities of siderophore cheat and wildtype strains grown as monocultures in the presence and absence of ancestral bacteriophages after 24h growth under iron-limited conditions. Phages caused a significant reduction in the density of wildtype (student's t test, $T_{7.515} = 5.68$, $p < 0.001$), and cheat phenotypes (student's t test, $T_{11.542} = 2.5$, $p < 0.05$). Consistent with previous work [35, 42] cheats reached significantly lower densities than wildtype bacteria in the absence of phages (student's t test, $T_{9.741} = 4.6822$, $p < 0.001$), however, in the presence of phages, this relationship was reversed and cheats grew significantly more than wildtype (student's t test, $T_{11.11} = 3.3051$, $p < 0.01$). The reduction in the density of wildtype bacteria brought about by cheats was significantly smaller than the reduction caused by the presence of phages (student's t test, $T_{6.725} = 4.0$, $p < 0.01$). Based on our simple model predictions, these ecological results suggest that phage-imposed selection for mutators is likely to be greater than that imposed by cheats. Competition experiments between wildtype SBW25 and SBW25Δ*pvdL* verified that cheats can invade cooperators under these experimental conditions (student's t test (alt=1), $T_5 = 2.5039$, $p = 0.05$), despite the poorer performance of cheats compared with cooperators as monocultures; i.e. pyoverdin production is an altruistic trait.

ii) Competition between mutators and wildtype over evolutionary time scales.

We predicted that phages should select for higher mutation rates, while selection for cooperation should select against mutators. Phage-imposed selection was manipulated by the presence versus absence of phages, whereas selection for siderophore-mediated cooperation was manipulated by changing iron availability (siderophores are required when iron is limited) in experimental metapopulations. While the frequency of mutators increased through time in the presence of phages and decreased in the absence of phages (LMER: timepoint x phage interaction, $\text{Chisq}_{1,5} = 9.334$, $p < 0.01$; Figure 2*)* iron availability had no impact on the frequency of mutators (LMER: $\text{Chisq}_{1,6} = 0.31$, $p =$

0.58). Moreover, there was no interaction between the presence of phage and iron availability (LMER: non significant phage x iron interaction, $\mathrm{Chisq}_{1,8} = 0.187, p = 0.67$). Selection for mutators via the presence of external viruses therefore was not affected by any conflicting selection for cooperation by iron limitation.

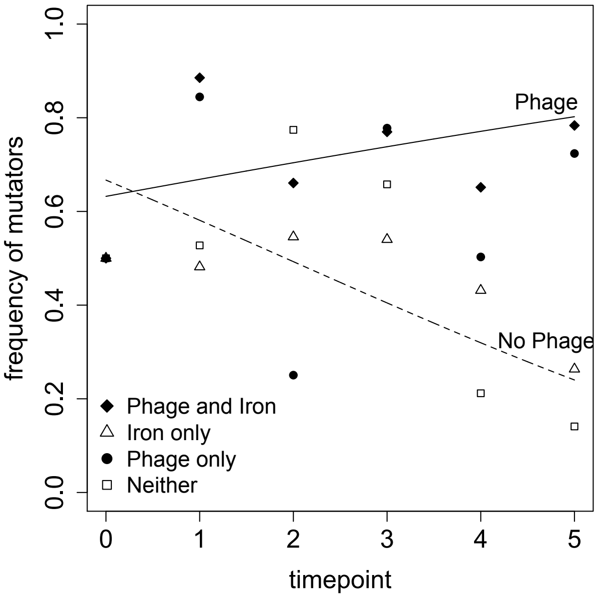

Figure 2: Changes in mutator frequency in a population through time is affected by the presence/absence of phage ($\mathrm{Chisq}_{1,5} = 9.3343, p < 0.01$). Data are plotted using predictions from minimal LMER model (*mutator frequency ~ time:phage + 1|population).*

iii) Evolution of siderophore cheats

The frequency of siderophore cheats increased through time in all treatments (LMER: $\mathrm{Chisq}_{1,4} = 53.939, p < 0.001$*)* but significantly more so when there was selection for cooperation by limiting iron availability (LMER: timepoint x iron interaction $\mathrm{Chisq}_{1,5} = 3.9498, p < 0.05$; Figure 3). Phage presence did promote the production of cheats through time, but this trend was not significant (LMER: non-significant phage x time interaction, $\text{Chisq}_{1,7} = 2.158, p = 0.14$). Furthermore, no interaction was observed between iron availability and the presence of phages in determining cheat frequency (LMER: non-significant phage x iron interaction $\text{Chisq}_{1,8} = 0.623, p = 0.43$). Cheat frequency increased with the frequency of mutators, but only in iron-rich populations (LMER: mutator frequency x iron interaction: $\text{chisq}_{1,5} = 8.148, p < 0.01$).

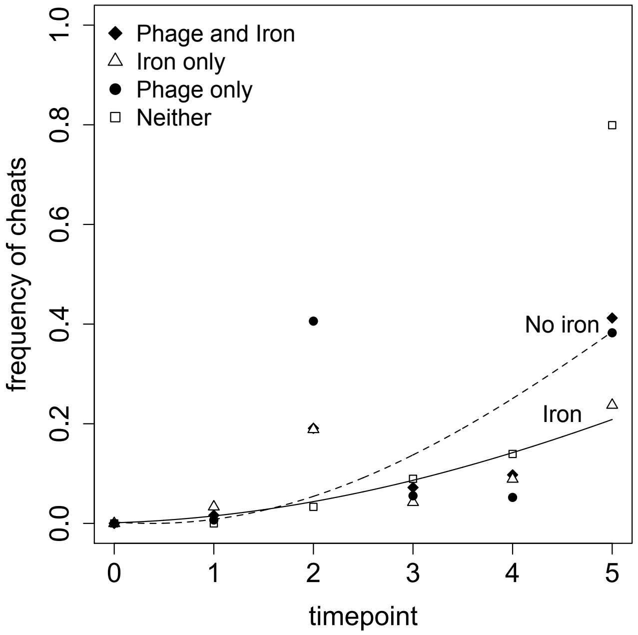

Figure 3: When Iron is limited, cheats reach higher frequencies through time than when iron is present ($\text{Chisq}_{1,5} = 3.9498, p < 0.05$). Data are plotted using predictions from minimal LMER model (*cheat frequency ~ time:iron* + 1*|population*.

A5.6. Discussion

Here, we investigate both theoretically and empirically how selection imposed by inter-specific parasites (bacteriophages) and intra-specific parasites (social cheats) affects the evolution of mutation rates in bacteria. Theoretically, selection for mutator bacteria decreases when there is selection for cooperation because mutators are more likely to generate social cheats, and increases in the presence of phage because mutators are more likely to generate phage resistance mutations. Our short-term growth rate experiments suggest that phages impose a much greater growth rate cost than social cheating in this experimental context. Moreover, the impact of phages on density was less for cheat than wildtype populations. Feeding this back into our simple model suggests that the strength of phage-imposed positive selection on mutators will be greater than cooperation-imposed negative selection on mutators. Our evolution experiment was consistent with this prediction: mutator frequency increased in the presence of phages but was not influenced by whether or not there was selection for cooperation.

That coevolution with phages favours mutator bacteria is consistent with previous work using this system [22, 24, 25]. Pal *et al.* [24] found that after approximately 170 bacterial generations mutation rates had increased 10- to 100- fold in 9 out of 36 populations in the presence of coevolving phages, whereas no significant change in mutation rate was observed in populations evolving in the absence of phages. Qualitatively identical results were observed when the wildtype and an isogenic mutator were competed at equal ratios in the presence and absence of phages. However, in these experiments bacteria were cultured as single populations. Our results reveal similar phage-imposed selection for mutation rates in high-relatedness metapopulations. Arguably, such metapopulation structure presents a more ecologically relevant scenario for bacteria, where microcolonies are founded by single bacteria, which then go extinct or go on to colonise new patches [44].

The lack of association between selection for siderophore-mediated cooperation and mutator frequency is initially surprising, as a previous study showed such a relationship using the related bacterium, *Pseudomonas aeruginosa* [27]. Specifically, in an evolution experiment of a similar duration to the current study (approximately 250 generations), mutator frequency was significantly lower in iron-limited metapopulations when patches were established with single colonies (high relatedness, hence strong selection for cooperation) compared with multiple colonies. In both studies, cheats of both species show reduced growth under iron-limited conditions [this study, 36, 42, 45]. The discrepancy between these results is likely to have arisen because i) different species and media were used, and hence the cost of cheating may be different, ii) selection for cooperation in the current study was manipulated by iron-availability as opposed to number of colonies founding a patch and iii) we used the mutL mutator strain rather than mutS as in previous studies [24, 27], and differences in genetic effects of the specific gene may account for discrepancies. However, studies in *Escherichia coli* suggest these mutations have very similar effects [46].

Our model and ecological experiment suggests a simple but potentially general interaction between social cheating and parasitism: parasites have less of an impact on reducing cheat population density, and hence the relative impact of cheating on population growth is reduced in the presence compared with the absence of parasites. This is a simple extension of the more general finding that enemies typically reduce high density populations relatively more than low density populations e.g. [47, 48]. In addition to weakening selection against mutators, this reduced costs of cheats also suggests that cheats may be relatively favoured in the presence versus absence of parasites. By contrast, recent theory and experiments suggest cheating may be less favoured in the presence of parasites, because cheats are likely to be initially rare in newly colonized patches, and hence less likely to evolve resistance mutations [35, 49]. Both mechanisms are not mutually exclusive, with the former relevant when cheats are at sufficiently high frequency to affect growth and the latter when cheats are initially rare. The simultaneous operation of both mechanisms may explain our finding that phages had no net effect on the presence of cheats.

High mutation rates can result in rapid resistance to antibiotics and bacteriophage, and clinical studies have shown hypermutators to be associated with poorer lung function in CF patients [50]. Therefore it is crucial to develop an understanding of how different selection pressures involved in their persistence interact with each other. Given the ubiquity of infectious phages in the natural environment e.g [51] and clinical infections [52, 53], our results suggest that phages are likely to be more important than social interactions in determining mutation rate evolution in natural environments.

A5.7. Acknowledgements

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